Zhengguo Wang
Jianxin Jiang
Editors

Explosive Blast Injuries

Principles and Practices

PEOPLE'S MEDICAL PUBLISHING HOUSE

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Preface

Blast injuries are characterized by high incidence rate, occurrence among the masses, and difficulty in prevention whether in wartime or in peacetime. Such explosions usually result in multiple critical injuries, high infection rates, treatment difficulty, and high mortality. Along with changes in combat styles and extensive application of various explosive weapons, blast injuries have become the main injury category in modern warfare, accounting for more than 70% of total injuries. In peacetime, frequent terrorist bombings and various explosions have caused a large number of casualties. Since the "September 11 attacks," terrorist bombings around the world have become increasingly rampant, and serious and vicious incidents with hundreds of casualties have become more frequent. Such terrorist bombings constitute a major issue facing the international community today and a top priority for world security. Therefore, it is of far-reaching military and social significance to strengthen the research on blast injury and improve protection and treatment of such injuries.

As the name suggests, blast injury is mechanical damage caused by various kinds of explosions. Its presence is closely related to gunpowder, one of the four great inventions of China. The black powder was invented as early as the beginning of the Western Han Dynasty. According to historical records, during the process of refining medicinal pills, ancient Chinese alchemists discovered that the mixture of nitrate, sulfur, and charcoal could burn and explode, thus giving birth to gunpowder. Close to the end of the Tang Dynasty at the beginning of the tenth century, gunpowder came to be used in warfare. Early gunpowder weapons did not explode with much power and were mainly used in ignition. In the thirteenth century, gunpowder was introduced to Arab countries by merchants through India, and then made its way to Europe. In the 1860s, Alfred Bernhard Nobel invented what is now trinitrotoluene, also known as nitroglycerin. The widespread use of gunpowder and gunpowder weaponry is a turning point in the world history of weapons, after which the military operation has undergone tremendous transformation, and armies transitioned from cold weaponry into the era of hot weaponry. In the future of IT-based warfare, not only will explosive weapons become the main combat force, but various types of new explosive weapons with greater lethality will also continue to be developed and play greater roles in winning conflicts against enemies.

Although warfare has already ushered in the age of hot weapons as early as the nineteenth century, research on blast injury really was only started after World War II. In August 1945, the United States dropped two atomic bombs on the Japanese cities of Hiroshima and Nagasaki. The whole world was shocked by their destructive power as hundreds of thousands of people were killed and injured, while huge parts of the cities fell into ruins. After World War II, the U.S. military took the lead in launching international research on blast injuries due to concerns about the emergence of nuclear warfare. The research on blast injury in China began in the late 1970s. Academician Zhengguo Wang, as the founder of blast injury research in China, mainly conducted early blast injury researches during nuclear tests. Through a large number of animal field experiments, he has successively summarized and compiled the monographs such as *Nuclear Weapon Injuries to Personnel and Its Protection, Nuclear Blast Injuries,* and other related works, which laid the foundation for systematic research on blast injuries at the later stages. In 1984, the former Institute of Field Surgery of the Third Military Medical University

established the first and also so thus far the world's largest and the most advanced bio-shock tube laboratory in China, opening a new era in which blast injury research may be carried out in labs. In spite of the late start of blast injury research in China compared to the United States and the former Soviet Union, Chinese blast injury research has been advancing rapidly since the 1980s, having solved a series of worldwide challenges in the field of blast injury treatment and becoming an international leader in blast injury research in one fell swoop. In the 1980s, Academician Zhengguo Wang wrote the first international monograph *Blast Injury.*

In recent decades, with the frequent occurrence of various explosion incidents and consequently the increasingly serious threats to society, blast injuries have garnered widespread attention, and considerable efforts are being committed to related research as people have begun to truly understand the severity of injuries caused by such explosions and the necessity of proper treatment. To better respond to the medical rescue needs of various explosion incidents, further improve the national awareness, and enhance the level of blast injury prevention and treatment, we have brought together 61 well-known national experts in trauma surgery, field surgery, coal mine medicine, anti-terrorism medicine, explosion physics, and other fields for the compilation of the book *Principles and Practices of Explosive Blast Injury,* which is in fact an updated version and expansion of the monograph *Blast Injury*. The book presents a systematic summary on the series of achievements of blast injury research and new progress in injury treatment in China and related international developments over the past 30 years. It not only includes methods to deal with various conventional injuries inflicted by explosive weapon and nuclear explosion commonly seen in modern warfare, but also systematically covers injuries caused by various explosion accidents (such as coal mine gas explosions and chemical explosions) and terrorist bombing. It also offers a comprehensive introduction to knowledge and injury theory about explosive shock waves, prevention and treatment of various explosion blast injuries and their complications. It is regarded as the only monograph that systematically expounds various types of blast injuries in peacetime and wartime, reflecting the highest level of research and treatment of blast injuries in the world today. Containing a wide variety of contents, the book is very practical as many segments are dedicated to elaborate on the causes of various types of explosion and impact injuries, as well as protection and treatment measures. This book serves not only as an important basis and technical support for military health service support in modern warfare, but is also extremely important in providing practical value in peacetime disaster prevention, mitigation, and relief and embodies great military and social significance for promoting the construction of a world-class army and propelling socio-economic development.

While the *Principles and Practices of Explosive Blast Injury* awaits publication, I would like to express my sincere gratitude to the experts who have participated in the compilation of this book, to whose great support and efforts that the debut of this book largely owes to. At the same time, my sincere thanks go to the People's Medical Publishing House for the guidance and scrutiny for this book. As this book covers a wide range of disciplines with relatively complex content, I hereby implore readers to put forth valuable criticisms and suggestions on any inadequacies.

Chongqing, China — Zhengguo Wang
Chongqing, China — Jianxin Jiang
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About the Editors

Zhengguo Wang is one of the first batch of academicians, researcher, doctoral supervisor of the Department of Medicine and Health of the Chinese Academy of Engineering, academic leader of field surgery, first-level professor of the Chinese People's Liberation Army Special Medical Center, editor-in-chief of the Chinese version of *Chinese Journal of Traumatology*, deputy director of the Military Medical Science and Technology Committee. He is one of the main founders of blast injury, wound ballistics, and traffic medicine research in China, the academic leader of the national key discipline — field surgery, and the first doctoral supervisor of the discipline. He has been engaged in blast injury research since 1970, has entered the nuclear blast zones eight times, visited the front line in Yunnan twice, participated in field tests of weapon explosion, and frequented scenes of accident more than ten times to observe injuries caused by various explosives for investigation and gathering of related first-hand information on blast injury. He has also conducted a large number of animal experiments. In 1984, in coordination with the Institute of Mechanics of Chinese Academy of Sciences, he helped develop the first bio-shock tube in China to simulate indoor shock waves generated by the explosion of different equivalents of explosives, thus enabling the performance of laboratory shock injury research for the first time in China. He also carried out systematic researches on lethal effects of shock wave, dose-effect relationship, safety standards and protection, etc., and he is the first in the world to systematically clarify the shock wave injury mechanism (overdraft effect theory). He put forward the principle of diagnosis and treatment, especially the use of perfusion, casting, freeze-etching, morphometric measurement, molecular biology, and other technologies for the first time, benefitting innovative studies on lung blast injuries. He also proposed a new pathological classification method for lung blast injuries. Regarding the traditional understanding that fluid infusion therapy for pulmonary shock injury would aggravate pulmonary edema and worsen the injury, after in-depth research, he put forward the treatment principle of "sufficient fluid supplementation and monitoring," providing a strong basis for clinical treatment of pulmonary shock injuries. In 1992, as the person in charge of the "development and application of a series of biological shock tubes," he won the first prize of State Scientific and Technological Progress

Award; and then the second prize of State Scientific and Technological Progress Award in 2005 with "Research on the Mechanism and Prevention Measures of Lung, Eye and Brain Shock Injuries." He has published more than 200 papers as first author, written and edited 39 monographs, and participated in the editing of more than ten books. He has won one first prize, five second prizes, four third prizes of the State Scientific and Technological Progress Award, and one third prize of the National Invention Award. He was also a winner of the Science Technology Progress Award by Ho Leung Ho Lee Foundation, the Mikael Debakey International Military Surgeons Award, the TKK Science Awards, and the Guanghua Engineering Science and Technology Prize.

Jianxin Jiang is an Academician of the Division of Medicine and Health of the Chinese Academy of Engineering, researcher, doctoral supervisor, and specialist in field and trauma surgery. He is also the Director of the State Key Laboratory of Trauma, Burns and Combined Injuries, Director of the War Injury Treatment of the Field Surgery Research Department of the Army Special Medical Center of Army Medical University, as well as Director of Key Laboratory of Combined Injuries in Military Field. Throughout the years he has also been concurrently holding various academic posts including the East Asia Regional President of the International Traffic Medicine Association, Secretary General of Asian Trauma Society, Member of the Discipline Appraisal Group of Academic Degree Commission of the State Council, Chairman of Traumatology Society of Chinese Medical Association, Chairman of Trauma Medicine Branch of China International Exchange and Promotive Association for Medical and Healthcare, Vice Chairman of Tissue Repair and Regeneration Society of Chinese Medical Association, Editor-in-chief of the *Chinese Journal of Traumatology*, etc. He has also been elected as a deputy to the 13th National People's Congress. He is mainly engaged in study on the treatment of high explosive weapon injury and wound infection in the field of war trauma and has presided over the completion of more than 30 scientific research projects including the National Basic Research Program (973 Program) and major military projects. He has pioneered studies in the molecular genetics of sepsis in modern explosive weapons wounds and trauma, established modern explosive weapon injury theory, solved the problem of injury mechanism and protection caused by explosive shock wave, and realized the preventable and treatable facts of explosion injury. He has uncovered endogenous infection as a significant pathway of critical injury complicated infection and the mechanism of immune escape infection by pathogenic bacteria, put forward new mechanism such as "trauma sensitization" of trauma sepsis and molecular genetics, established a new technical system for diagnosis and treatment of wound infection, and realized early warning identification and precise prevention and treatment of trauma infection, significantly enabling China to reach an inter-

national advanced level in the prevention and treatment of severe sepsis. He is the winner of four second prizes of State Scientific and Technological Progress Award, Science Technology Progress Award by Ho Leung Ho Lee Foundation, Wu Jieping Medical Innovation Award, Military Outstanding Professional Technical Talent Award, Outstanding Contribution Award for Western Development of China Association for Science and Technology, and Chongqing Outstanding Talent Award. He has also been selected into the New Century Millions of Talents Project, as well as the first batch of Military High-level Scientific and Technological Innovation Talent Project. He is the chief editor of seven monographs including *Trauma Infection Studies* and *Chinese Trauma Intensive Medicine Studies*, among others.

Part I

General Introduction

Introduction and Epidemiology

Zhengguo Wang, Jihong Zhou, and Zhihuan Yang

1 Overview of Explosive Blast Injury

When high explosives or nuclear weapons detonate, a tremendous amount of energy is released at an instant, and the pressure and temperature at ground zero soar in a dramatic fashion. The explosive force then rapidly spreads in all directions through surrounding media (e.g., air, water, soil, steel sheets), forming a high-pressure and high-speed energy wave, and this is explosion shock wave (blast wave). The abrupt movement of high-pressure gas from the firing of a cannon, supersonic flight, explosions from gas leaks, shock tube experiment, and other instances also generate similar shock waves. Bodily injuries caused by shock waves are hereinafter referred to as "explosive blast injury."

In clinical context, "explosion blast injury" usually refers to primary injury caused directly by shock waves in air or water. Injuries caused by shock wave in a solid (e.g., deck of a warship), or mechanical trauma from objects thrown by shock wave or other indirect effects (e.g., collapse of structures) may be categorized as "blast injuries" but are usually not called "explosive blast injuries."

In modern warfare, belligerents might adopt carpet bombing strategy and drop a huge number of large bombs in densely populated cities, or use bombs that mainly cause destruction through shock wave such as aerosol bomb or fuel-air explosive bomb, which are more likely to result in blast injuries. Take for example an equivalent to a 5-megaton nuclear weapon, the shock wave could injure personnel exposed on the ground surface within an area of over 800 km. This is only the direct kill zone, and if the indirect impacts of shock wave are taken into account, the area of effect would enlarge by one to two folds. Shock wave is one of the primary destructive elements that cause injury and damage in the use of a nuclear weapon. In August 1945, some 70% of injuries in the atomic bombings of Japan resulted from shock wave. At Hiroshima, among the deaths early on, 60% were attributed to blast injury. Among victims of moderate and severe injuries that survived after the first day of detonation, those afflicted by blast injuries accounted for 36.6%. In conventional warfare, shock wave is one of the primary destructive elements essential to the different types of explosive weapons. For instance, among the 1303 cases of patients severely wounded by explosion treated at the former Yugoslav Academy of Military Medical Sciences, 51.0% resulted from blast injuries. In skirmishes along the border in southwestern China, among a group of 166 persons injured by artillery and mines, 22.3% resulted from blast injuries.

Outside of the battlefield, many have been injured or killed from explosions in weapon factories, munition depots, chemical plants, mines, or other areas, not to mention victims of terrorist attacks. Bombings account for approximately 75% of terrorist attacks, and blast injury is one of the most common types of injuries caused by such attacks.

With advancements in explosive production and technology, explosives become increasingly diverse and powerful. Some common types of explosives include black powder, ammonium nitrate, nitroglycerine gelignite, TNT, RDX, Composition C and other plastic explosives, emulsion explosives and liquid explosives, to name but a few. In recent years, Composition C plastic explosive has risen as the weapon of choice for terrorist bombing. For example, Composition C plastic explosives were used in two series of bombings in Indonesia, respectively at tourist district on the island of Bali in 2002 and at upscale hotels in the business district of capital Jakarta in 2009.

Explosive devices are becoming smaller and smarter, and less metals are being used. Some explosives are made to look like toys, toothpaste, and other daily items, while others convert cameras, radios, and other objects into small bombs. Methods of detonation have also diversified from safety fuse to electrical, mechanical and chemical means, and even methods like remote control, temperature control, light control, or sound control. On July 9th, 2007, a bombing in Jinan

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shocked the nation, as perpetrator used remote control to detonate an explosive device planted inside a car.

Thus, it can be seen that blast injury is not only a matter crucial to military medicine, but also a type of injury and emergency commonly seen in hospitals.

In addition, it should also be pointed out that most vulnerable to the typical blast injury (as in what is frequently referred to as "explosive blast injury") are auditory apparatus and organs, in particular the lungs because of its high content of air. Many victims meanwhile do not exhibit obvious signs of injury on the surface. In the early phase of injury, the vital signs (e.g., breathing, circulation) of the injured might appear normal because of the body's natural tendency to compensate, but soon after the situation would quickly deteriorate. At the same time, blast injuries might be accompanied by other injuries (i.e., burns or other mechanical injuries), or bodily damage might manifest as multiple trauma. Prompt diagnosis and corresponding treatments are mandatory so as not to miss the best opportunity for medical intervention, otherwise the consequence could be dire or might even result in death.

2 Physical Parameters and Biomechanical Mechanisms of Injury from Blast Wave

2.1 Physical Parameters of Injury from Blast Wave

Main physical parameters of injury from blast wave include: peak value of shock wave pressure, duration of positive pressure, impulse, duration of pressure increase, etc.

1. Peak value of shock wave pressure. Peak value of shock wave pressure refers to the highest value of pressure in a blast wave and may be categorized as overpressure peak, negative pressure peak, and dynamic pressure peak. It is measured in kilopascal (kPa). A blast wave's pressure peak is the main parameter that causes blast injury, and it positively correlates with severity of injury. In other words, the higher the peak value of the shock wave at the site of explosion, the more severe the injury.
2. Duration of positive pressure. Duration of positive pressure refers to the length of time of positive pressure caused by shock wave in the pressure zone. It is measured in millisecond (ms) or seconds (s). Within a certain length of time and under the same peak pressure, the longer the duration of positive pressure, the more severe the injury.
3. Impulse. Impulse refers to the sum of the value of instantaneous pressure within the duration of pressure, or in other words, the integral of the force of pressure with respect to time. It is measured in kPa/s or kPa/ms. Impulse consists of two parameters, i.e., pressure peak and positive pressure duration, and it is more accurate and suitable in describing the relationship between a blast wave and the severity of injuries caused. In particular, for blast injuries that happen underwater, pressure peak is high but positive pressure duration is short; or for composite blast injuries that occur inside enclosed spaces such as that of a tank or armored vehicle, impulse can better explain the relationship between the shock wave's physical parameters and the resulting blast injuries.
4. Duration of pressure increase. Duration of pressure increase refers to the length of time starting when pressure acts on a certain point on the body until reaching its peak value. It is measured in millisecond (ms) or second (s). Duration of pressure increase reflects the rate at which pressure rises on a point affected by the shock wave, and when other conditions are constant, the shorter the duration of pressure increase, the faster the rate of pressure increase, the more severe the blast injury.

2.2 Biomechanical Mechanisms of Injury from Blast Wave

Blast wave injuries mainly result from the direct effect of blast wave, the indirect effect from the displacement of objects caused by the blast, and being thrown and collided against other objects due to dynamic pressure. Injury mechanism behind shock wave can be simplified as either a direct or indirect result of the shock wave. The biomechanical mechanisms, however, are not completely understood, particularly with regard to the effects of the overpressure and negative pressure from a blast wave.

1. Direct effects of blast wave refers to injury arising from the pressure of a blast wave (overpressure and negative pressure). Such injuries are called primary blast injury or pure blast injury, and are chiefly manifested in injuries to air-filled organs such as lungs, gastrointestinal tracts, and auditory apparatus, in addition to possible bleeding in some more solid organs. Strong overpressure on the human body could cause rupture in organs and fractures in bones such as ribs and ossicles, but usually does not cause direct injury on the surface. At present, there is general agreement that direct shock wave injury mechanisms mainly include:
   (a) Implosion: When a shock wave propagates through a liquid medium that contains bubbles or air pockets, the overpressure of shock wave would cause the compressible air to compress drastically, while liquids and solids would not be compressed nearly as much. The shock wave's overpressure is followed by negative pressure, which would cause the compressed

air bodies to expand immensely, much like many mini explosions that release energy in all directions and injure tissues in their surroundings. Implosion-induced injuries usually occur in tissues of air-filled organs such as the lungs and gastrointestinal tract.

(b) Pressure differential: When pressure on two sides of a tissue differ, such pressure differential could directly injure said tissue. Therefore, when a shock wave propagates and as it reaches a certain tissue or organ, the tremendous difference in pressure at a local area within a split instance caused by the high pressure on one side and ambient pressure on the other side could directly injure said tissue or organ. For instance, eardrum rupture caused by overpressure is the outcome of pressure differential. Another example happens in the lungs, when shock wave hits a body, pressures rise in both the liquid (blood inside vessels) and gas (air in pulmonary alveoli), but pressure rises more in liquid, and the massive pressure difference between liquid and gas would rip apart the capillaries, resulting in blood flowing into pulmonary alveoli and pulmonary hemorrhage.

(c) Overtension: When air-filled organs in the body are hit by a blast wave, during the pressure-decreasing and negative pressure phases, these air-filled organs could change from being under pressure to being expanded, as in tissues changing from being compressed to being inflated and extended, and these tissues have to bear tensile strain and tension stress arising from such inflation. In most cases, tissues can withstand much more compression than extension, and when tensile strain reaches a certain point, microvascular endothelial cells and alveolar epithelial cells would become more permeable, which would result in edema and bleeding. Worst, when tensile strain exceeds the limits of the tissue's capacity, more serious edema and bleeding would occur as the tissues and blood vessels rupture. During the course of pressure decrease, the higher the pressure peak and the shorter the duration of pressure decrease (i.e., faster rate of pressure decrease), the more obvious the overtension and the more serious the injury.

(d) Spalling (fragmentation): When a blast wave propagates through the body from a compact tissue into loose tissue, reflections take place at the interface between compact tissue and loose tissue. This type of reflected wave could cause a sudden rise in local pressure in the compact tissue, leading to injuries such as alveolar laceration and bleeding, subendocardial bleeding, and bladder mucosal bleeding.

(e) Inertia: When the same shock wave acts on two tissues with different densities, the two tissues accelerate and decelerate at markedly different rates, and this difference causes tremendous shear stress on the interface between the two tissues, resulting in laceration where the two connect. For examples, rib and intercostal tissue lacerations and bleeding, intestinal and mesenteric tissues lacerations and bleeding are both attributed to shock wave inertia.

(f) Negative pressure: Immediately following the overpressure of a blast wave is negative pressure. The speed at which pressure drops, the duration of negative pressure and peak value of negative pressure are the chief parameters in injury, of which, negative pressure peak value plays the biggest role. Negative pressure could result in severe injuries to the lungs, such as widespread pulmonary hemorrhage and edema. Worth noting is that the negative pressure peak value needed to cause such severe injury is much less than overpressure peak value.

(g) Hemodynamics: After a blast wave's overpressure acts on the body, the pressure pushes against the soft abdominal wall, causing pressure inside the abdominal cavity to rise rapidly, in turn pressing the diaphragm upward, causing blood in the superior vena cava to abruptly rush into the heart and lungs, sharply increasing blood volume in these organs. At the same time, the shock wave's overpressure also presses against the chest cavity, decreasing the volume of the space behind the chest, and since the thoracic cage is relatively harder than the abdomens, the pressure increase in the chest cavity is relatively delayed, resulting in subsequent rush of blood toward the head and sharp increase in blood volume inside the head. Right after overpressure is negative pressure, and the retraction due to pressure decrease would cause the abdominal cavity and thoracic cage to enlarge. This kind of rapid compression and expansion generates huge hemodynamic changes, resulting in injuries to the heart, lungs, and distant vascular tissues (such as that of the brain).

2. Indirect effects of blast wave: Indirect and secondary injuries caused by projectiles and other elements resulting from the dynamic pressure of a blast wave are collectively known as indirect blast wave injuries. Indirect injury effects of blast wave mainly include:

(a) Secondary projectiles: Not only does the dynamic pressure of a blast wave turn fragments and shrapnel of a shell into projectiles that could injure the human body, but also imbues other objects (e.g., glass, stone) with kinetic energy and turns them into damaging projectiles. Bombing investigations and statistical data acquired after the atomic bombings of Japan show that the majority of different kinds of open wounds were caused by these secondary pro-

jectiles. In cities, industrial sites, and residential areas, most secondary projectiles are glass shards from windows, in open spaces meanwhile rocks and even dust or dirt could be "weaponized" as projectiles.

(b) Throw and displacement: When dynamic pressure is strong enough, it could manifest as an impact force or projection force. When the dynamic pressure of a blast wave hits a human body, the person could be displaced or thrown high in the air, and then land from a high altitude or impacted against another solid object, resulting in injury. Injuries due to being thrown or displaced are similar to traumas from falling or traffic accident, such as skin abrasion, contusion of subcutaneous tissue, internal organ bleeding and rupture, and bone fracture.

(c) Crush and collision from the collapse of structure: Blast wave often causes a portion or an entirety of structures or fortifications on the ground surface to collapse, crushing or burying people within, leading to surface soft tissue and internal organ injuries alongside bone fractures, with crush injuries and crush syndrome appearing in the more severe cases. When fortifications covered in dirt collapse, people within might be buried and even die from suffocation.

(d) Other concurrent injuries: During the course of an explosion, often times there are other injury causes such as flash, fire, poisonous gas, dust, drowning, radioactive substance, virus, and other pathogens, which could lead to corresponding injuries to the human body.

3 Types of Blast Injuries

Due to the varying metrics systems and standards, blast injuries could be classified using different methods. For instance, methods could be based on blast injury cause, shock wave propagation medium, or body part and organ injured, etc.

3.1 Classification of Blast Injury Cause

Classification based on the biomechanics behind blast injury is a method based on the dynamics of how people are injured by a blast wave. In this regard, most classification methods used in China and abroad are based on the method developed by Zuckerman during World War II. This method classifies blast injury into four types: primary blast injury, type II blast injury, type III blast injury, and type IV blast injury, of which, the last three blast injuries are also known as secondary blast injuries.

1. Primary blast injury refers to injury directly caused by physical factors such as a shock wave's overpressure, dynamic pressure, or negative pressure, and may be called a pure blast injury. Since air is easily compressed and expanded, primary blast injuries are often seen in the lungs, middle ear, gastrointestinal tracts, and other air-filled organs.
2. Type II blast injury refers to bodily injuries caused by projectiles like shrapnel, fragment, broken glass and rock launched by the force of a blast wave. Such injuries are mostly penetration or laceration wounds, and could be seen on any part of the body from the surface and internal organ to the limbs.
3. Type III blast injury refers to collision injuries when people are being thrown by the force of a blast wave, or being struck or crushed by the collapse of structure and fortification. These could result in penetration wound, blunt trauma, bone fracture, traumatic disjunction, crush injury, and crush syndrome on any part of the body.
4. Type IV blast injury refers to any other injuries or diseases related to an explosion but not classified as either primary, secondary, or tertiary blast injury. This miscellaneous group includes bodily harms from flash, fire, toxic gas, dust, drowning, psychological factor, and other issues caused by an explosion, and may afflict any part or organ of the human body.

3.2 Classification of Shock Wave Propagation Medium

Since any blast wave-induced injury to the human body may only occur through some sort of medium, how an explosion causes injury is closely associated with shock waves in different media, including characteristics of propagation, features of injury causes, exposure–response relationship and outcomes. Therefore, classifying blast injury based on the shock wave propagation medium has many merits. Generally speaking, shock wave propagation medium are classified as either air blast injury, underwater blast injury, or solid blast injury.

1. Air blast injury refers to injuries to the body from blast wave propagated through the air. The term "blast injury" predominantly refers to air blast injury. Air blast injury is not only associated with the shock wave parameters discussed before, but also the wavelength and frequency of shock wave in the air. When shock waves in the air have relatively short wavelength and generate high-frequency "cracking" sounds, the number of shock waves that hit the human body is higher per unit of time, which translates into higher probability of injuring the human body. On the contrary, when shock waves in the air have

relatively long wavelength and emit low-frequency "boom" or "thud," usually only a single wave would strike the human body, which in turn means that the probability of harms to the human body is much lower.

At high altitudes, where the air is thin and atmospheric pressure is low, the same shock wave with the same force would cause more severe blast injury than at a lower altitude.

The author's laboratory conducted an experiment using BST-II bio-shock tube to study how rats would be injured by blast waves under different atmospheric pressures (53.99 kPa, 61.33 kPa, and 96.60 kPa). Results show that when overpressure peak value (190.40 kPa) and positive pressure duration (10 ms) remain constant, lower atmospheric pressure leads to significant increase in fatality rate and significant rise in lung injury severity. After 6 h, the fatality rate of the three groups of rats were respectively 36.8%, 25.0%, and 0%, area of pulmonary hemorrhage were respectively (653.21 ± 652.25)mm^2, (313.50 ± 357.25)mm^2, and (63.75 ± 69.01)mm^2, and lung volume indices were respectively 1.51% ± 0.77%, 1.31% ± 0.65%, and 0.93% ± 0.21%, indicating that lower atmospheric pressure raises fatality rate and exacerbates lung injuries.

In addition, BST-I bio-shock tube and decompression chamber were used to replicate and model the rats' high-altitude blast injuries so as to observe morphological and hemorheological changes. Results indicate that pulmonary hemorrhage and edema were more severe compared with low-altitude injuries, blood viscosity elevated significantly and remained heightened even 6 h after injury.

2. Underwater blast injury refers to injury to people in water due to blast wave generated in the subsurface explosion of bombs, missiles, or other explosive devices and propagated through water. Naval warfare is one of the major battlespaces in the future, which is why underwater blast injury has become one of the focal points of modern blast injury research, which take into full account the characteristics of shock wave propagation and injury causes in water.

The physical properties of underwater shock wave vary markedly from shock waves in the air, and therefore, injuries also differ vastly. Some of the main differences include: (1) Increased speed of propagation (usually three to four times faster than in the air); (2) propagated relatively farther, and area of effect of blast wave in water is almost ten times larger than that in the air; (3) no compression zones or rarefaction zones, and water molecules also do not move as much as air molecules as a shock wave propagates through them; (4) when underwater shock waves reach the interface between the water surface and the air, reflection occurs and creates unique reflected waves, or tensile waves. The tensile waves propagate in directions different from the incident waves, and serve to reduce the incident waves (Fig. 1). The closer the point of action to the water surface, the more the incident wave is reduced (Fig. 2). In other words, when there is an underwater explosion, people closer to the water surface would be less severely injured.

Clinical features of underwater blast injury are as follows: (1) There are extremely few injuries to the surface of the body. In an underwater explosion, usually there won't be a large amount of secondary projectiles, and seldom would people be thrown against some sort of hard, solid object. Thus injuries to the surface of the body are rare. (2) Injuries to air-filled organs are severe while injuries to liquid-containing organs are light. The former may be explained by implosion, while the latter is caused by similar density between liquid and soft tissues. Such an experiment has been performed previously: Isotonic saline-filled animal intestine was subjected to an underwater explosion, and no damage to the intestine was observed, even if the intestine was placed near the explosive. However, when there was even a tiny amount of air left inside the intestine, holes could be seen on the intestinal wall right after an explo-

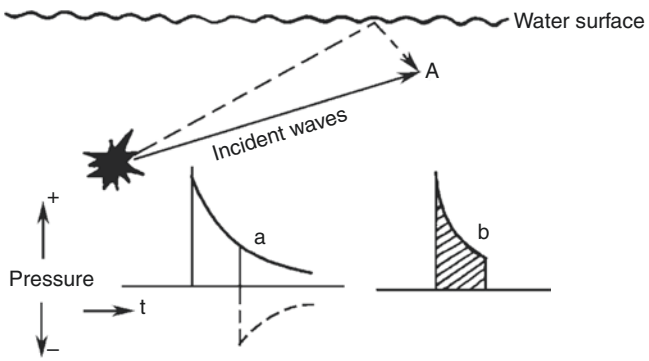

Fig. 1 Formation and action of tensile waves. (**a**) Incident waves before reaching point A; (**b**) reduced incident waves when reaching point A; *A* action point, *t* time

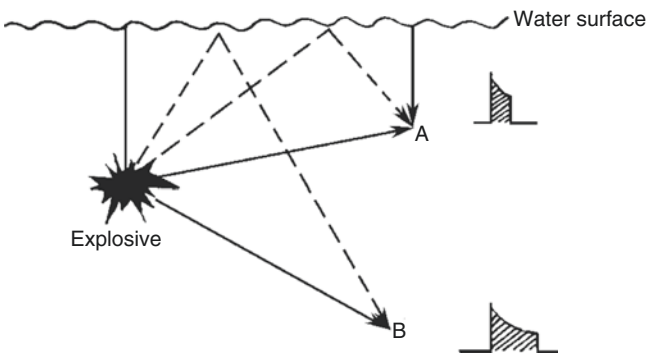

Fig. 2 Different effects of various tensile waves. *A* Action point, *B* Action point

sion. (3) Most head injuries are light. This is because during an underwater explosion, the majority of victims are near the surface and their heads are above water. (4) Most abdominal injuries are severe. For people underwater or floating on the surface, the abdomen is in direct contact with water, and the abdominal wall is relatively soft, which is why when an underwater explosion occurs, organs in the abdomen (gastrointestinal tract for the most part) are more prone to severe injuries compared with an air explosion. (5) The fatality rate is relatively higher. One report indicates that 47 victims out of 118 in an underwater blast died, a fatality rate of 39.8%. Another document reports that 9 victims out of 13 in an underwater blast died, a fatality rate of 69.2%. Meanwhile, in general 90% of air blast injury victims are not severely injured.

In order to understand the characteristics of underwater blast injury and the exposure–response relationship between shock wave strength and injury severity, the author's laboratory carried out the following experiment: 37 mixed-breed dogs anesthetized, floatation devices were attached to their necks so that their heads would stay above water while the bodies and limbs would be underwater perpendicularly to the surface. The dogs were placed on either side of the 3.5 m and 17.5 m in a distance from explosion center (Figs. 1, 2, and 3), TNT explosives ranging from 0.2 to 1.0 kg in quantity were placed 3 m underwater, high-pressure instantaneous detonator was used for detonation, and the survival conditions and pathological changes in the animals were observed on-site and 6 h thereafter. Result shows that (1) the features of physical parameters of underwater shock wave are: high peak pressure value but short duration, as in several hundred microseconds, far shorter than the several or tens of milliseconds in the duration of shock wave in air generated by an explosion. In addition, the duration of pressure increase is measured in microseconds, which is extremely short particularly when compared with the 1 ms-range of explosions in the air. Therefore, cause of injury by underwater shock wave can't be determined simply with using overpressure peak value, and in this regard impulse is more suitable. Preliminary exposure–response relationship analysis indicates that impulse ranges respectively for light, moderate, severe, and extremely severe injuries are 121.1–14.0 kPa/ms, 142.0–214.3 kPa/ms, 247.8–322.6 kPa/ms, and 322.6–579.8 kPa/ms. (2) The lethal radii for 0.2 kg, 0.5 kg, and 1.0 kg TNT explosions are respectively 5 m, 8.75 m, and 12.5 m from the blast center, much farther than the lethal range of mid-air explosions with the same amount of explosives. The lethal radius of 0.5 kg TNT underwater explosion (8 m) is approximate to 40.0 kg of TNT explosion in mid-air. (3) The fatality rate is high. Of the 37 dogs, ten died on-site (two had collapsed lung and pneumonia before injury, and thus were not counted), and no death was recorded 6 h after injury, registering the final fatality rate of 27%. This result may be attributed to the fact that explosions from the same quantity of explosives at different ranges underwater generate much stronger shock waves when compared to explosions in the air. (4) Lung injury is the most common (83.7%) and most serious, with the majority of on-site deaths associated with severe pulmonary hemorrhage and edema, coronary artery air embolism arising from ruptured lung and pulmonary injury were even observed in some animals. (5) There is a high rate of injury to the intestine, with 29.73% afflicted by small intestine injury and 51.35% afflicted by colon injury, far higher than those caused by mid-air explosions. Colon injury was more common because it contains more air. (6) The rate of injury to solid organs is low. Other than three cases of slight bleeding in the pancreas and one case of ruptured liver, no injuries were found in spleens, kidneys, and filled bladders. (7) There are no injuries on the surface of bodies.

Fig. 3 Placement of test animals for underwater blast injury experiment

3. Solid blast injury refers to injuries to the body from blast wave propagated through solids. The propagation of blast wave in solid is vastly different from propagation in air or water, specifically, the relatively smaller amplitude and shorter time of the shock wave's action (usually within a few milliseconds), but also an immensely faster acceleration. Solid blast injuries are often seen when battleships, tanks, or armored vehicles are hit by an explosive, and the blast wave and secondary shock wave thereafter act on the structure, deck and armor of the struck vessel/vehicle, then propagate in the form of flexion wave. This results in two types of motion: First is the slight displacement and acceleration of solid, and second is the subsequent bending, vibration, and other obvious macroscopic motions. The first type of motion would injure body parts that are in contact with the solid, usu-

ally injuries to the lower limb, especially damage to the ankles. This is considered primary injury of and the general definition of solid blast injury. The second type of motion might cause victims to be thrown against other objects and injured, which would be deemed secondary solid blast injury.

Main characteristics of solid blast injury include: Mostly injuries to bones and joints in the lower extremities, and this kind of bottom-up impact might result in closed fracture and injury to the heel bone, phalanges, shinbone and lower part of calf bone and ankle joint, with heel bone fracture being relatively frequent. Analysis of information about 50 victims of solid blast injuries shows that 18 suffered from bone fractures in the ankles, with the majority being multiple, comminuted fractures, including 11 victims with heel bone fractures affecting 15 limbs. Body part injured is clearly associated with body position, and the majority of injuries occur on one side of the body. For example, a particularly lower extremity is more easily injured when standing, while the spine is more likely to take damage when sitting. Injuries to solid organs in the abdominal cavity are also quite common, and one possible reason is that the acceleration of the shock wave induces deformation and displacement of internal organs, leading to crushing, collision, retraction, and other injuries when organs interact with bones, muscles, and ligaments. Injuries to the liver and spleen are the most frequent. Indirect injuries happen often, mostly manifested as damages such as soft tissue injury, bone fracture, cerebral concussion arising from sudden acceleration and movement of brain matter caused when a person is thrown or horizontally displaced then struck against something. Of the 50 hospitalized cases, 32 showed loss of consciousness, which resulted from head injuries when they were thrown or displaced.

3.3 Classification of Body Part and Organ Injured

Classification based on body part and organ injured is a method that focuses on the specific body part and organ injured by a blast wave. Some common categories are brain blast injury, thoracic blast injury, abdominal blast injury, blast injury of the spinal cord, and blast injury of the extremities. Moreover, blast injuries may also be categorized as injury to the lungs, heart, brain, gastrointestinal tract, liver, auditory apparatus, and other body parts. Among them, blast injuries to the lungs, gastrointestinal tract, and auditory apparatus occur on a more frequent basis.

Through body part and organ-based classification, diagnosis can quickly pinpoint the area(s) injured by blast wave, and carry out corresponding diagnosis, prevention and research methods based on injury mechanisms and characteristics.

3.4 Classification of Injury Severity

1. Pathological classification: Yelverton, an American scientist, recently introduced an injury scoring system that may be used as basis for judging severity of injury. The main points include: (a) First of all, calculate the overall score of an individual injury including its scope, severity, type, depth, or wound condition; (b) divide the score of said injury by the worst case scenario-score for that type of injury to obtain the ratio score of said injury; (c) add the ratio scores of all individual injuries to find their sum; (d) add scores of pathogenic factors (e.g., pneumothorax, hemothorax, hemoperitoneum, coronary artery air embolism or cerebral vascular air embolism); (e) multiply the score by two if the victim dies; and (f) to determine the level of non-auditory apparatus injury, subtract the ratio score for auditory apparatus injury from the Severity of Injury Index (SII) to obtain the Adjusted Severity of Injury Index (ASII). This methodology is detailed and relatively accurate, but also somewhat complex.
2. Clinical classification
   (a) Light: General injuries to auditory apparatus, light internal organ contusions (intraplaque hemorrhage), scratches on the surface of the body, etc.
   (b) Moderate: Relatively large-scale internal organ contusions (patchy hemorrhage or hematoma), relatively light pulmonary edema, large swaths of soft tissue injury, dislocation, rib fracture with no obvious dislocation, cerebral concussion, etc.
   (c) Severe: Ruptured internal organ, fractured bone (thigh bone, spine, cranial base, and multiple fractured ribs), relatively severe pulmonary edema, pulmonary hemorrhage, etc.
   (d) Extremely severe: Extremely serious or fatal injuries such as severe cerebral and spinal cord injury, ruptured chest cavity or abdominal cavity, widespread and serious pulmonary hemorrhage or pulmonary edema, ruptured artery, amputation with severe bleeding, etc.

4 Epidemiological Features of Blast Injury

Blast injuries differ quite markedly from other kinds of injuries in terms of aspects such as injury causing condition, injury mechanism, on-site environment and treatment. Therefore, the injury characteristics and epidemiological features of blast injury are also quite different from other types of injuries.

4.1 General Features of Blast Injury

Since both the direct and indirect effects of a blast wave act on the body during the course of a blast injury, things can get very complicated when injured tissues and organs, injury mechanisms and processes are all taken into consideration. The situation is further compounded due to the varying environments and conditions where injury occurred. Therefore, blast injuries are characterized by features unlike those seen in other types of injuries. In general, the features of blast injury include:

1. Complicated injury: The overpressure, negative pressure, and dynamic pressure of a blast wave all could cause injury on their own or when acting together, both directly and indirectly. This diversity in injury causes and methods means that blast injury types and conditions are complicated. Blast injuries are complicated for the following reasons: Most blast injuries are multiple injuries or injuries to multiple body parts, and external and internal injuries to numerous organs and body parts could all happen at the same time. Most blast injuries are combined injuries such as blast-fragment combined injury, burn-blast combined injury, and radiation-blast combined injury. Blast injuries often include different kinds of injuries such as blunt trauma and penetration wound, or contusion and rupture on the same body, or edema and hemorrhage simultaneously. In particular, external injuries caused by explosion are often accompanied by relatively serious infections due to contact of open wounds with disease-causing agents. Thus, many medical professionals naturally associate blast injury victims with substantial infection.
2. Blast injuries mostly affect specific, target organs. Although blast injuries could damage any part or tissue of the body, because of the features of the shock wave itself and of its propagation medium, most blast injuries affect specific organs. Air-containing tissues and organs are the primary victims of air blast injury or underwater blast injury, which is why injuries to the eardrum in the middle ear, the lungs, and gastrointestinal tract are almost unavoidable. Solid blast injury meanwhile almost always affects body parts in direct contact with the solid medium of shock wave propagation, or body parts that the propagated shock wave act on longitudinally. Thus, identifying the target organ damaged in blast injury is of utmost importance.
3. Light external injury and severe internal injury. This characteristic is the result of the blast wave's mechanisms and modes of action. When a shock wave acts on the body, injury on the surface often appear light, especially injuries caused only by overpressure or negative pressure, whereby the body surface might not even have any obvious sign of damage. However, the target organs inside, such as the lungs or gastrointestinal tract, might have already sustained heavy damage, or in other words, light external injury and severe internal injury.

   At the site of major bomb explosion experiment, when an animal is closer to the center of explosion (distance varies with respect to explosion equivalent), there might be visible signs of external injuries to the body surface and limbs, but injuries to the internal organs are always more severe, and most of the times the primary cause of death. For animals within the range of injury but farther from the center of explosion, there are relatively fewer and lighter injuries to the body surface and extremities, but internal injuries could still be dire and are even the main cause of fatality.
4. Rapid deterioration of injury. For severe or worst blast injuries, there is a relatively stable compensatory period within a short span right after the injury, but if treatment is not administered promptly, the situation would rapidly worsen. In particular, brain injury, pulmonary hemorrhage, pulmonary edema, or other organ injury would further speed up the deterioration.

   During the atomic bombings of Japan in the Second World War, there were a relatively few number of victims recorded as severe or worse blast injuries, which might be because the condition exacerbated rapidly for many such badly injured victims, resulting in casualty. At the site of explosion experiment, it has been observed that some animals initially showed decent conditions and normal movement in a short span after the explosion, but soon they would exhibit difficulty in breathing, shock, and then death. Dissection shows that these animals were mostly afflicted by serious pulmonary hemorrhage, pulmonary edema, or ruptured organs liver, spleen and other internal organs.

4.2 Incidence Rate and Fatality Rate of Blast Injury

With advancements in modern, hi-tech and high-explosive weaponry, and changes in the mode and method of armed conflict, modern warfare has seen an increasingly abundant use of various kinds of hi-speed and high-explosive weapons (including improvised explosive devices), and the ratios of injuries and casualties from blasts and explosions have continued to rise.

Take military actions for instance. Between 2001 and 2014, more than 6700 American soldiers in Afghanistan and Iraq have been killed by explosions, and over 50,000 wounded. For the US forces in Iraq between March 2003 and October 2011, improvised explosive device (IED) alone killed around 2200 American soldiers and injuring another 22,000. During the

two military campaigns in Iraq, the number of deaths and casualties among the Iraqi troops and normal citizens are even more difficult to count or even estimate.

In the past several decades, there have been many acts of violence perpetrated through explosives around the world, with terrorist bombings being especially devastating. In Israel alone, nearly 20,000 terrorist attacks were perpetrated between September 2000 and December 2003, resulting in approximately 900 casualties, of which suicide bombings have killed 412, accounting for 45.78% of total. Report from the UNESCO Center for Peace shows that between September 11th, 1993 and September 10th, 2009, across the globe there were 624 terrorist bombings that resulted in multiple injuries and deaths, with casualties amounting to a total of 26,073 victims (an average of 42 killed in each attack). The September 11th attacks in 2001 were a watershed moment, with 68 major terrorist bombings (including 9/11 itself) having been committed in the previous 8 years, resulting in the deaths of 3921 persons; meanwhile 556 major terrorist bombings took place in the next 8 years, killing a total of 22,152 persons, with the number of occurrences and casualties being respectively 8.2 times and 5.7 times more than the first 8 years. Clearly, terrorist attacks around the world have been on the rise, and their destructiveness to the worldwide community is only getting worse.

At present, there is not yet a relatively uniform database about bombing-related injuries anywhere in the world. In addition, due to the difficulty in gathering data about explosion-related injuries in warfare, there is still no report or analysis, at least with a relatively comprehensive scope, on worldwide data about the epidemiology of blast injuries. Most blast injury data available originate from the analysis of data of individual explosions, or data from regional databases or research centers such as the US-based Terrorism Research Center and the Global Terrorism Database. Therefore, no one has an accurate idea about the overall incidence rate and fatality rate of explosion-related blast injuries, and we can only make estimates through investigations and data analysis.

1. Blast injury incidence rate: At present, the atomic bombings of Hiroshima and Nagasaki in 1945 by the USA remain the only use of nuclear weapons in warfare, and some of the earliest statistics and data for analysis of relatively detailed extent also came from these atomic bombings. Post-war info on injury and casualty shows that among the moderately and severely wounded, 36.6% were blast injuries, 60% of early casualties in Hiroshima died from injuries due to blast wave, nearly 70% of the wounded (70% for Hiroshima, 64.3% for Nagasaki) that have survived after 20 days of the bombing were afflicted with combined injury that included blast injury. These wounded obviously suffered from blast injuries coupled with some other injuries (e.g., burns, radiation). Due to the limited understanding about and diagnostic capacity for blast injury at the time, it is possible that some others wounded by blast injuries were not tallied. Thus, conservative estimate of blast injury incidence rate for the atomic bombings should be above 70%.

In the modernization of weaponry, development in explosive weapon is one of the fastest and most obvious. From simple artillery shells, bombs, mines and cluster bombs, to high-explosive squash head (HESH) projectiles, shaped charges and weapons based on augmented shock waves (e.g., fuel-air explosives), even large atomic bombs and hydrogen bombs, the destruction to equipment and structures and fatality to personnel caused by the blast wave of these weapons are increasingly terrifying. In other words, shock wave is destined to be one of the most important and dangerous cause of injury and death in armed conflicts in the future, whether nuclear or conventional.

The USA deployed thermobaric weapon (fuel-air explosives) for the first time during the Vietnam War, and report of data analysis about 101 cases of wounded personnel shows that blast injury incidence rate was 50.4%. Another report indicates that blast injury incidence rate during the First Chechen War in Russia was 30%. When tallying the 1303 cases of patients severely wounded by explosion treated at the former Yugoslav Academy of Military Medical Sciences, it was discovered that blast injury incidence rate was 51.0%. In skirmishes along the border in southwestern China in the 1980s, among a group of 166 persons injured by artillery and mines, blast injury incidence rate was 22.3%. In a research about a certain model of fuel-air explosive carried out by the author's organization, it was discovered that of the animal deaths caused by said model of fuel-air explosive, the incidence rate of blast injury was a whopping 100%, while that for severely injured animals exceeded 90%.

Various types of bombs and improvised explosive devices have become the weapon of choice in terrorist attacks, and blast injuries from these explosives constitute the main cause of injury and death in victims. Analysis of statistics of 647 wounded terrorist attack victims tallied after their arrival at hospital shows that blast injury incidence rate was 29.8%. Analysis of a group of 3357 victims in another terrorist bombing points out that of those that died on-site, blast lung injury alone accounted for 47.0%.

In terms of distribution of body parts injured by blast wave, figures from different reports vary. In general, among blast wave survivors, about 10% have injured eyes, 9–47% have damaged auditory apparatus, 3–14% have obvious blast injury to the lungs, while only 0.3–0.6% suffered from blast injury in the gastrointestinal tract.

Clinically speaking, although incidence rate of abdominal injury from blast wave is not very high, the fatality rate from such injury is relatively high. Analysis of 61 articles and papers between 1966 and 2009 shows that average incidence rate of abdominal blast injury was 3.0% (lowest was 1.3%, highest was 33.0%). Primary blast injury incidence rates in open space and enclosed space are respectively 5.6% and 6.7%.

2. Blast injury fatality rate: Compared with other types of injury, blast injuries are characterized by more complications and more severe injuries, as well as a higher fatality rate. In most cases, death rates of blunt trauma or penetration wound exhibit a classic three-phase distribution, while that of blast injury is characterized by a two-phase distribution, namely a relatively higher rate of instantaneous death, and relatively lower fatality rate later on.

Instantaneous death rate is contingent on a myriad of influential factors such as intensity of explosion equivalent, distance from center of explosion, potential number of victims, structural collapse of buildings, and whether environment is open or enclosed. When other conditions are the same, structural collapse of buildings and fortifications, and whether environment is open or enclosed, are relatively more influential on blast injury severity and fatality rate.

When buildings and fortifications collapse due to an explosion, death rate of blast injury significantly rises. For example, analysis of blast injury data about 29 groups of explosion victims shows that instantaneous death rate is as high as 25% when there is structural collapse. In addition, explosions in enclosed spaces would lead to a larger number of and more severe primary blast injuries, along with a significant increase in instantaneous death rate. Research report shows that in explosions that occurred in enclosed spaces, death rate fluctuates between 8.3% and 15.8%, whereas fatality rate in open space explosions is merely 2.8–4%. Other experiment and research outcomes also demonstrate that with the same explosion equivalent and same density of animal distribution, primary blast injury incidence rate would reach as high as 78% and death rate 49% in enclosed space. Meanwhile, in open space, primary blast injury incidence rate drops to 34% and death rate merely 7.8%.

At the explosion site, victims with light or moderate primary blast injuries usually appear similar to those unwounded persons due to the absence of external injuries, but it is extremely difficult to diagnose and identify any damage to internal organs on-site. In addition, the fatality rate among victims with light or moderate primary blast injuries is very low, which drags down the death rate of blast injuries and can't truly reflect the severity of blast injuries. Therefore, some researchers use death rate from critical injury to reflect the seriousness of blast injury of an explosion and rescue performance level. In most cases, critical blast injury may be categorized as requiring immediate surgery, ICU care, or endotracheal intubation due to acute problems in windpipe, breathing, circulatory system, or nervous system. Death rates of critical blast injury as seen in documents and reports range from 9% to 22%.

5 Principles for Treatment of Blast Injury

In order to promptly and effectively perform emergency rescue, diagnosis, evacuation and transportation to hospital, and treatment, first and foremost it is necessary to determine injury severity before carrying out corresponding measures.

5.1 Light Blast Injury

Such injuries are mainly light cerebral concussion, light pulmonary hemorrhage, general auditory apparatus injury, cuts and scratches on the surface of the body, among others. Usually this category of victims are the most numerous, accounting for roughly half of all blast injury victims. Due to the lack of obvious internal organ damage or body-wide symptoms, this class of injury does not seriously affect victim's capacity and does not mandate special treatment.

5.2 Moderate Blast Injury

Such injuries are mainly relatively serious cerebral concussion, light pulmonary edema, serious auditory apparatus injury, internal organ intraplaque hemorrhage or patchy hemorrhage or hematoma, and large swaths of soft tissue injury, among others. Clinical symptoms are relatively obvious, usually accompanied by body-wide symptoms. Hemoptysis is common 1–3 days after moderate lung injury, auscultation might discover occasional rale and crepitus, and similar symptoms to other injuries for soft tissue injury and single dislocations. Situations for some victims might worsen because of combination of other injuries or inadequate protection during the evacuation process, but in general there won't be shocks or life-threatening risks. Most victims would eventually recover rather well, and only a small percentage would experience worsened situation due to other injuries.

5.3 Severe Blast Injury

Such injuries are mainly cerebral contusion, relatively serious pulmonary edema or hemorrhage, ruptured or perforated internal organs (i.e., liver, spleen, stomach, intestine, and bladder) and bone fracture (i.e., thigh bone, spine, cranial base and multiple fractured ribs), among others. Cerebral contusion might result in unconsciousness and increase in intracranial pressure. Lung injury might lead to dyspnea and hemoptysis, percussion of the chest may produce dull sounds, and auscultation might discover wide areas of moist rale. Ruptured abdominal organs might result in abdominal pain, abdominal wall tension, pain, pressing pain, rebound tenderness, and other peritoneal irritations. Ruptured liver or spleen might result in serious internal bleeding or shock, while gastrointestinal rupture or perforation might lead to diffuse peritonitis. Bone fractures have similar symptoms as other injuries and should be treated as per other injuries.

5.4 Extremely Severe Blast Injury

Such injuries are mainly multiple severe injuries like severe cerebral and spinal cord injury, chest, abdominal and spine injury, ruptured organ, serious pulmonary edema or hemorrhage, ruptured artery, serious crushing injury to soft tissue and amputation, among others. In addition, such victims might also suffer from serious burns or radiation injury. Victims in this category are mostly located near ground zero, and often die within a short period due to excessive injury. Most deaths early on result from serious cerebral and spinal injuries, ruptured organs resulting from serious hemorrhage (hemorrhagic shock) and multiple fractures (fat embolism). Deaths later on are chiefly attributed to perforation peritonitis, bronchopneumonia, septicemia, and other secondary infections. Serious cerebral injuries and multiple internal organ ruptures have clinical symptoms similar to other injuries and should be treated as general injuries. Most of the extremely severely wounded victims die within a day.

Explosion Physics

Tong Liu

1 Basic Knowledge About Explosives

1.1 Types of Explosives

Explosive is a kind of semi-stable substance that exhibits different levels of chemical reaction (release of heat in the form of deflagration, explosion, and detonation) when initiated by external energy. Substances that are merely explosive but unstable can't be called explosives, they are only considered some sort of explosive substance. In recent years, the general consensus is to use the term "energetic material" to denote materials that exhibit powerful chemical reaction and generate substantial quantity of heat and gas under certain conditions.

Categorized according to the scope of application and based on the reaction initiated, the form of transformation and explosion, and how explosion is manifested, explosives may be classified as primer (primary explosive), high explosive (secondary explosive), gun propellant (gunpowder, booster), and pyrotechnic composition. These substances all fall within the definition of the term energetic material.

Primer is highly sensitive in most cases, and striking with a pin (impact) or spark (fire) can both cause it to explode. Since the time between ignition and detonation is extremely short (10^{-8} to 10^{-6} s), its explosion transformation is usually detonation and primarily functions as a device responsible for initiating the detonation (deflagration) of other explosives. Several of the most common primers include: mercury fulminate $Hg(ONC)_2$, lead azide $Pb(N_3)_2$, trinitroresorcinol $C_6H(NO_2)_3O_2Pb \cdot H_2O$, dinitrodiazophenol or DDNP $C_6H_2(NO_2)_2N_2O$ and tetracene $C_2H_8N_{10}O$, among others.

High explosive is relatively less sensitive and requires a primer's blast wave or high-speed impact from a metallic object (speed ≥1000 m/s) to detonate. The explosion transformation of high explosive is usually detonation. Since low-energy initiation usually isn't enough to cause an explosion, it is relatively safe and convenient to use. However, high explosive does explode, it is very destructive to surrounding medium, which is why it is commonly applied in scenarios that require explosion or brisance. Based on the composition, high explosive may be classified as either single-compound explosive, or multi-compound explosive based on a single-compound explosive. Some of the more common single-compound explosives include: trinitrotoluene or TNT ($C_7H_5N_3O_6$), triamino-trinitrobenzene or TATB ($C_6H_6N_6O_6$), hexanitrostilbene or HNS ($C_{14}H_6N_6O_{12}$), Royal Demolition Explosive or RDX among other names ($C_3H_6N_6O_6$), octogen or HMX ($C_4H_8N_8O_8$), tetryl or CE ($C_7H_5N_5O_8$), pentaerythrite tetranitrate or PETN ($C_5H_8N_4O_{12}$), nitroglycerin or NG ($C_3H_5N_3O_9$) and nitrocellulose explosives or NC ($C_{12}H_{16}N_4O_{18}$), among others. Common multi-compound explosives (if categorized based on main constituent explosive or special additive) include: hexolite (a mixture of TNT and RDX), octol (a mixture of TNT and HMX), ammonal (a mixture of TNT and ammonium nitrate), aluminized explosive (a mixture of high-energy single-compound explosive and powdered aluminum) and polymer-bonded explosives (powdered high-energy explosive as main body, mixed with additives such as polymeric binder), among others.

Propellants react to heat, but are relatively insensitive to initiation from other forms of external energy. The explosion transformation of propellant is usually stable laminar burning, and substantial quantity of high-temperature exhausted gas substances and propulsion forces are created. Propellants include explosives used for producing gas in barrel-launched systems, or rocket propellants that generate propulsion in a rocket engine. Some common propellants include: gunpowder, single-base smokeless powder (nitrocellulose powder), double-base smokeless powder (nitroglycerine powder) and triple-base smokeless powder (Trail Boss powder), among others. Common rocket propellants include: liquid rocket propellants (liquid oxygen/liquid hydrogen, liquid oxygen/kerosene), solid rocket propellants (HTPB, CTPB, etc.), and hybrid solid/liquid propellants (HTPB/liquid oxygen, etc.).

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Based on physical form, explosives may be categorized as solid explosive, liquid explosive, or gas explosive.

Based on application, explosives may be categorized as military explosive or civil/industrial explosive.

1.2 Characteristics of Explosives

The chemical reaction of explosive is a stimulated/initiated reaction (only reacts when needed), and may be categorized as either thermal decomposition, deflagration, or detonation. Thermal decomposition of explosive is similar to normal organic substance. Explosives decompose at an extremely slow rate in room temperature and may be safely stored for an extensive period. However, the deflagration of explosive is unlike the combustion of normal organic substance. Explosives do not need external source of oxygen to burn, and combusts also faster than normal organic substance, the rate of which hinges on environment temperature and pressure. Detonation is a chemical reaction unique to explosives. Detonation is a rapid reaction, and the high temperature and high pressure generated are unrivaled by other forms of chemical reactions.

The exothermicity of reaction, the speed of reaction, and the substantial volume of gases generated are the three major characteristics of explosive. (1) Exothermicity of reaction: For most commonly used high explosives, the heat of explosion generated ranges from 3.71 to 7.53 MJ/kg, while temperature during explosion could reach as high as 3000–5000 °C, and the heat of explosion is symbolic of how an explosive releases its energy and works externally. (2) Speed of reaction: The speed of propagation of detonation could reach several thousand meters per second, and one way to put it is that all the potential energy before the explosion are contained within the explosive, meaning that the released energy density is extremely high. (3) Substantial volume of gases generated: This is equivalent to volume expansion by a thousand times. Before the explosion, these gases are forcefully compressed within a volume about the size of the original explosive at the instant of explosion, which is why the result generated could become high-pressure, high-temperature gas several hundred thousand times the pressure of the atmosphere. The potential energy in an explosive is instantaneously transformed into mechanical energy of the blast, which is how it releases its energy and works externally in a powerfully destructive manner.

Explosives are also characterized by their relative instability and high energy density. Explosives are comprised of oxygen elements such as O and F, and combustible elements like C, H, Si, B, Mg, and Al. The redox reaction between combustible elements and oxygen elements releases heat, and therefore, the performance and explosion process of an explosive are contingent on the proportion of combustible elements and oxygen elements in the explosive. To demonstrate the proportionate relationship between combustible elements and oxygen elements in an explosive, the concept of oxygen balance was formulated. Oxygen balance refers to the surplus or shortage of oxygen, measured in grams, contained within 1 g of the explosive itself needed to completely oxidize all combustible elements within said explosive. Oxygen coefficient A is used to illustrate the oxygen saturation level of explosive molecules:

When $A = 1$, there is just enough oxygen in the explosive to completely oxidize all combustible elements, this is said to be zero oxygen balance and this type of explosive is called zero oxygen balance explosive.

When $A > 1$, there is more than enough oxygen in the explosive to completely oxidize all combustible elements, this is said to be positive oxygen balance and this type of explosive is called positive oxygen balance explosive.

When $A < 1$, there is not enough oxygen in the explosive to completely oxidize all combustible elements, this is said to be negative oxygen balance and this type of explosive is called negative oxygen balance explosive.

When synthesizing or formulating a new explosive, it is necessary to take into consideration oxygen balance.

Generally speaking, the following five parameters are used to determine the overall performance of an explosive: heat of explosion, critical temperature of thermal explosion, volume of explosion gases, explosive velocity/detonation velocity and detonation pressure.

The heat of explosion refers to the amount of heat released by a unit of explosive when it explodes. Since the explosive reaction is extremely fast, usually the constant volume method is employed to determine the heat of explosion. It is expressed as Q_v and measured in kJ/kg. Charge density has a relatively obvious influence on the heat of explosion of negative oxygen balance explosives such as picric acid and tetryl. In order to elevate the heat of explosion, zero oxygen balance is the most ideal. The addition of powdered metals such as powdered aluminum or powdered magnesium will generate a second exothermic reaction and markedly increase the heat of explosion.

Critical temperature of thermal explosion is the highest temperature that the product of explosion reaches because of the heat of explosion. It is expressed as T_B and measured in K. In equation $T_B = T_0 + \frac{Q_v}{\overline{c_v}}$, T_0 denotes the initial temperature of the explosive, and has a value of 298 K; $\overline{C_v}$ denotes the average specific heat capacity of molecule of product of explosion. Clearly, adjusting oxygen balance and adding powdered metal that can generate high heat into the explo-

sive can raise the critical temperature of thermal explosion. However, if the measure adopted results in an increase in Q_v that is less than the increase in $\overline{c_v}$, the desired outcome would not be achieved.

Volume of explosion gases refers to the volume of gaseous products generated by the explosive reaction of 1 kg of explosive under standard conditions (0 °C and 100 kPa). It is expressed as V_0 and measured in L/kg. Volume of explosion gases reflects the efficiency at which the heat from an explosive reaction converts into mechanical force.

Explosive velocity or detonation velocity is the velocity at which the shock wave front propagates through a detonated explosive, and is expressed as D_{CJ}. If the explosive diameter is much larger than the critical diameter, and charge density has reached the highest theoretical density, then the detonation velocity would only be affected by the explosive's chemical composition and structure, and not contingent on external conditions. This would be called ideal detonation velocity. In actual conditions, the charge density, charge diameter, particle size of explosive, constraints of the charge, and other aspects all have an impact on detonation velocity.

Detonation pressure is the peak dynamic pressure of the shock wave fronts of shock waves, as in the pressure of the CJ plane of the detonation. It is expressed as P_{CJ}. Experience shows that in $P_{CJ} = \frac{1}{4}\rho_e D_{CJ}^2$, when ρ_e represents charge density, for some explosives there is a linear relationship between P_{CJ} and D_{CJ}: $P_{CJ} = 93.3D_{CJ} - 456$.

1.3 Application of Explosives

Today, explosives have found widespread applications in many areas from national defense and arms industry to various aspects of the economy, and play a crucial role in promoting advancements in civilization and society. Although explosives have a large number of military applications, from the perspective of scholars and technicians engaged in the research and production of explosives, they hope that their achievements could serve the progress of modern civilization and economic development. The Nobel Peace Prize, conceived by founder of the Nobel Prize and inventor of dynamite Alfred Nobel, epitomizes this belief. In terms of the scope of application, explosives may be classified as either military usage, civil usage, or military-civil usage.

1.3.1 Military Usage

Due to the complications in the purpose of usage and environment of battlefield, usually explosives for military usage have very high requirements in terms of performance and safety. Different ammunitions with different purposes have different charge requirements. For examples: The jet penetration capacity crucial to anti-armor shaped charges is proportionate to the explosive's detonation pressure, and therefore requires explosives with high detonation pressure; anti-ship missile warheads require penetration of the hull armor of vessel before detonation, and therefore require explosives that are not only powerful but also have specific demands in terms of sensitivities to impact and shock wave; bunker buster warheads are designed to penetrated targets buried deep underground, and therefore have even more stringent demands in terms of sensitivities to impact and shock wave; weapons used for underwater detonations usually desire more powerful shock wave and gas bubble, thus aluminized explosives with high heat of explosion and detonation velocity are chosen; blast and fragmentation warheads have to account for damage created by both blast and fragmentation, and therefore the explosives used commonly have to possess high charge density, high heat of explosion and detonation velocity; for explosives used in nuclear weapons, safety is of utmost importance, and must be extremely retarded against heat, mechanical force, and shock wave, with TATB-based polymer-bonded explosive (PBX) being the most frequent option.

1.3.2 Civil Usage

The quantity of explosives deployed for civil usage in China is enormous, upwards of millions of tons and still growing at a rapid pace. Civil or industrial explosives include emulsion explosives, powder emulsion explosives, expanded nitramine explosive and modified AN/FO, among other choices. Industrial explosives are often used in mining and construction, with the majority of around 80% deployed in the mining of coal, metals, and non-metal resources. Coal mines have to guard against gas explosion, and usually emulsion explosives or colloidal nitroglycerine explosives with good water-resistance, low heat of explosion, and shortened explosion duration are optimal; metal mining often takes place at sites with hard rocks, and therefore cheap and powerful explosives are desirable; for civil engineering, it is necessary to minimize impacts on surrounding people and structures, which is why non-explosive demolition agents are frequently used; moreover, explosives have also been widely applied to assist in oil and gas well drilling and to create seismic waves for investigation about the Earth's structure and interior. China is administering increasingly strict requirements in the safety, reliability, and environment friendliness of industrial explosives. Effective from June 30th, 2018, China has banned the production, sales and usage of blasting fuse, flash detonator, and explosives contained ammonium nitrate and trinitrotoluene.

1.3.3 Military-Civil Usage

Many applications of explosives can be used in both the military and civilian sector. The propellants and initiator

explosive for rockets could be military or civil, depending on whether the payload is intended for a military purpose or for peaceful purpose. The processing of explosives (synthesize, compress and connect, cut, form, etc.), emergency escape and ejection system, airbag and other applications may also fall under either military or civil usage depending on the intended user.

2 Basic Knowledge About Detonation

2.1 The Detonation Process

In the broad sense, explosion includes physical explosion, chemical explosion, and nuclear explosion. Characteristics of an explosion: A tremendous amount of energy is rapidly released or converted from a limited volume, leading to a jump and abrupt rise in the pressure and temperature of the medium surrounding the center of explosion. This kind of abrupt jump in pressure is the fundamental force behind the destructive power of an explosion. The detonation pressure of vapor cloud or dust explosion is usually measured in the MPa range and temperature around (3–5) × 10^5 K; for condensed explosives, detonation pressure is usually measured in the GPa range and temperature around (3–5) × 10^5 K; within a nuclear blast zone, detonation pressure is usually measured in the 1000 TPa range and temperature around 10^7 K; this book mainly focuses on the chemical explosion of explosives. The detonation process of an explosive is an extremely complicated process of chemical reaction accompanied by the rapid and violent release of energy, but may be roughly divided into two phases: In the first phase, the internal energy of the explosive is being released rapidly, and the high-temp, high-pressure detonation product starts to forcefully compress the medium in the surrounding, and this phase could be deemed a stage in which the internal energy of the explosive converts into the compressed energy of the detonation product; and in the second phase, the detonation product propagates through the surrounding medium in the form of blast wave, and this phase may be considered a stage in which the compressed energy is released, and expands and works externally. Since the propagation of detonation is the propagation of blast wave, it is therefore mandatory to first gain an understanding about the basics of shock waves and blast waves.

2.2 Basics About Shock Waves and Blast Waves

2.2.1 Basics About Shock Waves

A medium turns into a wave when disturbed, or to put it differently, a wave is the propagation of the disturbance in the medium. Take for example piston motion in a one-dimensional pipe filled with gas. When the piston moves with increasing speed and compresses the gas, it creates disturbances in the gas in the tube that are increasingly faster and stronger. Subsequent disturbances catch up to and add to previous disturbances, ultimately leading to abrupt jumps in the parameters of the state of the medium (pressure P, density ρ, and temperature T) and particle velocity u, creating a strong discontinuity surface. The strong discontinuity surface of a shock wave propagates through the medium, and this strong discontinuity surface causes the parameters of the state and particle velocity of the medium on the two sides to change abruptly, as in:

$$\left.\frac{\partial P}{\partial x}\right|_A = \infty, \quad \left.\frac{\partial \rho}{\partial x}\right|_A = \infty, \quad \left.\frac{\partial T}{\partial x}\right|_A = \infty, \quad \left.\frac{\partial u}{\partial x}\right|_A = \infty$$

This strong discontinuity surface is the shock wave front. Its identifying property is the abrupt changes in the region of the front, and abrupt changes in the parameters of the state of the medium at the wave front are highly destructive.

Shock waves propagate through medium but can also propagate as a field (i.e., electromagnetic field).

1. **The relationship between the physical quantities upstream and downstream of the shock wave front**. Assuming that the front of the shock wave is a plane, since the shock wave propagates extremely quickly, we can ignore the viscosity and heat transfer of the medium, or in other words, we can consider this propagation process an adiabatic process. The symbol D denotes the speed of propagation of the shock wave in the medium, subscript "0" represents the physical quantities upstream (in front) of the shock wave front, while subscript "H" indicates physical quantities downstream (behind) of the shock wave front (Fig. 1). For the sake of research convenience, coordinates are set on the shock wave front, as in

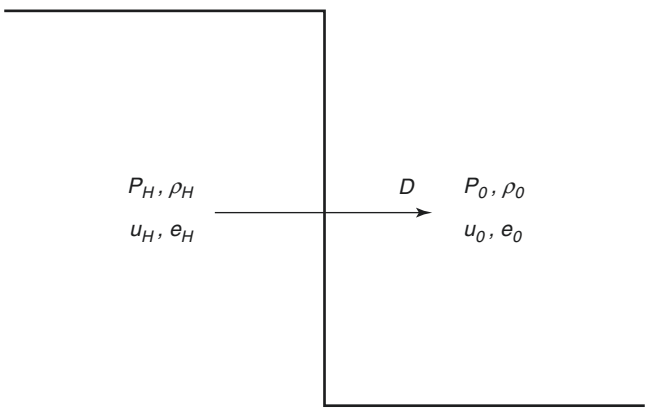

Fig. 1 Relationship of physical quantities on the two sides of shock wave front

the use of stationary relative coordinates on the shock wave front. In this coordinates system, the speed of the incoming shock wave is $D - u_0$, while the speed of the outgoing shock wave is $(D - u_H)$, meaning that the physical quantities upstream and downstream of the shock wave front obey the conservation laws of mass, momentum, and energy. Assuming that $u_0 = 0$, then the relationship may be expressed as:

Equation of mass conservation:

$$\rho_H (D - u_H) = \rho_0 D \tag{1}$$

Equation of momentum conservation:

$$P_H - P_0 = \rho_0 D u_H \tag{2}$$

From Eqs. (1) and (2), particle velocity u_H and shock wave velocity D may be respectively obtained:

$$u_H = (v_0 - v_H)\sqrt{\frac{P_H - P_0}{v_0 - v_H}} \tag{3}$$

$$D = v_0 \left(\frac{P_H - P_0}{v_0 - v_H}\right)^{1/2} \tag{4}$$

Equation of energy conservation:

$$P_H u_H = \rho_0 D\left[(e_H - e_0) + \frac{1}{2}u_H^2\right] \tag{5}$$

In the equation: ρ is density, u is particle velocity, P is pressure, and e is internal energy.

In combination with outcomes from the two aforesaid conservation laws, specific energy increases caused by compression of the shock wave or the Hugoniot energy equation may be derived:

$$e_H - e_0 = \frac{1}{2}(P_H + P_0)(v_0 - v_H) \tag{6}$$

In the equation, $v = 1/\rho$. The three conservation equations include the five parameters of P_H, v_H, e_H, D, and u_H5. To close the system of equations, two more equations are needed, with one being that of the state of the material. In order to avoid adding new variables, only equation related to the thermodynamics of the quantities of the state of the materials P, v, and e is used. It is expressed as $P = f(e, v)$.

For ideal gas, the formula for its state is:

$$P = \rho RT \tag{7}$$

The other equation needed is the adiabatic relationship of the shock. For an ideal gas that satisfies polytropic adiabatic process $Pv^{\gamma} = \text{constant}$, the internal energy function may be expressed as:

$$e = \frac{Pv}{\gamma - 1} \tag{8}$$

In the equation: γ is the polytropic index, $\gamma = \frac{c_P}{c_v}$, equation transformation (6) can obtain the polytropic gas shock adiabatic line, or the Hugoniot adiabatic line:

$$\frac{P_H}{P_0} = \frac{\frac{\gamma_H + 1}{\gamma_H - 1}\frac{\rho_H}{\rho_0} - 1}{\frac{\gamma_0 + 1}{\gamma_0 - 1} - \frac{\rho_H}{\rho_0}} \tag{9}$$

For a solid material, the Gruneisen equation of state is used:

$$P - P_K(v) = \frac{r(v)}{v}(e - e_K) \tag{10}$$

In the equation: r is the Gruneisen parameter, and the subscript "K" represents absolute zero.

Shock adiabatic relationship of solid material $P_H = f(v_H)$ was obtained through experimentation.

Equation for isentropic state is also a common equation of state.

$$P = A(S)\rho^n + B \tag{11}$$

For a specific material, A, B, and n are constants. Thus, in the isentropic state equation, the material's pressure P is only associated with density ρ.

2. **Rayleigh line, Hugoniot curve, and isentropic line** may be obtained from equation transformation (4)

$$P - P_0 = -\frac{D^2}{v_0^2}(v_1 - v_0) \tag{12}$$

This is expressed as a straight line with slope $-\frac{D^2}{v_0}$ that passes by the points (P_0, v_0) on the plane (P, v). This is called the Rayleigh line or velocity equation of the waves. D will show straight lines with different slopes, as in the higher the shock wave velocity D, the steeper the straight line (Fig. 2).

As mentioned above, different material states of different medium have different equations, and therefore the Hugoniot adiabatic line also differs. $\frac{dP}{dv} < 0$ and $\frac{d^2P}{dv^2} > 0$ can be proven, showing how the Hugoniot adiabatic line is a concave curve on the plane (P, v), which is why the line is called the Hugoniot curve. For the same medium, the Hugoniot

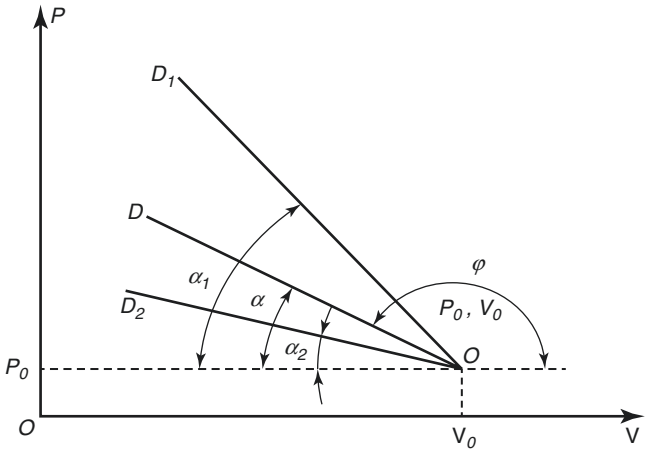

Fig. 2 Velocity line of shock wave

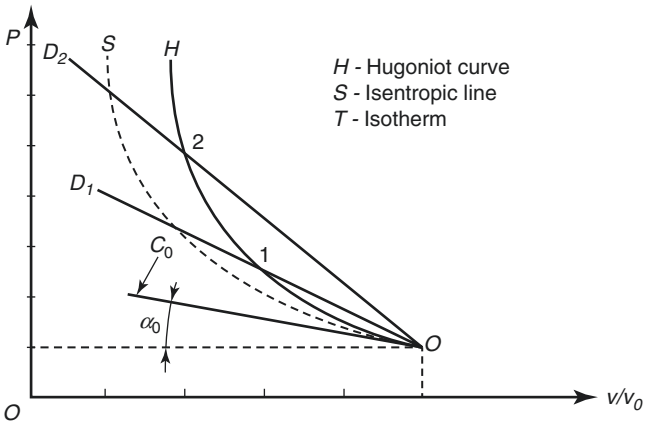

Fig. 3 Rayleigh line, Hugoniot curve, and isentropic line of shock wave

curve reflects the total of downstream states (P_H, v_H) of the shock wave for the corresponding upstream state (P_0, v_0). Therefore, the Hugoniot curve is not a line of process.

Equation (6) should be modified as:

$$H(P,v) = e_H - e_0 + \frac{1}{2}(P_H + P_0)(v_H - v_0) \tag{13}$$

In combination with the first law of thermodynamics, it can be proven that at point (P_0, v_0), $\left.\frac{dS}{dv}\right|_0 = 0$, $\left.\frac{d^2S}{dv^2}\right|_0 = 0$ meanwhile $\left.\frac{d^3S}{dv^3}\right|_0 > 0$. In the equation, S represents entropy, indicating that upstream and downstream of the shock wave, the increase in entropy (S) takes place in third order small quantities, and the isentropic state equation (11) may be applied.

The isentropic state equation (11) mirrors the course of changes in the isentropic state, and this is called the isentropic line. $\frac{dP}{dv} < 0$, $\frac{d^2P}{dv^2} > 0$ can also be proven, indicating that the isentropic line on plane (P, v) is also a concave curve. The isentropic line is a process line that reflects changes in the state, and for the same upstream state (P_0, v_0), different entropy (S) value corresponds to different isentropic line (Fig. 3).

3. **Relationship between Hugoniot curve, isentropic line, and isotherm line.** Thermodynamics theories prove $\left[\frac{dP_H}{dv} - \frac{dP_S}{dv}\right]_0 = 0$, $\left[\frac{d^2P_H}{dv^2} - \frac{d^2P_S}{dv^2}\right]_0 = 0$, and $\left[\frac{d^3P_H}{dv^3} - \frac{d^3P_S}{dv^3}\right]_0 \neq 0$ at point (P_0, v_0) on the $P - v$ plane, demonstrating that Hugoniot curve and isentropic line from the same initiate state [point (P_0, v_0)] are tangent at second order. Due to the rise in entropy during the shock's compression, the A in the corresponding Eq. (11) increases, and when specific volume is the same, the pressure of Hugoniot curve is higher than that of the isentropic line, which is why the Hugoniot curve is located above the isentropic line. Meanwhile, the temperature of the isentropic line increases but that of the isotherm line remains the same. The work along the isentropic line is higher than that of the isotherm line, which is also why the isentropic line is located above the isotherm line. Theoretically speaking, under low pressure conditions, the Hugoniot shock adiabatic line and the isentropic line are very close, and during experimentation, due to some uncertainties in measurement, the Hugoniot shock adiabatic line and the isentropic line were difficult to distinguish under the pressure of 20 GPa as measured in actual experiment (Fig. 4).
4. **Relationship between shock wave velocity D and particle velocity downstream of the wave u_H.** Theoretical analysis and empirical experiment show that shock wave velocity D and particle velocity downstream of the wave u_H share a linear relationship under a rather broad spectrum of pressure in many different materials.

$$D = c_0 + \lambda u_H \tag{14}$$

In the equation, c_0 and λ are constants. Table 1 shows the ρ_0, c_0, and λ values of several common materials.

5. **Shock wave front structure**. When inferring the shock wave front upstream and downstream physical qualities in the above segment, the viscosity and heat transfer of the medium were ignored, holding that the parameters of states and parameters of motions on the front of the shock wave do not exhibit tiered jumps with slope, and considering the front of the shock wave as a plane with abrupt jump in pressure (Fig. 5). However, shock waves in the

real world do not behave as such, and parameters of states and parameters of motions on the front of the shock wave do actually exhibit tiered jumps due to the influences of the viscosity (internal friction) and heat transfer of the medium, it's just that the tiers are extremely steep. Therefore, shock wave fronts in the real world are not perfect planes but possess a narrow transition zone with width d (Fig. 6). Using system of molecular dynamics equations that take into consideration heat transfer and viscosity, in tandem with measurements from actual experimentation, it can be shown that the width d of shock wave front and mean free path γ of molecules upstream of the shock wave exist on the same order of magnitude, demonstrating that the transition zone is very narrow (roughly several γ). The patterns of changes of the various physical qualities upstream and downstream of the shock wave front also differ. Figure 7 reflects the distribution of electron temperature T_0, ion temperature T_H, and density ρ upstream and downstream of shock wave fronts in the real world.

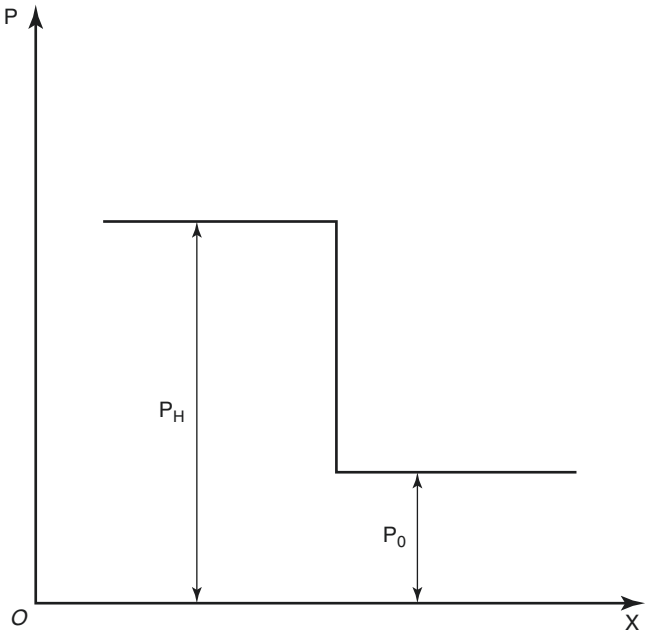

Fig. 5 Pressure jump in ideal shock wave

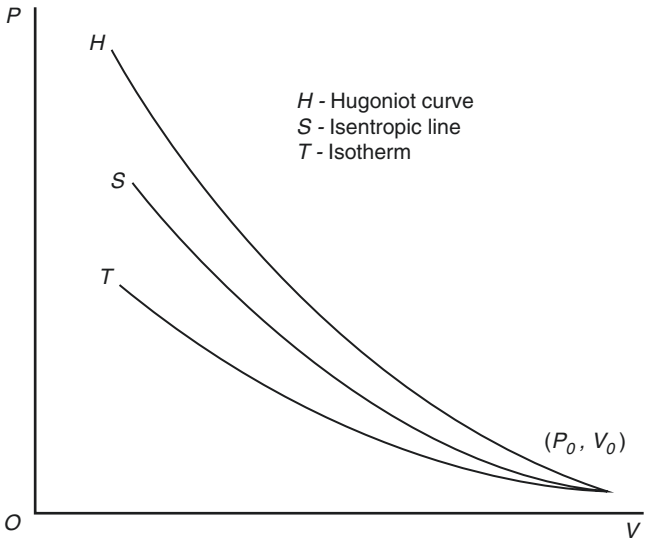

Fig. 4 Hugoniot curve, isentropic line, and isotherm

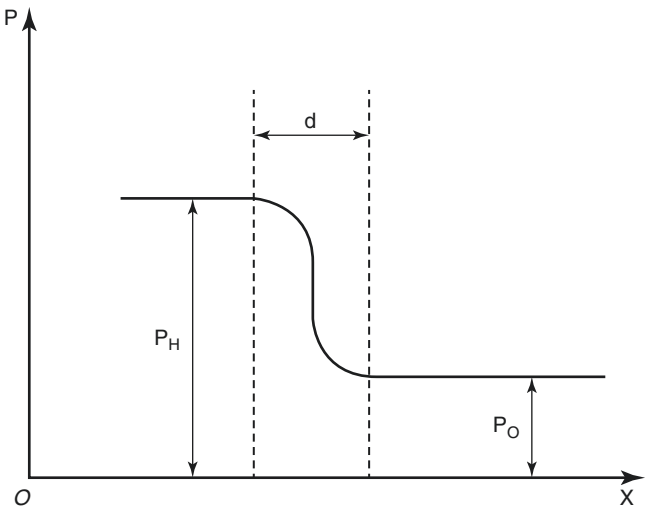

Fig. 6 Pressure jump in real shock wave

Table 1 ρ_0, c_0, and λ values of several common materials

Materials	ρ_0/(g · cm^{-3})	c_0/(mm · μs^{-1})	λ	Applicable range 10^4 ba
Organic glass	1.19	3.16	1.25	6–37
Al	2.79	5.44	1.34	22–180
Cu	8.466	3.94	1.47	50–270
W	19.2	4.049	1.215	30–450

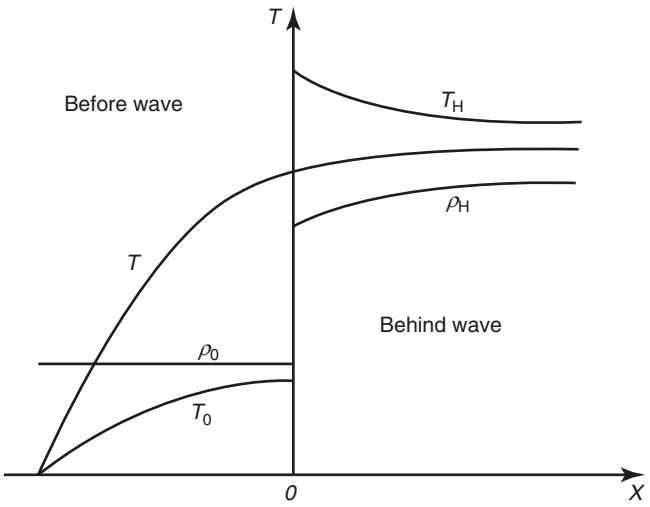

Fig. 7 Temperature and density distribution of shock wave

The purpose of expounding the structure of shock wave front is to explain that the relationships between physical qualities upstream and downstream of the shock wave front established in the above segment are only applicable to the state of medium upstream and downstream of the shock wave front. To study about the changes in states of medium in the transition zone, it is necessary to also take into full account the influences of the viscosity and heat transfer.

6. **Relationship between shock wave front velocity and sound velocity**. The velocity of sound is the speed of propagation of micro disturbances. The process of propagation of the velocity of sound is an isentropic process, and in a polytropic gas, sound velocity may be expressed as:

$$c = \sqrt{kRT} \tag{15}$$

In the equation, k denotes the polytropic index of the isentropic equation of the polytropic gas. By applying (15) to the relational expression for physical quantities upstream and downstream of shock wave front, it can be demonstrated that relative to undisturbed medium (upstream of wave):

$$D > u_0 + c_0 \tag{16}$$

The front of the shock wave moves at supersonic velocity, and the front can catch up to any disturbances propagating ahead. After a shock wave propagates through a medium, the medium obtains the same velocity that moves in the same direction as the propagation of the wave, as in $u - u_0 > 0$, and relative to the disturbed medium (downstream of wave):

$$D < u_H + c_H \tag{17}$$

The front of the shock wave moves at subsonic velocity, and any disturbance behind the shock wave front could catch up to the front and change its power.

2.2.2 Basics About Detonation Wave

1. **The Chapman-Jouguet theory about detonation wave**. Results from studies about disastrous gas explosions in coal mines between the end of the nineteenth century and the start of the twentieth century formed the foundation for classical theory of detonation wave fluid dynamics. In order to explain why different ignition conditions in experiment resulted in massive differences in the velocity of the propagation of flame inside channels filled with flammable gas that ranged from several meters per second to several thousand meters per second, Chapman (in 1899) and Jouguet (in 1905) respectively put forth the notion to simplify the detonation process into a one-dimensional propagation of strong discontinuity surface that includes chemical reaction, and they referred to this strong discontinuity surface as the detonation wave. The Chapman-Jouguet theory, or the C-J theory, is a fluid dynamics theory about detonation wave that simplifies the detonation wave into a strong discontinuity surface that includes chemical reaction. The C-J theory holds that detonation occurs on an infinitely thin shock wave front at an instant, it is unnecessary to account for chemical reaction process, the laws of conservation are still satisfied upstream and downstream of the wave front, effects of chemical reaction are summarized as an external and added energy, which is reflected in the fluid dynamics energy equation as the thermal effect at the termination state of the reaction. Thus, a detonation wave is a powerful shock wave that has a chemical reaction zone and that propagates as supersonic velocity.
   (a) **Basic relational expression of detonation wave:** The C-J theory simplifies the detonation wave into a strong discontinuity surface that includes chemical reaction, and may be understood as a kind of strong shock wave that propagates within the explosive medium. The mass conservation and momentum conservation relationships in physical quantities upstream and downstream of shock wave front are also applicable to detonation wave, but the difference is that the powerful impact and compression generate high temperature and high pressure, which in turn cause chemical changes in the explosive medium. The energy released in chemical reaction maintains and guides the shock wave's self-sustaining propagation inside the explosive. In terms of the relationship of conservation of energy, it is necessary to consider the heat of reaction in the products of the chemical reaction (detonation products).

Similar to the analysis method for the front of shock wave, the origin of coordinates is set on the front of detonation wave, which means using stationary relative coordinates established on the detonation wave front (Fig. 8). With the propagation velocity of detonation wave established as D, in this coordinates system the velocity of incoming detonation wave is D, while the velocity of the outgoing detonation wave is $(D-u_{\mathrm{H}})$, meaning that the physical quantities upstream and downstream of the shock wave front obey the conservation laws of mass, momentum, and energy.

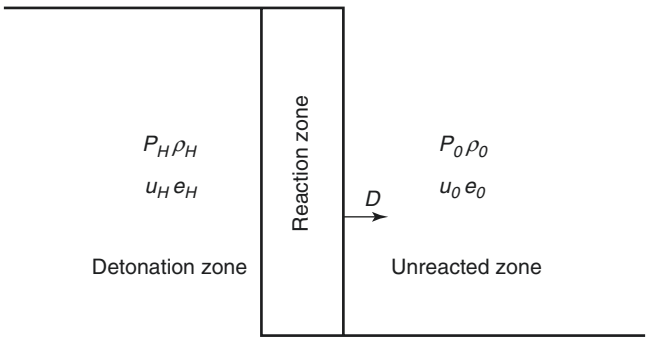

Fig. 8 C-J detonation model

$$\rho_{\mathrm{H}}\left(D-u_{\mathrm{H}}\right)=\rho_0 D \tag{18}$$

$$\rho_{\mathrm{H}}\left(D-u_{\mathrm{H}}\right)^2+P_{\mathrm{H}}=\rho_0 D^2+P_0 \tag{19}$$

$$\left(\frac{P_{\mathrm{H}}}{\gamma-1}+\frac{1}{2}\rho_{\mathrm{H}}\left(D-u_{\mathrm{H}}\right)^2-Q\rho_{\mathrm{H}}+P_{\mathrm{H}}\right)\left(D-u_{\mathrm{H}}\right)=\left(\frac{P_0}{\gamma-1}+\frac{1}{2}\rho_0 D^2+P_0\right)D \tag{20}$$

$$u_{\mathrm{H}}=\left(v_0-v_{\mathrm{H}}\right)\sqrt{\frac{P_{\mathrm{H}}-P_0}{v_0-v_{\mathrm{H}}}} \tag{21}$$

Then remove u_{H} in the equation of mass conservation and energy conservation equation to obtain the Rayleigh line:

$$\rho_0^2 D^2-\left(P_{\mathrm{H}}-P_0\right)\left(\frac{1}{\rho_0}-\frac{1}{\rho_{\mathrm{H}}}\right)^{-1}=0 \tag{22}$$

Since $\rho = 1/v$ and v is specific volume, the above equation may be rewritten as

$$P_{\mathrm{H}}=P_0+\frac{v_0-v_{\mathrm{H}}}{v_0^2}D^2 \tag{23}$$

$$\text{Or rewritten as}: \quad D=v_0\left(\frac{P_{\mathrm{H}}-P_0}{v_0-v_{\mathrm{H}}}\right)^{1/2} \tag{24}$$

The Rayleigh line of detonation wave does not include energy, and its nature is identical to the Rayleigh line of shock wave. Based on the constancy hypothesis of the detonation process, D is constant, Eq. (23) is expressed as a straight line with slope $-\frac{D^2}{v_0}$ that passes by the points (P_0, v_0) on the plane (P, v). This is called the detonation wave's Rayleigh line or velocity equation of the waves. For the same state upstream of the wave (P_0, v_0), different shock wave velocities D will show straight lines with different slopes, as in the higher the shock wave velocity D, the steeper the straight line. When $D = 0$ the straight line is horizontal, and when $D = \infty$ the straight line is vertical, as in an instantaneous explosion.

When u_{H} and D are removed from Eq. (20), and when the other two conservation conditions are applied, then the Hugoniot adiabatic line of the detonation wave may be obtained:

$$e_{\mathrm{H}}\left(P_{\mathrm{H}},v_{\mathrm{H}}\right)-e_0\left(P_0,v_0\right)=\frac{1}{2}\left(P_{\mathrm{H}}+P_0\right)\left(v_0-v_{\mathrm{H}}\right)+Q \tag{25}$$

In the equation, e_{H} is the specific internal energy of the products downstream of the reaction zone; e_0 is the specific internal energy of the explosive; and Q is the heat released by the explosive of a unit mass, equating to the specific heat Q_{Pv} released by chemical reaction under constant pressure ($P = P_0$) and constant volume ($v = v_0$).

(b) **Hugoniot curve of detonation wave**: The Hugoniot adiabatic line of the detonation wave is a concave line on the plane (P, v), and is also known as the Hugoniot curve of the detonation wave. For the same medium, Hugoniot curve of a detonation wave reflects the total of downstream states (P_{H}, v_{H}) under the effects of the detonation wave for the corresponding upstream state (P_0, v_0).

Although the Hugoniot curve of a detonation wave and the Hugoniot curve of a shock wave look similar, they are completely different in terms of physical significance. The Hugoniot curves of shock

waves always begin at (P_0, v_0), but the right side of Eq. (25) includes the release of chemical energy, and the Hugoniot curve of a detonation wave expresses the curve of the state of energy increase in the detonation products. Therefore, it is located above the Hugoniot curve of shock wave, which does not possess chemical reaction, and does not necessarily pass through the point (P_0, v_0). Since the impact compression provides an activation energy that can initiate a reaction, when the explosive medium in a non-reaction layer is impacted and compressed by detonation wave from the layer above, it would be activated from its initiation state at point A (P_0, v_0) to point C (P_1, v_1) in the intermediate state of the shock wave Hugoniot curve, then undergoes chemical reaction along the Rayleigh line of the detonation wave. Heat of reaction is released (heat of explosion Q), and the chemical reaction final state point B (P_2, v_2) of this layer of explosive is the tangent point between the Hugoniot curve of the detonation wave and the Rayleigh line of the detonation wave (Fig. 9). When the chemical reaction of this layer of explosive finishes, it would activate chemical reaction in the next layer of explosive. Therefore, energy released in chemical reaction maintains and guides the shock wave's self-sustaining propagation inside the explosive.

Next, let us discuss about the physical significance of the various branches of the Hugoniot curve of detonation wave. In accordance with the previous analysis, the Hugoniot curve of a detonation wave reflects the total of downstream states (P_H, v_H) under the effects of the detonation wave for the corresponding upstream state (P_0, v_0). Upstream states (P_0, v_0) are graphed as vertical line and horizontal line, and they respectively cross the Hugoniot curve at the points A and B (Fig. 10). Then graph two straight lines from point (P_0, v_0) that are tangent to the Hugoniot curve respectively at the points C and D.

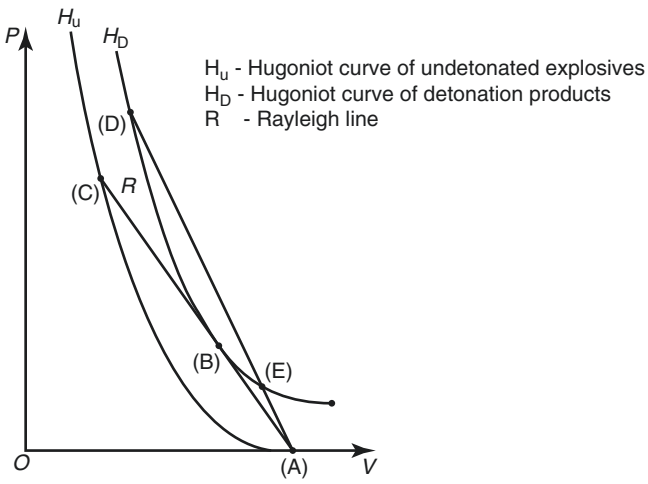

Fig. 9 Hugoniot curve of detonation wave. (A) Initiate state of unreacted explosive; (B) state of reaction product of explosion; (C) jump conditions of impact and compression that did not initiate explosive reaction; (D) Hugoniot state of product; and (E) Hugoniot state of product when pressure decreases and volume increases

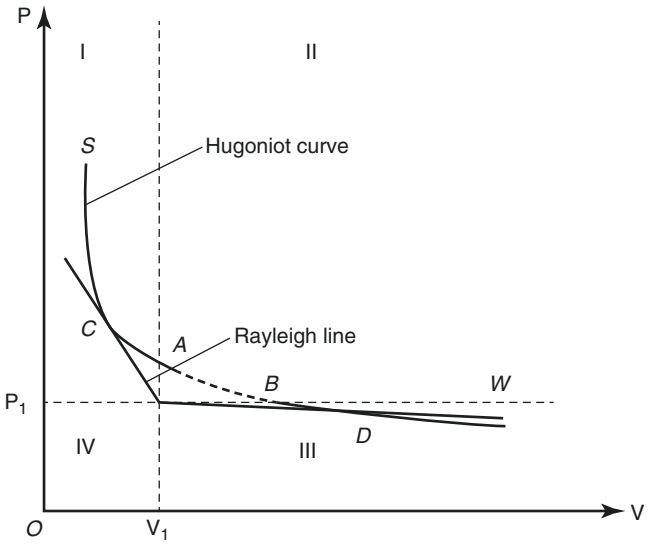

Fig. 10 Hugoniot curve branches of detonation wave

At point A, with $v_H = v_0$ and Eq. (24), it can be known that $D \to \infty$ corresponds to specific volume detonation.

At point B, with $P_H = P_0$ and Eq. (24), it can be known that $D = 0$ corresponds to specific pressure combustion.

At point C, with $\left(\frac{dP}{dv}\right)_H = \left(\frac{dP}{dv}\right)_S = \left(\frac{P_H - P_0}{v_H - v_0}\right)_R$, the subscripts "H," "S," and "R" respectively denote the Hugoniot curve, isentropic curve, and Rayleigh line. Point C is the point of common tangent between the three lines, and since it corresponds to C-J detonation, it is generally referred to as the C-J detonation point.

For area $P_H > P_0$, $v_H < v_0$ above point C of detonation wave Hugoniot curve, with (21) and (24) it can be known that $D > 0$, $u > 0$, direction of motion of products is identical to the direction of propagation of detonation wave, and it is a detonation state known as detonation branch. Detonation branch may be sub-divided into strong detonation branch and weak detonation branch. At the segment CS, $P_H > P_{CJ}$ is called strong detonation branch, and at segment CA, $P_H > P_{CJ}$ is called weak detonation branch.

Point D, $\left(\frac{\mathrm{d}P}{\mathrm{d}v}\right)_{\mathrm{H}} = \left(\frac{P_{\mathrm{H}} - P_0}{v_{\mathrm{H}} - v_0}\right)_{\mathrm{R}}$ corresponds to C-J combustion point.

For area $P_{\mathrm{H}} < P_0$, $v_{\mathrm{H}} > v_0$ above point B of detonation wave Hugoniot curve, with (21) and (24) it can be known that $D > 0$, $u < 0$, direction of motion of products is opposite to the direction of propagation of detonation wave, and it is a combustion state known as combustion branch. The combustion branch too is divided into the strong combustion branch and the weak combustion branch. At the segment DW, $v_{\mathrm{H}} > v_{\mathrm{CJ}}$ is called strong combustion branch, and at segment DB, $v_{\mathrm{H}} < v_{\mathrm{CJ}}$ is called weak combustion branch.

At segment AB, $P_{\mathrm{H}} > P_0$, $v_{\mathrm{H}} < v_0$, with (21) and (24) it can be known that D and u are imaginary numbers, and do not correspond to any actual constant process (Fig. 11).

(c) **Conditions for steady propagation of detonation wave**: Detonation branch is sub-divided into strong detonation branch and weak detonation branch, and the main difference between the two is the velocity of propagation. From O (P_0, v_0), mark three Rayleigh lines with detonation velocities D_{W}, D_{CJ}, and D_{S}, and these three Rayleigh lines respectively does not intersect, is tangent to, and intersects with the Hugoniot curve (Fig. 11). The corresponding relationships are as below:

- In case of detonation velocity $D_{\mathrm{W}} < D_{\mathrm{CJ}}$, there are no intersection point and no solution.
- In case of $D_{\mathrm{S}} > D_{\mathrm{CJ}}$, there are two intersection points and two solutions:
- Given a "strong" solution S: $u_{\mathrm{H}} + c_{\mathrm{H}} > D$, detonation wave velocity is subsonic compared to the medium downstream, and this is called strong detonation.
- A "weak" solution W: Given $u_{\mathrm{H}} + c_{\mathrm{H}} < D$, detonation wave velocity is supersonic compared to the medium downstream, and this is called weak detonation.
- This means that two different chemical reaction states in the explosive can achieve a uniform detonation velocity, which does not comply with physics principles.
- In case of detonation velocity D_{CJ}, there is only one point of tangent, and the only solution C: $u_{\mathrm{H}} + c_{\mathrm{H}} = D$. In other words, the flow of products downstream of the shock wave is equal to the local sonic velocity, and usually that point is called the C-J point. Following the Hugoniot curve of the detonation products, entropy reaches its smallest value at C-J point, as in $\mathrm{d}S = 0$ and also $u_{\mathrm{H}} + c_{\mathrm{H}} = D$. This reflects the state of the constant detonation wave and is called the C-J condition.
- Strong detonation is actually a kind of overcompressed detonation state. Due to $u_{\mathrm{H}} + c_{\mathrm{H}} > D$, the rarefaction wave downstream of the detonation wave will catch up to the wave front, causing unsteady motion in the wave front. Detonation at this phase is unsteady, until $u_{\mathrm{H}} + c_{\mathrm{H}} = D$, when a steady state could be maintained downstream of the wave front. Weak detonation is actually a kind of undercompressed detonation state. Due to $u_{\mathrm{H}} + c_{\mathrm{H}} < D$, the disturbance downstream of the detonation wave cannot catch up to the wave front, and the shock wave ahead won't receive continual energy supply. The strength and velocity of the detonation wave will continue to weaken, and this is an unsteady state of detonation propagation. Only the C-J point can maintain a steady state downstream of the wave front, and this is the C-J condition that is a requisite for steady detonation propagation.

(d) **Calculation of detonation wave parameters**: The six physical quantities that describe the explosive's detonation process are P_{H}, ρ_{H}, T_{H}, e_{H}, u_{H}, and D, and they are calculated using the following six basic equations:

$$e_{\mathrm{H}} - e_0 = \frac{1}{2}\left(P_{\mathrm{H}} + P_0\right)\left(v_0 - v_{\mathrm{H}}\right) + Q \tag{26}$$

$$D = v_0\sqrt{\frac{P_{\mathrm{H}} - P_0}{v_0 - v_{\mathrm{H}}}} \tag{27}$$

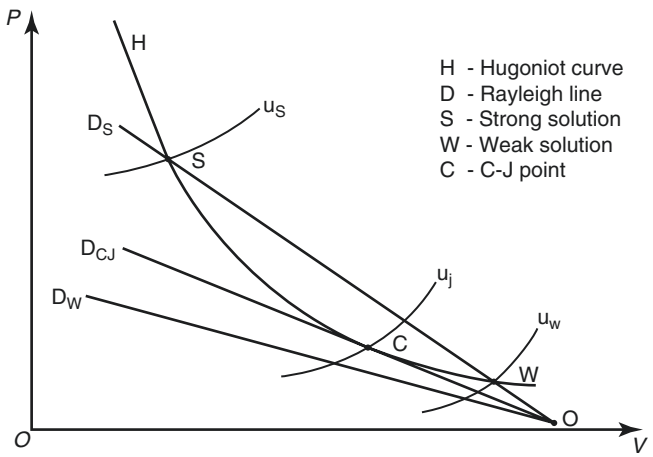

Fig. 11 C-J conditions of detonation wave propagation

$$u_{\mathrm{H}} = \left(v_0 - v_{\mathrm{H}}\right)\sqrt{\frac{P_{\mathrm{H}} - P_0}{v_0 - v_{\mathrm{H}}}} \tag{28}$$

$$\frac{P_{\mathrm{H}} - P_0}{v_0 - v_{\mathrm{H}}} = \left(-\frac{\mathrm{d}P}{\mathrm{d}v}\right)_{\mathrm{S}} = \frac{kP_{\mathrm{H}}}{v_{\mathrm{H}}} \tag{29}$$

$$P_{\mathrm{H}} = F\left(\rho_{\mathrm{H}}, e_{\mathrm{H}}\right) \tag{30}$$

$$P_{\mathrm{H}} = f\left(\rho_{\mathrm{H}}, T_{\mathrm{H}}\right) \tag{31}$$

The last two equations are state equations for the detonation products, and depend on the medium through which the detonation wave propagates. For example, when a detonation wave propagates through a mixture of ideal gases, then the ideal gas state formula may be used:

$$P_{\mathrm{H}} = \rho_{\mathrm{H}} R T_{\mathrm{H}} \tag{32}$$

Due to $R = C_{\mathrm{v}}(\gamma - 1)$, the above equation should be rewritten as:

$$P_{\mathrm{H}} = \rho_{\mathrm{H}} T_{\mathrm{H}} C_{\mathrm{v}}\left(\gamma - 1\right) \tag{33}$$

Meanwhile, due to the isentropic line equation $Pv^{\gamma} =$ constant for product, thus $e = Pv/(\gamma - 1)$, $-\mathrm{d}P/\mathrm{d}v = \gamma P/v$ and Eq. (26) should be rewritten as:

$$\frac{1}{\gamma - 1}\left(P_{\mathrm{H}} v_{\mathrm{H}} - P_0 v_0\right) = \frac{1}{2}\left(P_{\mathrm{H}} + P_0\right)\left(v_0 - v_{\mathrm{H}}\right) + Q \tag{34}$$

When $P_{\mathrm{H}} \gg P_0$, $e_{\mathrm{H}} \gg e_0$, from the above relational expression, it may be obtained that:

$$D = \sqrt{2\left(\gamma^2 - 1\right)Q} \tag{35}$$

$$P_{\mathrm{H}} = 2\left(\gamma - 1\right)\rho_0 Q = \frac{\rho_0}{\gamma + 1} D^2 \tag{36}$$

$$\rho_{\mathrm{H}} = \frac{\gamma + 1}{\gamma}\rho_0 \tag{37}$$

$$T_{\mathrm{H}} = \frac{2\gamma}{\gamma + 1}\frac{Q}{C_{\mathrm{v}}} \tag{38}$$

$$u_{\mathrm{H}} = \sqrt{\frac{2\left(\gamma - 1\right)}{\gamma + 1}Q} = \frac{D}{\gamma + 1} \tag{39}$$

Insert the state equation $T_{\mathrm{H}} = \dfrac{P_{\mathrm{H}} v_{\mathrm{H}}}{P_0 v_0} T_0 = \dfrac{P_{\mathrm{H}} v_{\mathrm{H}}}{C_{\mathrm{v}}\left(\gamma - 1\right)}$ into the above equation to obtain:

$$D = \frac{\gamma + 1}{\gamma}\sqrt{\gamma n R T_{\mathrm{H}}} = \frac{\gamma + 1}{\gamma} c_{\mathrm{H}} \tag{40}$$

It can be seen that the velocity of detonation is $(\gamma + 1)/\gamma$ times the sonic velocity c_{H} of compressed detonation product. γ is the polytropic index, and it is associated with the pressure of expansion when the detonation product is in a high-pressure state. For most explosives, γ is 1.3–3 (Fig. 12).

The P_{H} here is the pressure right after the detonation transition zone. Due to the expansion and exothermic effect of the reaction products downstream of the detonation wave, the pressure is already reduced by half compared with the pressure at the shock wave front ahead. C-J condition parameters are only associated with equations for initial state of unreacted explosive and fully reacted products. For most condensed explosives, the C-J point is approximately $\gamma \approx 3$.

Taylor devised a flat, one-dimensional explanation for the flow field of products behind a C-J detonation wave, and this is called the Taylor wave. The flow field upstream of a C-J detonation wave is simplified into simple wave flow, comprised of the C-J detonation wave front ①, central rarefaction wave (fan-shaped zone) ②, constant area ③ (Fig. 13). The central rarefaction wave is comprised of a system of characteristic line that intersect at the point of origin. The equation for characteristic line is:

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \frac{x}{t} = u + c \tag{41}$$

Solution for the Taylor wave in a flat, one-dimensional detonation product flow field:

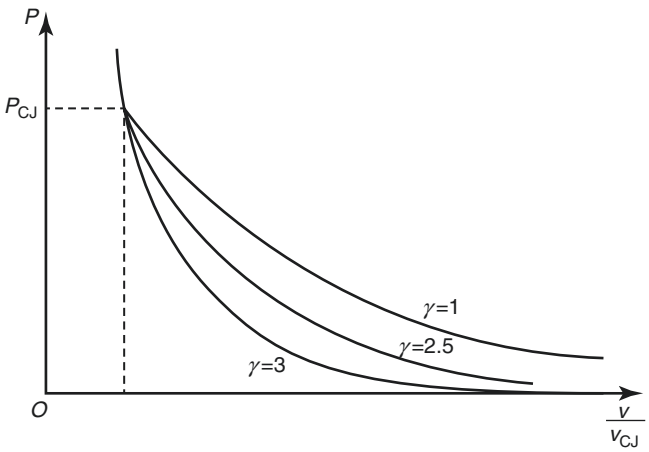

Fig. 12 Different values of gas expansion γ under high pressure

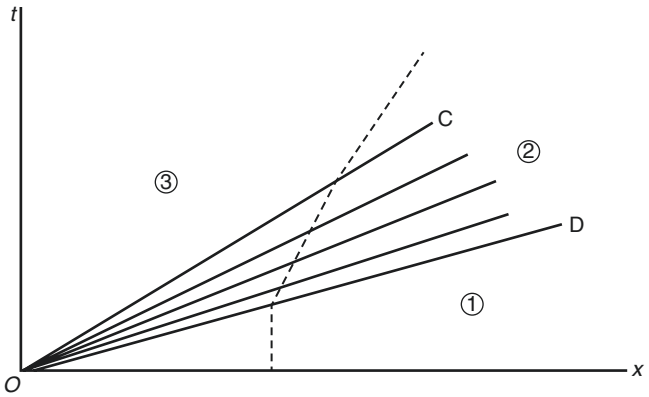

Fig. 13 Flow fields behind the CJ detonation wave

$$u = \frac{2}{\gamma+1}\frac{x}{t} - \frac{D_{\mathrm{CJ}}}{\gamma+1} \tag{42}$$

$$c = \frac{\gamma-1}{\gamma+1}\frac{x}{t} + \frac{D_{\mathrm{CJ}}}{\gamma+1} \tag{43}$$

The pressure, density, and other physical quantities of product may be obtained through sonic velocity c.

2. **Detonation wave's steady structure — ZND model**. Following advancements in empirical experimentation techniques, people discovered that there exists rather glaring differences in data analyzed and calculated in the aforesaid C-J theory versus data obtained in experiment, to the point that some experiment phenomenon could not be explained. For example, the C-J theory could neither explain the process of weak detonation, nor describe detonation in relatively wider chemical reaction zone. Therefore, it was necessary to carry out studies about the internal structure of detonation wave, and take into full account the energy release process of a detonation wave's chemical reaction. Upon the basis of the C-J theory, Zeldovich, von Neumann, and Döring added considerations on limited reaction time. They argued that detonation wave is comprised of the shock wave ahead (shock wave front is still assumed to be strong discontinuity) and a continuous and irreversible chemical reaction zone that progresses at a limited speed. This is the ZND model (Fig. 14).

Compared with the C-J theory, the ZND model introduces reactivity λ, a thermodynamics quantity that expresses the reaction process. $\lambda = 0$ represents unreacted explosive; $\lambda = 1$ represents fully reacted explosive, and Q denotes heat of reaction released; incompletely reacted state is $0 < \lambda < 1$, and the heat of reaction released is λQ.

Energy equation should be rewritten as:

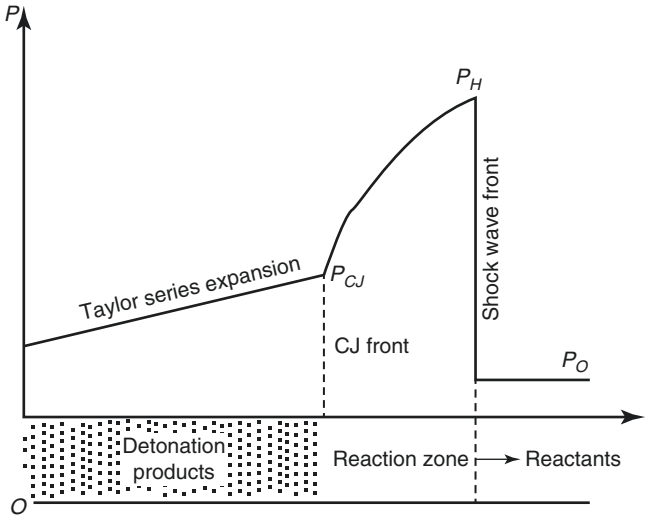

Fig. 14 ZND model of detonation wave

$$e_{\mathrm{H}} - e_0 = \frac{1}{2}\left(P_{\mathrm{H}} + P_0\right)\left(v_0 - v_{\mathrm{H}}\right) + \lambda Q \tag{44}$$

If the polytropic gas state equation is used, then the above equation should be rewritten as:

$$\frac{1}{\gamma-1}\left(P_{\mathrm{H}}v_{\mathrm{H}} - P_0 v_0\right) = \frac{1}{2}\left(P_{\mathrm{H}} + P_0\right)\left(v_0 - v_{\mathrm{H}}\right) + \lambda Q \tag{45}$$

Unlike the C-J theory, the shock wave's Hugoniot curve and detonation wave's Hugoniot curve are not the only Hugoniot curves on the plane (P, v), but a family of Hugoniot curves associated with the functions of λ may be obtained (Fig. 15). Given a specific λ to draw the corresponding Hugoniot curve, any of $0 < \lambda < 1$ is known as a frozen Hugoniot curve. $\lambda = 0$ corresponds to Hugoniot curve of a non-exothermic reaction, and it is actually the Hugoniot curve of the shock wave; $\lambda = 1$ corresponds to Hugoniot curve of a fully exothermic reaction, and this is called balanced Hugoniot wave or final state Hugoniot wave.

The C-J condition is also applicable to the ZND model. Steady detonation is the propagation process of a self-sustaining wave. When $\lambda = 1$, the point of final state of steady detonation is the C-J point, and a steady detonation propagates at a constant speed. Explosion is a special detonation phenomenon and is known as an unsteady detonation.

3. **Detonation product state equation**. In accordance with the first law of thermodynamics, the equation of the state of a material includes the four thermodynamics parameters of pressure P, volume V, temperature T, and mass m, and a systematic state equation may be written as:

$$\pi\left(P,V,T,m\right)=0 \tag{46}$$

When three parameters are known, the function of the fourth may be obtained, and therefore a broader expression should be:

$$\pi\left(P,V,T\right)=0 \tag{47}$$

It is very difficult to provide a solution that solves a theoretical model, and most of the time an approximate solution is used.

Equation for an ideal gas state:

$$PV=nRT \tag{48}$$

In this equation: P is the pressure of the gas, and V is the volume of the gas; n is the quantity of mass of gas; and R is the molar gas constant, which is 8.314 J/(mol · K).

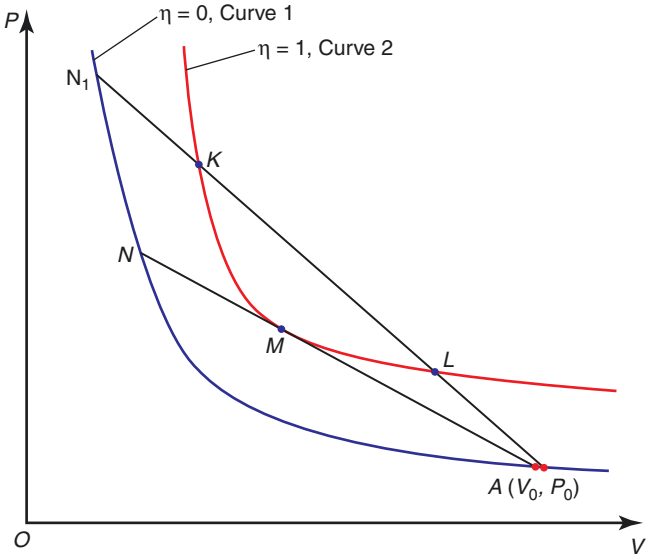

Fig. 15 Frozen Hugoniot curves

For real gas in high-pressure state, gas molecules have the largest volume (Fig. 16). Modify the equation for ideal gas state, subtract molecule-occupied volume b in mixture volume from gas volume V, then obtain the equation for the state of real gas:

$$P\left(V-b\right)=nRT \tag{49}$$

Another expression of the equation for real gas state:

$$P=\frac{\rho nRT}{1-b\rho}-a\rho^2 \tag{50}$$

In the equation, a denotes parameter that controls the intermolecular attraction within the gas, while ρ represents density, and $\rho=\frac{1}{v}$.

If ρ is expressed as a polynomial, then:

$$P=\rho nRT\left(1+bp+0.625b^2\rho^2+0.287b^3\rho^3+0.193b^4\rho^4+\cdots\right) \tag{51}$$

The virial equation for expressing the state of real gas:

$$PV=RT\left(1+\frac{B_2}{V}+\frac{B_3}{V^2}+\frac{B_4}{V^3}+\cdots\right) \tag{52}$$

In the equation: B_i represents the coefficient in the i order.

(a) **JWL equation of state**: The JWL or Jones–Wilkins–Lee equation of state is expressed as:

$$P\left(E,v\right)=A\left(1-\frac{\omega}{R_1v}\right)e^{-R_1v}+B\left(1-\frac{\omega}{R_2v}\right)e^{-R_2v}+\frac{\omega E}{v} \tag{53}$$

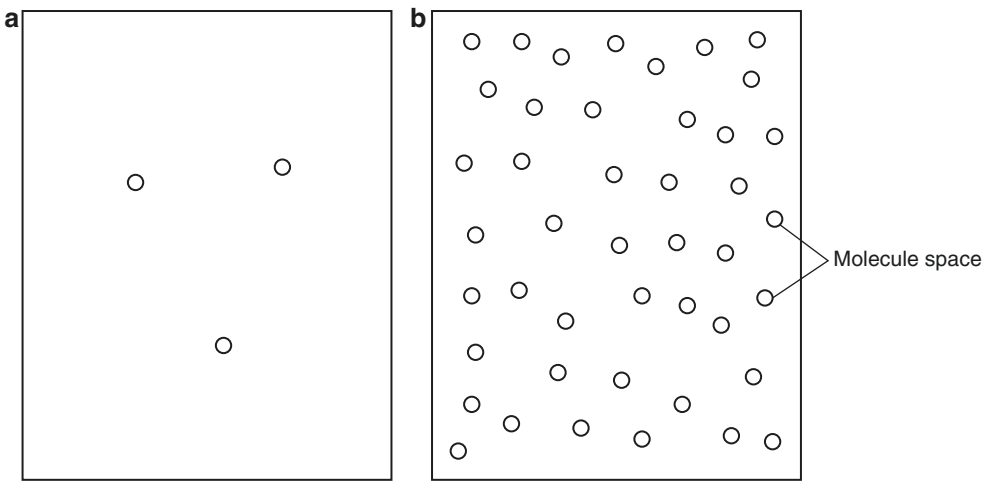

Fig. 16 Volume occupied by molecules in real gas under two pressure states. (**a**) Low pressure; (**b**) High pressure

In the equation, P is the pressure of product of detonation; v is relative specific volume, $v = \rho_0/\rho_H$; ρ_0 is the initial density of the explosive; ρ_H is the density of the detonation product; E is the internal energy in a unit volume; and A, B, R_1, R_2, and ω are undetermined parameters in the Jones–Wilkins–Lee equation of state. For C-J condition, Hugoniot condition, and isentropic condition, the JWL equation of state may be respectively written as:

$$AR_1 e^{-R_1 v_{CJ}} + BR_2 e^{-R_2 v_{CJ}} + C(1+\omega) v_{CJ}^{-(\omega+1)} = \rho_0 D_{CJ}^2 \tag{54}$$

$$\frac{A}{R_1} e^{-R_1 v_{CJ}} + \frac{B}{R_2} e^{-R_2 v_{CJ}} + \frac{C}{\omega} v_{CJ}^{-\omega} = E_0 + \frac{1}{2} P_{CJ}(1 - v_{CJ}) \tag{55}$$

$$A e^{-R_1 v_{CJ}} + B e^{-R_2 v_{CJ}} + C v_{CJ}^{-(\omega+1)} = P_{CJ} \tag{56}$$

Plus undetermined parameter C, although there are six undetermined parameters, only three are independent. The usual method is to give a specific group of values for R_1, R_2 and ω, then use the equation below to find the corresponding A, B and C. Then insert this group of A, B, R_1, R_2, ω and C parameters into fluid dynamic program for calculations in cylinder experiment or hemispherical shell experiment. If the calculated cylindrical wall velocity and steady-state flight time coincide with cylinder experiment and hemispherical shell experiment, then this group of parameters may be confirmed. Otherwise, it would be necessary to calculate with a new group of parameters until the required precision is satisfied. For the majority of explosives, $R_1 = 4$–5, $R_2 = 1$–2 and $\omega = 0.2$–0.4.

The JWL equation of state is applicable to C-J isentropic line that describes detonation product when it expands from the C-J point to the 10^{-1} GPa range of pressure, but is not applicable to describe states that diverge from the C-J isentropic line.

(b) **BKW equation of state**: The BKW equation of state of detonation product uses an exponential polynomial format:

$$P = \rho nRT\left[1 + \rho z(T+\theta)^{-\alpha} \exp \beta\rho z(T+\theta)^{-\alpha}\right] \tag{57}$$

$$z = k\sum x_i k_i \tag{58}$$

In the equation: P is pressure, ρ is the density of gas product, R is the molar gas constant, and T is temperature; x_i is the molar value for the gaseous components in the i order; k_i is the geometrical residual capacity for components in the i order; and α, β, k, and θ are parameters confirmed through empirical experience. Table 2 shows the benchmark results for BKW parameters for the explosives RDX and TNT as given by Mader.

Table 2 BKW parameters for RDX and TNT

Explosive type	α	β	k	θ
RDX	0.54	0.181	14.15	400
TNT	0.50	0.09585	12.685	400

(c) **VLW equation of state**: The first form of the virial equation corresponds to ideal gas; the second form takes into consideration the mutual actions between two molecules; the third form takes into consideration the mutual actions between three molecules, and so forth. When the detonation product gas mixture exists in a high-pressure state, it is necessary to consider the simultaneous collision between multiple molecules. Actual solution to virial coefficient of a high order is enormously complicated. Based on the virial theory and similarity theory, Wu Xiong expresses virial coefficient of a high order as a second virial coefficient, subsequently obtaining VLW detonation product state equation:

$$\frac{Pv}{RT} = 1 + B^*\left(\frac{b_0}{v}\right) + \frac{B^*}{T^{*1/4}}\sum(n-2)^{-n}\left(\frac{b_0}{v}\right)^{(n-1)} \tag{59}$$

In the equation, the Lennard-Jones 6–12 pair potential is used to express the second virial coefficient B^*, as in $B^* = \left[-\frac{2^{j+1/2}}{4j}\Gamma\left(\frac{j}{2} - \frac{1}{4}\right)T^{*-(2j+1)/4}\right]$, and B^* may also be used to modify the Buckingham potential (Exp-6 potential) expression; T^* is dimensionless temperature, and $T^* = \frac{k}{\varepsilon}T; b_0 = \frac{2}{3}\pi N\sigma^3$; N is the Avogadro constant; k is the Boltzmann constant; and ε and σ are Lennard-Jones potential parameters. The VLW equation of state uses the parameters of detonation product gas component's potential in its description, and holds that virial coefficients at different orders are similar under high temperature, of which, virial coefficients at higher orders are obtained through second virial coefficient. This kind of simplification has a certain degree of impact on the precision of the description of the thermodynamics state of detonation product gas component under high temperature.

2.3 Sensitivity of Explosives to External Effects

In previous section about knowledge on explosives, it was mentioned that primary explosives may be detonated with either pin (impact) or spark (flame), while condensed explosives require the shock wave from primary explosive explosion or high-speed impact of a metallic object to detonate. The sensitivity of an explosive reflects the lowest initiation energy required for set explosive to combust and detonate under certain conditions. It should be noted that reaction is very complicated, and even if charge conditions are given for the same type of explosives, the lowest initiation energy is not an invariant. In order words, sensitivity is not absolute, and other than the chemical and physical properties of the explosive, other influential factors include the type of the initial stimuli and how the energy of a given load is distributed in the explosive. Generally speaking, it is believed that a successful reaction requires energy from external stimuli to be concentrated on the explosive within an extremely short span. In contrast, if external energy is distributed evenly across the explosive, then a reaction would be difficult. An explosive's sensitivity to external stimuli can be divided into three categories: heat sensitivity, mechanical sensitivity, and shock sensitivity.

2.3.1 Heat Sensitivity

If the heat released from a reaction is larger than heat loss, the accumulation of heat would induce a rise in temperature, and in turn speeding up the reaction and causing an explosion. The heat required for heat reaction might come from two sources: One is the use of flame, spark, or other heat to provide localized heat to an explosive, and this local heat pulse initiates local reaction of the explosive, which should have a self-sustaining reactive property to allow the reaction heat to spread to other parts of the explosive. The second source is the heating of the whole explosive (without clear flame), and at critical temperature the explosive would decompose in accordance with the rule of thermal explosion. When the heat balance is broken, as in the heat generated by the explosive is more than the heat loss in the environment, the explosive would explode.

Classic thermal initiation theory holds that the relationship between the heat release process of chemical reaction and the heat transfer process to surrounding medium (heat dissipation) determines whether or not a thermal initiation of the explosive could be achieved under the effect of heat, and the properties in the span between exothermic chemical explosion to the explosive reaction. System of equation for describing heat conduction in the heat process and chemical kinetics:

$$c_{\mathrm{P}}\rho\frac{\partial T}{\partial t}=\lambda\nabla^2 T+Q\frac{\partial \Lambda}{\partial t} \tag{60}$$

$$\frac{\partial \Lambda}{\partial t}=k_0\mathrm{e}^{-\frac{E}{RT}}\varphi\left(\Lambda\right) \tag{61}$$

In the equation, c_{P}, ρ, and λ respectively denote the constant-pressure specific heat, density, and coefficient of heat conduction of the energetic material; T is temperature; Q is heat of decomposition per unit volume; Λ represents the percentage of already reacted energetic material; $\frac{\partial \Lambda}{\partial t}$ is rate of chemical reaction; k_0 is constant; R is the molar gas constant; E denotes activation energy; $\lambda\nabla^2 T$ equation expresses the heat inflow or outflow due to heat conduction, and $Q\frac{\partial \Lambda}{\partial t}$ shows chemical reaction energy released from unit volume of energetic material within unit time; $\varphi(\Lambda)$ represents rule of reaction in isothermal condition, and when $\varphi(\Lambda) = 1$, Eq. (60) changes to the Arrhenius equation of rate:

$$\frac{\partial \Lambda}{\partial t}=k_0\mathrm{e}^{-\frac{E}{RT}} \tag{62}$$

Any explosive has its own lowest explosion temperature, and when that explosive reaches that temperature, it doesn't react right away, but there's a delay before its explosion in what is called the explosion delay. The heat sensitivity of an explosive may be determined by using Wood's metal bath experiment to measure the explosion delay time τ, Wood's metal bath experiment thermodynamics temperature T, and use the Arrhenius equation to calculate activation energy E:

$$\tau=A\mathrm{e}^{-\frac{E}{RT}} \tag{63}$$

In the equation, R is the universal gas constant and A symbolizes the frequency factor that depends on the explosive; take the log of both sides of the above equation, and the above equation changes to:

$$\ln\tau=\ln A+\frac{E}{RT} \tag{64}$$

When $\ln\tau$ and $(1/T)$ are on a plane, the result is almost a straight line, and then calculate E/R to obtain activation energy E.

Another qualitative index associated with heat sensitivity is flash point, which is the temperature required for the explosive to explode during a 5 s or 10 s delay period.

2.3.2 Mechanical Sensitivity

The mechanical sensitivity here refers to dynamic actions other than shock wave effects. Mechanical actions include impact sensitivity, friction sensitivity, and others. Understanding mechanical sensitivity is vital to both the safe production and usage of explosive, and reliable and desired detonation.

1. **Impact sensitivity**. All instrument (equipment) that measures impact sensitivity work under pretty much the same principle, which is the drop hammer test. The dropped steel hammers respectively weigh 10 kg, 5 kg, 2 kg, and 0.6 kg. Main methods for expressing the mechanical sensitivity of explosives are:
   (a) Explosion percentage expression method: Change drop-height while hammer weight remains unchanged. Carry out ten attempts at each height and calculate the percentage of explosion initiated. The relationship between the different drop-heights of same weight and explosion percentage is expressed through an impact sensitivity curve.
   (b) Drop-height expression method: For a drop-height with 50% chance of explosion is recorded as H_{50}; the lowest drop-height at which explosion initiation is 100% is marked as H_{100}; and the highest drop-height at which explosion initiation is 0% (meaning that no explosion could be induced) is denoted as H_0.
   (c) Impact energy expression method: Usually expressed as the energy for an impact with 50% chance of explosion:

$$E_I = M_d Hg \tag{65}$$

   In the formula, E_I is the impact energy; M_d is mass of the dropped hammer; H is height; and g is gravitational acceleration.
   (d) Relative impact sensitivity expression method: Usually TNT is used as the reference explosive, and the impact sensitivity of the tested explosive is compared against TNT:

$$O_R = \frac{E_{IX}}{E_{ITNT}} \times 100 \tag{66}$$

   In the formula, O_R is relative impact sensitivity; E_{IX} is impact energy imparted on the tested explosive; and E_{ITNT} is impact energy of TNT.

   The impact sensitivity tests stipulated in *Explosive Test Method* GJB772A-97 include the drop hammer test, Susan test, and slide test.

   At present, the majority of studies opine that impact induces explosion through one of the four mechanisms below to convert mechanical energy into heat, creating localized hot spot in explosive for initiation: (1) Hot spot generated in local shear band created by impact initiates explosion; (2) hot spot generated in adiabatic compressed gas space; (3) hot spot generated from friction when the impacted service enters the explosive's interior and rub against explosive crystals and/or impurity particles; and (4) hot spot generated in the viscosity between the impacted surface and particles when the surface of the explosive quickly protrudes after impact.

   The general consensus is that the temperature, size, and sustained duration of the hot spot that causes ignition or detonation usually are characterized by: (1) temperature no less than 700 K; (2) hot spot diameter 0.1–10 μ_0; and (3) sustained time 10^{-5} to 10^{-3} s.

   During the production or utilization process, if the generated hot spot's temperature was lower than 700 K, and the generated hot spot's diameter was smaller than 0.1 μ_0, while the sustained duration was less than 10^{-5} s, then ignition or detonation would not occur, and at most an unsustainable localized decomposition would take place.
2. **Friction sensitivity**. During the production and utilization process of an explosive, it is common to see friction between particles within the explosive, or between the explosive and contact surface of other materials. This kind of friction could generate hot spot and ignite or detonate the explosive. Yet, whether or not a hot spot can ignite depends on the state of imbalance between the heat generated in chemical reaction and heat loss. As mentioned above, this kind of imbalance state is contingent on the temperature, size, and sustained duration of the hot spot. If a material melts, friction would no longer produce heat. Since an explosive's melting point is always lower than ignition temperature, therefore, pure friction between explosive and other materials is not enough to cause an ignition. The common consensus is that the friction between particles with high melting point included in a charge, or the friction between particles and substrate with high melting point, could most possibly result in ignition.

   Main instruments and methods for testing friction sensitivity used in China and abroad include: pendulum friction test, BAM friction device, Bowden-Kozlov device, torpedo friction test, solid explosive friction test, and liquid explosive friction test. The instrument stipulated in *Explosive Test Method* GJB772A-97 is the Bowden-Kozlov device, and the measurement taken was friction sensitivity of powdered explosives. To measure the fric-

tion sensitivity of packaged explosives, the USA created a friction sensitivity experiment and measurement method using small pieces of samples in order to ascertain the friction sensitivity of insensitive explosives. China is also undertaking similar studies.

2.3.3 Shock Sensitivity

The effect of a shock wave can initiate an explosion reaction in the explosive. Using the lowest shock wave pressure required for complete detonation of explosive as measurement of shock sensitivity, the *Explosive Test Method* GJB772A-97 stipulates the use of the card gap method to measure shock sensitivity.

1. **Initiation of explosion in homogeneous explosives by shock wave**. Homogeneous explosive refers to air explosive, homogeneous liquid explosive (without air bubble or solid impurities), and single-crystal explosive. After a shock wave enters the explosive and evenly compresses and heats the explosive molecules, chemical reaction would be initiated. When the incident wave has a relatively high pressure, the complete reaction time of the explosive is extremely short, and thermal detonation would occur near the incident surface where the shock wave enters, creating a strong detonation. The detonation wave propagates with a velocity higher than a steady-state detonation, and when the strong detonation catches up to the initial incident shock wave, the unreacted explosive would gradually turn into steady detonation. When the incident wave has a relatively low pressure, if the sustained duration is long enough, then the reaction process upstream of the shock wave front would take place at a low rate. Thermal detonation would occur somewhere between the shock wave's incident surface and the shock wave front, usually toward the end of the charge.

Figure 17 is a time-space diagram of detonation wave velocity based on analysis of shock initiation of nitromethane. Line *OA* is nitromethane's loaded interface track, and line *OD* is track of incident wave, with velocity that is pretty much constant; line *OA* is the trajectory of nonreactive gap surface, as in the trajectory of mass point of explosive behind the shock wave front; line *AD* is the trajectory of strong detonation wave; and line *DB* is the trajectory of steady-state detonation wave. As the incident shock wave propagates through the explosive, it pre-compresses and heats the explosive. After explosion delay, strong detonation takes place at point *A*, then propagates at a velocity higher than steady detonation within the pre-compressed nitromethane, catching up to and overtaking incident shock wave, subsequently evolving into a steady detonation.

In the explosive compressed by shock, the propagation velocity of the subsequent detonation is associated with the density of the compressed liquid and the increase in velocity at local mass points:

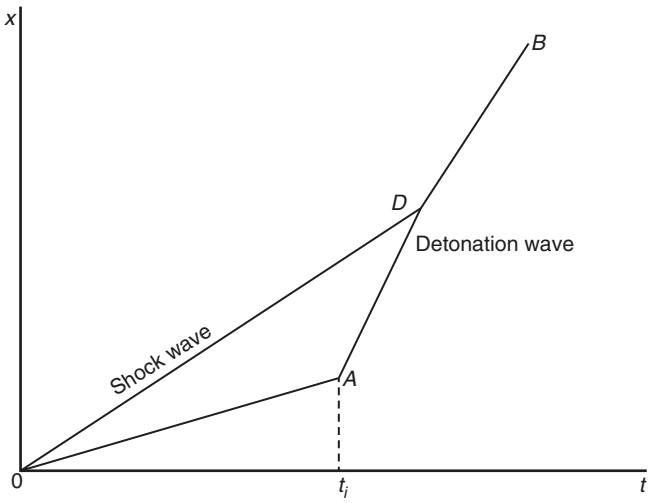

Fig. 17 Time-space diagram of detonation wave velocity of homogeneous explosive under shock wave action

$$D = 6.30 + 3.2\left(\rho - \rho_0\right) + u_{\mathrm{P}} \tag{67}$$

In the equation, ρ is density of compressed liquid; ρ_0 is undisturbed initial density; and u_{P} is mass point velocity, which corresponds to line *OA* in the diagram. This relational expression shows that for each 1 g/cm^2 increase in density, there is a corresponding 1 g/cm^2 increase in velocity. When the pressure of shock wave reaches 80 kPa, measured detonation velocity reaches 10 km/s. Pressure on ultra high-speed shock wave front could reach upward of 250 kPa, while pressure upstream of the shock wave front would drop rapidly because of the dispersal of detonation products, which is a conclusion that complies with the detonation product one-dimensional dispersion theory.

Reaction rate for homogeneous explosive described per the Arrhenius theory would be:

$$r = \frac{\mathrm{d}\lambda}{\left(1-\lambda\right)\mathrm{d}t} = Z\exp\left(-\frac{E_0}{RT}\right) \tag{68}$$

In the equation, λ is mass fraction of the reaction product; Z is frequency factor; E_0 is activation energy; T is reaction product temperature; and R is gas constant.

At present, shock ignition mechanisms related to single-crystal explosive include: dislocation ignition mechanism of hot spot generated from dislocation pile-ups and dislocation slide; ignition mechanism based on molecular entanglement structural transformation resulting from shear steric hindrance; and ignition mechanism of hot spot generated at crystal molecule adiabatic shear band.

2. **Initiation of explosion in heterogeneous explosives by shock wave**. Bubbles, gaps, and impurities formed during an explosive's casting, pressing, crystallization and other processes create discontinuous internal structure and uneven density in the explosive. In general, solid explosives used in real life are all heterogeneous explosives.

   When a shock wave enters a heterogeneous explosive, the bubbles and gaps inside the explosive are compressed adiabatically, creating hot spots inside the compressed bubbles with temperature higher than the crystal. Under the action of shock wave, the explosion processes of homogeneous explosive and heterogeneous explosive differ vastly. Figure 18 is a time-space diagram of detonation wave velocity of heterogeneous explosive under shock wave action. Compared with Fig. 17, it can be seen that in homogeneous explosive, the incident shock wave's propagation is basically a process with constant velocity, but in heterogeneous explosive, incident shock wave's propagation is a process that accelerates; in homogeneous explosive, strong detonation is formed in a jump manner, but no strong detonation has been observed in heterogeneous explosive; in homogeneous explosive, detonation usually occurs near the interface between the shock wave gap and the explosive, in heterogeneous explosive meanwhile, detonation is usually believed to take place near the front of the shock wave. Moreover, research shows that relative to homogeneous explosive, heterogeneous explosives are more sensitive to shock wave due to the existence of bubbles, gaps, and impurities that could form hot spots. Meanwhile, compared with heterogeneous explosive, the shock wave initiation process of homogeneous explosives is more sensitive to initial temperature and changes in the shock wave's pressure. In Fig. 18, point D represents steady detonation occurrence. The intersection point between the reverse extension line of detonation trajectory and time axis is Δt, known as excess propagation time. t_D and x_D are respectively time and distance to detonation.

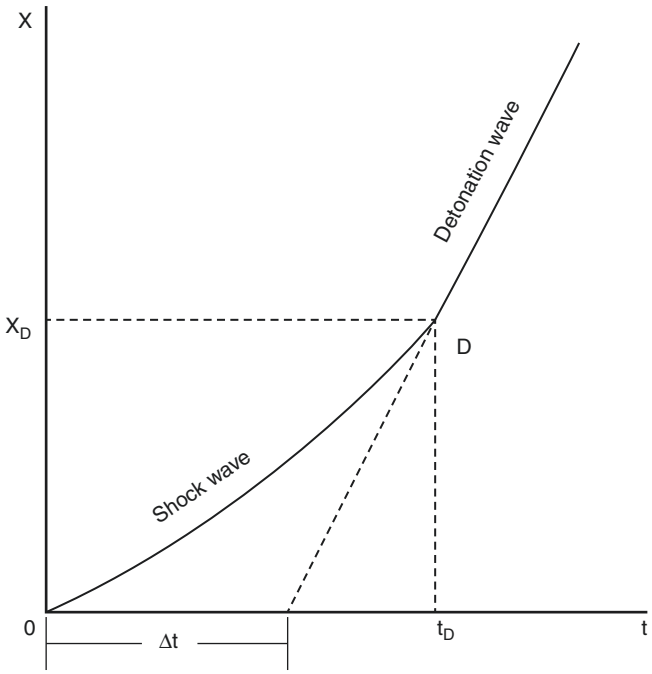

Fig. 18 Time-space diagram of detonation wave velocity of heterogeneous explosive under shock wave action

3. **Shock detonation model**. It is generally believed that the explosion initiation of heterogeneous explosive is caused by hot spots created by shock wave in the explosive, which in turn gradually develop into detonation wave. There are five possible mechanisms for the generation of hot spot as listed below: (1) Hot spot generated from adiabatically compressing bubbles or gaps in the explosive; (2) hot spots generated in localized shear bands; (3) hot spots generated from friction between impurity particles inside the explosive; (4) hot spots generated from dislocation and imperfection in crystal; and (5) hot spots generated from cavity elasto-viscoplastic collapse.

   Models related to shock initiated detonations in explosives include: Forest-fire model, JFT model, HVRB model, Lee–Tarver model and Kim model, among others. Here, we will take a look at the more popular Lee–Tarver model.

   The earliest Lee–Tarver model included two parts, namely ignition and growth. The reaction rate equation is:

$$\frac{\mathrm{d}F}{\mathrm{d}t} = I\left(1-F\right)^b \mu^x + G\left(1-F\right)^b F^g p^z \tag{69}$$

In the equation, $\mu = \rho/\rho_0 - 1$, F is mass fraction of the reacted explosive; and I, b, x, G, g, and z are coefficients. This is a two-part reaction rate model, and the first part on the right is the ignition part, which is assumed to be proportional to some compression capacity. The value of index x is associated with the assumed method of hot spot generation. Some methods hold that ignition is related to the square of particle velocity u_p, other methods believe that ignition is related to the square of pressure P. Since very similar relationships exist between P and μ^2, and between u_p and $\mu^{3/2}$, which is why the values of 3 or 4 is frequently used for x in most calculations. The second part on the right is pressure related to combustion rate of layered particles, and pressure index z usually falls within the 1–2 range. Factor F^g is associated with combustion surface, and for spherical hot spot that burns outwardly, index is 2/3. Proportionality coefficient G needs to be confirmed through experiments on layered particle combustion rate. Factor $(1 - F)^b$ was inserted in order to

ensure that when the proportion of solid explosive neared zero, reaction rate equals zero. When given index b is 2/9 and when F is 3/4, $(1-F)^b F^g$ reaches its greatest value.

Results of explosives calculated from said model fit closely with many experiments' data. These experiment data include data obtained from embedded manometer, particle counter, velocity interferometer system for any reflector (VISAR) and card gap test, as well as data of detonation failure. Yet, when simulating short-pulse duration ignition, the reaction growth coefficient G in the model needs to be adjusted with two to three pressure-related factors. In order to adjust the model to more accurately fit ignition processes with a wider range of input pressure, growth duration, and pulse duration, the model was modified and a three-part reaction rate model was built:

$$\frac{\mathrm{d}F}{\mathrm{d}t} = I\left(1-F\right)^b\left(\mu-a\right)^x + G_1\left(1-F\right)^c F^d p^y + G_2\left(1-F\right)^e F^g p^z \tag{70}$$

In the equation, I, G_1, G_2, a, b, e, d, e, g, x, y, and z are 12 undetermined coefficients; compared with the original model, the growth part was further divided into two parts. The first part describes the relatively slow reaction process of particles similar to deflagration, and at this time hot spots exist independently, and pressure index may be set at 1, while the second part describes hot spots coming together, and the remaining unreacted explosives decompose rapidly, and at this time pressure index may be set at 2 or 3. Another modification is changing the outward void combustion model to the inward particle combustion model so as to better fit the experiment results. In order to better restrict the respective application scopes of the three parts on the right, three constants have been introduced: F_{igmax}, F_{G1max}, and F_{G2min}. When $F > F_{\mathrm{igmax}}$, ignition is set at zero when $F > F_{\mathrm{G1max}}$, the first growth part is set at zero; and when $F < F_{\mathrm{G2min}}$, the second growth part is set at zero. Table 3 lists the Lee–Tarver model parameters for some explosives.

4. **Explosive detonation from fragments**. In experiments where fragments were used to detonate explosives including LX-04, TNT, PBX9404, and Comp B, the following relational expression was obtained:

$$\frac{P^2\tau}{\rho_{\mathrm{e}}U_{\mathrm{e}}} = \text{constant} \tag{71}$$

In the equation, P represents shock wave pressure; τ is the time that shock waves propagate back and forth in fragment; U_{e} is velocity of shock wave in explosive; and ρ_{e} denotes density of explosive.

If impacts from changes in U_{e} are taken into account, the above equation may be evolved into criteria for one-dimensional plane short-pulse ignition:

$$p^n\tau = \text{constant}, n > 2.3 \tag{72}$$

If impacts from changes in U_{e} are ignored, criteria for heterogeneous explosive detonation from fragments is obtained:

$$p^2\tau = \text{constant} \tag{73}$$

Flat-face steel hammers with different diameters were used to deliver impact to PBX9404 explosive with diameter 25.4 mm, and the front of the explosives were covered with metal plates of varying thicknesses. Experiment results show that explosion initiation threshold velocity for explosive rises as diameter of projectile decreases or as covering plate thickness increases. In other words, thinner covering plate reduces the area of loading of explosive, or to put it differently, similar to using a projectile with smaller diameter to strike a cover-less explosive. However, at certain covering plate thickness the shock wave no longer acts like a flat plane, and it would be necessary to account for issues in detonation of shock wave with curved plane.

Criteria for velocity of projectile direct impact on explosive with cover plate:

$$\frac{v_{\mathrm{d}}}{2} = \left(1+k\right)\left[A + \frac{Bh}{d}\right] \tag{74}$$

Table 3 Lee–Tarver model parameters for some explosives

Explosive	PBX9404	LX17	Propellant[a]
I/propellant^{-1}	7.43×10^{11}	4.00×10^6	40
a	0.0	0.22	0.0
b	2/3	2/3	2/3
x	20	7	4
G_1/GPa^{-y}μs^{-1}	0.031	0.006	0.031
c	2/3	2/3	2/3
d	1/9	1/9	1/9
y	1	1	1
G_2/GPa^{-z}μs^{-1}	0.04	0.0004	0.0018
e	1/3	1/3	1
g	1	1	1/9
z	2	3	2
F_{igmax}	0.3	0.5	0.015
F_{G1max}	0.5	0.5	0.12
F_{G2max}	0.0	0.0	0.0

[a] Propellant is comprised of AP, Al, HMX (12%), and binder

Table 4 Experiment and calculated values of steel ball impact detonation of explosive charges with cover plate

Angle of impact/°	Diameter of steel ball/mm	Thickness of cover plate/mm	Experiment value/(km thickness $^{-1}$)	Calculated value/(km thickness $^{-1}$)
0	16.67	6.0	1.917 explosive with cover plate	1.93
0	16.67	12.0	2.657 explosive with cover plate	2.64
15	16.67	6.0	1.967 explosive with cover plate	2.00
45	16.67	6.0	2.377 explosive with cover 7	2.73
45	18.34	6.0	2.127 explosive with cover 7	2.51
60	16.67	6.0	3.007 explosive with cover 7	3.86

In the equation, A is coefficient related to explosive and projectile material; B is coefficient related to cover plate material; k is coefficient related to projectile shape; h represents thickness of cover plate; and d refers to diameter of projectile.

Criteria for detonation threshold velocity obtained in experiments in which T/R (40/60) explosives covered with steel plate were detonated through impact from being struck with steel ball at different angles:

$$\frac{v_{\mathrm{d}}}{2}=\left(1+k\left(\theta\right)\right)\left[A+\frac{Bh}{d\cos\theta}\right] \tag{75}$$

$$k\left(\theta\right)=0.5+0.2\left(\frac{1}{\cos\theta}-1\right) \tag{76}$$

In the equation, θ denotes the included angle between the trajectory of projectile and normal line of cover plate. In direct impact, when $\theta = 0$ and $k = 0.5$, the above equation devolves to (74). For experiment results, please see Table 4.

3 Propagation of Detonation

3.1 Propagation of Detonation in Condensed Explosive

The ZND fluid dynamics model for detonation assumes: The front of detonation wave is a flat plane, the flow in the reaction zone is one-dimensional, the terminal point of steady detonation is the C-J point, and detonation is a perfect detonation with no energy loss. Velocity of the detonation wave primarily depends on the energy released in the chemical reaction zone upstream of the shock wave ahead. However, in real life the velocity of detonation wave is also affected by shape of charge (cylindrical, flat, etc.), size (diameter, thickness, etc.), restricting conditions (shell, etc.), and other properties (density, structure, particle size, evenness, etc.) of the charge (condensed explosive).

Therefore, the detonation of a charge in real life is not perfect: The front of detonation wave is not a flat plane but curved, flow in the reaction zone is not one-dimensional, and the terminal point of steady detonation is not the C-J point. Studies on non-perfect detonation mostly look at situations with size limitations, for instance when the size of charge approaches infinitely big or when explosive is located in an ideal environment with rigid restrictions, detonation wave front may still be considered a flat plane, and flow in the reaction zone may still be treated as one-dimensional.

When detonation wave propagates in a cylindrical charge with a certain diameter, sideward rarefaction waves should cause certain explosive medium undergoing reaction to disperse outward from the chemical reaction zone, leading to loss of some energy that ought to support the front of detonation wave. When the charge diameter is large enough, the aforementioned energy loss would not have much of an influence, and detonation wave would basically propagate at an ideal detonation velocity. As the diameter of the explosive decreases, impact from energy loss increases, and when the diameter of the explosive shrinks pass a certain size (critical diameter), detonation velocity would drop significantly with any reduction in diameter. When charge diameter reaches the critical charge diameter, energy loss in the chemical reaction zone would prevent the self-sustaining propagation of detonation, resulting in unsuccessful detonation. The influence of charge diameter on detonation propagation is called the diameter effect (Fig. 19).

Sideward rarefaction wave in charge with limited diameter affects the reaction zone of detonation, causing detonation front to change from a flat plane to a curved plane. The timing of the end of chemical reaction inside the detonation front and the time for rarefaction wave to reach the central axis of the charge affect the curvature of the wave front. In other words, the diameter effect is related to the width of chemical reaction zone and diameter of charge (Fig. 20).

Assuming that $\frac{\partial u}{\partial t}=0$ is the critical condition of constant propagation of a self-sustaining detonation, as in unsuccessful detonation would occur when velocity gradient of particles behind the shock wave front is zero, analyzing the flow field in the chemical reaction zone of the explosive behind the shock discontinuity would obtain the critical diameter's expression:

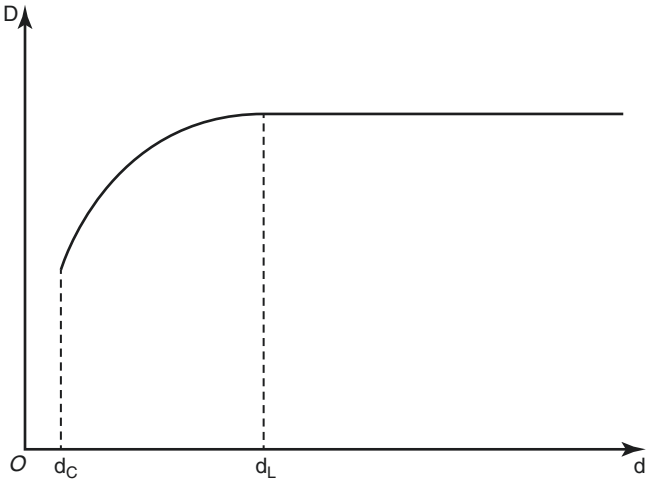

Fig. 19 Relationship between detonation velocity and diameter

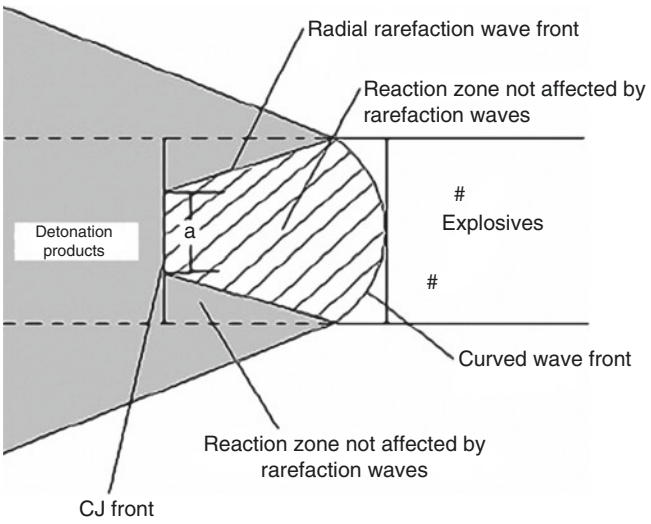

Fig. 20 Effect of detonation wave propagation diameter

$$d_{cr} = \frac{4uc^2 \cos\phi_c}{Q_{Pv}\Gamma W} \tag{77}$$

In the equation, u is particle velocity upstream of the constant detonation wave front; c is the sonic velocity of impacted and compressed explosive, and is known as "frozen sound speed"; ϕ_c is sound velocity angle; Q_{Pv} is heat of reaction under isobaric and isovolumetric conditions; Γ is the Grüneisen constant; and W is initial decomposition speed of explosive right after the shock wave discontinuity. Sonic velocity of impacted and compressed explosive c may be obtained from the equation below:

$$c = \frac{(D-u)(D+\lambda u)}{D} \tag{78}$$

In the equation, λ is empirical constant.

The properties of the explosive, material of the shell containing the charge, initial temperature of the charge, particle size of the explosive, charge density, and other factors also affect critical diameter.

3.2 Propagation of Detonation in Gas Phase and Mixed Phase Explosives

Detonation propagation velocity and detonation pressure are associated with the composition of gas phase and mixed phase explosives. Each kind of gas phase and mixed phase explosive has a best composition ratio, and under such optimal ratio detonation propagation velocity and detonation pressure would reach their maximums.

There are upper and lower limits for the concentration of oxygen or combustibles in the gas phase mixture, and detonation cannot propagate steadily beyond the upper and lower limit concentration. Table 5 shows the concentration limit for several mixtures.

Table 5 Concentration limit for detonation propagation (P_0 = 0.1 MPa, T = 293 K)

	A/%			*A*/%	
Mixture	Lower limit	Upper limit	Mixture	Lower limit	Upper limit
$H_2 + O_2$	15.5	92.9	$C_2H_4 + O_2$	3.5	93
H_2 + air	18.2	58.9	C_2H_4 + air	5.5	11.5
$CH_4 + O_2$	17.0	90.7	$C_3H_6 + O_2$	2.5	50.0
$C_3H_8 + O_2$	8.25	55.8	C_2H_2 + air	4.2	50.0
$D_2 + O_2$	2.50	42.5	$Si(CH_3)_4 + O_2$	1.8	48.0
$C_4H_{10} + O_2$	2.05	37.95			

Note: *A* is proportion of oxygen or combustibles in the air

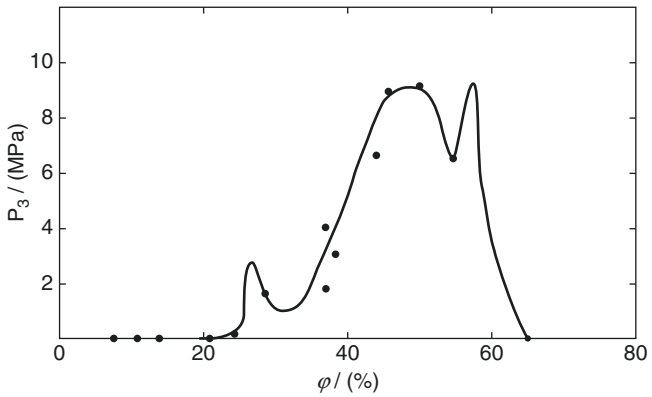

Fig. 21 Relationship between deflagration (or detonation) pressure P of the gaseous mixture of nitromethane and oxygen and the content of nitromethane φ

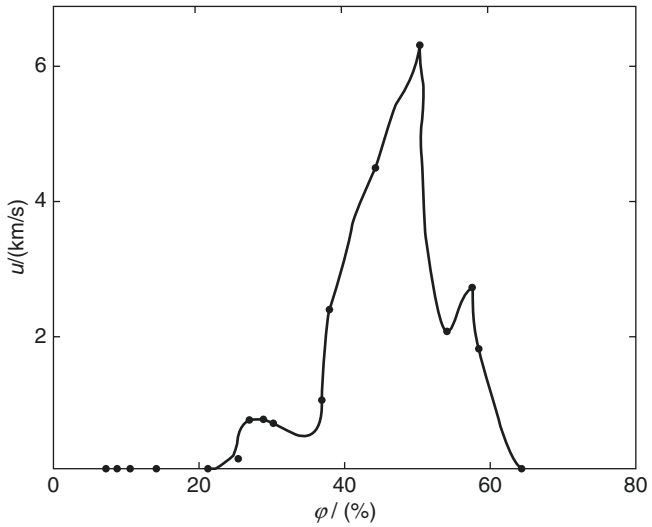

Fig. 22 Relationship between deflagration (or detonation) velocity u of the gaseous mixture of nitromethane and oxygen and the content of nitromethane φ

Unlike experimental results from normal gas phase mixtures (e.g., hydrogen, *n*-heptane), the nitromethane and oxygen gas mixture's deflagration (or detonation) pressure P and speed u have multiple extreme values following increase in nitromethane concentration (Figs. 21 and 22). The predicted reason is perhaps that the energy that breaks the bond between the nitro group and the upper body is relatively high, and only when a certain energy level is achieved would steady and rapid detonation reaction be initiated.

4 Effects of Detonation

4.1 Drive and Loading of Detonation

4.1.1 Drive of Metal Cylinder by Internal Explosion Load

When an internal charge explodes, metal cylinder rapidly expands until breakage under the action of detonation product, creating a field where fragments disperse in all directions and in high speed. Thirty percent of the energy released from the explosion of an explosive is used to break apart the shell and drive the fragments. Taylor proposed the tensile fracture criterion in 1944, and he was of the opinion that cylindrical shell expands radially under the load from explosion of explosive contained inside. Initially, circumferential stress is in a state of compressive stress within the whole range of the thickness of the wall, and as the metal cylinder expands, inner wall pressure is reduced, and areas near the outer wall turn to a state of toroidal tensile stress. The inner wall areas meanwhile remain in a state of compressive stress, and inside the cylindrical shell exists a neutral surface with zero circumferential stress. Cracks emerge in the tensile stress areas on the outer surface of the cylindrical shell. It is assumed that radial cracks propagate only in the circumferential tensile stress area of the shell and can't spread in areas with compressive stress. At first, the neutral surface is located on the exterior of the cylindrical shell, and thereafter, as the shell expands, the neutral surface moves inward, while cracks extend inward from the exterior of the cylindrical shell. When circumferential stress of the shell is fully stretched, neutral surface moves into the inner surface of the shell, or in other words cracks that propagate through the cylindrical shell (Fig. 23).

R.W. Gurney derived a calculation formula for fragment initial velocity driven by shell charge explosion based on the energy conservation equation:

$$v = \sqrt{2E_g}\left(\frac{m_e / m_c}{1 + 0.5\left(m_e / m_c\right)}\right)^{1/2} \tag{79}$$

In the equation, E_g is Gurney energy, which is an assumption of kinetic energy of metal and expansion of detonation product directly converted from chemical energy inside the explosive charge prior to detonation, and reflects to a certain extent the capacity of explosive in driving objects; and m_e and m_c are respectively the mass of the charge and mass of the shell.

Fragment velocity calculated from this equation is usually higher than initial fragment velocity actually measured, and this difference may be analyzed in a simplified manner using physical images of explosion-driven shell expansion and rupture: Blast wave that sweeps over the shell provides the shell with its initial acceleration, and for the cylindrical steel shell, when the shell's radius expands to 1.2 times that of its initial radius, elastoplastic expansion would occur. At this time, the radial velocity of the shell reaches 60% of Gurney velocity. When fragments reach 1.6–1.8 times the shell's initial radius, the fragments stop accelerating and reach their highest velocity, roughly 95–100% of Gurney velocity. And at this time, detonation product shoots out from cracks on the shell, and thereafter a continuously expanding cloud erupts from the ruptured shell. When fragments reach 20 times the warhead's radius, the fragments also reach the last phase of their terminal flight, once again shooting out from the cloud of detonation product. However, due to the deceleration action of the resistance of the cloud, fragment velocity would be dragged down to 90% of Gurney velocity. Subsequent deceleration of fragment is attributed to continual resistance of medium in the environment. Therefore, Gurney velocity is the greatest velocity during the acceleration phase of fragments, and at this time, fragments of the expanded warhead shell reach a location about twice the initial radius of the warhead.

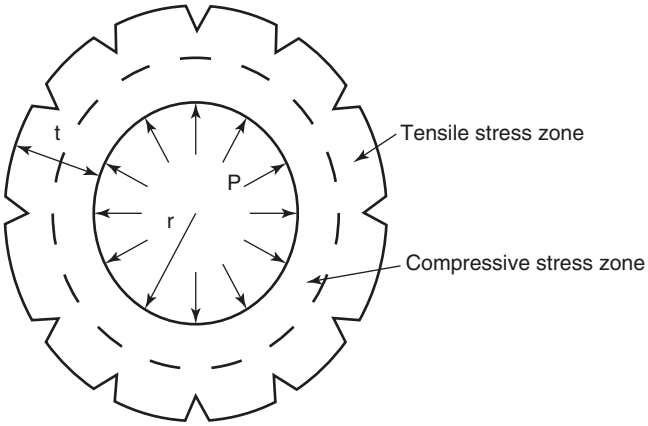

Fig. 23 Tensile stress zone and compressive stress zone in expansion loop

Gurney energy calculation method is usually associated with detonation parameters only, and does not take into consideration neither the effects of the expansion law of detonation product during the expansion process, nor the effects of the shell's metal material on the expansion process. Based on the detonation product's JWL equation of state and Taylor's rupture criterion, Wang Xinying et al. proposed a Gurney energy calculation method that accounts for energy conversion of metal cylinder driven by detonation products, as derived from law of energy conservation with the introduction of parameters from the JWL equation of state and yield strength of metal material. Figure 24 shows curves of cylindrical experiment results and calculation results for pressed TNT cylindrical charge used respectively in oxygen-free copper, type 6061 aluminum and #45 steel.

With high enough detonation pressure, shear band is formed when inner wall of the cylinder is compressed by impact and shear stress is generated. The shear band extends

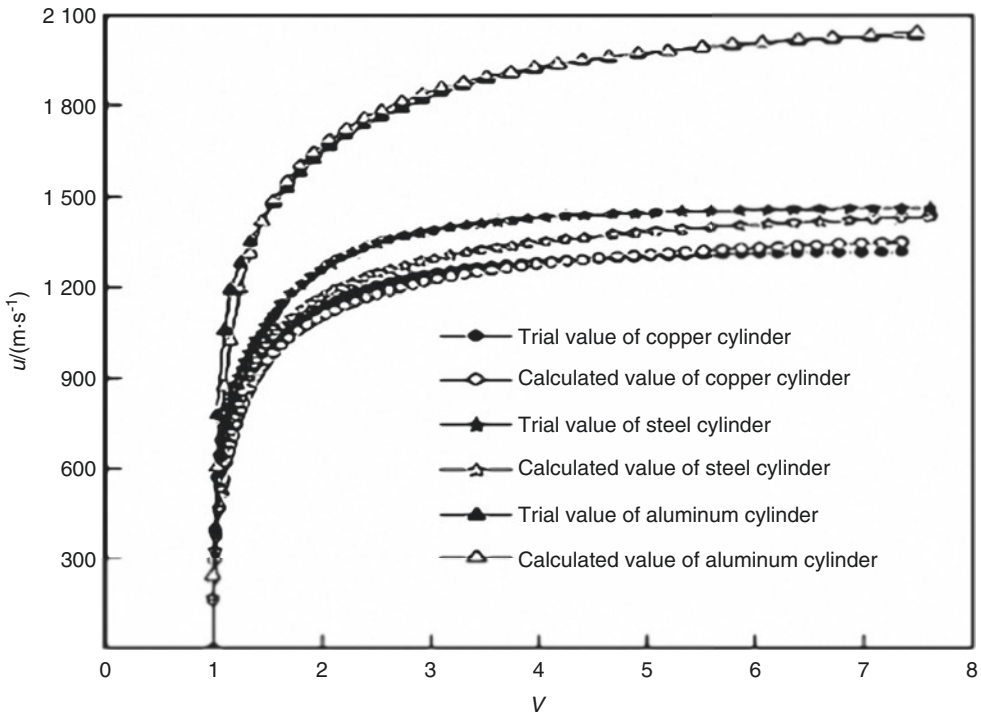

Fig. 24 Expansion velocity and relative volume curves of different material cylinders

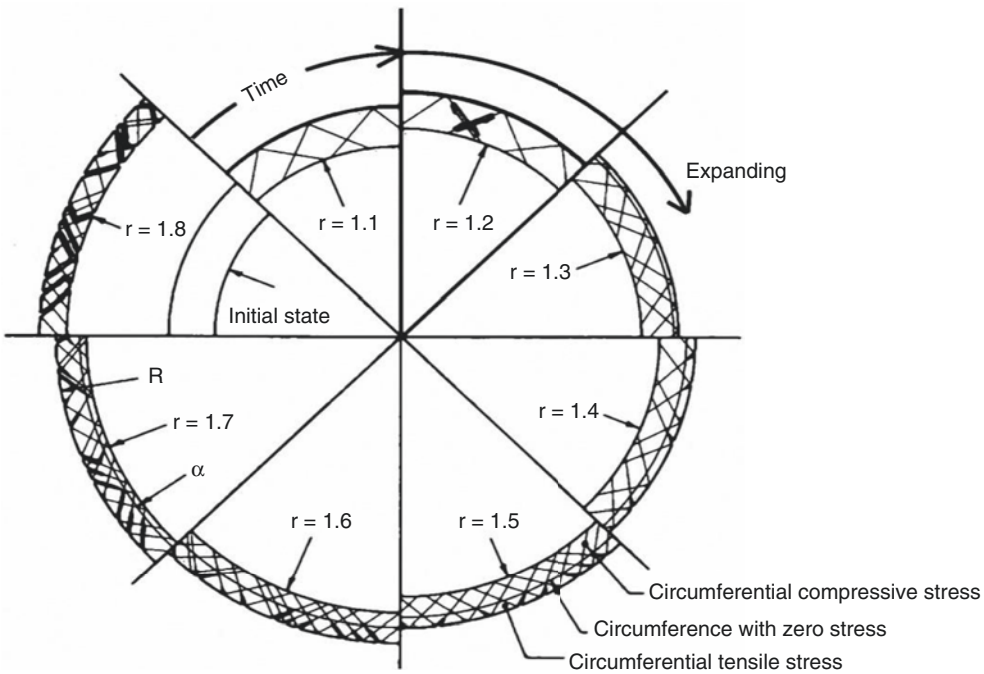

Fig. 25 Shear failure of cylindrical shell

toward the outer wall of the shell as the cylinder expands, and tensile stress causes cracks to form on the outer wall of the cylinder, with the cracks following the formed shear band and extending inward to the inner wall (Fig. 25). When the inner wall's circumferential compressive stress and circumferential tensile stress are equal, and when the two sides of the shear band change from a state of compressive stress to a state of tensile stress, the cylinder would fracture and break. When detonation pressure is relatively low, there is no time for unsteady shear band to form on the inner wall of the cylinder, and Taylor's tensile fracture criterion would be obeyed.

An increasing number of studies show that the destruction models of cylinder are tensile fracture, mixed tensile-shear fracture, or even purely shear fracture, but these depend on the detonation load pressure, size of cylinder, and expansion radius. Some materials show strain rate effect, and even plasticity peak during dynamic fracture.

When steel cylinder reaches a strain rate of around 10^4/s, fracture strains reach max value in what is known as the plasticity peak phenomenon (Fig. 26). Principle of fractures of cylindrical shell:

Assuming that flow stress satisfies the viscosity and flexibility relationships $\bar{\sigma} = \overline{\sigma_0} + \eta\dot{\varepsilon}$, then steel cylinder fractures should satisfy:

$$\mu^2\dot{\varepsilon}^2\varepsilon\left(\varepsilon+2\right)/2+\dot{\varepsilon}\left(2\mu\varepsilon-\alpha\right)+\ln\left(\varepsilon+1\right)=0 \tag{80}$$

In the equation, $\mu = \frac{\eta}{\bar{\sigma}_0}; \alpha = \frac{4E\lambda}{3C\bar{\sigma}_0^2}$. When ε 1, the above equation may be simplified as:

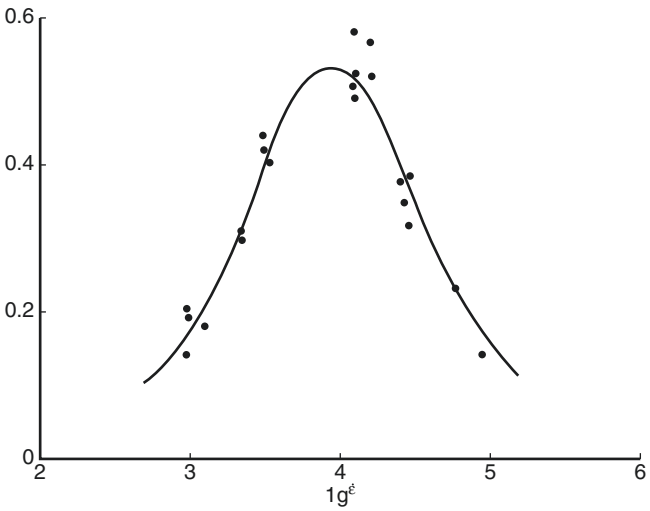

Fig. 26 Plasticity peak in dynamic fracture

$$\varepsilon = \frac{\dot{\varepsilon}\alpha}{\left(1+\dot{\varepsilon}\mu\right)^2} \tag{81}$$

Fracture principle has existing strain ε and strain rate $\dot{\varepsilon}$, and when $\dot{\varepsilon} = \frac{1}{\mu}$, fracture strain is at maximum, as in $\varepsilon = \frac{\alpha}{4\mu}$.

The Gurney equation calculates average initial velocity of detonation-driven fragments, but to calculate the distribution

of initial velocity of fragments, a modified Gurney equation may be adopted:

$$V_x = (2E)^{1/2}\left[F(x)m_e / M\right]^{1/2}\left[1+\frac{1}{2}F(x)m_e / M\right]^{-1/2} \quad (82)$$

In the equation, $F(x)$ is correction factor; d denotes charge diameter; L symbolizes charge length; and x represents specific location along the axis of the warhead. Figure 27 shows initial fragment velocity distribution of American projectiles of four different calibers.

Fragment velocity distribution based on the principle of impulse distribution:

$$V_\alpha = V_{\max}\left(i_\alpha / i_{\max}\right)^n, \quad 0<n<1 \quad (83)$$

In the equation, i is explosion impulse; subscript α expresses relative position of observation; n is empirical correction factor; for the common form of detonation initiated from one end, explosion impulse is distributed along the length of the axis of the charge:

$$i_\alpha = \frac{i_0}{8}\left[1+6\alpha(1-\alpha)+\frac{3\alpha}{2}\ln\left(\frac{3-2\alpha}{\alpha}\right)+6\alpha(1-\alpha)(2\alpha-1)\ln\left(\frac{3-2\alpha}{2-2\alpha}\right)\right] \quad (84)$$

In the equation, $i_0 = (8/27)\rho_0 lD$ represents the impulse per unit area on the surface of end with the charge; ρ_0 is initial density of charge; and D detonation velocity of charge. Relative position α of maximum impulse $i_{\max}$ may be obtained based on $\frac{di_\alpha}{d\alpha}=0$:

$$6-12\alpha+\frac{3}{2}\left(\ln\frac{3-2\alpha}{\alpha}-\frac{3}{3-2\alpha}\right)+6\left[\ln\frac{3-2\alpha}{2(1-\alpha)}\cdot\left(-6\alpha^2+6\alpha-1\right)-\frac{2\alpha^3-3\alpha^2+\alpha}{(1-\alpha)(3-2\alpha)}\right]=0$$

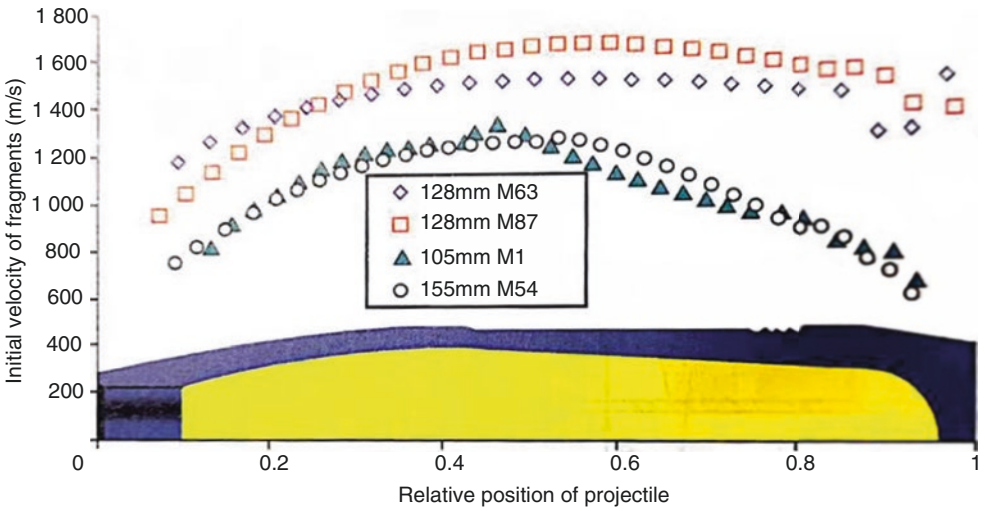

Fig. 27 Initial fragment velocity distribution of projectiles of four different calibers

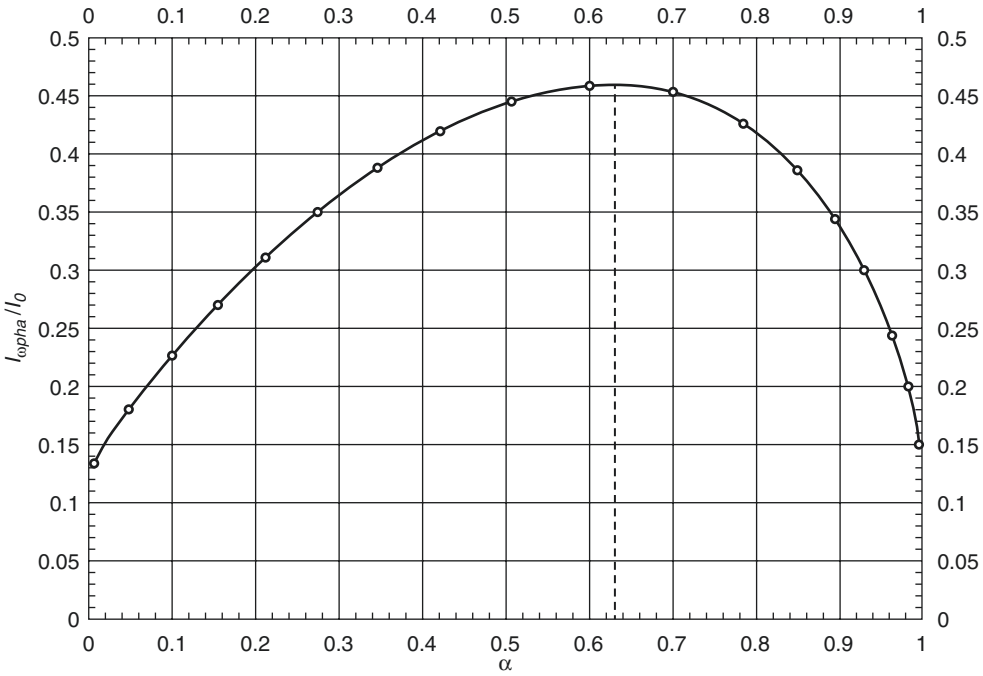

Fig. 28 Distribution of explosive impulse along the axis

The said equation has no explicit solution, and approximate solution obtained from numerical method is $\alpha = 0.62927$. Figure 28 illustrates distribution of explosive impulse along the axis.

4.1.2 Drive of Flat Plate by Detonation

1. **One-dimensional ejection of flat plate by detonation product.** Assuming that flat plate is a rigid object, that detonation wave reflected from interface of flat plate is weak shock wave, and that adiabatic index of equation of state of detonation product is 3, then the relationship between motion distance and time of detonation-driven one-dimensional ejection process of flat plate is (Fig. 29):

$$x = Dt\left(1+\frac{\theta-1}{\eta\theta}\right) \tag{85}$$

In the equation, x is distance of movement of flat plate; t denotes time; $\eta = \dfrac{16m}{27M}$, of which, m is mass of charge, $m = \rho_0 lS$, ρ_0 is density of charge, l is length of charge, and S represents cross-sectional area of flat plate; M is mass of flat plate; and $\theta = \left\{1+2\eta\left[1-2\eta\left(1-\dfrac{l}{Dt}\right)\right]\right\}^{1/2}$. From $u = \dfrac{\mathrm{d}x}{\mathrm{d}t}$, flat plate's movement velocity u may be obtained:

$$u = D\left(1+\frac{\theta-1}{\eta\theta}-\frac{l\theta}{Dt}\right) \tag{86}$$

Detonation-driven flat plate ejection velocity given by the Taylor model is:

$$u = \sqrt{2E_g}\left(\frac{\left(1+2\dfrac{m_p}{m_e}\right)+1^3}{6\left(1+\dfrac{m_p}{m_e}\right)}+\frac{m_p}{m_e}\right)^{-1/2} \tag{87}$$

In the equation, m_p is mass of flat plate; and m_e is mass of charge.

2. **Two-dimensional ejection of flat plate by detonation product**. The one-dimensional ejection model can only analyze the movement velocity of flat plate, but if to understand the movement process and movement posture of ejected flat plate, then the flat plate can no longer be treated as a rigid object. Assuming that the flat plate is approximate to an incompressible fluid, that the thickness of the plate remains the same during its movement, that explosion product satisfies polytropic index equation $Pv^{\gamma} = \text{constant}$, and that the flat plate is large enough that effects from rarefaction wave along the edges of the plate could be ignored (Fig. 30), then it may be derived that:

$$V_p = 2D\sin\frac{1}{2}\left\{\theta_k\left[1-\exp\left[-\frac{m_e t}{\theta_k t_e D m_p(\gamma+1)}\right]\right]\right\} \tag{88}$$

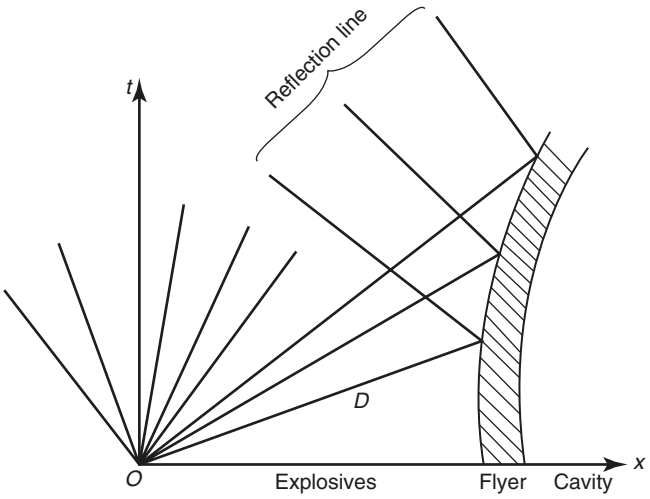

Fig. 29 One-dimensional ejection of flat plate by detonation product

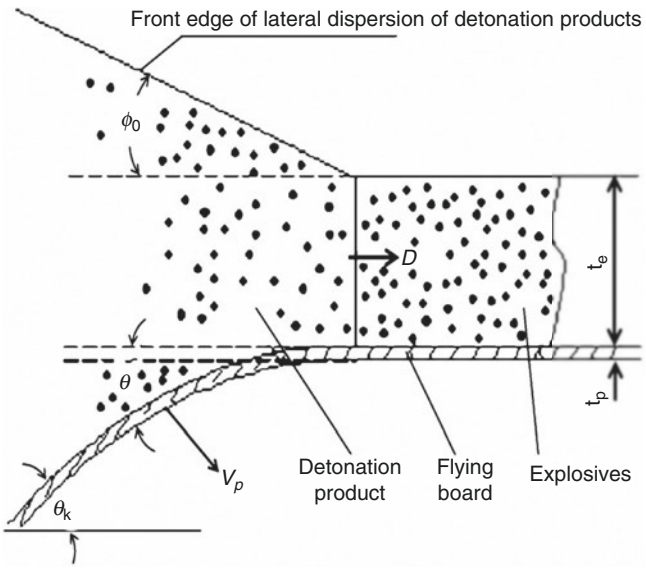

Fig. 30 Two-dimensional ejection of flat plate by detonation product

$$V_{p_{max}} = 2D\sin\frac{1}{2}\left(\frac{1}{b+\bar{c}m_p/m_e}\right) \tag{89}$$

In the equation: γ denotes polytropic isentropic index of detonation product; m_p is mass of flat plate; and m_e is mass of charge; $b=\dfrac{\sqrt{3}}{4}\dfrac{1}{\sqrt{1-\gamma\sqrt{\gamma^2-1}}}$; and $\bar{c}=\dfrac{\sqrt{3}}{2}\sqrt{\dfrac{\gamma^2-1}{\gamma^2-\gamma\sqrt{\gamma^2-1}}}$.

Law of changes of deflection angle θ following changes in time t:

$$\theta=\theta_k\left\{1-\exp\left[-\frac{m_pDt}{\theta_k t_e m_e(\gamma+1)}\right]\right\} \tag{90}$$

Relationship between flat plate's radial displacement x and deflection angle θ:

$$x=(\gamma+1)\frac{\theta_k t_e m_p}{m_e}\int_0^{\theta}\frac{\cos\theta}{\theta_k-\theta}\mathrm{d}\theta \tag{91}$$

4.2 Explosion in Air

4.2.1 Formation of Blast Wave

After an explosive explodes in the air, explosion product rapidly expands, and when the blast wave propagates to the interface between the explosive and the air, the air would abruptly compress and create a strong shock wave. When the detonation product stops expanding, the air shock wave separates from detonation product and independently propagates forward. When the shock wave reaches a certain point, pressure would suddenly rise to overpressure peak value. At the same time, particle velocity and dynamic pressure, as well as other parameters of the medium, also jump because of the shock wave front and reach their peak values.

During the propagation process of a shock wave, the wave front propagates at supersonic velocity, while the tail of the positive pressure zone moves at a sonic velocity relative to static atmospheric pressure. Therefore, the positive pressure zone is continually being extended. What actually transpires is that after reaching peak value, overpressure would decline to zero at a rate approximate to exponential law, while dynamic pressure drops even faster. Expansion of air near the center of explosion causes the pressure here to continue to decline, so much so that pressure falls below static atmospheric pressure of wave front, thereby creating negative pressure. Particle velocity meanwhile changes from its original direction with shock wave movement to direction opposite of shock wave motion. Negative pressure gradually decreases to negative pressure peak value, and particle velocity running in the opposite direction also reaches near peak value, then gradually returns to zero, as in restoring the state of static atmospheric pressure. Dynamic pressure decreases even faster than overpressure, but at a specific location, the duration of action of dynamic pressure wind in the direction of propagation lasts longer than the positive pressure of overpressure (Fig. 31).

Shock wave propagates at supersonic velocity, and as the shock wave's spherical front enlarges, energy is continually being depleted, and the velocity, pressure, and energy of the shock wave also attenuate very quickly as distance increases. The pressure, density, and other aspects upstream

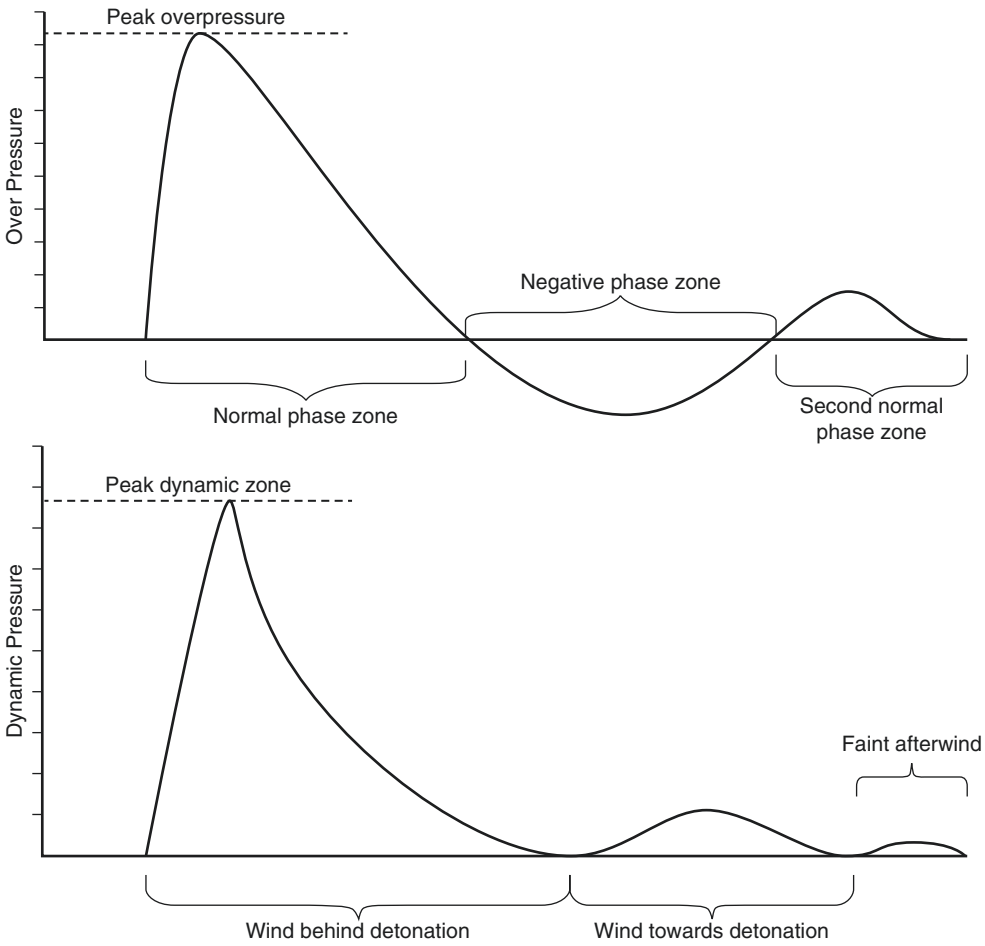

Fig. 31 Changes in shock wave and dynamic pressure of air explosion over time

of the shock wave front gradually decline, and pressure and density a certain distance behind the shock wave decrease to the pressure and density of the pre-disturbance medium, may be even falling below the pressure and density levels of the pre-disturbance medium. At (10–15) r_0 location away from the center of explosion, pulse propagation velocity nears the speed of sound (Fig. 32).

4.2.2 Similarity Theory in Explosion

When an explosive detonates in the air, due to the extremely short span of the detonation process, the influences of the medium's viscosity and thermal effect on shock wave strength can be ignored, and the overpressure of the shock wave may be expressed as:

$$\Delta P = f\left(E_0, P_0, \rho_0, \kappa, R\right) \tag{92}$$

In the equation, E_0 is energy of explosive; P_0 denotes initial pressure of the air; ρ_0 is density of the medium; κ represents adiabatic index; and R is distance from location of explosion.

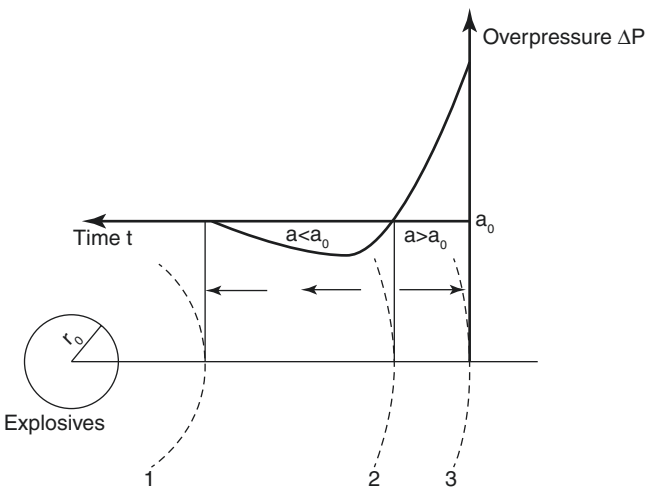

Fig. 32 Pressure distribution of shock wave. Dotted line 1 represents the interface of product of explosion; dotted line 2 denotes interface between compressed zone and rarefaction zone; dotted line 3 is front of the shock wave; c_0 symbolizes sonic velocity of undisturbed medium; and arrow is direction of particle movement upstream of the shock wave front

Based on the π law, choose the respective unit of the mutually independent quantities of E_0, P_0, and ρ_0 as basic units, and derive the following equation:

$$\Delta P = f\left(\frac{m_{\mathrm{R}}^{1/3}}{R}\right) \tag{93}$$

Expand the above equation in a polynomial manner:

$$\Delta P = A_0 + A_1\left(\frac{m_{\mathrm{R}}^{1/3}}{R}\right) + A_2\left(\frac{m_{\mathrm{R}}^{1/3}}{R}\right)^2 + A_3\left(\frac{m_{\mathrm{R}}^{1/3}}{R}\right)^3 + \cdots \tag{94}$$

Boundary conditions: $R \to \infty$, $\Delta P = 0$, and $A_0 = 0$.

In the equation, m_{R} is TNT equivalent of the explosive. Given $Z = \dfrac{R}{m^{1/3}}$, Z is called scaled distance. From the perspective of engineering, if a three-phase polynomial equation is obtained, then precision requirement is satisfied. Therefore, the above equation can be changed to:

$$\Delta P = \frac{A_1}{Z} + \frac{A_2}{Z^2} + \frac{A_3}{Z^3} \tag{95}$$

In the equation, A_1, A_2, and A_3 are coefficients confirmed through experiment. Shock wave peak value overpressure ΔP is only associated with scaled distance Z.

For spherical charge that explodes in infinite air medium, different researchers have put forth different suggestions with regard to the value of A_1, A_2, and A_3 coefficients due to differences in test and analysis method. The following may be considered:

$$\Delta p = \frac{0.082}{Z} + \frac{0.26}{Z^2} + \frac{0.69}{Z^3}, \quad Z \le 1 \tag{96}$$

$$\Delta p = \frac{0.076}{Z} + \frac{0.255}{Z^2} + \frac{0.65}{Z^3}, \quad 1 < Z \le 15 \tag{97}$$

Similar to overpressure ΔP, a mid-air explosion blast wave's positive pressure duration t_+ and specific impulse i also satisfy similarity rate. Similarity theory of explosion is thus obtained. In other words, when explosives of the same type with similar geometrical shapes but different sizes explode under identical atmospheric conditions, they generate self-similar blast waves at equal scaled distance. According to similarity theory of explosion, outcomes of experiments conducted with small charges can predict the properties of blast waves generated from larger charges.

Finite reflex impulse measurements show that when $Z < 0.16$ m/kg$^{1/3}$, similarity theory of explosion might become inapplicable.

The properties of blast waves from condensed, high-energy explosives are clearly similar to those generated by TNT, and the explosion parameters of other explosives may be calculated using explosives with explosion effects similar to spherical TNT. This is called TNT equivalent. In general, the equivalent factor is used in relative comparisons, and data are sourced from comparisons of air explosion data of different high-energy explosives. The changes in these data are not related to scaled distance, and do not rely on peak value overpressure or lateral impulse. When actually comparable explosion data do exist, averaging these data can confirm a specific TNT equivalent figure. When these data are unavailable, compare the values of heat of explosion Q of TNT and the target explosive to predict said explosive's TNT equivalent. Table 6 lists TNT equivalent coefficients for the explosion of some explosives in infinite air medium, to be used for calculating overpressure and impulse. Pressure

Table 6 TNT equivalent coefficients for different explosives

Explosive	TNT equivalent coefficients (overpressure)	TNT equivalent coefficients (impulse)	Pressure range/psi
Ammonium nitrate-fuel oil mixture	0.82	–	1–100
A-3 explosive	1.09	1.076	5–50
Composition B explosive	1.11 1.20	0.98 1.3	5–50 100–1000
C-4 explosive	1.37	1.19	10–100
Cyclotol (70/30)	1.14	1.09	5–50
HBX-1 aluminized explosive	1.17	1.16	5–20
HBX-3 aluminized explosive	1.14	0.97	5–25
H-6 aluminized explosive	1.38	1.15	5–100
Minol II	1.20	111	3–20
Octol (70/30, 75/25)	1.06	–	–
Polymer bonded explosive-9404 (PBX-9404)	1.13 1.7	– 1.2	5–30 100–1000
Polymer bonded explosive-9010 (PBX-9010)	1.29	–	5–30
Pentolite	1.42 1.38 1.50	1.00 1.14 1.00	5–100 5–600 100–1000
Composition D explosive	0.90	0.93	–
Tetryl	1.07	–	3–20
Tetrytol (75/25, 70/30, 65/35)	1.06	–	–
TNETB	1.36	1.10	5–100
TNT	1.00	1.00	Base value
Tritonal	1.07	0.96	5–100

generated from movement of the air's mass point itself, as in dynamic pressure of blast wave, is:

$$p_{\mathrm{d}} = \frac{1}{2}\rho u^2 \tag{98}$$

In the equation, ρ denotes air density, and u is blast wind (mass point) velocity. Figure 33 shows overpressure and dynamic pressure of 1 kg TNT explosion.

Experiments of impact of detonation parameters of RDX/Al and HMX/Al mixed explosives with different aluminum contents on overpressure of blast wave from air explosion show that the heat of explosion, detonation velocity, and volume of explosion gases have identical influences on air explosion blast wave overpressure. Overpressure is expressed as below:

$$\Delta P_{\mathrm{m}} = a\left(\frac{QVD}{Q_{\mathrm{T}}V_{\mathrm{T}}D_{\mathrm{T}}}\right)^{\frac{1}{3}}, \quad 1.8 \le Z \le 4.5 \tag{99}$$

In the equation, ΔP_{m} is shock wave overpressure of mixed explosive; Q, V, and D are respectively heat of explosion, volume of explosion gases, and detonation velocity of mixed explosive; Q_{T}, V_{T}, and D_{T} are respectively heat of explosion, volume of explosion gases, and detonation velocity of TNT; Z is scaled distance and denotes constants related to explosive type and detonation parameter calculation method, with a = 1 for TNT, a = 1.053 for RDX/Al, and a = 1.073 for HMX/Al.

4.2.3 Obstacle's Reflection, Transmission, and Diffraction of Blast Wave

The blast wave air propagation circumstance given above does not include any obstacles, but when blast wave propagation in the air reaches obstacles, then reflection, transmission, and diffraction would occur just as any other waves would.

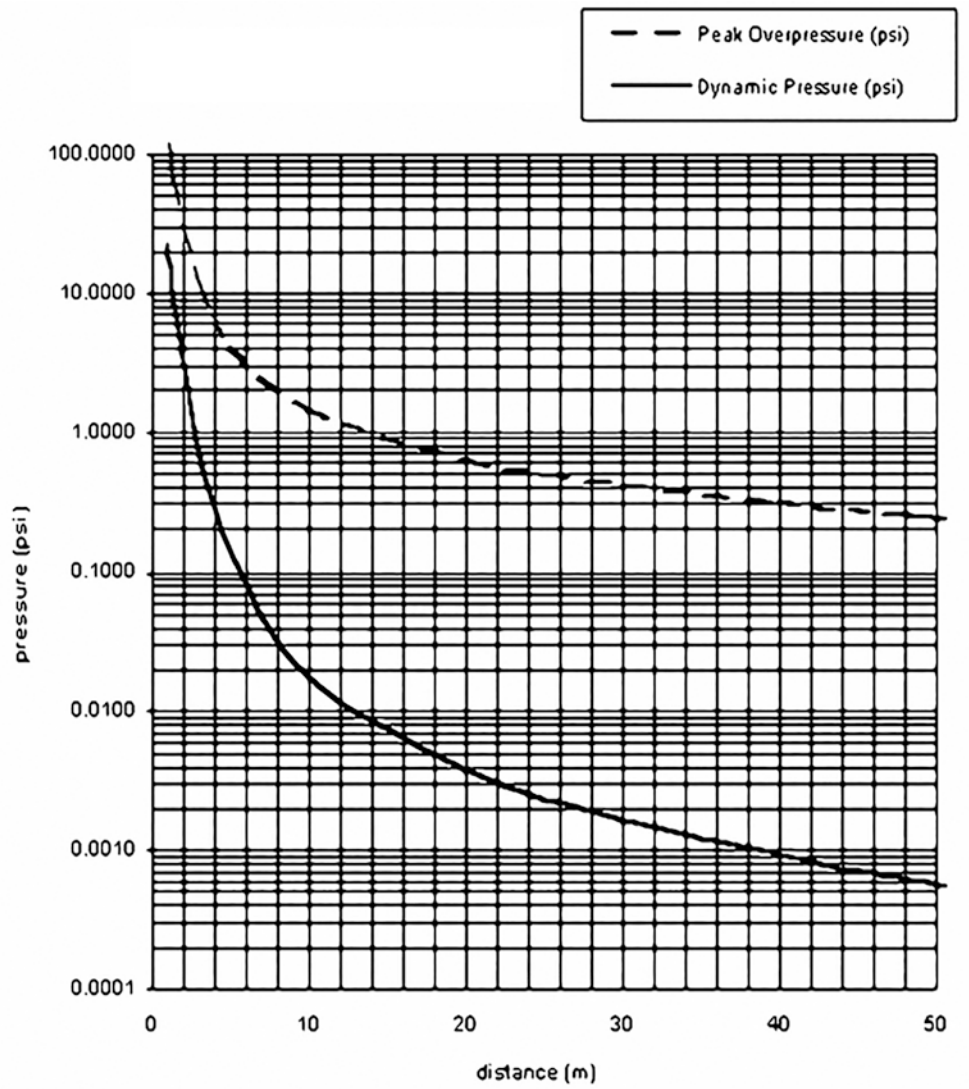

Fig. 33 Overpressure and dynamic pressure of 1 kg TNT explosion

1. **Reflection of shock wave.** Reflection of shock wave might be a positive reflection, oblique reflection, or Mach reflection.

The first type of reflection is positive reflection. When a shock wave vertically enters the plane of a rigid wall (normal direction of the incident wave front is perpendicular to the surface of the obstacle), the air mass point velocity at the surface of the wall would plunge to zero, air mass point would converge on the wall surface, causing rapid rise in pressure and density, and when these reach a certain extent, mass point would propagate in the opposite direction and create positive reflection (Fig. 34). Assuming that incident wave and reflection wave are constant, that parameter of air before disturbance is P_0, ρ_0, $u_0 = 0$, that parameter of incident wave front is P_1, ρ_1, u_1, and that incident wave velocity is D_1, then incident wave overpressure is $\Delta P_1 = P_1 - P_0$; and assuming that parameter of reflection wave front is P_2, ρ_2, u_2, that rigid wall's boundary condition is $u_2 = 0$, that reflection wave velocity is D_2, and that its direction of propagation is opposite to incident wave, then reflection wave overpressure is $\Delta P_2 = P_2 - P_1$.

Overpressure of reflection shock wave:

$$\Delta P_2 = 2\Delta P_1 + \frac{6\Delta P_1^2}{\Delta P_1 + 7P_0} \tag{100}$$

For weak shock wave, since $P_1 - P_0 \ll P_0$, so $\frac{\Delta P_2}{\Delta P_1} \approx 2$; for strong shock wave, $\frac{\Delta P_2}{\Delta P_1} \approx 8$. In other words, under ideal conditions, when an air explosion's blast wave encounters a rigid wall in its process of propagation, the generated reflection wave would have an overpressure two to eight times the overpressure of the incident wave. However, if influences of the high temperature and high pressure generated by strong shock wave are taken into account, air can no longer be treated as a perfect gas. Moreover, if dissociation, ionization, and other effects of the actual gas are also considered, $\frac{\Delta P_2}{\Delta P_1}$ would be much larger, sometimes 20 times or even higher.

The second type of reflection is oblique reflection. When a shock wave does not vertically enter the plane of a solid wall and instead enters at an angle of incidence (included angle between normal direction of the incident wave and the surface of the reflecting solid wall), there exists a critical angle of incidence φ_{cr}. When $\varphi_1 < \varphi_{cr}$, an oblique reflection of the shock wave would occur (Fig. 35).

In the diagram, φ_1, φ_2 represents angle of incidence and angle of reflection, and region (1) represents undisturbed region; (2) is region where incident wave has already propagated through, but not yet reached by reflection wave; and (3) denotes region where reflection wave has already propagated through. Overpressures of oblique reflection ΔP_2 and incident wave ΔP_1 are related to incident angle φ_1:

$$\Delta P_2 = \left(1 - \cos\varphi_1\right)\Delta P_1 + \frac{6\Delta P_1^2}{\Delta P_1 + 7P_0}\cos^2\varphi_1 \tag{101}$$

Under normal circumstances, the value of reflection angle φ_2 does not equal angle of incidence φ_1.

The third type of reflection is Mach reflection. When oblique reflection $\varphi_1 > \varphi_{cr}$, the solid wall first pushes the shock wave to a certain distance away from the wall, the incident wave and reflection wave combine to form the Mach wave in a phenomenon known as Mach reflection (Fig. 36). Wave combined on a vertical surface in front of a solid wall is called the Mach wave. The incident wave, reflection wave, and Mach wave's point of intersection O_1 is called triple point. The triple point is located at a certain distance from the wall surface, and slip line past the triple point is the boundary of area with the same pressure.

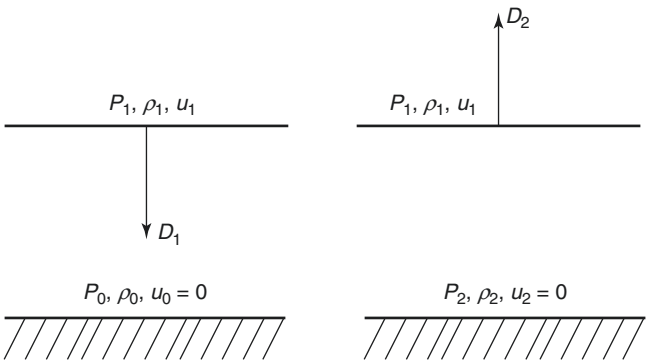

Fig. 34 Normal reflection of shock wave

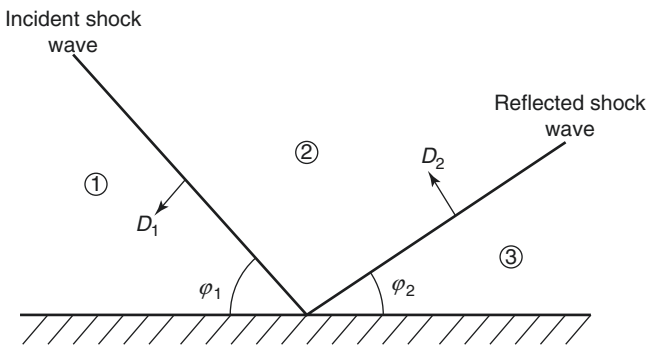

Fig. 35 Oblique reflection of shock wave

Figures 37 and 38 respectively show how the angle of incidence influence of reflected pressure peak value $P_{r\alpha}$ and reflected impulse $i_{r\alpha}$, of which, straight entry into the wall has an angle φ_1 of 0° (as in normal direction), and propagation parallel to the wall has angle of 90°. A normal direction reflected blast wave usually has features that restrict the upper limit of explosion load on structure, and situations regarding oblique load should also be considered.

Blast wave generated a certain distance from the ground should have angle of incidence that changes from normal direction to one with a tilt. Figure 39 shows reflection of strong shock wave at a reflection surface. I_1, I_2, and I_3 are expanding shock waves, and the contour lines of "R" respectively represent reflections of the various shock waves from plane. When I_1 just came into contact with plane S, the strength of the reflection wave is twice that of the incident wave. As the shock wave continues to propagate outward, the respective intersections between each I and the corresponding R is shown as dotted line. Reflected shock wave and incident shock wave combine to form the Mach stem. As the shock wave expands, the Mach stem also continually elongates, ultimately surrounding the reflected shock wave and incident shock wave above.

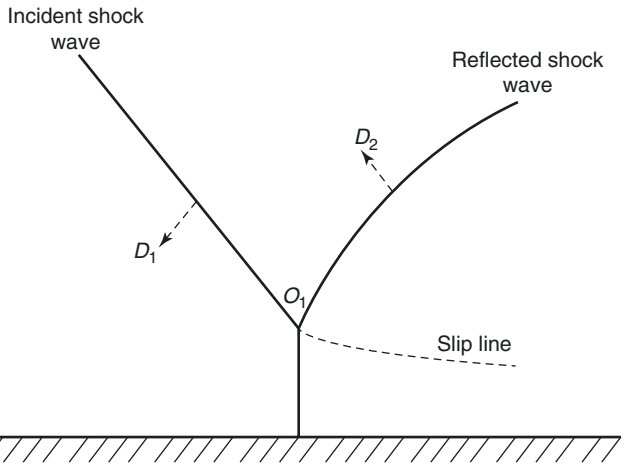

Fig. 36 Mach reflection of shock wave

Figure 40 also shows influence of reflection surface on shock wave, and illustrates shock wave waveforms at typical positions.

Critical angle of incidence φ_{cr} is not fixed because it is a function of the incident wave, or inversed proportional explosion height (Fig. 41).

When the explosion occurs on the ground or near the ground surface, calculation of blast wave parameters ought to account for the intensification effect from the ground reflection. A simple approximation calculation method is based on the hardness of the ground surface, namely multiplying the charge quantity in blast wave parameter equation by a factor of 1.8–2.

2. **Transmission of shock wave**. If a shock wave encounters an obstacle that is not a solid wall but a medium with a certain degree of density during its propagation, the shock wave would transmit into said medium and create transmission wave. Based on the obstacle medium's wave resistance, reflected shock wave or reflected rarefaction wave might be formed from the wall surface toward the original medium of propagation.

Under certain conditions, the transmission of air blast wave in a medium will create shock wave overpressure

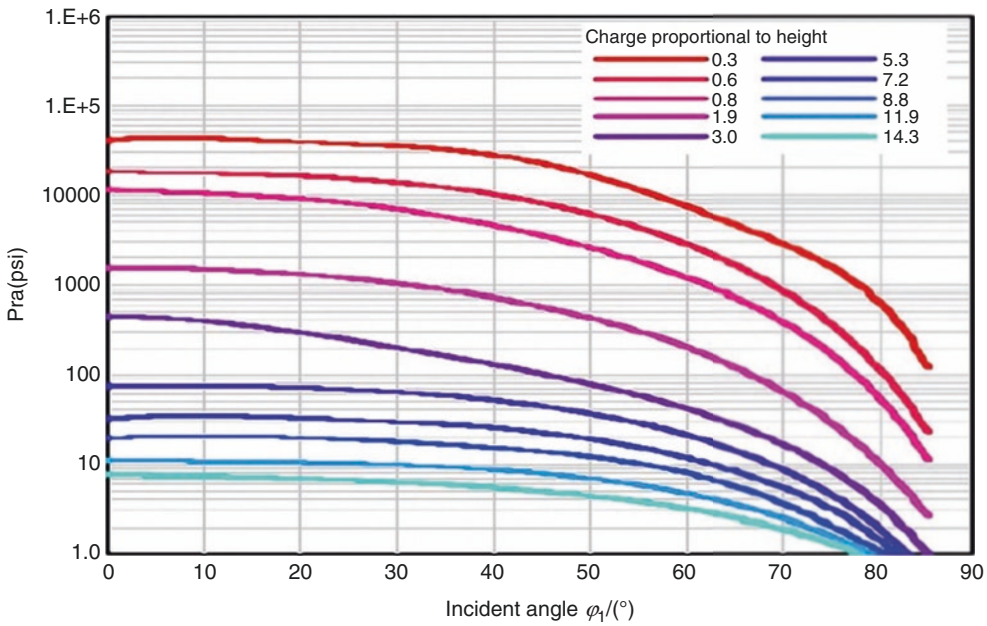

Fig. 37 Relationship between reflected pressure and incident angle

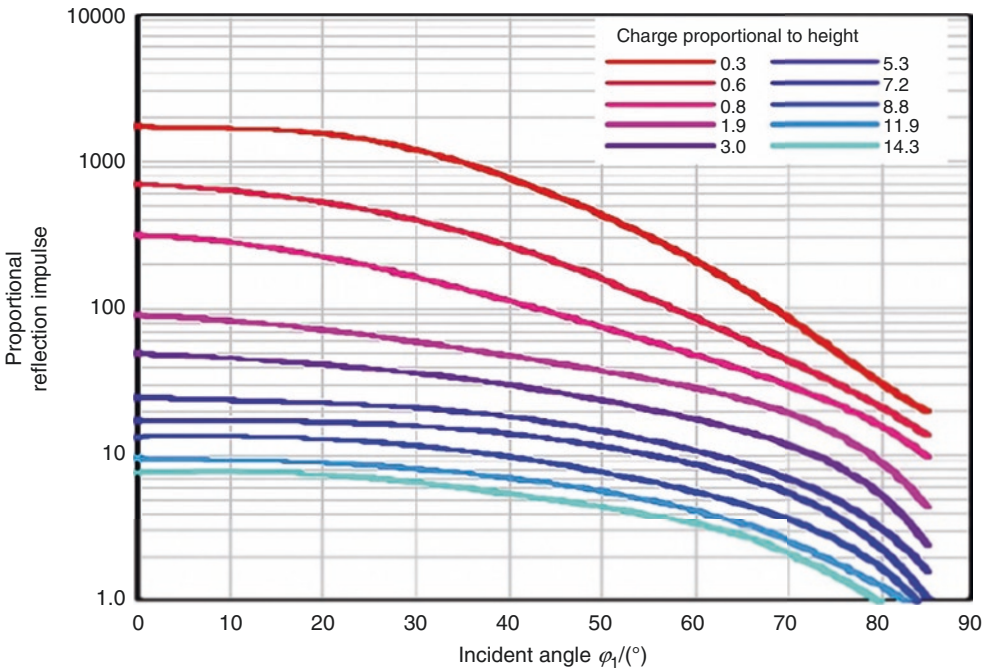

Fig. 38 Relationship between proportional reflection impulse and incident angle

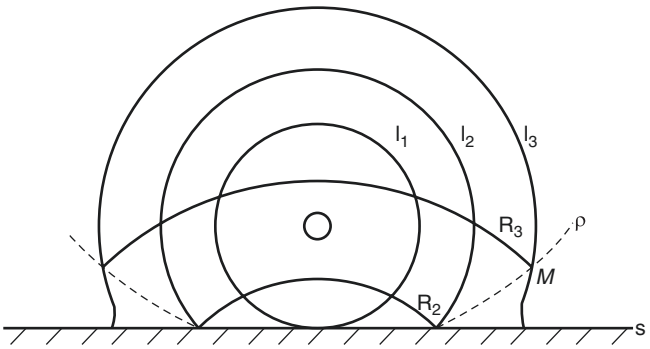

Fig. 39 Effect of reflection front on strong shock wave

peak value in the cavity behind the surface of (inside) the target that could result in blast injury.

3. **Diffraction of shock wave**. The reflections of shock wave mentioned previously assume that the obstacle is of infinite size. If during its propagation, a shock wave encounters an obstacle that is not of infinite size, other than creating reflected shock wave, the shock wave would also bend around the obstacle and create a diffracted shock wave (Fig. 42).

If a shock wave acts on a wide but not tall obstacle, a shock wave that enters the obstacle vertically would reflect upon hitting the front wall of the obstacle, causing the overpressure on the front wall to abruptly increase. However, when the incident wave does not encounter any more obstruction beyond the top edge of the front wall, overpressure would not increase, in turn creating overpressure difference, leading to generation of air flow and wave. While air flows from the high-pressure zone at the front wall toward the low-pressure zone outside the edge of the front wall, simultaneously air in the high-pressure zone is gradually attenuated from the edge area inward, creating rarefaction wave. Under the action of rarefaction wave, air at the surface of the wall moves upward, and the direction of this movement is changed because of air behind the incident wave at the top of the obstacle. This in turn creates moving vortex and circulation that propagates forward (Fig. 42a). When the circulation bends behind the diffracting obstacle and continues moving, it would collide with incident shock wave and result in higher pressure. Collision between diffracted shock wave and incident shock wave would create a new shock wave that continues to propagate. At this juncture, the circulation further develops, bends around the top of the obstacle and follows the back of the wall as it moves downward. Then, the back wall is put under increasing pressure, and due to the effect of the rarefaction wave at the front wall, pressure upstream of the reflection wave drops rapidly (Fig. 42b).

Circulation follows the back wall and continues to travel downward until reaching the ground. Thereafter, circulation follows the ground and moves forward, and forms Mach reflection behind the obstacle's back wall at a distance roughly equal to two times that of the height of the obstacle (Fig. 42c).

On the other hand, if the shock wave acts on a high but not wide obstacle, circulation would occur at both sides of the obstacle. The two circulation flows that bend behind the obstacle would collide, and pressure in the area of collision would rise (Fig. 43). In the diagram, 1 is

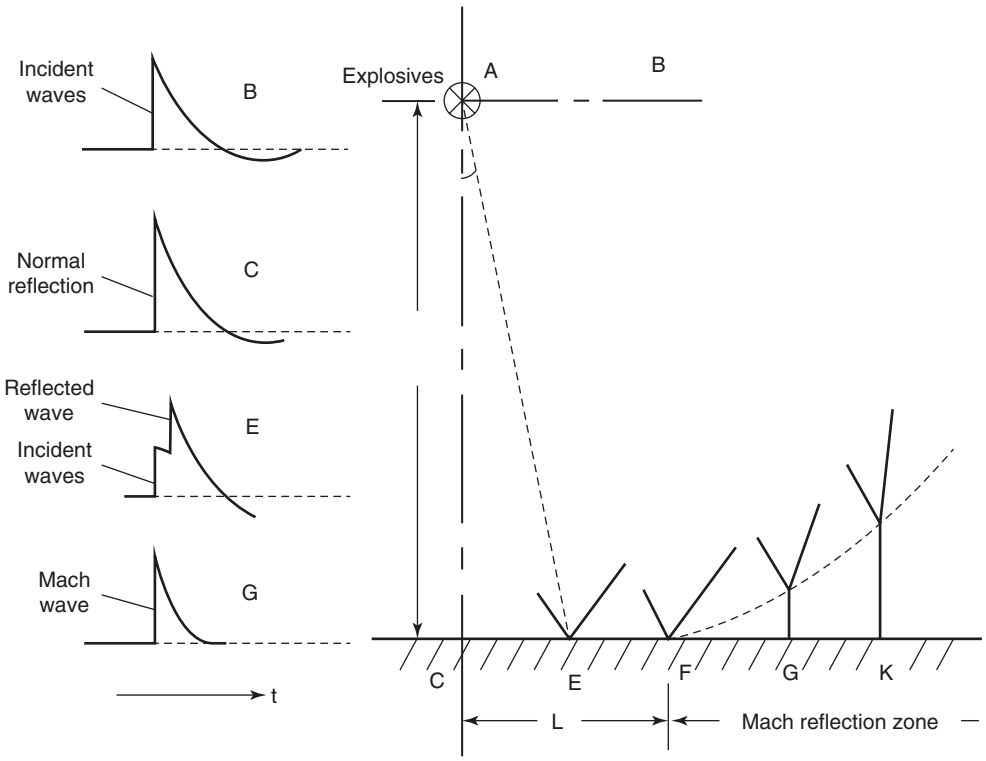

Fig. 40 Reflection on ground of shock wave of air explosion

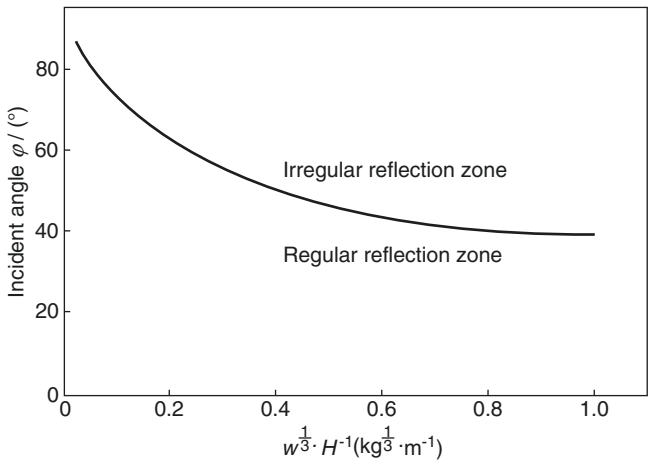

Fig. 41 Relationship curve between the critical incident angle and inversed proportional explosion height

the shock wave, 2 is the vortex, 3 is the reflection shock wave, and 4 is the rarefaction wave.

For an obstacle that is neither high nor wide, diffraction would simultaneously occur at the top and two sides of the obstacle. An area where the three waves collide will build up very high pressure and appear at a certain distance behind the back wall of the obstacle.

In light of the above, diffraction(s) of shock wave would generate a high-pressure region at a certain distance behind the obstacle, and the damage of the shock wave within a certain area behind the obstacle might be even more powerful than if there was no obstacle. Therefore, when choosing obstacle to use as defense against shock wave, it is necessary to pay attention to the shape and size of obstacle, as well as the position of where personnel take shelter.

4. **Effects of external blast wave on architectural structures**. When an explosive explodes on the ground a certain distance away from a structure, it would create a strong shock wave that propagates outward. The shock wave first arrives at the structure's blast load face (the side of the structure facing the direction of the blast). From the previous section, it is known that the reflection of the ground would intensify the strength of the shock wave compared with an unobstructed blast in the air. The action of the shock wave's load on the structure's blast load face generates a pushing thrust on the wall surface and load-bearing structure. The shock wave would shatter glass, and the wall surface of the building might be damaged. A complex series of shock wave entry, reflection, and diffraction would occur inside the building, but the overall effect is that when the shock wave enters the interior, it would create upward pushing thrust on the floors and roof. Once the blast wave surrounds the whole building, the roof would have to bear downward pressure, while the four walls and load-bearing structure of the building have to handle inward pressure (Fig. 44). Generally speaking, blast wave would cause local failures

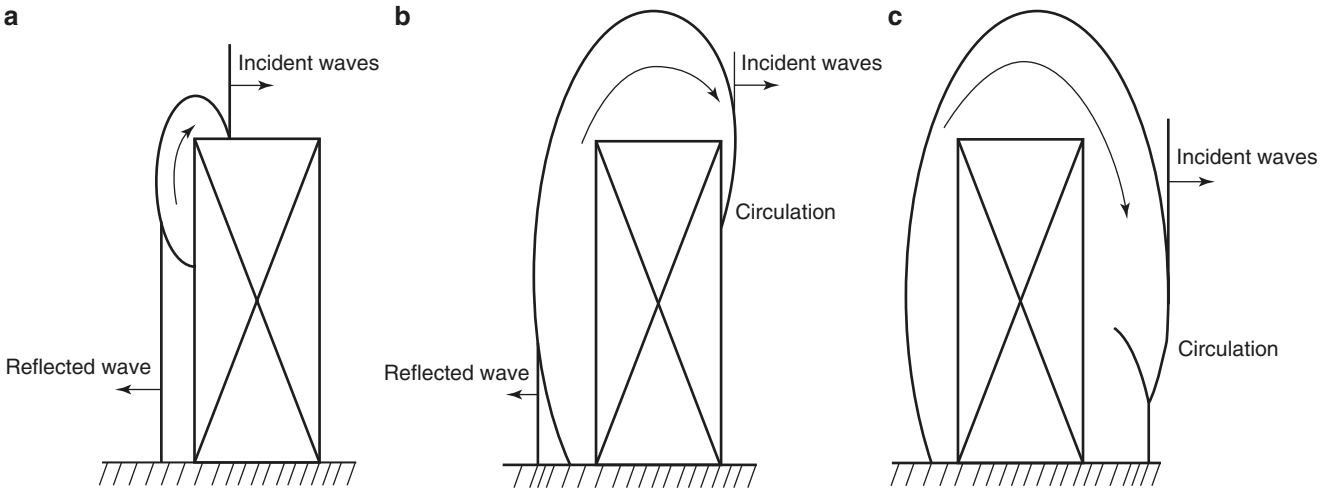

Fig. 42 (**a**–**c**) Diffraction effect of wide but not high obstacles on shock wave

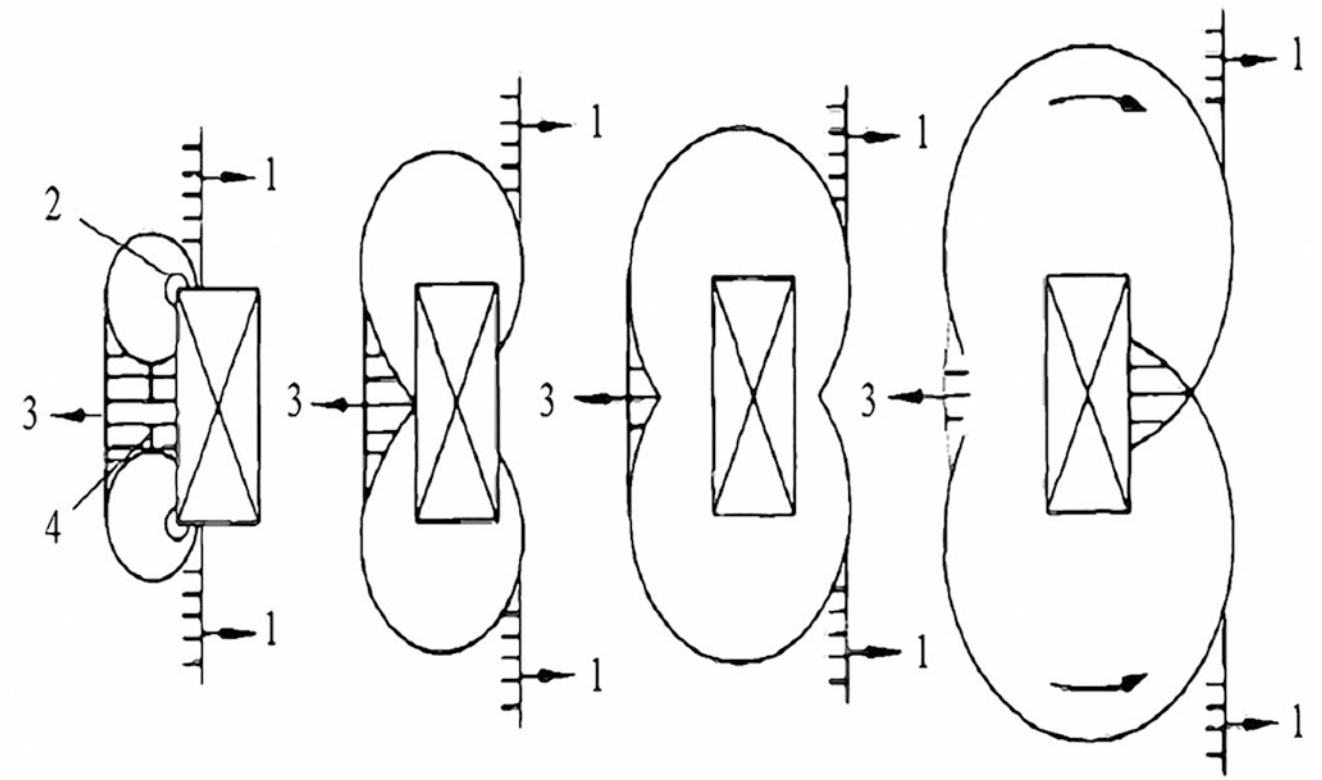

Fig. 43 Diffraction effect of high but not wide obstacles on shock wave

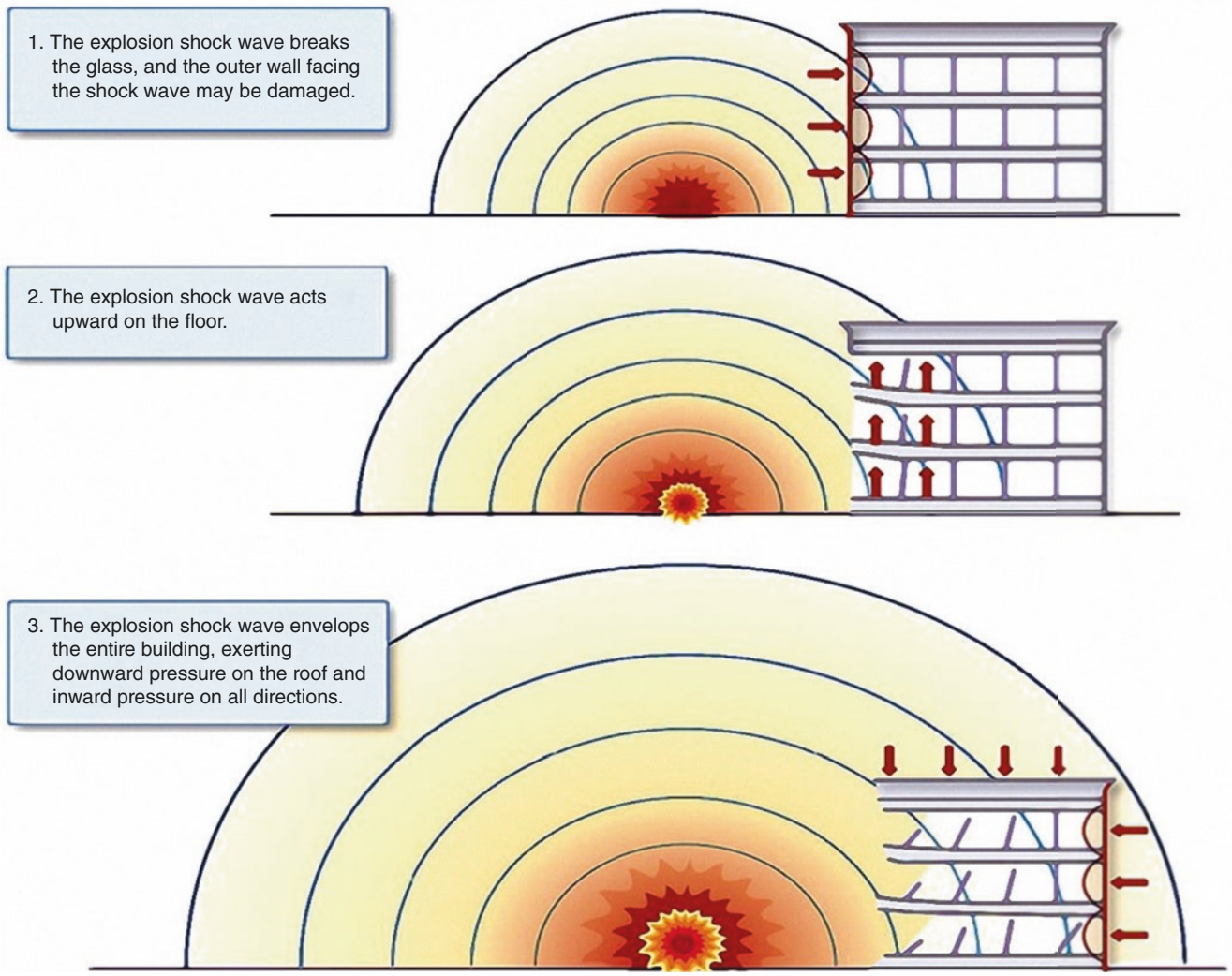

Fig. 44 Effect of shock waves of ground explosion on buildings

of structural components, and this is acceptable as long as such local failures do not lead to the collapse of the entire building.

4.3 Underwater Explosion

1. **Basic circumstances of underwater explosion**. When a charge explodes underwater, high-temperature and high-pressure detonation product is generated. Detonation product quickly expands outward, compresses the water medium and creates water shock wave, which propagates into the surrounding water environment at an extremely rapid manner while losing energy. This part of energy is termed shock wave energy, and makes up about 53% of the energy of the explosion. The rapid expansion of the detonation product thrusts the surrounding water to flow radially, expanding outward in the form of gas bubble while consuming energy. This part of energy is called bubble energy, which accounts for roughly 47% of energy of the explosion. When the internal pressure of a bubble exceeds the static pressure of the surrounding water medium, the bubble would continue to expand, the pressure meanwhile continues to drop as the bubble expands, until the bubble's internal pressure falls to the same as the static pressure of the water medium. The bubble ought to stop expanding, but due to the effect of inertia it remains in a state of expansion until reaching its largest radius. Since the internal pressure of the bubble is below that of the pressure balance of the surrounding water medium, under the action of the external pressure, the surrounding water turns to move internally. The bubble also turns from expanding outward to contract inward, and as the bubble continues to contract, internal pressure inside the bubble increases until the pressure inside the bubble elevates to the static pressure of the surrounding water medium.

Similarly, due to the inertia of water flow, the bubble ought to stop contracting, but continues to shrink until reaching its smallest radius. At this juncture, the pressure inside the bubble would again be greater than the static pressure of the surrounding water medium, which is why the bubble would once again expand, then repeat the previous expansion-contraction process. This is referred to as bubble pulsation, and this repeated cycle will also create a continuous pressure wave. The bubble gradually rises to the surface during this pulsation process of repeated expansion and contraction, until finally popping out of the water surface. How many times a bubble undergo the pulsation process is highly correlated to the depth at which the underwater explosion occurred.

Energy consumption during the course of the first pulsation is greatest, as roughly 60% of the explosion's energy is transferred to the main shock wave. During the second pulsation, about 25% explosion's energy is transferred to the second shock wave, while that for the third pulsation is approximately 8%. When calculating energy of an underwater explosion of an explosive, usually only the sum of shock wave energy and bubble energy from the first pulsation are taken into consideration. Figure 45 shows the time domain curve of a certain pressure point P in an underwater explosion, and the time domain curve of gas bubble pulsation radius R, along with the expansion-contraction process of its corresponding bubble.

With regard only to the effects of underwater blast wave alone, vessels on the surface are not impacted by the direct action of underwater shock wave, but also surface and bottom reflected shock wave, or even tsunami from the seabed due to the explosion, and the tsunami would in turn act on the vessels on water surface in the form of tidal wave (Fig. 46).

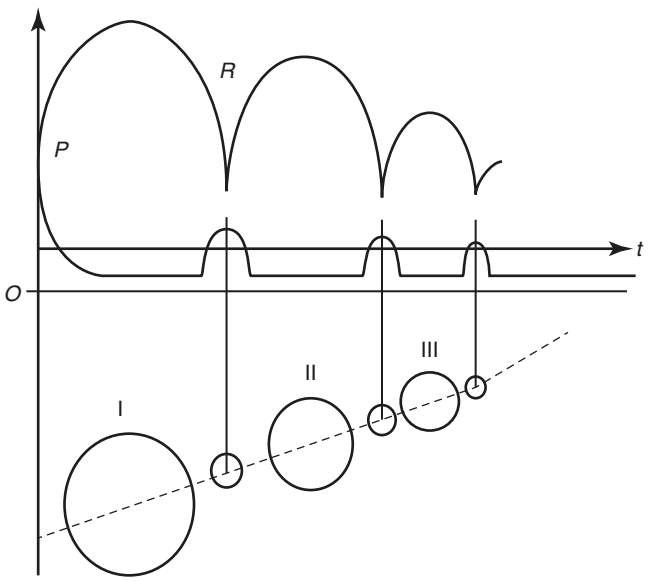

Fig. 45 Shock waves and bubble pulsation of underwater explosion

2. **Overpressure of underwater explosion shock wave**. The equation for overpressure of underwater explosion shock wave is:

$$\Delta P(t) = \Delta P_{\mathrm{m}} \mathrm{e}^{(1-1/\theta)\left[1-(R/c_{Z0})\right]} \sigma_0\left(t - \frac{R}{c_{Z0}}\right) \tag{102}$$

$$\theta = 10^{-4} \sqrt[3]{m_{\mathrm{e}}} Z^{0.24} \tag{103}$$

$$\sigma_0\left(t - R/c_{Z0}\right) = \begin{cases} 1, & t > R/c_{Z0} \\ 0, & t < R/c_{Z0} \end{cases} \tag{104}$$

In the equation, ΔP_{m} is peak overpressure in MPa; θ is time constant confirmed through experiment, shown in s; c_{Z0} denotes sonic velocity and is approximate to velocity of the front of the shock wave, shown in m/s; m_{e} is the explosive's TNT equivalent and shown in kg; R is distance, shown in m; and $Z = R/\sqrt[3]{m_{\mathrm{e}}}$.

When the charge is not located at a very great depth (static water pressure less than 1 MPa), the overpressure of shock wave front could be expressed as:

$$\Delta P(t) = \Delta P_{\mathrm{m}} \begin{cases} \exp(-t/\theta), & t < \theta \\ 0.368\theta/t, & \theta < t < (5 \sim 10)\theta \end{cases} \tag{105}$$

In the equation, ΔP_{m} is the overpressure of shock wave, shown in MPa; and θ is an exponential decay constant, shown in s.

3. **Similarity theory of underwater explosion**. Like air explosion, similarity theory also exists for underwater explosion. Similar to the format of expression for peak overpressure of shock wave in air explosion, the polynomial equation for underwater explosion shock wave may be written as:

$$\Delta P = \frac{355}{Z} + \frac{115}{Z^2} - \frac{2.44}{Z^3}, \quad 0.05 \le Z \le 10 \tag{106}$$

$$\Delta P = \frac{294}{Z} + \frac{1387}{Z^2} - \frac{17.83}{Z^3}, \quad 10 \le Z \le 50 \tag{107}$$

P. Cole provided an empirical equation for peak overpressure of underwater explosion shock wave:

$$\Delta P = K\left(\frac{1}{Z}\right)^{\alpha} \tag{108}$$

In the equation, K and α are both empirical coefficients for commonly used high-energy explosives, with K being

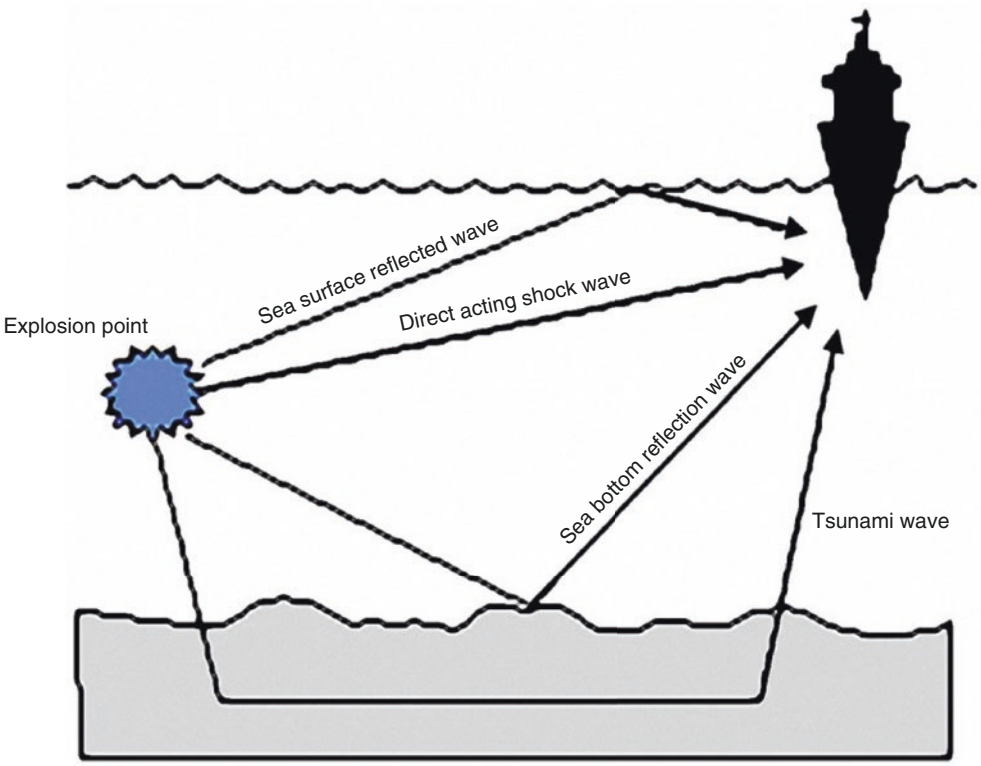

Fig. 46 Effect of underwater explosion shock waves on surface warships

around 50–60, while α is 1.13–1.14, and Z is scaled distance.

4. **Energy of underwater explosion.** Energy released by underwater explosion of an explosive converts into bubble energy, shock wave energy, and heat loss energy during its course of propagation. The relative energy assessment method for explosion of an explosive as given by GJB 7692-2012 is:

Equation for calculating bubble energy E_b:

$$E_b = \left(0.6842 P_h^{5/2} \rho_w^{-3/2} T_b^3\right) / \left(m \times 10^6\right) \tag{109}$$

In the equation:

E_b—bubble energy, shown in MJ/kg.

P_h—sum of static water pressure at the center of explosion and local atmospheric pressure during time of experiment, shown in Pa.

h—underwater depth of center of explosive, shown in m.

T_b—difference between time corresponding to peak dynamic pressure of first bubble pulsation and time of arrival of shock wave, known as bubble pulsation period and shown in s.

Equation for calculating shock wave energy E_s:

$$E_s = \frac{4\pi R^2}{\rho_w C_w m} \int_{t_a}^{\tau} P^2(t)\,\mathrm{d}t \tag{110}$$

In the equation:

E_s—shock wave energy a distance away from center of charge at location R, shown in MJ/kg.

ρ_w—density of water, and value for fresh water at normal temperature is usually 1.0 g/cm^3.

C_w—sonic velocity of water, and value for fresh water at normal temperature is usually 1460 m/s.

m—mass of explosive being experimented (including explosive equivalent mass of primer), shown in kg.

Explosion energy E is equal to sum of initial shock wave energy E_{s0} and bubble energy E_b, calculated as per the equation below:

$$E = E_{s0} + E_b = \mu E_s + E_b \tag{111}$$

In the equation:

μ—correction factor of shock wave.

4.4 Explosion in Rock and Soil

The rock and soil medium is a type of medium that varies greatly in properties, and one that is uneven and filled with a large number of gaps. Therefore, analysis of explosion in rock and soil is much more difficult than air or underwater explosion.

Effects of explosion in rock and soil also satisfy similarity theory of explosion and are mainly associated with mass

of explosive and buried depth of charge. Through the use of dimensional analysis, the relationship between buried depth and cube root of mass of charge is obtained:

$$Z = \frac{d}{m_{\mathrm{TNT}}} \tag{112}$$

In the equation, Z represents scaled buried depth; d is charge's buried depth; and m_{TNT} represents TNT equivalent of charge.

Studies show that there is a critical scaled buried depth Z_{cr}, and when $Z \geq Z_{\mathrm{cr}}$, all energy of the explosion is absorbed by rock and soil. This type of explosion is usually called hidden explosion or enclosed explosion. The value of Z_{cr} is related to the property of the rock and soil. Z_{cr} is 2 m/kg$^{1/3}$ for dry loess or sandy soil, while Z_{cr} is 2.5 m/kg$^{1/3}$ for more packed soil.

1. **Creation of cavity from enclosed explosion**. During an enclosed explosion, the blast wave and high-pressure, high-temperature detonation product forcefully compress the surrounding rock and soil medium, creating a cavity roughly several hundred or even a thousand times the size of the volume of the charge. The result is called an explosion cavity. Water content and cavities in the rock and soil medium immediately adjacent to the explosion cavity are forcefully compressed, and particle structures in the earth body are completely destroyed, creating a powerful compression zone also known as destruction zone. Shock wave outside the destruction zone has diminished into a stress wave, and though it can no longer destroy particle structures in the earth body, it can still cause radial displacement of rock and soil medium to a certain extent, instigating the formation of radial cracks. At the same time, unloading wave created from the rapid drop in expansion pressure of detonation product in the surrounding rock and soil medium would generate relatively strong radial tensile stress in the rock and soil medium, forming tangential cracks in a fracture zone crisscrossed with cracks and fissures. Outside the fracture zone, the stress wave diminishes to a seismic wave that propagates at the speed of sound, and it could only stimulate vibration in the rock and soil medium, but not enough to cause any structural destruction in said medium. Due to the relatively far distance of the seismic wave, this "vibration zone" is relatively large in area.

   Radius of the explosion cavity may be obtained through quasi-static theory analysis:

$$R = 0.794 P_{\mathrm{k}}^{0.139} \rho_{\mathrm{w}}^{1/9} D^{2/9} \left(p_{\mathrm{a}} + g \sum_{i=1}^{n} \rho_i h_i \right)^{-1/4} r \tag{113}$$

   In the equation, R denotes radius of the explosion cavity, shown in m; P_{k} is expansion pressure of detonation product when it expands and reaches conjugate point k, shown in Pa; ρ_{w} represents charge density and is shown in kg/m^3; D is detonation velocity of the explosive and is shown in m/s; P_{a} is atmospheric pressure and shown in Pa; ρ_i symbolizes natural density of the i layer of earth body, and is shown in kg/m^3; h_i is the thickness of the i layer of earth body, and is shown in m; g is gravitational acceleration, shown in m/s^2. P_{k} can be obtained via Hugoniot equation of the detonation wave, or more simply using the TNT explosive's conversion ratio e:

$$e = \frac{p_{\mathrm{kTNT}}}{p_{ki}} \tag{114}$$

   In the equation, P_{kTNT} is pressure of TNT at conjugate point k, $P_{\mathrm{kTNT}} = 2.8 \times 10^8$ and shown in Pa, while P_{ki} denotes pressure of the i type of explosive at conjugate point k, shown in Pa. For the e of industrial explosives, please see Table 7.

   Similarity theory of explosion may also be used to obtain empirical equation for radius of explosion cavity:

$$R = kr_0 \quad \text{or} \quad R = k^* m_{\mathrm{TNT}}^{1/3} \tag{115}$$

   In the equation, R denotes explosion cavity and is shown in m; r_0 is radius of charge and is shown in m; m_{TNT} represents TNT equivalent of charge and is shown in kg; and k and k^* are respectively scale factors obtained during explosion of No.9 ammonium nitrate.
2. **Creation of crater from explosion**. When $Z < Z_{\mathrm{cr}}$, the values of Z from big to small respectively create shallow buried explosion and ejection explosion. Since the interface between the rock and soil medium and the air medium is a free surface, when the radial compression wave generated by the explosion in the rock and soil medium reaches the free surface, it would turn into a reflected rarefaction wave. The destructive power generated from the overlapping of the compression wave and rarefaction wave creates a funnel-shaped fracture zone above the charge, and when coupled with the expansion action of the detonation product, the free surface of the

Table 7 Conversion ratio for common industrial explosives

Explosive	e
TNT	1.00
Ammonium nitrate-fuel oil mixture	1.18–1.56
No.2 ammonal for rocks	1.06–1.33
Colloidal nitroglycerin	0.95–1.05
No.4 waterproof ammonal for rocks	1.00–1.04
No.1 water-gel explosive for rocks/no.1 emulsified explosive for rocks	0.89–1.18

rock and soil medium would break up or bulge and protrude. If no rock and soil medium is ejected, this would be called a shallow buried explosion. For soil that is relatively less resistance to shearing forces, the empty cavity created by a shallow buried explosion would likely create explosion craters as the result of the weight of the soil itself and the collapse of the wall of the cavity. If Z further reduces, the fractured rock medium would be ejected upward and to the sides by the detonation product, creating an explosion funnel termed ejection explosion (Fig. 47).

There are numerous empirical equations for predicting diameter of explosion crater, and if influences of the earth body's density and strength parameters are taken into account, explosion crater diameter predictions are usually accurate with 10% deviation. Through dimensional analysis, an approximate opinion is that the scaled explosion crater diameter D/d is a function of $W^{7/24}/d$, and the explosion crater diameter function relationship may be expressed as:

$$\frac{D}{d} = F\left(\frac{(QW)^{7/24}}{\rho^{7/24} G^{1/8} c^{1/3} d}\right) \tag{116}$$

In the equation, D represents diameter of explosion crater; d is depth of charge buried; QW denotes energy of the explosive; ρ is earth body density; G is horizontal acceleration parameter; and c denotes velocity of propagation of seismic wave in said earth body.

Figures 48, 49, 50, and 51 depict numerical simulations of different explosion and destruction effect of explosives with same mass and size on multiple layers of medium (from top to bottom the layers are respectively concrete, rubble, rammed earth and natural soil) buried at different depths. The Euler calculation method is adopted

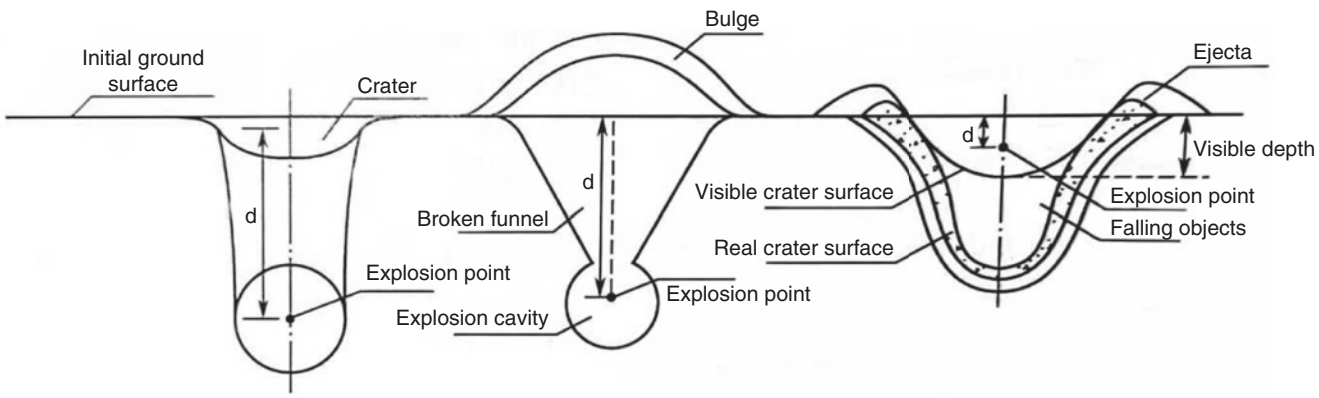

Fig. 47 Diagram of formation of explosion crater. d is the distance from center of explosion to ground surface

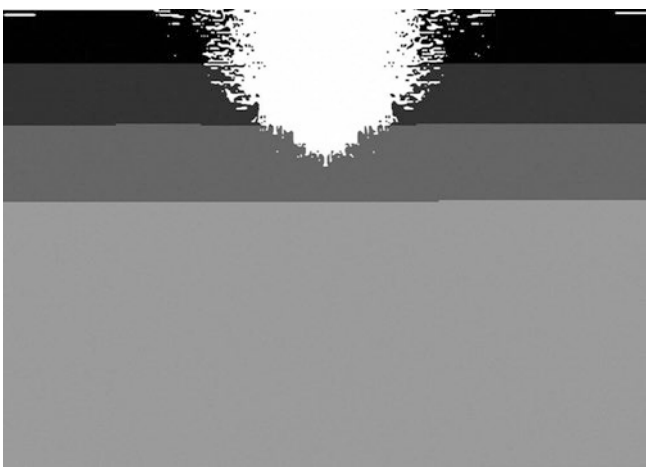

Fig. 48 Destruction of explosion at a depth of 0.35 m

Fig. 49 Destruction of explosion at a depth of 0.75 m

Fig. 50 Destruction of explosion at a depth of 1.25 m

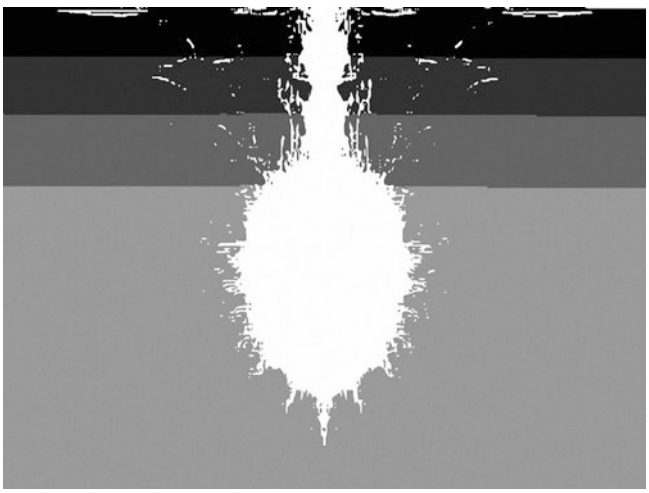

Fig. 51 Destruction of explosion at a depth of 2.00 m

for explosives and their products, and Lagrange calculation method is adopted for multi-layer board.

3. **Earthquake effects of explosion**. When the blast wave of an explosive's explosion propagates in the rock and soil medium, the energy of the shock wave near the point of explosion is immense, and the shock wave front has a steep head that propagates at supersonic velocity. During its propagation process, the shock wave loses a tremendous amount of energy and quickly diminishes into a stress wave. The front edge of the stress wave becomes less steep and propagates at sonic velocity, while energy loss is relatively low and diminishes at a slower rate. As the propagation distance increases, disruption energy gradually weakens, and stress wave diminishes to a periodic seismic wave and diminishes at an even slower rate. The seismic wave has low pressure, and its propagation energy only makes up 2–6% of the total energy of the explosion. The seismic wave causes periodic vibration in the medium, but does not destroy the internal structure of the medium. Waveforms at different stages of propagation (Fig. 52).

Due to the existence of gaps and holes in the earth body, the explosion product and shock wave propagation conditions in earth bodies differ vastly from propagation conditions in liquid medium. Attenuation law of shock wave with propagation distance is:

$$P = \sigma_r = P_0 \left(\bar{r}\right)^{-a} \tag{117}$$

In the equation: P_0 denotes initial detonation pressure, $\bar{r} = r / r_0$, r is distance from center of explosion, r_0 is diameter of blast hole, σ_r is radial peak pressure, and a represents attenuation coefficient.

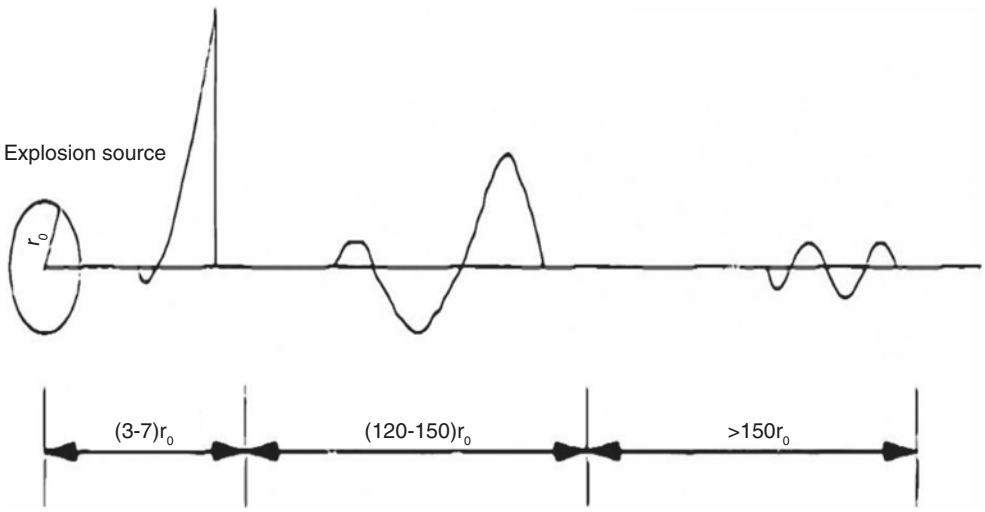

Fig. 52 Wave shape at different stages

After a shock wave diminishes into a stress wave, although it can no longer destroy the particle structure of the earth body, it can still cause radial displacement of rock and soil medium to a certain extent. The stress wave propagates at the sonic velocity of the medium, and this velocity has nothing to do with wave amplitude. Attenuation law of stress wave with propagation distance is:

$$\sigma_r = \sigma_0 \left(\bar{r}\right)^b \tag{118}$$

In the equation: σ_0 denotes initial peak pressure, and b represents stress attenuation coefficient, but it should be noted that this coefficient is different from the coefficient in the shock wave attenuation law.

Compared with natural seismic waves, a seismic wave created by an explosion has less energy, higher frequency, shorter wavelength, acceleration that is higher but also diminishes faster, and shorter vibration time. Blasting applications usually have to account for safety hazards on surrounding environment and structures caused by seismic waves of a blast explosion. Assessment of safe distance of blasting earthquake stipulated in China's *Safety Regulation for Blasting* uses peak particle velocity. In general, the Sadovski empirical equation is used to calculate the peak velocity of particle vibration:

$$v_s = k_s \left(Q^{\frac{1}{3}} / R \right)^{a_s} \tag{119}$$

In the equation, k_s and $a_{s\text{-}}$ are respectively blast earthquake attenuation coefficient and attenuation index, and are respectively associated with site of blast and properties of rocks; Q is charge quantity and is shown in kg; and R is distance from center of explosion, shown in m.

Calculation equation for vibration frequency of particle at ground surface caused by a blast's seismic wave:

$$f = k \left(Q^{1/3} / \log R \right)^{1/2} \tag{120}$$

In the equation, k represents a coefficient, of which, k for coyote-hole blasting has a value of 0.8–5.0; k for bench blasting has a value of 5.0–50; and k for demolition blasting has a value of 1.0–100. When explosive quantity is big, use a smaller coefficient, and vice versa.

The most common opinion at present is that the vibration intensity and vibration frequency of a blast are the two main factors that affect safety of structures, therefore, the criteria for judging safety of blasting vibration should consider the joint effects of vibration velocity and frequency. Figure 53 shows safety standards for blasting vibration as stipulated by the USMB and OSMRE. The blasting vibration safety standards of Germany (BRD-DIN4150) divides structures into three categories, namely industrial building, residential building, and sensitive building (Fig. 54).

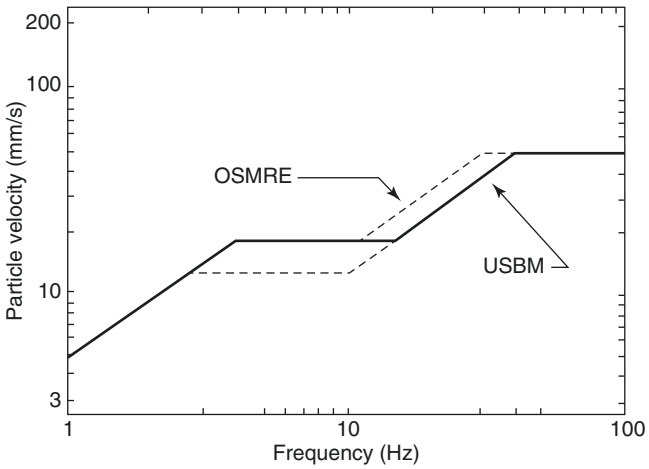

Fig. 53 USMB and OSMRE safety standards

4.5 Thermal Effects of Explosion

4.5.1 Temperature of Detonation Product

The chemical reaction in explosive releases thermal energy, which sustains the steady propagation of detonation wave. The detonation reaction zone has extremely high temperature, reaching several thousand degrees. However, detonation also has an extremely short reaction duration (about 10^{-7} s) and the temperature drops very rapidly. The equation for the decay of detonation product temperature with time is expressed as:

$$T = \exp\begin{bmatrix} 7.5722 - 0.443278\ln v + 0.09328338\left(\ln v\right)^2 \\ +0.002578583\left(\ln v\right)^3 - 0.003187935\left(\ln v\right)^4 \end{bmatrix} \tag{121}$$

In the equation, $\ln v = -\ln\left(0.33616 + 59.70427\,e^{-\tau/0.5832}\right)$ and $32.85152 \le \tau \le 50.88161$.

Figure 55 shows decay of detonation product temperature with time.

4.5.2 Temperature Increase Effect of Adiabatic Compressed Air Behind Blast Wave

After the explosion of an explosive, the surrounding air is abruptly impacted and compressed, and parameters for medium at the wave front change and jump. As entropy in medium elevates during the formation process of the shock

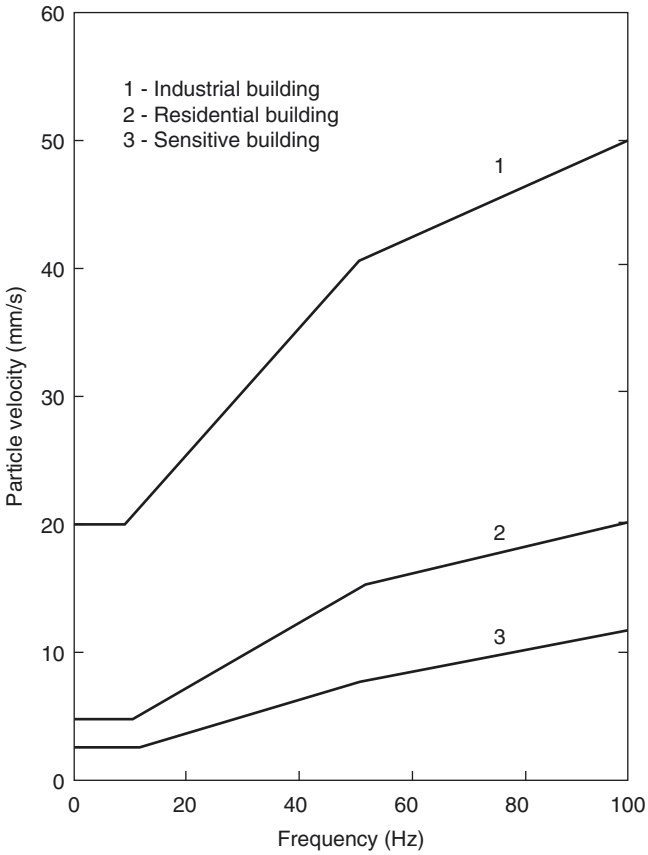

Fig. 54 DIN4150 blasting vibration safety standards

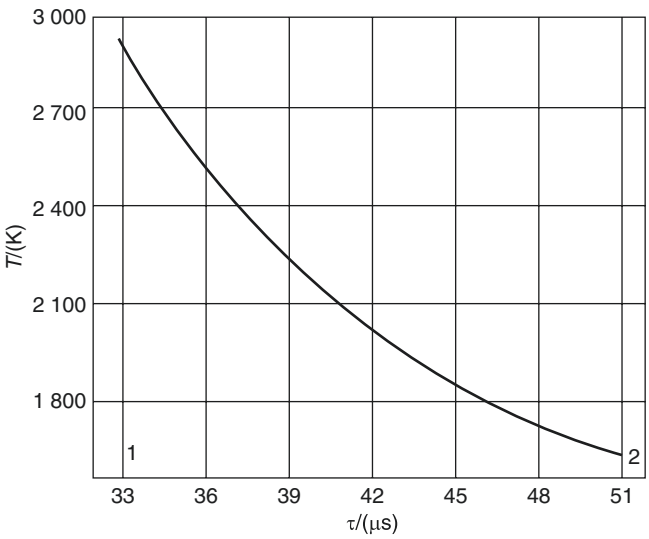

Fig. 55 Attenuation of detonation product temperature with time

wave, the extent of temperature increase with the rise in pressure far exceeds that of the isentropic process. For an ideal gas:

$$\frac{T_{\mathrm{Hsw}}}{T_{\mathrm{His}}}=\left(\frac{P_{\mathrm{H}}}{P_0}\right)^{1/k}\frac{\dfrac{k_0+1}{k_0-1}+\dfrac{P_{\mathrm{H}}}{P_0}}{\dfrac{k_{\mathrm{H}}+1}{k_{\mathrm{H}}-1}\dfrac{P_{\mathrm{H}}}{P_0}+1} \tag{122}$$

In the equation, the subscript "sw" represents parameters of the medium at the front of the shock wave; the subscript "is" represents parameters of gases in the isentropic process; and k_0 and k_{H} are isentropic indexes on the two sides of the shock wave front. For a strong shock wave, due to the dissociation and ionization processes in gases, the isentropic indexes on the two sides of the shock wave front are not equal. For a shock wave that is not too strong, $k_0 = k_{\mathrm{H}} = k$ and the above equation may be simplified as:

$$\frac{T_{\mathrm{Hsw}}}{T_{\mathrm{His}}}=\left(\frac{p_{\mathrm{H}}}{p_0}\right)^{1/k}\frac{k-1}{k+1} \tag{123}$$

Table 8 lists related parameters upstream of an air shock wave.

From data in Table 8, it is abundantly clear that temperature of gases upstream of the front of a shock wave that propagates in gases rapidly rises. However, compared with the damages caused by the overpressure and specific impulse of a shock wave, the temperature increase duration of blast wave of condensed explosive is very short, and its scope of effect is limited. In addition, due to the relatively fast decay of temperature in the air, thermal effect might not be that obvious.

Table 8 Related parameters upstream of an air shock wave

P_{H} (MPa)	T_{H} (K)	ρ_{H} (kg/m^3)	u_{H} (m/s)	v_{D} (m/s)
0.196	336	2.107	175	452
0.490	482	3.665	452	608
0.784	618	4.567	627	875
0.980	705	5.108	725	978
1.96	1126	6.223	1095	1369
2.94	1522	6.958	1364	1676
3.92	1898	7.448	1594	1930
4.90	2260	7.801	1795	2150
5.88	2660	8.144	1978	2350
7.84	3210	8.693	2300	2705

Note: Initiation conditions: $P_0 = 9.8 \times 10^4$ Pa, $\rho_0 = 1.2936$ kg/m^3, and $T_0 = 273$ K

4.5.3 Thermal Radiation Effects of Fuel/Air Mixture Explosion

Unlike the explosion of condensed explosive that occurs at an approximate spot, the explosion of fuel/air mixture could take place within a large geometric space of fuel and air mixture, and could happen in different kinds of forms such as rapid flame propagation or stationary combustion. For gas explosion, on the one hand, the spread of high-temperature detonation product and temperature increase effect of adiabatic compressed air behind blast wave could generate high-temperature thermal effect within a large geometric space surrounding the blast field, and on the other hand, it would also create massive explosion fireball that emits radiant flux, or thermal radiation, consisting of ultraviolet waves (wavelength less than 0.38 μm), visible lights (wavelength between 0.38 and 0.78 μm) and infrared waves (wavelength longer than 0.78 μm).

1. **Shape and duration of fireball**. At present, existing calculation models for shape and duration of fireball pretty much ignore the fireball's dynamic formation process, and does not include energy loss in the atmosphere. In other words, the models assume that the greatest diameter, the height and surface heat flux of the fireball all occurred at an instant, and that they remain the same during the duration of the fireball's existence. Almost all empirical equations derived from experiments consider fuel quantity M as a function of the fireball's greatest diameter R, the fireball's height H, and the fireball's duration of existence t:

$$R = A \times M^a \tag{124}$$

$$t = B \times M^b \tag{125}$$

$$H = C \times M^c \tag{126}$$

A, B, C, a, b, and c are coefficients related to the type of fuel.

Empirical equation derived from explosion of the Mars V rocket (fireball temperature around 3600 K):

$$D = 3.86 M^{0.32} \tag{127}$$

$$t = 0.299 M^{0.32} \tag{128}$$

In the equation, M is mass of propellant and is shown in kg; and t is the duration of existence of fireball, shown in s. This empirical equation is applicable to explosion of liquid propellant with fuel mass larger than 20 kg.

Empirical equation derived from explosion of liquid propellant with fuel mass less than 10 kg (fireball temperature around 3600 K) is:

$$D = 5.25 W^{0.314} \tag{129}$$

$$t = 1.07 W^{0.181} \tag{130}$$

Empirical equation derived from deflagration and detonation of diesel, kerosene, and gasoline within concentration range of 20–30 g/m^3:

(a) For deflagration of diesel and kerosene

$$D = 2 \times (26 \pm 1) M^{0.33 \pm 0.02} \tag{131}$$

$$t = (4.63 \pm 0.1) M^{0.177 \pm 0.012} \tag{132}$$

(b) For deflagration of gasoline

$$D = 2 \times (23.4 \pm 0.5) M^{0.34 \pm 0.01} \tag{133}$$

$$t = (4.77 \pm 0.19) M^{0.086 \pm 0.015} \tag{134}$$

(c) For detonation of diesel, kerosene, and gasoline

$$D = 2 \times (34 \pm 4) M^{0.32 \pm 0.04} \tag{135}$$

$$t = (1.8 \pm 0.3) M^{0.33 \pm 0.05} \tag{136}$$

Fireball's height H usually refers to the distance between the ground surface and the center of the fireball, and may be calculated using this empirical equation:

$$H = 6.48 M^{0.35} \tag{137}$$

2. **Thermal radiation parameters**. Generally speaking, thermal radiation parameters include heat flux density q and thermal dose Q. Heat flux density refers to heat that passes over a unit area within a unit time, and is measured in W/m^2. Heat flux density is also known as thermal flux. Thermal dose may be understood as the cumulative heat flux density over a unit area within a certain period of time and is measured in J/m^2. Heat flux density q chiefly hinges on type of fuel and combustion mechanism and is not directly related to quantity of fuel.

Heat flux density may be expressed as:

$$q = c_{\mathrm{p}}(T) \rho(T) T u \tag{138}$$

In the equation, $c_{\mathrm{p}}(T)$ represents specific heat at constant pressure; ρ(T) is density of medium inside the fireball; T denotes temperature of the fireball; and u is heat flux propagation velocity.

Thermal dose may be expressed as:

$$Q = \int_0^t c_{\mathrm{p}}(T) \rho(T) T u \tag{139}$$

Also ignoring the fireball's dynamic formation process, and not including energy loss in the atmosphere, empirical equation for thermal radiation propagation is:

$$\frac{q}{T^4} = \frac{G\dfrac{D^2}{R_2}}{F + \dfrac{D^2}{R_2}} \tag{140}$$

$$\frac{Q}{(bG)M^{1/3}T^{2/3}} = \frac{\dfrac{D^2}{R_2}}{F + \dfrac{D^2}{R_2}} \tag{141}$$

In the equation, q is heat flux density, shown in W/m^2; T denotes fireball's temperature, shown in K, and is given a value of 2200 K for unconfined vapor cloud explosion; D is diameter of fireball, shown in m; R is distance to center of fireball, shown in m; G represents a constant, of which Baker gave a value of $G = 5.26 \times 10^{-5}$, while some Chinese scholars opine that the value ought to be $G = 0.958 \times 10^{-7}$; F is a constant, $F = 1617$; Q denotes thermal dose, shown in J/m^2; bG is a constant, $bG = 2.04 \times 10^4$; and M symbolizes mass of fuel consumed in the fireball, and is shown in kg.

Under the same experiment conditions as mentioned above, the fireball is deemed a gray body, and the gray body radiant relationship equation is obtained:

$$q = q_0\varepsilon = q_0\left[1 - \exp(-kx)\right] \tag{142}$$

In the equation, $q_0 = \sigma T^4$, T denotes blackbody's temperature, σ is the Stefan–Boltzmann constant, ε is radiation coefficient (level of emissivity), x represents size of flame (optical thickness, and k is extinction coefficient.

Data from the thermal imagery are fitted using the above equation to obtain heat flux density's empirical equation:

$$q = (110 \pm 10)\left[1 - \exp(-2.6R)\right] \tag{143}$$

In the equation, the unit for q is kw/m^2, and the unit for R is m.

Based on the equation above, calculation results for heat flux density q are: For deflagration of diesel and kerosene, q = 80–200; for deflagration of gasoline, q = 150–300; and for detonation of diesel, kerosene and gasoline, q = 200–350.

Total thermal radiation energy E may be empirically expressed as:

$$E = F \times M^f \tag{144}$$

In the equation, the unit for E is J, and F and f are coefficients related to the type of fuel. From Eq. (145) it can be seen that as long as the fuel type and fuel quantity are confirmed, the total thermal radiation energy E listed for the three situations above won't deviate significantly.

3. **Thermal radiation damage and injury criteria**. Next, let's look at three common criteria related to thermal radiation damage and injury:
   (a) The q criterion: The q criterion uses heat flux density to evaluate the damage and injury effect on target, as different heat flux density causes different damage and injury. Table 9 lists damage and injury threshold values on humans based on experiment experiences.
   The q criterion is applicable in situations when time of effect of heat flux density is longer than the time it takes for target to reach thermal equilibrium.
   (b) **The Q criterion**: The Q criterion uses heat dose to evaluate the damage and injury effect on target, and can be applied to assess damage caused by heat dose in situations when time of effect of heat flux density is so short that radiated target doesn't have enough time to lose heat (Table 10).
   (c) **The q–Q criterion**: The q–Q criterion takes into account damage and injury effects from both heat flux density and heat dose. Specifically, this method respectively plots heat flux density and heat dose as x and y coordinates, and then on this on the q–Q plane, the target's critical damage status curve, known as critical damage curve, is graphed.
4. **Dynamic model for calculation of fireball thermal radiation consequence**. The fireball's actual course of development is a dynamic process, and so too is the course of effect of fireball thermal radiation. Dynamic model for calculation of fireball thermal radiation consequence may be expressed as:

$$I_{\text{dose}}(x) = \int_0^{t_d} (x,t)\,\mathrm{d}t = \int \tau(x,t)F(x,t)E(x,t)\,\mathrm{d}t \tag{145}$$

In the equation, τ is atmospheric transmittance rate, F denotes the target's maximum geometric view, E represents radiation energy on the fireball's surface, t is the

Table 9 Damage and injury threshold values on humans

Heat flux density (kW/s^2)	Damage and injury effect
37.5	100% fatality rate within 1 min, 1% fatality rate within 10 s
25.0	100% fatality rate within 1 min, severe burn within 10 s
16.0	Severe burn above 5 s
12.5	100% fatality rate within 1 min, first-degree burn within 10 s
6.4	Pain threshold of 8 s, second-degree burn within 20 s
5.0	Pain threshold of 15 s
4.5	Pain threshold of 15 s, second-degree burn
4.0	Pain after 20 s
1.75	Pain threshold of 1 min
1.6	No discomfort after prolonged exposure

Table 10 Damage and injury effects of heat flux

Heat dose (kJ/s^2)	Damage and injury effect
1030	Firewood
592	Death
392	Severe injury
375	Third-degree burn
250	Second-degree burn
172	Light injury
125	First-degree burn
172	Pain

duration of the fireball's existence; and I is thermal radiation dose, shown in KJ/m^2.

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Explosion and Injuring Factors

Jianxin Jiang and Zhengguo Wang

1 Types of Explosion

Explosion refers to mechanical energy imparted on surrounding medium converted from rapid expansion of gas powered by the energy of the system itself due to rapid physical and chemical changes of the substance system. This release of energy is accompanied by powerful exothermic effect, light emission, sound emission, and other reactions. Explosion is a common phenomenon, such as explosion of boiler, explosion of tires of automobile or bicycle, and explosion of explosives from firecrackers to atom bomb and hydrogen bomb. With regard to the nature of explosion, the three main categories are physical explosion, chemical explosion, and nuclear explosion, all of which are capable of causing blast injury.

1.1 Physical Explosion

This type of explosion refers to changes in the form of the substance, while chemical composition and properties do not change. Explosions of steam boiler/generator and high-pressure gas cylinder are common cases that belong to this category. The former is the outcome of rapid conversion of overheated water into overheated steam, leading to pressure so high that exceeded the vessel's resistance, resulting in an explosion. The latter is because too much gas was delivered, resulting in pressure that exceeds the tolerance of the vessel, resulting in fracture and explosion of the gas cylinder. Other examples of this category of explosion include those instigated by earthquake, or strong electrical discharge (lightning) or high-voltage current conducted through metal.

1.2 Chemical Explosion

This type of explosion refers to those caused by high temperature and high pressure generated from extremely rapid chemical changes. Chemical explosions not only change the form of the substance, but also alter its chemical composition and properties. Explosion of explosives is the typical chemical explosion. In addition, explosion caused by coal fines suspended in the air, and explosion of mixture of air with a certain ratio of methane or acetylene, are also regarded as chemical explosion.

Chemical explosions may be further divided into three sub-categories based on the chemical changes: (1) Simple decomposition explosion: The explosive substance that causes simple decomposition explosion does not necessarily undergo deflagration reaction when exploding, and the heat needed for explosion is generated during the decomposition of the substance itself. Examples of this sub-category include lead azide, silver acetylide, copper acetylide, nitrogen triiodide and nitrogen chloride, among others. These substances are very dangerous and could explode even with very slight vibration. (2) Complex decomposition explosion: The explosive substances linked to this kind of explosion are relatively less dangerous than those that cause simple decomposition explosions, and all explosives are such substances. Combustion accompanies explosions from this type of substances. Oxygen required for combustion is supplied by the decomposition of the substance itself. Various kinds of hydrogen and chlorine oxides, and picric acids are such substances. (3) Explosive mixture explosion: Explosions caused by flammable gas, steam, dust, and gaseous mixtures belong to this sub-category. Explosion from such substances require

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certain conditions, such as the content of the explosive substance, oxygen content and initiation energy, among other factors. Therefore, the risk of this sub-category is relatively lower than the other two kinds, but it is also very common and could cause very severe harm.

1.3 Nuclear Explosion

Nuclear explosion refers to the course of the abrupt release of an immense amount of energy during nuclear fission (such as that of an atom bomb) or nuclear fusion (such as that of a hydrogen bomb). To facilitate comparisons with common explosives, the yield of a nuclear power detonation, as in the energy released by its explosion, is expressed as a unit known as "TNT equivalent," defined as the weight of TNT explosive that would cause an explosion of the same power.

When an atom bomb detonates, the action of neutrons in the uranium^{235} or plutonium^{239} in its payload can cause and sustain fission chain reaction within less than 1 s, which would release an enormous amount of energy. The energy released from complete fission of 1 kg of uranium or plutonium is roughly equivalent to energy released from the explosion of 20,000 tons of TNT. When a hydrogen bomb is detonated, the hydrogen isotopes such as deuterium, tritium, and lithium in its payload undergo fusion reaction under the joint action of neutrons in and the extremely high temperature generated from nuclear fission detonation. This fusion reaction releases a massive amount of energy. The energy released from complete nuclear fusion of 1 kg of deuterium is roughly equivalent to energy released from the explosion of 60,000 tons of TNT. In other words, the explosion of complete nuclear fusion of 1 kg of deuterium has a TNT equivalent of 60,000 tons.

During a nuclear explosion, the inside of the bomb could reach several million degrees in temperature and several tens of billions of atmospheric pressure in pressure, which is why high-temperature, high-pressure gas is created, which rapidly expands in all direction, creating the shock wave of a nuclear explosion.

1.4 Common Types of Explosives

An explosive may be classified as either single-compound explosive based on the composition, or classified as primer, high explosive, propellant (gunpowder) or pyrotechnic explosive based on its function. The latter classification is more common in actual application.

1. **Primer** is an explosive usually used to fill a detonator device (e.g., blasting cap, shock tube detonator). The primer is characterized by its high sensitivity (explodes easily) and the capacity to reach maximum velocity of detonation within an extremely short period of time. Some typical primers are mercury fulminate and potassium chlorate. Damage and injury caused by the explosion of primers are usually minor.
2. **High explosive** is usually used in the charges of munitions such as shells and missile warheads. High explosive is characterized by relatively good stability, and most of the times require a primer to detonate. However, upon detonation, they generate higher velocity of detonation and more destructive power. There are many different kinds of high explosive. Common single-compound explosives include different kinds of nitrate (e.g., nitroglycerin, also known as glycerin trinitrate), nitro compounds (e.g., TNT, cyclotrimethylenetrinitramine a.k.a. RDX), and others. Multi-compound explosives include those based on nitroglycerin (i.e., blasting gelatin with 88–93% nitroglycerin and 7–12% collodion-cotton), AN/FO (i.e., amatol, which is 80% ammonium nitrate and 20% TNT), and liquid-oxygen explosive (various powdered organic absorbents impregnated with liquid oxygen). Most blast trauma resulting from the explosion of explosives are caused by this type of explosive.
3. **Propellant (gunpowder)** is mainly used to propel different kinds of munition. This type of explosive deflagrate when ignited by primer. The high-temperature and high-pressure gas and flame generated during deflagration can propel a munition from the muzzle. Common propellants are nitro-cotton, and some smokeless propellants emulsified through the inclusion of additives.
4. **Pyrotechnic explosives** are usually mixtures of oxidizer, flammable organic compound or powdered metal, along with a trace amount of binding agent. In terms of military applications, pyrotechnic explosives are pyrotechnic composition in flares, smoke composition in smoke grenades, incendiary composition in incendiary weapons, as well as tracers, napalm, signaling systems, and others. In these explosives, some might also detonate, such as signal systems that nitroglycerin use chlorate as oxidizer.

Table 1 lists data related to some common explosives.

2 Formation and Progression of Shock Wave

The shock wave created by an explosion is a three-dimensional shock wave, and it propagates outward in a spherical or semi-spherical manner from the center of explosion. As the radius expands, the area of the front of the shock wave increases while overpressure gradually diminishes.

Table 1 Data related to some common explosives

Name of explosive	Molecular formula	Molecular weight	Specific volume (L/kg)	Explosion (Kcal/kg)	Temperature of detonation (°K)	Velocity of detonation (m/s)
TNT	$C_7H_5O_6N_3$	227	700	1090	3350	7138
Picric acid	$C_6H_3O_7N_3$	229	725	1020	3650	7245
Tetryl	$C_7H_5O_8N_5$	287	748	1210	3950	7460
Hexogen	$C_3H_6O_6N_6$	222	907	1300	4150	8390
Pentaerythritol tetranitrate (PENT)	$C_5H_8O_{12}N_4$	316	780	1430	4330	8340
Nitroglycerin	$C_3H_5O_9N_3$	227	715	1510	4600	
Ammonium nitrate	NH_4NO_3	80	980	378	1650	
Ethylene glycol dinitrate (EGDN)	$C_2H_4O_6N_2$	152	737	1630	4640	

Specific volume: Volume of gaseous product in standard form (0 °C, 1 atm) generated after 1 kg of explosive explode, and is shown in L/kg
Heat of detonation: Heat released upon complete detonation of 1 kg of explosive explode, and is shown in kcal/kg
Temperature of detonation: Highest temperature reached by explosion product after explosion, and is shown in absolute temperature (°K)

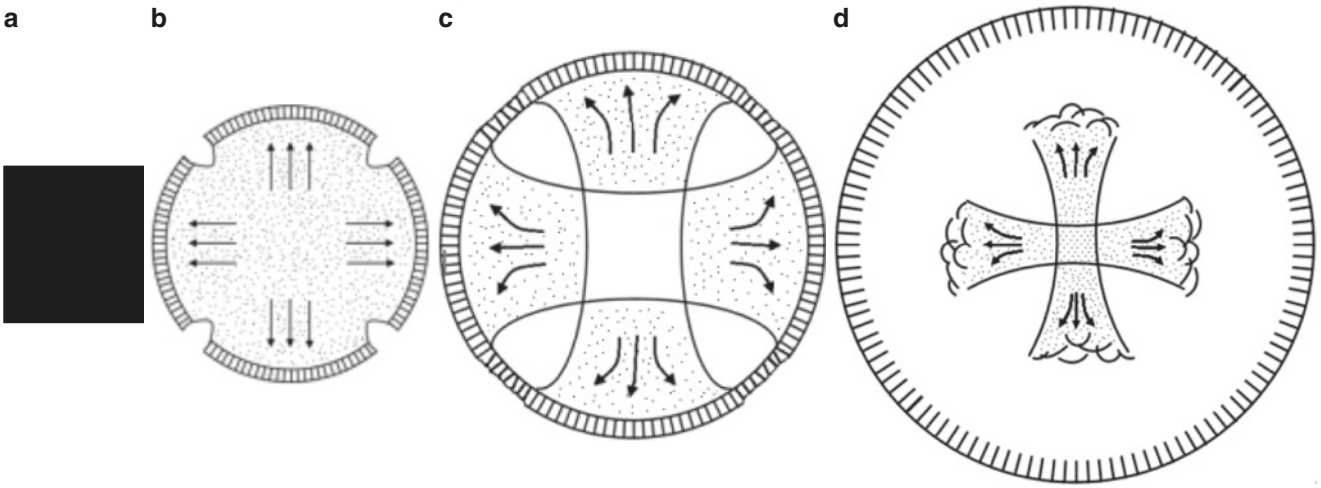

Fig. 1 Formation of explosion shock wave. (**a**) Before detonation. (**b**) After detonation with a large number of detonation products produced, the initial shock wave is formed at the edge. At this time, the shock wave has not yet formed a spherical shape. (**c**) The detonation products expand at a high speed, and the energy further transmits to the surrounding medium edge shock wave basically forming a spherical shape. (**d**) The expansion of detonation products ends, the shock wave separates from the products and spreads in the air alone

2.1 Formation of Shock Wave When Explosive Explodes

When an explosive explodes, substantial quantity of detonation products are generated at an instant, namely high-pressure gas, while releasing a tremendous amount of heat. The high-temperature and high-pressure gas rapidly expands outward and transfers its energy to the surrounding air medium, thereby creating the initial shock wave around the detonation product (Fig. 1). At this time, the initial shock wave has yet to become a complete spherical form. Immediately after, since the detonation product continues to expand at a rapid pace and further transfers its energy to the surrounding medium, it is only then would the initial shock wave emerge into its complete spherical form. Within this distance (as in near the center of explosion), the front end of the detonation product converges with the shock wave, becoming a unified entity, moving at the same velocity and causing destruction to the surrounding together. But within this distance, the density of detonation product is 20 times higher than the air at the front of the shock wave, which is why during this period destruction is mainly caused by detonation product. During the detonation product's expansion process, energy is continually transferred to the shock wave, which is why its own pressure and energy continue to dwindle. In turn, its rate of expansion also correspondingly slows down, until expansion ceases altogether. Based on calculation of relationship between detonation product's pressure P and its expansion radius ($P \approx 1/r^9$), when radius of detonation product doubles due to expansion, its pressure drops to 1/512 of its original pressure. Meanwhile, when detonation product expands to its maximum volume, its radius is only 10–15 times of the radius of the charge. When detonation product stops expanding, shock wave would separate from

the unified entity and independently propagates in the air. Since the shock wave obtained about 75% of detonation product's energy, it still has a lot of energy and destructive power as it propagates through the surrounding.

2.2 Formation of Shock Wave in Nuclear Explosion

During a nuclear explosion, due to the tremendous amount of energy suddenly released, all substances surrounding the center of explosion turn into extremely hot, high-pressure gases, and create a high-temperature fireball with even internal temperature. As the high-temperature and high-pressure gas rapidly expands through its surrounding, a compression wave is formed and then quickly transforms into shock wave with a very steep front. At first, the front of the shock wave is located behind the fireball. But soon its wave front would surpass the surface of the fireball. As it propagates throughout its surrounding, the overpressure at the front of the shock wave continues to decline. After a certain distance, negative pressure starts to appear behind overpressure. This is the typical nuclear explosion shock wave.

2.3 Formation of Shock Wave in Shock Tube Experiment

The shock tube is a unique type of shock wave generator and is used in laboratories to conduct research related to blast wave. Usually a shock tube is a long tube comprised of several steel tubes connected together, and a diaphragm is installed to divide the whole tube into two sections: The "high pressure section" is filled with high-pressure compressed gas, while the other "low pressure section" is connected with the atmosphere and has relatively lower pressure. The "low pressure section" may be further divided into the transition section (stable shock wave forms after this segment), test section (where pressure measurements are taken), and a tail experiment section (usually for bioexperiments). When the diaphragm bursts open because it can no longer withstand the gas pressure in the high pressure section, high-speed flow of gas immediately occurs inside the tube. Rarefaction wave is created in the high pressure section, and its vector of movement is opposite from the direction of the diaphragm. Shock wave is created in the low pressure section, and its vector of movement is directed toward the tail of the tube, as in opposite from the direction of the rarefaction wave. Although the shock waves in a shock tube might differ in wave form compared with shock waves from nuclear explosion or explosive explosion, they are identical in nature.

The length ratio between the high pressure section and the other low pressure section of a shock tube is usually 1:5–1:10, and distance of test section from diaphragm needs to be at least 30 times more than the diameter of the shock tube in order to ensure that the measured shock wave is steady and even. The duration of the shock wave is dependent on the length of the high pressure section. For instance, a longer high pressure section is required to simulate the shock wave of a nuclear explosion. Generally speaking, each meter of high pressure section can generate 5 ms of platform effective time (referring to duration of stable state of sustained peak value). Length of the low pressure section has to be appropriate to ensure the formation of steady shock wave. If the low pressure section is too long, not only will the shock wave continue to weaken, platform effective time will also shorten, while total effective time will extend, shock wave might even take on a triangular waveform. Therefore, common design plans require high pressure section to be 2.4–6 m long, and low pressure section to have a length of 15–30 m. For bioexperiments, usually A00 aluminum foil is used as diaphragm because this kind of material doesn't shatter into fragments upon breaking, thereby minimizing chances for flying fragments that might damage sensor or injure the tested animal. Under most circumstances, shock tube uses a buildup of high pressure from compressed gas as the main driving force.

2.4 Movement of Shock Wave

As a shock wave propagates in the air, two zones that resemble double-layered spheres are formed. The outer shell is the compression zone, and the inner shell is the rarefaction zone (Fig. 2). Inside the compression zone, pressure exceeds normal atmospheric pressure because of the compressed gas, and at the same time gas flows forward. The portion of pressure that exceeds normal atmospheric pressure is called

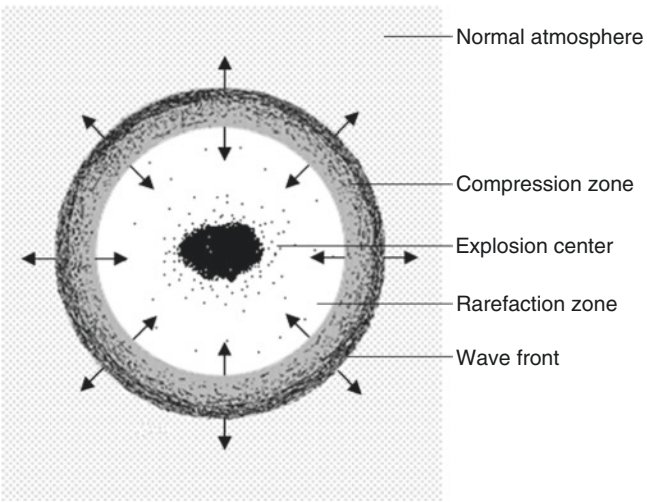

Fig. 2 Shock wave movements

overpressure, and shock wave pressure generated by gas flow is called dynamic pressure. Overpressure and dynamic pressure are at the highest on the front of the shock wave, and these are respectively called peak overpressure and peak dynamic pressure. Usually what are referred to as overpressure value and dynamic pressure value are their respective peak values, which are measured in kg/cm^2.

Inside the rarefaction zone, due to the vacuum created after air is compressed, air in this region is highly rarefied, pressure is less than normal atmospheric pressure, and air flows in the opposite direction (toward the center of explosion). The portion of pressure that is lower than normal atmospheric pressure is called the negative pressure of the shock wave. The highest value of negative pressure is known as peak negative pressure, and when people speak of negative pressure, they are usually referring to peak negative pressure. Inside the rarefaction zone, dynamic pressure is usually quite low because of the relatively slow pace of air movement. Generally speaking, the destructive power of negative pressure is less than that of overpressure and dynamic pressure.

When a shock wave reaches a certain spot, the air at said spot is suddenly compressed and pressure instantaneously reaches maximum value, while at the same time the air rapidly moves forward. After the front of the shock wave passes by said spot, air pressure and flow rate gradually decline. When the tail end of the compression zone passes by said spot, air pressure would have dropped to normal atmospheric level and air would have stopped flowing. Immediately after, the rarefaction zone passes by said spot, and at this juncture air pressure falls below normal atmospheric level, creating negative pressure while air flows toward the center of explosion. After the rarefaction zone leaves said spot, the air at said spot returns to normal. At the beginning, the shock wave propagates to its surrounding at an incredibly high speed (several thousand meters per second). As the distance of propagation increases, pressure at the front of the shock wave drops swiftly, and velocity of propagation also falls rapidly. When its velocity slows to the speed of sound (about 340 m/s at standard atmospheric pressure and temperature of 15 °C), the shock wave turns into a sound wave.

3 Similarities and Differences Between Shock Wave of Nuclear Explosion and Explosive Explosion

3.1 Similarities

Both nuclear explosion and explosive explosion are outcomes of rapid release of tremendous amount of energy within a limited space. Due to the sudden release of energy, temperature and pressure rise quickly, turning all detonation products into hot and high-pressure gases that expand to its surrounding. Shock waves are formed when these energies are transferred to the surrounding medium (i.e., air and water).

Whether nuclear explosion and explosive explosion, the shock waves generated share similar basic physical properties and change patterns, and their injury factors (such as dynamic pressure, overpressure, and negative pressure) and injury principles are identical. In addition, regardless of nuclear weapons or conventional bombs, their destructive effects (referring to damage caused to structures, fortifications, bridges, cars, weapons, and other objects) are chiefly attributed to shock waves.

3.2 Differences

1. **Method of generating energy**. The shock wave generated from the explosion of explosive is the result of energy released from rearrangement of atoms (i.e., hydrogen, carbon, oxygen, and nitrogen atoms in TNT) in the explosive substance. The energy in a nuclear explosion however originates from rearrangement or recombination of protons and neutrons in the nuke. In other words, this kind of energy comes from mutual effects between specific nuclei.
2. **Ratio of damage and energy**. A nuclear explosion releases several thousand times or even several million times more energy than the explosion of a conventional bomb, and the shock wave thereby created is naturally much larger than that generated from the detonation of a regular bomb. In terms of the direct area of damage, shock wave from a nuclear explosion could cover an area of several or even up to a thousand square km. Conventional bombs meanwhile usually cause damage only within an area of no more than 0.1–0.2 km^2.

   In terms of energy ratio, the two differ drastically. Generally speaking, about 50% of a nuclear explosion's energy is transformed into shock wave, 35% turned into optical radiation, 5% into early nuclear radiation, and 10% into radioactive contamination (or nuclear fallout). When a conventional bomb explodes, all or nearly all energy is converted into shock wave.
3. **Effective destructive power**: The pressure of shock wave near the center of a nuclear explosion is extremely high. Take a megaton nuclear weapon for example, when it explodes upon contact with the ground, the overpressure and dynamic pressure of its shock wave are both higher than 100 kg/cm^2 within 0.4 km of ground zero. When a conventional bomb explodes meanwhile, pressure around ground zero is much lower. For instance, when 1000 kg of TNT explodes on the ground surface, overpressure at a distance of 10 m from the center of

explosion is 10.54 kg/cm^2, and plunges to 0.118 kg/cm^2 at a distance of 100 m. For the destruction of shallow-buried targets or those located on the ground surface (e.g., regular fortifications, structures, tank clusters), usually an overpressure of 3–6 kg/cm^2 is sufficient. Pressure needed to cause injury and death is even lower. Overpressure of 1–2 kg/cm^2 or dynamic pressure over 0.5 kg/cm^2 is powerful enough to be fatal to humans. It would appear that a certain portion of energy from shock wave of nuclear explosion is ineffective. If compared to an equivalent of conventional explosives, its destructive power is actually a lot more minor.

4. **Pressure increase duration and positive pressure effective duration**: Theoretically speaking, both types of explosion, if occurring in a wide, open space, would cause ambient pressure to surge to maximum value within 1 μs (1 ms = 10^{-6} s). However, empirical tests show that when 40 kg of TNT explodes, pressure increase duration is about 0.5 ms, while that of a nuclear explosion takes several to more than 10 ms. In terms of positive pressure effective duration, the differences between the two are even more pronounced. When conventional explosive explodes, positive pressure effective duration lasts anywhere from several tenths of a millisecond to several tens of milliseconds, for instance the explosion of conventional bomb or torpedo has positive pressure effective duration of 3–15 ms. The positive pressure effective duration of a nuclear explosion, on the other hand, could stretch from several tenths of a second to more than 10 s (1 s = 1000 ms). And this is precisely the reason why nuclear explosion can cause the same degree of injury as conventional explosive explosion but with lesser pressure.
5. **Pressure attenuation:** After conventional explosive explodes, pressure diminishes at a faster rate with distance compared with that of a nuclear explosion. Take for instance the explosion of 1000 kg of TNT in the air above ground. Overpressure could reach 10.54 kg /cm^2 at a distance of 10 m from ground zero, but reduces to merely 1.97 kg/cm^2 at a distance of 20 m, and down to less than 0.20 kg/cm^2 (safety threshold) at a distance of 70 m. By comparison, pressure attenuation of a nuclear explosion proceeds at a much slower rate (see Table 2).

Table 2 Comparison of pressure attenuation between conventional explosive explosion and nuclear explosion

Distance from ground zero/m	Explosion in mid-air above ground	Kiloton nuclear explosion (relative altitude 120)
0		10.2
10	10.54	10.02
20	1.97	9.5
30	0.84	8.5
40	0.49	7.5
50	0.33	6.7
60	0.25	6.0
70	0.2	5.0
80	0.16	4.5
90	0.14	3.8
100	0.12	3.6
200	0.050	1.3
300	0.031	0.79
400	0.023	0.48
500	0.018	0.36
800	0.011	0.17
1000	0.0087	0.12
1500	0.0057	0.066
2000	0.0043	0.045
2500	0.0034	0.034
3000	0.0028	0.027

4 Factors That Decide and Influence Shock Wave Injury Capacity

4.1 Physical Parameters That Influence Shock Wave Injury Capacity

The overpressure and dynamic pressure during the positive pressure effective duration of a shock wave are the main causes of injury and death, though negative pressure can also be very lethal. Physical parameters that influence a shock wave's injury capacity include its peak pressure, pressure effective duration, pressure increase duration, negative pressure, impulse, compression wave, and seismic wave in the ground, among other factors. A shock wave's injury capacity is usually measured using peak pressure, pressure effective duration, pressure increase duration, and impulse.

1. **Peak pressure** refers to the maximum value of the pressure of a shock wave (overpressure or dynamic pressure) and is shown in either kg/cm^2 or kilopascal (kPa), with 1 kg/cm^2 roughly equal to 97.98 kPa. Under most circumstances, peak pressure is the main determinant of severity of injury, with the higher the peak pressure, the more serious the injury. The common belief is that the lowest peak pressure needed to cause light blast trauma (such as ruptured ear drum or minor hemorrhage of internal organ) is around 0.14–0.351 kg/cm^2, and lowest peak pressure that is fatal ranges from 1.0 to 2.6 kg/cm^2.
2. **Pressure effective duration**: Pressure effective duration includes both the duration of positive pressure and negative pressure. Positive pressure duration refers to the time for which a shock wave's compression zone passes by a certain point of action (such as a human body), while negative pressure duration refers to the time for which a shock wave's rarefaction zone passes by a certain point of action. Both are measured in milliseconds or seconds. The longer the pressure effective duration, the more seri-

ous the injury. When a conventional bomb or explosive blows up, positive pressure duration usually lasts for only several milliseconds or several tens of milliseconds, but positive pressure duration of a nuclear explosion could last for several hundred milliseconds to more than 10 s. Thus, for a conventional bomb to cause the same degree of injury, usually a higher peak pressure is needed when compared to a nuclear explosion.

Table 3 lists the overpressure value under positive pressure effective duration required to reach 50% fatality rate (LD_{50}) for six types of animals. Based on the experiment results involving six types of animals, using weight as basis for projection, with a positive pressure effective duration of 400 ms, the pressure required to reach 50% fatality rate (LD_{50}) for 70 kg persons within 24 h is 3.7 kg/cm^2. Upon this basis, the peak overpressure required to reach 1%, 10%, 50%, 90%, and 99% fatality rate (as in $LD_{1,\,10,\,50,\,90,\,99}$, please see Table 4) for 70 kg persons within 24 h of injury under various positive pressure effective duration was also projected. According to reports and literature, when positive pressure effective duration is relatively longer (such as a large nuclear explosion with greater TNT equivalent), the overpressure required to cause the same extent of injury is usually less than positive pressure effective duration (such as a small nuclear explosion with lesser TNT equivalent).

Regardless of air explosion or ground explosion, the positive pressure effective duration of ground shock wave follows this rule: At the same distance, the higher the TNT equivalent, the longer the positive pressure effective duration. At the same relative altitude, and at a distance with the same overpressure and dynamic pressure, positive pressure effective duration and TNT equivalent cube root share a positive correlation. For example, during the ground explosion of a kiloton nuclear weapon, at a spot when overpressure is 0.45 kg/cm^2 and dynamic pressure is 0.066 kg/cm^2, its positive pressure effective duration is 0.296 s. And during the ground explosion of a megaton nuclear weapon, at a spot with the same overpressure and dynamic pressure, its positive pressure effective duration is 2.98 s. This is equal to a thousand-fold increase in TNT equivalent and ten-fold increase in positive pressure effective duration.

3. **Pressure increase duration**: This refers to the duration it takes from the onset of shock wave effect at a certain point of action until reaching peak pressure, and is shown in millisecond or second. In general, the shorter the pressure increase duration, the more serious the injury. At a wide open space, the pressure increase duration could be extremely short (usually within 1 ms for explosion of explosive, and several milliseconds to more than 10 ms for nuclear explosion). However, in an enclosed space, such as the inside of a building or a tank, pressure increase duration would prolong markedly (sometimes exceeding 100 ms). Under the latter circumstance, even when peak pressure value is higher than at the same distance in a wide, open space, and even though positive pressure effective duration is also longer, people are obviously less severely injured due to the extension of pressure increase duration.

   As reported in a document, with peak overpressure at 1.52–1.64 kg/cm^2 and positive pressure effective duration at 1.04–1.10 s, but due to the relatively long pressure increase duration (200–237 ms), none of the six tested dogs suffered any blast injury. Animal experimentation using shock tube also illustrates that when peak pressure value is reached instantaneously and when positive pressure effective duration is 400 ms, pressure for LD_{50} of dogs is 3.5 kg/cm^2. However, when pressure increase duration was prolonged to 30–150 ms, even when positive pressure effective duration sustained for 5–10 s, and with peak pressure value at 10.6–12.0 kg/cm^2, there was no animal death and their injuries were light. Therefore, the length of pressure increase duration plays an important role in a shock wave's injury capacity.
4. **Negative pressure**: Negative pressure refers to the portion of pressure within the rarefaction zone that is lower than normal atmospheric pressure, and the highest value of negative pressure is known as peak negative pressure. Negative pressure effective duration is often several times or ten times longer than positive pressure effective duration. People once believed that shock wave injuries occur with peak pressure or during pressure increase duration, and not negative pressure effective duration. In the early 1990s, the author's laboratory discovered for the first time that serious lung injury could also occur when negative pressure reaches a certain level.

Table 3 Positive pressure effective duration required to reach LD_{50} for six types of animals

Type of animal	Average weight/g	Overpressure for LD_{50} under different positive pressure effective duration/(kg cm^{-2})					
		400 ms	60 ms	30 ms	10 ms	5 ms	3 ms
Mouse	22	2.04	2.04	2.04	2.04	2.04	2.04
Rat	192	2.54	2.54	2.54	2.54	2.54	2.54
Guinea pig	445	2.40	2.40	2.40	2.40	2.40	2.40
Rabbit	1970	2.35	2.35	2.35	2.35	2.35	2.35
Dog	16,500	3.45	3.45	3.45	4.25	5.61	7.48
Goat	22,200	3.75	3.75	3.75	4.80	6.76	9.70

Table 4 Overpressure for different fatality rate of people under different positive pressure effective duration/(kg cm^{-2})

Positive pressure effective duration	LD_1	LD_{50}	LD_{99}
400 ms	2.6	3.7	5.1
60 ms	2.9	4.1	5.6
30 ms	3.2	4.5	6.2
10 ms	4.9	6.9	9.5
5 ms	9.2	13.0	17.6
3 ms	21.9	30.4	42.3

5. **Impulse**: Impulse is affected by peak pressure and pressure effective duration and refers to the sum of the different instantaneous pressures during the effective duration of different pressure. Although impulse is an indicator that reflects the relationship between a shock wave's physical quantities and its biological injury capacity, due to the difficulties involved in its measurement, it is less frequently used, and peak pressure and pressure effective duration are substituted in to represent impulse.
6. **Compression wave and seismic wave in the ground**: When a shock wave propagating in the air reaches the ground, it would compress the ground and create compression wave in the ground. When a bomb explodes upon hitting the ground, the ground burst would also directly create seismic wave in the ground, generating powerful vibrations in the ground near the center of explosion. When a compression wave propagates in the ground, its energy diminishes at a much faster pace than when the shock wave propagates in the air. Take for instance propagation in gravel soil. 2.7 kg/cm^2 of overpressure at ground surface would reduce to 1.12 kg/cm^2 at 3 m underground, and further dive to merely 0.4 kg/cm^2 at 4.5 m beneath the surface. Therefore, fortifications built deep underground are much better protected from or could wholly avoid shock wave damage, and personnel inside such fortifications would also be spared from serious or even any injury.

   When a compression wave in the ground acts on the top of a shallow-buried fortification or tunnel, reflection would occur. The overpressure of the reflection wave could be two times or more powerful than the overpressure of the incident wave, which would cause different degrees of vibration and damage to the struck fortification. Seismic wave generated from a ground burst of an explosive device hitting the ground could also cause quite serious damage to subterranean structures near surface zero. Sometimes, although a struck fortification remains undamaged, personnel inside could have been injured, and equipment and internal structural components inside the fortification might have been damaged as well.

4.2 Influence of Explosion Conditions on Shock Wave

1. **Type of explosive substance**: Although nuclear explosion and conventional explosive explosion share many similarities in generating shock wave and causing blast injury, they also differ in many other aspects. One key difference is the length of positive pressure effective duration. During a nuclear explosion, the positive pressure effective duration of the shock wave could last anywhere from several tenths of a second to more than 10 s. This prolonged effective duration is several dozen to several hundred times longer than explosion of conventional explosives, and therefore, even with the same peak pressure nuclear explosion could clearly cause much more severe injury.
2. **Weight or equivalent of explosive substance**: When the weight or equivalent of the explosive differs, the peak pressure and positive pressure effective duration generated also differ, and so would the injury have caused. Clearly, the bigger the quantity of explosive, or the higher the equivalent of a nuclear weapon, the higher the peak pressure at the same distance, and the more serious the injury caused. Similarly, under the same peak pressure condition, the bigger the quantity of explosive or the higher the equivalent of a nuclear weapon, the longer positive pressure effective duration. For example, with peak pressure condition at 0.07 kg/cm^2, when 22.7 kg of TNT explodes, positive pressure effective duration is 2 ms; when 1816 kg of explosives explode, positive pressure effective duration is 10 ms; that for a kiloton nuclear weapon (equals to 1000 tons of TNT) is 400 ms; that for a ten kiloton nuclear weapon is 900 ms; that for a hundred kiloton nuclear weapon is 2 s; and that for a megaton nuclear weapon (equals to one million tons of TNT) is 2.4 s Therefore, under the same peak pressure condition, the explosion from a larger nuclear weapon or a larger quantity of explosives would cause markedly more severe injuries than the explosion from a smaller nuclear weapon or a smaller amount of explosives underwater.
3. **Explosion method**: Different kinds of explosion methods also influence the injury capacity of a shock wave. An explosion in the air creates resultant wave, overpressure at ground zero is highest, and the wave expands outward while overpressure decreases with distance. The dynamic pressure of a ground explosion on the other hand is zero at ground zero then increases with distance until reaching its maximum at the Mach point, then gradually diminishes farther out. Therefore, overpressure-induced injuries are the worst at the center of explosion, while those caused by dynamic pressure are most severe at the Mach point. Since injury severity of personnel exposed on the

ground surface is primarily contingent on the effects of dynamic pressure, therefore, generally speaking blast injuries are relatively less severe at ground zero, and worst at the Mach point, then decreases with farther distance. Injury severity based on distance exhibits a bell curve (Fig. 3). There is no resultant wave in a ground explosion, and both overpressure and dynamic pressure are at their highest at the center of explosion, then gradually decrease with the increase in distance. Thus, in general, injuries are worst at ground zero and less serious with distance, and injury severity based on distance exhibits a slope (Fig. 4). This is why the range of injury of shock wave from an air explosion is larger than that of a ground explosion.

The relative height of explosion, also known as the relative altitude, is one of the main factors that determine the method of explosion (Fig. 5). Relative altitude is the ratio between the actual height of explosion (m) and the cube root of TNT equivalent (kg). Even for two air explosions, when relative altitude differs, effects of damage and injury also vary. When TNT equivalent varies but relative altitude remains the same, overpressure at ground zero also remains the same. Peak dynamic pressure at a distance about one time that of height of explosion also remains roughly the same, and thus the damage and injury capacity at that area also remains roughly the same. For example, when relative altitude is 120, the overpressures at the respective ground zeros of a kiloton and a megaton nuclear weapon are both 10.2 kg/cm^2; and when relative altitude is 60, overpressures at the respective ground zeros are both 128 kg/cm^2. This is because at the same relative altitude, when TNT equivalent increases so too does the height of explosion correspondingly, which is why pressure at the ground zero is also the same. When TNT equivalent remains the same while relative altitude decreases, damage and injury capacity of shock wave near the center of explosion clearly increases, but overall range of damage would decrease. For example, when a megaton nuclear weapon detonates at a relative altitude of 120 (actual height of explosion is 1200 m), maximum dynamic pressure at the ground could surpass 5 kg/cm^2 (max value of 5.15 kg/cm^2, at 1054 m from ground zero on the ground surface), and readings of ground surface dynamic pressure above 1.50 kg/cm^2 were recorded in an area within a range of approximately 1 km (600–1600 m from ground zero). Personnel exposed on the surface within this range might have limbs severed or be thrown several hundred meters away due to the powerful dynamic pressure. In an area within a range of approximately 2 km (300–2200 m from ground zero), readings of ground surface dynamic pressure above 0.6 kg/cm^2 were recorded, and personnel exposed on the surface within this range might be afflicted with ruptured cavities and revealed organs, and/or thrown several tens of meters away. However, if the same megaton nuclear weapon were to detonate at a relative altitude of 200 (actual detonation height of 2000 m), then peak dynamic pressure on the ground surface would be less than 0.6 kg/cm^2 (max value of 0.57 kg/cm^2, at 2240 m from ground zero on the ground surface), and personnel exposed on the surface are spared from or extremely unlikely to suffer serious injury. Such persons might only be wounded by internal organ (such as liver or spleen) rupture and bone fracture and are much less likely to be thrown, even then the distance of displacement would also be much shorter.

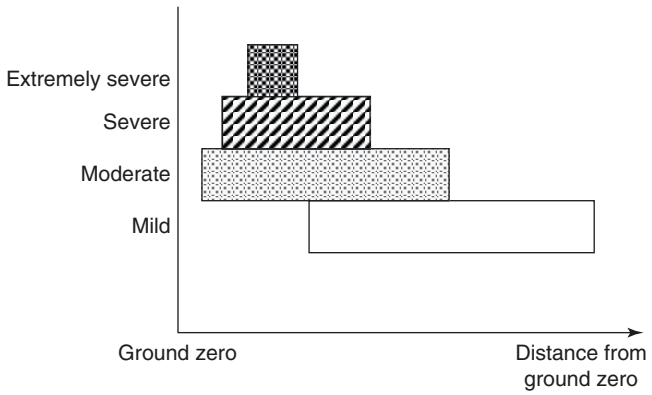

Fig. 3 Distribution of various blast injuries in the damage zone during an air explosion

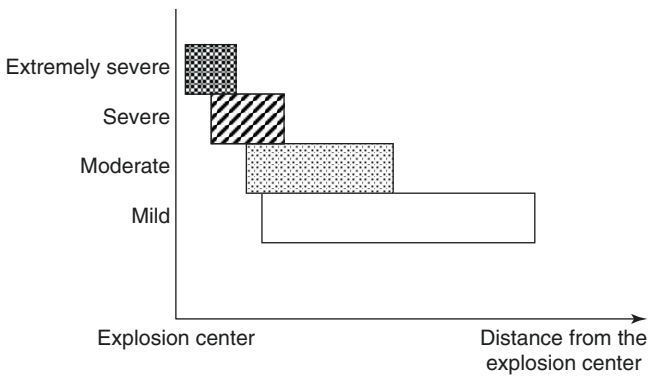

Fig. 4 Distribution of various blast injuries in the damage zone during a ground explosion

In terms of overpressure, at a relative altitude of 120, maximum overpressure on the ground surface (at ground zero) was 3.55 kg/cm^2, and at a relative altitude of 200, maximum overpressure on the ground surface was 2.66 kg/cm^2. From this, it may be determined that when relative altitude is lower, injury from overpressure will also worsen.

In terms of damage radius, at a relative altitude of 120, shock wave from the explosion of a megaton nuclear weapon is directly lethal for a distance of up to 8.2 km,

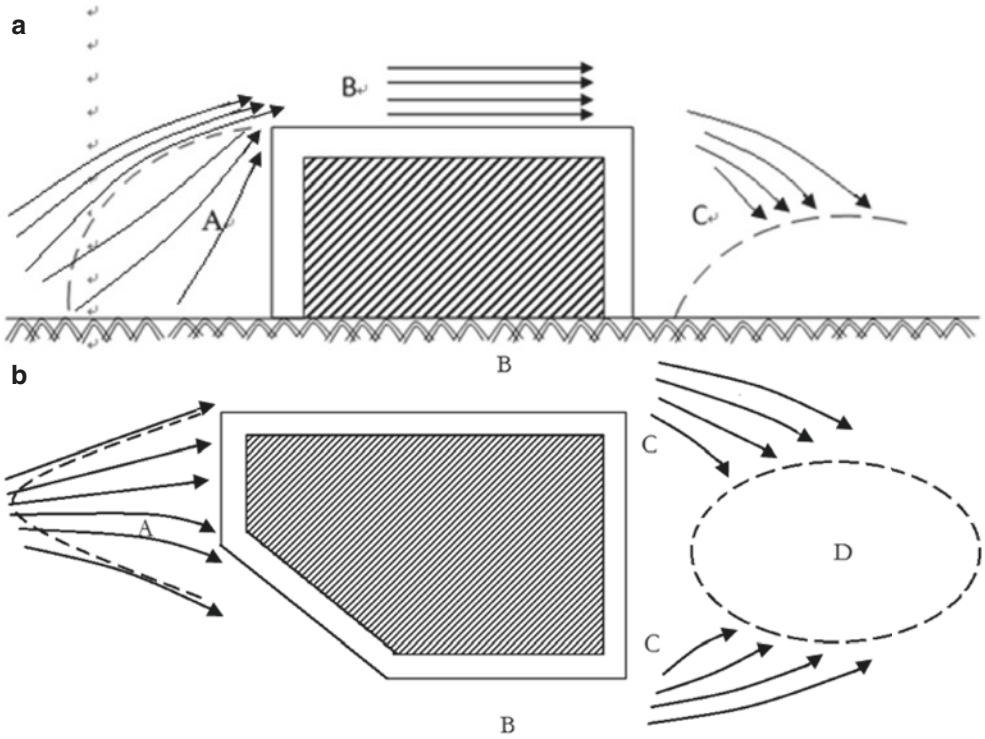

Fig. 5 Shock wave diffraction. (**a**) Viewed from the side of terrain and ground surface feature. (**b**) Viewed from the top of the terrain and ground surface feature

Table 5 Relationship between explosion method and relative altitude of 200

Explosion method	Relative altitude
Ground explosion	0~ < 60
Air explosion	
Low altitude	60~ < 120
Mid altitude	120~ < 200
High altitude	200~ < 250
Ultra-high altitude	Actual height usually several km above
Water surface explosion	0~60
Underwater explosion	Beneath water surface

and at a relative altitude of 200, the distance would increase to 9.4 km (Table 5).

4. **Formation of thermal layer due to explosion**: During a nuclear explosion, a layer of air hotter than normal temperature known as "thermal layer" would appear in an area near ground zero because of powerful optical radiation. At a relative altitude below 300, air on the ground would form a thermal layer. If the thermal layer was formed before the shock wave arrives, then thermal effect would occur after the shock wave enters the thermal layer, manifested as: reduction in the shock wave's overpressure, increase in dynamic pressure, and slight extension of positive pressure effective duration. Under the influence of the thermal layer, a ground shock wave's overpressure could reduce by as much as around 30%, while dynamic pressure on ground surface in the regular reflection region of an air explosion would soar by three to four times. Inside the irregular reflection region however, since temperature of the thermal layer isn't very high, dynamic pressure does not change as much. Thermal layer would not form on the ground if a nuclear explosion were to occur on ground surface or at ultra-high altitude above ground surface with large areas covered in ice or snow.

4.3 Influence of Personnel Conditions on Blast Injury

1. **Protection and shelter**: Under the effects of a blast wave, protection or the lack thereof makes a huge difference in injury severity. Personnel sheltered in different forms of fortification might suffer mild or no blast injury.
2. **Body position**: During an explosion, body position of personnel within the damage zone has a certain degree of influence on injury severity. When directly facing the center of explosion, the larger area of the body's frontal plane means that a person might be thrown a longer distance; when facing the center of explosion from the side, the smaller area of the body's longitudinal plane means that a person would be thrown a shorter distance. When lying down, the plane is the smallest (only about 1/5 of when standing) and usually no displacement would occur, meaning that the person is basically spared from injury arising from dynamic pressure.
3. **Other factors**: Different physical conditions also affect greatly tolerance for shock wave. For example, if the

same shock wave were to strike a young and health individual and an old and feeble individual, the injuries they suffer could vary markedly. For those with existing heart or lung conditions or other internal organ diseases, injuries are likely more severe. Animal experiments also show that weaker dogs in the same vicinity as stronger dogs also suffer more serious injuries and higher fatality rate. On a different note, when there is displacement involved, heavier individuals usually suffer worse injuries when thrown. In terms of types of animals, in general small animals have less tolerance for shock wave than large animals, while pressure effective duration's influence on injuring small animal is not obvious.

4.4 Influence of Environmental Conditions on Blast Injury

1. **Terrain and ground surface features**: When a shock wave propagates along the ground surface and encounters high ground, hill, or mountain, the frontal slope facing the center of explosion would resist the shock wave and generate reflection, resulting in a rise in overpressure (Fig. 5A). When the shock wave bends around the two sides and the top of high ground, hill, or mountain, both its overpressure and dynamic pressure would lessen to a certain extent, creating a reduced pressure zone (Fig. 5C). In areas outside the reduced pressure zone, when shock waves converge, overpressure would increase and result in a pressurized area (Fig. 5D). In general, the higher the grade the frontal slope of the high ground (as in steeper), the bigger the rise in overpressure; and the steeper the back slope, the bigger the reduction of the overpressure and dynamic pressure inside the reduced pressure zone. Taking advantage of this terrain characteristic, personnel, weapons, equipment, and other subjects should take shelter in the back slope of a high ground, i.e., the reduced pressure zone, in order to minimize or wholly avoid injury and damage from shock wave.

   Valleys also have a certain degree of influence on the propagation of a shock wave. When a valley is perpendicular to the direction of propagation of a shock wave, both overpressure and dynamic pressure would decrease. If a shock wave were to propagate along the valley, due to the reflection generated by the flanks of the valley, both the shock wave's overpressure and dynamic pressure would increase.

   In areas with dense population or structures, there is a much higher probability for people to be mutually wounded by indirect blast injuries; personnel exposed in wide, open areas are much more likely to suffer direct blast injuries. Forests, alpine areas, hills, valleys, and other landforms can weaken the effect of a shock wave to different degrees, thereby reducing injury severity of personnel situated in these locations.
2. **Environment pressure**: Environment pressure at the time a shock wave passes by also has a huge impact on the injury capacity of said shock wave. In an experiment, mice are placed in an explosion test chamber respectively at environment pressure of 0.49, 0.85, 1.27, 1.69, and 2.96 kg/cm^2 before explosion. Post-explosion pressure was quickly adjusted to the pre-explosion pressure, then the animals were placed in normal atmospheric pressure an hour later. The result indicates that pressure needed to cause 50% fatality rate in the different pressure levels are respectively 1.43, 2.20, 3.13, 3.89, and 6.46 kg/cm^2, demonstrating that the higher the environment pressure, the higher the tolerance for shock wave in the animals. For humans, most exist in standard atmospheric pressure prior to being struck by a blast wave, and as such there is no need to account for the environment pressure factor. However, for people in high-altitude areas where atmospheric pressure is comparatively lower, the lower pre-explosion pressure might result in more severe post-explosion injury.
3. **Climatic conditions**
   (a) Influence of wind: Wind speed increases with altitude. During a nuclear explosion, pressure is reduced in upwind location, and on the contrary, pressure is heightened in downwind direction. Since wind speed is always less than a shock wave's propagation velocity, thus the wind only has minimal influence on the shock wave's pressure. In areas where overpressure exceeds 0.1 kg/cm^2, strength of wind does not have much of an impact on seriousness of blast injury, and thus there is no need to account for wind.
   (b) Influence of air temperature: Air temperature is highest at ground surface, and decreases as altitude increases. This relationship is particularly pronounced during a summer afternoon. At such a time, a shock wave from a long distance away will diminish more obviously; on a cold winter day or a summer evening, the opposite might be true, as in temperature of ground surface air increases as altitude increases, and at such a time a shock wave from a long distance away will not diminish as quickly. In most cases, changes in air temperature do not have a significant effect on extent of injury of personnel inside the damage zone.
   (c) Influence of rainy weather: Shock waves might propagate a shorter distance on rainy days, and injury of personnel inside the damage zone might also be less severe.

(d) Focus of weak shock wave: After a nuclear explosion, sometimes very loud cracks could be heard several tens of kilometers or even several hundred kilometers away, and they might be loud and powerful enough to shatter windows. This kind of phenomenon might be the outcome of the "focus" of weak shock wave, as in reflection of shock wave that bounced back from the atmosphere. Therefore, it can be seen that whether or not such focus phenomenon occur, the location of focus and intensity of effect all hinge on atmospheric conditions several tens of kilometers above the explosion.

5 Evaluation of Injury Effect of Shock Wave on Humans

5.1 Criteria of Damage of Shock Wave

Criteria pertaining to damage caused by a shock wave mainly include the criterion of overpressure, criterion of impulse, criterion of overpressure-impulse, etc., of which the most commonly adopted is the criterion of overpressure. Quantified analysis of injury and damage effects of a blast wave firstly begins with ascertaining the relationship between the shock wave overpressure and blast energy generated from the explosion, then proceeds to analyzing the energy and subsequent injury and damage effects of explosions under different circumstances.

1. **Criterion of overpressure**: This criterion holds that when the overpressure of a shock wave exceeds or equals to a certain critical level, it would cause a certain degree of damage or injury to the target (structure, equipment, facility, personnel, etc.). Its applicable range is: $\omega T+ > 40$, and in the equation, ω is the target's response angular frequency (1/s), and T+ denotes duration of positive phase (s). The criterion of overpressure is only applicable to explosion originating from condensed explosive as source of explosion. Different explosion sources have different damage effects even when overpressure remains the same. For example, unconfined vapor cloud explosion is much more devastating compared with explosion of explosive even if the two create the same overpressure. The biggest shortcoming of the criterion of overpressure is that only overpressure is considered, but not the sustained duration of overpressure. Theoretical analysis and empirical experiment both indicate that same level of overpressure with different effective duration would result in different damages. Sustained duration is related to energy of explosion (Table 6).
2. **Criterion of impulse**: Damage effect is not only conditional on the shock wave's overpressure, but also directly related to the sustained duration of overpressure, which is why some people have proposed the use of impulse I (Pa, s) as a parameter for judging the damaging effect of a shock wave. This is the criterion of impulse. The definition of impulse is $\int_0^{T+} \Delta P(t)\,\mathrm{d}t$, among which $\Delta P(t)$ is overpressure. The criterion holds that when shock wave's impulse acting on a certain target reaches a certain critical level, the target would receive the corresponding level of damage. Since this criterion simultaneously accounts for overpressure, the sustained duration of overpressure and waveform, it is more comprehensive than the criterion of overpressure. However, said criterion neglects one situation, namely when overpressure is less than a certain minimum critical level, even if sustained duration is relatively long and impulse is really high, the target won't sustain any damage. The reality is that the criterion of impulse is only applicable within the range of: $\omega T_+ < 0.4$. Furthermore, different blast waveforms also cause different extents of damage even at the same impulse.

Table 6 Criteria of injury to the human body caused by overpressure of blast wave

Overpressure	Extent of injury
20~30 kPa/0.2~0.3 kg/cm^2	Mild contusion
30~50 kPa/0.3~0.5 kg/cm^2	Medium injury
50~100 kPa/0.5~1.0 kg/cm^2	Severe wound
>100 kPa/>1.0 kg/cm^2	Extremely severe, death probable

Note: 1 kg/cm^2 = 98 kPa

3. **Criterion of overpressure-impulse**: In the 1970s, the Naval Ordnance Lab and Ballistic Lab of the U.S. Navy carried out a substantial amount of experimental and theoretical studies, and gradually formed an overpressure-impulse damage model. Said model holds that effect of damage is jointly determined by overpressure ΔP and impulse I. If different combinations of these factors satisfy the below conditions, then they would generate the same effect of damage. In the diagram, P_{cr} and I_{cr} are respectively critical overpressure and critical impulse for damaging the target, C is constant, and they are all associated with the nature of the target and level of damage. On the ΔP–I plane, the equation represents a damage curve. $\Delta P < P_{cr}$ or $I < I_{cr}$ are safe areas, and all other areas are damage areas. The closer the location to the upper right hand corner of the plane, shock wave at coordinate (ΔP_a,I_a) results in larger damage (Fig. 6). Most believe that the criterion of overpressure-impulse is applicable to shock waves created by condensed explosive, and also usable with unconfined vapor cloud explosion, dust explosion, and other instances.

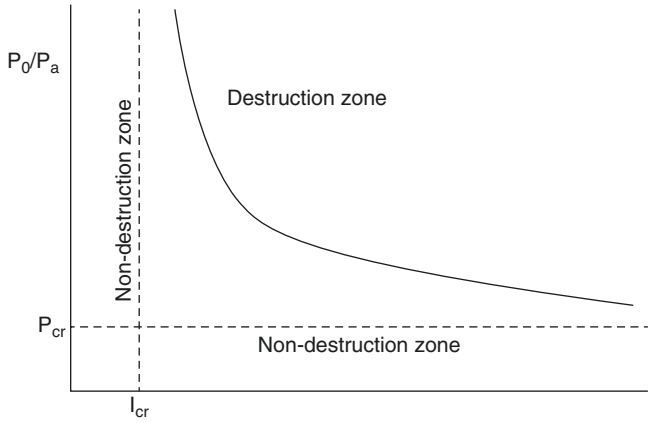

Fig. 6 Overpressure-impulse damage criterion of explosion shock wave

Table 7 Immediate effects of blast wave

	Mechanism of injury	Effect of injury
Primary blast injury	Direct effect (overpressure and negative pressure)	Eardrum rupture, lung injury, cavity and internal organ rupture
Secondary blast injury	Shrapnel and fragments from explosion	Penetrating wound and fragment wound
Tertiary blast injury	Being tossed and collapse of buildings	Head and brain trauma, crush injury, blunt injury, penetrating wound, bone fracture, and severed limb
Quaternary blast injury	High temperature, toxic gas, and lack of oxygen	Burns, suffocation, and poisoning

5.2 Injury Effects of Blast Wave on Humans

Injury effects of blast wave on humans include direct injury effects, displacement injury effects, explosion fireball's thermal radiation injury effects, structural collapse injury effects and high-speed explosion fragment injury effects, among others. Based on the cause of injury, blast injuries are usually categorized as one of four types (Table 7), as in primary blast injury directly resulting from the shock wave, secondary blast injury resulting from projectiles like shrapnel, tertiary blast injury resulting from being thrown by the force of the shock wave, and quaternary blast injury resulting from miscellaneous causes such as burns, suffocation, or toxicity. The type of blast injury sustained is closely related to the environment in which the explosion happened.

1. **Direct injury effects of shock wave** refer to injuries or deaths caused when a shock wave directly acts on the human body. Air-filled organs such as the lungs, auditory apparatus, and gastrointestinal tract are the main target organs of this type of blast injury. Overpressure could also cause internal organ rupture, bone fracture, and other issues. This kind of blast injury is the direct result of the shock wave from a blast. Victims of this type of pure blast injury might appear uninjured on the outside, but already suffered different degrees of internal organ damage. Thus, this type of injury is characterized by less severity outside and more severity inside. When this kind of blast injury is compounded by other types of injury, usually injuries on the surface would be very apparent, while internal injuries might instead be neglected, frequently leading to missed diagnosis and misdiagnosis. The lung is a vital organ most easily directly injured, while the ear is the non-vital organ most easily directly injured. Therefore, an analysis of this kind of direct injuries should commence with the lungs and ears.

   (a) **Lung blast injuries**: The lung is a vital organ most easily directly injured. During an explosion, the chest cavity and pulmonary alveoli are compressed, and after the effect of positive is the effect of negative pressure. On the one hand, the overexpansion of the lungs collide against the thoracic cage, causing the typical parallel bands of bleeding on the surface of the lungs. On the other hand, compressed gases inside the pulmonary alveoli expand rapidly, causing abrupt changes in blood flow dynamics inside the lung, rupturing the alveolar walls and causing hemorrhage of the lung parenchyma. Therefore, the author's research group proposes the theory of "over-tension" in shock wave-induced lung injuries.

   The extent of shock wave-induced human body injury is dependent on not only the properties of the shock wave (e.g., waveform, overpressure, impulse), but also conditional on environment pressure, the geometric position of the human body in relation to the blast wave, the weight and age of the victim, other objects and obstacles near the victim, and numerous other factors. In the real world, most people are standing, and the shock wave's position in relation to a victim's body usually falls into either one of two types: (1) The person is standing on the ground, the shock wave's propagation direction is perpendicular to the person's height, and there are no obstacles near the person. This is the most common scenario. At this juncture, the sum of overpressure P acting on the human body is: $P = P_a + 5P_a^2/(2P_a + 14 \times 10^6)$. P_a is peak overpressure of incident wave. (2) The person is standing on the ground, the shock wave's propagation direction is perpendicular to the person's height, and the person is near a vertical obstacle (which would function like a reflection plane). Persons exposed in this scenario are most at risk. At this juncture, the sum of overpressure P acting on the human body is: $P = (8P_a^2 + 14P_a \times 10^6)/(P_a + 7 \times 10^6)$.

Pathological changes of lung blast injuries include hemorrhage of pulmonary alveoli and lung parenchyma, hematocele in alveolar or interstitial edema, subpleural emphysema, lung rupture, and other issues, which could lead to pneumothorax, hemothorax, and atelectasis. Clinical manifestations include: chest pain, chest tightness, cough, hemoptysis, and other symptoms after injury, and those with serious injuries will exhibit obvious difficulty breathing, cyanosis and hemoptysis with frothy sputum, then worsen to acute respiratory dysfunction syndrome (ARDS) within 24–48 h. Auscultation might discover weak breathing sound, moist rale, and crepitus. Chest X-ray might reveal more noticeable lung markings, and spots or swaths of shadows. Chest ultrasound can help diagnose fluid accumulation in the lungs.

(b) **Auditory apparatus blast injuries**: The ear is the non-vital organ most easily directly injured, as the ear can perceive even the slightest of changes in pressure. Serious shock wave-induced injuries of the ear include ruptured eardrum, hematotympanum, severed ossicle chain, and others. There may also be oozing blood, bleeding, cochlea structure disorders, and other issues in the inner ear. Clinical manifestations include deafness, tinnitus, earache, dizziness, headache, and other signs. Deafness is usually conductive, but could also be mixed hearing loss. Fluid and bloody liquid might flow from external auditory canal. As of present, studies conducted on auditory apparatus blast injuries number far fewer than those carried out for lung blast injuries, and different researchers have produced conclusions that drastically differ. Some opine that 44 kPa of overpressure in the incident wave is enough to rupture eardrums 50% of the time. Other studies indicate that incident wave overpressure must reach at least 103 kPa for a 50% chance of causing ruptured eardrums. The relational expression for eardrum rupture probability and peak overpressure is: $P_r = -12.6 + 1.524$ In Pa. Said equation can project eardrum rupture probability for a person a certain distance away from the source of explosion. For example, when $P_a = 400$ kPa, the probability of ruptured eardrum is 98%.

(c) **Abdomen blast injuries**: When a shock wave's overpressure acts on the abdomen, gastrointestinal tract and bladder might rupture. Immense overpressure and dynamic pressure could also rupture or cause bleeding in mesenteric vessels, and solid organs such as the liver and spleen. Main clinical symptom is abdominal pain, accompanied by problems like nausea and vomiting. Abdominal examination might show signs of peritonitis such as pain upon contact, rebounding pain, and muscle tension. Serious peritonitis and hemorrhage might trigger shocks. Abdominal X-ray might reveal free intraperitoneal air from perforated abdominal organ. Perforation might cause leakage of gastrointestinal tract content, urine, blood, or other substances from abdominal organs.

(d) **Head and brain blast injuries**: A shock wave might transfer inside the skull and subsequently change intracranial pressure, and might even cause blood in the trunk to rush from the jugular vein and vertebral vein to the brain. Main pathological changes include congestive spotty hemorrhage and edema of the brain and meninges. When combined with lung blast injury, the outcome could be cerebral vascular embolism. When combined with mechanical trauma, the outcomes could be skull fracture, intracranial hematoma, cerebral contusion, and other problems. Clinical manifestations are: loss of consciousness that could sustain from several minutes to several days. After waking up, the victim might exhibit signs such as emotional apathy, depression, anger, insomnia, and memory loss. Severe cases might show intracranial hypertension and other local/focal symptoms. Electroencephalogram might show abnormal waveforms.

2. **Indirect injury effect of shock wave**: Shock wave's indirect injuries constitute the absolute majority of injuries from the explosion of a nuclear weapon or that of a relatively large conventional bomb. Indirect injury effects of shock wave chiefly fall into two categories: One is injury caused from projectile in the form of shrapnel of the detonation product, or object (e.g., fragment, sand, pebble) that has been given kinetic energy due to the dynamic pressure of the explosion's shock wave; and the second is being struck with parts of structures or fortifications collapsing or breaking off because of the force of the shock wave.

(a) **Injury from projectiles**: Investigations into many large-scale bombings and statistics from the atomic bombing of Japan illustrate that many and different types of open wounds were created from secondary projectiles. In cities, industrial sites and residential areas, most secondary projectiles are glass shards from windows, in open spaces meanwhile rocks and even dust or dirt could be "weaponized" as projectiles. An on-site investigation of an explosion of 4462 kg of ammonium nitrate shows that a crude wooden shed within a distance of 20 m from the center of explosion was completely obliterated, and wooden furniture, clothes, and other items in the shed were turned into fragments, and the body of the dead was damaged to such a degree as to render it impossible to identify the victim. All glass panes of win-

dows and doors at a three-story staff dormitory 185 m from the center of explosion were shattered, some windows and doors were thrown several meters away, and many persons inside were afflicted with multiple fragment wounds or other external injuries. Some glass panes of windows and doors at another employee dormitory 300 m from the center of explosion were shattered, some windows and doors were damaged, and a small portion of persons inside were afflicted with fragment wounds. Some glass panes of windows and doors at an office building 1000 m from the center of explosion were shattered, and some windows and doors were also damaged, but no one was injured because the building was empty at the time of the explosion (nighttime incident). Nuclear weapon detonation test sites show that animals placed at wide, open areas with relatively high post-explosion dynamic pressure are afflicted with multiple external injuries from "weaponized" sand and rocks, and wounds are sometimes clumps of dots. Sand and rocks with relatively high kinetic energy might penetrate into an animal's body.

(b) **Thrown and collision effects from shock wave's dynamic pressure**: When dynamic pressure reaches a certain power, it becomes a force of impact or projection, which, when acting on the human body, could displace a person (without leaving the ground) or throwing a person (leaving the ground) up in the air before he or she hits the ground again. Such movements would cause different kinds of injuries, and foreign papers usually call this tertiary blast injury.

When a body is being thrown, the initial state is acceleration, and then abruptly decelerates as the body lands on the ground or collides with an object. Hi-speed photography materials depict that when a person is thrown, initially it is affected by the rate of acceleration, manifested as obvious displacement of the body and its organ relative to each other. When the body hits the ground, it is affected by an extremely sudden deceleration. The severity of the wound sustained is determined by the trajectory of the projection, speed before collision, and a myriad of other random elements such as the position and angle of collision, the characteristics of the surface of collision, duration of deceleration, and a plethora of other factors. The thrown human body could suffer injuries both during the acceleration and deceleration phases, but the majority of injuries occur during the latter. Injuries arising from displacement or being thrown are chiefly manifested as superficial abrasion, subcutaneous soft tissue contusion, internal organ bleeding or rupture, bone fracture, and other problems, very similar to those sustained from falling or car accident.

Why does the body get thrown under the effect of dynamic pressure? This is because when the shock wave acts on the body, the side of the body facing the center of explosion has to sustain the sum of overpressure and dynamic pressure, and the pressure received by both sides of the body equal to the overpressure at the front of the shock wave, while the opposite side of the body has a lot less pressure. Due to the difference in pressure between the four sides of the body, thus the body is displaced parallel to the ground and toward the side away from the center of explosion. When the body is "blown" by the blast wind, the air above scatter more than the air below, thereby creating an uplifting force. The body is therefore thrown by the joint upward and forward forces. As witnessed after the explosion of two tons of nitramine, some personnel close to center of explosion were thrown several tens of meters away, some flying as far as over a hundred meters. A body was found to have been tossed on a roof 70 m away, and created a huge hole after crashing through the roof before landing inside the locked room. Some severed limbs were found on the roof of an office building 120 m away.

During a nuclear explosion, dynamic pressure creates even stronger blast wind. As commonly known, a force 12 typhoon can bring down houses and injure people, and usually windspeed would reach 40–50 m/s. But during a nuclear explosion, when dynamic pressure reaches 0.1 kg/cm^2, air velocity would increase to around 100 m/s, and surpass 300 m/s when dynamic pressure tops 1 kg/cm^2. Under such circumstances, exposed personnel would very likely sustain injury from being thrown or displaced.

Based on theoretical projection, a 72 kg person could be injured when displaced at a speed of 3.048 m/s. Generally speaking, if other conditions (such as hardness and smoothness of area of landing) remain roughly the same, the farther the distance thrown, the higher the chance and seriousness of injury. People of different weights also suffer different degrees of injury upon being tossed. Usually heavier persons are injured more severely than lighter persons.

When dynamic pressure is relatively low, the body might not be thrown or displaced, but could still be injured after falling from being "blown." In most cases, an injury arising from being "blown" down is much milder than being tossed or displaced, but the chance of occurrence is also far higher.

(c) **Crushing and collision effects of falling structures**: Above-ground structures or simple underground fortifications near the center of explosion might partially

or wholly collapse due to the effect of a powerful shock wave, which could crush or collide with personnel within, causing injuries such as surface soft tissue and internal organ trauma, bone fracture, and other afflictions. More severe cases might be crush syndrome. When a fortification covered under a thick layer of earth collapses, people inside might be buried, then suffocate because of excessive amount of dirt clogging the respiratory tract. If a nuclear weapon were to detonate in an urban area, secondary injury from indirect damages caused between objects could be widespread and victims could be numerous. This problem is one of the prominent issues in the deployment of nuclear weapon. Based on investigations of the Japanese cities of Nagasaki and Hiroshima, among all blast injury victims, those injured indoor accounted for 80.3% of total, of which the absolute majority suffered from this kind of secondary injury.

5.3 Blast Wave Damage and Injury Zones

In order to predict injury and casualty in an explosion, area of effect may be classified into four zones based on distance from the center of explosion. The following equation uses overpressure ΔP based on horizontal distance from source of explosion to identify the zones:

$$\Delta P = \left(0.1 < \Delta P\left(\text{atm}\right) < 10\right)$$

$$\Delta P = \left(\Delta P\left(\text{atm}\right) > 5\right)$$

In the equation, $Z = R/(E/P_0)^{1/3}$, R denotes horizontal distance (m) between target and source of explosion, E is total energy (J) of source of explosion, and represents P_0 environment pressure.

1. **Fatality zone**: Personnel inside this zone who lack protection are expected to suffer severe injury or death without exception. Its internal radius is zero and external radius is $R_{0.5,}$ indicating that personnel at the circumference have a 50% chance of dying from pulmonary hemorrhage because of effects of the shock wave, and the relationship between it and explosion energy is confirmed via the following equation: $R_{0.5} = 13.6\left(\frac{W_{\text{TNT}}}{1000}\right)^{0.37}$, wherein W_{TNT} denotes TNT equivalent (kg) of energy of source of explosion.
2. **Severe injury zone**: Personnel inside this zone who lack protection are expected to suffer severe injury, and an extremely small minority might suffer death or mild injury. Its internal radius is $R_{0.5}$ and external radius is $Re_{0.5}$, indicating that personnel at the circumference have a 50% chance of suffering from eardrum rupture, and its shock wave peak overpressure requirement is 44 kPa.
3. **Mild injury zone**: Personnel inside this zone who lack protection are expected to suffer mild injury in the absolute majority of cases, and a small minority might suffer severe or no injury, while death is extremely unlikely. Its internal radius is $Re_{0.5}$ and external radius is $Re_{0.01}$, indicating that personnel at the circumference have a 1% chance of suffering from eardrum rupture, and its shock wave peak overpressure requirement is 17 kPa.
4. **Safety zone**: Personnel inside this zone who lack protection are expected to suffer no injury in the absolute majority of cases, and the probability of death is almost zero. Its internal radius is $Re_{0.01}$ and external radius is infinitely big.

Table 8 displays simulations of damage and destruction radii of several kinds of common explosions calculated using the method above and under the assumption that the environment pressure is 101.3 kPa. When the same mass of TNT, nitro-cotton, and propellant explodes, TNT has the largest damage and destruction radius, nitro-cotton has the second largest damage and destruction radius, and propellant has the smallest damage and destruction radius. For instance, when the mass is ten tons, the respective damage and destruction radii of TNT, nitro-cotton, and propellant are 39.6 m, 38.0 m, and 34.9 m, and the respective property damage and loss radii are 144.4 m, 138.6 m, and 126.8 m. As the mass of explosive substance rises, damage and destruction radius also increases significantly. For example, when 10 t, 20 t, and 30 t of TNT explode, the respective damage and destruction radii are 39.6 m, 51.2 m, and 59.5 m, and the respective property damage and loss radii are 144.4 m, 184.1 m, and 211.3 m.

Table 8 Simulations of damaging effects of explosions of different kinds of explosives

Explosive	TNT equivalent/T	Heat of detonation/ (kJ kg^{-1})	Death radius/m	Severe injury radius/m	Mild injury radius/m	Property damage and loss radius
TNT	10	4520	39.6	100.6	180.6	144.4
	20	4520	51.2	127.2	228.2	184.1
	30	4520	59.5	145.5	260.5	211.3
Propellant	10	3197	34.9	89.9	160.9	126.8
Nitro-cotton	10	4040	38	97	174	138.6

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Injury Principles and Mechanisms of Shock Wave

Zhengguo Wang, Zhihuan Yang, and Haibin Chen

1 Injury Principles of Shock Wave

When a shock wave propagates through the air, two zones that resemble a double-layered sphere are formed: The outer shell is the compression zone, and the inner shell is the rarefaction zone. Inside the compression zone, pressure exceeds normal atmospheric pressure because air is compressed, and the portion of pressure that exceeds normal atmospheric pressure is called overpressure. Force of impact created by shock wave during its high-speed movement is known as dynamic pressure. The front of the compression zone is called the front of the shock wave, and at this front both overpressure and dynamic pressure are at their peaks, which are respectively termed peak overpressure and peak dynamic pressure. Inside the rarefaction zone, due to the vacuum created after air is compressed, air in this region is highly rarefied, and air flows toward explosion center in the opposite direction. Air in this zone has less pressure than normal atmospheric pressure, and the portion of pressure that falls below normal atmospheric pressure is called the underpressure of the shock wave, while the maximal underpressure is termed peak underpressure. The overpressure and dynamic pressure of the shock wave are the main causes of injury, and underpressure can also lead to apparent injury.

Physical parameters related to blast injury are introduced below:

1. Peak pressure. This refers to the maximum value of the shock wave's overpressure or dynamic pressure, and in the past the unit of measurement was either kg/cm^2 or psi (pound per square inch), but today the standard unit has been changed to kPa (1 kPa = 0.0102 kg/cm^2 or 0.145 psi). This is the main parameter to consider when judging a shock wave's injury capacity. The higher the peak pressure, the more serious the injury. A pressure of 34.5 kPa is capable of causing mild injuries (ruptured eardrum), and a pressure of approximately 690.6 kPa is enough to cause death (referring to injuring/killing effect of shock wave on an exposed person within a very short period).
2. Positive pressure effective duration. This refers to the time for which a shock wave's compression zone passes by a certain point of action (such as the human body surface), and it is measured in seconds or milliseconds. Within a certain time limit, the longer the pressure effective duration, the more serious the injury. When a conventional bomb or explosive blows up, positive pressure effective duration usually lasts only several milliseconds or tens of milliseconds, but positive pressure effective duration of a nuclear explosion could last for several hundred milliseconds to more than 10 s. Thus, under the same peak pressure, a nuclear explosion would cause more severe injury than that of a conventional bomb.

   Pressure increase duration refers to the duration it takes from the onset of shock wave effect at a certain point of action until reaching peak pressure, and it is measured in millisecond or second. Other conditions being equal, the shorter the pressure increase duration, the more serious the injury. For example, pressure increases slower inside a building or a tank and requires a longer duration, while pressure increases at a much faster pace in a wide and open space. Therefore, if peak pressure remains the same for the two above instances, people inside the relatively enclosed space would be less severely injured.

2 Injury Mechanisms of Shock Wave

The injury mechanisms of shock wave are comparatively complex. The mechanisms of subsequent blast injury and tertiary blast injury are similar to general mechanical trauma, but primary blast injury is more unique.

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1. Direct effects of overpressure and underpressure
   (a) Changes in hemodynamics: After a shock wave's overpressure acts on the body, on the one hand, the pressure pushes against the abdominal wall, causing pressure inside the abdominal cavity to rise, in turn pressing the diaphragm upward, causing blood in the inferior vena cava to abruptly rush into the heart and lungs, sharply increasing blood volume in these organs. On the other hand, the shock wave's overpressure also presses against the chest cavity, decreasing the volume of the space behind the chest, resulting in an abrupt increase of pressure inside the chest cavity. Right after overpressure is underpressure, and the pressure decrease would cause the chest cavity to enlarge. This kind of rapid compression and expansion generates huge hemodynamic changes in the chest cavity, resulting in vascular injuries in the heart and lungs. The authors and others have witnessed in animal experimentation that during the moment of injury when struck by shock wave, the chest cavity pressure inside the struck animal (dog) rose by approximately 650 mmHg (86.7 kPa), while pressure in pulmonary arteriole climbed by more than 400 mmHg (53.3 kPa). This abrupt surge in intravascular pressure inevitably causes hemodynamic disorders (Fig. 1).
   (b) Pressure difference: When a shock wave acts on the body, pressures in both the liquid of the lungs (blood) and air (alveolar air) would rise, but pressure in the liquid would increase more, creating a massive pressure difference between the two. The high-pressure liquid flows toward the low-pressure air, creating tears in capillaries and leading to lung hemorrhage. The pressure difference then quickly reverses; the air portion of the lungs has higher pressure than the liquid portion, forcing the air into the liquid, resulting in air embolism. Eardrum rupture can also be explained by the difference in pressure between the external auditory canal and the tympanic cavity (Fig. 2).
   (c) Spalling effect: When a pressure wave propagates from a relatively dense medium into a relatively loose medium, reflection would occur at the interface between the two. The sudden rise in local pressure on the surface of the relatively dense medium causes damage, manifested in injuries such as that of the alveolar wall.

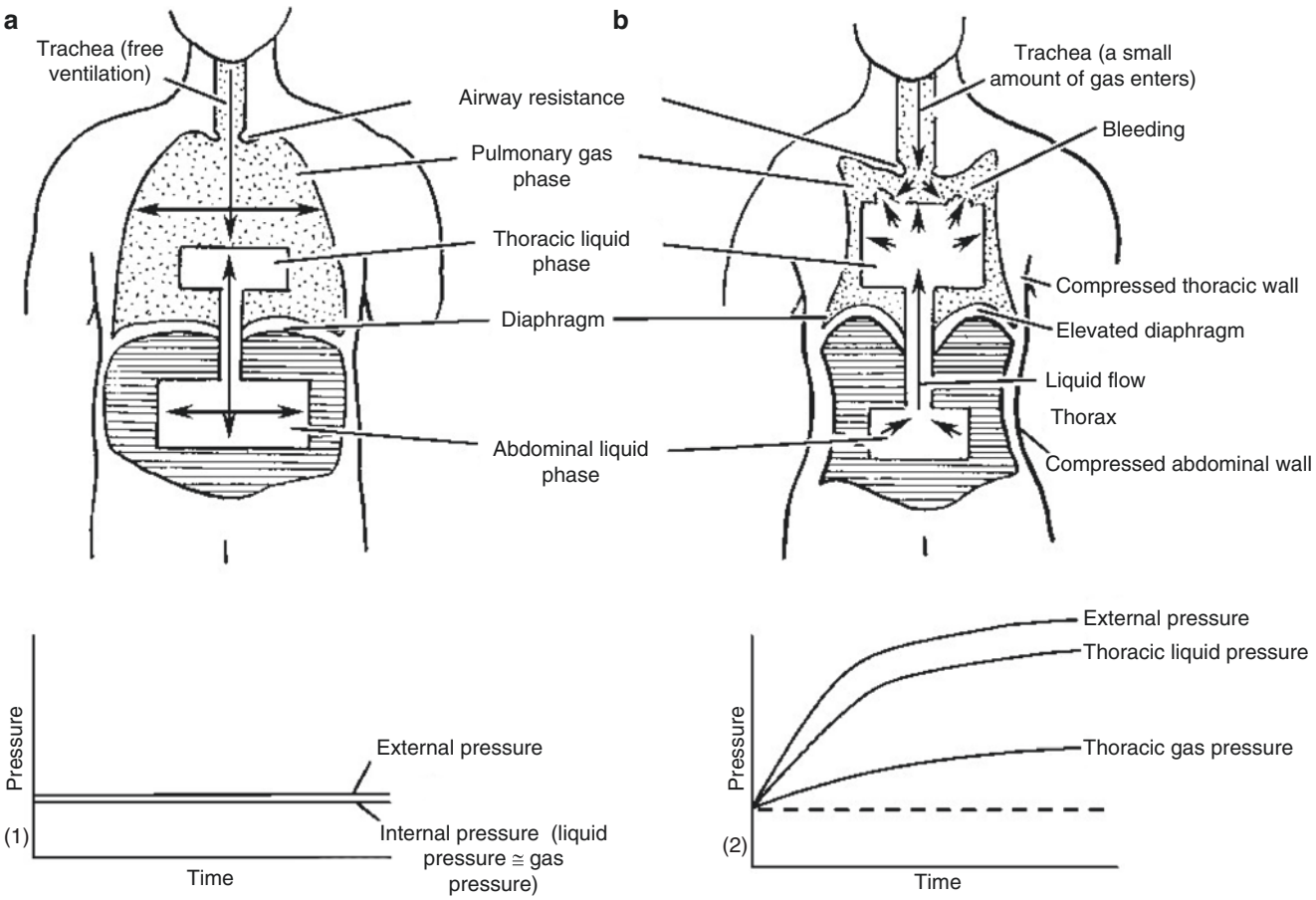

Fig. 1 Hemodynamic changes in blast injury. (**a**) Normal hemodynamics; (**b**) Hemodynamics after overpressure

(d) Implosion effect: When a pressure wave passes by a human body, the liquid inside is not easily compressed, but air compresses by a much larger extent. The effect of overpressure is followed immediately by the effect of underpressure, and the previously compressed air would rapidly expand, much like an internal explosion, damaging surrounding structures such as the alveolar wall (Fig. 3).

(e) Inertia effect: When pressure wave acts on tissues of different density, it moves at different velocity due to difference in inertia. Movement is faster in areas with low density and vice versa. Therefore, areas of connection between tissues of different density are prone to tearing, for instance, bleeding between the ribs and intercostal muscles.

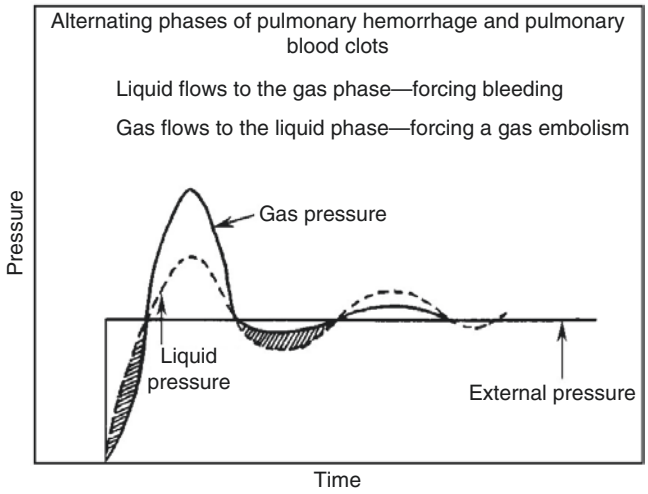

Fig. 2 Injury mechanism of pressure difference caused by shock wave

With regard to injury mechanisms of overpressure and underpressure, a number of new discoveries and new concepts have emerged in recent years, particularly those related to biomechanical effects of pressure. In the past, most believed that underpressure does not cause much injury, a point of view substantiated by the limited extent of changes in underpressure and the ceiling of 98.06 kPa (1 atm) on peak underpressure. However, recent studies have found that underpressure is highly capable of causing severe injuries like those arising from overpressure, including hemorrhage, edema, alveolar rupture, and microthrombosis. Injury parameters include pressure decrease rate, peak underpressure, and underpressure effective duration, of which, peak underpressure is the most important. In addition, sometimes the ratio of absolute pressure values between atmospheric pressure and reduced pressure might be more useful. Experiments indicate that as peak underpressure increases, incidence rate of lung injury in rats would also elevate, along with increases in the ratio of lung weight to body weight multiplied by 100% and the area of pulmonary hemorrhage.

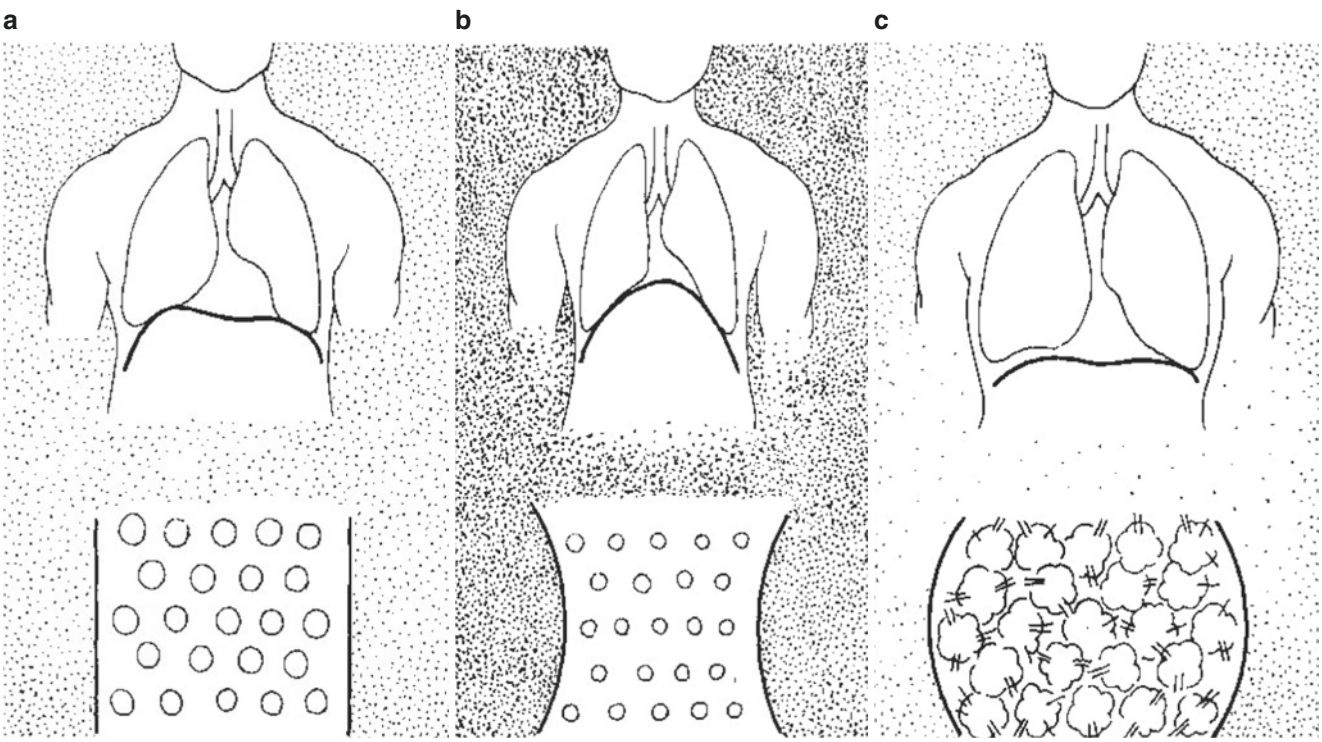

Fig. 3 Schematic diagram of implosion effect. (**a**) Normal atmospheric pressure; (**b**) Overpressure action; (**c**) After action

The author's laboratory once used underpressure generator of shock wave to simulate pure blast underpressure, in an effort to observe dynamic responses in rats, isolated lung system and air-filled swim bladder under blast underpressure. Hi-speed photography revealed that under the effects of blast underpressure, the expansion, speed, and acceleration of isolated lung system were higher than the corresponding expansion, speed, and acceleration of rat's chest. Swim bladder expanded very obviously and also experienced rupture during the expansion, demonstrating expansion-induced injury of pulmonary alveoli under blast underpressure. It can therefore be deduced that the effects of blast underpressure induce rapid expansion of the thoracic wall and lung tissue, and at a certain point, lung tissue expands at a faster rate than that of the thoracic wall. Given the difference in movement of the two, lung tissue is likely to collide against the thoracic wall, resulting in bleeding on pulmonary surface.

In addition, micro acceleration sensors and piezoresistive pressure sensors were employed to respectively measure the rabbit's acceleration of thoracic wall and intrapleural pressure under the effects of blast underpressure. A portion of the curvilinear integral is used to obtain the speed and displacement curve. Experiment results show that under the effects of blast underpressure, thoracic wall outward movement could reach the 100 g-level in acceleration, expansion speed was roughly 0.5 m/s, and displacement from expansion was about 1 mm. Thoracic wall movement did not show obvious recompression, and pleural cavity internal pressure started with underpressure, and followed by positive pressure of a certain intensity, revealing possible collision between the lungs and thoracic wall.

Another experiment shows that injuries from shock wave mostly occur during the lung tissue expansion. For instance, if the chest of a rabbit is wrapped with single-layer nylon strip (5 cm wide and 20 cm long) before hit by shock wave in order to restrict excessive expansion, the extent of post-shock wave lung hemorrhage in the animal was far less compared to animal without the wrapping of the nylon strip. This case illustrates that limiting the rapid expansion of the lungs is an obviously effective protection against shock wave, as injuries from underpressure are precisely induced by excessive expansion of lung tissue.

Moreover, shock wave biomechanics research indicates that the body's response to shock wave mainly undergoes three biological stages:

(a) The body surface's rapid response to impact load of the shock wave: The load of the shock wave acting on the body's surface is known as impact load. The side of the body facing the wave source receives the biggest impact load, and the geometric shapes of the body structure and tissues may cause the shock wave to diffract or focus. The load borne by some partially open enclosed structure (such as the pulmonary alveoli) is much greater than load borne in open areas. After the impact load acts on the body, tissues and organs would deform.
(b) Organ deformation and tissue stress: The rapid displacement of the thoracic wall may compress certain lung tissue, but the energy could not be readily released through the air passage, resulting in stress imparted on the lung tissue. In another scenario, when the abdominal cavity is suddenly subjected to pressure, some air-filled parts of the gastrointestinal tract would collapse, creating stress on the intestinal wall.
(c) Tissue stress and injury: A certain level of stress can cause tissue hemorrhage or damage, and the severity of injury hinges on composition, structure of tissue, and how energy is applied. When tissue retracts and elongates to 150% of its original length, stress would quickly escalate, resulting in tear, and this part of the energy dissipates in the tissue. Based on the physical process of how the body reacts to shock wave as explained above, American scientists Stuhmiller et al. used finite element modeling (FEM) to simulate how different organs respond to the effects of shock wave. The experiment revealed unevenness in distribution of pressure inside the lungs and focal points of stress inside tissue (as in areas more prone to injury), but was not able to uncover the relationship between stress inside tissues and deformation of tissue. Therefore, the concept of tensile strength (resistance of a material to breaking under tension) remains inapplicable in determining severity of injury.

Overexpansion effect or decompression effect: Based on both Chinese and foreign papers, and experiments conducted in the author's laboratory, we propose a new notion that lung injury from shock wave does not occur during the compression stage, rather during the decompression section and underpressure section, which we term overexpansion effect or decompression effect. To prove this notion, we independently studied and developed a staged shock wave simulation cabin to investigate the injury effects from shock wave during its compression stage, decompression section, and underpressure section.

The simulation cabin is comprised of the high-pressure cabin, diaphragm rupture section, low-pressure cabin and corresponding components (Fig. 4). They respectively simulate injury effects from shock wave during its compression stage, decompression section, and underpressure section. Its usage method and results are as described and shown below:

Compression wave experiment: Test animal is placed in Cabin B, which has normal pressure, and a certain

amount of oxygen is injected; Chamber A is filled with compressed gas, and upon reaching peak pressure, diaphragm would rupture, gas in Chamber A would quickly flow into Chamber B, causing the latter's pressure to rapidly rise. When pressure in Chamber B reaches peak value of 0.32 Mpa over the duration of 1–2 ms (Figs. 5 and 6), thereby subjecting the test animal in Chamber B to the effects of the compression wave, and after maintaining this state for 1 min, pressure is gradually reduced to normal pressure at a rate of 0.037 MPa/min (pre-experiment already proves that this kind of slow decompression itself does not cause any injury to the lungs). Result: No obvious injury to the lungs of the animals (common rabbit and rat) were caused by the compression wave (Table 1).

Decompression wave experiment: Test animal is placed in Chamber A, and a certain amount of oxygen is injected. Pressure in Chamber A is slowly elevated at a rate of 0.07 MPa/min, and upon reaching predetermined level of high pressure (pre-experiment already proves that this kind of slow pressure increase itself does not cause any injury to the lungs), diaphragm ruptures, gas in Chamber A would quickly flow into Chamber B, thereby subjecting the test animal in Chamber A to the effects of the decompression wave. Result: Under the effects of decompression wave, no injury, mild injury, or severe injury may result in the lungs of rabbit depending on the duration of decompression (Figs. 7 and 8, Table 2). As decompression duration shortens, lung injury score (IS) rises.

Underpressure wave experiment: Test animal is placed in Chamber B, which has normal pressure, while air in Chamber A is sucked out to create an approximately vacuum environment, causing the diaphragm to rupture. Then gas in Chamber B would quickly flow into the near-vacuum of Chamber A, creating underpressure wave in Chamber B (Figs. 9 and 10), thereby subjecting the test animal in Chamber B to the effects of the underpressure wave. Result: Rabbits suffered a certain degree of injury (Table 3).

The various shock wave stages simulated in this experiment are somewhat different than those in real shock waves, for instance when compression wave pressure

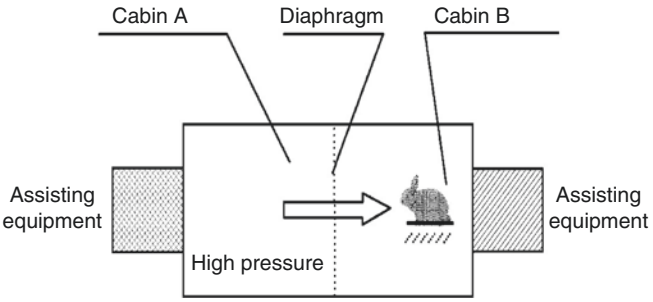

Fig. 4 Segmented simulation cabin of shock wave

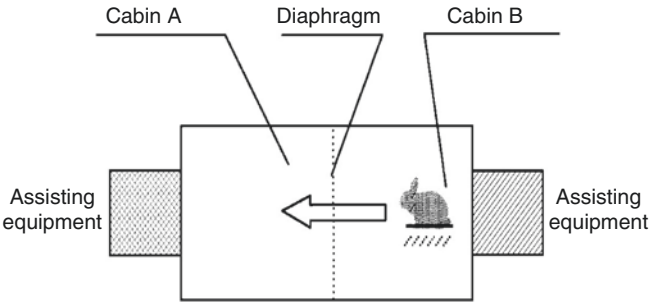

Fig. 5 Simulation cabin in compression section of shock wave

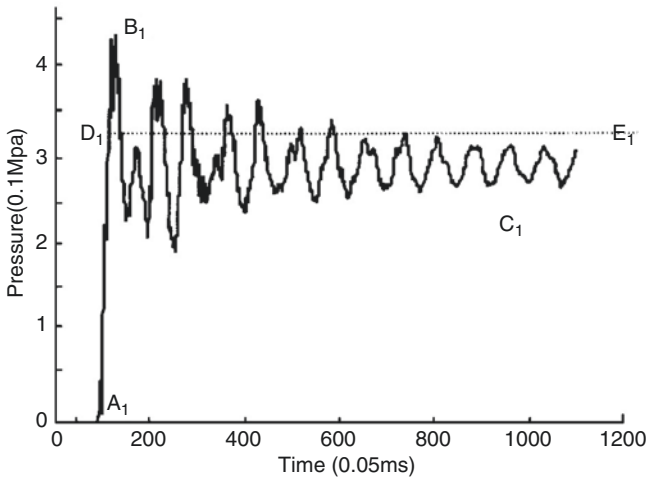

Fig. 6 Compression wave in the compression section of the simulated shock wave. (A_1) Starting point of compression wave; (B_1) Pressure peak value of compression wave; (C_1) Steady-state pressure value of compression wave; (D_1–E_1) Parallel line corresponding to the pressure value of 0.32 MPa

Table 1 Injury effects of compression wave on the lungs $(\overline{\chi} + S)$

Group	Type of animal	Number of animals	P_{B1-A1}/MPa	t_{B1-A1}/ms	Lung injury score (IS)
R_1	Rat	10	0.41 ± 0.03	2.43 ± 0.39	0.1 ± 0.3
R_2	Rat	10	0.40 ± 0.02	2.80 ± 0.32*	0△
R_3	Rabbit	6	0.39 ± 0.03	4.90 ± 0.30	0
t value				2.319	01.054

Note: *$P < 0.05$, △$P < 0.5$, P_{B1-A1}: pressure difference between the points B_1 and A_1 as shown in Fig. 6, t_{B1-A1}: time difference between B_1 and A_1 as shown in Fig. 6

increase time might be a bit too long, while decompression wave pressure decrease time might be a tad short, but in general experiment result should provide some value as a reference. The aforesaid experiment further cemented our belief that the overexpansion and decompression effects are the main culprits responsible for causing injury to the lungs. In other words, when a shock wave acts on the human body, the physical properties of moving fluids (i.e., the pressure, flow velocity, temperature, and density of water and air) abruptly change when flow velocity exceeds the speed of sound, thereby creating overexpansion of lung tissue, consequently causing injury. The specific process is as described below:

During the compression stage, there is abrupt change in pressure on the surface of the body; even though the lungs and thoracic wall are packed against each other, due to the dampening effects of the thoracic wall and lungs, this kind of pressure is mainly borne by the thoracic wall, meanwhile the compression-driven displacement in the movements of the thoracic wall and lungs occur over a gradual span. In the end, due to the effects of inertia, they move beyond a point of balance and arrive at a position of maximal compression-driven displacement.

After the compression stage, pressure on the body surface rapidly reduces, and the elastic force causes the thoracic wall and lungs to rebound together. During this rebounding phase, the thoracic wall might rebound back to position ahead of lung tissue because the former's dynamic response capacity is much stronger than the latter's. Previously, the two were packed tight against each other, and now the two are separated and vibrating on their own.

The thoracic wall vibrates at a faster frequency but smaller amplitude, and on the contrary, lung tissue vibrates at a slower frequency but with bigger amplitude.

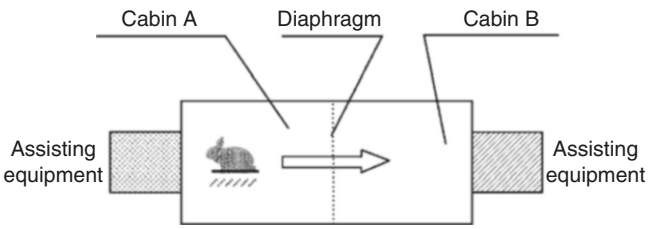

Fig. 7 Simulation cabin for decompression phase of shock wave

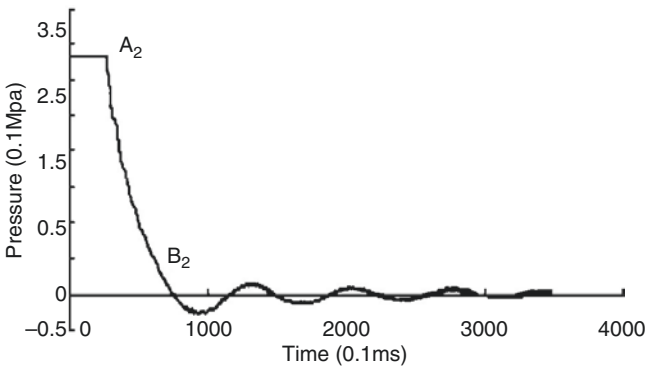

Fig. 8 Decompression wave in the decompression section of the simulated shock wave. (A_2–B_2) Decompression wave section

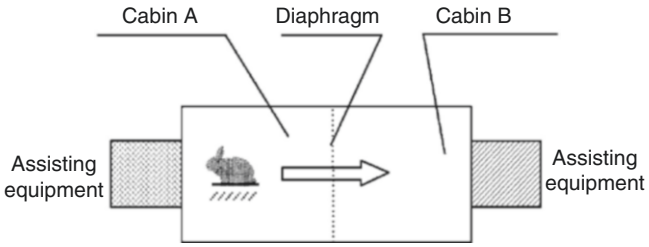

Fig. 9 Simulation cabin for negative pressure phase of shock wave

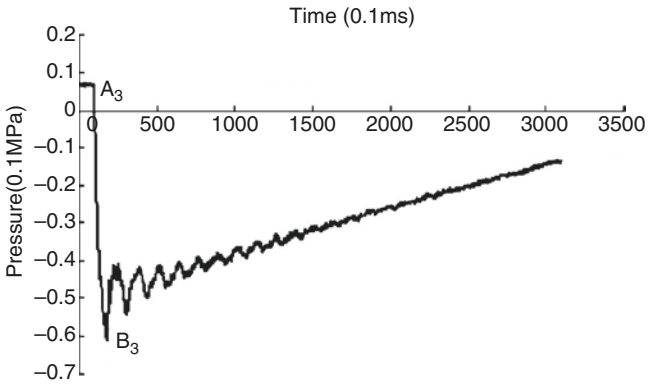

Fig. 10 The negative pressure wave in the negative pressure section of the simulated shock wave. (A_3–B_3) Negative pressure wave section

Table 2 Injury effects of rapid decompression on the lungs

Group	Number of common rabbits	Decompression value/MPa	Sustained duration/ms	Lung injury score (IS)
D1	6	0.32 ± 0.03	8 ± 0.1	4.0 ± 0.0
D2	6	0.32 ± 0.02	17 ± 1.27*	2.0 ± 0.6*
D3	6	0.32 ± 0.01	30 ± 2.1*	0.6 ± 0.7*
D4	6	0.32 ± 0.02	53 ± 2.6*	0*
D5	6	0.39 ± 0.01	12 ± 0.9	2.4 ± 0.9
D6	6	0.41 ± 0.01	64 ± 1.3	0△

Note: *P<0.001 compared with group D1, △P<0.001, comparison between group D6 and group D5

Table 3 Injury effects of underpressure on the lungs

Group	Number of common rabbits	$P_{B3–A3}$/kPa	*t* value	$t_{B3–A3}$/ms	*t* value	Injury score (IS)	*t* value
U1	6	−25.0 ± 3.1		16.0 ± 1.4		0	
U2	6	−61.1 ± 3.0*	20.498	6.7 ± 1.1*	12.795	0.9 ± 0.35*	6.299
U3	6	−55.5 ± 2.0*	20.251	30.0 ± 2.0	14.047	0	

Note: *P<0.001 compared with group U1, $P_{B3–A3}$: underpressure difference between the points B_3 and A_3 as shown in Fig. 10, $t_{B3–A3}$: time difference between B_3 and A_3 as shown in Fig. 10

Therefore, it is very probable that the thoracic wall and lungs collide against each other, and the force of collision is conditional on the speed at which the surface of the lungs strikes against the thoracic wall. The higher the collision speed, the bigger the impact, and when the impact surpasses the lung tissue's threshold, injuries such as hemorrhage and edema would appear on the surface of the lungs. This kind of effect may be termed overexpansion effect.

During vibration, pulmonary alveoli are compressed and expanded; during expansion, alveolar wall has to bear tensile strain and tension stress. When tensile strain reaches a certain level, the pulmonary microvascular endothelial cells' and alveolar mesenchymal epithelium's small solute permeability levels surpass their critical values, resulting in alveolar edema. When tension stress exceeds the maximum strength of alveolar wall, alveoli would rupture. When alveolar wall tears apart, so too would alveolar capillaries, leading to pulmonary parenchymal hemorrhage. In other words, this is the over-volume expansion effect during the expansion process.

The overexpansion and decompression effects are manifested in the injury mechanisms of excessive rapid expansion effect and over volume expansion effect. The overexpansion effect mostly occurs during the shock wave's decompression section. The higher the peak pressure, the shorter the decompression duration; the more obvious the overexpansion effect, and the more severe the lung injury. The compression stage might not directly harm the lungs, but it energizes the overexpansion effect.

The difference in shock wave injuries between humans and animals has long been studied, but the underlying mechanisms remain unclear. Radojicic et al. of the former Yugoslavia reported that inbred strain mice (BALB/c, C57BL/6, CBA, and AKR) showed clearly different fatality rates under identical injury factors (burn injury, mechanical injury, local blast injury, and radiation injury). Of which, the C57BL/6 strain exhibited rather strong tolerance against all the injury factors. Feng Gang et al. conducted further research. They systematically observed the heterogeneity (or difference) in how the BALB/c and C57BL/6 strains of mice responded to shock waves, then used DNA chip, suppression subtractive hybridization technique, and candidate gene approach to carry out comparative analysis on the gene expression profiling of the tissues of the brain, liver, and lungs, which are closely associated with response after subjecting to the effects of shock wave. The main conclusions are as below: (1) Confirmed that when the BALB/c and C57BL/6 mice are subjected to the same type of full-body shock wave, the C57BL/6 mice exhibited stronger tolerance against injury, while no significant difference was observed between male and female mice of the same strain; (2) DNA chip experiment discovered that liver tissue gene expression profiles of BALB/c and C57BL/6 differ vastly during the early stage after being subjected to full-body shock wave, primarily manifested as obvious heterogeneity in genes involved in stress response, inflammatory response, tissue injury and restoration, cell signal transduction, biological oxidation, and substance metabolism. Early on after the onset of injury, the C57BL/6 mice demonstrated rather good stress response, which might be attributed to its relatively strong tolerance for trauma; (3) DNA chip experiment also revealed that lung tissue gene expression profiles of BALB/c and C57BL/6 also differ greatly, primarily manifested as obvious heterogeneity in genes involved in tissue injury and oxidative injury, inflammatory response, apoptosis, and cell signal transduction, of which the high gene expression related to tissue injury in BALB/c mice and the high gene expression related to oxidative injury in C57BL/6 mice might be associated with their heterogeneity in lung injuries; (4) suppression subtractive hybridization technique was used to select 37 differentially expressed genes and gene fragments in brain tissues of test subjects early on after the onset of injury, including 31 genes with known functions and six expression sequence tags (EST) fragments with unknown functions. These genes with known functions are chiefly involved in functions early after injury including mutual brain tissue and cell interaction, nerve cell damage, protein synthesis, biological redox reaction, and Ras signaling pathway activation. This is manifested as active functionality of cells inside brain tissue early after injury, and an increase in mutual effects between the cells that clearly entered stress state; (5) alpha-enolase and cytochrome c oxidase subunit III (COX3) genes have drastically different expressions in the brain tissues of the two mice strains early after injury, which might be related to heterogeneity in response after shock wave injury; (6) full-length cDNA of a new gene GBI, which exhibited

differential expression in brain tissue early after injury, in the BALB/c and C57BL/6 mice were selected, cloned and registered with the GenBank. Preliminary study proves that the GBI gene has specific expression in brain tissue, where this gene might be involved in cell signal transduction as suggested by bioinformatics analysis; (7) it was discovered that IFNγ has vastly different expressions in the two strains of mice early after injury, and analysis indicates that the difference might be attributed to heterogeneity in lung injury pathological processes of the two respective strains. In light of the above, the heterogeneity of response to post-shock wave injury in mice with different genetic backgrounds is related, to a certain extent, to respective activation methods and levels of stress response, inflammatory response, tissue injury and oxidative injury, and cell signal transduction. It can be confirmed that "injury response is unrelated to genetic factors" viewpoint is no longer established.

2. Effects of throwing and collision due to dynamic pressure. Dynamic pressure can injure a person by throwing (leaving the ground) or displacement (without leaving the ground), and the abrupt deceleration upon falling and hitting the ground or colliding against another object is the predominant cause of mechanical trauma.

When dynamic pressure reaches 9.8 kPa, wind speed equals approximately 100 m/s, which is about twice as powerful as a force 12 storm. When dynamic pressure reaches 98 kPa, wind speed is higher than 300 m/s. When a sizable quantity of explosive or a nuclear weapon explodes, dynamic pressure near ground level might even eclipse this figure, which would throw exposed personnel a very far distance. It was witnessed during a nuclear experiment that some test animals (dogs) were tossed more than 500 m away by shock wave. When the human body or animal is "blown" by the blast wind, the air above scatters more than the air below, thereby creating an uplifting force. The body is therefore thrown or ejected by the joint upward and forward forces.

At areas where dynamic pressure is very high, different parts of the human body bear different pressure, and the impact force of dynamic pressure might result in tears on the body surface, or even separation of limbs from body.

In a relatively enclosed environment (such as an indoor space or a tank), complex shock wave formed from multiple reflections and overlap of the original shock wave would result in an even more complicated injury mechanism. Under such circumstances, there exists a good linear relationship between the movement speed of the thoracic wall and the severity of injury (including lungs, upper respiratory tract, gastrointestinal tract, and solid organs in the abdomen). Damage threshold is at thoracic wall movement speed of 4 m/s; LD_1 is at 8 m/s; and LD_{50} is at 12 m/s. Therefore, the movement speed of the thoracic wall can also function as projection index for non-auditory apparatus injury caused by complex shock waves. In addition, upon being struck by a medium-intensity complex shock wave (170 kPa, 1 ms), there was transient increase in the concentration of both the neuroprotein and neuro-specific enolase in cerebrospinal and glial cell marker s-100 in the rats, demonstrating a rise in leakage of protein from nerve cells and glial cells. Brain tissue has already been damaged, while obvious injuries have yet to be observed in other tissues.

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Mechanical Mechanisms and Simulation of Blast Wave Protection

Zhuang Zhuo and Zhanli Liu

1 Mechanical Mechanisms of Blast Wave and Protection Requirements

A typical blast wave as an effective duration of 2–10 ms, and the features of a shock wave in a free field are illustrated in Fig. 1. When explosives or other explosive substances explode, immense energy is generated. When materials are compressed by the impact, compression wave is formed, which quickly spread and propagate in all directions, thereby creating drastic damage to surrounding materials and abruptly elevating pressure to the point of overpressure. A non-linear shock wave includes a discontinuous overpressure front, and pressure, density, and temperature behind the front usually decline according to an index until reaching negative pressure. Thereafter the figures gradually return to baseline, followed by a vacuum phase (cavitation), which then swiftly spread and propagate in all directions, thereby creating drastic damage to surrounding medium. The peak overpressure of a blast wave is related to the quantity of explosives, surrounding environment conditions, and other factors. Structures and solids are subjected to loads that may include many cycles comprised of such periodic overpressures and cavitation. Once a vertical shock wave (particle velocity parallel to wave velocity) hits a solid structure, without a doubt it would create some kind of shear (non-zero part of particle velocity perpendicular to wave velocity). In various isotropic materials, this kind of composite force is easy to dissipate. However, in various anisotropic materials, stress wave would create many qualitatively different transformations, which increase the difficulty of predicting responses in such solids. At the same time, in this kind of material, new mechanisms (dissipation, resonance, etc.) may be introduced to effectively defend against stress waves.

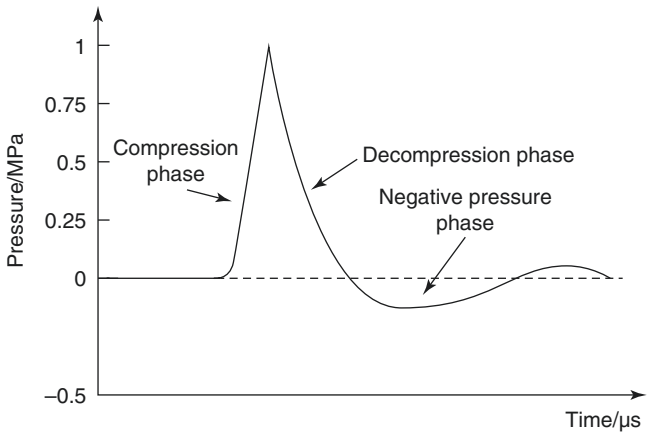

Fig. 1 Typical waveform of explosion shock wave

Modern day local analysis of battle wounds show that injury consisting of a combination of explosion fragments and shock wave overpressure is one of the biggest threats for soldiers on the battlefield. Its defining feature is that even if a fragment or projectile does not penetrate defensive equipment, it could still cause damage serious enough to injure or even kill an individual soldier. This also manifests a feature of modern weapons in that the capacity to wound or kill has switched from direct contact to indirect contact. For instance, fuel-air explosive and thermobaric weapon in modern warfare are characterized by long duration of shock wave

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impulse that can enter tunnels, enclosed spaces, and other areas that fragments cannot penetrate, and their long negative pressure duration gives them extra destructive power. At present, the Personnel Armor System for Ground Troops (PAGST) can hardly protect them from injuries to the brain and chest, areas easily injured by shock wave overpressure, and neither are passengers in armored vehicles spared from injury or death due to blast waves. Currently available methods to assess protective effect cannot provide the technical support necessary for the research and development of effective protection.

For a long time, research and development of the Personnel Armor System for Ground Troops have been predominantly focused on using the lightest material that can maximize defense against penetration by bullets or fragments. Ultra-high molecular weight polyethylene fiber, high strength para-aramid, light-weight bullet-resistant ceramic, and other new types of ballistic materials not only reduce the weight of PAGST but also help protect soldiers against bullets or fragments that fly with higher energy, as the anti-penetration capacity of personnel armor continues to enhance. Yet, overpressure of shock waves generated from an explosion can propagate through PAGST and act on the wearer because its propagation and dissipation mechanisms differ drastically from the relatively local energy dissipation mechanism of bullet penetration. Blast wave energy can reach places fragments cannot and might even strengthen due to reflection from the surface of other protection gears. As early as the 1980s, US military research already concluded that regular Personnel Armor System for Ground Troops could not effectively avoid or mitigate blast wave's injuring effects on the human body and might actually worsen the situation. However, since the blast wave did not contribute to many military casualties, the US military didn't vigorously pursue research in this area.

Since the turn of the century, military conflicts have erupted in different parts of the globe, and military injury and casualty analysis indicates that personnel wounded or killed by explosion surged to 70% of the total, of which shock waves account for approximately 60% of injuries and casualties from bomb explosions. A large number of explosions that combine explosive fragments with blast waves have led to a lot of damage on the battlefield, with an increasing number of both soldiers and civilians afflicted by traumatic brain injury (TBI) and lung injury. Traumatic brain injury differs from penetrating craniocerebral injury and closed craniocerebral injury in that even when wearing a protective helmet, a brain injury could still occur since the helmet cannot effectively protect against or allay the effect of a shock wave. This is war-related post-traumatic stress disorder, and symptoms could range from short-term loss of consciousness to serious coma. Severe traumatic brain injury from blast injury is characterized by widespread edema and blood congestion in the brain that develop quickly, oftentimes within an hour of injury. Subarachnoid hemorrhage signifies severe injury and portends more serious edema and blood congestion in the brain, as well as delayed cerebral vasospasm. This kind of vasospasm usually triggers delayed neurologic impairment and is more common among blast wave-induced TBI patients than other types of craniocerebral injuries. A recent evaluation from the American think tank Research and Development Corporation (RAND) points out that 320,000 or 20% active American soldiers suffer from blast wave-induced TBI, and in the past 5 years, some 40,000 US troops have been diagnosed with post-traumatic stress disorder.

Traumatic brain injury is difficult to diagnose (or treat) on the battlefield, and the situation worsens in 40% of victims a few days or a few weeks after onset of the problem (secondary injury) due to a series of mechanical and biochemical processes. The latest research from the University of California San Diego about military and civilian patients with mild TBI indicates that there exists a certain connection between measurements of anisotropic damage taken using diffusion tensor imaging (DTI) and abnormally slow brain waves measured through magnetoencephalography (MEG). In DTI images, anisotropic damage is a clear structural change in the brain's white matter. It is believed that this change is caused by dissociation resulting from the mechanical shearing of bundles of white matter fibers from the brain's gray matter. At present, the ARO/JIEDDO project in the USA is developing very detailed models of the brain to identify cell injury mechanisms. The goal is to use the research results and the outcomes of other projects to ascertain the effective injuring range of blast waves and explore technical pathways to reduce such injuries.

As blast injuries occur at rising frequency, hot topics in the research on Personnel Armor System for Ground Troops have shifted to minimizing traumatic injuries from overpressure of shock waves. Numerous European and North American countries have set up a raft of research plans pertaining to mechanisms of head injury and lung injury caused by shock wave overpressure, anti-blast wave equipment, and other related matters, having achieved some research outcomes. Our research in protective gear also needs to adapt to changes on the battlefield and the weapons deployed by other militaries, and the challenge herein is the study on the control mechanisms and injury mechanisms behind the energy (and temperature) of different stages of the shock wave.

2 Shock Wave Energy Dissipation Mechanisms in Multiscale Microstructure of Composite Materials

The wavelength of a blast wave is measured in a micrometer or millimeter, and traditional constitutive models and homogenization theories of composite materials based on longwave approximation cannot explain the dynamic response of micro/nanoscale materials and structures under the action of a shock wave. Therefore, it is necessary to develop constitutive models for multiscale composites at high strain rates, realize effective thermodynamic parameters that can be calculated at the component level, and establish the protective mechanism of polymer composites against shock wave energy. There are two key issues to be addressed. The first is to establish a dynamic constitutive model of multiscale polymer materials under large deformation and high strain rate, with particular attention to focus on the relationships between viscous, hyper-elastic, modulus, and other macroscopic factors and the dynamic response of microstructure. The second is to conduct experiments to measure material parameters in the stress wave shape calibration constitutive relationships, such as Hopkinson's notched bar impact testing, light-gas gun or explosion-driven plate impact experiment, and shock tube experiment. Stress waveforms that expand on a nanosecond scale may be used to determine the constitutive relation of viscoelastic materials, and this method is also applicable for blast waves with wavelength ranges as listed in Table 1.

A typical characteristic of shock wave propagation in multi-structural phase polymer composites is dispersion, as in variations in group velocity and phase velocity with the input frequency. Dispersion is the main inducement of deformation localization, scattering, and resonance. Consequently, a drastic reduction in stress waves, especially when local resonance occurs, stress wave could be completely shielded in a certain frequency band. When the deformation size and stress wavelength are equivalent to the microstructural size of the composite material, the local waves instigated by these microstructures reflect and diffract on a microscale in a way that extensively affects the dispersion and dissipation of the larger waves.

The patterns of mutual action between shock wave and multiscale microstructures are complex, and Table 1 lists the relationships between blast wave wavelength and structural scale. The wavelength of a blast wave is measured in micrometer or millimeter, and microstructural phase or material scale that dissipates or absorbs shock wave is usually on a scale one level lower than the wavelength, as in a scale ranging from several hundred nanometers to several hundred micrometers. By designing microstructure at a subwavelength scale, it would be possible to capture and dissipate a shock wave's energy at the front of the wave. For example, a blast wave, its wavelength is within the range of 10 μm–1 mm and its frequency in the range of 2–200 MHz. The design's corresponding soft coating, hard, hollow, thin, and long-fillers, and piezoelectric and magnetostrictive fillers ought to be within the 1–100 μm scale to mitigate, redirect, and absorb blast wave. Materials comprised of several filament segments can capture one wavelength's movement of the stress wave. If the material scale is smaller than 1 μm, atomic and molecular mechanical energy at the nanoscale could be captured and dissipated at the front of the shock wave, thereby mitigating, redirecting, and absorbing the shock wave. The conclusion is that for stress waves with relatively wider wavelengths, it would be impossible to use materials at one scale to achieve different protective functions, and effective protective material has to consist of multiscale components. "Multiscale" here refers to a combination of millimeter-scale polymer, micrometer-scale fiber and granular particle filler, and nanoscale molecular structure and matrix/composite interface. This kind of multi-structural polymer material can defeat stress waves under high pressure and broad-spectrum shock wave frequency and amplitude.

Recent research by the Office of Naval Research (ONR) of the U.S. Navy indicates that there are effective applications of polyurea and polyurethane in allaying body armor failure against explosions. Polyurea is a kind of segmented copolymer with a glass transition temperature (T_g) at −60 to −50 °C and can be easily manufactured and sprayed onto the surface of metal or other materials. Its mechanical property is affected by temperature, pressure, and strain rate. Research shows that said kind of polymer material has high-stress resistance, low tensile strength, and high energy-dissipation capacity. Its basic properties include bat-like yield trajectory and tensile gap yield (Fig. 2). If other materials are included, the main material's tensile gap may be changed. For instance, inserting rubber particle filler can stabilize existing gaps so

Table 1 Relationship between stress wave wavelength and structural scale

Wavelength λ	Frequency	Structural scale	Application	Design method
Less than 10 μm	More than 200 MHz	Less than 1 μm (nanoscale)	Shock wave: At the front, capture and dissipate nanoscale mechanical energy	Control molecule scale, mobility, and electrical property, soft/rigid segment; insert functional nanofiller
10 μm~1 mm	2~200 MHz Blast wave on battlefield	1~100 μm	Blast wave: Mitigate, redirect, and absorb; independently or externally adjustable material	Soft coating, hard, hollow, and thin and long-fillers; piezoelectric and magnetostrictive fillers

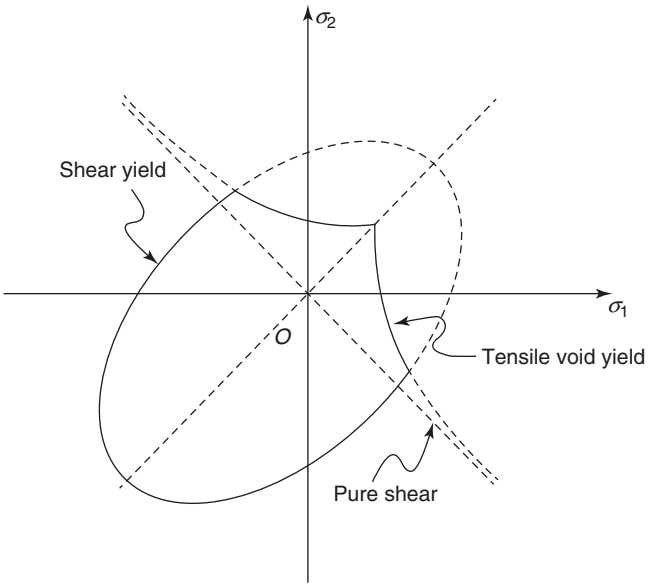

Fig. 2 Yield trajectory of amorphous glassy polymer

that they won't turn into real cracks, thereby enhancing the toughness and impact resistance of the armor material substantially. At the same time, said kind of polymer can be applied in many other areas after chemical modification and nanostructure modification. Through design optimization, their mechanical and physical properties could be augmented immensely and are particularly suited for redirecting, capturing, and dissipating effective stress waves in various frequency bands created by the explosion.

3 Resistance Property Matching and Chemical Design of Material Interface Related to Blast Wave Propagation

The reflection and transmission of incident impulse energy of shock waves on the interface of material are very complex. The shock wave first invades the polymer substance in the form of a stress wave, then turns into and propagates as both stress wave and shear wave upon encountering interface between layers or other fillers. Therefore, the mechanical resistance property of the matrix and fillers are determining parameters crucial to distributing and dissipating the incident impulse energy. Researches on the underlying mechanisms further shed light on how the chemical structure, length, and composition of segmented copolymer affect polymer composite material structure and how they affect the material's main properties such as storage and loss modulus and macroscopic anisotropy. Such revelations are vital to understanding the principles in matching the appropriate resistance properties beneficial to stopping the propagation of shock wave propagation in different anisotropy materials with various moduli and densities.

Research about mechanisms behind how chemical properties and microscopic heterogeneous structures of materials respond to shock waves. The wavelength of a blast wave is measured in a micrometer or millimeter, and interactions between different polymer chains, chemical bonds, and molecules have varying responses to shock waves. In combination with polymer composite material's multiscale theory and calculation model, and through designing the molecular structure and aggregated state microscopic cluster structure, efforts are made to research and produce composite materials with anti-shock wave capacity, multiple soft and hard chain structures at the nanometer, micrometer and millimeter scales, and various energy dissipation mechanism, so that it possesses functions including adjusting and controlling the path of the shock wave, and properties of matrix and filler interfaces. This kind of material has a protection mechanism that can diffract, reflect and absorb shock waves.

The design and chemical modification of multi-structure polymer material need to emphasize researches based on multiple scales ranging from nanometer to micrometer and based on a combination of both soft and hard segments. The macromolecular structure of polyurea is shown in Fig. 3. By selecting different molecular structures and microphase separation structures and the use of different polymer conditions, the material's mechanical strength can be adjusted. The focus should be placed on modulus and viscoelasticity parameters, and the relationship between molecular structure and aggregated structure versus shock resistance of materials should be summarized through shock wave-resistance tests. The flexibility of molecular chains, microphase separation scale, and modulus are some examples. These findings should provide guidance for molecular structure optimization and aggregated structure adjustment and control so as to help identify the best composite materials in terms of energy dissipation.

By changing the relative content and chain length of different components, the size of elastomer microphase separation structures at the nanoscale may be adjusted. In terms of matching microscopic structural moduli, structures such as hard-over-soft, soft-over-hard, or hard-over-soft-over-hard may be obtained. Through the adoption of the dielectric relaxation spectrum method, the viscoelasticity of materials at high frequencies may be characterized. Or employ the room temperature equivalence principle to measure viscoelasticity at low temperature and low frequency so as to obtain viscoelasticity at room temperature and high frequency by extrapolation. By mixing two kinds of polymers, the sizes of their respective structures are adjusted at the micrometer scale. The elastomer foaming method can also be used to produce foam materials that contain pores, or inject microcapsules filled with low-modulus high-viscosity liquid into the elastomer. Studies are conducted on the effi-

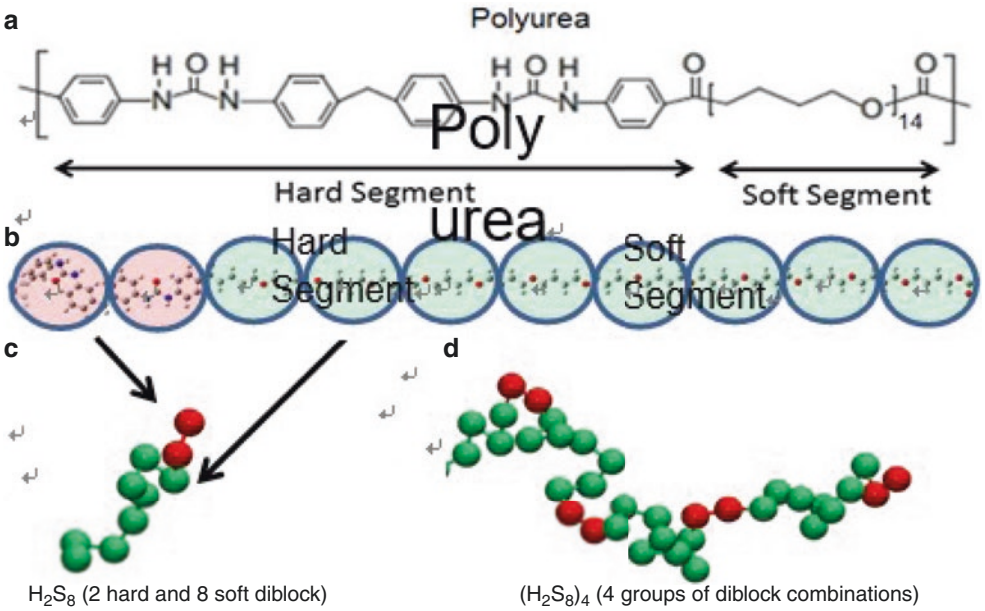

Fig. 3 Schematic diagram of crosslinking of soft and hard segments of polyurea macromolecular structure. (**a**) Molecular formula of polyurea. (**b**) Schematic diagram of coarse-grained model of polyurea. (**c**) Diblock coarse-grained molecular chain, H_2S_8. (**d**) Multi-block coarse-grained molecular chain, $(H_2S_8)_4$

ciency of energy dissipation of methods such as matrix deformation and interfacial debonding, microcapsule breakage and rupture, matrix yield, intermolecular friction, destruction and reconstruction of nanoparticle clusters, shear thickening, and piezoelectric processing.

4 Injury Mechanisms and Tolerance Thresholds of Blast Wave

There are already a copious amount of studies and broad consensus about the injury mechanisms behind blast waves. When the overpressure of a shock wave acts on the human body, the first-level incident wave is firstly reflected off of the surface of the body, thus creating diffraction waves at the sides and back of the body. The most damaging are stress waves that enter and propagate throughout the body in the forms of compression waves, tensile waves, and shear waves. When a stress wave penetrates the interfaces of materials with different densities, the three aforementioned types of waves could all appear, thus injuring body tissues and organs in various forms such as compression, tension, and lesion. The most vulnerable parts of the body against shock waves are tissues and organs with cavity and relatively high air content. Since the head, neck, and lungs are the most sensitive to the effects of shock wave overpressure, at present, a broadly accepted shock wave overpressure threshold value is the human lungs and head shock wave overpressure threshold value proposed by Bass (Fig. 4) based on a massive amount of biological experiences. The lung area with chest armor as outlined in the blue rectangle can obviously withstand higher peak overpressure compared to the lung area with no chest armor as outlined in black rhombus, and in Fig. 4, the difference equates to at least two levels. Said threshold value is also an assessment standard for the protective performance of gears for the torso and head.

The aforesaid studies on shock wave injury mechanism and studies on tolerance threshold of the human body indicate that numerous research feats already address the shock wave overpressure injury issue. However, two aspects still require in-depth research: First is whether or not some safety threshold values obtained through processes of low-speed impacts on the human body still hold true with relatively high-speed impacts from projectiles. Second is whether or not the overpressure's course of effect would change when the subject is wearing protective gear, and whether or not the protective gear might actually worsen the injury.

Protective material and protective structure are the main focal points in studies related to anti-projectile and anti-explosion blast wave technology for personnel armor. Of them, how the protective structure controls the propagation of stress waves generated by direct projectile penetration and blast wave overpressure so that the energy they carry can be absorbed and dissipated within armor layers with limited thickness and mass is the key to minimizing the transferal of the impact force onto the human body. Research shows that whether the injury is a BABT (behind armor blunt trauma) or shock wave overpressure, energy mostly transfers to the human body through two mechanisms: (1) Primary fast wave, referring to bullet or overpressure that acts on the protective equipment in the form of elastic stress wave and deformation wave, then reaches the surface of the human body at sonic velocity or supersonic velocity; (2) deformation wave, referring to changes in elasticity and plasticity of protective layers in the protective equipment during the course of the bullet's penetration, causing the side away from the bullet's entry to protrude and come into contact with the body surface. Specifically, shock wave overpressure trans-

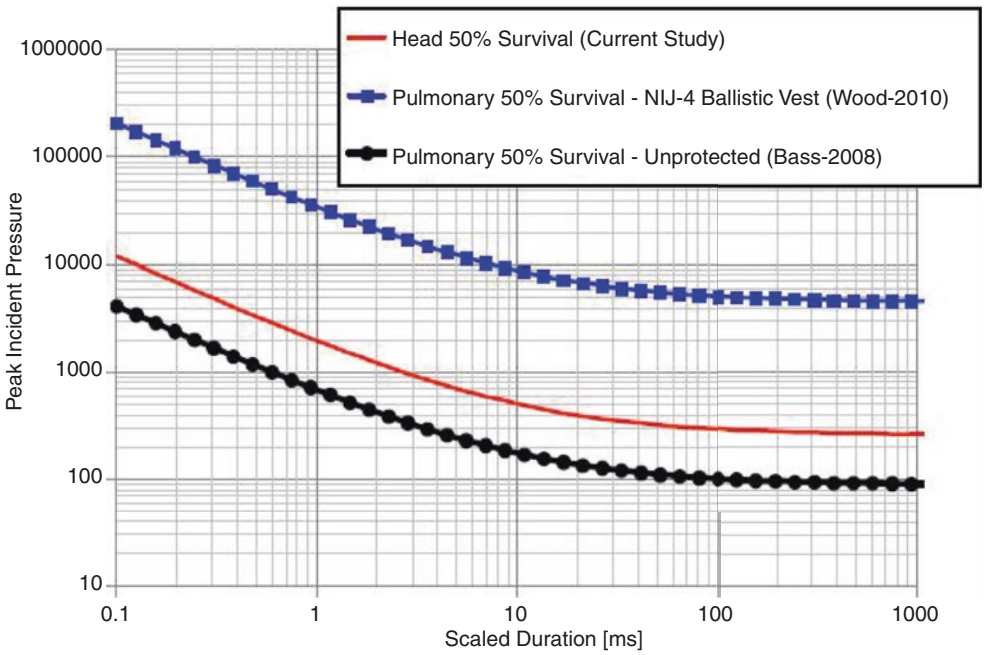

Fig. 4 Overpressure tolerance curve of shock wave in human head and lung

fers to the human body predominantly as a primary fast wave. The two mechanisms have different courses and duration of effect. As such, Ken-An Lou proposed a four-layer protection structure to defend against non-penetration wounds, of which the two middle layers namely barrier and compression mitigation layers are intended to be the main energy absorbers and dissipator. Upon this basic structure, various combinations of matching materials with different functions have been designed. Digital simulation analysis is used to simulate performance against overpressure with 345 kPa in peak pressure and 5 ms in duration, and it is concluded that such a structure is effective. Viano D.C et al. established a mechanics analysis model based on the features of bulletproof vest, opining that during the course of action of shock wave overpressure, stress wave with relatively high peak pressure and short impulse would first act on the relatively dense bulletproof layer, creating compression wave that would transfer to the foam layer, then transfers through the foam layer to interfaces of different densities and disperses. The compression wave's peak pressure would be reduced while duration would be prolonged, thereby lowering damage caused to the chest area.

5 Mechanical Model and Simulation of Blast Wave Protection

Targeting the scientific issues stated above, it is necessary to establish a multiscale calculation model that looks at the nanometer, micrometer, and millimeter scales so as to reveal shock wave energy dissipation mechanisms behind the local deformation and dispersion in macro, small, and microstructures; develop constitutive theory and homogenization methods for multiscale heterogeneous materials; and assess the extent of shock wave decline in order to provide guidance in material selection and chemical modification. The objective is to provide guidance beneficial to the design and production of specific microscopic structures that can be applied to weaken and guide shock wave energy and to enable composite materials to possess frequency-selective properties.

Establish theories and calculation models for polymer composite materials with microstructures at specific sizes. These efforts should mainly include: (1) Under necessary space and time scales, use coarse-granulation and homogenization methods in parallel to simulate dynamic mechanical properties of elastomers; under wave propagation and dynamic loading conditions, develop effective digital simulation and calculation tools (develop spectral elements and fluid-solid coupling methods).

Targeting the multiscale characteristic of polymer composite materials, and based on multiscale simulation at multiple levels, establish a calculation framework whereby four spatial scales are interrelated:

1. **Nanometer scale**. Molecular dynamics (MD) calculation and simulation (characteristic scale of $<10^{-9}$ m). For polymer, the molecular dynamics model is already a coarse-granulation model. In other words, radical groups are usually represented as coarse-grained particles with interacting forces. By establishing potential energy model of macromolecular chain with hard and soft chains, use

molecular dynamics simulation to analyze the molecule-level structure–function relationship of polymer materials, then obtain memory modulus and loss modulus of polymer clusters, with storage modulus reflecting viscosity of the polymer, while the loss modulus can demonstrate the frequency dissipation of polymer.

2. **Sub-micrometer scale**. Quasi continuum model with characteristic scale between 10^{-8} and 10^{-6} m. With polymer clusters obtained by nanoscale analysis, and based on memory modulus and loss modulus, establish a coarse-granulation (CG) model in order to establish the physical and constitutive parameters of polymer cells.
3. **Micrometer scale**. Continuum model with characteristic scale between 10^{-5} and 10^{-3} m. Considering energy density as a variable in input and output, establish momentum equation and the weak form of the virtual power term, and through the use of discretization by finite element, generate finite element models of polymer composites containing microstructures. In particular, a representative voxel model can help obtain material's dynamic response behavior, which would be helpful to guide the material selection and chemical modification.
4. **Millimeter and above scale**. A continuum model for a bigger characteristic scale ($>10^{-3}$ m). With a new method of solid coupling, simulation dynamic responses of materials and structures under shock wave action would provide results with engineering and application value.

The objective is to develop theories and calculation models for polymer composite materials containing microstructures at specific scales in order to predict a material's resistance against shock waves. Each spatial scale is also related to the temporal scale, and thus there are related frequencies and wavelengths. Dynamics calculation at the nanometer scale is a key step. Analysis results of structure–function relationship of polymer material at the molecular level would provide guidance for optimization of the design of the new generation of anti-blast wave polymer materials.

By using modeling and simulation of cutting-edge polymers at different scales, calculate the various structural, thermal, and dynamic properties of composite materials. Targeting the studies of these models, the following key issues need to be addressed: (1) How the chemical structure, length, and composition of segmented copolymer affect the structural form of polymer composite materials; (2) how does this form affect the material's main properties such as glass transition temperature, storage, and loss modulus, among other features. Simulation and calculation research will work closely with tasks related to polymer synthesis and expression.

At the same time, numerical simulation was also used for analytical purposes. Results indicate that for both strapped or strapless helmets worn by troops, peak overpressure and impulse energy at the side of the helmet and occipital bone area inside the helmet are both significantly higher than when no helmet was worn. Specifically, the increase is highest at the top of the head, with peak overpressure surging by three folds (Fig. 5a). The ACH helmet has a padding structure, but numerical simulation research shows that upon the action of shock wave overpressure, the gaps in between the different padding components of the helmet have higher peak overpressure than other parts of the helmet (Fig. 5b). The reason is multiple shock wave reflections and overlaps that occur at the gap between the helmet's interior surface and the wearer's head, thereby elevating peak overpressure and worsening injury to the head. Moreover, when the eyes and ears are unprotected, they would also be afflicted by more serious blast injuries (Fig. 5c).

The analyses stated above illustrate that protection against blast waves requires targeted and systematic design and research to improve personnel armor systems currently fielded.

6 Temporal and Spatial Propagation Mechanisms of Blast Wave

Research about the anti-blast wave mechanism of polymer composite materials entails the design of material at the macro, mesoscale, and microscale, and then material samples need to be produced. The purpose of such materials is to absorb, redirect, and dissipate stress wave energy, convert impact pressure wave into a shear wave, and alter the direction of propagation away from sensitive areas. To deal with the complications in amplitudes and strengths in different stages of a high-pressure blast wave such as pressure increase, sharp decrease, and negative pressure, as well as the effective duration and biological injury mechanisms, the anti-blast wave mechanisms of multi-structure polymer composite material are put forward, for example, the proposal of subwavelength microstructure dissipated shock wave energy model, obtainment of patterns of how material anisotropy guides the propagation of shock waves, and formulation of methods for assessing the effectiveness of biological injury protection.

To deal with stress waves of different frequencies, it is necessary to study how the multiscale microstructure distribution, microstructure phases, and mechanical properties of polymer substrates affect the overall modulus, then design

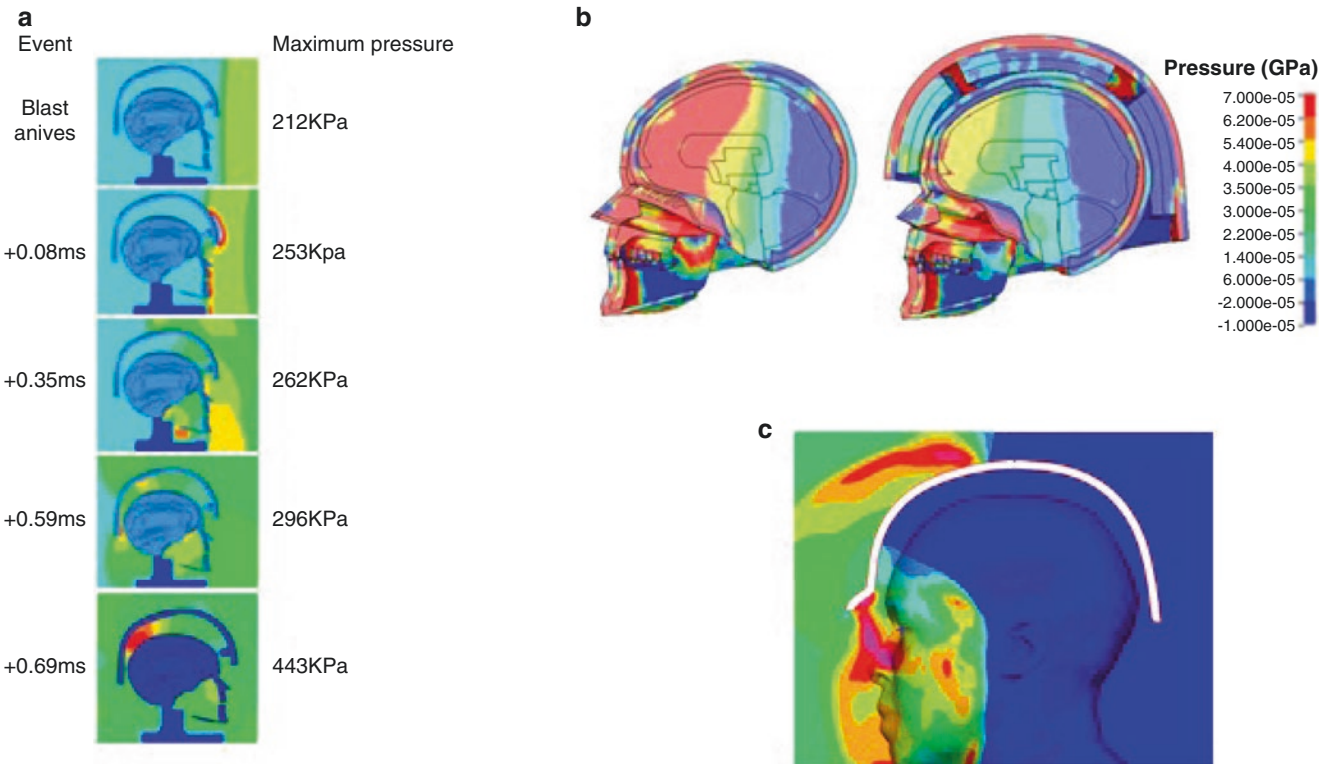

Fig. 5 Simulation of the interaction between helmet and head under the action of the explosion shock wave. (**a**) Spread of shock wave overpressure inside the helmet when the helmet and head are not lined; (**b**) Distribution of shock wave overpressure on the head when the helmet is worn and not; (**c**) Influence of the shape of the helmet on the shock wave overpressure on the head

composite materials with frequency-selective properties by way of using storage and dissipation moduli as a function of frequency so as to protect the most vulnerable parts from the shock wave. As shown in Fig. 6, when a blast wave enters the equipment from the left and passes through polymer composite materials and structural system, stress wave is redirected, absorbed, deflected, and reflected, thereby consuming energy and filtering out high-frequency waves. The result is a weakened outgoing wave on the right side, successfully shielding the protected subject (middle of diagram) from the effects of pressure, shear wave, and stress wave instigated by the shock wave.

Systematic research is carried out targeting geometric dispersion, viscoelasticity dispersion, and the accompanying localized deformation and energy dissipation mechanisms instigated by factors such as the shape of microstructure and form of the polymer substrate. When the deformation scale matches shock wave wavelength and scale of composite material's microscopic structure, the reflection and diffraction of localized wave instigated by microscopic structure phase have profound effects on the dispersion and dissipation of the larger wave. A typical characteristic of stress wave propagation in multi-level polymer composite material is dispersion.

Molecular properties and composite structure's mechanism for controlling wave absorbing capacity of the material are studied, producing polymer composite materials with highly efficient energy absorption and dissipation capacity by designing the molecular structure of the polymer and adjusting multiscale microstructure. Methods are explored to control and adjust viscoelasticity of molecular structure and multiscale aggregated structure of materials, and produce polymer materials with controllable and adjustable viscoelasticity and microphase scale. It is focused on observing the viscoelasticity of materials, the different shock wave energy dissipation methods in materials, and structural design for combining these materials. The relationship between chemical composition, aggregate structure, and macroscopic combination method versus material's shock wave resistance is summarized, which is used to guide and optimize material design, and select materials with high efficiency in dissipating and redirecting shock wave.

By using multicomponent reaction and composite construction of multiscale polymers, and by utilizing specific interactions between different molecules such as hydrogen bonds, ionic bonds, and coordination bonds, realize protection against shock wave and reveal how viscoelasticity affects a material's performance in dissipating shock wave energy. Traditional anti-shock wave materials rely on interactions

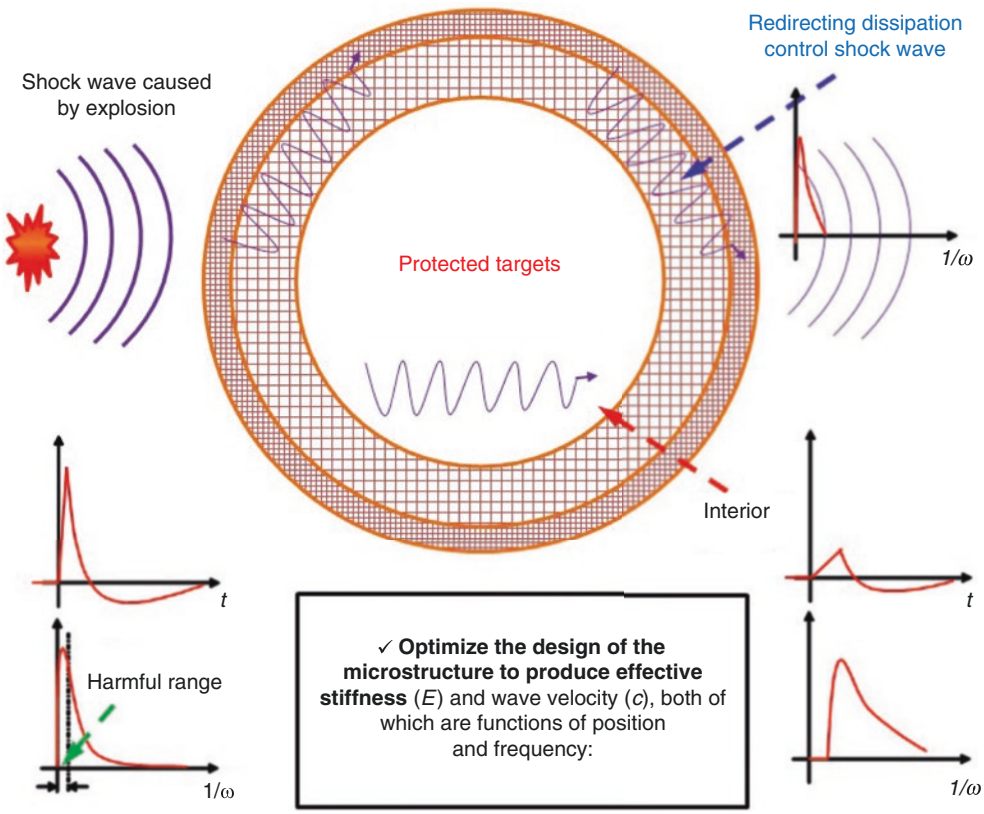

Fig. 6 Schematic diagram of protection against shock wave

between hydrogen bonds, and their functionality are rather limited. Such polymer structures are difficult to further modify or enhance, and require a rather complicated process when combining them with nanoparticles into composite materials. Through the nanometer-scale and micrometer-scale structural design of multicomponent and multiphase polymer materials, and adjusting and controlling the viscoelasticity of different components, make comprehensive use of crystal fragmentation, phase transformation, shear thickening piezoelectric dissipation and a multitude of other energy dissipation methods, produce polymer material samples with outstanding shock wave energy dissipation capability.

During research, theories, calculations, empirical experiments and studies in shock wave dynamics, material study, chemistry, and military medicine should be taken into account, to grasp the mechanisms that dictate how the multilevel microstructural form, interface property, spatial distribution, chemical property, and other factors of polymer composite materials mitigate blast wave energy, and establish constitutive models of materials with various microstructural scales. These efforts should help realize the prediction of response patterns of polymer composite materials under blast wave effect on macroscopic structure scale, assessment of the extent of decline of shock wave, provision of guidance on material selection and chemical modification, design and production of new types of samples of materials for equipment, and establishment of battle-related biological injury severity evaluation standards. Achieve breakthrough in polymer material design from nanometer scale to macro scale, and control propagation properties of stress waves with different frequencies and wavelengths so that materials possess functions such as wave absorption and wave redirection. Research results achieved will be put to use in the research and production of a new generation of personnel protection equipment, which can defend against blast waves of different strength, broad-spectrum frequencies and amplitudes so as to reduce war injuries and casualties as well as post-war disorders, and enhance the survivability of troops on the battlefield.

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Biological Shock Tube

Haibin Chen

Ever since the World War II, in particular after the emergence of nuclear weapon, research on effects of explosion has greatly risen in significance. Experiments of explosion effects may be carried out either in the field or in the laboratory. Field tests offer higher authenticity, but environment conditions are dire and costs are high, making them more suitable for general or final evaluations. Experiment conditions in laboratories are much easier to control and manipulate, and measurement accuracy is high, thus laboratory experiments are more fitting for basic research endeavors. The explosion simulator is the key device when conducting explosion effect experimentation in labs. Of the different types of explosion simulators, the shock tube has numerous clear advantages. The development of shock tube as an explosion simulator has become a priority in many countries.

A shock tube with a diameter of 2 m and a length of 60 m was already built back in the 1940s. To use this kind of early instrument, an explosive is hung at one end of the tube, while the test subject would be placed at the other end for experimentation and observation. Simulation considerations were rather simple and crude. In the early 1960s, the Lovelace Medical Education and Research Base in the USA constructed shock tubes with different combinations comprised of segments ranging between 42 and 72 in. in diameter (1 in. = 0.0254 m) and 12, 24, and 40 in. in variable cross-sections. Reflection shock waves at the tube ends were used to carry out biological shock wave injury experiments. However, the stress waveforms created were irregular, and using the reflection to conduct experiments could not simulate airflow effect. At the same time, the Mach Research Institute in West Germany researched and developed a kind of isobaric shock tube. The design of this device was ingenious. When one end was opened, it could create blast wave stress waveform with very long effective duration. However, just like the aforesaid instrument, this device could only simulate static pressure effect. In a period of about a decade starting from the end of the 1950s, the U.S. Naval Ordnance Laboratory built a massive conical shock tube. The working principle of said device was very direct and obvious, namely cutting out a small, three-dimensional cone in a symmetrical and spherical explosion space for experimentation. This setup can reduce quantity of explosives required and correspondingly lower impact from surrounding environment. Its airflow conditions were approximate to on-site experiments. The main shortcoming was the enormous size, and even then positive pressure effective duration is still rather limited. In the 1970s, focus was placed to address the disadvantages of the conical shock tube, namely size and cost reductions. Amann from the Mach Research Institute in West Germany and Gratias from the d'Etudes Center in France attempted to use driving segments of many different lengths to mimic the driving segment of the conical shock tube, while the driven segments were modified into cross-section tubes. This kind of new shock tube structure relied on air as driver, resulting in shortened structural length that could deliver rather long positive pressure effective duration. To prevent horizontal airflow from interrupting planar shock waves, all driver segment diaphragms were required to burst at the same time. Amann insisted that the diaphragm rupture time difference should be less than 5 μs, posing immense challenges for both diaphragm rupturing technology and actual operation. Even more troublesome was the different lengths of the driver segments, which made it difficult to create both the ideal stress waveform and the number of crests and troughs that corresponded to the number of driver segments. During this period, the former No.3 Research Institute of the Engineering Corps of the General Staff Headquarters of the People's Liberation Army of China also constructed a domestic shock tube dedicated to conducting explosion effect experiments.

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A noteworthy point is that blast injury is rather common in industrial explosion accidents, terrorism worldwide, conventional warfare, and nuclear strike. In order to discuss the occurrence mechanism behind and protection measures related to blast injury and formulate different kinds of injury standards for both military and civilian usage, countries like the United States, Sweden, and China utilized shock tubes to carry out large-scale simulated explosion experiments and promoted the development of shock wave dynamics as an academic discipline, as well as its collaborations with other subject matters. In general, research focal points are mainly concentrated in the following areas: (1) Propagation, reflection and refraction of shock waves, and their mutual effects on each other; (2) supersonic and hypersonic flow; (3) physics and applications of detonation; (4) flow simulation and empirical experiment test technology; (5) digital simulation research; (6) empirical experiment equipment and measuring technology; and (7) multi-disciplinary collaborations and applications of the shock wave. By convention, people refer to shock tubes suitable for laboratory animal testing as biological shock tubes. As an "injury-causing origin" device, biological shock tube has played a crucial role in the research studies mentioned above.

1 Basic Knowledge About Biological Shock Tube

1.1 Shock Wave and Rarefaction Wave

To understand biological shock tubes, first and foremost, it is necessary to explain shock waves. Let us use daily life examples to illustrate this matter. It is common knowledge that when there is a slight pressure change at some location in the air, this sort of change would propagate at the speed of sound. Then let us assume that at some location in the still air or inside a small space, this change occurred in an abrupt and powerful manner, like an explosion. The propagation of stress waves from this change would travel at a speed greater than the speed of sound, the rate of which depends on the strength of the explosion. One characteristic of this kind of wave is that when the front of the wave reaches some sort of spherical surface, the physical properties of air on the said sphere will change suddenly. Meanwhile, the air in the areas ahead of the wave front would not be affected. Pressure and density upstream of the wave are higher than the pressure and density of still air, and particles upstream of the wave would also begin flowing. We refer to this kind of wave as a shock wave, or more precisely, a forward-moving shock wave.

There is another kind of shock wave that is difficult for us to notice in daily life. For instance, when a conical bullet flies through the air at supersonic speed, there is actually a shock wave in front of it. To prove this point, we can put a bullet in a supersonic wind tunnel, and a special photography technique can capture the type of shock wave described above, as depicted in Fig. 1.

Let us assume that the shock wave has no thickness, even though strictly speaking, molecules in the air can't just suddenly change on a surface without thickness. In other words, shock waves do have a certain thickness, and molecular changes occur on thickness equal to the molecular mean free path. In normal circumstances, the molecular mean free path is very small, which is why for calculation purposes, the physical property of shock wave is considered a discontinuous surface (Fig. 2).

Riemann was the first to calculate the relationship between physical forms downstream and upstream of a shock wave, but he made a theoretical mistake. Rankine and Hugoniot respectively carried out their own rectifications and obtained the following equation for calculating shock wave: Assuming that shock wave is stationary, and assuming that P_1, $\rho 1$, T_1, and M_1 represent pressure, density, temperature, and Mach number upstream of the shock wave, while P_2, ρ_2, T_2, and M_2 denote pressure, density, temperature, and Mach number downstream of the shock wave, and γ is constant pressure

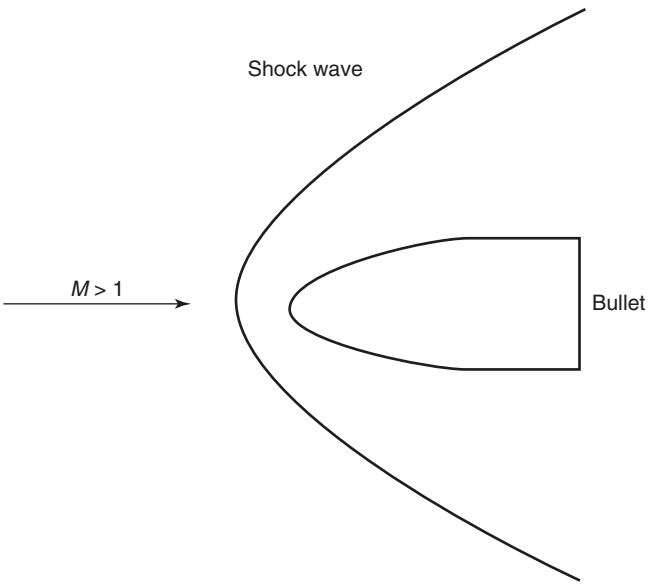

Fig. 1 Shock wave in front of a supersonic bullet

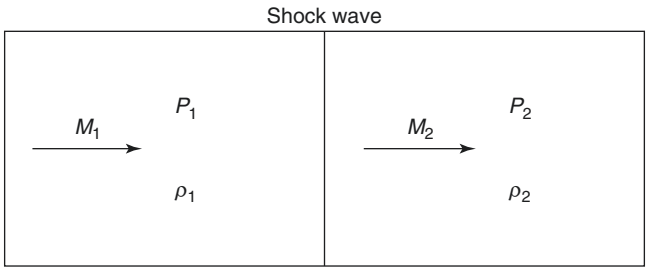

Fig. 2 Discontinuous surface of shock wave

specific heat of gas and ratio of constant volume to heat, then:

$$M_2^2 = \frac{1+\frac{\gamma-1}{2}M_1^2}{\gamma M_1^2 - \frac{\gamma-1}{2}} \quad (1)$$

$$\frac{P_2}{P_1} = \frac{2\gamma}{\gamma+1}M_1^2 - \frac{\gamma-1}{\gamma+1} \quad (2)$$

$$\frac{\rho_2}{\rho_1} = \frac{(\gamma+1)M_1^2}{2+(\gamma-1)M_1^2} \quad (3)$$

Since there is also a rarefaction wave in the shock tube, other than clarifying what a shock wave is, a rarefaction wave should also be explained. Let us take a look at two experiments illustrated in Fig. 3.

Assuming that a piston is moving toward the right at a certain constant speed inside a long tube (Fig. 3a), a rightward shock wave would be generated. As mentioned earlier, when a shock wave reaches a certain area, the air in that area would be compressed and begin flowing to the right. Air downstream of the shock wave meanwhile would remain unaffected and still. This experiment further explains shock wave. On the contrary, if the piston retreats toward the left at a certain constant speed, a leftward rarefaction wave would be generated as depicted in Fig. 3b. When the piston begins to retreat, the air isn't influenced immediately, and only air in the area at the front of the wave is affected. The front of the wave travels at the velocity of sound toward the right, and the particle speed in the air at the front of the wave is still zero but is already starting to accelerate because of the effects of the rarefaction wave. In other words, when a rarefaction wave passes by a certain particle, it accelerates said particle. The closer a particle is to the tail of the wave, the higher the acceleration, until it reaches the tail of the wave, when the particle's acceleration would reach the same velocity as the piston's retreat and stop accelerating. The velocity of particles between the tail of the wave and the piston is the same as the speed of retreat of the piston. When the piston's retreat velocity exceeds a certain level (this level of velocity is called escape velocity), a vacuum would be created between the tail of the wave and the piston, which is why the former is called incomplete expansion, and the latter is called complete expansion.

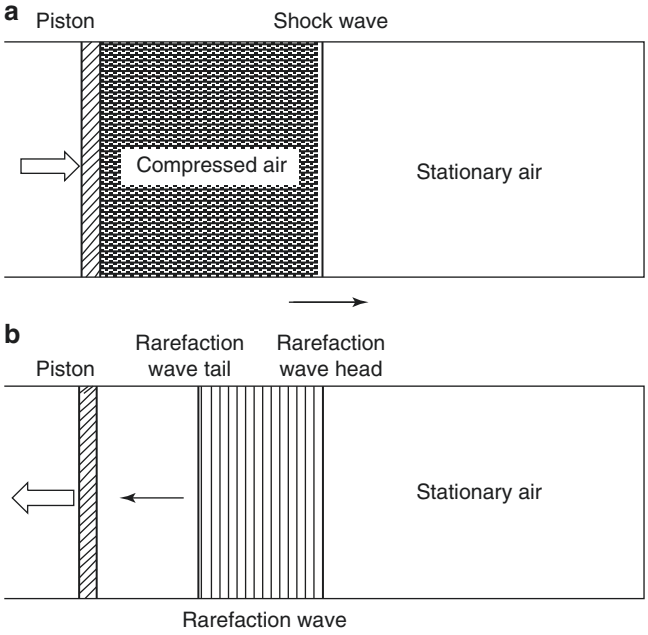

Fig. 3 Shock wave and rarefaction wave. (**a**) Shock wave moving forward to the right; (**b**) Rarefaction wave moving backward to the left

1.2 Theoretical Equations of Shock Tube

A shock tube is just a long tube with both ends closed and the high-pressure and low-pressure chambers separated by the diaphragm. In general, both chambers are to be filled with air, sometimes with specific gas such as hydrogen, argon, helium, nitrogen, or oxygen. When the diaphragm is punctured, a wave is created due to the pressure difference between the high-pressure chamber and the low-pressure chamber. Let us first analyze what happens in the low-pressure chamber. The low-pressure chamber is akin to the piston's forward movement in the tube. Molecules near the diaphragm are compressed into shock wave, which then moves at a supersonic velocity along with the low-pressure chamber. Physical states prior to the shock wave are not affected by the rupturing of the diaphragm. The air between the upstream of the shock wave and contact plane are compressed, and they obtain mean velocity.

Now let's analyze what happens in the high-pressure chamber, which is akin to the piston's backward retreat in the tube. At the high-pressure chamber, a rarefaction wave is formed near the diaphragm. The front of the rarefaction wave propagates toward the left at sonic velocity. To the left of the front of the rarefaction wave is high-pressure gas yet to be disturbed, and the thickness of the wave is conditional on the pressure ratio between the two sides of the shock wave. The molecules between the rarefaction wave and contact plane have already undergone the spreading process and obtained mean velocity. Their particle velocity is the same as the ones in front. Since the gas between the rarefaction wave and contact plane has been expanded, so its temperature reduces. Meanwhile, the gas between the shock wave and contact plane has been compressed, so its temperature rises. Therefore, the former has a lower temperature than the latter. Consequently, the former's sonic velocity is slower than the latter, and the result is that the former has a higher Mach number than the latter.

Experiments show that results calculated from the simple wave and uniform flow field method stated above are very correct. The results are listed below. Of which, α represents sonic velocity, P is pressure, u is particle velocity, w is shock wave velocity, x is distance between shock tube and diaphragm, ρ is density, C_p is constant pressure specific heat, C_v is specific heat of constant volume, M is Mach number, T is temperature, $E_{ij} = (C_vT)_i/(C_vT)_j$ (i, j refers to physical states in certain flow fields in the flow inside the shock tube), $M_i = u_i/\alpha_i$, $P_{ij} = P_i/P_j$, $T_{ij} = T_i/T_j$, $u_{ij} = u_i/\alpha_j$, $W_{ij} = w_i/\alpha_j$, $\alpha_i = (\gamma_i + 1)/(\gamma_i - 1)$, $\beta_i = (\gamma_i - 1)/2\gamma_i$, $\gamma = (C_p/C_v)$, $\Gamma_{ij} = \rho_i/\rho_j$, ($\vec{S}$) = shock wave propagating toward the right, ($\vec{R}$) = rarefaction wave propagating toward the left, and ($\vec{C}$) = contact plane propagating toward the right.

1. Shock wave strength (P_{21})

$$P_{14} = \frac{1}{P_{21}}\left[1-\left(P_{21}-1\right)\sqrt{\frac{\beta_4 E_{14}}{\alpha_1 P_{21}+1}}\right]^{\frac{1}{\beta_4}} \tag{4}$$

$$\text{If } P_{14} \text{ is very small,} \quad P_{21} \approx 1+\frac{\alpha}{\beta_4 E_{14}} \tag{5}$$

2. Density ratio

$$\Gamma_{34} = \left[P_{14}P_{21}\right]^{\frac{1}{\gamma_4}} \tag{6}$$

$$\Gamma_{21} = \left(1+\alpha_1 P_{21}\right)/\left(\alpha_1 + P_{21}\right) \tag{7}$$

3. Temperature ratio

$$T_{34} = \left[P_{14}P_{21}\right]^{\beta_4} \tag{8}$$

$$T_{21} = \frac{P_{21}\left(\alpha_1 + P_{21}\right)}{1+\alpha_1 P_{21}} \tag{9}$$

4. Shock wave velocity and Mach number

$$M_{11} = \left[\beta_1\left(1+\alpha_1 P_{21}\right)\right]^{\frac{1}{2}} \tag{10}$$

5. Particle speed of contact plane or uniform flow field

$$U_{34} = \left(1/\gamma_4\beta_4\right)\left[1-\left(P_{14}P_{21}\right)^{\beta_4}\right] \tag{11}$$

6. Mach number of uniform flow field

$$M_3 = \left(1/\beta_4\gamma_4\right)\left[\left(P_{14}P_{21}\right)^{-\beta_4}-1\right] \tag{12}$$

$$M_2 = \left(P_{21}-1\right)/\gamma_1\left[\beta_1 P_{21}\left(\alpha_1 + P_{21}\right)\right]^{\frac{1}{2}} \tag{13}$$

7. Rarefaction wave velocity

$$\text{Front of wave} \quad C_{14} = -1 \tag{14}$$

$$\text{Tail of wave} \quad C_{34} = \frac{1}{\gamma_4\beta_4}\left[1-\left(P_{14}P_{21}\right)^{-\beta_4}\right]-\left[P_{14}P_{21}\right]^{\beta_4} \tag{15}$$

From the above equation, it can be seen that by measuring the physical states of the air inside the tube before the rupturing of the diaphragm, one could calculate the flow in the shock tube.

1.3 Shock Tube Technologies in Shock Wave Protection Research

1.3.1 Key Issues in Shock Wave Protection

It is widely known that nuclear weapons injure and kill via four damage and destruction factors: Light radiation, shock wave, early nuclear radiation, and radioactive contamination. The truth is, people have also realized that the destructive power of electromagnetic pulse also can't be ignored. Of these damage and destruction factors, a shock wave is the most powerful. In nuclear weapon defense research, protection against shock waves occupies a central role. Shock waves can wound and kill people in direct and indirect manners. The so-called direct injury and fatality refer to people that are injured or killed due to the effects of the shock wave. Indirect injury and fatality, meanwhile, refer to mechanical trauma caused by structures and objects that collapse when struck by a shock wave or different projectiles like weapons, sand, stone, brick, tile, and glass fragments that are launched by the force of the shock wave. Nuclear blast injury is classified as either mild, medium, severe, or extremely severe. Different levels of blast injuries caused to exposed personnel on the ground have corresponding shock wave overpressures, and for severe or extremely severe injuries, there are also corresponding dynamic pressures. Under the same relative altitude of an explosion, an air detonation of a 20-kiloton nuclear weapon can cause medium injury within about 1000 m of ground zero. Meanwhile, that of a megaton nuclear weapon can cause medium injury within about 4000 m of ground zero, and so its area of damage is very big. When Japan was hit with nuclear bombs in World War II, 70% of those injured

suffered blast injuries. After Hiroshima was nuked, 60% of early casualties were attributed to blast injuries.

Nuclear blast wave has positive pressure effective duration that is quite long, from 0.3 to 2–3 s. This allows shock waves to penetrate into the interiors of defensive fortifications from different holes and crevices, damaging, wounding, and killing equipment and personnel contained and hidden inside. How to prevent shock waves from entering the interior of fortification through the air inlet, air outlet, smoke exhaust, sewage outlet, door, and other openings, in other words fortifying these holes and gaps, is a top priority for defense engineers and researchers.

As nuclear weapons become increasingly powerful, fortifications are also buried deeper underground in order to maximize the earth's natural load-bearing capacity. The main structure of the North American Aerospace Defense Command (NORAD) has a natural protection layer 420 m thick. In recent years, civil defense structures have become increasingly important in nations around the globe. The beliefs that the "atomic era is an underground era" and "Europe goes underground" are common in Europe, and Switzerland has deeply buried bunkers and shelters that can accommodate 80% of the country's total population. This brings us to the next question of how shock waves propagate in rock and soil mediums.

Shock wave protection involves a multitude of subject matters related to mechanics. Some key subjects include: calculating parameters of a shock wave from a nuclear explosion; reflection, diffraction, and other issues when shock wave encounters different kinds of obstacles; dynamic load borne by structures and objects on the ground surface; propagation and decay of shock waves that entered tubes or tunnels; propagation of shock waves in rock and soil medium; mutual effects between load, underground structure, and medium. In short, many issues require further study.

1.3.2 Application of Shock Tube Technology

Defense research involves not only a multitude of theoretical and calculation problems, but it is also mandatory to prioritize empirical research. Between 1945 and 1972, the United States carried out 193 atmospheric nuclear tests, underground 358 nuclear tests, and 5 underwater nuclear tests. Nuclear tests were conducted predominantly to study technical issues pertaining to the nuclear weapons themselves. But at the same time, other experiments related to the effects of nuclear weapons were also performed, including shock wave protection. However, since nuclear test sites and climate conditions affected the destructive factors of nuclear weapons, which limited what participating personnel could do, and thus direct experimentation could not fully solve the problems. Pursuing cost performance, reliability, and replicability, people researched and developed a plethora of indoor experimentation instruments to simulate blast waves.

Shock tube technology was invented back in the 1880s, but its application in blast-resistance experiments and research did not take place until the past 50 to 60 years. In 1953, Sweden became the first country to complete the construction of a shock tube with a diameter of 1.0 m and length of 11.5 m. Toward the end of the 1950s, the U.S. Army, Navy, and Air Force built different kinds of shock tubes, of which the U.S. Naval Ordnance Laboratory constructed the largest conical shock tube, which had a diameter of 0.4 m at the tip, a diameter of 7.2 m at the base, and total length of 736.4 m. China began using shock tubes for shock wave protection research in the 1960s. What kind of shock wave protection experiments and studies can be done with shock tubes?

1. **Experiments and studies about shock wave entry into pipes, tunnels, and other kinds of entry and exit openings**. When a shock wave enters a pipe or a tunnel, if the incident wave's direction differs from the included angle of the axis of the pipe or tunnel, the new shock wave that forms inside the pipe or tunnel has a different strength. When a shock wave propagates inside a pipe, due to influences of the boundary layer and expansion wave, the shock wave's strength will decrease. When the cross-section of the axial direction of the pipe changes, the shock wave's strength could both decrease or increase. If the area of the pipe shrinks, the shock wave's strength would increase; if the pipe curves, and reflections, refractions, and other waves are generated during the shock wave's propagation, the overpressure would increase. The increase in overpressure instigated by the geometric shape of the pipe is always less than the overpressure of the direct reflection of the shock wave. When a shock wave propagates along a pipe with an uneven cross-section or a non-straight pipe, the shock wave's imagery is rather complex. Using wave theory to make the calculation is complicated and practically unnecessary. Empirical and semi-empirical calculation formulas proposed both in China and abroad are chiefly the result of empirical trials. U.S. Air Force design manual clearly states that information mostly comes from research conducted in modeled tunnel systems based on shock tubes. China has also conducted "experiment and research on different kinds of tunnel entry and exit methods," "experiment and research on pressure distribution of civil defense fortification entry and exit," and other such kinds of tests. Through the use of shock tubes in experiments, scholars have been able to obtain pressure distribution and pressure on doors related to different types of entry and exit. As long as the model design is conducted in accordance with appropriately approximate conditions, experiment results are applicable. If nuclear explosion effect experiments could be checked and corrected, their results could be more reliable in the real application.

2. **Experiments and studies about dynamic load borne by structures and objects on the ground surface**. The dynamic loads borne by structures and objects are not only associated with the incident wave's free field parameters (e.g., wind speed, pressure, direction) but also properties of the structure or object, such as shape and surface characteristics. Usually, the dynamic load is calculated by multiplying dynamic pressure with the resistance coefficient. However, the resistance coefficient measured in a constant speed wind tunnel can't be directly applied on blast-resistant structures and objects on the ground surface. This is because the wind speed upstream of the shock wave changes instantaneously. The influence of free field parameters on resistance is indicated as Reynolds number (R) or Mach number (M). The Reynolds number indicates how airflows around objects: A low Reynolds number means laminar flow, while a high Reynolds number points to turbulent behind the structure or object, and the turbulent boundary layer passes the weaker resistance to the target. Airflow inside the range of critical Reynolds numbers is unstable and could change at any instant. Mach number may be divided into three ranges: The subsonic speed range is approximately $M < 0.5$, and the air around the structure or object flows at subsonic speed; transonic speed range is about $0.5 < M < 2$, and the air around the structure or object flows at supersonic or subsonic speed, and the supersonic speed range is about $M > 2$, and the air around the structure or object flows at supersonic speed. In order to study dynamic load borne by structures on the ground surface, Chinese scientists have also used blast-resistant shock tubes to perform experiments related to ground surface structure destruction and ceiling load capacity of civil air defense fortifications attached to other structures, load distribution of cones, and influence of terrain on shock wave.
3. **Experiments on blast-resistant engineering and structures**. There are numerous contents related to experiments on blast-resistant engineering and structures, and Chinese scientists have carried out many related tests. Generally speaking, such experiments may be divided into two categories. One category is anti-blast wave equipment such as anti-blast valve, blast door, wave reduction troughs, and filtering equipment. The other category is protective structure, mostly shallow-buried structures such as beams, arches, and domes. Experiments on anti-blast wave equipment mostly aim to test the strength of the equipment and whether anti-blast wave performance satisfies design standards and requirements. Experiments on protective structures face the challenge of totally replicating conditions of the structure, and thus usually, small-scale tests are performed to verify theoretical calculation methods and explore patterns in how the structure reacts to dynamic effects. Chinese scientists have carried out many experiments in these two categories using blast-resistant shock tubes. When performing small-scale experiments for shallow-buried structures, there are two points that demand attention. First is the placement position of the structure, ensuring that soon after the peak pressure of the compression wave passes by the structure, as in before the occurrence of the structure's greatest displacement, the reflection wave from the front wall of the box and the rarefaction wave at the back wall of the box do not act on the structure. Second are issues related to the analysis of measurement results of structure deformation parameters. For all measured parameters, mark the starting time of all records so as to facilitate reading any parameter values at any given instant. The key to the analysis should be how the structure reacts when the peak value of the compression wave passes by the whole structure or the instant when the structure's greatest displacement occurs. Researchers should refrain from analysis of all waveforms because that would include influences from reflections and rarefaction off of the side walls. In order to study the influences of the side walls of the box, optical photography of the waves of the air shock waves in the simulation box was carried out by optical imaging instrument. Experiment results indicate that positions that satisfy experiment requirements can be chosen. Some are doubtful whether shock tubes can be used to test shallow-buried structures, and the root of the issue lies in influences from reflections off of the side walls and analysis of all waveforms. However, it should be noted that a steady flow period is often the portion used for aerodynamics model tests conducted in shock tubes. Calibrations of boxes with sand medium indicate that range of influence of reflections from box bottom is not that big. If energy-dissipation pads are installed, the influence of reflections from the box bottom could be further lessened. This demonstrates that using shock tubes to conduct tests about shallow-buried structures is viable.
4. **Experiments on dynamics properties of soil and propagation of stress waves in soil**. The shock wave generated by the shock tube is a strong discontinuity surface, and it loads suddenly and at great speed, and thus conducting testing on the dynamic properties of soil is quite appropriate. In order to raise peak pressure, often times reflection from the surface is used for testing. A shock wave produced by an even cross-section shock tube is one-dimensional. Connect cylindrical-shaped soil at one end of the tube to conduct a test, but it is necessary to compensate for influences from frictions with the side of the tube. With the proper setup, it is viable to conduct testing and research about the propagation of one-dimensional waves in soil. In experiments on dynamics

properties of soil and propagation of stress waves in soil conducted abroad, the majority have been carried out in vertical shock tubes.

5. **Experiments on blast injuries on organisms**. After the appearance of nuclear weapons, blast injury has risen as a key research topic in protection medicine, and materials from both China and overseas indicate that studies on the source of injury of "blast injury" are done using shock tubes. Biological effects can be observed qualitatively and measured quantitatively, and systematic studies on blast injury's early diagnosis, pathological anatomy, and experimental treatment may all be undertaken. Indoor experiments may be performed repeatedly, which is helpful for observation. Participants may also carry out experiments and tests without the need to prepare protection measures, which is conducive to studying patterns and laws related to the matter at hand. However, it is necessary to combine tests with nuclear effect experimentation in order to uncover the differences and associations between the two. Only by doing so would researchers be able to determine the similarity and applicability of indoor experiment results.
6. **Tests on the dynamic performance of pressure sensors**. Tests on the dynamic performance of pressure sensors mainly include calibration curve of pressure value, frequency characteristics, damping characteristics, etc. To test the dynamic performance of pressure sensors, people have researched and developed some devices such as the shock tube, which has been put to effective use. The shock tube can generate a strong discontinuity surface, which ensures a steep pressure front of the shock wave, allowing the signal to possess characteristics of step-function signals. There is a certain steady flow zone behind the shock wave, and further back is the unsteady flow zone, with the whole duration lasting up to 110 ms. The waveform of pressure waves created in the shock tube has a relatively wide frequency band. It is an ideal instrument for testing the dynamic performance of pressure sensors. The pressure value may be converted using the shock wave's Mach number. Measurement system of shock wave velocity, when coupled with high-precision digital voltmeters with instantaneous value, could produce relatively reliable pressure calibration values. A large number of pressure calibration experiments demonstrate that using shock tubes to calibrate overpressure sensors can produce accurate and reliable results, with relative error within ±2%. With regard to testing the performance of dynamic pressure sensors, experiments show that analyzing the response curve of a sensor can reveal diffraction property, damping property, drag property, etc. With regard to calibration of dynamic pressure value, since the velocities of airflow in front of and behind the contact plane inside the steady flow zone are the same, densities are different. Thus dynamic pressure values vary and exhibit a tiered-step distribution. Experimental values and theoretical values of dynamic pressure often deviate when overpressure is relatively high. More research efforts are needed to analyze value conversion, error, and other aspects of the shock tube when utilized as a dynamic pressure calibration instrument.

In light of the above, the shock tube has many uses and functions, making it an irreplaceable device in blast-resistance and protection research. As a load device, it is a rather mature technology. However, how to put this device into an application and more effectively carry out different kinds of blast-resistance engineering testing requires further efforts in research experimentation theories and experimentation technical issues, for instance, the law of similarity in blast-resistance engineering testing, technical means to reduce box friction and box reflection, or pressure increase measures for and analysis of errors in high-pressure calibration. All in all, any kind of experiment must be based on an experiment plan designed in accordance with experiment requirements and device performance. Sometimes, it might also be necessary to modify existing equipment according to experiment aims. Experiment instruments can only provide a certain degree of performance for experimentation. How to make the most out of these conditions to address one's own research topic also requires researching relevant technologies.

2 Categories and Features of Biological Shock Tubes

2.1 Technical Requirements of Biological Shock Tubes

Mechanical properties of materials in biological tissues vary quite markedly from those seen in non-biological materials and engineering. Therefore, shock tubes used for biological experiments and research have some unique requirements compared to those used in other engineering fields.

1. **Generation of shock waves with typical waveforms**. When using shock tubes to simulate the generation of blast waves of biological experimentation, the exponential wave, and not the "platform wave," is the waveform required for the first reflection and rarefaction wave that catches up to the front of the shock wave.

   It should be noted that different weapons (i.e., guns and cannons), explosion type (i.e., chemical explosion, nuclear explosion), and TNT equivalent generate different waveforms that result in different injuries. For example, cannoneer auditory apparatus and non-auditory

apparatus injuries caused by shock waves from the muzzle of cannon, cumulative injury effects from firing weapons repeatedly, and how a shock wave from a bullet traveling in water affects cell metabolism and form are all directly associated with shock wave waveform parameters. Explosions inside fortifications, shelters, and tunnels, or those inside enclosed or semi-enclosed spaces like that of an armored vehicle or battleship, generate complex waves because of multiple reflections of the initial shock wave, and the injuries caused are also unique.

When conducting biological experiments, test animals are placed at the front of the opening of the shock tube's driven segment and are injured using a shock wave that flows out from the tube's opening. At the same time, it is also imperative to prevent unnecessary waveforms created by the shock tube itself. This is very important for various kinds of studies that aim to research the relationship between blast waves and biological injury.

2. **Utilization of different types of biological inspection technologies and test instruments**. When studying the effects of shock waves on the whole body, specific organ, tissue, or cell, it is necessary to adopt different and often sophisticated techniques such as exposure of the entire body, exposure of specific body parts, filling specific organs or tissues inside or outside the body, or cell cultivation. Blood oxygen and system, temperature control device, respiratory tract, and other items need to be placed near the test animals or connected to animals via tubes while ensuring that the items won't be damaged by shock waves inside the tube.

   Research matters such as shock wave pressure–injury severity relationship, instantaneous changes in the volume of the animal chest or abdominal cavity upon the action of a shock wave, diaphragm motion, displacement of the chest or abdominal wall in response to shock load, and its rate of change, the time difference in the propagation of shock wave in tissues versus blood vessels, the centripetal transmission of pressure waves in the vascular system and effects of wave interference, measurement of electrophysiological indexes and regulation of the respiratory state, or stress concentration or focus created by stress wave reflections from different interfaces or when stress wave propagates to certain tissue or structure, require the placement of different types of sensors in the shock tube, sometimes on the surface of animals or inside the test animals. In addition, hi-speed cameras or hi-speed X-ray cameras might be placed in the surroundings to record related parameters and their changes. Shock tube design is required to be able to ensure the implementation of the aforesaid methods, including sensors and test instruments, remains undamaged.
3. **Capacity to adjust the pressure inside the shock tube**. When closing the ends of the shock tube for usage, reflection pressure may be raised, but the high-pressure environment inside the tube in the span of several seconds to several minutes after the shock wave disappears might cause the test animal to suffocate. From another perspective, a strong shock wave causes test animals to die rapidly, and the main cause of death is coronary or cerebral artery embolism due to gas from the lungs that enters the pulmonary veins. If high-pressure chamber treatment is administered, the fatality rate could be reduced, and the high-pressure environment inside the tube might also offer curative and preventative functions. These two situations complicate the injury factors and perplex the difficulty of analyzing experiment results because such conditions usually don't exist in explosions in the real world.

   When investigating the relationships between explosions and injuries encountered by personnel at high altitude in the air or those underwater, it is necessary to increase or decrease pressure in the driven segment. Subject animals to the effects of shock waves in environments with different pressures, and quickly raise or reduce environment pressure after causing injury. Then maintain the status for a certain duration.

   Therefore, the shock tube has to have tube segments that can be sealed adequately and equipped with devices that can increase or decrease pressure swiftly, or the capacity to rapidly seal and isolate the experiment segments, so as to ensure the speed of pressure increases or decrease.
4. **Others**. TNT equivalent, and distance between animal and center of the explosion, are directly related to injury severity. Impulse-related injuries are caused by small TNT equivalent and close distance to the center of explosion (i.e., high peak overpressure, and short effective duration), while pressure-related injuries are caused by opposite conditions. Researching the differences between and protections for these two kinds of injuries require shock tubes to be able to create a rather wide range of shock wave peak overpressure and effective duration. However, when positive pressure effective duration surpasses 5 ms, usually wave peak overpressure doesn't need to exceed 600 kPa because this is already the threshold value for 100% fatality in common test animals.

   When inducing a chemical explosion, a substantial quantity of toxic gases like carbon monoxide might be generated, and these could suffocate or poison animals to the extent of death. Such circumstances are unfavorable to exploring the injury or fatality effects of any single element (such as overpressure) and the underlying mechanisms. When performing dose-response research or trying to determine damage and injury area or death rate, the shock tube's driving force needs to create toxic environments like those resulting from the explosion of explosives or air-fuel bombs.

2.2 Categories of Biological Shock Tubes

Based on the length and diameter of the driver segment and driven segment, biological shock tubes may be categorized as large, medium, small, or micro. Based on length of effective duration, categories include long-duration, medium-duration, and short-duration shock tubes. By the amplitude of peak overpressure, categories include high-pressure, medium-pressure and low-pressure duration shock tubes. However, none of the above categories can comprehensively encompass all the features of biological shock tubes. For example, a micro shock tube could create a positive pressure effective duration lasting up to 1 s. Based on currently available literature and reports, only the USA and China have been able to construct biological shock tube systems.

Over the span of 11 years since the 1950s, the USA has built four multi-purpose shock tubes ranging from 0.31 to 1.83 m in diameter at the Lovelace base in New Mexico City. Using nearly a hundred different assembly methods, more than 10,000 experiments have been conducted on 12 kinds of animals (mouse, rabbit, pig, dog, goat, etc.) to study the animals' tolerance for shock wave load and species-specific association, then extrapolated related injury standards for the human body.

2.3 Variable Cross-Section Shock Tube

1. **"1.07/1.83" variable cross-section shock tube**, total length of 54.56 m. Driver segment has an internal diameter of 1.07 m and length of 4.75 m and is connected to the 38 m-long diffuser segment, and the 2.74 m-long cone segment is an experiment segment with a diameter of 1.83 m and length of 9.14 m. The ends are sealed with blocking boards to carry out research on an overpressure-induced injury.

   Its structural features are: (a) The surface of the cone segment has seven square holes that amount to 0.65 m^2; (b) the cone segment is situated far away from the driver segment; (c) the bottom has a rail system to facilitate the operation of the diaphragm and changing tube segments. Through the holes on the cone segment, rarefaction waves may be oriented to avoid multiple reflections between the blocking board and cone segment, while the rail system can adjust the position of an animal inside the experiment segment, and these designs all enable "modification" of a waveform to select the desired waveform parameter.
2. **"0.61/1.03" variable cross-section shock tube**, total length of 21.34 m. The driver segment has an internal diameter of 1.03 m and length of 5.31 m; the area near the diaphragm is conical, and the diameter changes to 0.61 m. Diffuser segment (internal diameter 0.61 m and length 9.14 m) and cone segment (length 0.91 m) are attached to the diaphragm in that sequence, thereby enlarging the internal diameter to 1.03 m. Then a 6.71 m-long uniform cross-section experiment segment is attached, and the end is sealed with a blocking board to carry out research on an overpressure-induced injury.

   Its structural features are: (a) End of the driver segment is semi-spherical in shape, which is unfavorable to the formation of reflected rarefaction wave and can help extend the positive pressure effective duration of the shock wave; (b) driver segment has a nozzle shape, which expands the volume inside the driver segment and reduces diaphragm material usage; (c) three trapezoidal holes evenly distributed on the top of the cone segment near the experiment segment amount to 0.19 m^2 in area, which can be used to introduce rarefaction wave to quickly transform "platform-shaped" shock wave into blast wave in exponential form, which can facilitate more sufficient use of peak overpressure compared with attaching a cone segment to the driver segment; in addition, these holes can rapidly release pressure after shock wave disappears, and swiftly reduce amplitude of multiple shock wave reflections that occur between blocking board and cone segment; (d) cone segment is semi-spherical in shape, and shock wave reflection off of the spherical surface is focused instead of reflecting to test animals 9 m away, so that the waveform of shock wave that acts on the surface of the animals more closely resembles those of single explosions; and (e) positive pressure effective duration can last up to 400 ms.

   This equipment was modified later. A tube 0.91 m in length and 0.61 m in diameter was installed as a driver segment to lower the volume of gas that enters the driven segment. The previous driver segment, with 1.03 m in diameter and 5.31 m in length, was turned into a gas cylinder or vacuum cylinder, connected to the driver segment and the driven segment via pipe and valve. This setup enables adjustment of pressure inside the experiment segment before and after rupturing the diaphragm to simulate environment pressure in high-altitude mid-air and underwater explosions. Several animal cages can be directly affixed to the blocking board at the end of the driven segment. This method has proved that raising pressure inside the tube before and/or after rupturing the diaphragm and sustaining such pressure for an hour can reduce the fatality rate of animals.

2.3.1 Uniform Cross-Section Shock Tube

1. **Shock tube with diameter of 0.61 m** and total length of 43.6 m. The driver segment has an internal diameter of 0.61 m and length adjustable between 1.52 m and 3.05 m; the 29.56 m-long diffuser segment in front has a circular uniform cross-section, and behind is a 9.75 m-long segment with a flat bottom and semi-circular cross-section,

while the remaining 4.27 m-long segment also has a circular cross-section. The experiment segment has a length of 5.18 m and is located 10.67 m from the diaphragm. Five containers 20.3 cm × 20.3 cm × 20.3 cm in dimension are installed on the pipe wall of the experiment segment, which is where the animals are placed. By affixing fillers of different sizes and shapes in the containers, scientists could change the area and height of exposure of the animal contained inside, thereby simulating injury conditions of personnel in different postures when exposed to shock waves inside a shelter. Five square steel plates 43.18 cm × 43.18 cm in dimension are located at the bottom of the 9.75 m-long segment with a flat bottom, where animal models of different shapes are placed. If an animal model is placed behind the plate, scientists could study how shock waves flow around the plate and injure the subject behind, which is a simulation of injuring conditions of personnel hidden behind walls.

2. **Shock tube with diameter of 0.31 m** is similar to "shock tube with diameter of 0.61m." The driver segment has an internal diameter of 0.61 m and a length of 0.76 m. During operation, when the diffuser segment has its end opened, the driven segment has a length of 9.14 m, or a length of 5.18 m when the diffuser segment end is closed. Pipe may be attached to the diffuser segment to raise or reduce pressure inside the diffuser segment before or after the diaphragm ruptures so as to simulate how environment pressure changes affect injury severity (0.5–1.5 standard atmospheric pressure).

2.3.2 Combination Shock Tube

In order to generate shock waves with different waveforms and various effective duration, pipe segments with varying diameters are used to create numerous combinations. This is called a combination shock tube. Its structural features are as below:

1. Change the length of the driver segment or open holes on the cone segment to obtain different waveforms during positive pressure effective duration.
2. At the diaphragm and/or middle of the diffuser segment, reserve a gap of 8–25 cm at the segment connected with a flange, or place the end of the relatively thinner upstream tube near the end of the relatively wider downstream tube with a gap of a certain width in between them, creating a separated type of combination. When the "platform-shaped" shock wave from upstream arrives at the gap, it would quickly rarify into a blast wave with exponential form, then reflect off of the blocking board behind the animals. The wave then rarifies again at these gaps, and after reflecting off of the blocking board at the end of the driver segment, it is rarified once again at these gaps. The shock wave's strength has weakened after rarifying three times, and the animal would be pretty much subjected to the effect of a single wave with exponential form.

   The diffuser segment uses relatively thinner tubing, and the experiment segment uses relatively wider steel tubing and opens holes on the top of the cone segment in front of the experiment segment. By using methods that generate typical blast waves or by modifying waveform through separated attachments, experiment results indicate that the diffuser segment's diameter could be shrunk and total length could be shortened, while waveform factors could be significantly improved. These methods are easy to apply and are valuable references in the design of biological shock tubes.
3. Movable blocking board installation: Using a uniform cross-section shock tube with a diameter of 0.61 m, install animal containers on the tube wall near the end of the diffuser segment. The depth of the container should be approximate to the transverse diameter of the chest cavity of the animal. The blocking board at the end of the diffuser segment can be moved to change the time difference between the incident shock wave and peak reflection that act on the surface of the animal. This setup simulates how shock waves act on personnel in different locations inside a shelter or bunker or situated at different distances from a rigid wall, enabling scientists to explore how this time difference influences injury severity. Experiments have proven that when the test animals are subjected to the effects of shock waves, the longer the time difference, the higher their tolerance.
4. To simulate the shock wave effective duration that acted on animals placed inside troop shelter at the nuclear testing site in Nevada, a butane tank with a length of 6.02 m, the maximum internal diameter of 1.03 m, and volume of $3.82m^3$ was used as driver segment. Said driver segment as a semi-spherical end and a conical end, connected by two cone segments with identical taper degree, reducing the internal diameter to 0.31 m. A diaphragm may be inserted at the 0.31 m spot or in between the two cone segments. The experiment segment has a maximum internal diameter of 1.03 m, a length of roughly 2 m, and an internal volume of around 1.1 m^3. It is cylindrical in shape with spherical ends, and steel pipes with a diameter of 0.31 m are welded to the two spherical ends, with one end sealed using a steel plate with size-adjustable holes and the other end connected to the driver segment in a linear or T-shaped configuration using a pipe tee fitting with a diameter of 0.31 m. The lengths of the three outlets of the pipe tee fitting may be adjusted. Spherical blocking boards are set inside the experiment segment, which can be moved to change the velocity and direction of the crossflow. Test animals are placed within the concavity of the sphere boards. Shock wave peak overpressure, pressure increase, and decrease rate acting on the test animals

may be adjusted through modifying the lengths of the three outlets of the pipe tee fitting, size of holes, and location of the movable blocking boards. Pressure may be raised from 690 to 1380 kPa within 10–155 ms, positive pressure effective duration could last anywhere from 5 to 20 s, and it is capable of simulating positive pressure effective duration equivalent to the blast wave of a ten megaton nuclear weapon.

The aforesaid shock tubes all use compressed air as driving force, soft polyester film as a diaphragm, and gas pressure or gunshot as a diaphragm rupturing method.

2.3.3 Explosive-Driven Biological Shock Tube

When Clemedson et al. studied the changes in pulmonary compliance in rabbits, experiments were undertaken using variable cross-section shock tubes. Animals were placed at the end of the experiment segment (with an internal diameter of 2.3 m and length of 12 m) right next to the block boards at the end. The explosion segment had an internal diameter of 0.7 m and length of 4 m, behind which were connected thin exhaust pipe and sound muffler. The size of the thin exhaust pipe opening may be changed to adjust the amplitude and effective duration of reflection pressure. Explosive is placed between 2 m-long cone segments and can simulate the explosion of 7–30 g of TNT, with peak overpressure and positive pressure effective duration respectively reaching up to 360 kPa and 15 ms.

Read et al. reported a near-conical shock tube with a total length of 72.5 m and an experiment segment internal diameter of 4.9 m. This device can simulate peak overpressure and positive pressure effective duration equal to the explosion of 20 tons of TNT, with waveforms very similar to real-world chemical explosions. Phillips et al. used an explosive-driven shock tube with a length of 36.6 m and an internal diameter of 3.0 m to conduct a pressure-injury experiment on bulletproof vest-clad sheep. Animals were placed near the blocking board, the other end of the tube was opened, and peak overpressure was set at 420 kPa.

2.3.4 Micro Shock Tube

In recent years, a micro shock tube has risen as one of the preferred devices for scholars researching the relationship between localized shock wave exposure and injury and the relationship between localized shock load and impact injury. During car accidents, localized blunt impact trauma is one of the frequent injuries. Yen et al. built a micro shock tube to study how the lungs respond to shock load. The driver segment has an internal diameter of 2.5 cm, uses compressed gas as driving force and paper as a diaphragm. When pressure is built up to 207–276 kPa, a gas-driven needle will burst the diaphragm.

Jaffin et al. designed a similar micro shock wave generator to study localized blast injury. The driver segment has a volume of 150 mL and can withstand pressure from 10 to 25 MPa, and uses one or several 0.36 mm-thick aluminum foils as diaphragms, to be burst with built-up air pressure. Animals are placed in front of the opening of the driven segment, and adjusting the distance between the animal and tube opening can change shock wave strength. Effective duration is always 340 μs, and a high-speed X-ray camera is used to observe and record the Mach disc that the shock wave creates on the body surface of the animal and imagery of impact from high-speed airflow. The unique feature here is the ability to measure electrocardiogram changes in the animal under direct observation. Another feature is the ability to adjust the shock wave's direction of action by connecting a high-pressure soft tube at the end of the driver segment and attaching a diaphragm segment at the other end of the soft tube.

The waveforms generated in the shock tubes mentioned above are not smooth. Some scholars employed the pressure relief principle and researched and developed pressure relief-based micro shock that can produce regular, exponentially decaying waveforms with an effective duration of up to 1 s. Animals are placed at the end of the diffuser segment, and the end is sealed using a blocking board with pressure-relief holes. A delayed timer is used to control the size of the opening of pressure-relief holes and the rate at which they open. After rupturing the diaphragm and when the shock wave reaches the animal, the pressure-relief holes are opened. Since the driven segment is very short after the shock wave disappears, high-pressure gas fills the whole tube, creating a high-pressure environment, and the size of pressure-relief holes is increased in exponential micro-enlargement, allowing pressure inside the tube to reduce in exponential form. Adjusting the gas-release rate of pressure-relief holes can change the curvature of the falling edge of the pressure waveform, enabling positive pressure effective duration to last up to 1 s, equivalent to the time of the detonation of a 10 kiloton nuclear weapon. Soft and uneven wave absorbers are placed at both ends of the tube to prevent reflection wave and rarefaction waves from affecting the smoothness of shock wave curvature, with the resulting exponential waveform being quite satisfactory.

2.4 Features of Biological Shock Tube

2.4.1 Advantages

The biological effects of shock waves generated in shock tubes are similar to those generated in chemical explosions or nuclear explosions. A shock tube offers numerous advantages for blast injury research.

1. Shock waves with a long effective duration can be produced economically, facilitating the simulation and repli-

cation of some waveforms. This is a basic requisite for acquiring substantial biological samples for statistical purposes.
2. Instrumentation and unique direct or X-ray cameras for recording animal's physiological changes inside may be placed near the shock tube, enabling the performance of functions and dynamic measurement right at the instant of injury or soon after the injury, which is difficult or impossible to achieve at the site of an actual chemical explosion. In addition, results are consistent and replicable.
3. Adjusting the shock tube's assembly method can change waveform parameters, and waveforms may also be modified through various means.
4. Animal injury can be replicated, allowing the testing of various indices at the same time.
5. Experiments may be conducted indoor or near laboratories, without the need to travel long distance to the site of a chemical explosion. This cuts down the labor and financial costs, and experiment processes and results are not impacted by external conditions like weather.

2.4.2 Disadvantages

1. **Limitation of the shock tube as a "source of injury" equipment**. During a large chemical explosion or nuclear explosion, negative pressure effective duration is much longer than that of positive, sometimes twice or even ten times longer, and studies have pointed out that negative pressure can also be very damaging. In a shock tube, negative pressure amplitude and effective duration depend on whether or not gas pressures at the tail of reflected rarefaction waves are lower than initial pressure inside the driven segment. Compared with initial pressure gas pressures, the lower the gas pressure at the tail of the rarefaction wave and the longer the sustained duration, then the longer the negative pressure effective duration and bigger the negative pressure amplitude. However, the working principles and structures of the existing cylindrical shock tube are not conducive to the production of powerful rarefaction waves, and thus negative pressure parameters can't achieve the standards stated above. Perhaps discussing shock tube working principles and structural design that can produce long negative pressure effective duration is already a new research topic.

   During a chemical explosion or nuclear explosion, waveforms of the generated shock wave are not nearly as smooth and regular as those produced in a shock tube. Scholars have hypothesized that lung injuries are caused because the lower frequencies of blast waves are close to the existing frequencies of the lungs. However, since waveforms of shock waves generated in shock tubes do not have that wide spectrum of frequencies in the shock wave of an actual explosion, injuries might be caused to a certain degree. Under the same chemical explosion conditions, influences such as weather, terrain, architectural structure, protection measures, body position, and other factors could all create rather big differences in the relationship between waveform and injury. The limited number of shock tubes can't totally simulate these conditions.

   When conducting dose–response relationship research, the range of explosive quantity can be rather large, from 1 g to 2000 tons. Sometimes chest and abdomen models or mannequins are placed inside the experiment venue to research the biological effects of combined shock waves in closed environments. Often times tunnels, armored vehicles, and such venues are used to test and discuss how blast wave dynamic pressure affects secondary blast injury, tertiary blast injury, and other matters. To fully replicate the aforementioned situations would be remarkably challenging for any laboratory or even impossible.

   In light of the above, biological shock tubes can't completely replace chemical explosion or nuclear explosion experiments.
2. **Shock tubes are permanent fixtures**. A completely constructed shock tube can only simulate blast waves to a certain degree, and it is necessary to build other supplementary systems like add-on components and compressed gas sources to satisfy normal experimental needs. Thus, blast wave injury studies are difficult to conduct in regular labs, and one-time experiment investments can be rather sizable. From design and production to installation, commissioning, and actual operation, the process might take several years to complete. For research projects with tight time constraints, a chemical explosion is a more suitable option. For a completely constructed shock tube, it can only simulate chemical explosions to a limited extent and won't offer a very broad scope of application. Moreover, some specific biological experiment projects might not last very long, and equipment would be left idle. These all lower the usage frequency of the shock tube.
3. **Danger and environmental pollution**. Compressed air-driven shock tubes are often outfitted with compressed air container facilities. At least, that would be the case for the entire duration of some experiments. Shock tubes are often built inside or near the lab, which poses a certain degree of risk to personnel and the environment. Unlike chemical explosions conducted in wide, open areas where personnel stay a safe distance away, explosive or compressed air-driven shock tubes generate rather considerable vibrations and noise more than a hundred decibels. When shock tubes are used with ends sealed, it is inconvenient to install a sound suppression system, which is somewhat harmful to operating and test personnel, as well as the surrounding environment.

3 Development and Application of Biological Shock Tubes in China

3.1 Design Principle and Technical Proposal for the First Biological Shock Tubes in China

3.1.1 Design Proposal

With assistance from the Institute of Mechanics of the Chinese Academy of Sciences, the Institute of Field Surgery of the Third Military Medical University began to construct the first shock tube for animal blast injury experimentation during the second half of 1983. The technical parameters for said shock tube include experiment segment inner diameter of 1 m, overpressure of 2 kg/cm^2, and positive pressure effective duration of more than 20 ms. Shock wave front has to be steep, peak value has no plateau, and waveforms have to be regular. Other than technical requirements, a key demand was the low cost of production and construction.

Considering that blast effects are not only present in the positive pressure zone, especially since this shock tube is designed for biological blast injuries. Therefore negative pressure also can't be ignored. Thus, other than the aforesaid demands, negative pressure simulation was also required.

To satisfy the various needs stated above, replicating any existing equipment would not get the job done. It was necessary to pursue innovations in working principles in order to research and develop a new instrument with a simple structure, low technical requirements, and high performance.

3.1.2 Principles Behind Generation of Blast Wave

The gargantuan conical shock tube constructed by the U.S. Naval Ordnance Laboratory in the 1960s might have been the first instrument that could fully simulate blast wave and airflow upstream of a blast wave. It is a small piece of the explosion space. Other than influences from tube wall friction and reflection wave from the tube end, it is essentially a kind of on-site experiment. Due to the complexity in manufacturing conical tubes, compounded with the enormous size, said the equipment was expensive to build, and thus widespread adoption was unviable. In the 1970s, the Germans and French made changes to traditional conical shock tubes in two regards: First was changing the driven segment from cone to cylinder, and secondly was simplifying the conical driver segment into a tiered tube, and then further simplified to multiple cylindrical tubes of varying lengths. These improvements simplified the production process, but the last modification was marred by increased synchronized diaphragm rupture difficulties and waveform fluctuations. In a shock tube with a conical driver segment, when a shock wave formed upon the diaphragm rupturing, continuous rarefaction waves created from the conical wall of the driver segment would instantly catch up to the shock wave. The shock wave's strength (density) would gradually decay, and airflow pressure upstream of the shock wave would also gradually decline. This is the waveform of a blast wave.

For a common uniform cross-section shock tube, as long as the ratio between the lengths of the driven segment and driver segment reaches a certain value, the rarefaction wave reflecting off of the end of the driver segment would catch up to the shock wave. Flow behind said cross-section resembles flow in driver segment of conical shock tube and can similarly produce pressure waveform of a blast wave. Overpressure is highest at the spot where rarefaction wave catches up to shock wave. Since the said spot is quite a distance from the diaphragm, therefore, there is a certain positive pressure effective duration. Peak overpressure in the driver segment of a conical shock tube is located near the diaphragm. Thus positive pressure effective duration is short. The experiment segment is downstream, and positive pressure effective duration could be extended, but this would not facilitate sufficient use of peak pressure.

When gas pressure at the tail of the reflected rarefaction wave is lower than the initial pressure of the driven segment, a negative pressure zone would be created.

Blast wave waveform parameters including peak overpressure (ΔP_+), the greatest negative pressure (ΔP_-), driving pressure of driver gas (P_4), dimensionless length of a driven segment at the spot where reflected rarefaction wave catches up to shock wave, and positive pressure effective duration (T_+) could be calculated from shock tube theoretical equation. Details are omitted here due to the complexity of the calculation.

3.1.3 Other Technical Proposals

1. **Choice of driving gas**. Constructed explosion effect experiment shock tubes predominantly use explosive or combustible gas as a driver, with the use of compressed gas being a minority. Combustible gas has high sonic velocity and is easy to obtain strong blast waves or high peak pressure. The shortcoming is that it is an anoxic combustible gas and would combust again upon contact with the atmosphere. Combustion at the interface would disrupt the flow field, and combustion at the exit of exhaust would increase noise. For biological experiments, combustion would also burn and suffocate test animals. Compressed gas has low sonic velocity and is suitable for the creation of low- or medium-power overpressure. In addition, when other conditions remain the same, positive pressure, the effective duration is also relatively long. In accordance with the requirements of this equipment, compressed gas was chosen as the driver.

2. **Exit wave elimination proposal**. When the blast wave propagates to the exit at the end of the shock tube, if its reflection wave travels back into the experiment segment, the stress wave waveform would be disrupted. Secondly, when a relatively powerful blast wave travels outside the tube, it could damage the surrounding structures and impact the environment. As such, it was necessary to adopt some sort of wave reduction and elimination measures.

   The first proposal was wave elimination at the exit at the end of the tube. Based on the unsteady wave propagation features, a wave would reflect homogeneous waves from the closed end; compression wave or rarefaction wave would reflect heterogeneous wave from an opened end. If the exit was downsized to a certain area, the reflection wave could be reduced or even eliminated. The main function of this wave elimination method is to minimize the reflection wave's disadvantageous influence on the experiment zone. However, since the impact force at the exit is great, reflector structure at the end is cumbersome, it would be difficult to make adjustments. When experiment parameters change, wave elimination performance also differs quite markedly.

   Another wave elimination proposal was to install multi-layered hole boards. The kinetic energy of the reflection and vortex interference generated by a shock wave or blast wave propagating within would be gradually converted into thermal energy. This type of wave elimination proposal is very effective in decreasing strong waves, but not so much against weak waves. Another problem with this setup was the inconvenience in the placement of animals, which was why this proposal was not adopted.

   This third wave elimination proposal was to use hole tubes. When a shock wave or blast wave passes through a tube with holes, some of the gas will leak from the holes on the sides. Thus wave strength would gradually decrease. The wave would be discharged at the end exit when strength decays to an acceptable level. The device adopted this method, and the wave elimination performance was ideal, but sound suppression performance was unsatisfactory. When considering different proposals, perhaps decision-makers did not know that there were residential areas nearby, and so sound suppression did not become a priority.
3. **Tube movement problem**. Shock tubes generate massive recoil when used. If the tube is fixed to a foundation, then a substantial quantity of concrete would be needed. This equipment uses a flexible installation method. Since an open-tube and increased mass design was adopted, the device didn't move too much every time it operated. It is only necessary to make one adjustment after multiple uses.
4. **Variable cross-section tube**. Uniform cross-section tube with large caliber also requires membranes of large caliber and membrane potential holding machine of larger size. The larger the membrane potential holding machine, the higher the cost. The internal diameter of the driver segment was reduced to slash costs. Based on existing material, it was decided to create an internal diameter of 346 mm. With a smaller driver segment internal diameter, correspondingly driver pressure condition is lowered, and so too the strength of the shock wave created. Existing gas supply sources can satisfy demands. Construction costs and experiment expenditures could be lowered. In addition, the negative pressure of variable cross-sections could be increased during low-intensity experiments.

3.2 Research, Production, and Application of Serial Biological Shock Tube

Between 1985 and 1988, Zhengguo Wang et al. of the Third Military Medical University of Chinese P.L.A. (now Army Medical University) researched and produced the country's one and only series of large, medium-sized, and small (micro) biological shock tubes for blast injury research. A considerable amount of animal experiment results indicate that this set of devices can impart different levels of injuries, from a mild wound to on-site fatality, upon test animals like goat, dog, rabbit, guinea pig, and rat, and can even create localized injuries such as eye injuries. Thus, the system is an ideal instrument for undertaking blast injury research and experimentation. Related research, production, and application situations are as described below.

3.2.1 Research, Production, and Design Principles of Serial Biological Shock Tube

1. **Large biological shock tube (BST-I)**. The shock tube has a length of 39.0 m, comprised of components such as the driver segment, diffuser segment, transition segment, experiment segment, wave elimination segment and accessory equipment, gas compressor, and compressed gas cylinder. It has a dual-diaphragm structure. The driver segment has a length of 1.41 m and an internal diameter of 0.348 m. The diffuser segment has a length of 1.0 m and an internal diameter adjustable between 0.348 m and 1 m. The transition segment and experiment segment have a total length of 24.0 m, while the wave elimination segment has a length of 11 m and an internal diameter of 1 m. Experiment results indicate that when the end of the experiment segment is opened, the experiment segment can produce overpressure up to 0.219 MPa, positive pressure effective period of 32.7 ms, and negative pressure of 0.9 MPa. When the end of the experiment segment is closed, it can produce overpressure up to 0.63Mpa and

positive pressure effective period of 24.5 ms. Therefore, the device has the capability of simulating the blast waves from air explosion of TNT weighing from several dozen kilograms up to 6000 kg.

Design principles: A rarefaction wave reflecting off of the blocking board at the end of the driver segment is used to allow the rarefaction wave and shock wave travel in the same direction in the shock tube. When the rarefaction wave catches up to the shock wave, the shock wave's pressure would rapidly drop. When the pressure at the tail of the rarefaction wave falls below atmospheric pressure, a negative pressure phase would be achieved. Based on this circumstance, it would be possible to replicate blast waves with both positive pressure and negative pressure. In addition, by repositioning the movable blockboard to alter the length of the driver segment, the pressure inside the driver segment may be adjusted, including adjustments to the peak overpressure and peak negative pressure, as well as corresponding effective duration. This flexible system allows scientists to control experiment parameters. Using compressed gas (as opposed to explosive) as driver helps prevent poisoning test animals. Aluminum foils are used as diaphragms, and a dual-diaphragm tiered gas pressure bursting technique is utilized, which enables accurate control of diaphragm bursting pressure and prevents diaphragm fragments from injuring test animals. Movable boards at the end of the experiment segment allow for simulation of explosion injury conditions in both wide, open space and limited, enclosed space.

2. **Medium-sized biological shock tube (BST-II)**. The driver segment has five basic configurations with internal diameters of 77 mm, 100 mm, 200 mm, 350 mm, and 600 mm, and can respectively simulate high-altitude plateau blast wave, underwater blast wave, explosion-related decompression, high-speed airflow impact, and other effects and conditions. Overpressure can be set to between 2.52 and 650 kPa, and the positive pressure effective period can last anywhere from 0.2 to 2000 ms. It can also adjust injuring conditions at increments of ±1 dB and ±1 ms.

   Design principles: Rarefaction waves in the driver segment can catch up to the shock wave. This is a prerequisite for simulating the positive pressure effective duration of the blast wave from the detonation of a 10 kiloton nuclear weapon. The driver segment uses a sequenced connection combination method for a total of 12 different lengths, greatly improving the device's capacity and range of blast wave simulation. By changing the distance between the blocking board at the end and the cone segment at the cross-section location where the shock wave changes, scientists could create various time differences between the incident wave and reflection wave. To simulate how two blast waves act differently on a person located at different distances from a reflector wall, this device features a closed design: There are negative pressure, positive pressure controllers, and water and air supply systems installed at the top of Chamber A and Chamber B. Prior to bursting the diaphragm, the pressure inside the chambers could be maintained at a low-pressure level to simulate the conditions of high-altitude plateau battlefields or mid-air explosions. If high pressure was built up in the chambers, or the water-based compression method was used, then the device could simulate underwater explosion conditions, such as those encountered by scuba divers. After causing injury, Chamber A or Chamber B, where test animals are located, could quickly return to pre-injury conditions so as to raise the realism of the simulation. The end of the driver segment is a closed-end (with the other end open) with a semi-spherical shape, which can prevent rarefaction waves from reflecting synchronously, thus allowing for the generation of blast wave waveform with an effective duration of up to 100 ms even inside a relatively short device. If the test subjects are placed at the end of the test section, scientists could study the effects of focused waves and augment injury severity.

3. **Small (micro) biological shock tube (BST-III)**. The shock tube has a total length of 0.5 m and a designed pressure load of 68.6 MPa. The experiment segment has nine cross-sections with internal diameters ranging from 2 to 10 mm and could produce peak overpressure and positive pressure effective duration, respectively, ranging between 26.8 and 477.0 kPa, and between 0.062 and 16.8 ms. Said equipment is used to produce point explosion blast waves and allows for exposure to blast waves at specific distances, with specific areas and positions.

   Design principles: Blast wave is obtained as shock wave propagates to the mouth of the tube and then rapidly rarifies in the air. To enhance the capacity of producing blast waves of different strengths, steel fillers could be placed to change the volume or pressure of the driver segment. The strength of the blast wave that acts on the test animals could be adjusted by changing the distance between the tube opening and the position of the animals. In addition, by adjusting the diameter of the holes on the protective cover, scientists could study localized blast injuries to specific positions and with specified areas. An adjustable tube rack allows scientists to change the direction of a shock wave, adding more convenience and flexibility in experiment design. Said device permits the use of measurement instruments for pressure, acceleration, displacement, and other aspects, allowing scientists to record the dynamic responses of the test subjects at the instant of blast wave action.

3.2.2 Biological Experiments

Ever since their completion, the serial biological shock tubes have so far conducted experiments on blast injuries to the whole body or localized regions (eyes, ears, head, chest, and abdomen) of 1459 test animals, including 757 rats, 105 guinea pigs, 335 rabbits, 240 dogs, and 22 goats. Test results indicate that this set of equipment can cause various degrees of injuries, from mild injury to the auditory apparatus to instant death. When the blocking board is closed, overpressure-induced injury experiments can be done, and when the board is opened, experiments on injuries caused by both overpressure and dynamic pressure may be performed. Selected portions of animal testing results are reported below.

1. **Animal experimentation based on the BST-I shock tube**. BST-I shock tube has been used to cause injuries to 52 adult male mongrel dogs weighing (12.3 ± 1.8) kg. Of these 52 test dogs, seven died within 5 min of injury, and of these fatalities, four died because of severe pulmonary hemorrhage and edema, two died from internal bleeding caused by ruptured liver or spleen, and one died from a coronary embolism.
2. **Animal experimentation based on the BST-II shock tube**. Fifty domestic rabbits were divided into five groups of ten. Bandages were wrapped around the thoracic cage of animals in groups one and three in order to limit the expansion of the thorax after shock wave action for the purpose of limiting lung injury. Animals in groups two and four were injury controls, and those in group five were normal controls. Test results indicate that the bandaged animals in groups one and three had significantly smaller areas of lung hemorrhage compared to unbandaged animals in groups two and four ($P < 0.05$).
3. **Animal experimentation based on the BST-III shock tube.**
   (a) Blast injuries to the eyes. Sixty rabbits were placed 4 cm and 2 cm from the opening of the tube, and the eyes of the rabbits were exposed to the tube either straight on or from the side. Respective shock waves with peak overpressures of (477.0 ± 42.4) kPa and (236.7 ± 22.0) kPa, and positive pressure effective duration of (8.2 ± 0.3) ms and (0.062 ± 0.23) ms were applied to cause eye injury. After an injury, contracted pupil was observed in 97.5% of subjects, increased intraocular pressure was observed in 80% of subjects, contralateral eye cases were respectively 22.5% and 35%, and differences between the two eyes were significant ($P < 0.05$). Pathological inspection discovered injuries to the cornea, lens, retina, and tunicae media oculi. Ruptured eyeball and separation of the visual pathway were seen in some of the more severe cases, and even eyeballs that were ejected from the body. A note worth mentioning is that in some of the lens and retina injury cases, no obvious change was observed in the subjects' cornea or anterior chamber.
   (b) Localized blast injuries to chest and abdomen: Fifty rats weighing (234 ± 25) g were used as test animals. The rats were divided into five groups, with those in groups one and three subjected to a chest injury, groups two and four subjected to abdomen injury, and group five was the control group. Exposure distance for animals in groups one and two was 15 mm, and that for groups three and four was 135 mm. Experiment results indicate that when the chest was exposed, only chest injury occurred (pulmonary hemorrhage), and no organs in the abdomen were wounded. Meanwhile, when the abdomen was exposed, organs in both the chest and abdomen were injured, which might be attributed to sudden uplift of the diaphragm when pressure acted on the abdomen.

3.3 Others

When carrying out empirical experiments related to blast injuries, the explosion caused by an explosive was the preferred method for tests in the past. Although this method is closer to real-world situations, it is difficult to acquire accurate test data and also quite challenging to conduct functional and other inspections of the animals early on. In addition, the injuries caused are not consistent and are tough to replicate. Meanwhile, injuries caused by shock tubes can quite sufficiently circumvent the aforesaid shortcomings.

The USA and Sweden respectively researched and produced small biological shock tubes for use in laboratories in the 1950s. These devices have an internal diameter of 0.1 m and lengths ranging from 1 m to several meters. They consist of driver segment and experiment segment, separated by a film in between. These instruments were used to carry out studies related to topics such as the relationship between pressure value and mouse death rate.

After the 1960s, American scientists such as Richmond et al. researched and produced five types of large or medium-sized biological shock tubes. These instruments were used to research how various peak pressure conditions and positive pressure effective duration conditions injure or kill different animals, and the results were projected for effects on the human body.

In 1987, American scientists Jaffin et al. designed a micro shock wave generator. Its driver segment has a volume of 15 mL and can withstand pressure from 10 to 25 MPa. One or several aluminum foils 0.36 mm in thickness could be used as the diaphragm(s), ruptured using the natural compressed gas pressure bursting method. This device could conduct tests on small animals.

The shock tubes developed by Zhengguo Wang et al. of the Third Military Medical University of Chinese P.L.A. were improved upon based on the works of previous scientists, with the main innovations manifested in (a) Large, medium-sized and small models could all be used in the same laboratory, thus basically achieving the serialization of the shock tube instrument. (b) The ingenious design principles (for instance, the BST-I shock tube uses a blocking board at the end of the driver segment and enables reflected rarefaction wave to catch up to shock wave, and when the pressure at the tail of rarefaction wave is lower than atmospheric pressure, the device could create negative pressure wave) allow for typical blast wave waveforms. Most shock tubes overseas can't produce typical negative pressure waves, and therefore their simulation capacities are relatively poor in this regard. (c) The dual-diaphragm structure permits better control of pressure. (d) The BST-I shock tube has a multifunctional combination design and could simulate high-altitude plateau blast wave, underwater blast wave, explosion-related decompression, high-speed airflow impact, and other effects and conditions. (e) The shock tube system could carry out a wide range of tests because of its flexible design and numerous accessories. It could induce full-body injury to animals large or small (goat, dog, rabbit, mouse, etc.) and could also produce localized wounds; it could use overpressure to injure, and use both overpressure and dynamic pressure to wound; it could cause mild injuries and could kill instantaneously. In light of the above, one may conclude that the aforesaid series of biological shock tubes are advanced and relatively capable of meeting the needs in various blast injury research and experiment at high levels.

Approximately since the 1950s, the shock tube has developed into an experimental instrument with numerous purposes and functions. The shock tube has become an effective tool for studies about the fundamental theories and applications in fields such as aerodynamics, hypersonics aerodynamics, chemical kinetics, physical mechanics (in particular high-temperature physics), explosion dynamics, condensation effect, chemical fluid dynamics, electromagnetic fluid dynamics, cryomechanics, astrophysics, and other related disciplines. It has risen as a tool of choice for researchers engaged in areas like aeronautics, astronautics, physics, chemistry, astrology, blasting, defense engineering, environment protection (particularly noise pollution), and measurements. At the same time, due to the low cost and investment for construction and operation, it is also welcomed by institutions of higher education and research institutes. However, it is still a weak link in China and requires additional attention and more substantive support.

Biological shock tube refers to those that are specially dedicated to or primarily used for animal testing. The shock waves created by this kind of device can simulate the blast waves produced in the detonation of nuclear weapons or the explosion of conventional explosives. Laboratory at the Third Military Medical University of Chinese P.L.A. has already completed the construction of a series of biological shock tubes. The devices have been configured in nearly a hundred different combinations to perform more than a thousand explosion simulation experiments on rats, guinea pigs, rabbits, dogs, and sheep. Test results indicate that the varieties and capabilities of shock tubes can basically meet biological research demands, and the instrument has emphatically fueled the development of the shock wave dynamics discipline and its collaboration with other disciplines.

It is worth mentioning that foreign nations have used shock tubes to study the injury effects of blast waves for more than four decades. However, this is still a relatively new pathway in China, and there is much to do in terms of both theoretical and empirical efforts. More in-depth discussions about the theories and design features of shock tubes for biological experiments are highly crucial for blast wave research. Such endeavors will also be beneficial to fostering progress in disciplines like mechanics, biomechanics and biomedicine, and strengthening the cooperation and exchanges between them.

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Defense Against Blast Injury

Ce Yang

Without a doubt, providing effective protection against blast injury is much more beneficial than administering effective rescue and treatment. According to nearly a decade of data from the U.S. Army Institute of Surgical Research (USAISR), a total of 4596 American soldiers died on the battlefields of Iraq and Afghanistan, and 73.7% of these casualties arose from blast injury. 87.3% of victims died before arriving at a medical establishment, and those fortunate enough to survive on the road to hospital are usually afflicted by a combination of wounds. Existing medical care techniques are often incapable of dealing with such complexities. In a recent period of more than a decade (January 2000 to April 2017) in China, production and work-related explosions reported by the State Administration of Work Safety alone totaled 2013 cases, injuring or killing 26,019 victims, and averaging one explosion every 3 days. Therefore, attempts to reduce the fatality rate and disability rate of blast injury, and to improve post-injury recovery of the wounded, ought to underscore protection. Of which, most focus should be placed on shock wave as the main cause of blast injury. During the propagation process of the tremendous energy of a blast wave, the key to effective protection is how to avoid or reduce the human body's rapid absorption of the load imparted by the shock wave itself and its encirclement. Using traditional Chinese philosophy to analyze the matter, countermeasure should focus on using softness to counter hardness, "mutual overcoming" of different phases and elements, maximize advantage and avoid disadvantages, and prevent problems from occurrence. This mentality is equally valuable in terms of defense against blast injury. This section will use blast injury treatment as point of view to contemplate about the status quo and practical needs in defense against blast injury. Looking into status quo of the research and development of blast injury protection materials and equipment, this section will also discuss precision and suitability of blast injury protection strategies. The aim of this section is to provide references and revelations conducive to blast injury protection endeavors in China.

1 History and Status Quo of Blast Injury Protection

Blast injury protection is inseparable from gunpowder, one of the Four Great Inventions of ancient China. Gunpowder was invented by the Chinese as far back as the early stage of the Western Han dynasty, and various explosives based on the gunpowder as main content constitute the earliest source of blast injury. In the several thousand years of subsequent history, explosions never stopped due to reasons both related and unrelated to warfare. Humankind's concept of blast injury protection arose from the fear and grievance of explosion-induced injury and death. People learned to take precautions, such as isolating or covering combustible and explosive substances in venues where explosion might occur. Shields, helmets, armors, and such protective gear used in battles and conflicts became the earliest blast injury protection equipment. Therefore, the development of the notion of blast injury protection began with the inclination to avoid injury or death and people's survival instinct, which arose from behaviors during warfare and skirmishes such as being aware of explosions, staying away from explosions, shielding oneself, and using defensive and protective tools.

In modern history, especially after Alfred Nobel invented the dynamite, explosive has become the main content in high-energy weaponry and production-related blasting and has found widespread utility in aspects from worldwide and regional warfare and terrorism to industrial production. At the same time, of the injuries and deaths from explosive and gas explosions in mining, blast injury constitutes a substantial portion. Yet, limitations in scientific and technological progress have hampered advancements in blast injury protection, and even the injuring mechanisms behind explosions remain unclear, while no effective breakthroughs have been effectively achieved in protective equipment or materials.

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Therefore, in the turbulent course of modern social development, limited shielding, proactive isolation, and passive avoidance are still the predominant yet passive measures to defend against blast injuries.

Since the end of World War II, in particular in the past half a century or so, although no large-scale warfare has erupted on a global scale, still regional conflicts, blasting operation, terrorist attacks, and explosions of hazardous substances have led to serious injuries and deaths because victims were often unprotected or could not find protection in time. At present, several countries have developed their individual nuclear arsenal. The detonation of a nuclear weapon produces powerful impact and vibration that can seriously damage subterranean works several hundred meters beneath the ground surface. People near the center of explosion have a slim chance of survival, but those far away from explosion center differ in their injury severity because of different conditions, making them the target for protective measures. In the past decade, serious explosions have occurred with increasing frequency both in China and abroad. The majority of blast injury remedy is focused on elevating treatment standard, although scholars have actively undertaken studies in blast injury protection, and a raft of data have been accumulated through research and experimentation. People are gradually realizing that when an explosion takes place, different environmental conditions affect the injury severity of target organs. Due to the complexity in the simultaneous existence of overpressure, underpressure, dynamic pressure, noise, vibration, and other factors, injury severity vary drastically. Therefore, to protect against blast injury is in essence an exploration of effective measures that can convert and dissipate energy from the force of an explosion. Past methods include anhydrite sheath, rubber garment, cotton garment and earplugs, among numerous other examples. In accordance with the people-first philosophy, the construction of fortifications should have already fully accounted for shockproof performance for the benefits of personnel and should aim to effectively avoid personnel injury and fatality. At the same time, research efforts related to protective sites and body positioning conducive to protection have been undertaken. In addition, based on experiment, research, and digital simulation, notable feats have been achieved in advancing theoretical understanding and in-depth exploration about protective material, protective equipment, and protection strategy. In particular, the various types of shock wave isolation and energy dissipation measures proposed in recent years including protection principles such as lowering peak pressure, filling wave troughs, conversion of dynamic pressure into inertia and transforming great energy into lesser energy, further strengthened sense of protection and revolutions in protective materials, as well as enhancement in protection methods. Without a doubt, these advancements are immensely valuable from both a theoretical and practical standpoint since it would be impossible to completely prevent explosions in any foreseeable future.

2 Key Factors in Blast Injury Protection

Blast wave can injure or kill people either directly or indirectly. The so-called direct injury and fatality refers to people that are injured or killed due to effects (overpressure, underpressure, dynamic pressure, noise, shock) of the shock wave (Table 1). Indirect injury and fatality meanwhile refers to mechanical trauma caused by structures and objects that collapse when struck by shock wave, or different projectiles like weapon, sandstone, brick, tile, and glass fragment that are launched by the force of the shock wave. Moreover, an explosion is often accompanied by heat sources or chemical sources that can cause injury. These factors compound and worsen the injury and lethal effects of the shock wave itself, thus they can't be ignored and constitute key factors that demand corresponding protection measures. To sum up, to prevent or reduce injuries caused by shock wave, it is necessary to adopt effective measures in order to ensure that overpressure and overpressure duration of a shock wave from a certain distance away can be reduced to below the safety threshold of the human body.

Clinical experience pertaining to blast injury in recent years indicates that blast injury victims not related to warfare have many different characteristics, and injuries are

Table 1 Physical parameters related to blast injury protection

Physical parameters	Concept	Protection target
Overpressure	Pressure above normal atmospheric pressure within the shock wave's compression zone, no directionality	Reduce
Underpressure	Pressure below normal atmospheric pressure within the shock wave's rarefaction zone, no directionality	Increase
Dynamic pressure	Impact force created by high-speed air movement during the shock wave's movement, specific direction	Reduce
Noise	Unharmonious sounds generated by the discordant changes in sound pitch and intensity during an explosion	Reduce
Vibration	One or several intermittent vibration(s) within a short period generated by an explosion	Reduce
Fragment	Fragments or shrapnel moving at high speed because of forces generated by an explosion	Block
Heat source	High temperature energy generated by an explosion and manifested in air flow or fragments	Isolate
Impulse	Product of shock wave pressure and time	Reduce

often very complicated due to a bevy of factors involved (combination of fragments, burns, collision with other objects, crushing injury, etc.), making treatment very difficult and elongating recovery period. The root of the problem is that when the body is exposed during an explosion without the ability to control the quantity of explosive material, the body lacks effective and comprehensive protection measures. For the small number of victims that did have protection measures during the explosion, their injury status might be hard to assess quantitatively after the explosion because of the lack of quantitative evaluation parameters (e.g., blast wave overpressure, pressure duration, cumulative explosion frequency) about exposure to shock wave. Therefore, how to ensure active and effective blast injury protection and how to acquire personalized parameters pertaining to exposure to blast wave are key topics in blast injury protection.

In light of the above, it could be said that blast injury protection involves the three aspects of physical, chemical, and biological injuring factors. In terms of the universality and urgency in protection, it is necessary to earnestly ensure protection against shock wave overpressure and dynamic pressure, protection against fragments, protection against mechanical crushing and collision injuries, protection against heat source, toxic chemicals, radiation, etc., in that order. Upon satisfying the basic protection requirements outlined above, further thoughts should be dedicated to self-restoration of protective material, intelligent warning, thermal isolation and flame retardation, protection against bacterium and toxins, resistance against radiation, corrosion tolerance, etc. Lastly, after satisfying the aforementioned performance foundations, the wearability of protective materials and protective equipment, such as weight and comfort, should also be taken into full consideration.

3 Blast Injury Protection of Organs

3.1 Easily Injured Organs

It is extremely common for organs to be injured by the blast wave. A consensus in the past is that when the body is exposed to a blast wave, air-filled organs (lungs, gastrointestinal tracts, etc.) are the most easily wounded because of the fact that their connection with the air and air content make them vulnerable to injuries caused by air pressure. Therefore, a huge number of experiment results have indicated that air-filled organs are the primary focus of concern in blast injury protection. Second of all, auditory apparatus and the eyes have contact with the air, which also make them easily wounded target organs. Thirdly, due to the physical complexities of blast injury, and the heterogeneity in the human body, along with the fact that shock waves are often accompanied by other injuring factors, therefore, solid organ injuries are not uncommon. A large amount of studies have proved that blast wave might create air pockets or micro bubbles in the anatomically complicated structure of the brain, and these might cause actual damage to the brain parenchyma under the effects of mechanisms such as implosion. At the same time, brain displacement and other craniocerebral injuries arising from the person being thrown or colliding with an object due to the shock wave's dynamic pressure will further exacerbate craniocerebral injury. Therefore, the brain is one main target for primary and secondary blast injuries. Injuries to other solid organs such as the spleen, liver, kidney, adrenal gland, thymus, heart, and eye have also been reported on numerous occasions, and therefore, the key areas for protection against exposure to shock wave ought to be the head, chest, and abdomen. With regard to protection for the auditory apparatus, eyes and limbs, injuries to these body parts are not lethal, therefore the proper use of effective protective materials can also achieve the desired results.

3.2 Defense Against Blast-Induced Traumatic Brain Injury

At present, the majority of studies about injuries due to exposure of the brain to shock wave largely focus on forced or passive hospitalization and treatment after injuries or psychological symptoms are discovered in victims. With regard to how much shock wave exposure occurred during a victim's craniocerebral injuring process, especially with regard to blast injury victims with no visible wound but that exhibit brain function disorder, how to carry out quantitative assessment of blast-induced traumatic brain injury demands more attention. After an explosion occurs, at present medical care institutions are unable to obtain dynamic injury data regarding blast-induced traumatic brain injury. Therefore, medical personnel can't make judgments about dose–effect and time–effect relationship between traumatic condition and pressure, particularly in cases of repeated exposures to low-intensity shock waves. It is quite difficult to make blast-induced traumatic brain injury judgments and administer relevant treatments in a highly personalized manner. In light of this situation, other than exploring effective protection measures for both peacetime and wartime use, it would be highly valuable in terms of diagnostics to study and develop the "black box" around the brain. Such endeavor would be helpful to conduct accurate assessment of the intensity and frequency of shock wave(s) to which a victim was exposed to, which are crucial information that underpin objective evaluation and prognosis of blast-induced traumatic brain injury, improvement to psychological condition, and promotion of brain function recovery.

At present, blast-induced traumatic brain injury assessment involves changes of physiological indices of brain function and the corresponding scoring methods such as the Glasgow Coma Scale. Of which, physiological abnormalities primarily involve changes in consciousness, memory, and mental status after injury, and/or focal nerve damage. The chief complaints put forth by victims are often more complicated and varying than clinical diagnostic indices, especially when clinical imagery diagnosis shows no abnormality. However, neuropsychiatric symptoms might have already appeared and continue to exist, with serious cases resulting in self-mutilation or injuring others. Looking at the root of the problem, other than stimulation in specific scenarios (i.e., injury and fatality of troops on battlefield), at present most argue that cumulative exposure to frequent or a certain intensity of shock wave is extremely likely the main reason for pathological changes in brain function. Hence, before a victim exhibits craniocerebral pathological changes clinically, dynamic monitoring of frequency and intensity of shock wave(s) to which a victim's brain was exposed to is a prerequisite for blast-induced traumatic brain injury assessment and protection in both peacetime and wartime. Current shock wave monitoring chiefly utilizes imported equipment, and main restrictions include: Monitoring is focused mostly on blast waves related to semi-static facilities and fixed animals; devices are cumbersome and not easy to carry around; do not have strong interference-resistance; large deviation between the measurement range of blast wave and the shock wave exposure threshold of the survivors; and uncertainty about location of monitoring instrument (i.e., whether near the body, which part of the body) when the shock wave strikes the brain of injured individual (civilian, worker). Therefore, monitoring data might exhibit significant bias due to the difference in medium between the shock wave and the device, making it difficult to objectively reflect the intensity of the pressure imparted upon the brain of the victim. In addition, research data indicates that 10–50% of patients with traumatic brain injury also have some sort of eye disease. Blurred vision, insensitivity, diplopia, sore eyes, difficulty reading, headache, tunnel vision, and other symptoms have been reported in such patients. Data from FG.Hirsch shows that when shock wave's overpressure reaches 34 kPa, rapidly and slowly elevated shock wave could respectively cause tympanic membrane injury nearly 96% and 65% of the time.

Therefore, considering actual situations at explosion site or excavation blasting site, dynamic monitoring device of brain shock wave exposure must meet the following requirements: First of all, brain protection gears (such as helmet) are compatible with monitoring instrument, as the said instrument can fit inside helmet; second of all, monitoring instrument must be small enough to be easily carried around or installed inside the brain protection gear; third of all, it can monitor shock wave impact to the brain with waterproof and fire-resistance properties; fourth of all, it can withstand blast waves with different pressure intensities and in different closed and open environments; fifth of all, the design of brain protection gear (such as helmet) should extend to cover the eyes, ears, and cheeks, and an ideal protective equipment should not obstruct operational performance or combat effectiveness while providing full protection for the entire face of the wearer.

3.3 Chest and Abdomen Blast Injury Protection

For protection against blast injury to the chest and abdomen, protective equipment are similar to those designed for protecting the head, and attention must be paid to early warning ability. In addition, focus should also be placed on the area, material, accessory, and other aspects of the equipment.

On the one hand, considering that the lungs are the most frequently exposed target organs in shock wave injury, chest strap devices are often used in research. To explore how different devices affect blast injuries to the lungs, chest straps are used to restrict expansion of the thoracic cage, or air bag might be utilized to limit thoracic expansion and attempt to lessen air expansion in the lungs. The chest strap group wrapped straps around the chests of test animals. Chest strap used was fabricated from high-strength and low-elasticity nylon cloth, and weaved into a fan-shape to fit the shape of the thoracic cage, which has a narrower top and wider bottom. Bottom edge of chest strap is in line with the costal margin. It was discovered through the experiments that lung surface bleeding area and lung body index (lung wet weight/body weight) of both the chest strap group and/or air bag protection group were significantly lowered than those in the injury group, with protective effects in the air bag protection group being the most ideal. These results indicate that chest strap can restrict the inertia of the thoracic cage's excessive outward movement and lessen the lung tissues' tensile strain, which can significantly reduce blast injury to the lungs, and further disapprove of the importance of the "drag effect" in the mechanism behind lung blast injury. At the same time, in light of the outcomes described above, lung blast injury protection equipment principles are proposed below:

1. Use high-strength, low-elasticity and soft materials to maintain a certain degree of tension.
2. Soft protection is preferred, and those with gas as main content provides relatively sound protection performance.
3. Bottom edge of thorax is a key area for protection for the purpose of effectively limiting expansion of the thoracic

cage. Therefore, protective gear should not be excessively wide and may take the form of bands.

On the other hand, researchers have discovered that the diaphragm clearly conducts stress upon the chest cavity above when the abdomen is pressed by the pressure of a shock wave when the person is exposed, if the person is wearing chest protection equipment, there would be stress wave that travels and spreads from the abdomen cavity to the chest cavity, which would be highly detrimental to the lungs. Therefore, to ensure the safety of blasters, developing chest & abdomen protective equipment would be more effective than ones that protect only the chest (such as chest strap). Since blast-induced traumatic brain injury mechanisms are not yet fully understood today, when a person is exposed to a shock wave, whether or not shock wave pressure to the chest and abdomen causes stress damage to the brain via specific pathways (circulatory system or body cavities) remains unclear and demands more exploration. Analyzing from the point of view of treatment however, the chest and abdomen ought to be protected as a whole, and protective equipment for either part only might provide protection at the cost of the other part, thus lowering the overall effectiveness of shock wave protection. At the same time, considerations should be taken to meet clinical treatment needs. Since conscious disturbance has been observed in a certain portion of those injured due to exposure to shock wave, it is challenging or even impossible to provide basic vital information and actual conditions required for emergency rescue (e.g., blood type, weight, age, allergy), in terms of the development of intelligent protective equipment (such as the use of flexible and wearable health sensor), other than overcoming existing static configuration environment restrictions in shock wave pressure and time monitoring, and improving precision of and sensitivity to craniocerebral data responses in specific areas when struck by a blast wave, the memory chip used could contain the wearer's basic information as described above, so that medical personnel can obtain information necessary for diagnosis during emergency rescue as soon as possible because every second counts.

4 Blast Injury Protection Materials

In the research and development of blast injury protection equipment, not only is it necessary to select effective protection materials, especially composite materials that can defend against shock waves with high peak pressure, short effective duration and wide bandwidth, it is also a must to create a scientific protection structure based on combination of materials. Elements to consider include material thickness, order of placement and method of composition, among others. The core purpose is to highly efficiently control the transfer of stress wave produced by the direct penetration of a projectile and the overpressure of a blast wave, so that the energy they carry can be absorbed and dissipated within protection layers with limited thickness and mass. This is the crux to minimizing the transfer of shock wave energy to the wearer's body.

4.1 Material Composition

Blast injury protection material acts as a medium placed between the human body and the shock wave. An ideal blast injury protection material should be able to defend against or weaken the shock wave's direct effect on the human body. Such material should provide protection through effective dispersion of blast loading; protect through absorbing energy generated from impact and related energy products (fragments, heat source, etc.); considering the safety and comfort of protection material on the human body, the material should transfer as little stress to the wearer's body as possible when struck by a blast wave; and protection equipment fabricated from the protective material should not interfere with the wearer's performance. Therefore, selecting the best blast injury protection material that meets the requirements stated above is a scientific question, which has garnered the sustained interest in fields including medicine, material science, and biomechanics.

Early studies have shown that materials like plaster and plastic have some degree of protection against air blast injury. Thereafter, artificial leather, foamed plastic, rubber, nickel foam, aluminum foam (ALF), polyurethanes, polyurea, polycarbamate, and other materials have shown some impact-resistant and pressure-reduction properties against shock waves and could lower the death rate of test animals. Phillips et al. used Kevlar bulletproof vest in shock wave protection testing, and the outcome showed that not only bulletproof vest offered no protection, it actually worsened blast injury (Table 2).

Researchers also discovered that foam materials constitute a major group of ideal candidates for blast injury protection. Philip A, a Canadian scholar, conducted simulation analysis and research on strained condition of helmeted head under various explosion circumstances. The "helmet–foam vibration absorber layer–head" model was developed. Through analysis of the parameters of different foam materials, the conclusion is that aluminum foam is an ideal vibration absorber because it has relatively low density coupled with a certain degree of strength, in addition to its unique foam structure capable of absorbing a considerable amount of energy upon impact. High-density aluminum foam boasts a higher shock wave decay coefficient than low-density aluminum foam. A downside is that aluminum foam affects the

Table 2 Comparison of performance of blast injury protection materials

Blast injury protection material	Chemical composition	Protection performance		
		Shock wave	Fragment	Heat source
Plaster	Calcium sulfate hydrate	Moderate	Moderate	Excellent
Artificial leather	Fabric base coated with synthetic resin and various plastic additives	Poor	Poor	Poor
Cotton	Fibrin 87–90%, water 5–8%, other materials 4–6%	Moderate	Good/ moderate	Poor
Plastic	Resin and additive	Good/moderate/ poor	Good	Poor
Nickel foam	Nickel	Good	Moderate	Poor
Aluminum foam	Aluminum	Excellent/good	Good	Poor
Rubber	Natural rubber and synthetic rubber	Good	Moderate	Poor
Polyurethane	Synthesized with either diisocyanate or polyisocyanate, and a dihydroxy or polyhydroxy compound	Excellent/good	Excellent/ good	Excellent/ good
Polyurea	Synthesized with isocyanate and amine	Excellent	Excellent/ good	Excellent/ good
Kevlar	Poly (*p*-phenylene terephthalamide)	Poor	Excellent	Good
Glass fiber	Silicon dioxide, aluminum oxide, calcium oxide, boron oxide, magnesium oxide, sodium oxide, etc.	Good	Poor	Excellent
Zeolite	Synthetic zeolite: Sulfonated polystyrene Natural zeolite: Sodium aluminosilicate	Excellent	–	–
Water packs	Water	Excellent	–	Poor

comfort of the equipment. Moreover, researchers at the U.S. Army Natick Soldier Systems Center carried out studies on the performance of various types of foam materials under dynamic compressions at relatively high strain rates and proved that different foams exhibit different energy absorption characteristics when dealing with different shock wave waveforms. Foams with specific structure might demonstrate the best and optimal energy absorption and energy dissipation capacities against certain shock wave waveforms, but might perform much less ideally against other waveforms, or even accelerate the rate at which the thoracic wall deforms, worsening blast injury severity. Similarly, the U.S. Naval Research Laboratory undertook studies about the energy dissipation capacities of polymers chiefly consisting of polyurea/polyurethane elastomer and arrived at similar conclusion. Therefore, the non-universal revelations from the shock wave-resistance capacities of the materials mentioned above indicate that restructuring or reengineering of basic material (such as polymers with multi-structure phase), addition of supplemental materials, or combinations with other materials might be helpful to augmenting a material's protective performance against the overpressure from a broad band of shock waves.

In recent years, the selection and performance of composite materials (such as nickel foam, aluminum alloy-sponge combination, polyurea-polyurethane, polyurea-elastic fiber, polyurea-glass fiber, zeolite absorber/micro-nanofluidic technology, etc.) in capturing shock waves have garnered widespread interest, and has emerged as a mainstream in studies pertaining to blast wave protection. Especially worth noting is that US military researchers have discovered that polyurea is an ideal basic material for use in shock wave defense. Some years back, the U.S. Air Force once carried out an experiment by wrapping a structure's wall with polyurea several millimeters thick to test anti-fragment capacity. The Research Institute of Field Surgery of the Third Military Medical University used biological shock tubes for experiments and proved that polyurea materials offered sound protection against severe lung blast injuries in rats (Fig. 1).

Under identical explosion load, polyurea elastomer interlayer outperformed protection materials with no interlayer or rubber interlayer in terms for both deformation and energy absorption. Chemical structure analysis reveals that polyurea is a segmented copolymer with glass transition temperature (Tg) or −60 to 50 °C, which can be easily applied on the surface of metals or other materials by spraying and its mechanical properties are affected by temperature, stress and strain rate. Polyurea-clad material's impact resistance involves transformation from a rubber-like state into glass-like state under high deformation rate loading conditions, as well as shock impedance mismatch, strain delocalization, rupture mode transformation, and other mechanisms. At present, mainstream opinion is that polyurea-clad material's impact resistance mechanisms involve two aspects, namely shock wave dissipation and ballistic protection. Of which, shock wave dissipation is mainly contingent on control of the hard segment of polyurea, including the following aspects. (1) Shock wave-induced hard segment orderliness, extent of impact reduction, and proportionality of hard segment volume ratio. (2) Shock wave-induced crystallization/densification of hard segment. This kind of microstructural change allows for the absorption and dissipation of shock wave's

Peak pressure (kPa)	Duration (ms)	Impulse (kPa.ms)	TNT equivalent (Kg)	Equivalent explosion center distance (m)
477.5±8.9	44.3±2.1	5684.5±315.9	4436.9±631.74	65.7±3.0

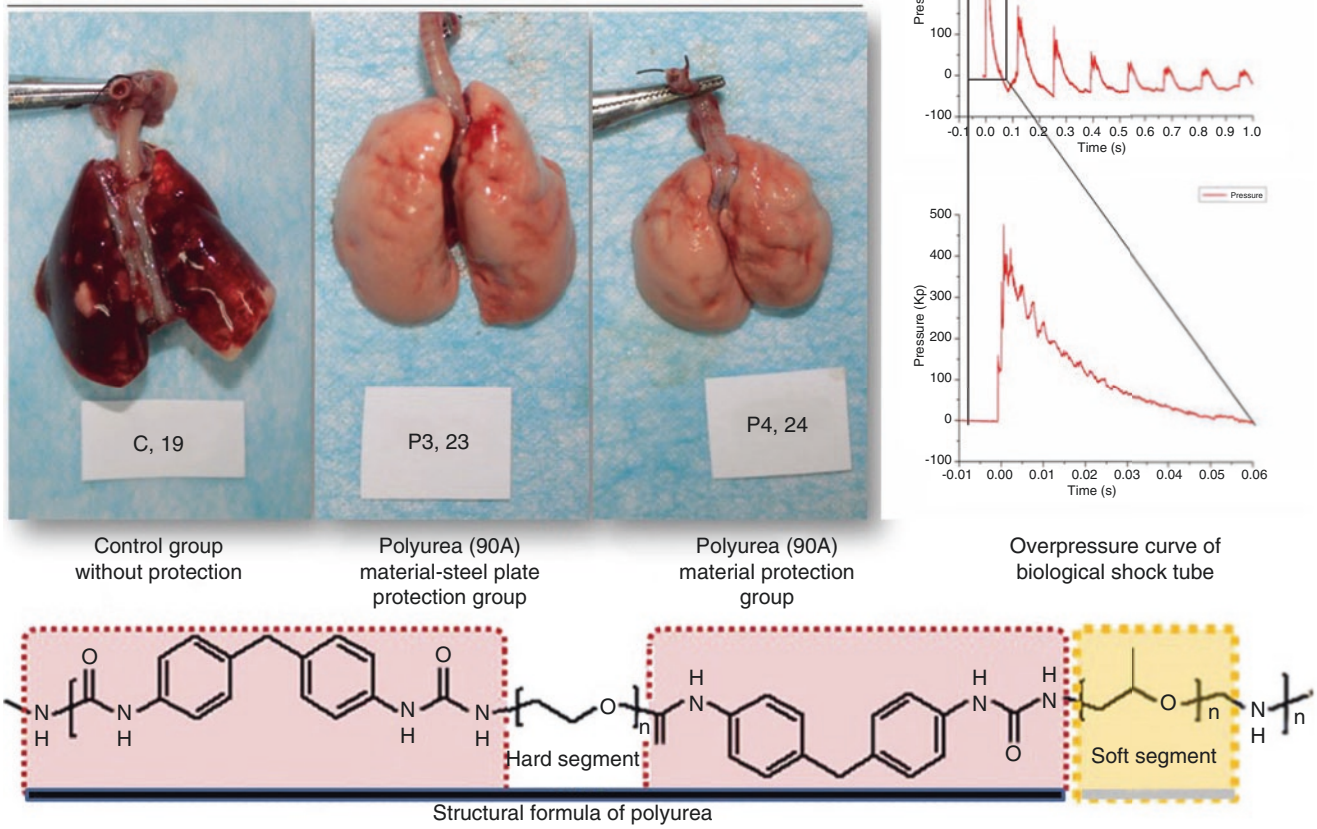

Fig. 1 Protective effect of polyurea material on the lung blast injury caused by biological shock tube in rats

kinetic energy. (3) Shock wave-induced hydrogen bond breakage. This refers to breakage and rearrangement of microstructural phase-bidentate bond between the urea linkages, in turn forming more hydrogen bonds in the hard segment favorable to absorption and dissipation of shock wave's kinetic energy. (4) Relaxation of viscoelastic stress at the interface of substrates between the hard and soft segment. In terms of shock wave's capture and neutralization, the point of view of ballistic protection mechanism holds that soft substrates play the central role. The key here is to instigate polyurea to transform from a rubber-like state into glass-like state under high strain rate conditions in order to dissipate the ballistic energy. Furthermore, Huang Weibo et al. used comparative studies to prove that the mechanical properties (tensile strength and elongation rate before breakage) of structurally stable polyurea are significantly better than polyurethane. Scanning electron microscope shows that the microstructure of polyurea is denser than that of polyurethane. Said type of polymer material is characterized by high compression-resistance, low tensile strength, and high energy dissipation capacity, and has bat-shaped yield locus and tensile gap yield. The addition of other materials could change the material's stretching gaps, such as rubber particle inclusions, which would stabilize existing gaps, prevent formation of cracks and dramatically strengthen the material's toughness and impact resistance. At the same time, chemical modification and nano restructuring could substantially augment the mechanical and physical performances of said type of polymer, and make the material especially suitable for redirecting, capturing, and dissipating blast waves in a multitude of frequency ranges. At present, China has already developed mature polyurea spraying technology, which functions as a solid foundation for modification and restructuring of materials.

Moreover, scientists at the Massachusetts Institute of Technology studied and developed a series of new materials: Polyurethane elastomers modified using fluids with shear thickening properties, supplementation of multilayer

thermoplastic elastomer structure with fluid layers consisting of glycerin, water and other liquids, and modification of thermoplastic elastomer using micron-level glass microspheres and carbon nanotubes, among other innovations. Studies about the compression characteristics and energy absorption characteristics of these polymer materials with scale structural units tested under relatively high strain rate demonstrate that they have better energy absorption performance than regular thermoplastic elastomer materials. These materials are highly valuable to blast injury protection. In 2015, Grujicic M proposed the use of zeolite absorption/micro-nanofluidic technology to defend against shock wave. Zeolite is a microporous solid medium. When water enters the micropores of the material, water clusters are formed because of the densification of the material and slow flow caused by localized hydrophobic effect. The consequent time-phase conversion results in impact momentum linearization, meaning that the load spends more time on the target, thereby lowering peak pressure and maximum acceleration. The research projects mentioned above all illustrate that the key to blast injury protection is the effective absorption, dissipation, and redirection of shock wave so as to reduce the amount of the shock wave's impulse received by the body, accomplished through mechanisms such as lowering peak overpressure, shortening pressure effective duration, extending pressure increase duration, etc.

In the real world, blast waves have wavelengths between the micrometer and millimeter level. Analyzing from the mechanics angle, small-scale materials are flat, not uneven, when subjected to vibrating stress waves. The combination of numerous small-scale line materials can capture and dissipate stress wave of one wavelength. Therefore, the choice in material for shock wave energy dissipation or absorption should be one scale lower than the intended shock wave wavelength, as in a range between several hundred nanometers to between several hundred micrometers. For instance, blast waves on the battlefield have wavelengths between 10 μm and 1 mm, and frequency between 2 and 200 MHz, and thus material design should incorporate soft coating, hard, hollow and thin and long inclusions, and piezoelectric and magnetostrictive inclusions correspondingly at scales in the 1–100 μm range, which would be beneficial to mitigating, reorienting, or absorbing blast waves. For shock waves with wavelength shorter than 10 μm and frequency higher than 200 MHz, if material scale was less than 1 μm (nanometer scale), then it would be advisable to use nanometer-scale functional inclusions or control the size, mobility, and electrical property of molecules, with the purpose of mitigating, reorienting, or absorbing blast waves by capturing the front of the shock wave and dissipate the mechanical energies of molecules and atoms at the nanometer scale. Therefore, materials that offer effective protection are always polymers that feature multiple scales and multi-structural phases. This is a crucial principle in the selection of material to protection against shock waves.

4.2 Material Structure

As of present, personnel in both China and abroad have already conducted a large number of designs and researches on explosion- and impact-resistant materials with composite structures. Analyzing from the perspective of material combination structure and the pattern of their influences on performance, materials with capacity to defend against blast waves involve composite structures founded upon base materials such as cement concrete, metal, and organic materials. Researchers have discovered that using a single material as defensive structure against explosion or impact is difficult to achieve both lessening the damage to structure caused by high-speed impact loading and reducing shock wave. A multilayer material with composite structure consisting of high-strength material on the outside and buffer materials with energy absorption capacity in the interlayers can realize the goal of explosion- and impact-resistant functionality. In other words, the combination of hard and soft materials can defend against explosion and impact. The combination can capitalize on the strengths of each individual material while supplementing the weaknesses of the other, thereby augmenting overall protective performance. High-strength materials include the plethora of high-strength concrete, metal plates and such, while interlayer materials are predominantly porous materials or various kinds of soft materials.

4.2.1 Two-Layer Material Combination Structure

1. **Steel plate-reinforced concrete combination structure**. Attaching a thin steel plate behind reinforced concrete slab can significantly lower projectile and explosives' penetration depth due to membrane effect. Although there is still damage inside the concrete, collapse or breakdown would be less probable. Profiled steel plate-square steel tubing-reinforced concrete combination structure can reduce structural deformation, prevent concrete at the bottom from cracking too early, while at the same time offering relatively good margin safety factor. This is a rather ideal explosion-resistant defensive structure.
2. **Aluminum foam-reinforced concrete slab combination structure**. Explosion-resistance studies conducted on aluminum foam-reinforced concrete slab combination structure revealed that increased aluminum foam thickness significantly decreased deflection deformation in reinforced concrete slab, along with relatively large reduction in the acceleration of the impact. Aluminum foam protective layer can effectively enhance the explosion-resistance capacity of reinforced concrete slab, and its thickness has a rational value of calculation.
3. **Aluminum foam-regular steel plate combination structure**. Aluminum foam combined with regular steel plate is a new type of protective structure. Compared with

regular steel plate door, peak pressure in this combination structure has been dramatically lessened, as aluminum foam can effectively lower the peak pressure of the blast wave. Using independently designed gas explosion experiment tube, explosion-suppression tests and studies were carried out on metal wire mesh, ceramic foam with different parameters and their combinations. Outcomes indicate that the combinations boast clear advantage in explosion overpressure reduction compared with individual materials, and the anti-explosion materials suffered significantly less damage.

4. **Polyurea-regular steel plate combination structure**. Protective performance is highly conditional on the thickness and position of polyurea cladding. Specifically, when the polyurea is attached to the surface of the material and faces the shock wave, protective performance is poor. Meanwhile, when the polyurea of appropriate thickness is attached to the back of the material and farther away from the shock wave without tacking on extra weight, protective performance against shock wave is ideal, demonstrating significant reduction in instantaneous effect of impact loading.
5. **Steel tube-filler combination structure**. Finite element software is used to analyze the dynamics responses of three types of tubes with cores under different explosion loads, and the explosion-resistance performance of five types of tubes with cores. It was discovered that these tubes with cores performed better than solid cylinders of the same weight. For tubes with cores, the thickness of the wall of the internal core should be less than the thickness of the wall of the external tube, which would reduce the overall deformation of the whole tube while also capitalizing on the energy absorption advantage of the aluminum foam core. Digital simulation was conducted to study aluminum foam-filled steel tube structures with different tube wall thickness, and the outcomes were compared with results calculated via empirical equations. It was revealed that underwater blast wave pressure and values calculated via empirical equations were approximate to each other, and as tube wall thickness increases, steel tube's anti-deformation performance also rises, and the pressure and kinetic energy in aluminum foam continued to decline. Cheng Tao et al. used digital simulation method to study instantaneous energy absorption properties of thin-walled square titanium tubes filled with aluminum foam and circular titanium tubes under constant-speed impact load effect. They discovered that aluminum foam-filled thin-walled square titanium tubes showed better energy absorption performance than circular titanium tubes, as the buckle wavelength of square titanium tubes filled with aluminum foam shortened, contrary to what was observed in the circular titanium tubes. Cheng Tao et al. studied polygonal metal tubes filled with aluminum foam, discovering that the metal shell plays a crucial role, as different geometric shapes and structures significantly affected the energy absorption rate and energy absorption distribution of the filled tubes. As tube wall thickness increased, energy absorbed by aluminum foam decreased. Therefore, when designing this kind of steel tube-aluminum foam combination, it is necessary to identify the appropriate tube wall thickness so that the respective resistance advantages of both steel pipe and aluminum foam are maximized.

   Through undertaking static compression tests on honeycomb-enhanced plastic foam and calculating the stress-strain curves under different parameters, it was discovered that under the same conditions, all stress figures in composite structure were larger than the sum of stress in honeycomb and plastic foam. As the sides of honeycombs shortened and thickness of sample increased, the composite effect improved significantly. Upon this basis, a simulation model of honeycomb-enhanced plastic foam composite structure was built.
6. **Aluminum foam-filled soft material structure combination**
   (a) **Epoxy resin-filled aluminum foam**: Aluminum foam is filled with epoxy resin modified using silicone, resulting in a composite material with extraordinary energy absorption properties that may be used as a new type of high-performance energy absorption protective material. Static compression test outcomes showed an obvious rise in the aluminum foam's yield plateau.
   (b) **Silicone-filled aluminum foam**: Scientists have conducted studies on aluminum foam filled with silicone, which was then used in conjunction with aluminum pipes and steel pipes to create multilayer pipes. Researchers found out that this type of composite structure offers even stronger buckling fold load and delays the onset of buckling fold. The material has a longer plateau range, which is why it has an elevated energy absorption capacity, and this outcome was even more prominent in the layered steel pipe structure than the layered aluminum pipe setup.
   (c) **Polyurethane-filled aluminum foam**: After filling aluminum foam with polyurethane, there was a significant increase in buckling strength and compression strain, and showed obvious oscillations in stress-strain curve. As strain increased, strain also gradually rose. Some researchers used Hopkinson bar to carry out dynamic impact experiments under the atmospheric pressures of 0.3 MPa, 0.4Mpa, and 0.6 MPa. By analyzing energy absorption curve and ideal energy absorption efficiency curve, scientists discovered substantial improvement in energy absorption properties. Yang Yi et al. discovered that

the use of cushioning material like polyurethane-aluminum foam or polyurethane honeycomb cardboard as energy absorption layer can effectively defend against or weaken a shock wave.

4.2.2 Three-Layer Material Combination Structure

The three-layer "sandwich structure" has excellent buckling stiffness to weight ratio and strength to weight ratio, making it a potentially ideal energy absorption structure. Sandwich structures with cylindrical materials at the center offer excellent impact load-resistance and collision performance. In general, this type of structure consists of two layers of high-strength, thin, face plates sandwiching a light, soft core material that has a relatively weak load-bearing capacity, joined together through welding or adhesives. The face plates are usually metals such as aluminum, copper or steel, or ceramic, hard plastic or fiberglass reinforced plastic, and the core are often materials like plastic foam, corrugated sheet metal, honeycomb sheet metal, polymer or asbestos. Adjusting the distance of the upper and lower face plates can increase the moments of inertia of the structure's cross-section and raise bending stiffness, enabling the face plates to maintain relatively good flexibility and stability when bearing stress. Forms of the core include options such as foam, honeycomb, octahedral grid, prisms, waves, etc. Of which, foam and honeycomb are the two earliest and most widely adopted forms, while trusses, four-sided pyramids, and folding plates offer unique advantages in areas such as lightweight and thermal conductivity, and in recent years a new generation of multifunctional materials have risen, including the likes of lattice structure and hexagon netted honeycomb with concave holes.

1. **Combination structure with porous material in the middle**. Placing highly porous material in between high-strength material can result in a composite blast-resistant structure that can provide anti-impact, anti-explosion and shock wave weakening function. When blast wave acts on the composite structure, plastic deformation and compression occur in the porous material, which could markedly weaken the strength of stress wave.
   (a) **Porous metallic material**: Aluminum foam core material not only offers high impact resistance, but also has a certain degree of penetration resistance. Of which, penetration-resistance performance largely depends on strength of face plate and the combined strength of face plate and core material. Both Chinese and foreign scholars have conducted a plethora of studies on the penetration-resistance performance of aluminum foam core materials. Theoretical analysis was conducted on aluminum foam sandwich plate under a certain unit of impulse so as to find the qualitative relationship between deformation extent and other related physical quantities of the structure, geometrical size of structure and material properties. Finite element software was used to analyze how plate thickness, core thickness, relative density of core layer, and different bullet shape affect the penetration resistance of the sandwich board, as well as its energy dissipation mechanism. Digital simulation method was used to analyze the differences in mid-span displacement and core material compressive strain between aluminum foam sandwich beams with either pure industrial aluminum or stainless steel as surface material under different explosion loads. It was found out that surface material does not make a big difference on compressive strain of aluminum foam sandwich beams. Digital simulation method was used to analyze air shock wave overpressure properties on three types of structures, namely a structure without any protective layer, structure with only one steel plate protective layer, and structure with steel plate and aluminum foam composite protective layer. Digital simulation method was also used to carry out comparative studies on six types of sandwich structures in terms of aspects like mode of deformation, dynamic response, and energy absorption properties, and it was discovered that density of aluminum foam material has a rather large influence on dynamic response of sandwich structure. Researchers used digital calculation model to study the pattern of stress wave propagation in aluminum foam sandwich plates under explosion load action. Comparative analysis was performed on the material's buffering, energy absorption and stress wave weakening properties, discovering that when overall density remains the same, a tiered structure boasts better buffering performance. Since multilayered aluminum foam offers better protection and higher energy absorption efficiency than single-layer structure, researchers studied the protective performance of multilayered aluminum foam plate with three layers of aluminum foam cores sandwiched between steel plates. They discovered that the bottom plate showed lesser lateral deflection in a structure with aluminum foam layers that had progressively decreasing density, compared with layers that had progressively increasing density. Therefore, aluminum foam layers arranged in a configuration with progressively decreasing density sequence can augment the blast-resistance capacity of the overall structure.

   Researchers found out that bullets under different speeds give rise to different forms of damage against

pure aluminum plates versus aluminum plate-aluminum foam sandwich plate. Other researchers studied how bullet impulse, face plate thickness, core layer thickness, and different forms of core affect the blast-resistance performance of sandwich plate. Initial reverse penetration test on aluminum foam sandwich plate under impact load shows that aluminum foam mostly bears local stress, and strain hardening was observed in aluminum foam prior to damage to top face plate. Research on blast-resistance capacity of aluminum alloy face plate-tiered aluminum foam core-steel armor plate sandwich structure demonstrates that aluminum foam sandwich structure with aluminum foam cores with tiered densities has clearly better blast resistance than sandwich structure of the same mass but with aluminum foam cores with the same density. Multi-target optimization can further strengthen the structure's overall blast resistance. Ren Xinjian et al. analyzed the blast-resistant mechanism of the steel plate-metal foam-steel plate sandwich structure and pointed out that the material's improved blast-resistance capacity is chiefly attributed to the boundary effect and focusing effect generated by a shock wave's reflection, scattering, and interference.

(b) **Cement-based porous material**: Sandwich structure with steel plate and concrete core is noted for strengths such as optimal structure stress, high load-bearing capacity, high stiffness, outstanding anti-seismic properties and dynamic properties, variable cross-section forms, and easy construction and installation. Researchers discovered that cellular concrete structure boasts obvious energy absorption capacity, and as density decreases, blast wave's peak stress and strain dramatically reduce upon passing through said layer. In this composite protective structure, blast resistance and energy absorption are realized by sacrificing cellular concrete to preserve the inner structure.

(c) **Porous polymer material**: Polymer foam is arguably the most common core component, and some materials include polyvinyl chloride, polystyrene, polyurethane, and polyetherimide. Researchers used adhesive to prepare steel plate-high polymer-steel plate laminated composite material. Results show that increasing the roughness of the steel plate surface and raising the bonding pressure to a certain extent can enhance the formability of the laminated composite material. Through falling weight impact test, researchers studied the dynamic response characteristics of polymer foam and metal foam sandwich materials under low-speed impact load, discovering that modes of damage to polymer foam sandwich structure include stripping of surface material, localized shear cracks, and fractured cores, among others. Other researchers used reinforced concrete and steel plate-concrete structure as top and bottom panels, with rigid polyurethane foam placed in between, in an attempt to adjust the distribution method of blast load within this structure. Specifically, the goal was to turn partial load into load distributed across the whole structure so as to reduce and mitigate blast wave and minimize damage to structure.

2. **Combination structure with honeycomb in the middle**. Honeycomb sandwich panels have some sort of honeycomb core placed in between panels. Usually the panels bear bending and deformation load and constitute the main load-bearing function, while the core links the two panels and together bear external loads. Some of the more common categories are aluminum panel-aluminum honeycomb core structure, carbon fiber panel-aluminum honeycomb core structure, and fiberglass panel-fiberglass honeycomb core structure, among other varieties. Honeycomb cardboard features two layers of cardboards at the top and bottom, and hollow honeycomb structure sandwiched in between them, and the holes in the honeycomb are filled with polyurethane foam. This is a new type of composite structure buffer material.

Yoshiaki Yasui studied the dynamic impact compression behaviors of even and conical honeycomb sandwich panels and concluded that conical honeycomb sandwich panels perform better in terms of energy absorption. Dear JP et al. studied sandwich material consisting of sheet molding compound, thermoplastic glass felt and honeycomb core panel, concluding that said material can absorb a relatively large amount of impact energy and is noted for lightweight and high strength. Chen Changhai et al. were the first to use finite element software to simulate the responses of sandwich panels under impact load. They then analyzed the blast-resistance capability of such material from the perspective of energy absorption and obtained a type of optimized sandwich panel model. Other researchers carried out comparative analysis on the impact-resistance capacity of three types of sandwich panels featuring pyramid truss, square honeycomb and folding boards. At present, it is widely accepted that honeycomb sandwich panel structure offers stability against crushing load and relatively long effective distance under horizontal impact load, demonstrating excellent energy absorption property. Structure density is the key factor that determines that structure's collision-resistance capacity, while the height of the core layer does not have much of an influence on collision-resistance capacity. Other researchers are of the opinion that fiberglass honeycomb sandwich composite material is an ideal choice for the construction of shallow-buried blast-resistant structures.

3. **Combination structure with heterogeneous structure in the middle**. Heterogeneous core materials are noted for flexibility in optimization, and cores could have different structures like lattice, I-shaped, O- shaped, V-shaped, wave or other structures. Zenkert and Burman studied relative strength of sandwich panels with the same structure, discovering that increase in the height of V-shaped cores, while adding very little to core layer mass, could drastically enhance the sandwich panel's hardness and bend-resistance. D. D. Radford et al. studied responses of fixed sandwich beams featuring cone-shaped stainless steel core, wavy stainless steel core, and foamed aluminum alloy under impact load. Their experiment discovered that sandwich beams with cone-shaped core had the worst impact-resistance capacity, but all sandwich beams performed better than holistic beams in terms of impact resistance. Other researchers constructed analytical model for 3D-Kagome lattice sandwich panel under ideal impact load. After redesigning and optimization, the outcome is a new type of light structure with higher stiffness, better impact resistance, and more energy absorption and dissipation.

   Metal sandwich structure is a type of holistic sandwich structure featuring metal top and bottom plates that sandwich metal cores in wave, honeycomb, truss, or other forms, with the components joined together using laser welding technique. European and North American countries have conducted a considerable number of studies on material performance, design, manufacturing, and other regards. In China, studies in these areas are not yet systematic, and there are rooms for improvements in aspects such as theoretical calculation, experiment, research, engineering, and application. Wang Guo et al. found out that metal-based fold-type sandwich panel offers excellent impact-resistance capacity. In addition, under blast load of underwater explosion, Y-shaped, laser-welded sandwich panel showed film tensile deformation in bottom plate, and crushed deformation in core layer, which together help buffer the top plate, thereby reducing damage and deformation of top plate, augmenting the structure's energy absorption efficiency and exhibiting fantastic blast-resistance capacity. Other researchers experimented with square honeycomb, I-shaped core and wave-structure metal sandwich panels in underwater explosions, discovering that form of response depends on the relative time of compression of core and the size of air bubbles in the water. Yi Jiankun et al. analyzed the theoretical analysis model of blast-resistant process of lattice metal sandwich structure, deformation and failure forms of sandwich structure, blast-resistance and energy absorption properties, and other related influential factors.
4. **Other sandwich combination structures**. Researchers have proposed a novel type of sandwich structure comprised of fiber reinforced polymer (FRP) and aerated lightweight concrete, and research result showed that said composite structure has significantly augmented bend-resistance performance. Tian Zhimin et al. conducted blast-resistance experiments to study the blast-resistance load-bearing capacity and typical forms of damage on rebar-enhanced composite sandwich material. Researchers also found out that double-layer steel plate sandwiching cement-fiber board in the middle has obviously better blast resistance than double-layer steel plate sandwiching plain cement board or double-layer steel plate sandwiching foam board. The side facing explosion mostly exhibits crushing failure, and the side back to explosion indicates tension failure, while the core layer shows "layered fractures." In addition, aerated sandwich structure also performs very well against explosions, and can occupy a crucial position in key buildings and civil defense structures.

4.2.3 Multilayer Combination Structure

Through multilayer combination of rigid and flexible materials, it would be easier to realize certain design goals. However, there are relatively few literature pertaining to experiments on blast resistance and impact resistance of composite structures consisting of three or more layers of materials, with the majority focused on penetration resistance testing. Some researchers simulated the course of penetration of steel/ceramic/aluminum composite target board by cylindrical shrapnel 8 mm in diameter. Results showed that, under the condition of same area density, decreasing board thickness while increasing ceramic plate and aluminum back plate thickness can significantly enhance the composite target board's ball-proof performance. In terms of selection of composite structure for materials used for safety suits, Ken-An-Lou et al. proposed a four-layered protection structure (Fig. 2) to deal with non-penetrating injuries. Of

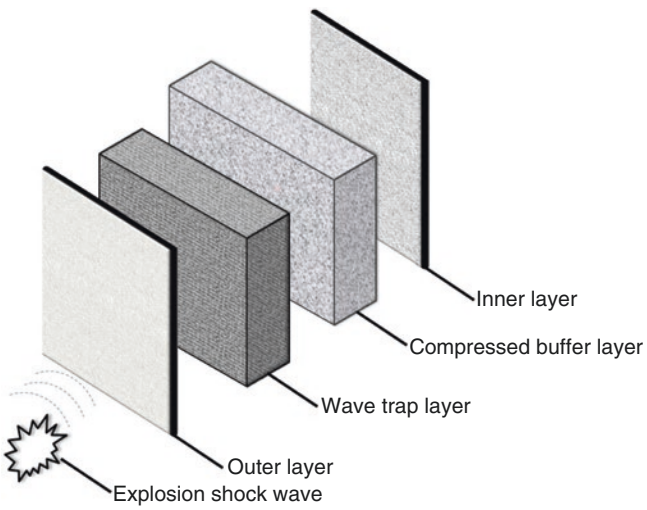

Fig. 2 Four-layer protection structure against non-penetrating injuries

which, the two central layers are the barrier and compression buffer that are predominantly responsible for energy absorption and dissipation. Based on this structure, a myriad of material performance combinations were designed, and it has been proved that this type of combination is an effective structural model to defend against blast injury.

4.2.4 Fiber-Wrapped Composite Enhanced Combination Structure

Wrapping fiber around the surface of the material structure can change the model of damage imparted on the structure under explosion load, resulting in obvious improvement of the material's dynamic performance. Horizontal wrappers not only restrict the expansion deformation of the structure of the internal materials, but also greatly improve axial bearing capacity and anti-shearing capacity, exhibiting bending ductility reaction under explosion load. Tonatiuh Rodriguez-Nikl et al. used quasi-static load to simulate blast impact on carbon fiber-wrapped reinforced concrete pillar. Outcomes demonstrated that damage caused to the reinforced concrete pillar shifted from brittle shear failure to bending ductility failure. Ha Yue et al. designed the Whipple protection structure, in which basalt fiber cloths were configured in different manners within the structure. Researchers found out that when penetration damage occurred, the fracturing and shearing of fiber bundles at the penetration point and the deformation of the fiber bundles around the penetration point dissipated and absorbed the force of impact of the projectile. Through conducting tests on concrete beam reinforced with carbon fiber cloth and high-strength glass fiber cloth, it was discovered that the bearing capacity and ductility resulting from two layers of high-strength glass fiber cloth and one layer of carbon fiber cloth were respectively 7.8% and 10.9% better than beam reinforced with two layers of carbon fiber cloth, yet stiffness was 10% inferior. Bao Yuming et al. realized that fiber cloth reinforcement can raise bearing capability by 200%, and under blast load, bending could decline by 40–70%.

All in all, multilayer composite materials comprised of both rigid and flexible materials offer obvious technical advantages and can more easily achieve integrated protective and functional structure design goals encompassing blast resistance, energy absorption, sound suppression, and thermal insulation. However, the structures of these multilayer composite materials have not been widely applied in actual protection structures because the majority of core materials are still being studied. Some functional features remain unclear, especially response under dynamic load. Moreover, the performance of composite materials largely hinges on the method of connection between the core materials and surface plates. Since welding and adhesive are the primary methods at the current stage, and because there are many differences in core materials and surface plate materials, there are still many challenges in realizing sturdy connections between the different materials, hampering the functionality and stability of material structure.

5 Blast Injury Protection Equipment

Existing protection equipment for troops mostly emphasizes the use of lightweight protective materials that can achieve high effectiveness against penetration by bullets and shrapnel. In recent years, equipment research and development personnel have tested ultra-high molecular weight polyethylene fiber, high-strength para aramid, lightweight bulletproof ceramics, and other new types of bulletproof materials, and they have clearly improved anti-bullet and anti-shrapnel performance upon effective weight reduction. Yet, research results showed that regular bulletproof equipment is not only helpless in defending the penetration of blast wave, but shock wave that reaches the surface of the protection equipment might even increase damage because of reflection and superposition. In particular, for explosions inside an enclosed space, shock wave might be even more damaging due to reflecting multiple times between wall and protection equipment surfaces and prolonged underpressure effective duration. Therefore, to reduce bodily injury caused by blast wave, it is mandatory and urgent to enhance material's protection efficacy against shock wave under the condition of maintaining bulletproof performance and controlling weight. At present, it is necessary to continue studying and developing protection equipment (List 1) with ideal material composition and structure to protect organs and body parts that are most easily injured by explosion so as to reduce injuries and deaths of wearers caused by blast wave, fragment, and related crushing injuries. Data of injuries and deaths of American troops in Iraq and Afghanistan in the past 10 years indicate that shock wave reduction (absorption and redirected dissipation) and protection against impact from projectile are crucial to saving 95.8% of injuries and fatalities, and the chief pathway to reducing death is to improve blast injury protection equipment and stopping blood loss.

List 1 Blast Injury Protection Equipment Required for Different Body Parts

Body part injured	Main organs and tissues affected	Protection equipment
Brain	Cerebrum, cerebellum, and brainstem	Helmet
Chest	Lung, heart, and thymus	Safety suit and safety vest
Abdomen	Liver, spleen, kidney, adrenal gland and gastrointestinal tract	Safety suit

Body part injured	Main organs and tissues affected	Protection equipment
Pelvis	Urinary tract, femoral artery, and gastrointestinal tract	Pelvis blast resistance equipment
Limbs	Muscles, bones, and blood vessels	Safety suit
Auditory apparatuses	Eardrums and auditory ossicles	Ear muffs and earplugs
Face	Eyeballs and jawbone	Face mask, eyeshade, goggles, and lower jaw protection equipment
Neck	Large blood vessels and cervical cord	Neck protection equipment
Hands	Muscles, bones, and nerves	Safety gloves
Feet	Muscles, bones, and nerves	Safety shoes and safety boots

5.1 Protection Equipment for Head

Since blast wave could form air pockets or mini bubbles inside the head, implosion and other mechanisms could injure the brain parenchyma. Thus, it is necessary to use effective materials to increase shock wave absorption and dissipation outside the brain. At the same time, dynamic pressure generated by the shock wave could cause the head displacement, being thrown or colliding against something else, further aggravating craniocerebral injury. Therefore, it is necessary to reduce peak acceleration during the head collision. Protective helmet and supportive components are no doubt the premier choice when it comes to decreasing blast injury to the head.

Chinese troop helmet is born from the GK80 helmet, fabricated mostly from bulletproof steel. However, shock wave could reflect and superpose multiple times on the surface of the helmet and in the gap between the helmet and the head, thereby elevating peak overpressure and possibly worsening head injury. Thereafter, the QGF series of helmet was studied and developed, using kevlar bulletproof composite material as base material. However, both the metal and non-metallic helmets mentioned above have much room for improvement in terms of protecting the wearer against blast-induced traumatic brain injury. At present, a helmet is comprised of the protective body and the suspension system. The former is mainly focused on preventing penetration by bullets and fragments, while the latter bears the main responsibility for protecting the wearer against non-penetrative injuries caused by shock wave. Developing lightweight polymer material with shock reduction and energy absorption capacity, and multilayer shock reduction structure are urgent needs. Data indicates that The HYS-1 impact-resistant helmet developed by the Chinese military can reduce peak impact acceleration by 72–82% under a medium-strength shock wave, with average acceleration slowed down by 50–71%, as verified in explosion experiments. In addition, tank helmet also offers a certain degree of protection against blast injury to the head and brain.

In reality, it took several centuries for the combat helmet to evolve from protecting against external wounds caused by blades and clubs to defending against injuries from bullets and fragments. In essence, the majority of combat helmets of the twentieth century were designed for usage in trenches or foxholes. In trenches or foxholes, most ballistic threats come from above. It was only in the past few years did the nature of warfare change, accompanied by changes in the types of external injuries on the battlefield, with a rising number of troops afflicted by external head injury and traumatic brain injury caused by blunt force, explosion or impact from bullet. Of which, improvised explosive device (IED) are often seen in terrorist attacks and roadside bombing. According to a 2009 report released by the US military, all troops in a brigade deployed in Iraq were outfitted with bulletproof helmets, but 1292 members out of 3973 had been injured, of which 907 soldiers (22.8%) suffered from traumatic brain injury as diagnosed by clinical physician. In most cases, these injured personnel would be afflicted by post-war headache and/or dizziness and might even exhibit outbursts of rage or memory loss as time passes by. Therefore, it is an urgent need to provide better protection against this sort of blast injury. It is also necessary to consider the gap between the edge of the helmet and protective vest, because the lack of protection in this region means high risk of injury. In addition, due to feedback from battlefield injuries and fatalities data, foreign military equipment research and development departments currently are more inclined to promote equipment that protect the entire head, including safety goggles and jaw shield with fragment-proof capacity so as to reduce injuries to the eyes and ears caused by shock waves. At the same time, for shock waves that enter the helmet, full protection helmets also decrease injuries caused by reflection and superposition waves that occur in the gaps around the head.

Not long ago, regular helmets issued by foreign militaries to their troops could only provide limited protection against hi-speed bullet in some instances. Meanwhile, there is still no solution that can balance between both lightweight and impact/explosion protection. At present, foreign militaries have proposed the research and development of a comprehensive head protection system based on the form of helmet. This kind of helmet features a modular design with add-on accessories such as night vision system, eye and lower jaw protection system, as well as additional armor layers to protect the wearer against impact from more powerful explosions and projectiles.

The British Armed Forces used the Mk.6 helmet supplied by NP Aerospace. Its design was introduced in the mid-1980s and is notable for exceptional performance against

blunt trauma compared with counterparts at the time. NP Aerospace adopted ergonomics principles and redesigned its product, giving rise to the Mk.7 helmet, which kept many of the advantages of the Mk.6 helmet, including substantial use of internal cushioning and head-shaped support framework. Starting from 2009, to satisfy urgent operational requirement, the Mk.7 helmets were issued to troops stationed in Afghanistan.

NP Aerospace also designed the model for the AC900/ICH (integrated combat helmet), which provides cover for the gap between the bottom edge of helmet and bulletproof vest, where vulnerability is high and fatal because the major cervical vessel, brain stem, and cervical spinal cord are located here. Lower jaw protection device and full face mask may be attached to the AC900/ICH. These accessories are affixed through a unique design, and the frame of the face mask can also accommodate night vision goggles. The patented multi-axial shell structure allows the AC900/ICH to weigh 10% less while maintaining the same level of protection, which can compensate for the additional burden when lower jaw protection device is attached.

The US military announced in February 2011 its search for an improved helmet design called enhanced combat helmet (ECH). This kind of helmet needed to have better bulletproof performance than the existing advanced combat helmet (ACH). The latter was issued in large quantities to US troops in 2003 and is still being used. At first glance, the ECH might not appear to be much more different than the predecessor it is intended to replace, but person in charge of the soldier protection and individual equipment (SPIE) program stated that the two helmets are drastically different in performance. The ECH is a bit thicker than the helmet issued to the majority of U.S. Army troops, but lighter in weight. It has been known that the ECH offers a whole new level of protection for the head, especially in protecting the wearer against projectiles. Research data indicate that the ECH shows at least 35% better performance in bulletproof capacity compared to the ACH. Therefore, the enhanced combat helmet plays a crucial role in reducing traumatic brain injuries.

The ECH is designed by Ceradyne Diaphorm and is named the seamless ballistic helmet (SBH). This kind of headgear uses a patented thermoplastic composite helmet molding technology to augment protection against projectiles and light weaponry. The manufacturer claims that the US military has finally been given a combat helmet that can stop rifle shots. It has been learned that the shell of the helmet adopts a seamless ballistic helmet technology using a type of multilayered and laminated material, which does not require cutting or stitching together any single layer. After the shell is formed, there is pretty much no folding in the laminated material, which ensures the shell's performance and thickness uniformity. Ceradyne also declares that the thermoplastic composite material not only improved the bulletproof capacity of the material used for the main shell of the combat helmet, but also dramatically impacted the production process. It has been projected that the performance of this type of helmet would not be affected in different combat environments and factors such as seawater, drastic temperature changes, high-altitude area, salt spray, and outdoors. This is the result of the type of chemical properties of the material (in particular the thermoplastic resin and fiber materials in the main parts of the helmet) used to manufacture the helmet.

In Canada, the Department of National Defence has already commenced tests on the soldier integrated headwear system (SIHS), which includes the advanced modular multi-threat protective headwear system as one of its crucial components. Starting from 2007, the research and development task force of the Canadian Department of National Defence already began developing and enhancing combat helmets, with the aim of addressing weaknesses (such as protecting the gap between the helmet and bulletproof vest), adding protection for the face, and ensuring protection against not only the threats of bullets but also the dangers of blast waves.

In November 2011, Mark Rutley from the Soldier System Integration Group once mentioned that the helmet in the advanced modular multi-threat protective headwear system features a modular design that includes an inner shell, a bulletproof external shell, a lower jaw protection device, a face mask, and a neck protection device. In recent experiments, Canadian efforts focused on testing soldiers and new helmet designs in environments as close to real battle scenarios as possible. The concept of the advanced modular multi-threat protective headwear system was tested to ascertain the genuine efficacy of the design. The advantage of this kind of equipment (modular helmet) is the ability to reconfigure the device based on the specific demands and conditions of each mission. Based on the requirements of the mission at hand, the basic helmet may be used during regular missions, while protection may be maximized by attaching the lower jaw protection device and face mask during combat situations so as to effectively safeguard the wearer against mild traumatic brain injury (MBTI) and facial injury.

Canadian bulletproof goggle manufacturer Revision Military, a supplier of combat eyewear for numerous NATO states, has expanded its business into the modular helmet sector, hoping to address the demands for countries and militaries in need. Revision Military unveiled its revolutionary BATLSKIN model at the DSEI in London in 2011. Thereafter, Revision Military revamped its BATLSKIN model into a fully modular design that consolidates trauma liner, communication system, heads-up display, anti-chemical, anti-biological, anti-radiation and anti-nuclear face mask, and enhanced night vision goggles, and attachable face mask and lower jaw protection device that can bol-

ster defense against blunt force, explosion, and bullet (compared against helmet shell alone). Based upon extensive research and development of headgear, Revision Military's new type of helmet and head protection system can help reduce brain injury while lightening weight and offering superior bulletproof performance. Therefore, by taking advantage of new materials and new processes, and by sustaining the design, development, and iteration of expandable and modular helmet systems for soldiers both in vehicles and on foot, efforts should be aimed at providing headgear with better impact resistance compared to current helmets, while consolidating electronic devices and power supply. Such equipment would also allow troops to customize their own gear based on the various conditions including protection needs of the assigned mission.

Project engineer Don Lee with the U.S. Army Natick Soldier Systems Center mentioned: "The current situation is that soldier-centric design mentality still has not yet been adopted in the design of platforms that integrate head protection and functionality. This hinders optimization between the weight, balance and components." Speaking of the advantages that Revision Military brings, Don Les said: "This development contract supports the upgradeable army technology objective, an electronic helmet equipment and display system. It is anticipated that the integrated helmet technology will include upgradeable bullet and impact protection, face mask that consolidates anti-chemical, anti-biological, anti-radiation and anti-nuclear functions, full facial protection and integrated heads-up display and sensor input that can provide actionable info to soldiers and operators." Researchers hope that the final product combines a set of tools and technologies that allow for continual expansion and optimization of functions, so that the helmet system can provide critical protection against explosions on the battlefield. Such a system can provide better protection to the head while accounting for enhanced situational perceptions, thereby raising mission performance and giving an extra edge to troops in action.

5.2 Protection Equipment for Chest and Abdomen

Protection equipment for the torso are mostly protective suits produced to defend the torso against shock waves, with the purpose of stopping or weakening the direct effects that shock waves have on the thoracic and abdominal walls so as to lessen injuries of internal organs caused by the shock wave. In the 1980s, the US military conducted shock wave protection performance tests on kevlar bulletproof vests. Results indicated that due to the gaps that existed between the layers, peak overpressure transmitted to the body of someone wearing the soft bulletproof vest was actually higher than a person not wearing a vest, including worsened blast injuries to the lungs. Research showed that nickel foam of a certain thickness can attenuate peak overpressure of a shock wave by 26% while prolonging the shock wave pressure increase duration. Using this as the main component in a protective vest with composite material could significantly reduce the injury suffered by test animals and lower fatality rate. Other reports stated that sailors wearing air-filled compressed foam material separate the body's air-filled parts (chest and abdominal cavities) from the surrounding water, which could effectively lessen underwater blast injuries and other related damages. However, it should be pointed out that although current bulletproof suits or bulletproof vests offer decent protection against fragments, both clinical results and empirical research show that they provide no protection against blast injuries, and might even worsen blast injuries. This is an area worthy of attention in the prevention and treatment of blast injuries. Analyzing from a clinical treatment point of view, protecting organs easily affected by shock wave also needs to consider the wearability, flexibility, smartness, fire resistance, and other factors of the protection equipment so as to achieve both precision and foresight upon the basis of defending against blast waves. Actually, in 2017 the Japanese Ministry of Defense officially began conducting new explosion and impact experiments that research protective suits, face masks, and other gears capable of preventing bomb's shock waves from entering the human body.

5.3 Protection Equipment for Pelvis

According to injury and fatality data of US troops in Iraq and Afghanistan between 2001 and 2011, ground troops on patrol were frequently targets of IED explosions, and victims largely suffered from injuries in the abdomen, pelvis, and urinary tract. Pelvic blast protective equipment is a new type of protection system designed to protect the pelvis and femoral artery and will replace two existing protection systems: base layer and exterior garment. Soldiers usually prefer simple protection systems that fit the body shape, and feedback info also indicate that said system offers higher mobility.

5.4 Protection Equipment for Auditory Apparatus

Wearing different kinds of earplugs or earmuffs can decrease blast injuries on auditory apparatuses. Take for instance the American EAR earplugs, they can reduce 30–48.9 dB within the frequency range of 63–8000 Hz if used in strict accordance with experimental conditions, and reduction for normal usage falls within 14–25 dB, while the Type-69 earmuffs

developed in China could reduce sounds by 14.7–40.3 dB. The protective helmets issued to tank operators could reduce sound by 30–40 dB if no headphones were worn.

5.5 Protection Equipment for Feet

The wedge-shaped metal-plated shock-reduction shoes issued to US troops can protect against blast injuries to the feet for those on naval vessels. The shock-resistant shoes for use on naval vessels developed by the Chinese military could reduce acceleration on the heels by 61.1% under an impact with peak acceleration of 92G, with the performance still maintained at a reducing acceleration by about 50% when peak acceleration reaches 120–170G. At present, this type of footwear is suitable for operators on minesweeper vessels. Different anti-mine shoes developed based on pressure-reduction principles and explosion mechanics principles can help prevent or decrease injuries caused by mine explosion. For instance, the FLX-1 anti-mine shoes developed by the Chinese military can attenuate 77% of energy of an air shock wave.

6 Protection Measures Against Blast Injury

6.1 Planning, Design, and Protection Education

6.1.1 Planning and Design of Storage for Explosives

With regard to site selection for explosive substance plants and storage buildings, these should be constructed in independent areas far away from urban areas with dense population. These structures need to maintain safe distance from key facilities such as water conservation facilities, transportation hubs, bridges, tunnels, high-voltage transmission cables, communication lines, oil pipelines, etc. At the same time, when confirming the overall planning and design of these structures, it is necessary to separate work zones in strict accordance with the respective production natures and functions, and ensure enough isolation distance between the different work zones and distance from important external locations.

The definition and determination of blast safe distance should include both internal safe distance and external safe distance. To ensure that damage and injury to structures and relevant personnel do not exceed a certain level of destruction, it is necessary to maintain enough safe distance between the production zones, main storage zones, destruction and disposal zones, and other zones related to explosive articles. This is internal safety distance. It is mandatory to maintain enough distance between the production zones, main storage zones, destruction and disposal zones, and areas of civilian activities such as villages, residential buildings, factories, towns, and roadways. This is called external safe distance. Specific safe distance must abide by code for safety design.

In terms of layout of production processes conducive to protection against explosions, the following principles should be adopted: (1) In terms of production process and technique, maximize the use of new and intelligent technologies to achieve human-machine separation so that people stay far away from actual operation. (2) During the actual production process, it is necessary to separate the processes for hazardous and non-hazardous items, and placing the two in separate buildings would be advisable. (3) With regard to specific layout of a building, it would be best to keep production of dangerous items at an isolated location with few pedestrians nearby, then configure less dangerous productions outward. Temporary storage for dangerous articles should also be situated at an isolated location. (4) In terms of layout of sites of dangerous item production and storage, they should have simple rectangular footprints, which facilitate emergency evacuation in case of explosion accidents. (5) For processes or equipment with explosion venting requirements, vent direction should be configured away from buildings and main roadways. (6) Configuration of explosion-resistant rooms should meet safety protocol requirements.

Rapid fire sprinkler: Explosive substances like gunpowder ignite and burn extremely quickly, and could cause an explosion within mere seconds, which is why extensive placement of rapid fire sprinklers and other fire protection devices are necessary in production sites of explosive substances. Rapid fire sprinkler system is mainly comprised of photosensitive detection system and water piping network. When there is a fire in the factory building, illumination would increase, photoresistor's resistance value decreases, while electricity current in the control system increases, which passes by electronic amplifier and electric relay to open up electromagnetic valve. With valves opened, pipes would deliver water to sprinkler heads to extinguish fire with the purpose of stopping explosion.

In addition, regarding blasts required in production, avoid blasting in the morning, evening, outdoor, or other conditions favorable to air shock wave propagation. Try to refrain from blasting with uncased explosives or outdoor blasting with detonation cord. If these can't be avoided, cover items with sand and dirt.

6.1.2 Education About Protection from Explosives and Dangerous Items

In indoor situations, use wall corner, wall or table as cover and lie or sit down; for those with fortifications nearby, use

them; and for those not quick enough to enter a fortification, protect oneself using terrain or ground surface features. When there are large ground surface features, lie down horizontally; when ground surface features are small, lie down with head toward the center of explosion; and when the terrain is flat, lie down with head away from the center of explosion. Other crucial actions: Keep arms crossed in front of chest, keep eyes closed, keep abdomen tight, and keep mouth closed; straighten legs and keep them together, lower head and hold breath. This is the position most helpful to minimizing the effects of blast wave in most situations.

1. **Simple defensive actions**
   (a) Hide immediately: The propagation of a shock wave is much slower than that of light radiation. When a nuclear weapon detonates, in most cases it would take quite some time after the flash of the explosion before its blast wave reaches a certain distance. For instance, it takes 26 s for the shock wave from a mid-air detonation of a hundred kiloton nuclear weapon to reach a distance of 10 km. Therefore, after seeing the flash of explosion, immediately enter a fortification or hide behind terrain or ground surface features to avoid or decrease injuries caused by shock wave.
   (b) Lie down on the ground: For people in the direct kill zone of shock wave, especially those close to the center of explosion, they might be thrown, displaced, or "blown down" by the dynamic pressure of the shock wave. If these people could not make use of landscape or ground surface feature or fortification, the immediate course of action is to lie down on the ground with head away from the center of explosion. In other words, when the length of the body is parallel to the propagation direction of the shock wave, keep the head as far away from the source of explosion as possible. When lied down, the windward area of the body is only 1/5 of that when standing, thus beneficial to reducing total impulse and substantially weakening the injuring effect of the shock wave's dynamic pressure. Lying down can also help prevent the chest and abdomen from directly bearing pressure to a certain degree.
   (c) Stay away from doors, windows, and walls: When an explosion occurs in urban residential areas, boards and glass panes of doors and windows are often shattered, which would turn into fragments that could cause indirect injuries, often times even fatal injuries. Therefore, when indoor personnel don't have time to exit the building and hide, at least try to stay away from doors and windows, and lie down and hide by foot of wall, foot of building, beneath table or bed. Considering how shock waves could reflect off a surface, people near an explosion should stay away from walls when conditions permit, quickly relocate to an open space.
   (d) Other actions: Keep mouth opened and ears covered during an explosion could help decrease auditory apparatus injuries (such as eardrum rupture) to a certain extent. Personnel on operating minesweeper vessels should try to walk around as little as possible, and it would be best to tip toe while walking to avoid or lessen solid blast injury caused by underwater non-contact explosion. When explosion from an underwater weaponry, missile, or cruise missile is imminent, personnel working in the water should try to reach the surface of the water as quickly as possible while keeping body exposed to water surface. For those unable to react quick enough, lie flat on the water surface to avoid or decrease underwater blast injury.
2. **Terrain and ground surface features**. Terrain and ground surface features could shield or strengthen a shock wave. When a shock wave propagates along the surface of the ground, upon encountering highland, hill, or mountain, the shock wave would reflect from the slope facing the center of explosion, thereby increasing overpressure. When a shock wave propagates around the top and two sides of highland, hill, or mountain, the overpressure and dynamic pressure behind the land feature would decrease, creating a decompression zone. In a region a certain distance away from the decompression zone, the parts of the shock wave would converge, increasing overpressure and creating a compression zone. Generally speaking, the steeper the slope facing the explosion, the more the overpressure increases, and the steeper the slope away from the explosion, the lower the overpressure and dynamic pressure in the decompression zone. Therefore, by taking advantage of the characteristics of the terrain and ground surface features explained above, upon seeing the flash of an explosion, quickly enter the decompression zone of the slope behind the highland, hill, or mountain away from the source of explosion, which would be helpful in avoiding or decreasing injuries caused by shock wave.

Ridge, pit, sidewise culvert and archway under bridge, roadbed facing away from center of explosion and underground sewage also offer a certain amount of protection. However, when trying to use a terrain or ground surface feature, remember to stay away from structures that could easily collapse (e.g., tall chimney, high-rise and thin building), and valleys that face the center of explosion (overpressure could increase due to convergence of the different parts of a shock wave).

Therefore, in terms of the factory zone or site selection for explosive materials, first and foremost site planning should take into account the production process, safe distance and the features of different zones, and make sufficient use of beneficial and safe natural landforms within the chosen area so as to engender effective protection against blast waves. Second of all, main storage zone of explosive and flammable items should be situated far away from residential and urban areas with high population density. If conditions permit, this type of warehouses should be located inside valleys or other such regions with natural barriers. Similarly, destruction and disposal zones should choose naturally beneficial landforms like valley, hill, or river shoal. Upon the premise of meeting safe distance requirements, determine the location of destruction and disposal zone and relevant buildings.

6.2 Establishment of Man-Made Barriers

6.2.1 Explosion-Proof Retaining Walls

In narrow passages, the "shielding" technique could be employed to weaken the strength of an air shock wave. For instance, setting up brick walls, sandbag walls, stone walls, or other types of walls near explosive zones can help stop blast waves. Some nations use water bags made from high-strength artificial membrane as material for such walls. Bags filled with water are packed tightly against walls of the passage. When a shock wave reaches the bags, pressure in the water bags increases, which is passed to the sides of the passageway. This setup enhances shock wave-resistant capacity. Such water bags are economical and easy to set up as shock wave-resistant walls, not to mention their effectiveness, usually capable of weakening shock wave by ¾ or more while also reducing dusts and harmful gases from the explosion. Therefore, man-made retaining walls can decrease damage caused to buildings by the shock waves and fragments from the explosion of explosives. Constructing such retaining wall is one of the methods employed in explosion-proof design.

6.2.2 Bubble Curtain

During underwater blasting, bubble curtain technology is an effective method in lowering the power of an underwater blast wave. This entails setting up a bubble curtain device between the source of explosion and subject(s) of protection. Generally speaking, steel pipes are used to create two rows of little holes on the two sides. After injecting compressed air into the device, a large amount of tiny bubbles would shoot out from the little holes. Due to buoyancy, the clusters of bubbles would continuously rise to the surface, creating a bubble curtain. This bubble curtain can effectively weaken the peak pressure of shock wave and thus protect the target subject(s). Engineering tests have been conducted to verify the efficacy of this technology.

6.2.3 Impact-Proof and Shock-Absorbing Device

Steel spring damper is a classic impact-proof and shock-absorbing device. Under external impact, the damper first absorbs the kinetic energy of the impact, then converts it into internal elastic potential energy, and finally releases this potential energy and converts it back to kinetic energy at a relatively slower pace. At the same time, some of the kinetic energy is converted by damping action into heat, which is then dissipated. A significant portion of impact energy may be dissipated after repeating several such cycles. The Cheyenne Mountain Complex in the USA has been outfitted with more than 1300 large-scale steel coil springs, which provide holistic protection for 15 steel-structured buildings that do not have contact with the internal surfaces of the mountain chambers.

Magnetorheological damper is a proactive shock absorber that adjusts according to external impact. The self-adjusting damping force and system stiffness together optimize shock protection performance.

6.3 Protection of Fortifications

Fortification is a basic method of protection. To help save lives, various impact-resistance and energy dissipation methods are adopted to convert kinetic energy into potential energy, lessen big impact into small impact and to ensure safety. This is the last line of defense that must be upheld in blast protection. The various kinds of permanent fortifications and air raid shelters underground can effectively shield personnel against the dynamic pressure and overpressure of a shock wave, thereby sparing the subjects from blast injury, or at least substantially lowering the seriousness of injury. Different kinds of open-air fortifications such as trenches, bunkers, ear-shaped caves, and ear-shaped caves could pretty much shield personnel from the injuring effects of dynamic pressure, while that of overpressure would also be greatly weakened (marked prolongation of pressure increase duration). Thus, these offer different degrees of protection.

Battlefield fortifications include open-air fortifications (trenches, communication trenches, and various bunkers), covered fortifications (ear-shaped caves and shelters), along with covered machine gun posts, observation posts, and other structures. This type of fortifications, which come in numerous forms, can be built with great speed using materials readily available and simple equipment, and offer a certain extent of protection against blast wave.

6.3.1 Open-Air Fortifications

Within open-air fortifications, overpressure is usually higher than that in an open space. The overpressure of the shock wave front increases due to reflecting multiple times within the fortification. The pattern of overpressure increase includes bigger increase at fortifications close to the center of explosion than fortifications farther from the center of explosion, bigger increase from walls facing the center of explosion than walls not facing the center of explosion, and bigger increase in deeper fortifications than shallower fortifications.

Although overpressure in such open-air fortifications is higher than on open ground surface, blast injuries suffered by personnel in fortifications are one to two levels milder than those exposed on ground surface. The crux of the matter is that fortifications basically nullify the effects of dynamic pressure. In addition, large peak overpressure reductions and shortened effective duration in battlefield fortifications such as ear-shaped caves might also contribute to this outcome. Therefore, it requires higher overpressure in such open-air fortification to cause the same severity of injury afflicted in an open space.

Under the effect of a violent blast wave, open-air fortifications might collapse, or covering materials might fall off, thereby causing indirect injuries to personnel stationed inside. In fuel-air explosive experiments, it was observed that communication trenches and other battlefield fortifications closer to the center of explosion collapsed from the explosion, burying all the test animals placed in such areas.

6.3.2 Covered Fortifications

Covered fortifications offer protection from shock waves because they have enough resistance and can stop shock waves from entering the interior of the structure, thereby sparing personnel inside from injury or lessening the severity of such injuries. Test results indicate that overpressure inside covered fortifications is merely 5–10% of that measured in open spaces. Bunkers are relatively weaker in terms of reducing shock wave, gunnery and observation fortifications have many openings and are even worse in terms of defending against shock waves, with overpressure equal to roughly 30–40% of that of ground surface. If blast door is damaged, then overpressure inside fortification would rise. On the contrary, if blast door is strong enough, the fortification's blast resistance would improve drastically, and overpressure inside fortification might only be 2–3% of that of ground surface.

A shock wave powerful enough could damage a covered fortification such as plugging up entrance and exit, blasting open cover plates of caves, damaging blast doors, deforming, displacing or fracturing the main structure, etc., which could cause indirect injury to personnel inside. Therefore, to prevent or reduce indirect blast injury, it would be advisable to enhance the strength of hole covers and blast doors, personnel should stay away from blast doors, and anti-pressure capacity of the main structure should be raised.

6.3.3 Air Raid Shelter

Most cities in China have been outfitted with air raid shelters. In normal times, these structures may be considered ordinary underground buildings and put to use so as to maximize their economic benefits. During wartime, they provide protection against blast wave and other injuring factors. In addition, air raid shelters provide rather sound protection against the shock wave and other injuring factors of a nuclear explosion. Common forms of air raid shelters include those dug into the ground, passageways, underground tunnels and as an annex to other buildings.

Air raid shelters dug into the ground can obviously weaken the effect of a shock wave. Within such a fortification, peak overpressure is lowered, pressure increase duration is prolonged, and form of pressure wave inside the fortification is changed. The entry/exit is often the weakest part of a fortification. Many air raid shelters have main structures featuring strong pressure-resistance capacity, but entry/exit is easily damaged by shock wave. Therefore, when designing air raid shelters, it is necessary to include protective cover plates of adequate strength and adopt design that ensure entry/exit would be not blocked.

An air raid shelter's shock wave energy dissipation capability largely hinges on its internal structure. When a shock wave passes through a passageway or tunnel and changes direction at a junction or bend, its overpressure would propagate along the different directions and attenuate. Experiments have proved that when a shock wave changes direction upon passing by a single bend, the overpressure on its front would reduce by a modicum. When the shock wave changes direction upon passing by two bends, its overpressure would be slashed significantly. The reduction and expansion in the cross-section of the passage that a shock wave passes by both have a clear influence on the shock wave's overpressure. When the cross-section of a passageway suddenly shrinks, a reflection of the shock wave would occur, producing a compressed air layer. When this layer flows toward the passage's reduced cross-section, it would create a shock wave with higher overpressure compared with that at passageway with larger cross-section. On the contrary, when a shock wave enters a passageway that suddenly expands in its cross-section, overpressure would drop quickly due to the sudden enlargement of the shock wave front. In addition, the rougher the surface of passageway and the smaller the cross-section of passage, the faster the shock wave's impulse attenuates.

6.3.4 Permanent Fortifications

Permanent fortifications refer to sturdy and durable fortifications constructed during normal times. These fortifications

are characterized by strong resistance, tight protection of holes and openings, and relatively comprehensive provision of equipment inside. Permanent fortifications are mostly situated underground and are mainly used as sites for battle command and providing shelter to personnel. Some other permanent fortifications are semi-underground or aboveground, and these are chiefly utilized as arsenals. Different types of permanent fortifications can effectively prevent shock wave from entering inside because of the configuration of one or multiple blast doors, along with shock wave reduction devices. The ventilation and filtering space is a weakness in such fortifications. Overpressure in these regions are usually higher than other areas, and personnel in these spaces are more exposed and easily injured by blast wave. Furthermore, surface burst could generate seismic wave that would directly transmit underground. At such times, personnel inside underground fortifications might suffer blast injury due to direct contact with the interior wall or floor of the fortification.

6.4 Protection of Weaponry

Things like armored vehicles and vessels are fitted with a variety of firepower and armament, and therefore they may be categorized as large-scale weaponry. At the same time, they are also characterized by a certain degree of shielding and enclosure, which can provide some amount of protection against a blast wave.

6.4.1 Armored Vehicles

Tanks are very enclosed and it is difficult for shock waves to enter, which is why overpressure inside a tank is lower than that of an open space. Due to the broad differences in tank model and equipment, overpressure inside tank could be somewhere from 7% to 70% of that of an open space. The pressure increase duration is slowed down from several or around ten or so milliseconds in an open space, to several hundred milliseconds inside a tank. There isn't much difference in the positive pressure effective durationDue to the relatively longer pressure increase duration, it requires higher pressure in a tank to cause the same severity of injury afflicted in an open space. Dynamic pressure usually doesn't act directly on crew members inside a tank, but dynamic pressure powerful enough could impart impact acceleration upon the armor, which would cause injury to body parts in direct contact with armor. Moreover, powerful dynamic pressure could displace or even flip over a tank, which might indirectly injure the crew members inside. Based on experiment observations, during a nuclear explosion, blast injuries suffered by test animals inside a tank are usually one to two levels milder than those injuries suffered by animals in the open field. Animals inside tank usually won't suffer from extremely severe injury, and the radius of injury and fatality also shrinks significantly.

6.4.2 Naval Vessel Compartment

Effects of a shock wave in such areas depend on pressure-resistance and seal performance. Other than having a lower overpressure than ground surface, the pressure increase duration inside vessel compartments is also longer, as slow as several hundred milliseconds to 1 s. Pressure effective duration is extended, usually at least double that of area outside compartment. Due to the aforementioned protection performance, shock wave injury and fatality range for test animals inside outside compartment is significantly shorter than ground surface, around 10–90% of the latter.

7 Blast Injury Protection Bottlenecks

As of present, explosion accidents are still hard to predict, and the power of the explosive and the relative distance from people are crucial limiting factors that determine the prognosis of victims. Therefore, blast injury protection should first and foremost start with ensuring rational cognition about risks of explosion among persons exposed to such risks. This is crucial to the effective dissemination and application of protection equipment. For military personnel and blasting workers, protection equipment are standard. However, for ordinary citizens that can't truly perceive the threat, they are often victims of severe injury or unable to protect themselves when a lethal explosion occurs because of personal financial reasons, lack of attention of limitations in available protection equipment. Therefore, analyzing from the perspective of social safety, people should be repeatedly informed and educated about any environment at risk of explosion, whether in production or daily life. In blast injury protection, sense of safety and protective performance of equipment material are equally important. Obviously, prevention is better than treatment.

A considerable amount of data has shown that in blast injury diagnosis, the power of explosive and distance of victim from center of explosion are the injuring factors that must be identified first. Next, environment of explosion (enclosed space or open space) and environment medium (air or liquid) are key factors that determine blast injury severity. And then, shock wave action upon solid interface (wall or fortification) could increase the destructive effect of the shock wave's pressure. In addition, the existence of obstacle or the lack thereof needs to be considered as an independent variable in making judgments about victim status. When conditions permit, ascertaining the factors outlined above would be highly valuable to the targeted and personalized configuration of blast injury protection material and equipment. In addition, knowledge about these factors are

also vital to determine the occurrence and severity of internal organ injury.

Past blast injury data indicate that in most weapon-related cases, blast injury occurs within a span of 2–10 ms. In times of peace, situations such as actual needs in production and the drastic increase in the quantity of dangerous chemicals in storage, once an uncontrolled explosion occurs, the duration of explosion is often quite long. Therefore, blast protection methods that target short-duration explosions in the past can't fully satisfy the blast injury protection needs in times of peace, it is necessary to adopt evidence-based medicine founded upon laboratory experiment outcomes, in conjunction with digital simulation and empirical assessment method, to make accurate judgments on efficacy and performance of protection mechanisms against long-duration explosions.

Upon realization of effective protection against blast injury, it should be noted that some types of such injuries are still hard to defend against because of the complicated internal relationships between force of explosion and severity of injury. In the real world, when personnel closer to the center of explosion didn't suffer serious injury does not necessarily mean that those farther away also do not suffer serious injury. Similarly, within the same distance, if certain personnel didn't suffer serious injury, it does not necessarily mean that those nearby were also spared from serious injury. Logically speaking, blast injury protection could cover up certain clinical signs of a victim's internal organ injuries. Therefore, to obtain accurate and comprehensive diagnosis, it is necessary to ensure objective and accurate descriptions provided by victim or accompanying persons, with the objective of first dealing with urgent, life-threatening injuries. In terms of physical inspections (respiratory rate, oxygen saturation, medical imaging, air embolism indications, etc.), perform dynamic observations if the need arises, and stay cautious against missed or delayed diagnosis, in particular paying attention to delayed medical signs. At the same time, remember preemptive diagnosis of complications associated with blast injuries (those related to genes, and biomarkers) and nonhereditary data of victim (age, gender, physiological condition, basic health status, etc.). Upon the basis of administering treatment to body injuries, diagnosis and treatment of psychological trauma require effective and appropriate measures and techniques (i.e., eye movement desensitization and reprocessing, Naikan therapy, reliefs for other psychological problem, among others).

However, the best treatment is prevention, and the even the best treatment isn't as effective as a preventative mentality. It is necessary to use new types of biological and chemical materials that can satisfy iterative attempts and repeated validations upon the basis of achieving lightweight, soft, fire-proof and thermal isolation functions so as to continuously enhance the blast wave energy absorption, redirection, and dissipation capacities of materials, which should provide effective protection by dealing with three aspects of shock wave, namely peak overpressure or underpressure, pressure effective duration, and pressure increase duration. The effectiveness, safety, and comfort of existing protective materials have much room for improvement.

In terms of protection against lung blast injuries in individuals, it would be advisable for workers to focus not only on effective personal protection, but also pay attention to potential explosives in the operation environment (dust, gas, flammable liquids, mobile phone, etc.), as these also play equally important roles in reducing how strongly and how long a shock wave acts on the body. These require scalability of protective materials that can realize economical application and adoption. Looking at past blast injury experiences, wounds from fragments or projectiles often result from a shock wave, therefore, in terms of protection efficacy, the necessity to not reduce or even improve fragment protection upon the foundation of effective shock wave protection means an inclination toward composite materials that offer projectile/fragment protection performance. Furthermore, in order to achieve quantified assessment of injury, it is necessary to obtain parameters of shock wave that impacted a victim, such as frequency and pressure. In this regard, it is an urgent need to research and develop intelligent preemptive warning devices.

Therefore, the difficulty in anticipating blast injury, the unpredictability of explosion environment, the complexity in injuring factors, wide range of variances in individual victims and differences in time and efficiency of rescue and treatment all attribute to the complexities in the translational research of blast injury protection. In particular, it is difficult to create appropriate composite materials. Meanwhile, injury evaluation indicators are urgently in need of optimization due to the ocean of data required in preemptive diagnosis, assessment, and rescue. These factors all compound the already immense challenge in blast injury protection.

8 The Future of Blast Injury Protection

As of present, there have already been a myriad of breakthroughs in the research and development of blast injury protection materials and equipment. In particular, the rapid advancements and cross-disciplinary efforts in material science, chemistry, biomechanics, and medicine will no doubt continue to elevate protection capacity against blast waves. Yet, since human perceptions of the material world will always be limited by depth and breadth of knowledge, compounded by subjective imaginations and cognition limitations about the unknown, obtaining an understanding about blast injuries infinitely close to the objective

truth is no walk in the park. In the real world, animals and plants have developed more effective protective tissues and structures compared to humans due to eons of evolution and the survival of the fittest in nature. These might provide inspirations and revelations for our own blast injury protection. Observations indicate that woodpeckers chisel woods at an impact speed of 6–7 m/s and an acceleration of approximately 10,000 *g* in order to find food and drill holes. Such behaviors bring about impact to the brain a thousand times its own weight, yet woodpeckers do not suffer head trauma. Research proves that other than a lower beak that is longer than the upper beak, and a "safety belt-like" hyoid bone, the sponge-like bone trabecula in the skull provides the "elasticity" that may buffer collision and absorb impact. Cartilage and muscle tissues outside the skull also function as shock absorbers. Similarly, many birds accidentally ram head-on into glass when flying, but they would shake it off as if nothing happened and keep on flying. In addition, the impact-resistance capacity of grapefruit is also worthy of investigation. As the largest citrus fruit, grapefruits can usually grow to a diameter of 15–25 cm and weighs up to 6 kg. Yet, even when dropped from a height of 10 m, grapefruits seem to be undamaged on the outside. Research proves that the unique structure in grapefruit peel can absorb tremendous amount of mechanical energy and bear impact in the range of thousands of dynes. The secret lies in the combination of two different kinds of biological tissues: an external layer that contains sebaceous glands, and a relatively thicker middle layer that looks like white sponge. In addition, the density of grapefruit peel layers change from the outside to the middle. This kind of tissue layer configuration with gradually changing density effectively prevents sudden changes in tissue composition, structure and mechanical performance, in turn lowering the possibility of tearing when tissue is exposed to impact. Therefore, efforts to lighten the weight of blast protective material structures may adopt bionics and microanatomy angles to explore the revelations from the above natural plant and animal impact-resistance structures.

It can be anticipated that within the current power of human beings, the foundation in blast injury protection is first and foremost to establish and cement a sense of safety and awareness, coupled with tireless, round-the-clock monitoring, prevention and control mechanism. As humans progress, it is inevitable that explosives increase in their diversity, and thus, injuring factors might become increasingly complicated. To deal with such "spears," we need to design the right "shields." Blast injury protective material and equipment need to stay updated and continually upgraded. In particular, with the prevalence of precision-guided, high-payload and deep-penetration munitions, only by employing comprehensive protection with multilayered structure and energy dissipation at every layer could the best blast impact protection effect be achieved. In addition, as material science and electronic technology continue to advance at a breakneck pace, defensive philosophies like smart protection and preemptive warning will certainly thrust to the forefront in blast injury protective material and equipment research. Without a doubt, any material and equipment revolution will be based upon mitigating or reducing injury, and the people-first protection mentality is the bedrock. The crux of the matter is to stick with a scientific mentality founded on practice, logical deduction and mathematical methods. Although the conversion and application of blast injury protection research outcomes still have much work to be done, the prospects are promising.

9 Conclusion and Vision

Blast injury and protection constitute a battle between "spears" and "shields." Following the rapid advancements in science and technology, new forms of opposition might emerge, but based on the philosophical viewpoint of "everything has its vanquisher," we must stay abreast of the latest developments, maximize strengths, minimize weaknesses, strive for dynamic balance and adjust on the fly as new types of explosives, in particular high-energy explosives and projectile explosives, emerge in the future. The purpose of diagnosis is to treat primary injuries, prevent complications, and restore victims to their full physical and psychological health. Protective strategy on the other hand is an expansion and forward extension of clinical treatment, and a "warning post" or form of proactive treatment driven by natural and unscalable human fear for the existing risks of blast injuries. The research, development, and upgrade of protective equipment need to actively take advantage of the world's new types of cutting-edge, multi-scale biological and chemical materials (polyurea composite or modified materials, nanomaterial with low environmental health risks, addition of high polymer, etc.) while considering preemptive warning based on protection parameters, types of organs to be protected, and assessment of protection performance. We firmly believe that explosion source monitoring and control form the basis of protection, and upon this foundation, the research and development of new types of equipment, and timely feedback and upgrade of such equipment, could possibly realize protection-treatment synergy and joint protection-administration progress in dealing with threats such as production accident, terrorist attack, and gas or mine explosions. It is necessary to earnestly elevate blast injury protection level in China from both the theoretical and technical approaches, and effectively improve the establishment of comprehensive blast injury protection system.

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Diagnosis of Blast Injury

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Blast injury is a variety of injuries caused by energy release when the overpressure of shock waves directly imposed on the body after explosion. It is a type of primary explosive injury. The range of a shock wave depends on the explosion intensity and surrounding environment. Blast injury appears mild outside but severe inside, with multiple injuries and commonly combined injuries, and severe visceral contusions. Such injury develops rapidly and is difficult to diagnose. Specifically, air-filled hollow viscera, such as lungs, gastrointestinal tracts, and acoustic organs are susceptible to injury, which is the key point of this chapter.

Blast injury mentioned here only refers to the first-level blast injury (i.e., primary blast injury). There may also be secondary blast injury (caused by projectiles), tertiary blast trauma (caused when casualties propelled and thrown up), and quaternary blast injury (all other explosion-related injuries except for the aforementioned three injury mechanisms) during the explosion. Among them, the quaternary blast injury includes burns, radiation exposure injuries, chemical injuries, inhalation injuries, suffocation, crush injuries, angina, high blood pressure, and psychological disorders, among others. Therefore, in clinical practice, medical history of the wounded from an explosion should conclude information about not only the primary blast injury (i.e. the first-level blast injury) but also a comprehensive assessment of injuries that may be caused by various mechanisms, including: (1) the secondary blast injury, an open injury formed by projectiles penetrating skin and into the deep soft tissues which may affect all parts of the body, such as primary projectile injuries like gunshot wounds and shrapnel wounds, as well as secondary projectile injuries caused by glass shattered by shock waves, fragments from the ruptured chamber, and objects raised by explosions; (2) the tertiary blast injury occurs when casualties are thrown up by shock waves which may affect all parts of the body commonly including fractures, traumatic amputations, craniocerebral injuries and trunk injuries; (3) the quaternary blast injury, including burns, radiation exposure injuries, chemical injuries, inhalation injuries, suffocation, crush injuries, angina, high blood pressure, and psychological disorders, etc.

1 Medical History Collection of Blast Injury

1.1 Medical History Collection of Blast Injury

The range of a shock wave depends on the explosion intensity and surrounding environment. Blast injury has a clear direction. Its severity is subject to explosion intensity, surrounding environment, and the distance between the wounded and the explosion point. The pressure amplitude produced by explosion is inversely proportional to the square of the distance from the explosion point. When collecting the trauma history, practitioners should obtain detailed information of the explosion site and the conditions at the time of injury, including the features of explosives, the distance between the casualty and the explosion center, the environment, whether there is potential exposure to toxic materials, etc. Explosion in open space can cause more serious injury in the lung nearer the explosion point, while an explosion in closed space or underwater can cause more serious bilateral lung injury and intestinal injury. Auditory organs are susceptible to blast injury, and the most vulnerable organs in an explosion as relatively low pressure can cause damage to the tympanic membrane. Therefore, medical workers should identify the position of the head and ears relative to the explosion point, whether there is cerumen or external protective equipment, etc. In addition to the clinical manifestations of the injury, it is important to know the management at the scene, in transit, and in other hospitals, including the confirmed or suspected injuries, the treatment given and its effect, etc.

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1.2 Clinical Manifestations of Blast Injury in Various Organs

1.2.1 Blast Lung Injury

Clinical manifestations of blast lung injury (BLI) depend on the degree of impact injury. The patients with BLI who survived and arrived at the hospital may be relatively stable in the early stage after the injury, and dyspnea or discomfort may occur within 2 h but may develop rapidly within a few hours to 24 h after the injury. Some patients may further evolve into acute respiratory distress syndrome. Blast lung injury often combines with pneumothorax, hemopneumothorax, and multiple rib fractures with corresponding symptoms and signs.

The clinical manifestations of BLI include symptoms of cough, hemoptysis, chest pain, dyspnea, and apnea, as well as signs of bradycardia, low blood pressure, cyanosis, dullness on percussion, low breath sounds with crackles, dry rales, and retrosternal friction sound audible, etc., ranging from temporary chest pain, chest tightness or suffocation in mild cases to cough, hemoptysis, or bloody sputum to severe symptoms and signs like dyspnea, cyanosis, restlessness, convulsion, asphyxia, a large amount of bloody frothy or bloody liquid in the mouth and nose, partial dullness found during percussion, weakened breath sounds, and a wide range of moist rales audible. The typical triad of "apnea, bradycardia and low blood pressure" may occur in some patients after injury, lasting for 30–120 s.

BLI can be accompanied by air embolism. According to the location of the embolism, patients could develop hemoptysis, blurred vision or blindness (retinal artery embolism), persistent chest pain, focal neurological deficit, epileptic seizure, coma, confusion (cerebral embolism), and changes in mental state. A huge embolism can lead to stroke, myocardial infarction, spinal cord infarction, cerebral infarction, intestinal ischemia, even death, and other serious consequences.

1.2.2 Gastrointestinal Blast Injury

The main results of abdominal blast injury are abdominal hemorrhage and peritonitis. Bleeding mainly occurs when the solid organs rupture, especially the rupture of the liver and spleen and injuries of blood vessels. Peritonitis is caused by the rupture of hollow organs with gastrointestinal contents overflowing into the peritoneal cavity. The following manifestations are different due to the different locations and conditions of injury.

1. **Abdominal pain**. It is the most common symptom which occurs immediately after injury, starting at the injured location and spreading to the whole abdomen. The perforation of the stomach, upper intestine, and gallbladder often causes diffuse acute pain, while the pain caused by colon perforation is mild and limited but likely to cause septic shock. In the treatment of delayed gastrointestinal perforation, abdominal pain may occur or worsen, accompanied by nausea, vomiting, and other gastrointestinal symptoms.
2. **Nausea and vomiting**. Nearly half of casualties with abdominal blast injury develop transient or persistent nausea and vomiting after injury.
3. **Peritoneal irritation signs**. Casualties with visceral rupture may develop peritoneal irritation signs such as tenderness, rebound pain, and abdominal muscle rigidity. Signs caused by internal bleeding are generally mild. Because of the overflowing bile, peritoneal irritation signs are more severe in liver rupture than that in spleen rupture. Peritoneal irritation signs are even more serious when the stomach and small intestine are perforated.
4. **Shock**. Shock can occur due to massive hemorrhage in the abdominal cavity or severe diffuse peritonitis, with hemorrhagic shock most common after injury. Septic shock may occur after a few days when special attention should be paid to delayed gastrointestinal perforation with more obvious peritoneal irritation signs of peritonitis.
5. **Others**. Hematuria can occur when kidneys and bladder are injured. Dark purple or bloody tarry stool may appear when intestinal mucosa is damaged or bowels are perforated. Fresh blood flowing from the anus indicates injuries in the colon or rectum. Patients with gastrointestinal perforation may develop subdiaphragmatic gas accumulation, pneumoperitoneum, and obliterated dullness margin of the liver, accompanied by bowel sounds disappearing, fever, and rapid pulse. Pelvic organ injury can stimulate the rectum causing frequent defecation desires. For casualties with abdominal blast injury caused by an immersion blast, many are accompanied with temporary lower extremity paralysis, possibly due to small blood vessel injury in the spinal cord.

Noteworthily, symptoms and signs such as abdominal pain and peritoneum irritation may not be obvious if analgesic drugs have been used during transit. What's more, additional attention is needed to the trauma of areas except for the abdomen, which obviously covers up the symptoms of abdominal injury. For example, unconscious patients due to brain injury cannot provide subjective symptoms of the abdomen. For a patient with combined injury of chest and abdomen, abdominal examination findings may be neglected due to attention fixed on the chest for its wound and dyspnea. Fractures of long bones of limbs often cover up the abdominal injury as well.

1.2.3 Auricular Blast Injury

In the event of hearing loss, tinnitus, earache, dizziness, bleeding in the external auditory canal, etc., auricular blast injury should be considered. Tinnitus occurs in most casualties with blast injuries in hearing organs and persists for a long time. Middle ear injuries are often unilateral, and inner ear injuries are mostly bilateral. Vertigo develops more commonly when the middle ear is damaged but lasts transiently, ranging from several minutes to several hours, while it is not common the inner ear injury. A small number of casualties may have symptoms such as transient nausea and vomiting or vestibular dysfunction.

1. **Traumatic tympanic membrane perforation**. It manifests as earache, tinnitus, hearing loss, bloody or watery secretions, even inflammatory secretions, and dizziness, etc. Tympanic membrane perforation is generally located in the tension part in an irregular shape. Conductive hearing loss may be more severe when combined with damage to the ossicular chain. Sensorineural hearing loss could also occur due to the rupture of the round window and the oval window caused by blast injury. Bleeding of the external auditory canal could develop, accompanied by tinnitus and cerebrospinal fluid otorrhea when combined with a temporal bone fracture.
2. **Blast-induced hearing loss**. Hearing loss is the most common symptom. Its severity is closely related to the proximity of the patient to the detonation source, detonation times, and detonation intensity. Conductive hearing loss may occur when the middle ear is damaged by explosive blast waves, such as the tympanic membrane rupture. Sensorineural hearing loss may develop when the inner ear and the auditory nerve are injured. Combined injuries of the middle ear, inner ear, and auditory nerves may result in mixed hearing loss. The hearing loss (transient or permanent) occurs asymmetrically and immediately after the explosion, which could gradually heal spontaneously in mild cases. In the early stage, ear discomfort, earache, and tinnitus appear, where tinnitus is mostly bilateral, persistent, and high-pitched, while deafness is progressively exacerbated, gradually evolving from the high-frequency area of 4 kHz to the speech frequency area. Sound waves induced by a blast act on the inner ear, damaging the outer hair cells first, and the inner hair cells degenerate due to ischemia, resulting in degeneration of cells and fibers of the Corti organ. Tinnitus is the main complaint of patients with such hearing loss, which seriously affects their quality of life.

In addition, due to the impact of explosive airflow, the body lifts up and falls down, resulting in ear contusion when it hits hard objects. A mild contusion is only manifested as skin contusion and subcutaneous ecchymosis. In severe cases, a subcutaneous hematoma can form, with auricle swelling and pain of varying degrees which may result in auricle cartilage infection and necrosis if not treated in time. Auricular cuts and lacerations could lead to varying degrees of auricular tear defects or complete dissection, manifested by local pain and bleeding. Massive bleeding is mostly caused by a break or rupture of the superficial temporal artery and the posterior auricular artery. Auricular burns are often combined with maxillofacial burns.

1.2.4 Clinical Manifestations of Other Blast Injuries

1. **Ocular blast injury**. It is mainly presented as visual impairment or loss. In mild cases, there may only occur burning sensation, photophobia, and blurred vision, while in more severe cases, temporary blindness may occur. In more severe cases, blast injury could immediately cause permanent blindness.
2. **Other thoracic blast injuries**. Perforation caused by esophageal blast injury could appear as chest pain, dyspnea, and subcutaneous emphysema after the explosion. Coronary insufficiency should be considered when coronary dysfunction symptoms occur, such as severe precordial pain, chest tightness, suffocation, cold sweating, etc. Patients with severe cardiac blast injury can develop acute left heart failure, which is manifested as sudden orthopedic breathing, cyanosis, frothy or pink sputum, and diffuse dry and moist rales audible in both lungs. Patients with coronary air embolism may develop various signs of acute myocardial infarction, such as severe precordial pain, shock, left heart failure, weakening of the first heart sound in the apical area, or the diastolic galloping rhythm audible.
3. **Blast-induced traumatic brain injury**. Blast-related traumatic brain injuries can range from mild dysfunction to complete loss of reactivity, but most patients have normal GCS (Glasgow Coma Scale) scores.
   (a) Blast-induced mild traumatic brain injury: It is mainly manifested as different degrees of deficits in higher cerebral functions, attention, perception, memory, expression, or executive ability, and in neurological specialty examination, varying degrees of impairment in cognition, speech, or logic. Persistent symptoms similar to post-traumatic brain syndrome or post-concussion syndrome manifested as headache, nausea, vomiting, dizziness, blurry vision, sleep disturbance, irritability, depression, fear, anxiety, emotional instability, etc. These symptoms are usually improved within a few hours or days after the injury, but the fear may last for several days or even longer.

(b) Blast-induced moderate traumatic brain injury: There is usually a long-term loss of consciousness and/or neurological function, possibly accompanied by long-time symptoms such as nightmares, insomnia, hypervigilance, and susceptibility to fright.
(c) Blast-induced severe traumatic brain injury: Clinical manifestations could be headache, vomiting, gradual increase in blood pressure, slow and shallow breathing, slow and strong pulse, and other symptoms of acute intracranial hypertension. Brainstem function may be exhausted when pathological respiration, rapid and weak pulse, and decreased blood pressure occur. When there are manifestations of traumatic shock, such as pale complexion, fast and weak pulse, undetectable blood pressure, irritability, etc., it is necessary to consider whether it is combined with other organ injuries. Local localization symptoms may also occur, including paralysis, motor, and sensory disturbances, aphasia, visual field defects, etc., caused by different parts of cerebral parenchyma.

2 Imaging and Laboratory Examination

2.1 Imaging and Laboratory Examination of Blast Lung Injury

2.1.1 Imaging Examination

Except for trauma history, symptoms, and signs, following assistant examinations are beneficial to the diagnosis of blast lung injury.

1. **Chest X-ray scan**. If conditions permit, all patients who have experienced an explosion should undergo a chest X-ray scan. The incidence of abnormal chest radiographs ranges from 52% to 91.7%. Lung lesions can appear in chest X-ray images within 4–6 h after injury, mainly manifested as typical patchy butterfly lung opacities. Diffuse lung infiltrates usually begin to dissipate gradually 24–48 h after injury. If the chest opacity expands after 48 h, it often indicates complications such as ARDS or pulmonary infection.
2. **CT scan**. It could show ground-glass opacities of unclear margins in lungs, which can be fused, and lung consolidation is noted sometimes. CT images are clearer than X-ray images, which can quantify the scope of lung injury. Studies suggest that mechanical ventilation is required when lung injury exceeds 28% of the whole lung, and mechanical respiratory support is needed for an average of 7 days when lung injury exceeds 45%, while mechanical ventilation is not needed with lung injury less than 18%.

In addition to diagnosing blast lung injury, images such as X-ray and CT could also assess its severity and find out abnormal signs like rib fractures, pneumothorax, hemothorax, foreign bodies, pericardial effusion, subcutaneous emphysema, and free gas under the diaphragm.

3. **Ultrasonography**. Ultrasonography can be used to diagnose hemothorax, pericardial hemorrhage, etc. Transesophageal echocardiography and Doppler technology are the most sensitive methods to diagnose air embolism, which could cooperate with the end-tidal partial pressure of CO_2 ($PETCO_2$), mean pulmonary artery pressure (mPAP), or transcutaneous oxygen tension ($PtCO_2$).

2.1.2 Laboratory Examination

1. **Arterial blood gas analysis**. In patients with severe blast lung injury, it is often seen a decrease in SaO_2, a significant reduction in PaO_2, a downward trend of $PaCO_2$ mostly, while for patients accompanied with chest wall injury, $PaCO_2$ increases. Sometimes, a decrease in SaO_2 can be detected even before the patient has any symptoms.
2. **Intrapulmonary shunt volume**. There are significant changes in the early stage after injury, the degree of which is basically consistent with the severity of the injury. The intrapulmonary shunt volume is mostly within 5% before the injury but increases up to more than 20% after the injury.
3. **Examinations for air embolism**. By ophthalmoscopy, bubbles could be noted in the blood vessels of the retina. The tongue is pale or ecchymosis, and the skin of the extremities is red and blue mottled. The electrocardiogram shows arrhythmia or myocardial ischemia. Brain CT scan sometimes shows low-density bands in the cerebral arteries.

Pizov et al. have combined chest radiographic presentation, oxygenation index (PaO_2/FiO_2), and whether there is a bronchopleural fistula to determine the severity of blast lung injury (Table 1).

Table 1 Severity grading of blast lung injury

Monitoring indicators	Severe blast lung injury	Moderate blast lung injury	Mild blast lung injury
Oxygenation index (PaO_2/FiO_2) (mmHg)	<60	60~200	>200
Chest X-ray	Massive bilateral lung infiltrates	Bilateral or unilateral lung infiltrates	Local lung infiltrates
Bronchopleural fistula	Yes	Yes/No	No

2.2 Imaging and Laboratory Examination of Gastrointestinal Blast Injury

2.2.1 Imaging Examination

1. **Abdominal X-ray (anterior–posterior supine/erect view)**. It can determine whether there is pneumoperitoneum. If pneumoperitoneum is found, gastrointestinal perforation can be diagnosed. Especially with a large amount of gas, there may be penetrating trauma in the stomach and colon. However, without signs of pneumoperitoneum, gastrointestinal perforation cannot be excluded. One of the signs of abdominal blast injury is dilated stomach and small intestine by gas on plain film, with an incidence rate of 8.1%.
2. **B-ultrasonography**. Focused assessment with sonography for trauma (FAST) mainly examines the pericardium, right upper abdomen, left upper abdomen, and pelvic cavity, by which a positive result is defined as 250 mL of free fluid noted and free fluid after trauma is regarded as hemorrhage. In the case of blunt injuries such as abdominal blast injuries accompanied by hypotension, FAST assessment could be so accurate that it can even determine whether emergency surgery is required. However, for patients with stable hemodynamics, if there is a high risk of potential injury in the abdominal cavity, a CT examination should be performed even if FAST has been performed. Nevertheless, ultrasonography is often limited due to gaseous dilation after abdominal primary blast injury (PBI) and is dependent a lot on ultrasound technicians.
3. **CT scan**. There are no studies that specifically explain the role of CT in diagnosing abdominal blast injury, while images mostly show blunt abdominal trauma caused by other reasons. CT is the preferred imaging evaluation method for patients with severe trauma and stable hemodynamics. For these patients, CT scans should cover the head downwards to the middle of the thigh, including neck, chest, abdomen, and pelvis, identify and quantify the injury and hemorrhage of solid viscera in the abdominal cavity, guide the non-surgical management of solid organ injury, and improve the diagnosis rate of hollow organ injury through abdominal effusion, extravasation of intestinal contrast agent, intra-abdominal or retroperitoneal gas, etc. The accuracy of multi-slice spiral CT is higher than that of abdominal X-rays in the diagnosis of most abdominal injuries. Increasing attention has also been paid to Its application in the diagnosis of abdominal blast injury.

2.2.2 Laboratory Examination

1. **Complete blood count (CBC)**. It is helpful for diagnosis. Progressive internal hemorrhage results in a gradual decrease in hemoglobin; peritonitis is linked to a sharp increase in neutrophils with a left shift.
2. **Liver function tests**. Serum alanine aminotransferase activity increased sharply within 2 h after liver rupture and reached two to five times that before injury at 12 h after injury, and remained at a high level 72 h after injury.

2.3 Imaging and Laboratory Examination of Auricular Blast Injury

2.3.1 Imaging Examination

CT scan could diagnose the location, type, and severity of temporal bone and ossicle fractures. The three-dimensional reconstruction technique of the ossicles is relatively intuitive for ossicle trauma, of great diagnostic value.

2.3.2 Laboratory Examination

Patients with severe blast injuries are generally more seriously injured. Therefore, the foremost is to save their life. After the condition is stabilized, an audiometric assessment is needed for hearing damage.

1. **Otoscopy and electric otoscopy**. For external ear canal injury and traumatic tympanic membrane perforation, the position, area of eardrum perforation, and effusion and bleeding in middle ears can be visually seen through electric otoscopy.
2. **Auditory function examination**. Pure tone audiometry often shows a conductive hearing loss in traumatic tympanic membrane perforation and sensorineural hearing loss in inner ear injury. Otoacoustic emission could reflect the functional status of the outer hair cells of the cochlea. The detonation shock wave and the noise generated by the explosion may damage the outer hair cells of the cochlea, causing the acoustic reflection threshold to be less than the background baseline. Electrical response audiometry is used to detect various biological potentials generated during the transduction of sound waves in cochlear hair cells and their transmission through auditory nerves and auditory pathways to the auditory cortex. Auditory brainstem response (ABR) combined with pure tone audiometry can reflect the hearing loss caused by blast injury more objectively.
3. **Vestibular function examination**. The trauma teams can objectively assess the vestibular damage of patients with blast-induced hearing loss by vestibular function examinations with great significance for determining the degree, scope, and management of blast-induced auditory organ injury.

2.4 Imaging and Laboratory Examination of Other Blast Injuries

2.4.1 Imaging and Laboratory Examination of Ocular Blast Injury

1. **CT scan**. It is the gold standard for diagnosing ocular blast injuries, especially penetrating trauma. Its diagnostic sensitivity is 71–75% for concealed open ocular injuries. Explosive fragments are not necessarily made of metal so that they may not be displayed in CT images. The patient should always undergo a CT scan of the eyes earlier than MRI to avoid serious damage caused by concealed metal foreign bodies during the MRI.
2. **Ultrasonography**. It can also provide a rapid diagnosis of eye injuries. Since extra pressure acting on the open eyeball may cause further damage, it is recommended to limit its application in penetrating eye trauma.
3. **Ophthalmoscopy and slit-lamp microscopy**. Changes like conjunctival edema, congestion and hemorrhage, pupil reduction, and corneal opacity can be seen in the anterior segment of the eye. Retinal hemorrhage, edema, and exudation can be seen in the fundus, and manifestations like retinal detachment can be seen in severe cases.
4. **Fluorescence fundus angiography**. In the early stage after injury, it can be noted that the choroid was filled slowly with or defective in fluorescence at the posterior pole of the injured eye. At 3 and 6 h after the injury, capillaries on the surface of the optic disc are dilated with fluorescein leaked, and the optic disc fluorescence gradually increases. During the later period, a high degree of fluorescence infiltration can be seen in the vitreous, and the optic disc shows a strong fluorescence phase. At the same time, corneal opacity and edema aggravate with obvious corneal fluorescence staining.
5. **Electroretinogram (ERG)**. There is a significant decrease in the amplitudes of the a-wave and the b-wave, and prolonged peak latent time of the a-wave of the injured eye, indicating suggesting that the visual function is obviously impaired.

2.4.2 Imaging and Laboratory Examination of Other Thoracic Blast Injuries

1. **Barium meal examination**. Barium meal examination could help for diagnosis when esophageal damage is suspected.
2. **Electrocardiography**. After the cardiac blast injury, the electrocardiogram (ECG) shows a decreased heart rate and prolonged systolic and diastolic periods, which is maintained at about 60% of the normal heart rate within 1 week and recovers significantly half a month after the injury. Presentations like pathological Q wave and ST-T wave changes can be seen in patients with myocardial infarction to identify the location and extent of the infarction and observe the therapeutic effects.
3. **Myocardial injury markers**. For patients suspected of myocardial infarction, markers can be detected, such as serum aspartate aminotransferase, creatine phosphokinase (CPK), and its isoenzymes (CPK-MB), of which the increased activity combined with typical changes in ECG can help diagnose cardiac blast injury.

2.4.3 Imaging and Laboratory Examination of Blast-Induced Traumatic Brain Injury

For patients with blast injuries with obvious body surface trauma, attention should be paid to whether they have a blast-induced traumatic brain injury. If conditions permit, patients should undergo relevant diagnostic examinations as soon as possible to identify the diagnosis and grade of the injury, including X-ray scan, CT scan, and MRI scan of the brain, lumbar puncture, etc. If necessary, laboratory tests could also be of great reference significance in assessing cerebral function and the overall conditions after the injury.

1. **X-ray scan of the brain**. Cerebral and cervical X-rays can reveal fractures, intracranial gas, metal foreign bodies, etc.
2. **CT scan of the brain**. It is mainly used to determine the extent and depth of fractures and the relationship with the dura and brain parenchyma; at the same time, it can be applied to clarify the severity of brain contusion, the location, and size of the hematoma. CT angiography can also be used to diagnose cerebrovascular damage. It is suitable for diagnosing severe blast-induced traumatic brain injury, while head CT images are normal in 85% of mild cases.
3. **MRI scan of the brain**. It is suitable for diagnosing mild blast-induced traumatic brain injury, with the positive detection rate (sensitivity) 50% higher than that of a CT scan. MRI can show a small amount of bleeding in the corpus callosum or cerebral cortex, better than CT in the diagnosis of diffuse axonal injury. T2-weighted MRI and fluid-attenuated inversion recovery (FLAIR) imaging of MRI are helpful for the diagnosis of non-hemorrhagic brain injuries. Susceptibility weighted imaging (SWI) is more valuable for the detection of microhemorrhage. Diffusion tensor image (DTI) can quantitatively analyze the extent and severity of brain white matter damage. Functional MRI (fMRI) can record the recovery process of the brain.
4. **Evoked potential examination**. It provides a basis for the objective evaluation of the nervous system functional status of patients with a blast-induced traumatic brain injury without positive signs in the nervous system or abnormal manifestations in the CT scan.

5. **Electroencephalogram (EEG)**. The diagnostic effect of EEG is not affected by coma, sedatives, or muscle relaxants, playing an important role in determining the prognosis of patients.
6. **Lumbar puncture and intracranial pressure monitoring**. Lumbar puncture is conducted to measure intracranial pressure, and it can also be used to diagnose and treat subarachnoid hemorrhage and intracranial infection. According to the objective data of intracranial pressure, practitioners can detect intracranial hematoma and cerebral edema early to guide further management.
7. **Laboratory examinations**. Laboratory examinations are of certain value in assessing cerebral function and overall conditions after injuries, such as relevant biochemical indicators (S-IOOB, lactic acid, free radicals, excitatory amino acids, creatinine, blood transaminase) in arterial blood gas analysis, electrolytes, cerebrospinal fluid (CSF), and plasma.

3 Key Points for Diagnosis of Blast Injury

3.1 Assessment of Severity of Blast Injury

Severe blast injuries can only be manifested as minor external trauma. The goal of on-site triage is to sort out patients with severe blast injuries based on the conditions on-site. Tympanic membrane rupture was once considered a marker of severe blast injury, but data from the US military during the Afghanistan and Iraq wars show that tympanic membrane rupture has a poor correlation with the prognosis. Tympanic membrane rupture is typical of blast injury. Eardrum perforation represents a blast injury related to the intensity of the detonation and the direction of the ear at the detonation time. Tympanic membrane rupture is significantly related to the consciousness disturbance caused by cerebral concussion. In the absence of resources, patients with simple tympanic membrane perforation can be discharged after undergoing a chest X-ray and a period of medical observation, but the ideal observation period has not yet been determined.

In addition to tympanic membrane perforation, some other signs have also been used to predict the blast injury, including subpharyngeal petechiae or ecchymosis, gas embolism of retinal artery in the fundus examination, and subcutaneous emphysema. Current studies suggest that more accurate evidence for predicting severe internal blast injuries includes the following: four or more body surface injuries, burns over 10% of body surface area, cranial or facial fractures, and penetrating trauma of brain or trunk.

3.2 Key Points for Diagnosis of Common Blast Injuries

3.2.1 Blast Lung Injury

The lung is one of the most vulnerable target organs of blast injury. Shock waves can pass through the chest wall and airways, which is the main mechanism of pulmonary blast injury (PBI). The incidence of blast lung injury among survivors was 0.6–8.4%, while the incidence of blast lung injury in the Iraq war was 7.3%, and that in the Afghanistan war was 11%. Blast lung injury has the characteristics of mild external manifestations but severe internal conditions, rapid development, etc., seriously threatening patients' life with a mortality rate of 11%. Common blast lung injuries are lung contusions and barotraumas that can cause pneumothorax, pneumomediastinum, interstitial or subcutaneous pneumatosis.

According to the blast injury history and clinical manifestations such as cough, chest pain, hemoptysis, and dyspnea after the injury, blast lung injury should be suspected. The decline in oxygen saturation can be seen after the injury or as late as 48 h after the injury. Blood gas analysis suggests progressively decreasing blood oxygen partial pressure and blood oxygen saturation. All the casualties of blast injury should undergo a chest X-ray or CT scan. Blast lung injury is typically manifested as batwing-like infiltrates, which can be initially diagnosed.

3.2.2 Gastrointestinal Blast Injury

Gastrointestinal blast injury occurs in 0.3–0.6% of survivors, and the terminal ileum and cecum are the most commonly affected organs. The injured may not suffer any abdominal pain at all at first, but in clinical practice, we should be careful to exclude abdominal injuries, including those with the intact tympanic membrane. Delayed gastrointestinal perforation can occur 14 days after the blast injury, most commonly at 3–5 days after the injury. In most cases, diagnosis can be confirmed based on trauma history, clinical manifestations, and auxiliary examinations. Even today, when imaging technology has been highly developed, the abdomen is still the last black box. The key to diagnosis is to perform clinical examinations dynamically, including physical examination, abdominal X-ray (supine/erect), ultrasonography, CT scan, etc. The accuracy of peritoneal irritation is only 50% due to injuries to other parts like limbs and sedatives. For patients with progressive aggravation of abdominal signs and unstable hemodynamics, delayed gastrointestinal rupture should be highly suspected, and laparotomy or laparoscopic exploration is needed for final diagnosis.

1. **Diagnostic abdominal paracentesis**. This examination can be done when the blunt abdominal injury is suspected.

A positive result is defined as bloody fluid aspirated with a positive rate up to 83.0–97.7%.

2. **Diagnostic peritoneal lavage**. When there is a small amount of blood or exudates in the abdominal cavity, abdominal paracentesis is often negative, and peritoneal lavage can be used at this time. It is regarded to be positive when the lavage fluid is light red or under the microscope, the red blood cell count above 0.1×10^{12}/L, or the white blood cell count above 0.5×10^{9}/L, or findings of bacteria, bile, or vegetable fiber in the lavage fluid. The diagnosis rate of this method can reach 97%.
3. **Urinary catheterization**. Urinary catheterization should be done when a bladder injury is suspected. Clear and bloodless urine exported indicates that there is no damage to the bladder, while a large amount of bloody urine suggests injuries to the bladder, ureter, or kidney; if there is no urine or only a small amount of bloody urine exported, 50–100 mL sterile isotonic solution can be injected through the catheter, and aspirated after a few minutes. If the aspiration volume is significantly less than the injected volume or there is blood in the aspiration fluid, it proves that there is a rupture of the bladder.
4. **Laparoscopy**. It is suitable for adult patients with stable vital signs that require exploratory laparotomy. Stable vital signs mean that the systolic blood pressure is above 90 mmHg (1 mmHg = 0.133 kPa), the infusion volume is less than 2 L, and the GCS is more than 12; the need for laparotomy means that a clearly diagnosed or highly suspected visceral injury in the abdominal cavity by clinical presentations and auxiliary examinations. Abdominal viscera can be observed under direct vision by laparoscopy, and if used when conditions permit, it can enhance the diagnosis rate and reduce the negative rate of exploratory laparotomy.

3.2.3 Auricular Blast Injury

Blast-induced hearing impairment is easy to be neglected despite its high incidence rate. All casualties who have experienced the explosion should undergo an otological assessment and otoscopy. The injury depends on the direction of the ear at the time of the detonation. Tympanic membrane perforation is the most common injury to the middle ear. Signs of ear impairment usually appear during the initial evaluation. Any patient presenting with hearing loss, tinnitus, earache, dizziness, external auditory canal hemorrhage, tympanic membrane rupture, or purulent otorrhea should be suspected of ear injury. The symptoms of auricle injury are more apparent, which is not difficult to diagnose. Injuries to the inner ear and the middle ear are relatively hidden. Such patients should undergo related audiological, neurological, and imaging examinations according to the specific conditions to initially identify the location of the injury. Otoscopy and audiometry are conducive to the diagnosis.

3.2.4 Other Blast Injuries

1. **Ocular blast injury**. Although the surface area of the eye is relatively small (close to 0.1% of the total body surface area), eye trauma after detonation is not hard to see. There are 10% of survivors with apparent ocular injuries. The diagnosis is mainly based on the trauma history and clinical symptoms and signs. Ultrasonography, CT scan, ophthalmoscopy and slit-lamp microscopy, fluorescent fundus angiography, electroretinogram, and other examinations can be performed, if necessary. The most common are foreign bodies in the eye, corneal abrasions, eyelid or periorbital abrasions, retinal detachment, orbital fractures, and eyeball rupture.
2. **Other thoracic blast injuries**. Cardiac blast injury can be diagnosed based on the trauma history, clinical presentations, electrocardiogram, aspartate aminotransferase test, creatine phosphokinase, and its isoenzymes test, etc.
3. **Blast-induced traumatic brain injury**. The incidence of blast-induced traumatic brain injury is about 3%, ranging from mild to fatal. More obvious injuries include subarachnoid hemorrhage, subdural hemorrhage, brain, and meningeal hyperemia. The diagnosis is mainly based on the trauma history, clinical symptoms, and signs, CT scan, etc. Since most patients with blast-induced traumatic brain injury present with mild brain injury or cerebral concussion, they often develop many symptoms but few or no signs. In addition to the neurological examination, a neuroelectrophysiological examination and a psychological evaluation are necessary if possible. A few patients with blast-induced traumatic brain injury are severely injured and need to undergo necessary neuroimaging examinations, such as head CT or MRI scan, according to the individual conditions.

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Medical Treatment in Echelons of Blast Injury

Zhaohui Huang

Medical treatment in echelons, also known as step treatment, is used when there are batches of the wounded and under unstable treatment environment, referring to an organization form that the treatment activities for the wounded are carried out continuously and in stages.

With the evolution of weapons of war, the factors that cause weapon injuries are also changing where modern warfare injuries and blast injuries caused by or combined with explosions have attracted attention due to the increase in the incidence of injuries and deaths. The theory of medical treatment in echelons for blast injury in wartime is proposed in the context of war, and has been fully verified and developed in the existing war practice, which reveals the basic characteristics and laws of the medical evacuation work of the injured in wartime, playing a significant role in improving the efficiency and effectiveness of the medical evacuation of the injured during the war.

Blast injuries are usually caused by terrorist activities or production accidents, and the organization principles and forms of treatment for the injured also follow the law of medical treatment in echelons. Medical treatment in echelons for the injured, in view of the increasing uncertainty of the detonation site and the relatively abundant resources of medical evacuation, should be conducted based on the thinking method of echeloning and labor division and the work method of continuous medical security. The overall echelon, based on the two levels of the on-site hospital, with the in-hospital labor division organized flexibly according to the characteristics of the hospital's business division, should be organized and implemented according to the principles of organizational classification, division of labor and continuous proceedings.

To learn the theory of medical treatment in echelons of blast injury, it is necessary to grasp the theory significance in organization and guarantee. Organization theory mainly includes the classification rules of the treatment system, the division rules of the task system, the general rules of the organization work system and work methods, etc.; the guarantee theory mainly includes the organization rules of the treatment technology, and the general rules of guarantee mechanism and technical support.

1 Summary of Medical Treatment in Echelons Theory

1.1 History of Medical Treatment in Echelons Theory

The emergence of medical treatment in echelons is closely related to the development of war patterns. In the cold weapon era, treatment was generally implemented on site, while when entering the firearm era, the injured were transported to the hospital for treatment. During the Franco-Prussian War (1870–1871), the staged medical treatment and evacuation has been formed where the injured were sent to the hospital in the rear of the battlefield after being simply treated in the battlefield. In 1916 (during the First World War), the idea of step treatment proposed by the Russian surgeon, V·A·Opel, has become a common therapeutic strategy adopted by the armies of the world since the Second World War. In 1965, the academic circles of our military renamed "step treatment" as "medical treatment in echelons" with the same essence and connotation.

Medical treatment in echelons is a product of conflicts between the treatment conditions in wartime and the requirements for management of the injured. There are many wounded personnel in wartime, with complicated and

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severe injuries which need timely and good treatment urgently; however, due to the limitations of wartime conditions, well-equipped medical institutions are not convenient to approach the front line, and it is impossible for a large number of the injured to stay near the battlefield for long periods of time for treatment. Therefore, the wounded in wartime cannot be treated by one medical unit from admission to discharge as usual, while, instead, the complete treatment process must be separated in time and space, and carried out by several treatment agencies together. The injured are initially treated by a medical organization close to the front line, and received complete and appropriate treatment step by step as they are evacuated, until the entire treatment is completed. It can be seen that medical treatment in echelons refers to an organizational form with grading, division of labor, and continuous proceedings in management, which is gradually improved from a low level to a high level in technique. The emergence of medical treatment in echelons is a great development and contribution to the medical evacuation system, which organically combines medical treatment and evacuation, so that the injured can receive complete management step by step during the evacuation process, until the entire treatment process comes to an end. This unifies the contradiction between the special conditions in wartime and the requirements for the treatment of the injured.

1.2 Main Content of Classic Medical Treatment in Echelons Theory

According to the characteristics of the wounded in wartime, the battlefield environment, and the special requirements for the treatment of war wounds, the whole process of the wounded treatment must be divided and implemented in stages. Emergency treatment should be carried out in the fire line, and complete and thorough treatment should be carried out in the rear hospital.

When organizing the step treatment of the wounded, military characteristics must be taken into consideration such as military operations, troop formation, and battlefield environment. The medical treatment in echelons should be consistent with combat missions and military deployment, coordinated with the combat style and characteristics of the military action, connected with the establishment system for troops, and compatible with the terrain and traffic conditions of the battlefield.

The treatment and evacuation of the wounded in wartime should be continuous and successive. The previous medical institutions should create conditions for further management in subsequent medical institutions so that the injured can finally receive complete and thorough treatment. Besides, the treatment and evacuation of the wounded must be organized and implemented under continuous medical monitoring without interruption.

1.3 Basic Viewpoints of Medical Treatment in Echelons Theory

1.3.1 Certain Applicable Environment and Conditions for Medical Treatment in Echelons

The organizational form of medical treatment in echelons is not required under any environmental conditions, but an organizational form and guarantee principle adopted under special conditions, which can usually be summarized as unstable treatment environment, mass casualties, and limited health resources.

1. **Unstable treatment environment**. The environment of the treatment site is complicated and unstable. On the one hand, the natural and geographical environment of the scene is various, with changeable meteorological and climatic conditions, complex topographical and geomorphic features, which cannot meet the medical requirements for thorough treatment; on the other hand, the treatment place may be damaged and attacked at any time (especially on the battlefield), so that the treatment area needs to be changed frequently.

   Unstable on-site environment is the basic prerequisite for medical treatment in echelons, that there are many unstable factors at the scene of explosion, no matter in peacetime or wartime the wounded need to be evacuated quickly after emergency treatment on the scene.
2. **Mass casualties**. Mass casualties occur suddenly and intensively with complicated injuries, various injury conditions, and urgent treatment tasks, so that it is impossible to treatment all the injured simultaneously at the scene.

   The casualties with blast injury in wartime often occur in batches, while the number of the injured is determined by the power of the explosives and the intensity of the personnel in peacetime. There are usually multiple wounded at the same time, which puts pressure on the medical capacity.
3. **Limited health resources**. It is mainly targeted for wartime, when health resources at the treatment site are limited, medical equipment is incomplete, high-level treatment medical personnel and equipment is difficult to reach the scene, and medical staff can only carry a few medicines and equipment, and many examinations and diagnostic measures are difficult to implement. Under such environment and conditions, the wounded must be organized and treated according to the medical treatment in echelons.

1.3.2 The Basic Characteristic of Medical Treatment in Echelons Is Step and Continuous Organization and Implementation with Labor Division

According to the characteristics of the explosion and treatment environment of the injured, as well as the special requirements for the treatment of the wounded, the work of injury treatment and protection should contain following characteristics.

1. **Hierarchical deployment**. Phased management should be adopted for organization with hierarchical deployment, when the organization system of the injury treatment is established.
2. **Division of labor during treatment**. Medical institutions at all levels should be divided according to different levels of functions, following the principle of level management and level-by-level improvement, when the technique system of the injury treatment is established.
3. **Continuous proceedings**. It is a continuous and seamless process of organization and implementation from the front to the rear, from the unstable environment to the stable environment, from on-site first aid to early treatment, to specialist treatment.

1.3.3 Medical Treatment in Echelons Is a Theoretical Principle

In the practical application of the theory of medical treatment in echelons, the following two aspects should be emphasized.

1. **Medical treatment in echelons, as a theoretical principle, should be used flexibly in practice**. The medical treatment in echelons, especially for blast injuries, does not refer to a specific organizational form. Echelon is an organizational principle, the regulatory requirements and guidelines for organizational work, which must be followed. Designing echelons is a specific method of organizational work, which is formed under specific environmental conditions. There are various organizational methods in various situations.
2. **Flexibly grasp the application of treatment to give full play to its effectiveness**. In medical treatment in echelons, the complete activity and process of treatment should be guaranteed by different people in stages, which is another basic principle. However, the technical scope and specific measures of each stage need to be flexibly determined according to the actual situation. From the perspective of the law of the application of medical science and technology, if the "principle" is regarded as the "rule," it is easy to simplify the medical technology problems and even lead to rigid application.

1.3.4 Determine Echelons Based on the Technical System as the Main Line

In the practical application of medical treatment in echelons, there are two echeloning ideas and methods. The traditional approach is to echelon and standardize the medical support organization system as the main line, that is, to design assignments according to organizations. The modern theory of medical treatment in echelons advocates the echeloning and standardization based on the treatment system as the main line, that is, to determine organizations according to assignments. Treatment agencies at all levels respectively undertake one of the above-mentioned treatment tasks according to the battlefield environment and support capabilities, and the specific division of tasks shall be determined by the leadership. The advantages of such an organization and standard method include better reflecting the essence of the treatment work due to its need-oriented idea, better corresponding to the law of treatment for the wounded by involving the basic process from emergency treatment to rehabilitation, suiting the complex and varied medical environment, and effectively avoiding problems of over-echeloning and low efficiency, which is more flexible, better satisfying the requirements of technical specifications.

1.3.5 During the Medical Treatment in Echelons, First Aid Is the Key, and Definitive Treatment Is the Foundation

Whether in the wartime or peacetime, in the medical treatment in echelons, on-site first aid is the foundation of the entire treatment, which is the key to reducing deaths, improving the cure rate and the overall quality of the treatment. Definitive treatment is designed to completely eliminate the causes and complications of the wounded, which is a fundamental measure to maintain the lives of the wounded and restore their functions. Multiple previous treatment measures can also be regarded as a foundation to provide opportunities for definitive treatment, which is the real solution to the problem that completely eliminates the causes and complications of injuries, so that medical personnel must regard it as the core and a key point of the treatment of the wounded.

1.4 Basic Requirements for the Medical Treatment in Echelons of Blast Injuries

According to the characteristics of medical treatment in echelons, the relationship between the complete treatment requirements of the wounded and the implementation of labor division at all levels must be correctly handled in order to reduce the influence of evacuation on the timely implementation of treatment during the organization and implementation of medical treatment in echelons of blast injuries.

Therefore, the following three basic requirements should be followed.

1.4.1 Rescue the Wounded Quickly and Timely for Early Recovery

The rapid and timely treatment can effectively prevent the deterioration of the injury so as to save the lives of the critically injured, and promote the recovery of the injury, striving for a good prognosis and an increased healing rate. (1) On scene care by quickly transporting and evacuating the injured. It is necessary to strengthen the organization schedule of evacuation, including improving transportation conditions by using multiple means of transportation (such as ambulances, armored ambulances, and helicopter ambulances) to eliminate various factors that may affect timely evacuation. (2) In wartime, set up rescue and treatment institutions as close as possible to the frontline for timely treatment of the wounded as per the combat requirements and with due regard to safety conditions. (3) Strengthen the internal organization of the rescue and treatment institutions, simplify the rescue and treatment operations, improve work efficiency, and shorten the time for the casualties to stay at each rescue and treatment unit.

1.4.2 Make Processes Continuous to Ensure the Quality of Treatment

In order to ensure the quality of medical treatment in echelons, in addition to sufficient technical and medicinal guarantees, it is also necessary to ensure that the treatment work at all levels is carried out successively, so that the entire treatment work is not interrupted and the treatment at all levels is not repeated. Each process should make a good preparation for its next process, to create opportunities and time; the next process should supplement the uncompleted work on the basis of the previous level, and adopt new measures to make the treatment connected closely and gradually expanded and improved.

The above are achieved by (1) reinforcing military medical training and unifying the academic views; (2) requiring rescue and treatment institutions at all levels to establish a holistic concept, earnestly observing the rules of treating blast injuries, and correctly implementing the scope of treatment; (3) filling out the medical documents of a uniform format in accordance with provisions to ensure coherence.

1.4.3 Complement Each Other by Combining Medical Treatment And Evacuation

Medical treatment and evacuation are complementary and indispensable, which must be organically combined, and their relationship must be dealt with dialectically. In terms of the outcome of the wounded, medical treatment is the leading factor while evacuation is auxiliary, where active medical intervention is a must in order to completely heal the wounded, especially for the wounded in need of emergency treatment, active medical treatment must be implemented. Evacuation is for medical treatment, which will be meaningless without subsequent medical work. Therefore, on the whole, medical treatment is the dominant aspect of medical evacuation. But such primary and secondary conflicts may also change. Before the wounded receives definitive treatment, medical treatment is only to ensure the safe evacuation of the wounded. Besides, in different battles, at different stages, or under specific circumstances, evacuation may become the main contradiction. Health service leaders must take advantage of the times and circumstances to organically integrate medical treatment and evacuation, instead of stick to the accustomed rules.

2 Types of Medical Treatment in Echelons for Blast Injury

2.1 Kinds of Medical Treatment of Classic War Injury

In order to meet the needs of medical treatment in echelons, the treatment of the wounded is classified according to the nature and complexity of treatment technical measures, namely the kinds of medical treatment, which is the main basis for the management in echelons of war wound treatment technology. Our army currently conducts the management in echelons in accordance with five kinds of medical treatment, that is, on-battlefield (on-site) first aid, medical treatment, forward resuscitative surgery, definitive treatment, and rehabilitative treatment.

2.1.1 On-Battlefield (On-Site) First Aid

On-battlefield (on-site) first aid refers to the initial temporary first aid taken near the injured location in order to save the lives of the wounded, improve the injury condition and prevent complications, mainly including ventilation, hemostasis, bandaging, fixation, transportation, and basic life support. First aid is the starting point of medical treatment in echelons, which directly affects the prognosis of the wounded and the effect of subsequent treatment. The first aid in wartime is a heavy task, and often carried out under direct threats such as firepower from the enemy, radioactive contamination, and chemical pollution, combined with medical treatment of health personnel of company and battalion and self-treatment and mutual treatment of commanders and fighters.

2.1.2 Medical Treatment

Medical treatment refers to emergency treatment measures taken to save the lives of the wounded, prevent the deterioration of the injury, and ensure the safety of evacuation, which

is a supplement to and correction of first aid, and is essentially first aid, but a more specialized primary first aid. It concludes triage and professional first aid for different injuries on the basis of the six major technologies, including some simple surgeries. It is usually completed by medical sergeants and military doctors of units below the brigade on the battlefield (site) or regiment or equivalent treatment institutions.

2.1.3 Forward Resuscitative Surgery

Forward resuscitative surgery is a formal treatment carried out on the basis of a clear diagnosis, which is a treatment mainly based on emergency surgery, including a series of relatively complete emergency treatment measures, playing an important role in the treatment of the wounded. It is usually completed by the brigade or equivalent treatment agencies.

2.1.4 Definitive Treatment

Definitive treatment refers to the treatment of the wounded by specialists with specialized technical means in the hospital. It is a fundamental treatment to completely eliminate the cause of injury and threats of life in a relatively stable environment and complete equipment conditions, which, as a decisive link in the treatment of the wounded, plays an important role in improving the cure rate and reducing the disability rate. In our military, the definitive treatment is usually organized and implemented by strategic rear hospitals, specialized hospitals deployed in base military stations behind the battle, general hospitals with finer divisions, and requisitioned designated local hospitals. In addition, field hospitals strengthened by higher-level specialist medical teams and surgical teams can also provide corresponding definitive treatment.

2.1.5 Rehabilitative Treatment

Rehabilitative treatment is a kind of comprehensive treatment carried out on the basis of definitive treatment, aimed to improve the quality of life and function. It usually refers to the functional restorative treatment, training and rehabilitation performed in a nursing home, under conditions of stable vital signs and during the recovery period of injury, which is the last link in the treatment of war injury, and a concrete manifestation of the "people-oriented" thinking with great significance to the rehabilitation of a large number of disabled people after the war.

2.2 Classification of Treatment for Blast Injury During Wartime

The wartime environment is characterized by the contradiction between supply and demand, especially the greater limitation of medical resources so that the types of treatment for blast injury in wartime are divided finely. It needs to be emphasized that medical treatment in echelons should be organized flexibly according to the principles instead of rigidly application. The following types of treatment for blast injury can be further merged or split according to actual conditions in the organization and implementation of the treatment.

2.2.1 On-Battlefield (On-Site) First Aid for Blast Injury

1. The wounded who are in shock without obvious trauma but with hearing damage, chest pain, abdominal pain, dyspnea, irritability, hematuria or hemoptysis, should be treated as visceral injuries.
2. Prevent and manage traumatic suffocation. Remove mouth and nasal secretions, keep the respiratory tract unblocked. Improve respiratory function and encourage the sober wounded to cough and expectorate, and perform mouth-to-mouth artificial respiration for the wounded who have stopped breathing. Do not squeeze the chest.
3. Severely injured persons with ruptured tympanic membrane, nose and mouth bleeding or expectorate bloody foamy sputum should be transported in the head-high prone position instead of walk with assistance.

2.2.2 Medical Treatment for Blast Injury

1. For the wounded in shock, the amount of infusion should be appropriately limited especially for the traumatic chest where the infusion speed should be controlled.
2. For the wounded with severe dyspnea, a tracheostomy should be performed in time to remove the secretions in the trachea, as well as oxygen inhalation and unobstructed airway maintenance.
3. For chest pain, intercostal nerve block can be used for analgesia, while morphine and pethidine drugs are prohibited.
4. Lying still. Evacuate the wounded immediately when the blood pressure is stable.
5. A large dose of corticosteroids can be applied to the critically wounded in the early stage.

2.2.3 Forward Resuscitative Surgery of Blast Injury

1. Keep continuous oxygen supply. After rib fractures and pneumothorax are excluded, pressurized oxygen inhalation, and infusion of hypertonic glucose and mannitol can be used to meliorate pulmonary edema and reduce intracranial pressure. After the blood pressure is stable, furosemide (furosemide) or etacrynic acid (diuretic acid) can be used for diuresis, and intravenous aminophylline can be used to prevent and treat bronchospasm. Tracheostomy

is performed on the wounded who are unconscious, difficult to expectoration, or suffocated. Cool the head to treat brain edema.
2. For tympanic membrane perforation or tympanic hemorrhage, the secretions in the external auditory canal should be removed to keep it dry, with cotton loosely filling it. Washing and drug dripping are prohibited.
3. Take chest X-rays. Monitor the heart function and give cardiotonic drugs if necessary.
4. Thoracic puncture and drainage should be performed on the wounded with hemothorax; closed drainage should be performed for massive hemorrhage; chest laparotomy can be performed for progressive massive bleeding.
5. When an abdominal organ injury is suspected, a laparotomy should be carried out in time (according to the treatment principles of abdominal injury).
6. Use broad-spectrum antibiotics to prevent and treat infections. When diffuse intravascular coagulation (DIC) is combined, fresh plasma and platelets can be infused as appropriate. Potassium chloride can be infused intravenously to correct hypokalemia.

2.2.4 Definitive and Rehabilitative Treatment of Blast Injury

Stay away from the battlefield and treat symptomatically.

2.3 Classification of Treatment for Blast Injury in Peacetime

Blast injury mainly occurs in the auditory apparatus, heart, lung, brain, and abdominal viscera with milder external injury but more severe internal injury, which develops rapidly, so that it should be closely observed and treated as soon as possible to prevent missed diagnosis and mistreatment. In the peacetime with sufficient medical resources, it is particularly important to provide immediate treatment and prompt in-hospital treatment. The treatment of blast injury in the peacetime are divided into three parts, namely on-site first aid, key pre-hospital treatment, and comprehensive in-hospital treatment, where on-site first aid is the prerequisite for the life support of the wounded, while the key pre-hospital treatment is the core of the life support, and the in-hospital comprehensive treatment is the guarantee for the physical recovery of the wounded. Regarding different levels of hospitals, on-site first aid is usually completed by the emergency unit, and the key pre-hospital treatment can be completed by the first aid unit with strong capacity or by the inpatient unit. In-hospital comprehensive treatment is completed by the inpatient unit and the rehabilitation unit.

2.3.1 On-Site First Aid for Blast Injury

1. **Pre-judgment of blast injury**. At the detonation site, all the wounded in shock without obvious trauma, especially those with extensive burns that occurred during the nuclear war, should be suspected of visceral blast injury.
2. **Unobstructed airway maintenance**. Encourage the wounded to cough and expectorate to clear the secretions in mouth and nose. Mouth-to-mouth artificial respiration should be performed for those who have stopped breathing, while chest compression breathing is prohibited. The unconscious wounded with tongue falling back can be managed with tongue traction fixation, or trachea or nasopharyngeal ventilation tube to keep smooth ventilation. When possible, tracheotomy should be performed on the wounded with severe dyspnea or a relative long time of coma in order to remove the secretions in the trachea to keep the airway unobstructed.
3. **Hemostasis**. Pressure bandage to stop bleeding if there is bleeding from the wound. A tourniquet can be used to stop bleeding from the arterial bleeding of the limbs, and the obvious mark should be added, and the wounded should be evacuated in priority.
4. **Prevention and treatment of pneumothorax**. Chest wounds need to be tightly wrapped with thick dressings. Tension pneumothorax should be treated with thoracocentesis and gas drainage.
5. **Pain relief**. Analgesics can be taken orally or intravenously to prevent shock. For patients with chest pain, intercostal nerves can be blocked for pain relief. Analgesics such as morphine or pethidine with central nervous system inhibition are prohibited.
6. **Rehydration**. For hypotension or shock due to blood loss, low-molecular dextran or plasma substitutes can be infused, and those who can drink can supplement fluid orally.
7. **Anti-infection**. Antibacterial drugs should be given.
8. **Evacuation**. Prevent bumps during transportation and limit activities. Head-high prone position should be adopted to evacuate those severely wounded patients with ruptured tympanic membrane, mouth and nose hemorrhage or coughing up bloody foamy sputum, while the patients shouldn't walk with assistance.

2.3.2 Key Pre-hospital Treatment for Blast Injury

1. **Visceral injury**. Those with suspected closed visceral injuries should be carefully examined, diagnosed as early as possible, and treated with appropriate measures in time, while for the casualties suspected of visceral injury, exploratory laparotomy should be performed in a timely manner, and ether anesthesia is prohibited. For severe

head injury, chest-abdominal joint Injury, open fractures or large vessel injury, emergency surgery can be performed according to the requirements of various specialties.

2. **Multiple injury**. According to the principle of serious condition first, priority is given to debridement of wounds that affect the respiratory and circulatory function, keep bleeding, or bandaged by a tourniquet. Meanwhile, if there is shock, debridement is usually done after the injury is stable, but when there is active bleeding, surgery should be performed to stop the bleeding simultaneously with anti-shock treatment.
3. **Prevention and treatment of pulmonary edema and brain edema**. Keep continuous oxygen inhalation. Infuse hypertonic glucose and mannitol to meliorate pulmonary edema and reduce intracranial pressure. After the blood pressure is stable, furosemide or etacrynic acid can be used for diuresis, and intravenous aminophylline can be used to prevent and treat bronchospasm. Cool the head to treat brain edema. Tracheostomy should be performed on the wounded who are unconscious, difficult to expectoration, or suffocated.
4. **Hearing injury**. For tympanic cavity hemorrhage, foreign bodies in the external auditory canal need to be removed to keep it dry, while dripping of oily liquid and washing should be prohibited, as well as blowing the nose forcefully and pouring raw water into the ears. Antibacterial drugs should be given to prevent otitis media and systemic infections.
5. **Hemothorax**. After the injury is stable, thoracentesis should be performed to drain blood. If there is a large crack noted in the chest wall, sutura can be used.

2.3.3 In-Hospital Comprehensive Treatment for Blast Injury

Comprehensive treatment should be implemented according to the requirements of various specialties.

3 The Main Work of Medical Treatment in Echelons of Blast Injury

3.1 Organizations for Medical Treatment in Echelons of Blast Injury

3.1.1 Organizations for Medical Treatment in Echelons of Blast Injury in Wartime

1. **The head of the company and battalion**. Special personnel should be assigned to lead the treatment and evacuation of the wounded in the line of fire. The evacuation of the wounded in the regiment and above treatment agencies must be implemented in the logistics support plan under the unified leadership of the heads of logistics at all levels, and the support of the relevant departments inside and outside the military and the people must be actively sought.
2. **The brigade and regiment treatment agencies and hospitals at all levels**. Special personnel and organizations (evacuation group or classified evacuation group) should be assigned to take charge of the evacuation of the wounded, including arranging the distribution of transportation, handling evacuation procedures, and assigning escorts, organizing the wounded to board the vehicle, etc. Each treatment team and room mainly assists the evacuation team to prepare for the evacuation of the wounded. The evacuation indications should be strictly controlled, and the evacuation review system should be implemented.
3. **The leading organs of health services at all levels**. Special personnel should be designated to take charge of the organization and coordination of the wounded evacuation, mainly including the followings.
   (a) Before the war, according to estimates of health attrition, sufficient transportation capacity was planned from various aspects, so that dedicated transportation capacity should be combined with other transportation capacity, and internal military transportation capacity should be combined with local transportation capacity that supports the front.
   (b) Formulate an organizational plan for evacuation. After the battle, a car evacuation medical team, a health train medical team, an air transport medical team, and a hospital ship medical team should be established to be responsible for the evacuation of the wounded.
   (c) Define the procedures and requirements for the evacuation of the wounded, determine the evacuation indications, and clarify the evacuation method.
   (d) During the battles, it is necessary to keep abreast of the evacuation of the lower-level wounded, keep close contact with the transportation department, dispatch transportation vehicles in time, organize the wounded for successive evacuation, and request the head of the Synthetic Army to dispatch personnel and vehicles in emergency situations.

   When the number of wounded in the rear of the battle is large and the evacuation task is heavy, it should also be recommended that war service, medical service, military transportation and transportation departments, and local front support agencies participate in the wounded evacuation command structure to strengthen the organization and leadership of the evacuation work.

3.1.2 Organizations for Medical Treatment in Echelons of Blast Injury in Peacetime

1. **Hospital emergency treatment team**. Medical institutions at all levels have formulated emergency treatment plans for public health emergencies and pre-programmed emergency treatment teams. Once an explosion occurs in the responsible area where there may be casualties, the hospital directly responsible for the emergency public health incident shall immediately initiate the emergency treatment plan, and set up an emergency treatment team.

   The team usually has the dean as the team leader and the business dean as the deputy team leader. The members are composed of related department directors, medical and nursing backbones, and personnel from related departments of auxiliary diagnosis, pharmacy, and logistics. The team can be divided into several echelons. Usually the director of the emergency department serves as the leader of the first echelon, leading the medical staff of the emergency department to the scene of the accident for on-site first aid, and quickly classifies and organizes the evacuation of the wounded; the second echelon is headed by the director of the general surgery or the main surgery, and the members are from the surgeons. It is composed of senior clinical physicians, whose main tasks are to carry out further examination and classification, organization inspection, hospitalization, and emergency surgery for the wounded who were admitted to the hospital; the third echelon is led by the head of the medical department, and its members are mainly staff from auxiliary clinics, pharmacy personnel and related logistics support personnel with the main task of assisting the clinical departments in examinations, ensuring the safety of medicine transportation, equipment and goods supply vehicles, keeping communication lines unblocked, and contacting with relevant local departments.
2. **Treatment command organizations for the medical treatment in echelons of blast injury**. Treatment command organizations for the medical treatment in echelons of blast injury can be composed of four levels, namely the national emergency command center (the first level, including national ministries and special emergency command centers), the provincial (city) emergency command center (the second level), the prefecture-level city emergency headquarters (the third level), and the county-level city emergency headquarters (the forth level), to implement command in echelons.

   The first-level emergency command center is located in the State Council and consists of the emergency management office of the State Council, the emergency command center of national government departments, and the special emergency command center. The emergency command center of government departments is a command department established by various ministries, commissions and bureaus of the State Council to deal with emergency incidents within the business scope of the department. The special emergency command center is a command department jointly established by several ministries, commissions and bureaus to deal with special emergency incidents.

   The second-level emergency command center is the provincial (city) emergency command center. It is composed of the emergency management office of the provincial (city) government office and the prefecture-level city emergency command center and the special emergency command center under the management of the emergency committee, responsible for the organization and command of the emergency response of the province (municipality) and the special business department.

   The third-level emergency command center is the prefecture-level city emergency command center, Being responsible for the organization and command of the emergency response of the county-level city under the management of the emergency management office of the county-level city government.

   The fourth-level emergency command center is the county-level city emergency command center, which handles emergencies under the management of the prefecture-level city government emergency command center.

3.2 On-Site Treatment Organization for Blast Injury

The organization of on-site treatment of blast injury focuses on the organization during wartime.

3.2.1 Organize Treatment in the Killing Zone to Minimize Deaths

Treatment of the blast killing zone, the starting point for the treatment of the wounded, is carried out under the direct threat of enemy artillery fire, air strikes, and radioactive and chemical contamination, which is the most arduous and difficult part of the task. Managing it well can reduce deaths in battle and lay a foundation for further treatment at all levels. Treatment experience has proved that in addition to the immediate death due to vital organ injury, the mortality is closely related to the timeliness of the treatment, the quality of the treatment and the evacuation time of the wounded. Therefore, it is necessary to carefully organize the killing zone treatment.

1. **Establish a treatment organization and strengthen the organization and leadership of the on-battlefield**

(on-site) treatment. Under the leadership of the company and battalion leaders, the combat detachment must be established with health personnel as the backbone and recruit relevant personnel to form a line of fire treatment team; during the nuclear combat, troops above the brigade or regiment level must establish treatment teams in the killing (contamination) zone to distribute treatment medicines, equipment, stretchers, etc. to the nuclear and chemical wounded.

2. **Strengthen war treatment training in peacetime**. War treatment training has been included in the regular military training. The health service department must implement it as required in peacetime. Self-treatment and mutual treatment should also be rehearsed before the war. Combat treatment training that is needed urgently should be arranged targeted to the actual conditions during the interval of wartime and peacetime.
3. **Carry out mass self-treatment and mutual treatment widely**. The treatment task on the line of fire during war is too arduous to complete by medical personnel alone, so that extensive mass self-treatment and mutual treatment is necessary which must rely on the majority of officers and soldiers and give full play to the role of health soldiers.

3.2.2 Give Priority to the Treatment of the Critically Wounded, and Strive to Reduce the Early Deaths of the Wounded

The critically wounded mainly refer to those who suffered shock, massive hemorrhage, suffocation, and severe visceral damage, who are the main targets of early death among the wounded, where shock occurs most. Therefore, it is necessary to strengthen the prevention and treatment of shock among the wounded and the treatment of the critically wounded.

Comprehensive measures are taken to prevent shock. If there is no infusion condition, give the wounded water, hot soup, or tea. Application of sedatives and analgesics, prevention from cold and heatstroke, mental comfort, and local treatment of wounds should be implemented to ameliorate shock and strive for evacuation time. Treatment organizations at all levels above the regiment should establish treatment organizations to resist shock or treatment the critically wounded. Those in shock or critically wounded should be treated actively.

The critically wounded with suffocation, massive hemorrhage, or major visceral injury, should be treated rapidly in priority from the on-site first aid, based on medical conditions at all levels. When the wounded arrive at brigade treatment organizations or field hospitals, emergency operations must be performed first on the critically wounded.

3.3 Evacuation Organization of the Wounded with Blast Injury

The evacuation of the wounded is a transfer to the treatment institutions, a component of the medical evacuation, and an important means to realize the medical treatment in echelons of the wounded. In modern wars, there are a large number of casualties with blast injury. Only the rapid and safe transfer to treatment institutions at various levels can ensure their timely and proper treatment, which is not only directly related to the efficiency of the treatment, but also affects the maneuver of the troops and the treatment institutions. Evacuation is not only a professional work of health services, but also an important part of logistics command. Therefore, it must be included in the logistics support plan under the unified leadership of logistics leaders at all levels, who must earnestly carry out the evacuation of the wounded.

3.3.1 Evacuation Method

There are two basic methods for the wounded to evacuate, namely forward connection and backward transfer.

1. **Forward connection**. Forward connection is an activity in which the upper-level treatment organization organizes to pick up the wounded from a lower-level treatment organization. Its advantage is that the superiors control the transportation force and coordinate the overall situation of evacuation of the wounded, where the transportation force could be used more rationally according to the occurrence of the wounded in each unit, to avoid uneven assignment distribution so as to improve the efficiency of transportation. However, its disadvantage is that when the communication between the superior and the subordinate is not smooth, the injured cannot be evacuated in time.

   The forward connection can be divided into forward stepwise connection and forward leapfrog receiving.

   (a) Forward stepwise connection is commonly used that the upper-level treatment institution fetch the wounded from a lower-level treatment institution according to the organizational system (Fig. 1).

   (b) Forward leapfrog connection is a method generally adopted when the lower-level institution has no capacity to receive the wounded or prepare to transfer,

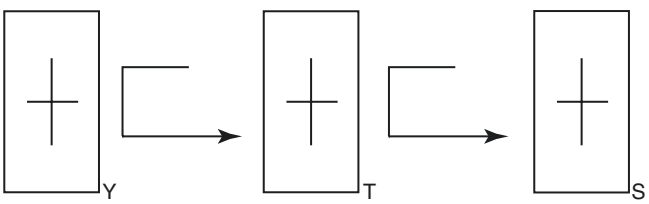

Fig. 1 Schematic diagram of forward stepwise connection. *Y* battalion, *T* regiment, *S* division

or during the air transportation, that the upper-level treatment institution picks up the wounded bypassing the treatment institution one or two level lower to (Fig. 2).

2. **Backward transfer**. Backward transfer is an activity in which the lower-level treatment institution organizes its own transportation to transfer the wounded to the upper-level treatment institution. The advantage is that each unit has its own means of transportation, which facilitates timely arrangements for the use of transportation forces, and masters the initiative of the evacuation of the wounded of the unit, while its disadvantage is that the transportation force is scattered so that the superior cannot coordinate. In this case, sometimes, there may occur uneven busyness of various units, which is inconvenient for mobile use, making it more difficult to deal with unexpected situations. Therefore, backward transfer is more commonly used in unstable battle conditions where the troops are frequently maneuvering, with a small number of the wounded, or sufficient transportation forces.

   Backward transfer can also be divided into backward stepwise transfer and backward skip-level transfer.

   (a) Backward stepwise transfer is that the lower-level treatment institution transfers the wounded to the upper-level institution (Fig. 3).

   (b) Backward skip-level transfer is that the lower-level treatment institution transfers the wounded to the institution one or two levels higher. When the upper-level treatment institution has no capacity to take in the wounded or prepare to transfer, or the evacuation road is blocked or damaged, the backward skip-level transfer may be initiated as the instructions of the superior (Fig. 4).

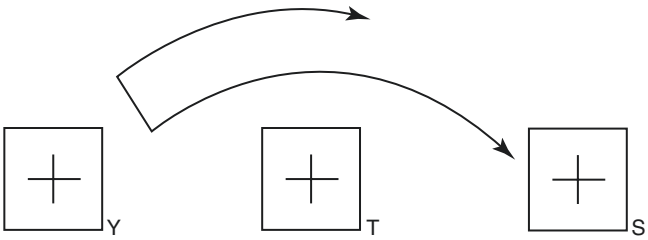

Fig. 2 Schematic diagram of forward leapfrog connection. *Y* battalion, *T* regiment, *S* division

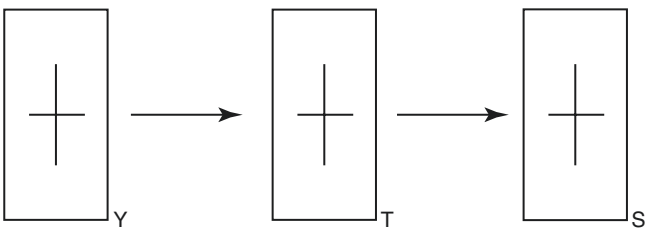

Fig. 3 Schematic diagram of backward stepwise transfer. *Y* battalion, *T* regiment, *S* division

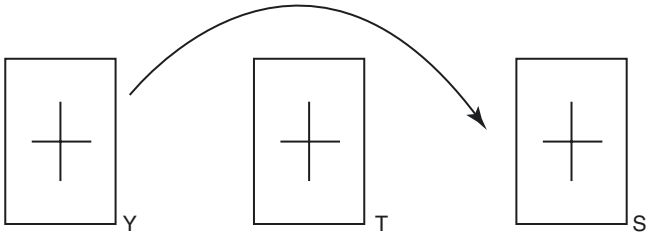

Fig. 4 Schematic diagram of backward skip-level transfer. *Y* battalion, *T* regiment, *S* division

In previous wars of our army, transferring the wounded to the upper-level medical institution was mostly adopted, while receiving the wounded from the lower-level medical institution was adopted only from the end of the War to Resist US Aggression and Aid Korea. Which method should be adopted depends on specific conditions. However, the evacuation method is not only to make clear who controls the evacuation method and the transportation force, but also to clarify the responsibility of the evacuation of the superior and the subordinate. Therefore, there must be principled regulations to follow. According to the analysis of the above two methods and the experience of various countries, the evacuation of the wounded in our army should usually be conducted based on the forward stepwise connection, and the combination of the forward connection and backward transfer. In individual cases, forward leapfrog connection and backward skip-level transfer could be considered.

Which form to adopt depends on the specific situation. There must be clear regulations on evacuation methods before the war so that they can be followed during the war. At present, the evacuation form of the wounded in our army is predominated by forward connection, and the combination of the forward connection and backward transfer. Meanwhile, forward leapfrog connection or backward skip-level transfer is adopted when conditions permit.

3.3.2 Evacuation Tools

Evacuation tools include vehicles, boats, aircrafts, and various equipment used to evacuate the wounded, which are the material basis for organizing evacuation of the wounded.

1. **Classification of evacuation tools**. Evacuation tools can be divided according to their application space into ground, water, and air evacuation tools.

   (a) Ground evacuation tools mainly include long-, medium-, and short-distance evacuation tools. Short-distance evacuation tools include standard stretchers, folding stretchers, sled stretchers, amphibious

stretchers, pack-type stretchers, ship stretchers, mesh stretchers and armored ambulances, etc.; medium-distance evacuation tools include light ambulances, medium-sized ambulances, transport vehicles for the wounded with additional devices, etc.; long-distance evacuation tools include modified transport vehicles for the wounded and medical trains.

(b) Water evacuation tools mainly include rubber boats, medical ambulance boats, medical station pad ships, medical transport ships, etc. Medical ambulance boats and hospital ships can also be used as evacuation tools when necessary.

(c) Air evacuation tools include ambulance helicopters, medical aircraft, and transport aircraft.

The above-mentioned evacuation tools, except the stretcher, should be equipped with first aid medicines, equipment and medical oxygen, etc., for the treatment of the wounded on the way. In wartime, in order to make up for the lack of evacuation, transportation vehicles with additional devices for evacuation of the wounded, or empty return vehicles could be used as a supplement to the evacuation tool.

2. **Function of the evacuation tool**

(a) Military stretcher: Military stretcher is the standard tool for the army to carry the wounded in prone position, divided into general stretcher and special stretcher. A general stretcher, made according to uniform specifications, is suitable for lying down and changing various means of transportation for the wounded. A special stretcher can be used for naval ships, mountains, swamps, snow, and water to transport the wounded. According to the application condition and properties, special stretchers have a variety of structural forms, such as folding stretchers, Robinson stretchers, ship stretchers, horse-drawn stretchers, sled stretchers, scoop stretchers, air cushion stretchers, basket rigid stretchers, etc., mainly used for on-battlefield or on-site first aid and short-distance transportation of the wounded.

(b) Ambulance: An ambulance is a special vehicle that transports the wounded, where first aid can be carried out on the way, which can be divided into the ordinary ambulance and the armored ambulance. Ordinary ambulances are usually equipped with special stretchers and shock-absorbing fixed support devices, emergency resuscitation equipment and medical supplies, as well as ventilation, heating, cooling, lighting, communication and other equipment. A small ambulance can carry 1–3 wounded in prone position, or 4–6 wounded in sitting position. A large-scale ambulance can transport 8–12 wounded in prone position, or about 30 wounded in sitting position.

There are two types of armored ambulances, namely the wheeled and the tracked armored ambulance, which can protect the passengers from bullets and shrapnel, used for treatment and evacuation of wounded in the line of fire in wartime. It can usually carry 4 wounded in prone position or 6–8 wounded in sitting position.

(c) Medical train: Medical train is a special railway train that evacuates the wounded and provides treatment and living supplies on the way, characterized by a large carrying capacity, a high speed, stable driving, and the ability to transport a large number of wounded in a short time. In wartime, it is usually used to evacuate the wounded in battles or strategic rear and peacetime treatment and disaster relief. The various carriages of the medical train are arranged and grouped in a certain order, equipped with corresponding medical personnel, medical materials, nursing supplies and communication equipment to facilitate treatment on the way.

A standard medical train with a maximum of 19 carriages, including a traction locomotive, a command car, an intensive care vehicle, a logistics support vehicle, a camping car, 2 surgical emergency vehicles, and 12 wounded transport vehicles, where except for surgical emergency vehicles and intensive care vehicles that are developed specially for medical application, other functional compartments are quickly retrofitted by 25G hard sleepers and dining carriages. Each section of the wounded transportation vehicle can carry 38 patients in prone, the intensive care vehicle can carry 20 severely injured patients, and the medical train can load about 476 patients in prone at one time.

(d) Ambulance helicopter: An ambulance helicopter is a helicopter used to treatment and evacuate the wounded, which can be divided into special-purpose ambulance helicopter and dual-purpose ambulance helicopter. The special-purpose ambulance helicopter is modified specifically for air transportation of the wounded; the dual-purpose ambulance helicopter is temporarily equipped with stretchers and portable medical and health equipment when needed for treatment work. The ambulance helicopter is mainly used for air evacuation of various wounded in peacetime, and medical treatment of the wounded in natural disasters and major traffic accidents (including aviation, navigation, railway, highway accidents), as well as treatment of persons in danger at sea, jungle, desert, cold areas and other areas. The aircraft is equipped with health personnel who are responsible for the on-site first aid of the wounded and the medical care on the aircraft during the evacuation. The

ambulance helicopters used by our army mainly include Mi-8, Mi-17, Zhi-8, Super Hornet, and Black Hawk, which can carry 12, 12, 15, 15, and 4 wounded patients, respectively.

(e) Medical aircraft: A medical aircraft is a special aircraft that transports the wounded and provides medical care in flight. A medical aircraft can be used for emergency treatment and disaster relief, transportation and treatment of the wounded in remote areas and other situations, and rapid medical evacuation of the wounded in wartime. The aircraft is equipped with health personnel who are responsible for the medical care of the wounded during the flight. Medical aircrafts used by our military are modified from military transport aircrafts, mainly including An-26, Yun-5, and Yun-8, of which Yun-5 can carry 6 wounded in prone position or 12 wounded in sitting position, and Yun-8 can carry 60–96 wounded.

(f) Evacuation ships: Evacuation ships are service vessels used to evacuate the wounded and provide medical care during the evacuation, where the medical equipment of the medical transport ship is relatively simple, and the medical personnel are few, which are mainly used to ensure the safety during the evacuation of the wounded. During the war, local civilian ships are mainly mobilized for use as medical transport ships. Medical ambulance boats are mainly equipped with medical and surgical emergency equipment and professional health personnel, which are mostly used for treatment and evacuation of the wounded near shore. The medical transportation ship and the medical ambulance boat can be used together, or alone for the treatment and evacuation of the wounded, according to the number and type of the casualties, the distance between the sea areas, the situation and style of the battle. In order to meet the needs of treating the wounded in future naval battles, a complete sea-air combined evacuation system composed of the evacuation ship, the ambulance helicopter and the water ambulance aircraft.

(g) Evacuation tools mainly include additional devices for the casualty evacuation and the casualty transfer tools.

The casualty transfer tool is used for transportation and carrying of the wounded during the transfer to another type of an evacuation tool. The transportation device can be divided into vertical transmission device and horizontal transmission device according to the transportation space, mainly including marine transportation device and land-air transportation device. The former is used for vertical and horizontal transfers between medical ships at sea, mainly including ropeways, pulleys, and lifting equipment. The latter is used for transfers between helicopters and land, mainly including aerial hoisting equipment and supporting equipment. Carrying tools include hanging baskets, hangers, slings, navy stretchers, and inflatable boats.

The additional device for casualty evacuation is a detachable stretcher fixing device installed on the transportation tool for the evacuation of the wounded, which can be installed on transport vehicles, helicopters, transport aircraft, and ships. The additional equipment for the wounded evacuation equipment equipped by our army includes the evacuation equipment for the transport vehicle and the fixed equipment for the stretcher for air transportation. Additional equipment for transporting the wounded can be installed on Jiefang CAl0BE, Dongfeng EQl40, Dongfeng EQ240, and other transport vehicles, which can carry 6–9 casualties in prone position. The fixed device of the stretcher for air transportation. After installation of the stretcher fixture, the Mi-8 helicopter can carry 12 wounded in prone position, the Zhi-8 helicopter can carry 15 wounded in prone position, the An-26 transport aircraft can carry 24 wounded in prone position, and the Yun-5 transport aircraft can carry 6 wounded in prone position.

3.3.3 Main Measures for Safe Evacuation

The main measures for safe evacuation involve strict control of evacuation indications, selection of suitable means of transportation, determination of appropriate evacuation position, do a good job of treatment work during the evacuation, pay attention to strict camouflage and vigilance during wartime to ensure safety Protection.

1. Strictly grasp the evacuation indications and make various medical preparations before evacuation. In order to reduce the confusion of evacuations and ensure the safety of evacuations, we should adhere to the system for determining the evacuation based on the evacuating indications and taboos and the review system before evacuation. Before evacuation, check the whole body and local conditions of the wounded carefully to determine whether they meet the evacuation indications and whether the medical evacuation documents are complete. The wounded in coma, with suffocation or other dangers during the evacuation, or after the surgery shall be observed for a certain period of time in accordance with the regulations. Evacuation of the wounded in shock is prohibited in principle, while when the evacuation is a must, the helicopter should be used as much as possible, with continuous anti-shock measures on the way. Certain treatments and preventive measures should be taken for the wounded who must be evacuated, with sufficient preparations of medicinal materials for first aid and nursing on the way.

2. Choose rapid and safe evacuation tools and maintain a proper evacuation position. Generally, severely injured patients should be evacuated by stretchers, ambulances and helicopters. Casualties with abdominal injury without surgery or those with thoracic injury with or without surgery should be evacuated by stretchers and ambulances. Generally speaking, there is no absolute contraindication for air evacuation.
3. Observe and treat the wounded well on the way, and arrange the wounded to rest and take food in time. For a large number of wounded or critically wounded, health personnel should be assigned to escort them during the evacuation according to actual situations. A special escort organization must be established when the wounded in batches are evacuated by the hospital. Escort and transport personnel should keep an eye on the wounded, especially on them in shock, with suffocation and hemorrhage, in order to perform first aid in time. The transportation hub and other transfer places that often pass the wounded should be set up rest places for the wounded. Between two levels of treatment institutions that are far apart, a wounded transit agency can be set up on the way according to the situation for the wounded to transfer, in order to provide space for temporary rest, eating and drinking, heating, and first aid.
4. Pay attention to safety protection on the way to prevent accidental injury. During the evacuation, it is necessary to prevent mechanical trauma of the wounded due to transportation, roads and weather, or cause secondary bleeding and shock, and aggravate the injury. For this reason, in addition to taking anti-vibration measures before evacuation, the speed of the general vehicle should be appropriately reduced when evacuation; special attention should be paid to the cold and warm work of the wounded when evacuation in winter. Enemy fire strikes often cause damage to evacuation roads, and evacuation personnel must follow the rules of enemy fire strikes and camouflage to ensure the safety of evacuation.

3.4 Organization of the Relay Treatment of Blast Injury

The medical treatment in echelons of blast injury, which should not be rigidly applied, should be applied flexibly according to different treatment types and actual conditions in organizational practice. The relay treatment refers to the medical treatment that the wounded received after the battle (on-site) emergency treatment before receiving the professional comprehensive treatment. Defined from the scope of medical technology, it roughly corresponds to the scope of emergency treatment and early treatment of explosive shock injuries and wartime treatment, while corresponding to the usual treatment of explosive shock injuries, it can be understood as the more complicated part of the on-site emergency treatment and a simple part of the pre-hospital key treatment.

The essence of relay treatment is more involving the participation of a number of institutions or groups. The content of the treatment is inherited and complemented each other, and the organization is flexible and diverse in coordination. Affected by the differences in peacetime and wartime and the uncertainty of evacuation environment, distance, and time, relay treatment may be completed by several medical subjects, or by a certain medical unit, or even with tools.

3.5 Definitive Treatment and Rehabilitative Treatment of Blast Injury

3.5.1 Effective Control of Shock

Measures include immediate establishment of multiple (up to four) venous channels for rehydration administration, rapid rehydration, and blood replenishment for shock with massive bleeding, up to 200 mL in 5 min, or 800 mL in 30 min, proper use of hypertonic saline against shock to shorten shock time for surgery, and routine application of naloxone and other drugs to inhibit sympathetic nerve activity, improve microcirculation, and promote recovery of consciousness.

3.5.2 Focus on Prevention and Treatment of Wound Infections with Debridement Operations at Appropriate Times

Blast injury is often accompanied by multi-site open injuries, almost all of which are contaminated by primary bacterial, which is an important cause of late death of the wounded. The increase in multiple injuries and multiple injuries in modern warfare not only increases the chance of trauma infection, but also makes trauma infection more serious and complicated, requiring higher treatment technology. Therefore, early prevention and treatment must be performed.

1. **Early debridement surgery**. Those with mild skin injury should undergo thorough debridement at an early stage; for those with severe skin damage or more skin defects, it should be “effective” instead of “complete,” and second-stage surgery is feasible. When debridement on the severely wounded, it is necessary to grasp the degree and function of the damage of each important organ, and comprehensively measure the relationship between the pros and cons. During debridement, the medical staff should pay attention to the patient’s vital signs, and focus on the tolerance of the whole body, instead of attending to one thing and losing another, missing the more important

because of a trifle. The traumatic shock and the potential visceral hemorrhage should not be neglected. Besides, dealing with skin and limb injuries too much or for a long time may delay the condition.

2. **Timely application of anti-infective drugs**. Our army stipulates that anti-infective drugs should be used from the camp, and the brigade ambulance and field hospitals should continue to take orally or inject antibiotics. Once the infection develops, treatment agencies at all levels should confirm the diagnosis as soon as possible, and take effective antibiotics and other effective measures to control it. In order to prevent tetanus, on the basis of the usual toxoid injections, tetanus toxoid injections are generally administered before the battle. For those who have not been vaccinated, tetanus toxoids and anti-toxic serum.
3. **Isolation and treatment of the wounded with specific infections, such as tetanus and gas gangrene**. Once the tetanus and gas gangrene wounded are found at all levels, special rooms should be opened for isolation, and special tools will be sent to the specialist department or guidance hospital. The hospital conducts treatment and management. The secretions, contaminated equipment, appliances, rooms, vehicles, etc. of the wounded with specific infections should be strictly disinfected to prevent cross-infection.

3.5.3 Proper Treatment According to the Characteristics of Blast Injury

For the organ injuries associated with blast injuries, such as lung injury, craniocerebral injury, kidney injury, spleen injury, liver injury, etc., it should be anticipated in advance, detected at the bedside in time, and active symptomatic intervention.

Blast injury is most likely to cause damage to the lungs, tympanic membrane and other hollow organs. The severely wounded should use a ventilator to assist breathing. When the ventilator supports assisted breathing, the principles of "early ventilation and early removal" and "individualization" should be followed. Generally, protective mechanical ventilation is adopted, with a low tidal volume (6–8 mL/kg). For the wounded with fast breathing rate (25–30 breaths/min) and hypercapnia ($pH \geq 7.25$), pressure-controlled ventilation (platform pressure $\leq$ 30 cmH_2O, peak inspiratory pressure ≤ 40 cmH_2O) is used to prevent progressive damage to normal lung cells. For the wounded with tympanic membrane perforation, the small perforation is allowed to heal by itself, and the larger one is applied with gelatin sponge, and the second-stage surgical repair is performed depending on the situation.

3.5.4 Focus on Psychological Trauma to Promote Both Physical and Mental Health

Measures include humanity care, psychological treatment, follow-up guidance, recovery promotion, and accident prevention. High explosives not only cause great harm to the human body, but also bring great trauma to their psychology, especially when they are not mentally prepared. Due to the horrible scene of the explosion, the wounded were generally fearful and had poor sleep. Some wounded dared not face the reality of physical disabilities and were silent and pessimistic. In the definitive treatment and rehabilitative treatment of the wounded, attention should be paid to humanistic care and psychological treatment. For the wounded who have recovered and recuperated, regular follow-up guidance should be adopted to achieve good treatment results.

First Aid Techniques for Blast Injury

Zhaowen Zong

Explosions are more common in wartime, while in peacetime they often occur in terrorist attacks and accidents, which are a physical reaction process caused by rapid changes in pressure and temperature, directly or indirectly acting on the human body through shock waves, projectiles, heating power, toxic gases, etc. In addition to common lung and auditory injuries, it can also cause life-threatening conditions such as airway damage and massive hemorrhage, which need effective first aid in the shortest time to save the lives of the wounded. This chapter discusses on-site first aid assessment and first aid techniques for blast injury.

1 On-Site Assessment and First Aid Principles of Blast (Battlefield) Injuries

The explosion shock wave is generated by the rapid expansion of the high temperature and high pressure gas produced by the explosion and conduction to the surroundings. When an explosion occurs in an open space, the pressure wave grows rapidly in an instant, forming an overpressure, then decays to form a negative pressure, and then gradually rises to return to atmospheric pressure. Under the action of shock waves, various injuries to the body are called blast injury (i.e., explosive injury). Blast injury mainly affects internal organs and auditory organs, especially air-bearing tissues (like lungs and gastrointestinal tract), and the body surface of the wounded is often intact, manifested as complex injuries with mild external injury but severe internal injury which progresses rapidly. It should be noted that during the explosion, in addition to the shock wave, which is the injury mechanism, damage may also be caused by projectiles, heating power, toxic gas, throwing up, etc., which can cause airway damage, hemorrhage, and other life-threatening conditions, which need timely diagnosis and management.

First aid for blast injury follows the principle of advanced trauma life support (ATLS), that is, judging the injury by fast and accurate methods as soon as possible, clarifying the existence of life-threatening conditions and dealing with them as soon as possible. At the battlefield first aid and disaster rescue site, it is recommended to use simple triage and rapid treatment (START), "ABCDE," and "MARCH" methods for injury assessment.

1.1 On-Site Assessment of Blast Injury

1.1.1 Simple Triage and Rapid Treatment (START)

START is widely used when evaluating batches of wounded in disaster relief. According to the severity of the disease, patients are often divided into four grades, which are marked with four colors of black, red, yellow, and green. Among them, the green mark represents minor injuries, where the wounded has no life-threatening injuries, and the condition is relatively stable even without medical assistance within a few hours or days; the yellow mark represents the wounded that can be delayed for treatment, although the patient's injury is more serious, it will not be deteriorated further when the treatment is postponed slightly; the red mark indicates that the patient is seriously ill and requires immediate emergency treatment; and the black mark indicates that the patient has died or the injury is bound to lead to death.

In general, the evaluation method is the "30-2-can-do" rule, where 30 refers to whether the respiratory rate exceeds 30 breaths/min, 2 refers to whether the capillary refilling time is greater than 2 s, and can-do refers to whether the wounded can walk as the command. The wounded with less than 30 breaths per minute, capillary refilling time less than 2 s and able to walk according to instructions are classified as lightly wounded. The wounded that meet the above criteria but cannot walk are classified for delayed treatment. The wounded that are unconscious,

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have a rapid breathing rate, prolonged capillary filling time, or disappearance of radial artery pulsation are classified for immediate rescue.

1.1.2 "ABCDE" and "MARCH" Method

In peacetime trauma treatment, it is generally recommended to use the "ABCDEF" sequence to conduct on-site examination of the wounded, which can make quick judgments on the diagnosis that threatens the patient's life and limb safety, and will not miss the diagnosis. Among them, "A (airway)" refers to whether the airway is unobstructed, whether inspiratory dyspnea occurs due to obstruction, whether there is throat sound due to incomplete obstruction, where the throat sound of severely wounded is a life-threatening sign; "B (breathing)" refers to whether breathing is normal, whether there are tension pneumothorax, open pneumothorax, abnormal breathing, orthopnea, dyspnea, cyanosis, etc.; "C (circulation)" refers to the circulatory state, including pulse, blood pressure, skin color, capillary filling time; "D (disability)" refers to the central nervous system, including indicators such as the pupil size, light reflection, limb mobility, degree of consciousness disturbance, and the Glasgow score; "E (exposure)" refers to exposure of the patient's body in order to fully estimate the condition; and "F (fracture)" refers to whether there is a fracture.

This sequence can still be followed during wartime. However, the experience of the US military in the wars in Afghanistan and Iraq shows that the sequence of examinations needs to be changed. They recommend using the "MARCH" sequence for triage, where "M (massive hemorrhage)" refers to whether there is a major hemorrhage, so that if there is a life-threatening hemorrhage, it should be dealt with immediately. A tourniquet should be applied to the limbs, but for open bleeding in the abdomen and chest, where a tourniquet is not possible, it is recommended to use hemostatic gauze to cover and bandage the wound. "A (airway)" is the same as the A in the "ABCDE" sequence. The "R (respiration)" is the same as the B in the "ABCDE" sequence, which refers to whether there is tension pneumothorax or open pneumothorax, which need decompression and closing the chest wound. The "C (circulation)" is the same as the C in the "ABCDE" sequence. The "H (hypothermia)" refers to whether there is hypothermia, where keeping warm or reheating is needed with an insulation blanket, etc. It can be seen that examinations and the control of massive hemorrhage are prior to the airway examination in the sequence, because from the data analysis of the wars in Vietnam and the wars in Iraq and Afghanistan, there is no change in the causes of death due to war injuries, that is, in the order of trunk injury (35%), central nervous system injury (31%), multiple organ failure (12%), massive limb hemorrhage (9%), tension pneumothorax (5%), and airway injury (1%). However, the top three causes of preventable death are limb hemorrhage (>70%), airway injury (14%), and tension pneumothorax (1%). Therefore, the U.S. military recommends that the triage and control of massive hemorrhage should be the first priority.

Another difference between the "MARCH" triage sequence and the "ABCDE" triage sequence is the addition of hypothermia examination. This is because hypothermia is a very important independent factor that constitutes the "lethal triad" of severe trauma patients, and its treatment also has a vital impact on the prognosis in pre-hospital care.

Regardless of the application of the "ABCDE" or the "MARCH" triage sequence, it is necessary to judge in a relatively short period of time whether there is life-threatening conditions in the wounded. At present, the US military generally recommends the triage time of each wounded to be 20 s or so. After the triage, a life-threatening condition (such as massive hemorrhage, tension and open pneumothorax, airway obstruction, etc.) is required emergency treatment.

1.2 First Aid Principles of Blast Injury

Whether in wartime or in peacetime, on-site assessments and first aid must be carried out after ensuring the safety of surrounding environment and the wounded. For example, during wartime, it is necessary to suppress or destroy the enemy's firepower and ensure the safety of the battlefield environment. In peacetime, if the environment of the detonation site is unstable with possible secondary disasters such as secondary explosions and exposure to chemical hazardous materials, the on-site first aid of blast injury, like other disaster rescues, should follow the principle of "guaranteeing the safety of the medical personnel themselves prior to the wounded" to ensure the safety of rescuers as the first priority. In the same way, the injured should be removed from the danger zone as soon as possible after searching to avoid secondary injuries.

Afterwards, according to the specific situation, rescuers should treat the wounded after judging the existence of any life-threatening condition by the above-mentioned on-site assessment methods. Specific treatment measures are detailed in the following sections.

2 Airway Management of Blast Injury

There are various reasons for airway obstruction in blast injury, which can be directly caused by firearm injuries or secondary injuries, mainly including maxillofacial injury, craniocerebral injury, inhalation burns, chest blast injury; coma, neck hematoma, soft tissue displacement, fracture fragments due to thoracic blast injury, and tracheal compres-

sion, intratracheal injury, and foreign bodies caused thereby, etc. The head tilt-chin lift or jaw thrust maneuver should be used to open the airway of the unconscious wounded. If the wounded can breathe spontaneously without airway obstruction, the nasopharyngeal airway should be established during the further airway management. If the wounded remains conscious, the oropharyngeal airway opening is easier to tolerate, but the nasopharyngeal airway opening is more stable during transportation. In addition, occlusion (jaw clenching) usually occurs in patients with head injuries, which makes oropharyngeal airway opening difficult. The unconscious wounded should be placed in a resuscitation position in a semi-prone position to prevent inhalation of blood, mucus, or vomitus. For those who cannot relieve dyspnea after using nasopharyngeal ventilators, thyrocricocentesis can be used to temporarily relieve airway obstruction, and the wounded should be evacuated immediately for tracheal intubation or cricothyrotomy by medical institutions and other institutions.

The most important first aid measures for airway obstruction in the event of an explosion shock injury are to open the airway and ventilate. The methods of opening the airway include removing foreign bodies in the respiratory tract and relieving glossocoma, etc. The nasopharyngeal airway is mainly used for ventilation, and if necessary, thyrocricocentesis or incision is used.

2.1 Removing Foreign Bodies in the Respiratory Tract

1. **Fingering out**. It is suitable for oral foreign bodies obstructing the airway, including oral and maxillofacial injuries. The rescuer should first tilt the head back, use the index finger and thumb to pull out the wounded tongue, and lift up together with the lower jaw to separate the tongue from the pharynx, partially unblock the obstruction, and then extend the index finger of the other hand to take the foreign bodies (including bone fragments, blood clots, secretions, mud, etc.) out of the throat. When conditions permit, the fluid in the mouth can be sucked, the tongue can be pulled out of the body or fixed in the pharyngeal airway/nasopharyngeal airway, and the wounded can be placed in a lateral or prone position before sending. It should be noted that when removing foreign bodies with fingers, the performer should avoid pushing the foreign bodies down into the airway.
2. **Knocking back**. It is applicable to the wounded with foreign matter obstruction in the airway (including blood clots or tissue fragments). The rescuer slams the wounded back between the shoulder blades four to five times with the palm of the hand. The awake wounded shall be seated or standing, while the unconscious wounded shall be placed in a prone or semi-prone position. The rescuer shall support the wounded sternum with the other hand for support.
3. **Abdominal compression**. It is applicable to foreign matter obstruction of trachea and large bronchi. The rescuer should stand behind the wounded, wrap the arms around the waist of the wounded, with one hand a fist grasped by the other. Then the rescuer should press the thumb side of the fist against the abdomen, with the middle of the fist slightly higher than the belly button, but lower than the lower edge of the sternum, and press the abdomen back and forth quickly with the fist. It should be noted that compressions should be intermittent with a significant motion, abdominal compressions should be continuous until the wounded is free of obstruction or has no response. If the wounded does not respond, seek rescue with the airway of the wounded kept opening, and perform respiratory first aid, including mouth-to-mouth resuscitation.

2.2 Glossocoma Relief

Glossocoma can directly block the airway and obstruct the ventilation, which is one of the most common causes of airway obstruction for comatose patients. It should be noted that the foreign matter in the oropharynx should be removed before the airway obstruction caused by the glossocoma is relieved. In addition, the injured should be checked for cervical spine injury to prevent high paraplegia due to aggravated neck trauma. The airway obstruction caused by glossocoma can be relieved by the following methods.

1. **Neck support method (head backward, neck lift)**. Lie the wounded in a supine position with a small pillow placed under the shoulders. Then the rescuer puts one hand on the top of the forehead of the wounded, and the other hand lifts the neck to make the head back so that the airway obstruction will be relieved.
2. **Lower jaw support method (head backward, lower jaw lift)**. Lie the wounded on the back with head tilted back. Then the rescuer puts both hands on both sides of the lower jaw, lift the lower jaw with both hands and push them forward so that the airway obstruction will be relieved. During this process, if the wounded is suspected of cervical spine injury, only the mandibular lift method can relieve the airway obstruction.
3. **Chin-lifting method**. Put the wounded on the back with head slightly tilted back. Then the rescuer lifts the chin of the wounded with the thumb opening the lower lip. It should be noted that this method is not applicable to the wounded with maxillofacial injuries (including mandibular fractures).

2.3 Nasopharyngeal Airway Ventilation

Nasopharyngeal airway tube can effectively relieve the airway obstruction of the wounded where the rescuer should put the wounded on the back with the head upright, then lubricate the nasopharyngeal airway tube and the nasal cavity of the wounded, gently push the tip of the nose to put the nasopharyngeal airway tube with the sloping surface facing the nasal diaphragm, slowly rotate the nasopharyngeal tube forward and insert it into the nostril until its distal edge reaches the nostril. It should be noted that gasoline-based or non-water-based lubricants cannot be used to avoid damage to the nasal cavity or throat tissues and increase the risk of infection; it cannot be forced into it during insertion, and the nostrils can be replaced if there is resistance. This method cannot be used for the wounded with cerebrospinal fluid otorrhea or rhinorrhea, when obvious fluid flows out of ears or nostrils of the wounded.

2.4 Thyrocricocentesis

This is a simple, fast, safe, and effective method, applicable to the injured with suffocation or emergency situations when the above methods cannot bring effective ventilation. The rescuer lies the wounded on back with head tilted back to fully expose the neck, and then stands on the right side of the wounded, fixes the cricoid cartilage with the thumb and index finger of the left hand, and pierces the cricothyroid membrane with a syringe needle or a thicker lumen needle in his right hand. Therefore, ventilation with a needle can relieve the suffocation. This method only provides temporary relief, and then a cricothyrotomy should be performed. Usually, thyrocricocentesis can be performed by combat personnel or combat healthcare workers and professional healthcare workers.

2.5 Cricothyrotomy

Generally, simple thyrocricocentesis can only relieve airway patency in a short time, and it is necessary to perform cricothyrotomy as soon as possible to establish a stable airway. Usually, cricothyrotomy is performed by a healthcare worker or military doctor. The simple steps of cricothyrotomy include cutting the skin about 3 cm in length between the thyroid cartilage and cricoid cartilage by a sharp blade; cutting the cricothyroid membrane about 1 cm in length after full exposure and hold the incision open with a knife handle (or hemostatic forceps); inserting the tracheal tube and fix it properly after sucking out blood and secretions in the trachea.

2.6 Tracheal Intubation

Whether to use tracheal intubation to relieve airway obstruction on the battlefield is a controversial topic. The usual trauma treatment experience shows that if the airway obstruction continues to develop or exist after opening the airway with nasopharyngeal ventilation and other measures, a more deterministic airway opening method should be used, where tracheal intubation is the most effective and quickest. However, tracheal intubation in the traumatic environment of the battlefield has many disadvantages. For example, the weak light in the war environment makes it difficult to use the laryngoscope. Most medical soldiers and military doctors have never intubated the living wounded or even the corpse. Maxillofacial injuries make intubation extremely challenging, etc. It is difficult for even experienced medical staff to correctly perform tracheal intubation. Therefore, it is not advisable to try tracheal intubation on the battlefield. However, in the peacetime rescue, if an experienced anesthetist or intensive care physician, tracheal intubation can be tried.

3 Massive Bleeding Control Technology and First Aids for Shock Due to Blast Injury

Wartime bleeding is the most common injury, often caused by severe damage to the limbs, damage to the large arteries, and the thoracic and abdominal parenchymal organs (such as liver rupture, etc.). Statistics on the treatment of war wounds in previous wars show that hemorrhage is the second cause of all war casualties (15–18%), and the primary cause of avoidable war casualties (up to 80–91%). Therefore, controlling the bleeding of the wounded in first aid on the battlefield can save the lives of countless wounded.

There are many types of hemostasis methods, including direct compression hemostasis, drug hemostasis, tamponade hemostasis, surgical hemostasis, tourniquet hemostasis, etc. Each has its advantages and disadvantages, and different hemostasis measures need to be combined with the different treatment steps of our army.

3.1 Evaluation of Hemorrhage and Shock

In the first aid phase of the battle scene, it is necessary to quickly evaluate the bleeding of the wounded, including the nature of the bleeding (venous bleeding or arterial bleeding), the bleeding site (whether internal bleeding or external bleeding), the amount of bleeding, and whether there is shock. Among them, the most important thing is to determine whether there is a fatal hemorrhage that requires first

aid on the battlefield for the bleeding wounded, so as to adopt necessary first aid measures to save lives as soon as possible. In early treatment institutions, B-ultrasound and CT examinations can assist in the diagnosis of bleeding in the chest and abdomen.

3.1.1 Determine Whether There Is Fatal Hemorrhage

The most important thing on the battle scene is to judge whether there is any external bleeding that is threatening to the limbs. For example, the wounds of the limbs, maxillofacial, chest, and abdomen open injuries are constantly bleeding, gushing or pulsating jet bleeding, prompting the wounded is suffering from the threat of hemorrhage. At this time, a tourniquet or a hemostatic dressing should be used urgently to control bleeding. For specific methods, see later in this chapter.

3.1.2 Judgment of Bleeding Volume

Secondly, it is necessary to evaluate the amount of blood loss, with comprehensive consideration of the situation of the place the wounded was found, the local situation of the injury, and the general situation, which all can affect the assessment of the blood loss amount.

1. Whether there are blood stains, and their ranges around the wounded, clothing or on the ground. Usually, when the camouflage clothing around the wound is wetted, and there is blood stains on the ground around it, the blood loss can reach 700–800 mL.
2. Check the wounded's consciousness, pulse, temperature of the extremities, lip color, breathing state, etc., to make a comprehensive judgment on the blood loss volume. The method of consciousness examination is to observe the change of consciousness of the wounded by simple talks with the wounded, such as asking the wounded "Are you okay?" If he/she answers the questions with a normal speed and expression, it indicates that his/her consciousness is relatively normal; if he/she is nervous with answers like "I can't do it anymore," "Help me now," etc., it indicates that he/she is irritable; if the wounded responds to irrelevant answers such as "You are so handsome," or does not answer with an indifferent expression, it indicates unconsciousness Pulse is usually examined on the radial artery by recording the number of pulsations within 1 min, and feeling its strength. When the radial artery pulsation cannot be touched in time, it can be replaced with the pulsation of the common carotid artery. Also, the temperature of the extremities can be used by touching the hands and feet of the injured, where low temperature indicates poor blood supply to the ends of the hands and feet, and the possibility of massive blood loss and shock.

On the battlefield, the blood loss of the wounded can be estimated mainly through the wounded's pulse and consciousness. Generally, when the pulses of the radial artery of the wounded is within the normal range of 80–90 bpm, the blood loss is usually less than 750 mL; when the pulse of the radial artery is accelerated to 90–120 bpm, and the wounded is nervous and excited, it indicates a blood loss volume of 800–1500 mL; when the radial artery pulse of the wounded increases to 130–160 beats/min, which is too weak to feel clearly, and the wounded is confused and indifferent, it indicates that the wounded has a bleeding of 1500–2000 mL; when the radial pulse is so weak that it cannot be felt, and the wounded is in a coma, it indicates a blood loss volume of the wounded beyond 2000 mL with severe hypotension and shock. When the bleeding volume of the wounded is judged as more than 1500 mL, it is necessary to request the help of the healthcare worker and the military doctor for urgent infusion, and prior evacuation.

3.1.3 Recognition of Shock

Recognition of shock on the battlefield is generally done by battlefield lifeguards and healthcare workers, but battlefield commanders need to master the method of identifying shock in order to identify shock patients as early as possible and arrange their emergency evacuation. The above methods of judging the bleeding amount of the wounded are helpful to judge whether the wounded is in shock. On the battlefield, it is recommended to use a simple, effective, and rapid method of judgment, including two observation indicators, that is, consciousness, and the pulsation of the radial artery. If the consciousness of the injured is abnormal and/or the radial artery pulses significantly increase to more than 120 beats/min, which is weakened or disappeared, in the absence of head trauma, the injured is judged as in shock, so that if possible, the injured should be arranged to evacuate urgently.

In disaster relief in peacetime, comprehensive indicators such as blood pressure measured by a sphygmomanometer can be used to assess shock.

3.2 On-Site First Aids for Severe Hemorrhage Caused by Blast Injury

During the battlefield (on-site) first aid phase, tourniquet, tamponade, and finger pressure can be used to stop bleeding, where the former two have the best effects.

3.2.1 Tourniquet Application for Bleeding Control

The practical experience in the treatment of war injuries in recent years has shown that the active and correct use of tourniquets on the battlefield can save lives more effectively, and the death rate of the wounded has been significantly reduced. Common tourniquets include rubber tourniquets, pneumatic tourniquets, cassette type tourniquets, and spinning tourniquets. In the peacetime, tourniquets are only used for active

bleeding in the upper arm, forearm, thigh or calf that endangers the life of the wounded. On the battlefield, tourniquets are mainly used for temporary hemostasis for fire rescue, and generally should not be used for more than 2 h. In subsequent treatments, the tourniquet will be removed (which can still be used if necessary). Long-term bandage (>2 h) may cause serious complications such as avascular necrosis of the limbs.

Spinning tourniquets are preferred when possible. In the absence of standard tourniquets, ties, sphygmomanometer cuffs, triangle bandages, and wooden sticks can be used as substitutes for temporary hemostasis, which is also an effective treatment.

1. Indications. The indication for the tourniquets at the battle site is fatal hemorrhage in the extremities. How to judge it: constantly blood gushing or pulsating jet bleeding from the wound. It is most common in dismemberment or destruction of limbs.

In case of a large foreign matter in the wound which cannot be removed and the bleeding still continues, a tourniquet can also be used at the proximal end of the wound to control the bleeding.

2. Contraindications. When the bleeding is not fatal with a small amount, the tourniquet should not be used blindly; otherwise it will increase the probability of limb necrosis. How to judge it: there is no continuous large amount of blood gushing, outflow, or jet-like bleeding from the wound. How to deal with it. If the battlefield conditions permit when the enemy's firepower is suppressed and the surrounding environment is relatively safe, bandage the wound with a hemostatic dressing or ordinary dressing.
3. Spinning tourniquet operation. When one of the upper limbs of the wounded is not injured, hemostatic operations that require a tourniquet can be performed by themselves or their comrades in arms. Bandage by tourniquets in mutual rescue.
   (a) Check the injured limb and determine the location of the tourniquet. Take the spinning tourniquet from the pocket. Put the limbs of the wounded into the loop of the self-retaining belt. If there is no threat of enemy fire, a tourniquet should be tied 5–10 cm near the wound, directly on the skin surface, but not at the joints. When threatened by the enemy's firepower, or under other urgent situations, a tourniquet can be placed directly outside the underwear of the proximal limbs, and the tourniquet can be improved after the local fire threat is removed.
   (b) Pass the tourniquet tail through the opening of the buckle, tighten it, and then stick it around the limb. Tighten the short stick until the bleeding stops, and fix the short stick on the short stick clamp. At the smaller extremities, wrap the remaining self-retaining belt around the winch clamp. For thicker thighs, rectangular ribbed non-slip buckle must be used. Tighten the self-adhesive tape, and then stick it on the Velcro to end the tourniquet tying.
   (c) Reassess and record the condition of the distal end of the injured limb and check the effectiveness of the tourniquet. Check whether the pulse of the distal arteries disappears or whether the bleeding of the limbs has stopped. If the arterial pulsation does not disappear, or venous stasis occurs, it indicates that the tourniquet has not achieved the desired effect.
   (d) Record and mark the time of the tourniquet with an oily marker, and ensure that it is handed over to the staff of the receiving institution, indicating that the wounded has been tied with the tourniquet.
   (e) Wrap the wound with the sterile dressing in the first aid kit.
4. Precautions for bandage by tourniquets
   (a) When using a temporary tourniquet, be sure to mark the time of use of the tourniquet, and the markings need to be waterproof and scratch-proof; continue to check and treat to ensure that the bleeding of the wound is under control, and treat other life-threatening conditions simultaneously; record the treatment and the wounded evacuation.
   (b) The ischemic tolerance time of the distal limbs after the tourniquet is tied is about 2 h, and definitive hemostasis measures must be implemented before then. After 2 or more hours, necrosis of limb tissues, especially muscle tissues, begins to appear. After 6 or more hours, the necrotic tissue will release a large amount of toxins. At this time, the tourniquet cannot be loosened at will, otherwise the toxins at the distal end will flow back into the blood circulation of the whole body, which is life-threatening.
   (c) After the tourniquet is tied up in the limbs, discomfort and pain will usually occur. The tourniquet should not be loosened just because of pain.

3.2.2 Hemostatic Dressing Packing and Compression for Bleeding Control

Hemostatic dressing refers to loading hemostatic drugs on gauze, which can stop bleeding after covering the wound. When there is no special hemostatic dressing, ordinary gauze or oil gauze dressing can also be used to stop bleeding. The indications mainly include the followings. (1) For bleeding in the limb junction area (such as the groin area and axilla) and open chest and abdominal cavity, where tourniquets cannot be used, hemostatic dressings can be used to stop bleeding. (2) Massive bleeding in extremities which cannot be effectively controlled by tourniquets, hemostatic dressings can be used additionally to stop the bleeding. (3) It is also applicable to non-fatal wound bleeding.

How and when to use it. When using a hemostatic dressing, 2–3 min of pressing is needed to get an effective hemostatic effect. Therefore, this method of hemostasis can only be used when the enemy's firepower is effectively suppressed and the surrounding environment is relatively safe, otherwise the rescuer is more likely to be exposed to enemy fire for a long time, causing unnecessary personal injury. If there is obvious injury to the oral cavity, it is advisable to fill it with a dressing and press for a few minutes, and fix it with a bandage.

3.2.3 Direct Compression Hemostasis

Direct compression hemostasis is applicable to rapid temporary hemostasis. The superficial blood vessels mentioned above are usually chosen as the pressure points for stopping blood pressure. Rescuers or the wounded themselves can press the blood vessels to the back bone or soft tissues by their hands or fingers, dressings or knees, so that the vascular cavity could be compressed and closed to prevent blood flow to achieve hemostasis. In order to achieve effective compression, two hands should be used with force, and the wounded should be placed on a hard ground to ensure that the effective pulsatile pressure exists, and the pressure should be maintained until the wounded is sent to a place where the blood vessel can be surgically repaired. During the transportation of the wounded on a stretcher, direct pressing is usually ineffective. Be sure to continue to press the wound directly, rather than repeatedly and intermittently stop pressing to check the bleeding point. For all these reasons, tourniquets and hemostatic dressings are a good way to control bleeding on the battlefield. Because it is difficult to maintain the necessary pressure, and there usually are more than one blood vessel ruptures in the wound, direct compression hemostasis can only be used as a temporary hemostasis method when no other hemostasis measures are available, or as a temporary measure in the case of compression bandaging. Commonly used compression hemostasis points include the followings.

1. Common carotid artery. The left common carotid artery originates from the aortic arch, and the right common carotid artery originates from the trunk of the head and passes through the sternoclavicular joint, ascends along both sides of the esophagus, trachea, and larynx, and separates into the internal carotid artery and the external carotid artery at the level of thyroid cartilage. The former vertically ascends into the brain to innervate the blood supply of brain tissue; the latter ascends through the parotid gland to the mandibular neck into the superficial temporal artery and the maxillary artery.

Place the fingertips of the index and middle fingers about 3 cm next to the injured person's Adam's apple, and compress this position which has a certain hemostatic effect on maxillofacial and head bleeding.

2. Mandibular artery. It is a branch of the external carotid artery which is distributed in the anterior muscles and other tissues. When hemorrhage occurs in the face, pulses about one transverse finger in front of the mandibular angle can be touched, on which compression can stop the bleeding.
3. Superficial temporal artery. The superficial temporal artery is sent out through the upper back of the mandibular neck in the parotid gland to the front of the tragus, crosses the surface of the zygomatic arch root to reach the temporal region, and its branches are distributed in the parotid gland and the soft tissue of the temporal region. Its anatomical location is the surface of the zygomatic arch root in front of the tragus. The position on the skin surface can be determined by touching its pulse about 2 cm directly in front of the opening of the ear, on which compression can stop facial bleeding.
4. Brachial artery. The brachial artery is the continuation of the axillary artery, which is the main blood supply artery of the upper limbs. Positioning method. It locates in the inner side of the biceps brachii in the middle of the upper arm, on which compression can stop bleeding.
5. Radial artery. Radial artery originates from the brachial artery, walks along the thumb side of the forearm, and its terminal branches are distributed in the hand. When examining the pulse of the radial artery, place the tip of the index finger and the middle finger on the thumb side of the injured wrist, on which compression can stop bleeding in hands.
6. Femoral artery. The femoral artery is the main trunk of the lower extremity artery, which continues from the external iliac artery and goes down to the popliteal fossa where it is renamed the popliteal artery. It is palpable and pulsating at the junction of the inner thigh and the lower third and upper two-thirds of the groin on which compression can stop bleeding in the lower limbs.
7. Posterior tibial artery. The posterior tibial artery, a direct continuation of the popliteal artery, walks between the superficial and deep muscles of the calf, passes through the back of the medial malleolus and enters the plantar, which can be divided into plantar medial and lateral arteries to nourish the ankle joints and feet. When examining the pulse of the posterior tibial artery, place the fingertips of the index and middle fingers about 1.5 cm behind the injured medial malleolus, on which compression can stop bleeding in the ankle.
8. Arteria dorsalis pedis. It is a continuation of the anterior tibial artery and provides blood supply to the foot together with the terminal branch of the posterior tibial artery. Positioning method. It locates in the vertical extension line of the midpoint of the middle of the back of the foot and the inner and outer ankles.

3.2.4 Hemostatic Techniques for Closed Massive Abdominal Hemorrhage

Statistics from the early days of the Iraq war showed that massive bleeding and airway obstruction were the main causes of avoidable casualties. In recent years, with the improvement of pre-hospital emergency technology and the widespread use of tourniquets and hemostatic dressings, the casualty rate caused by massive limb bleeding and airway obstruction in pre-hospital emergency has significantly decreased, while the casualty rate caused by closed abdominal hemorrhage has increased significantly, which becomes one of the main causes of avoidable casualties. Closed abdominal hemorrhage accounted for 51.5% of avoidable casualties during the war in Afghanistan, while data from the US Level I Trauma Center showed that closed abdominal hemorrhage accounted for 42.3% of avoidable casualties, and it was increasing year by year.

Closed abdominal hemorrhage is mostly caused by blunt trauma leading to abdominal organ damage, which cannot be packed to stop bleeding during transportation like open abdominal hemorrhage, and it is often necessary to go to the hospital for surgery to effectively control the bleeding. However, a considerable part of the patients suffered shock due to the inability to control abdominal hemorrhage during the transit, which led to death. Therefore, it is necessary to develop a hemostatic device that can effectively control the closed abdominal hemorrhage during the pre-hospital emergency treatment, so that the wounded can be safely transported to the hospital for definitive hemostasis and reduce its casualty rate. The U.S. military now uses an injectable hemostatic device, where two polypropylene chemicals are injected into the abdominal cavity, and chemically react so that the volume expands to more than 30 times, which can effectively compress and temporarily stop the bleeding. In our previous work, we developed an injection hemostatic device that can temporarily control closed abdominal injuries. It has a double-cavity structure, one cavity is pre-filled with Xstat, an "expandable" agglutinant hemostatic agent and a compressed sponge that can swell more than ten times after absorbing water, so as to concentrate local platelets and coagulation factors, as well as compress the bleeding point; the other cavity is loaded with salmon thrombin-fibrinogen (STF), a procoagulant hemostatic agent, which can directly activate the coagulation system to trigger coagulation. During use, after injecting the hemostatic drug into the abdominal cavity through a small incision under the navel, it can exert the compression and pro-agglutination effects of Xstat. At the same time, STF uses the expansion of the sponge to increase its hemostatic area, which can effectively control the fatal closed abdominal hemorrhage, and transport the wounded to the hospital for definitive treatment. This design also has the advantages of portability, convenience, cheapness, and safety without toxic side effects.

3.3 First Aids for Shock

When it is judged that the wounded is in shock on the battlefield/on site, the wounded should be moved to a safe area after immediately ensuring the battlefield environment is safe. Fluid resuscitation or oral rehydration for anti-shock treatment should initiate as soon as possible after controlling the fatal bleeding, and the wounded should be evacuated first.

3.3.1 Oral Rehydration

U.S. military experience in the Afghan war showed that some shock victims did not receive any form of infusion when they arrived at the field hospital, and the delay in their evacuation caused severe dehydration of these wounded. Therefore, they recommend oral rehydration for the wounded with clear consciousness, normal swallowing function, and no gastrointestinal injury, as an auxiliary rehydration method for anti-shock. At the same time, we recommend oral rehydration as soon as possible when there is no condition for intravenous or intraosseous infusion, or there are so many wounded personnel that intravenous channels cannot be established in all the patients in shock, and the wounded have no contraindications such as gastrointestinal injury.

Salt-containing beverages are generally selected for oral rehydration with 3 g of salt, 1.5 g of sodium bicarbonate, and 10 g of sugar in 1000 mL of drinking water to make a drink for burns, or 5% glucose and sodium chloride injection. Oral administration should be done in small amounts but multiple times. Too much and too fast oral rehydration can cause vomiting, abdominal distension, and even acute gastric dilatation. When frequent vomiting or gastric retention occurs, oral rehydration should be stopped, and intravenous and intraosseous channels should be established as soon as possible for rehydration.

3.3.2 Initiate Fluid Resuscitation Intravenously or Through Marrow Cavity Infusion Channels, If Conditions Permit

The earlier fluid resuscitation is initiated, the better the anti-shock effect is. I have participated in the Tactical Combat Casualty Care (TCCC) training of the German and US forces in 2014, which both requested to establish intravenous or marrow cavity infusion channels on the battlefield. Therefore, if conditions permit, intravenous or intraosseous channels can be established from the battlefield for fluid resuscitation, especially when it is estimated that evacuation will be delayed. If it is estimated that the wounded can be evacuated quickly, and can be sent to the next level of treatment facility within 30 min, the infusion channel can be established in the emergency treatment facility and the fluid resuscitation process can be initiated then.

In the process of fluid resuscitation, it should be noted that the wounded with blast injury usually suffer from lung injuries so that they cannot tolerate rapid and large amounts of fluids infusion. Otherwise, heart failure is prone to occur. Infusion speed should be tightly controlled and the average arterial pressure should be controlled at 80 mmHg.

3.3.3 Other Treatments

Other comprehensive treatments include loosening collars, belts, shoelaces, keeping the airway unobstructed, and keeping warm.

4 Treatment of Pneumothorax and Hemothorax in Blast Injury

4.1 Recognition and Management of Tension Pneumothorax of Blast Injury

4.1.1 How to Identify Tension Pneumothorax On Site

Different severity degrees of tension pneumothorax varies in symptoms and signs. The patient may manifest fear and discomfort, usually with chest pain and dyspnea. As the condition deteriorates, dyspnea will be more and more severe. In severe cases, cyanosis and apnea may occur. The most classic physical examination findings are that the trachea deviates from the injured side, the breath sounds on the injured side are weakened, and the percussion sounds are drum sounds. However, in the noisy environment during wartime, this sign is not easy to find.

In wartime, the wounded with tension pneumothorax can be identified based on the following signs.

1. There is a history of chest injury with chest wounds, while some patients may not have obvious wounds.
2. Dyspnea progressively worsens, manifested as an increased respiratory rate, which can reach 20–40 times/min, with difficult breathing.
3. Breath sounds on the injured side are weakened or disappeared.
4. The chest on the injured side is uplifted compared to the opposite side, subcutaneous emphysema, distended jugular vein, and weak pulse.
5. As the pressure in the chest cavity continues to increase, tachycardia and shortness of breath become increasingly prominent, which eventually leads to hypotension and shock.

4.1.2 First Aid for Tension Pneumothorax

Tension pneumothorax is a life-threatening emergency and requires puncture decompression, especially in situations such as exacerbated dyspnea, decreased or disappearing unilateral breath sounds, and shock, urgent puncture decompression is needed. Detailed steps are as follows.

1. Take out the thoracic puncture catheter needle from the first aid kit. Generally, a puncture needle with a larger diameter (10–16G) should be selected with the length of at least 8 cm, because a needle that is too short is difficult to penetrate the chest wall, or because the catheter may be buckled after the removal of the needle which will interfere with air drainage.
2. The puncture position is located in the second intercostal space (between the second and third ribs) along the midclavicular line of the injured side of the chest, because this position is easy for punctuation for pre-hospital first rescuers, when generally, the injured is lying on his/her back with his/her arms close to the sides; besides, the injured lung collapses and shifts to the opposite side so that during the operation, the lung would not be injured by the needle. Positioning: two horizontal fingers down from the midpoint of the clavicle.
3. If time permits, wipe the area to be punctured with disinfectant.
4. Insert the needle at the insertion point at the top of the third rib at a 90° angle, and stop inserting the needle once the hissing sound of air is heard. When the needle enters the chest cavity, a sense of breakthrough can be felt, or air can escape. Note: Inserting the needle too deeply may damage the underlying lung tissue or other vital organs.
5. Fix the catheter and remove the needle core.
6. Fix the catheter firmly with tape.
7. Production of puncture needle tail valve: Whether or not to make a valve on the battlefield is still controversial. Opponents believe that since the diameter ratio of the puncture decompression catheter is much smaller than that of the patient's airway, the air circulation through the catheter cannot significantly affect the respiratory movement, even if the valve is not used, that is, punctuation without a valve placement will only transform a tension pneumothorax into negligible open pneumothorax. We support the use of gloves to make a one-way valve to improve the decompression effect when conditions permit on the battlefield, especially when the evacuation time is estimated to be long or may be delayed.
8. The catheter can be placed in situ or flushed with saline every 2 h to keep it flow smoothly. If conditions permit, the catheter can be removed, and then the patient can be closely monitored to find signs of increased tension and "re-puncture" if necessary. Note: Once a patient develops a tension pneumothorax, his/her condition will continue to deteriorate unless the wounded receives a thoracic tube and closed drainage. Therefore, when symptoms such as dyspnea of the wounded after decompression become

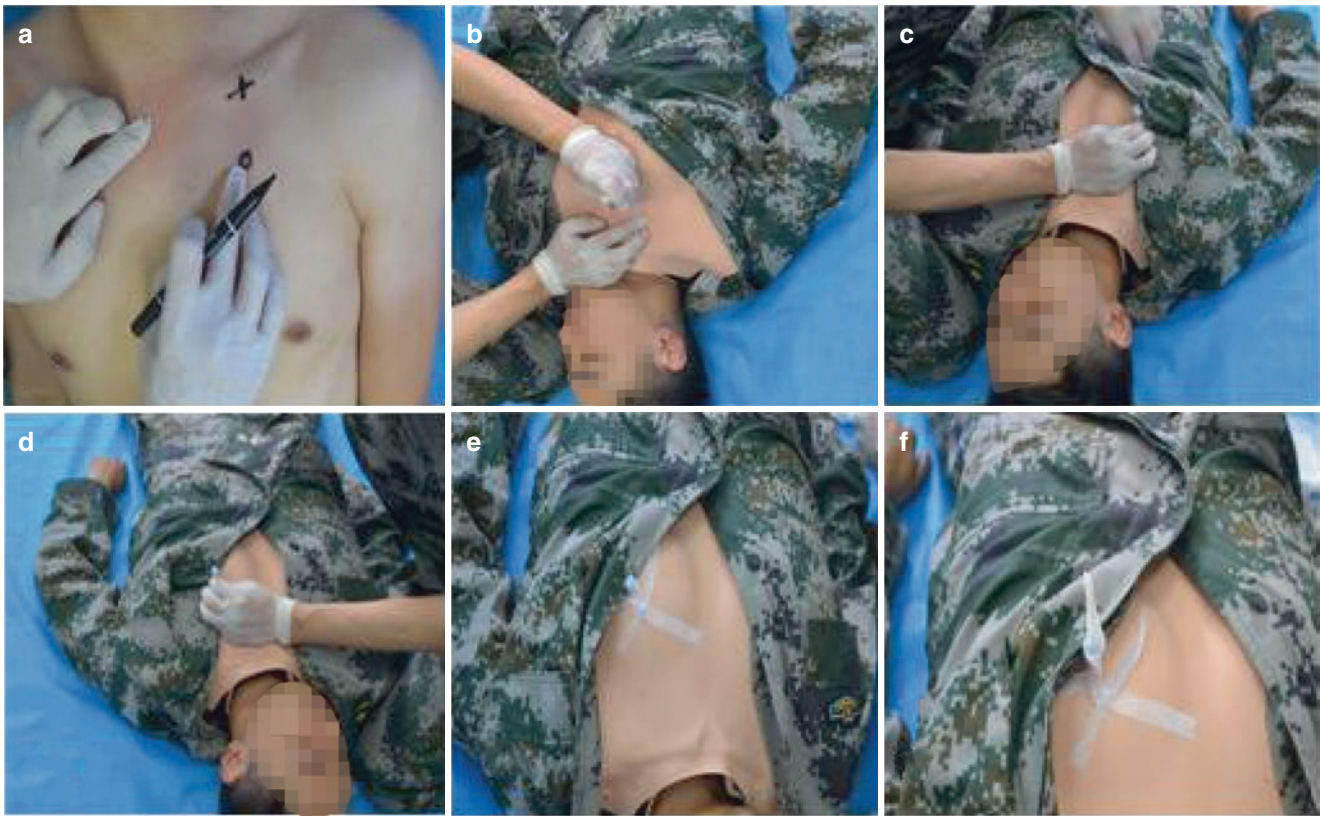

Fig. 1 Treatment of tension pneumothorax. (**a**) Determine the puncture position; (**b**) Pierce the puncture needle vertically; (**c**) Pull out the needle core; (**d**) After pulling out the needle core; (**e**) Fix the exhaust tube; (**f**) Create a single-direction valve with glove

worse, the commander should arrange for an emergency evacuation (Fig. 1).

4.2 Recognition and Treatment of Open Pneumothorax of Blast Injury

4.2.1 How to Identify Open Pneumothorax On Site

The wounded with open pneumothorax can be identified on the battlefield based on the following special signs.

1. History of trauma.
2. A wound on the chest wall.
3. A sucking or hissing sound on the chest wall. Air enters the wound during the inspiration of the wounded with open chest injury, while exit during the expiration. Air currents sometimes cause sucking or hissing sounds. Due to such unique physical signs, it is called a sucking chest wound.
4. Dyspnea. The main manifestations include abnormal breathing rate beyond 10–30 breaths/min, shortness of breath, respiratory distress (deepened and increased respiratory amplitude), and obviously hypoxic presentations in tissues (blue lips, cyanosis).
5. Visible penetration wound on the chest (front or back) or penetrating objects protruding from the chest wall.
6. A lot of air bubbles in the blood of the wound. The wounded can cough up bright red blood or foamy blood.
7. The chest cannot rise as normal during inspiration.

4.2.2 On-Site First Aid for Open Pneumothorax

One of the purposes of treating open chest injuries is to prevent outside air from entering the chest cavity through the wound, which can prevent lung collapse, or at least slow it down. The dressing used for wounds must be placed between the wound and clothing in order to effectively prevent air from entering the chest cavity through the dressing and wound. The Chinese army's third-generation first aid kit is equipped with a standard chest patch. The following is the procedure of using chest patch to close open pneumothorax (Fig. 2): (1) Clean around the wound with gauze as much as possible to increase adhesion; (2) apply tincture of iodine around the wound to increase adhesion; (3) tear the back of the dressing to expose the adhesive surface; (4) tell the casualty to exhale and hold his or her breath to make the gas in the chest partially expelled from the body; (5) put the dressing to the wound around the patient's chest wall. The transparent material allows better observation of the wound; and (6) wrap the wound with battle dressing.

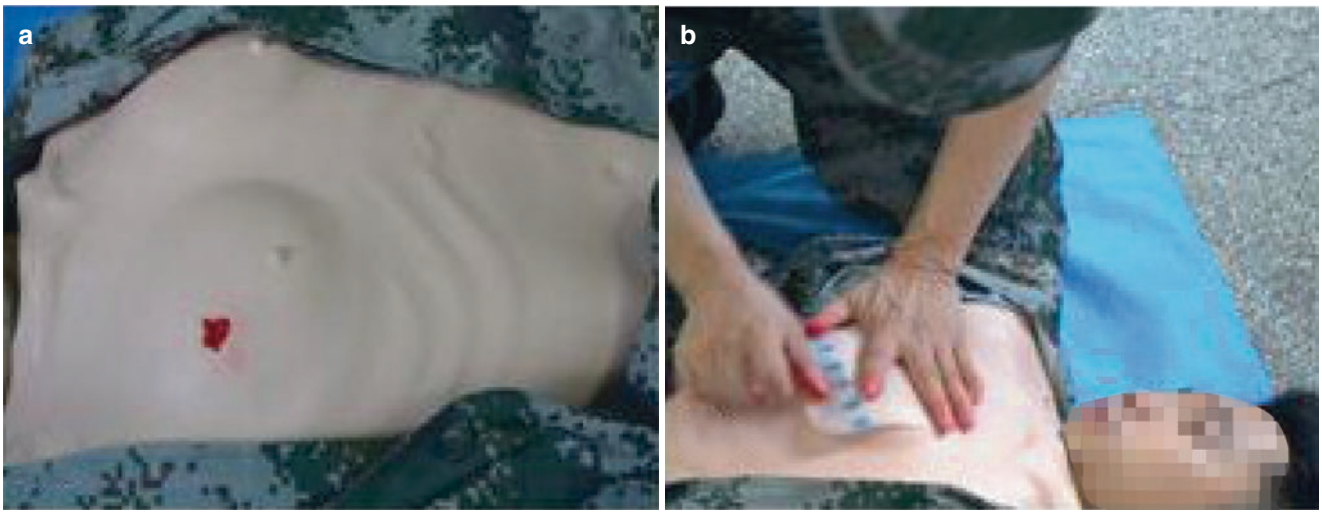

Fig. 2 Chest sealing for open pneumothorax. (**a**) Open injury on the right chest; (**b**) Ask the wounded to breathe out and hold breath, and then quickly close the wound

[Precautions]

1. If there is a piercing object protruding from the chest wound, a wide dressing can be used to wrap the wound without covering or moving the object. Then, stabilize the penetration by placing a clean dressing around the protrusion.
2. Position of the wounded. Place the wounded in a comfortable position when he/she is conscious. Most people may ask to sit up, which is allowed if tactical conditions permit. If the wounded cannot protect their airway unconsciously, place the wounded in the recovery position.
   (a) Lateral position: A lateral position can help open the airway and help fluid drain from the mouth of the wounded. When lying on the injured side, the ground plays a fixed role and helps to relieve pain. The lungs on the uninjured side are not restricted and can be fully expanded during inhalation.
   (b) Sitting position: The injured may wish to sit up. If he/she breathes more smoothly in a sitting position than in a lying position, ask him sit up to lean against a tree, wall, or other stable support. If the injured becomes tired, place him/her in a recovery position.
3. Use substitutes for temporary filling. If the above-mentioned standard pneumothorax dressing is not available, a convenient device with better airtightness can be used to seal the open pneumothorax wound, such as the plastic wrap of the first aid kit, poncho, metal foil, etc., and then fix it with tape.
4. Pay attention to closely observe the condition of the wounded. If the wounded becomes more difficult to breathe after the wound is closed, which indicates that a tension pneumothorax may occur, Emergency puncture and decompression can be performed, or the wounded has a large amount of hemothorax that needs urgent treatment, emergent evacuation to a suitable treatment facility is needed.

4.3 Recognition and Treatment of Massive Hemothorax of Blast Injury

4.3.1 How to Identify Massive Hemothorax

Two prominent signs are chest pain and shortness of breath, usually accompanied by symptoms of severe shock, such as tachycardia, shortness of breath, confusion, pallor, and low blood pressure. Breath sounds on the injured side is weakened or even disappear, but dullness can be heard on percussion (contrast with drum sounds of pneumothorax). Pneumothorax may be combined with hemothorax, which will exacerbate the damage to the heart and lungs. Because of the decrease in circulating blood volume, the distended jugular vein will disappear. On the battlefield, it is difficult to make an accurate judgment on the hemothorax. For patients with chest trauma accompanied by shortness of breath and other symptoms, if the symptoms of emergency puncture decompression are not relieved, it should be suspected that there is massive hemothorax.

4.3.2 On-Site First Aid for Massive Hemothorax

Such patients all need adequate oxygen supply, where a mask can be used to supply oxygen when the airway is obstructed, or tracheal intubation can be performed when conditions permit and physical signs indicate the need. For those with shock, active fluid resuscitation should be

Fig. 3 Closed-tube thoracostomy. (**a**) Positioning; (**b**) Tube placement

initiated. Patients with massive hemothorax need thoracic catheterization with following operation steps (Fig. 3).

1. Place the injured person in a supine position, with the arm on the affected side raised above the head.
2. Choose the anterior axillary line and the fourth or fifth intercostal junction as the insertion point. After routine disinfection, a 2–3 cm transverse incision is made at the selected position and extends down to the intercostal muscles. Insert the large forceps into the upper intercostal muscle of the skin incision, pierce the pleural wall with the tip of the forceps, and then slightly open the forceps by 1.5–2 cm, and then enlarge the hole.
3. Use one finger to go deep into the incision to remove any adhesions, blood clots, etc., when withdrawing the fingers, clamp the tip of the chest tube with forceps, and insert the tip of the chest tube into the incision. Send the chest tube inside until the last side hole is also sent into the chest wall, reaching 2.5–5 cm deep.
4. Connect the other end of the chest tube to the one-way exhaust valve, and fix the chest tube with sutures, and then use an occlusive dressing to wrap the incision site.
5. Evacuate the patient in priority.

5 Bandaging and Fixation of Blast Injury

Due to the various equipment and methods used for bandaging and fixation, this section mainly explains the wound dressing methods of typical parts with the most practical battlefield dressings, and briefly introduces the methods of dressing wounds by triangle bandages and conventional bandages. In terms of fixation, this section focuses on the method of using standard splints to fix long bone fractures.

5.1 Open Neck Injury

It is not necessary to fix the cervical spine for penetrating neck injuries, because under enemy fire, the potential risks of cervical spine fixation to rescuers and injured persons outweigh the potential benefits. Although it is a standard procedure for civilian injuries to fix the cervical spine before transporting the wounded who may injure the spine, this operation is usually not applicable in a battlefield environment. According to a study that specifically analyzed the value of cervical spine fixation for the penetrating neck wounded in the Vietnam War, results show that only 1.4% of such casualties may benefit from this operation. Even a skilled rescuer needed 5.5 min to complete the fixation of the cervical spine. Therefore, for open neck injuries, cervical spine fixation is not recommended on the battlefield.

For the wounded with obvious blunt contusion and neck pain, the possibility of cervical spine injury should be suspected. At this time, cervical spine fixation is still necessary, unless there is a situation where enemy fire poses a huge threat. Fixation steps: When the injured person is suspected of cervical spine injury (neck pain with or without loss of limb sensory movement), use a neck brace to fix the neck of the injured person; if there is no neck brace, use convenient equipment to fix the neck, if available, for example, army filled with sand can be placed on both sides of the wounded for fixation (Fig. 4).

5.2 Open Brain Injury

When the brain tissue bulges out from the brain injury, do not reinstate it. Cover it with a large sterile dressing soaked in isotonic saline, and then buckle a clean bowl to prevent the brain tissue from protruding further, and then bandage and fix it. At the same time, place the wounded to lie on the side, and remove the secretions, mucus or blood clots in the oral cavity to keep the airway unobstructed.

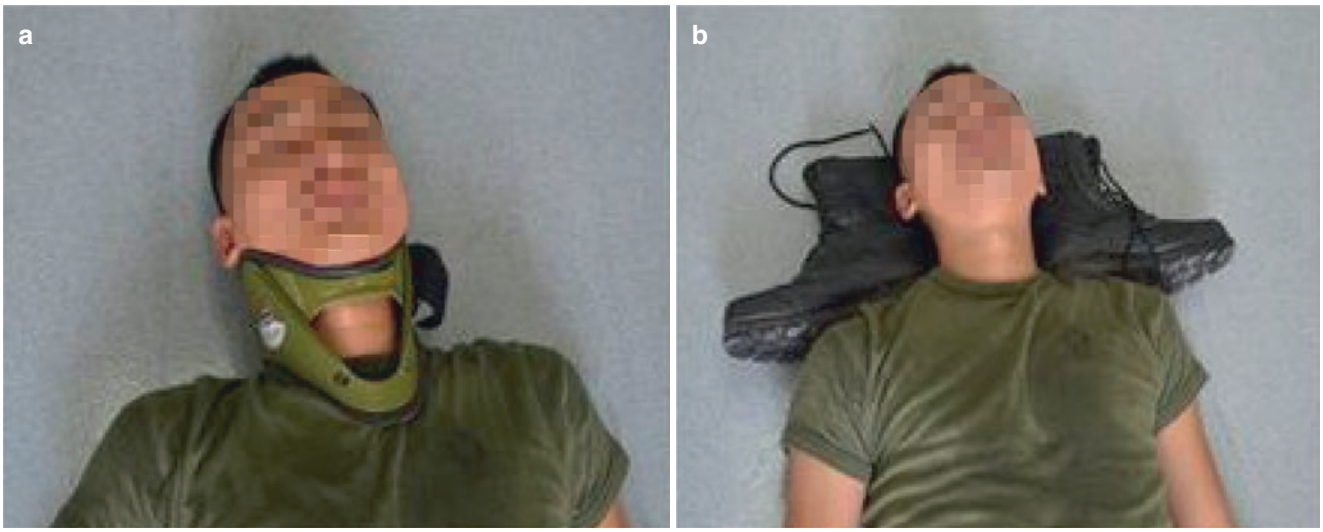

Fig. 4 Cervical spine fixation at the battle scene. (**a**) Fix the cervical spine by cervical brace; (**b**) Fix the cervical spine by military boots

5.3 Bandaging of Eye Trauma

Pressure on the eyeball will cause the contents of the eyeball to be squeezed out, leading to permanent damage and blindness. Therefore, the most important thing for eye trauma is to protect the eye from further damage. The processing steps are as follows.

1. Quick on-site vision test (after leaving the enemy's direct fire). Ask the wounded to count your fingers, read a paragraph of text casually, or distinguish brightness and darkness.
2. Cover the eyes with a hard eyeshade. For eye injury, a hard eyeshade or other convenient equipment (such as bottle caps) can be used to protect eyes as shown in Fig. 5, in order to avoid putting any pressure on the eyes. If a foreign matter is inserted into the eyeball, it is strictly forbidden to pull it out. It is best to fix the foreign matter with hard goggles or other findable protectors, then wrap with the sterile dressing and a bandage.
3. Bandage. Then the H-shaped battlefield dressing or Israeli dressing can be used to bandage the eye. Be careful not to apply excessive pressure. A triangle bandage or bandage can also be used to wrap the ocular injury. It should be noted that when one eyeball is seriously injured, both eyes should be covered and fixed, because the two eyes are moving together, so that bandage on both eyes can reduce the probability of further damage to the injured eye. But on the battlefield, if the enemy situation is still not resolved, the wounded still need to participate in the battle. At this time, the uninjured eyes must be exposed so that the wounded can see the outside world.

5.4 Bandaging Methods at the Junction of the Trunk and Limbs

1. Shoulder and axillary bandaging. It is easy to bandage the shoulder and axillary wounds with the H-shaped battlefield dressing. Place the aseptic side of the dressing on the wound, and then wrap the bandage around the shoulder and then wrap a bandage on one branch of the H-shaped compression buckle, change the direction of the bandage, wrap around the chest, and then pass the other branch corner of the H-shaped compression buckle to wrap around the shoulder. Finally, stick the end of the bandage on the dressing to complete the fixation (Fig. 6).
2. Inguinal and perineal bandaging. The inguinal and perineal bandaging is similar to that of the shoulder (Fig. 7). Place the aseptic side of the dressing on the wound, and then wrap the bandage around the thigh for a circle before wrapping the bandage on one branch of the press buckle to change the direction of the bandage and then go around the abdomen once, then change the direction of the bandage on the other branch of the H-shaped press buckle, wrap around the thighs, and stick the bandage on the dressing to complete the fixation.

5.5 How to Bandage a Wound Filled with Foreign Bodies

When shrapnel, glass, wooden sticks and other foreign bodies are found in the wound, be careful not to move the foreign bodies to avoid additional damage. At this time, gauze can be used to protect the foreign matter first, and then bandage the wound with a battle dressing (Fig. 8).

Fig. 5 Dressing method of eye injury. (**a**) Protect eyes by hard goggles; (**b**) Cover eyes by ordinary bandages; (**c**) Protect eyes by hard goggles; (**d**) Cover a single eye by Israel bandages

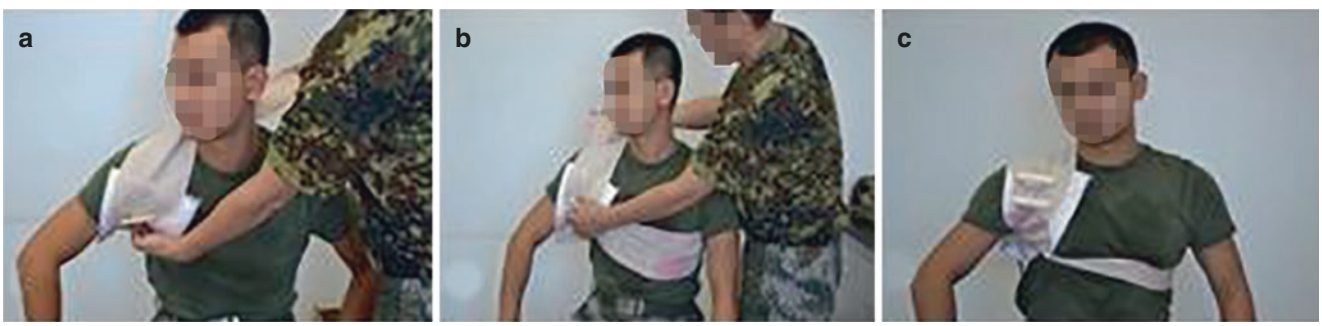

Fig. 6 Dressing method of shoulder injury. (**a**) Place the sterile side of the battle dressing on the wound; (**b**) Complete the bandage winding on the shoulder and chest one by one; (**c**) Complete the dressing

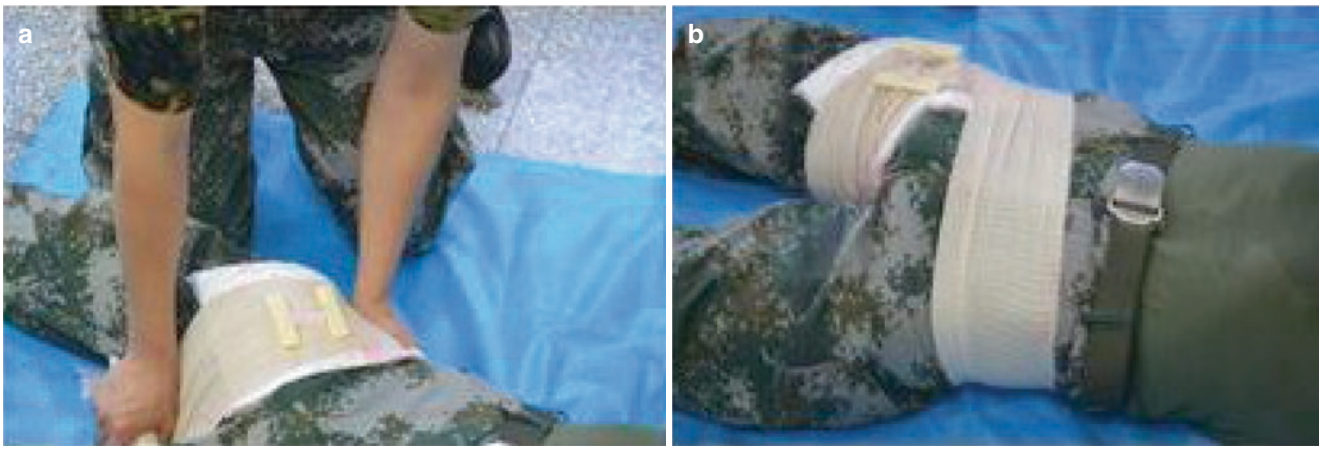

Fig. 7 Dressing method of inguinal injury. (**a**) Place the sterile side of battle dressing on the wound; (**b**) Complete the bandage winding on the thigh and abdomen one by one

Fig. 8 Bandage a wound with foreign bodies. (**a**) There is a steel strip penetrating into the wound of the left thigh with unknown depth; (**b**) Use gauze to protect the steel strip; (**c**) Bandage the wound with battle dressing

5.6 Abdominal Viscera Prolapse

For abdominal trauma with internal organs prolapsing, do not reinstate it, and cover it with a large sterile dressing soaked in isotonic saline, and then buckle with a sterile dressing bowl or sterile bowl to prevent the intestines and other internal organs from furthering prolapse, and then bandage and fix. If the prolapsed intestinal tube has been ruptured, use bowel forceps to clamp the perforation and rupture and wrap it in the dressing together. Note that the dressing that directly covers the internal organs must be soaked with isotonic saline to avoid adhesion, intestinal serosal or other internal organ damage, intestinal obstruction or other long-term complications.

5.7 Fixation of Long Bone Fractures

A simple way to identify a fracture on the battlefield includes signs such as obvious pain in the wound, deformity such as angulation, or activity (abnormal activity) in a place where movement should not occur, etc., indicating a fracture. If there is a wound with the bone out of the wound, it is an open fracture, which is easy to judge. If fractures of long bones of the limbs are not properly fixed, there will be many harms during the transportation and evacuation, such as severe pain, movement of the fractured end, resulting in increased bleeding, shock, or additional neurovascular damage. At this time, the wound should be wrapped up with an H-shaped battlefield dressing, and then roll splints should be placed on both sides of the injured limb to properly fix the injured limb (Fig. 9). It should be noted that when using a roll splint to fix the limbs, it is necessary to achieve "cross-joint" fixation. For example, when fixing the femur, the proximal end of the splint should exceed the hip joint, and the distal end should exceed the knee joint.

In early treatment institutions or emergency treatment institutions when conditions permit, external fixation brackets can be used to fix long bone fractures.

5.8 Fixation of Pelvic Fracture

In the first aid phase of the battle scene, when unstable pelvic fractures are estimated, the pelvis can be fixed with handy equipment such as pelvic straps or sheets. After arriving at the early treatment facility, the pelvis was fixed with an external pelvic fixator.

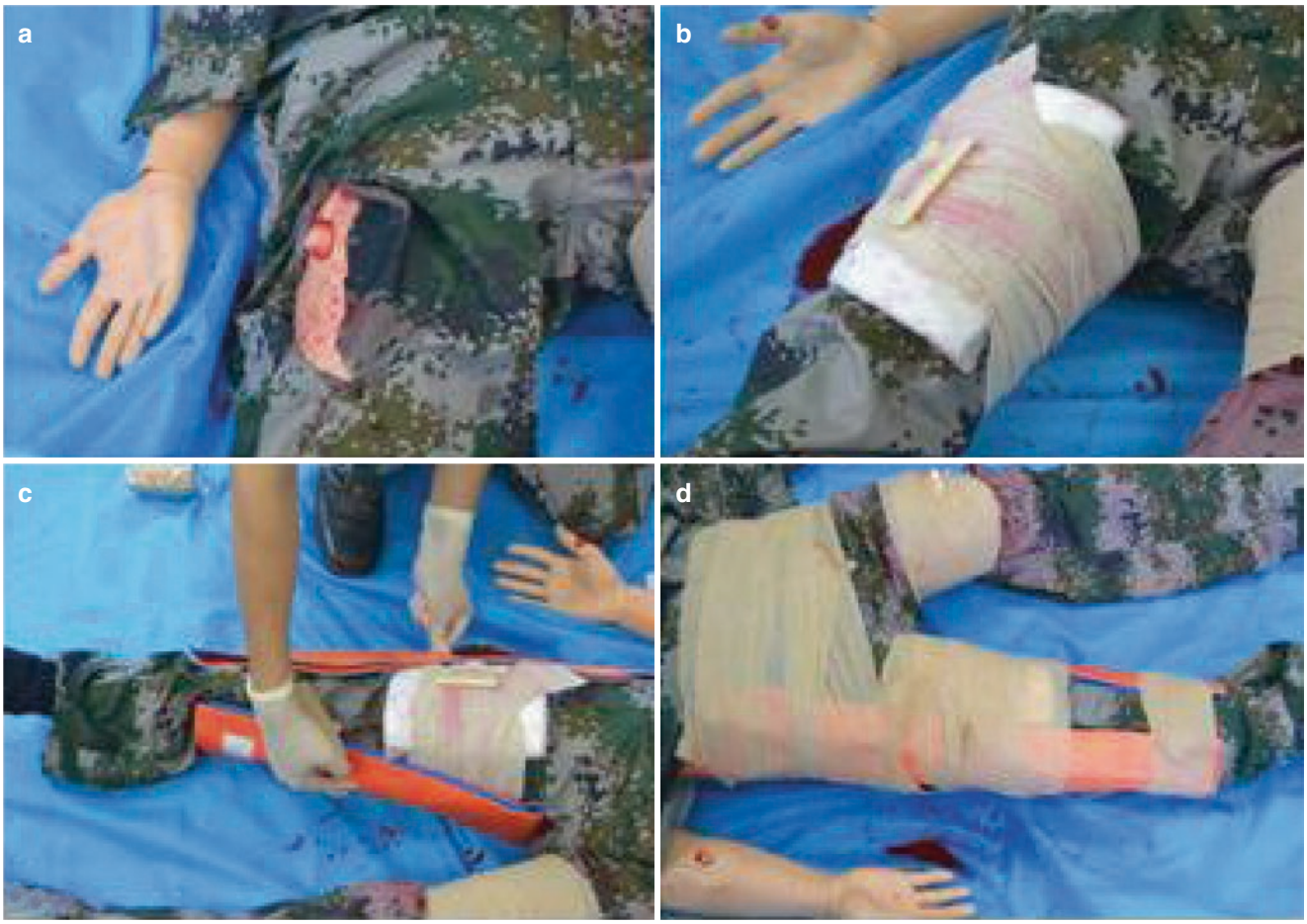

Fig. 9 Dressing and fixation of open limb fractures. (**a**) Open fracture of the right femur; (**b**) Bandage the wound properly with battle dressing. (**c**) Place splints on both sides of the injured limb. (**d**) Fix the injured limb with elastic bandage

6 Transportation Techniques in Blast Injury

In wartime, it is necessary to evacuate the wounded from the line of fire to a safe place such as a concealed place or a bunker, or move the wounded from the line of fire to a medical institution. Correct transportation techniques can reduce additional injuries to the wounded. In peacetime, a stretcher can be used to carry the wounded.

6.1 Carry by Crawling

Moving the wounded should be fast and safe. Therefore, it is necessary to make full use of the favorable terrain and choose the appropriate transportation method and posture. When the wounded is in the enemy's firepower range or the enemy's firepower has not completely suppressed at the line of fire, the piggyback transportation and carry by crawling sideways are generally used to move the wounded to a hidden place.

During the carry by crawling sideways, the rescuer crawls sideways to the wounded, adjusts the wounded to a back-to-side position, lifts the waist belt, and pads the waist and hips of the wounded on the flexed thighs of the rescuer (Fig. 10a). Then place the wounded's hands in front of the chest, and the rescuer's upper arm passes through the upper armpit of the wounded and wraps around the chest to hug the lower edge of the wounded upper arm deltoid muscle, close to the wounded's body (Fig. 10b). The rescuers' lower forearms and elbows are propped on the ground and kicked forward. Transport the injured to a safe place (Fig. 10c). This method is fast and relatively safe, where most of the injured person's body is under the protection of the carrier. But the disadvantage is that the carrier feels more laborious, which is not conducive to long-time transportation, and it is not suitable for when the injured part is on the lower back.

Piggyback transportation includes steps as follows. The rescuer prostrates to the wounded in a low posture, lies on the side of the wounded in the same direction and close to the wounded's body, and then pulls the wounded's upper arm from the upper shoulder with the lower hand and grasp the wounded's buttocks with the upper hand, turns the wounded up, crawls forward in a low posture, and moves the wounded to a hidden place. In the process of carrying, if the rescuer's strength is insufficient, the upper foot can be used to hook the

Fig. 10 Carry by crawling sideways. (**a**) Approaching the casualty; (**b**) Put the casualty at lateral position and onto the first aider's leg; (**c**) Move the casualty forward

Fig. 11 Drag. (**a**, **b**) Two-person clothes drag; (**c**) One-person drag

wounded person on the same side, and force with both hands to complete the carrying action quickly and effectively.

6.2 Drag

Drag is a commonly used method of transporting the wounded on the battlefield, including two-person clothes drag and one-person drag. The fastest way to transport the injured is the two-person clothes drag, in which two paramedics drag along the long axis of the injured body (Fig. 11a), applicable to situations such as in buildings, shallow water areas, snowy areas, and downstairs, which can be done when the rescuers are standing or crawling. The use of items such as tactical vests, pulling ropes, ponchos, clothing or temporary harnesses on the wounded can make this method of transportation easier (Fig. 11b). But in fact, it is enough to grab the wounded from under the injured arm. One-person drag can be used for short-distance transportation of the wounded, but compared with two-person clothes drag, one-person drag is more difficult for ambulance personnel, slower, and more difficult to control (Fig. 11c).

The biggest disadvantage of drag is that the injured personnel's body directly touches the ground, leading to additional damage when the injured person is transported in rugged terrain. When tactical conditions permit, it is a better choice to lift the wounded. Hawes carry is a method that can be used to quickly transport the wounded by one person (Fig. 12). If the wounded can maintain an upright position, the paramedics stand in front of the wounded and squat down, place the wounded's arm loop on the paramedic's neck, and hold it firmly. After that, the ambulance crew stood up and leaned forward, bearing the weight of the wounded and moving towards the designated location.

The members of team three of the Navy SEALs designed a two-person carry method and it has been put into use. In this method, each of two paramedics put one arm of the wounded on their shoulders, and lifted the wounded with their waist belt that was close to the wounded and grabbed the waist of the wounded (Fig. 13). If the wounded has conscious and both arms can hold two ambulance personnel, then the free hands of the ambulance personnel can use their weapons when necessary.

Fig. 12 Hawes carry. (**a**) Front view; (**b**) Side view

Fig. 13 SEAL Team Three carry. Method used by U.S. Navy SEALs to move casualty. (**a**) Put the arms of the casualty onto the shoulders of the rescuers, and grab the belt of the casualty; (**b**) Move the casualty forward

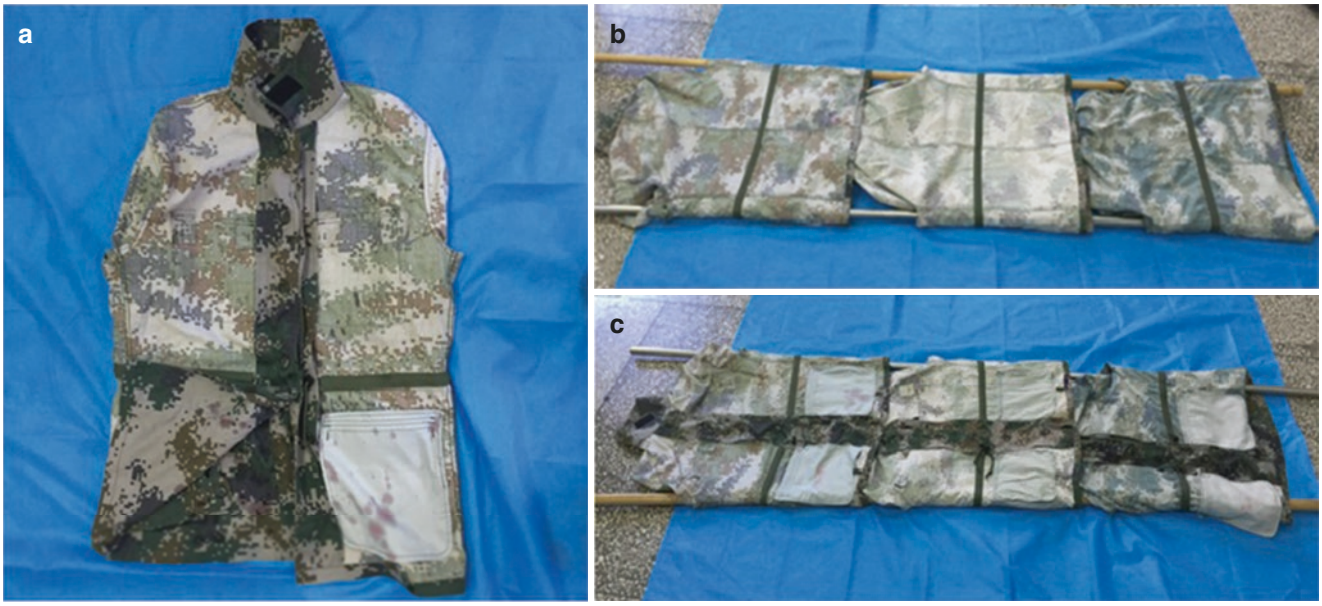

Fig. 14 Make a simple stretcher with expedient equipment. (**a**) Reverse the camouflage uniform and fasten the buttons; (**b**, **c**) Pass two bamboo poles through two to three camouflage uniforms and prepare a simple stretcher

6.3 Stretcher Transportation

When the wounded are rescued and organized for evacuation, a stretcher can be used for transportation. Here is a four-fold stretcher issued by our army to describe its transportation method: a transporter approached the wounded and turned the wounded over, taking care to keep the wounded body from twisting. At this time, another transporter put the stretcher behind the wounded, and then rolled the wounded on the stretcher. After adjusting the wounded to a suitable position, fasten the straps on both sides of the stretcher to fix the wounded. After unifying the password, the two stretchers lifted the wounded at the same time. If there is no standard stretcher, camouflage uniforms and bamboo poles can be used to make temporary stretchers according to the battlefield environment (Fig. 14).

When other parts of the spine are suspected of injury, the longitudinal axis of the injured person should be kept in a straight line to avoid distortion (Fig. 15).

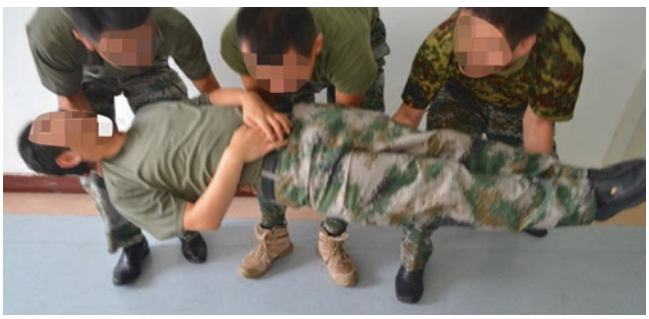

Fig. 15 Carry of the wounded suspected of spinal and spinal cord injury

7 Cardiopulmonary Resuscitation Techniques for Blast Injury

7.1 Indications for Cardiopulmonary Resuscitation

The purpose of cardiopulmonary resuscitation is to enable patients with cardiac arrest to maintain a certain level of vital organ perfusion and oxygen supply before receiving further life support treatment. Although cardiopulmonary resuscitation is helpful for heart patients with intact anatomical structures, it is of little value to the wounded who are severely injured on the battlefield causing cardiac arrest, respiratory arrest, or other vital signs disappearing. A large number of clinical data show that pre-hospital resuscitation of trauma patients in cardiac arrest is futile, even in some urban areas close to trauma care centers. In a recent study, Rosemurgy and his team reported 138 cases of war wounds that had undergone pre-hospital resuscitation, and none of them survived. Because of the threat of the harsh battlefield environment and the consistent failure of resuscitation, the editor does not recommend cardiopulmonary resuscitation for the wounded in the theater or even ordinary civilian patients.

Therefore, we do not advocate cardiopulmonary resuscitation on the war wounded at the battle scene, especially when under threat of enemy fire, but only on the war wounded

who are not in the head, chest, heart, heartbeat, and respiratory arrest, as well as hypothermia and drowning. Cardiopulmonary resuscitation for the wounded with apnea caused by drowning, freezing, and electric shock. In the peacetime disaster rescue, it is recommended to perform cardiopulmonary resuscitation for all the wounded with cardiac apnea.

7.2 Steps of Cardiopulmonary Resuscitation

1. Place the wounded in a supine position, on a flat ground or table, and the rescuer should kneel or benefit the upper body side of the wounded.
2. Clean up the respiratory tract. Quickly remove foreign bodies by fingers such as vomit, blood clots, secretions, mud, sand or water plants that block the wounded's respiratory tract.
3. External chest compression. On the left side of the midpoint of the wounded sternum and two nipples, the rescuers overlap the palms of the hands, press the base of the palms on this part, straighten both arms, and use upper body strength to press vertically downwards regularly. Compression depth: 5 cm. Compression speed: 80–100 times/min.
4. Mouth-to-mouth resuscitation. When there are two rescuers for the first aid at the same time, mouth-to-mouth resuscitation or simple respirator-assisted artificial ventilation together with external chest compression is recommended; if there is only one person for first aid, it is recommended to perform two cycles of mouth-to-mouth resuscitation after every 30 external chest compressions; external chest compression and mouth-to-mouth resuscitation should be performed in cycle without interruption until the spontaneous heartbeat and breathing is restored or the treatment is confirmed to be ineffective.

The indicators of effective CPR (cardiopulmonary resuscitation) are: (a) the pupils change from large to small; (b) the face turns from cyanotic to ruddy; (c) recovery of carotid artery pulsation; (d) active eyeball movement; (e) recovery of spontaneous breathing.

5. Evacuate the injured urgently after the heartbeat and breathing recover, and keep the airway unobstructed on the way.
6. After arriving at the early treatment facility, pay attention to maintain the stability of the heart function and internal environment. (a) Promptly replenish blood volume, maintain effective circulatory function and normal heart rate; (b) correct arrhythmia, microcirculatory disorders, electrolyte disorders and acid-base imbalance; (c) maintain respiratory function: tracheostomy and preoxygenation should be carried out if there are many respiratory droplets or the patient is unconscious for a long, and assisted ventilation is required when necessary; (d) defibrillation by electric shock is required if there is ventricular fibrillation

Blast Injury Management and Treatment in ICU

Hao Tang and Dongpo Jiang

Blast injuries have multiple injury mechanisms, characterized by externally minor but internally severe injuries involving multiple organs, rapid development, difficult diagnosis, and critical conditions. Intensive care unit (ICU), as a specialized department in the hospital for intensive monitoring and managing critically ill patients, could provide timely systematic and high-quality medical monitoring and treatment technology for patients with dysfunction of one or more organs or systems to threat patients' life, or with potentially high-risk factors caused by blast injuries. In the ICU, the wounded could be observed continuously and dynamically, qualitatively, and quantitatively through advanced diagnostic, monitoring and treatment equipment and technologies, and for severely wounded patients with blast injuries, standardized and high-quality life support could be provided through effective intervention measures to improve the quality of life.

1 Intensive Monitoring

1.1 Basic Vital Signs Monitoring

1. **ECG monitoring**. Clinicians could obtain the ECG activity changes through ECG monitoring, one of the basic contents of intensive care monitoring, which could monitor the patient's ECG activity continuously by a monitor, so that appropriate measures can be taken as soon as possible to deal with possible life-threatening malignant events.
2. **Noninvasive blood pressure monitoring**. In principle, noninvasive blood pressure should be monitored for all critically wounded patients as a routine monitoring item. The monitoring frequency should be adjusted according to the condition. For critically wounded patients or patients with significantly unstable hemodynamics, it should be replaced with invasive blood pressure monitoring. Current monitoring methods include manual cuff pressure measurement and electronic automatic pressure measurement.
3. **Pulse oxygen saturation monitoring**. The monitoring methods of oxygen saturation are usually divided into electrochemical method and optical method. The pulse oxygen saturation (SpO_2) is commonly monitored by an optical method with a good correlation with the arterial partial pressure of oxygen, which, meanwhile, could significantly reduce the number of arterial blood sampling, characterized of rapid, dynamic, and continuous monitoring so that its application clinically has been increasingly wide.
4. **Body temperature monitoring**. To determine the trend of the critically wounded patient's conditions, practitioners could dynamically monitor his/her body temperature, including the skin temperature and the core temperature and their difference. Currently commonly used thermometers include mercury thermometers and electronic thermometers. Temperature can be read directly and measured remotely by electronic thermometers meeting the needs to monitor the body temperature continuously. For patients with fever, the first is to find out the cause and then to control it aggressively. Meanwhile, cooling treatment should be actively given to reduce the patient's oxygen consumption and energy metabolism, including physical cooling, drug cooling, etc. Hypothermia may occur in severely wounded and extremely exhausted patients. Patients with blast injuries often suffer from hypothermia. When a shock is accompanied by hypothermia, the fatality rate will increase significantly.

1.2 Hemodynamic Monitoring

Hemodynamics include a series of indicators that reflect the functions of the heart, blood vessels, circulation volume, and tissue oxygen supply and oxygen consumption, providing a

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digital basis for clinical monitoring and clinical management. Generally, hemodynamic monitoring can be divided into two categories: noninvasive hemodynamic monitoring and invasive hemodynamic monitoring. During the noninvasive hemodynamic monitoring, indicators of various cardiovascular functions are obtained through methods without mechanical damage to the body so that it is safe, convenient, and acceptable for patients. During the invasive hemodynamic monitoring, the cardiovascular function parameters can be acquired directly by inserting various catheters and probes into the heart cavity or the blood vessel cavity through the body surface so that more comprehensive hemodynamic parameters can be obtained, which is conducive to an in-depth and comprehensive understanding of the condition, especially suitable for the diagnosis and treatment of critically wounded patients. However, it is harmful to the body to some degree, where improper operation can bring about complications. Hemodynamic evaluation methods include clinical manifestations, general monitoring, CVP, Swan-Ganz catheter, PiCCO monitoring, and volume responsiveness assessment based on the dynamic changes in monitored parameters. To determine the specific monitoring methods in the clinical practice, patients' conditions and treatment needs should be taken into consideration. When selecting monitoring methods, the pros and cons should be fully weighed, and the indications should be met properly.

1. **Invasive blood pressure monitoring**. As early as the eighteenth century, Hales inserted a catheter into the femoral artery of a horse to measure its blood pressure. Afterwards, there have been multiple studies on noninvasive blood pressure measurement. The oscillatory blood pressure monitoring technology described by Roy and Adami in 1890 has gradually matured, laying a theoretical foundation for current automatic noninvasive blood pressure monitoring. In the past two decades, invasive blood pressure monitoring has been the main method of hemodynamic monitoring in critically ill patients. The radial artery is commonly used for arterial puncture; others include the dorsal foot artery and femoral artery other than the brachial artery, generally. Invasive arterial blood pressure monitoring can provide clinicians with accurate, reliable, and continuous arterial blood pressure data.
2. **Central venous pressure monitoring**. In the late nineteenth century, people have realized the importance of right atrial pressure measurement through animal experiments. By the 1950s and 1960s, central venous pressure monitoring had been widely used clinically to assess blood volume, cardiac preload, and right heart function. To puncture the central vein percutaneously, the main paths are the internal jugular vein and subclavian vein through which the catheter was inserted into the superior vena cava; in addition, the femoral vein or cubital vein can also be selected via which a longer catheter will be used to insert into the upper or inferior vena cava. At present, such a technique has been widely used in patients with heart diseases and critical illness, which is generally safe. However, if the operator is not skilled enough, complications such as pneumothorax and bleeding may also occur. The reference value of CVP is 5–12 cmH_2O. CVP of lower than 5 cmH_2O indicates hypovolemia, while CVP of higher than 15 cmH_2O indicates cardiac insufficiency, excessive venous vasoconstriction or increased pulmonary circulatory resistance, and CVP of greater than 20 cmH_2O indicates congestive heart failure.
3. **Pulmonary artery floating catheter**. The emergence of pulmonary artery floating catheters is a milestone in the history of hemodynamics, which has brought a revolution in cardiovascular monitoring and made bedside monitoring of critically ill patients possible. By the Swan-Ganz catheter, practitioners can not only measure pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP), central venous pressure (CVP), right atrial pressure (RAP), and right ventricular pressure (RVP), but also measure the cardiac output by thermodilution and take mixed venous blood samples, which makes hemodynamic indicators more systematic and feeds back and guide treatment.

   The hemodynamic parameters available through the Swan-Ganz catheter mainly include three aspects: pressure parameters (including right atrial pressure, pulmonary artery wedge pressure, and pulmonary artery pressure), flow parameters (mainly cardiac output), and oxygen metabolism (mixed venous blood samples). Based on these parameters, combined with routine clinical examinations, more relevant parameters can be obtained through calculations. Commonly used hemodynamic parameters and reference normal ranges are given in Table 1.
4. **Pulse indicator continuous cardiac output measurement**. Pulse indicator continuous cardiac output (PiCCO) is a new technology combining the pulse contour method for continuous cardiac output combined with the transpulmonary thermodilution for cardiac output, which analyzes the arterial pulse contour and calculates the aortic compliance accompanied with thermodilution measurement. Individualized indicators, stroke volume (SV), cardiac output (CO), and stroke volume variation (SVV), based on the corrected arterial pulse contour formula, are calculated to achieve the purpose of monitoring hemodynamic changes with multiple data combined.
5. **Impedance cardiography**. Impedance cardiography (impedance cardiography, ICG) based on thoracic electrical bioimpedance (TEB) provides a safe, convenient,

Table 1 Common hemodynamic parameters

Parameter	Abbreviations	Unit	Calculation method	Normal range
Mean arterial pressure	MAP	mmHg	Direct measurement	82–102
Central venous pressure	CVP	cmH_2O	Direct measurement	5–12
Pulmonary artery wedge pressure	PAWP	mmHg	Direct measurement	6–12
Mean pulmonary artery pressure	MPAP	mmHg	Direct measurement	11–16
Heart rate	HR	bpm	Direct measurement	60–100
Hemoglobin concentration	Hb	g/dL	Direct measurement	12–16
Cardiac output	CO	L/min	Direct measurement	5–6
Stroke volume	SV	mL/beat	CO/HR	60–90
Cardiac index	CI	$L \cdot min^{-1} \cdot (m^2)^{-1}$	CO/BSA	2.8–3.6
Stroke volume index	SVI	$mL \cdot beat^{-1} \cdot (m^2)^{-1}$	SV/BSA	30–50
System vascular resistance index	SVRI	$dyne \cdot s/cm^5 \cdot m^2$	79.92 (MAP~CVP)/CI	1760–2600
Pulmonary vascular resistance index	PVRI	$dyne \cdot s/cm^5 \cdot m^2$	79.92 (MPAP~PAWP)/CI	45–225
Right cardiac work index	PVSWI	$g \cdot m^{-1} \cdot (m^2)^{-1}$	SVI (MPAP~CVP) · 0.0143	4–8
Left cardiac work index	LVSWI	$g \cdot m^{-1} \cdot (m^2)^{-1}$	SVI (MAP~PAWP) · 0.0143	44–68
Oxygen delivery index	DO_2I	$mL \cdot min^{-1} \cdot (m^2)^{-1}$	$CI \cdot CaO_2 \cdot 10$	520–720
Oxygen consumption index	VO_2 I	$mL \cdot min^{-1} \cdot (m^2)^{-1}$	CI (CaO_2~CvO_2) · 10	100–180
Oxygen extraction ratio	O_2ER	%	(CaO_2~CvO_2)/CaO_2	22–30

accurate, reliable, low-cost, real-time, and continuous hemodynamic parameter monitoring method to monitor hemodynamics and assess the myocardial function. After more than 30 years of improvement, at present, hemodynamic parameters are acquired by superimposed average signal processing technology and ZMARC algorithm, through which low accuracy and poor repeatability of the earlier Kubicck method have been solved. Various indicators can be measured by ICG, such as thoracic fluid content (TFC), ventricular acceleration index (ACI), pre-ejection index (PEP), left ventricular ejection time (LVET), heart rate (HR), blood pressure (BP), and then multiple hemodynamic parameter scan be calculated, such as cardiac output (CO), stroke volume (SV), cardiac index (CI), systemic vascular resistance (SVR), and left cardiac work (LCW).

6. **Doppler ultrasound**. Doppler ultrasound is commonly adopted to monitor the heart function changes in ICU, especially in patients with heart failure or cardiac dysfunction, where the quantitative analysis of ventricular systolic and diastolic functions has great clinical significance to monitoring conditions, guiding treatment, and understanding prognosis. As a noninvasive method to evaluate cardiac function, heart ultrasound often includes two-dimensional echocardiography, M-mode echocardiography, a volumetric method using geometric models, Simpson method, tissue Doppler imaging, Tei index, and three-dimensional echocardiography. Cardiac function measurement includes methods such as left and right ventricles, including the systolic function and diastolic function, of which the left ventricle function is of the greatest clinical importance.

2 Respiratory Support for Blast Injury

The main pathological features of blast lung injury include alveolar rupture and interalveolar hemorrhage mainly, and pulmonary edema and emphysema secondarily, sometimes accompanied by pulmonary rupture, resulting in acute respiratory distress syndrome (ARDS). ARDS is the acute hypoxic respiratory dysfunction or failure caused by diffuse pulmonary interstitial and alveolar edema due to the damage of lung capillary endothelial cells and alveolar epithelial cells in the process of non-cardiogenic diseases such as severe infection, shock, trauma, and burns. Its pathophysiological characteristics involve reduced lung volume, declined lung compliance, and severe ventilation/perfusion mismatch, presented as progressive hypoxemia and respiratory distress in clinical manifestations, and heterogeneous exudative lesions in lung imaging.

2.1 Oxygen Therapy

Patients with blast injuries should receive oxygen therapy in time to improve the gas exchange function, ensure oxygen delivery, and prevent cell hypoxia. The basic goal of the treatment is to improve hypoxemia and enhance PaO_2 to 60–80 mmHg, but the inhaled oxygen concentration should be lower than 60%. If a higher concentration of oxygen is required, the inhalation time should be less than 24 h as far as possible, and once the patient's oxygenation improves, the inhaled oxygen concentration should be adjusted as soon as possible. The oxygen therapy method should be adjusted

according to the improvement degree of hypoxemia and the treatment response of the patient. A nasal catheter can be chosen at first; an oxygen adjustable venturi mask or a non-rebreather mask with a reservoir bag can be used if a higher oxygen concentration is required. Patients with blast injuries are often accompanied by severe hypoxemia, where the conventional oxygen therapy hardly works for most patients once it is diagnosed, and mechanical ventilation is still the most important means of respiratory support.

2.2 Noninvasive Positive Pressure Ventilation

Noninvasive positive pressure ventilation (NPPV) can avoid complications caused by tracheal intubation and tracheotomy, which has been widely used in recent years. However, there are many controversies about the application of NPPV in acute hypoxic respiratory failure caused by blast injury.

When the patient is conscious with stable hemodynamic indicators and there are conditions for close monitoring and trachea intubation, NPPV can be tried. If the hypoxemia and the systemic condition are improved after NPPV are used for 1–2 h, NPPV can be continued. If hypoxemia cannot be improved or the systemic condition deteriorates, indicating that NPPV has failed, invasive ventilation should be promptly applied instead.

NPPV can prevent some ARDS patients accompanied with immunosuppression from invasive mechanical ventilation, thereby avoiding the occurrence of ventilator-associated pneumonia (VAP) and improving the prognosis. In immunosuppressed patients with ARDS, NPPV can be tried in the early stage.

2.3 Invasive Mechanical Ventilation

1. **Timing of mechanical ventilation**. In ARDS patients caused by blast injury with hypoxemia which cannot be improved even after a high concentration of oxygen is inhaled, the tracheal intubation should be performed timely for invasive mechanical ventilation. For patients whose breathing efforts are increased significantly, manifested as severe dyspnea, early mechanical ventilation with tracheal intubation can reduce the work of breathing and improve dyspnea. Although no RCT studies currently have evaluated the significance of early endotracheal intubation in the treatment of ARDS, it is generally believed that tracheal intubation and invasive mechanical ventilation can more effectively improve hypoxemia, reduce the work of breathing, relieve respiratory distress, and improve systemic hypoxia to prevent damage to organs except lungs.
2. **Lung-protective ventilation**. When ARDS occurs after the blast injury, a large number of alveoli collapse and the lung volume is significantly reduced, so that conventional or high-tidal-volume ventilation can easily lead to excessive alveolar expansion and overhigh airway plateau pressure, which aggravates the damage to lungs and other organs. A low tidal volume (VT) mechanical ventilation strategy is required according to the pathophysiological results of ARDS. At present, it is believed that the tidal volume should be set to about 6 mL/kg (ideal body weight), and it is recommended to maintain the airway plateau pressure <30 cmH_2O.

   Because the lung volume of an ARDS patient is significantly reduced, in order to limit the airway plateau pressure, sometimes the tidal volume has to be lowered, allowing $PaCO_2$ to be higher than the normal range, but maintaining pH > 7.20, which is called as permissive hypercapnia. Permissive hypercapnia is the result of lung-protective ventilation strategies but not the therapeutic goal of ARDS.
3. **Recruitment maneuver**. The main means to correct hypoxemia and ensure the effect of PEEP is an adequate recruitment maneuver to aerate collapsed alveoli caused by ARDS after blast injury. The small tidal volume ventilation forced to limit the airway plateau pressure is often not conducive to inflating the collapsed alveoli of ARDS, and the effect of PEEP to maintain the re-expansion depends on how much the alveoli inflate during inspiration. Moreover, the recruitment maneuver could reduce the shear damage caused by repeated opening and collapse of alveoli. The recruitment maneuver commonly used in present clinical practice include sustained inflation, increments of PEEP, and pressure-controlled ventilation (PCV). Among them, sustained inflation adopts constant pressure ventilation, and the recommended inspiratory pressure is 30–40 cmH_2O, and the recommended duration is 30–40 s.

   The effect of the recruitment maneuver strategy is affected by multiple factors, including the pressure and set time, which have a significant impact on the effect of the recruitment maneuver, different recruitment techniques, and the causes of ARDS. Generally, patients with extrapulmonary ARDS always respond better to recruitment maneuvers than those with pulmonary ARDS and the ARDS phase, where recruitment maneuvers function better at the early stage of ARDS.

   It is worth noting that the lung recruitment maneuvers may reduce the cardiac output so as to affect the circulation and may induce pneumothorax so that those patients should be closely monitored during the whole process.
4. **The selection of PEEP**. The extensive alveolar collapse of ARDS caused by blast injury can not only lead to stubborn hypoxemia but induce or aggravate ventilator-related

lung injury because of the shear force produced by a part of inflatable alveoli that periodically collapse and open. An appropriate PEEP should be applied after adequate re-aeration of collapsed alveoli to prevent end-expiratory alveolar collapse, improve hypoxemia, avoid shear forces, and prevent and treat ventilator-related lung injury. Therefore, practitioners should select the lowest PEEP that can prevent alveolar collapse.

How to determine the optimal PEEP for ARDS is still controversial. Generally, PEEP is set at 5–15 cmH_2O with the reasonable selection goal to minimize the adverse effects of PEEP on the body while avoiding alveolar collapse as much as possible. During actual practice, doctors could enhance PEEP gradually while keeping the inhalation pressure constant and observing the changes in tidal volume and circulation. Some scholars suggest that PEEP can be determined according to the pressure of the lower inflection point from the static lung pressure–volume (P–V) curve. Research by Amoto and Villar showed that when PEEP was set as the pressure of lower inflection point from the static P–V curve +2 cmH_2O, accompanied with a low tidal volume ventilation strategy, the mortality rate of ARDS patients, as a result, was significantly lower than that treated with conventional ventilation. If possible, PEEP should be determined according to the pressure of the lower inflection point from the static P–V curve +2 cmH_2O. Some scholars also suggest determining the optimal PEEP value by oxygenation, referring to after an adequate lung recruitment maneuver, directly setting PEEP to a higher level (such as 20 cmH_2O), and then reducing PEEP by 2 cmH_2O every 5–10 min until the oxygenation index decreases to <400 mmHg or by >5% (indicating the alveoli collapse again), next, performing the recruitment maneuver again, and then adjusting the PEEP value to the PEEP when the oxygenation index is reduced +2 cmH_2O for ventilation, which is the optimal PEEP.

5. **Spontaneous breathing**. During spontaneous breathing, the active contraction of the diaphragm can increase ventilation in the gravity-dependent area of the lungs in patients with ARDS and improve the ventilation and perfusion mismatch, as well as the poor oxygenation. Keeping spontaneous breathing as much as possible is a more important trend in invasive ventilation. A prospective controlled study showed that compared with controlled ventilation, the sedative dosage, the mechanical ventilation time, and the ICU stay in patients with spontaneous breathing remained were significantly reduced. Therefore, for ARDS patients with stable circulatory and respiratory functions and good human-machine coordination, spontaneous breathing should be retained during mechanical ventilation.
6. **Semi-reclining position**. VAP often further aggravates the lung damage for patients with ARDS caused by blast injury, so the prevention of VAP is of great clinical significance. VAP may occur when tracheal intubation or tracheotomy disables the closure function of the glottis so that the patient's gastrointestinal contents are easy to flow up and aspirate into the lower respiratory tract. The semi-reclining position with an angle below 30° is an independent risk factor for VAP. Therefore, unless there are contraindications for postural changes such as spinal cord injury, mechanically ventilated patients should maintain a semi-reclining position (30–45°), which can significantly reduce the development of VAP in patients with mechanical ventilation.
7. **Prone ventilation**. Prone ventilation significantly improves oxygenation by reducing the pressure gradient in the chest cavity, promoting secretion drainage and fluid movement in the lungs. If there are no obvious contraindications, prone ventilation can be considered. Gattinoni et al. used prone ventilation for 7 h a day for 7 consecutive days. The results showed that prone ventilation significantly improved the oxygenation of ARDS patients but didn't affect the mortality rate significantly. However, when making a stratified analysis of patients based on the PaO_2/FiO_2 ratio, researchers found that the mortality rate of patients with PaO_2/FiO_2 < 88 mmHg significantly declined after prone ventilation. In addition, a stratified analysis based on the simplified acute physiology score (SAPS II) showed that in patients with a SAPS II score higher than 49, the mortality rate was significantly reduced after prone ventilation, superior to the supine position. A recent RCT study on prone position ventilation for 20 h a day showed that prone ventilation tended to reduce the mortality of patients with severe hypoxemia. Therefore, for patients with severe ARDS, when conventional mechanical ventilation is ineffective, prone ventilation can be considered.

   Relative contraindications for prone ventilation include severe hypotension, ventricular arrhythmia, facial trauma, and untreated unstable fractures. There is no doubt that complications such as accidental shedding of tracheal intubation and central venous catheters may occur when the patient's position is changing, which needs proper prevention measures, but serious complications are not common.
8. **Sedation, analgesia, and muscle relaxation**. Sedative analgesics should be considered for patients with mechanical ventilation to relieve anxiety and pain and reduce excessive oxygen consumption. Appropriate sedation and analgesia can ensure patient safety and comfort and improve human-machine synchronization.

   Before the administration of sedatives during mechanical ventilation, practitioners should make a sedation plan first, including sedation goals and criteria for sedation effect evaluation. It is suggested to regard Ramsay scores

of 3–4 as the goal of sedation, wake up the patient every day, and if necessary, analgesics can be used in combination to make the patient comfortable. After the application of muscle relaxants in critically injured patients, the time of mechanical ventilation may be prolonged, leading to alveolar collapse and increasing the incidence of VAP which may extend the length of hospitalization. Muscle relaxants should be avoided as much as possible for ARDS patients with mechanical ventilation. During the application of muscle relaxants, the degree of muscle relaxation should be monitored to guide the dosage in order to prevent the development of diaphragmatic dysfunction and VAP.

2.4 Liquid Ventilation

Partial liquid ventilation is a technique based on conventional mechanical ventilation by injecting perfluorocarbons equivalent to the functional residual capacity into the lungs through tracheal intubation to reduce the alveolar surface tension and promote the re-aeration of collapsed alveoli in the gravity-dependent areas of lungs. Studies have shown that after 72 h of partial liquid ventilation, lung compliance of patients with ARDS can be improved, as well as the gas exchange without significant changes in circulation, but the prognosis of all the patients did not improve significantly, with a fatality rate still as high as about 50%. Partial liquid ventilation can improve the patient's gas exchange and enhance lung compliance, which can be an option for patients with severe ARDS when conventional mechanical ventilation is ineffective.

2.5 Extracorporeal Membrane Oxygenation (ECMO)

The strategy of extrapulmonary gas exchange following the establishment of extracorporeal circulation can reduce the burden on the lungs, which is beneficial to the recovery of lung function. Uncontrolled clinical studies have suggested that the survival rate of patients with severe ARDS was 46–66% after ECMO. However, RCT studies have shown that ECMO did not improve the prognosis of patients with ARDS. With the improvement of ECMO technology, further large-scale researches are needed to confirm the status of ECMO in the treatment of ARDS caused by blast injury.

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Blast Trauma Care

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1 On-Site Assessment and Management of Blast Injuries

Blast injuries are common, whether it is a war or a peaceful era, accidental or deliberate, which cause huge losses to people's lives and property, and pose great challenges to the work of medical staff. To save the wounded to the utmost extent, different departments should collaborate closely, medical resources should be allocated properly, and the basic principles of emergency treatment should be followed, given the complex environment at the detonation site, the large number of casualties, and the severe injuries. Nurses work together with other professionals on the front line of rescue in various rescue activities, who play an important role that cannot be ignored.

1.1 Assessment of the Detonation Site

1. **Priority at the detonation site**. The explosion could destroy the foundations of tall buildings and others, to make the main structure collapse and unstable. The chaotic and noisy scene, with widespread panic, could cause a large number of casualties. In this condition, it is necessary to immediately establish a command organization to ensure the safety of all people at the scene, as well as to guarantee smooth medical rescue. Followings are the work focus at the scene in order, including establishing a command organization, ensuring people's safety, ensuring smooth communication, assessment at scene, triage, treatment, and transportation. As one of the medical rescue team, nurses must, like all the other rescue personnel, wear personal protective equipment before entering the scene to work after ensuring their own safety. Before entering the scene, nurses must ensure that it is safe to enter the scene. They can enter the scene for rescue only if the scene is safe and the scene commander's permission is obtained. Follow the 1-2-3 safety guidelines: Am I safe? Is the site safe? Is the injured safe?

   Rescuers on the scene should first avoid secondary disasters, such as damage caused by scattered debris and collapse of buildings after the explosion. Besides, contamination should also be considered, including chemical, biological, and radiological pollution from the environment and the injured. Self-protection measures are needed, such as appropriate decontamination, if necessary. If it is a terrorist attack, be wary of a second explosion, and the radio should be off to avoid a radio-controlled secondary explosive device. Relevant data indicate that the second explosion was usually set to 30–100 min after the first one in terrorist attacks, mainly targeted at emergency rescuers, firemen, and police. Terrorists should be identified, who sometimes may monitor the scene somewhere, and detonate a second explosion at a distance, or use high-energy weapons to kill the rescuers. Rescuers should also be alert that the injured may have weapons or explosive devices.

   In addition to preparing sufficient supplies and mobilizing spare ambulances, medical rescuers must also be fully prepared for various situations at the scene, such as deformed remains, mangled extremities, massive bleeding, and severely damaged wounded, etc.
2. **Casualty assessment at the detonation site**. During the on-site rescue, medical personnel can evaluate the intensity of the explosion and the overall casualties based on some evidence at the scene in order to guide the follow-up preparation of related supplies, rescue teams, and in-hospital medical management. Explosive craters, damage to nearby personnel, and collapsed buildings are important evidence of blast wave intensity. Explosive craters could occur with the explosive overpressure exceeding 300 psi (2 MPa), which could cause lethal tearing of the

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human body. Some buildings may collapse when the external overpressure reaches 10 psi (68 kPa). The intensity of blast waves that cause tympanic membrane rupture is about the same as that shattering the windshield of a car, shaking a telephone pole, or shattering a brick wall. First-aid personnel can estimate the general situation of the incident through the car collision and the object damages after the explosion, as well as the characteristics and the total number of casualties when combined with the location of the detonation, the range of the affected area, and the time of detonation.

Unlike general emergencies, most of the explosions caused injuries to a large number of casualties. The management of a huge number of the wounded often exceeds the rescue capacity of a hospital or a region. Fixed rules exist in batches of the wounded after the explosion, that the closer to the explosion, the higher the mortality rate, and vice versa, the further from the detonation center, the weaker the impact. If the explosion occurs in a relatively closed space (such as vehicles, mines, buildings, subway stations, etc.), the casualties will be more serious; if a building collapses, the mortality rate will increase significantly. Analysis of a number of explosions shows that the total mortality rate at the scene was 25%, most of which occurred at the time of the explosion or within a short period after the explosion, where most of the survivors required medical intervention, 30% required hospitalization, and many without any physical injury suffered from severe mental disorders and disorientation. The overall assessment of the wounded includes looking for wounds or burns caused by debris and shrapnel. Explosives may produce high-humidity air currents that can cause severe burns when in contact with the wounded, resulting in a large number of burn patients. Severe burns have become the focus of attention of clinicians. Although burns may be fatal, deaths caused by potential trauma cannot be ignored.

1.2 Emergency Management at the Detonation Site

1. **Injury assessment**. Explosion injuries are divided into four types. The primary blast injury is the direct effect of the blast wave passing through the body. Due to the violent vibrations at the air–liquid interface, the air-filled organs of the human body are particularly vulnerable to damage. When the explosion occurs in the air, the lungs are easily damaged; when it occurs in the water, the risk of gastrointestinal injury is high. The secondary blast injury is caused by fragments from the explosion hitting the human body. When casualties are lifted or thrown by the explosion blast waves against relatively stationary objects, it could lead to the tertiary blast injury. Other indirect effects of explosions are categorized into the quaternary blast injury, including inhalation of harmful substances, burns, and crush injuries, etc. Rescuers may face multiple traumas after the explosion, where the most common are penetrating injury, blunt injury, thermal injury, and blast injury. Blast injury is mostly manifested as multiple injuries and combined injury, which is complicated with damages to both internal and external organs, but mild external wounds and severe internal injuries, which are easy to miss the diagnosis. Meanwhile, negative mentality, such as panic, anxiety, irritability, is common. The severity of the injury is affected by the strength of the explosion, the surrounding environment, and the distance between the injured and the detonation center. The severity of the survivor's injury is inversely proportional to his/her distance from the detonation center. However, debris or other objects can cause fatal secondary wounds.
2. **Triage**. In disaster rescue, triage of the injured is a key factor for successful on-site treatment in order to provide the greatest assistance to the most injured when resources are limited. Although there are a variety of methods for triage, there is no uniform standard for the classification of injuries for pre-hospital treatment of batches of wounded patients. After understanding the general situation of the detonation site, caretakers should classify the injured according to the priority order of treatment. Triage sieve can be used first to quickly identify the people most in need of medical treatment, while triage sort needs more detailed examinations, but generally only with sufficient medical resources and anatomical and physiological parameters. During the triage sort, over-prioritizing usually exists, which disperses the limited medical resources and increases the mortality of the injured who need the treatment most, although it can enable the injured to receive more treatment at once. Due to the limited ability of each medical institution to meet the needs of the injured, the negative impact of over-prioritizing intensifies with the increasing total number of the injured.

   Among the triage strategies, START (simple triage and rapid treatment) has been widely used which was first adopted as a triage method in medical rescue in the USA, with a purpose of simple triage and rapid treatment, mainly for the triage of mass casualty incidents with insufficient medical resources. Through STAT, rescuers should quickly judge the severity of the injury by assessing mobility, breathing, circulation, and consciousness and then make decisions about the treatment priority of the injured. In this system, the injured are quickly divided into four categories through a simple clinical assessment, which are green (mildly injured/walking wounded), yellow (moderately injured/delayed treatment), red (severely

injured, immediate treatment), and black (expectant treatment). Its application in the triage of blast injury is given in Table 1. What's simpler is to divide the injured into emergency and non-emergency treatment. Immediate treatment is required for those in emergency treatment with single or multiple site injury, limited to simple management, such as opening airways, controlling bleeding by compression, etc. For those in need of delayed treatment with stable hemodynamics, their survival will not be affected if they are not treated or simply treated for a certain period of time. Expectant treatment is only be used for mass casualty incidents. The probability of survival and the available resources are the foundation to judge whether the injured should receive expectant treatment. The injured who are sorted as expectant treatment are injured seriously with a survival rate, so that rescue and treatment will consume a lot of time and resources, which will reduce the chances of survival of those with a high probability of survival, such as those with systemic smashed wound, and traumatic limb dismemberment. However, the former are not left untreated but given comfort and analgesic treatment. When signs of improvement occur, they should be actively treated. In the management of batch wounded, CPR is not recommended due to limited on-site resources.

Table 1 Triage strategy for explosion blast injuries

Type (by color)	Requirements	Examples
Minor injuries (green)	Move quickly from the triage area to other staff	Psychological trauma, simple auricular injury (no lung injury), small area injury
Delayed (yellow)	Simple management is needed within hours	Burns to the epidermis or part of dermis. Extremity injury without large blood vessel injury combined. Stable condition after chest drainage
Immediate (red)	For those with severe trauma but a high probability of survival, immediate treatment, and rapid intervention are needed in a short period of time to relieve respiratory and circulatory dysfunction caused by airway obstruction and tension pneumothorax	Airway obstruction, hypotension, active extremity bleeding, penetrating trunk trauma, vascular trauma, extensive damage to the limbs, deep or full-layer skin burns
Expectant (black)	Treatment may waste limited resources where the probability of survival is low even with sufficient resources For complicated and time-consuming cases, proper measures should be taken to make the injured feel comfortable	Limb dismemberment (damaged by blast waves instead of fragments); open skull fractures with spillage of brain contents; dilated pupils with no vital signs (CPR is not recommended at the rescue site)

As a dynamic process, triage needs to be repeated throughout the entire treatment and further transportation. When the condition of the injured deteriorates or improves, another evaluation is needed to ensure that all the injured are treated correctly. For the injured exposed to chemical, biological, or radioactive materials, the triage sort is based on the medical or surgical treatment they need, not on whether they need decontamination. After the explosion, many walking wounded or uninjured people tend to go to nearby medical institutions for treatment by themselves, while the severely injured usually stay in place for examination and triage. Current studies suggest that more accurate evidence for predicting severe internal blast injuries includes the following: four or more body surface injuries, burns over 10% of body surface area, cranial or facial fractures, and penetrating trauma of brain or trunk. In the suicide bombing in Istanbul, Turkey, 52% of the 184 wounded who arrived at the trauma center within 1 h were not medically transferred but went by themselves. In many cases, walkable wounded walked to medical institutions on their own, causing a lot of congestion, thus affecting the management of really serious wounded.

3. **Management strategy**. In the process of blast injury treatment, it is very important to clarify the specificity of blast injury treatment and confirm the specific problems related to the explosion. Emergency treatment includes resuscitation, stabilization of the severely wounded, identification, and prevention of blast injury complications. In the early management of blast injury, from group triage to individual treatment, the initial assessment and treatment are the same as trauma, following the principles of ABCDE, that is, A (airway), stabilizing the cervical spine, and opening the airway, B (breathing) maintaining effective ventilation and breathing, C (circulation) controlling bleeding and stabilizing circulation, D (disability), assessing neurological function, E (exposure/environment), exposing and keeping warm. ABC should be first focused on. Although ABC is commonly used for advanced life support for traumas, previous experience and evidence of battlefield rescue show that 10% of deaths on the battlefield are caused by extremity bleeding. According to data analysis of the Vietnam War, among the preventable deaths on the battlefield, greater than 50% was caused by extremity bleeding, while limb tourniquets can prevent 7% of battlefield deaths. Therefore, ABC was corrected to <C>ABC, where <C> means catastrophic or fatal bleeding. Lives can be saved by quickly identifying fatal external bleeding, controlling the bleeding, and applying tourniquets early, especially

for those with severe multiple injuries. For high-energy trauma, there are six potentially fatal situations be clarified in the initial assessment, namely airway obstruction, pressure pneumothorax, open pneumothorax, massive bleeding, floating chest (flail chest), and cardiac tamponade. During the reassessment, rescuers should focus on the head, chest, abdomen, and extremities damaged by blast waves, about whether there are thermal burns, contusions, or penetrating fragments, or shrapnel injuries. Through the assessment, medical staff should discover and deal with the injury that endangers the injured person's life in time, understand his/her problem, how to be treated at present, the focus of further treatment, and the hospital most suitable to accept him/her.

Those with blast injury may suffer from inhalation burns, which will cause pharyngeal edema within a few minutes, so tracheal intubation should be used as soon as possible to open the airway; and oxygen should be given, whenever possible. If the injured suffer from breathing effort, abnormal auscultation, and definite evidence of chest trauma, high-flow oxygen should be given, and the corresponding injury should be assessed and treated immediately. During chest assessment, the focus should be laid on pneumothorax, hemothorax, or pressure pneumothorax (caused by rapid changes in air pressure of blast waves). For those with chest trauma suffering from respiratory distress, which is suspected of tension pneumothorax, thoracic puncture should be given for decompression; and if symptoms do not improve, closed thoracic drainage should be given instead. As for the abdomen, it is important to note that hollow organs (stomach, spleen) may be damaged by rapidly changing air pressure. For spleen rupture, tenderness in the left abdomen and other typical signs of bleeding can be found. If an injury in solid organs is suspected, rescuers should also be alert for hollow organ damage.

The tympanic membrane is very sensitive to pressure. Those severely affected by blast waves generally have suffered from tympanic membrane injury or rupture, which can be diagnosed by bleeding in the external auditory canal or hearing loss. If the injured has bleeding in one or two auditory canals or sudden hearing loss, he/she may have been near the detonation center and should be considered as severely injured (yellow tag) until the next deep evaluation. When the injured has bleeding in the external auditory canal or bloody foamy secretions in the mouth and nose, or he/she is in shock without obvious trauma noted, or he/she suffered from dyspnea, irritability, hemoptysis, chest pain, or abdominal pain, rescuers should contact the hospital and transport the injured as soon as possible. Blast lung injury is the most common fatal injury for early survivors. Medical treatment outside the hospital can follow the following simple principles. (1) Early treatment is the same as a conventional trauma treatment. (2) Quick transportation can improve the survival. (3) Follow the principles of disaster management, namely no treatment during the triage. (4) Because of the number and the survival rate of the injured, on-site resuscitation should be avoided as little as possible. For those with abdominal blast injury combined with hemorrhage, aggressive resuscitation is needed, and low-pressure resuscitation is feasible (to keep systolic pressure 80–90 mmHg) in order to avoid excessive resuscitation, which may aggravate lung damage. If continuous intracranial hemorrhage occurs, the massive intravenous infusion should be avoided, while if signs and symptoms of hypovolemia appear, such as deterioration of consciousness, an appropriate fluid supplement should be given. A tourniquet or similar is used to bandage a bleeding limb, and for those with unstable hemodynamic parameters without external bleeding, the pelvis should be wrapped and fixed. In the beginning, attention should be paid to reducing heat loss and preventing hypothermia. As for screening radioactive contamination, if radioactive materials are detected, decontamination equipment is needed, and the hospital that received the injured should be notified.

It is not most suitable to assess or treat trauma before arriving at the hospital, and the injured should be safely transferred to an ambulance as soon as possible. No matter at the rescue scene or during the rapid transportation in the ambulance, the principle of "no further harm" should be observed, that is the absence of "faults of inaction" and "faults of acting." For example, the airway should be open before transporting the injured to ensure that the airway of the injured is unobstructed, the circulation should be stable, and the fracture should be fixed. Intravenous infusion is a routine process in the hospital, but not as a routine requirement before arriving at the hospital because this will prolong the transportation time, and intravenous channels should be established in the ambulance as needed. For those who need fluid resuscitation for a good prognosis, a large venous channel should be established, and low-pressure resuscitation is required. Faults of inaction refer to unnecessary venous passage established before the hospital, unnecessary secondary physical examinations, unnecessary diagnostic tests performed before the hospital, and some measures that are not important to survival but delay the rescue of the critically injured, which will aggravate the injury.

2 In-Hospital Management and Care for Common Blast Injury

Terrorist attacks have increased in modern society, and accidents have occurred from time to time. Blast injuries that were previously only seen in wars can now be seen at ordinary times. Pre-hospital rescuers and in-hospital medical staff should be familiar with relevant knowledge, signs and

symptoms, diagnosis, and treatment of blast injury, which will help the triage and treatment of the blast injured. General treatment is according to the standard procedure of trauma. After the injured is stabilized, corresponding organs should be carefully evaluated, focusing on the fatal injuries of the chest and abdomen. Among survivors, penetrating and blunt wounds of the body surface are the most common. The mortality rate is high in blast lung injury and abdominal blast injury, where among early survivors, blast lung injury is the most common fatal injury.

2.1 Key Points of In-Hospital Emergency Management

2.1.1 Management Plan

1. **Preparation of emergency medicines, equipment, and personnel**. Prepare materials, equipment, medicines, and manpower in a foreseeable manner. According to the needs of treatment, the Care staff prepares corresponding equipment and items according to the key points of emergency treatment to ensure that they are kept in good condition and ready for use.
   (a) **Respiration**. Prepare tracheotomy, tracheal intubation, closed chest drainage, oxygen inhalation device, simple respirator, ventilator, etc.;
   (b) **Circulation**. Hemostatic materials, medicines, and equipment, simple fixing devices, infusion devices, blood products, etc.;
   (c) **Body temperature and pain**. Insulation and rewarming items and equipment, painkillers, etc.;
   (d) **Manpower**. Initiate the emergency rescue manpower contingency plan, recall the rest of personnel, mobilize the emergency and critical care reserve personnel of the hospital, organize teams, and allocate the work.
2. **Initiation of the emergency treatment plan**. If a batch of wounded is accepted, the emergency treatment plan for the batch of wounded should be activated immediately. Form teams with the predetermined system and personnel to make sure each member carries out the tasks assigned in advance simultaneously and orderly so as to provide fast and effective treatment. It is very important for team members to clarify the tasks of themselves and others. Each person's specific tasks can be displayed by post boxes, reminder notes, etc., to avoid interference from other tasks and confusion of responsibilities. Shunt patients in the hospital, adjust the treatment site and set up various functional areas, such as emergency command, personnel reception, press release, decontamination, triage, and graded treatment areas.

   Before the injured arrive, all members of the rescue team must be in place and wear protective gloves, plastic aprons, goggles, lead suits, etc. If the injured are contaminated by radioactive materials, medical staff should take standard protective measures and wear personal protective equipment, including long white coats, surgical masks, waterproof shoe covers, eye masks, and double-layer gloves. Severely injured patients should be actively treated before they are formally removed from the contamination. Rescuers should ensure a smooth flow of the wounded, contact the first-aid personnel in the hospital to understand the condition, type, and quantity of the injured. The injured transferred out of the emergency room should be treated as far as possible to meet the needs of emergency rescue in priority. Divide the site to treat the wounded of different severity, and each area should be equipped with correspondingly competent medical staff. Slightly injured patients should be discharged as soon as possible after simple treatment to avoid occupying the hospital's medical resources. The severely injured who need emergency treatment should be transferred out of the emergency room after emergency treatment to the ICU, an operating room, or a specialized ward for further management. For the injured who need emergency surgery, a green channel should be opened, and the nurse should prepare relevant documents, coordinate relevant personnel, and send them to the operating room. The injured who are severely injured but not fatal should be treated aggressively and, at the same time, be watched closely in order to adjust the treatment step at the proper time.

2.1.2 Emergency Management Strategy

1. **Principles of early management**. Follow the hospital and local disaster emergency plans. The severity may be reversed, where the severely injured may arrive at the hospital later than the slightly injured. Estimate the total number of the first batch of wounded roughly as twice the number of the wounded arriving in the first hour. If buildings collapse, the injury will be more severe, and the injured will arrive later. In batch wounded treatment, the general principle is that the wounded should receive the "minimum acceptable treatment," that is, relatively brief damage control treatment and short-term stable injury treatment, rather than a few hours of definitive treatment. "One-way treatment principle" should be followed, that the injured should be transferred from the emergency room to the operating room, ICU and then discharged, not returning to the emergency room halfway.
2. **Injury assessment and management**. Because of the complicated mechanism of the blast injury, re-evaluation in the emergency room is particularly important, focusing on ABC, which is opening the airway while fixing the cervical spine, maintaining ventilation, controlling bleeding, and stabilizing circulation. Treat life-threatening injuries urgently. Severely injured patients with

unstable hemodynamics should receive concentrated red blood cells, frozen plasma (1:1), and platelets; if possible, fresh whole blood should be given as soon as possible. For the injured with severe trauma, cryoprecipitate and recombinant factor VIIa can be used. If the injured suffers from a traumatic brain injury, it is essential to prevent hypoxia and hypotension. Because lung contusion may progress within a few hours, close observation and repeated radiographs are required, as well as definitive airway establishment and respiratory support. If abdominal pain persists with vomiting, the injured should be sent to the emergency room for observation. Common treatment operations include chest drainage, fracture fixation, gastrointestinal decompression, bladder decompression, wound coverage, application of broad-spectrum antibiotics, injection of tetanus antitoxin, and pain relief. Routine radiographic examinations, MRI, CT, angiography, laboratory examinations, radiological diagnosis, etc., are not recommended because they will interfere with the shunt of the injured. For the injured with traumatic brain injury, if the GCS is low or worsening, CT should be performed to rule out the large-scale injury and decide whether or not to take a surgical intervention. For the injured around the detonation center, FAST is recommended, especially for those with signs of primary blast injury, where FAST can be used to determine whether there is abdominal effusion and important abdominal injury.

2.1.3 Routine Care Measures

1. Assess the condition of the injured, open the airway, keep the airway unobstructed, assist the physician in performing tracheal intubation and tracheotomy when necessary, and routinely give oxygen inhalation.
2. Immediately establish dual venous channels to expand blood volume to prevent the development and deterioration of shock. Peripheral veins of the upper limbs are recommended first. And large blood vessels and large indwelling needles (#14 or #16) should be selected. Generally, for the fluid therapy, isotonic saline or balance solution should be given first, followed by concentrated red blood cells or whole blood, and then crystallized solution, albumin or plasma if needed. For the injured with uncontrolled bleeding, restrictive fluid resuscitation is advocated. In other words, the blood pressure is controlled below the normal level, but the tissue perfusion can be satisfied, and fluid supplementation can be strengthened after the bleeding is controlled.
3. Indwell catheter. Urine volume is a sensitive indicator of the blood volume state and the renal perfusion of the injured. A urinary catheter should be indwelled as soon as possible when conditions permit. When a urethral injury is suspected, it is contraindicated to insert the catheter through the urethra, where an emergency bladder fistula is recommended.
4. Closely observe the changes in the vital signs of the injured and report and treat any abnormalities immediately.
5. Preoperative preparation. Blood samples should be taken for the corresponding examination of the injured who need surgical treatment, and the skin preparation of the operation area should be performed.
6. Safe transfer. Before transfer, the condition of the injured should be fully assessed to make sure the vital signs are stable. Before transfer, caretakers should communicate with relevant departments and bring necessary first aid supplies and equipment.

2.2 Care of Common Blast Injuries

The interface between gas and tissue is most susceptible to direct blast injuries. Gas-rich organs, such as the lungs, gastrointestinal tract, and auditory system, are the most vulnerable. When the overpressure exceeds 35 kPa, the auditory system will be damaged. When the pressure is 75–100 kPa, lung and gastrointestinal tracts are more likely damaged. Other systems that can be damaged by overpressure include the central nervous system, musculoskeletal system, and, relatively rarely, visual and cardiovascular systems. The main work of caretakers is to evaluate and observe the condition of the injured, understand the treatment principles of blast injuries in each system, prepare for and assist in treatment in advance, actively prevent various complications, and provide care, help, encouragement, and humanistic care to the injured.

2.2.1 Care of Blast Lung Injury

1. **Injury assessment**. Except for the auditory organs, the lungs are most susceptible to blast injury, which is caused by the blast waves affecting structures such as the chest cavity and airways. Primary blast lung injury occurred in 47% of the deaths from explosions; 44% of hospitalized injured patients and 71% of hospitalized severely injured patients suffered lung injuries. The incidence of lung injury could increase by three times under ultra-high pressure or a rapidly increasing pressure (such as an explosion in a closed space). It was previously believed that patients with tympanic membrane perforation should be hospitalized for observation because it was believed that blast lung injury might occur 24–48 h after injury. However, it is now believed that manifestations of blast lung injury do not appear after a period of time, but all appear significantly shortly after the explosion; most of the acute lung injuries that occur 48 h after the explosion are related to Systemic Inflammatory Response Syndrome

or sepsis, rather than blast lung injury. Common blast lung injuries include lung contusion, pneumothorax, pneumomediastinum, interstitial or subcutaneous emphysema. Apnea, bradycardia, and hypotension are the typical clinical trial of lung impact injury. Patients with the following symptoms should be suspected of blast lung injury, that experienced dyspnea, cyanosis, cough, hemoptysis, chest pain, etc., after the explosion. Generally, an explosion in a confined space could cause bilateral lung injury, while in an open space, the lung near the explosion is damaged more severely. It is recommended that all personnel who have experienced an explosion undergo a chest radiograph, where blast lung injury can be confirmed when the butterfly sign is noted.

2. **Care strategy**. Blast lung injury ranging from scattered ecchymosis in the lungs to obvious active bleeding usually manifests as hypovolemic shock, respiratory distress, air embolism, etc., with a mortality rate of 11%. The surviving wounded must be diagnosed immediately and resuscitated quickly. If the injured suffers from unstable conditions or requires emergency tracheal intubation immediately, positive pressure ventilation can be used to correct respiratory distress and improve hypoxia, but it can also cause air pressure injury or arterial air embolism. For those with severe blast lung injury, it is necessary to increase the positive end-expiratory pressure (PEEP) on the basis of positive pressure ventilation, but a high PEEP can aggravate lung parenchymal damage, causing pneumothorax and other complications. Therefore, for blast lung injury, it is necessary to adopt the lung-protective strategy to reduce peak airway pressure as much as possible during mechanical ventilation. If sufficient oxygenation of the injured cannot be maintained by conventional mechanical ventilation, advanced respiratory support techniques are required, such as different types of pressure-controlled ventilation to overcome hypoxia without significantly increasing PEEP. The prone position helps to improve oxygenation, but it is difficult to implement in an emergency. It is now generally believed that bilateral chest drainage is not recommended in the absence of pneumothorax, while for those suspected of blast lung injury who need general anesthesia or air transport, it is suggested to undergo preventive chest drainage.

2.2.2 Care of Gastrointestinal Blast Injury

1. **Injury assessment**. The incidence of gastrointestinal impact injury is lower than that of the tympanic membrane or lung blast injury, and the incidence rate among survivors is 0.3–0.6%. Similar to other blast injuries, gastrointestinal injury is more likely to occur in a confined space and more serious underwater because blast waves are more likely to spread in water. Because of the interaction of the blast waves with the gas–liquid interface of the intestine, intestinal contusion, rupture, and perforation will appear. Shear forces at the junction between the free part and the fixed point of the intestine, such as the liver and splenic flexure of the intestine, can cause intestinal volvulus. If the moving debris penetrates the abdomen, fatal abdominal bleeding may occur. Abdominal blast injury should be considered if people who experienced the explosion suffered the following symptoms, including abdominal pain, diarrhea, nausea, vomiting, hematemesis, bloody stool, the disappearance of bowel sounds, passive position, rebound tenderness, tenesmus, testicular pain, unexplained hypovolemia, or other signs of acute abdomen. However, in many cases, the clinical signs of abdominal blast injury are not obvious before it progresses to acute abdomen and sepsis.
2. **Care strategy**. The emergency treatment of gastrointestinal blast injury is similar to that of common abdominal traumas. First, the wounded should be resuscitated with an advanced trauma life support plan and assessed whether surgical intervention is needed. If there is a major hemorrhage in the abdominal cavity, it must be confirmed and treated immediately, and usually, a laparotomy is required. Intestinal contusion, which may be secondary to intestinal perforation, requires active management. Studies have shown that many secondary intestinal perforations occurred 3–5 days after injury and occasionally after 2 weeks; if small intestinal contusion smaller than 15 mm and colon contusion smaller than 20 mm are found during laparotomy can be treated with traditional methods. Esophageal perforation may also occur, often manifesting as chest pain, dyspnea, and subcutaneous emphysema, which can be diagnosed by esophageal X-rays. The most common treatment is one-stage repair, which, however, in many cases, it is not needed. Patients with abdominal injury should fast before diagnosis. Those with paralytic intestinal obstruction should undergo gastrointestinal decompression before an effective bowel movement is restored to reduce the risk of delayed bowel perforation and vomiting and to reduce the oppression on the diaphragm, which is conducive to lung ventilation. Patients with a non-perforated abdominal injury should be observed for another 1 week (at least) after symptoms are relieved by conservative treatment to determine whether there are signs of delayed perforation.

2.2.3 Care of Tympanic Membrane Blast Injury

1. **Injury assessment**. The hearing apparatus is so fragile that the incidence of auricular blast injury is high. Life-threatening injuries must be treated first during the rescue, instead of nonfatal hearing damage, which is often overlooked. After the life-threatening injury has been managed, the hearing damage should be assessed. Signs

of ear damage could appear at the initial evaluation, often including hearing loss, tinnitus, earache, dizziness, bleeding through the external auditory canal, tympanic membrane rupture, and otorrhea. Basic hearing tests and otoscopy should be routinely performed. The most common type of middle ear injury is tympanic membrane perforation. Hand-held otoscope examination, which is simple and easy to perform, can be used to observe the injury of the external auditory canal and tympanic membrane. Factors that affect tympanic membrane damage include blast wave pressure, the position of the head and ears relative to the detonation center during the explosion, whether there is earwax, whether the injured has worn protective equipment, ear infection history, past illness history, and injury history, etc. For children whose tympanic membrane owns better compliance, the probability of tympanic membrane perforation of children is lower than that of adults at the same distance from the detonation center. In addition, the cerumen in the external auditory canal can protect the tympanic membrane like earplugs.

2. **Care strategy**. Patients with tympanic membrane perforation are mainly treated with conservative therapy, disinfection and rinse, and removal of a foreign object to prevent further damage. If the tympanic membrane is invisible due to earwax or blood clots, ask an otolaryngologist to carefully suck and clean the perforation. Keep the ears clean and dry when no professional is present. Non-ototoxic antibiotic drops should be used for tympanic membrane perforation and ear canal tears, which can help flush and clean ear canal debris. Generally, quinolone pharmaceutical preparations such as ciprofloxacin and ofloxacin should be used instead of ototoxic local ear drops. If the tympanic membrane ruptures more than 1/3, surgical repair is recommended. Irregular perforation combined with everted tympanic membrane wings, recombination repair, can promote healing. The prognosis is good for most patients with tympanic membrane perforation, which can heal without treatment, but 30% of the injured will suffer from permanent hearing loss. Tympanic membrane perforation increases the incidence of cholesteatoma, especially for large perforation, which has no tendency to heal itself, so that follow-up screening is required. The management of outer ear injury is the same as other soft tissue injuries, that is, removal of foreign bodies, flushing and cleaning wounds, drainage of ear hematomas, and soft tissue repair for broken auricle and exposed ear cartilage. Due to tinnitus, hearing loss, temporary or permanent deafness, and other reasons, medical staff should pay attention to communication skills and methods when communicating with the injured and use writing boards, hearing aids, and other auxiliary appliances when necessary.
3. **Screen primary blast injury**. Those with tympanic membrane perforation should be highly suspected of primary blast injury to the lungs and abdomen, which are generally delayed. The tympanic membrane perforation was previously believed as a clear sign of an explosion, while other blast injuries were common but not easy to identify. Therefore, the empirical approach is to closely observe the blast injured patients with tympanic membrane perforation overnight to make sure whether there is delayed dyspnea caused by occult lung injury. Although its necessity is yet to be proven, this principle is followed in many practices. In 2009, Harrison et al. conducted a landmark study. Of the 167 U.S. blast wounded in Iraqi field hospitals, 16% suffered from tympanic membrane perforation, and 7% suffered from blast injuries such as pneumothorax, pneumomediastinum, lung contusion, nasal sinus injury, or intestinal perforation, however, only half of whom had tympanic membrane perforation at the same time. This result was repeatedly confirmed by other studies, indicating that tympanic membrane perforation is just an insensitive sign of blast injury. Therefore, it is now recommended that if there is simple tympanic membrane perforation without any evidence of other trauma, the injured should be closely observed for 6–8 h, including blood oxygen saturation monitoring and chest X-ray examinations. The injured without symptoms can be discharged after preventive measures for lung and abdomen injuries. Supportive treatment is needed if there are positive findings in radiological examinations or clinical symptoms. Primary blast injury can be excluded if the injured has no damaged tympanic membrane without respiratory and abdominal symptoms and complaints.

2.2.4 Care of Other Blast Injuries

1. **Blast-induced traumatic brain injury**. Blast-induced traumatic brain injury is more common than previously thought. Through a retrospective cohort study in 2011, Dougherty et al. found that 37% of the 2254 U.S. bombing casualties in Iraq had varying degrees of nerve damage. It is also reported that among the 3000 casualties caused by the recent terrorist bombing, brain injury is the main cause of early and late death. Blast-induced traumatic brain injury varies from mild to severe. The main symptoms include headache, tinnitus, noise intolerance, degenerative or anterograde amnesia, and post-traumatic stress disorder (PTSD), which were previously known as “bomb shock,” “bomb concussion,” and “combat fatigue”, etc., with clinical manifestations ranging from mild dysfunction to complete loss of response, but normal GCS scores for most of the injured. Care staff should closely monitor the brain signs and intracranial pressure of the injured, properly manage temperature, and limit fluid

infusion, which can reduce intracranial hypertension and maintain cerebral oxygenation.

2. **Cardiac blast injury**. Cardiovascular injury often occurs in a small part of the injured close to the detonation center. Those injured may fall into severe shock even in the absence of bleeding or other common causes of hypotension, which does not respond to resuscitation. Shock is considered to be a direct effect of shock waves, leading to decreased cardiac index and bleeding, systemic vasoconstriction, and tachycardia without compensation. In addition, the bleeding caused by the explosion can also cause hypotension in the injured with cardiovascular injury. Air embolism is more common in patients with cardiovascular injury, manifested as stroke, myocardial infarction, acute abdomen, blindness, deafness, spinal cord injury, and claudication. The greatest risk of air embolism occurs within 24 h after the blast injury and increases during positive pressure ventilation. Therefore, the peak airway pressure should be decreased to reduce the risk of embolism for the injured who requires positive pressure ventilation. If air embolism is suspected, empirical treatment, mainly supportive treatment, should be initiated. Nursing staff can place the injured lying on the left with feet higher than the head to limit the gas to the apex of the left ventricle to prevent subsequent air embolism. Caretakers can place the injured lying on the left with feet higher than the head to limit the gas to the apex of the left ventricle to prevent subsequent air embolism. For the injured with air embolism, their oxygenation generally reaches the lowest point within the first 24 h, so that they need to inhale 100% oxygen. Hyperbaric oxygen therapy is useful in some cases. Interpleural syndrome, another rare cardiovascular complication, is characterized by a sharp drop in blood pressure during positive pressure ventilation, due to limited cardiac function caused by cardiac tamponade induced by mediastinal edema or hematoma, where thoracotomy is recommended.
3. **Musculoskeletal blast injury**. Musculoskeletal blast injury is very common in blast injuries. Secondary injuries are more common than primary ones. Above 80% of the operations of the survivors were related to the musculoskeletal system. Great attention should be paid to crush injuries during the rescue of the blast injured patients because, with non-timely treatment, it could develop into rhabdomyolysis, acute renal failure, acidosis, and metabolic disorders, and even death. Rhabdomyolysis may also occur without obvious crush injury, such as when the injured has been forced to stay in a small enclosed space for a long time after a building collapses. Treatment includes active hydration, urine alkalization, mannitol for diuresis, and hemodialysis when renal failure appears. Among the blast injured patients, any limbs dismemberment is a sign of multi-system injuries. Traumatic limb dismemberment and avulsion injuries occur in 1–3% of the blast wounded, which have a poor prognosis, especially those with the proximal dissection of the wrist and ankle joints, because serious internal organ injury may be complicated. Management of skeletal injury caused by explosions includes X-ray to assess fractures and foreign bodies, injection of tetanus antitoxin, and application of broad-spectrum antibiotics if the fracture is open. Many fracture injuries require early fixation, and the gap syndrome should be considered during external fixation of extremity injury. The treatment of wounds caused by small residual shrapnel has been controversial. Some studies have pointed out that such wounds should be treated conservatively in the following cases, that only involve soft tissues, but not the peritoneum, pleura, or major blood vessels, less than 2 cm in diameter, without significant infection, and not caused by a coal mine explosion.
4. **Ocular blast injury**. Ocular blast injuries are usually divided into two categories. One is caused by shock wave shear force, which is manifested as hemorrhage, retinal detachment, or eyeball rupture, and the second is more common to be caused by explosive projectiles. The incidence of ocular injury after the explosion is very high. Although the injury is less than 0.1% of the body surface area, 10% of the survivors suffered from significant eye injuries, mainly caused by projectiles, commonly manifested as intraocular foreign body, corneal abrasions, lacerations around the eyelids or orbits, retinal detachment, orbital bone fractures, and eyeball ruptures. Treatment principles: Routine eye examinations are necessary, and further in-depth examinations are recommended for vision loss. Protect the exposed eyeballs to prevent the eyeballs and wounds from drying out. Bilateral eye bandaging is needed for all eyeball perforation and rupture. Wear metal eye masks to prevent accidental injury. Mannitol can be given intravenously to reduce high intraocular pressure, and debridement and suture or eyeball removal should be performed if necessary.

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Psychological Intervention and Therapy After Blast Injury

Zhengzhi Feng

Like earthquake, mine accident, or major car accident, an explosion accident is an acute, severe, and major traumatic stress event. The harms of an explosion would bring about a series of physiological, psychological, and behavioral changes to the different kinds of people that experienced the explosion, impacting their psychological health in different aspects. These impacts might exist only in the short term but could sustain for the long term and lead to serious psychological issues, stress disorder or adaptation disorder, or even psychological disorder. Serious cases might even suicide or hurt others. Therefore, carrying out psychological intervention and therapy in an effective and prompt manner has significant importance.

1 Overview on Psychological Crisis and Intervention

1.1 Definitions of Psychological Crisis and Intervention

An explosion is an abrupt and extraordinary disaster that brings about mental trauma difficult to heal. Psychological crisis refers to acute emotional, cognitive, and behavioral dysfunctions in individuals that experienced the mental stress of an explosion and that are unable to use normal methods and one's own abilities or resources to deal with the issue at hand.

When an individual experiences a psychological crisis, it is necessary to perform effective crisis intervention on said individual. The so-called psychological crisis intervention entails the application of rapid, clear, and effective measures on an individual undergoing psychological crisis to help him or her overcome said crisis and return to normal mental health. This is a kind of short-term psychological assistance process.

Post-explosion psychological intervention refers to the timely provision of appropriate psychological counseling to survivors of explosion, families of the dead, and rescue personnel that are experiencing psychological crisis, so as to help them overcome such psychological troubles as soon as possible.

1.2 Models, Techniques, and Implementation Steps for Psychological Intervention and Therapy

At present, the three main approaches in psychological crisis intervention, namely the balance model, cognitive model, and social conversion model. During the early stages of crisis intervention, the balance model is mostly adopted, and in the later stages of intervention, the cognitive model and social conversion model may be applied. The three different approaches form the foundation for various kinds of in psychological crisis intervention strategies and methods.

Corresponding psychological intervention and therapy techniques should be employed for different psychological crises and conditions. Generally speaking, there are two major categories of techniques: support techniques and intervention techniques. Support techniques refer to emotional or psychological support provided to the subject in question, which serves to prepare the subject for further intervention and therapy procedures. Intervention techniques are founded upon changing the subject's cognition as a premise and apply a range of methods to help the person recognize and understand the psychological crisis'course of development and the inducement relationship, as well as teaching the individual skills and methods to address and deal with the problems.

Although psychological crisis intervention and therapy do not have a fixed procedure, the following basic steps are usually the same. Step 1 is to perform a sufficient assessment

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of the psychological crisis, step 2 is to formulate a crisis intervention and therapy plan, and step 3 is to execute the intervention plan. Step 3 is the actual implementation process and constitutes the staple in the entire course of intervention and therapy. In general, the following areas are the main focus of this endeavor: Stabilize the subject's emotions; facilitate the subject to realize and accept the crisis at hand; provide methods and techniques for dealing with this crisis, and encourage the subject to more actively participate in other activities.

1.3 Necessity of Psychological Intervention and Therapy

PTSD, or post-traumatic disorder, is a common consequence of an explosion injury and a source of psychological crisis. When a psychological crisis occurs in an individual, it is highly crucial to administer emergency psychological intervention and therapy. This should ideally be performed within 2–10 days but could also be within 3–4 weeks for victims of a major disaster. There are two reasons for emergency interventions and therapies for psychological crisis: The first is to avoid the victim from hurting oneself or others and the second is to restore psychological balance and motivation.

2 Psychological Intervention and Therapy for Survivors of Blast Injuries

Blast injuries come in the form of physiological harm, which people have fully acknowledged and extensively studied, based on which corresponding treatment strategies have been devised. However, psychological wounds suffered by explosion survivors and the pattern of their recovery still lack systematic, scientific, and effective research and measures. In the upcoming section, the focus will be placed on discussing general psychological characteristics, causes of psychological issues, and the strategies and methods for intervention and therapy for different stages, with the goal of providing some theoretical and methodological references in psychological assistance for blast injury survivors.

2.1 Psychological Characteristics of Survivors

The onset of psychological crisis reaction instigated by blast injury is sudden and might take place as soon as extraordinarily powerful stimulation is imparted. Such reactions are characterized by tremendous changeability and diversity. Psychological characteristics of survivors can be generally summed up as follows.

1. **Adverse emotional conditions**. Surveys indicate that poor emotional conditions exhibited by blast injury survivors include depression, loneliness, fear, irritation, anger, and anxiety.
2. **Adverse physiological reactions**. These are mostly bodily discomfort, sleep loss, and poor diet or unwillingness to eat or drink.
3. **Cognitive disorder**. These include different degrees of attention deficit, loss of interest, and reduction in self-performance.
4. **Increase in adverse coping behaviors**. These include forcing, avoidance, indifference, suicide, self-blame, guilt, withdrawal, fantasizing, etc.

2.2 Causes for Psychological Issues in Survivors

Based on intensity, an explosion may be classified as either a high-order explosion or a low-order explosion, and different order of explosion causes different kinds of bodily injuries, but serious, multi-organ, composite injuries are very common. Since explosions always occur suddenly and unexpectedly with destructive consequences, there are often many victims that die on the spot or soon afterward due to critical injury, and these could lead to mild or severe psychological issues in survivors. Main causes of issues include the following.

1. **Fright from the disastrous scene of the explosion**. The gruesome and disastrous scene of the explosion often seriously impacts the normal psychological activities of people on-site, which could lead to a dramatic deterioration in the mental state of survivors after they return to normal life. Their emotional state might fluctuate wildly, followed by a series of negative emotions.
2. **Body injuries caused by the explosion**. Although survivors might have escaped with their lives, the worst blow comes from having to deal with the consequence of survival, including serious bodily injuries and disabilities such as amputation, loss of sight, and disfigurement. At such junctures, survivors would often ask themselves, "how could I deal with this for the rest of my life?"
3. **Pain from loss of family**. While many survivors are shocked by the event, they might also have to bear the pain of losing loved ones. These serious impacts and losses that all occur abruptly inflict enormous psychological trauma on survivors that are difficult to surmount. The loss of loved ones results in extreme states of emotional loss such as depression, guilt, anxiety, anger,

tiredness, helplessness, loneliness, fear, longing, etc. Other than dealing with such pains, some might be haunted in their sleep or blame themselves for the death of their loved ones.

4. **Impact from damage to home and property**. Explosions often cause huge losses in assets and property, instantly changing the material foundation and condition upon which people rely in their daily life. Such sudden and colossal changes would leave strong psychological effects on people.

2.3 Psychological Intervention and Therapy Techniques for Survivors

Post-blast injury psychological intervention and therapy entails the swift provision of psychological support to survivors so as to allay the early stages of pains caused by blast injury and help them cope in the short term and long term. This area includes the following aspects:

1. **Contact and connect**. Use an uninterrupted, sympathetic, and constructive attitude to respond to the contact or the initiation of such contact from a survivor.
2. **Security and comfort**. Restoring the sense of security is an urgent and crucial objective after a blast injury. Provide emotional comfort and support to survivors, as improving their sense of security is one of the most integral parts of psychological intervention. Show care about their bodily comfort, encourage them to partake in social activities, avoid worsening their traumatic experience, and avoid things associated with this trauma.
3. **Emotional stability (if necessary)**. Most individuals that have suffered blast injury do not need "special" measures to stabilize their emotions. Forceful, numbing, or anxious emotional states are normal reactions caused by the stress of a traumatic event. However, an extremely strong and sustained internal state of arousal, numbing, or anxiety might affect the survivor's normal performance and function, and in such cases, application of appropriate interventive measures should be considered in order to help the survivor stabilize his or her emotional breakdowns and adapt to the circumstance mentally. If these interventive measures still could not stabilize their emotions, then it might be necessary to ask a psychologist to carry out some medication therapy.
4. **Substantive aid**. After experiencing a disaster, people often feel lost or hopeless. Providing people with things they need can bolster their confidence, hope and help them restore their dignity. Therefore, helping survivors deal with issues at hand or anticipated in the future is a core component of psychological intervention.
5. **Social support**. Social support is crucial to stabilizing the emotions of survivors of blast injuries and helping them return to shape. Assisting survivors to foster and maintain social contact play a crucial role in helping their restoration. For most survivors, timely care is to enable them to make contact with people they care about most (i.e., spouse/partner, children, parents, friends, etc.). Use tangible methods (i.e., face-to-face meetings, phone calls, the internet, or email) to help survivors contact these persons important to them.
6. **Information support**. Blast injury could cause a sense of bewilderment, confusion, or loss, hampering survivors' faith in their ability to deal with the current situation. Therefore, providing relevant information about the stress response, discussing common psychological reactions to traumatic experience or losses, and offering a variety of methods (such as teaching them behaviors to adapt, simple techniques of relaxation or anger management skills, etc.) can help survivors deal with stress responses and more effectively handle problems.
7. **Sustained and steady assistance relationship**. It is very meaningful for most survivors to maintain rapport with their earliest helpers. Under most circumstances, helpers would not be able to continue maintaining contact with survivors, but losing communication with the first post-disaster rescuer might induce a sense of abandonment or rejection in the mind of the survivor. To avoid or minimize the occurrence of this type of situation, provide survivors with contact info for local health institutions, public psychological support service organizations, or other authorized organizations and volunteers. This would establish a sense of continuous care in the mind of the survivor.

3 Psychological Intervention and Therapy for Families of Explosion Casualties

3.1 Psychological Characteristics of Families of Accident Casualties

Investigative survey results indicate that people with or without experience in accidental death in the family differ significantly in several personality dimensions such as affiliation with others, trust, calmness, and optimism, demonstrating that the loss of family in an accident changed several of their personality traits to a certain extent. In terms of adverse emotions and coping methods, compared to people without experience in accidental death in the family, those that have such experiences tend to exhibit adverse emotions such as depression, loneliness, fear, irritation, anger, and anxiety, and are more prone to coping methods such as forcing, avoidance, indifference, suicide, self-blame, guilt, withdrawal or fantasizing.

3.2 Psychological Intervention and Therapy for Families of Accident Casualties

It is inevitable that those alive suffer from the loss of family, and such pain is a normal psychological and physiological reaction to such serious losses. Therefore, the goals of performing intervention and therapy for family members of accident casualties include helping them endure and come out from the normal distress response process so that they can face the pain, express their emotions for the dead, and find new objectives in life.

For an individual that has lost family members, the first and foremost action should be the establishment of a supportive relationship and assessment of the individual's psychological status, then formulate a psychological intervention plan. Specific psychological intervention.

1. **Guide the bereaved to accept the reality of the family lost**. Helping the bereaved recognize, face, and accept the loss of family is the first step to successful intervention.
2. **Educate the bereaved about the psychology of mourning**. Helping the bereaved understand what is "normal" mourning behaviors can help alleviate the worry among the bereaved that they are going insane and accept their seemingly abnormal behavior at the present time.
3. **Encourage the bereaved to use language to express their feelings and memories about the dead**. When dealing with the pain, helping the bereaved discover, accept and express the complicated emotions during the mourning process is key. The intervener may use chiefly open-ended questions to inquire the bereaved about his or her feelings toward the deceased family member(s), create an appropriate emotion venting environment for the bereaved, including chatting, expressing, crying, remaining silent, and remembering with the individual, and providing suitable feedback.
4. **Encourage the bereaved to bid farewell to the dead ritually**. After the bereaved expresses his or her experiences and painful emotions, the intervener may encourage the bereaved to search for tokens for family remembrance, guide the bereaved to bid farewell to the dead in a ceremonial fashion, and discuss with the bereaved about issues with the remains of the dead.
5. **Improve the social support system**. Disastrous events dramatically impact the stability of the social support system, which could increase the probability of post-traumatic stress disorders. During the psychological intervention process for individuals that have lost family members, improving and completing his or her social support system is the most important and effective aspect in helping the person return to normalcy from the pains of the disaster.
6. **Provide active coping methods**. This abrupt death of the family thrusts the bereaved into a sort of emotional imbalance, and existing coping mechanisms and problem-solving methods cannot meet the needs at hand. Therefore, the focal point of psychological intervention and therapy ought to emphasize stabilizing the emotions of the bereaved so that he or she can return to a state of balance. The intervener may instruct the bereaved to recall effective methods of dealing with negative emotions he or she has utilized before, affirm and solidify his or her response, sum up the essences and encourage continual usage. The intervener may also help the bereaved identify passive and negative coping methods, as well as the adverse impacts they cause. In addition, the intervener should provide an array of methods to help the bereaved adapt to the coping behaviors and also teach the individual some simple relaxation techniques.
7. **Rebuild a positive cognitive model**. The mentality of people who have experienced the loss of family would often change dramatically, with cognitive model distortion not uncommon, resulting in a pessimistic outlook that might even lead to suicidal thoughts. Intervener needs to help the bereaved realize his or her irrational thoughts, correct excessive guilt, and self-blame so that the person may return to a rational state of mind and state of self-approval, face and recognize the changes, and adapt to life.

4 Psychological Intervention and Therapy for Rescuers in Explosions

After an explosion occurs, rescuers have to work with constant and immense pressure over extensive hours, all the while compounded by a swath of gruesome and destructive scenes littered with bodies of the dead and resonating with the moans of the injured and wailing of the bereaved, which could easily lead to various kinds of psychological reactions and vicarious trauma. When this type of psychological trauma surpasses the tolerance limit, the outcome is a serious psychological issue. Obviously, rescuers also need to be rescued through psychological intervention and therapy.

4.1 Psychological Characteristics of Rescuers

1. **Adverse emotional reactions**. Fear is the most easily triggered, most common, and most spreadable type of emotional reaction in such a rescue situation. In addition, anxiety and obsession are also caused by the exigence of the mission and the prolonged duration of a stressful state of mind. Some survivors might die in the hands of a rescuer during the rescue process, which might cause the rescuer to blame oneself.
2. **Physiological maladaptive response**. Due to the extreme tiredness of both the body and mind, some physical responses include a drop in performance, dizziness, bodily discomfort, loss of sleep or nightmare, poor diet, or eating disorder.
3. **Cognitive ability and efficiency issues**. These include attention deficit, difficulty in making decisions, memory loss, and flashbacks. Some might exhibit a lack of interest, reduction in the sense of the efficiency of one's own performance, or even emotional exhaustion.
4. **Changes in personality**. Some might show withdrawal or avoidance of contact with others, indifference, lack of self-control, irritation, or nervousness, or stress in terms of managing relationships with others.

4.2 Psychological Intervention and Therapy for Rescuers

Carry out psychological health knowledge training for rescuers before they head out on the mission so that they can master the basic skills related to psychological health and improve their own psychological conditioning. In addition, assessing the psychological health of rescuers beforehand is an effective preventative measure.

Try to ensure that the normal diet, sleep, and rest of the rescuers can be maintained, address their difficulties or demands in a prompt manner, take timely measures to prevent the onset of disease, and help them keep in touch with their families.

Strengthen communication between rescuers themselves, and augment the internal synergy and mutual assistance within the rescue team.

Raise the sense of pride in rescue work, and post-mission commendations and awards are also favorable to enhancing the psychological health level of the rescuers.

Formal psychological counseling needs to be undertaken after the rescue mission ends. Counseling concerns include education for the normalization of psychological reactions, incident stress debriefing, relaxation training, etc. Carry out psychological therapy and/or drug therapy for rescuers that demonstrate stress disorder.

Part II

Key Complications and Their Treatment

Hemorrhagic Shock

Liangming Liu

1 Pathophysiological Characteristics and Mechanisms of Hemorrhagic Shock Caused by Blast Injury

1.1 Pathophysiological Characteristics of Shock Due to Blast Injury

Shock is a common syndrome of organ dysfunction caused by insufficient effective circulating blood and reduced tissue perfusion due to various serious pathogenic factors, such as severe war injury, trauma, blood loss, infection, cardiac dysfunction, and allergy.

With the use of high-energy and highly explosive weapons in modern war, increasingly frequent terrorist attacks, urban explosions, and gas explosions in industrial and mining enterprises, explosive injuries have become common injuries in wartime and peacetime. Due to the variety of injury factors caused by blast injury, the condition is more serious and complex than general gunshot injury and trauma. Blast injury often causes severe contusion of tissues and organs. At the same time, shrapnel and debris produced by explosion can hit large blood vessels, often leading to massive blood loss and hemorrhagic shock. In general war trauma, the incidence of shock is 10–15%, and the incidence of explosive shock can be as high as 39%. Because explosive shock is often accompanied by damage to other organs, its mortality is higher than that of general shock. According to the characteristics of blast injury, the shock caused by blast injury is mostly hemorrhagic shock, sometimes accompanied by burn shock, and later secondary infection may be accompanied by septic shock. Therefore, we should pay close attention to whether there is shock after explosive injury. Once there is shock, we should actively and properly deal with it, including hemostasis, anti-shock, and organ function protection.

1.2 Cardiovascular Dysfunction and Mechanisms of Shock Induced by Blast Injury

1.2.1 Hemodynamic Disorder and Mechanism

Hemodynamics is a comprehensive index reflecting cardiac and vascular functions. Blast injury-induced shock is mainly hemorrhagic shock, with the main hemodynamic changes of decreased blood pressure and cardiac function. The marked indicator of impaired cardiac function is the decrease of cardiac output (CO). Cardiac output is a comprehensive index reflecting cardiac pump function. For example, it is calculated by unit body surface area, which is called cardiac index (CI). Cardiac output is determined by heart rate and stroke output, which depends on preload, afterload, and myocardial contractility. In the process of hemorrhagic shock, CO or CI decreases absolutely or relatively. The normal value of CO in adults is 3.5–5.5 L/min, and CO is often lower than 2.5 L/min in cardiac insufficiency and failure. Under normal conditions, the contractility of myocardium is determined by the volume of myocardium and the activity of sympathetic adrenal system. After hemorrhagic shock, tissue ischemia and hypoxia can damage myocardial contractility and reduce cardiac output. In addition to the decrease of heart function, the reduction of blood volume and the vasomotor dysregulation are also the main factors.

1.2.2 Cardiac Dysfunction and Mechanism

1. **Predisposing factors of cardiac dysfunction**
   (a) Insufficient blood perfusion and abnormal distribution of myocardial tissue. Myocardial tissue consumes the most oxygen in the human body. Generally, the tissue absorbs 20–30% of oxygen from arterial blood, and the oxygen absorbed by myocardial tissue can be as high as 65–70% of arterial oxygen content. After the severe hemorrhagic shock, coronary blood flow decreased significantly, resulting in myocardial ischemia and hypoxia, myocardial metabolic disorder, and structural damage, followed by insufficient

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energy supply of myocardial cells, decreased myocardial contractility, and cardiac pump dysfunction.

Heart rate increases, myocardial oxygen consumption increases, and the sympathetic catecholamine system is excited during shock when β adrenoceptors increase heart rate and myocardial contractility. When the heart rate is accelerated within a certain range, it has compensatory significance because it can improve cardiac output. However, when the heart rate is too fast, it can lead to insufficient ventricular filling and reduced cardiac output. On the other hand, tachycardia can increase myocardial oxygen consumption and aggravate myocardial hypoxia. Each time the heart contracts, the myocardial oxygen consumption is 5–15 mL/(min · 100 g) tissue, and each relaxation consumes about 2 mL/(min · 100 g) tissue. Therefore, when the heart rate increases from normal 75 beats/min to 100 beats/min, the myocardial oxygen consumption can increase by 113%. The faster the heart rate, the higher the myocardial oxygen consumption.

(b) Role of myocardial depressant factor and inflammatory factors. As early as 1966, a substance was discovered to inhibit myocardium in the plasma of hemorrhagic shock cats, which became the myocardial depressant factor (MDF). Later, it was reported that this substance also exists in patients with septic, traumatic, and cardiogenic shock. Research suggests that MDF may be two kinds of substances with different molecular weights: one is low molecular weight MDF that may play an early rapid inhibitory effect on the myocardium, and the other is high molecular weight MDF which plays a late, delayed inhibitory effect on the myocardium. In addition, many inflammatory factors and cytokines after shock, such as tumor necrosis factor alpha (TNF-α) Interleukin-1β (IL-1β) can induce damage to cardiac function.

2. **Mechanism of cardiac dysfunction**

(a) Receptor desensitization. The maintenance of normal cardiac function depends on the joint regulation of the central nervous system and endocrine system. The dysfunction of the adrenergic receptor system plays an important role in cardiac dysfunction in hemorrhagic shock.

Adrenoceptors include α (α_1,α_2) receptors and β (β_1, β_2) receptors, involved in the regulation of most organ functions in vivo. The receptors distributed on the myocardial cell membrane mainly include β_1, β_2, and α_1, where the β_1 receptors are mostly distributed in the myocardial sinoatrial node and coronary vessels, accounting for 70–80% of the total receptors, β_1 and α_1 receptors are mainly distributed in cardiomyocytes, such as the vascular wall, endocardium, adventitia, and conduction system, accounting for 20–30% of the total receptors. During shock, norepinephrine (NE) in sympathetic nerve and myocardial sympathetic nerve endings and norepinephrine levels in circulating blood increase. In the early stage of shock, norepinephrine can pass through the β adrenoceptor information transmission system enhances myocardial contraction. But in the middle and late stages of shock, the β_1 receptor was down-regulated after long-term exposure to high concentration NE, whereas β_2 and α_1 receptors are mainly distributed in non-myocardial tissue and thus less affected by high NE and they change little. During shock, β adrenoceptor and all links of its signaling pathways are significantly inhibited, resulting in decreased sensitivity to catecholamine and decreased cardiac function.

(b) Calcium homeostasis imbalance: The regulation of Ca^{2+} concentration in cardiomyocytes holds the key to determining myocardial diastole and contraction by various calcium transport systems on the myocardial membrane, mitochondria, especially the sarcoplasmic reticulum membrane. When cardiomyocytes are excited, firstly, the voltage-dependent calcium channel of the myocardial membrane on the myocardium is opened, which makes extracellular calcium much higher than that in cells flow into cells through the L-type channel, and induces sarcoplasmic reticulum to release a large amount of Ca^{2+} into the cytoplasm. When its concentration increases rapidly, Ca^{2+} binds to regulatory proteins, resulting in configuration changes, exposing the action point of the myosin cross-bridges, and forming an effective cross-bridge. At the same time, Ca^{2+} activates myosin ATPase to decompose ATP and release energy, resulting in myocardial contraction. During the repolarization of cardiac muscles, the uptake of Ca^{2+} by the sarcoplasmic reticulum calcium pump and outward transport of Na^{+}-Ca^{2+} exchange can reduce Ca^{2+}concentration leading to myocardial diastole. In the coupling of myocardial excitation–contraction and repolarization diastole, membrane calcium channel, and sarcoplasmic reticulum play a key role in the regulation of intracellular free calcium concentration. During shock, the uptake and release of calcium by membrane calcium channel and sarcoplasmic reticulum can be changed, resulting in the disorder of calcium homeostasis and decline of myocardial contractility.

(c) Calcium desensitization. The normal function of the myocardial contractile protein and regulatory protein is needed to maintain the normal systolic and dia-

stolic function of the myocardium, in addition to the signaling pathway system with β-receptors and the balance and stability of Ca^{2+} in cardiomyocytes. During myocardial ischemia and hypoxia damage, due to local or diffuse necrosis of the myocardium, a large number of myocardial contractile components are lost, which can weaken the contractility of the ventricle. Our laboratory found that during shock, especially in the late stage, due to myocardial ischemia and hypoxia, the effects of various cytotoxic substances and their metabolites on myocardium can be through various ways and mechanisms, which reduces the binding force between Ca^{2+} and calcium-binding protein (such as H^+ and Ca^{2+} competing for binding calcium protein sites), or weaken the response of myofibrils to Ca^{2+}, or turn myocardial chemical energy into mechanical energy disorder due to insufficient ATP and reduced ATPase activity, or lead to the decline of myocardial diastolic and systolic function due to the destruction of contractile protein structure and function.

(d) Mitochondrial dysfunction. Shock can seriously damage the energy processing factory of cells, that is, mitochondria. Myocardial mitochondrial dysfunction exists in both hemorrhagic shock and septic shock, which is mainly manifested in the destruction of mitochondrial ultrastructure, the disorder of respiratory function, the reduction of oxygen utilization capacity, the inhibition of energy production, and the decline of cardiac function.

In short, factors and mechanisms of cardiac dysfunction and changes in diastolic and systolic performance during shock are extremely complex, which is not only related to the type, development stage, and severity of the shock but also related to different inducing factors acting through organ, cell, subcellular and molecular levels, respectively, or at the same time.

1.2.3 Vascular Dysfunction and Mechanism

The vascular function includes a vasomotor, barrier, and material exchange function, and the damage of vasomotor and barrier function after shock is the most significant. The impairment of vasomotor function after shock mainly includes vascular hyporeactivity. The damage of vascular barrier function is mainly presented as vascular leakage. Vascular hyporeactivity and vascular leakages are important pathological processes and key complications of severe trauma, shock, and multiple organ dysfunction syndromes (MODS). Their occurrence will seriously affect the occurrence, development, and treatment of trauma and shock, which is a major problem perplexing the treatment of severe clinical diseases such as shock. In view of the mechanism, great progress has been made in recent years.

1. **Vascular hyporeactivity**

(a) Characteristics and rules of vascular hyporeactivity in shock. There are biphasic changes and organ differences in vascular reactivity after shock. The vascular reactivity increases in the early stage, which is manifested by the response of a variety of arteries, including superior mesenteric artery, renal artery, and pulmonary artery to norepinephrine (NE) systolic response increased. With the extension of shock time, vascular reactivity decreased gradually and decreased significantly at 1, 2, and 4 h after shock. Vascular reactivity is different in various organs after hemorrhagic shock. In other words, the vascular reactivity changes differently in different organs after shock where in the celiac artery and left femoral artery, and the vascular reactivity decreases the most, followed by superior mesenteric artery and renal artery, which is related to the different expressions of nitric oxide synthase, cytokines, and endothelin-1.

The vascular reactivity of rats after shock showed different in age. A study found that the vascular reactivity of 7-week-old rats after shock was the highest. With the increase of age, the vascular reactivity of rats after shock gradually decreased. Compared with normal rats of the same age, the vascular reactivity decreased most significantly after shock at 7 and 10 weeks, both of which were more than 30%, with the increase of age, The loss of vascular reactivity gradually decreased after shock. The vascular reactivity of 24-week-old rats decreased by about 10% of that of normal 24-week-old rats. In addition to age differences, there are gender differences in post-shock vascular reactivity. Compared with male rats, female rats showed stronger resistance to endotoxemia caused by bacterial lipopolysaccharide (LPS) and less impaired vascular reactivity. The same trend was noted in hemorrhagic shock; that is, the loss rate of vascular reactivity in female rats was lower than that in male rats. The results showed that there were differences in shock time, organ, age, and gender.

(b) Inducing factors of vascular hyporesponsiveness after shock: multiple factors can induce shock vascular hyporesponsiveness. The initial study showed that acidosis and energy metabolism were the main causes of shock vascular hyporeactivity. Correcting acidosis and supplementing energy had a certain effect on restoring shock vascular hyporeactivity, but the effect was limited; Subsequently, it was found that nitric oxide (NO) and endothelin (ET) play an important role in inducing shock vascular hyporesponsiveness, where NO has been more studied in the occurrence of shock vascular hyporesponsiveness. The inhibitors of

no and et have a certain effect on the prevention and treatment of shock vascular hyporesponsiveness.

With the deepening of research, in recent years, it has been found that in addition to the above factors, cytokines, endogenous opioid peptides, and adrenomedullin also play an important role in the occurrence of shock vascular hyporesponsiveness, where cytokines have attracted more attention in the occurrence of shock vascular hyporesponsiveness, and cytokines changed the vascular reactivity time-dependently. The short-term effect is mainly vasoconstriction, while the long-term effect is vascular hyporesponsiveness stimulated by cytokines. In the late stage of shock, the large release of cytokines plays an important role in the occurrence of vascular hyporesponsiveness, which participates in the occurrence of vascular hyporesponsiveness by causing adrenergic receptor desensitization. In addition, studies have found that endogenous opioid peptides and adrenomedullin also play an important role in the occurrence of vascular hyporesponsiveness in shock. Endogenous opioid peptides may inhibit adrenergic receptors. It regulates the large-conductance calcium-dependent potassium channel (BK_{Ca}) of vascular smooth muscle cells and regulates vascular reactivity after shock. Adrenomedullin participates in the occurrence of vascular hyporeactivity after shock by inducing no production.

(c) Shock vascular hyporesponsiveness. Scholars at home and abroad have done a lot of research on the mechanisms of shock vascular hyporesponsiveness. Existing studies suggest that the mechanisms involved in the occurrence of vascular hyporesponsiveness in shock include receptor desensitization, membrane hyperpolarization, and calcium desensitization.

- Receptor desensitization: Membrane receptor desensitization mechanism refers to the reduction of the number of adrenergic receptors and the decrease of receptor affinity caused by the stimulation of high concentration of cytokines, receptor agonists, endogenous opioid peptides, and NO, resulting in receptor desensitization, resulting in the occurrence of vascular hyporesponsiveness.
- Membrane hyperpolarization: Membrane hyperpolarization mechanism refers to the over the opening of BK_{Ca} channel and KATP channel of vascular smooth muscle cells due to the reduction of ATP and stimulation of some inflammatory factors after shock, resulting in membrane hyperpolarization of vascular smooth muscle cells, inhibition of voltage-dependent calcium channel and insufficient calcium influx, resulting in vascular hyporesponsiveness.
- Calcium desensitization: Although receptor desensitization and membrane hyperpolarization theory explain the mechanism of vascular hyporesponsiveness after the shock to a certain extent, with the deepening of research, it is found that they cannot fully explain some phenomena of vascular hyporesponsiveness after shock. Their central idea is that the occurrence of shock vascular hyporesponsiveness is due to the insufficient increase of calcium in vascular smooth muscle cells after shock, but in severe shock or late shock, vascular smooth muscle cells are not less calcium, but more calcium, and even calcium overload, but there is still the problem of reduced vascular reactivity. Based on this phenomenon, the author proposed the calcium desensitization mechanism of vascular hyporesponsiveness after shock; that is, there is calcium desensitization of muscle contractile protein in vascular smooth muscle cells after shock. Calcium desensitization may play an important role in the occurrence of vascular hyporesponsiveness after shock. It was found that Rho kinase and PKC are the main pathways regulating calcium sensitivity of vascular smooth muscle cells after shock. The change and regulation of their activity after a shock are the main mechanism of calcium desensitization of vascular smooth muscle cells after shock.

2. **Vascular leakage**. Vascular hyperpermeability is an important pathological phenomenon in the process of shock. Vascular hyperpermeability and subsequent tissue edema can not only aggravate microcirculation disorder and tissue cell ischemia and hypoxia but also aggravate the damage to tissue cells, including microvessels and microlymphatic vessels.

The main sites of increased vascular permeability are capillaries and venules. It was previously thought that the reason for the increased vascular permeability during shock was the formation of an intercellular gap due to cytotoxicity in a pathological state. Recent studies have shown that the increased vascular permeability after shock is activated by endothelial cells in addition to the destruction of intercellular junction and widening of the intercellular gap, enhanced transcellular transport also plays a major role. Vascular leakage in the early stage after shock is mainly due to the effect of inflammatory mediators such as thrombin, histamine, bradykinin, and leukotriene B4 on endothelial cells, which belong to non-genotype reaction in signal transduction effect. The continuous exudation in the later stage is due to cytokines in immune response (such as IL-1, TNF, and γ-interferon),

belonging to genotype response in signal transduction effect.

1.2.4 Microcirculation Dysfunction and Mechanisms

As a part of the systemic circulation, microcirculation is regulated by the neuroendocrine system, immune system, nutrition, metabolism, and other internal environmental conditions. However, the current research shows that the regulation of microcirculation function in the course of shock is mainly local factors, which are the factors that can promote microcirculation disorder at the tissue and cell level, mainly including the following aspects:

1. **Microcirculatory vasomotor dysfunction**. Normal microvessels have automatic rhythmic vasomotor movement. Vascular vasomotor dysfunction is characterized by excessive contraction or expansion of arteries and veins, uncoordinated contraction and expansion between arteries and veins, or a large number of anastomotic branches of arteries and veins. When a shock is caused by severe trauma, massive blood loss, or fluid loss, microcirculatory vasomotor dysfunction is often the first pathological change.

   Both systemic and local microcirculation vasomotor dysfunctions lead to microcirculation disorder. In the local tissue, if both arterioles and venules contract, the capillary network tends to close, and the tissue cells in the controlled area are ischemic and hypoxic. If it lasts too long, it will cause ischemic cell damage; when the contraction of arterioles is more obvious, the return of intercellular fluid at the venous end can be increased due to the decrease of internal capillary pressure; when the microvascular contraction is more obvious, due to the increase of internal capillary pressure, the production of intercellular fluid can be increased, resulting in edema and blood concentration. It can also slow down the blood flow in microcirculation, resulting in congestion or blood flow stop. If both arterioles and venules are dilated, the capillary network will be opened, and the blood flow of microcirculation will increase; when the expansion degree of arterioles and venules is significantly different, due to the significant impact on the internal pressure of capillaries, the imbalance of fluid exchange inside and outside blood vessels, and the change of blood flow velocity can occur. The changes in systemic circulation function can directly or indirectly affect the blood supply of various organ systems, the metabolism, and the function of tissues and cells. For example, extensive contraction of arterioles and arterioles can increase cardiac afterload and blood pressure, resulting in the reduction of organ blood supply; extensive dilatation of arterioles and arterioles can reduce cardiac afterload, but it is not conducive to maintaining normal blood pressure; extensive venule and venule expansion can significantly reduce the amount of return blood and cardiac output, resulting in the reduction of effective circulating blood volume and blood pressure.
2. **Hemorheological changes**. Hemorheological changes are not only the result of microcirculation disturbance in shock but also one of the important factors to promote the injury and aggravation of shock microvessels. During the transition of microcirculation from contraction and ischemia to expansion and congestion, the changes of microblood flow often show different flow patterns such as linear flow, linear particle flow, granular linear flow, granular flow, slow granular flow, granular swing flow, and blood flow stagnation. The blood flow in microvessels changes from normal "pill flow" (red blood cells are suspended in plasma, and one line passes through microvessels) to "skimming flow" (red blood cells are separated from plasma, plasma flows, but no red blood cells enter), leading to vascular endothelial injury, leukocyte aggregation activation, platelet aggregation, coagulation system activation, blood flow stagnation, and aggravating microcirculation disorder and tissue cell injury. The hemorheological changes in microcirculation are a comprehensive result of the changes of various blood cells and plasma properties, with the following influencing factors.
   (a) Reduction of erythrocyte aggregation and deformability. Erythrocyte aggregation is the earliest manifestation of erythrocyte flow disorder during shock. Severe erythrocyte aggregation significantly reduces the cell surface area bound to oxygen. At the same time, large agglomerates formed by aggregation can block microvessels and aggravate hypoxia. The causes of erythrocyte aggregation include: (1) increased concentration of abnormal proteins such as fibrinogen in plasma, which adsorbs on the surface of erythrocytes and obscures the electronegative groups on the surface of erythrocyte membrane, resulting in a decrease in the negative charges on the surface of erythrocytes; (2) increased hematocrit (HCT), which causes blood flow to slow down or even stagnate, and increases the collision probability of blood cells, easy for aggregation; (3) decreased blood pressure during shock, which slows down blood flow, and reduces the shear stress and shear rate of blood flow, prompts erythrocyte aggregation.

   The reduction of erythrocyte deformability is another important manifestation of erythrocyte flow disorder. The red blood cells with reduced deformability are stiff and cannot pass through capillaries smoothly, affect the blood perfusion of microcirculation, and even block microcirculation, resulting in tissue and organ ischemia. The reasons for the decrease of erythrocyte deformability during shock

include: (1) the lack of ATP makes the erythrocyte round, that is, the change of geometry (the change of cell surface area/volume ratio), resulting in the decrease of erythrocyte deformability; (2) Acidosis and osmotic pressure increase in red blood cells, which increases the viscosity and decreases the deformability of red blood cell fluid; (3) During shock, the molecular structure of erythrocyte membrane changes. For example, the configuration of erythrocyte membrane skeleton protein (blood shadow protein) changes after LPS stimulation, which reduces the viscoelasticity and fluidity of the erythrocyte membrane and leads to the decrease of deformability.

(b) Leukocyte seizure and capillary impaction. Leukocyte seizure and capillary impaction refers to the phenomenon that the deformation speed of leukocytes slows down, the time of passing through capillaries prolongs, and even impaction of capillaries, which is involved in the occurrence of the no-reflow phenomenon in the congestion stage and refractory stage of shock. It can also directly damage cells by releasing a variety of toxic substances such as free radicals, lysosomal enzymes, and leukotrienes. It is one of the factors of multiple organ dysfunction syndrome (MODS) in the later stage of shock. The mechanisms include: (1) the deformability of leukocytes decreases, which is manifested by the increase of hardness, the increase of leukocyte volume and roundness, which is an important factor in the initial stage of leukocyte seizure, but its signal transduction pathway is unclear; (2) the decrease of blood pressure reduces the perfusion pressure driving the flow of leukocytes, resulting in the impaction and seizure of leukocytes in capillaries; (3) during shock, cells lack oxygen, energy, and acidosis, resulting in swelling of capillary endothelium, narrowing of the capillary lumen and leukocyte impaction; (4) after shock, leukocytes express leukocyte adhesion molecules (Leu-CAMs), endothelial cells express intercellular adhesion molecule-1 (ICAM-1) and endothelial leukocyte adhesion molecules (ELAM). The role of these adhesion molecules increases the adhesion between leukocytes and endothelial cells and prevents leukocytes from passing through capillaries.

(c) Platelet adhesion and aggregation. In the early stage of shock, the adhesion and aggregation of platelets begin to increase. Platelet aggregation can start the process of intravascular coagulation, cause microthrombosis, block the entrance of venules, capillaries and arterioles, cause microcirculatory blood flow stagnation, and release β-platelet globulin (β-thromboglobulin, βTG), thromboxane A_2, and neuropeptide Y (NPY), β-platelet globulin can inhibit the production of prostaglandin I_2, thromboxane A_2 and neuropeptide Y by arterial vascular endothelial cells, and has a strong vasoconstrictive effect, thus affecting the vasomotor function of microvessels; platelet aggregation can release serotonin and produce platelet-activating factor, activate granulocytes, and cause granulocyte dependent platelet adhesion to vascular endothelial cells; it can also release serotonin, ADP, histamine, prostaglandin E_2, and cationic protein, which can directly damage vascular endothelial cells.

The mechanisms of platelet adhesion and aggregation include: (1) microvascular endothelial cell injury causes the exposure of collagen and microfibrils at the platelet adhesion site under endothelial cells, at the same time, the production of prostaglandin I_2, NO, and ecto-ADP enzyme by endothelial cells decreases, and the release of ADP and Ca^{2+} increases, resulting in platelet aggregation; (2) shock produces a variety of humoral factors, including strong platelet agonists such as collagen, platelet-activating factor, and thromboxane A_2, as well as weak agonists such as ADP, epinephrine, and serotonin, which act on the corresponding receptors on platelet membrane to cause platelet activation and aggregation; (3) after the blood flow slows down, the aggregated red blood cell mass pushes the platelets to the side flow with high shear stress in the blood vessel. Coupled with the effect of shear stress, the configuration of platelet membrane glycoprotein IIb-IIIa (GPIIb-IIIa) and platelet membrane glycoprotein Ib (GPIb) changes, resulting in platelet aggregation. Shear stress can also cause platelet aggregation by promoting the release of ADP from erythrocytes.

2 Assessment and Monitoring of the Hemorrhagic Shock Caused by Blast Injury

2.1 Diagnosis and Assessment of Hemorrhagic Shock

It is not difficult to diagnose hemorrhagic shock, which is triggered by trauma or blood loss or present with at least one of the following manifestations: (1) inducement of shock like trauma or blood loss; (2) abnormal consciousness; (3) thready and rapid pulses, more than 100 times/min or not palpable; (4) wet and cold extremities, positive results of finger test on the skin at the sternal area (refilling time >2 s after acupressure), i.e., skin marks, pale or cyanotic mucous membranes, urine output <30 mL/h or no urine; (5) systolic

Table 1 Assessment of shock

Indicators	Mild	Moderate	Severe
Blood loss	15–20%	20–40%	>40%
Blood pressure	Systolic blood pressure is low or close to normal	Systolic blood pressure ranges 60–80 mmHg, pulse pressure <20 mmHg	Systolic blood pressure is <60 mmHg or cannot be measured
Heart rate	Rapid but powerful	Rapid and weak pulse	The pulse is weak and cannot be palpable
Consciousness	The injured is conscious and may be anxious or irritable	The injured is indifferent with slow response	Coma
Skin mucosa	Pale facial skin, clammy limbs	Pale skin and mucosa	Cyanosis
Urine volume	Reduced urine	Oliguria or anuria	Anuria

pressure <80 mmHg; (6) pulse pressure <20 mmHg; (7) systolic pressure drops more than 30% from the original base in hypertensive patients. Shock can be diagnosed if (1) and any two of (2), (3), (4) or any one of (5), (6), (7) are met.

Assessment of hemorrhagic shock. Clinically, hemorrhagic shock can be divided into mild, moderate, and severe (Table 1).

2.2 Monitoring of Functions of Organs in Shock

In order to grasp the shock process in time and formulate or revise the diagnosis and treatment plan, it is necessary to closely monitor the shock. The basic monitoring indexes of shock include basic vital signs, hemodynamics, tissue perfusion and oxygenation, biochemical blood test, etc.

2.2.1 Vital Signs Monitoring

Shock is a clinicopathological state characterized by insufficient tissue perfusion. Therefore, as a traditional monitoring index of circulatory dynamics, blood pressure, heart rate, and urine volume are still the basic indicators of shock monitoring. Combined with the patient's consciousness, respiration, and limb terminal temperature, we can understand the tissue perfusion so as to evaluate the amount and speed of bleeding and formulate the treatment plan. These indexes reflect the functional state of the blood circulation system to a certain extent. They are suitable for decompensated shock characterized by low blood pressure, tachycardia, and oliguria, but they have obvious limitations for compensatory shock characterized by abnormal tissue blood flow and oxygen supply.

Shock blood pressure refers to arterial systolic blood pressure <90 mmHg (domestic setting is <80 mmHg), pulse pressure <20 mmHg. Systolic blood pressure in patients with hypertension decreases by more than 30% compared with the original level, indicating that the amount of return blood is seriously insufficient. Blood pressure should be correctly understood in diagnosis. As there is usually a drop in blood pressure during shock, hypotension is an important index to judge shock, but hypotension is not the only standard to judge shock and the degree of shock because hypotension is not necessarily a shock, and normal blood pressure cannot rule out hypoperfusion of tissue organs. If some patients with hypertension are accompanied by high tension dehydration, their blood pressure is often high, but they are actually in a state of low perfusion. In addition, blood pressure itself is not sensitive. Experiments have proved that when the cardiac output decreases significantly, the blood pressure decreases at least 40 min later, and when the cardiac output fails to fully recover, the blood pressure first returns to normal.

In contrast, changes in heart rate and urine volume are more sensitive than blood pressure. Heart rate is the most concise and fast index. Through heart rate, we can judge the condition of shock and guide the application of fluid resuscitation and vasoactive drugs. Urine volume is an important index to judge the perfusion of kidneys and other visceral systems. The normal value of urine volume is 0.5–1 mL/(kg h), or the 24-h urine volume of adults is not less than 700 mL and not less than 30 mL/h. In shock, the decrease of renal perfusion flow reduces glomerular filtration pressure and leads to the decrease of urine volume. Conversely, the decrease of urine volume may also be due to the decrease of renal perfusion flow, suggesting that the maintenance of blood pressure is insufficient and the shock has not been fundamentally improved. During the shock, the urine volume often decreases before the decrease of blood pressure and after the increase of blood pressure.

2.2.2 Hemodynamic Monitoring

Hemodynamic monitoring during shock mainly includes blood pressure, cardiac output (CO), central venous pressure (CVP), pulmonary wedge pressure (PCWP), systemic circulation resistance, pulmonary circulation resistance, etc.

1. **Arterial blood pressure**: monitoring blood pressure is the most important and basic monitoring method in shock. Peripheral arterial blood pressure is very useful in acute trauma monitoring and can provide evidence for

significant blood loss. The most common is to monitor peripheral arterial blood pressure with a cuff sphygmomanometer. However, due to peripheral vasoconstriction during shock, manual blood pressure measurement and noninvasive automatic blood pressure oscillography are not accurate. Even if the blood loss reaches 30% of blood volume, the measured blood pressure may be normal. Moreover, these techniques cannot quickly and continuously detect the hemodynamic changes of unstable patients. Therefore, for patients with severe shock and unstable blood pressure, direct invasive blood pressure monitoring is more effective and safer. Arterial catheterization is considered to be an accurate method to measure systolic blood pressure and mean arterial pressure (MAP) under normal blood flow. However, in hypovolemic shock, due to the increase of small vessel resistance, the rebound wave can enter the large artery where the catheter is placed, resulting in the false increase of the measured systolic blood pressure, while the intra-arterial measurement of mean arterial pressure is less affected by small vessel contraction. Therefore, the accuracy is higher in hemorrhagic shock with low blood flow.

2. **Cardiac output**: cardiac output (CO) refers to the amount of blood emitted by the heart per minute. It is an important index reflecting the cardiac pump function. The calculation formula is CO = stroke output × heart rate. The normal value is 4–8 L/min, which is affected by many factors such as return blood volume, myocardial contractility, heart rate, cardiac output resistance, oxygen demand, and oxygen consumption. Monitoring cardiac output is helpful to diagnose the type and period of shock and judge the curative effect and prognosis. When the cardiac output is less than 4 L/min, it indicates hypovolemic shock. Low cardiac output is a dangerous signal, while in septic shock, the cardiac output can be higher than the normal value. Impedance cardiogram, Doppler, and pulmonary artery catheter thermodilution methods are often used to measure cardiac output. The pulmonary artery catheter thermodilution method is invasive, but the accuracy is high.
3. **Central venous pressure and pulmonary wedge pressure**
   (a) Central venous pressure (CVP) refers to the blood pressure of the right atrium and the great veins in the chest, which reflects the right cardiac preload and right cardiac function, as well as the dynamic changes between blood volume, return blood volume, and right ventricular ejection function. The normal value is 5–12 cmH_2O, which is affected by many factors such as blood volume, venous tension, right ventricular ejection capacity, thoracic or pericardial pressure, and venous return blood volume. The change during shock is generally earlier than the change of arterial pressure, and the dynamic observation of the trend of central venous pressure is more meaningful than measuring a single value. In hypotension, a central venous pressure lower than 5 cmH_2O indicates insufficient blood volume. If it is higher than 15 cmH_2O, it indicates cardiac insufficiency, excessive contraction of venous vessels, or increased pulmonary circulation resistance. If it is higher than 20 cmH_2O, it indicates congestive heart failure. Central venous pressure can be used to distinguish different types of shock, such as decreased central venous pressure in low-volume shock and increased central venous pressure in cardiac tamponade. However, central venous pressure cannot accurately evaluate the left ventricular preload in critically ill patients, and its significance is limited in the presence of valve disease and increased thoracic and abdominal pressure.
   (b) Pulmonary artery wedge pressure (PAWP) represents left ventricular preload, reflecting pulmonary circulation resistance, and left ventricular filling pressure. The normal value is 6–12 mmHg, not more than 18 mmHg. If PAWP is less than 8 mmHg, indicating insufficient blood volume, the accuracy is higher than central venous pressure; If PAWP is greater than 20 mmHg, it indicates the left ventricular dysfunction. If PAWP is equal to or greater than 30 mmHg, it often indicates pulmonary edema. If the pulmonary artery wedge pressure has increased, even if the central venous pressure is not high, the excessive infusion should be avoided to prevent pulmonary edema, and the reduction of pulmonary circulation resistance should be considered. Pulmonary wedge pressure is an important method to distinguish cardiogenic shock from non-cardiogenic shock, but its measured value is affected by valve disease, myocardial compliance, and ventricular rate.

   Central venous pressure and pulmonary wedge pressure can reflect the adequacy of blood volume when cardiac function is normal. When the blood volume is normal, it can reflect the functional state of the heart and blood vessels. Although these parameters can be used to guide fluid resuscitation, if there is cardiac dysfunction, they cannot accurately predict acute blood loss. Moreover, both central venous pressure and pulmonary wedge pressure reflect the preload of the heart by replacing volume with pressure, so they are affected by ventricular compliance. Low blood volume will reduce ventricular compliance, increase central venous pressure and pulmonary wedge pressure, and make their measured values unreliable. Direct measurement of left and right ventricular end-diastolic volumes under ultrasound is considered to be the most effective method to

accurately reflect cardiac preload, which can be used to determine cardiac preload when other monitoring methods are in doubt.

4. **Systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR)**. The systemic vascular resistance (SVR) can be calculated according to the mean arterial pressure (MAP), central venous pressure (CVP), and cardiac output (CO), and the formula is SVR = (MAP-CVP) × 7.5 × 80/CO, the normal value is 700–1500 dsc^{-5}. Pulmonary vascular resistance (PVR) can be calculated from pulmonary arterial pressure (PAP), pulmonary wedge pressure (PAWP), and cardiac output (CO), and the formula is PVR = (PAP-PAWP) × 7.5 × 80/CO, and its normal value is 100–250 dsc^{-5}. In clinical practice, systemic circulation resistance is usually used as the main index to monitor left ventricular afterload, and pulmonary circulation resistance is used as the index to monitor right ventricular afterload.

2.2.3 Tissue Perfusion and Oxygenation Monitoring

Due to the compensatory mechanism of the body, cardiac output, mean arterial pressure, and cardiac perfusion pressure can also be maintained in a certain range of blood loss. Therefore, simple hemodynamic changes are not enough to evaluate whether patients have hemorrhagic shock, and determining the cumulative oxygen debt is of great significance for correctly evaluating the condition and resuscitation effect of patients and preventing multiple organ failure. Oxygen debt, organ oxygen consumption, and tissue acidosis are the main indexes to evaluate tissue perfusion and oxygenation.

1. **Blood oxygen saturation**. Blood oxygen saturation is an important index to evaluate tissue blood perfusion, including mixed venous oxygen saturation ($SmvO_2$) and central venous oxygen saturation ($ScvO_2$). $SmvO_2$ refers to the average value of mixed venous oxygen saturation from the systemic vascular bed. At this time, the blood liquid oxygen partial pressure at the venous end of capillaries in the tissue is balanced with the tissue oxygen partial pressure. Therefore, the venous oxygen partial pressure and blood oxygen saturation of these tissues can reflect the balance of systemic oxygen delivery (DO_2) and oxygen consumption (VO_2), as well as the oxygenation state of the tissue and its normal range is 60–80%. Clinically, $SmvO_2$ measurement is generally used as a method to monitor tissue oxygenation, and pulmonary artery blood extracted by Swan Ganz catheter is used as a test sample. During shock, the oxygen transport is insufficient and the oxygen uptake of tissues and cells increases, resulting in the decrease of $SmvO_2$. If $SmvO_2 < 60\%$, it indicates that the oxygen supply of the whole body is insufficient or the oxygen consumption is increased. If $SmvO_2 < 50\%$, it indicates that anaerobic metabolism and acidosis occur. If $SmvO_2 < 40\%$, it indicates that the compensation has reached the limit. If $SmvO_2 < 30\%$, it indicates that it is on the verge of death. If $SmvO_2 > 80\%$, it indicates that the oxygen supply or oxygen consumption is increased, generally not more than 90%.
2. **Oxygen delivery and oxygen consumption**: oxygen delivery (DO_2) refers to the oxygen delivered by the heart to peripheral tissues every minute, which is determined by hemoglobin (HB) level, arterial oxygen saturation (SaO_2), and cardiac index (CI, = CO/body surface area). The formula is $DO_2 = CI \times 13.4 \times Hb \times SaO_2$, the normal value of resting-state is 520–720 mL/(min · m^2). Oxygen consumption (VO_2) refers to the actual oxygen consumption of the body per minute. It needs to be multiplied by the difference between arterial oxygen saturation (SaO_2) and mixed venous oxygen saturation ($SmvO_2$). The formula is $VO_2 = CI \times 13.4 \times Hb \times (SaO_2 - SmvO_2)$, the normal value of resting-state is 100–180 mL/(min · m^2). Under normal conditions, oxygen consumption reflects the oxygen demand of the body but does not represent the actual oxygen demand of the tissue. Oxygen extraction rate (ERO_2) refers to the utilization rate of oxygen per minute, that is, the ability of the tissue to absorb oxygen from the blood. The formula is $ERO_2 = VO_2/DO_2$. The oxygen uptake rate reflects the internal respiration of tissue and is related to microcirculation perfusion and the function of mitochondria in cells. The normal value is 20–25%, and the maximum limit value is 75%.

   Oxygen uptake rate (O_2ER) is a more sensitive index to evaluate the balance of oxygen supply and demand than DO_2 and VO_2 alone, which can judge the prognosis of patients. $O_2ER > 0.4$ indicates insufficient oxygen supply and accumulation of oxygen debt. If O_2ER is close to 0.5 in critically injured patients, it indicates that it is very dangerous. Within a certain range of cardiac output and blood pressure, if DO_2 decreases, O_2ER can increase to keep VO_2 stable (i.e., not affected by DO_2). However, if DO_2 falls below the critical value, O_2ER cannot meet the needs of aerobic metabolism even if it increases. At this time, VO_2 decreases linearly with the decline of DO_2, accompanied by hyperlactatemia and other manifestations of hypoxia. This state is called oxygen supply dependence. At this time, the DO_2 value is called the critical value of oxygen transport [330 mL/(min · m^2)], that is, the most hypoxic demand for maintaining tissue and cell aerobic metabolism. In addition, in the hypermetabolic state of sepsis, there is a phenomenon of "pathological oxygen supply dependence," which shows that VO_2 still depends on DO_2 even if DO_2 is normal or increases, suggesting that O_2ER decreases, tissue oxygen supply is

insufficient, and oxygen debt exists. However, some studies believe that such data reflecting systemic perfusion and oxygenation are meaningful in the prognosis of a large number of critically ill patients, but the significance of individual patients is still controversial.

3. **Serum lactate and base deficiency**. Serum lactate and base deletion are the most common serum markers for shock diagnosis and resuscitation monitoring, which can reflect the information of systemic perfusion, oxygenation, and anaerobic metabolism in trauma patients.
   (a) Serum lactate. As a product of glycolysis, serum lactate can indirectly reflect oxygen debt. It can reflect tissue hypoperfusion and acidosis before hemodynamic changes. It is a reliable index to evaluate tissue hypoperfusion and tissue oxygen debt. It can indirectly reflect the severity of shock and is a good index to evaluate the prognosis of shock patients. The normal value of arterial serum lactate is 0.1–1 mmol/L, which is allowed to reach 2.0 mmol/L in critically ill patients. If arterial serum lactate is beyond 2 mmol/L, it is hyperlactatemia, and if it is beyond 4 mmol/L, it is lactic acidosis. During shock, hypoxia leads to the increase of arterial serum lactate concentration, which is often accompanied by acidosis. Some data show that if serum lactate concentration is less than 4 mmol/L, the patient can be cured, while if it is higher than 4.0 mmol/L, only 11% could survive, and if it is higher than 8.0 mmol/L, few can survive. If the serum lactate concentration decreases rapidly to the normal level within 12–24 h, it often indicates that shock resuscitation is ideal, tissue perfusion and oxygenation have been improved in a short time. More and more studies have shown that serum lactate can be used as an indicator of the end point of shock resuscitation.
   (b) Base deficiency (BD). Base deficiency reflects the level of anaerobic metabolites such as lactic acid during tissue hypoperfusion and can quickly and sensitively reflect the degree and duration of tissue hypoperfusion and acidosis. In compensatory shock, base deficiency is more sensitive in reflecting the actual loss of volume than other physiological indexes (such as heart rate, mean arterial pressure, cardiac output, mixed venous oxygen saturation). In patients with insufficient circulation volume, ischemia, and hypoxia, the continuous reduction of base deficiency level is often closely related to organ failure and death in critically ill patients. Davis et al. have found that base deficiency can accurately reflect the severity of shock and the effect of resuscitation and is closely related to the incidence and mortality of adult respiratory distress syndrome and multiple organ failure. They observed a large number of trauma patients with base deficiency ≤ -6 within 1 h after injury, and found that the base deficiency value of survivors generally began to recover within 4 h after injury and reached normal within 16 h; the base deficiency value of deaths was still at a low level 24 h after injury. Therefore, the patients with shock were divided into three degrees by the base deficiency value, -5~2: mild, -14~6: moderate, and ≤ -15: severe. Based on this, the average arterial pressure and the amount of fluid required for resuscitation were estimated. In addition, it was found that 65% of the patients with the continuous decline of base deficiency value during resuscitation suffered active bleeding. Therefore, it was considered that base deficiency was an important index to evaluate the severity and duration of microcirculation perfusion insufficiency, and base deficiency was used to judge the end point of resuscitation.
4. **pH in gastric mucosa**. The pH value in the gastric mucosa (pHi) is a sensitive index reflecting ischemia and hypoxia of gastric mucosa. In routine clinical application, its normal value is 7.32–7.44, while pHi less than 7.32 suggests acidemia in gastric mucosa and insufficient splanchnic blood perfusion; and maintaining pHi above 7.35 can improve the survival rate. Intra-gastric mucosal pH is closely related to systemic and organ oxygen consumption, organ failure, and prognosis of critically ill patients. When pHi is corrected, the survival rate can be improved. Besides, it can be the target of shock resuscitation and an important index to test the effectiveness of resuscitation. Studies have shown that the pH in gastric mucosa is very sensitive as an indicator of tissue hypoxia. Even when there are no abnormalities in other indexes of shock and perfusion (such as serum lactate, base deficiency, cardiac output, etc.), the pH in gastric mucosa decreases. After shock resuscitation, even if the mean arterial pressure returns to normal, the pH in gastric mucosa is still lower than normal. Moreover, gastric mucosal pH is the only way to diagnose "recessive compensatory shock" (which means that the general traditional monitoring methods do not clearly show, but the local tissues and organs are indeed in the state of ischemia and hypoxia) and guide resuscitation, and it predicts the prognosis more accurately than other indicators. Some even support that the pH in gastric mucosa is the only reliable index to predict the mortality of multiple organ dysfunction 24 h after admission. However, if the pH in gastric mucosa is according to Henderson–Hasselbach formula, $pH = 6.1 + \log[HCO^{3-}/(0.03 \times PCO_2)]$, then the arterial blood HCO^{3-} used in the formula will reduce the specificity of pH in gastric mucosa as a gastrointestinal parameter, and the treatment

information may be provided too late. If the pH in gastric mucosa is directly detected by inserting a nasogastric tube, the procedures will be complicated, and the exchange balance between the salt solution and gastric mucosa takes 1 h.

Recent studies have shown that gastric mucosal PCO_2 can also accurately reflect the changes in gastrointestinal ischemia and hypoxia. The difference between gastric mucosal PCO_2 and arterial blood PCO_2 is an indicator of gastrointestinal mucosal oxygen metabolism. Studies have found that there is a good correlation between subcutaneous tissue PO_2, percutaneous PO_2, gastric mucosa PO_2, and PCO_2, which can accurately reflect the degree of blood loss. Other studies have found that when systemic oxygenation is normal after shock resuscitation, gastric mucosal PO_2 is still low, indicating that gastric mucosal PO_2 is more sensitive to ischemia than systemic PO_2 and hemodynamic parameters, but the clinical relationship between gastric mucosal PO_2 and acute stage treatment is subject to further studies. Moreover, monitoring gastric mucosal PO_2 is troublesome, and it is unlikely to be carried out in the early stage of resuscitation, which has little to do with the treatment of the emergency department and trauma department. In recent years, using optical fiber sensor probes to directly measure PO_2 and PCO_2 in gastric mucosa can significantly shorten the measurement time (PCO_2 changes can be displayed within 60 s), which is expected to provide a direct basis for the treatment of critically ill patients.

In addition, other researchers measured PCO_2 at other locations outside the gastrointestinal tract, such as esophageal PCO_2 and sublingual mucosa PCO_2 (Psl CO_2). Povoas et al. have found that there was a good correlation between PCO_2 in sublingual mucosa and tissue oxygenation. With the aggravation of shock, PCO_2 in sublingual mucosa increased; When the shock was corrected, the PCO_2 of sublingual mucosa also decreased to normal, and the PCO_2 of sublingual mucosa was highly consistent with the change of arterial blood lactate. Therefore, it is considered that continuous monitoring of PCO_2 in sublingual mucosa is of guiding significance for shock resuscitation. Weil et al. considered that the PCO_2 of sublingual mucosa was higher than 70 mmHg, suggesting the existence of clinical shock. Compared with PCO_2 of gastric mucosa, the monitoring of these indexes is noninvasive and simple and is expected to become a useful means of clinical application.

5. **Cerebral perfusion**. The brain is the most sensitive organ to hypoxia, and compared with other tissues, the recovery ability after cerebral ischemia is poor, and the cells after infarction are difficult to regenerate. Therefore, the monitoring of cerebral perfusion is particularly important in patient treatment.

Brain tissue oxygen partial pressure ($PbtO_2$). Local brain oxygenation is mainly obtained by directly measuring brain tissue oxygen partial pressure, which can find the existence of brain tissue hypoperfusion in the early recovery stage of trauma patients. Studies have proved that the determination of $PbtO_2$ has a strong clinical predictive value. Although this is the most accurate cerebral perfusion monitoring method, its clinical application is limited because it is invasive and needs to be directly close to the brain tissue itself.

Jugular venous oxygen saturation ($SjvO_2$): it is the primary index reflecting brain oxygen consumption, brain tissue perfusion, and oxygenation. Focal edema, increased intracranial pressure (ICP), decreased mean arterial pressure, decreased cerebral perfusion caused by anemia, and tissue hypoxia can lead to the decrease of jugular venous oxygen saturation. However, the effect of jugular venous oxygen saturation monitoring on the treatment results of patients with head trauma is not clear. Indwelling jugular vein balloon (JVB) catheter can continuously measure oxygen saturation with a Spectrophotometer in situ. It is widely used in late resuscitation, neurosurgery, and cardiovascular surgery.

3 Prevention and Treatment of Hemorrhagic Shock Caused by Blast Injury

3.1 Hemostasis and Fluid Resuscitation

According to the characteristics of blast injury, special attention should be paid to the injury of peripheral blood vessels and visceral blood vessels in the first aid at the scene of blast injury, and active hemostasis and damage control should be paid to at the scene. Because blast injury-induced shock is mostly uncontrolled hemorrhagic shock and often accompanied by lung injury, restrictive fluid resuscitation and damage control should be used for early resuscitation. If there is blast lung injury, the amount of infusion should be reduced.

The traditional principle of resuscitation is to advocate active and rapid resuscitation and timely use of positive inotropic or vasoactive drugs to restore blood pressure to a normal level as soon as possible, that is, the so-called active (positive pressure) resuscitation or immediate resuscitation. However, in recent years, with the in-depth study of shock pathophysiology and tissue fluid and oxygen metabolism, these traditional concepts of shock fluid resuscitation are being challenged. Some new resuscitation concepts were put forward, including limited/hypotensive fluid resuscitation, delayed fluid resuscitation, and hyperthermic resuscitation. These new early resuscitation concepts and methods brought new measures for the early

treatment of war trauma shock patients. It is increasingly valued and accepted by clinicians. At present, European and American guidelines for the treatment of massive hemorrhage have been incorporated into these new ideas and measures.

1. **Permissive hypotensive resuscitation**. The traditional concept of rapid recovery of blood pressure after shock mainly comes from the controlled hemorrhagic shock model raised by Wiggers. However, clinically, especially in war injury (trauma) shock, most of them are an uncontrolled hemorrhagic shock. Recent studies have shown that for patients with uncontrolled hemorrhagic shock, a large number of rapid fluid resuscitation before complete hemostasis can increase blood loss and cause dilute coagulation dysfunction and metabolic acidosis. At the same time, a large number of rapid liquid infusions can affect the vasoconstrictive response, leading to thrombus translocation or wound rebleeding. The results of the author's laboratory and other experimental studies show that the target resuscitation pressure of permissive hypobaric resuscitation is to control the systolic blood pressure at 90 mmHg and the average arterial pressure at 50–60 mmHg. The time of hypobaric resuscitation should not be too long, preferably no more than 90 min. If it exceeds 90 min, organ function protection measures should be considered, otherwise it will aggravate the ischemic and hypoxic injury and affect the resuscitation effect. Although this new method has achieved good results in laboratory and clinical resuscitation and traumatic shock, more clinical studies are needed to further verify the effectiveness, safety, and scope of application of this method.
2. **Delayed resuscitation**. It is traditionally believed that if the blood pressure is low after war trauma shock, fluid resuscitation should be carried out immediately, and vasoactive drugs should be used to increase the blood pressure as soon as possible. However, recent studies have found that severe war trauma shock, especially uncontrolled hemorrhagic shock, if vasoactive drugs or a large amount of liquid are used prematurely to increase blood pressure before complete hemostasis, it cannot improve the survival rate of patients. In fact, it is dangerous to increase mortality and complications. Based on the results of laboratory and clinical studies, a new concept of delayed resuscitation for severe war trauma shock, especially uncontrolled hemorrhagic shock, has been put forward in recent years, that is, for patients with traumatic hemorrhagic shock, especially those with active bleeding, immediate massive fluid resuscitation is not advocated before complete hemostasis, but a small amount of balanced salt solution is given to maintain the basic needs of the patients before reaching the operating room for complete hemostasis. A large amount of fluid for resuscitation is carried out after thorough surgical treatment, which will have a better resuscitation effect than immediate active resuscitation. However, the specific amount of liquid to be given before operation (or during evacuation) and which liquid is appropriate need to be further studied and clarified.
3. **Hypothermic resuscitation**. Hypothermic resuscitation has always been a controversial topic. Long-term deep hypothermia will affect body metabolism, coagulation function, and cardiovascular function. However, more and more studies show that short-term mild hypothermic resuscitation can enhance the effect of low-pressure resuscitation for severe traumatic hemorrhagic shock. Our laboratory research shows that short-term (1 h) mild hypothermia (34 °C) from injury to complete operation can significantly enhance the effect of low-pressure resuscitation, reduce the metabolic rate of tissue and cells, reduce the body's demand for oxygen, prolong the golden rescue time of shock, and prevent the increase of capillary permeability. However, what needs to be further studied in the future is how to implement low-pressure resuscitation, what methods to reduce body temperature, and how to cooperate with restrictive fluid resuscitation. It is worth noting that the therapeutic and controlled hypothermia mentioned here is different from the spontaneous and uncontrolled hypothermia in trauma patients. The former is beneficial to the treatment of trauma patients, while the latter is harmful.
4. **Damage control resuscitation**. In recent years, in addition to limited fluid resuscitation, the concept of damage control resuscitation has been put forward to manage patients with severe trauma. Damage-controlled resuscitation refers to the use of non-surgical treatment strategies to prevent or reverse a series of measures to reduce damage and improve the effect of resuscitation in the first 24–48 h, including blood loss anemia, coagulation dysfunction, acidosis, and hypothermia (spontaneous). These measures include the application of permissive low-pressure resuscitation, prevention and treatment of hypothermia with both passive and active heating methods, acidosis correction by an exogenous buffer, etc., direct application of 1:1 fresh frozen plasma and erythrocyte suspension, early use of platelets, early use of recombinant cytokine VIIa, etc. Although damage control resuscitation has been widely used in the early treatment of war trauma shock, some specific schemes and measures, especially the treatment of coagulation dysfunction, need to be further studied to improve the treatment effect.

3.2 Resuscitation Fluid Selection

Resuscitation fluids are usually divided into crystal liquid and colloidal liquid. Crystal liquid is also divided into isotonic liquid and hypertonic salt liquid. The colloidal liquid includes albumin, dextran, gelatin, and hydroxyethyl starch. They have their own advantages and disadvantages (Table 2).

An ideal fluid for war wound resuscitation should meet the following requirements, that (1) it can quickly restore plasma volume and improve microcirculation perfusion and oxygen supply, (2) it can carry oxygen, (3) it causes no obvious side effects, such as immune response, (4) it can protect cells, and (5) it is easy to store, transport, and cheap. Obviously, the current clinical fluids cannot meet these requirements. Therefore, great efforts have been made to solve this problem. (1) Modified hemoglobin solution has been studied, trying to use human waste blood and animal blood to develop hemoglobin that can simulate human body through artificial modification or intermolecular crosslinking, eliminate its immunogenicity, eliminate allergic reaction, and avoid cross matching and infection. In recent years, the USA, Japan, Canada, and other countries have been spending a lot of money on the research and development of such products. Although much progress has been made in technology, many products have entered phase III clinical practice, and some products have been listed in South Africa, Mexico, and other countries. However, because some toxic reactions (such as vasoconstriction, nephrotoxicity, and oxidative damage) have not been overcome, such products have not been put on the market on a large scale, and further research is needed to overcome these problems. (2) Efforts have been made to study functional liquids with cell protective effects to prevent tissue and cell ischemia and hypoxia damage caused by war trauma shock or reperfusion injury caused by fluid resuscitation after shock, but there is no such products for clinical use. (3) Recent studies have shown that a large amount of lactate Ringer's solution (LR) can activate neutrophils (PMN) and lead to tissue damage. Studies have confirmed that D-lactic acid in LR is the main reason for its activation of PMN. LR contains 14 mmol/L of L-lactic acid and 14 mmol/L of D-lactic acid. With LR containing 28 mmol/L of L-lactic acid, the activating PMN effect is significantly reduced; if lactic acid is completely replaced by ketone in LR, the results are similar, indicating that D-lactic acid is related to the activation of PMN. Therefore, the U.S. military proposes to improve the current LR, remove D-lactic acid, reduce the total amount of L-lactic acid, and add ketones as energy materials. At present, a keto Ringer solution has been developed and proved to have a good anti-shock effect.

Table 2 Advantages and disadvantages of different resuscitation fluids

Name	Advantage	Insufficient
Isotonic salt solution	Easy to store and cheap	Low efficiency (only 25% of the whole blood), large infusion volume, easy to cause hemodilution, edema, and coagulation dysfunction
Hypertonic saline	A small amount, high efficiency (450%), increasing myocardial contractility, longer action time than that of normal saline	Excessive use can cause perchloric acid poisoning
Albumin	Strong volume expansion that can replace blood with 1:1	Excessive use will leak into the tissue to affect the tissue function
Dextran	Long expansion time	It can affect coagulation and blood matching and induce allergic reactions
Gelatin	It has little effect on coagulation	Shorter volume expansion time but the higher allergic rate
Hydroxyethyl starch (HES)	Volume expansion efficiency 150–200%	It has no obvious side effects. It is suitable for early first aid of traumatic hemorrhagic shock, but it should be used with caution in the later stage, especially with renal dysfunction

At present, the consensus view on the early stage of hemorrhagic shock is that both crystal solution and colloidal solution should be supplemented at the same time. The data show that the 2:1 ratio of Ringer's lactate solution and hydroxyethyl starch compound solution has a better effect during uncontrolled bleeding permissive low-pressure resuscitation.

3.3 Vasoactive Drugs

With the deepening of the research on the pathophysiology of shock, it has gone from the overall and organ level to the cellular, subcellular and molecular level. Therefore, the treatment of shock, especially anti-shock drugs, has developed significantly, and many new anti-shock drugs have emerged, such as new adrenergic agonists, opioid receptor antagonists, calcium channel blockers, arachidonic acid metabolite inhibitors, phosphodiesterase inhibitors, shock cytokine antagonists, and endotoxin antagonists. It shows a broad prospect for the treatment of shock.

1. **Vasoconstrictor drugs**. In the past, vasoconstrictor drugs were commonly used to improve patients' blood pressure. The more commonly used vasoconstrictor drugs were norepinephrine, metaraminol, ephedrine, etc. Most shock patients had higher blood pressure and improved clinical symptoms. However, tissue perfusion decreased

significantly, and the increase of arterial blood pressure was obtained at the cost of reducing tissue perfusion, which was only an expedient measure. In wartime or disaster accident scene first aid, it can only be used when the blood pressure drops sharply and endangers life. First, use vasoconstrictor drugs to win time for blood transfusion and infusion. When it must be applied, a small dose and low concentration should be used. Stop bleeding, blood transfusion, and infusion as soon as possible to restore effective blood volume.

2. **Vasodilators**. The purpose of vasodilators is to appropriately expand the anterior sphincter of capillaries on the basis of full infusion and blood transfusion so as to increase the blood volume of microcirculation and make the peripheral tissues fully perfused.

   The commonly used vasodilators include adrenergic β receptor stimulants (isoproterenol), adrenergic α, β receptor stimulants (dopamine), adrenergic α receptor blockers (benzylamine, benzimidazoline, tolazolin), anisodamine drugs (atropine, anisodamine, scopolamine), balanced vasodilators (sodium nitroprusside), etc.

   Indications for vasodilators are as follows:

   (a) After intravenous infusion, the central venous pressure has risen above the normal range, but the clinical symptoms of shock have not improved.
   (b) The patient has clinical signs of sympathetic hyperactivity (pale skin, limb chills, small pulse pressure, insufficient capillary filling, etc.).
   (c) Cardiac output is difficult to meet the needs of normal or increased peripheral resistance.
   (d) Late hypovolemic shock leads to heart failure. Cardiac output decreased, total peripheral resistance, and central venous pressure increased.
   (e) Patients in shock have presentations of pulmonary hypertension and left heart failure.

   It should be noted that after the use of vasodilators, the perfusion pressure of abdominal organs (including kidneys) decreases, and the perfusion flow decreases; the oxygen consumption decreases but the oxygen debt increases, which may aggravate acidosis. Therefore, when using vasodilators, various indexes such as blood gas and cardiac function should be monitored in time, and corresponding measures should be taken in time when necessary.

3.4 Heart Function Enhancement

Hemodynamics and blood pressure cannot be improved after fluid resuscitation and an appropriate amount of vasoactive drugs in war trauma shock. When cardiac insufficiency is suspected, cardiac function improvement drugs can be considered. Commonly used drugs include the following:

1. **Isoproterenol**. Isoproterenol is a powerful adrenergic β receptor agonist, which excites the cardiac β1 receptor, resulting in significantly faster heart rate, faster conduction, stronger contractility, and increased cardiac output. Isoproterenol can also excite β2 receptors, mainly dilate blood vessels of skeletal muscle and skin, dilate visceral blood vessels such as heart and mesentery, and reduce peripheral resistance. As a whole, it can increase the systolic blood pressure and reduce the diastolic blood pressure to enlarge the pulse pressure. It can be used to treat hemorrhagic shock and septic shock at a dose of 1–5 μg/min, 1 mg in total, added to 500 mL sugar saline.
2. **Dopamine**. Dopamine also belongs to catecholamines. It can stimulate α and β adrenergic receptors and dopamine receptors. Dopamine can increase myocardial contractility, increase cardiac output, increase myocardial oxygen consumption, and expand coronary arteries, renal vessels, and mesenteric vessels. Dopamine can expand the blood vessels of the kidney and mesentery. At the same time, it can constrict the blood vessels of skeletal muscle and skin and distribute the blood to vital organs. Therefore, it makes the blood distribution more reasonable in shock. Isoproterenol dilates most of the blood vessels in the whole body, making the blood distribution unreasonable. This is an important reason why dopamine is superior to isoproterenol and has attracted clinical attention. The effect of low-dose dopamine on reducing peripheral resistance and blood pressure is generally not significant, but it can significantly reduce blood pressure in patients with insufficient blood volume, so dopamine should also be used on the basis of fluid resuscitation. 20 mg dopamine can be added to 250 mL 5% glucose solution for intravenous drip, 15 drops per minute. If the effect is not obvious, the dose can be gradually increased.
3. **Dobutamine**. Dobutamine is a dopamine derivative, which mainly plays an anti-shock role by acting on adrenergic β1 receptor, increasing cardiac function, relaxing peripheral blood vessels, increasing tissue oxygen supply and oxygen intake, and improving tissue oxygenation function. The common dose is 2.5–10 μg/(kg · min), with a total amount of 5–20 mg, add 250 mL of 5% glucose solution for intravenous drip.
4. **Digitalis preparation**. Digitalis preparation has a positive inotropic effect so that it shows great effects in treating shock complicated with congestive heart failure, where it can increase the output of the failed heart, slow down heart rate, reduce ventricular end-diastolic volume, and save cardiac oxygen consumption. 0.2–0.4 mg of Lanatoside C is commonly used, and 20 mL of 50% glucose solution is added for intravenous slow injection. Because the heart always has a certain degree of hypoxia during shock, it is particularly sensitive to this kind of drug and is prone to arrhythmia after medication. This

kind of drug should be used slowly and carefully, the dose should be smaller than usual, and ECG monitoring should be carried out.

5. **Glucagon**. Glucagon is a 29 amino acid peptide secreted by islet A cells. It can moderately improve myocardial contractility, has no obvious effect on peripheral resistance, and is not easy to cause arrhythmia. The commonly used dose is 1–3 mg every time or 3–4 mg/h.

3.5 Microcirculation Improvement

Improving microcirculation is very important in the treatment of shock by the following measures: (1) appropriate application of vasodilators; (2) use of low-molecular dextran that can dilute blood, prevent red blood cell agglutination and coagulation, and have a good effect when used together with vasodilators; (3) use of appropriate doses of heparin. In case of DIC tendency, add 0.5–1.0 mg/kg heparin to 250 mL glucose solution as early as possible for intravenous drip once every 6 h to prolong the clotting time by one time; in case of excessive application with bleeding tendency, use fisetin for neutralization purpose.

3.6 Acidosis Correction

During shock, tissue perfusion is insufficient, anaerobic metabolism is enhanced, lactic acid production is increased, and intracellular potassium is lost. Acidosis and hyperkalemia often occur. Sodium bicarbonate can be used to correct metabolic acidosis caused by excessive accumulation of lactic acid. The first choice is 5% sodium bicarbonate, 24 h dosage: 300–400 mL for mild acidosis and 600 mL for severe acidosis; 3.5% aminobutanol can be used for the wounded with cardiac and renal insufficiency or avoiding sodium. The mild dose is 300–400 mL, and the severe dose is 500–800 mL. Hyperkalemia should also be actively corrected. In addition to sodium bicarbonate drip, calcium gluconate intravenous drip can also be used to antagonize the toxic effect of potassium ions on the heart. In addition, glucose, insulin, and sodium bicarbonate can be combined intravenously to make K^+ in blood enter cells to reduce blood potassium.

3.7 Recovery of Vascular Hyporesponsiveness in Shock

As mentioned above, vascular hyporesponsiveness exists in severe clinical cases such as severe trauma and shock, which seriously affects the treatment of trauma and shock. At present, effective prevention and treatment measures are being sought for the inducing factors and mechanism of shock vascular hyporesponsiveness. Our laboratory found that the combination of low-dose terlipressin and a certain dose of norepinephrine can better improve the vascular hyporesponsiveness of traumatic hemorrhagic shock and septic shock. The clinical effect is significant. It not only improves the resuscitation effect of septic shock but also reduces the dosage of norepinephrine and the side effects of high concentration norepinephrine. Other studies have found that L-NAME, an inhibitor of NO synthase, pd142893, an antagonist of ET-1, ICI174, 864, and nor BNI, specific antagonists of opioid receptors, glibenclamide, an inhibitor of KATP channel, and genistein, an inhibitor of tyrosine kinase, also have good anti-shock vascular hyporeactivity, but the effects of these drugs are only studied in the laboratory, Whether it can be used clinically needs further research.

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Infection and Sepsis

Huaping Liang and Jun Yan

1 General Concepts

Infection is the main cause of death 5 days after injury, and the cause of death only second to shock. Although considerable progress has been made in debridement, tissue repair, and antibiotics, infection is still a common complication of trauma patients. If not handled properly, it can cause multiple organ dysfunction syndrome and death. According to statistical data, the infection rate of soft tissue trauma, colon injury and multiple injuries with open femoral fractures are about 12%, 8% and 90%, respectively. Infection depends not only on the wound, but also on the type of wound. For example, the infection rate of colon firearm injury can reach 58%.

No matter what type of wound infection, its prevention and treatment measures mainly rely on proper surgical treatment at each stage, and antibiotics only play an auxiliary role. Therefore, the prevention and treatment of trauma infection must not rely too much on antibiotics and neglect the correct surgical treatment.

For the convenience of description, the following basic concepts should be clarified first.

1.1 Infection

It refers to the inflammatory reaction caused by microorganisms invading the body.

1.2 Surgical Infection

It refers to the infection requiring surgical treatment, including trauma, burn, operation, and other concurrent infections.

1.3 Bacteremia

The presence of living bacteria in the circulating blood is called bacteremia. Also, there are several similar concepts such as viremia, fungemia and so on.

1.4 Toxemia

A large number of toxins rather than pathogens enter the blood circulation, causing severe systemic responses, such as endotoxemia.

1.5 Septicemia

In the past, the definition of septicemia refers to the systemic reaction caused by the presence of bacteria in the circulating blood or various toxins produced by them, but its concept is easy to be confused. Therefore, the American College of Chest Physicians (ACCP) and the Society for Critical Care Medicine (SCCM) suggest abandoning this name.

1.6 Systemic Inflammatory Response Syndrome (SIRS)

A series of systemic inflammatory reactions caused by various infectious and non-infectious pathogenic factors are called SIRS. SIRS can be found in many clinical situations, such as infection, pancreatitis, ischemia, multiple trauma, organ damage caused by immune response, the effects of TNF, IL-1, and other mediators. If developed further, it can lead to acute lung injury, renal dysfunction, shock, and multiple organ dysfunction syndrome (MODS).

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1.7 Sequential Organ Failure Assessment (SOFA)

SOFA is a scoring system developed by the working group on infection related issues of the European Society of Intensive Care Medicine (ESICM), mainly on the severity of respiratory system, nervous system, cardiovascular system, coagulation system, liver and kidney and organ failure. With clear evaluation indicators and simple and easy process, the score system can sequentially evaluate the organ dysfunction, properly evaluate conditions and prognosis of critically ill patients, reflect the changes of condition and therapeutic effects during dynamic monitoring, which is also convenient for retrospective analysis. In the third international consensus on the definition of sepsis and septic shock, SOFA has become the clinical standard for determining sepsis. At present, the expert group recommends that on the basis that the basic SOFA value is assumed to be 0, SOFA $\geq$2 points represent organ disorders.

1.8 Sepsis

In the past, sepsis was defined as SIRS in hosts caused by infection. In addition to the presence of infection in the entity, symptoms and signs of SIRS also appear clinically. However, the definition of SIRS often ignores the body's inflammatory response and adaptive response to inflammation. At the same time, its traditional definition is too broad with low specificity. Sepsis 3.0 redefines sepsis as a dysfunctional host response to infection, resulting in life-threatening organ dysfunction. Organ dysfunction refers to a new SOFA $\geq$2 after infection.

1.9 Systemic Infection

Some scholars advocate using the term "systemic infection" instead of "sepsis." Because the expression of "sepsis"is not accurate, it is easy to be understood as the toxin produced by abscess formation and pyogenic bacteria. In fact, systemic infection cannot be accompanied by abscess formation, and "toxin" mainly does not come directly from pyogenic bacteria, but refers to cytokines and inflammatory mediators produced by the body's defense system stimulated by bacteria and their toxins. However, the expression of "systemic infection" is far from perfect, which is easy to be misunderstood as infection in all systems of the whole body. In fact, over the years, "surgical sepsis"has been conventionally defined, that is, the clinical manifestations of severe surgical infection with systemic inflammatory response (such as burn sepsis) are not necessarily related to "purulence and toxins." Since the term "sepsis"is still widely used in the literature at home and abroad, it is impossible to abolish it in the short run. At present, we only need to know that these two expressions refer to the same clinical syndrome, which are commonly used. Let its advantages and disadvantages judged by time.

1.10 Multiple Organ Dysfunction Syndrome (MODS)

Since the traditional definition of multiple system organ failure (MSOF) is relatively vague, ACCP and SCCM put forward a new concept of MODS, that is, the clinical syndrome of two or more organ dysfunction caused by acute diseases (where trauma and infection are often the initiating factors), and organ dysfunction can occur simultaneously or sequentially. According to different pathogenic factors, MODS can be divided into primary and secondary MODS. Primary MODS is the direct result of etiology, while secondary MODS is mainly caused by abnormal host inflammatory response. Although some organ diseases related to the end-stage and pathogenesis of chronic diseases also involve multiple organs, they do not belong to the category of MODS.

2 Main Pathogens of Trauma Infection

2.1 Evolution of Major Pathogens

For decades, the main pathogens of trauma infection have undergone significant changes. *Streptococcus* was the main pathogen of trauma infection in the 1930s; in the 1940s, it was mainly penicillin-sensitive *Staphylococcus*; in the 1950s, a large number of penicillin-resistant staphylococci appeared; since the 1960s and 1970s, gram-negative bacilli represented by *Escherichia coli* and *Pseudomonas aeruginosa* have gradually replaced gram-positive cocci represented by *Streptococcus* and *Staphylococcus aureus* as the main pathogens of trauma infection. According to statistics abroad, from 1945 to 1956, two-thirds of the pathogenic bacteria of wound infection were gram-positive cocci. From 1957 to 1974, the wound infection rate caused by gram-negative bacilli increased 14 times. From the 1970s to 1980s, spore free anaerobes increased significantly in trauma infection, and some new opportunistic pathogens and "non-pathogenic bacteria" in the past continued to appear, such as various fungi, *Serratia marcescens*, *Klebsiella*, *Aerobacter*, cloacae bacilli, and *Acinetobacter*. It had been noted that the number of mixed infections involving anaerobic bacteria and fungal infections (such as *Candida albicans*, *Aspergillus*, *Mucor*, etc.) was increasing. Since the 1990s, gram-positive cocci represented by *Staphylococcus aureus* have made a comeback. The proportion of its infection has exceeded 50% of clinical infection cases, gradually replacing gram-negative

bacilli and becoming the main pathogen of trauma infection. For example, methicillin-resistant *Staphylococcus aureus* (MRSA) infection has posed a clinical threat and attracted much attention. In recent years, infections caused by multidrug-resistant bacteria such as *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Klebsiella pneumococci* have increased significantly. The evolution of pathogens of trauma infection is related to at least the following factors:

1. **The wide application of antibiotics** is an important reason for the evolution of pathogens. With the continuous development and application of new antibiotics, although they can effectively kill bacteria sensitive to antibiotics, they also cause the reproduction of drug-resistant strains. In addition, the abuse of antibiotics can cause the imbalance of human normal physiological flora, which is easy to lead to endogenous infection.
2. **The progress of microbiological detection technology** enables some new pathogens that clinicians are not familiar with to be found. For example, *Serratia marcescens* was recognized as a harmless bacterium in the 1960s, but it was later proved that it not only causes disease, but also death. With the improvement and application of anaerobic culture technology, it has been found that the proportion of anaerobic bacteria in trauma infection is increasing. At the same time, it also shows that in the past, traumatic anaerobic infection may be missed only because of the limitations of culture and detection methods.
3. **The improvement of surgical treatment** also leads to the evolution of pathogens. For example, in the early stage of World War I, Clostridium infection was quite common, but with the improvement of debridement technology, this kind of infection has decreased significantly.
4. **The application of new medical equipment and technologies**, such as respiratory devices, elastic dressings, transplantation of various arteriovenous catheters, sensors, and artificial materials, can often cause iatrogenic infection, such as fungal infection.

Thus, the main pathogens of trauma infection will change constantly. In different regions, the evolution process may be inconsistent, for which we should have a clear understanding.

2.2 Origin and Invasive Route of Trauma Infection Pathogens

During trauma, the main invasive route of pathogenic bacteria is brought in by injury causing instruments and projectiles, and then brought in by clothes, soil, and other dirt. This kind of infection is called exogenous infection. Another source is the resident bacteria of the human body itself, mainly distributed in the sweat glands, hair follicles, oropharynx, respiratory tract, gastrointestinal tract, and urogenital tract. Under physiological conditions, these normal flora do not cause disease, but form a symbiotic and mutually beneficial ecological balance with the human body. When the skin and these cavities are injured and damaged, bacteria can invade; If the structure is not damaged while its defense barrier function is reduced, bacteria can also pass through the skin and mucous membrane and enter the deep tissue to cause infection. This kind of infection is called autoinfection or endogenous infection. After the invasion of bacteria or other microorganisms from exogenous or endogenous sources, they mostly invade lymphatic vessels and blood vessels, or cause specific site and even systemic infection along natural pores. Minor injury and less serious simple trauma or burn mostly occur exogenous infection. While in the case of severe trauma and burn, both exogenous infection and endogenous infection, especially intestinal infection, can occur.

2.3 Clinical Significance of Bacterial Count

A major advance in the study of wound infection is the recognition that the growth level of bacteria in wound or the surface of a wound is more important than the existence of bacteria. Generally speaking, the more bacteria contaminate the wound or the surface of a wound, the greater the chance of infection. At present, it is recognized that the critical number of bacterial infection is 10^5–10^6 bacteria per gram of tissue or per milliliter of liquid. This "threshold" is suitable for any bacterium. Some non-pathogenic bacteria, such as *Salmonella*, *Staphylococcus epidermidis*, and *Bacillus subtilis*, can also cause infection if the number in tissues or body fluids exceeds the above critical limit. It is worth mentioning that this "critical value" is not absolute. On the one hand, when the microbial virulence is particularly strong, such as group A beta-hemolytic streptococci can also cause infection when it is less than 10^5 tissues/g. On the other hand, when the overall resistance of the wounded decreases and the local conditions are conducive to bacterial breeding rather than killing bacteria, even bacteria less than this "critical value," such as 10^2 bacteria/g tissue, will cause infection. On the contrary, in some special cases, this "critical value" may also increase. For example, as the author reports, the critical number of bacterial infection in plateau area is 10^8/g of tissue.

The quantitative examination of bacteria in wound tissue can not only be used as one of the basis to judge wound pollution and infection and guide the rational application of antibiotics, but also an objective index to guide debridement and suturing and predict the success or failure of wound treatment. Where the bacteria is less than 10^5/g, the wound can be sutured immediately after debridement, without future wound infection and with a high healing rate. However,

if the bacterial count exceeds 10^5/g tissue, even after thorough debridement, the wound infection rate after early suture is still very high, sometimes more than half.

3 Main Types of Trauma Infection

3.1 Post-traumatic Suppurative Infection

Common pyogenic bacteria include *Staphylococcus aureus*, *Staphylococcus epidermidis*, pyogenic *Streptococcus*, *Enterococcus*, *Pseudomonas aeruginosa*, *Escherichia coli*, etc. Pyogenic infection can be divided into local wound purulent infection, visceral and body cavity purulent infection and systemic purulent infection due to different location and scope.

3.1.1 Clinical Manifestations

1. Local symptoms include wound pain, swelling of surrounding tissues, redness, fever, and local tenderness of the skin near the wound. The wound is covered with purulent exudates or necrotic tissues of different numbers and colors, and affected by organ dysfunction.
2. When local infection is formed, the whole body also has different reactions. If no bacteria invade the blood, the whole body symptoms are only caused by toxemia caused by bacterial toxin. If bacteria invade the blood, grow and reproduce in the blood and produce toxins, they can develop into systemic infection and sepsis. Some have multiple organ dysfunction and septic shock.

3.1.2 Diagnosis

1. It can be diagnosed according to clinical symptoms and signs: the diagnosis of acute infection (wound suppuration, cellulitis around the wound, etc.) is made based on clinical manifestations, and generally there will be no difficulty. If there is no obvious purulent exudate in the wound or wound, bacterial culture can be counted. The critical number of bacterial infection is 10^5–10^6 bacteria per gram of tissue or per milliliter of liquid. Bacteremia can be diagnosed if viable bacteria are cultured from the blood.
2. When the disease is comparatively serious (such as combined anaerobic infection, biochemical purulent lesions in internal organs and bones) and when the infection spreads to the whole body, in addition to routine smear microscopic examination, wound secretion and blood culture, the diagnosis must also rely on some modern equipment, such as ultrasound, CT, magnetic resonance, or laboratory methods, such as gas chromatography.

3.1.3 Treatment Plan and Principles

1. The most important is undoubtedly to carry out radical surgery in time. Medical staff must extensively inspect the affected area, remove all lifeless tissues, cut open abscesses and swollen areas and drain them fully. Drainage can be carried out by suction flushing, running water flushing or wound gauze loose packing, plus hypertonic ointment with water-soluble matrix. After the infection was controlled, the wound should be closed as soon as possible by secondary suture, skin grafting or adjacent flap transfer.
2. When there are many secretions from infected wounds, wet compress can be used. Common wet compress solutions include bleach boric acid solution, hydrogen peroxide solution, hypertonic or isotonic saline, antibiotic solution, etc. Wet dressing with hypertonic saline could be applied for edematous granulation tissue. The affected limb should be raised without motion. Hypertonic ointment of water-soluble matrix, carbon fiber, and other absorbent, helium containing dressing, proteolytic enzyme fixed on different matrix, etc. can also be used.
3. Broad-spectrum anti-infective drugs may be used; and then select effective anti-infective drugs according to the results of bacterial culture and drug sensitivity test of secretions.
4. Active in vitro detoxification methods can be initiated to reduce the circulating concentration of toxic components in blood, lymph, and interstitial fluid. The most commonly used in vitro detoxification methods include intestinal absorption, blood absorption and lymph absorption, hemodialysis, plasma exchange, ultraviolet irradiation, and indirect electrochemical oxidation of blood and plasma.
5. Systemic support therapy can be used to enhance the resistance of the wounded, keep the balance of liquid, electrolytes and acid-base, correct metabolic disorders, supplement nutrition, etc., and when conditions permit, immunomodulatory therapy shall be implemented as appropriate.

3.2 Post-traumatic Tetanus

Tetanus has been considered to be associated with previous wars since ancient times. Tetanus occurs six to seven times in every 10,000 trauma. According to the statistics of the World Health Organization (WHO), more than 160,000 people die from tetanus every year. The mortality among young people is 25–50%, while that of the elderly is as high as 70–80%. Due to the development of urbanized society, coupled with a large number of casualties caused by natural disasters and man-made accidents, the number of trauma cases is increasing in peacetime. Therefore, tetanus is not simply a war problem.

Tetanus is a special type of wound infection. Tetanus bacilli may invade the open wound of human body, proliferate, and secrete toxin, resulting in a series of clinical symp-

toms and signs. It has few local symptoms, but the central nervous system is seriously affected, manifested in worsening tonic spasm, hypoxia, and cardiopulmonary dysfunction.

3.2.1 Etiology and Pathogenesis

Tetanus bacillus is a gram-positive anaerobic clostridium, which is an absolutely anaerobic, gram positive, and 3–5 μm long, with two forms of propagules and spores. Propagules which have flagella around them, can move, but cannot form capsules so that they are easy to be killed; spores are round and located at one end of the thallus, so tetanus bacillus with spores has a drumstick-shaped appearance. Spore is the survival form of bacteria in adverse environment. It has strong resistance to the outside world. Its spore is extremely stubborn. It can survive for 1 h in boiling and 150 °C dry heat. Under normal conditions, it can survive for decades.

Tetanus bacilli are widely distributed in nature. It can be found in the intestines of herbivores such as cattle, horses, and sheep and 2–30% of adults. Tetanus bacilli can also be found in the surface layer of soil and dust polluted by feces. Therefore, feces and soil are important infectious sources of the bacillus. The rate of wound infection is very high, which can reach 20–80%, but the incidence rate of tetanus is only 1–2% of the polluters. This is because after entering the traumatic tissue, tetanus bacilli can grow and reproduce in a certain hypoxic environment. That is to say, spores can be transformed into propagules and produce exotoxins to cause disease. The trauma injuries of tetanus usually come from deep stab wound, bullet wound, animal bite, open fracture, crush injury, large-area burn, serious wound pollution or mixed infection. Its common characteristics are deep wound, many necrotic tissues and heavy pollution. A few of the wounded were slightly injured and did not attract the attention of the wounded, so they need to be carefully inquired and examined. Umbilical cord infection caused by local baby delivery can cause neonatal tetanus. Unclean delivery or induced abortion can also cause maternal tetanus infection. Some of the wounded may get tetanus after surgical removal of metal foreign bodies (bullets and shrapnel) that have remained in the body for many years. In addition, cases of tetanus caused by insect bite, tooth extraction, unclean injection or surgery, skin ulcer, furuncle, otitis media, paronychia, and bedsore have also been reported. Cryptogenic tetanus is defined as the wounded without obvious wound or exact trauma history, which occurs among 10–20% of the wounded.

The symptoms and signs of tetanus are caused by the strong exotoxin produced by tetanus bacilli. There are two kinds of exotoxins, namely tetanospasmin and hemotoxin. The former can damage the nervous system, while the latter can damage red blood cells. Tetanospasmin has special affinity for the central nervous system and is the direct cause of muscle tension and spasm. The main pathogenic mechanism is that the toxin binds to gangliosides of gray matter synaptic body membrane to prevent synapses from releasing inhibitory media, resulting in the loss of control of α and γ motor nervous system, resulting in characteristic spasm and rigidity of systemic striated muscle and uncoordinated movement. In addition, tetanospasmin may also block neuromuscular junctions in the periphery and can directly act on muscles to produce muscle contraction.

3.2.2 Clinical Manifestations and Diagnosis

Tetanus can be divided into systemic and local ones. The latter is rare, accompanied by long-term tetanus on the injured limb side, which is not life-threatening. This is because the tetanus will subside by itself with the treatment of the wound, but it should be remembered that some kinds of local tetanus, such as Rose tetanus facialis and Brunner cephalotetanus, can lead to death due to laryngeal spasm. The clinical stages of systemic tetanus are as follows.

1. **The incubation phase** varies in length, most of which are 5–14 days. Some of the wounded are less than 1 day or as long as several months or even years, or they only get sick when removing the foreign bodies left for many years. The shorter the incubation period, the more acute and severe the course of disease, and the worse the prognosis. If symptoms appear within 2–3 days after injury, the mortality is very high. The incubation period of neonatal tetanus is generally 5–7 days.
2. **The prodromal phase** is mostly between 12 and 24 h, and its symptoms include general fatigue, dizziness, headache, irritability, weak chewing, local muscle tension, pulling pain, jaw stiffness, inconvenient opening of mouth, dysphagia, tension or soreness of masticatory and cervical muscles, etc.
3. **The attack phase** usually occurs 24–72 h after the initial symptoms, and the affected muscles show clonic spasm. Masticatory muscles were first involved, and the teeth closed tightly; subsequently, facial expression muscles, neck, back, abdomen, and limb muscles were involved; finally, diaphragmatic and costal muscles; due to the continuous contraction of facial muscles, a characteristic "wry smile" can be formed, and the wounded frown and quarrel shrink down; the neck is stiff, the head is tilted back, and the back and abdominal muscles contract at the same time. Because the neck back muscle is stronger than the ventral side, the trunk is twisted into an arch, combined with the spasm of the neck and limbs, forming "angular arch tension" or "lateral arch tension"; there is a "typical tetanus triad," i.e. closed teeth, dysphagia, and neck muscle rigidity. Myospasm often leads to muscle rupture. When the diaphragm is affected, it can cause respiratory disorder, aggravate cough, and may inhale vomitus by

mistake. When the diaphragm spasm is serious, it can cause respiratory arrest. Spasm can also lead to cardiovascular dysfunction, manifested as unstable pulse, blood pressure, and heart rhythm. Any slight stimulation such as light, sound, vibration, drinking water, injection, etc. can induce strong spasms. The duration of each attack varies from a few seconds to a few minutes. Muscle tension persisted during both episodes. However, whether in attack or remission, the consciousness of the wounded is always clear.

4. **The convalescent phase** lasts generally 3–4 weeks, and more than 6 weeks in severe cases. After the second week, the symptoms gradually decreased with the extension of the course of disease. For a long time after tetanus is cured, some muscle groups can still have tension and hyperreflexia.
5. **Complications**. Atelectasis and pneumonia are common complications. Pneumonia is the cause of death in 50–70% of the wounded. It is also possible to have asphyxiating crisis, a fatal respiratory arrest, in a spasm attack. The direct causes of spastic asphyxia are laryngeal spasm and spastic contraction of diaphragm. Sudden and strong muscle spasm can cause muscle tear, bleeding, fracture dislocation, and tongue bite.

Tetanus symptoms are typical, which are not difficult to make a diagnosis. If there is a history of trauma and post-traumatic muscle tension, closed teeth, stiff neck, paroxysmal generalized muscle spasm, the possibility of this disease should be considered. When there are only some prodromal symptoms in the early stage, the diagnosis is difficult, and we should pay close attention to the changes of the disease.

Clinical manifestations of some diseases are often similar to tetanus, which should be differentiated. Temporomandibular arthritis, tonsil or posterior pharyngeal wall abscess, tooth, and gingival lesions can cause mouth opening difficulty due to local swelling and pain. Diseases of spine and muscles can cause local muscle rigidity. Encephalitis often has neck rigidity and general convulsions, but the consciousness of the wounded is not clear, and the examination of cerebrospinal fluid is abnormal, which is different from tetanus. The symptoms of strychnism are similar to those of tetanus, which is called pseudotetanus. However, the characteristics of muscle relaxation during spasm interval, history of medication and disappearance of symptoms 24–48 h after drug withdrawal can be helpful for differential diagnosis. Sometimes the clinical manifestation of hysteria is very similar to that of mild tetanus. Careful dynamic observation can find the inconsistency between hysteria and tetanus. In addition, children with low calcium tetany and rabies have their own characteristics, which is not difficult to identify clinically.

3.2.3 Prevention

1. **Wound treatment**. The wound with serious trauma and pollution must go through radical debridement. It can be washed repeatedly with 3% hydrogen peroxide solution and metronidazole solution to remove all necrotic and inactive tissues, remove foreign bodies, and open up the wound. Small and deep wounds should be fully expanded and drained.
2. **Active immunization** is an effective method to prevent tetanus. Tetanus vaccine is a toxoid produced by tetanus bacilli after several generations of special culture. It produces antibodies after injection into human body, which can produce more stable immunity. Specific methods: a total of 3 times before and after injection, each time 0.5 mL. The first subcutaneous injection interval is 4–8 weeks, and then the second injection can obtain "basic immunity." The third injection after half a year to 1 year can obtain more stable immunity. This immunity can be maintained for more than 10 years, and sufficient immunity can be maintained if an additional injection (0.5 mL) is given in the next 5 years. For those who have obtained "basic immunity," 0.5 mL tetanus toxoid should be injected after injury to prolong the time limit of active immunity.
3. **Passive immunization**. For those who did not receive active immunization before injury, joint immunization measures should be taken as soon as possible after injury. In addition to tetanus toxoid, 1500–3000 U of tetanus antitoxin (TAT) should be injected subcutaneously as soon as possible. After injection, the antibody titer in blood can increase rapidly, but it can only last for about 10 days. Due to the long incubation period of tetanus, the patients with deep trauma and serious pollution can be injected again after 1 week. Tetanus antitoxin is a horse serum preparation, which is prone to allergic reaction. Intradermal sensitivity test must be performed routinely before injection. If positive, desensitization method shall be used for injection.

3.2.4 Treatment

The treatment principle of tetanus is to control spasm; keep respiratory tract unobstructed to prevent asphyxia; neutralize free toxins as soon as possible; prevention of complications.

1. **Controlling and relieving muscle spasm** is the central link of treatment. The wounded shall be isolated in a quiet dark room to reduce the stimulation of sound and light. According to the condition, the following sedative and antispasmodic drugs can be used to reduce and control the occurrence of spasm:
   (a) **Diazepam** is suitable for those with mild symptoms. It has the advantages of rapid action, no interference with respiration and circulation, no obvious toxic and

side effects, large dosage range and safety. It is recognized as the first choice of sedative and antispasmodic drugs at present. Usually 10 mg intramuscular injection or intravenous drip, four to six times a day.

(b) **Chlorpromazine** has a good sedative and hypnotic effect. The common dose for adults is 50 mg intramuscular injection or intravenous drip, once every 6–8 h.

(c) **Chloral hydrate**: chloral hydrate 10 mL or 30 mL retention enema can be used. It is suitable for the combined application of sedative and antispasmodic agents in patients with mild or serious symptoms.

(d) **Hibernation therapy**: it is suitable for the wounded with severe spasm, especially those with high fever. It is usually injected with half amount of hibernation I (chlorpromazine 25 mg, promethazine 25 mg, and pethidine 50 mg) intramuscularly every 6–8 h, which can effectively reduce muscle rigidity and muscle spasm.

(e) **Thiopental sodium**: it is suitable for the wounded with severe spasm and convulsion. Intravenous therapy of thiopental sodium 0.1 g can quickly relieve spasm. The disadvantage is that it makes the wounded unconscious and has respiratory inhibition.

(f) **Muscle relaxants**: they have a good relaxation effect on the bones of the whole body and paralyze the respiratory muscles. Therefore, it can only be used under the condition that there is a ventilator to control breathing. It is only used for the wounded with severe symptoms and frequent respiratory spasm. Commonly used drugs include l-tubocurarine, succinylcholine chloride, imbretil, galaiodonium, hanjisong, etc.

2. **Keep the respiratory tract unobstructed and prevent asphyxia**. The wounded with severe tetanus should be given tracheotomy as soon as possible. On the one hand, tracheotomy can prevent asphyxia caused by laryngeal spasm, on the other hand, it can also prepare for the application of muscle relaxants and ventilator in case of respiratory spasm, so as not to be overwhelmed in case of asphyxia. The commonly used drug is thiopental sodium 0.5 g dissolved in 20 mL glucose solution, intravenous therapy of 2–4 mL, which can immediately relieve respiratory spasm, supplemented by short-term artificial respiration, and the wounded can resume spontaneous breathing. If respiratory spasm occurs frequently, dissolve the required sodium thiopental in advance, but the storage time shall not exceed 24 h. Muscle relaxants and respirators can be used to control breathing if conditions permit. At the same time, pay attention to suck out secretions, clean the catheter, inhale atomized gas, and drip antibiotic solution regularly.

3. **Neutralize free toxins**. In principle, TAT should be a small dose, with a total amount of 50,000–100,000 IU. After debridement and high-dose penicillin injection, 100,000 IU, 70,000 IU and 50,000 IU were injected, respectively, according to the severe, medium, and light wounded. The blood concentration gradually increased 6 h after TAT intramuscular injection, so intravenous administration is better. However, intravenous medication cannot effectively penetrate the blood–brain barrier, often combined with subarachnoid injection (intrathecal injection). Intrathecal injection has the advantages of fast control of convulsion, short course of treatment, and less medication. Generally, 5000–10,000 IU of TAT is used. In order to avoid nerve damage and inflammatory reaction caused by a small amount of toluene and phenol in TAT preparation, cerebrospinal fluid can be diluted and adrenal cortical hormone can be added during injection. Human tetanus immunoglobulin (TIG) can be used if conditions permit. It has been popularized in overseas countries, but it is rarely used in China due to drug source. Its curative effect is much better than that of TAT, and there is no risk of allergic reaction. It is not suitable for intravenous therapy, because it can cause elevated blood pressure. By deep intramuscular injection of 3000–6000 IU, the effective antibody titer can be maintained for 8–12 weeks, so it only needs to be used once.

4. **Prevention of complications**

(a) Pulmonary infection. Due to respiratory spasm, difficulty in expectoration, application of sedatives and long-term bed rest, tetanus wounded are often complicated with pulmonary infection and even respiratory failure. The key points of controlling pulmonary infection are effective application of antibiotics, strengthening nursing after tracheotomy, oxygen administration through tracheotomy, sputum suction, atomization inhalation or drug dropping.

(b) Heart damage. Long-term sympathetic hyperfunction and myocardial damage caused by hemolytic toxin can lead to cardiac failure. Therefore, the wounded with tachycardia and high blood pressure can be given propranolol orally or intravenously. Occasionally, it can be seen that the wounded in the recovery period suddenly have heart failure or even sudden death after getting out of bed. Therefore, the monitoring of the cardiac function of the wounded should not be ignored even in the recovery period.

(c) Malnutrition and disorder of water electrolyte balance. The wounded consume a lot due to frequent muscle convulsions, massive sweating, and infection, and can't eat normally for a long time. Therefore, nutrition maintenance is very important for the wounded with tetanus. Diet should be high-calorie, high-protein, and high-vitamin. Patients with mild

illness can be fed by mouth or tube, and those with frequent convulsions can be given total parenteral nutrition support.

3.3 Post-traumatic Gas Gangrene

Gas gangrene occupies a special position in trauma infection, because it is characterized by particularly serious infection, high mortality, and high disability rate among rehabilitated patients. During the Second World War, gas gangrene accounted for about 1.5% of all the wounded, and the mortality was as high as 60%. Among the recovered patients, 50% were disabled due to amputation. Gas gangrene, also known as Clostridium myositis or muscle necrosis, is an acute specific soft tissue infection caused by Clostridium. It is often seen in severe open contusion of muscle tissue after trauma.

3.3.1 Etiology and Pathogenesis

The pathogenic bacteria of gas gangrene are a group of gram-positive *Clostridium*, mainly including *Clostridium perfringens*, *Clostridium septicum*, Clostridium malignant edema, *Clostridium perfringens*, and *Clostridium histolyticum*, but *Clostridium perfringens* is the most common and important, with biological characteristics of easy-proliferation in hypoxia and inactivated tissues. This kind of bacteria grows and reproduces all year round in the human gastrointestinal tract, bile duct, and vagina. Its outstanding feature is that it has the ability to form spores, and spores are very tolerant to environmental conditions. Therefore, they widely exist in soil and human and animal feces, which is very easy to pollute wounds. Under appropriate conditions, they can grow and reproduce locally and produce a variety of exotoxins and enzymes to damage human body. All kinds of Clostridium can secrete exotoxins, which can cause hemolysis, vascular thrombosis, kidney damage, and muscle damage. The main characteristic of Clostridium toxin is to destroy connective tissue and muscle and make it necrotic. Its biochemical structure is very complex and consists of many components, such as α toxin (lecithinase C) β toxin (hemolysin) κ toxin (collagenase), n toxin (hyaluronidase), μ toxin, plasmin, and neuraminidase, among others. Each component has a certain pathogenic effect.

3.3.2 Clinical Manifestations and Diagnosis

Trauma complicated with gas gangrene generally occurs 1–4 days after injury, but it could also be as short as 6 h.

1. **Local manifestations**. Local severe pain of the wound is the earliest symptom. In the early stage, the wounded limb is heavy. Later, due to the rapid infiltration of gas and liquid into the tissue to the increase of pressure, there is swelling and cracking like severe pain, which is ineffective with painkillers. What can be noted include edema around the wound, pale, tense and shiny skin, and marble like markings on the skin surface, as well as a lot of foul smelling serous or bloody exudates and bubbles in the wound. Crepitus can be palpable in the limbs (also known as snow holding feeling). The wound muscle is massively necrotic, brick red, and inelastic, which does not shrink or bleed during cutting, and finally turns to black carrion.
2. **Systemic manifestations** mainly include severe toxemia caused by toxins. Soon after the local symptoms appear, the wounded can appear pale lips and skin, rapid and weak pulse, indifferent expression, trance, irritability, shortness of breath, irregular heart rhythm, where body temperature and pulse are not directly proportional, and the body temperature is not high, but with rapid pulse. Later, due to the aggravation of toxemia, the body temperature can reach more than 40 °C, and then coma, severe anemia, and multiple organ failure may appear.
3. **Laboratory examination** shows a large number of gram-positive short but thick bacilli and few leukocytes in the smear of the wound exudate. The blood routine test shows that the wounded is obviously anemic, the red blood cell count decreases to $(1.0–2.0) \times 10^{12}$/L, the hemoglobin decreases by 30–40%, and the white blood cell count increases, but generally does not exceed $(12–15) \times 10^{9}$/L. The urine analysis shows hemoglobinuria. Diagnosis can be confirmed by the anaerobic culture, but it takes a long time (2–3 days), which is not helpful for early diagnosis.
4. **Diagnosis**. Early diagnosis is very important. As the disease progresses very rapidly, delaying diagnosis for 24 h is fatal. The three main bases for early diagnosis include crepitus around the wound, gram-positive bacilli noted in the smear of wound exudate, and gas accumulation shadow in muscles in the X-ray scan. Indirect immunofluorescence can also be used for early diagnosis. In diagnosis, it should be noted that clinically, interstitial pneumoconiosis is not limited to Clostridium infection and should be distinguished. Anaerobic streptococcus and fragile bacteroides can also produce gas in infected tissues. Subcutaneous emphysema, twisting, and even fascia necrosis can also occur in physical examination, but the disease develops slowly, the symptoms of pain and systemic poisoning are mild, and the prognosis is also good. *Streptococcus* and gram-negative bacilli can be found in wound exudate smear examination.

3.3.3 Treatment

1. **Surgery therapy**. Once the diagnosis is established, emergency surgery shall be performed immediately. Even if the wounded is on the verge of death, surgery should be performed immediately while rescuing shock. The key to

treatment is complete debridement and drainage, maximum resection of necrotic tissue, and incision of fascia decompression. Preoperative intravenous administration of a large number of antibiotics (penicillin + metronidazole), blood transfusion, and infusion to correct the acid-base balance. The preoperative preparation time shall be shortened as far as possible, generally no more than 30–45 min. General anesthesia (such as intravenous ketamine) is used for the operation, and tourniquet is strictly prohibited for the injured limb. The surgical method is to conduct extensive and multiple longitudinal incisions in the lesion area, and quickly removes all necrotic and bloodless tissues until the normal tissues with normal color and good bleeding. Because the scope of infection often exceeds the scope of macroscopic lesions, the whole muscle should be removed, including its starting and ending points. If the infection is limited to a certain fascia space, the affected muscles and muscle groups can be removed from the starting point to the ending point. If the muscles of the whole limb have been involved, high amputation shall be performed at the healthy part, and the stump shall be open without suture. The wound cavity was repeatedly rinsed with a large amount of 3% hydrogen peroxide solution or potassium permanganate solution (concentration of 1: 4000) to improve the anaerobic state. The wound remained open after operation, and was loosely covered with gauze soaked in hydrogen peroxide and potassium permanganate solution. It was replaced several times a day until the wound infection was controlled.

2. **Antibiotics therapy**. The high-dose penicillin and metronidazole should be used continuously after the surgery. Antibiotics have a special therapeutic effect on this kind of infection, because such infection belongs to acute diffuse infection. The cultivation of anaerobic bacteria, especially the drug sensitivity test, requires special equipment and technology, which is difficult to achieve generally, and the time is not allowed. According to the materials of most laboratories, penicillin, metronidazole or other broad-spectrum antibiotics can be selected among the existing antibiotics. The dose of penicillin should be large, more than 10 million U/day. Aminoglycoside antibiotics (such as kanamycin, gentamicin, etc.) have proved ineffective against such bacteria.
3. **Hyperbaric oxygen therapy** should be used at the earliest stage after operation, which aims to increase the oxygen content between tissues to create an environment not suitable for bacterial growth and proliferation. It can be used as an adjuvant therapy for surgery. By the hyperbaric chamber of the Third Military Medical University, 11 cases of gas gangrene (aged 21–50 years old) confirmed by bacteriology were treated with hyperbaric oxygen (while local thorough debridement and systemic use of high-dose penicillin). Methods: to inhale oxygen for 20 min at an interval of 8 h under pure oxygen at 3 atm, for three times in the first 24 h and once per 12 h for 3 days. Results: six cases showed markedly effective; four cases were significantly improved; one case showed ineffective (this case was 50 years old, admitted late and in coma). Therefore, those with proper conditions should undergo hyperbaric oxygen treatment.
4. **Other therapies**. Hydrogen peroxide solution (hydrogen peroxide) is continuously injected into the wound to increase the oxygen content between tissues. The method is to place a catheter in the depth of the wound, fix it on the edge of the wound with a wire, and connect it to the infusion bottle containing 1% hydrogen peroxide isotonic saline, drip it continuously at the rate of 8–10 drops per minute, and wet compress the wound with hydrogen peroxide gauze, so as to maintain the local aerobic environment and facilitate flow diversion. It generally lasts 3–5 days until the wound infection is controlled. In addition, systemic support therapy includes multiple small blood transfusions; maintain water, electrolyte, and acid-base balance; give three high (high calorie, high protein, and high vitamin) diet; protect the functions of heart, lung, liver, and kidney to keep the daily urine volume above 1500 mL to facilitate the excretion of toxins. Gas gangrene antiserum has poor control effect and allergic reaction, so it is not used now.
5. **Anaerobic cellulitis**. The prognosis is good with timely incision, decompression, sufficient drainage, removal of certain necrotic tissue, and antibiotic treatment. The gas dispersion range can be quite wide, but it is not necessary to cut too much according to the gas dispersion range, let alone amputate rashly.
6. **Pollution disposal**. The pollution and polluted dressing contacted by the wounded shall be collected separately or disinfected or discarded (fire burning). Those disinfected with spore bacteria should be boiled for more than 1 h.

3.3.4 Prevention

Attention should be paid to the trauma prone to such infection, such as open fracture with extensive muscle injury or crush injury of thigh and hip; patients with important vascular injury or secondary vascular embolism; patients with a history of suture with tourniquet for too long, plaster too tight or incomplete early debridement. The key to prevention is complete debridement as soon as possible; including removing the inactivated and ischemic tissues, completely removing foreign bodies, especially non-metallic foreign bodies, and fully opening the drainage for deep and irregular wounds to avoid the existence of dead cavity; for those with increased tension under fascia, fasciotomy, and tension reduction should be carried out early; for open fractures with extensive soft tissue injury, early suture should not be carried

out after debridement. In addition, it is difficult to determine the vitality of contused and crushed soft tissue in the early stage. During this period, we should closely observe it. For penetrating abdominal injury, especially colon, rectum, and perineum trauma, we should be alert to the occurrence of such infection, because these bacteria are resident bacteria in human intestinal tract. Early use of large doses of penicillin or metronidazole has its indications for the above wounded. According to the US military report, the incidence of gas gangrene was 1.5% in World War I; during World War II it dropped to 0.7%; in the Korean War it was further reduced to 0.08%; in the Vietnam War it has declined again. The main experience is early full debridement and blood circulation reconstruction, which shows that the disease focuses on prevention and can be prevented.

3.4 Invasive Streptococcal Infection

If the wound of war wound is treated improperly or delayed, it can cause invasive streptococcal infection. The initial manifestation is local cellulitis around the wound, and then it can quickly develop into systemic poisoning symptoms. The characteristics of the disease are: any part of the skin can be infected, and the lesions are not easy to be limited, spread rapidly, there is no obvious boundary between the pathological tissue and the normal tissue, and the symptoms of systemic poisoning are obvious. However, local tissues generally do not have obvious necrosis and dissolution, so there are no traces after recovery.

3.4.1 Etiology

According to whether the streptococcus is hemolytic after growing and multiplying on the blood medium and its hemolytic nature, the streptococcus can be divided into three categories: (1) α-hemolytic streptococcus: there is 1–2 mm wide grass green hemolytic rings around the colony (aka α hemolysis). This kind of streptococcus is mostly opportunistic pathogen. (2) β-hemolytic streptococci: a 2–4 mm wide, well-defined, completely transparent colorless hemolytic ring forms around the colony (aka β hemolysis). This type of bacteria is also called as hemolytic streptococcus, strong in pathogenicity, often causing a variety of diseases in humans and animals. (3) γ-streptococcus: it does not produce hemolysin, and there is no hemolytic ring around the colony, which is also known as γ or non-hemolytic streptococcus. The bacteria are not pathogenic, often present in milk and feces, and occasionally cause infections.

Invasive streptococcal infection of war wound is usually caused by beta-hemolytic streptococcus, which is widely distributed in nature. It exists in water, air, dust, feces, and the oral cavity, nasal cavity, and throat of healthy people and animals. It can be transmitted through direct contact, air droplets or wound infection through skin and mucous membrane. Aerobic or partly anaerobic bacteria, spherical or oval, 0.6–1.0 m in diameter, arranged in chains, varying in length, composed of 4–8 to 20–30 bacterial cells. It does not form spores, and it is flagellum-free, easily colored by common alkaline dyes, Gram-positive, or Gram-negative after aging cell culture or phagocytosis by neutrophils. The resistance of the bacterium is generally not strong where it can be killed at 60 °C for 30 min. It is sensitive to common disinfectants, but it can survive for several months in dry dust. It is sensitive to penicillin, erythromycin, chloramphenicol, tetracycline, and sulfonamide.

3.4.2 Pathogenesis

The pathogenicity of β-hemolytic streptococci is related to the toxins produced and their invasive enzymes, mainly including: (1) Streptolysin: there are two kinds of hemolysins, O and S. O is a protein containing –SH, which is antigenic, and S is a small molecular polypeptide with a small molecular weight, which is thus not antigenic. (2) Fever-causing exotoxin: once known as erythrotoxin or scarlet fever toxin. It is the main toxic substance of human scarlet fever, which can lead to local or generalized red rash, fever, pain, nausea, vomiting, and general malaise. (3) Hyaluronidase, aka spreading factor or invasin. It can break down the hyaluronic acid in the intercellular matrix so as to increase the invasive power of bacteria and make the germs easy to spread in tissues. (4) Streptokinase, aka streptococcal fibrinolysin. It can turn plasminogen into fibrin ferment (plasmase), and facilitate the diffusion of bacteria in tissues. The enzyme is heat resistant, and can still maintain activity at 100 °C for 50 min. (5) Streptodornase, aka streptodornase or streptococcal DNA enzyme, which can rarify the pus, and expedite the spread of bacteria. (6) Leukocidin: it can make leukocytes unable to move, become spherical, and finally swell and rupture.

Because the pathogen produces toxin and invasive enzyme, and the texture of the invaded tissue is loose, its acute suppurative inflammatory lesions expand rapidly. The lymph nodes on the lesion side are often infected and often have obvious toxemia or bacteremia. Histopathological examination showed extensive acute suppurative inflammatory changes in dermis and subcutaneous tissues, and the infiltrating cells were mainly lymphocytes and neutrophils. Skin appendages were destroyed, blood vessels and lymphatic vessels dilated or embolized, and granuloma formation was seen in the later stage. It is often accompanied by lymphadenitis, lymphangitis, gangrene, metastatic abscess, and even sepsis.

3.4.3 Clinical Manifestations

1. **Local symptoms**. Cellulitis can occur in any part of the skin, more commonly in limbs and face.
   (a) General subcutaneous cellulitis. The wounded with skin injury often have cold, fever, and systemic dis-

comfort. The affected part is swollen and painful, and the epidermis is red. It can fade slightly after finger pressure. The boundary of redness and swelling is unclear. The color of the central part is dark and the surrounding color is light. Those with shallow infection site and loose tissue have obvious swelling and diffuse pain; When the infection site is deep or the tissue is dense, the swelling is not obvious, but the pain is severe. The lymph nodes on the lesion side are often swollen and painful, for example, swollen and painful axillary lymph nodes in case of cellulitis in the forearm, and swollen and painful cervical lymph nodes in case of cellulitis in the face. When the lesion worsens and expands, the skin can blister, part of it turns brown, or bursts into pus.

(b) Acute submandibular cellulitis: between various tissues of the maxillofacial region, such as subcutaneous tissues, muscles, salivary glands, and jawbones, there are varying amounts of loose connective tissues or fats with blood vessels, nerves, lymphatic tissues, and salivary gland ducts. Physiologically, this structure has the function of buffering the tension and pressure generated by movement; anatomically, it is a potential gap, connected by adjacent gaps. When the infection invades these potential spaces, it can cause the dissolution and liquefaction of loose connective tissue, and the obvious space appears when the inflammatory products are filled. Infection can originate from contaminated wounds in the mouth or face. Local manifestations include redness, swelling, heat, and pain, which often spread downward with comparatively severe systemic responses. After the infection affects the connective tissues in the platysma muscle, laryngeal edema and compression of the trachea can occur, resulting in dyspnea and even asphyxia. Sometimes inflammation can also spread to the mediastinum, causing mediastinitis and mediastinal abscess.

2. **Systemic symptoms**. Patients with acute infection may suffer high fever, chills, headache, fatigue, and systemic discomfort. Some may be accompanied by lymphadenitis, lymphangitis, gangrene, metastatic abscess or severe sepsis.
3. **Signs**. The lesion is locally red and swollen with obvious tenderness. The local redness and swelling in the patients with deep lesions are not obvious, often only local edema and deep tenderness.
4. **Complications**. They mainly include septic shock and sepsis.

3.4.4 Diagnosis

1. Clinical manifestations and signs. Diagnosis can be made according to typical local and systemic clinical manifestations and signs.
2. **Laboratory examination**
   (a) Peripheral blood routine test
   - White blood cell count: In general infection, the white blood cell count is beyond 10×10^9/L. If the white blood cell count is above (20–30) $\times 10^9$/L, or less than 4×10^9/L, or immature leukocytes exceeds 0.1%, or toxic particles are noted, septic shock and sepsis should be closely watched.
   - White blood cell classification and count. The increase of white blood cell count is often accompanied by the increase of neutrophil ratio.

   (b) Bacteriology
   - Bacterial culture. For patients with multiple and repeated infections, the pus can be directly extracted from the abscess for bacterial culture.
   - Drug sensitivity test. The drug sensitivity test should be carried out with the bacteria culture of pus. The results can provide a scientific basis for clinical drug treatment.
3. **Radiology examination**. It is helpful to judge the type of disease in the early stage and understand the severity of local tissue damage.
   (a) B-scan ultrasonography. The local tissue structure of the focus is disordered, with uneven medium and low echo shadow in the center, edema in surrounding tissues, and unclear boundary.
   (b) X-ray. Widened mediastinum with high density can be seen when the cellulitis in the floor of mouth, jaw, and neck causes mediastinal abscess.
   (c) CT. Edema in the peripheral tissues and liquefaction in the center. The mediastinum is widened with high density, indicating mediastinal abscess.
4. **Differential diagnosis**
   (a) Erysipelas. It refers to infection caused by hemolytic streptococcus invading skin and reticular lymphatic vessels. Local manifestations include crimson patches, fade after finger pressure, mild skin edema, slightly raised edges, and clear boundaries. Infection spreads rapidly, but does not suppurate. There is little tissue necrosis and is easy to occur repeatedly. If it is found repeatedly in the lower limbs, there may be subcutaneous lymphatic obstruction.
   (b) Necrotizing fasciitis. It is often a mixed infection of aerobic and anaerobic bacteria. Necrotizing fasciitis occurs rapidly with severe systemic symptoms, and not obvious local symptoms. The infection may spread rapidly along the fascia and a large number of necrosis of fascia and subcutaneous tissue. Patients often have anemia and toxic shock. Ulcers and thin pus can be seen on the skin, and a variety of bacteria can grow in pus culture.
   (c) Aerogenic cellulitis: it occurs after skin injury, and the germs are anaerobic, such as Enterococcus, facul-

tative *E. coli*, bacteroid, facultative bacillus proteus, or *Clostridium perfringens*. The inflammation is mainly in the subcutaneous connective tissue, and does not affect the muscular layer. The initial performance is similar to general cellulitis. It is characterized by rapid expansion and can touch the subcutaneous twist. There can be odor after collapse, and the whole body condition deteriorates rapidly. CT scan shows different degrees of subcutaneous pneumatosis and deep soft tissue emphysema.

(d) Gas gangrene. It is common in severe trauma deep to muscles, accompanied by injured limbs or physical dysfunction. In the early stage, the local skin is bright, tense, and twisted, and the lesion can involve the deep part of the muscle. The secretions have some kind of fishy odor, and Gram-positive bulky bacilli can be detected on smear. The muscle is stained and necrotic, myoglobinuria may be present, and free gas between the muscles can be seen on the X-ray film.

3.4.5 Treatment

1. **Local therapy**

(a) Incision and drainage. In the early stage of general cellulitis, incision and drainage should be carried out in time to alleviate the expansion of subcutaneous inflammation and reduce skin necrosis. If the finger is cellulitis, it should be cut and decompressed early to prevent phalanx necrosis. For cellulitis at the bottom of the mouth and under the jaw, if the short-term active anti infection treatment is ineffective, it should be cut and decompressed as soon as possible to prevent laryngeal edema from compressing the trachea and causing asphyxia. Once the inflammation is limited to form an abscess, it should also be cut and drained in time. Multiple smaller incisions can be made and the wet gauze strip can be used for drainage.

The indications of incision and drainage include local swelling, jumping pain and obvious tenderness, local depression edema with wave motion, or pus extracted by puncture, septic necrotic infection, the abscess which has been punctured, but the drainage is not smooth. The purpose of incision and drainage is: to make the pus and necrotic infections drain quickly and reduce the absorption of toxins; ease local swelling, pain and tension, relieve the pressure on the airway and pharyngeal cavity, and avoid asphyxia; prevent the spread of infection to the adjacent interstitial space, as well as the skull, mediastinum and blood, and avoid serious complications; prevent the occurrence of marginal osteomyelitis.

(b) Local wet dressing therapy. Fifty percent magnesium sulfate or normal saline can be used, and then wrap the wound with 10% fish fat ointment.

(c) Physical therapy. The early application of ultraviolet and infrared rays can limit abscesses and promote inflammation to subside; after the discharge of pus, diathermy can be selected, such as ultrashort wave and microwave, which can promote local blood circulation, granulation tissue growth, and accelerate wound healing.

2. **Systemic therapy**

(a) Early administration of sufficient and efficient antibiotics. The first choice is 4.8–8 million U/day penicillin, intravenous drip; if the patients is allergic to penicillin, erythromycin 1–1.5 g/day intravenous drip can be used. Or ciprofloxacin, 0.2 g each time, twice a day, intravenous drip. Oral ofloxacin, 0.2 g each time, twice a day. Xianfeng V 6G/day intravenous drip or cephalosporins with wide antibacterial spectrum can also be used. The general course of treatment is 10–14 days, which should be maintained for a period of time after the skin lesions subside.

(b) Systemic support therapy. It is to ensure that patients have a full rest. Severe infection cases should be properly nourished with calories and proteins, appropriate amount of fresh blood or plasma input, and vitamin supplements like vitamin C and vitamin B complex.

(c) Symptomatic treatment. Drugs can be used like analgesic and antipyretic drugs, such as antipyretic analgesic tablets (APC) and somitol tablets.

(d) Anti-shock therapy. Patients with septic shock should be given active fluid replacement and volume expansion, improve microcirculation and corresponding symptomatic treatment, and pay close attention to the patient's urine volume, blood pressure, heart rate, and peripheral circulation. The effect of dopamine intravenous drip is good for patients with hypotension. During rehydration, the concentration of glucose solution should be limited to avoid covering up oliguria symptoms due to osmotic diuresis, resulting in the illusion of sufficient rehydration.

3.4.6 Prognosis

If there are no serious complications, the prognosis is comparatively good after active and standardized treatment. People with impaired immunity and diabetes have the possibility of recurrence.

3.5 Necrotizing Fasciitis

Necrotizing fasciitis, also known as "flesh-eating bacteria" infection, is an acute necrotizing soft tissue infection caused by bacterial invasion of subcutaneous tissue and fascia. This disease is rare clinically, but has an acute onset, rapid pro-

gression, high destructive power, and high mortality, and can cause severe disability. The clinical manifestation is the infection spread along the deep and superficial fascia, the formation of thrombosis in the involved blood vessels, and the necrosis of the corresponding subcutaneous tissue, skin, and fascia. It can occur in all parts of the body, mostly in the limbs, especially in the lower limbs; Followed by perineum, neck, face, abdominal wall, back and hip, etc. In severe cases, the internal tissue of the infected part is completely exposed outside the body, and the necrotic part forms a depression, just like being eaten, which is very terrible.

3.5.1 Etiology

Necrotizing fasciitis is often a mixed infection of a variety of bacteria, including Gram-positive hemolytic streptococcus, *Staphylococcus aureus*, Gram-negative bacteria and anaerobic bacteria. With the development of anaerobic culturing techniques, it has been confirmed that anaerobic bacteria are an important causative agent and necrotizing fasciitis is often the result of the synergistic action of aerobic and anaerobic bacteria.

Trauma patients with comorbidities such as diabetes, nephrosis, obesity, peripheral vascular disease, malnutrition, or long-term use of immunosuppressive agents such as corticosteroids are more likely to develop necrotizing fasciitis.

3.5.2 Pathogenesis

Necrotizing fasciitis is the result of the synergistic action of aerobic and anaerobic bacteria. After immune damage occurs in the whole body or local tissue, a variety of bacteria invade the subcutaneous tissue and fascia. Aerobic bacteria first consume the oxygen in the tissue, reduce the redox potential, and enhance the reducibility of the system. At the same time, enzymes secreted by bacteria break down the hydrogen peroxide in the tissue, creating a less aerobic environment suitable for the survival and reproduction of anaerobic bacteria. Bacterial infection spreads rapidly and widely along the fascia tissue, causing extensive inflammation, congestion and edema of the infected tissue, and then inflammatory embolism occurs in the skin and subcutaneous small vascular network. Tissue nutritional disorder leads to ischemic tunnel like necrosis and even circular necrosis of the skin. Microscopic examination showed that there were obvious inflammatory manifestations in the blood vessel wall, neutrophil infiltration in the deep dermis and fascia, fibrous embolism in the blood vessels in the affected fascia, and cellulose necrosis in the arteriovenous wall. Gram staining could find pathogens in the damaged fascia and dermis, but there was no damage to the muscle tissue. An important feature of necrotizing fasciitis is that the infection only affects the subcutaneous tissue and fascia and does not involve the muscular tissue at the site of infection.

3.5.3 Clinical Manifestations

1. **Local symptoms.** It occurs acutely, with early local signs often hidden so that the patients always ignore them, which can affect the whole limb within 24 h.
   (a) Patchy redness, swelling, and pain. In the early stage, skin can be red and swelling, which is purplish red, patchy, and painful with unclear boundary. In this case, the subcutaneous tissue is already necrotic, and lymphadenitis and lymphadenitis seldom occur because the lymphatic pathways have been rapidly destroyed. Infection can affect the whole limb within 24 h.

   Individual cases can start slowly and be latent in the early stage. The affected skin is red or white, edematous, and painful by palpation, and the lesions are poorly defined and present as diffuse cellulitis.
   (b) Pain relief and numbness of the affected area: early infection is localized with severe pain due to the stimulation by inflammatory substances and the invasion of germs. When the sensory nerve at the site of lesion is destroyed, the intense pain may be replaced by numbness or anesthesia, which is one of the characteristics of the disease.
   (c) Bloody blister. Due to the destruction of nutrient vessels and vascular embolism, the color of the skin gradually turns purple and black, and blisters or bullae containing bloody liquid appear.
   (d) Strange smelly bloody exudate. Edema can be noted in the subcutaneous fat and fascia, as well as sticky, turbid, and blackened exudate which finally becomes liquefied and necrotic. The exudate is bloody and serous with strange odor. Necrosis spreads widely and is undermined, sometimes producing subcutaneous gas. Crepitus can be heard by examination.
2. **Systemic toxic symptoms**. Early in the disease, when the local infection symptoms are still mild, the patient has severe systemic toxic symptoms such as fear of cold, hyperpyrexia, anorexia, dehydration, impaired consciousness, hypotension, anemia, and jaundice. If not treated in time, diffuse intravascular coagulation, toxic shock, and multi-organ failure may occur.

3.5.4 Diagnosis

The disproportion between the severity of local signs and systemic symptoms is the main feature of necrotizing fasciitis.

1. **Diagnostic criteria**. Fisher proposed six diagnostic criteria, which have certain reference value.
   (a) Extensive necrosis of subcutaneous superficial fascia with extensive concealed tunnels diffused into the surrounding tissues.
   (b) Moderate to severe systemic poisoning symptoms with mental changes.

(c) No muscle involved.
(d) No Clostridium found in wound and blood culture.
(e) No significant vascular obstruction.
(f) Debridement and histopathological examination: extensive leukocyte infiltration, focal necrosis of the fascia and adjacent tissues, and microvascular embolization are detected.

2. **Laboratory examination**
   (a) Complete blood count (CBC)
      • Determination of red blood cell counts and hemoglobin: 60–90% of the patients experience a minor to moderate decrease in red blood cell counts and hemoglobin due to inhibition of bone marrow hematopoiesis by bacterial hemotoxin and other toxins.
      • Leukocyte count: a leukemia-like reaction with elevated white blood cells mostly between (20 and 30) × 10^9/L, shifting to left in nuclear index, with toxic granules.
   (b) Serum electrolytes: hypocalcemia may be present.
   (c) Urine examination
      • Urine volume and urine specific gravity: oliguria or anuria when fluid supply is adequate, constant urine specific gravity, etc. are helpful to determine early damage to kidney function.
      • Urine protein characterization: positive urine protein indicates the presence of damage to the glomerulus and tubules.
   (d) Bacteriologic examination
      • Slide review: take the secretion and vesicular fluid from the edge of the lesion.
      • Bacterial culture. Take secretion and blister fluid for aerobic and anaerobic culture, respectively, and no Clostridium is found, which is helpful to judge the disease.
   (e) Serum antibody: the presence of streptococcal-induced antibodies in the blood (hyaluronidase and deoxyribonuclease B released by streptococci can induce the production of antibodies with high titers) is helpful for diagnosis.
   (f) Radiology
      • X-ray radiography: there is gas in the subcutaneous tissue.
      • CT: a small bubble shadow is found in the tissue.
   (g) Biopsy: Frozen section of fascia tissue is also helpful for the diagnosis of necrotizing fasciitis.
   (h) Differential diagnosis
      • Erysipelas: patchy erythema, no edema, clear boundary, and often lymph node and lymphangitis. The patient can suffer fever, but relatively mild systemic symptoms without characteristic manifestations of necrotizing fasciitis.
      • Streptococcal necrosis: Streptococcal necrosis is caused by beta-hemolytic streptococcal infection. It is manifested by skin necrosis without involving fascia. In the early stage, the local skin is red and swollen, and then becomes dark red, with blisters, containing bloody serous and bacteria. Skin necrosis is followed by a dry, burn-like eschar.
      • Bacterial synergistic necrosis: Bacterial synergistic necrosis is mainly skin necrosis, rarely involving fascia. Pathogenic bacteria include non-hemolytic streptococci, *Staphylococcus aureus*, obligatory anaerobic bacteria, bacillus proteus, enterobacteria, etc. The patient's systemic poisoning symptoms are mild, but the wound pain is severe. The center of the inflammatory area is purplish red induration, the surrounding is flushed, the central area is necrotic and forms an ulcer, the skin edge sneaks, and there are scattered small ulcers around.
      • Gas gangrene: Gas gangrene is an infection of obligate anaerobic bacteria, which often occurs under the condition of wound pollution. In the early stage, the local skin is bright, tense, and twisted, and the lesion can involve the deep part of the muscle. A smear of the secretion can detect Gram-positive bulky bacilli. Myoglobinuria may be present in stained and necrotic muscles, and free gas between the muscles may be detected by actinographema.
      • Clostridium anaerobic cellulitis: it is a severe skin tissue necrosis resulting from clostridia with extensive gas formation, mostly in areas of stained or incompletely debrided wound, especially the perianal region, abdominal wall, buttocks, and lower extremities that are easily contaminated. Its clinical manifestations are similar to those of necrotizing fasciitis, represented by the sudden appearance of red, swollen, and painful skin, which soon develops into a plaque with a black center gradually becoming gangrenous, with fever and chills, but there is also a number of anoxic gangrenes whose secretions are black and foul-smelling, often containing lipid droplets, with obvious crepitant rales around the lesion. There is much gas seen in the soft tissue via X-ray examination, but none in the patients infected with mixed anaerobic flora.
      • Fournier gangrene: It is a severe gangrene that occurs in the male penis, scrotum, perineum, and abdominal wall. It may be caused by Enterobacter, gram-positive bacteria or anaerobic bacteria infection. It is commonly seen in patients with diabetes, local trauma, incarcerated phimosis, urethral

fistula or genital surgery. The clinical manifestations include sudden redness and swelling of local skin, and many develop into dark red patches and ulcers in the center. The edge of the ulcer is latent, with serous exudation on the surface, severe tenderness, and often fever.

3.5.5 Treatment

Necrotizing fasciitis is a critical surgical emergency, which develops rapidly. Once diagnosed, extensive incision and drainage should be carried out immediately. It has been reported that the time of incision and drainage is directly related to the mortality. Necrotizing fasciitis spreads along the fascia. Sometimes the fascia has necrosis, but the skin is normal. Therefore, the incision and debridement should not take the affected skin as the edge, but should be cut to the normal fascia. If the involved area is too large, it is necessary to make multiple incisions for decompression and repeatedly flush the incision with hydrogen peroxide solution to eliminate the anaerobic bacterial growing environment. Apply high-dose antibiotics systemically as early as possible by starting with high-dose penicillin injections or cephalosporin antibiotics. Simultaneously apply glucocorticoids for severe systemic symptoms. Reinforce supportive therapy and symptomatic (or expectant) treatment.

The treatment principle of necrotizing fasciitis include early diagnosis, debridement as soon as possible, application of a large number of effective antibiotics and systemic support treatment.

1. **Debridement and drainage**. Thorough debridement and adequate drainage are the key to successful treatment. The necrotic fascia and subcutaneous tissue should be completely removed until the tissue cannot be separated by fingers. Commonly used methods:
   (a) Remove the healthy skin from the infected area for backup: remove the necrotic tissue and clean the wound; perform free skin grafting to cover the wound surface. This method can prevent a large amount of serous exudations from the wound surface and help maintain the postoperative fluid and electrolyte balance.
   (b) Make multiple longitudinal incisions on healthy skin: remove the necrotic fascia and adipose tissue, flush the wound with 3% hydrogen peroxide solution, metronidazole solution, 0.5–1.5% potassium permanganate solution, etc. to create an environment unfavorable to the growth of anaerobic bacteria; then, apply a gauze soaked with antibiotic solution (i.e. metronidazole, gentamicin) as wet dressing, and change the dressing once every 4–6 h. When changing the dressing, probe for the separation of skin, subcutaneous tissue, and deep fascia to determine whether further expansion of drainage is needed.
   (c) Pick a date for skin grafting: when the skin defect is large in area and difficult to heal on its own, pick a date for dermepenthesis after the inflammation has subsided.

   Attention should be paid to the protection of healthy fascia during operation, which is easy to cause the spread of infection after injury. Metronidazole local wet compress can delay skin growth and should not be used for a long time.
2. **Antibiotics**. Necrotizing fasciitis is a mixed infection of a variety of bacteria (various aerobic and anaerobic bacteria). Systemic poisoning symptoms appear early and the condition is serious. Antibiotics should be used in combination. Metronidazole is highly effective against bacteroides fragilis, which can be controlled by application of clindamycin at the same time; aminoglycosides (e.g. gentamicin, tobramycin, amikacin), can control enterobacter spp.; ampicillin is sensitive to enterococci and anaerobic peptostreptococci; cephalosporins such as cefotaxime and ceftriaxone have a broad antimicrobial spectrum and are effective for both aerobic and anaerobic bacteria.
3. Supportive therapy. Actively correct water and electrolyte disorders. In terms of anemia and hypoproteinemia, transfuse fresh blood, albumin or plasma; ensure adequate caloric intake via nasal feeding or hyperalimentation.
4. Hyperbaric oxygen therapy. In recent years, there is a growing number of concomitant anaerobe infections among surgical infections, and hyperbaric oxygen is effective for obligatory anaerobic bacteria. It is important to note that hyperbaric oxygen therapy can never replace surgical debridement and antibiotic therapy, though it may reduce the mortality of necrotizing fasciitis and the need for additional debridement.
5. Observation of complications. Closely observe the blood pressure, pulse, and urine volume observed throughout the treatment of necrotizing fasciitis; perform examinations such as hematocrit, electrolytes, coagulation mechanism, and blood gas analysis; immediately treat cardiac and renal failure, and prevent disseminated intravascular coagulation and shock.

3.6 Post-traumatic Sepsis

Sepsis is one of the most serious surgical infections. There are more than 18 million new cases of sepsis in the world every year, and the number of patients increases at a rate of 1.5–8.0% every year. About 14,000 people die of sepsis every day. Foreign epidemiological surveys show that the mortality of sepsis is higher than that of acute myocardial infarction. More than 350,000 people die of sepsis in Europe and the USA every year, and the treatment cost is as high as

USD 25 billion. Among them, there are about 750,000 new cases and 215,000 deaths in the USA every year. Although China is short of detailed clinical epidemiological data, according to the previous research results and the current clinical information, it is estimated that there are three to four million new sepsis patients and more than one million deaths every year. In the field of field surgery, sepsis can be divided into trauma sepsis, burn sepsis, and ordinary sepsis (sepsis in daily life such as soft tissue, odontogenic, and otogenic ones). Among them, the chance of sepsis in severe trauma and burn patients are increased, and sepsis is closely related to the occurrence and development of multiple organ dysfunction syndrome (MODS). In recent years, significant progress has been made in the pathogenesis, prevention and treatment of sepsis, and it has become one of the important research directions in the field of trauma infection.

3.6.1 Pathogenesis

Pathogenic microorganisms and their toxins are the trigger factors of post-traumatic sepsis. There is evidence that bacterial cell wall components such as lipopolysaccharide, peptidoglycan and phosphoteichoic acid, gram-positive bacterial exotoxins such as streptolysin O, *Staphylococcus aureus* enterotoxin B and toxic shock syndrome toxin (TSST-1) can participate in the pathogenic process of sepsis. However, the occurrence and severity of sepsis depend on the reactivity of the body to a greater extent. The essence of sepsis is the individual response to inflammatory substances, which is very complex and widely involves neuroendocrine immune network, complement, coagulation, fibrinolysis, kinin system, and vascular endothelial cell system, in which the immune system and vascular endothelial cell system play a particularly important role.

The characteristic of post-traumatic sepsis is that it gradually develops from a progressive, continuous high dynamic and high metabolic state to a process of decline of visceral function, and pulmonary function is often damaged first. Visceral damage is often the result of two blows. When the body receives the first blow or primary injury (trauma, major surgery, infection, etc.), immune cells such as neutrophils, monocytes macrophages, lymphocytes, and endothelial cells are activated and in an "excited state." When there is a second blow (secondary infection, surgery, iatrogenic error or stimulation, etc.), even if the degree is not serious, it is easy to cause the immune cells and endothelial cells in the excited state to have a super reaction and release excessive humoral media, that is, the so-called amplification effect. However, these humoral mediators are only the primary products of body reaction. When the target cells are activated, they can also produce "secondary," "tertiary," and even more secondary products, namely waterfall effect. The mediators involved in inflammatory response can be broadly divided into two categories: (i) directly cytotoxic: lysosomal enzymes, elastase, myeloperoxidase, cationic proteins, and oxygen radicals, which can directly kill target cells. (ii) cytokines: tumor necrosis factor(ii) (TNFα), interleukin 1 (IL-1), interleukin 6 (IL-6), interleukin 8 (IL-8), interferon-α (IFN-γ), platelet activating factor (PAF), granulocyte-macrophage colony-stimulating factor (GM-CSF), arachidonic acid metabolites, etc. The above humoral media can have adverse effects on the body. The main manifestations are: high dynamic circulatory state of "high excretion and low resistance," myocardial inhibition, endothelial injury and increased vascular permeability, blood hypercoagulability and microthrombosis, mandatory and "autophagy" hypermetabolism. These changes will eventually lead to the impairment of multiple organ function. In addition to direct cell damage, it is mainly ischemic damage. Especially when the heart and lung function are damaged, it will accelerate and aggravate the damage process of various organs of the body.

3.6.2 Diagnosis

The third international consensus on the definition of sepsis and septic shock published in the Journal of the American Medical Association JAMA in February 2016 divides the types of sepsis into two categories: sepsis and septic shock. This is based on the further understanding of sepsis pathology and the results of clinical big data analysis, and forms a new diagnostic standard.

1. **Diagnosis of sepsis.** The latest definition of sepsis is the life-threatening organ dysfunction caused by the maladjusted host response to infection. It not only emphasizes the importance of unsteady host response caused by infection (which has exceeded the possible lethality of direct infection itself), but also emphasizes the necessity of timely diagnosis.

   Although the international consensus in 2001 proposed extended diagnostic criteria and could indicate the existence of inflammation or organ dysfunction through bedside and routine laboratory tests, there is still no clinical method to reflect the concept of dysregulated host response.

   Critical medicine research shows that the prognosis of critically ill patients is closely related to the number of failed organs and the degree of functional failure, and the evaluation of organ function of critically ill patients is helpful to evaluate their prognosis. Recent big data clinical studies show that, the effect of using SOFA system to predict the risk of death (the area under the working curve of the subject is 0.74, 95% CI: 0.73–0.76) is better than that of SIRS standard (the area under the working curve of the subject is 0.64, 95% CI: 0.62–0.66); the overall mortality risk of infected patients with SOFA score of more than 2 is 10%, which is higher than that of myocar-

dial infarction patients with ST elevation (8.1%); the death risk of infected patients with SOFA score above 2 is 2–25 times higher than that below 2. The above results suggest that the clinical criteria involved in SOFA score are more suitable for the judgment of sepsis in infected patients, that is, on the basis that the basic sofa value is assumed to be 0, SOFA ≥2 indicates organ disorder. This can become the diagnostic criteria for determining patients' sepsis and be included in the third international consensus on the definition of sepsis and septic shock.

Practice has proved that sofa and its derived quick SOFA (qSOFA) system are conducive to the screening of sepsis. A clinical model determined that two of the following three items met, which was similar to the complete SOFA score: Glasgow score less than 13; Systolic blood pressure is less than 100 mmHg (1 mmHg = 0.133 kpa); respiratory rate is more than 22 times/min. This model has been verified in out of hospital, emergency and ward (data outside the USA and non--U.S.). For patients suspected of infection in ICU, the SOFA score is better than this model, and can well reflect the correction effect of intervention measures (such as vasopressor, sedative, mechanical ventilation). Increasing blood lactate measurement does not improve the prediction effect, but it can help identify patients at moderate risk. This new measure, qSOFA, can provide a simple and rapid bedside standard for judging the possible poor prognosis of adult infected patients. Although qSOFA in ICU is not as effective as SOFA score, it does not need experimental examination and can be evaluated quickly and repeatedly. Therefore, qSOFA standard is used for clinicians to timely identify and further investigate possible organ dysfunction, start or upgrade treatment, consider intensive care treatment or increase the frequency of monitoring. In addition, patients who have not considered infection but meet the positive qSOFA criteria should pay attention to the possibility of infection.

2. **Diagnosis of septic shock**. The latest definition of septic shock is a form of sepsis, characterized by obvious abnormalities of circulation and cell metabolism, which significantly increases the mortality. This definition emphasizes the difference between septic shock and simple cardiovascular dysfunction and the importance of abnormal cell metabolism. Compared with simple sepsis, septic shock is more serious and has a higher risk of death.

   According to the Delphi consensus process and the test results of actual patients, the variables of septic shock were determined as: hypotension, elevated blood lactate, and continuous use of vasopressor. The combination of hypotension and high lactic acid can reflect cell damage and cardiovascular dysfunction, which has been accepted by most experts (72.2%). In other words, if sepsis patients still have continuous hypotension after full fluid resuscitation, and need to use vasopressors to maintain the average arterial pressure above 65 mmHg and blood lactate above 2 mmol/L, they meet the diagnostic criteria of sepsis shock, and their clinical mortality exceeds 40%. Therefore, sepsis and septic shock can be diagnosed hierarchically by using SOFA score and three variable indexes of septic shock.

3.6.3 Prevention and Treatment

According to the methods recommended in the current international guidelines for sepsis related treatment, the treatment measures for sepsis mainly include early infection control and organ support treatment, and put forward many precautions such as "no basis for goal orientation, no recommendation for early goal directed therapy (EGDT), targeted blood glucose control and many hazards of deep sedation," and suggested the direction of molecular targeted treatment.

1. **Early goal directed therapy**. Active early resuscitation treatment should be used for sepsis and septic shock, and even advance the resuscitation phase to when the wounded is in the emergency department. The goal of resuscitation is not only to make the common indicators such as central venous pressure, blood pressure, and urine volume basically full, but also to make the mixed venous oxygen saturation ≥70%. Therefore, infusion, vasoactive drugs, and blood transfusion can be used to achieve the goal.
2. **Low tidal volume ventilation**. The ventilation concept of ARDS and acute lung injury has changed from normalizing the blood gas indicators to normalizing the blood gas indicators plus protecting the lungs. Low tidal volume ventilation can avoid excessive expansion of damaged lungs, so as to reduce "secondary lung injury." The results show that the ventilation strategy of AVC mode, TV 6 mL/kg, and platform pressure ≤30 cmH_2O has better prognosis than classical ventilation (TV 12 mL/kg; platform pressure <50 cmH_2O).
3. **Medium dose glucocorticoid**. The valuable literature report has completely denied the high-dose and short-term glucocorticoid strategy (hydrocortisone 30 mg/kg, 1–2 days), and advocated the use of medium-dose and long-term treatment scheme (50 mg/kg, once every 6 h for 7 days).
4. **Blood glucose control**. Hyperglycemia in sepsis is not a simple adaptive response or reduced receptor affinity. There is evidence of islets β. The function is damaged, so it is reasonable and necessary to give insulin. Hyperglycemia inhibits immune function and leads to increased susceptibility to infection. Therefore, it is of great clinical significance to control hyperglycemia.
5. **Proper antibiotics application**. Although only half of the blood cultures of sepsis are positive, infection is still

an important factor. Therefore, antibiotics should be used prophylactically.

6. **Anticoagulant therapy**. Low molecular weight heparin, urokinase, antithrombin III, etc., can be used.
7. **Plasma exchange**. It can remove both endotoxin and cytokines. If conditions permit, the hospital can use plasma exchange to treat the wounded with severe sepsis.
8. **Remove the infected foci**. Debridement and drainage must be carried out in time for the definite infection focus.
9. **Traditional Chinese medicine application**. The application of Xuebijing injection has been successfully developed in China, which can antagonize both endotoxin and the uncontrolled release of TNF alpha. The combination of antibiotics and Xuebijing can play the role of "simultaneous treatment of bacteria/endotoxin/inflammatory mediators."

3.6.4 Early Warning

The following indicators are mainly used to predict the development of post-traumatic sepsis: (1) epidemiological information of the injured: e.g. age, sex, race, injury severity, mechanism/site/number of injuries, and physiological score; (2) biochemical and immunological indicators: lactate clearance rate, procalcitonin (PCT), IL-6, IL-18, neopterin, Gc globulin, N-terminal pro C-type natriuretic peptide (NT-proCNP), kynurenine/tryptophan ratio, human leukocyte antigen DR of monocytes (HLA-DR), etc. Clinical studies show that only PCT among the above indicators can be used to identify sepsis and non-infectious SIRS patients, and guide the use of antibiotics (shorten the use time of empirical antibiotics), which has been included in the early warning and auxiliary diagnostic indicators of sepsis.

At present, the following indicators are mainly used to predict the adverse outcome of sepsis: (1) routine clinical indicators; (2) scoring system; (3) acute phase reactive protein; (4) cytokines and adhesion molecules; (5) immune function; (6) vasoactive and immunomodulatory neuropeptides; (7) coagulation system indicators; (8) myocardial damage markers, etc. It is very difficult to accurately predict the occurrence and outcome of sepsis due to the different causes and backgrounds of patients (such as age, basic disease or basic state). In terms of infection/sepsis caused by trauma, there are few predictive studies. Some scholars have found that the relative risk of postoperative sepsis in children with penetrating abdominal injury was >2, including age >10 years old, gunshot injury, number of abdominal organs >2, colon injury, injury severity score (ISS) >15, penetrating abdominal trauma index Other data showed that the SIRS score of trauma patients from 3 to 7 days was a predictor of nosocomial infection and length of hospital stay, and the continuous SIRS until the seventh day would significantly increase the risk of death (relative risk was 4.7). For patients with blunt trauma, early detection of serum C-reactive protein (CRP) and IL-6 levels is not helpful for the diagnosis of sepsis. Some literature have confirmed that the migration rate of polymorphonuclear leukocytes in peripheral blood of patients with multiple injuries decreased, and the positive predictive value, negative predictive value, sensitivity, specificity and likelihood ratio of predicting infection were 0.72, 0.93, 0.88, 0.82, and 5.0, respectively. It is suggested that peripheral blood polymorphonuclear leukocyte migration rate is a highly sensitive predictor of infection, and its early determination will guide active anti-infective treatment. Hyperglycemia is related to the high mortality of trauma patients. The incidence of infection complications such as pneumonia, urinary tract infection, wound infection, and bacteremia in hyperglycemia group is also significantly higher. Multiple logistic regression analysis showed that under the control of age, gender and ISS, hyperglycemia is an independent predictor of death, hospital stay, ICU stay and infection complications. Recent studies have confirmed that the increase of plasma procalcitonin (PCT) level in patients with severe trauma indicates an increased risk of sepsis. Although the level of PCT in trauma patients is better than IL-6 in predicting post-traumatic sepsis, if both levels increase in the early stage of trauma, it indicates that the risk of developing post-traumatic mods increases sharply.

Due to the limited biomarkers for early warning and diagnosis of sepsis, the use of genomics, proteomics, metabolomics, and bioinformatics to find biomarkers in the early stage of sepsis is gradually gaining attention. Some scholars have found that the changes of protein expression profiles in liver and heart of septic rats are related to energy metabolism. Some scholars have tried to detect the changes of serum proteomics and whole blood cell gene expression profile in patients with sepsis. Unfortunately, the research of this technique in the field of traumatic sepsis has not been reported.

By predicting the occurrence of post-traumatic sepsis, trauma patients can be divided into high-risk group and low-risk group, If early intervention treatment measures can be implemented for patients in the high-risk group of sepsis (this measure is undoubtedly more advanced than the treatment measures after being diagnosed with sepsis), it can effectively curb the development process of sepsis in the "embryonic" state, so as to reduce the incidence of sepsis. Patients with sepsis can also be divided into high-risk group and low-risk group by predicting their adverse outcomes (such as MODS and death). If strict monitoring and treatment measures can be implemented for patients in high-risk group, the prognosis of patients with sepsis can be effectively improved and the mortality of sepsis can be finally reduced. Pusajo et al. calculated the abdominal reoperation predictive index (ARPI) of the wounded accompanied with sepsis after the surgery, including the following eight parameters affecting the outcome of patients with sepsis, that is, the

first operation as an emergency operation (3 points), respiratory failure (2 points), renal failure(2 points), intestinal obstruction 72 h after operation (4 points), abdominal pain 48 h after operation (5 points), wound infection (8 points), consciousness change (2 points), and symptoms that occur 4 days after the surgery (6 points). The integral value of the index is accumulated after scoring each patient. Patients with an index of 1–10 should be observed conservatively. If the symptoms persist, further laboratory and imaging examinations should be performed. For the positive results of the examination, the second operation shall be performed (for the negative results, the observation shall be continued); if the index is 11–15, the laboratory and imaging examination shall be performed immediately, and for the positive results, the second operation shall be performed (for negative results, continue to observe. If the symptoms persist during the observation period, perform the second operation); if the index is 16 or above, perform the second operation immediately. The ARPI is based on the parameters affecting the outcome of sepsis patients after abdominal surgery. After clinical application, it not only reduces the interval between two operations and ICU stay time, but also reduces the mortality of reoperation patients from 67% to 45%.

The author recently proposed the sepsis predictive score post-trauma (SPSPT) for the first time by analyzing the data of nearly 3000 trauma cases in multiple centers in China. According to the injury severity score (ISS), the LD50 of ISS (the ISS value that causes half of the deaths in a certain age group), the wound surface/track initial contamination degree, and the systemic inflammatory response syndrome (SIRS) score, the clinical values are described below: (i) SPSPT < 4.25 indicates a low probability of sepsis, routine treatment is recommended; (ii) SPSPT ≥ 4.25 indicates a high probability of sepsis, and early intervention is recommended; (iii) SPSPT ≥ 6.45 indicates an extremely high probability of sepsis and a high mortality, suggesting early and aggressive definitive intervention. The SPSPT values (4.25 and 6.45, respectively) were accurate (84.6% and 82.1%, respectively) in predicting the occurrence of sepsis and the death from sepsis among trauma patients during hospitalization. The scoring system is simple and practical. It can be calculated according to the blood routine test results and relevant simple scores. There is no need for complex biochemical index detection. It is suitable for the early warning of trauma sepsis in grass-roots hospitals, but its accuracy still needs to be verified by prospective multi-center and large sample size.

4 Principles of Antibiotics

Wounds are contaminated. A wound bacteriology survey conducted by the Research Institute of Field Surgery of the Third Military Medical University showed that a few hours after injury, the early wound bacteria before debridement were complex, and 29 kinds of aerobic bacteria and 16 kinds of anaerobic bacteria could be detected. These bacteria were the same as those in the soil of the combat area. After debridement, the number of bacterial species decreased, but the positive rate was still as high as 66.7–75%. According to statistics, the infection rate of soft tissue trauma is about 12%, that of colon injury is about 8%, and that of multiple injuries with open femoral fractures is about 90%. Infection depends not only on the wound, but also on the type of wound. For example, the infection rate of colon firearm injury can reach 58%.

The basic principles of wound infection prevention and treatment are still active treatment of wounds, rational application of antibiotics, symptomatic and supportive treatment.

4.1 Basic Principles of Antibiotic Prophylactic Application

4.1.1 Timing of Administration

A large number of studies have shown that 6 h after wound contamination is the key period. With the passage of time after injury, the number of bacteria increases exponentially. Therefore, the most effective measure to resist infection is early debridement 6–8 h after injury. Although the application of antibiotics cannot replace debridement, a considerable part of the wounded may delay the initial surgical treatment due to the poor environment and the limitations of transportation tools. Therefore, early anti infection treatment is very important. Early application of antibiotics can prevent bacterial growth and invasion to deep tissues. A retrospective analysis of penetrating abdominal injury found that the incidence of postoperative infection complications was 7% in those who used antibiotics before operation, while 33% and 30% in those who used antibiotics during and after operation. A meta-analysis showed that 669 of the 1241 patients with thoracic trauma who had placed thoracic duct were using prophylactic antibiotics, 572 patients were not used, and incidence rate of empyema was 2.1% and 6.8%, respectively. Meta-analysis showed that the risk of empyema after using these prophylactic antibiotics in these patients was about three times lower than that of non-users. The above experimental studies have proved the superiority of early use of antibiotics after trauma, that is, to reduce the risk of infection complications.

As for the specific time of prophylactic use of antibiotics, some scholars have confirmed through animal experiments that the application of antibiotics 1 h after injury can control the growth of bacteria within 12 h after injury, so as to win time for debridement. It was also reported that 90% of the wounds were not infected when penicillin was given to experimental animals contaminated with *Staphylococcus*

aureus 2 h after injury. Some scholars recommend that a single dose of oral, intravenous or intramuscular antibiotics should be given as soon as possible within 3 h after trauma. However, the preventive use of antibiotics for contaminated wounds has no obvious therapeutic value 3 h after injury. The reason is that the extravasated fibrin at the wound can wrap around the invading bacteria and form a barrier that antibiotics cannot cross. At present, most scholars believe that the "golden time" of preventive medication is within 3 h after injury, which is also the acute reaction period of the body. The local hyperemia reaction is conducive to the diffusion of drugs and play its bacteriostatic or bactericidal role. Delayed use, such as the use of antibiotics after 6 h after injury, will significantly increase the risk of infection.

4.1.2 Antibiotic Selection

The types of prophylactic use of antibiotics after trauma are not the more the better, but should be determined according to the situation. A study showed that there was no difference in the incidence of sepsis, organ failure, length of hospital stay and mortality between the use of single antibiotics (time <24 h) and multiple antibiotics (time >24 h).

After trauma, especially war injury, it is controversial whether to choose broad-spectrum antibiotics or narrow-spectrum antibiotics for preventive use of antibiotics. The U.S. military recommends the use of broad-spectrum antibiotics, especially when surgical treatment cannot be accepted in an emergency. In October 1993, the US military launched the largest urban ground attack after the Vietnam War in Mogadishu, the capital of Somalia. In December 1998, the United States Special Operations Command convened relevant experts and personnel to summarize the problems and lessons encountered in the treatment of the wounded in this urban war. In this battle, due to the delay of operation time, the incidence of infection is very high. Therefore, it is necessary to give antibiotics as soon as possible. Experts believe that cefoxitin has a wide antibacterial spectrum and good effect, so it should be the first choice; ceftriaxone is the second. Although its antibacterial spectrum is narrower and more expensive than cefoxitin, it is only needed once a day, which has great advantages in the case of extended evacuation time. The amount of antibiotics it penetrates into interstitial fluid can reach 92%, and the half disappearance time in vivo is 8 h. From 1981 to 1989, it was used in 140 units and 22,901 wounded patients. The failure rate was only 5.49%. The dosage was 2G once a day, which could prevent infection of contaminated wounds for 48 h. In addition, fluoroquinolones have a wide antibacterial spectrum and can be absorbed quickly after oral administration, so they are suitable for use under harsh conditions. In wartime, in addition to considering the antibacterial spectrum, the selection of antibiotics allocated to individual soldier kits and health worker kits should also be comprehensively considered in combination with factors such as their bactericidal ability, tissue penetration, safety, stability, convenience of use and whether they are limited by storage conditions. At present, the U.S. military distributes oral moxifloxacin to individual medicine boxes and medical kits. The antibiotic is 8-methoxyfluoroquinolones.

A British study shows that penicillin based therapy is sufficient for the prevention of limb injury infection. For the prevention of war wound infection, the British Army used relatively narrow-spectrum antibiotics, typically penicillin plus β-lactamase inhibitors. During the Falklands war, the British Army required all the wounded with open wounds to be injected with ampicillin intravenously, sulfamethazine for head penetrating injury, and gentamicin and metronidazole for abdominal injury.

The International Committee of the Red Cross recommends that penicillin be used as much as possible before hospitalization for the war wounded, because its biggest infection killer is beta-hemolytic streptococcus and Clostridium, and penicillin is still the best antibiotic for these pathogens.

Whether broad-spectrum or narrow-spectrum, antibiotics should be selected for most contaminated wound sites. The selected antibiotic antibacterial spectrum should cover bacteria that may pollute the wound (such as normal skin and intestinal flora, *Staphylococcus aureus*, *Escherichia coli*, and anaerobic bacteria in digestive tract). There are different kinds of pathogens in different parts of trauma. For example, after abdominal trauma, almost all pathogens come from the intestine, and a large part of the infection is a mixed infection caused by the cooperation of aerobic and anaerobic bacteria. Therefore, antibiotics or antibiotic compatibility that can cover both aerobic and anaerobic bacteria should be selected. Do not directly initiate preventive treatment against multidrug-resistant bacteria. After all, multidrug-resistant bacteria such as *Acinetobacter baumannii*, *Pseudomonas aeruginosa* and *Klebsiella pneumococci* are not representative bacteria of the wound at the time of injury. Empirical use of vancomycin to prevent methicillin-resistant *Staphylococcus aureus* (MRSA) infection is not necessary. For some wounded, such as those with deep wounds, many necrotic tissues and heavy pollution, tetanus immunoglobulin, and tetanus toxoid should be given appropriately.

The use of "appropriate" antibiotics usually means narrowing the antibacterial spectrum or stopping antibiotics. The unrestricted use of broad-spectrum antibiotics makes the infection of multidrug-resistant bacteria and opportunistic bacteria more common. Considering the drug resistance associated with the application of broad-spectrum antibiotics, the application of narrow-spectrum antibiotics may bring

greater long-term benefits, but this needs to be confirmed by a lot of practice.

4.1.3 Dosage

It is found that insufficient or excessive use of antibiotics will lead to bacterial drug resistance, which will be complicated with various drug-resistant bacterial infections, such as ventilator-associated pneumonia, catheter-related infection, and fungal infection. Therefore, the maximum allowable dose should be used for the first time in the prophylactic use of antibiotics after trauma.

4.1.4 Treatment Course

The traditional view of prophylactic use of antibiotics after trauma is about 1 week, but the current view is that the duration of antibiotic use should be minimized, that is, the time of antibiotic administration should be reduced from 5 to 7 days to 3 days or 1 day, or even one prophylactic dose. Prospective studies have shown that prophylactic use of antibiotics for 1 day was equivalent to the traditional recommended use of antibiotics for 5 days. Long-term administration of antibiotics to prevent infection after injury does not bring benefits. Studies have shown that there is no difference in the incidence of complications between short-term ($\leq$24 h) and long-term (>24 h) prophylactic use of antibiotics in abdominal trauma. Severe trauma is not a reason to prolong the preventive use of antibiotics. The study found that the preventive use of more than one antibiotic in severe trauma for more than 24 h cannot prevent the occurrence of organ failure and sepsis, nor reduce its mortality. On the contrary, it increases the possibility of infection with drug-resistant bacteria. For war wounded, under the ideal conditions of emergency evacuation, early pre-hospital first aid and sufficient basic health conditions, antibiotics can be used in a single dose or no more than 24 h. However, under the conditions of limited medical resources, such as unsatisfactory health environment and delayed evacuation, antibiotics can be used for up to 5 days until the delayed initial suture. The course of prophylactic use of antibiotics was different in different parts of trauma, which was recommended in relevant literature.

4.1.5 Administration Route

Intravenous infusion should be the first choice for the administration of post-traumatic prophylactic antibiotics, especially in patients with hemodynamic instability, intravenous therapy of antibiotics is better than intramuscular injection. Of course, not all traumatic patients need systemic medication, and burn patients do not need systemic medication unless there is a clear systemic infection or combined with other trauma. Some scholars believe that local medication is better than systemic medication because it is less affected by metabolism in the body, and the probability of systemic allergic reaction and toxic and side effects is significantly reduced. It can also avoid the imbalance of flora caused by systemic use of antibiotics, especially is suitable for removing bacteria colonized on the skin. For example, applying antibiotic powder directly to the wound can achieve higher drug concentration level and longer duration. When dealing with the wounded in the Pearl Harbor attack, the U.S. military not only emphasized early debridement, but also stipulated the routine use of sulfonamides on the wound surface, which greatly reduced the infection rate. Since then, sulfa powder has been carried in American backpacks. In addition to vascular plug, the range of blood circulation disorder in patients with deep burn is very wide. After systemic medication, the effective dose cannot reach the local area. Switching to local medication can achieve a certain effect. Therefore, many burn wound drugs have been developed, such as silver sulfadiazine (zinc), silvadene–silver nitrate, 1% gentamicin, and so on. Some scholars recommend that patients with burns should be wet applied with silver sulfadiazine or mafenide acetate. During the Vietnam War, the USA gave antibiotics to some of the wounded who could not go through early debridement (including tetracycline or neomycin, bacitracin, polymyxin, etc.), and the wound infection rate was also significantly reduced (from 39% to 16.3%) compared with those without medication. Therefore, as long as the local medication is reasonable, it can work. For drug selection, first consider the drugs that are not prepared for systemic use, but pay attention that the concentration should not be too high and the wound should not be too large to avoid drug absorption and poisoning. In addition, abscess, intraperitoneal medication and airway atomization are also different forms of local medication. In recent years, antibiotics are made into biodegradable polymers by drug controlled release technology. Foreign countries have made some progress in the research of in the wound antibiotic sustained-release capsules or sustained-release beads. Its advantages are local high concentration, high efficiency and small side effects. The disadvantage is that local administration is difficult to reach the deep part of the wound. There is evidence that the application of antibiotic sustained-release beads is appropriate for those open fracture patients waiting to be evacuated from the theater, but it is not accurate for the patients who are evacuated to the definitive treatment institution 1–3 days after the injury. It is suggested that the combination of systemic and local medication will have a better effect.

Post-traumatic infection is caused by many factors, including the immune response of the injured body, the environment, mechanism and location of the injury. Prophylactic use of antibiotics is to reduce post-traumatic infection and its complications. Antibiotics should be selected based on practical guidelines. In case of infection, antibiotics should be selected empirically according to the local antibacterial spectrum before the results of bacterial culture and drug

sensitivity test, and unnecessary empirical broad-spectrum antibiotics should be avoided, because unreasonable preventive antibiotics will also lead to the occurrence of bacterial drug resistance. The study found that about 50% of patients had high resistance to ampicillin and sulbactam due to inappropriate empirical antibiotic treatment. In the process of diagnosis and treatment, the rational use of antibiotics needs specific analysis, corresponding treatment plans according to different environments and individual conditions, and pays attention to the drug type, course of treatment, dose, and timing of administration. In conclusion, reasonable selection and use of antibiotics can reduce the occurrence of drug side effects and bacterial resistance, shorten the length of hospital stay and save medical resources.

A recent systematic literature review by the U.S. Surgical Infection Society (SIS) on whether prophylactic antibiotic application affects the incidence of infection after open fractures has supported some important and practical conclusions: (1) early and short course of first-generation cephalosporins after injury, along with the implementation of prompt and modern treatment measures for fracture, will greatly reduce the risk of infections; (2) there is inadequate evidence to support that other usual means of treatment is effective, such as extended courses or repetitive short-course application of antibiotics, coverage of the antibiotic spectrum to Gram-negative bacilli or Clostridium spp., and topical application of antibiotics like sustained-release beads; (3) extensive randomized and blind trials are needed to prove or disprove the value of these traditional routes.

4.2 Basic Principles of Antibiotic Therapy

The confirmed bacterial infection should be comprehensively considered according to the type and severity of wound infection, the general condition of the wounded, the type of pathogenic bacteria, the sensitivity of bacteria to drugs, the permeability and effective concentration of drugs in tissues, maintenance time and side effects. Before obtaining the results of bacterial culture, clinicians need to make preliminary strain judgment and drug selection. The following information can be used as a reference for empirical medication:

4.2.1 Bacterial Strain Analysis According to the Wound Site

There are both exogenous and endogenous bacterial sources of trauma infection, and the latter has attracted more and more clinical attention in recent years. It is important for clinicians to be familiar with the resident bacteria in different parts, because the pathogenic bacteria of trauma infection are often consistent with the resident bacteria in the adjacent parts of trauma.

1. **The superficial wounds around the skin, subcutaneous tissue, mouth and nose** are often dominated by gram-positive cocci, such as *Streptococcus*, *Staphylococcus*, etc.
2. **Infection with extensive muscle damage**. In addition to gram-positive cocci, anaerobic infection should be focused on particularly.
3. **Infected fracture wounds**. In addition to *Staphylococcus*, *Proteus* infection is also quite prominent.
4. **Abdominal (especially gastrointestinal penetrating injury), perineum, perianal and thigh root injury**. The common pathogenic bacteria are intestinal flora. The intestinal flora is complex, mainly including three types: intestinal anaerobic bacteria, intestinal gram-negative bacilli and fecal streptococcus.
5. **Complex oral injury**. In addition to gram-positive cocci, anaerobic infection often occurs in.

As for the common pathogenic bacteria of intracranial and thoracic trauma infection, there is no certain law due to different reports.

4.2.2 Bacterial Strain Analysis Combined with Local Conditions

1. ***Streptococcus***. The inflammation is obvious and diffuse rapidly which is easy to form Peritraumatic cellulitis and lymphangitis. The pus is thin and sometimes bloody.
2. ***Staphylococcus***. The local suppurative reaction is severe with thick pus which is easy to form focal destruction.
3. ***E. coli* infection**. The pus can be comparatively thin. In the past, it was thought that there was fecal odor, which was actually the fault of intestinal anaerobic bacteria.
4. ***Pseudomonas aeruginosa***. The infected dressing is green, and when it coexists with necrotic tissue, it has musty smell or sewer odor.
5. ***Anaerobic* bacteria**. It varies with strains. In case of gas gangrene, due to protein decomposition and fermentation, it often has special odor such as hydrogen sulfide and ammonia, local gas production or subcutaneous emphysema and tissue corruption; Gram positive bacillus can be found by secretion smear staining. It should be noted that no matter what kind of anaerobic infection, it often grows aseptically in ordinary bacterial culture (other bacteria can grow in mixed infection).

4.2.3 Bacterial Strain Analysis Combined with Conditions

For those with rapid onset, rapid deterioration, rapid occurrence of hypothermia, low leukocyte and hypotension, and dyspnea, gram-negative bacilli and anaerobic bacteria infection are common. For those with relatively slow development, mainly presented as high fever and metastatic abscess, gram-positive cocci are common. *Candida* infection should be considered in patients with prolonged course of disease,

persistent fever and poor response to general antibiotic treatment.

4.2.4 Antibiotics Selection for Pathogenic Bacteria

As the bacterial drug sensitivity may be different in different regions and units, drug sensitivity tests should be carried out if conditions permit. Here, only the general situation of bacteria sensitive to drugs is used as a reference for drug selection.

1. ***Hemolytic streptococcus***. Penicillin is the first choice. Although it has been used clinically for more than 50 years, penicillin resistant hemolytic streptococcus is still rare. The wounded who are allergic to penicillin can choose lincomycin, erythromycin, etc.
2. ***Staphylococcus aureus***. Penicillin has strong vitality and good tissue dispersion among antibiotics, which can still be used for non-drug resistant *Staphylococcus aureus* infection. For penicillin resistant *Staphylococcus aureus* infection, semi synthetic penicillin, cephalosporin, lincomycin, clindamycin, ampicillin, erythromycin, Tylenol, etc. can be selected. Vancomycin can be used for multi-drug resistant *Staphylococcus aureus*.
3. **Intestinal gram-negative bacilli**. The most common in aerobic bacteria are *Escherichia coli*, *Klebsiella*, aerogenic bacilli, proteus, etc. The sensitivity of these bacilli to drugs is similar, such as gentamicin, amikacin, polymyxin B, the third generation cephalosporin, and Tylenol.
4. **Anaerobic bacteria**. Anaerobic bacteria that are common to see include Bacteroides, Clostridium anaerobes, anaerobic streptococcus, Clostridium, etc. The cultivation of anaerobic bacteria is difficult, and the drug sensitivity test is more difficult. The latter still lacks standard methods. If the drug sensitivity test needs to be carried out on the wounded one by one, it not only requires high conditions, but also takes time. Therefore, at present, it is more necessary to borrow the materials of some special laboratories to assist in drug selection. Almost all aminoglycoside antibiotics (such as amikacin, gentamicin, neomycin) and polymyxin B are not sensitive to anaerobic bacteria. Penicillin is mostly sensitive to the above anaerobic bacteria, with the exception of fragile bacilli. The antibacterial spectrum of lincomycin is similar to that of penicillin. It can be used when the wounded are allergic to penicillin. Among the commonly used antibiotics, chloramphenicol, clindamycin, and metronidazole are available. In recent years, many pharmaceutical departments have paid attention to the development of broad-spectrum antibiotics that can take into account both aerobic and anaerobic bacteria, such as the second and third generation cephalosporins, Tienam (imipenem/cilastatin), soproxen (Cefoperazone/sulbactam), piperacillin/tazobactam, and so on.
5. ***Pseudomonas aeruginosa***. The drugs available include gentamicin, polymyxin B, amikacin, Tylenol and the third generation cephalosporins. It was also found that some strains were quite sensitive to chloramphenicol.
6. **Fungal infection**. Amphotericin B and fluconazole can be used for fungal infection.

4.2.5 Drugs Selection According to Its Tissue Distribution

The drug sensitivity tests adopted in clinic are based on the effective inhibitory concentration in serum, which does not reflect the effective concentration of drugs in different tissues. For example, due to the blood–brain barrier, the drug concentration in cerebrospinal fluid is often significantly lower than that in serum. Besides, there are obvious differences in the ability to penetrate the blood-brain barrier of different kinds of antibiotics. For example pathogenic bacteria of intracranial infection are highly sensitive to gentamicin, kanamycin and polymyxin B in vitro, which can hardly penetrate into cerebrospinal fluid, so that they should not be used. In contrast, chloramphenicol, tetracycline, sulfadiazine, and ampicillin are better; lincomycin and cephalosporin can also be considered. In addition, there are significant differences in drug concentrations in the prostate and bile. Ampicillin is often used in clinic in case of biliary tract infection, because it is involved in the enterohepatic circulation so that if the biliary tract is not blocked, the bile concentration can reach several times of the serum concentration. Cephalosporin has a good effect on bone and soft tissue infection, which is also related to its good diffusion effect on the above tissues. Therefore, in the selection of antibiotics, in addition to the sensitive ones, the distribution of the bacteria to relevant tissues should also be considered.

5 Principles of Medical Treatment in Echelons of Trauma Infection

5.1 Medical Treatment in Echelons of War Wounds

Anti-infection strategy in the medical treatment in echelons of war injuries. The treatment rules of war injuries issued by our army in 2006 defines the medical treatment in echelons of war injuries, including five basic treatment links according to the treatment technical system, that is, battlefield (on-site) first aid, emergency treatment, early treatment, definitive treatment, and rehabilitative treatment. Article 16 of Section II (basic requirements for treatment) of Chapter II (organization of war wound treatment) stipulates that the wounded shall take anti-infective drugs from the battlefield (on-site) first aid. When conditions permit, anti-shock treatment such as fluid infusion can be taken in the battlefield (on-site) first aid. From the rescue center of

regiment and brigade level units of arms, debridement, injection of tetanus toxoid or antitoxin serum and anti-shock treatment based on fluid infusion and blood transfusion shall be carried out. In the emergency treatment stage described in Chapter III (technical scope of war wound treatment), it is stipulated to debride muscle and superficial tissue; bandage the wound as soon as possible and take oral antibiotics. If possible, intravenous antibiotics shall be given to the wounded with serious wound pollution; in the early treatment stage, it is stipulated that relatively complete debridement operation shall be carried out as well as broad-spectrum antibiotics injected intramuscularly or intravenously; tetanus toxoid and tetanus antitoxin serum shall be supplemented for the wounded who have not received tetanus automatic immunization"; in the definitive treatment, it is stipulated to continue systemic anti-infection therapy. Chapter 8 (treatment of the wounded in special environment operations) also puts forward requirements for anti-infection measures for the wounded in special operation environment. For example, for war injuries combined with seawater immersion, it is proposed to use broad-spectrum anti-infective drugs locally in the early stage. For the treatment of war wounds in mountains and forests, it is proposed to thoroughly debridement and give anti infection treatment as soon as possible. For plateau war wound (open wound), timely debridement is emphasized, and primary suture is not suitable. For war wounds in Gobi desert, debridement and washing should be carried out as soon as possible and covered with sterile dressing. For war injuries in humid and hot environment, first aid should be given on site and broad-spectrum antibiotics should be taken orally.

5.2 Management Principles for Prevention and Treatment of War Wound Infection

1. **Early debridement**. The prevention and treatment of war wound infection mainly depends on good early surgery, while antibiotics only play an auxiliary role. Debridement should be performed as soon as possible after injury, which should generally be performed within 6 h after injury, and should not exceed 72 h at the latest. Normal saline and sterile water without additives can be used as the lavage solution for flushing the wound. If sterile water cannot be obtained, the wound can be flushed with drinking water. It is not recommended to use the liquid added with antibiotics for wound lavage. In addition to the eyes, brain and spinal cord, debridement in other parts can completely remove necrotic tissue and foreign bodies. For infected wounds, necrotic tissue and foreign bodies should be removed as much as possible and drainage should be established. The wounded who undergo debridement unconditionally shall be evacuated as soon as possible with the support of continuous anti shock and anti-infection measures.
2. **Delayed suture**. After debridement, initial suture or positioning suture shall be performed in special parts such as head, face, hand and vulva. For penetrating wounds of skull, chest, abdomen, and joint cavity, the thoracic peritoneum, dura mater, and joint capsule must be sutured. After debridement of wounds in other parts, only sterile dressing shall be used for dressing or covering, and initial suture is prohibited. For the wound with clean surface, fresh and neat granulation tissues, and no purulent secretion, or redness, swelling and tenderness in the wound edge, delayed suture should be performed 4–7 days after debridement, generally. If the wound is infected or the timing of delayed suture is missed, after the infection is controlled, the wound is cleaned and the granulation tissue is healthy, and the secondary suture is generally carried out 8–14 days after debridement. When induration formed at the bottom of the granulation of the wound affects the healing, the induration tissue should be removed before suture (late secondary suture). If the larger wound cannot be sutured at one time, suture the part that can be sutured at first, and cover the rest with skin transplantation. For the wound that cannot be sutured at a later time or secondary suture, skin grafting, flap transfer with vascular pedicle or flap transplantation with anastomotic vessels can be performed as appropriate.

   In the self-defense counterattack against Vietnam, many serious suppurative infections and gas gangrene of limbs were caused by primary suture in violation of the most basic principle of delayed suture. Among the 199 wounds immediately sutured after debridement, 198 wounds (99.3%) had serious infection, including four cases of gas gangrene. After the Wenchuan earthquake on May 12, 2008, when many wounds were cleaned and sutured in the disaster area and transported to major hospitals in Chengdu, Chongqing, and other places, almost all of them were infected and had to be cleaned again.
3. **Usage of antibiotics**
   (a) Timing: the sooner the better. Even if delayed, antibiotics should be used within 3 h after injury. The longer the delay, the higher the incidence of infection.
   (b) Administration route. Intravenous infusion is the first choice, followed by intramuscular injection and oral administration.
   (c) Medication course. The International Committee of the Red Cross recommends that under the conditions of rapid evacuation, early pre-hospital first aid and adequate health infrastructure, a single dose of antibiotics is usually selected or the preventive use of antibiotics is limited to 24 h. In the case of limited medical resources and delayed evacuation, anti-

biotics are usually given for 5 days until delayed suture.

(d) Antibiotics types. The U.S. war wound infection prevention guidelines recommend the use of broad-spectrum antibiotics (such as cephalosporins) at least for those who cannot receive rapid surgical treatment. Because the use of broad-spectrum antibiotics is easy to cause subsequent drug-resistant bacterial infection, the excessive use of broad-spectrum antibiotics should be avoided. The main infection threats faced by a large number of war wounded are gas gangrene, tetanus and invasive streptococcal infection, which can be treated with relatively narrow-spectrum antibiotics. Penicillin and metronidazole are the first choices recommended by the International Committee of the Red Cross. Gentamicin should be added if there are systemic symptoms of suppurative infection.

4. **Dynamic monitoring**. The treatment of batch wounded is a continuous process, which is not interrupted by the evacuation and transfer between steps. Similarly, the continuous implementation of wound infection prevention and control measures depends on the dynamic monitoring of contaminated wounds. A large amount of evidence shows that the bacteria of wound pollution and subsequent infection evolve over time. At the moment of injury, there is a risk of multiple microbial contamination, especially Clostridium and beta-hemolytic streptococcus, followed by contamination from its own skin and gastrointestinal flora, and finally the intervention of hospital-acquired infection bacteria.

   Do not perform routine preoperative or postoperative microbial culture unless there are clear signs of clinical infection. Because the wound bacterial culture results cannot fully indicate the subsequent infection or the pathogen of infection, the bacterial culture results may mislead the use of unnecessary antibiotics or abuse of broad-spectrum antibiotics. Dynamic observation of wound is a simple and feasible method. The transparent dressing developed by foreign scholars provides convenience for wound dynamic monitoring.

5. **Comprehensive intervention**. Comprehensive anti-infection strategies include lavage, debridement, drainage, antimicrobial therapy, dressing and stabilization for fractures. Secondary interventions include controlling bleeding, reducing hyperglycemia, providing adequate oxygen, reducing blood transfusion, and avoiding hypothermia. Other measures to prevent nosocomial infection are followings. Medical staff shall perform operations in a special operating room with strict sterilization, keep good hand hygiene habits, change the dressings in the ward regularly, isolate infected patients, and make proper cleaning, disinfection, and sterilization of hospital facilities.

5.3 Problems to Be Solved

1. **Specification for the use of antibiotics**. Due to the lack of randomized controlled trials on the application of antibiotics in batch wounded, many suggestions are based on a large number of expert opinions and publications. The two main issues debated are: whether to use the broadest spectrum of antibiotics after injury, and what is the duration of prophylactic antibiotic use. This needs to be proved by large-scale, randomized and double-blind clinical trials.
2. **Early warning and intervention measures of sepsis**. If wound infection is not handled properly, it can cause sepsis, septic shock, multiple organ dysfunction syndrome (MODS), and death. Sepsis refers to systemic inflammatory response syndrome (SIRS) caused by infection. The condition is dangerous, and the incidence and mortality have been high. Although modern medical methods such as vaccines, antibiotics, and intensive care have made progress, sepsis is still the main cause of death caused by infection, and its in-hospital mortality is as high as 30–60%. The key reason is that once the pathological process of sepsis starts in the body, the "SIRS → MODS" pathological process with sequential development is difficult to be effectively contained. If we can accurately predict (not diagnose) the occurrence of "SIRS → MODS" caused by post-traumatic sepsis and implement early intervention measures, it is expected to interrupt this pathological process at an early stage. However, at present, the treatment in echelons and time effective treatment strategies for the prevention of sepsis of batch wounded have not attracted great attention in the academic community.

The infection of batch wounded in wartime has been and will still be an important problem in the field of trauma surgery. The future development trend is: on the premise of following the traditional principle of "early debridement and delayed suture," we should follow the basic law of treatment in echelons of anti-infection, advocate the concept of dynamic monitoring and comprehensive prevention and treatment, and implement the strategy of "early warning in echelons and continuous intervention," so as to effectively reduce the incidence and mortality of war wound infection of batch wounded.

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Trauma-Induced Coagulopathy

Su Liu and Liyong Chen

1 Overview

Severe trauma has become a serious issue in modern society. More than three million people die of trauma each year worldwide, as the main cause of death for people under 44 years old, where, what's more, uncontrollable bleeding is the direct or indirect cause of death in the first 48 h after trauma. Coagulation disorders occur in about 1/3 of severely injured patients when they arrive at the hospital. The injured mainly died of massive hemorrhage in the early stage, while sepsis and multiple organ failure in the late stage. Massive traumatic hemorrhage can lead to hypothermia, acidosis, and blood coagulation disorders, which will promote each other to exacerbate the condition. Therefore, for massive traumatic hemorrhage, simple treatment of blood loss is not enough, but comprehensive management on a series of pathophysiological changes in the body caused by massive traumatic hemorrhage and subsequent multiple organ dysfunction, so as to reduce the mortality of patients.

Although the concept and relevant studies of trauma-induced coagulopathy have been put forward for nearly a century, its pathogenesis still needs clarification, because of multiple factors involved, such as anemia, blood dilution, hypothermia, acidosis, hemorrhagic shock, and the severe trauma itself. About 70 years ago, difficult hemostasis was noted in patients during major surgery, cardiac arrest, severe hemorrhage, shock, burns, emergency obstetric diseases, lung surgery, massive blood or fluid infusion, and tumor metastasis, which was named variously, such as severe bleeding tendency, defibrillation syndrome, consumption coagulopathy, etc., but all can be explained as hyperfibrinolysis, according to the observation and research at that time. In 1946, MacFarlane and Biggs, who summarized the research in the field of hematology believed that although the normal function of the hemostatic system was not clear, it could be activated during shock and proposed that shock was a common feature of different forms of hemorrhage. The predictions made by MacFarlane and Biggs more than 70 years ago marked a turning point in our understanding of hemostasis that the complex and in-depth issues revealed by their insights still arouse various thinking. In the 1970s, US military surgeons discovered the phenomenon of diffuse exudative coagulopathy during the rescue of the injured in the Vietnam War; trauma surgeon Hirsch described the phenotype of exudative hemorrhage accompanied by a shock that he had observed on the battlefield in Vietnam as a fatal and unchangeable outcome; Stefanini called such a hemorrhagic disorder as diffuse intravascular coagulation with fibrinolysis. In the 1980s, it was believed that early hemorrhage and coagulation disorders were secondary to the consumption, loss, or dilution of coagulation factors and platelets, which was exacerbated by acidosis and hypothermia. In 1982, Moore's research team called this coagulation disorder a vicious hemorrhage circle, and other researchers called it together with low temperature and acidosis as the lethal triad. In 2003, Brohi et al. introduced the term acute traumatic coagulopathy (ATC); others tended to use acute coagulopathy of trauma (ACoT), acute coagulopathy of trauma/shock (ACoTS), trauma-induced coagulopathy (TIC), or early trauma-induced coagulopathy (ETIC). In order to avoid unnecessary confusion in the literature, it is necessary to make a consensus on the name and abbreviation. According to the existing research materials on the pathophysiological changes of post-traumatic coagulation disorders, TIC seems to be the appropriate choice, which was also accepted by the National Institutes of Health (NIH) in 2010.

Although there is still no exact and accepted definition of TIC at present, the more agreed view is that TIC consists of multiple coagulation disorder processes, a clinic syndrome mainly manifested as coagulation dysfunction, caused by tissue damage after severe trauma or major surgery.

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The survival of injured patients depends on the regulation of these two opposing conditions, namely early hemorrhage and late thrombosis. Massive blood transfusion is commonly used to prevent the death of severely injured patients with massive hemorrhage in the early phase of trauma, but it can also negatively affect the outcome of trauma patients. The latest research shows that changes in coagulation function in the injured patient occurred before the consumption, loss, and dilution of coagulation factors or other traditional hypothetical factors, but the mechanism was not very clear. Therefore, understanding the pathophysiological changes and pathogenesis of TIC is of great significance to the management and blood transfusion of severe post-traumatic hemorrhage, which is also crucial to confirm the diagnosis, modify therapeutic strategies, and improve patient outcomes.

2 Pathogenesis

TIC is a syndrome characterized by blood exudation on the mucosa and serosal surface of non-surgical areas. The initiation of post-traumatic hemostasis concludes physiological changes and pathological changes (Table 1). The latter include initial endogenous pathology (directly caused by the trauma itself and/or traumatic shock) and secondary external pathology.

Small blood vessels contract immediately after the trauma in the early phase when manual hemostasis methods are recommended, such as compressing the wound and ligating the blood vessels. If hemorrhage and blood exudation occur in the non-traumatic areas besides the wound, coagulation dysfunction begins. The coagulation disorder directly caused by the trauma itself and/or traumatic shock is so different from the above-mentioned causes, such as blood dilution, hypothermia, and acidosis, that, sometimes, minor trauma can also cause extensive blood oozing. Therefore, it is important to make clear the difference between traumas in physiology and pathology to understand the pathophysiology of trauma-induced coagulopathy and improve therapeutic strategies.

Table 1 Changes in post-traumatic hemostasis [1]

Physiological changes	Pathological changes
• Hemostasis and wound healing	• Early changes induced by endogenous factors – Disseminated intravascular coagulation (DIC) Activated coagulation Insufficient anticoagulation Hyperfibrinolysis (early phase) Consumptive coagulopathy – Acute coagulopathy of trauma/shock (ACoTS) Coagulation inhibition mediated by activated protein C Hyperfibrinolysis mediated by activated protein C • Exogenous secondary DIC and ACOTS – Anemic coagulopathy – Hypothermic coagulopathy – Acidosis coagulopathy – Dilutive coagulopathy – Other factors

2.1 Classic Trigger Factors

2.1.1 Hemodilution

It has been believed for years that trauma-related coagulopathy is mainly caused by acidosis, hemodilution, and hypothermia after resuscitation with crystalloids. As the guide of advanced trauma life support (ATLS), patients with hypotension at admission could be infused with 2 L of crystalloid solution. In the early stage of hemorrhage, the small amount of fibrinogen and platelets stored in the body can be quickly lost; crystalloid resuscitation can further dilute the coagulation factors, thereby reducing blood viscosity. Resuscitation of pre-hospital crystalloids in severe trauma patients can worsen coagulation dysfunction, acidosis, and hypothermia and inhibit thrombin production. In 2013, a study by Cohen's team showed that severe trauma with an INR value greater than 1.3 was an independent risk factor for coagulopathy. Maegele, who has analyzed 8724 patients in the German Trauma Registry in 2007, has found that coagulopathy occurred in over 40% of those with crystalloid infusion greater than 2000 mL, more than 50% of those with crystalloid infusion greater than 3000 mL, and over 70% of those with crystalloid infusion greater than 4000 mL. Therefore, the amount of crystal resuscitation fluid should be limited during trauma resuscitation to reduce the dilution effect.

2.1.2 Hypothermia

The core body temperature of the severely injured is often lower than 36 °C. Hypothermia could promote trauma-induced coagulopathy. Heat production could be damaged by the rapid loss of a large amount of blood and the accompanying hypoperfusion. Besides, the production of thrombin will be slowed down, and its function will be inhibited below 36 °C. Mitrophanov analyzed the effect of low temperature and blood dilution on coagulation with a computer model and found that the prothrombin time of blood at 36 °C was prolonged to three times that of the normal.

2.1.3 Acidemia and Hypoperfusion

The impact of acidemia must be considered when blood clots are formed in the body. Engström et al. conducted an in vitro experiment in which a lower pH of a normal human blood sample combined with hydrochloric acid could slow down

the speed of thrombus formation with maximum clot strength, and the clot formation time with the pH value of 6.8 was extended by 168% when compared with that when the pH value was 7.4. Acidemia is the result of both crystalloid resuscitation (hyperchloremia) and hypoperfusion (hyperlacticaemia), which are difficult to separate. In the state of shock, when the perfusion for normal cell metabolism is insufficient, in such hypoxic conditions, the lactic acid produced by the anaerobic metabolism of cells helps the recovery of metabolic acidosis when the liver functions as normal. The over-physiological chloride concentration in normal saline, which leads to hyper-chlorinated metabolic acidemia, could aggravate the existing acidemia due to hypoperfusion in the injured. Hypoperfusion is also an independent factor that affects coagulation function. Simmons et al. reported that 777 patients with war trauma in shock state (alkaline deficiency BD<−6 mEq/L or systolic blood pressure < 90 mmHg) showed an abnormal increase in INR upon admission.

With the in-depth understanding of and attention to TIC, recent studies have shown that the role of these classic triggers has been exaggerated. A study by Brohi in 2003 found that TIC appeared before resuscitation, that 1/3 of patients with multiple injuries was diagnosed with coagulation disorders by routine laboratory examinations upon admission, with a few pre-hospital fluid infusion, no hypothermia, and no consumption of coagulation factors. Subsequently, Moore and Floccard also found, respectively, that there were 29% of patients suffering from coagulation disorders at 15 min after trauma and 56% of the patients at 25 min. In 2014, MacLeod also found that about 11% of patients with early trauma-induced coagulopathy were mildly injured (ISS < 16, normal RTS score, no fluid resuscitation therapy, stable vital signs). These studies and findings question the conventional beliefs that blood dilution, hypothermia, and acidosis trigger blood coagulation disorders. Attention has been paid to the possible relationship between the endogenous induction mechanism of early post-traumatic coagulation disorders and the location and features of the trauma.

2.2 Early TIC Mechanism

Regarding the mechanism of TIC, conventional views have focused on the consumption and dilution of coagulation factors, hypothermia, and acidemia. So far, it is still considered to be a component of the pathophysiology of TIC, but it is not the primary cause of TIC. The possible mechanism of TIC has been gradually revealed, which is a hypothesis that involves multiple factors, more complex than conventional doctrines. The current hypotheses of the mechanism of TIC include (1) DIC-fibrinolysis, (2) activated protein C, (3) glycocalyx, and (4) fibrinogen-centric hypotheses. TIC is a dynamic process changing over time. These hypotheses intersect with each other so that no single hypothesis can fully explain the various manifestations of coagulation dysfunction.

2.2.1 DIC-Fibrinolysis Hypothesis

DIC-fibrinolysis hypothesis believes that bleeding tendency is secondary to hypoperfusion/shock and endothelial injury, accompanied by prolonged PT, increased thrombin production potential, low antithrombin level, consumption of coagulation factors, decreased fibrinogen, increased fibrinogen degradation products (FDP), and increased FDP/D-dimer ratio. An excessive increase in plasmin activity instead of thrombin activity is considered hyperfibrinolysis.

For the same injury, the pathology of DIC and systemic fibrinolysis sometimes coexist, so it is called DIC with fibrinolysis phenotype. In addition to the secondary fibrinolysis caused by DIC, tissue-type plasminogen activator (t-PA) is released from the Weibel–Palade body of endothelial cells due to insufficient tissue perfusion caused by trauma, leading to systemic fibrinolysis. The levels of plasminogen activator inhibitor-1 (PAI-1) are almost the same in patients with or without DIC, while the levels of t-PA and plasmin α2-plasmin inhibitor complex, as the markers of plasmin production, are significantly higher than those without DIC, because of the extreme imbalance between t-PA and PAI-1, that the time is short from t-PA releasing from endothelial cells to its concentration reaching the peak, while several hours are required for the induction and expression of PAI-1 mRNA so that hyperfibrinolysis occurs immediately after trauma and lasts several hours.

Although fibrinolysis plays an important role in TIC, Letson et al. recently found in a rat model of hemorrhagic shock that a small volume of intravenous therapy of 7.5% adenosine/sodium chloride, lidocaine, and magnesium ions can fully recover the damaged coagulation function in 5 min, reverse hemorrhage in 20 min, and reverse shock in 60 min, indicating that TIC can be reversed because the animal models with early TIC did not suffer from DIC-consumptive coagulopathy. In another prospective cohort study, Rizoli et al. found that clinical manifestations of DIC did not occur in the severely injured assessed as DIC by scores according to the pathological results within 24 h after trauma. Therefore, the author believes that DIC is rare in severe trauma, and the ISTH (International Society of Thrombosis and Hemostasis) score is not a reliable prognostic indicator of DIC. Therefore, the early TIC, which is not DIC, has a distinctive feature of diffuse intravascular fibrin deposition. Although the in vitro thrombin generation potential is enhanced and/or fibrin and fibrin degradation products are increased, which all suggest the possibility of DIC, there is no clinically significant thrombosis state in vivo.

2.2.2 Activated Protein C Hypothesis

The activated protein C hypothesis also believes that hemorrhage is secondary to hypoperfusion/shock, vascular endothelial injury, and prolonged clotting time. However, hemorrhage is mainly stopped by activated endothelial protein C receptor (EPCR), thrombomodulin (TM), and TM-thrombin complex through activating the protein C (APC) pathway. APC inhibits the production of thrombin through the proteolytic inactivation of FVa and FVIIIa (enhanced by the cofactor protein S) to inhibit coagulation and also leads to a higher concentration of t-PA by inactivating PAI-1 so as to reduce fibrinogen to enhance the production of FDP and D-dimer, which ultimately promotes hyperfibrinolysis. In the first stage, when platelets and fibrinogen are relatively sufficient, microvascular thrombosis or DIC will not occur; the later thrombotic DIC is a unique clinical manifestation, which is an exhaustion of APC, clotting factors, and microvascular clots.

In addition to antithrombotic and antiplasmin effects, APC also has many important cytoprotective effects, such as anti-inflammation, anti-apoptosis, and endothelial barrier stabilization. The clinical results of multiple research teams support the protein C hypothesis.

The activated protein C has many properties that can explain TIC, but the basic mechanism is still unclear because APC is only moderately increased in TIC patients, indicating that it seems unlikely to be the only determinant or activator. In addition, the central principle of this hypothesis is that APC induces systemic anticoagulation by inactivating FVa and FVIIIa. It is difficult to observe an increase in thrombin production in TIC patients upon admission. However, it is possible that APC is a key point in the evolution of TIC over time.

2.2.3 Glycocalyx Hypothesis

The glycocalyx degradation hypothesis puts particular emphasis on the hypoperfusion/shock of endothelial injury and the trend towards DIC-fibrinolysis and activated protein C hypotheses. This hypothesis proposes to protect the negatively charged glycocalyx mesh, 0.1 μmol/L to 1 μmol/L in thickness, on the lumen side of the endothelium from damage or shedding. It is estimated that the destruction of glycocalyx may be caused by increased proteoglycan 1, resulting in systemic spontaneous heparinization leading to an anticoagulation state, which can be reversed by heparinase in some injured patients. Glycocalyx degradation may further lead to the activation of endothelial cells and the subsequent imbalance between coagulation and inflammation, including local thrombin formation, fibrinolysis, leukocyte, and platelet dysfunction. Increasingly severe trauma and endothelial injury (increased proteoglycan 1) are manifested as prolonged APTT, enhanced sympathetic activity, exhausted protein C, increased soluble TM, hyperfibrinolysis, and inflammation.

In the early 1960s, Willoughby diagnosed a few post-catastrophic cases of postpartum hemorrhage with heparinized blood instead of de-fibrinolysis syndrome. This is still attractive even at present because this early hemorrhage may involve widespread endothelial damage, which supports the glycocalyx degradation hypothesis. The latest research indicates that endothelial damage may be reversible in a short period of time, which makes it a potentially valuable therapeutic target. However, except for the changes in proteoglycan 1, this hypothesis needs to be directly verified by intravital, electron, or confocal microscopy. The latest data from intravital microscopy support this hypothesis.

2.2.4 Fibrinogen-Centric Hypothesis

The fibrinogen-centric hypothesis is a relatively conventional hypothesis. Its roots can be traced back to the mid-1940s, but today it still attracts the attention of clinicians such as trauma, obstetrics, and cardiac surgery doctors. The focus of this hypothesis is that the loss of fibrinogen is the main driving factor of TIC, which reduces the amplitude of viscoelastic clots and increases FDP and D-dimer. Acute traumatic hypofibrinogenemia occurs when the breakdown of fibrinogen is faster than its synthesis, and the degree of fibrinogen loss depends on the severity of the trauma, shock, and the amount of infused fluid. Low fibrinogen may also change the platelet function and increase the activation of EPCR and TM-thrombin/protein C, which exacerbates the hemorrhage.

The fibrinogen-centric hypothesis is clinically relevant, especially in moderate to severe hemorrhage and shock. However, similar to the APC hypothesis, there are still various questions about how to link with the hemorrhage mechanism, such as the time of fibrinogen consumption, the role of FDP and soluble fibrin monomer, and the cause of possible defects in the cross-linking of fibrin through FXIII. The potential shortcoming of the current fibrinogen-centric hypothesis is that it ignores the number and function of platelets and erythrocytes when evaluating and treating coagulation disorders.

Although there are many hypotheses, animal models of different trauma types are still needed to further study their basic mechanisms, and prospective randomized controlled trials are urgently needed.

2.3 Others

Erythrocytes participate in hemostasis through four mechanisms, namely the rheological effect, adenosine diphosphate (ADP) release, production of eicosanoids regulated by platelets, and activation of the intrinsic coagulation pathway. Therefore, the reduction of hematocrit plays an important role in trauma-induced coagulopathy.

Red blood cells flow in the center of blood vessels, push platelets to move toward endothelial cells, increase shear force, and activate platelets. A low hematocrit value, regardless of the platelet count, can significantly reduce the platelet deposition in endothelial cells. When the platelet count is 50×10^9/L, and the hematocrit is 20%, there is almost no platelet deposited, but it can be compensated by increasing the hematocrit. Blajchman et al. have demonstrated that thrombocytopenia and low hematocrit levels can independently predict prolonged bleeding time and have believed that the formation of thrombi destroys erythrocytes and releases ADP. In addition, red blood cells participate in the production of thrombin through the exposure of procoagulant phospholipids. Red blood cells stimulate platelet to reactively induce eicosanoid production (thromboxane A2) to promote thrombosis. Factor XII or XI triggers the activation of the intrinsic coagulation pathway, leading to the stepwise activation of factors IX and X, inducing the production of thrombin. Intrinsic coagulation requires the activation of factor IX, which is activated by the erythrocyte elastase located on the erythrocyte membrane, and its activation can be exaggerated by increased hematocrit. The reaction of red blood cells on the platelet membrane plays an important role in the subsequent propagation of the coagulation waterfall. Experiments have shown that when a hematocrit reaches 33%, the bleeding time is improved, and thrombosis reappears, so it is generally believed that the hematocrit with the best hemostatic effect is 27–35%.

In short, although the basic mechanism of TIC cannot be explained well and uniquely, its main initiation is still related to tissue hypoperfusion, endothelial injury, and inflammation, various types and severity of trauma increase the complexity of its manifestations. The TM-thrombin complex and the fibrinolytic system of the endothelial cell are attractive therapeutic targets because they can act as a switch from fibrinolysis to anti-fibrinolysis. In-depth research will open up new diagnostic tools to reveal potential etiological factors and treat endothelial injury or other pathophysiological changes with new drugs. It is a challenge for early post-traumatic diagnosis and treatment to find the right balance point in the body.

3 Diagnosis

There are significant limitations in defining TIC according to laboratory standards. First, the pre-hospital emergency management of the injured is not entirely consistent. Many studies have excluded the patients who had received a large amount of crystalloid infusion before arriving at the hospital so that those with severe injuries may not be included leading to a data bias. Secondly, the laboratory equipment used to diagnose TIC does not meet the standards. Not every hospital has thromboelastography (TEG, Haemonetics, USA) or rotational thromboelastometry (ROTEM, Pentapharm, Germany). What's more, the laboratory testing of certain indicators takes longer. For example, in a study of 325 trauma patients, Davenport et al. found that the laboratory provided the electronic results of PT to clinicians for an average of 78 min. Finally, the patient's low hematocrit and the temperature of the POCT detection device may affect the accuracy of point-of-care testing (POCT). In other words, the difference between POCT and laboratory testing may lead to false-negative TIC. Nevertheless, there are still some laboratory indicators and clinical manifestations that help clinicians to judge.

3.1 Laboratory Diagnosis

3.1.1 Classical Coagulation Tests (CCT)

In 2010, Frith et al. retrospectively analyzed several major trauma centers in Europe and found that when shock and hemorrhage existed simultaneously, prothrombin time (PT) and activated partial thromboplastin time (aPTT) both increased significantly. In order to use these parameters to limit TIC functionally, they recommended the diagnostic criteria for TIC as the prothrombin time ratio (PTR, PTR = tested plasma PT value/normal reference plasma PT value) > 1.2. The patient's mortality and the need for blood transfusion were significantly increased above this threshold. The predictor, PTR, was helpful to discover 42.9% of patients with PTR > 1.2 that required a large amount of blood transfusion, with a negative predictive value of 98.4%. It was easier to find patients in need of massive blood transfusion by setting PTR >1.2 instead of 1.5 (17% more in probability).

However, the latest evidence shows that the classic coagulation test indicators, such as the international normalization (prothrombin time) ratio (INR), PT, aPTT, and fibrinogen (Fg), cannot accurately describe the complex process of acute trauma, because these classic tests are performed on platelet-poor plasma (PPP) samples so that the intensity of hemostatic clots cannot be assessed, leading to an increased false negatives incidence. These tests can provide fibrinogen content and platelet count, but their performance or function cannot be determined, as well as the condition of coagulation disorders, such as fibrinogen exhaustion, platelet dysfunction, or hyperfibrinolysis.

3.1.2 Viscoelastic Hemostatic Assays (VHA)

As the further understanding of the TIC triggering mechanism, laboratory tests of viscoelasticity have become more and more popular. Viscoelasticity tests, such as TEG or ROTEM, use whole blood and provide a result of the entire coagulation process, including information on the development, stability, and dissolution of blood clots, which reflects homeostasis.

Research by Jeger and Doran showed that there was a difference in sensitivity between the classic coagulation tests and TEG. Doran's data shows that 64% of the injured with coagulation disorders can be identified with abnormal description in the thromboelastogram in battle injuries, while the classic test (PT > 18 s) only has a 10% identification rate. Cotton et al. conducted a prospective study on 583 patients and compared the difference in time response between rapid TEG (rTEG) and classical coagulation test (CCT). Different from standard TEG, rTEG used tissue factor and kaolin added to the reagent to reduce the coagulation time by about 10 min. In Cotton's research, it was found that the results of R and K point in rTEG were displayed graphically within 5 min, and the results of MA value were visible within 15 min. There was a strong clinical correlation between activated clotting time (*R*-value in rTEG) and K-time with PT and aPTT, and a good correlation between α angle and MA with PT, aPTT, and platelet count. The activated clotting time measured by rTEG can determine whether there is a need for massive blood transfusion in the first 6 h after trauma.

By ROTEM, researchers have determined that the intensity of thrombi is weakening during TIC, which is weakened to 42% at 5 min, and such a situation continues until the blood clot is formed. Davenport studied 325 traumatic patients and found that the intensity of the clot was weakened, and the time for clot formation was shortened in ROTEM. With the threshold value of clot amplitude <35 mm within 5 min (CA5 < 35 mm), ROTEM can predict 77% of TIC, with a false-negative rate of 13%. Compared with the traditional coagulation test (PTR > 1.2), ROTEM can identify more TIC patients (18% vs. 8%). Davenport's research also shows that CA5 < 35 mm has a strong correlation with PRBC and FFP transfusion, where CA5 < 35 mm was used in 71% of the subjects to determine whether massive blood transfusion was needed, and PTR was used in 43%. Tauber has studied 334 injured patients with ISS > 15, with the results showing that ROTEM could accurately diagnose TIC and predict mortality and blood transfusion well, where the incidences of slowing down clot formation, reducing clot intensity, and impairing fibrin polymerization were 23%, 22%, and 30%, respectively. The viscoelasticity test appears to be superior to CCT in the management of TIC due to its diagnostic sensitivity and ability to guide clinical decision-making.

Although the viscoelasticity test analyzes the coagulation component dysfunction or defects through the indicators of whole blood coagulation, so that it has various advantages in guiding clinical treatment, it still has some limitations. Some parameters of standard TEG can be used in experimental research, such as functional fibrinogen (FF) to determine clot intensity, which is not routinely used in routine clinical practice. Similarly, rTEG is faster than standard TEG. In addition, the analysis software is often placed in the laboratory so that it is not convenient to provide real-time detection in the operating room, which limits its practicality in decision making in a rapidly changing environment. Finally, requires trained personnel are needed to operate the bedside TEG which is not daily examinations in the hospital, instead.

3.1.3 Platelet Function Analysis

Although conventional platelet count is a feature of post-traumatic platelet dysfunction, it has been replaced by another promising indicator, impedance aggregometry (IA), which has been validated by existing platelet function tests and bedside rapid analysis. The working principle of IA is to measure impedance through a stationary silver-coated copper wire in whole blood. If certain platelet agonists are added to whole blood, changes in impedance determine the functional activity of platelets. Commonly used agonists of platelet aggregation are collagen, adenosine diphosphate (ADP), arachidonic acid (AA), and thrombin receptor activating peptide (TRAP). The results can be obtained in about 10 min, and each test requires 300 μL of the blood sample. Research conducted by Kutcher indicates that although the platelet count was normal, the platelet function still has abnormal characteristics after trauma, and the platelet graph can reflect 45.5% of those with abnormal platelet function. Platelet graph or impedance aggregation will be a useful tool in the diagnosis of TIC in the future.

3.2 Clinical Diagnosis

The common properties of TIC-related studies have been quantified and graded through laboratory testing, and detailed laboratory data analysis of TIC is related to efficacy. The scope and investigation of coagulopathy phenotypes have been limited by the lack of a standardized clinical scoring system for coagulation disorders and criteria that can determine whether coagulation disorders affect mortality after injury. Although the clinical diagnostic criteria are still unclear, a resuscitation strategy for this clinical symptom has emerged. The definition of this clinical symptom currently relies on the subjective assessment of surgeons and the demand for blood products/transfusion. The establishment of a standard scoring system that can quantify, verify, and test the reliability of scorers for key clinical definitions is critical to clinical disease progression. NIH funded the establishment of the Trans-Agency Consortium for Trauma-Induced Coagulopathy (TACTIC), which contacted nationwide heart, lung, and blood institutes. The alliance

formed by TACTIC conducts research from large-scale multi-site clinical studies to basic mechanism laboratory investigations through the cooperative research between NIH and the Department of Defense on the issue of trauma-induced coagulopathy. In the design of a multi-center study, it is necessary to establish a scoring system that can quantify the severity of coagulopathy with criteria of significant clinical variables taken into account. The quantification of impaired coagulation and hemorrhage caused by coagulopathy rather than surgical bleeding (uncontrolled arterial or venous destruction) sometimes carries the risk of controversy and misclassification. In order to provide a balanced assessment and avoid assessing potential subjectivity, the scoring system adopts the five-point Likert scale (definitively positive, positive, possibly positive, suspected, and negative), and the observer must believe in the score of each stratification. Therefore, the following quantitative scoring system is proposed to evaluate TIC (Table 2).

One of the goals of the scoring system is to distinguish whether the injury that requires hemostasis is a mechanical trauma alone or hemorrhage with coagulopathy. Therefore, for a coagulation disorder with a score higher than I, the trauma surgeon must judge that the hemorrhage caused by severe trauma can not only be stopped by general hemostatic intervention. Besides, in order to further study coagulopathy, hemorrhage should be subdivided according to its source. The revised content includes the injury type with p for penetrating stab injury and b for blunt injury; the location of the injured should be considered when calculating the score, such as emergency department (ED), operating room (OR), or intensive care unit (ICU). Table 3 illustrates the examples of scoring different cases.

It is recommended that the attending trauma surgeon determine the evaluation score immediately or as soon as possible after the surgical hemostasis so as to obtain an initial impression of whether the patient has severe coagulopathy before resuscitation. Clinicians must make a clear distinction between bleeding from the injury site (uncontrollable surgical bleeding) and that due to coagulopathy in order to limit the assessment scores for trauma-induced coagulopathy. It is expected that the reliability of the score will increase as the injury time increases. The focus of the scoring system should be on controlling the bleeding amount after surgical hemostasis.

Table 2 Clinical scores of coagulopathy (I ~ V) [2]

Score	Description
I	Normal hemostasis. (No coagulopathy)
II III	Mild coagulopathy needs direct pressure or temporary gauze padding without other intervention required. (Coagulopathy is suspected.)
IV	Coagulopathy that is difficult to stop bleeding with direct pressure requires advanced hemostasis techniques (such as electrocautery, hemostatic agents, or sutures). (Coagulopathy is suspected.)
V	Although wound hemostasis has been performed, auxiliary blood products or systemic treatments are still needed for continuous bleeding. (Coagulopathy exists).
Sub-category	There are multiple diffuse and persistent hemorrhages far away from the trauma (such as tracheal intubation site, venous indwelling needle site, chest drainage tube, etc.) (Coagulopathy exists definitely.)
A	Isolated traumatic brain injury
B	Neck/chest/abdomen/pelvis injury
C	Extremity injury
D	Polytrauma[a]
E	Polytrauma + isolated traumatic brain injury

[a] The current consensus definition of polytrauma is a serious injury with AIS score > 3 points, two or more AIS anatomical parts, combined with one or more variables (systolic blood pressure ≤ 90 mmHg, Glasgow coma score ≤ 8 points, BE ≤ 6, age ≥ 70 years/o). The nature of the injury should be revised by an attending trauma surgeon, including blunt injury (b) or penetrating stab injury (p) after the mechanical bleeding is controlled

4 Treatment and Outcome

The two main causes of coagulopathy are surgical hemorrhage and non-surgical hemorrhage caused by trauma. Critical surgical hemorrhage is prone to induce hemorrhagic shock accompanied by extensive tissue damage. Their synergistic effects may cause DIC of the fibrinolytic phenotype. Therefore, the two main goals of TIC management are to control the hemorrhagic shock caused by severe trauma and severe hemorrhage, respectively. Early attention should be paid to the identification of high-risk factors, especially for the wounded with severe injury, severe traumatic brain injury, shock, active hemorrhage, and expected massive blood transfusion. The underlying disease should be treated actively, i.e., the trauma itself and shock caused by the trauma, because this is the main cause of systemic hyperfibrinolysis in the early stage of trauma. DIC could be controlled in the early stage of trauma, where it could be quickly reversed when hemorrhage and shock caused by trauma are controlled by surgery. Therefore, the multidisciplinary management of severe hemorrhage is the cornerstone of optimizing the treatment of injured patients.

The biggest controversy about TIC management is whether following the established protocol or the point-of-care (POC) goal-oriented treatment. The USA advocates established protocols to guide treatment, while Europe advocates POC goal-oriented to guide resuscitation. In addition, early and appropriate application of various hemostatic drugs is also advocated.

Table 3 Examples of clinical cases with TIC scoring system [2]

Clinical cases	Intervention/resuscitation	Location	Clinical scoring
1. Motorcyclists without helmets, severe traumatic brain injury; GCS: 3 points; CT: Intracranial hernia, active hemorrhage involves sites of scalp laceration, tracheal intubation, venous puncture site, and mucosa. 2. The patient was injured by a knife on a single limb with massive venous hemorrhage in the emergency department and was immediately sent to the operating room for surgery. After surgical hemostasis, there was continuous hemorrhage noted from an unknown part, which could be stopped after a few minutes of direct pressure on the suspected bleeding area. 3. The patient suffered from closed injury of multiple systems, grade III liver injury and moderate TBI (GCS 10 points), sent to ICU for non-surgical treatment. The third-level examination revealed a large area of scalp laceration with persistent non-pulsatile hemorrhage. The laceration continued to bleed after direct compression and suture. Due to a brief drop in the blood pressure, the patient was given several units of fresh frozen plasma and platelets because of the suspected continuous hemorrhage in the wound and abdominal injury. After the blood products were transfused, the bleeding on the scalp stopped, and the hemoglobin level remained stable after rechecking.	Manual compression to stop hemorrhage, application of blood products Only manual compression for hemostasis Blood products, wound suture	Emergency Department Operating room ICU	V-A(b)(ED) [Coagulopathy exists definitely.] II-C(p)(OR) [Coagulopathy is suspected.] IV-E(b)(ICU) [Coagulopathy exists.]

4.1 DIC with Fibrinolytic Phenotype

The key to DIC management is the preventive treatment of potential disorders, that is, the treatment of trauma itself and hemorrhagic shock. The main cause of hemorrhage induced by DIC with a fibrinolytic phenotypic is coagulation factor consumption and hyperfibrinolysis when anticoagulants are forbidden. The alternative therapy for coagulation factor consumption is to supplement concentrated platelets, fresh frozen plasma, and fibrinogen concentrate or cryoprecipitate, which can forcibly maintain the normal platelet count and coagulation level including antithrombin and protein C. At present, the infusion of FFP to maintain protein C and antithrombin may be a reasonable strategy for the treatment of post-traumatic DIC, which is important to inhibit the development of the fibrinolytic phenotype to the thrombotic phenotype. But the dosage of activated protein C, recombinant human thrombomodulin, and antithrombin needs to be noted, which, when exceeding normal levels, can cause hemorrhage.

Tranexamic acid, which can reduce the risk of hemorrhage and death in the injured, should be given as early as possible. Regarding the application of antifibrinolytic drugs in the injured, a global multi-center study is currently underway, of which the results may increase the theoretical evidence for antifibrinolytic therapy in patients with fibrinolytic DIC. However, within 24–48 h after the trauma, DIC with a fibrinolytic phenotype rapidly changes to DIC with a thrombotic phenotype, where patients should not be treated with antifibrinolytic drugs. In the randomized controlled trial (CRASH-2) conducted in 2011, 20,211 patients received tranexamic acid within the first 8 h after trauma. The results showed that the application of tranexamic acid within 3 h after trauma could reduce the mortality rate, while the application after 3 h may not be beneficial but cause harm.

Table 4 Bundling of trauma resuscitation [1]

Treatment of DIC with a fibrinolytic phenotype	Damage controlled resuscitation
• Following disorders – Surgical hemorrhage caused by or secondary to trauma – Hemorrhagic shock • Coagulation factor consumption • Hyperfibrinolysis	• Damage control surgery • Permissive hypotension – Restrictive infusion – Sufficient perfusion pressure • Hemostasis and resuscitation – Fixed blood transfusion ratio – Fibrinogen concentrate – Prothrombin complex – Reorganization FVIIa – Tranexamic acid

4.2 Severe Hemorrhage and Hemorrhagic Shock

In the past decade, based on the experience of military medicine and the progress of TIC research, the method of massive blood transfusion in severe hemorrhage has been greatly improved, which is now called damage control resuscitation. Damage-controlled resuscitation usually consists of damage-controlled surgery, permissive hypotension, and hemostatic resuscitation; however, during the damage-controlled resuscitation, which should be considered as a bundled type from the trauma site to the emergency room, operating room, and ICU, the traumatic resuscitation should cover the following therapeutic protocols during the treatment (Table 4).

4.2.1 Damage Control Surgery and Permissive Hypotension

Damage control surgery includes simple surgery, primary suture of the surgical site (primarily in the abdomen), correction of hypothermia and coagulopathy, and reoperation plan (allowing the injured to recover physiological reserve func-

tion before surgery). Although there are no randomized controlled trials to evaluate the procedure of damage control surgery, this concept has now become the guiding principle of trauma surgery.

Due to a non-randomized trial conducted in the mid-1990s, the concept of permissive hypotension (defined as restricting infusions until the hemorrhage is controlled) spread across the world. However, a Cochrane review showed no difference in mortality between early and delayed (restrictive) resuscitation. There is no evidence to support the mandatory use of permissive hypotension and sufficient cerebral perfusion pressure in patients with traumatic brain injury. The primary goal of the management of severe hemorrhage and hemorrhagic shock is to deliver oxygen to peripheral tissues and organs to counteract the oxygen debt and restore oxygen supply. Oxygen transport is to maintain adequate cardiac output through proper blood volume and perfusion pressure, and it is also coordinated by oxygen carried by hemoglobin and dissolved in plasma. The problem with permissible hypotension is that using this term makes the specific definition of restrictive infusion unclear. In order to maintain adequate perfusion pressure, this misleading term should be more accurate, so that it is currently recommended to describe it as the mean arterial pressure $\geq$ 80 mmHg.

4.2.2 Goal-Directed Hemostatic Resuscitation

The prevention and treatment of coagulopathy has gradually received attention since whole blood resuscitation in the 1970s to the extensive application of massive crystalloid solution and oxygen resuscitation, especially the current damage-controlled surgery (DCS) theory to trauma resuscitation, where the most representative new theory is early goal-directed coagulation therapy (EGCT). The embryonic form of this diagnostic, guided therapy is to guide component blood transfusion based on the results of viscoelastic hemostatic assays (VHA), then it developed into goal-oriented hemostatic resuscitation and finally formed EGCT. The short time required for VHA provides the possibility of rapid diagnosis and individualized treatment, and dynamic detection can be used to continuously evaluate the treatment effect and further optimize the treatment plan to minimize the side effects of treatment. POC treatment is promoted in the guidelines published by the European Task Force for Advanced Bleeding Care in Trauma in 2013, where it was realized that early intervention could improve the abnormal coagulation indicators, reduce the overall demand for red blood cells, FFP, and platelets, and reduce the incidence of post-traumatic multiple organ failure.

It has become a common method to guide massive blood transfusion by laboratory tests or viscoelastic equipment to avoid excessive or harmful use of blood products, which is called goal-directed hemostatic resuscitation. Many studies have suggested the application of viscoelastic devices in this strategy, which can provide a more comprehensive observation of the coagulation function of the injured. It has been proved that rapid TEG at admission has great advantages compared with conventional coagulation indicators (PLT, PT, PTINR, aPTT, and fibrinogen). However, a Cochran report confirmed that for patients with severe hemorrhage, neither ROTEG nor TEG could improve their mortality. The DIC scoring systems of the International Society on Thrombosis and Haemostasis (ISTH) and the Japanese Association for Acute Medicine (JMMA) include the parameters of inflammation, platelets, coagulation, and fibrinolysis, which is beneficial to those who need hemostatic resuscitation in addition to hemorrhagic shock resuscitation. The DIC scoring system of JAAM at admission can independently predict whether the patient needs a massive blood transfusion, the morbidity, and the outcome of severe trauma patients. Therefore, the DIC scoring systems of ISTH and JAAM is promising method to guide goal-directed hemostatic resuscitation.

4.2.3 Fixed Proportion of Blood Product Transfusion

Hemostasis resuscitation, as a part of the massive blood transfusion strategy, is a treatment of severe hemorrhage and trauma-induced coagulopathy through the management of blood products to improve shock and hemorrhage and ultimately to control the development of DIC with a fibrinolytic phenotype to a thrombotic phenotype (DIC with a thrombotic phenotype is the reason for organ dysfunction). In spite of quite a few studies conducted in the past decade, there are few reports based on studies of hemostatic resuscitation.

Previous reports believed that sufficient plasma was important but did not provide an appropriate ratio. A fixed ratio of plasma and red blood cells of 1:1 in the treatment of coagulopathy due to war injuries, which has caused considerable controversy when proposed for the first time, has been accepted by many studies.

Recently, four systematic reviews and two meta-analyses have summarized the impact of a fixed proportion of blood transfusion strategies in trauma on mortality. The conclusions of the systematic reviews are almost the same that a high ratio of platelets or FFP to red blood cells can be associated with low mortality. The USA has summarized its experience through the wars in Iraq (Operation Iraqi Freedom, OIF) and Afghanistan (Operation Enduring Freedom, OEF) and recommended adopting a fixed ratio of packed red blood cells (PRBCs), fresh frozen plasma (FFP), and platelets (PLT) of 1:1:1 in the damage-controlled resuscitation for soldiers with acute trauma, according to the surgical research clinical practice guidelines of the Army Research Institute.

Based on the results of the prospective, observational, multi-center, major trauma transfusion (PROMMTT) study and retrospective analysis, a pragmatic randomized optimal

platelet and plasma ratios (PROPPR) trial was designed to assess the effects of different ratios of plasma, platelets, and red blood cells of 1:1:1 and 1:1:2 in predicting trauma patients who require a massive blood transfusion. There was no significant difference in mortality between the two groups. However, in the 1:1:1 group, more patients received hemostasis within 24 h and experienced fewer deaths after blood loss. This modified whole blood transfusion did not reduce the volume of blood transfusion in severely traumatic patients who had been predicted to require a massive blood transfusion.

Blood products containing plasma and platelets should be used in the early stage instead of a large number of crystalloids and red blood cells to replace the lost blood, which can reduce the occurrence of dilutive coagulopathy to a minimum; meanwhile, hemostatic resuscitation should be individualized based on the actual needs of the injured, according to the results of TEG. These two measures can not only reduce tissue perfusion insufficiency caused by inadequate blood transfusion and excessive hemorrhage caused by trauma-induced coagulopathy, but avoid increasing the risk of acute respiratory distress syndrome, acute lung injury, sepsis, and multiple organ dysfunction due to excessive blood transfusion. For patients with polytrauma but below the diagnostic criteria for hemorrhagic shock or without an indication for massive blood transfusion, DCR is not appropriate because the massive transfusion of plasm and platelets will increase the risk of multiple organ dysfunction but will not enhance their survival rate.

The amount and timing of FFP transfusion have been a topic of much controversy in recent years, mainly focusing on the optimal infusion ratio of FFP and RBC. Although many studies support the active application of FFP in the early stage of trauma, the optimal ratio of FFP to RBC is still inconclusive. In particular, the trauma of different severity and causes can directly affect the optimal ratio of FFP to RBC. Although there are no randomized studies on the application of DCR, many medical centers have adopted this view. But it is very difficult to accurately achieve this ratio, and the optimal ratio is still unknown. Some studies believe that compared with whole blood, this proportional blood component treatment (PRBCs:FFP:PLT = 1:1:1), which is similar to whole blood, provides lower hematocrit, fewer platelets, and lower coagulation factor activity. Therefore, many people believe that the most suitable resuscitation fluid is whole blood in patients with severe trauma who require a massive blood transfusion, which could also alleviate hemorrhagic shock and coagulopathy. At present, the only guideline that supports the application of whole blood before hospitalization is the Tactical Combat Casualty Care guidelines developed for tactical evacuation care.

4.2.4 Fibrinogen Concentrate

Fibrinogen can decrease to a very dangerously low level in the early phase of trauma. European treatment guidelines recommend that the fibrinogen of the injured should not be less than 1.5–2.0 g/L, below which fibrinogen concentrate (3–4 g) or cryoprecipitate (50 mg/kg or 15–20 U) should be used. In severely injured patients with hypofibrinogenemia, FC transfusion can reduce the application of other blood products and decrease the occurrence of hemorrhagic shock and MOF.

Fibrinogen, as the direct precursor of fibrin, is transformed by activating aggregation mediators of thrombin and platelet, playing the basic role of hemostasis. European guidelines define the critical value of fibrinogen as below 1.5 g/L before the critical value of platelet count, PT and aPTT appear within the first 24 h after the patient arrives in the emergency room. The fibrinogen level of patients with DIC decreased significantly when they arrived in the emergency room. Compared with patients without DIC, the former dropped to the critical value more quickly. The level of fibrinogen when arriving in the emergency department is an independent predictor of massive blood transfusion and death after trauma, and its optimal predictive value is 1.9 g/L or <1.5–2.0 g/L. These results clearly indicate that not all injured patients need to be treated with fibrinogen concentrate, and only those with DIC with a fibrinolytic phenotype should use fibrinogen concentrate.

After eliminating the cause of DIC, a fibrinogen concentrate is an important option for the treatment of hemorrhage induced by coagulopathy, including in trauma. However, there are still various contradictions and controversies in the use of fibrinogen in severe hemorrhage. Although there is a lack of evidence for the use of fibrinogen concentrate in injured patients with massive hemorrhage, and there are many methodological flaws in the included studies, some studies still show that fibrinogen concentrate management can reduce the demand for blood products. In the perioperative period, the effectiveness and safety of fibrinogen concentrate still need randomized clinical trials to solve this problem.

Fibrinogen concentrate does have the advantage of reducing the application of allogeneic blood products. Fibrinogen replacement therapy is being explored as a therapeutic strategy, such as fresh frozen plasma, other plasma products, or coagulation factor concentrates (such as prothrombin complex concentrate and recombinant FVIIa).

4.2.5 Prothrombin Complex Concentrate (PCC) and Recombinant Human Activated Factor VII (rFVIIa)

Prothrombin complex concentrate (PCC) is a concentrated product containing three (factors II, IX, and X) or four (factors II, VII, IX, and X) vitamin K-dependent coagulation fac-

tors. PCC contains no or very small amounts of anticoagulant proteins such as protein C, protein S (free), and fibrinogen-free antithrombin. In addition to the use of PCC to treat or prevent vitamin K antagonist-induced hemorrhage, PCC has been used in traumatic hemorrhage.

Many studies have confirmed increased thrombin production and lower levels of protein C, protein S, and antithrombin noted in DIC from immediately to a few days after the trauma. The production of thrombin in the circulation of such patients mainly depends on the reduction of antithrombin. Therefore, in theory, PCC, which has a very high ratio of procoagulant/anticoagulant, can induce thrombosis and DIC. In fact, PCC increases thrombin production with a decrease in platelet count and antithrombin levels, as well as prolongation of PT and APTT, which are similar to the progression of DIC. So it is recommended that PCC should be used cautiously based on the DIC score and the monitored antithrombin level.

The results of two randomized controlled trials on rFVIIa have been published. In the first study, it could only reduce the need for blood transfusion in patients with blunt trauma, but this study contained many methodological flaws; the second was terminated due to lower mortality than expected and ineffectiveness. A recent Cochrane systematic review concluded that the effectiveness of rFVIIa as a hemostatic drug had not been proven, while it can increase the risk of arterial diseases. Clinical trials should be restricted to using rFVIIa beyond the permitted scope.

Recombinant factor VII (rFVIIa) is a promising drug. A prospective study by Boffard et al. found that rFVIIa can reduce the amount of red blood cell transfusion in patients with blunt wounds, but there is no statistical significance in its effect on those with penetrating injuries and mortality. The available evidence suggests that there is no significant difference between patients treated with rFVIIa or placebo, and the former can also increase the probability of worsening DIC. Therefore, it is necessary to further study the efficacy and safety of rFVIIa in patients with fibrinolytic DIC.

4.3 Other Drugs

Considering that inflammation can trigger TIC, it is currently being studied to give patients non-steroidal anti-inflammatory drugs (NSAIDs) in the early post-traumatic phase. Prehospital use of NSAIDs can reduce the incidence of TIC. The conclusions drawn from these data are retrospective and observational, so further randomized controlled trials are needed to verify the significance of NSAIDs and whether they can develop another treatment for TIC.

Desmopressin is a synthetic arginine vasopressin analog that can promote the release of vWF from endothelial cells, increase the number of glycoprotein receptors on the platelet surface and the concentration of factor VIII in the blood, but there is no report on its application in trauma patients.

In terms of treatment, in addition to the above methods, we should also be alert to the hypercoagulable state and thrombosis secondary to TIC. An early study found that coagulopathy at admission is an independent predictor of venous thrombosis in trauma patients. Therefore, it is necessary to pay close attention to the risk of venous thrombosis and pulmonary embolism in such patients in the later phase.

5 Conclusion

Trauma-induced coagulopathy is an endogenous host reaction that occurs immediately after severe trauma. The activation of protein C, endogenous heparinization, and glycocalyx degradation are important initiating factors for TIC, and fibrinogen consumption and platelet dysfunction in hemorrhage must also be considered. The severity of trauma is related to the degree of coagulation. Classic coagulation tests cannot fully describe the complex process of TIC, so its value is limited. At present, it is mostly supported to adopt bedside coagulation function tests through TEG or ROTEM for early diagnosis. Early management includes rapid transportation from the scene to the hospital, surgical operation to control hemorrhage, avoiding excessive crystal fluid for resuscitation, and early identification of TIC. Protocol-guided treatment and PRBC:FFP: PLT = 1:1:1 is a promising strategy that has been widely used worldwide and has been proven to reduce mortality. Existing drugs, such as TXA which is now a standard treatment, should be used early. With our increased understanding of TIC, it is expected to establish standards for early diagnosis and treatment of trauma.

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Multiple Organ Dysfunction Syndrome

Jian Zhou

Multiple organ dysfunction syndrome (MODS) or multiple organ failure (MOF) refers to a potentially progressive syndrome of reversible dysfunction of two or more organ systems as a result of dysregulated host defense, inflammation system, coagulation system and their pathological interaction after the body suffered fatal damage such as severe trauma, infection, major surgery. It has been more than 40 years since the concept was first introduced in the 1970s. MODS has always been the main cause of death in surgical critically ill patients and patients with severe trauma (except early exhausting hemorrhage and severe brain trauma). Although the research on the pathophysiological changes of MODS has come a long way in recent years, there is still a lack of effective treatment, so it is the focus and research hotspot to take active measures to prevent MODS in the early stage.

1 Evolution of Concepts

In the early stage of World War II, hemorrhagic shock once became the main problem threatening the lives of the wounded. The problem has been solved by the progress of blood transfusion and infusion knowledge and the improvement of surgical technology. By the end of the World War II, post-traumatic renal insufficiency (damage) once became the main obstacle to the treatment of the wounded and continued till the Korean War. The in-depth understanding of the pathophysiological changes of shock and the improvement of fluid resuscitation strategy properly addressed this issue. Then the problem of "shock lung" or "large white lung" arose in the Vietnam War. In 1967, Ashbaugh first reported adult respiratory distress syndrome (ARDS) and pointed out that the early use of positive pressure ventilation can reduce pulmonary insufficiency. In the mid-1970s, with the emergence and development of ICU in western countries, the organ function support technology was significantly improved, and the death of patients or wounded due to single-organ dysfunction decreased day by day. At the same time, it was found that as a sequelae of active shock resuscitation, more and more patients died of progressive and sequential multiple organ dysfunction. Tilney and Baue successively reported cases of multiple organ dysfunction in the 1970s, which is called sequential system failure (sequential system failure, SFF). Baue's article emphasizes the interaction between organs. In other words, the prognosis of patients depends on the interaction between organ systems. The injury of one organ can cause the dysfunction of other organs. On the contrary, the recovery of the first failed organ function after treatment can reduce the risk of dysfunction of other organs, and different organ dysfunction has different risks of death. In 1977, Eiseman first proposed the concept of MOF. Shortly thereafter, Fry reported the famous linear relationship between the number of failed organs and mortality in critically ill patients, with a mortality of 30% after single-organ failure and mortality of 100% after four or more organs.

At the beginning of the concept of MOF, people once believed that infection was the main cause, and the traditional theory developed a local infection into a systemic event, also known as "sepsis." Seeing a series of other symptoms such as fever, rapid breathing, and heart rate, clinicians realized that pathogen invasion and subsequent changes in the internal environment of the body not only affected the local infection but also affected the whole body. For example, in the 1970s, most of the wounded in the United States were penetrating injuries), half of whom suffered intraabdominal infection (IAI). The report of the American "Knife & Gun Club" believes that "MOF is a fatal manifestation of uncontrolled infection," which urges the research to focus on the surgical treatment and periop-

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erative management of abdominal infection. Through efforts, the incidence of abdominal infection decreased significantly, but MOF did not decrease. Almost in the same period, more and more studies found that other non-infectious clinical events can also make the body have clinical manifestations similar to the above "sepsis" and eventually MOF, such as the wounded after severe blunt trauma, but these patients have not found a clear infection focus. Since then, a large number of clinical, experimental studies and autopsies have confirmed that not all patients with MOF have an infection. Under laboratory conditions, they can even replicate the clinical and inflammatory manifestations of MOF without the involvement of infectious factors.

Some scholars have noted the role of the gastrointestinal tract in MOF and pointed out that gastrointestinal bacteria could cause MOF in the absence of identifiable infection, and, therefore, raised the hypothesis of "MOF gastrointestinal engine" that the excessive growth of gastrointestinal bacteria and the impairment of mucosal barrier function promote the translocation of bacteria, resulting in the occurrence of MOF. However, the clinical application and experimental research of selective digestive decontamination have not confirmed its effectiveness, suggesting that this hypothesis still needs further research.

With the gradual deepening of research in the 1990s, the general view was that infection was a sufficient condition rather than a necessary condition for MOF. Both infectious and noninfectious factors could lead to MOF, and its main mechanism was to stimulate the body's self-destructive uncontrolled immune-inflammatory reaction rather than the damage to the body of the toxicity of pathogenic microorganisms: Goris' study confirmed that the massive release of inflammatory mediators caused by serious tissue damage and infection is the cause of MOF. Marshall et al. pointed out that the severity of inflammatory response (rather than the type and degree of infection) is correlated to the prognosis of patients.

Based on the above research, the American College of Chest Physicians/Society of Critical Care Medicine (ACCP/SCCM) proposed the concept of systemic inflammatory response syndrome (SIRS) at the joint meeting in 1992, and pointed out that SIRS is the systematic activation of inflammatory cascade caused by infectious or noninfectious factors. It was advised to replace MOF with MODS and pointed out that MODS/MOF was a complication of SIRS. It is clear for the first time that SIRS and MODS are different stages of a pathophysiological process. It was thought that MODS was a static, all or none syndrome. The new definition described MODS as a dynamic pathophysiological process, which greatly changed the perspective of understanding and studying MODS.

2 Epidemiology

MODS is the main cause of death in critically ill patients. Due to different types of injuries and different diagnostic criteria, the incidence is also quite different. Studies have confirmed that the incidence of MODS among ICU patients is 19%, and 50–80% of ICU deaths are related to MODS; a retrospective study of 31,154 cases from 2002 to 2011 in the German Trauma Registry disclosed that the incidence of MODS was 32.7% by sequential organ failure assessment (SOFA), which was deemed as the diagnostic criterion, and the 30-day hospital mortality of MODS and non-MODS patients was 34.1% and 7.5%, respectively, suggesting a high statistical difference; Minei et al. reported that of 916 patients with serious blunt trauma, the incidence of MODS was 29%, according to the diagnostic criterion proposed by Marshall.

Any biological stress-induced inflammatory response can lead to SIRS, which puts the body at risk of MODS. At present, it is known that some external pathological damage, the health condition of the body itself, and whether there are comorbidities are the risk factors of MODS. Studies have revealed that ischemia/reperfusion injury is the most common risk factor for MODS, followed by sepsis and shock caused by any cause. According to the research of Sauaia et al., the probability of MODS is 46% when the injury severity score (ISS) is $\geq$25 plus the need to infuse more than 6 units of erythrocyte suspension within 24 h. However, Cryer found that as long as ISS $\geq$ 25, regardless of the need to infuse erythrocytes, the incidence of MODS is 66%. As for the body's own factors, old age and a variety of comorbidities are the risk factors of MODS. Compared with Caucasians, blacks have a higher risk of infection, sepsis, MODS, and death. The risk of MODS in newborns and young children is significantly higher than that in older children because their own defense mechanism is not perfect. Diseases or pathological processes prone to MODS include sepsis, multiple trauma, burn, severe acute pancreatitis, aspiration, hemorrhagic shock, massive blood transfusion, ischemia-reperfusion syndrome, major surgery, ischemic necrosis of organs or tissues, cytokine release syndrome (cytokine/anti-cytokine therapy), etc.

In recent years, great progress has been made in the research on the relationship between human gene polymorphism and disease. The existing data show that the increased susceptibility of the body to MODS is due to the existence of genes that can amplify the intensity of natural immune and inflammatory response when the body suffers from disease or trauma. At present, single-nucleotide polymorphisms (SNPs) have screened a series of receptors related to pattern recognition (pattern recognition receptors, PPRs), genes and peptides related to signal transduction molecules, effector cytokines, and coagulation factors. However, due to the

diversity of functions, it is difficult to establish the causal relationship between them and MODS. It is believed that with the development of precision medicine and in-depth research, the relationship between the above SNPs and MODS will be further revealed.

2.1 Pattern Recognition and Signal Transduction Receptor

Angiopoietin 2 gene (ANGPT2), calcitonin gene (CALCA1), heat shock protein A1B (HSPA1B), heat shock protein a1l (hspa1l), interleukin 1 receptor-related kinase-1 (interleukin-1 receptor-associated kinase-1, IRAK-1), interleukin-1 receptor-associated kinase-4 (IRAK-4), lipopolysaccharide-binding protein (LBP), mannose-binding lectin (MBL), mitochondrial ND1 gene (ND1), CD14, myelin and lymphocyte protein, myeloid differentiation 2 gene (MD2), myosin light polypeptide kinase (MYLK), toll interleukin 1 receptor domain-containing adapter protein, TLR1, TLR2, TLR4.

2.2 Cytokine

IκβA, INFγ, IL-1α, IL-1β, interleukin-1 receptor antagonist, IL-6, IL-10, macrophage inhibitory factor, TNF-α, TNF-β.

2.3 Coagulation Factor

Coagulation factor V, fibrinogen, plasminogen activator inhibitor-1 (PAI-1), thrombin-activated fibrinolysis inhibitor.

3 Pathogenesis and Pathophysiology

At present, there are three theories about the occurrence mechanism of MODS, i.e., one hit, two hit, and sustained hit. When the initial damage suffered by the body is serious enough (such as severe burn, multiple severe injuries, severe acute pancreatitis), it will directly cause the corresponding organ dysfunction, which is the "one hit" theory. When the body withstands the first hit, and the homeostasis of the body gradually recovers, due to the influence of more serious stress events such as accidental infection, bleeding, and major surgery, the inflammatory response is out of control again and leads to MODS, which is the "two hit" theory. The "sustained hit" theory refers to the continuous action of stress factors on the body (such as ventilator-associated pneumonia caused by multidrug-resistant bacteria) leading to MODS. In clinical practice, the above three mechanisms are difficult to be completely separated and often interact and work together.

Due to the different functional and structural characteristics of organs, the tolerance to ischemia and hypoxia is also different. Generally, heart is the most vulnerable organ and the earliest organ with dysfunction. Respiratory dysfunction is often seen in the early stage, while organ system dysfunction such as liver, blood, digestive tract, and kidney often occurs in the late stage of the disease. Due to the strong compensatory ability of the liver, the early manifestations of functional impairment are often hidden. Once there are obvious abnormalities, it indicates that the severity of the damage exceeds the abnormal degree of clinical test indicators. In addition, clinical observation found that the mortality risk of some organ dysfunction combinations was higher, such as liver and lung combination, kidney and lung combination, blood and heart combination, etc.

Many studies have proved that the immune system plays an important role in the pathophysiological process of MODS and has experienced two distinct functional changes. It has become one of its characteristic manifestations: the first stage is the upregulation and enhancement of innate immunity function (manifested in the uncontrolled inflammatory response, production of a large number of pro-inflammatory factors, increased endothelial permeability, extensive microvascular embolism, accelerated apoptosis, and destruction of parenchymal cell integrity); the second stage is the enhancement of anti-inflammatory response and adaptive immunity. The downregulation of (adaptive immunity) function was weakened (manifested by increased production of anti-inflammatory factors, immunosuppression, and increased risk of infection). These changes are called immunologic dissonance.

3.1 Inflammatory Response and Tissue Injury

The innate immune system is the first barrier to the body's defense function. Tissue injury, ischemia-reperfusion injury, and pathogen invasion all activate it to produce a nonspecific inflammatory response. It can not only protect the body but also play the role of identifying and presenting antigens. It is an important premise for the body to produce specific cellular and humoral immune response. If the body's defense function is strong enough to limit and remove the damaged or invading pathogens locally, only local inflammatory reactions will occur. On the contrary, many pro-inflammatory bioactive substances in local inflammatory foci can enter the blood circulation through multiple ways to cause a systemic inflammatory reaction, resulting in fever, rapid breathing and heart rate, and other typical clinical manifestations of SIRS.

1. Under severe stress, the homeostasis of the body is unbalanced, the tissue is hypoxic, the inflammatory reaction system in the body is activated, and the uncontrolled inflammatory reaction occurs, resulting in tissue damage. The early stage of inflammatory response is mainly the activation of macrophages and the release of cytotoxins and cytokines. Cytotoxins are mainly secreted by $CD8^+$ cells (cytotoxic T lymphocytes and dendritic cells) as co-receptors of MHC class II antigens on the cell surface, which destroy the cell wall and tight junction locally. Cytokines are secreted not only by immune cells (helper T cells, regulatory T cells, monocytes, macrophages, and dendritic cells) but also by platelets or endothelial cells. They mainly mediate the local or systemic reaction of $CD4^+$ cells as MHC class I antigen co-receptors. Although cytokines can be classified according to their functions, the reactions they cause are complex and diverse, overlapping with each other. A factor can activate different effector cells and cause reactions of different properties (pro-inflammatory or anti-inflammatory). The maintenance of homeostasis benefits from the self-regulation and balance of pro-inflammatory and anti-inflammatory responses. Once this balance is broken, it will cause damage to the body. Due to the huge and complex immune and inflammatory response network, although huge human and material resources have been invested in extensive research from the basic and clinical aspects in the past decades, no breakthrough has been made. How to regulate the inflammatory response and restore the body's own homeostasis as soon as possible is still a hot and key research direction in the future.
2. The aggregation of polymorphonuclear (PMN) leukocytes in target organs is another main feature of MODS. Autopsy of patients who died of ARDS showed a large number of leukocyte infiltration in the pulmonary stroma; ARDS animal model experiments also confirmed that removing or inhibiting the function of leukocytes can reduce the pathological damage of the lung. Studies have proved that leukocyte activation (respiratory burst) can produce a large number of reactive oxygen species, nitrogen substances (oxygen-free radicals, nitric oxide), and lipid mediators (prostaglandins, leukotrienes), destroy the tissue structure of target organs, and increase the permeability of capillaries. Clinically, it is often manifested as increased production of acute-phase protein (AP), such as C-reactive protein (CRP), α2-macroglobulin, fibrinogen, α1-antitrypsin, and complement increased, resulting in edema in many parts of the body.
3. The immune system also plays an important role in the pathological process of MODS. Immune cells interact with conserved pathogen-associated molecular patterns (PAMPs) on pathogens through pattern recognition receptors (PRRS) to transmit the information of invasion. In addition to recognizing PAMPs, PRRS can also recognize endogenous damage-associated molecular patterns (DAMPs) mediated by toll-like receptors (TLRs) and produced by activated or necrotic cells when the body is invaded by pathogens, also known as endokines or alarmins. DAMPs include high mobility group box-1 protein (HMGB-1), HSPs, uric acid, altered matrix proteins, S100 proteins, etc. Infectious stress mainly causes the generation and release of HMGB-1 and S100, while HMGB-1 is actively generated and released by identifying PAMPs during infectious stress, rather than passively released by ischemic or damaged necrotic cells during noninfectious stress. DAMPs cause receptor-dependent signal transduction and activation of nuclear factor kappa beta (NF-kB) through the recognition of receptors for advanced glycation end products (RAGE) and TLRs. Animal models of infectious and noninfectious inflammatory response have confirmed that RAGE can also act as the endothelial adhesion receptor of leukocyte integrins, promote leukocyte aggregation in the vascular endothelium of target organs, aggravate the damage of target organs, and inhibit the signal transduction of RAGE can reduce the inflammatory response. Existing studies have also revealed the mechanism of the inflammatory response of the body's natural immune system caused by aseptic injury: as a widely distributed nuclear protein, HMGB-1 mediates the production and release of cytokines and the damage of tissues and organs through TLR signal transduction pathway, so as to play a core role in the production of inflammatory response. RAGE-HMGB-1 complex can also affect the function of the self-stabilizing system by enhancing autophagy, programmed cell survival, and inhibiting apoptosis, programmed cell death.
4. Coagulation system also plays an important role in the occurrence and development of MODS. It is well known that inflammatory response is closely related to the change of coagulation function. The uncontrolled inflammatory response is often accompanied by extensive microthrombosis. TNF-α can activate tissue factors and start a coagulation cascade. In turn, the thrombin receptor activates NF-κB which will increase the transcription of pro-inflammatory genes and produce and release a large number of pro-inflammatory factors. Microthrombosis and tissue hypoxia will prolong the duration of the inflammatory response, so coagulation dysfunction in the early stage of inflammation will greatly increase the risk of MODS.

3.2 Anti-Inflammatory Response

The initial stage of MODS is characterized by the destruction of homeostasis, tissue and organ damage, and systemic inflammatory response. The subsequent stage is characterized by impaired adaptive immunity and increased susceptibility to infection. This phenomenon is called compensatory anti-inflammatory response syndrome (CARS). The final result is that the body's immune function is paralyzed (immunoparalysis). At this stage, characteristics include increased anti-inflammatory cytokines, such as IL-10, IL-13, and TGF-β, decreased antibodies which is the marker of humoral immune function, and inhibited T-lymphocyte function, which is the marker of cellular immune function.

In many sepsis patients, it can be found that the failure of the body's immune system to remove pathogens in time often indicates the continuation of the immunosuppressive state. Its characteristic performance is that the body is complicated with a nosocomial infection on the basis of failure to remove the primary infection. Its mechanism is multifactorial, including but not limited to the reduction of lymphocytes and dendritic cells caused by mass apoptosis. The expression of HLA-DR cell surface antigen presentation complex decreased, and the expression of negative costimulatory molecular programmed death 1, cytotoxic T-lymphocyte-associated antigen 4, B-cell T-cell attenuation factor, and its corresponding ligands increased. The number of regulatory T cells and myelogenous inhibitory cells increases. The cell phenotype of pro-inflammatory Th1 could change to anti-inflammatory Th2 cells, resulting in the production of IL-10. The exhausted $CD8^+$ cells and disabled $CD4^+$ cells in the blood suggest that both natural immunity and adaptive immunity are seriously inhibited.

As another explanation of the pathogenesis of MODS, the theory of system biology holds that the occurrence of MODS is the result of the destruction of the normal communication between organs or cells. This theory regards every organ and system in the body as a stochastic biological oscillator, and their functional activity state changes periodically with time. The functional state of each organ must adapt to its system. The functional activities between organs are interconnected through a variety of mechanisms, in a good coordinated state, and the body can maintain health. When the body is in a normal homeostatic state, the variability of the oscillatory body's functional activities can be kept stable and controllable through the regulation of input mechanisms such as machinery, nerve, hormone, and immunity (cytokines and prostaglandins). After the body suffers serious physiological damage, the contact between biological oscillators (organs and systems) loses coordination, which can lead to the deterioration of organ and system functions. The most studied in this field is the heart: the loss of normal heart rate variability indicates that the communication between organs and systems is out of coordination, and MODS is easy to occur. For example, clinical studies have reported that heart rate variability in healthy volunteers increased significantly after endotoxin injection, and the same changes were also seen in emergency patients with sepsis. Other studies also observed that low heart rate variability was associated with mortality in patients admitted to ICU or suffering from MODS. Therefore, heart rate variability monitoring is expected to become a noninvasive, accurate, and effective means to predict the prognosis of critically ill patients.

Although the disorder of communication between random biological oscillators may become a new theory to clarify the pathogenesis of MODS, and some studies have used this theory to explain the failure of MODS anti-mediator treatment trials that have invested a lot of human and material resources, more research is needed to enter the clinical application and provide practical and effective treatment options for MODS treatment, including the fundamental update of the current definition of MODS (from the current theory to the biological oscillator imbalance theory).

3.3 Persistent Inflammation, Immunosuppression, and Catabolism

In previous clinical studies on MODS, the analysis of the Denver MODS database in 1996 revealed that the occurrence of MODS showed a bimodal phenomenon, and after the body withstood the initial damage, some patients developed MODS soon, which is the first peak, accounting for about 1/3 of patients with MODS. After that, the patients experienced a relatively stable stage. Until the nosocomial infection began, some patients developed MODS, the second peak, accounting for about 2/3 of the patients with MODS. At that time, the immunological view was that after the initial damage, the body suffered from the pro-inflammatory response caused by the enhancement of natural immune activity, namely SIRS, and its effector cells were mainly neutrophils. If this reaction is strong enough, it can lead to the occurrence of early MODS and form the first peak; in order to protect itself from the damage of self-destructive pro-inflammatory reaction, the activity of adaptive immunity will decrease, and CARS will occur, and its effector cells are mainly lymphocytes. If CARS continues to exist, the immune function of the body will be damaged, prone to nosocomial infection, and form the second peak of MODS. However, based on this theory, a large number of clinical trials aimed at the early antagonizing inflammatory response, controlling SIRS and CARS, and preventing late nosocomial infection in the 1990s ended in failure. In recent years, more and more studies have questioned the SIRS/CARS theory. It was found

that blocking the early pro-inflammatory response failed to affect the anti-inflammatory response and adaptive immunosuppression in the animal model of sepsis by cecal ligation and perforation. In addition, pro-inflammatory and anti-inflammatory cytokines were produced almost at the same time. It is worth noting that in the study by Glue Grant, through the genome-wide expression analysis of peripheral blood leukocytes of patients with severe trauma, the researchers found that in addition to the increased expression of pro-inflammatory genes, the expression of genes related to T-lymphocyte activity and antigen presentation function was downregulated and the expression of anti-inflammatory genes was upregulated. These results are consistent with that observed in animal experiments.

During this period, as the main population of MODS, the clinical treatment of trauma patients has made a lot of progress, which is reflected in the following aspects.

The concept of initial treatment of patients with severe bleeding has changed fundamentally in order to reduce the occurrence of abdominal compartment syndrome (ACS), including (1) formulation and implementation of massive transfusion protocols (MTPs), (2) rapid control of bleeding, (3) early resuscitation to limit the use of crystalloid fluid as much as possible, and (4) that an active resuscitation strategy-oriented by oxygen delivery cannot be adopted.

Standard operating procedures (SOPs) have been adopted based on evidence-based medicine in the professional trauma center to avoid iatrogenic damage, including (1) avoiding high tidal volume ventilation, (2) avoiding the use of open blood transfusion strategy, (3) avoiding early parenteral nutrition, and (4) avoiding intermittent hemodialysis.

The management of infection control is strengthened to reduce the risk of late nosocomial infection, including (1) hand disinfection, (2) full barrier protection during deep vein puncture, (3) mechanical ventilation cluster scheme, and (4) formulating an antibiotic application plan.

Thanks to the above progress and the development and improvement of intensive support technology, most severe patients can safely survive the high-risk period of death, and the two death peaks of common patients with severe post-traumatic MODS gradually disappear. Yet, among the surviving patients, some will enter chronic critical illness (CCI), which is characterized by a prolonged stay in ICU (more than 14 days), the function of important organs continued to maintain a low level of normal or slightly abnormal state, peripheral blood lymphocytes decreased, function decreased, granulocytes increased, even immature granulocytes appeared, serum IL-6 level increased, C-reactive protein representing acute phase reaction increased, prealbumin decreased, the body was in a catabolic state, lean constitution continued to lose, and wound healing ability disorder cannot be reversed even through active nutritional support intervention. This state has become a new manifestation of MODS in the twenty-first century, which is known as persistent inflammation, immunosuppression, and catabolism syndrome (PICS). These patients often need to be admitted to long-term acute care facilities (LTACs) after discharge. Their condition is difficult to recover. They often need to be repeatedly admitted to ICU due to infection or organ dysfunction, and the final prognosis is poor. It is believed that with the aging of the population and the continuous improvement of perioperative treatment levels, PICS will become the main manifestation of MODS in the twenty-first century and pose a severe challenge to the treatment of critically ill patients.

4 Pathological Changes

4.1 Lungs

The most significant changes of MODS often occur in the lungs, manifested as weight gain and the following pathological forms. (1) The incidence of bronchopneumonia is as high as about 82%. (2) Pulmonary hemorrhage, with an incidence rate of about 65%, is mostly bilateral, multiple, focal pneumonia, with high congestion and edema, extensive bilateral hemorrhage, and sometimes "blood edema," or liquid exudation in the alveolar cavity with hemorrhage, accompanied by infiltration of a large number of inflammatory cells. (3) Pulmonary congestion and edema. Mild congestion and edema can also be near-death changes. However, high congestion and edema often occur in patients with MODS.

Respiratory dysfunction has a high incidence in MODS, which occurs early and can occur 24–72 h after onset. The clinical manifestations are acute lung injury (ALI) and ARDS. Due to the severe systemic inflammatory reaction, there are a large number of inflammatory mediators in the circulation that can enter the pulmonary circulation, resulting in the destruction of the alveolar-capillary wall and increased permeability. Water, protein, and neutrophils exude and accumulate in pulmonary stroma and alveoli. Neutrophils and other inflammatory cells release a large number of inflammatory mediators, resulting in edema and effusion in pulmonary stroma and alveoli, affecting gas exchange diffusion function. Hypoxia, acidosis, endotoxin, and vasoactive substances cause spasm and contraction of pulmonary arterioles, adhesion, and aggregation of platelets and microthrombosis, which will lead to embolism of the pulmonary capillary network and pulmonary ischemia. Ischemia, hypoxia, and inflammatory mediators can induce metabolic disorders of alveolar type II epithelial cells, resulting in insufficient production and composition changes of alveolar surfactant, increasing alveolar surface tension, reducing the pressure of pulmonary stroma and perivascular tissues, promoting the

transfer of fluid to stroma and alveoli, and aggravate pulmonary edema. Alveolar surface tension increased, lung compliance decreased, resulting in diffuse alveolar collapse and atelectasis, and intrapulmonary partial flow increased. The compression of pulmonary vessels by pulmonary embolism, contraction, and interstitial edema reduces the blood flow through the alveoli, resulting in dead space ventilation. Pulmonary interstitial edema and hyaline membrane on the alveolar surface lead to diffusion dysfunction, imbalance of ventilation blood flow ratio (V/Q), and increase of intrapulmonary partial flow, which together lead to persistent hypoxemia.

4.2 Heart

Bleeding and small necrotic foci can be seen in the endocardium, banded damage in the myocardium, the disappearance of local transverse striations, cell swelling, vacuolar degeneration, and myocardial rupture, showing intercalated disc damage and disappearance of mitochondria. Histochemistry showed that succinate dehydrogenase disappeared in a bag shape and myocardial microvascular stasis.

Endotoxin, inflammatory mediators, and free radicals can all have toxic effects on the heart. It can show impaired systolic and diastolic function before heart injury. MODS involves the release of PGI_2, histamine, bradykinin, NO, and other vasodilator substances, as well as TXA_2, endothelin, and other vasoconstrictor substances, resulting in disordered secretion of vasoconstrictor and vasodilator substances. On the one hand, a large number of short-circuit blood vessels are opened, resulting in low resistance of peripheral blood vessels. On the other hand, the increase of microcirculation resistance leads to the insufficient blood supply of tissue cells, resulting in high output and low resistance hemodynamic manifestations, combined with ischemia and hypoxia of peripheral tissue cells, increased oxygen saturation of mixed venous blood, decreased oxygen differential pressure between dynamic and static veins, and increased blood lactic acid level. High cardiac output is mainly achieved by the increase of heart rate, but the ejection fraction decreases. High dynamic circulation runs through the whole course of the disease. According to the statistics of clinical cases, the incidence of cardiac dysfunction is lower than that of other organ system dysfunction. Once it occurs, it is often accompanied by shock, which is characterized by decreased cardiac output, increased left ventricular end-diastolic pressure (LVEDP), increased pulmonary capillary wedge pressure (PCWP), large fluctuation of blood pressure, and decreased responsiveness of the cardiovascular system to positive blood vessels and myocardial active drugs.

4.3 Microcirculation

The microvessels in the whole body are highly stagnant, mostly red blood cells overlap, and there are many small vacuoles near the free edge of the endothelium in the vascular cavity, which is confirmed by an electron microscope. Having experienced endothelial cell swelling, vacuolar degeneration or even falling off, plasma infiltration and eosinophilic enhancement can be found in endothelium and basement membrane. Perivascular collagen fibers also have a certain degree of plasma infiltration, and cellulose infiltration can be found in severe cases. The eosinophilia of stroma is generally enhanced, which may be the phenomenon of extracellular acidosis. This situation is not eosinophilic hyperchromatism but the change of extracellular pH with increased acidity. The yellow-green change of red blood cells in microvessels of patients who died of MODS may be the manifestation of dying or dead red blood cells.

Trauma, hemorrhage, and infection can induce shock, resulting in insufficient effective circulating blood volume, decreased cardiac output, disturbance of microcirculation, and decreased tissue perfusion. Primary factors initiate a series of changes, such as activated coagulation factors and activated platelets cause local microthrombosis in microcirculation, activated mast cells produce vasoactive substances, promote local microcirculation vasodilation, improve vascular permeability, increase local blood flow, and slow down blood flow velocity, resulting in blood flow deposition. Complement activation can activate the coagulation system and cooperate with inflammatory cells to produce inflammatory mediators. Cells adhere, migrate, and release damaging substances such as lysozyme and oxygen-free radicals locally, resulting in tissue damage.

4.4 Gastrointestinal Tract

When the body is severely hit by trauma, shock, and infection, the circulatory function is unstable, and the visceral blood vessels contract selectively to ensure the blood perfusion of important organs such as the heart and brain. In this case, the blood vessels of the gastrointestinal tract often contract, and the digestive tract is prone to ischemic injury. After resuscitation, local blood vessels recover blood flow, and reperfusion injury may occur. Therefore, it can be seen that the gastrointestinal mucosal epithelium is ischemic and shed, the permeability is increased, and non-gastrointestinal nutrition will also lead to gastrointestinal mucosal atrophy. The use of acid inhibitors decreased gastrointestinal peristalsis function, and bile reflux will make pathogenic bacteria multiply. The decrease of secretory IgA can decrease the local anti-infection ability. Clinically, the irrational use of

antibiotics can cause intestinal flora imbalance, bacterial and endotoxin translocation to the abdominal cavity through gastrointestinal mucosa, and then enter the systemic circulation through mesenteric lymph nodes and portal system. Bacterial translocation is an important cause of sepsis. Clinical manifestations include toxic intestinal paralysis, stress ulcer, and intestinal nutritional intolerance.

4.5 Liver

Some data have confirmed that most patients with MODS have liver enlargement. The reasons are as follows: (1) cholestasis: bile thrombus is formed at the edge of hepatic lobule with cholestasis, which may lead to intrahepatic bile excretion disorder, especially interlobular bile duct (Hering tube) obstruction to the bile duct, and bile duct transition; (2) blood flow stagnation: MODS cases generally show high expansion of the hepatic sinus, up to three to four times the original size, blood flow stagnation, degeneration, necrosis, and bleeding in the central lobular zone, and sometimes degenerative free hepatocytes or cell clusters can be seen in the central hepatic vein, inferior lobular vein, or hepatic sinus, which is the phenomenon of hepatocyte mobilization; (3) Glisson sheath cell infiltration: mainly lymphocytes, plasma cells, a small number of neutrophils, and eosinophils can also be seen, which is nonspecific hepatitis, related to immune response; (4) other lesions: when liver cirrhosis is accompanied by gastrointestinal bleeding and esophageal variceal bleeding, the liver may have varying degrees of ischemic necrosis.

Severe trauma, shock, and infection can lead to the decrease of ATP level of hepatocytes, the disorder of energy metabolism, injury of hepatocyte parenchymal cells and biliary cells, injury of capillary bile duct caused by endotoxin, obstruction of bile drainage, cholestasis, and damage of liver excretion function. The activity of the P450 enzyme system in liver tissue decreased, and the ability of drug metabolism was impaired. Bilirubin metabolism disorder is clinically manifested as jaundice. Endotoxin can stimulate liver Kupffer cells to release inflammatory mediators, affect hepatocyte clearance of inflammatory mediators and reduce acute-phase protein synthesis. Liver metabolic dysfunction, abnormal levels of blood glucose, blood lipid, amino acid, bilirubin, urea, and lactic acid, and affected the uptake and clearance of endotoxin, bacteria, and their toxic metabolites.

4.6 Kidneys

In kidneys, significant turbid swelling and increased weight can be noted. The specific changes were as follows: (1) Renal tubular changes. The most significant changes in the kidney were in the renal tubules, especially in the proximal tubules, showing varying degrees of turbid swelling and degeneration, filled with protein tubules. Some cases had significantly expanded renal tubules and renal vesicles. Some cases have oliguria a few days before death and no urine 1–2 days before death. In some cases, the renal tubular epithelium becomes flattened, and in severe cases, the renal tubular epithelial cells become necrotic, and the morphological changes are similar to extrusion syndrome. In patients with obvious jaundice, bile duct type can be seen in the distal convoluted tubule, which is like biliary nephropathy. (2) Interstitial changes. The interstitium could be edematous in varying degrees, most with scattered focal lymphocyte infiltration, plasma infiltration, and sometimes, severe cellulose infiltration.

In SIRS and sepsis, toxins and inflammatory mediators directly act on renal tubular cells, endothelial cells, and mesangial cells. ATP production decreases, Na^+-K^+ ATPase activity decreases, free radical production increases, and clearance decreases, reducing glutathione decreases, and intracellular calcium overload will cause damage to cells and lead to renal dysfunction. When shock and low blood volume exist, the sympathetic nerve is excited, renal entry and exit arterioles contract, renal vascular resistance increases, blood flow decreases, and glomerular filtration rate decreases. The increase of antidiuretic hormone (ADH) secreted by the pituitary promotes the reabsorption of water in renal tubules and collecting ducts; the increase of renin-angiotensin-aldosterone system activity promotes the reabsorption of Na^+. The increase of endothelin release and the decrease of prostaglandin synthesis, the imbalance of vasoconstrictor and diastolic substances also lead to the further decline of renal blood flow, the redistribution of intrarenal blood flow, and the ischemia of the renal cortex. The clinical manifestations of the above conditions are rapid deterioration of renal function, change of urine volume, metabolic disorder, and so on.

4.7 Nervous System

Neurological changes include nerve cell swelling, vacuolar degeneration, Nissl body reduction, and disappearance, even layered necrosis, glial cell proliferation, gangliophagocytosis, glial cell swelling, and fuzzy cell boundary. Patients with MODS affecting the nervous system often fall into a coma. If the cortex is seriously damaged and the brain stem function remains normal, they enter the state of plant survival. Once the disease seriously affects the brain stem, the patient will have irreversible brain death.

Central nervous system dysfunction is often evaluated by the Glasgow Coma Scale (GCS), which is important to predict the mortality of patients. Neurological dysfunction is related to sepsis, intracranial hemorrhage, brain edema, liver

and kidney diseases, acidosis, etc. In addition, neuromuscular diseases are also quite common, up to 50–70%, which are related to neurotoxic drugs, malnutrition, long-term braking, toxic effect, tissue hypoxia, intraneural edema, and other neuroaxonal degeneration and nerve injury. Patients show fatigue, sensory loss, and respiratory muscle weakness in severe cases.

5 Clinical Manifestations and Diagnosis

5.1 Clinical Manifestations

Although the uncontrolled inflammatory response shows a clear impact on the body's organs, on the whole, organ dysfunction rarely occurs alone. In addition, it should be noted that organ dysfunction is often the result of multiple factors in addition to the inflammatory response. For example, acute kidney injury (AKI) is the result of a combination of factors such as low blood volume and the use of nephrotoxic drugs. The inflammatory reaction can widely affect multiple organs of the body, resulting in different clinical manifestations. In the heart, it may be myocardial inhibition; in lungs, it may be ARDS; in kidneys, it can be acute tubular necrosis and acute kidney injury (AKI); for the digestive system, there may occur cholestasis, erosive gastritis, intestinal obstruction, non-calculous cholecystitis, dysfunction of the hepatic reticuloendothelial system and translocation of intestinal flora; in the central nervous system, there may be toxic encephalopathy, multiple neuron disease or neuromuscular dysfunction; for the blood system, thrombocytopenia and coagulation dysfunction may occur.

Although shock is often the initial cause of SIRS and MODS, the inflammatory response itself can also lead to cardiac dysfunction. Inflammatory mediators tumor necrosis factor, reactive oxygen species, and nitric oxide can inhibit the contractility of the myocardium. The inflammatory reaction can also increase the permeability of endothelial cells and vasodilation, reducing the amount of return blood and vascular resistance. In this way, the main factors affecting blood pressure, preload, myocardial contractility, and afterload are affected, resulting in hypotension and poor response to simple supplementary volume, and often need to be combined with vasoactive drugs.

In MODS, the main manifestation of lungs is the impaired gas exchange function, which is mainly presented as hypoxemia with nonobvious carbon dioxide retention in the early phase. The most serious type of lung damage is ARDS, mainly manifested as PaO_2: $FiO_2 < 200$, and diffuse infiltration shadow of bilateral lungs on the chest X-ray scan without evidence of increased left atrial pressure and excessive fluid.

There are many manifestations of gastrointestinal function injury in patients with MODS, including gastric motility disorder after surgery, intestinal and colonic obstruction, and intractable diarrhea after sepsis. It is reported that about 50% of patients are with the gastric emptying disorder during mechanical ventilation, and this proportion can rise to 80% in patients with increased intracranial pressure after brain injury. The intestinal tract receives dual innervation of visceral intrinsic nerve and external autonomic nerve (mainly parasympathetic nerve). The causes of gastrointestinal motility disorder may be innervation disorder, inflammation, ischemia-reperfusion injury, drug influence, electrolyte disorder, or sepsis. In most cases, it is the result of the comprehensive action of many factors. Ischemia-reperfusion injury can lead to stress gastric mucosal bleeding (stress ulcer) or acute non-calculous cholecystitis. It can also destroy the integrity of intestinal mucosa, and the displacement of intestinal flora can lead to intestinal infection (intestinal engine theory).

Studies have proved that the increase of intestinal permeability is closely related to the occurrence of SIRS and MODS, but the current routine clinical examination methods cannot accurately judge its changes. The non-protein amino acid citrulline almost completely comes from intestinal cells. The determination of citrulline concentration in serum is expected to be a means to judge the function of intestinal cells.

Cholestatic jaundice is another important characteristic manifestation of liver damage, in addition to the increase of related enzymes in serum (represented by alanine aminotransferase ALT and aspartate aminotransferase AST). The toxic effect of proinflammatory mediators can destroy the tight junction between hepatocytes, make bilirubin leak out of the Diehl space between hepatocytes and finally return to the blood vessel. During the systemic inflammatory reaction, the liver first shows an increase in the synthesis of positive acute-phase reactive protein (CRP, ferritin) and a decrease in the synthesis of negative acute-phase reactive protein (albumin, transferrin), followed by a decrease in the synthesis of coagulation factors, which cannot be corrected by vitamin K supplementation, and coagulation dysfunction can occur clinically. The liver has a strong compensatory reserve function. The clinical manifestations of liver injury caused by the inflammatory reaction are generally not obvious and cannot be found by conventional laboratory examination methods. Jaundice often indicates that normal liver function has been seriously lost, up to 80%. Therefore, measuring the metabolite concentration of serum metabolites through the liver may become an early and quantitative method to measure liver function damage.

The most common causes of AKI are hypoxia and ischemic damage. Other causes include rhabdomyolysis, the use of nephrotoxic drugs (iodine-containing contrast agents, antibiotics, etc.), and inflammatory mediators (TNF-α) acti-

vating the renin-angiotensin aldosterone system, etc. Clinical manifestations include oliguria, azotemia, and accumulation of liquid and metabolic waste. In addition to rhabdomyolysis, most of the early electrolytes in other patients were normal. Kidney has a strong reserve capacity. AKI is common in the middle and late stages of the course of MODS. If it occurs, it indicates that the condition is serious and the mortality and disability rate are high. Therefore, it can be used as an index to evaluate the prognosis of patients. Due to IL-1, TNF-α, TGF-β, and other inflammatory mediators can inhibit the synthesis and function of erythropoietin, and patients with AKI can also develop anemia.

The nervous system damage of MODS shows the change of state of consciousness or loss of consciousness. At present, the degree of consciousness disorder is still expressed by Glasgow Coma Score (GCS). Studies have shown that the GCS score is closely related to the prognosis of patients with MODS. However, it must be noted that GCS is not a scoring system specially developed for critically ill patients. The accuracy of critical patients in ICU will be limited by factors such as limited speech expression (patients with endotracheal intubation) and affected arousal assessment (anesthesia, labor pain, and sedative drug use). At present, we are actively looking for a more suitable method for the functional evaluation of the nervous system in critical patients.

In the pathophysiological process of MODS, tissue hypoperfusion, extensive microvascular embolism, and brain edema are the main causes of neurological damage. The manifestations of the nervous system should include muscle lesions in addition to neuropathy. In recent years, it has been observed that many critically ill patients with altered consciousness also have fatigue and muscle weakness, need long-term mechanical ventilation support, and finally develop mods. It is called critical illness polyneuropathy syndrome or ICU acquired weakness. Its severity can be quantitatively evaluated by neuromuscular electrophysiological examination and is not affected by anesthesia, analgesic, and sedative drugs. Its risk factors include long-term bed rest, sepsis, hormone use, and so on. With further research, critical polyneuropathy may replace critical encephalopathy and become a sign of neurological damage in MODS.

5.2 Diagnosis

Early studies believed that organ failure was a binary phenomenon of "all or nothing." If an organ needed medical support measures (such as ventilator-assisted respiration, vasoactive drugs to maintain blood pressure, renal replacement therapy, etc.) to maintain its basic function, it was considered that there was a functional failure. Once organ failure occurs, there is no difference in its impact on the body regardless of its severity and duration. The prognosis of patients is only related to the number of failed organs, not the degree and duration of the failure. With the deepening of basic and clinical research and the gradual accumulation of clinical cases, researchers found that MODS is not simply "all or nothing," but a dynamic process gradually produced and formed in the process of continuous changes in physiological organ functions. Organ dysfunction does not necessarily lead to functional failure. The damage degree of different organs in MODS due to different primary causes varies too. The severity of organ dysfunction should be measured by grading method. The prognosis of patients is not only related to the number of organs with dysfunction but also closely related to the grading severity and duration of organ dysfunction. Therefore, several scoring systems have been developed to quantitatively evaluate the degree of organ dysfunction in MODS. The scoring system mainly includes the following organ systems: (1) respiratory system, (2) cardiovascular system, (3) kidney, (4) liver, (5) blood system, and (6) central nervous system. The focus of each scoring system is to grade and score the organ function by using the objective detection indicators commonly used clinically that can reflect the changes in organ function. The sum of each organ score is the patient's MOD score. A large number of clinical studies have verified that there is a good correlation between patients' MOD scores and prognosis. The higher the score, the worse the prognosis. Because it is difficult to find objective and reliable indicators to reflect its functional status, gastrointestinal and endocrine systems are not included in the scoring system. Now, Marshall's multiple organ dysfunction score, a representative scoring system, is introduced as follows (Table 1).

Table 1 Multiple organ dysfunction score

	Score				
Organ system	0	1	2	3	4
Respiration (PaO_2: FiO_2, mmHg)	>300	226–300	151–225	76–150	≤75
Kidney (serum creatinine, mol/L)	≤100	101–200	201–350	351–500	>500
Liver (serum bilirubin, μmol/L)	≤20	21–60	61–120	121–240	>240
Cardiovascular system (pressure-adjusted heart rate)	≤10	10.1–15	15.1–20	20.1–30	>30
Blood (platelet count, $\times 10^9$)	>120	81–120	51–80	21–50	≤20
Glasgow coma scale (GCS)	15	13–14	10–12	7–9	≤6

Note: heart rate after pressure adjustment = heart rate × CVP/MAP

The above scoring system was designed and developed by Marshall et al. based on the review of MEDLINE related to MODS clinical research from 1969 to 1993. The maximum score of each organ system is 4 points, and the maximum score of the system is 24 points. If the prognosis is judged by the single-organ score, the mortality corresponding to any single-organ score of 0 is less than 5%, and the mortality corresponding to a score of 4 is more than 50%. If the prognosis is judged by the total score, the mortality rates corresponding to the scores of 9–12, 13–16, 17–20, and >20 are <25%, 25–50%, 50–75%, and 75–100%, respectively. The system is simple and easy to use, which provides an objective evaluation basis for the occurrence and development of MODS, the dynamic changes of the disease, and the prognosis of patients.

In the past three decades, researchers have made many efforts to find biomarkers related to inflammation and infection to guide diagnosis and treatment. However, due to the natural heterogeneity of the disease process, different patients with the same disease can have a variety of clinical manifestations. The determination of biomarkers has a short duration or does not appear in the blood circulation. It is difficult to find a meaningful correlation with the pathophysiological process of the disease due to the mismatch of blood collection time points, so the results found so far are limited. For example, nearly 40 screened biomarkers have been evaluated in clinical research on pancreatitis in recent years. Due to the complexity and diversity of methodology and diseases, no indicators of guiding significance for clinical practice have been found. A little progress has been made in the study of blood lactate, IL-6, and PCT: it has been confirmed that early clearance of blood lactate suggests that tissue hypoxia is corrected during shock resuscitation in patients with severe trauma and sepsis, which is related to the lower serum concentration of proinflammatory mediators and lower risk of MODS and can be used as a marker of beneficial clinical outcome. In trauma patients, the increase of serum IL-6 concentration suggests an increased risk of organ dysfunction, but the relationship between infection and prognosis is still unclear. As a marker of infection and inflammatory response, the dynamic changes of PCT are helpful to the differential diagnosis of infection and non-infection and can guide anti-infection treatment. The relationship between PCT and patient prognosis remains to be further studied.

6 Treatment Principles

The management of MODS is mainly twofold, i.e., prevention and treatment. The corresponding clinical treatment focuses on the support treatment of organ function and reducing the degree of systemic inflammatory response. The key to prevent MODS is to timely deal with the primary lesion or trauma, the further deterioration of tissue condition or injury, accurately and dynamically evaluate organ function, early identify the deterioration of organ function, and take effective support means (including drugs or instruments), timely and effective resuscitation, correct tissue hypoxia as soon as possible, and effectively maintain organ function. At present, the measures supported by evidence-based medicine include low tidal volume mechanical ventilation in patients with ARDS, renal replacement therapy in patients with AKI, appropriate anti-infection treatment, and blood glucose control.

It should be noted that although hypoxia of tissues and cells plays an important role in the early pathophysiological process of MODS, the implementation of supernormal oxygen resuscitation strategy by inhaling high concentration oxygen, using vasoactive drugs or active blood transfusion cannot reduce the occurrence of MODS and improve the prognosis of patients. In fact, it may aggravate organ function damage, which must be avoided. The reasons why supernormal oxygen resuscitation aggravates organ and tissue injury are as follows: (1) very high oxygen content in tissue is conducive to the production of cytotoxic reactive oxygen species and aggravates tissue injury. (2) The "storage loss" characteristic of red blood cells causes the loss of high-energy phosphate of stored red blood cells: the content of 2,3-diphosphoglyceride in red blood cells stored for more than 48 h decreases due to the loss, resulting in the left shift of oxygen departure curve and the difficulty of red blood cells in releasing oxygen. After storage for more than 14 days, adenosine triphosphate on the erythrocyte membrane is exhausted, which increases its brittleness and reduces its deformation ability. It cannot enter microcirculation through deformation, resulting in obstruction of oxygen release. Therefore, a large number of stored red blood cells cannot improve the body's oxygen supply.

Timely, appropriate, and anti-infective treatment is the basic guarantee for the success of the treatment of sepsis and septic shock. Despite the numerous recommendations and guidelines on the dosage of antibiotics in patients with renal insufficiency, there is little literature on the dosage of antibiotics in patients with MODS. The most likely pharmacokinetic parameters to change in patients with MODS are distribution volume and clearance. First, the disease itself and fluid resuscitation often increase the distribution volume of patients. After using antibiotics according to the conventional recommended dose, the plasma drug concentration of MODS patients is lower than that of other non-MODS patients, at least on the first day of treatment. Second, organ dysfunction will lead to the reduction of drug clearance, which is prone to toxic reactions caused by drug accumulation. Therefore, the selection of antibiotic maintenance dose should be adjusted according to the degree of dysfunction of drug excreting organs (liver or kidney). For drugs with large

toxic and side effects or narrow treatment windows, therapeutic drug monitoring (TDM) can also be carried out to accurately guide the adjustment of antibiotic dosage.

As for anti-inflammatory treatment, the focus of intensive research in recent two decades is to regulate the uncontrolled immune and inflammatory response in MODS. Early attempts were made to systematically inhibit the body's immune response, including the use of nonsteroidal anti-inflammatory drugs, hormones, and the addition of fish oil to food, but the results of many clinical trials were disappointing. Among them, nonsteroidal drugs can also reduce renal blood perfusion and destroy the integrity of gastrointestinal mucosa, thus aggravating the damage of organ function. With the deepening of the research on the pathophysiological mechanism of the inflammatory response, the role of various cytokines in inflammatory response has been gradually explored, and the focus of treatment research has shifted to the target treatment guided by inflammatory mediators. So far, nearly 100 clinical trials have been carried out, and we tried to confirm the inhibitory effects of anti-inflammatory mediators monoclonal antibodies (TNF antibodies, endotoxin antibodies) and inflammatory mediators receptor antagonists or inhibitors (IL-1 receptor antagonists, tissue factor pathway inhibitors, TLR-4 antagonists) on the inflammatory response. However, the results of these studies aimed at neutralizing inflammatory mediators are also disappointing. So far, no treatment or drug targeting inflammatory mediators has been approved for clinical application. At the same time, the interaction between inflammation and the coagulation system has also attracted attention. Studies have found that the early inflammatory response is always accompanied by the activation of the coagulation system, and the risk of extensive microthrombosis is significantly increased. Therefore, recombinant human-activated protein C (rhAPC) was developed at the end of the twentieth century and the beginning of the twenty-first century. This is a coagulation factor with anti-inflammatory, anticoagulant, and fibrinolytic activity. The results of pre-market clinical studies show that the risk of 28-day death can be reduced by 19.4% in patients with severe sepsis, and the average SOFA score has also been significantly improved. However, from the clinical observation after listing in 2001, it was found that the incidence of life-threatening severe bleeding increased significantly. Therefore, it was delisted from the world in 2010, and the upsurge of anticoagulant treatment of sepsis came to an end.

In conclusion, the treatment of MODS can be summarized into the following aspects.

6.1 Removal of Primary Cause

Removing the primary etiology is crucial and fundamental to the success of the treatment of MODS. If the primary etiology is not removed in time or effectively controlled, even if other treatment measures are sufficient and powerful, the prognosis of patients is poor, especially those with a serious infection and a large number of tissue necrosis (such as severe burns). Therefore, all timely and effective measures should be taken to actively address the primary cause.

6.2 Removal or Antagonism of Endotoxins

Clinical studies have confirmed that before using anti-infective drugs, the concentration of free endotoxin in plasma of patients with Gram-negative bacilli sepsis is roughly proportional to the number of bacteria. After the application of antibiotics, the number of bacteria in blood flow decreases, and the concentration of endotoxin increases. Therefore, in the correct selection of antibiotics, we should not only prevent the disorder of flora but also take measures to remove endotoxin. At present, the measures to antagonize endotoxin include (1) traditional Chinese medicine, such as honeysuckle, dandelion, Folium Isatidis, Houttuynia cordata, and other traditional Chinese medicine which can relieve internal heat and toxin; (2) endotoxin mAb, including E_5, which is obtained from mouse spleen cells sensitized with J_5 mutant *Escherichia coli*, an IgM that reacts to lipid A, and HA-IA, a human IgM antibody, which can specifically bind to lipid A. Early studies have confirmed that E_5 and HA-IA can combine with the endotoxin of a variety of Gram-negative bacilli and can reduce the mortality and the incidence of organ dysfunction under specific disease conditions. E_5 was effective in cases without shock, regardless of bacteremia; HA-IA is only effective for bacteremia, regardless of whether the patient is complicated with shock, but it is not effective for other types of infection. However, subsequent large-scale, multicenter randomized controlled studies failed to prove the clinical efficacy of E_5 and HA-IA. According to the available data, anti-endotoxin preparations cannot improve the prognosis of MODS patients, so we cannot expect too much about the clinical application prospect of anti-endotoxin preparations.

6.3 Neutralization and Antagonism of Inflammatory Mediators

This field has developed rapidly, including almost all the clarified media. It was once thought that the combination of bacteria endotoxin inflammatory mediators would be a new strategy for the treatment of MODS, but it was ineffective for inflammatory mediators that had been bound to the cell membrane and played a role. In addition, it can just clear the inflammatory mediators in the circulation and still has no effect on tissue damage caused by other complex mechanisms. Preparations mainly include (1) monoclonal antibod-

ies against TNF-α, IL-1, phospholipase A_2, C5a, etc. and adhesion molecule inhibitor; (2) receptor antagonists such as TNF-α, IL-1, PAF antagonist, etc., (3) prostaglandins, such as PGE_2, PGI_2, etc., (4) other inflammation inhibitors, such as C1 inhibitors, C5 blockers, arachidonic acid inhibitors, including cyclooxygenase inhibitors, thromboxane synthase inhibitors, lipoxygenase inhibitors, bradykinin inhibitors, oxygen-free radical scavengers, and protease inhibitors, and (5) thrombin regulators, such as antithrombin III, activated protein C, thrombomodulin, hirudin α_1-antitrypsin, aprotinin, soybean trypsin inhibitor, plasminogen activator, etc. The following is a brief introduction of inflammatory factor antagonists with clear effects.

6.3.1 TNF-α Monoclonal Antibody

Animal experiments and phase I clinical trials suggest that TNF-α monoclonal antibodies can improve the outcome of Gram-negative and -positive bacterial sepsis. However, the popularization and application of the preparation are limited by the following factors: (1) Because the half-life is short (14–18 min), TNF-α can be detected in the blood of only about 1/3 patients with septic shock, giving TNF-α to these patients may be too late. (2) Some studies have shown that elevated TNF-α levels alone do not cause shock. (3) TNF-α McAb is not effective in all cases. (4) At least low concentrations of TNF-α are a part of the normal body's defense function. The input of antibodies may cause more damage than benefit. The clinical application timing and precautions of TNF-α monoclonal antibody are worthy of in-depth study. At present, it has been successfully developed in China to antagonize both endotoxin and TNF-α Xuebijing, a traditional Chinese medicine injection with releasing effect, combined with antibiotics which can play the role of bacteria endotoxin inflammatory mediators.

6.3.2 IL-1 Receptor Antagonist

IL-1 is a polypeptide with a similar effect to TNF-α in the pathogenesis of septic shock. It is a vascular endothelial toxin, which can improve tissue sensitivity to TNF-α. In addition, IL-1 can activate cells and cause serious damage to the adrenal gland, intestine, and joints. The level of IL-1 in the blood of patients with sepsis has increased. It has been confirmed in animal and volunteer studies that low-dose IL-1 can induce sepsis. IL-1 has at least two forms, namely IL-1α and IL-1β. In sepsis, it is usually the increase of IL-1β. Their biological activities can be regulated at the receptor level. IL-1α and IL-1β receptor is a widely distributed cytoplasmic membrane glycoprotein. Any substance that can bind to the receptor can inhibit the effect of IL-1. At present, recombinant IL-1 receptor antagonist (IL-1ra) has been produced by recombinant technology and entered the stage of clinical verification after animal experiment verification. In the animal model of inflammation, where IL-1β was injected into rabbits, results showed that recombinant IL-1ra could inhibit IL-1 such as leucopenia, leukocyte aggregation, hypotension, and other biological effects of IL-1β dose-dependently. But so far, the randomized controlled clinical trials of IL-1ra have not obtained beneficial results.

6.3.3 PAF Receptor Antagonists

There are natural and synthetic preparations at present. Animal experiments show that these two kinds of preparations can prevent and treat endotoxin-induced pulmonary hypertension, oliguric renal failure, gastrointestinal damage, and brain blood flow reduction. WEB2086 has completed the second phase of the volunteer test and is undergoing clinical trial.

6.3.4 Arachidonic Acid Production Inhibitors

They can block the synthesis of TXA_2, which has been proved in animal experiments that can improve the survival rate of the endotoxemia model. It was found in healthy volunteers that it can inhibit the increase of body temperature and heart rate caused by endotoxin. In patients with severe sepsis, it can improve blood pressure, heart rate, body temperature, minute ventilation, and peak airway pressure and increase the probability of shock reversal. Ibuprofen has been used most, but the biggest limitation is its potential nephrotoxicity and the risk of gastrointestinal ulcers. At present, large-scale observation is being carried out to evaluate the efficacy and safety of ibuprofen. Montelukast sodium and zallukast approved by FDA are commonly used this year. They are powerful leukotriene receptor antagonists and belong to competitive inhibition. They can reduce the increase of vascular permeability caused by leukotrienes, reduce airway edema and eosinophil infiltration, and produce antiasthmatic effects. The second is nimesulide, which highly selectively inhibits the activity of cyclooxygenase II (COX-2) related to inflammatory prostaglandin synthesis without affecting cyclooxygenase I (COX-1) related to physiological prostaglandin synthesis in the stomach, kidney, and other organs, so as to ensure the anti-inflammatory effect and reduce the side effects on gastrointestinal tract and kidney.

6.3.5 Medications to Inhibit or Resist the Release of Toxic Mediators by Neutrophils

It is theoretically beneficial to inhibit or resist the release of toxic mediators by neutrophils because sepsis is endothelial damage and subsequent organ damage, which is related to activated neutrophil release mediators. At present, the most studied are pentoxifylline, ulinastatin, and neutrophil endothelial cell adhesion inhibitor, which can inhibit the activation of monocyte macrophages caused by stress response and reduce TNF-α and IL-1 secretion, inhibit the activation of neutrophils, indirectly reduce the adhesion reaction with

endothelium, reduce the production of oxygen free radicals, and inhibit platelet aggregation. The above mechanism can ensure the blood perfusion of important organs in sepsis and maintain the good function of vital organs, which may help to improve the survival rate of patients.

6.3.6 Antithrombin III

It can inactivate thrombin and inhibit the effects of plasmin, vasopressin, and coagulation factors IXa, Xa, XIa, and XIIa. Since thrombin can weaken the regulatory effect of PAF, stimulate the release of EDRF, PGI_2, and endothelin-1, and work as a chemoattractant of neutrophils, the effect of antithrombin III has exceeded its role in reducing contact and coagulation system. In the animal experimental sepsis model, thrombin III can reduce the damage of lung, metabolism, and blood system and improve the survival rate of animals. In the early small sample treatment of septic shock, it was confirmed that thrombin III could improve the generation rate of patients, but the subsequent large sample randomized controlled trials failed to prove its effectiveness.

6.3.7 Plasminogen Activators

Studies have explored the effect of plasminogen activators on enhancing fibrinolysis in patients with sepsis. For example, 30 ARDS patients with severe trauma and septic shock were treated with streptokinase (patients responded to oxygen supply and mechanical ventilation). As a result, the mean arterial oxygen partial pressure increased by 217 mmHg, and 14 of them survived. In addition to the risk of bleeding, the use of such preparations can cause the rebound of thrombin production.

6.3.8 Growth Factor

The generation of growth factors is synchronized with the development of internal organs, including liver, kidney, gastrointestinal tract, etc., where the embryonic development needs the stimulation of a variety of growth factors such as insulin-like growth factor (IGF), many cells in some mature organs such as liver and brain do not proliferate after maturation, but still maintain strong proliferation ability so that they can repair the injured organ with growth factors. Studies have confirmed that growth factors are widely distributed in various tissues, and their release is significantly reduced in tissue injury. Exogenous supplementation can promote the repair of tissue cells damaged by acute inflammation, ischemia and hypoxia, and ischemia-reperfusion.

6.4 Glucocorticoid

SIRS/MODS is considered to be a manifestation of excessive and severe inflammatory response. Glucocorticoids were tried clinically many years ago. Early studies found that the use of glucocorticoids in the treatment of patients with septic shock and severe ARDS can increase mortality due to secondary infection and other complications. In recent years, some trials have reached different conclusions. The treatment with low-dose and long-term glucocorticoid can not only quickly improve the symptoms but also significantly reduce mortality. Some scholars believe that glucocorticoids can protect the body from excessive inflammation and stabilize the internal environment by turning off the body's defense response. Inflammatory mediators such as TNF-α and IL-1 can activate the hypothalamic-pituitary-adrenal axis and promote glucocorticoid secretion. However, in SIRS patients, excessive immune defense response often makes glucocorticoid secretion relatively insufficient. A variety of inflammatory mediators can cause glucocorticoid resistance in a concentration-dependent manner. Activated hypothalamus pituitary adrenal can produce macrophage migration inhibitory factor (MIF), which can also inhibit the anti-inflammatory effect of glucocorticoid. The main basis for long-term glucocorticoid therapy is to supplement glucocorticoids that are relatively insufficient and play a declining role in the body, improve the response of target organs to glucocorticoids, regulate the level of intracellular transcription factors, and change the uncontrolled immune defense state of the body into controllable. However, there is no clear pharmacological evidence to determine the dose required for the so-called long-term glucocorticoid treatment, which can only be judged by experience. Here, there are also risks caused by hormone excesses, such as gastrointestinal bleeding and secondary infection. Further clinical research is needed to clarify the position of glucocorticoid in the treatment of SIRS/MODS. At present, most scholars believe that the combined use of seven to ten dexamethasone 10 mg/day, methylprednisolone 80–240 mg/day, or hydrocortisone 100–400 mg/day is helpful to reduce the excessive inflammatory response of patients and may improve the prognosis of patients.

6.5 Prevention and Treatment of DIC

At present, it is agreed that DIC plays an important role in the pathogenesis of MODS. Anticoagulation has become an important measure for the prevention and treatment of MODS. It should be used in the early stage of onset. At the same time, attention should be paid to correcting shock, supplementing blood volume, and protecting the functions of important organs.

6.5.1 Heparin or Low Molecular Weight Heparin

They can be used to prevent and treat DIC, especially for pregnant women. The earlier it is used, the more obvious the effect is. Plasma rich in fibrinogen, mechanically collected platelets, and fresh frozen plasma should be supplemented after DIC control. Antifibrinolytic drugs can also be used in

the later stage of DIC, but they must be applied on the basis of full heparinization. Studies have shown that low molecular weight heparin is better than unfractionated heparin in the treatment of DIC.

6.5.2 Anticoagulation with Recombinant Human Activated Protein C (rhAPC)

Hypercoagulation is an important pathological process of sepsis and forms a positive feedback mechanism with inflammatory reactions. Therefore, anticoagulation is an important link in the treatment of sepsis. APC is the first research report to make breakthrough progress. The study was conducted in 164 ICUs in 11 European and American countries, and a total of 1690 cases of sepsis were included. The results showed that the 28-day mortality was 24.7% in the treatment group and 30.8% in the control group ($P = 0.006$). Stratified analysis showed that the more serious the disease, the greater the benefit. However, in the clinical observation after listing, it was found that the risk of life-threatening severe bleeding in patients with rhAPC was significantly increased, which prompted the drug to withdraw from the market.

6.6 Supportive and Symptomatic Treatment

6.6.1 Supply Adequate Nutrition and Keep Homeostasis

An important feature of SIRS/MODS is high catabolism, leading to autophagy. In addition to taking measures to control body temperature and reduce body exertion, nutritional support is also an indispensable and important means to control hypercatabolism. Unless there are contraindications, enteral nutrition is the first choice for nutritional support, supplemented by parenteral nutrition if necessary. Proper enteral nutrition can prevent the loss of intestinal barrier function and the translocation of intestinal bacteria and endotoxin. In addition to the traditional three nutrients, a nutritional treatment emphasized in recent years is the application of glutamine. In case of emergency, glutamine, which is highly sensitive to glucocorticoids, is released from muscle and plays a role in maintaining intestinal integrity, supporting the immune system, and inhibiting muscle decomposition. Glutamine also has an antioxidant damage effect. Glutamine consumption increased significantly under the conditions of long-term hunger, surgical trauma, and SIRS/MODS. Traditional nutritional support could not meet the demand and needed additional supplements. At present, there is no clear regulation on the dosage. It is generally believed that at least 20–30 g/D can be supplied under severe stress, which is acceptable through intravenous or intestinal routes. In addition to nutritional support, attention should also be paid to correcting acid–base balance disorder and maintaining water–electrolyte balance.

6.6.2 Correct the Imbalance between Oxygen Supply and Demand

It can solve the contradiction between oxygen supply and demand. Ensuring adequate oxygen delivery is the basic guarantee for maintaining the normal aerobic metabolism of body tissues and preventing MODS. But it is not that the higher the oxygen delivery, the better. A multicenter clinical trial confirmed that supernormal oxygen delivery could reduce the mortality of patients with MODS, but cannot reduce the incidence of organ failure. Therefore, as long as the size of oxygen transport can ensure that the body does not produce anaerobic metabolism and maintain the function of important organs, too fierce intervention measures should not be taken to pursue too high oxygen transport.

6.6.3 Organ Function Support

In addition to medications, various artificial organ function support technologies and means play an important role in organ function support, such as maintaining effective blood volume and heart function, ventilator treatment of acute lung injury and respiratory failure, blood purification treatment of acute renal function injury, artificial liver support, prevention of stress ulcer, etc.

The pathophysiological mechanism of MODS is complex, the clinical manifestations are diverse, and many organs and tissues are involved. Although great progress has been made in basic research and clinical treatment in recent two decades, it is still a serious threat to human health. According to foreign literature reports, from 1970s to 1990s, the incidence of MODS was 14% and the mortality was 60%. By the end of the 1990s, the case fatality rate had dropped to 53%. In 2005, a prospective clinical study including 79 ICUs and 7000 patients reported that the incidence and mortality of MODS were 17% and 43%, respectively. It is believed that with the in-depth study of the pathogenesis of MODS, new therapeutic targets, therapeutic methods, and medications will be found in the follow-up, so as to further improve the prognosis of patients.

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Stress Disorder After Blast Injury

Jie Gao and Yamin Wu

1 Epidemiological Characteristics of Stress Disorder After Blast Injury

Post-blast injury stress disorder is a psychogenic mental disorder and reactive psychosis instigated by the explosion and includes acute stress disorder, post-traumatic stress disorder and adjustment disorder. It is a type of psychological disorder caused directly by a powerful psychologically traumatic event that has sustained for a period of time. Explosion-related stress disorder is caused by an explosion event and is characterized by a clear pathogenic stimulus. The clinical signs of this illness are closely associated with fright and injury from the explosion. This illness is accompanied by corresponding emotional experiences and is easily misunderstood by others, but could be healed after appropriate therapy. After therapeutic cure, patient's psychological state would return to normal and not exhibit any personality defects.

An explosion event and/or strong psychological trauma, or sustained difficulty and hardship, could all be the direct culprit of this illness. Statistics indicates that most victims of terrorist bombings range between the ages of 15 and 44 years, with the majority suffering from complicated and severe injuries that require surgeries in most cases. In a bombing, most personnel caught near the center of explosion would be afflicted with critical injury and die instantaneously, while 10–15% of survivors suffer severe injury, but generally will not end up dying. Analyzing 3357 terrorist attacks around the globe, and by comparing the mode of injury between victims of terrorist bombings and those in non-terrorist explosions, it was discovered that 29% of terrorist bombing victims incur serious injury, a figure far higher than those caught in non-terrorist explosions. Generally speaking, terrorist bombings might be followed by attacks from snipers, as this is a popular tactic among terrorists. In addition, they often set off a second explosion after law enforcement and medical care personnel arrive on-scene in order to create more mayhem and destruction. Terrorist attacks are characterized by a high rate of casualty due to head injury, accounting for some 50–70% of total. For head injury survivors, even though they have escaped life-threatening injuries, many of them are very likely to end up with stress disorders. In general, these stimuli can be divided into the following several situations: (1) serious explosion event or terrorist bombing; (2) witnessing or participation in on-site rescue or clean-up of the scene of explosion; or (3) unavoidable, brutal warfare. According to reports in literature, more than 50% of the regular population has been exposed to at least one traumatic event in their life, and not all trauma survivors demonstrate stress disorder. Due to the wide variations in different research, application, and evaluation methods, sample collection methods, and the types of trauma and population involved, the occurrence rate of acute stress disorder is reported to be between 6% and 33%. Meanwhile, that of post-traumatic stress disorder (PTSD) in the normal population ranges from 7% to 12%, and females have a 51.2% rate of exposure to trauma and a PTSD prevalence rate of 6.07%, and males have a PTSD prevalence rate of 5%. The lifetime prevalence of PTSD in female victims is higher than that in male victims, which is related to gender difference and the type of traumatic event experienced before. Research reveals that gender differences in reaction to traumatic events cannot be explained by exposure circumstances of traumatic events and instead should be explained by gender-determined attribution to traumatic time. Male victims are more prone to substance abuse and anti-social personality disorders than female victims.

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2 Pathogenetic Mechanism of Stress Disorder After Blast Injury

Kaplan sums up and divides the consequence of sudden and immense stress into three phases. Phase 1 is the shock. When an individual is put under stress, he or she would be thrust into a state of loss and shock, manifested as some degree of disorientation and attention deficit that could last from several minutes to a couple of hours. Phase 2 is characterized by clear confusion, ambiguity, and changeability, accompanied by emotional disorders such as irritation, anxiety, and depression. Phase 3 is long-term reconstruction and return to balance. According to the Pavlov school of thought, the course of effect of a dramatic and powerful stimulus on higher nervous activity could excite or inhibit the over-nervousness and conflict in emotions. Meanwhile, in a bid to avoid further damage or breakage, the central nervous system would always resort to trans-marginal inhibition. This trans-marginal inhibition mechanism originated from protective inhibition. During the spread of the inhibition process, the functions of some lower-level parts in the central nervous system, such as certain unconditioned reflexes, would be released from inhibition. This would result in various forms of abnormal interaction between cortex and subcortical activities. Clinically, issues may manifest as emotional responses uncontrolled by consciousness, random behaviors without purpose, and primitive responses. In general, it is believed that hereditary factors are not influential in stress disorders.

In experiment and research, Selwyn et al. utilized hydraulic impact on rats to simulate blast injuries and used electron emission tomography to evaluate inhibition of glucose metabolism and uptake in the brain, discovering that peak inhibition occurred 1 day after explosion stress and returned to normalcy 16 days after stress. After adult rats experienced two explosion-related stress, the activations of microglia astrocytes, microglia cells of the nervous system, and astrocyte cells are closely associated with the second blast injury. Therefore, glucose metabolism in the brain has a direct impact on nervous system damage induced by an explosion. Another literature reported testing with injury caused by an explosion in mice, in which C57BL/6 mice were placed in shock tubes and afflicted with shock waves once or thrice at mild intensity (0, 2, and 24 h) as per requirements. It was discovered that the cerebellum showed a decreased density of cerebellar myelin after 1, 7, 30, and 90 days, and shrunken and deformed Purkinje cells were found after 90 days. Therefore, the authors opined that behavioral abnormalities and cognitive injuries might be the result of damaged cerebellum due to explosion. Hoffman et al. carried out an experiment, using hydraulic impact applied on adult male rats on one side of the head to create brain injury and simulate blast injury, and found out that electroconvulsive white noise could enhance the expression of immediate early genes in the lateral amygdala. This indicates that explosion-induced stress affects the processing of fear information and coding of traumatic emotions.

Grisbach et al. found out that the starting 2 weeks of mild traumatic brain injury usually constitutes the acute stress stage. Due to the high intensity, often times the patient would exhibit acute stress disorder. By afflicting mild blast injury on rats for experimentation, it was discovered that timely sports activities could effectively mitigate stress damage, as manifested in the normal function of the norepinephrotropic axis in the rats, without an increase in the secretion of a brain-derived neurotrophic factor in the hippocampus. Valiyaveettil et al. subjected rats to three repeated explosions and studied and discovered that cytoskeletal protein caspase-3/2 in the frontal lobe and temporal lobe exhibited abnormal expressions, and there was degradation in the contractile protein a-II, revealing that damage to cytoskeletal protein resulted in damage to axons, and consequently emotional and behavioral abnormalities.

3 Categories and Diagnosis of Clinical Stress Disorders After Blast Injury

The categories of clinical stress disorders after blast injury are basically the same as general stress disorders, with the majority being acute or subacute onset and a relatively short period before reaching the full development stage. The general categories are as listed below:

3.1 Acute Stress Disorder

Stress stimulus is clear, as in psychological impact directly induced by an explosion event. Due to the fierceness of the event, psychological symptoms would appear within a few minutes or several hours after the explosion stimulation. The duration is short, and usually, patients would recover within several days to a week's time, with a good prognosis.

3.1.1 Clinical Signs

1. Cognitive disorder: Patients exhibit different degrees of cognitive disorder, but deranged psychological state is very common. Disorientation and attention disorders may be observed, along with difficulty in carrying out dialogues, talking to oneself, disorganized and incoherent speech that are hard to understand, random and purposeless actions, and impulsive behaviors on occasions. After

recovery, a small percentage of patients might exhibit forgetfulness.
2. Psychological disorders: Patients might exhibit excitement or inhibition in psychomotor activities accompanied by strong emotional feelings. Excited psychomotor activities are characterized by lack of coordination, irritation, screaming, random and erratic motions, purposeless roaming, and speaking about things related to elements in the explosion event or personal experience. Emotional outbursts are also observed on occasions. There could also be limb twitches, similar to hysteria. Inhibited psychological activities are rarer, which may be expressed as avoidance, silence, emotional indifference, lack of response, sitting or lying down aimlessly for an extensive period, and lack of emotional expressions. These might be accompanied by increased heartbeat, sweating, flushing of the skin, and other nervous system symptoms.

3.1.2 CCMD-3 Diagnosis Standards

1. Symptom standard: Serious and extraordinary psychological stimulus that resulted in at least one of the following: (1) psychomotor excitement with an intense experience of fear and behaviors with a certain degree of aimlessness; (2) psychomotor inhibition of emotional retardation (such as reactive stupor) and could also demonstrate mild cognitive disorder.
2. Serious standard: Serious defect in social functions.
3. Disease course: Onset within a few minutes or several hours after stimulation, and a short disease course that usually lasts several hours to a week, then allayed and usually recovered within a month.
4. Exclusion criteria: Rule out hysteria, organic mental disorders, or mental disorders and depression caused by non-addictive substances.

3.1.3 Differential Diagnosis

1. Acute organic brain syndrome: Examples such as toxic psychological disorder or delirious state exhibit similar clinical signs, but this type of disorder mostly feature visual hallucinations and fluctuations in clear and unclear consciousness. In addition, there are corresponding positive signs, and abnormal laboratory tests (electroencephalogram and CT) may be used for identification.
2. Emotional disorder: Onset under a certain stimulus, manifestations of coordination psychomotor excitability and inhibition can be identified, the cognitive disorder is rare, disease course is long, the extensiveness of mania and depression can assist with identification.
3. Hysteria: Onset under certain social psychological stimuli, patients might exhibit pretentiousness and demonstrate a self-centric mentality before the illness. Illness is full of fantasizing and could repeatedly occur, and one key element is that patients are very suggestible.

3.2 Post-traumatic Stress Disorder

Post-traumatic stress disorder is a type of psychological and behavioral disorder related to war or blast injuries. In the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* issued by the American Psychiatric Association (APA), post-traumatic stress disorder (PTSD) is categorized as a trauma-related and stress-related disorder. Diagnosis emphasizes that PTSD must be associated with a clear traumatic experience. PTSD has three key types of symptoms: pathological re-experience (intrusion), avoidance, and increased alarm, and the patient's social and vocational capacity, interpersonal communication capacity, daily life capacity, and recreational capacity all suffer different degrees of serious damage. Sometimes other symptoms might be seen in accompaniment, such as pain in the torso, alcohol abuse, or a combination of other psychological disorders. Clinically, there are three categories: acute, with a disease course less than 3 months; chronic, with disease course ranging between 3 and 6 months; and delayed, which may also be called delayed psychogenic response. There is a latent period between the explosion experience and the onset of psychological symptoms, usually ranging from a few weeks to several months, or might not even begin to show 6 months after the explosion stress took place.

In a serious explosion, it is common for victims to die right on the spot, but survivors are often left with arrhythmia, nerve damage, retinal artery air embolism, and other injuries. Injuries might not be seen on the body surface of other survivors, and they might only be afflicted with wounds and problems like the pierced eardrum, flash blindness, and acute dyspnea, which might be coupled with non-critical external injuries. The injuries of these patients might stabilize early on, but medical personnel cannot relax because conditions might rapidly deteriorate. On-site rescues at explosion scenes indicate that related stress psychological disorders may be divided into two types, namely stress psychological disorder resulting from blast injury to the head, and stress psychological disorder resulting from blast injury to elsewhere on the body.

3.2.1 Post-traumatic Stress Disorder from Blast Injury to the Head

Traumatic brain injury is the most common injury that could lead to death, with 71% of victims dying in the early stage. Patients with brain injuries will exhibit a series of clinical signs afterward, such as loss of consciousness, agitation, excitement, irrational behavior, etc. The relatively long-term grade I post-injury complications and post-traumatic syndrome are related to traumatic brain injuries caused by an explosion. Mild traumatic brain injuries caused by the light explosion might not exhibit clinical signs, but as time progresses, cognitive function and cognitive could reduce,

particularly victims that have been repeatedly exposed to explosion site(s). This kind of repeated exposure and contact with explosion site(s) could lead to PTSD. Moderate and severe traumatic brain injuries will seriously affect the overall body condition of patients. In mild brain traumas, changes to short-term or long-term cognitive ability constitute a matter of utmost urgency and importance in times of war. Mild traumatic brain injuries could incur from blunt trauma to the head or overpressure of a blast wave, in particular history of repeated contact with shock waves. The US Department of Defense has devised Military Acute Concussion Evaluation (MACE) that can help commanders and medics carry out a preliminary assessment of the cognitive capacity of the injured. In conjunction with the bodily health status of the injured, MACE makes it possible to determine whether the injured can continue participating in combat from a tactical standpoint. European and North American military experiences indicate that head trauma is a common injury in battle. During the Vietnam War, 15–20% of US soldier casualties could be attributed to head injuries. Although improvements and upgrades had been made to protective measures, external injuries to the head still accounted for 15% of military injuries during the Gulf War. In modern society, head injuries caused by explosions may be divided into either closed-head injury or penetrating craniocerebral injury. In addition, there is also brain dysfunction that arises as a consequence of exposure to an explosion. This is mainly the outcome of the force of the explosion penetrating into and wounding the brain parenchyma. Overpressure, electromagnetic energy, sound, and other factors are potential causes of illness. It is also directly related to the intensity of high-power explosions. The severity of traumatic brain injuries can be divided into mild, moderate, or severe. A mild traumatic brain injury refers to loss of senses or consciousness for a short period (<5 min), and victims usually suffer from headaches, confusion, and memory loss. Other symptoms include difficulty concentrating, emotional fluctuations, sleep disorder, and anxiety. The aforesaid symptoms should allay within several hours or a couple of days. Signs of delayed consciousness syndrome might appear.

A concussion is a sub-type of closed-head injury and may be categorized as mild (grade 1), moderate (grade 2), or severe (grade 3), with grades divided based on increments of 15 min of injury. For many of the soldiers exposed to blast impact for the first time, they do not realize that they might have been injured and do not seek medical attention in a timely manner. It would take subsequent exposures to blast impact for them to learn that they have been injured. Most patients complain about symptoms typical of continuous exposure to shocks, such as headache, dizziness, short-term memory loss, or difficulty in concentration. Since the symptoms stated above are relatively hard to discern, patients should receive full-spectrum assessments by physicians or psychologists. Mild traumatic brain injuries, if coupled with PTSD, are difficult to cure. The signs and symptoms of the two are very similar clinically. Shared symptoms include difficulty in concentration, emotional fluctuations, sleep loss, and a tendency for anxiety. Therefore, soldiers need to receive an assessment from stress task forces or psychologists at the theater of battle. If a traumatic brain injury is suspected, the individual should be evacuated until his or her symptoms subside. The individual should also be evacuated to a high-level medical institution equipped with neuroimaging and other detailed assessment capacities.

Mild traumatic brain injuries arising from an explosion often overlap with PTSD. Reid et al. studied 573 active US troops that have experienced multiple explosions (four at the most), discovering a positive correlation between each individual's PTSD Checklist score and his or her cumulative explosion experience. Elder GA et al. subjected male rats to anesthesia and then repeated explosion shock and discovered PTSD symptoms in rats, including increased anxiety, increased fear for certain scenarios, and changes in odor identification. Therefore, the author is of the opinion that outcomes of blast injuries administered under anesthesia indicate explosions can induce PTSD without any psychological stress. This experiment is helpful in estimating and understanding the ratio of Iraq and Afghanistan veterans with moderate or mild PTSD.

3.2.2 Post-traumatic Stress Disorder from Blast Injury Without Head Injury

Blast injury is divided into five levels of severity, and multi-dimensional injury after an explosion refers to complicated blast injuries in which multiple parts of the body are affected, including blunt trauma and penetrating wounds arising from projectiles launched by the explosion, body part and organ displacement caused by the explosion, primary lung blast injury or intestine rupture, burns from the fireball of the explosion, composite effects from chemical or radioactive substances inside the bomb, etc. Multi-location, multilayer, and multi-organ effects dramatically worsen the severity of such composite injuries, which are always incredibly challenging for first-aid medics.

Injuring Mechanism

A considerable number of research studies have reported brain dysfunction due to explosion-related brain trauma, with the underlying mechanism being injury from the mutual effect between impact and the body (as in how the body was injured). Primary injuries are direct injuries to the head, and the other type is one shock wave acting on the entire body. After the whole body is afflicted with blast injury, gross anatomy outcomes reveal severe lesions in the lungs such as hemorrhage and edema, but no obvious organic changes were

found in the brain tissue. This indicates that the severity of central nervous system changes induced by exposure to full-body shock waves is clearly less than injuries incurred by air-filled organs (mostly the lungs), and possibly functional damage is the main problem. Rats could suffer from damage in cognitive capacity equal to emotional disorders in humans. The author deduces that a possible mechanism is an excess of nitric oxide in the brain after blast injury. Nitric oxide, as an important regulatory biomolecule, directly affects and regulates the presynaptic transmitters and postsynaptic reverse signaling in areas such as information transmission and synaptic plasticity. Mechanism of post-explosion injury to the blood–brain barrier (BBB) first starts with a direct effect of a shock wave, which destroys tight junction, leading to cerebral edema, exudation, and other vascular response changes, as well as degradation and damage to some brain tissues, resulting in necrosis in the minority. The plasma membrane on the inner surface of vascular endothelial cells is the initial target of the shock wave's effect on the brain. In particular, the shock wave's overpressure will seriously damage the tight junctions between the endothelial cells, destroying the blood–brain barrier and damaging the brain. As time elapses after the explosion (1/4 to 48 h), the blood–brain barrier gradually recovers, gaps between nerve shells slowly shrink, and water content in the brain tissue also reduces, demonstrating that cerebral edema is gradually subsiding, and the obvious time factor and reversibility of increase in permeability of blood–brain barrier. This outcome shows that if explosion victims are not treated promptly, reversible and unstable brain tissues might degenerate further or even result in necrosis, and consequently, further damage to the blood–brain barrier. This would worsen the chaos in the internal environment of the brain tissue, leading to secondary damage to the brain. As time progresses further after the explosion, damage to nerve cells in brain tissue would exacerbate, indicating that other than an increase in permeability of the blood–brain barrier, brain damage might also be associated with local tissue ischemia, biochemical changes due to hypoxia, changes in vasoactive substances, free radical production, and other reasons. In addition, the longer the blood–brain barrier stays open, the worse the brain damage.

Clinical Manifestations

Repeated traumatic experience, repeated occurrence of dreams and nightmares related to the explosion, unwillingness to make contact with others, lack of response to the surrounding environment, loss of sense of joy, and avoidance of memories of scenarios or activities related to the explosion are common. In addition, there could also be signs such as a state of high alertness and startle response, coupled with the loss of sleep, anxiety, or depression. Most patients can recover on their own, but for a minority, chronic illness might occur and could last for several years, long enough to alter personality. Due to the delayed onset of PTSD, its symptoms and signs only reveal themselves over time. In faster cases, symptoms and signs might appear within a few days, but others might take months, while the disorder itself could span a few years or even stay with the patient for life. Usually, a triad is the main manifestation: reexperience of a traumatic situation, a sustained state of high alertness and avoidance behaviors, selective forgetfulness about the traumatic experience, and loss of confidence for the future. Generally, requirements include patient to have experienced an explosion on-site and in-person, not demonstrating manifestations of parenchymal brain injury from an explosion, and typical symptoms observed continuously for at least 1 month (usually within 6 months). It is also necessary to rule out emotional and psychological disorders, other stress disorders, neurotic disorders, physical disorders, etc. At the same time, a clinical PTSD Checklist should be utilized. In China, the Chinese version of the PTSD Checklist translated by Prof. Li Lingjiang and Prof. Shi Jiaqi is reputable as highly effective. Moreover, event-related potentials (ERP), startled response, and other techniques may be employed to aid with diagnosis. During animal testing for PTSD research, the author discovered abnormal sensitivity to pain, showing that changes in pain threshold are also a relatively simple assistant indicator to help diagnosis.

Diagnosis Standards

Individuals should have experienced an explosion event and said event ought to be an extraordinarily traumatic situation or condition for the person. Repeatedly experiencing the trauma associated with the explosion (flashback) and meeting at least one of the following conditions: (1) involuntarily thinking about the tormenting experience of the explosion stress; (2) repeated appearance of nightmares with traumatic contents; (3) repeated occurrence of delusions and hallucinations; or (4) repeated emotional pain triggered by situations such as seeing an explosion, visiting the site of an explosion, or hearing about news or reports about related circumstances, leading to extraordinary pain and obvious physiological responses (palpitation, sweating, pallor, etc.). A sustained state of increased alertness and meeting at least one of the following conditions: (1) difficulty in falling asleep or unable to achieve deep sleep; (2) easy agitation; (3) difficulty in concentrating; or (4) excessively alarmed or anxious. Avoidance of similar stimuli or related scenario alertness and meeting at least two of the following conditions: (1) endeavoring to not think about people or events related to the traumatic experience; (2) avoiding participation in any activities that might trigger painful memories, or avoiding visits to places that might trigger painful memories; (3) unwillingness to interact with others, and showing unconcern to families; (4) narrowed area of interest, but remains interested in some activities unrelated to the traumatic experience of the explosion; (5) selective amnesia; or (6) loss of hope and confidence about the future.

Serious standard: Deficiency in social functions.

Disease course: Delayed occurrence of psychological illness (onset should take place a few days or several months after the trauma, and in rare cases, delays of more than half a year), and meeting criteria for at least 3 months.

Exclusion criteria: Rule out emotional, psychological disorders, other stress disorders, neurotic disorders, physical disorders, etc.

Differential Diagnosis

1. Depression: Having had a sad and painful experience and lack of emotion, but the two differ in that the depressiveness in depression usually encompasses a wide range of areas including numerous interests and hobbies, or the future prospect of the individual, instead of a fixed stimulus time or event. Depression is also accompanied by pessimism, self-abasement, or suicidal intentions and behaviors. Another telltale sign of depression is that it is usually more severe during the day and less so at night.
2. Anxiety neurosis: People with anxiety neurosis are often worried about one's own health and complain about physical problems. Some might even demonstrate hypochondriac tendencies. There is also no clear pathological factor for psychological trauma.

3.2.3 Post-blast Injury Adjustment Disorder

This order is mainly manifested as worries, depression, or other emotional disorders, along with behavioral disorder or physiological functional disorder of poor adjustment, arising from susceptible personality and weak adaptiveness to the stress of an explosion event. This illness results in a functional deficiency in areas such as learning, work, daily life, and interpersonal communication. The disease course is quite long, but usually no more than 6 months. Often the onset of the illness occurs within a month after the stressful explosion event or is accompanied by changes in daily life as a consequence of the said event. With elapse in time or changes in the environment, the patient would adjust as the stimulus alleviates or after making adjustments. In turn, psychological disorders would subside. Therefore, ICD-10 divides the disorder into eight child categories, namely short-term adjustment disorder with depressed mood, long-term adjustment disorder with depressed mood, adjustment disorder with mixed anxiety and depressed mood, adjustment disorder with other symptoms, adjustment disorder with disturbance of conduct, adjustment disorder with mixed disturbance of emotions and conduct, and adjustment disorder with unspecified details. This illness could occur in a person of any age group after experiencing an explosion, and there is no gender difference, although there are reports that state females are more susceptible.

Etiology and Pathogenesis

1. Stimulus from explosion event: Since the explosion event is clear, so the stimulus of adjustment disorder is clear. However, the severity of adjustment disorder is closely associated with the intensity, nature, timing, reversibility, and other factors of the explosion event, though the severity is more conditional on the individual's subjective understanding and experience of the explosion event.
2. Susceptibility of the individual: Personality deficiency, as in adjustment, is usually highly contingent on personality, and those with a sensitive, cowardly, stubborn, or suspicious nature often adjust more poorly. People with kind of personality often exhibit an adverse psychological reaction to stress in the face of stimulation from serious pressure, especially in life-threatening circumstances, resulting in poor adjustment behaviors.
3. Stress response capacity: In general, an individual's response to environmental stress is consistent. Therefore, those that lack stress response capacity might exhibit poor adjustment behaviors when dealing with a stressor.
4. Basic physiological health of the individual: The bodily health of an individual also has a huge impact on his or her adjustment to the environment. When an explosion occurs, due to intense physical, chemical, and situational stimuli, if an individual is already suffering cerebrovascular diseases or heart diseases, or if the person is already pregnant, is handicapped (which would hamper his or her ability to save oneself) or affected by other conditions, it would substantially hinder the individual's capacity to deal with the situation.
5. Other factors: Solid social support and love and care from the family can provide a beneficial sense of security. After a person experiences a major disaster, interpersonal relationships, support systems from society and family, and other factors all play crucial roles in the individual's ability to cope with the powerful psychological crisis.

Clinical Categories and Diagnosis

1. Adjustment disorder with depressed mood: After experiencing an explosion event, it is common to see the individual in a depressed mood, who might feel despair, cry, exhibit dejection, or other depressed states, though the severity is less than depression.
2. Adjustment disorder with anxiety: After experiencing an explosion event, the survivor mainly exhibits psychological over-sensitivity, annoyance, and agitation, nervousness, and restlessness with regard to different kinds of changes.
3. Adjustment disorder with mixed anxiety and depressed mood: An individual might exhibit a combination of depression, anxiety, and other emotional abnormalities, though the severity is less than anxiety disorder and depression.
4. Adjustment disorder with disturbance of conduct: An individual might behave inappropriately, such as playing

truant, skipping class, leaving work without permission, damaging public property, getting into fights, refusal to perform legal responsibilities, and other misconducts that run contrary to social norms and rules.
5. Adjustment disorder with mixed disturbance of emotions and conduct: Similar to subtype (4) stated above, coupled with emotional abnormalities.
6. Adjustment disorder with physical and bodily complaints: An individual might complain about headache, lethargy, back pain, and other discomforts throughout the body, and medical examination should be conducted to rule out any organic disease.
7. Adjustment disorder with work issues: Inability to work normally due to impacts from the explosion, but the patient does not show symptoms such as depression, anxiety, or fear.
8. Adjustment disorder with withdrawal: Individual demonstrates a tendency to withdraw from or avoid social activities but without emotional symptoms like anxiety or depression.

CCMD-3 Diagnosis Standards

Symptom Standard

1. Confirmed cause in a personal experience of explosion event: Using the effects of the explosion event to deduce manifestations in life and personality as the basis is crucial to a psychological disorder. Main manifestations are depressive mood, anxiety, fear, and other emotional symptoms and meeting at least one of the following conditions: (1) Behavioral disorder from poor adjustments such as withdrawal, lack of personal hygiene, irregular daily habits, etc. or (2) physiological function disorders such as poor sleep, lack of appetite, etc.
2. Emotional, psychological disorder (excluding delusion and hallucination), neurosis, stress disorder, somatoform disorder, or various symptoms of conduct disorder, but do not meet the aforementioned diagnosis standards.
3. *Serious standard*: Deficiency in social functions.
   (a) Disease course: Psychological disorder starts within 1 month after a social psychological stimulus (the consequence of the explosion event was not disastrous or extraordinary) and meets symptoms and standards for at least 1 month. After alleviation of stress factors, symptoms usually will not persist for longer than 6 months.
   (b) Exclusion criteria: Rule out emotional, psychological disorder, neurosis, somatoform disorder, conduct disorder, etc.

Differential Diagnosis

1. PTSD: Identify stress disorder after specific trauma. In some serious cases, patients might show intense response to anything in life, such as a flood, earthquake, or airplane crash. If the explosion event ended with three or more deaths, the symptom is serious. Recurrence probability is rather high.
2. Depression: Generally speaking, the depressive mood is very severe, including self-abasement and pessimism, or even suicidal tendency or behavior. In addition, clinically speaking, depression changes between day and night, and this could also be used as a reference for identification.
3. Anxiety disorder: Anxiety disorder is manifested as sustained and widespread worries, accompanied by symptoms of autonomic nervous system disorders, obvious sleep disorder, long disease course, and usually there is not a clear and strong stressor.
4. Personality disorder: It is a key factor in triggering adjustment disorder and could be aggravated by a stressor. Yet, a personality disorder is already obvious before maturity, and therefore, stressor is not the main cause of personality disorder. Abnormal expressions of personality disorder could continue to adulthood or even remain for the entire lifetime.

4 Post-blast Injury Stress Disorder in Special Environments

Based on the medium of propagation, blast injury could be classified as a gas explosion, underwater explosion, or solid explosion. An explosion could occur in locations like plateau, desert, underwater, high-altitude and cold areas, etc. These explosions in different environments and mediums differ in some characteristics. Injury caused by a mid-air explosion is air blast injury; blast waves from the underwater detonation of mine, depth charge, or underwater nuclear weapon detonation cause underwater blast injury. The injuring mechanism of underwater blast injury is more or less the same as air blast injury, but the speed of propagation and lethal range of a shock wave underwater is about three to nine times more than a shock wave in the air. Water is 800 times denser than air and could be compressed at least 10,000 times more. Personnel in the shallow area or sleeping would be affected directly by not only the explosion but also the impacts of reflection waves coming from the ground surface, seabed, or other surrounding structures. The power of an underwater explosion from the same explosive is three times more powerful than that on the ground, and the deeper the center of the explosion, the bigger the effect. Pulmonary hemorrhage is the most common, and then that of the intestines, while solid organs in the abdomen receive the least damage. Solid blast injuries are predominantly afflicted through the propagation of depth charge's blast wave through the structure of a vessel like a deck. Due to the high frequency of secondary displacement, head and brain injuries are common. High plateau blast injuries are those that occur

at areas 3000 m or more above sea level, where the human body is obviously affected biologically. High plateau blast injury is characterized by mild injury on the outside and severe injury on the inside. In experimentation, no injuries were found on the surface of all survived and killed rats, but visible lung hemorrhage or edema was seen in 97.5% of rats in the injured group. For instance, in recent years, such injuries frequently occur in enclosed environments such as buildings, public vehicles, or public venues (such as the Kunming Railway Station explosion on March 11, 2014), as well as car bombs set up by terrorists. This kind of blast wave in enclosed space generates a special kind of shock wave that leads to more serious injuries than regular blast injuries. Chinese scientists have experimented with simulated explosions involving armored personnel carriers and observed that although rats in the driver and passenger seats remained conscious, capable of movement, and unscathed on the surface within 30 min after the explosion, an hour later, physical action disorder was seen in some parts of the body, they still gradually recovered. Comparatively speaking, brain function effects from blast impact in open space only slowly emerged after 8 h, manifested as abnormal cerebral evoked potential. Based on animal experimentation, the author opines that waveform and energy of a series of complicated impulses created from blast wave are more directly transferred to those exposed inside an enclosed space than those at an open space, and this is an increasingly common circumstance at present because of the rising frequency of terrorist attacks using IEDs. From past terrorist attacks to recent cases of terrorist bombings in Paris and Turkish cities, the deadly outcomes and the consequent psychological disorders are worthy of the attention of medical care professionals. Therefore, stress disorders induced from explosion events in such environments demand even more timely treatment than other similar disorders.

5 Treatment of Post-blast Injury Stress Disorder

5.1 Treatment of Post-blast Injury Acute Stress Disorder and PTSD

5.1.1 Psychological Therapy

Stress disorder arising from blast injury is obviously stimulated by stress from the explosion, which is why psychological therapy is important. The therapist assigned needs to build a sound doctor–patient relationship with the individual in question, analyze the pathogenesis with the patient, then explain the situation, and instruct the patient on how to properly deal with the explosion stimulus. The therapist also needs to emphasize that powerful stimuli exist throughout daily life, with the crux of the matter being how to deal with them correctly, and at the same time, provide adequate social support, actively encourage the subjective impetus of the patient, help the person free himself or herself from troubles as soon as possible, and establish the conviction in combating and overcoming the illness so as to promote patient recovery. Techniques and methods such as cognition, simulated scenario, sand box, positive mentality, mindfulness, desensitization to exposure, virtual video, cognitive behavioral therapy, Eye Movement Desensitization and Reprocessing (EMDR), interpersonal psychotherapy (IPT), family therapy, hypnosis, and Gestalt psychology produce different efficacy depending on the individual. In addition, there are two other novel types of simple and applicable therapeutic techniques: Connect-the-dots and staircase training.

Connect-the-dots technique: Instruct the disaster survivor to draw a map of coordinates based on time sequence, with the *x*-axis being age. The map should clearly illustrate adjustments or maladjustment to challenges of past and present, including what methods were used, then select the positive coping methods, which would be conducive to the patient's recovery.

The staircase training technique is closely connected with the previous technique by building upon the lessons learned before and then envisioning the future. Specifically, instruct the survivor to draw a simple staircase with a couple of steps on a blank piece of paper or board. The platform at the top is the long-term goal, and the platform at the bottom is the short-term target, then sequentially add the intervention techniques in the middle steps. The entire plan must be realistic yet flexible and requires all members participating in the training to provide a meaningful plan to the other members. This approach allows them to review and learn from each other. This method is very helpful in strengthening the self-assessment, self-knowledge, and self-esteem of an individual.

The mindfulness-based stress reduction (MBSR) technique was invented by Dr. Jon Kabat-Zinn of the Center for Mindfulness in Medicine, Health Care, and Society at the University of Massachusetts Medical School. MBSR is employed as a supplemental tool (not a replacement) for regular medical treatment, and patients treated using this therapy are usually afflicted by their own different physiological or psychological illnesses. The aim of MBSR is to educate patients to utilize their own physical and mental power in fostering and cementing the correct mindset beneficial to one's bodily and emotional health. A 2015 article in the American *JAMA* magazine reports that 116 veterans in Minneapolis with PTSD were treated with mindfulness-based intervention methods, and the outcome showed PTSD improvements in 63.6% of the cases, markedly better than group therapy. The author proposes with reserve that the mindfulness-oriented approach might be more accepted by veterans with PTSD, which was why symptoms were allayed.

5.1.2 Environment Therapy

Strive to remove the patient from an illness-inducing environment or modify the environment, which would be hugely favorable to the whole therapy process. The other facet of environment therapy is to provide guidance and assistance helpful to the patient's future life and work. Specific aspects include rearranging the life pattern of the patient, fostering interest in work, and helping the individual improve interpersonal relationships, among other areas.

5.1.3 Drug Therapy

First and foremost is to ensure the patient gets adequate sleep, and anxiolytics and hypnotics may be prescribed to help those with anxiety or vexation so as to help improve their sleep and mitigate worries. Common drug prescriptions include alprazolam, estazolam, and clonazepam, among other medications. These are all applicable, but be mindful of long-term usage or excessive dosage.

For patients suffering chiefly from psychomotor excitement and the need to suppress delusion or emotional disorders, antipsychotic drugs or antidepressants may be prescribed carefully, with dosage-dependent on symptoms but usually not too big.

5.1.4 Transcranial Magnetic Stimulation

The stimulus location and treatment parameters of acute stress disorder are located at the dorsolateral prefrontal cortex. Stimulus parameter may be set at 1 Hz, 100% motor threshold, 60 s/string, 5 strings/day at 60 s intervals. Five days constitute one cycle and administer two cycles in total. Contraindications of this therapy include space-occupying lesion of the brain and other lesions that raises intracranial pressure. Those that have suffered from intracranial hemorrhage recently or those with heart attacks due to unstable heart functions are also unsuitable for this therapy, as are patients fitted with biomedical devices such as a cardiac pacemaker. Furthermore, high-frequency stimulation is not appropriate for people with epilepsy.

The stimulus location and treatment parameters of PTSD are located at the dorsolateral prefrontal cortex. Stimulus parameter may be set at 1 Hz, 100% motor threshold, 60 s/bunch, 5 bunches/day at 60 s intervals. Five days constitute one cycle and administer four cycles in total.

5.1.5 Other Therapies

Work and recreational therapy, biological feedback, and other techniques also offer a certain amount of benefits for emotion stabilization, sleep improvement, and amelioration of bodily discomfort, among other areas. In addition, for those that do not eat voluntarily or consume too little, actively provide support therapy such as IV injection or nutritional supplements. Recently, the US began the application of infrared/near-infrared light-emitting diodes (parameters of cluster LED include 5.35 cm diameter, 500 mW, and 22.2 mW/cm^2) around the scalp. LEDs are placed at 11 locations near the scalp for 10 min of stimulation each and results so far prove that this technique is effective in improving sleep and reducing post-traumatic stress disorder symptoms. This experiment is currently in an open protocol testing phase, and further progress is anticipated. On a different note, there have been reports that Danqi capsules and lycium barbarum polysaccharide, an extract of *Lycium barbarum* (Chinese wolfberry), can engender recovery from psychological disorders and cognitive function problems associated with brain trauma by promoting improvements in neuroprotection and neurodegeneration. Road et al. studied and concluded that the central nervous system's autoantibodies are released into the peripheral circulation and react with specific proteins, and its unique mode of effect has a lot of real-world prospects in providing effective treatment for nerve damage. In addition, it could be utilized as a candidate marker of pathological components to detect and assess the potential targets of central nervous system injuries of different extents.

5.2 Treatment of Post-blast Injury Adjustment Disorder

5.2.1 Psychological Therapy

Psychological therapy is the main approach for post-blast injury adjustment disorder and has positive effects on poor adjustment behaviors and improving social functions. Common techniques include cognitive therapy, behavioral therapy (relaxation system, system desensitization, etc.), and psychological Catharsis therapy, among other options, which may be performed in individual, collective, or familial modes.

5.2.2 Drug Therapy

If patients exhibit severe symptoms, such as suicidal behavior, violence, or other critical states, prescribe medication in conjunction with psychological therapy. Use minimum dosage whenever possible, and halt medication once symptoms subside. Common types of drugs include anxiolytics and antidepressants, and when necessary antipsychotic drugs are applied in accordance with specific symptoms.

5.2.3 Transcranial Magnetic Stimulation

The stimulus location and treatment parameters are located at the dorsolateral prefrontal cortex. Stimulus parameter may be set at 1 Hz, 100% motor threshold, 60 s/bunch, 5 bunches/day at 60 s intervals. Five days constitute one cycle and administer two cycles in total.

6 Prognosis of Post-blast Injury Stress Disorder

The Israelis looked into war-related stress responses in soldiers that participated in the 1973 Yum Kippur War. The researchers studied how these troops adjusted to military duties in the 9 years after the war, discovering that 94% of the subjects remained in service. The recurrence of combat stress reaction was observed in 80% of these troops that had adjusted to battle. Results indicate that soldiers could recover from combat stress completely and return to battle. The American military believes that soldiers with stress disorder would experience flashbacks of brutal scenes from the battlefield if and when they aged and coupled with health deterioration, troubles at work, unemployment, family conflicts, or other problems. Or they might see those scenarios in their dreams, succumbing to lifelong post-traumatic stress disorder. Some might be compounded with depression or loneliness, leading to alcohol abuse and causing health issues like cardiovascular diseases, digestive system or joint problems, diabetes, etc. The National Defence Medical Centre of Canada assessed 92 Canadian armed personnel before and during military deployment using PTSD Checklist, traumatic life events questionnaire, and Cambridge Neuropsychological Test Automated Battery (CANTAB) cognitive research software. Research results indicate lowered stress risk and reduction in cognitive function, biological and psychological predictors in 62% of the subjects, and serious maladjustment in 12%. Therefore, stress response enhancement is a very necessary part of training for military personnel.

Through the therapies outlined above, a patient could recover from acute stress disorder within a few days or about a week. The disease course of PTSD comes in waves. Though the majority of patients can recover, it might turn chronic in the minority, lasting up to several years. Some might exhibit personality changes that could impact work and study to a certain extent. All in all, it is possible to recover well from stress disorder, and only a small number of patients would suffer the illness for a length of up to several years or end up with other types of psychological disorders.

7 Prevention of Post-blast Injury Stress Disorder

7.1 Prevention of Post-blast Injury Acute Stress Disorder

Timely psychological intervention and self-rescue education after blast injury are highly necessary. After the catastrophic explosion in Tianjin, the media reported that the nation dispatched 176 professional psychological and mental therapists, demonstrating the importance of intervening post-explosion psychological damage.

The fatality rate from head wounds in terrorist bombings is high, accounting for 50–70% of the total, but 98.5% of survivors of external head wounds do not have critical injuries. Modern medical principles also suggest thorough and detailed inspection during early diagnosis of survivors that seem to have been spared from serious injuries, for the purpose of observing whether or not there are hidden injuries or to spot minute indications that existing injuries might worsen. The survival rate of injury solely from fragments in the explosion is relatively high, but in most cases, explosions cause injuries due to multiple factors. This kind of composite injury includes explosion impact, fragment penetration, and compression, burns and inhalation of toxic gas as a result of the explosion.

7.2 Prevention of Post-blast Injury Stress Disorder in War

Blast injuries have huge impacts on people exposed, firefighters, law enforcement and medical personnel involved in the rescue effort, and people responsible for event investigation and site clean-up. Therefore, researchers and decision-makers in nations across the globe are actively seeking effective protection measures.

1. During normal times or when proceeding to the site, the exposed personnel should be educated on knowledge about general psychological health and learn ways to identify possible psychological problems, as well as some effective coping methods. When a team member with an excessive stress reaction and related psychological issues return, refrain from any discrimination or rejection so as to reduce suggestibility about stressful reactions to explosion sites.
2. During normal times, it is suggested to take an active approach to a training regimen that strengthens psychological tolerance, including the implementation of processes that can enhance military, physical, and mental strength in extreme environments and situations that are close to real-life scenarios.
3. Enough sleep of personnel that participate and come into contact with relevant scenarios should be guaranteed. It is generally believed that 24 h of non-stop contact should be given at least 4 h of sleep. Troops and decision-makers in a sustained state of combat need at least 6 h of sleep. At the same time, try to ensure that participating personnel get enough rest and food, make sure that they are protected from temperature and weather changes so that they stay in a relatively sound state of mind and fitness.

4. An effective and reliable medical rescue system should be established to minimize fear in people that are involved with or come into contact with explosion site, and reduce post-injury worries to establish impregnable mental strength conducive to defending against stressful reactions in combat.
5. Strive to improve the on-site environment, remove bodies and pollutants on-site in a prompt fashion, and keep the environment as clean and neat as possible to help ease emotional burden. If personnel must be stationed on-site, carry out different kinds of recreational activities during off-time if conditions permit, so as to reduce the nervousness and fear in the ambiance. And physical exertion should be appropriate to avoid excessive tiredness. For teams in a dangerous environment or difficult position, try to rotate them out of the situation on a regular basis, which would be helpful to lower the chances of stressful reactions.
6. When the site is under a high-stress stage, soldiers usually do not have much appetite. Researchers at the US Army Natick Soldier Systems Center discovered that consuming high-protein foods with high content of tyrosine, or including tyrosine in drinks and foods, can allay stress. The author's own study found out that feeding lycium barbarum polysaccharide, an extract of *Lycium barbarum* (Chinese wolfberry), to rats under severe stress can effectively prevent and improve stress disorder symptoms in the animal.

8 Research on Protection Against Post-blast Injury Stress Disorder

With regard to research on blast injury, the common consensus is that the best way to protect the brain against an explosion event is to strengthen the protection of the head. Present literature points out that the American military is developing a new type of helmet that differs from conventional helmets in that a transparent polycarbonate faceplate is featured. Experiments indicate that said helmet can delay to a certain extent the time at which a blast wave reaches the head and can reduce the area of the brain impacted. Several American universities also published articles that look into related protection research and production philosophies. Emory University researched and developed a portable medical instrument that can provide a rapid assessment of explosion impact and can quickly evaluate cognitive function, balance, and eye movement. The University of California, Los Angeles (UCLA) developed a new type of energy absorption crystal material. A tie-up between the University of Miami and Pittsburgh University developed portable protective goggles that collect high-precision data on eye movement to assist in the actual identification of mild head trauma. The University of Washington developed a helmet with a new type of impact absorption structure that can reduce injuries to the head caused by an explosion. At the same time, the US military has developed the rate-actuated tether, which can lessen acceleration of head movement to lighten impact injury suffered by the head. Also, other literature points out that adequate protection of the chest can effectively reduce blast injuries to the head and abdomen, in turn providing new pathways to physiological and psychological defense against stress response to blast injury.

Looking at current social development trends, people have already become accustomed to seeing worldwide explosion events in the daily news, and from online news publications statistics between 2010 and 2016 alone, there were 172 explosion events in China in which three or more persons had been injured or killed during that period, with the catastrophe in Binhai New Area of Tianjin being the most influential and significant. According to news reports and information, after the Tianjin explosion, experts analyzed that ratio of stress disorder occurrence might hover around 30%. Thus, for medical personnel participating in on-site emergency rescues, the early diagnosis and treatment of explosion-related stress disorder and intervening in the processing of large-scale explosion events are of utmost importance. In the real world, explosion-induced stress disorder harms the patient's health either by causing premature death or capacity loss. Therefore, such disorder not only shortens the lifespan of patients but also clearly increases direct and indirect economic losses due to the illness and raises the consumption of medical care resources. This is why the illness is a burden for everyone, including the patient, his or her family, and society. Researchers compared patients with such disorder against control group patients of the same age, same gender, and same hospitalization duration and discovered that patients with such disorder spend 76% more on hospitalization expenses. For veterans with PTSD, each year, the US government has to spend USD 8300/person on therapy expenditures. Illnesses related to psychological symptoms arising from brain and head injuries are directly associated with the marked rise in medical expenses, and this is no doubt an urgent issue in the diagnosis and treatment of blast impact-related stress disorder. At present, the international community uses disability-adjusted life years as an indicator to measure quantitative and qualitative losses in life and health. Patients with progressive psychological stress disorder induced by blast waves might die before the average lifespan of the normal population because of reasons like the blast injury itself or possible comorbidity, resulting in a shorter lifetime and opportunity to serve society. This will surely become the main problem that demands attention from both blast wave event victims and society.

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Part III

Local Blast Injury

Blast Lung Injury

Bo Zhang

Blast lung injury refers to the mechanical damage to the target organs, mainly lungs, of the personnel in the detonation area by the blast wave. Therefore, the lung could be regarded as the primary target organ for blast injury. Various detonations can lead to blast lung injury, and the incidence shows a clear upward trend, such as explosive weapons (fuel air explosive or FAE, aerial bombs, mine explosions, etc.) in wartime, and explosive accidents in military factories, chemical plants, ammunition depots, mines, boilers, high-pressure gas cylinders, and increasingly frequent terrorist activities in peacetime.

Although blast wave can cause damage to various organs and parts of the body, such as auditory organs, eyes, heart, abdominal cavity, limbs, and spine, still its main "target organ" is the lung. This is because after the overpressure-underpressure of shock waves act on the human body, air-filled tissues can be damaged through the implosion. As an organ with relatively abundant air-filled tissues (such as alveoli), lung damage will bear the brunt. Compared with other organs, the lung, as the "target organ" of the shock wave, is more likely to be injured seriously, which often affects the development and outcome of the whole-body injury, becoming one of the main reasons for the early death of blast injury.

1 The Mechanism of Blast Lung Injury

The mechanisms of blast lung injury mainly include implosion effect, spalling effect, hemorheological effect and hemodynamic change, inertia effect, pressure difference effect, underpressure and alveolar expansion effect, and biomechanical effect.

1.1 Implosion Effect

After being compressed, the gas expands rapidly and causes injury (see Fig. 3 in chapter "Injury Principles and Mechanisms of Shock Wave").

1.2 Spalling Effect

When the dense medium enters the porous medium, reflection occurs on their interface, causing a sudden increase of local pressure and injury (Fig. 1).

1.3 Hemodynamic Effect

A large amount of blood flows from the abdominal cavity into the thorax, causing damages to the lung tissues (see Fig. 1 in chapter "Injury Principles and Mechanisms of Shock Wave").

1.4 Underpressure Effect

Although past studies believed that underpressure had little or minor killing effect, our studies show that the underpressure of shock waves can cause mild to fatal injuries with similar pathological characteristics of overpressure-induced injuries. There is a certain dose–effect relationship between shock wave underpressure and injury in animals, and its decompression multiple can better reflect the relationship between the pressure intensity of shock waves and injury than the peak pressure in animals.

1.5 Overexpansion Effect

The blast lung injury does not mainly occur in the compression period as previously thought, but in the decompression

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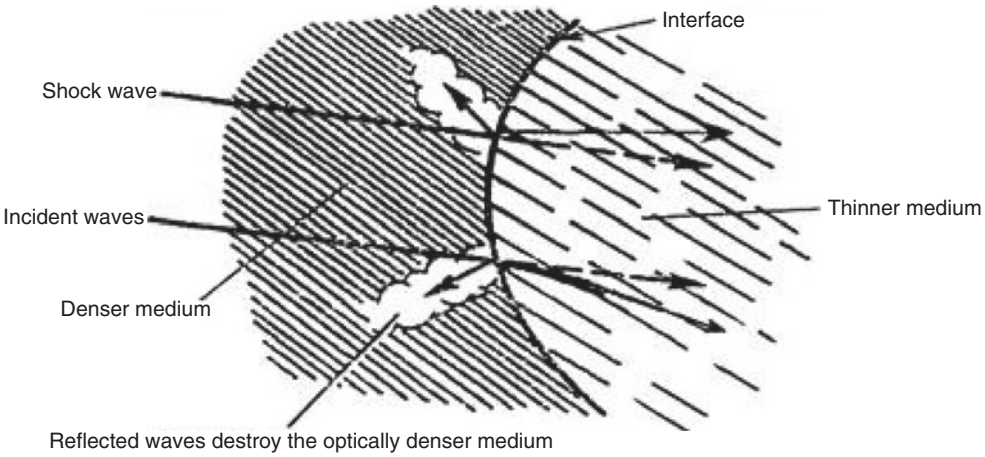

Fig. 1 Spalling effect

period and the underpressure period, that is, the effect caused by overexpansion. Under the action of shock waves, the alveoli experienced two phases of compression and expansion. During the expansion phase, tensile strain and tensile stress are generated on the alveolar walls. When the tensile strain reaches a certain level, the permeability of pulmonary microvascular endothelial cells and alveolar epithelial cells to small solutes can be increased to exceed its critical value, resulting in pulmonary edema. When the tensile stress exceeds the strength limit of the alveolar wall, the alveoli will rupture as well as the alveolar capillaries at the same time, causing pulmonary parenchymal hemorrhage, that is, excessive and rapid expansion effects occur during the expansion phase of lung.

2 Pathophysiological Changes of Blast Lung Injury

The main pathological features of blast lung injury include alveolar rupture and intra-alveolar hemorrhage mainly, and pulmonary edema and emphysema secondarily, sometimes accompanied by lung rupture. Pulmonary hemorrhage can vary from spotty to diffuse, and in severe cases, parallel strips of pulmonary parenchymal hemorrhage can be seen under the intercostal space. The rupture of blood vessels in the lung parenchyma can form hematomas, and even a blood clot that can block the trachea and cause rapid death. The bronchiolar epithelium separates from the basement membrane, which causes fluid/air membrane dysfunction so that blood or edematous fluid enters the alveoli, while air is forced into the pulmonary blood vessels. In mild cases of pulmonary edema, there is a small amount of fluid in pulmonary interstitium or the alveolar cavity, while in severe cases, a large amount of edematous fluid overflows into bronchi or even the trachea, often mixed with blood, i.e., bloody foamy fluid. Pulmonary hemorrhage and edema can lead to atelectasis. Emphysema can be interstitial or alveolar. In severe cases, pulmonary bullae containing blood and gas appear under the pleura, which can cause hemothorax or hemopneumothorax when the lung ruptures. When the lung tissues are torn, gas in the alveoli enters the pulmonary veins through the ruptured small blood vessels, causing air embolism. Alveolar and interstitial hemorrhage, edema, and inflammatory cell infiltration can be seen under the light microscope. Under the electron microscope, the capillary endothelium was swollen, the pinocytosis was enhanced, and myeloid bodies were occasionally observed. In the capillaries, there is red blood cell squeeze and deformation, platelet adhesion and neutrophil accumulation, degranulation, and cell membrane dissolution.

3 Clinical Manifestations and Diagnosis of Blast Lung Injury

The clinical characteristics of blast lung injury vary depending on the trauma. In mild cases, there may be only short-term chest pain, chest tightness, or suffocation. In slightly severe cases, patients may develop cough, hemoptysis, and sputum with blood streaks, while some casualties have short of breath with scattered moist rales audible sometimes during auscultation. The wounded with severe blast lung injury often suffer obvious dyspnea, cyanosis, and bloody foamy fluid in mouth and nose, and in physical examination, local dullness can be found on percussion, and breath sounds are weakened with a wide range of moist rales audible on auscultation, which is often accompanied by shock. Lung injury caused by dynamic pressure is often accompanied with pneumothorax, hemopneumothorax, and multiple rib fractures with corresponding symptoms and signs.

Other features: After a blast injury, changes in the internal tissues of lungs, the circular metabolism and other physiological indicators can be used to assess the severity of the blast lung injury by means of pulmonary shunt measure-

ment, arterial blood gas analysis, and chest X-ray. Studies have shown that obvious changes in chest X-rays often occur when there is hemorrhage and edema deep into the lung parenchyma. The typical X-ray findings include butterfly-like infiltrates around both hila, while atypically, there are only patchy or flocculent opacities. However, bleeding with mild lung injury cannot be shown on chest X-rays. In the blood gas changes of the body after blast lung injury, there are more significant changes in PaO_2 than the changes in $PaCO_2$ and pH. Studies have also shown that the pulmonary shunt flow is significantly increased at different time points after blast injury, but the changes in related blood gas indicators are not significant, suggesting that the pulmonary shunt flow is a more valuable diagnostic indicator when judging the severity of lung injury.

4 Classification Criteria for Blast Lung Injury

The classification criteria for blast lung injury are based on different injury characteristics. Various blast lung injuries have respective characteristics in pathology, clinical manifestations, and diagnosis, which are closely related to the severity of blast injury. For a long time, the relevant classification criteria of domestic and foreign studies are also designed according to the aspects above. But the content is relatively scattered without a comprehensive summary of the characteristics of blast lung injuries in various aspects, especially practical classification criteria.

4.1 Classification According to the Shock Wave Pressure Value and Impulse

The severity of lung trauma after a blast injury is related to factors such as the explosion equivalent, the proximity to the detonation center, etc., but can be briefly determined by the force value per unit area of the body (i.e., the shock wave pressure) or impulse. According to the standard of the relationship between shock wave pressure and injury severity reported by the America National Bomb Data Center, the pressure of 207.2–345.3 kPa can cause mild lung injury, while the pressure of 522.4–690.6 kPa can cause 50% of the wounded to develop severe lung injury. Yang Zhihuan et al. have studied the relationship between the severity of immersion blast injury in canine lungs and the shock wave pressure value, finding that impulses leading to extremely severe, severe, moderate, mild (including minor injury according to the pathological classification, which will be described in detail later), and no injuries were 248.3–848.2 kPa/ms, 190.9–324.1 kPa/ms, 146.3–246.8 kPa/ms, 106.6–213.5 kPa/ms, and 73.1–152.1 kPa/ms, respectively.

4.2 Classification According to Pathological Characteristics

The relevant domestic and foreign classification standards have been formulated for decades based on the pathological characteristics of blast lung injury. These two standards overlap in content but are not completely unified.

In domestic classification standards for blast lung injury, the scope of pulmonary hemorrhage has also been regarded as an important basis to assess the severity. The scope of hemorrhage is quantified as follows: pulmonary hemorrhage with the area less than 0.5 cm × 0.5 cm or as large as a soybean is called "spotted lesion," that less than 1.5 cm × 1.5 cm or as large as a broad bean is called "plaque," that less than 3 cm × 3 cm or as large as a watch is called "small patchy lesion," and that larger is called "large patchy lesion" or "diffuse lesion." According to this criteria, the injury severity is divided into four categories, respectively, mild, moderate, severe, and extremely severe. Among them, mild blast lung injury is defined as pulmonary hemorrhage mostly as large as soybeans accompanied with mild edema. The main manifestations include scattered spotted hemorrhage lesions or a few (less than two) hemorrhage plaques in one or several lobes, and a small amount of pink foamy fluid flowing out when pressing the cut surface of one or several lobes, which is also attached to the knife during cutting. Moderate blast lung injury is defined as pulmonary hemorrhage mostly as large as broad beans accompanied with moderate edema. The main manifestations include scattered hemorrhage plaques or a few (less than two) small patchy hemorrhage lesions in one or several lobes, and swollen surfaces of more than two lobes, in the section of which there is a small amount of foamy fluid accumulated in bronchioles and more flowing out when pressing it. Severe blast lung injury is defined as pulmonary hemorrhage mostly as large as watches and cracks in lung accompanied with severe edema. The main manifestations include small patchy hemorrhage lesions or a few (less than two) diffuse large patchy hemorrhage lesions in one or several lobes, foamy fluid accumulated in the trachea and swollen and shiny surface of more than three lobes, in the section of which there is a larger amount of foamy fluid accumulated in bronchioles and flowing out without pressing, as well as superficial cracks in some (less than two) lobes without massive blood accumulation in the thorax. Extreme severe blast lung injury is defined as large patchy pulmonary hemorrhage mostly and multiple cracks in lung accompanied with severe edema. The main manifestations include diffuse large patchy hemorrhage lesions in more than four lobes, and foamy fluid accumulated in the trachea, causing severe suffocation. Usually, the lung surfaces on both sides are significantly swollen and shiny. The lungs do not collapse after opening the chest cavity. A large

amount of foamy fluid can be seen extensively in the bronchiolar lumen, and they spontaneously flow out from the cut section of the lung. Multiple superficial cracks or a few (less than two) deep cracks (above 0.5 cm in depth) appear in the lobes.

Internationally, the Yelverton scoring is mainly used to quantitatively evaluate and then qualitatively classify blast lung injuries. Generally speaking, it simply divides blast lung injury into three categories: mild, moderate, and severe. Mild blast lung injury refers to spotted or scattered pulmonary hemorrhage with the total bleeding area less than 10% of the whole lung. Moderate blast lung injury refers to more spotted pulmonary hemorrhages, with patchy hemorrhage lesions with the total area of bleeding between 10% and 50% of the whole lung. Severe blast lung injury refers to more patchy pulmonary hemorrhage lesions, occasionally large patchy lesions or even lung consolidation with the total bleeding area greater than 50% of the lung surface area, and more bloody foamy fluid in the trachea.

5 Treatment Principles of Blast Lung Injury

Casualties with mild injuries can recover within a few days after rest and symptomatic treatment. For severe cases or those accompanied with other injuries, aggressive comprehensive treatment is required.

5.1 Rest

Anyone who is suspected of lung injury should avoid strenuous activities as much as possible to reduce the burden on the heart and lungs and prevent aggravation of bleeding.

5.2 Keep the Airway Unobstructed

Patients with shortness of breath should stay in a semi-sitting position. Those with bronchospasm can be treated with cervical vagal block or bronchodilators. The secretions of the airway should be sucked out in time.

5.3 Oxygen Therapy

For casualties with dyspnea or a decrease in PaO_2, a face mask or nasal cannula is required to deliver oxygen. If the decrease in PaO_2 cannot be corrected after oxygen therapy, or respiratory failure has occurred (mostly in 12–36 h after injury), mechanical assisted ventilation is required.

5.4 Positive Pressure Ventilation

It helps maintain good ventilation, remove trapped CO_2, increase the pressure in the alveolar cavity, in order to prevent lung collapse, and open up the collapsed alveolar. Besides, it increases the pressure in alveoli and interstitium and then reduce the effusion into alveoli, in order to relieve pulmonary congestion and interstitial edema, and rectify the imbalance between ventilation and blood perfusion. Continuous positive pressure ventilation (CPBB) can enhance functional residual capacity (FRC) to improve compliance.

5.5 Hyperbaric Oxygen Therapy

The wounded with air embolism can be treated with 607.9 kPa high pressure gas (of which the oxygen cannot exceed 253.3 kPa) for 2 h, followed by decompression, and 100% oxygen should be given immediately when the pressure is reduced to 283.7 kPa, and then given intermittently. This approach can shorten the time required for decompression, to improve tissue oxygenation, and to reduce the incidence of decompression sickness. In addition, mannitol can also assist in the treatment of air embolism. For those suspected of air embolism in need of an air transportation, the flying height should be reduced as low as possible, because air embolism is prone to occur under upper-level and low-pressure conditions. During transportation, the wounded suspected of air embolism should be placed in left lateral position, with head lower than feet, so that the air embolism can stay in the heart and enter the lower limbs.

5.6 Prevention and Treatment of Pulmonary Edema and Protection of Heart Function

When pulmonary edema occurs, oxygen can be inhaled through a 50% or 95% alcohol solution to reduce the surface tension of airway secretions or edema fluid. Dehydration therapy can also be used, such as aminophylline (dissolved in 50% glucose solution for slow intravenous therapy), and intravenous therapy of 20% mannitol and furosemide; intravenous therapy of hydrocortisone can treat interstitial pulmonary edema. Those with heart failure can be treated with digitalis, such as tabellae digitalis, digoxin tablets, deslanoside, and strophanthin K.

5.7 Prevention and Treatment of Hemorrhage and Infection

Various hemostatic agents can be used, such as carbazochrome, fibrin, and traditional Chinese medicines for pro-

moting blood circulation and removing blood stasis. For those with severe lung rupture accompanied by massive hemorrhage, surgery should be performed immediately to suture the rupture or remove the lobe. Antibiotics should be administered to prevent lung infections.

5.8 Sedatives and Pain Relievers

In order to relieve pain and irritability, pethidine or morphine hydrochloride can be administered, but morphine is contraindicated for those with poor respiratory function or brain contusion. Intercostal nerve blocks can be used to relieve pain in the chest wall. Besides, acupuncture therapy can be used as appropriate.

5.9 Blood Transfusion and Fluid Infusion

For those combined with other serious injuries (such as internal organ rupture, burns, etc.) that cause loss of whole blood or plasma, blood transfusion, and fluid infusion are required in time to restore blood volume and cardiac output. The ideal volume of supplemented fluid is to slightly raise the central venous pressure and increase the cardiac output. If the central venous pressure increases with no change or even decrease in cardiac output, it indicates that myocardial contractility is impaired. It should be noted specially that acute left heart failure is likely to occur if the patient continues to be infused a large amount of fluid. Pay attention to supplementing colloid during crystalloid solution infusion at the ratio of 1:1. Adequate fluids can be infused, when lung signs (whether there is a significant increase in moist rales) and urine output (whether it is too little) are closely monitored.

5.10 Choice of Anesthesia

Within 1–2 days after the injury, the wounded with blast lung injury are poorly tolerated to anesthesia, so surgery should be delayed as much as possible; if an emergency operation is necessary, nitric oxide anesthesia can be used, and oxygen should be inhaled during the operation.

6 Blast Lung Injury Under Special Circumstances

The explosion shock wave propagates to the surrounding through media (such as air, water, soil, rock, steel plate, etc.). According to different propagation media, blast injury can be divided into air blast injury, immersion blast injury, and solid blast injury. The density of the propagation medium can significantly affect the propagation velocity of shock waves and the severity of blast injury. Generally, the higher the density of the propagation medium is, the faster the propagation velocity of the shock wave is, and the longer the propagation distance is, the more severe the blast injury is. Special environmental blast injury mainly refers to plateau blast injury, immersion blast injury, and blast injury in limited spaces such as tanks, armors, and cabins.

6.1 Plateau Blast Lung Injury

The total plateau area of China is about 3.1 million km^2, accounting for 33% of the territory and 1/6 of the entire country. There are more than ten million people permanently residing in the plateau, ranking the first in the world. In addition, there are a large number of unexploited minerals, oil, and gas resources of great economic value in the plateau of China. There is a long defense line. Besides, its geographical characteristics also determine its significant strategic defense position in future national and regional conflicts.

Plateau blast lung injury refers to a blast injury that occurs in areas of 3000 m above sea level. The air in the plateau area at an altitude of 4000 m is as thin as 65% of that at the sea level, and the atmospheric pressure and the oxygen partial pressure at an altitude of 4000 m are as low as about 61% of that at the sea level. Plateau blast lung injury refers to the damage that occurs when organisms are directly or indirectly affected by shock waves in the high-altitude environment. The atmospheric pressure is low on the plateau and decreases with the increase in altitude. As the surrounding pressure declines and the lack of oxygen is aggravated, the tolerance of animals to shock waves decreases, manifested as that blast injury at a high altitude is more severe than that in plains and low-altitude areas. The mortality rate of plateau blast injury is very high with the main target organs causing injury/fatality of lungs, bowels, and other air-filled organs, while injuries rarely occur in solid organs such as liver and kidney.

The main pathological presentations of the plateau blast lung injury are pulmonary hemorrhage and edema. Massive blood components escape into alveoli of the injured region: alveolar walls collapse and fuse with each other to form bullae; in terms of ultrastructure, alveolar wall type I epithelium and capillary endothelium are destroyed, which seriously affects the respiratory and barrier function, leading to respiratory failure in severe cases. This is the main cause of early death.

6.2 Underwater Blast Lung Injury

Underwater blast lung injury is common during island-reef operations and landing operations. Underwater blast could be generated by underwater explosion of depth bombs,

mines, torpedoes or underwater nuclear weapons, as well as guided missiles, bombs, and artillery shells, which can cause underwater blast lung injury to people underwater.

The density of water is about 800 times that of air so that it can hardly be compressed. Compared with the air blast, the propagation velocity of underwater blast is further faster (about 3–4 times) with slower attenuation and longer propagation distance. Therefore, the intensity of shock waves generated by the underwater explosion is three times that of its explosion in the air. The pressure produced by TNT exploded on ground is about 200 times larger than that produced by TNT of the same mass exploded in air at the same distance. Underwater blast injury is much more severe than air blast injury, with significantly larger killing range and significantly higher mortality rate. As early as World War II, it was noted that there were thousands of underwater blast injuries that could lead to death, which were known as "immersion blast injuries" then. However, experimental studies on underwater blast injuries did not begin until the US military established an underwater test site in Kirtland in the late 1960s.

In addition to the same characteristics of complicated injuries, mild external trauma but severe internal injury, and rapid development as the air blast injury, underwater blast injury has its own features such as high incidence of injury, severe conditions, and close relation with severe pulmonary hemorrhage and edema, as well as coronary artery air embolism caused by lung rupture and lung injury.

7 Prevention of Blast Lung Injury

Currently, domestic and foreign studies on blast injury have focused on individual protection of strong shock waves, injury mechanisms of blast injury, and the injury effect of multiple low-intensity shock waves during weapon launching. After hearing organs were protected appropriately from blast injury, the problem of blast lung injury protection becomes particularly prominent. According to the protection studies on conventional bulletproof vests against shock waves, the vest can aggravate blast lung injury instead of providing protection. Therefore, in the early 1970s, Academician Zhengguo Wang studied protection against thoracic blast injury by plaster tubes and plastic products and found that plaster tubes could reduce blast lung injury. In the mid-1980s, some researchers developed anti-vibration suits made out of foam plastic for gunners, which were mainly aimed at the protection against weak shock waves but not effective in strong shock wave protection.

When developing protection equipment against thoracic and abdominal blast injury, which is similar to that against blast-induced traumatic brain injury, additional consideration is needed about materials, area, and auxiliary materials, besides early warning performance. On the one hand, given that the lung is the most common target organ for shock wave exposure, a chest strap device has been used in previous studies. The lung surface hemorrhage area and lung body index (wet lung weight/body weight) were significantly lower in animals of the chest strap group and the inflatable bag group than those in the injury group. In addition, the inflatable bag was most effective in protection. These results indicate that chest strap can restrict the inertia of the thoracic cage's excessive outward movement and lessen the lung tissues' tensile strain, which can significantly reduce blast injury to the lungs, and further disapprove of the importance of the "drag effect" in the mechanism behind lung blast injury.

At the same time, in light of the outcomes described above, lung blast injury protection equipment principles are proposed below:

1. Soft materials with high strength and low elasticity should be used to maintain a certain degree of tension.
2. Soft protection is recommended, and equipment performs better with gas as the main component.
3. The lower edge of the thorax should be protected mainly to limit the expansion of the thorax better. So the protection equipment does not have to be very wide and can be made into belts. On the other hand, studies have found that during the exposure to shock waves, significant stress was transmitted to the thoracic cavity above the diaphragm from the abdomen under the action of the shock wave pressure. Even under the protection of the chest protection device, there is still considerable stress waves propagating through the abdominal cavity to the thoracic cavity, generating a destroy effect to lungs. Therefore, in order to ensure the safety of explosive operations, greater protective significance could be attached to a thoracoabdominal one-piece protection device than a single chest protective device (such as a chest strap).

Hearing Damage Through Blast

Dejing Meng and Jichuan Chen

The rise of explosive terrorism attacks across the world has begotten a large body of blast injuries that were only seen in combat before. Many of the victims close to the explosive source die of multiple severe damages resulting from the primary blast injury. Often, the blast wave can easily heave the victim and throw any object when propagating in overheated air, which is the cause of penetrating and blunt injuries. A study on 3000 explosion victims found that the mortality was 13%, and 30% of the survivors needed hospital care [1]. As opposed to other patients, the patients with blast trauma are badly injured in most cases and require surgical treatment, intensive care, as well as further hospitalization and rehabilitation therapies [2]. Blast injuries are classified as primary, secondary, tertiary, and quaternary. Primary injury is the direct effect of the shock wave generated during an explosion upon human bodies, and gas-filled organs like the tympanic membrane, pulmonary alveoli, and GI tract are susceptible in general; secondary injury is fragmentation-caused penetrating wound; tertiary injury occurs usually when the victim is crashed into something hard under the impact of the shock wave or hit by falling objects; quaternary injury refers to other explosive injuries, including burns, apnea, radiation, toxins, and psychological trauma [3]. Human ears consist of the outer or external canal, middle ear, and inner ear. Explosions may cause single or combined damages to the ear. The detection and treatment approaches depend on the parts injured and the time of injury.

1 Blast Damage to the Outer Ear

The pinna is exposed to the head, and thus more likely get injured than other parts of the ear. The debris from explosion may cause burns and damages [4, 5]. The fragments from explosion may scourge the pinna through contusions, cuts, lacerations, burns, etc.

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1.1 Auricular Contusions

1.1.1 Clinical Manifestation and Diagnosis

Pinna bruises tend to occur when a person is first thrown up by the gas shock released in an explosion and then struck by a hard object after falling down. Mild contusions are present only in the form of local skin abrasion, swelling, and ecchymosis. In severe cases, the minute vessel beneath the skin and perichondrium ruptures to cause a hematoma, which, partially in purplish red hillock-shaped protrusions or circular swelling but without the signs of acute inflammation, gives a feeling of softness and motion when touched. Hematoma mostly occurs outside the auricle. Small hematomas are self-absorbed. Sometimes, the pinna is partially thickened and deformed by organized hematomas. Large hematomas are hard to self-absorb because there are too few subcutaneous tissues beneath the pinna to improve blood circulation and self-absorption. In addition, the conchal cartilage lacks nutrient vessels inside, and its nutrition supply primarily relies on the perichondrium. If the perichondrium is largely separated from the cartilage due to hematomas, it may result in chondronecrosis, exposure to continued infections, and ultimately, auricular malformation. "Cauliflower ear" is a common consequence of delayed hematoma treatment. The accumulation of hematomas may lead to secondary infections, and pseudomonas aeruginosa, for example, is the bane of suppurative perichondritis that causes massive cartilage necrosis.

The cartilage swells locally and appears dark red as a result of blood gathering. A sense of mild pressing pain and cyst is felt by palpation. The swelling part resembles an opaque shadow, which can be differentiated from an auricular pseudocyst.

1.1.2 Treatment Principles and Methods

The treatment principles of auricle contusions are thorough disinfection, prevention of infection and hemorrhage, and promotion of hematoma absorption or drainage. If the hematoma is difficult to absorb or continues to bleed, an incision

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parallel to the natural folds of the skin should be made on the surface of the hematoma without damaging the auricular cartilage. If the hematoma has been organized, it should be excised. The right amount of antibiotics is recommended to prevent infections and cartilage necrosis.

It is advisable to reduce hematopedesis by ice compress in the early stage (within 24 h) of pinna hematoma. If blood is oozing too much, draw the accumulated blood with a thick syringe needle after proper disinfection. First, finger press the swollen area until the blood is completely drained by the needle, but pay attention not to stab and wound the cartilage. Put a piece of gauze or an alcohol-soaked cotton ball that has been squeezed dry on the swollen area for pressure bandaging. In the meantime, use antibiotics as a precaution against infection. If puncture falls on stony ground, make an incision as small as 1 cm on the part of hematoma after disinfection to remove excessive blood. Then, dress the wound with a compression bandage, and prevent infection with the aid of systemic antibiotics 5–7 days later.

1.2 Auricular Cuts and Lacerations

1.2.1 Clinical Manifestation and Diagnosis

Auricular cuts and lacerations involve varying degrees of avulsion and defects or complete separation. In mild cases, there is only a fissure; in severe cases, the auricle may be torn and defective, or even completely severed. This type of trauma is often accompanied by damages to the jaw, brain, and other facial parts. Lacerations are characterized by uneven skin cuts and undamaged cartilages. If the cartilage is injured, the site of damage may not coincide with the location of the skin injury; cartilage injuries are often irregular. Cut wounds are neat, where the skin and cartilage are always compromised at the same time and in the same area.

By site, the injury is divided into upper auricle, middle auricle, helix, and earlobe. By degree, it is classified as skin injury, skin and full-thickness cartilage defects, partial auricle mutilation, and complete auricle mutilation. Traumatic wounds evoked by blast injuries are often serious, or accompanied by craniofacial and parotidean trauma, as well as facial nerve damage. Foreign bodies may be present at the site of injury.

1.2.2 Treatment Principles and Methods

Pay attention to associated injuries to other parts of the body, especially the brain, chest, and abdomen, so as not to delay the diagnosis and treatment of injured vital organs. Seek early debridement and suturing as soon as systemic conditions allow. The auricle is very important for cosmetic purposes, and reconstruction of an ear defect is not an easy job. It is still hard to restore the original shape or make the damaged auricle look as perfect as before even if advanced technology is available. This should be considered when dealing with blast-related ear injuries, and a piece of advice is to use as many intact auricular tissues as possible.

1. *Local treatment*. Debridement and suturing are recommended for broken auricle skin, or damaged cartilage, or cartilage skin that has been separated from most of the auricle skin but is still connected with the auricle. In terms of suturing time, the principle is the faster the better, optimally within 8 h of injury, and 24 h is acceptable only if the wound remains uninfected. For the wounds that remain uninfected or are slightly infected 48 h later, suturing can be performed shortly after debridement and local disinfection. If the infection is noticeable, systemic and local infection control is advised before wound repair. Before suturing, rinse the wound with normal saline, remove all the foreign bodies from the wound, and excise the irregular or mildly infected rims with a maximum of ear tissues left. If the margin of the cartilage is exposed due to the contraction of the skin at the edges of the lacerated wound, trim the exposed cartilage, and stitch it up to abate the tensile force arising from suturing. After debridement, disinfect the wound with hydrogen peroxide, benzalkonium bromide (Bromo Geramine), and 75% alcohol, and then sew it up 10–20 min later. This is very important to prevent postoperative infection. First, align the helix to the antihelix, then stitch up the auricle in the order of outer side and inner rear side using 3–0 silk sutures at an interval suitable to be 0.5 cm. If the stitches are too dense, it will affect the blood supply. Pay attention not to pierce the auricular cartilage. Fix the perichondrium with 6–0 noninvasive absorbable sutures if necessary, but take care not to penetrate the cartilage. Repair the skin at the opening of the external auditory canal, immobilize the skin flap with sutures if skin grafting is required, and compress them well with dressings. It is crucial to avoid the stenosis of the external auditory canal.

   Handling of disarticulated auricular tissues: examine the disarticulated auricular tissue block. If all or part of the tissue is available, first wash the tissue in normal saline, and place it in Ringer's solution containing heparin for cryopreservation. The reimplantation time of a severed ear is usually less than 24 h. The probability of viable reimplantation is very small if the ear is just debrided for apposition suture. The skin closure should not be too dense in case cutaneous dropsy leads to compression necrosis. Gently press the outside with a gelatin sponge soaked in an antibiotic solution and properly bandage it. Postoperatively, if the skin is heavily edematous, multiple small incisions can be made to facilitate the alleviation of tension and drainage. In addition to aggressive postoperative antibiotics for infection, vasodilators and anticoagulants can also be used to promote blood supply.

If the repaired tissue is red and swollen, purple-black, or blistered, it is a sign of ischemia. Do not rush to remove it. Local infrared radiation or hot compress helps increase blood flow to the ear. Disinfect the blister, and draw out the tissue fluid by needle aspiration. In most cases, part or all of the repaired tissue can survive. In the case of shedding due to dry gangrene, the tissue can be repaired after the wound has been occluded. If the auricular tissue becomes unavailable due to loss or separation, it can be repaired according to the defect. For small defects, a bridle suture can be performed immediately. For severe defects or if a repair cannot be done at once, the edges of the wound can be sutured, or the wound can be embedded in the skin behind the ear for secondary repair.

2. *Postoperative management and systemic therapy*. The wound is dressed and fixed for 3–4 weeks. Postoperative treatment with tetanus antitoxin and anti-infection is crucial, especially if there is cartilage damage. Once perichondritis has occurred, it often leads to cartilage necrosis and cicatrization, and the support for auricle is lost accordingly. Worse still, a "cauliflower ear" is formed, which requires further plastic surgery. Antibiotic treatment is given for 5–7 days, generally penicillin, or other antibiotics when needed. The auricle should be immobilized for a fortnight to avoid movement, as this may interfere with wound healing.

1.3 Auricle Burns

The auricle is exposed and prominent, and the skin is relatively thin and fragile. Blast injuries are often associated with burn injuries. Since the auricle is adjacent to hairs and connected to the external acoustic meatus, the surface is uneven and difficult to clean, making it susceptible to infection and postburn suppurative perichondritis.

1.3.1 Clinical Manifestation and Diagnosis

Auricle burns are classified into three degrees according to the traumatic condition. First degree: injuries to the epidermal layer, manifested by dermathemia, slight swelling, and mild pain. Second degree: injuries to the deeper layers of the skin or superficial dermis, manifested by local congestion, vesiculation, and baryodynia (sharp pain). In terms of superficial second-degree burns, there is significant post-injury swelling, edema, and vesiculation, but without deformity or proliferative scars after healing, and perichondritis rarely occurs. Deep second-degree burns, on the other hand, are vulnerable to infections in the course of crustal melting, and the incidence of perichondritis is highest. Third degree: the subcutaneous cartilage is injured. The skin is pale, and later eschars take shape; the auricle is partially or completely detached, accompanied by the lost sense of pain and other sensations. This can lead to scar contracture, mummification necrosis, and auricular detachment. With respect to first-degree burns and second-degree burns, there is no deformity according to prognosis.

Auricle burns are susceptible to infection due to factors such as susceptibility to pressure and moisture, head and face burn secretions and tears, which are often deposited in the auricular fossa when the patient is in the supine position, local swelling of the ear and poor blood circulation that lead to the ischemia of the auricular cartilage. The earliest symptom of suppurative perichondritis is localized persistent distending pain, which is difficult to relieve with analgesics. Localized redness, swelling, and pressure pain spread to adjacent areas in severe cases, and this may result in edema of the adjacent scalp and protrusion of the auricle; as the condition progresses, it softens locally, and seems to fluctuate. The pain will be relieved by incision and drainage or by self-diabrosis. If drainage is unsuccessful or there is a bony sequestrum, recurrent attacks may occur. In addition to local symptoms, there is also fever, loss of appetite, and leukocytosis. Pathological changes include extensive loss of cartilage matrix, purulent lacunae, lack of chondrocytes, neutrophilic infiltration, and the presence of bacteria.

1.3.2 Treatment Principles and Methods

First-degree burns do not require special treatment. Just keep clean. Like common burn wounds, superficial second-degree burns are treated by exposure or semi-exposure therapy: apply the suspension of silver sulfadiazine on partial wounds, or a layer of gauze or ribbon gauze daubed with polyvinylpyrrolidone iodine (PVP-I) ointment externally after debridement, keep the wound clean, and change the dressing once a day. Treatment of deep second-degree burns complicated by purulent auricular perichondritis: (a) use exposure or semi-exposure therapy by applying the suspension of silver sulfadiazine onto the wound locally or a layer of gauze or ribbon gauze daubed with PVP-I ointment externally after debridement; (b) keep the scab intact, avoid compression, and treat with far-infrared rays locally; (c) if conservative treatment is ineffective, perform surgery promptly. Make an incision large enough to drain the remarkably swollen area, the incision should be large enough, best in the shape of a fish mouth to avoid premature closure; (d) for limited lesions, excise part of the cartilage, and wait for the growth of granulation tissue before skin grafting; if the lesion is extensive, make a longitudinal incision along the helix, separate the anterior and posterior auricular flaps, remove the necrotic cartilage, place a ribbon gauze with antibacterial agents for drainage, wrap a piece of soft gauze around the pinna, change a fresh dressing once a day. After the pain and swelling have subsided, perform the surgical repair. After healing, there is generally a deformity, and the auricle can be reconstructed at a later date, if appropriate. (e) Concerning auricle burns, early

escharectomy for skin grafting is not recommended. It is better to wait for the eschar to separate and form granulation tissue. As for third-degree burns, use a sterile dressing or expose the wound to a sterile environment, and gradually sever the eschar a week before skin grafting. When the auricle becomes necrotic and detaches, there is no need to intervene surgically. Wait for the auricle to detach naturally. As the extent of detachment is often smaller than clinically estimated, the available cartilage support can be soaked in antibiotic solution as a backup. Also, the traditional Chinese treatment hydropathic compress is recommended.

The external auditory canal is exposed to burns as well. Due to local swelling and too much secretion, the auditory meatus is occluded, going against drainage, yet making for bacterial reproduction and infection, which in turn aggravates the swelling of the auditory meatus. It is a vicious circle. Patients may experience heavy headedness, local pain, and hearing loss, or worse, perichondritis and tympanitis. After healing, canal stenosis may occur due to scar contracture. In treatment, attention should be paid to cleanliness and dryness, removal of secretions, and unobstructed drainage. Penicillin and highly effective antibiotics are recommended for systematic infection.

Once infection occurs, take and culture secretions, and change the medication for sensitive strains. Be careful not to use ototoxic drugs as they may cause drug-related hearing loss. Intramuscular inject 1500 units of tetanus toxoid (if the skin test result is negative). After cicatrization, excise the scar for skin grafting when it becomes soft (it usually takes 3 months). Reshape the auricle in case of malformation.

1.4 Damage to the External Auditory Canal

Direct trauma to the external auditory canal is mostly soft tissue injury, such as skin abrasion and rupture. Indirect trauma is caused by fractures of the temporal bone or the temporomandibular joint trauma that affects the external auditory canal. Temporal bone fractures are predominantly longitudinal, and more common to damage the external auditory canal than transverse fractures. These injuries include fracture or malposition, and trauma to the external auditory canal may be accompanied by injuries to the tympanic membrane and auricle.

1.4.1 Clinical Manifestation and Diagnosis

Injuries to the external auditory canal are often concurrent with damage to the head and temporal bone. The skin of the external auditory canal is generally broken and swollen, and errhysis and hematoma are also seen. When complicated by temporal bone fractures, the wall of the external auditory canal may also be broken to clog the external auditory canal. The main symptoms are ear pain and hemorrhage, swelling of the external auditory canal, and epithelial erosion. It may cause hearing loss when the external auditory canal is blocked by blood clots or displaced bone fragments, and cerebrospinal fluid (CSF) otorrhea may occur if there is a temporal bone fracture. Secondary infection and excessive epithelial loss can result in scarring, stenosis, or osteophyte that impedes cerumen excretion and affects hearing.

1.4.2 Treatment Principles and Methods

The top priority is to prevent infection. Local disinfect the wound, but never rinse the external auditory canal. Remove the dirt, cerumen, and shredded broken tissue from the external auditory canal through suction or with a small curet or cotton swab. Try to keep the canal dry. For the sake of observation, it is better not to apply mercurochrome or methylrosanilinium chloride solution. After thorough clearing of foreign bodies, place a piece of iodoform gauze or silicone dilator in the ear canal to prevent stenosis and atresia. At the later stage, if atresia has developed, perform meatoplasty.

1. *Direct Treatment.*
   (a) For the external auditory canal (EAC) with only epidermis abrasions, disinfect with 75% alcohol and spray with antibiotic powder, or instill 2% phenol glycerin in the ear.
   (b) In the case of massive skin defects, press the EAC with a clean dressing or a dressing of 0.5% neomycin solution after disinfection until it is completely epithelialized. Compression facilitates epithelial repair and prevention of granulation and EAC stenosis.
   (c) If the EAC is narrowed by misalignment and bone fragments, reposition or take out the bone fragment.
   (d) If there is CSF otorrhea, take care not to block the EAC or use ear drops for fear of retrograde intracranial infection. Disinfect the EAC with 75% alcohol, and keep it unblocked. Let the patient lie in a semi-reclining position while avoiding coughing and constipation. If there is still cerebrospinal fluid after 2–3 weeks of observation, provide a referral to the neurosurgery for the repair of the ruptured meninges.
   (e) The duration of systemic antibiotic treatment depends on the traumatic condition and the treatment effect. Antibiotics or sulfonamides with high blood–brain barrier permeability are appropriate for patients with CSF otorrhea.
   (f) Significant infection or trauma caused by impurities requires concomitant tetanus antitoxin.
2. *Posttreatment***.** The EAC needs to be reconstructed when trauma has resulted in cicatricial stenosis or atresia.
   (a) If a small area is narrowed, excise the scar tissue until the EAC is slightly wider than normal, then dilate the EAC until it does not contract anymore.

(b) If there is extensive cicatricial stenosis or bony atresia, make an incision inside or behind the ear, separate the tissue, expose the stenosis or atresia, separate and excise the scar tissue. In the case of atresia or bony stenosis, isolate the closed flap from the narrowed flap, excise the dislocated bone fragment, remove the stenosis and fragment using an electric drill or osteotome. The EAC should be one times wider than normal after surgery. In cases where there is not much flap loss, reposition the flap, and compress for 1–2 weeks; if there is too much flap loss, repair it by skin grafting.

2 Blast Damage to the Middle Ear

2.1 Traumatic Tympanic Membrane Perforation

Tympanic membrane (TM) has the lowest pressure threshold and is most common in initial injuries. TM survivors account for 47% of initial injury survivors [6]. In addition to pressure amplitude, TM rupture is also related to head position, the cerumen in the external auditory canal, ear protection, and previous injury and infection. Researches show frequent TM hemorrhagic contusions within 50 m of explosion. In the past, TM rupture was often regarded as a marker of serious injury. However, recent researches have evidenced that TM rupture lacks sensitivity and specificity [7]. More accurate markers of severe blast injuries are burns greater than 10% of the surface area, facial fractures, and penetrating injuries to the head and trunk [8].

2.1.1 Clinical Manifestation and Diagnosis

In previous studies, many patients with tympanic membrane perforation had no symptoms or presented with only subjective dysaudia and tinnitus [9]. The symptoms of TM rupture chiefly include ear stuffiness and discomfort, tinnitus, hearing loss, bloody or watery secretion, pain, suppurative otitis media, and dizziness [10], which may lead to severe conductive deafness when complicated by the trauma to the ossicular chain. Blast injuries may also result in sensorineural deafness because of the damages caused to the round window and oval window [11]. When combined with temporal bone fractures, the external auditory canal may bleed and be accompanied by CSF otorrhea. Otoscopy and fiber optic otoscope are intuitive approaches to examining traumatic perforations evidenced by the blood and blood crusts in the external auditory canal and tympanic membrane. Tympanic membrane perforation is generally located in the tension part in an irregular shape [12]. Burst injuries often cause serious TM perforations and damages to both the tympanic ring and incus. Among a few patients, the symptoms are not obvious in the injured ear, but noticeable in the contralateral ear, so both ears must be diagnosed by endoscopic observation. Researches have lately found a strong association between TM rupture and unconsciousness [13]. Because of conductive deafness, concurrent inner ear trauma may result in sensorineural hearing loss, for which subjective hearing test (e.g., pure tone audiometry) and objective hearing test (e.g., brainstem auditory evoked potential, BAEP) can be performed. TM perforations may also present with varying degrees of tinnitus in variable duration, occasionally accompanied by transient vertigo. Blast damage to the tympanic membrane is often associated with varying degrees of otalgia at the time of or after getting injured, which is more pronounced in cases of EAC skin damage and infection.

2.1.2 Treatment Principles and Methods

First, remove the blood crust or foreign body from the external auditory canal, disinfect the EAC skin with alcohol, and keep it clean to prevent infection. After disinfecting the EAC again with alcohol, lightly plug the opening of the canal with a sterile cotton ball. Do not rinse or put antibiotic ear drops into the EAC for it may cause a secondary infection of the middle ear, but apply antibiotics systemically to prevent infection [9, 14]. If the wound is contaminated, tetanus antitoxin should be used. Let the patient first snuff up the mucus into the pharynx, then spit it out if necessary.

TM rupture is not an indication for tympanic membrane repair because of a high self-healing rate of 75% according to some researches, albeit up to 30% of high-frequency hearing loss. Patients whose tympanic membranes do not repair themselves for 3–6 months can be treated surgically. Recent studies have found that large tympanic membrane perforations with associated loss of surrounding tissue are difficult to heal [15]. Healing is also delayed around the curled edges of tympanic membrane perforations, and there is a high likelihood of cholesteatoma tympanitis [16]. Many studies have found that the main causes of delayed union of TM perforations are tympanosclerosis, malleus injury, and large perforation. Hence, topical application of epidermal growth factor and fibroblast growth factor may be a reasonably effective approach [17–20]. Surgical repair is possible in cases where the perforation is too large, or marginal, or repeated repairs have failed, or a follow-up visit is not possible. Early exploratory tympanotomy should be performed for patients with post-injury conductive hearing loss greater than 40 dB or symptoms of vertigo.

If a pyogenic infection has already occurred, treat it as suppurative otitis media. The most important treatment is to avoid further hearing impairment. In an Israeli study, 433 soldiers suffering from deafness and tinnitus were divided into two groups: one group was moved away from combat noise, whereas the other was left in the combat zone. In the first group, 30% of the patients had their hearing improved,

and this happened only to 8.7% of the patients in the second group [21]. So, to avoid further hearing damage, transferring injured patients to non-combat zones is an essential preventive and therapeutic measure.

In the case of tinnitus, sensorineural deafness, or vertigo, it can be treated with medications to improve microcirculation in the inner ear as well as neurotrophic drugs such as niacin, 654-2, vitamin B1, vitamin B12, ATP, coenzyme A, and cytochrome C. They are helpful in the reduction and cure of early tinnitus and sensorineural deafness.

For patients with CSF otorrhea, avoid blocking the ears and using ear drops. Let the patient lie in a semi-recumbent position, and apply systemic antibiotics to prevent infection. In cases of headache, nausea, emesis, obnubilation, and cranial nerve disorder, seek prompt diagnosis and treatment from the department of cerebral surgery.

2.2 Ossicles Trauma

In the circumstance of severe blast injuries, the pressure waves from the explosion can damage and break the ossicles in the middle ear that transmit sound vibrations or lead to fractures or joint dislocations, sometimes in conjunction with the rupture of the tympanic membrane. Like the tympanic membrane, ossicles can mitigate blast wave-induced damages to the inner ear. In addition, violent movement of the brain may damage the ossicular chain because of the inertial traction impact on the ossicles. Incus injuries are the most common damages to the ossicular chain, while the stapes and malleus are less likely to be injured. The ossicular chain can be broken due to the dislocation of the incus, or the fracture of the long process of the incus, which can also separate the stapes footplate from the oval window, or break the arch of stapes. Malleus damages are mainly characterized by manubrium mallei fractures. Trauma to the ossicular chain can result in 40 dB or above conductive deafness, or mixed hearing loss or complete deafness if combined with labyrinth damage, or craniocerebral injury if the trauma is severe.

2.2.1 Clinical Manifestation and Diagnosis

Two common types of clinical presentation are acute TM trauma and no signs of trauma to the tympanic membrane. Ossicular chain injury is usually followed by a conductive hearing loss, generally in the range of 50–60 dBHL, which is positive in Gelle tests, but the negative result does not exclude disruption of the ossicular chain. It may also be accompanied by vertigo and nystagmus. The adhesions of the dislocated articulations of ossicles are chiefly manifested by conductive deafness with predominantly low-frequency hearing loss, which is more severe than TM perforation. The air–bone gap is higher than 50 dB. When the patients with associated TM rupture are found having dislocated ossicular articulations after the ruptured tympanic membrane has healed, acoustic impedance is conductive to diagnosis, and the acoustic compliance value will increase significantly. The three-dimensional reconstruction technique of the ossicles is relatively intuitive for ossicle trauma, of great diagnostic value. The otoscope allows visualization of the ossicular chain and identifies the site of the lesion. The method of examination is as follows: let the patient lie in a supine position, and perform routine disinfection and draping. If the tympanic membrane is intact, the detection of suspected ossicular chain injury is usually confirmed by laboratory tests. For further morphological observation of the injury, exploration of the tympanic cavity by otoscope is recommended after tympanotomy, but only with the patient's permission. In the case of tympanic membrane perforations, it can be observed using an appropriate otoscope after surface anesthesia of the TM mucosa. Care should be taken to minimize damage to the skin of the external auditory canal or the mucosa of the tympanic cavity during the examination, and small amounts of bleeding can be stopped by compression with a gelatin sponge.

2.2.2 Treatment Principles and Methods

Reconstruction of the ossicular chain is performed according to different situations. In general, patients whose hearing does not improve within 3 months of injuries should undergo exploratory tympanotomy as soon as possible. Patients who develop vertigo and stapes invaginate fracture shortly after getting injured should be treated surgically as soon as possible, and facial nerve decompression can be performed at the same time if combined with facial nerve paralysis.

1. *Division of the incus and stapes*. It is the most common form of injury. In mild cases of separation, perform lysis on the ossicular chain to make the lenticular or long process attached to the head of stapes, then locally immobilize it with biogel and gelatin sponge. This method can be applied when the long process deficiency of the incus is not obvious. When it is impossible to reach the stapes, wedge a bone fragment between the incus and stapes to restore the connection, or take out the incus, and loosen and translocate the malleus until it is connected to the stapes.
2. *Stapes dislocation*. When the stapes footplate is mildly separated from the oval window, reposition the stapes by plucking under the microscope, then fix it with a gelatin sponge. When the stapes are almost completely out of the oval window or the footplate sags, remove the stapes stirrup, cover the oval window with gelatin sponge or fat, and place the stapes at the outer side of the oval window.
3. *Stapes arch fracture*. If the broken arch is close to the footplate, the chain of ossicles can be loosened, the stapes can be pushed downwards to press the arch against the

footplate. It should be tension-free; otherwise, the cicatrix can separate the arch from the footplate again. If the broken part of the arch is high, reconnection is no longer possible. The repair method is to take down the incus, and carve the body of the incus into a small pillar to connect the footplate and the manubrium.

Postoperative care: (a) Apply antibiotics for 5–7 days; (b) Prevent forceful nose blowing, limit head movements, and avoid the food that is too hard to bite.

2.3 Mastoid Process Injury

2.3.1 Clinical Manifestation and Diagnosis

Trauma to the mastoid process may be limited to the mastoid process in less severe cases, or the external auditory canal, tympanic cavity, and inner ear in severe cases, or accompanied by facial nerve palsy and brain injury.

2.3.2 Treatment Principles and Methods

Treatment depends on the extent and severity of the trauma. For mastoid trauma only, it is sufficient to protect hearing by removing the impurity, debris, and necrotic or septic mastoid cells; radical mastoidectomy is suitable for complications from the external auditory canal, tympanic cavity, and inner ear. The facial nerve can be explored intraoperatively, decompressed, or repaired later.

3 Blast Damage to the Inner Ear

The inner ear is vulnerable to brain injury-caused temporal bone fractures, explosion, detonation, and noise. Temporal bone fractures are often associated with damages to the middle ear, facial nerve, etc. (see Temporal Bone Fracture). Blast damage to the inner ear is caused by explosion pulses, noise, etc. Blast waves are pressure waves with a frequency of 10–20 Hz, which arise from accidental explosions in everyday life, during wartime, nuclear wars, blasts, bombing, and through the shooting of firearms of different calibers. Explosions generate pulse noise, which causes damage to the inner ear. The changes in the inner ear caused by blast waves and pulse noise are predominated by lesions of the spiral organ of the Corti ("spiral organ" for short), followed by the retrogressive process of the spiral ganglion and auditory nerve. The lesions of the spiral organ are manifested by the hemorrhage of the endolymph and spiral ligament, degeneration, dislocation, and atrophy of hair cells and supporting cells, degeneration and dislocation of the basilar membrane, the collapse of the vestibular membrane, and, in severe cases, dislocation of the otolith. In hair cell lesions, the invasion happens first to the lateral part of the outer hair cell, later the medial part of the outer hair cell, and finally the inner hair cell. The damage is most severe and occurs earliest around 4000 Hz in the injured area. The blast waves from explosions and the resulting noise can cause structural damages to the inner and outer ear hair cells, resulting in conductive deafness and/or vestibular dysfunction [22], which is primarily mechanical injury. The displacement of sensory cells and basilar membrane caused by blast waves may lead to temporary or permanent hearing loss [23]. Round and oval windows can also be damaged by blast waves to cause permanent hearing loss. Blasts mostly cause sensorineural deafness; the patients with sensorineural deafness account for 35% [24] of blast injury patients, and up to 64% suffer from progressive hearing loss [25]. According to audiometry, the hearing loss caused by blast waves is hearing impairment of at least one high frequency, ranging from mild to severe or complete deafness. The hearing loss resulting from acoustic trauma is generally around 4 kHz, whereas blast injuries produce an oblique high-frequency hearing loss, often affecting frequencies below 8 kHz.

3.1 Overview

3.1.1 Clinical Manifestation and Diagnosis

Clinical symptoms of inner ear injury include deafness, tinnitus, otalgia, bleeding, headache, dizziness, and vertigo. Deafness occurs immediately after the injury and is sensorineural. When it involves a temporary threshold shift (TTS), the damage can be partially or completely recovered within a week, but if not after 6 months, there is little chance of healing, which is called permanent threshold shift. Tinnitus has a very high incidence, up to 100%, either temporary or permanent, and sustained tinnitus is often unbearable. Vertigo is the result of labyrinth injury or brain injury, one of the sequelae of cerebral concussion. Otorrhagia, headache, and otalgia are signs of damage to the external auditory canal, middle ear, and skull base.

3.1.2 Treatment Principles and Methods

The sooner the injury is treated the better. According to the literature, treatment is most effective within 3 days of injury, satisfactory within a week, and effective within a month. It is more likely to be ineffective if treated 3 months later.

Treatment includes:

1. Sedative drugs such as diazepam and barbiturates, for a course of 1–3 weeks.
2. Vasodilators like niacin, rooted salvia, Kudzu root, and flunarizine.
3. Neurotrophic drugs, including vitamin B1, vitamin B12, adenosine triphosphate, coenzyme A, cytochrome C, etc.

4. Glucocorticoids. Early use can relieve edema and wound response, and help recovery. Dexamethasone and prednisone can be given in large amounts for a short period, and the course of treatment is 1–2 weeks.
5. Hyperbaric oxygen inhalation.
6. Inhalation of oxygen containing 7–10% carbon dioxide.
7. Tissue therapy. Inject 2–4 ml of placental tissue slurry intramuscularly, once a day or every other day, for about 3 months.
8. Traditional Chinese medicine that can promote blood circulation and remove blood stasis.
9. Tinnitus. Sedatives for antispasmodic treatment. Dissolve 1–2 mg/kg lidocaine in 200 ml normal saline as intravenous drips, or intravenous therapy of 0.5% solution. Administer 100 mg carbamazepine three times a day, gradually increase the dosage to 600–1000 mg/days for 2 months, and give phenytoin sodium for those sensitive to this drug. The masking method has some effect in suppressing tinnitus.
10. Symptomatic treatment in case of vertigo and headache.

3.1.3 Prevention

In most cases, inner ear injury occurs suddenly and unexpectedly, making it difficult to take precautions. Methods to mitigate or prevent inner ear injuries are introduced below:

1. When encountering blast waves, quickly lie in a prone position, keep the head away from the source of the wave, or hide in a protected and obstructed area to reduce the effect of the wave.
2. Plug both ears with fingers or other objects. Earplugs work best. Open your mouth and breathe at the same time.
3. Rapidly move into the shelter.

3.2 Inner Ear Barotrauma

Blast injury-induced inner ear barotrauma results mostly from rapid changes in ambient pressure. When the ambient pressure is greater than the middle ear pressure by more than 90 mmHg, the high air pressure compresses the Eustachian tube. It is not suitable to perform the Valsalva maneuver at the moment for it not only prevents the air from entering the tympanic cavity but also causes a sudden rise in cerebrospinal fluid pressure; the labyrinth window membrane is thereby broken along the direction of the tympanic cavity, resulting in sensorineural hearing loss. Light microscopy reveals deformation and degeneration of the cochlear Corti's organ, as well as hemorrhage of the endolymphatic duct in the vascular striae, tympanic canal, and vestibular canal. Electron microscopy shows the absence of hair cells, disorganization and lodging of stereocilia, partial loss of the cuticular plate, dilatation of the endoplasmic reticulum, and reduction of the mitochondrion.

3.2.1 Clinical Manifestation and Diagnosis

It is often accompanied by signs of middle ear damage, such as otalgia, a sense of ear occlusion, and hearing loss. Examination shows the tympanic membrane is invaginated or congested, and fluid or blood accumulates in the tympanic cavity. In severe cases, the tympanic membrane may rupture. If the inner ear is affected, the auditory and vestibular systems will be compromised. Presentation: otalgia, tinnitus, a sense of ear occlusion, hearing loss, and acute vertigo, which are combined with nausea and vomiting. Specialist checkup: pure tone audiometry, acoustic impedance, electrocochleogram, auditory evoked potential, and otoacoustic emission may show mixed or sensorineural hearing impairment, while vestibular function tests like spontaneous nystagmus and bithermal caloric tests may reveal abnormalities.

3.2.2 Treatment Principles and Methods

The principles of treatment are to manage to balance the pressure inside and outside the tympanic cavity, prevent infection, and eliminate the factors obstructing the Eustachian tube; improve middle ear ventilation, and clear middle ear effusion while giving symptomatic treatment such as analgesia, sedation, and rest; promote fluid absorption through local hot compresses and steam inhalation. For tympanic membrane perforations, disinfect with 75% alcohol, and cover with a sterile cotton ball. If ruptures of round and oval window membranes are suspected, perform exploratory tympanoplasty as soon as possible, locate the rupture under the otoscope or microscope, and repair with the fat of the earlobe or temporalis fascia.

3.3 Radiation Damage to the Inner Ear

The morphological damage to the cochlea and vestibular system caused by radiation is progressively exacerbated for some time after exposure. The reduction in vestibular functions does not occur immediately, but begins 2 weeks after exposure, suggesting that there is a delayed effect of radiation on vestibular dysfunction. The damage to the structure of the cochlea caused by radiation is also a direct cause of hearing loss.

3.3.1 Clinical Manifestation and Diagnosis

If the condition is serious, the patients may experience nausea and vomiting 15–30 min after exposure to a high dose of radiation. The onset time of vomiting is closely related to the total dose and the exposure dose rate, and also to the

sensitivity of the irradiated site. Symptoms within the next 24 h include fatigue, fever, diarrhea, etc. Often, blast wave-induced barotrauma is accompanied by manifestations of middle ear damage. Patients may present with ear pain, a sense of ear occlusion, and hearing loss. Examination reveals fluid in the tympanic cavity, and perforation of the tympanic membrane. Both blast waves and radiation can damage the inner ear, represented by otalgia, tinnitus, a sense of ear occlusion, dysacusis, and severe vertigo, accompanied by nausea and vomiting. However, there are cases where vestibular symptoms do not occur. The reason for this is that vestibular disorder is a slow-onset process that allows for central compensation. Because rays are penetrating, vestibular dysfunction caused by irradiation is bilateral, not unilateral.

3.3.2 Treatment Principles and Methods

1. In the case of tympanic membrane perforations, if the external auditory canals are clean and dry, follow-up observations demonstrate that most of them can heal spontaneously. If there are radioactive contaminants inside the external auditory canal, it should be washed and disinfected immediately, and applied with calamine lotion, antibiotic solution, or ointment as appropriate. Keep the surface of the tympanic membrane clean and dry, and repair the tympanic membrane if the tympanic membrane perforation has not healed within a month.
2. In the case of inner ear damage such as sensorineural deafness and vertigo, hormones, neurotrophic drugs, vitamins, and symptomatic treatment should be given as soon as possible.

3.4 Damage to the Vestibular System

The effects of explosion damage on the vestibular system are poorly understood. Many studies have found that vertigo and unbalance may be the damage to the central nervous system induced by traumatic brain injury [26]. However, it may also result from blast damage to the vestibular system.

3.4.1 Clinical Manifestation and Diagnosis

Damage to the vestibular system can occur in one or both ears, or affect the sense organ and vestibular nerve. Clinical manifestations are chiefly vertigo and unbalance. Some researches suggest that it may be caused by noise exposure [27]. Central vestibular injuries may be the result of a sequela of brain or brainstem trauma [28]. Patients with symptoms of either vertigo or unbalance should undergo a balance function test. As balance is maintained by multiple organs, so the test is not sensitive in assessing the site of the lesion [29]. Clinically, it is important to exclude other conditions that may cause vertigo, e.g., traumatic brain injury, orthostatic hypotension, cervical vertigo, visual defects, and side effects of ototoxic drugs. Screening tests for patients with possible vestibular impairment from blasts are recommended, including assessment of the cervical vertebra, eye movements, postural stability, gait, vertebral arteries. Peripheral vestibular function tests cover semicircular canal functions, through bithermal caloric tests or rotation; otolith tests involve vestibular evoked myogenic potentials (VEMP). The central vestibular function is assessed during eye movement tests and vestibular suppression tests.

3.4.2 Treatment Principles and Methods

Patients with balance disorders or semicircular canal dysfunction can be treated by vestibular rehabilitation therapy (VRT), or physiotherapy, i.e., changing the activity level, medication, and food habit; aggressive treatment of conditions such as eye disease and infections that may cause vertigo; or surgery. Benign paroxysmal positional vertigo (BPPV) is more difficult to treat. The adaptation process rarely occurs after an otolith injury, and researches have shown that conventional vestibular rehabilitation therapy for patients with BPPV is generally ineffective [30].

3.5 Perilymph Fistula

During an explosion, the blast wave causes an instantaneous rise in ambient pressure and a sharp rise in middle ear pressure, which can break the round window membrane, forming an abnormal passage between the perilymph and the middle ear from which the perilymph fluid leaks out. Temporal bone fractures and ear trauma arising from an explosion can also lead to perilymph fistula, and the most common injury is rupture of the oval window membrane, namely the annular ligament.

3.5.1 Clinical Manifestation and Diagnosis

Sudden sensorineural deafness and tinnitus are most often found on one side or, rarely, on both the sides. Sensorineural deafness often occurs suddenly, usually accompanied by tinnitus, and needs to be distinguished from sudden hearing loss. The degree of hearing loss varies. If treatment is delayed, the ability to hear will gradually decline. Some patients may also experience hyperacusis. Autonomic dysfunction such as rotatory vertigo, nausea, vomiting, pallor, and diaphoresis may occur immediately. After a period of compensation, these symptoms will gradually diminish, but a sense of shakiness and positional vertigo will remain for a considerable period. It may also be accompanied by symptoms of brain trauma, and damage to the cranial nerve and motor system. Violent changes in air pressure should be noted for hematological and cardiovascular symptoms. The tympanic membrane may be ruptured when observed with an

otoscope or fiber optic otoscope. Pure tone audiometry shows a sensorineural hearing loss curve of predominantly middle to high frequencies. Vestibular function tests may be positive for spontaneous nystagmus and Romberg's sign. Glycerol and Tullion tests may be positive too. Through acoustic impedance examination, it is found that there is little perilymph leakage if the tympanic membrane is not perforated, and the tympanogram is usually the type A. However, some patients may experience vertigo, nausea, vomiting, and nystagmus when the external auditory canal is pressurized or decompressed. The electrocochleogram SP/AP ratio may appear to be increased.

3.5.2 Treatment Principles and Methods

Early conservative treatment, with a head elevation of 30–40°, and absolute bed rest for more than half a month. Avoid motions that increase the pressure in the middle ear cavity and brain, such as forceful defecation, forceful nose blowing, and violent coughing. Symptomatic treatments, such as sedatives and anti-vertigo drugs, can be used in severe cases of vertigo. If conservative treatment is not effective, surgical exploration is required. Exploratory thoracotomy of the two window membranes is possible to repair the fistula. Avoid creating a new perilymph fistula during surgery.

3.6 Labyrinthine Concussion

An explosion can produce violent air oscillations, which are transmitted to the inner ear via the tympanic membrane and ossicles, causing trauma to the inner ear. The closer the detonation point, the more severe the damage, which can be accompanied by damages to the tympanic membrane and ossicles. Explosion-induced temporal bone fractures may also cause damage to the inner ear and result in a labyrinthine concussion.

3.6.1 Clinical Manifestation and Diagnosis

Usually, blast-induced labyrinthine concussion is accompanied by intracranial hemorrhage and cerebral concussion. Bilateral deafness is most often present after injury and may be accompanied by tinnitus. Post-injury rotatory vertigo associated with autonomic dysfunction such as nausea and vomiting occurs, which can last from a few days to several months. An otoscopic examination may reveal a perforated tympanic membrane with blood crusts or bleeding around the perforation. Pure tone audiometry often shows sensorineural hearing loss; if concurrent with middle ear damage, mixed deafness may be present.

Most patients acquire vestibular dysfunction according to the vestibular function examination of the injured ear.

3.6.2 Treatment Principles and Methods

Patients with craniocerebral trauma should be treated aggressively for craniocerebral disease and to maintain stable vital signs. In cases of hearing loss, treatment with neurotrophic drugs, vasodilations, and vitamin A is indicated. Patients with vertigo can be given symptomatic treatment, such as sedation and anti-dizziness medications. For those who do not improve and those whose vertigo persists in later stages and affects their quality of life, vestibular function training can be intensified to shorten the vestibular compensation time.

3.7 Acoustic Trauma

Many experimental studies have shown that the hearing system is most vulnerable to blast injuries. Blast-induced hearing loss, on the other hand, results from extensive cochlear damage, and exposure to terrorist or military explosions, which can lead to moderate to severe sensorineural deafness [31], permanent sensorineural deafness being the most common type, accounting for 33–78% [32]. In addition to mechanical damage to the auditory system in the explosion, cellular and molecular processes can also be triggered to cause long-term impairment by activating apoptotic pathways and inflammatory molecular mediators, which damages the outer hair cells and cochlear supporting cells, and thus results in degeneration of cochlear cells, and accumulation of lymphocytes and macrophages in the perilymph fluid of the tympanic canal. Some researchers have found that oxidative stress is related to both steady noise- and impulsive noise-induced hearing loss [33], so we believe that oxidative stress may also play a role in blast-induced hearing loss. Acoustic trauma varies with the intensity, frequency spectrum, time course, and type of detrimental acoustic stimuli, but also has some commonalities. Temporary damage manifests itself as a temporary threshold shift (TTS), namely change in the blood flow volume of the inner ear and damage to the organ of Corti arising from the relatively lower intensity of acoustic stimuli or short duration of exposure, which can gradually recover after the harmful acoustic stimulus disappears. Permanent damage manifests itself as a permanent threshold shift (PTS), possibly resulting from long-term exposure to harmful acoustic stimuli, or single or several intense sound stimuli. The most common type is noise-induced hearing loss, with the most serious case occurring at 3 kHz, 4 kHz, and 6 kHz. The frequency at which noise-induced hearing loss is most pronounced is related to the sound energy contained in the frequency one octave band lower. This means that hearing loss at 1 kHz and 2 kHz is associated with the sound energy in the 300–

600 Hz band, and hearing loss at 4 kHz is associated with the sound energy in the 2 kHz band.

3.7.1 Clinical Manifestation and Diagnosis

Hearing impairment is the most common symptom, the extent of which is closely related to the distance from the explosive source, the number of explosions, and the magnitude of the burst. Conductive hearing loss may occur when the middle ear is damaged by explosive blast waves, such as the tympanic membrane rupture. Sensorineural hearing loss may develop when the inner ear and the auditory nerve are injured. Mixed deafness is represented by the combined injury of the middle ear, inner ear, and auditory nerve. Hearing loss is always complicated by tinnitus, usually above the frequency of 4 kHz, but has no great impact on daily life. Tinnitus is the main complaint that has a strong impact on the quality of life. In terms of middle ear trauma, the tympanic membrane is mostly congested, ruptured, or perforated, and the patients may have symptoms of ear pain. Severe blast injuries may compromise the inner ear and vestibular system, which is manifested as rotatory vertigo, and balance disorder, concurrent with the symptoms of nausea and vomiting. Otoscopic examination reveals asymptomatic traumatic perforation, congestion, rupture, and hemorrhage of the tympanic membrane. The extent of hearing impairment is assessed via hearing screening, including pure tone audiometry, acoustic impedance, otoacoustic emission, and auditory brainstem response (ABR). Patients suspected of vestibular system damage undergo vestibular function examination. Damages to the middle ear, inner ear, and temporal bone are diagnosed by imaging. Acoustic impedance finds acoustic stimulation-caused damage to the middle ear transducer. Tympanogram "D" or "B" shows that part of all of the stapedius reflex is absent. If the acoustic trauma does not involve the transducer, the tympanogram is usually the type "A," and the stapedius reflex is partially or completely absent. In the early stages of electric response audiometry (ERA), the ABR threshold is elevated. ABR waveform is seldom found in the later stages, except for mid-latency response, such as a 40 Hz auditory event-related potential (AERP). Patients with chronic exposure to harmful stimuli often have difficulty eliciting transient evoked otoacoustic emission (TEOAE), and spontaneous otoacoustic emission (SOAE) or only some distortion product otoacoustic emissions (DPOAE) are found in early stages.

3.7.2 Treatment Principles and Methods

Patients with blast injuries should first be treated aggressively for life-threatening trauma, such as hemorrhage and craniocerebral injury, then moved away from the blast scene to avoid repeated exposure to blast noise. In the early stages of acoustic trauma, most of the patients can get well by rest and after leaving the harmful acoustic environment, or be treated with high doses of vitamins, vasodilators, traditional Chinese medicine, among other conventional methods. Patients without Eustachian tube injury can be given hyperbaric oxygen treatment. When the middle ear is traumatized, the first step is to remove all foreign bodies from the external auditory canal other than putting in ear drops. Usually, traumatic tympanic membrane ruptures are self-healing. Tympanic membrane repair can be considered for large perforations (>80% of the surface area) and those that do not heal in 3–6 months. The presence of infection and progression to suppurative otitis media should be treated as suppurative otitis media. In case of trauma to the inner ear, early treatment is required to restore hearing and control symptoms. Neurotrophic drugs, vasodilators, sedatives, and antidincis can be applied to improve hearing and for symptomatic treatment (CBT). Patients with tinnitus can be treated with tinnitus retraining therapy (TRT) or cognitive behavioral therapy. In recent years, researchers have made some progress in finding ways and means to treat and improve acoustic trauma. Some researchers have found that the human β-nerve growth factor (hNGFβ) can increase the activity of antioxidant enzymes, and protect against blast-induced hearing damage; furthermore, it can inhibit the activity of apoptosis-associated proteins, and protect the ear from blast-induced acoustic trauma. Adenovirus-mediated human β-nerve growth factor (Ad-hNGFβ) can be expressed at high levels in the cochlea damaged by an explosion, indicating a protective effect on cochlear spiral ganglion cells after blast exposure [34]. This study provides a new, viable therapy for acoustic trauma-caused hearing loss. Leupeptin is a calpain inhibitor able to restrain cell death and has been shown to reduce acoustic trauma-induced hearing loss when applied to the middle ear cavity [35]. Prompt application of corticosteroids and hyperbaric oxygen therapy for acute acoustic trauma can help control hearing loss to some extent [36]. The research of the above approaches and methods, though not yet mature, has offered an insight into the treatment of acoustic trauma.

3.7.3 Prevention

The central issue concerning acoustic trauma is prevention, the emphasis of which is trying to avoid harmful acoustic stimuli. To effectively avoid harmful acoustic stimuli, it is important to understand the relationship between the physical nature of acoustic stimuli and acoustic trauma. This will determine the type of acoustic stimuli that are likely to cause acoustic trauma. Since noise-caused injury is the most common form of acoustic trauma, understanding the relationship between the physical properties of the noise environment

and the hearing loss caused is the main basis for the prevention of noise-induced hearing loss.

4 Blast Damage to the Central Auditory Nervous System

Blast injuries to the central auditory nervous system often coexist with traumatic craniocerebral injury (or traumatic brain injury). There is a lack of diagnostic criteria. As a result, symptoms are often misdiagnosed as post-traumatic stress disorder, psychological health problems, and cognitive dysfunction, and it is difficult to logically test patients with multiple injuries. Brain concussion can also lead to audiological symptoms like hearing loss and vertigo because the auditory centers such as the temporal lobe, thalamus, and corpus callosum are susceptible to damage. The main mechanism of blast injuries to the central auditory nervous system is swelling and disruption of axonal connections resulting from shearing, stretching, and other acting forces applied to the axons and small vessels [37].

4.1 Clinical Manifestation and Diagnosis

Damage to the central auditory nervous system makes it hard to hear and localize sounds in background noise [38]. Injuries to primary auditory cortices on both sides namely cortical deafness manifest as temporary or persistent insensitivity to all nerves, speech and language comprehension disorder, and impairment in recognizing and discriminating environmental sounds. Whether or not the damage is caused to the peripheral or central auditory nervous system, the exact site of the damage needs to be determined. Only a few tests are currently available to assess central nerve injuries, and the association of existing test results with specific dysfunctions is imprecise. Hence, the collaboration among audiologists, cognitive psychologists, and neurologists is necessary for the diagnosis of injuries to the central auditory nervous system. For patients suspected of such injuries, a range of tests should be performed, such as staggered spondaic word (SSW), noise test slot, masking level difference, auditory and temporal processing and patterns, binaural speech, monaural low redundancy tests, binaural interactions, and electrophysiological detection, as well as mid and late latency responses, to know the reactivity of the auditory centers to auditory stimulation.

4.2 Treatment Principles and Methods

Two steps, including hearing training and general management, are advised by clinical supervision. The aim of hearing training is to exploit the plasticity of the auditory system to alter the neural encoding of sound and the subsequent timing of brainstem response. The training should start as early as possible to maximize brain plasticity individually, plus a series of examinations. General management strategies include the use of environmental tactics, such as frequency modulation, and compensatory strategy learning. Targeted remedies include phonological awareness, discrimination training, auditory closure training, rhyme training, text-to-speech, and improvement of information transfer between the two cerebral hemispheres through cross-channel activities. In addition to the above therapeutic measures, Frederick J. Gallun et al. find that low-gain hearing aids and remote microphone techniques are useful in the treatment of damages to the central auditory nervous system [39].

5 Temporal Bone Fracture

Petrous fractures are most common among the injuries to the petrous, squamous, and mastoid portions of the temporal bone as the petrous bone itself has irregular margins and lacks elasticity in its connection with the surrounding cranium, making it vulnerable to fractures when subjected to external forces. The inner ear is located within the petrous bone and is often injured due to temporal bone fractures. They are classified as longitudinal, transverse, and mixed fractures according to the relationship between the fracture line and the long axis of the petrous bone. Petrous bone fractures are usually complicated by traumatic brain injuries [40].

5.1 Clinical Manifestation and Diagnosis

1. *Longitudinal fractures*. The fracture line is parallel to the petrous part. This kind of fracture accounts for 70–80% of temporal bone fractures, and often damages the middle ear. The fracture line mostly crosses the squamous part via the posterior apex of the external acoustic meatus (bony canal) to the tympanic membrane, spans the tegmen tympani (aka tegmental wall or roof of the tympanic cavity), and goes inwards along the tensor tympani muscle until it arrives at the geniculate ganglion, or the foramen spinosum along the carotid canal, and faces toward the clivus. In severe cases, the fracture line can extend from the lacerated foramen to the opposite side via the base of the sphenoid bone. The areas passed by the fracture often cause fractures to the long process of the incus, the neck of the malleus, the arch, and the footplate of the stapes. Because of tegmental wall fractures, the meninges and tympanic membrane may rupture to cause CSF otorrhea. Clinical manifestations include:

(a) Constitutional symptoms: Temporal bone fractures are often combined with different degrees of craniocerebral injuries and other neurological symptoms.
(b) Hearing loss: The fractures parallel to the long axis of the petrous part ("axis" for short) mainly cause damages to the middle ear other than the labyrinth (rarely). So, hearing impairment is slight, mostly conductive. Intense blasts damage the cochlea, the auditory nerve, and even the auditory cortex, resulting in long-lasting deafness. There is usually no tinnitus or only low-frequency tinnitus.
(c) Otorrhagia: It is usually in small amounts and may last for several days. When the posterior wall of the external auditory canal is fractured, there will be soft tissue edema behind the ear, and ecchymoma; in cases of tympanic membrane rupture and tympanic cavity injury, blood will flow from the external auditory canal.
(d) CSF leak: Pink or clear liquid is flowing from the external auditory canal or nostrils. If there is no crust after coagulation, it suggests the possibility of cerebrospinal fluid.
(e) Peripheral facial palsy (PFP): The incidence is relatively low, about 20%. The injury is usually minor and able to recover gradually in most cases.

2. *Transverse fractures*. The fracture line is perpendicular to the axis and constitutes approximately 20% of temporal bone fractures. The line extends from the posterior cranial fossa to the middle cranial fossa and crosses the bony labyrinth in multiple forms. Usually, it runs to or approaches the foramen lacerum from the foramen magnum, jugular foramen, vestibule, and internal auditory canal; it often damages the inner ear structure, complicated by the symptoms of cochlear, vestibular, and facial nerve injuries, such as severe hearing loss, tinnitus, vertigo, balance disorder, facial palsy, and CSF otorrhea. Clinical manifestations include:
(a) Constitutional symptoms: Same as longitudinal fractures.
(b) Hearing loss: It can easily injure the vestibule of the inner ear and the internal acoustic meatus. The cochlea and the semicircular canals may also be compromised, but it less often happens to the middle ear. The hearing is badly impaired, usually sensorineural with persistent high-frequency tinnitus.
(c) Vertigo: Transverse fractures mostly cause damages to the labyrinth and vestibular nerve, resulting in severe rotatory vertigo, accompanied by autonomic dysfunction such as nausea, vomiting, and diaphoresis, tilting to the affected side, and spontaneous nystagmus on the unaffected side. The symptoms can last 2–3 weeks, and later vestibular function tests may show afunction.
(d) Peripheral facial palsy (PFP): As the fracture line is perpendicular to the direction of the facial nerve, the nerve is prone to compression or laceration to cause peripheral facial palsy, which accounts for 50% or so of the injuries, and is not easy to recover.

Mixed fractures are rare but serious, often combined with severe craniocerebral trauma. The fracture lines are distributed in multiple directions, both transversely and longitudinally, causing tympanic cavity fractures and labyrinth fractures, and may have clinical manifestations of middle and inner ear injuries. The diagnosis of temporal bone fractures can be made qualitatively and locally through the clinical presentation, history of trauma, otoscopy, audiometry, neurological examination, and imaging. Pure tone audiometry can be performed when the patient is awake to assess the degree of hearing loss, while auditory brainstem response (ABR) can be performed when the patient is unconscious to understand the hearing impairment. Early tests including nerve excitability, salivary gland secretion, and facial electromyography can be done to understand facial nerve injuries. Imageological examination like temporal bone X-ray photograph and CT can be performed to figure out the specific condition of temporal bone fractures. The X-ray film of the skull base is less likely to show longitudinal fractures, and the negative result does not rule out fractures. In general, traumatic brain injuries concurrent with CSF otorrhea suggest a petrous bone fracture, while CT scans reflect the propagation of the temporal bone fracture, and can also detect intracranial hematoma and pneumatosis. Quantitative tests of CSF otorrhea glucose and radionuclide scan can assist in making a definitive diagnosis.

5.2 Treatment Principles and Methods

Temporal bone fractures are generally associated with craniocerebral trauma and should be aggressively treated in collaboration with neurosurgeons to maintain stable vital signs [41].

1. Maintain airway patency, clear away secretions and foreign bodies in the upper respiratory tract. For unconscious patients whose tongue falls backwards (glossocoma), pull the tongue out of the mouth, and fix it outside the mouth with a pair of tongue forceps or a thick thread passing through the dorsum of the tongue from about 2 cm behind the tip of the tongue, or put in a ventilation tube, or perform tracheal intubation or tracheotomy if ineffective. Use a respirator or administer oxygen when needed.
2. Maintain the circulatory function. The fall of blood pressure is a sign of internal bleeding and requires fluid or blood transfusion to find out the bleeding point and stop it, and vasopressors should not be used.

3. In terms of cerebral edema, treat it by dehydration, intravenously inject 50% glucose, or intravenous drip mannitol, administer oxygen, and put ice packs on the brain, which are helpful to protect the brain tissue.
4. For CSF otorrhea patients, disinfect the external auditory canal with 75% alcohol (take care not to block it) and change the dressings if wetted by the cerebrospinal fluid. Remove the foreign bodies and blood from the external auditory canal under aseptic conditions, and be sure not to put ear drops when there is a tympanic membrane rupture. If the tympanic membrane has been perforated for more than 3 months, give surgical treatment to repair the tympanic membrane depending on the situation. Lift up the patient's head or make the patient in a semi-reclining position, and avoid upper respiratory tract infections, coughing, or straining to defecate. CSF otorrhea will stop spontaneously; if it does not stop 3 weeks later, the lacerated foramen can be repaired by covering the injured duramater with temporalis or fascia via the pathway from the ear to the brain. Antibiotics that can easily unblock the blood–brain barrier should be administered in time to prevent the ear infection and intracranial infection.
5. When associated with peripheral facial palsy (PFP), surgical decompression should be performed as early as possible if the condition permits. Minor injuries necessitate 6-month observation. If there is no sign of recovery, facial nerve exploration or facial nerve decompression is indicated.
6. Sensorineural deafness can be treated with prednisone, adenosine triphosphate, and vitamin B. Regarding conductive deafness and mixed deafness, the middle ear should be explored as soon as possible, and different tympanoplasties should be performed according to the lesion. Tympanic membrane perforations can be repaired using temporalis and fascia at the same time.
7. Patients with vertigo should be bedridden for 2–3 weeks and given sedative drugs such as dimenhydrinate (Dramamine), and vertigo recovers gradually in most cases.

6 Traumatic Facial Paralysis

There are two types of facial nerve injury: direct and indirect. Direct injury is caused by surgery, fractures, oncothlipsis, or inflammation that disconnects nerves or hinders conduction. Indirect injury is caused by viral infections or allergic reactions that make the facial nerve edematous and ischemic, which are further aggravated under pressure within the fallopian canal, thus creating a vicious cycle. Bacterial toxins and physical injuries result in the spasm of facial nerve blood vessels, affect the blood supply, and degenerate the facial nerve. Sometimes, trauma occurs without fractures, but the spasm of blood vessels caused by severe skull concussion can lead to nerve ischemia and hypoxia that affect the facial nerve functions. Facial nerve injury is generally divided into three types of pathological changes: (a) Neurapraxia: It is a temporary conduction disorder resulting from mild injuries to the facial nerve, usually due to nerve compression. This type of lesion often involves motor nerve fibers, proprioception, tactile sensation, temperature, pain sensation, and autonomic nerve fibers in order. There are no associated changes in axonal structure or disconnection of myelin sheaths or nerve fibers; innervated muscles are paralyzed but not atrophied; the neuromuscular function can be fully restored within a short time after the cause is removed. The order of recovery is the reverse of that described above, i.e., autonomic nerve fibers first and motor nerve fibers last. (b) Axonotmesis: The distal facial nerve, axon, and myelin sheath are degenerate while the myelin sheath is intact, and axonal degeneration often occurs 24–72 h after injury. The degenerated axon and myelin sheath are absorbed, so the axon can regenerate from proximal to distal along the hollow canalis vaginalis until the motor endplate, whereby nerve conduction is partially or fully restored. The recovery time is longer than that of the first type described above. It often takes months. (c) Neurapraxia: A complete transection of the nerve trunk with distal degeneration. The lacerated ends are generally unable to be fit together. Although the proximal end can regenerate, it can only form a neuroma due to the loss of myelin membrane guidance and must be surgically removed for anastomosis or grafting. Generally, this way of recovery is incomplete, with the sequela of coordinated muscular movements in varying degrees.

6.1 Clinical Manifestation and Diagnosis

In terms of unilateral peripheral facial palsy, there is asymmetry on both the sides of the face. The expressions and actions on the affected side are lost, and the patients are unable to frown or close their eyes. Over time, lower lid ectropion occurs, tears run down the face, and the conjunctiva and cornea are dry and inflamed due to prolonged exposure. The nasolabial groove on the affected side becomes shallower, and the labial angle droops and moves toward the healthy side, which is noticeable when the face is in motion. Air leaks from the mouth after cheek blowing and plosives like "b" and "p" cannot be pronounced. Liquids tend to escape from the corners of the mouth when the patients are eating. Solid food easily gets lodged between the cheek teeth. The patients with bilateral facial paralysis have stiff and expressionless faces.

1. *Toposcopy.*
   (a) Tear secretion experiment: It is a clinical method to test the functions of the greater superficial petrosal nerve. Place two pieces of 5 cm long and 0.5 cm wide filter paper with one end folded in the centers of the dried lower fornices without anesthesia. Five minutes later, compare the lengths of wetting. If the difference is more than double, the result is positive, suggesting that the injury is above the geniculate ganglion. It can be induced by the nasal mucosa stimulated due to fewer tear secretions.
   (b) Taste test: Compare tastes by applying sour, sweet, bitter, salty liquids to the anterior two-thirds of the tongue on both the sides or using an electrogustometer. The normal taste threshold is 50–100 mA, and a positive result of more than 50% greater on the affected side than the healthy side indicates that the damage is above the plane of the chorda tympani nerve. Test results vary greatly between individuals and are often unreliable among the elderly, smokers, and alcoholics.
   (c) Acoustic stapedius reflex (ASR): Acoustic reflex will not be elicited when the lesion on the affected side is above the level of the nervus stapedius.
2. *Identification of Injury Degrees.*
   (a) Facial palsy assessment: Refer to House's percentage method to evaluate facial muscle function. Level 1: normal (100%). Normal facial muscle functions in all regions. Level 2: mild dysfunction (99% to 75%), with mild deficiencies in facial muscle functions that can only be detected by close examination. When the facial muscles are relaxed, the tensile force is normal, and it is symmetrical; when the facial muscles are mobile, there is no problem with the movement of the frontal muscle, the eyelids can be closed, and the corners of the mouth are mildly asymmetrical. Detailed examination reveals mild dysfunction of facial muscles with the secondary onset of slight coordinated movements, but there is no facial spasm or hemifacial spasm. Level 3: moderate dysfunction (75% to 50%). The dysfunction of facial muscles is noticeable, but the face does not look ugly. Differences appear on both the sides of the face, and there is no severe dysfunction, extensive muscular movements, contractures, or hemifacial spasms. When the facial muscles are relaxed, both the symmetry and tensile force are normal. When the facial muscles are moving, there is only slight to moderate movement in the frontal muscle, and the eyelids can be closed with force. There is also a mild asymmetry in the forceful movement of the corners of the mouth. The presence of a few muscular movements, contractures, or hemifacial spasms is also considered level 3 facial palsy. Level 4: relatively severe dysfunction (50% to 25%), i.e., significant facial muscle dysfunction. The face is ugly and asymmetrical. When the facial muscles are relaxed, both the symmetry and tensile force are normal. When the facial muscles are moving, the frontal muscle remains motionless even with force, the eyelids cannot be fully closed, and the corners of the mouth are asymmetrical even when moved with force. Regardless of motility, this grade is reached when there are severe muscular movements, contractures, and hemifacial spasms that affect the function of facial muscles. Motionless, the eyelids are not fully closed, the mouth and lips move a little even when strained, and the corners of the mouth move slightly. Level 5: severe dysfunction (25% to 0%), with just a few movements observed. The facial asymmetry is observed when relaxing facial muscles, the corners of the mouth droop, the nasolabial groove becomes shallower or disappears; when the facial muscles are in motion, the frontal muscle remains motionless. There are usually no coordinated muscular movements, contractures, or hemifacial spasms. Level 6: complete paralysis (0%). The facial muscles are tensionless, asymmetrical, and motionless without associated movements, contractures, or hemifacial spasms.
   (b) Nerve excitability test: The facial nerve trunk below the stylomastoid foramen is stimulated transdermally with square wave electric pulses at a time course of 0.3 ms once per second to determine the minimum current intensity resulting in facial muscle contractures. A difference of more than 2 mA between the two sides is considered neurodegeneration on the lesion side. When stimulated by high intensity, it can be classified as mild, moderate, severe, or completely denervated, according to the extent of response, compared to the normal side. This method is simple, painless, and indicated for over 72 h of unilateral facial paralysis.
   (c) Electromyography: The latency, waveform, and amplitude of motor units are recorded by stimulation with electrodes inserted into the facial muscles. Normal muscles have no electrical activities at rest and show biphasic or triphasic potentials during slight voluntary movements, as evidenced by 50–1500 mV potentials every 30–50 ms. Interference waves appear after forceful contractions. Fibrillation potentials after nerve degeneration or muscle denervation often occur 14–21 days after nerve injury. Multiphase potentials appear after nerve reinnervation. The degree of nerve and muscle damages and the probability of recovery can be judged by the type of potential, latency, and amplitude change.

(d) Electroneurography: A 0.2 ms, 60–120 V rectangular electric pulse is applied once per second to stimulate the facial nerve trunk of the stylomastoid foramen via a bipolar surface electrode, while the compound muscle action potential of the muscle groups around the mouth is elicited with another similar electrode and recorded to measure the intensity of the current inducing potentials, the latency under maximum stimulation, and the superimposed wave amplitude. The ratio of the wave amplitude on the affected side to that of the unaffected side is approximately representative of the number of paralyzed nerve fibers.

3. *Topical Diagnosis.*
   (a) Nuclear damage: It is often associated with abducens nerve palsy, Millard–Gubler syndrome, and contralateral hemiparesis. Taste, tear, and salivary secretions are normal on the affected side.
   (b) Damage to the cerebellopontine angle segment: Facial palsy associated with nerve deafness and vertigo, loss of corneal and stapedius reflexes, normal or reduced taste, tear, and salivary secretions.
   (c) Labyrinthine segment injury: Facial palsy associated with nerve deafness, loss of stapedial reflexes, reduced or absent taste, tear, and salivary secretions.
   (d) Tympanic segment injury: Associated with conductive deafness, tinnitus or hyperechema, loss of stapedial muscle reflex, reduced taste and salivary secretions, but normal lacrimal gland secretions.
   (e) Mastoid segment injury: If above the chorda tympani nerve, it is only associated with the loss of taste and salivary secretions; if below the chorda tympani nerve, the results of topical diagnosis are normal.
   (f) Extratemporal segment injury: Involving paralysis of all the muscles innervated by the trunk, and paralysis of the corresponding muscles innervated by a branch.
   (g) Topical diagnosis items are all normal.

6.2 Treatment Principles and Methods

Nerve decompression is recommended after 2 weeks of facial paralysis if there is no response to direct current stimulation during electrical stimulation tests or electromyography shows myofibrillation. It has also been reported that after more than 10 years of paralysis, there is a possibility of partial recovery after surgery as long as the muscles do not atrophy completely. With respect to nerve compression or edema caused by trauma or surgery, nerve decompression aims to relieve the compression and alleviate the edema to prevent degeneration in the early stages, or to provide conditions for axonal regeneration for advanced degeneration [42].

Facial nerve (rerouting) anastomosis: It is appropriate for neurological deficits from any cause. The facial nerve is rerouted from the geniculate ganglion and can be made 5 mm shorter from the inner wall of the tympanic cavity to the stylomastoid foramen. Thus, the surgery can be performed for defects within 5 mm.

Facial nerve grafting: Appropriate for extremely long facial nerve defects or patients not suitable for rerouted suturing.

Facial-cranial nerve swap: It can be exchanged with the accessory nerve, glossopharyngeal nerve, or hypoglossal nerve, of which the facial-hypoglossal nerves are always swapped.

Cross facial nerve grafting: Appropriate when the facial nerves on one side are incapable of regeneration or there are extratemporal nerve trunk deficits.

7 Summary

Blast injuries can cause multiorgan dysfunction. The patients should be aggressively rescued according to their conditions as well as the order of priority through the joint efforts of multidisciplinary clinicians. Ear trauma includes damages to the outer ear, middle ear, inner ear, and central auditory system, and temporal bone fractures. Depending on patient conditions, audiological, neurological, and imaging examinations should be carried out to initially locate the injury, and targeted and specific treatment plans should be made to enable the patient to receive active and effective treatments. Varied and complex ear injuries necessitate a wide range of protective measures. Headphones and earplugs can go some way to prevent sound, pressure, and debris from damaging the outer and middle ears. Researches have proven that the use of antioxidants to scavenge free radicals and increase blood flow, and drugs to block signal factors in the cell death pathway help protect against hearing loss, which has broken a new path for prevention and treatment of hearing loss resulting from blast injuries. In the matter of auditory system injuries, early diagnosis and immediate treatment are important to heighten the rehabilitation effect, lessen cognitive dysfunction, and improve the quality of life.

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Blast-Induced Traumatic Brain Injury

Minhui Xu

1 Epidemiology

Blast-induced traumatic brain injury (bTBI) is an important research topic in military medicine, disaster medicine, and trauma medicine. With the increase in international terrorist activities and unexpected incidents (such as explosions of gas, gasoline, coal mine gas, etc.), as well as the rapid development and large-scale use of shock wave-enhancing weapons, bTBI is more common in peace and war time than before with a growing trend.

1. *Morbidity rate*. It is estimated that about 320,000 service personnel or 20% of soldiers have suffered from bTBI with the incidence of bTBI about 40–60% in recent military operations of the US military. Large-scale epidemiological surveys and screenings were conducted in soldiers on the battlefield or returning home, including various questionnaires, assessments and examinations, such as the post-deployment health assessment/reassessment (PDHA/PDHRA), military acute concussion evaluation (MACE), brief trauma brain injury screen (bTBIS), standardized assessment of concussion (SAC), traumatic brain injury (TBI)-related pituitary function screening, etc., of which compulsory screening is required for those within 50 m from the detonation. The above investigation reports have shown that more than 60% of the casualties caused by the explosion were bTBI, and 15–23% of the soldiers who participated in the war suffered TBI, while most of them were mild TBI (mTBI). It is reported that about 19.5% of soldiers participating in the war have the potential to develop TBI.
2. *Fatality and disability rate*. Statistics of the US military found that 63% of the casualties in the Iraq and Afghanistan wars from 2001 to 2007 were related to blast injuries, while 52% of the head injuries of the US Navy and Marine Corps in the Iraq war were caused by explosions. The blast-injured personnel occupied two-thirds of the transferred wounded and 88% of the wounded who needed to go to the secondary medical institution for further management. According to the report by Konlos et al., of the 22,203 soldiers participating in the Iraq/Afghanistan war, there were 2813 (12.7%) soldiers who had suffered from at least one type of mTBI, and 1476 (6.6%) who had clinical symptoms of post-traumatic stress disorder (PTSD), with a correlation in dose and response degree where repeated exposure to shock waves could induce permanent damage effects. Drake et al. have examined 7907 US Marine Corps recovery personnel and found that 9% of them had positive signs of TBI, and among the soldiers returning from Iraq/Afghanistan, those with mental disorders accounted for 19% and 11%, respectively (Hoge et al.). Besides, in the frequent terrorist attacks of homemade bombs and car bombs, the incidence of bTBI is also high. According to the study by Watson College of Brown University in the United States, it is estimated conservatively that at least 130,000 civilians died in the anti-terrorism war in Afghanistan/Iraq in the last decade, and, moreover, the civilians injured by the war and the long-term damage caused by the war cannot be counted.

2 Mechanism of Occurrence

The damage mechanics process of bTBI is extremely complicated, maybe including both wave propagation in the body directly affected by shock waves, and the response of the head to the dynamic pressure of shock wave impact, that is, the accelerated movement of the head, where the former leads to an increase in cranial pressure, and the latter generates cranial stress/strain.

1. *Primary impact effect*. Pressure waves act on the surface of the human body and are transmitted into the brain through local and systemic tissues. Meanwhile, local

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damage can also occur when intravascular pressure waves are transmitted to the brain. Tissues could be mechanically damaged due to the difference and mismatch of acoustic impedance of tissues of different densities, to which shock waves are transmitted to, that is, spalling effect. Shock waves cause a sudden change in intracranial pressure, which leads to the formation of bubbles, especially at the interface between cerebrospinal fluid and brain tissue. As a result, cavities will be formed in the brain tissue when it is penetrated, axonal channels will be destroyed, and capillaries are damaged.

2. *Secondary impact effect*. The projectile hits the head and causes a skull fracture; the pressure wave is transmitted to the brain tissue to destroy the tissue structure.
3. *Tertiary impact effect*. When the human body is thrown up and the head collides with a hard object, the wave front could generate acceleration/deceleration when it passes through the body, especially in relatively closed fortifications or buildings, where the pressure waves move reversely due to the reflected waves, leading to diffuse axonal injury (DAI), similar to the brain damage in a car accident.

3 Classification

Most patients with bTBI are milder and relatively hidden. A large number of animal experiments and clinical studies have shown that shock wave overpressure (none of the secondary, tertiary, or quaternary blast injuries caused by penetration, impact, poisoning, etc.) can cause changes in neurophysiology, pathology, biochemistry, and behaviors. Most mild head injuries (mTBI) belong to primary bTBI, which can be classified according to GCS score. Grade 0 (15 points): no coma, memory loss, headache, or vomiting after injury; Grade 1 (14 points): transient loss of consciousness (<5 min), with or without memory loss and vomiting; Grade 2 (13 points): loss of consciousness (<30 min) and neurological symptoms. The moderate to severe TBI and multiple injuries are mostly caused by the multiple effects of the four types of blast injuries.

4 Pathophysiological Characteristics

The most significant feature of bTBI is diffuse lesions described as the "pepper spray sign," including edema, intracranial hemorrhage, and vasospasm as the most common pathophysiological characteristics.

1. *Increased intracranial pressure*. Increased intracranial pressure often occurs in the acute phase of severe bTBI, accompanied by congestion and severe edema. Through the pathological examination, cerebral edema can be found as neuronal swelling, astrocyte proliferation and hypertrophy, fragments of myelin sheath, and high expression of aquaporin 4 (AQP4), which is one of the causes of the increased intracranial pressure. Such manifestations will be noticed usually when a subarachnoid hemorrhage is found on a CT scan. Basal cistern hemorrhage may predict an increase in intracranial pressure, but this situation may not appear until 2–3 weeks after the blast injury. In addition, an important injury factor for bTBI is the secondary neuronal damage caused by the destruction of the blood–brain barrier (BBB). In mice with closed brain injury, BBB destruction occurred 4 h after the injury and lasted until 30 days after the injury (brain edema had disappeared as early as 7 days after the injury), indicating that the BBB is not only sensitive to injury but also difficult to rebuild after injury, which, what's more, could cause damage to other systems.
2. *Cerebral vasospasm*. Traumatic cerebral vasospasm (TCV) caused by bTBI is considered to be the most common pathophysiological reaction. A study on cerebral angiography in patients with severe bTBI has found that 47% of patients had cerebral vasospasm, and 35% of patients had pseudoaneurysm, among whom, cerebral vasospasm can appear very early (within 48 h) after injury, with an average duration of 14 days and a maximum of 30 days. The cerebral vasospasm caused by bTBI lasted longer than that caused by closed brain injury, and the time of the latter was up to 14 days. Another study has found that cerebral vasospasm may last 10 days or longer after the injury, and the duration was related to acute traumatic subarachnoid hemorrhage. TCD ultrasonography as early as possible is helpful for intervention in time.
3. *Hemorrhage*. Cerebral hemorrhage is very common in blast injuries in animal experiments. The common injured locations involve midbrain, cerebellum, and brainstem. However, when the pig was dressed in the chest protective clothing, no symptoms of cerebral hemorrhage or abnormal blood vessels occurred with exposure to the shock wave of 131–366 kPa. It is speculated that the reason may be that the pressure waves which impact the thoracic cavity were transmitted to the cerebral blood vessels through the thoracic blood vessels.

5 Neuropathological Changes

5.1 Local Neuropathology

Shock waves can also cause damage to the neuron skeleton in multiple parts of the cortex, white matter, hippocampus, cerebellum, etc., including cell body degeneration and necrosis, as well as swelling, degeneration, and death of neurite,

especially the axonal injury, such as axonal retraction, dissolution, demyelination, etc.

1. *Cortex*. It is difficult to identify the symptoms of a simple cortical bTBI, because there is a complicated correlation between the cortex and other structures. It has been confirmed that bTBI can cause shrinkage of pyramidal cells and deformation of dendritic tips. In patients with bTBI, damage to both the cortex and white matter fiber tract presents diffuse axonal injury.
2. *White matter*. Diffuse axonal injury is very common in bTBI because bTBI could induce fibrotic degeneration, especially the conduction bundles in fronto-striatal circuits, fronto-parietal circuits, and fronto-temporal circuits. In an experiment where pigs with the chest cavity protected was exposed to shock waves of 131–538 kPa, axonal injury occurred around the cerebral ventricles, but no cerebral blood vessel was injured. In addition, for experimental primates, the exposure to shock waves of 80 kPa and 200 kPa could lead to necrosis of white matter oligodendrocytes and astrocytes under the cortex, as well as damage to the conduction pathway caused by myelin disorders.
3. *Hippocampus*. Exposure to shock waves of 80 kPa and 200 kPa could result in twist of the dendritic tips of pyramidal neurons, contraction of somatic cell in the CA3 area, and a significant decrease in the CA1 area, which may be due to the low oxygen content and local ischemia of pyramidal neurons. Diffusion tensor imaging (DTI) proved that patients with bTBI suffered from diffuse axonal injury (DAI) in the hook bundles and white matter nerve bundles.
4. *Cerebellum*. Diffusion tensor imaging (DTI) could find damage to the cerebellar fiber bundles in patients with bTBI. Besides, experiments have also showed that when primates were exposed to shock waves, the Purkinje neurons of the cerebellum would change, manifested as irregular nuclear shrinkage, as well as the dendrites from Purkinje neurons which degenerated, resulting in the change in the movement coordination.

The studies above showed that traumatic cerebral vasospasm and diffuse axonal injury are the basis for symptoms of patients with bTBI, such as memory disorders.

Diffusion weighted imaging (DWI) can be used to diagnose the damage of white matter nerve bundles, which is difficult to diagnose through MRI and CT. In the strictly confined white matter nerve bundles, the diffusion is limited and directional, with a low diffusion coefficient and a high anisotropy score. In the acute white matter injury, the cells swell, increasing the diffusion coefficient, and decreasing the anisotropy score, while in the chronic injury, cells lyse or die, resulting in a high diffusion coefficient and a low anisotropy score.

A variety of diffusion tensor imaging methods have been applied to diagnosis and assessment of bTBI. In patients with mild bTBI, through DTI, it can be found that the anisotropy scores in the injured area and white matter declined, while traditional techniques cannot identify such damage. When patients are exposed to multiple shock waves, DTI will show more anisotropy scores. The application of DTI and electroencephalography (EEG) can better distinguish the bTBI.

5.2 Cell Neuropathology

Previous experiments have shown that after bTBI, it could be noted not only the decrease of neurons (apoptosis or necrosis) but also the apoptosis of microglia, astrocytes, and oligodendrocytes.

1. *Astrocytes*. In the experiment, when the cortical area of a primate was exposed to shock waves, astrocytes proliferated, and the astrocytes in the ipsilateral hippocampus were activated. In another experiment, astrocytes in pigs also increased. There was a close relationship noted between the reactive astrocyte proliferation in the ischemic marginal zone and hippocampus after bTBI and the reduction of microcirculation perfusion.
2. *Neurons*. Axon microtubules reassemble and arrange after bTBI when receiving shock waves, leading to partial or complete destruction of the axon transmission system. Axonal fluctuations may indicate diffuse axonal injury. Beta amyloid preprotein (β-APP) is produced in neurons and transported rapidly by axons. If the axon is injured, β-APP will accumulate at the injured site, and axonal spheroids can be formed within 2 h after bTBI. According to the report by De Lanerolle et al., the accumulation of β-APP axons could be seen around the cerebral ventricles in pigs with bTBI.
3. *Oligodendrocytes*. When primates were exposed to shock waves, their oligodendrocytes in the subcortical white matter died, and the myelin produced by the cells decreased, leading to weakened nerve conduction.

5.3 Changes in Molecular Biochemistry

There is an increase in inflammation-related molecules, such as glial fibrillary acidic protein (GFAP), S100B (GFAP and S100B are both specific markers of astrocyte activation and related to early inflammation and glial scar formation), myelin-related protein 8 (MRP8, expressed by activated macrophages and microglia), osteopontin (a mucin chemotactic molecule that recruits microglia, macrophages, and astrocytes after ischemia), chemokines (recruiting mac-

rophages), and inflammatory factors, such as IL-1B, IL-6, TNF-α, etc., in the early stage of inflammation after bTBI, and some even last for 1 month.

Neurofilament heavy chain (NfH) and Pavlov's enterokinase (PE) may be biomarkers of bTBI. NfH increased with time and reached its peak 6 h after exposure to shock waves in experimental pigs. Pavlovian enterokinase acts on the 476 and 986 sites of NfH. The products from the NfH division can be quantified by cortical microdialysis. In traumatic subarachnoid hemorrhage, there are a lot of neurofilament heavy chains. The phagocytic Mato cells increase after the bTBI, leading to the metabolic changes of the brain. Meanwhile, bTBI could also suppress glucose metabolism, which can cause Alzheimer's disease or disorders.

6 Clinical Symptoms and Signs

Mild bTBI can lead to headache, amnesia, confusion, inattention, short-term memory loss, mood changes, sleep disorders, vertigo, and anxiety. Generally speaking, these symptoms may appear immediately after the injury, but be relieved after a few hours or days. But some patients with bTBI patients may not realize their blast-induced brain injury. Patients may gain awareness of the head injury after experiencing a second or more bTBI. Initial impact syndrome: Syndromes persist after concussion, such as headache, vertigo, short-term memory loss, and inability to concentrate or perform multiple tasks. Second impact syndrome: It concludes rapid loss of consciousness, malignant brain edema, increased intracranial pressure, and coma. Up to 50% of the mortality rate is related to the second impact syndrome.

For the wounded, the post-injury phobia may last for several days. The foremost symptoms of the injury may persist after the phobia, which usually respond well to placebos and specific drugs such as nonsteroidal analgesics, antimigraine drugs, and antidepressants. Since the early symptoms of most bTBI casualties are mild or covered by other head or face injuries so that it cannot attract enough attention from the casualties and practitioners, which could result in gradual exacerbation of the patient's conditions due to the lack of appropriate management in the early stage. Therefore, attention is still needed to the damage and secondary symptoms of the following organs after initial treatment.

1. *Vision*. Visual impairment is the most common cause of secondary damage after blast injury, where closed ocular trauma is more common than open ocular trauma. Serious nonpenetrating injuries include choroidal rupture, optic nerve injury, anterior chamber hyperemia, retinal detachment, and traumatic lens and vitreous hemorrhage. From August 2004 to October 2006, the Defense and Veterans Brain Injury Center (DVBIC) found that 66% of patients with traumatic brain injury in the Walter Reed Army Medical Center (WRAMC) also suffered from ocular trauma. CT imaging is an main method important to diagnose ocular trauma.
2. *Hearing*. The ear is a complex organ that can feel pressure waves and movement as well as make a collective response to gravity. Hearing loss is the most common damage caused by noise of explosives. A retrospective analysis of 10,431 veterans who had suffered from bTBI in *Operation Iraqi Freedom (OIF)/Operation Enduring Freedom in Afghanistan (OEF)* showed that 68.5% of them had hearing impair. Symptoms such as swollen ear, earache, tinnitus, abnormal hearing, sensory, vertigo, hearing loss, etc. that appear rapidly after bTBI are mainly caused by overpressure that impacts the tympanic membrane, leading to a significant pressure difference between the middle ear tympanum and the external auditory canal, resulting in tympanic membrane rupture, tympanic hemorrhage, broken ossicular chain, etc. Meanwhile, there may also be hemorrhage and cochlear structure disorders in the inner ear, but they are not common.

   Conductive hearing loss (CHL) is caused by impeded sound conduction or damage to the structure of the middle and outer ears, while sensorineural hearing loss (SNHL) could be attributed to the injury of the cochlea, auditory nerves, or auditory cortex, of which the common symptom is tinnitus. Previous studies showed that sensorineural hearing loss and CHL & SNHL hearing are more common in blast injury, while CHL is more common in non-blast injury. A retrospective analysis of hearing injury showed that 50% of SNHL is caused by blast injury. A small-scale cohort study of patients with bTBI has showed that 62% of them had suffered from impaired hearing, where more than half of SNHL patients had suffered from hearing loss.

   Through MRI or PET-CT, the changes can be detected in the structure of the auditory brainstem and the central auditory regions.
3. *Vestibule*. Blast injury may impact the vestibular function, including benign paroxysmal positional vertigo (BPPV), central or peripheral regulation disorders, vertigo, or post-traumatic Meniere's syndrome. BPPV is the most common balance disorder of bTBI. Scherer and Schubert have reported that bTBI can cause vestibular damage, resulting in imbalance and dizziness. In a cohort of 3973 soldiers returning from OIF, there were 797 cases of bTBI, with the main symptom of headache, where half of them were accompanied by dizziness and vertigo, and one quarter had suffered from balance disorders. Examinations of 24 bTBI patients have found that the incidence of vertigo caused by peripheral ves-

tibular dysfunction was high. Forty-two percent of OIF veterans suffered from vestibular center injury, manifested as nystagmus and/or eye movement dysfunction. Vestibular symptoms might last several weeks or months.

4. *Epilepsy*. It is not clear about bTBI-induced seizures yet. There are various forms of epilepsy. Post-traumatic epilepsy (PTE) is defined as two or more seizures with no apparent cause after brain trauma, which usually appear within 5 years after a traumatic brain injury. According to a PTE research on soldiers who participated in the Vietnam War, 53% of veterans with bTBI had suffered from PTE, which is of great significance in predicting the risk of PTE for those with brain injury. In current practice, diagnosis of PTE still requires video electroencephalogram (EEG) and MRI.

   In summary, bTBI has the following characteristics:

   (a) In most cases, bTBI is a part of the systemic blast injury caused by primary shock waves, with only overstimulation signs, such as headache, impaired memory and concentration, vertigo, anxiety, depression, insomnia, fatigue, irritability, and sensitivity to sound and light, which may not be accompanied with any significant manifestations of body surface or brain parenchymal injury. It is easy to be misdiagnosed without comprehensive examination and carefully observation.

   (b) In a part of severe bTBI, extensive cerebral edema and hyperemia are common and develop rapidly. Those with subarachnoid hemorrhage predict more severe cerebral hyperemia and edema, as well as delayed cerebral vasospasm.

   (c) The vital signs change early and rapidly. Animal bTBI experiments have shown that typical pathophysiological changes occurred immediately after bTBI, including apnea, heart rate reduction, and decrease or loss of mean arterial pressure and brain electrical activity.

   (d) The type and condition of the bTBI are complicated, involving the direct impact of shock waves on the body, throwing objects hitting the body, throwing body hitting an object, or burns, or inhalation of toxic smoke.

Therefore, patients with bTBI may suffer from injury not only to brain and hollow organs (e.g., gastrointestinal tract) directly caused by shock wave overpressure, but also to solid organs (such as liver, spleen, kidney) caused by throwing and displacement under dynamic pressure. Although sometimes bTBI is mild, the combined multi-site injury can aggravate the brain damage, which may be missed without careful screening, leading to serious consequences.

7 Auxiliary Examination

1. *MRI of head*. It can show a small amount of bleeding in the corpus callosum or the cerebral cortex, superior to CT in the diagnosis of diffuse axonal injury. T2-weighted and MRI fluid attenuated inversion recovery (FLAIR) imaging is helpful for the diagnosis of nonhemorrhagic brain injury, susceptibility weighted imaging (SWI) is more significant in detecting microhemorrhages, diffusion tensor imaging (DTI) can quantitatively analyze the range and severity of the brain white matter damage, and functional magnetic resonance imaging (fMRI) can record the recovery process of the brain.
2. *CT of head*. It is mainly used to determine the extent and depth of the fracture and the relationship with the dura and brain parenchyma; meanwhile, it can clarify the severity of brain contusion, and the location and size of the hematoma. CT angiography can also be used to diagnose cerebrovascular injury.
3. *Evoked potential test*. It can provide a basis for objectively evaluating the functional status of the nervous system of the wounded with bTBI without any positive signs in the nervous system examination and CT scans.
4. *Electroencephalogram (EEG)*. EEG could not be affected by coma, sedatives and muscle relaxants, of great importance in determining the prognosis of the injured.
5. *Lumbar puncture and intracranial pressure monitoring*. Lumbar puncture is mainly used to measure intracranial pressure and can also be used to diagnose and treat subarachnoid hemorrhage and intracranial infection. Through objective data of intracranial pressure, practitioners can discover intracranial hematoma and cerebral edema early as a treatment guide.
6. *Laboratory examination*. Monitoring arterial blood gas, electrolytes, related biochemical indicators (S-100B, lactic acid, free radicals, excitatory amino acids, creatinine, blood transaminase) in cerebrospinal fluid (CSF) and plasma, etc., is of a certain significance in assessing the post-traumatic brain function and general conditions.

8 Clinical Diagnosis

Since a majority of the wounded with bTBI have mild brain injury or concussion with multiple symptoms but few or no signs, neuroelectrophysiological examinations and psychological evaluations are required when possible, in addition to nervous system examinations. Therefore, clinicians need to formulate corresponding criteria based on the patient's symptoms and signs in order to better understand the severity of the brain injury. According to the American Congress of Rehabilitation Medicine (ACRM) of the Mild Traumatic Brain Injury Committee (1993), bTBI is defined as the

cause of consciousness loss, memory loss before or after injury, psychological changes during injury, and with or without focal neurological deficits. A few bTBIs, which are severe, require necessary neuroimaging according to the conditions, such as head CT or MRI, and general examinations, including monitoring blood pressure, pulse, breathing, consciousness, position and posture, etc., to determine whether there are life-threatening conditions like suffocation and shock, which can prevent neglecting significant information and improve the diagnosis efficiency. The key points for diagnosis of bTBI of different severity are as follows:

1. Mild bTBI: It is defined as consciousness loss less than 30 min, post-traumatic memory loss less than 24 h, GCS: 13–15. The principal manifestations are different levels of advanced neurological dysfunction, including deficits in attention, perception, memory, expression, or execution, with different degrees of damage in cognition, language, or logic function found in the special examination of the nervous system, and persistent symptoms similar to post-traumatic brain injury or post-concussion syndrome, involving headache, nausea, vomiting, vertigo, blurred vision, sleep disorders, irritability, depression, fear, anxiety, emotional instability, etc. These symptoms usually improve within a few hours or days after the injury, but the post-traumatic fear may last for several days.
2. Moderate bTBI: It is defined as consciousness loss >30 min but <24 h, persistent memory loss >30 min but <1 day, GCS: 9–12, with skull fracture, possibly. Patients with moderate bTBI usually suffer from a long-term consciousness loss and/or neurological dysfunction, as well as possible symptoms such as nightmares, insomnia, over alertness, and susceptibility to fright for a long time.
3. Severe bTBI: It is defined as consciousness loss >24 h, memory loss >1 day, GCS: 3–8, which may be accompanied by brain contusion, laceration, or intracranial hemorrhage. Its main clinical presentations are headache, vomiting, gradual increase in blood pressure, slow and shallow breathing, slow and strong pulse and other symptoms and signs of acute intracranial hypertension. Signs like pathological breathing, fast and weak pulse, and decreased blood pressure may indicate brain stem failure. When manifestations of traumatic shock such as pale face, fast and weak pulse, undetectable blood pressure, irritability occur, combination with injury of other organs needs to be considered. Meanwhile, local localization symptoms may also occur (according to different parts of the brain parenchyma, paralysis, motor and sensory dysfunction, aphasia, visual field defects, and other presentations may appear).

9 Differential Diagnosis

Post-traumatic stress disorder (PTSD) is a group of clinical syndromes, which refers to a disease caused by a patient's deep fear or helplessness after experiencing a life-threatening or health-threatening event, with symptoms including attention deficit, sleep disturbance, explosive anger, irritability, over alertness, and being easily startled. Although many similar clinical manifestations could also occur in mild bTBI, PTSD needs to be diagnosed based on six relevant diagnostic criteria, and supplemented by standardized test tools (structured diagnostic review, including structured clinical interview for DSM (SCID), clinician-administered PTSD scale (CAPS)), and self-assessment questionnaire (life event scale (IES), the PTSD checklist-civilian version (PCL-C), post-traumatic stress disorder self-rating scale (PTSD-SS), or post-traumatic stress disorder social support scale (PTSD-SSS)). These two diseases are easy to be misdiagnosed in clinical practice and must be distinguished carefully.

10 Treatment Principles

According to the characteristics of bTBI, the treatment schedule should be made immediately based on rapid diagnosis and injury assessment, with the principle of giving priority to solving the main contradictions, to ensure a rapid and orderly treatment for patients with traumatic brain injury.

1. *Treatment Principles of bTBI*. Generally, most manifestations of bTBI are occult, with mild external damage and severe internal damage. Practitioners should calculate the GCS score of the wounded during the injury examination and treat the patients corresponding to the severity and evolution of the injury.
   (a) For patients with mild bTBI and a GCS score of 15–13, according to the post-traumatic clinical symptoms, they can be divided into the followings: Grade 0 (15 points): no coma, memory loss, headache, or vomiting after injury; Grade 1 (14 points): transient loss of consciousness (<5 min), with or without memory loss and vomiting; Grade 2 (13 points): loss of consciousness (<5 min) with neurological symptoms. Specific treatment principles: (a) Patients of Grade 0 only with symptoms of scalp contusion, local headache, and dizziness can be observed for 6 h. If there are no neurological symptoms, the patient can rest in bed or take non-steroidal analgesics, anti-migraine drugs, and anti-depressants. There is no need for radiological examinations and other special treatments. (b) For patients of Grade 1, head X-ray or CT

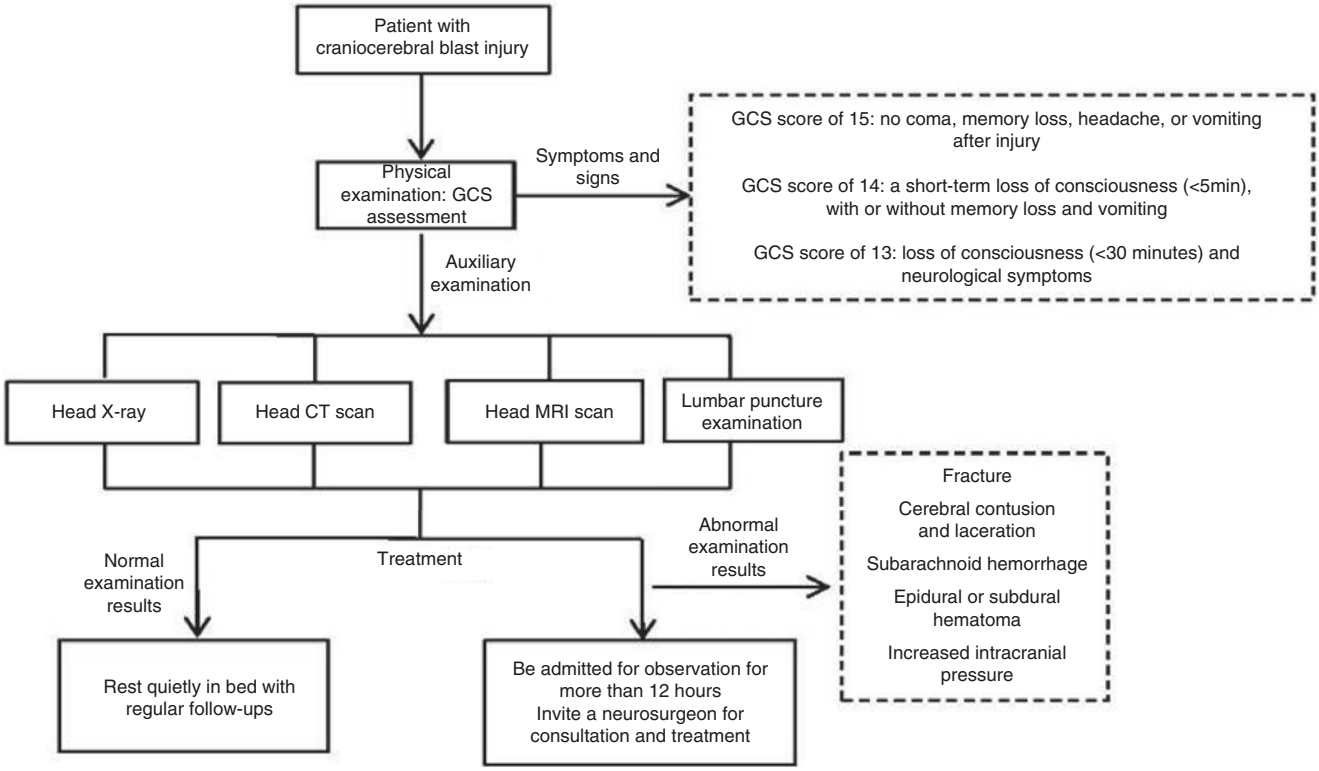

Fig. 1 Diagnosis and treatment process

scan can be chosen, and early head CT can find hematoma or rule out delayed hematoma. If symptoms occur, the patient should be observed for at least 24 h, rest in bed, keep the airway unobstructed, and take oxygen, while CT should be performed dynamically according to the patient's consciousness. (c) Patients of Grade 2 may develop intracranial hematoma so that close observation is needed. Practitioners should pay attention to screening out those who may develop intracranial hematoma and transfer them to the neurosurgery department for surgical management in time.

(b) The critically wounded should be treated orderly with keeping the airway unobstructed, supplying adequate cerebral perfusion, supplying oxygen, controlling intracranial pressure, and managing hypovolemic shock. Early decompressive craniectomy treatment should be implemented for those with severe closed brain trauma and penetrating wounds, to ensure the later treatment of critically injured patients.

2. *Treatment Principles for Penetrating Injury*. (a) Operation should be performed immediately if there is active bleeding from the brain wound. (b) The bone window should be expanded through the entrance wound, and then the hematoma should be removed near the injury channel for patients with hematoma and one or two dilated pupils. If the hematoma is far from the entrance wound, or located in the contralateral hemisphere, a separate craniotomy should be removed near the hematoma. (c) Perform operation as soon as possible for those with a large amount of cerebrospinal fluid discharged from the brain wound. (d) For patients with penetrating brain traumas, those with a longer injury time should be operated first. (e) Patients with penetrating brain injury should undergo surgery prior to those with nonpenetrating brain injury.
3. *Diagnosis and Treatment Process* (Fig. 1).

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Ocular Blast Injury

Nian Tan and Jian Ye

1 Overview

A variety of injuries to human eyes from the energy unleashed by blast waves are called ocular blast injuries (OBI). OBIs are common during warfare and in peacetime. According to the study of 199 globe penetrating wounds in the military operation in the southwest border area of China, the incidence of retinal blast injuries is 21.6%, while OBIs at ordinary times are more frequently seen in explosive accidents, and the incidence is up to 30–50% among people injured by setting off fireworks and the blast of small quantities of high explosives. The eye can be injured either from the effect of blast waves or due to foreign bodies (FB) thrown up by explosions, such as dirt, grit, stone, or fragments of explosive.

The OBI can cause extensive damages to a myriad of tissues in the eye: intraorbital soft tissue hematomas and emphysema, as well as orbital wall fractures; incarceration and rupture of extraocular muscles; hyperemia, edema, hemorrhage, and tearing of the eyelid and conjunctiva; corneal opacity, laceration, epithelial defect, ulceration, and even perforation; aqueous humor muddiness and hemorrhage; iridodialysis and hemorrhage; dislocation, opacity, and rupture of the lens; choroidal vasodilatation, stasis of blood cells and, in severe cases, choroidal rupture hemorrhage; retinal edema, exudation, hemorrhage, and more serious, retinal detachment, occasionally with air embolism in the retinal vessel; papilledema is seen in the optic nerve, or worse still, optic nerve breakage; in severe cases, eyeball rupture or proptosis.

The effect of blast waves on the eyeball is mechanical injury from dynamic, excess, and negative pressure, which is further transmitted to the interior of the globe where the blast wave–caused damage is often slighter on the outside than the inside. OBIs can be divided into closed ocular blast injuries (COBI) and open ocular blast injuries (OOBI) depending on the presence or absence of a full-thickness wound in the wall of the eyeball. OOBI can be subdivided into: globe penetration or perforation, intraocular foreign body (IOFD) injury, and eyeball rupture. OBIs are often caused by explosions, which, apart from the effect of shock waves, are concurrent with chemical injuries, burns, and radiation injuries according to the nature of the blasting materials acting on the eye.

1.1 Essentials of Diagnosis

1.1.1 History Taking

The diagnosis of an OBI is based primarily on the history of injury and clinical signs and symptoms. Comprehensive and accurate information is helpful in guiding physicians to do a rapid examination, directing initial management, and assessing prognosis. History taking should include the following:

1. *Time of injury*. The time of injury can help determine the likely progression of the injury. Endophthalmitis occurs 24–72 h after injury, and sympathetic ophthalmitis over 4–8 weeks of injury. For open ocular trauma, the longer the time of injury, the higher the probability of infection. So, earlier debridement and suturing are required. Adults with orbital fractures can undergo surgery within half a month to 2 months of injury; in terms of children whose orbits especially orbit floors are broken, the earlier the repair is performed, usually within 5–7 days of injury, the better the effect.
2. *Cause of injury and nature of injury-causing materials*. Understanding the cause of injury and the nature of injury-causing materials is an important reference for early management and assessing the state of the injury. Blast injuries are often associated with intraorbital and intraocular foreign bodies, or insidious posterior segmental and penetrating injuries; copper- and iron-bearing intraocular foreign bodies (IOFBs) can induce ocular chalcosis (chalcosis bulbi) and ocular siderosis (siderosis bulbi) that can seriously affect visual function; while

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IOFBs such as glass and plastics usually do not need to be removed and just require close observation. If these are associated with acid and alkali burns, it will lead to an immediate biological effect with the tissue, with protein coagulation necrosis and precipitation occurring after acid burns, often complicated by thermal burns; after alkali burns, alkaline substances are saponified with the lipids in the tissue cells to form a compound that is both lipolytic and hydrosoluble, enabling them to quickly penetrate the epithelial tissue and corneal stroma, and further inside.

1.1.2 Eye Examination

The eye examination includes a visual function test, external ocular examination, and eyeball examination.

1. *Visual Function Test*
   (a) Central visual acuity. Vision is the basic indicator of eye examination, and the severity and prognosis of ocular trauma can be indirectly determined by the loss of vision. The visual function is roughly evaluated when the injured cannot be examined with an eye chart, such as recording indexes, hand movement, and light perception. Regarding highly edematous eyelids where the palpebral fissure cannot be separated, visual acuity can be examined after the swelling of the eyelids has subsided.
   (b) Pupillary light reflex (PLR). This includes both direct pupillary reflex (DPR) and consensual pupillary reflex (CPR). Pupillary response to light is an objective reflection of the function of the optic pathway in the anterior segment. Relative afferent pupillary defect (RAPD) can occur when the optic pathway is traumatized, which is characterized by a narrowing of both pupils when a torch is shined on the healthy eye; the narrowing of the pupil in the affected eye is caused by the pupil's indirect response to light. Because of RAPD, both pupils are not narrowed or slightly constricted via the movement of the light flashed into the affected eye. When the torch is alternately shined on both eyes at the interval of 1 s, the pupil of the unaffected eye is narrowed, the other dilated.
   (c) Examination of the visual field (perimetry). It can reveal damages to the optic nerve, retina, and brain. For these with severe ocular trauma or injuries to other organs, a cursory examination of the peripheral visual field can be performed by shielding one eye of the patient in front of the examiner while asking the examined to look straight ahead and watching the examiner's finger moving from the periphery to the center via four quadrants from the corner of the blocked eye. The comparison of finger movement ranges seen by both the patient and the examiner helps make a preliminary judgment of whether the patient's visual field is affected.
2. *External Ocular Examination*. Examination of the eyelids and surrounding soft tissues includes the skin color and temperature of the eyelid, the edema and ecchymosis of the eyelid and periorbital tissues, and the presence of tissue tears, foreign bodies, ptosis, or ectropion.

   In the case of orbital fractures, conjunctival emphysema or periorbital skin crepitus may result from gas entering the conjunctiva or skin; in the case of significant enophthalmos, the diagnosis of orbital wall fracture is basically established; ocular motility disorders are possibly due to the edema of extraocular muscles or contusions, with the incarceration of extraocular muscles caused by orbital wall fractures being a more common cause.
3. *Eyeball Examination*
   (a) Conjunctiva. The conjunctiva is examined under good light and a slit lamp microscope for hyperemia, edema, hemorrhage, laceration, and foreign bodies. In terms of conjunctival laceration, the area beneath the conjunctiva is further explored with a cotton swab after surface anesthesia to see whether there are foreign bodies and scleral injuries. Conjunctival hemorrhage is usually fresh and localized. Extensive bulbar subconjunctival congestion in a dark red color with associated hypotonia suggests a possible scleral rupture. It is therefore important to avoid squeezing the eyeball during the examination. There is a likelihood of endophthalmitis in the presence of photophobia, lacrimation, and a highly congested, edematous bulbar conjunctiva with globe penetration.
   (b) Cornea: Cornea examination is in principle performed under a slit lamp microscope from epithelium to stroma, including corneal surface abrasion, epithelial peeling, deficit, edema, ulcer, laceration, and tissue impaction, foreign bodies, etc. Regarding deeper foreign bodies, their depth and full-thickness impaction in the cornea need to be determined under narrow slit light to decide whether the removal of these foreign bodies will result in corneal perforation, and where it is done, in the consultation room or operating room. Drops of 1–2% sterile sodium fluorescein solution onto the corneal surface can be applied for the following examinations under the cobalt blue light from the slit lamp microscope: (a) corneal abrasion. It can paint the tissue without corneal epithelium for easy observation under the slit lamp; (b) when it is not possible to determine whether a corneal laceration is a penetrating wound, the eyeball can be pressed gently through the lower eyelid to see whether there is bright stream-like, thread-shaped

liquid flowing from the wound namely aqueous humor leakage (Seidel test), whereby a full-thickness laceration can be identified; (c) the absence of aqueous humor leakage suggests a closed corneal wound.

(c) Sclera: Injuries to the sclera are often easily overlooked as it is covered by the conjunctiva. It is usually difficult to accurately locate the wound tract due to the small size and fast velocity of minor foreign bodies. However, when there is significant localized conjunctival hemorrhage and a history of penetrating trauma, surgical exploration of the sclera needs to be considered. In case of extensive, heavy subconjunctival hemorrhage after a blast injury, associated with hypotonia, or even worse, globe deformation, occult scleral rupture is highly suspected, which can be definitely diagnosed through CT scan.

(d) Anterior chamber: The anterior chamber is examined under a slit lamp microscope for binocular comparison, including the depth of the anterior chamber, the opacity of the aqueous humor, the exudation, hemorrhage of the anterior chamber, abscess and foreign bodies in the vitreous body, etc.

Deepening of the anterior chamber: the anterior chamber is deepened by the loss of support due to blast or penetrating injuries resulting in downward or backward subluxation of the lens, iridodialysis, and scleral rupture. Shallowing of the anterior chamber: the anterior chamber may become shallower due to the anterior subluxation of the lens, vitreous loss, aqueous humor outflow caused by corneoscleral laceration, severe choroidal detachment, etc.

(e) Iris and anterior chamber angle: Iris damage such as pupillary margining avulsion, iridodialysis, iris laceration, and iridodonesis caused by lens subluxation is examined using direct irradiation or retroillumination. Iridodialysis presents as a fissure or semilunar deficit in the iris at the corneal limbus through which the lens behind the iris can be seen, with the D-shaped pupil being obscured by the iris curled up due to severe iridodialysis. A torn pupil margin is manifested by moderate corectasis, serrated defects, or notches. Iridodonesis occurs due to the loss of support from the lens that is half dislocated under the effect of trauma.

The presence or absence of an iris perforation has a diagnosis value in assessing foreign body injuries. Iris perforations can be examined by direct light from a slit lamp, and small iris perforations with bright red light can be detected through the light reflected by the posterior pole after passing through the pupil. Approximately 80% of IFOBs enter the eye via the cornea. According to the sizes, locations, and formation angles of corneal and iris perforations, the approximate location of the foreign body in the eye and the degree of damage it may have caused to the eye can be initially determined.

Closed globe injuries can be detected through the gonioscopy of the anterior chamber angle (ACA). The anatomical structure of the eye makes the anterior chamber angle vulnerable to injuries such as recession and dissection from the blast injury of the eyeball. Gonioscopy can also detect the foreign bodies retained in the anterior chamber angle.

Angle recession: the moment the shock wave acts on the eye, the pupil is blocked, and the aqueous humor impinges on the root of the iris, squeezing the ACA tissue whereby the fibers of the circular and radial muscles are separated from the fibers of the longitudinal muscles, thus displacing the iris and medial ciliary body backwards to deepen and widen the ACA. The process is therefore called angle regression.

Angle dissection: the eyeball is subjected to an external force that causes a gap between the ciliary body and the scleral crest attached. Because of the gap, the white sclera is exposed, and completely separated from the ciliary body, resulting in an open suprachoroidal space (SCS).

(f) (Crystalline) lens: The examination of the lens focuses on the presence or absence of lens subluxation, rupture of the anterior and posterior lens capsules, traumatic clouding, edema, and cortical overflow of the lens, and the presence or absence of foreign bodies within the lens. Severe trauma may cause the lens to fall into the vitreous chamber (aka vitreous cavity or vitreous space), or detach completely from the eye, or enter the subconjunctival via the ruptured limbus or scleral break. The fracture of the lens capsules allows the lens cortex to spill out to cause phaco-anaphylactic uveitis, the clinical signs of which can easily be confused with suppurative endophthalmitis.

(g) Vitreous humor (also vitreous body), retina, and choroid: Examination of the vitreous humor mainly includes the clouding, hemorrhage, and organization of the vitreous humor. As trauma often results in corneal opacity, traumatic cataract, and vitreous hemorrhage that cannot be detected by direct ophthalmoscopy, a binocular indirect ophthalmoscope with a stronger light source and better stereoscopic effect to fully dilate the pupil is an indispensable instrument for the examination of the posterior segment of ocular trauma after excluding craniocerebral injury-induced neurological disorders. The indirect ophthalmoscope allows visualization of the vitreous hemorrhage or the location of the wound tract.

The examination of the retina and choroid includes commotio retinae, choroidal rupture, the position of foreign body in relation to the retina, macular hole and retinal tear, retinal and choroidal detachment, subretinal or subchoroidal hemorrhage, and proliferative vitreoretinopathy (PVR), among others. The examination of the retina should be thorough and careful. When a retinal tear is found, attention should be paid to whether there are multiple breaks in other areas of the retina, and whether it is associated with macular holes and dialysis of the ora serrata. Commotio retinae are closed ocular injuries featuring off-white edema in the posterior pole of the retina, possibly accompanied by a small amount of retinal hemorrhage, commonly due to blunt trauma. A choroidal rupture, on the other hand, often occurs in the optic disc, macular area, or the posterior pole, arc-shaped, with banded subretinal hemorrhage, accompanied by retinal edema; after the blood is absorbed, a well-defined white blackspot is left, and surrounded by hyperpigmentation. There is generally hemorrhage and vitreous traction around the retinal break with a well-defined margin, beneath which is the red choroid or blood. When the retina is detached, it is partially uplifted, grayish white, with lots of floaters. Retinal detachment from ocular trauma is accompanied by retinal tears and hemorrhage. For retinal detachment following trauma in highly myopic patients, macular holes and the dialysis of ora serrata are more common, in addition to retinal tears from penetrating injuries. Retinal impaction is a common sign of ocular injury that forms at the site of scleral injury or where the foreign body is inserted in the sclera. The retina concentrates toward the wound tract or foreign body impaction to produce large, fixed retinal folds; the foreign body and wound tract can be wrapped and covered by proliferated and organized tissues, often with local hemorrhage.

Intraocular pressure can be significantly lowered due to rupture of the eyeball, and the detachment of the retina and ciliary body, or the choroid is separated due to hemorrhage in the suprachoroidal space. A limited choroidal detachment presents as tiny longitudinal choroidal folds, while a more extensive choroidal detachment presents as a solid, dark red heave in an unchanged form, most often around the equator, which may not be associated with retinal detachment. The choroidal separation below the area of retinal detachment is a brownish-yellow bulge with a blurred surface resulting from retinal detachment and subretinal fluid (SRF), sometimes obscured by turbid vitreous humor or hemorrhage, and thus difficult to detect, unless otherwise by B-mode ultrasound.

(h) Optic nerve: Optic nerve injury is the third most common form of indirect brain injury, including direct and indirect injuries to the optic nerve, with traumatic effects mostly due to the fracture of the orbit and optic canal. The acting point of the injury is primarily located on the part where the forehead is close to the temporal side. The edema, hemorrhage, and color of the optic disc can be examined using a direct ophthalmoscope and three-mirror contact lens, with a sudden loss of vision to an exponent or even no light perception being the main clinical symptom. CT scan can reveal fractures of the optic canal, the relationship between the fracture and the optic nerve, and whether the fragment is compressing the optic nerve. In the early stages of indirect optic nerve injury, the fundus may be without abnormal changes, and optic canal fractures may not occur.

(i) Intraocular pressure (IOP): IOP examination has a great diagnostic value for patients with eye injuries. Significantly lowered IOP suggests a penetrating globe wound, or an occult scleral rupture. There may also be retinal, choroidal, or ciliary body detachment, which increases aqueous humor overflow and reduces IOP. Injury-induced ciliary epithelial detachment slows down aqueous humor secretion, leading to reduced IOP. The hyphema and the inflammatory response of the anterior chamber can cause an increase in IOP. Both the formation of the pupillary exudative membrane and ciliary body edema leading to the antedisplacement of the lens–iris diaphragm, the forward subluxation of the lens, or the incarceration of the vitreous body into the anterior chamber can block the pupil, impede the flow of aqueous humor, and raise the IOP; injuries may also cause mechanical damage to the anterior chamber angle (ACA) or angle recession. As a result, the channels through which aqueous humor flows are dwindled in number, leading to prolonged high IOP. Alternatively, massive hemorrhage in the vitreous body causes ghost cell glaucoma or hemolytic glaucoma.

All patients with ocular injury should have an IOP check in the absence of a definite rupture or penetrating injury to the globe, either by a pneumatic non-contact tonometer, Schiotz tonometer, or Applanation tonometer. If the patient has difficulty walking or is uncooperative, digital tonometry can be adopted. Notably, a low IOP indicates a penetrated or ruptured eyeball, but an increased IOP does not completely rule it out.

1.1.3 Diagnostic Imaging

Routine techniques such as X-ray, CT, MRI, and ultrasound are important in the diagnosis and differential diagnosis of ocular blast injuries and their complications.

1. *X-ray examination*. X-rays are the conventional technique of viewing the foreign bodies in the eye that show a complete picture of the orbit and the quantity of foreign bodies inside it, as well as the holistic morphology of high-density foreign bodies in the orbit or the eye. However, the density resolution of X-rays is poor and does not show radioparent foreign bodies and small roentgen opaque foreign bodies. Therefore, a negative plain radiograph does not exclude ocular foreign bodies.
2. *CT examination*. CT scan is more commonly used to examine orbital and intraocular foreign bodies, traumatic retrobulbar hematomas, and orbital fractures to name a few. CT examinations provide a clear and accurate picture of intraorbital and intraocular foreign bodies and their relation to the globes, extraocular muscles, and optic nerves, among other structures. With a higher density (contrast) resolution, it can also detect some materials that are less visible on X-rays, such as alloys, glass, and plastics. CT scan is the most accurate of all imaging examinations in showing the place, extent, and degree of orbital fractures and in locating the site of bleeding; has a high tissue resolution, and can fully visualize the structures within the eyeball while performing cross-sectional and coronal scans. The following are the signs of globe rupture on CT scan: (a) discontinuity of the limbal ring, which may be accompanied by local irregular thickening; (b) deepening of the anterior chamber, which is a vital sign of posterior scleral rupture; (c) globe rupture, often accompanied by trauma to the structures around the eyeball, such as orbital wall fracture, emphysema, and hemorrhage.
3. *MRI examination*. The MRI in the diagnosis of ocular injuries is more commonly used for the examination of traumatic orbital hematoma, traumatic vitreous hematoma, globe ruptures, intraorbital and intraocular nonmagnetic foreign bodies, etc. Nonmagnetic foreign body MRI is much better than CT scan in terms of display effect; the signal missing area formed by IOFBs is close to the long T1 signal of the vitreous body and aqueous humor, whereas the increased T2WI signals of the vitreous body and aqueous humor are in sharp contrast to the signal voids of foreign bodies, forming a signal-free black hole. Intraorbital fat is a high signal while orbital foreign bodies (OFB) are low signals or devoid of signals, which can be easily detected on T1WI and T2WI.
4. *Ultrasonography*. Ultrasound is of high value in the diagnosis of eyeball contusions, and orbital soft tissue contusions, also useful in the diagnosis of globe ruptures and IOFBs. Type-B ultrasound is mainly used for the detection of tissues behind the posterior lens capsule, such as the vitreous body, retina, extraocular muscles, optic nerve, and retrobulbar and orbital lesions. Ultrasound biomicroscopy (UBM) is also a type-B ultrasound scan, the transducer of which has a high frequency. Despite high resolution, it has a weak penetrating force and can only detect soft tissue structures at a depth of 4–5 mm. This technique is appropriate for the diagnosis of the lesions in the anterior segment of the eye, such as ciliary body detachment, and tiny foreign bodies in the anterior segment.

1.1.4 Special Examination of the Eye

1. *Fundus fluorescein angiography (FFA)*. The basic principle is to inject sodium fluorescein rapidly into the vein of the patient until it circulates to the blood vessel of the fundus, where it is excited by blue light to generate yellowish green fluorescence to observe and photograph. The dynamic process of fundus blood circulation is observed and photographed in time using a fundus camera equipped with a special light filter.
   (a) Blunt trauma to the optic nerve: in the early stage, the optic disc is mildly edematous with poorly defined borders. Angiography shows radial telangiectasis on the top layer of the optic disc, and later on, fluorescein leaks over the margin of the optic disc, making it blurred (the extent of telangiectasis varies). In the later stage of the injury, the optic disc appears partially or completely pale and light-colored, with retinal vascular sheathing. The angiogram shows capillary filling defects in the optic disc, either partially or completely, or reveals weak fluorescence.
   (b) Commotio retinae: shock waves act on the anterior segment of the eye, and the pressure waves propagate through the intraocular stroma and act on the posterior pole, resulting in macular edema and clouding, also known as Berlin's edema. The retinal and choroidal vessels begin to twitch acutely in response to the external force, followed by paralytic dilation, edema, and exudation. The fundus image is similar to the retinal protein edema and macular cherry red spot formed after the occlusion of the central retinal artery. FFA shows weak fluorescence in the macular area in the early stages and fluorescence accumulation and tissue staining in the late stages, similar to the macular edema found in the sick and wounded with diabetic maculopathy.
   (c) Traumatic subretinal hemorrhage: due to the impact of shock waves on the anterior segment of the eye, the damage is caused to the posterior pole of the retina. The fundus is initially dark red in and around the macula, with a hazy, cloudy surface, and gradually

turns grayish-yellow because of hemorrhage, followed by a faviform yellowish-white round or oval lesion that can gradually enlarge. FFA shows blocked fluorescence at the lesion, normal retinal artery and vein filling time without leakage or staining on the canal wall. Later in the course of the disease, there is patchy translucent fluorescence.

(d) Traumatic macular hole: when the anterior segment of the eye is impacted by external forces, the retina in the central avascular area of the macula breaks directly to form a hole, or because Berlin's edema persists, macular cystoid degeneration is induced, and the cyst ruptures to form a hole. A circular or quasi-circular retinal coloboma is seen under the ophthalmoscope. In the early stages of the macular hole, no abnormal change is seen under FFA, but later, due to pigment epithelial atrophy, the image appears as a translucent fluorescence, i.e., a window-like defect in the macular area.

(e) Choroidal rupture: it often occurs within 1–2 PD of the optic disc margin, and the laceration is usually crescent-shaped, and arranged in concentric circles with the optic disc because of external forces causing the choroid to slide on the inner surface of the sclera. The posterior polar choroid is relatively fixed in relation to the sclera, and near the back of the equator, there is little room for contraction or sliding due to the vortex vein. So, most choroidal ruptures form at the junction of sliding and nonsliding. The ophthalmoscope reveals a crescentic laceration, with the sclera exposed underneath in gray or white; the length of the laceration varies, and there may be hemorrhage or hyperpigmentation at the edge of the laceration. FFA shows early fluorescence loss of the artery, and later fluorescence staining of the exposed sclera, hemorrhage, and fluorescence blocking at the spot of hyperpigmentation.

(f) Traumatic choroidal ischemia: the choroidal artery branch is blocked by spasm after an external impact on the eyeball, resulting in ischemia of the distal choroid it supplies, and necrosis of the retinal pigment epithelium and the outer layer of the neuroepithelium on its inner surface due to interruption of blood supply. In the early stages of the disease, the retina is edematous and muddy, with scattered exudate and blood spots. Over time, the hemorrhage is gradually absorbed, the edema subsides, and a fan-shaped retinal choroidal atrophy with the tip facing the posterior pole is formed. The ischemic penumbra seen in FFA is weakly fluorescent in the early course of the disease, with a prolonged retinal arteriovenous phase and fluorescein leakage at the margins of the lesion. In the later course of the disease, there is a well-defined area of weak fluorescence.

2. *Indocyanine green angiography (ICGA)*. It chiefly reflects the choroidal condition and the morphologies of the large and medium retinal vessels.

   (a) Post-traumatic choroidal neovascularization: ICGA is clearly superior in imaging choroidal neovascularization. The choroidal neovascularization blocked by blood and cloudy fluid or occult neovascularization on FFA can often be detected by ICGA.

   A typical choroidal neovascular membrane (CNV) appears on the fundus as a subretinal gray lesion surrounded by secondary changes such as hemorrhage and exudation due to the presence of CNV. The pattern of CNVs is varied, punctate, lamellar, annular, antler-shaped, anemone strongly fluorescent or coarse vessels, and in number, one or more leaky spots. Through dynamic observation, depending on the activity of neovascularization, some new vessels are present in the early angiographic phase, choroidal neovascularization with a tendency to fibrosis is more evident in the late phase, and some CNVs may be associated with fluorescence accumulation or staining of the surrounding tissues in the late phase. The most striking feature is leakage during the imaging procedure, which is strongly supported by weak fluorescence or staining of the surrounding tissues due to hemorrhage.

   (b) Post-traumatic choroidal vascular filling defect: This weak fluorescence seen in ICGA is mainly related to altered choroidal perfusion. Vascular filling defects can be classified as physiological, secondary to vascular occlusion, and tissue atrophic. In ICGA, examples of physiological filling defects can occur in the early (choroidal watershed zone) and late (contours of the large choroidal vessels relative to background fluorescence) stages. These areas of weak fluorescence are caused by the fact that the dye present in the fundus varies from region to region.

3. *Optical coherence tomography (OCT)*. OTC is a noninvasive, high-resolution ocular tissue tomography technique that uses different tissues with different light reflectivities to form the image of tissue structures.

   (a) Traumatic macular hole (TMH): OCT images show the absence of the full-thickness retinal neuroepithelium in the macula, thickening of the retina around the hole, possible subretinal fluid, with or without posterior vitreous detachment (PVD), and sometimes the traction of the posterior limiting lamina of the vitreous body on the macula.

   (b) Traumatic lamellar macular hole (TLMH): The central fovea changes from gently sloping to steeply sloping, and the retinal neuroepithelium is partially absent.

   (c) Epiretinal membrane (ERM): This is manifested by a zone of separation between a layer of highly reflec-

tive tissue in the front of the retina and the retina, which is an epiretinal membrane separate from the retina. There is also a pre-retinal membrane that is immediately adjacent to the retinal surface and whose reflective properties are different from those of the retina beneath it, sometimes accompanied by the marginal rise of the membrane.

(d) Cystoid macular edema (CME): Retinal edema and thickening can be clearly measured with OCT, and CME manifests as a confined, nonreflective cystic space in the outer plexiform and inner nuclear layers.

4. *Visual Electrophysiology (VEP)*

(a) Electroretinogram (ERG): It is a set of electrical responses recorded in the light-stimulated retina prior to the electrical impulses in the axons of the retinal ganglion cells (RGC), reflecting the potential activities and changes in all layers of the retina from the optic cells to the ganglion cells. Blight found that the outer segments of the retinal photoreceptors were fragmented early after eyeball contusion, and then disappeared, only to recover weeks later. Hart and Blight also found that the ERG b-wave in the pig eye disappeared immediately after blunt injury and began to reappear gradually after 10–15 min, but with only half the amplitude of the pre-injury wave, indicating that not only the function of the part that showed retinal edema but also the normal retinal function are damaged. Wu Yongqiang et al. built two types of blunt injury models through animal experiments, namely light and severe, to observe the changes in a-wave, b-wave, and OPs wave, and to observe the changes in the blood–retina barrier (BRB) using lanthanum tracer. The results showed that there was a temporary decrease in ERG b-wave in terms of light contusions, but it recovered quickly, while the a-wave and OPs wave were normal, with mild disruption of the retina photoreceptor's outer segment, and unaffected BRB; in the group of heavy contusions, the outer segment of the retina photoreceptor is severely damaged, and accompanied by BRB disruption, whereas the ERG a-wave, b-wave, and OPs wave all appeared to be significantly reduced. Huang Qiumin et al. studied the early ERG a-wave and b-wave changes after experimental rabbit eye injuries and found that the ERG a-wave and b-wave amplitudes decreased significantly within 24 h of the mildly and heavily injured eyes, and the a-wave latency was prolonged. The a-wave and b-wave amplitudes in the contralateral healthy eye were also found to decrease.

(b) Visual evoked potential (VEP): It is an electrical response evoked in the visual center cortex in the occipital lobe when the eye is stimulated by light or graphics. VEP is mainly used for blunt injuries to the globe and optic nerve. VEP examinations are more commonly applied for optic nerve contusions because the clinical significance of ERG is greater than that of VEP in blunt injuries to the eye. The main manifestation of VEP in eyeball blunt injuries is prolonged P100 wave latency, with an abnormality rate of 74%, and reduced P100 wave amplitude, making up about 76%. Tang Jingqian et al. performed VEP examination on 74 eyes of 68 cases clinically diagnosed with optic nerve contusions, among which 4.00% had disappeared VEP waveforms, 71.62% lowered P100 wave amplitudes, 17.57% a prolonged wave latency, 6.76% a reduced P100 wave amplitude concurrent with a prolonged latency. When the P100 wave amplitude diminished, the margin of decrease exceeded the control value of the healthy eye by more than 80%, or for the case with the P100 wave amplitude disappeared, the post-injury visual acuity was poor, the treatment effect was not obvious, and the prognosis was bad; when the P100 wave amplitude decreased by 50–70%, the prognosis was good after treatment; when the P100 wave amplitude reduced by less than 25%, the visual acuity could be restored after treatment, and the prognosis was favorable.

1.2 First-Aid Principles

Prompt and appropriate early management of ocular blunt injuries can lead to timely control and better results while reducing complications. The following principles of first aid should be mastered:

1. When ocular blunt injuries are associated with cranial and systemic injuries, the treatment principle is life-threatening injury before the ocular injury, which can be treated at the same time as the cranio-cerebral and systemic injuries.
2. The first thing to determine is whether the ocular blast injury is closed or open. If it is closed, the focus should be placed on intraocular pressure and whether there is bleeding in the anterior chamber, which can be treated with hemostasis, corticosteroids, and by lowering the intraocular pressure; if open, further CT or X-ray examination is required to determine the presence and location of the foreign body. For management of the injured eye, first, cleanse the surface of the wound, explore the wound, and remove the foreign bodies from the eyelid, conjunctival sac, and the wound; second, reset the torn tissue. In general, try to reset the prolapsed tissue and do not cut it off easily. For the tissue prolapsed from the eyeball, such as uveal tissue, if there is no obvious contamination within

24 h, retract it after washed with antibiotics; if it is a vitreous loss, cut it off with a cotton pad until no incarceration. Finally, suture the wound, and first treat the globe rupture when the laceration of the eyelid and eyeball tissue is formed at the same time.

3. If the ocular blunt injury is associated with chemical burns such as acid and alkali, immediately flush with a buffer solution or saline. If conditions do not permit, flush with plenty of tap water or other clean water, by turning the eyelid, rotating the eyeball, and exposing the dome to thoroughly wash away the chemicals in the conjunctival sac, usually for 30 min at least.
4. If associated with thermal burns, leave the heat source or remove the injury-causing material immediately, and flush with saline or clean water to reduce the temperature.
5. In terms of radiation injuries to the eye, the focus is on prevention and protection. Once the damage is done to the eye, symptomatic treatment is generally the only solution.

2 Mechanical Ocular Injury Through Blast

2.1 Closed Ocular Blast Injury

When the shock wave acts on the eye, the dynamic pressure of the shock wave can strike directly at the globe, forming a mechanical injury. At the same time, the dynamic pressure and overpressure can also be transmitted backwards through the intraocular tissues and the wall of the eyeball, causing an indirect injury, even to the whole segment of the eye. Closed ocular blast injuries are formed when the force of the shock wave is not yet sufficient to crush the eyeball, and there is no foreign body piercing the eyeball.

2.1.1 Corneal Contusion

The cornea, located at the forefront of the globe, tough, and slightly elastic, is the first part of the eye to be injured through the blast. After high-pressure fluid or gas percussion, it is manifested by corneal edema and corneal lamellar rupture. Patients often experience pain, photophobia, lacrimation, ciliary congestion, and loss of vision. Minor corneal contusions induce localized corneal tissue damage, resulting in linear, latticed, and discoid clouding; severe contusions cause damage to the corneal endothelium, weakening or even disabling the water pump-out function within the stroma, which in turn leads to increased corneal water content, and massed or diffuse edema of the corneal stroma. In contrast, corneal laminar rupture is the laceration of interstroma collagenous fiber or elastic lamina caused by transient corneal contusions that result in the strong invagination of the cornea. Consequently, the aqueous fluid enters the corneal stroma to form diffuse corneal edema.

Treatment of corneal contusions: Corneal edema will go down within days or weeks as the corneal endothelium repairs; in terms of the corneal edema without corneal epithelial damage, topical corticosteroid eye drops or ointment can be applied. For corneal lamellar ruptures, if the laceration is small, and there is no tissue defect, sutures are not necessary; for larger ruptures with poor interlaminar alignment, interrupted sutures can be performed under surface anesthesia; regarding the patients with the corneal flap from corneal lamellar rupture, the foreign body under the corneal flap needs to be removed before anatomical alignment and suturing are performed to allow corneal flap resetting.

While throwing grit, iron filings, and other explosive debris to the eye, the shock wave can also cause keratoconjunctival epithelial abrasions, epithelial defects, corneal foreign body injuries, etc. Broad-spectrum antibiotic eye drops like levofloxacin, and those to promote corneal epithelial growth such as recombinant bovine basic fibroblast growth factors, are applied to prevent infection. Associated corneal foreign bodies can be removed with sterile wet swabs or forceps under surface anesthesia. If the foreign body is deeply located, and it is not possible to determine whether it is a full-thickness corneal penetration, the treatment is the same as that of corneal penetrating injuries; if the surface of the conjunctival sac is full of small foreign bodies, the conjunctival sac can be flushed with saline. If the area of corneal epithelial injury is large with apparent symptoms, a corneal bandage lens is used to reduce the symptom and facilitate corneal epithelial repair, but care should be taken to prevent infection.

2.1.2 Contusion of the Iris and Ciliary Body

The iris, as the forepart of the uvea, with the thickest being the pupillary margin, and the thinnest at its root, has only a layer of pigment epithelium and is particularly fragile. When the eyeball is subjected to a shock wave, its diameter expands from the middle, most notably at the corneoscleral ring, so that the root of the iris is prone to separate. When the iris sustains a blast injury, spastic pupil miosis occurs immediately, followed by mydriasis and cycloplegia (accommodation paralysis). The disturbance of tissue cell metabolism, along with the release of inflammatory mediators such as histamines, and increased prostaglandins in the aqueous humor, can cause vasospasm of the iris and ciliary body, followed by capillary dilation, hyperemia, and increased vascular permeability after an ocular blast injury. Clinical manifestations include decreased visual acuity and photophobia in the injured eye, positive aqueous flare, visible planktonic cells, fibrinous exudate, and gray dotted precipitum behind the cornea under the slit lamp. Gonioscopy shows cell and fibrin deposition. Treatment includes cortico-

steroid drops, nonsteroidal anti-inflammatory drugs, and tropicamide eye drops to activate the pupil and avoid synechia; oral administration of methazolamide or topical use of brimonidine tartrate eye drops (Alphagan) when the intraocular pressure is high. In less severe cases of iris diastasis, rest and observation without special treatment are advised, while in more severe cases, surgical repair is possible.

When the blast injury causes a separation of the longitudinal muscles of the ciliary body from the sclera at the scleral spur, it is called cyclodialysis cleft, which results in direct contact between the supraciliary cavity and the anterior chamber. When the ciliary body separates directly from the sclera but the longitudinal ciliary muscle is still linked to the scleral spur, it is called ciliary body detachment. Both can cause a state of low intraocular pressure due to a decrease in aqueous humor production and a concomitant increase in drainage resulting from ciliary epithelial edema. When the ciliary body detachment is found on UBM, it can be treated with corticosteroids and monitored if small and mild; generally, surgery is performed when the detachment is large and high, or there is a separation.

2.1.3 Anterior Chamber Hyphema (Hyphema)

The iris and ciliary body are richly vascularized and meticulously structured. When the shock wave acts on the eyeball, the anterior and posterior diameters of the globe are compressed, the central cornea is depressed, the eyeball equator is dilated, the pupillary sphincter is reflexively contracted, and the iris root is stretched. While the cornea is depressed, the aqueous humor is squeezed to the periphery and directly impacts the iris root. Hyphema is primarily due to the tearing of the anterior surface of the ciliary body, whereby the trunk and branches of the circle of Willis are broken, the ciliary vein and choroid are repeatedly fractured. Approximately 15% of hyphema is caused by the rupture of iris vessels, cyclodialysis cleft, and iris separation.

The amount of hyphema less than 1/3 of the anterior chamber volume and below the lower pupillary margin is classified as level I; occupying 1/2 of the anterior chamber volume and exceeding the lower pupillary margin is classified as level II; more than 1/2 of the anterior chamber volume and even filling the entire anterior chamber is level III.

Treatment of hyphema: resting in a semi-recumbent position with both eyes covered can adequately limit eyeball movement and prevent re-bleeding; resting in a semi-recumbent position can prevent blood from accumulating in the pupil and reduce venous congestion in the neck and eye; early systemic and local corticosteroids are recommended, along with hemostatic drugs such as oral administration of Yunnan Baiyao. Regarding mydriasis or miosis, there are different opinions. In terms of miosis, it is believed that the iris surface and anterior chamber angle can be enlarged, and the hyphema can be absorbed from the crypt on the iris surface and drained from Schlemm's canal; in terms of mydriasis, it is believed that 70% of hyphema is due to ciliary body tears and ciliary artery injuries and that after the pupil is dilated, the iris gathers at the root, but the blood vessels constrict and stop bleeding.

When there is much blood accumulated in the anterior chamber but no signs of absorption within 24 h, ocular hypotensive agents can be applied to prevent glaucoma or corneal blood staining, regardless of whether there is a rise in IOP. Systemic IOP-lowering medications include oral administration of methazolamide, intravenous drip of 20% mannitol, or topically with brimonidine tartrate eye drops (Alphagan), levobunolol hydrochloride eye drops (Betagan), etc.

The decision to treat surgically is based on IOP, corneal blood staining, and clot size and duration. Indications for surgery: anterior chamber puncture and irrigation should be done; when the blood is not absorbed over 5 days, anterior chamber infusion-aspiration should be performed when the blood is clotted other than absorbed over 5 days, IOP is elevated, or there are signs of corneal blood staining.

2.1.4 Lens Injury

1. *Traumatic cataract*. When the cornea is subjected to a blast wave, the iris is suddenly pressed against the anterior capsule due to the conduction of aqueous humor pressure, attaching pigment to the epithelium of the anterior capsule, which is called the iris ring (Vossius ring). This change is mainly at the pupillary margin. When the blast wave force is removed, the vitreous body and the lens rebound, again colliding with the iris, whereby the pigment and texture behind the pupil is reprinted on the anterior lens capsule, so that the injury to the anterior lens capsule appears in a double ring, which is more likely to occur among young people and usually fades away after a few weeks or months or persists for several years. The prognosis for vision is good.

   Traumatic disseminated subepithelial clouding, and traumatic rosette-like clouding: if the blast injury is mild, scattered pinpoint-shaped turbidness can occur in the subepithelium of the anterior part of the lens, primarily in the center or equator, either in large spreads or small areas; when the fibrous binding site under the epithelium of the anterior lens capsule is involved, the clouding looks like a feather or petal that disappears a few days or weeks after the injury, and occasionally lasts for a long time. As the lens epithelium continues to grow, the latter type of clouding is gradually moved to a deeper level, and the period of injury can be estimated according to the depth of the clouding. The prognosis is good for visual acuity.

   Diffuse contusion cataract: When a blast injury causes lens capsule rupture, the aqueous humor is absorbed, and the clouding spreads quickly, producing a cataract. In the

event of small tears, it is first closed by fibrin, and later an epithelium grows in; in the event of large tears, the clouding develops rapidly, and the fibers swell and protrude from the breach, either into the anterior chamber or into the vitreous cavity. In young patients, the cataract may be absorbed, leaving only the lens capsule; in older patients, it may be complicated by iritis and glaucoma.

Treatment: Regarding traumatic cataracts due to closed blast injuries, most are of the limited static type, and have no significant effect on vision; those with intact lens capsules do not require surgery. Atropine is given to the pupil twice daily to prevent the occurrence of posterior synechia; corticosteroid eye drops are given three times a day to control the inflammatory response. Surgery is feasible for more severe traumatic cataracts that have a significant impact on vision or lens capsule rupture.

Application of mydriatics and miotics: To use mydriatics or miotics for lens injuries depends on specific conditions. The principles of pupil constriction and pupil dilatation need to be grasped: (a) when the rupture of the lens capsule is not large and has not yet fully closed, it is advisable to use mydriatics, enabling the iris to cover the rupture, through which the contact reaction between the rupture and the iris produces a fibrinous exudate that will seal the capsule tear to prevent the aqueous humor from entering the lens; (b) if the lens cortex is dislodged into the anterior chamber to block the anterior chamber angle, there is a tendency for the intraocular pressure to rise but no obstruction in the pupil area or intraocular inflammation, miotics can be administered and closely monitored; (c) in the presence of iritis and adhesions between the iris and the lens capsule, mydriatics are appropriate; (d) if the condition of lens damage is unknown, weak pupil dilating agents such as 1% phenylephrine or 0.5% tropicamide eye drops can be used to clarify the diagnosis; (e) if the intraocular pressure is raised due to the obstruction of the pupil by lens cortex prolapse, mydriatics can be used.

Timing of surgery: For traumatic cataracts without lens edema or swelling, or cortical detachment, but there is partial lens clouding, and the eyeball is static, surgery can generally be performed 3–6 months, or 1 year after the injury. If the lens capsule rupture is large, the cortex rapidly becomes cloudy or even overflows into the anterior chamber within a few hours or days after the injury, or if complications arise, early cataract extraction is required. Otherwise, the lens cortex will absorb water and swell quickly, causing adhesions with the wound or damage to the corneal endothelium, or even phacoallergic uveitis or secondary glaucoma. If both the eyes are injured at the same time, and the vision is too poor that the patients are unable to take care of themselves, conditions should be created to perform an operation on one eye first when possible, enabling the patients to live independently.

Common surgical methods: Irrigation and aspiration are performed for young patients with soft cataracts and phacoemulsification for simple traumatic cataracts. Both can be implanted with an intraocular lens (IOL). For patients with aphakic eyes, the general conditions for second-stage IOL implantation are: 6–12 months of injury, stable injury, and disappearance of inflammatory reaction; no vitreous hemorrhage on B-scan ultrasonography and visual electrophysiological examination, no retinal detachment, normal retinal and macular functions; improvement of visual acuity through correction. To prevent amblyopia, IOL implantation should be performed as early as possible in children.

Indications for posterior chamber IOL implantation: The posterior capsule is intact or the extent of the posterior capsule defect is less than 1/3. The IOL is stable after implantation, and displacement or dislodgement of the IOL will not occur. If the extent of the posterior capsule defect is large, phakic toric intraocular collamer lens (TICL) implantation should be adopted. If the iris is defective, the residual iris needs to be able to cover the edge of the IOL; otherwise, an IOL with an artificial iris should be implanted.

If the posterior capsule defect is too extensive for a posterior chamber IOL, an iris-fixated aphakic intraocular lens or an anterior chamber IOL can be considered. But the following need to be met: basically normal corneal endothelial cells (CECs) and anterior chamber depth; the pupil smaller than 5 mm, the iris able to support the IOL stably; normal anterior chamber angle, no glaucoma; no significant cloudy lesions in the ocular tissue of the visual axis.

2. *Crystal dislocation*. After a blast injury to the eyeball, the pressure forces the eyeball to deform, the diameter of the middle segment of the eyeball expands, and the aqueous humor impacts the lens. Subsequently, due to the rebound force, the vitreous body jumps back and impacts the lens, so that the repeated impacts on the front and back of the lens can tear the suspensory ligament of the lens, causing subluxation or complete luxation. Some dislocate forward into the anterior chamber, while others prolapse backward into the vitreous cavity.

When the lens is dislocated forward into the anterior chamber, it may remain clear, or occupy almost the entire anterior chamber, or sink below the anterior chamber. When the iris is pushed backward, the anterior chamber deepens, and the pupil becomes smaller due to spasmodic constriction, which can lead to complications such as iridocyclitis and acute glaucoma. When the lens is dislodged into the vitreous cavity, the injured eye develops various symptoms due to the absence of the lens, such as deepening of the anterior chamber, iridodonesis, incarceration of the vitreous

body into the anterior chamber, loss of vision, and diplopia. The lens is still movable in the vitreous cavity in the early stage, but often fixed below in the late stage. Fundoscopy reveals tractional retinal detachment (TRD) from the disturbance of the vitreous body at the edge of the lens; the anterior lens capsule shows epithelial degeneration, the lens is in full opacity or even overripe to develop phacoanaphylactic endophthalmitis and phacolytic glaucoma.

It is sometimes not easy to detect mild, incomplete lens dislocation, with an increase in the anterior and posterior diameters of the lens, impaired distant vision and fair near vision, forming near-sightedness. Examination reveals a thickening of the lens, a forward protrusion, and a shallow anterior chamber, especially in young people who have more lens crystals, highly flexible, and more noticeable. If the lens protrudes anteriorly, the iris bulges, and the anterior chamber becomes shallow; while if the suspensory ligament is broken, the iris is sunken, the anterior chamber becomes deeper, and iridodonesis is seen in the part without the support of the lens. In severe incomplete dislocations, the pupil is slightly larger but not round, the iris surface is partly high and partly low, the anterior chamber has varying depths, and two arc-shaped light and dark parts are visible within the pupil area, with the bright part being the reflection of the dioptric media present in the lens, and the dark part being aphakia. Under transillumination, the lens area is dark, while the aphakic area is crescent-shaped red.

Treatment: If the lens is still transparent, and there is no serious visual impairment or complications such as iridocyclitis or secondary glaucoma, surgery is not required for lens subluxation; if the lens is significantly dislocated, or the resulting high refractive error cannot be corrected by wearing glasses, surgical extraction of the lens can be considered. If the lens is completely dislocated into the anterior chamber or embedded in the pupil area, it should be removed immediately; when the lens is completely in the vitreous cavity but there is no complication, it can be observed; in the event of uveitis, secondary glaucoma, or retinal detachment, vitrectomy is required to remove the lens.

2.1.5 Vitreous Hemorrhage

When the extrusion and fluctuation caused by a blast injury are transmitted to the retina and choroid, the retinal or choroidal vessels are broken, with blood flowing into the vitreous cavity. The degree of vision loss in the injured eye correlates with the severity of vitreous hemorrhage. Mild vitreous hemorrhage leads to a mild degree of vision loss; in patients with severe vitreous hemorrhage, the vision may sometimes be reduced to light perception. A small amount of vitreous hemorrhage can be detected by dilated eye examinations to understand choroidal and retinal injuries. B-scan ultrasonography is the most valuable adjunctive examination, not only reflecting the extent of vitreous hemorrhage, but also providing evidence of whether there are posterior vitreous detachment, retinal detachment, and choroidal hemorrhage.

Treatment principles: (a) retinal tears can be closely observed or given laser photocoagulation treatment; (b) mild vitreous hemorrhage can be treated with oral administration of Yunnan Baiyao; (c) regarding excessive vitreous hemorrhage, if no retinal detachment or choroidal hemorrhage is found on ultrasound examination, observation is required, and surgery should be considered if the blood is not significantly absorbed over 4–6 weeks; (d) in cases with retinal detachment or choroidal hemorrhage, surgery should be performed 1 week after the injury.

2.1.6 Traumatic Chorioretinopathy

1. *Choroidal rupture*. The injured choroidal vessel is vulnerable to breakage under the dynamic pressure and overpressure of the shock wave transmitted through the wall of the eyeball and vitreous body to the choroid. The choroidal rupture is irregular in shape, single or multiple, and after healing, a half-moon-shaped scar formed by tissue fracture is visible. The rupture is often followed by hemorrhage, possible tissue proliferation, and choroidal neovascularization. The rupture extending into the center of the macula will largely affect the vision. In general, choroidal contusions can be treated with anti-inflammatory, hemostatic, and absorption-promoting drugs according to the inflammatory response; in case of neovascularization and recurrent bleeding, laser therapy or intra-bulbar injection of anti-angiogenesis factor drugs can be used.
2. *Retinal contusion*. Slight contusions result in transient retinal edema and vision loss in the posterior pole of the fundus, and the edema will be absorbed and the vision recovered after a few days, leaving no obvious pathological changes, which is known as commotio retinae. Serious contusions can impair the outer barrier function of the retina, lead to extracellular edema and exudation, tissue necrosis, significant and irreversible loss of vision. In addition, severe blast injuries can result in retinal hemorrhage, necrosis or tear, with macular holes being the most common. Retinal contusion edema can be treated with topical or systemic glucocorticoids. Laser therapy is available for patients with retinal tears. Vitrectomy is required for patients suffering from macular holes or rhegmatogenous retinal detachment. In addition, severe blast injuries can result in retinal hemorrhage, necrosis or tear, with macular holes being the most common. Retinal contusion edema can be treated with topical or systemic glucocorticoids. Laser therapy is available for patients with retinal tears. Vitrectomy is required for patients suf-

fering from macular holes or rhegmatogenous retinal detachment.

2.1.7 Traumatic Optic Neuropathy (TON)

Shock waves, when transmitted to the optic nerve via the orbit or the brain, can cause direct contusion of the optic nerve, or ischemia and necrosis of the optic nerve. Traumatic optic neuropathy is usually divided into an anterior segment and posterior segment, with anterior TON arising from injury to the intraocular or anterior orbital segment of the optic nerve, and the posterior TON arising from injury to the intraorbital, intracanalicular, or intracranial segment of the optic nerve, often presented as a post-traumatic vision loss or blindness, or progressive vision impairment or delayed blindness, mostly on the injured eye, but also both the eyes. Among the patients, 43–56% have the eyesight declined to light perception or no light perception.

In mild cases, the direct light reflex is slow on the affected side, and there is an indirect light reflex; on the unaffected side, the direct light reflex is present, and the indirect light reflex is blunted. In severe cases, the direct light reflex on the injured side is absent, and the indirect light reflex is present; on the healthy side, the direct light reflex is present, and the indirect light reflex is absent. Funduscopic examination of anterior TON reveals optic disc edema, which may be surrounded by flame-like hemorrhage, and often varying degrees of retinal vein dilatation, whose manifestation is similar to retinal vein occlusion. Some cases may be associated with central retinal artery obstruction (CRAO), retinal pallor, and edema. In the posterior TON, the optic disc may appear to be normal in the early stages, and pallor will not occur until 4–5 weeks later.

Assistant examination including CT can be performed for optic canal fractures, and in some cases, varying degrees of diffuse thickening in the intraorbital segment of the optic nerve. Abnormal VEP waveforms are often recorded on visual electrophysiological examination. Perimetry is available for those with sufficient visual acuity, and mostly quadrantal in nature, with the lower half quadrant being the most common.

Currently, high-dose glucocorticoid pulse therapy is the most commonly used method, with dehydrating agents, vasodilators, and neurotrophic drugs given at the same time. The aim of treatment is to mitigate optic nerve edema, improve local blood circulation, increase optic nerve nutrition, and avoid further damage to the optic nerve. Medication should be administered as early as possible.

Surgical treatment may be considered in the following cases: for example, a foreign body in the orbit, hematoma, or fracture of the optic canal compressing the optic nerve. Optic canal decompression is best performed within a few hours of injury. In cases where high-dose glucocorticoid pulse therapy is not effective, optic canal decompression is sometimes effective. The purpose of surgical treatment is to remove the fracture fragments in and around the optic canal, relieve the compression or stabbing of the optic nerve, open the optic canal to alleviate the internal pressure, and improve local blood circulation.

2.2 Open Ocular Blast Injury

2.2.1 Penetrating Injury to the Eyeball

Blast waves are usually induced by an explosion. Penetrating injuries to the eyeball are full-thickness wounds in the wall of the eyeball caused after the fragments of explosives or projectiles excited by blast waves strike the eye directly, often due to the high-speed metal fragments and stones from explosions, occurring primarily in the anterior part of the eyeball, with the cornea, corneoscleral limbus, and anterior sclera being the most common.

When the penetrating injury is located in the cornea and slight, and the depth of the anterior chamber is not obviously reduced, the Seidel test can be performed to see whether the wound is airtight. From large or irregular corneal wounds, the aqueous humor can escape, making the anterior chamber shallower, the pupil deformed or displaced, and the iris usually incarcerated in the corneal wound.

Treatment: (a) Wound management. Neat, smaller than 3-mm wounds do not require suturing as they close spontaneously. To prevent the eyeball from further squeezing, and to support wound healing, a corneal bandage contact lens can be worn until the wound is firmly healed, which usually takes 3–6 weeks. For open corneal wounds that cannot be closed, with iris or vitreous incarceration, or extremely low intraocular pressure, immediate corneal suturing must be performed, and normal intraocular pressure established in principle. (b) Anti-infective treatment. For open corneal lacerations, topical eye drops are not advocated before suturing for fear of irritation and reflex squeezing of the eyeball. After suturing, antibiotics can be locally and systemically applied to prevent infection. (c) Prevention of intraocular hemorrhage. For patients with active bleeding, in addition to hemostatic drugs, one-eye or two-eye bandaging, fastening, and semi-recumbency can help reduce bleeding. (d) Tetanus treatment. Intramuscular injection of 1500 U tetanus antitoxin is for negative skin test of tetanus antitoxin, and the dose is doubled for severe wound contamination or over 12 h. Direct injection of desensitized tetanus antitoxin preparation is also possible.

Scleral penetrating wounds are concurrent with conjunctival hemorrhage, rupture and edema, ciliary congestion, corneal dehiscence or distortion, shallowing or loss, and hemorrhage of the anterior chamber, uveal incarceration or prolapse. Small scleral penetrating injuries or posterior scleral penetrating injuries are difficult to detect, especially

small wounds where only local conjunctival hemorrhage and edema are likely to be seen, and debridement and suturing require adequate cutting of the bulbar conjunctiva, for the purpose of carefully exploring the insertion of the wound; large scleral lacerations, due to massive hemorrhage and the prolapse of ocular contents, may have more manifestations, such as subconjunctival hemorrhage, anterior chamber hemorrhage, decreased intraocular pressure, ocular movement restriction, and severe vision impairment. Scleral wounds do not close easily, and irregular wounds that are larger than 2 mm require suturing.

2.2.2 Intraocular Foreign Bodies

Foreign bodies pierce the wall of the eyeball and remain in the eye as intraocular foreign bodies. The two full-thickness wounds caused by foreign bodies passing out through the wall of the eyeball are called globe perforation.

If the intraocular foreign body (IOFB) is an active metal like copper or iron, it can cause serious injury to the eye in the immediate future due to its toxicity and ocular siderosis in the long term, bringing extensive damage to the injured eye, which indicates bad prognosis. IOFBs, especially in seriously polluted environments of injury where the organics such as wooden foreign bodies exist, are also the most frequent cause of post-traumatic endophthalmitis. Therefore, once diagnosed, they need to be surgically removed as soon as possible. CT scans have excellent visualization of metallic foreign bodies, and MRI is suitable for the diagnosis of nonmagnetic foreign bodies. The removal of extraneous matters varies according to the property, size, magnetism, depth, and location of the foreign body. If the wound is large, and the location of the foreign body is shallow, the foreign body can be removed via the original wound during debridement and suturing. If the foreign body is magnetic, a magnet can be used to hold and draw the foreign body during the primary suture of the perforating wound, even though it is deeply located in the eye; if the foreign body is small and deep into the vitreous cavity, vitrectomy can be performed to take it out. The surgery is not just aimed at removing foreign bodies, but also preventing long-term complications like proliferative vitreoretinopathy (PVR). In case of endophthalmitis, antibiotics such as ceftazidime and vancomycin are injected into the vitreous space besides while removing the foreign body.

2.2.3 Eyeball (Globe) Rupture

When the overpressure of the blast wave is applied to the eyeball, it is subjected to tremendous pressure, and the intraocular pressure rises sharply. The acting force from the inside out, usually causes the wall of the eyeball to disrupt from the weakest area, either at or not at the force bearing point. Globe ruptures often occur at the corneoscleral limbus where the wall of the eyeball is weakest. Clinical statistics indicate that the most frequent sites of scleral rupture are between the posterior corneal limbus and the Tillaux spiral (the circle through the insertion of four rectus muscles) in the nasal superior quadrant, and between the Tillaux spiral and 5 mm behind the equator in the superotemporal quadrant, followed by the area between the posterior corneal limbus and the Tillaux spiral in the superotemporal quadrant. Globe ruptures are often severe, with large wounds and prolapsed ocular contents, and require careful intraoperative exploration to return as much of the uncontaminated contents as possible. The aim of suturing the globe rupture is to preserve the shape of the eyeball, reduce long-term complications, and restore as much vision as possible.

3 Ocular Blast Injuries Associated with Ocular Chemical Burns

3.1 Acid Burns

3.1.1 Features and Mechanisms of Acid Burns

Acids are water soluble, which, at low concentrations, do not easily penetrate the epithelial structures when in contact with the tissue, thus unable to further damage deep tissues. The epithelial tissue, rich in lipids, has a good shielding effect against aqueous liquids. High concentrations of acids, when in contact with the tissue, will result in the coagulation and denaturation of the proteins in the tissue. As the coagulated proteins are insoluble in water, they form a barrier on the surface of the injury, and to some extent prevent further penetration of the acids into deeper layers. But this is in comparison with alkali burns and refers to weak or diluted strong acids only. Clinically, the burns from high concentrations of strong acids (e.g., sulfuric and hydrochloric acids) can also lead to severe and devastating ocular injuries. In addition to the concentration and pH of the acid, the biological effects of the acid on the tissue vary considerably as well. Trichloroacetic acid, for example, causes tissue damage at the pH of 4.5, whereas hydrochloric acid has a damaging effect at the pH of 2.5. Concentrated sulfuric acid has a strong affinity for water and releases a lot of heat when exposed to water, causing both chemical and thermal damages to the ocular trauma, which may often be accompanied by damages to the eyelid and functional loss, and have a bad prognosis. Acid anhydrides, such as nitrogen dioxide, sulfur dioxide, and sulfur trioxide, are both water-soluble and lipid-soluble and can produce acids when encountering water. Once in contact with the eye, it quickly penetrates into the deep tissues, causing serious injuries similar to alkali burns. Hydrofluoric acid has a small molecular structure that makes it easy to penetrate tissues and also has a strong cell-damaging effect. Chromic acid is a strong corrosive agent, the solution and even the volatile gas of which can cause serious damages to the eye.

3.1.2 Classification and Prognosis

According to the ocular burn classification standard developed by the Ocular Trauma Classification Group, the contents include damage to the corneal epithelium, degree of stromal edema and clouding (indicating injury depth), and corneal limbus ischemia (whitening of the corneal limbus). Ocular burns are categorized by four degrees.

1. *First-degree burns*. It is manifested by corneal epithelial exfoliation, bulbar conjunctival edema, and congestion. The symptoms of irritation such as photophobia and tearing are obvious and subside in 1–2 days, and the corneal epithelium heals spontaneously without cicatrix.
2. *Second-degree burns*. It is characterized by edematous and cloudy corneal superficial stroma, indistinct iris texture, and sparse conjunctival vessels, and thin, dull black blood vessels, often with small hemorrhagic spots. The duration of corneal limbal ischemia does not exceed 1/4 week. With treatment, most recover, or a little pannus remains.
3. *Third-degree burns*. The deep stroma of the cornea is in a grayish white muddiness, through which only the outline of the pupil can be seen. The conjunctiva is present as white coagulated necrosis with loss of blood vessels. The corneal limbus is ischemic to within 1/2 week. It is often accompanied by intraocular reactions like iridocyclitis. Macula cornea and atretoblepharon (the globe and eyelid form an abnormal adhesion to one another) remain after proper and prompt treatment. If not treated in time, the cornea will continue to ulcerate and even perforate.
4. *Fourth-degree burns*. The cornea is porcelain-white, the conjunctiva presents as white or yellow necrosis, and the corneal limbus ischemia lasts for more than 1/2 week. At this point, there are no signs of irritation, but corneal perforation, pantankyloblepharon, and visual loss often occur.

Although the criteria and elements above are true with acid burns, the classification results recorded 24–48 h of injury are more reliable due to the fact that the immediate presentation of acid burns is more severe than expected.

3.1.3 Clinical Manifestations

After exposure to acids, a variety of subjective symptoms may occur, including burning pain, foreign body sensation, tearing, photophobia, blepharospasm, and blurred vision to name a few. In mild cases, there may only be conjunctival congestion and edema on optic examinations. Corneal injury is usually associated with epithelial exfoliation, corneal stromal edema, and clouding. In severe cases, the eye tissue may be charred and necrotic, with the complete destruction of the corneal limbus, or even corneal perforation and loss of vision.

3.1.4 Treatment

In principle, it involves fast removal of injury-causing materials, control of ocular inflammatory response and complications (prevention of infection, control of intraocular pressure, prevention of tissue lysis and adhesions, etc.), promotion of tissue repair, and efforts to preserve and restore the visual function.

1. *First-Aid Treatment*
   (a) Flushing: The injured area is thoroughly rinsed with water shortly after injury, and timely and continuous flushing is the key to emergency treatment. For severe burns, overemphasis on water quality is unnecessary, and the right thing to do is immediate rinse at the scene of injury. After initial flushing, when available, surface anesthetic drops are placed in the conjunctival sac before washing with saline. The flushing time is at least 1 h. This can be measured with a piece of test paper until the pH value is ≥ 7. If the injury-causing material is determined to be acidic, it can be neutralized with a 2% sodium bicarbonate solution. However, an alkaline solution should not be used to attempt neutralization without initial flushing, which can otherwise aggravate the condition.
   (b) Removal of solids: Especially residual foreign bodies that may be hidden in the conjunctival sac in the upper and lower fornices.
2. *Medication*. After emergency irrigation, a more detailed examination should be carried out, including visual acuity, lid, epithelial, and corneal rim involvement. The degree of corneal stromal edema clouding is initially difficult to determine due to clouding of the corneal epithelium, unless there is epithelial exfoliation.
   (a) Topical application of broad-spectrum antibiotics: The main aim is to prevent secondary infection.
   (b) Cycloplegics: For most patients, long-acting drugs such as homatropine or even atropine are preferable.
   (c) Corticosteroids: Early application can protect the tissue and reduce the inflammatory response, but just for 1–2 weeks; otherwise, they may inhibit the tissue repair process, activate collagenase, and accelerate ulceration and perforation.
   (d) Vitamin C: In animal experiments, 10% vitamin C solution administered orally or as topical drops has reduced the incidence of corneal ulcers. It is presumed to be beneficial for acid burns to human ocular surfaces as well, but there is no detailed clinical validation and experience.
   (e) Growth factor: Eye drops of epidermal growth factors (EGF) are currently available. EGF promotes cell proliferation and epithelial repair. Concerning those with corneal epithelialization followed by recurrent exfoliation, the fibronectin (FN) isolated

from autologous plasma is conductive to epithelial cell adhesion, and more effective if used in combination with EGF.

(f) β-Blocker: It is applied for secondary glaucoma and may require concomitant oral administration of carbonic anhydrase inhibitors if necessary.

(g) Analgesics: For severe pain, temporary oral use of analgesics is recommended.

3. *Surgical treatment*. Major acid burns to the ocular surface often cause extensive tissue necrosis, persistent corneal epithelium defects, corneal ulceration, or perforation. Later occurrence of corneal opacity, pannus, corneal limbus deficiency, atretoblepharon, and incomplete eyelid closure may require surgical treatment, such as amniotic membrane transplantation (AMT), keratoplasty, conjunctival autograft (CAT), limbal transplantation, and mucous membrane grafting (MMG).

3.2 Alkali Burns

3.2.1 Features and Mechanisms of Alkali Burns

Alkaline substances can saponify with lipids in tissue cells, forming compounds that are both lipolytic and water-soluble, enabling them to fast penetrate epithelial tissue, corneal stroma, and deeper. Therefore, unlike acid burns, the boundaries of injury zones are generally blurred clinically, making it difficult to accurately delineate the extent and depth of the injury zone. Alkali burns can be divided into the following courses of disease:

1. *Acute phase*. Within 1 week of burn, the most striking pathological change is tissue ischemia, edema, and extensive necrosis. The cells within the conjunctiva and cornea disintegrate and fall off, the coagulative necrosis of collagen fiber lamellae slowly occurs, and the corneal cells disappear as necrotic keratitis. Microvascular thrombosis is seen within the conjunctiva and sclera.
2. *Corneal melting phase (early repair stage)*. In the second to the third week of the burn, the tissue enters a pathological process of regeneration, ulcer deepening, enlargement, and interlocking, which is also the peak time of collagenase release from the tissue. Due to the action of large amounts of collagenase, most of the corneal parenchyma, epithelium, and endothelial cells undergo lysis and necrosis, and the cornea in the injured area is in a cell-free state, making it prone to corneal ulceration and perforation.
3. *Tissue repair phase (late repair stage)*. In the fourth week of the burn, the long period of epithelial failure to repair and increased tissue inflammatory exudation eventually lead to cicatricial atretoblepharon and corneal cicatricial vascularization.

Corneal alkali burns are different from other injuries: they take a long time to repair, do not heal for a long time, relapse, and have a poor prognosis.

3.2.2 Clinical Manifestations

Like other chemical injuries, the ocular surface may have subjective symptoms including burning pain, foreign body sensation, tearing, photophobia, blepharospasm, and blurred vision after exposure to alkaline substances. In mild cases, there may only be conjunctival congestion and edema on optic examinations. Corneal injury is usually associated with epithelial exfoliation, corneal stromal edema, and clouding. In severe cases, the corneal limbus may be completely damaged with ischemia. In corneal burns caused by calcium-bearing alkaline substances, many white-dotted granular calcium deposits are usually seen in the corneal stroma.

1. *Clinical features*. (a) Injury-causing materials permeate the tissue quickly, requiring prompt on-spot flushing and resuscitation. (b) The boundary of the injury zone is unclear, which is helpful in the initial determination and identification of acid and alkali burns. (c) Deep tissue damage often occurs, including the iris, lens, and ciliary body. Therefore, in cases where the anterior chamber cannot be seen due to corneal opacity, early salvage treatment must take into account the control of inflammation. (d) Increased intraocular pressure. In addition to secondary glaucoma from the mechanisms of inflammation and synechia, the rise in IOP in the immediate aftermath of alkali burns is presumed to be related to the contraction of collagen fibers. (e) The cornea is often not fully epithelialized. Persistent epithelial defects frequently lead to corneal indolent ulceration or even perforation. This is closely related to severe tissue destruction, increased local collagenase activity, and lack of vitamin C and nutrient supply. (f) The tissue inflammation of alkali burns induces massive neovascularization and cicatrization. (g) Conjunctivalization and vascularization resulting from the structural damage of the corneal limbus. (h) Late conjunctival sac constriction, atretoblepharon, incomplete closure, and dysfunctional tear secretion and distribution. Many of these elements pose difficulties for sight rehabilitating operations such as corneal transplantation and serious challenges in maintaining surgical effects.

3.2.3 Treatment

In principle, (a) remove the alkaline substances as soon as possible; (b) promote tissue repair, particularly regeneration of the ocular surface epithelium, to cover the cornea with corneal phenotype cells; (c) prevent ulceration, support collagen production by corneal cells, and reduce collagenase activity; (d) control the ocular inflammatory response and complications (including prevention of infection, control of

intraocular pressure, and prevention of tissue lysis and adhesion); (e) preserve and recover the visual function.

1. *Immediate First-Aid Treatment of the Burn Injury*
   (a) Flushing: The injured area is rinsed with plenty of water continuously and immediately on the spot rather than the undue emphasis on water quality. Then, a more detailed examination can be performed when surface anesthetic drops have been applied to the conjunctival sac, to see whether the visual acuity, eyelid, epithelium, and corneal limbus are involved. Next is the classification of corneal burns (see the acid burn classification standard).
   (b) Removal of solids: In particular, residual foreign bodies that may be hidden in the conjunctival sac of the upper and lower fornices.
   (c) Anterior chamber paracentesis: As alkaline substances can quickly enter the anterior chamber, if the exposure to high-concentration alkaline substances is prolonged, measures such as anterior chamber paracentesis and radial incision of the bulbar conjunctiva should be employed as appropriate after flushing to remove as much of the residual alkaline substances as possible from the deep tissues. Anterior chamber paracentesis is effective in draining the alkaline materials in the anterior chamber and regenerating the aqueous humor shortly after injury. It also relieves the transient increase in intraocular pressure caused by the burn. Radial incision of the bulbar conjunctiva is indicated in cases of serious conjunctival edema and may be helpful in alleviating tissue pressure and draining subconjunctival exudate.
   (d) Removal of devitalized tissue: It depends, as excessive clearing may cause large-scale tissue defects.
   (e) Topical broad-spectrum antibiotics: The main aim is to prevent secondary infection. For epithelial defects, application of antibiotic eye ointment and compression bandaging can be effective in promoting epithelialization of the ocular surface. The eye ointment also helps to lubricate the ocular surface and lessen adhesions.
   (f) Corticosteroids: Application in the early stages can help protect the tissue and lower inflammatory response. Hormonal eye ointments or drops containing antibiotics are currently available. Effective control of inflammation promotes epithelial healing and abates the reoccurrence of corneal neuroepithelial exfoliation.
   (g) Cycloplegics: For mild burns, short-acting mydriatics are available to move the pupil and avoid posterior synechia of the iris. For relatively severe burns, long-acting drugs such as atropine are required.
2. *Medication*. Depending on the cause stage of the alkali burn, corresponding treatment is given for the purpose of rebuilding and maintaining a healthy corneal epithelium, controlling the balance between collagen synthesis and collagen lysis, and reducing sequelae.
   (a) Acute phase: It includes topical and systemic application of antibiotics to prevent infection, and glucocorticoids to inhibit inflammatory response and neovascularization, but great care must be taken when applying topical glucocorticoids. This is because improper use may result in serious corneal damage and melting. Efforts should be made to improve the microenvironment of the conjunctival sac by using preservative-free artificial tears, ophthalmic solutions containing growth factors, or wearing hydrophilic, highly oxygen-permeable corneal contact lenses to accelerate epithelial healing and support repair; oral vitamin C and topical application of 10% vitamin C solution, or combined topical application of cysteine, acetylcysteine, and ethylenediaminetetraacetic acid disodium salt (EDTA) among other collagenase inhibitors to minimize ulceration. Cycloplegics and IOP-lowering medications are used concurrently. Oral analgesics can be administered when necessary.
   (b) Corneal melting phase (early repair stage): If the corneal epithelium has not yet been repaired, intensive use of lubricants, corneal contact lens, or even tarsorrhaphy is required, and the application of hormones needs to be gradually reduced and discontinued within 14 days of injury. Continual treatment is given with vitamin C, collagenase inhibitors, antibiotics, and intraocular pressure lowering. Mechanical separation in conjunction with topical lubricants is performed to prevent symblepharon.
   (c) Tissue repair phase (late repair stage): In addition to continued pharmacological treatment, surgery is the primary treatment modality at this stage. Surgical treatment including corneal contact lenses, amniotic membrane coverage and tarsorrhaphy, oral mucosa grafting, transplantation of corneal limbal epithelial cells, and corneal lamellar or full-thickness transplantation is chosen for local symptoms.

Common sequelae of late severe chemical burns include corneal opacity, atretoblepharon, entropion or ectropion, and increased intraocular pressure. The appropriate surgical procedure is chosen for specific conditions, such as tarsoplasty and reconstruction of the conjunctival sac, correction of entropion or ectropion, and surgical correction of atretoblepharon. If secondary glaucoma, complicated cataracts, or proliferative vitreoretinopathy (PVR) is present, it requires appropriate surgical and pharmacological treatments.

4 Blast Injuries in Combination with Thermal Burns of the Eye

Burns directly caused by flame are called flame burns, and thermal burns from hot liquids (i.e., molten metal) or solids (i.e., explosive fragments, glass, cinders) splashed into the eye are termed contact burns.

4.1 Flame Burns

1. *Flash burns*. They are commonly seen, superficial burns, mostly invading the face (including the eyes) and hands. In general flame burns, the eyeball is rarely invaded, because the protective effect of tightly closed eyelids due to blink reflex is sufficient to prevent or diminish the direct effect of the flame. However, the eyelashes and eyebrows are scorched in most cases. If the effect of heat is intense and long lasting, the eyelid can also be burnt, the cornea necrotic; in most severe cases, the entire eyeball and orbital contents are burnt, and the injured rarely survive.
   (a) Magnesium flash burns: Usually, only the superficial layer is burned. Flash bomb burns, for example, are caused when there is not enough time for the eyelid to close, resulting in small magnesium particles in the exposure splashed over the surface of the globe. If not removed in time, they can penetrate deep, piercing the cornea, and forming ulcers. If conjunctival ulceration occurs, it will partially adhere to the eyelid.
   (b) Phosphorus burns: They can be seen occasionally in wartime or peacetime. White phosphorus is often used as incendiary bomb filler. Phosphorus can spontaneously combust in contact with air and is particularly destructive. As phosphorus particles can be buried in the skin or on the surface of the eye, they will continue to burn if no action is taken. Its products can also generate phosphoric acid when in contact with water, further corroding the tissue. If the local burns are extensive, they can cause degenerative changes to parenchymatous organs because of systemic absorption, such as hepatotoxicity and nephrotoxicity, which can be fatal. The most effective emergency measure is washing the skin with 5% copper sulfate and putting drops of 0.5% copper sulfate in the conjunctiva, so that the phosphorus particles are coated with a layer of copper phosphate, thus preventing exposure to oxygen in the air, and becoming inert. The phosphorus particles on the surface of the eye must be removed with forceps, or dipped out with a wet cotton swab. After the phosphorus particles have been cleared, take a 2% sodium bicarbonate solution as a wet dressing or immersion cleaning to neutralize the phosphoric acid. If it is difficult to obtain copper sulfate for a while, the best way is to immerse the head and face in water, or to continually dip rinse the conjunctival sac in water or, in the absence of water, to apply a water-soaked pad to isolate it from the air and prevent continued spontaneous combustion. Oil-based dressings are prohibited as phosphorus dissolves quickly in oil. Subsequent therapy is the same as for general thermal burns. Phosphorus burns are generally slow to heal.
2. *Gunpowder (aka black powder) explosion burns*. They are transient, superficial burns, sometimes deeper, and unable to form ulcers, causing permanent corneal opacity, or even penetrating injuries to the eyeball. There are often innumerable "salt and pepper particles" on the cornea and bulbar conjunctiva. The explosion can also result in concussions and lacerations. The artillery may be burnt by hot gas when delivering fire, and in many cases, the gunpowder gas burns in the eye are accompanied by eyeball contusions.
3. Burning napalm bomb coagula adhere easily to the body. Those who try to extinguish them will get burning petrol on their bodies, which are not easy to get rid of. If you try to put out a burning gasoline clot on your body, you should not use your hands to put it out, but try to isolate the flame from the air, for example, by immersing the burning part in water, or by wrapping it in a mackintosh, quilt, or coat, or by taking off the burning clothing and throwing it away. Generally, the petrol clots that fall on the body result in extensive and deep burn injuries because they burn slowly at a high temperature and for a long time. The necrotic tissue from such burns is slow to regenerate, usually with local infections, and the scar is often in the form of keloids.
4. *Clinical Manifestations*.
   (a) Flame burns of the eyelid: Because of the blinking reflex effect of the eyelid, most flame burns involve the eyelid only, and a narrow strip of skin at the palpebral margin is often preserved from burns due to forceful eye closure. The superficial burns of the eyelid skin are manifested by eyelid congestion, edema, and inability to open the eyes. It peaks 12 h after injury and begins to decrease slowly at 36 h, gradually subsiding and healing. If the epidermis and partial dermis of the eyelid are involved, the swelling and edema not only enlarge the whole face, but there are also clear serous blisters of varying sizes on the surface of the skin. In cases involving the entire epidermis and dermis of the eyelid or even damaging the muscles and tarsal plate, the eyelid is yellow-brown

or black crusted, and the wounded surface is extremely difficult to heal. In severe cases, not only is the eyelid destroyed, but there is also deep and extensive tissue loss in the forehead and cheeks, which is often prone to infection. Separation of slough follows granulation; in the granulation tissue, there is fibroplasia, the contraction of which can make the face ugly and misshapen. The most important aspect of this procedure for the eye is severe eyelid ectropion. It exposes the conjunctiva and cornea, resulting in exposure conjunctiva and keratitis. In mild cases, vision is affected, and in severe cases, exposure corneal ulcers occur, with secondary infection or even perforation and blindness.

(b) Corneal flame burns: Mild epithelial damage, moderate superficial corneal stromal edema, severe full-thickness involvement, and invisibility of the iris. If a secondary infection develops, the cornea ulcerates and even perforates.

(c) Flame burns of the palpebral margin: Blockage of the tarsal gland drainage orifice, deformation of the skin–conjunctiva junction, and metaplastic lashes causing irritative keratoconjunctivitis; involvement of the puncta and lacrimal ductile in the medial canthus with fibrosis, resulting in sequelae such as epiphora from obstruction of the lacrimal passage.

4.2 Contact Burns

In industrial production, contact burns are frequently caused by molten metal, slag, solder, glass, or burning cinders splashing into the eye. In wartime, it is often caused due to the spattering of explosive fragments into the eye. There is often a hot substance attached to the wound.

1. *Factors determining the extent of thermal burns*. The severity of contact burns is determined by the size, temperature, and contact time of the hot object.

   The smaller the hot object, the less heat it carries, the quicker the tissue cools after contact, and the smaller and shallower the burn; conversely, the larger the hot body, the more heat it carries, the slower it cools, and the greater the damage caused.

   The higher the temperature of a hot object, the more heat it carries, and the heavier the tissue injury caused. High-temperature liquids or solids, if more than 1000 °C, can result in serious burns, such as molten iron (with a melting point of 1200 °C), molten glass (1300–1500 °C), and molten copper (1000 °C). Substances with a low melting point like lead (330 °C), tin (280 °C), and oil wax (grease) lead to slight thermal burns.

   The longer a hot object of the same temperature and size is in contact with the tissue, the more severe the damage caused.

2. *Clinical Manifestations.*

(a) Conjunctival and iris burns: The hot object is left in the conjunctival sac if it has come into contact with the eye before the eyes blink reflex and the eyelid subsequently closes. If it is a low-temperature object, it will be cooled by the tear film covering the surface of the eyeball once in contact with the eyeball, to rapidly generate a thin film; on the other hand, a hot molten metal will be left in the conjunctival sac, and all the heat carried in the process of cooling into a solid is transferred to the tissue, which will cause serious damages to the conjunctiva, iris, and cornea.

Degree I and II conjunctival burns do not leave a scar after healing, but for burns of degree III or above, not only will the conjunctiva be coagulated and necrotic, but the sclera underneath it will also be necrotic and even perforated, often with atretoblepharon after recovery.

(b) Corneal burns: According to animal experiments, above 45 °C hot objects can result in corneal thermal burns, 60 °C can cause corneal edema, and 80 °C can cause corneal endothelial damage.

Superficial corneal burns often occur in hot water, steam, sparks from burning coal, hot oil, etc. Low-temperature materials, when in contact with the cornea, lead to a visible gray-white necrotic membrane on the corneal surface that will recover over 1–2 days, leaving no trace, but a slight nubecula.

Deep corneal burns are commonly from hot molten metals, such as iron, copper, and lead. After the metal cools down and is attached to the surface of the cornea or conjunctiva, a burn wound of the same size and shape is formed, accompanied by corneal opacity and necrosis, infiltration of materials from the anterior chamber, miosis, as well as uveitis, pain, photophobia, and tearing. When the necrotic membrane falls off, the cornea becomes thinner, the corneal cloudiness is alleviated or even becomes transparent, which should not be mistaken for improvement, but is often a prelude to corneal perforation. If emergency measures are not taken at this point (e.g., lamellar keratoplasty), the cornea will be perforated. If scar repair with a large number of new blood vessels grows in, a membranous or thick vascular opacity (flesh-like vascular opacity) and cicatrix will form. Corneal scarring not only affects vision, but because of its weak resistance, often forms localized corneal staphyloma.

Severe corneal burns can lead to scorching, necrosis, and perforation of the corneal tissue, resulting in

endophthalmitis and blindness due to prolapse of the eye contents. If the palpebral conjunctiva is also burned, the upper and lower eyelids are often completely adhered to the eyeball to form eyelid atresia.

4.3 First Aid and Treatment of Thermal Burns

Thermal burns to the eye are usually associated with extensive burns to the face, neck, chest, upper limbs, and other parts of the body, whether in wartime or normal times. In the emergency treatment of patients with thermal burns, it is important to fully understand the whole body and local conditions, and both systemic and local treatment is desirable in the first-aid treatment of patients with severe burns.

1. *Systemic treatment*. Early systemic treatment should focus on the prevention of shock and infection according to the whole-body injury. Regarding large area, degree II, III, and IV burns, pain relief, fluid infusion, and prevention or salvage of shock come before prevention of local and systemic infections.

   In terms of a battlefield or on-spot rescue, the main focus is to protect the injured part, try to create opportunities to reduce shock and infection, and evacuate quickly.

   With regard to protecting the injured part, the first thing is to put out the fire. For localized phosphorus burns, it is required to quickly immerse the injured area in water, or cover it with waterlogged cloth to isolate the phosphorus from the air. Other measures include oral administration of morphine, necessary fastening of the injured part, and immediate delivery to the hospital.

   Regiment- and division-based medical aid stations and primary medical units should, according to systemic conditions, first administer subcutaneous pethidine, then tetanus antitoxin. If necessary, inject anti-shock fluids, and supplement with a high-protein, high-vitamin diet, orally for those who can eat. Give salt-containing drinks rather than boiled water to avoid water intoxication. For severe burns, give plasma or blood transfusions, and inject or orally administer antibiotics. If necessary, inject anti-shock fluids, and supplement with a high-protein, high-vitamin diet, orally for those who can eat. Give salt-containing drinks rather than boiled water to avoid water intoxication. For severe burns, give plasma or blood transfusions, and inject or orally administer antibiotics.
2. *Local Treatment.*
   (a) Degree I to II face (including eyelid skin) burns: The principle of treatment is exposure therapy. Rinse the face and eyelid with saline, drip antibiotic solution or antibiotic eye ointment, take a cage to protect the head and neck while dressing the cage with a sterile towel, or gently cover it with several sterile dressings. The advantages of this therapy are that the burning surface can come into contact with the outside air, good for drying and wound healing; the temperature of the exposed wound surface is lower than body temperature, and thus unfit for bacterial growth; it is easy to observe and simple to care for. This type of patients does not need to be hospitalized and can be healed in about 3 days.
   (b) Degree II to III eyelid burns: First scrub the healthy skin around the burn with soapy water, then rinse the wound with sterile saline while gently wiping away any dirt or residue in the wound with sterile wet cotton balls or gauze. Pierce the blister with a disinfected syringe needle to extract the fluid, then apply broad-spectrum antibiotic eye ointment to prevent and inhibit secondary infection and protect the wound without hindering epithelial growth. Attention should be paid to the prevention of systemic shock and infection in such case, promptly followed by transport to the hospital with the corresponding department for continued treatment.
   (c) Eyelid burns above degree III: Wet pack the necrotic area of the skin with the gauze immersed in a saline solution containing 0.25% gentamicin to make the eschar come off quickly. In healthy granulation tissue, perform early skin grafting and tarsorrhaphy to avoid ectropion and lagophthalmos, and protect the eyelid and its function.
   (d) Degree I to II conjunctival and corneal burns: Place mydriatics and antibiotic ointment to the injured eye; apply drugs that promote corneal epithelial repair and regeneration, such as recombinant basic fibroblast growth factor (Beifushu eyedrop), basic epidermal growth factor (Yibei eye drops), corneal nourishing drugs (deproteinized calf blood extractives, an ophthalmic gel), and nerve growth factor (NGF) that can promote corneal epithelial cell proliferation and migration, and regulate the differentiation and proliferation of corneal stem cells to avoid corneal stroma opacity and facility stromal closure.
   (e) Severe corneal burns: The aim of treatment is to keep the eyeball intact and prevent serious complications. Observe the injured eye carefully. If the necrotic tissue is shed, and the cornea becomes thin and transparent, it is a precursor to corneal perforation, which requires urgent transplantation of total cornea with circular lamellar sclera to prevent perforation and prolapse of ocular contents. Before the materials of keratoprosthesis are found, give antihypertensive medication, apply cyanoacrylic adhesive, or wear soft corneal contact lenses to temporarily protect the cornea, while giving oral cough suppressants and

intramuscular injection of lytic cocktail 1 so that the patient can sleep quietly. Perform therapeutic corneal transplantation with circular lamellar sclera as soon as the corneal material is found.

(f) Severe conjunctival and scleral burns: If partial, remove the necrotic tissue completely and graft the supratemporal bulbar conjunctiva of the healthy eye by fixing it to the scleral surface of the defect. If both the eyes are burnt, or the burn is so extensive that atretoblepharon cannot yet be prevented using the above method, only early transplantation of lip mucosa can be adopted to repair the conjunctival defect, prevent adhesion, improve the condition of the conjunctiva, and facilitate corneal healing.

(g) Burns deep to all layers of the sclera: In this case, the injured eye is at risk of perforation, and it is best to leave the eye untreated, allowing the lid tissue adhering to the eyeball to promote wound repair. Plastic surgery is desirable at a later stage.

5 Blast Injuries Complicated by Radiation Damage to the Eye

Radiation injuries include ionizing radiation and nonionizing radiation damages. Ionizing radiation damages are induced by far-ultraviolet rays (short wavelength), X-rays, γ-rays, and nuclear radiation rays. These rays are increasingly strong in energy as the wavelength decreases and are emitted by neutrons, atoms, and protons among other particles changing their state of motion. They can penetrate into tissues at different depths, producing a biological effect within the tissue, which is a high-energy photonic effect. Nonionizing radiation damages, on the other hand, include injuries caused by near-ultraviolet light, visible light, infrared light, microwaves, etc. These electromagnetic waves, emitted by electric oscillators, feature long wavelengths and weak energies and create photochemical or thermal effects within the tissues.

1. *Injuries through infrared rays*. Infrared light is usually produced by hot objects, and the damage it caused to the eye is mainly thermal. This is due to the increased rate of molecular movement in the tissue and the rise in temperature resulting from the absorption of the energy propagated via infrared vibration by the tissue. Common ocular damage from infrared rays includes chronic blepharitis and thermal cataracts due to prolonged exposure to low-energy short-wave infrared rays (e.g., blast furnace and glass workers), and solar eclipse burns of the retina caused by observing a solar eclipse.
2. *Ultraviolet damage*. UV light is a part of the radiation, categorized by two types: long-wave ultraviolet (300–400 nm) and short-wave ultraviolet (180–300 nm). The wavelengths of 315–400 nm have a mild effect on tissue; the wavelengths of 280–315 nm have a strong effect on the skin; the wavelengths of 200–280 nm have a destructive effect on tissue proteins and lipids, and cause hemolysis; the wavelengths of 250–320 nm can result in electric ophthalmia, with the wavelengths of 265–280 nm being the most serious. Of 375–400 nm, a very small portion can invade the fundus; the wavelengths of 300–375 nm can reach the lens; for 300 nm or less, the depth of invasion does not exceed the cornea. They can cause electric ophthalmia (snow blindness), cataract, and membrane damage, respectively. The light from welding, plateaus, snow and water surface reflections, UV lamps, and atomic bomb explosions is generally short-wave ultraviolet, with a wavelength of about 290 nm, which can cause ocular ultraviolet damage, namely electric ophthalmia. It usually strikes 3–8 h of exposure, with a strong feeling of foreign bodies, stinging pain, photophobia, lacrimation, and blepharospasm, mixed conjunctival congestion and punctate peeling of the corneal epithelium. The symptoms begin to diminish 24 h later.

   Treatment and prevention: The aim of treatment is to ease the patient's pain, facilitate recovery from the injury, and prevent secondary infection. Early cold compresses or bandages on the injured eye can relieve blepharospasm and attenuate symptoms. Topical application of 0.5–1% tetracaine eye drops once can immediately alleviate the eye pain. As anesthetics inhibit the regeneration of the corneal epithelium, they should only be used as a stopgap measure for first aid and should not be used repeatedly; cocaine can damage the corneal epithelium and should be contraindicated.

   To relieve the discomfort induced by hippus, compound tropicamide eye drops can be topically used to dilate the pupil. Basic fibroblast growth factors can promote healing of the corneal epithelium. Both indomethacin ophthalmic solution and dexamethasone suspension have anti-inflammatory effects and are able to shorten the course of the disease. In addition, sunglasses can reduce photaesthesia.
3. *Ionizing radiation damage*. It includes those caused by X-rays, γ-rays, and nuclear radiations, all belonging to the ionizing radiation, and unable to cause damage to various eye tissues, including the conjunctiva, cornea, lens, uvea, retina, and optic nerve, with the sclera being less sensitive. The active mechanism of ionizing radiation injuries is generally considered to be of three types: one is the direct action of radiation on tissue cells, causing abnormal cell growth or death; the second is vascular damage to the tissue that results in secondary injury; the third is the entry of large amounts of cell disintegration products into the bloodstream, triggering a systemic toxic reaction, i.e., radiation-induced shock.

Clinical manifestations are erythema, dry or wet desquamation (blister plus exfoliation) of the eyelid skin, and damage to hair follicles resulting in loss of eyebrows or eyelashes. In high doses, radiation dermatitis can occur; long-term chronic radioactive irritation can lead to skin cancer induced by radiation or necrotizing skin ulcers. Damage to the lacrimal gland often results in less tear secretion, leading to symptoms of "dry eye." Conjunctival damage is characterized by conjunctivitis, conjunctival edema, and, in severe cases, conjunctival necrosis, eventually leading to atretoblepharon or conjunctival xerosis. Corneal injuries are tarnished in minor cases and result in varying degrees of keratitis in severe cases, which can lead to corneal necrosis and perforation, with the irritative symptoms of photophobia and tearing. There is no obvious pain because of corneal hypoaffectivity. The iridociliary can cause acute iridocyclitis at high doses of exposure. X-ray damage can induce the sudden onset of incurable glaucoma.

Treatment and prevention: The damage to the skin, conjunctiva, cornea, etc. is supportively and symptomatically treated to prevent secondary infections. The ionizing radiation cataract can be treated in accordance with the treatment principles of other types of cataracts. For ionizing radiation-induced retinal damage, vitamins, energy mixtures, vasodilators, and hormones can be used.

For prevention, lead shields and safety goggles of different thicknesses can be used according to the nature and energy of different radiation sources. PMMA glasses can screen X-rays, and lead glasses protect β-rays and γ-rays.

4. *Laser-induced damage*. The effects of lasers on living organisms are categorized by photochemical, thermal, electromagnetic, and mechanical effects (including shock waves). The most important of these is the thermal effect. Clinical manifestations are: (a) corneal injury: coagulative burns; (b) lens injury: mainly thermal injury, clouding the posterior subcapsular cortex of the lens, thus forming cataracts; (c) vitreous injury: making the vitreous body turbid; (d) retinal injury: facing up to the laser beam, it can cause severe damage to the retina and choroid of the macula, with significant diminution of central vision, and even blindness. The fundus manifestation is similar to that of solar retinopathy, and macular edema is obvious, accompanied by hemorrhage.

   Medication. The principles of early treatment are to stop bleeding, inhibit inflammatory reactions, use antioxidants and free radical scavengers, and increase ocular tissue nutrition. In the later stage, it is mainly to promote absorption of bleeding and exudation as well as tissue repair. In the later stage, it is mainly to promote absorption of bleeding and exudation as well as tissue repair.

   (a) Hemostasis: Carbazochrome and oral Yunnan Baiyao can prevent excessive blood from entering the vitreous body. To help the absorption of retinal and vitreous hemorrhage, 1.5 mg iodized lecithin can be taken orally, 1 tablet each time, 3 times a day.
   (b) Inhibition of inflammatory response: It is generally believed that glucocorticoid therapy for laser-induced fundus injury can inhibit the inflammatory response, reduce leakage and retinal edema, increase blood flow, lessen ischemia, and prevent subretinal neovascularization.
   (c) Vitamins and energy mixtures: They help repair laser-induced damage.
   (d) Antioxidants and free radical scavengers: The commonly used desferrioxamine, dimethyl thiourea, vitamin E, vitamin C, carotene, traditional Chinese medicines including ginseng, gallnut, panax notoginseng, angelica, rooted salvia, etc., also have the same effects.

5. *Microwave-induced damage*. The microwave frequency is between 3000 and 3 million MHz. The higher the frequency, the weaker the tissue penetration, but the greater the energy absorbed by the tissue, which can cause burns to the eyelid, conjunctiva, and cornea; the microwave with a low frequency has strong penetrating power and a small absorbing capacity, mainly resulting in lens opacity. Microwave-induced cataracts are treated the same as other types of cataracts. Microwave operators need to wear safety goggles and undergo regular health checkups for early detection, prevention, and treatment.

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Heart Blast Injury

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Heart blast injury is divided into primary and secondary ones. Primary heart blast injury refers to injury caused by overpressure; secondary heart blast injury refers to heart trauma caused by dynamic pressure such as throwing, impact, etc., including the injury to pericardium, myocardium (papillary muscle), valves, and endocardium directly caused by the high pressure of the lung–heart tissue interface (air-/non-air-filled tissue), and coronary air embolism caused by alveolar pulmonary venous fistula; meanwhile, cardiac tamponade and reflex vagus nerve stimulation induced by active hemorrhage in the pericardial cavity, and hypoxemia and malignant arrhythmia resulting from combined lung injury, could further aggravate myocardial injury, worsen the circulatory system, and even lead to death. In addition, the combined blast injury of heart and other parts (such as pulmonary contusion, head and abdomen injury, pelvic or long bone fracture, etc.) is always fatal with a high incidence.

1 Diagnosis of Heart Blast Injury

1.1 Clinical Manifestations

The clinical manifestations of heart blast injury vary greatly, ranging from no symptoms, no ECG changes to cardiogenic shock, and even death. Besides, lack of standardized diagnostic criteria results in its unclear incidence. Therefore, heart blast injury should be suspected for all the patients with a history of blast injury and chest trauma and changes in heart function that cannot be explained by common causes. Compared with adults, children with blast injury may present milder clinical symptoms and signs.

All patients with a history of impact injury should undergo a careful physical examination, especially focusing on assessing vital signs, and related signs of head, neck, and heart and lungs. Rapid deterioration may occur when accompanied symptoms occur, including low blood pressure, engorged jugular vein, chest pain, distant heart sounds, and heart murmurs. Critically ill patients may experience consciousness disturbances, bradycardia, and hypotension.

1.2 Auxiliary Examination

At present, there is no high-specificity and high-sensitivity criteria to diagnose heart blast injury. ECG, chest X-ray imaging, serum biochemical markers of myocardial injury, ultrasonic cardiogram (UCG), and myocardial perfusion imaging (MPI) can be used to exclude heart blast injury and evaluate the injury condition.

1. *ECG*. A 12-lead electrocardiogram examination is required for any patient who is suspected of heart blast injury. Most of the ECG changes usually appear within 24 h after injury. Heart blast injury may cause multiple transient ECG changes or various complex malignant arrhythmias caused by cardiovascular dysfunction, where sinus tachycardia and nonspecific ST segment elevation are most common (occurring in 35–80% of patients), and others include transient supraventricular tachycardia, ventricular premature beats, atrial fibrillation, ventricular tachycardia, ventricular fibrillation, and degree II or III atrioventricular block. In most patients, the right ventricle close to the back of the breastbone is the main involved part of heart blast injury, so that there may be mild ST segment changes in the precordial leads. A past history of cardiovascular diseases, pulmonary contusion, post-traumatic acid–base electrolyte disturbance may affect the interpretation of electrocardiogram. Although ECGs of patients with heart blast injury are rarely normal, normal ECG cannot completely rule out complications related to heart blast injury.

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2. *Chest X-ray imaging*. Chest X-ray imaging is used to evaluate the comorbidities of chest trauma, including rib fracture, hemopneumothorax, and mediastinal widening. Signs of combined chest trauma could be found on the chest X-ray scan in about 60% of patients with heart blast injury. For patients with unstable hemodynamics such as hypotension or heart failure, emergent bedside chest X-ray should be performed for rapid diagnosis and immediate management.
3. *Echocardiography*. Two-dimensional echocardiography can be used to observe the changes in the structure and function of the heart directly. Therein, transthoracic echocardiography is a simple, fast, and practical noninvasive examination for diagnosis of myocardial injury and assessment of its severity, which can clearly show abnormalities in the cardiac structure and function, including ventricular wall motion abnormalities, intramyocardial hematoma, cardiac chamber dilation, abnormal valve shape and movement, myocardial rupture or cardiac tamponade, etc. However, transthoracic echocardiography may be limited in patients with PEEP mechanical ventilation, mediastinal emphysema or extensive subcutaneous emphysema, and severe chest wall injury. When used to diagnose myocardial injury and its complications, transesophageal echocardiography, as an ideal examination method in evaluating myocardial injury, can fully observe the changes in heart with clear images and high sensitivity, which has overcome the limitations of transthoracic echocardiography, so as to be suitable for various critical situations. On the other hand, the real-time three-dimensional imaging can display the instantaneous dynamic cardiac anatomy and hemodynamic status, abnormal motion of a certain segment of the ventricular wall, the stereoscopic range and orientation of the valve regurgitation branch, the morphology and position of the chordae and papillary muscles, etc., providing strong evidence to further diagnosis and therapeutic schedules of heart diseases in clinical practice.

   In recent years, the focused assessment with sonography in trauma, or FAST, an important bedside ultrasound assessment for severe trauma, has showed its great clinical application value in accelerating the process of trauma treatment and avoiding delays, especially in the large-scale on-site triage under field conditions, with high sensitivity to and specificity for free blood accumulation in the pericardial, thoracic, and abdominal cavity, as well as high specificity for the injury of the abdominal solid organs. However, due to the relatively low incidence of hemopericardium in blunt traumas, and the limited value of FAST in evaluating the structure of heart chambers, the research of FAST on heart blast injury is still in the accumulation phase.

   In spite of the great value of CT and MRI in diagnosis, especially in the assessment of coronary artery injury and large blood vessel injury, they may delay the rescue time for critically wounded patients due to transferring and examination. They can be considered for application, if the patient's condition is stable and diagnosis cannot be confirmed by above routine examinations.
4. *Myocardial perfusion imaging (MPI)*. MPI-related technologies have been making progress, including radionuclide ventriculography, multiple-gated acquisition (MUGA) scan, positron emission imaging (PET), ^{99m} Tc pyrophosphate hot spot myocardial imaging, etc., but they are of limited value in diagnosis of blunt myocardial injury, and, besides, their high price, long examination time, and complex procedure limit their application in clinic.

1.3 Laboratory Examination

Serum enzymes have been used to diagnose myocardial injury for a long time, but their specificity is not high. Serum creatinine phosphate kinase (CPK), serum glutamic-oxaloacetic transaminase (SGOT), and lactate dehydrogenase (LDH) all increase significantly after injury, but these increases are nonspecific, which can also occur in injury of other organs, such as liver, kidney, brain, and skeletal muscles. The increase in CPK-MB isoenzyme (CPK-MB) is regarded as a specific and sensitive indicator of cardiomyocyte damage, which usually reaches its peak at 6–24 h after myocardial injury and gradually returns to normal at 72 h. CPK or CPK-MB must be measured every 8 h within the first 24 h or 48 h after hospitalization. If CPK-MB/CPK $\geq$ 5%, myocardial injury should be highly suspected. But it should be noted that CPK-MB could also be released due to other muscular injuries with the cross-reaction rate of 20%, so false-negative and false-positive reactions are likely to occur. The LDH assay has certain value in the diagnosis of myocardial contusion. However, LDH and its isoenzymes can also be increased in the case of kidney diseases and hemolysis, while LDH does not increase in skeletal muscle injury.

Cardiac troponin (cTn) concentration is a specific indicator of myocardial injury. It has been found that cTn includes three types, that is, cTnT, cTnI, and cTnC, and the first two are mainly used in clinical practice. Increased serum cTnT and cTnI concentrations, both myocardium-specific antigens, are specific signs of myocardial injury. Compared with other indicators to diagnose myocardial injury at present, cTn has the advantages of early occurrence in blood, high sensitivity, high specificity, and long duration, so it is the most sensitive and specific indicator for the diagnosis of cardiomyocyte injury currently. It is generally believed that the

serum cTnT concentration of normal adults is (0.18 ± 0.1) μg/L, and the normal range of cTnI is <301 μg/L. The cTnI assay is applied more commonly to current clinical practice.

2 Special Manifestations of Heart Blast Injury

2.1 Pericardium Injury

Pericardium injury refers to the rupture and hemorrhage of the visceral and/or parietal layer of the pericardium caused by violence. Blood accumulation in the pericardial cavity is called hemopericardium. The blood accumulated in the pericardial cavity rapidly increases, forming tension, which affects venous return and causes circulatory dysfunction, resulting in acute cardiac tamponade. When crack in the pericardial wall is so large that a part of heart may herniate out of the chamber, or be incarcerated in the pericardial crack, it may result in dislocation or incarceration of heart, leading to severe heart dysfunction.

2.2 Coronary Artery Injury

Complications of coronary artery injury can be roughly divided into three types: (a) coronary artery thrombosis and occlusion, (b) coronary artery rupture, and (c) coronary fistula. Therein, the causes of coronary thrombosis and occlusion are more complicated, possibly related to the increased blood coagulation (prethrombotic state) on the basis of trauma; or the torn-off in-situ atherosclerotic and calcific plaques in the coronary artery by external force may cause the coronary artery blockage, or local coronary artery spasm; or coronary artery stenosis caused by compression by hematoma in adjacent myocardial contusion tissues results in secondary thrombosis. The coronary artery rupture or fistula is always caused by relatively great violence, which is often accompanied by heart rupture and/or ventricular septal perforation.

2.3 Heart Rupture

Heart ruptures are more common to see in the free walls of the ventricles and atria, where right atrial ruptures can also occur at the entrances of the superior and inferior vena cava, relatively fixed locations. In the case of acute heart rupture, because the blood rapidly flows into the pericardial cavity through the heart crack, the patient may die due to acute cardiac tamponade; meanwhile, if the pericardium is torn so that the blood enters the pleural cavity, the patient may die from hemorrhagic shock. The incidence of heart rupture accompanied with pericardial tear is 10–33%. A torn pericardium can temporarily relieve the symptoms of cardiac tamponade and prolong the patient's survival time. In addition, when the blood clot of the heart crack falls off, or the myocardial contusion softening lesion is necrosed and perforated, leading to the secondary heart rupture, which develops rapidly with poor prognosis, those patients with left ventricular rupture may die within several minutes, and those with right ventricular rupture may die within 30 min. When a blood clot temporarily blocks the heart perforation, the patient can survive for a longer period of time, thereby obtaining an opportunity of diagnosis and treatment.

The following conditions may indicate a heart rupture: (a) mismatch of the clinical manifestations of severe hypotension and hypovolemia and the severity of trauma; (b) no response to blood transfusion and fluid infusion, i.e., no increase of the blood pressure, and no improvement in the condition; (c) no alleviation of any hemothorax signs, in spite of the placement of an intrapleural tube drainage which has drained a large amount of accumulated blood; (d) no improvement in metabolic acidosis despite adequate fluid replacement; (e) hypotension with elevated central venous pressure or engorged jugular vein.

2.4 Ventricular Septal Rupture

A closed chest trauma can cause ventricular septal perforation, which is usually single, often located in the muscular septum near the apex. Acute ventricular septal rupture, mostly linear, is mainly caused by the shear and torsion induced by trauma, as well as the sudden increase in intraventricular pressure; delayed rupture is caused by contusion or infarction of the ventricular septal muscle, which appears mostly in 1–2 weeks after the injury with an irregular shape, and it can be multiple ruptures.

Such injury is often accompanied with myocardial contusion, tricuspid regurgitation, atrial septal defect, thoracic aortic tear, etc. Similar to congenital ventricular septal defect, a significant shunt can occur immediately from the left ventricle to the right ventricle. However, the difference lies in that the ventricular septal defect mentioned could rapidly develop to the right heart failure because a large amount of blood is accumulated suddenly in the right ventricle through the left-to-right shunt, leading to signs of hepatomegaly, edema in lower extremities, and pulmonary congestion. In severe cases, the condition may deteriorate quickly even to death. If extensive ventricular contusion is accompanied, especially with injury of the intracardiac structure, hemodynamic disturbance will be more serious, and hypotension and cardiogenic shock will occur earlier with a higher mortality rate.

2.5 Heart Valve Injury

Simple heart valve injury is relatively rare in blast injury, and the injury rate of each valve is in order of aortic valves, mitral valves, and tricuspid valves. Semilunar valve avulsion often damages the stent tissue such as the valve leaflet junction; the atrioventricular valve injury often leads to rupture of the papillary muscles or chordae, causing acute valve insufficiency and regurgitation.

Aortic valve injury should be considered, when signs of aortic valve insufficiency are noted after the blast injury such as diastolic murmurs audible at the left sternal border or the aortic valve area. If systolic murmurs with a blowing character are audible in the precordial or apical area, and accompanied by clinical manifestations of atrioventricular valve insufficiency, the corresponding atrioventricular valve injury should be considered, and further examinations are required for such patients. Two-dimensional echocardiography and color Doppler ultrasonography are specific noninvasive methods to diagnose heart valve injury, which could indicate the location, extent, and regurgitation of the injured valve according to its structure and reflux condition.

2.6 Great Vessel Injury

Thoracic aorta injury is mainly located in the aortic isthmus distally underneath the left subclavian; other less common locations include the aortic arch, the proximal ascending aorta, and the descending aorta at the diaphragm level. It occurs in relation to the fixed position of the aortic isthmus, fragile structure, and compression by bony structure (sternum and thoracic spine before and after the aorta) caused by blast injury.

When combined with other chest injuries, aortic injury is often overlooked. Routine chest X-ray images may show the thoracic aorta injury, such as widening mediastinum, blurred aortic knobs, displaced trachea and left and right main bronchi, and a large amount of fluid accumulated in the left pleural cavity. Therefore, when a patient is suspected of thoracic aortic injury based on the chest injury mechanism, combined injuries, chest radiography, etc., further imaging examinations are needed, including chest CT, transesophageal ultrasonography, etc., to confirm or exclude the diagnosis. Emergency CT angiography (CTA) of large vessels can clarify the type and range of the injured large vessel, providing a basis for follow-up diagnosis and treatment.

3 Treatment of Heart Blast Injury

3.1 Treatment Principles

Nonsurgical treatment is mainly adopted for patients with general heart blast injury. For patients without hemodynamic changes, only analgesia, bed rest, and ECG monitoring are required. It is important to predict and early identify the "high-risk" wounded personnel who will develop complications of myocardial contusion through continuous dynamic monitoring of vital signs and clinical manifestations, so as to prevent and manage possible life-threatening complications and sequelae.

3.2 Therapies

For patients with suspected myocardial injury, management includes admission for observation of 24 h, full bed rest, and examinations of electrocardiography, serum enzymes, and echocardiography. If auxiliary examinations are normal with stable hemodynamics, the monitoring will be removed after 24 h. Only those of chest trauma with suspicious positive results of electrocardiography, echocardiography, or serum enzyme tests need to be admitted to the intensive care unit (ICU) for further monitoring and treatment. For patients with clinical manifestations of low cardiac output or low blood pressure, positive inotropic drugs should be given routinely, and if necessary, central venous pressure should be monitored, or pulmonary capillary wedge pressure should be monitored through Swan-Ganz catheter, and the blood volume should be increased appropriately to avoid fluid overload, and when heart failure occurs, cardiotonic and diuretic drugs should be administered. Enough attention should be paid to observe and treat complications. Patients with arrhythmia, cardiac tamponade after pericardial laceration, coronary artery injury, heart rupture, intracardiac structure injury, and other complications which may not appear until 48-72 h or longer after the injury, need close observation and corresponding treatment in time.

1. *Pericardial laceration*. When acute cardiac tamponade is suspected, pericardiocentesis should be performed, which, with the blood drained, could not only improve the hemodynamic disorder of the wounded but also further confirm the diagnosis. Continuous new signs of hemorrhage during or after pericardiocentesis may indicate heart or great vessel injury, which is an indication for early surgical exploration.
2. *Coronary artery injury*. Generally, there are no specific clinical symptoms or signs of coronary artery injury, which, besides, are often covered by severe myocardial contusion. For patients without a past history of heart diseases, if continuous heart murmurs are audible and/or signs of myocardial infarction found on ECG after the injury, coronary artery injury is indicated. For patients highly suspected of coronary artery injury or coronary thrombosis, selective coronary angiography can be performed as soon as possible. For patients with unstable circulation, emergency exploratory thoracotomy should be performed to repair myocardial laceration and manage coronary artery injury at the same time as aggressively

managing acute cardiac tamponade or rescuing hemorrhagic shock.

3. *Heart rupture*. When the heart rupture is highly suspected, it is not advisable to do any more unhelpful examinations. Instead, emergency surgical exploration should be performed without hesitation, during which the surgeons could make the final diagnosis and differential diagnosis.

   For those with suspected cardiac rupture or manifestations of cardiac tamponade after examinations, pericardiocentesis or exploratory thoracotomy should be performed immediately to avoid losing the opportunity for surgery. During the preparation for the surgery, pericardiocentesis or subxiphoid pericardiotomy should be performed, because for those critical patients, sometimes, cardiac compression can be relieved temporarily and the patient's tolerance to surgery can be enhanced by even draining 20–30 ml of blood from the pericardial cavity. Meanwhile, anti-shock treatment should be performed immediately, including venous and arterial infusion channel establishment, blood transfusion, fluid infusion, and close monitoring of arterial pressure and central venous pressure. Blood sources should be prepared sufficiently with preparations for autologous blood transfusion. With supine position and median sternum incision, the four heart chambers and the ascending aorta can be well exposed, and the incision can be extended down to the upper abdomen when abdominal exploration is required.
4. *Ventricular septal rupture*. For those with mild ventricular septal rupture, small shunt, no significant clinical symptoms, and stable circulation, medication should be considered first. The disadvantages of early emergency surgery include that: (a) such patients often suffer from injuries to other parts as well as myocardial contusion with low cardiac output and arrhythmia; (b) tissues around the ventricular septal rupture are difficult to repair, which are so fragile with severe hemorrhage and edema, so that residual shunt often occurs after the operation. The ventricular septal defect can be repaired in about 3 months when there is no sign of spontaneous closure of the crack during medication. For patients with persistent or progressive heart dysfunction and pulmonary hypertension, surgical repair should be performed as soon as possible within 2 weeks after the injury. If the acute traumatic reaction is improved during this period, safety of surgery can be enhanced.

   A comprehensive examination must be performed before surgery to prevent missed diagnosis and misdiagnosis and to manage combined injuries appropriately. For those with acute or progressive heart failure in the early post-injury period, hemodynamic indicators should be monitored through Swan-Ganz catheter, and cardiotonic and diuretic therapy should be strengthened. For the wounded with low cardiac output or cardiogenic shock, dopamine inotropic drugs, vasodilators, or intra-aortic balloon pump should be applied before surgery to maintain the blood pressure and improve the cardiac function, so as to keep coronary arteries and systemic tissues perfused satisfactorily.

   Cardiopulmonary bypass and potassium-containing cardioplegia should be applied to induce cardiac arrest during the operation. The ventricular septal defect can be repaired selectively as per conventional methods. Emergency ventricular septal defect repair is often performed after trauma, when myocardial contusion still exists with a relatively large breach located at the ventricular septal muscle adjacent to the apex. A small incision parallel to the left anterior descending branch in the apical region with few vessels of the left ventricular anterior wall can be the first choice for ventricular septal defect repair, through which the location and size of the ventricular septum rupture can be explored through the left ventricular surface to assess the range and severity of the myocardial contusion and then to determine the repair method. At present, in order to avoid the difficulty in repairing, some have adopted interventional ventricular septal defect closure or occluder implantation under direct vision (if other intracardiac surgery is required simultaneously), while its efficacy needs to be further evaluated.
5. *Heart valve injury*. For patients with no severe valve injury, small or mild to moderate regurgitation, and relatively stable conditions after medication, traumatic response can be waited first, including re-evaluation after the recovery of myocardial contusion. For the wounded with severe valve insufficiency, progressive heart dysfunction immediately after injury, and no significant effects after medication, surgery should be performed as soon as possible or emergently.

   Since the hemodynamic change of heart valve injury is the severe overload of the left or right heart caused by valve orifice regurgitation, so that heart dysfunction and/or heart failure will occur almost sooner or later, it is necessary to treat such patients aggressively and improve their heart function, including proper administration of inotropic drugs and vasodilators, maintaining stable circulation, and seizing the opportunity for emergency surgery. Preoperative preparation for selective surgery can refer to general heart valve surgery.

   For aortic valve injury, most patients need valve replacement, while commissural annuloplasty can be tried only for a small number of patients with valve junction avulsion; if the result of the latter operation is not satisfactory, the aortic valve replacement should be performed immediately. For mitral and tricuspid valve injury, the specific operation method, whether valve replacement or valvuloplasty, should be determined during the surgical exploration. In general cases, valve repair or plasty should be considered, which should be changed to valve replacement surgery immediately for those with severe valve injury, especially in the mitral valve.

6. *Great vessel injury*. The initial management of blunt aortic injury includes establishment of two large-caliber peripheral venous channels for medication and fluid resuscitation, respectively. For patients with hypertension, the systolic blood pressure should be controlled at approximately 100 mmHg, and the target heart rate should be maintained at 100 bpm. Based on the principles of advanced traumatic life support, the wounded with unstable hemodynamics should undergo emergency surgical exploration in an operating room to determine the possible bleeding source in the chest and/or abdomen. When hemodynamic instability is caused by closed thoracic aortic injury, it should be managed immediately.

It was believed previously that if the aortic injury is not life-threatening, delayed repair of the aorta may be more beneficial (damage control, DC). For hematoma in the aorta wall, instead of repair surgery of the aorta, conservative treatment is recommended, including active medication management, systolic blood pressure control of approximately 100 mmHg, and the target heart rate maintaining of 100 bpm. Meanwhile, a series of imaging evaluations are needed. For acute aortic dissection or aortic pseudoaneurysm, if the risk of surgery is low, the repair surgery is recommended. For patient with severe coexisting injuries but relatively stable hemodynamics, the surgery can be delayed appropriately. During the waiting, blood pressure and heart rate should be actively controlled. However, with the advancement of aortic endovascular treatment technology and the development of material technology, the management of aortic diseases becomes more minimally invasive and diversified, which makes the surgical timing of aortic injury earlier and flexible, greatly improving the patient's prognosis.

3.3 Diagnosis and Treatment Process

See Fig. 1.

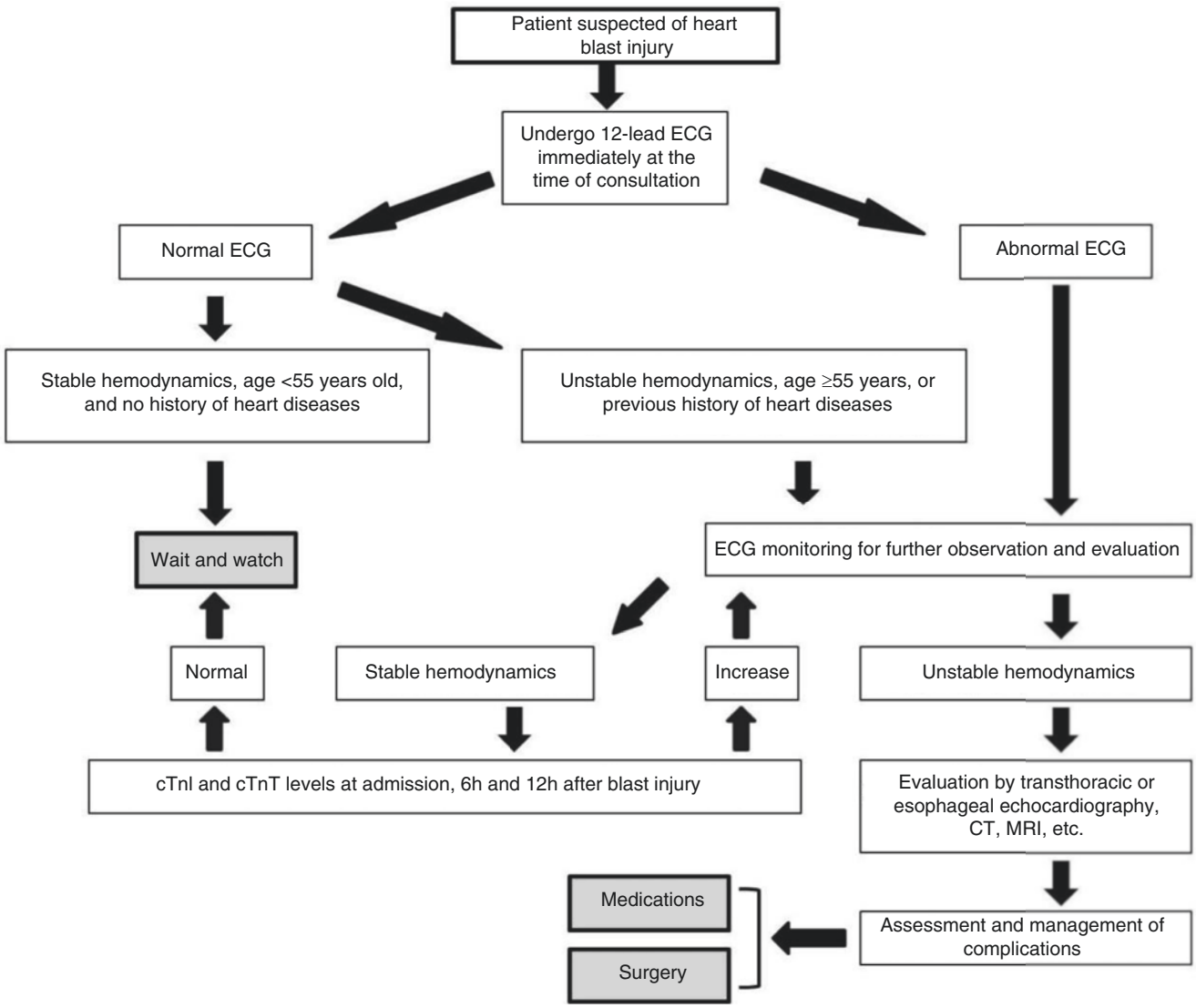

Fig. 1 Diagnosis and treatment process

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Abdominal Blast Injury

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1 Overview

Blast injury is a variety of injuries caused by the release of energy after the blast wave formed by detonation acts on the human body, which is mainly caused by shock wave overpressure and negative pressure, that is, primary injury or simple blast injury. The generalized blast injury also includes the injury caused by the direct action of the shock wave on the human body or the throwing and displacement of the human body, and the injury caused by the secondary projectile formed by the shock wave hitting the human body or the collapse of the building hitting the human body. Abdominal blast injury refers to all kinds of injuries caused by blast wave on human abdomen, often compound injury. Due to the large abdominal surface area and large number of organs, the incidence of abdominal injury caused by shock wave is high, the injury condition is complex, and most of them are closed abdominal injury, with strong concealment and high missed diagnosis rate, which greatly affects the treatment efficiency of the wounded.

1.1 Classification

Abdominal blast injury can be divided into open injury and closed injury according to the presence or absence of body surface wound. Closed injuries are mostly caused by overpressure, negative pressure, and other primary injuries due to shock waves, while open injuries are mostly caused by secondary impacts due to shock waves.

According to the injured organs, they can be divided into solid organ injury and hollow organ injury. Among the abdominal blast injuries caused by air blast wave, liver and spleen and other solid organ injuries are the most common. In case of underwater explosion, air-filled organs, such as intestines, are more likely to be damaged.

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1.2 Pathogenesis

A large number of studies have shown that the possible mechanism of shock wave–induced abdominal blast injury is tissue deformation and displacement caused by overpressure. After the explosion, the overpressure shock wave passes through the peritoneum and produces high-speed and low-frequency stress wave (transverse wave) and low-speed and high-frequency shear wave (longitudinal wave). Stress wave mainly causes the change of micro level, while shear wave causes the movement of the whole abdominal wall and viscera. Due to the different densities of various organs, tissues, and abdominal wall, the moving acceleration caused by shear wave is also inconsistent, resulting in dissociative damage to abdominal organs and vascular structure attached tissues. The small intestine and cecum with long mesentery in the gastrointestinal tract are more likely to be damaged by traction effect or inertial impact on the posterior abdominal wall, also known as inertial effect. Stress wave mainly causes tissue injury through fragmentation effect and implosion effect. The fragmentation effect is common in tissue junctions with different densities and gas–liquid interfaces. At these junctions, stress waves are partially reflected, resulting in damage to the surface and edge of dense tissue due to the sudden increase of local pressure. The implosion effect refers to that under the action of stress wave, the gas in the tissue is compressed and the local pressure increases. After the impact of overpressure, other compressed gases expand rapidly and release more energy to the surrounding in a radial manner. Each compressed air mass is equivalent to a small explosion source, and the implosion causes damage to the surrounding tissue.

1.3 Diagnosis

By asking the wounded, we can learn more about the environment at the time of injury, the category of explosives and the specific state at the time of explosion, which is helpful to

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fully and comprehensively understand the injury. Physical examination, especially comprehensive and dynamic observation of signs, is helpful to judge the development of injury and adjust the treatment plan in time. The items of auxiliary examination shall be determined according to the injury condition of the wounded and the stability of hemodynamics and shall not be omitted as far as possible.

1. *Symptoms and signs*. Abdominal blast injury mainly causes intraperitoneal hemorrhage and peritonitis. Bleeding mainly occurs when the solid organs rupture, especially the rupture of the liver and spleen and injuries of blood vessels. Peritonitis is caused by the rupture of hollow organs with gastrointestinal contents overflowing into the peritoneal cavity.

   Due to different injury sites and conditions, there are the following manifestations:

   (a) Abdominal pain. It is the most common symptom. It begins at the injury site and then spreads to the whole abdomen. The perforation of the stomach, upper intestine, and gallbladder often causes diffuse acute pain, while the pain caused by colon perforation is mild and limited but likely to cause septic shock.
   (b) Nausea and vomiting. Nearly half of the wounded with abdominal blast injury have nausea and vomiting after injury, ranging from short to continuous.
   (c) Shock. Shock can occur due to massive bleeding in the abdominal cavity or severe diffuse peritonitis.
   (d) Peritoneal irritation signs. The wounded with visceral rupture may have abdominal irritation signs such as tenderness, rebound pain, and abdominal muscle tension.
   (e) Others. Hematuria can occur when kidney and bladder are damaged. Dark purple or bloody tarry stool may appear when intestinal mucosa is damaged or bowels are perforated. Fresh blood flowing from the anus indicates injuries in the colon or rectum. In case of perforation of stomach and intestine, there may be accumulation of gas under the diaphragm, disappearance of pneumoperitoneum and liver dullness boundary, disappearance of intestinal ringing, fever, and pulse frequency. Pelvic organ injury can stimulate the rectum causing frequent defecation desires. Many patients with abdominal blast injury caused by underwater explosion are complicated with temporary lower limb paresis, which may be caused by small blood vessel injury in the spinal cord.

   It is worth noting that some wounded have used painkillers during evacuation, and their symptoms and signs may not be obvious. In addition, multiple injuries need to be noted, because the symptoms of abdominal injury are often obviously covered up by the symptoms of injuries in other parts. For example, the unconscious wounded due to craniocerebral injury cannot provide the conscious symptoms of abdomen; combined thoracoabdominal injury can focus on the chest and ignore the examination of the abdomen due to chest wound and dyspnea; limb long bone injuries and fractures often mask abdominal injuries.

2. *Auxiliary Examinations*.

   (a) X-ray and CT examination. Abdominal X-ray examination can determine whether there is gastrointestinal perforation, but its application is relatively limited. When renal and ureteral injury is suspected, intravenous pyelography can be used. At present, it is considered that the diagnostic rate of multi-slice spiral CT in most abdominal injuries is higher than that of abdominal X-ray, and its application in the diagnosis of abdominal blast injury has attracted more and more attention.
   (b) Diagnostic abdominal puncture. It can be performed when closed abdominal organ injury is suspected. If bloody liquid is extracted, it is positive. This method is simple and rapid. For closed trauma, the positive rate can reach 83–97.7%.
   (c) Diagnostic peritoneal lavage. When there is less blood or exudate in the abdominal cavity, abdominal puncture is often negative. At this time, lavage can be used. If the lavage solution is light red or the microscopic red blood cell count is $>0.1 \times 10^{12}$/L, or white blood cell count is $>0.5 \times 10^{9}$/L, or if bacteria, bile, vegetable fiber, or fecal residue is noted in the lavage solution, it is positive. The accuracy of this method can reach 97%.
   (d) Ultrasonography. Ultrasonography is simple, rapid, and noninvasive. It can be carried out at the bedside or repeated dynamic observation. It has great diagnostic value for the injury of liver, spleen, kidney, pancreas, and other substantive organs, the existence of retroperitoneal hematoma and abdominal fluid.
   (e) Laparoscopy. Laparoscopy is not only an examination technology but also some therapeutic operations, but its application is relatively limited. Due to the lack of touch, there are deficiencies in fully exploring abdominal organs.
   (f) Catheterization examination. If the exported urine is clear and free of blood, it indicates that there is no damage to the bladder; if there is a large amount of hematuria, it indicates that there is damage to the bladder, ureter, or kidney; if there is no urine or only a small amount of hematuria, 50–100 ml sterile isotonic fluid can be injected into the catheter and sucked out after a few minutes. If the sucked out amount is significantly less than the injected amount or with blood, it indicates that there is bladder rupture.

(g) Laboratory examinations. Blood routine test, liver and kidney function assay, arterial blood gas analysis, urine and gastric juice analysis are of great significance to judge the local and systemic function of the wounded.

1.4 Treatment Principles

1. Treatment Principles. First deal with the injuries that pose the greatest threat to life, such as to keep the respiratory tract unobstructed, control obvious external bleeding, etc. Not all the wounded with abdominal blast injury need surgical treatment. For the wounded with nonsurgical treatment, continue to observe for 1–2 weeks and pay attention to the risk of delayed perforation of digestive tract. For the wounded that have not been diagnosed or are in the observation period and cannot be determined whether to operate, they need fasting and water deprivation, and cannot be given analgesic treatment at the same time. For the wounded who have been diagnosed and need to wait for surgery, analgesia can be used to relieve the pain of the wounded. The choice of operation time should be determined according to the injury of the wounded, especially for those complicated with other organ injuries, it should be decided whether to operate by stages or at the same time. Abdominal exploration should take the rectus abdominis incision or median incision. It must be fully explored to avoid missed diagnosis. Especially for some gastrointestinal myometrial hematomas and perforation of mesangial margin, repeated and careful examination should be carried out. For ballistic injuries, we cannot just be satisfied with finding a wound. The operation should be rapid, safe, accurate, without omitting visceral injury, and adequate drainage should be provided after operation.
2. Treatment Procedure.
   (a) Make a quick systemic examination to judge whether there is abdominal visceral injury and combined injury in other parts. In case of respiratory and circulatory dysfunction, carry out limited initial resuscitation. If necessary, perform endotracheal intubation, emergency tracheotomy, or chest wall drainage to relieve respiratory tract obstruction and keep the respiratory tract unobstructed. Prevent air embolism during positive pressure ventilation, and then manage the abdominal injury. If necessary, surgery is required at the same time.
   (b) Forbid food and water, place a gastric tube, keep continuous gastrointestinal decompression, and observe the hemorrhage.
   (c) Place the catheter and record the urine volume.
   (d) Supplement blood volume. For those suspected of visceral injury, blood should be drawn quickly for blood group cross test and blood matching. For those with more bleeding and severe shock, two or even three to four infusion channels should be established for simultaneous infusion. Patients in critical condition should undergo exploratory laparotomy while resisting shock.
   (e) Early apply broad-spectrum antibiotics and metronidazole against infection. Those who have not used tetanus toxoid should be injected with tetanus antiserum.
   (f) For all abdominal wall injury, exploratory laparotomy should be prepared at the same time.
   (g) Exploratory laparotomy. For severe abdominal trauma, focus should be paid on early rescue surgery, especially the rescue of hemorrhagic shock. For those with particularly serious conditions, we should race against time to win time and save lives. Early exploratory laparotomy is the key and difficult point in the treatment of critical trauma. We should not unilaterally emphasize the correction of hypotension and delay the rescue time, resulting in the death of the wounded. The operation should be simple, short, and effective, with the aim of repair, hemostasis, drainage, closure of the abdominal cavity, and damage control.

2 Blast Injury of Abdominal Wall

Blast injury of abdominal wall can be divided into closed injury and open injury, which are mainly treated symptomatically.

Closed injuries of abdominal wall, such as hematoma of abdominal wall, are mostly caused by the rupture of blood vessels in abdominal wall muscles caused by blast injury. The treatment method can be cut at the most obvious part of the hematoma, remove the hematoma and necrotic tissue, ligate the obviously damaged blood vessels, suture the broken muscles, build each layer of abdominal wall, and decide whether to place drainage according to the intraoperative situation. After operation, pay attention to abdominal signs and be aware of possible abdominal visceral injury.

For open abdominal wall injury, if it is nonpenetrating injury, debridement can be given, and the wound can be fully drained. The primary suture or secondary suture can be determined according to the injury time. It should be noted that after the injury is treated, such patients should closely observe the changes of abdominal conditions, be alert to the occurrence of delayed perforation and delayed splenic rupture, observe while treating, and make preparations for laparotomy. For penetrating abdominal wall injury, explor-

atory laparotomy should be performed regardless of the acute abdomen signs. The right rectus abdominis incision is generally used to avoid the original wound as far as possible, and the original wound will be debridement after the operation is accepted. The abdominal cavity should be closed as much as possible after operation. For those with large abdominal wall injury, temporary abdominal closure (TAC) can be used to temporarily close the abdominal cavity with the help of skin and artificial materials (such as VSD negative pressure sealing drainage device). After the patient's condition is stable, the second stage of deterministic abdominal wall closure operation could be performed.

3 Hollow Organ Blast Injury

Gastrointestinal (especially intestinal) injury is the main affected organ of impact injury of hollow organs. The main pathophysiological changes are tube wall hematoma, serosal tear, and lumen perforation. There are two main points to note. First, there may be more than one injury; second, the injury may be delayed or aggravated.

3.1 Stomach and Duodenum

3.1.1 Clinical Manifestations

The clinical manifestations of impact injury of stomach and duodenum are different, commonly including abdominal pain, nausea, vomiting, hematemesis, etc. Severe cases may be accompanied by shock.

Physical examination. For simple contusion of gastroduodenal wall, only discomforts occur, such as epigastric tenderness. For patients with gastric and duodenal perforation, epigastric tenderness, rebound pain, and muscle tension can occur due to the strong stimulation of gastric acid, bile, and pancreatic juice.

3.1.2 Diagnosis

In addition to the above trauma history and signs, laboratory examinations are of great significance. Blood routine test shows that the leukocyte count increases. The abdominal X-ray (standing and lying position) can indicate the free gas accumulated below the diaphragm. The diagnostic abdominal puncture can extract the turbid peritoneal effusion. Abdominal CT is of great significance to clarify the perforation of hollow organs and investigate the substantial organ injury.

3.1.3 Treatment

For simple gastric and duodenal wall contusion, fasting, gastrointestinal decompression, and symptomatic treatment can be given. For those suspected of gastric and duodenal wall bleeding, gastrointestinal decompression can be performed. If gastric and duodenal perforation is clear, it can be comprehensively evaluated according to the following conditions. If the vital signs of the wounded are stable, the symptoms are light, the abdominal signs are relatively limited, and the wounded are injured under fasting state, they can receive conservative treatment, active anti-infection, acid inhibition, fasting, water deprivation, and gastrointestinal decompression and closely observe the changes of the condition. If the wounded has a rapid heart rate, fever, severe abdominal signs, and total peritonitis, active surgical exploration should be carried out. During the operation, attention should be paid to fully expose the hidden parts such as gastric fundus, posterior gastric wall, descending segment, and horizontal segment of duodenum, which cannot be satisfied with finding only one lesion. For those with serous membrane rupture, suture the seromuscular layer with silk thread. If there is perforation, it is necessary to remove the inactivated tissue, completely stop bleeding, repair the perforation, flush the abdominal cavity with a large amount of sterilized water, fully drain, and perform gastrointestinal decompression at the same time. Partial gastrectomy is only used for patients with large-area gastric damage, while patients with severe duodenal damage need duodenal end-to-end anastomosis, gastroduodenal anastomosis, or duodenojejunostomy.

3.2 Small Intestine and Mesentery

3.2.1 Clinical Manifestations

The main clinical manifestations of small bowel and mesenteric blast injury are small bowel rupture and mesenteric hematoma. Typical symptoms are abdominal pain, abdominal distension, nausea, vomiting, and shock.

Physical examination. There is a typical peritoneal irritation sign. Early tenderness can be limited to the injured part, and then gradually spread to the whole abdomen. Bowel sounds disappear. Abdominal puncture can extract blood turbidity.

3.2.2 Diagnosis

In addition to the typical history, symptoms and physical examination of impact injury, abdominal CT, and abdominal X-ray are still of great significance in the diagnosis of blast injury of small intestine and mesentery. Especially for mesenteric hematoma, CT has great diagnostic value. It is worth mentioning that if there is no gas accumulated below the diaphragm on abdominal X-ray, it cannot be concluded that there is no possibility of intestinal perforation based on this result alone.

3.2.3 Treatment

Whether small bowel and mesenteric blast injuries need surgery depends on the patient's condition. Simple small bowel wall hematoma or stable mesenteric hematoma can be treated

conservatively. For such patients with acute abdomen or hemorrhagic shock, active surgical exploration and treatment are needed. During the operation, all the intestines and their mesentery should be carefully explored. Multiple injuries of the small intestine are one of the characteristics of small intestinal impact injury, and the range of lesions is often scattered and multiple. The intestinal injury at the mesenteric margin of the small intestine is often hidden and difficult to find. Careful observation must be observed during the examination. For small perforation, intestinal perforation repair can be performed after removing necrotic tissue. When the damage is serious or the bleeding point of mesenteric vessels is difficult to control, intestinal segment resection and anastomosis are required. The scope of small bowel resection should be as close to the wound as possible, and more small intestine should be preserved to avoid the occurrence of short bowel syndrome.

3.3 Colorectal and Anal Canal

3.3.1 Clinical Manifestations

The blast injury of colorectal is more than that of small intestine, and the injury of transverse colon and sigmoid colon is the most common. The main symptoms were peritoneal irritation caused by fecal fistula, severe abdominal pain, nausea and vomiting, and early manifestations of septic shock. The blast injury of rectum and anal canal at a lower position can have the symptoms of anal distension, frequent defecation, anal bleeding, and so on.

Physical examination: Abdominal tenderness, rebound pain, muscle tension, disappearance of bowel sounds, and blood stains on digital rectal examination. At the same time, the patient may be accompanied by high fever, rapid and weak pulses, etc.

3.3.2 Diagnosis

In addition to the history, symptoms, and signs of blast injury, laboratory examinations in auxiliary examinations can indicate the increase of inflammatory indicators. Abdominal X-ray may find free gas accumulated below the diaphragm. Abdominal CT has certain reference value, which can find peritoneal effusion and free gas in abdominal cavity. Diagnostic abdominal puncture can extract purulent turbid fluid or fecal fluid, which is of great significance.

3.3.3 Treatment

Once the blast injury of colorectal and anal canal is clear, surgery should be carried out as soon as possible. The exploration principle is the same as that of small intestinal impact injury. The operation method depends on the intraoperative situation. Most minor injuries can free this segment of colon and pull it out of the abdominal cavity to directly repair the external wound or remove the damaged segment of colon and perform double lumen stoma. For more extensive colorectal injury, intestinal segment resection, distal colonic open placement, and proximal colostomy are considered. For the extensive injury of the right colon, one-stage ileal transverse colon anastomosis can be performed while removing the right colon. Repeated irrigation and adequate drainage are the key points of the whole operation.

4 Solid Organ Blast Injury

4.1 Liver

Hepatic blast injury is the most common injury in abdominal blast. The main pathological changes are subhepatic hemorrhage, hematoma, and rupture, often accompanied by varying degrees of intraperitoneal hemorrhage.

4.1.1 Clinical Manifestations

It is often manifested as acute abdominal pain, which can be accompanied by nausea and vomiting, hematochezia, irritability, and testicular pain. In severe cases, shock may occur, such as increased pulse, pale complexion, cold sweat, thirst, irritability, dizziness, panic, cold limbs, etc. Underwater blast injury can sometimes be accompanied by conduction inductance and transient lower limb paralysis. Physical examination often shows tenderness, rebound pain, and muscle tension, as well as whole or local peritoneal stimulation signs.

4.1.2 Diagnosis

According to the injury condition, the injured organs can be judged by the path of the wound and the injured object; it is difficult to diagnose closed blast injury, especially those with coma. It is necessary to quickly judge whether there is liver blast injury; whether there is intraperitoneal hemorrhage; whether there are combined injuries of other abdominal organs; whether emergency surgery is required. Diagnosis is generally based on the following clinical features.

1. Blast injury history.
2. Above clinical manifestations.
3. Intraperitoneal puncture or diagnostic intraperitoneal lavage. For hepatic blast injury, the positive rate of intraperitoneal puncture can reach up to 89–95%. 0.1 ml fluid without coagulation is of diagnostic value. Peritoneal lavage can be considered when abdominal puncture cannot be used for diagnosis.
4. Ultrasonography is simple, rapid, and noninvasive, which can be carried out at the first aid site and can be observed repeatedly. So it is the first choice for first aid.

5. Abdominal CT also has the advantages of simplicity, rapidity, and noninvasive, with high resolution and clear anatomical relationship. It is a more objective examination than ultrasonography. It is an important method in the diagnosis of hepatic blast injury, but it is not suitable for the wounded with unstable hemodynamics.
6. Selective hepatic arteriography can determine the location and degree of intrahepatic vascular blast injury. The rupture of hepatic vessels showed the overflow of contrast medium; vascular rupture or occlusion can also be manifested as a wedge-shaped filling defect with the tip of the liver parenchyma pointing to the anus; when the liver parenchyma is broken or there is hematoma, filling defects and cracks or vascular compression and displacement can be seen.
7. Hemoglobin decreased gradually when progressive internal bleeding occurred in blood and biochemical examination; in peritonitis, neutrophils increase sharply and the nucleus moves to the left.

It should be emphasized that the main means of clinical diagnosis of liver blast injury are still physical diagnosis and laboratory examination. Severe injury and delayed treatment are two major factors leading to death of liver blast injury.

4.1.3 Treatment

1. Nonsurgical treatment. It is applicable to patients with minor injuries classified by the American Association of Traumatic Surgery (AAST) as grade I to II. Closed blast injury, consciousness, with objective conditions to closely observe the changes of the disease; there was no moderate or severe shock, and the hemodynamics was stable; there was no obvious peritoneal irritation sign; there is no blast injury of other abdominal organs; CT examination shows that the injury is not serious and conservative treatment is feasible.
2. Surgery.

   Surgical indications. Unstable hemodynamics even after 1000 ml blood transfusion; combined blast injury of other organs; peritonitis; hematoma bleeding more than 250 ml; progressive enlargement of hematoma; abscess of infection secondary to hematoma. If the vital signs cannot be maintained by massive blood transfusion and infusion, it is necessary to enter the "green channel."

   Surgical method: according to the site, degree, and pathological type of the blast injury to the liver, different management methods are used: (a) control hepatic blood inflow, and confirm the source of bleeding; (b) debride the site of blast injury, explore the damaged vessels and bile ducts, and give treatment such as suturing, caulking, ligation, and resection; (c) pad the wound with hemostatic materials or pedicled greater omentum; (d) drain the perihepatic area.

4.1.4 Management of Complications

1. Bleeding. Bleeding includes secondary bleeding of liver wound, biliary bleeding, and stress ulcer bleeding. In case of re-bleeding in a short time after operation, the necrotic tissue should be removed again, the bleeding point should be ligated, fully drained, or the wound should be filled with gauze. Biliary bleeding and gastric and duodenal stress ulcer bleeding can be secondary about 10 days after operation. Conservative treatment is ineffective and re-operation is needed.
2. Infection. Infection includes subphrenic abscess, liver abscess, intraperitoneal infection, and incision infection. It is related to improper abdominal drainage, packing gauze, combined with explosive impact injury of gastrointestinal tract, etc.
3. Bile leakage. Bile leakage can heal by itself after full drainage. For large bile duct fistula, the common bile duct can be cut and T-shaped tube can be placed for drainage.

4.2 Biliary Tract

4.2.1 Clinical Manifestations

Blast injury of extrahepatic biliary tract accounts for about 3–5% of explosive blast injury of intra-abdominal organs. Simple extrahepatic blast injury of biliary tract is rare, and its symptoms are often covered by blast injury of other organs, mainly manifested as abdominal pain, abdominal distention, jaundice, etc., a large amount of bile and pancreatic juice leak out or occur simultaneously with bleeding.

4.2.2 Diagnosis

1. There is a clear history of blast injury, and the injury site is the right chest or abdomen.
2. The clinical manifestations are in line with the above, and some are diagnosed only after jaundice, peritoneal effusion, and clay stool in the later stage.
3. Intraperitoneal puncture and peritoneal lavage. The operation is the same as that of blast injury of liver. If bile-like fluid is extracted, it can be diagnosed.
4. B-mode ultrasound. If the damage is small, it is easy to miss diagnosis.
5. Radiology examinations: Abdominal X-ray sometimes shows gas accumulated below the diaphragm. CT, ERCP, PTC, and MRCP can find the integrity destruction of biliary tract, or stenosis or even occlusion, which is helpful for clinical diagnosis.

4.2.3 Treatment

1. In the early stage of cholecystectomy, simple rupture of gallbladder with small fissure and neat edge can be repaired and sutured with double layers.

2. Cholecystostomy is mostly used for minor laceration of gallbladder, and the general condition is more critical. On the basis of gallbladder repair, cholecystostomy is performed at the bottom to drain bile.
3. Cholecystectomy is suitable for all types of blast injury of gallbladder.

All the above procedures require indwelling drainage tube.

4.2.4 Postoperative Complications

1. Anastomotic hemorrhage. It is mostly caused by incomplete hemostasis after intestinal wall incision at the anastomotic site. Hemostatic drugs can be used appropriately. If there is more bleeding, it is necessary to operate again to stop bleeding.
2. Anastomotic leakage. It is rare, which is often caused by infection caused by incomplete debridement of the injury caused by bile duct explosion and large tension at the anastomotic site. Small fistula can be healed by double cannula drainage; in case of gallbladder wall or common bile duct duodenal anastomotic fistula with excessive bile and intestinal fluid, fasting, somatostatin, and double cannula negative pressure drainage should be used.
3. Anastomotic stenosis. It is the most common, mostly within a few months or 2 years after operation. It is characterized by abdominal pain, jaundice, bile duct stones and recurrent cholangitis, and even biliary cirrhosis in the late stage.

4.3 Pancreas

The incidence of blast injury of pancreas is low in peacetime and wartime, accounting for 1.0–6.0% of abdominal trauma. Due to the special location of pancreas, the injury is often accompanied by blast injury of other organs (50–98%), the injury is complex and serious, and the mortality is as high as 20%. Early diagnosis is difficult and treatment is complex; it can further cause serious complications such as tissue necrosis, peritonitis, rupture, and bleeding of large blood vessels.

4.3.1 Clinical Manifestation

1. There is a small amount of tissue damage, blood seepage, or pancreatic juice leakage in the local part of pancreatic injury caused by mild pancreatic explosion and impact injury. The wounded only has mild upper abdominal discomfort or mild peritoneal irritation. However, a pseudocyst of the pancreas may be formed in a few weeks or months or even years, with symptoms of upper abdominal mass or gastrointestinal compression.
2. Severe pancreas blast injury. It can cause shock or collapse. The wounded complained of severe pain in upper abdomen, radioactive pain in shoulder or scapula, abdominal distension, nausea, vomiting, hiccup, and other symptoms. Localized rectus abdominis muscle rigidity and tenderness, sometimes irregular ecchymosis around the umbilicus or waist skin.

4.3.2 Diagnosis

1. Medical history. The patient with upper abdominal penetrating injury should undergo exploratory laparotomy during which diagnosis can be made clearly. For patients with closed blast injury, the action mode and position of shock wave should be learned.
2. Physical examination. Pay attention to whether there is peritoneal irritation sign, lumbar tenderness, etc. If there are different degrees of tenderness in the upper abdomen, special attention should be paid.
3. Amylase assay. Pancreatic injury should be suspected when the blood and urine amylase increase, and the diagnosis is more meaningful with persistent and progressive increase of blood and urine amylase. Abdominal puncture or peritoneal lavage and amylase determination in peritoneal fluid have a high diagnostic value. Perforation in other parts of the upper gastrointestinal tract can cause the increase of amylase in blood and peritoneal puncture fluid due to the flow of intestinal content into the abdominal cavity.
4. Color Doppler ultrasonography can show the size, shape, and continuity of the pancreas, which is helpful to clarify the accumulation of liquid around the pancreas and the existence of pancreatic pseudocyst.
5. CT has high diagnostic value for pancreatic injury, which can clarify the accompanying duodenal injury, pancreatic splenic effusion, and abnormal pancreatic morphology. It is applicable to the diagnosis of pancreatic injury 24 h later and late postoperative complications.
6. Diagnostic peritoneal lavage. The amylase in peritoneal lavage fluid increases, and the positive rate of pancreatic retroperitoneal injury is not high.
7. Endoscopic Retrograde Cholangiopancreatography (ERCP) can show the injury of the main pancreatic duct, the overflow or aggregation of contrast media. The injury of the main pancreatic duct is the absolute indication of abdominal exploration, which is helpful in decision-making of surgery.
8. Laparotomy. It is difficult to diagnose pancreatic blast injury and accurately estimate its location and severity before operation. Exploratory laparotomy should be actively performed for those who are highly suspected of explosive impact injury of pancreas, obvious peritoneal irritation sign, or have been identified as blast injury of other abdominal organs.

4.3.3 Treatment of Pancreatic Blast Injuries

Comprehensive medical measures based on surgery are the most important means to treat pancreatic blast injury. Timely surgical exploration is the key to reduce complications and improve the cure rate.

1. *Management Principles*.
   (a) Strict hemostasis of pancreatic injury: The most common complication after pancreatic blast injury is secondary postoperative bleeding that requires strict hemostasis of each bleeding point. After the necrotic pancreatic tissue is removed, the bleeding point of the pancreas should be sutured parallel to the wound with intermittent mattress suture.
   (b) Excision of necrotic pancreatic tissue: Thorough debridement and preservation of maximal pancreatic functions must be taken into account during surgery, and when partial removal of the pancreas is required, the preservation of endocrine and exocrine pancreatic functions should be considered. If the two cannot be fully considered, complete debridement and resection of the dead pancreatic tissue.
   (c) Adequate peripancreatic drainage: Adequate and effective abdominal and peripancreatic interstitial drainage is a key measure to ensure the treatment effect of pancreatic injuries and to prevent and control complications. Drainage can reduce the accumulation of peripancreatic juice, reduce the digestion and corrosion of pancreatic juice to its own tissues, and prevent serious infection in the abdominal cavity and the occurrence of peripancreatic cysts.
   (d) In case of severe pancreatic injuries, additional percutaneous transhepatic cholangial drainage (PTCD) should be performed, and when necessary, laparoscopic choledocholithotomy T-tube drainage (LCHTD) to drain most of the bile out of the body via the bypass to reduce the secretion of pancreatic juice and promote healing.
   (e) Correct treatment of other combined organs and blood vessels injuries: Associated blast injuries to the liver, spleen, empty organs, and large vessels complicated by intra-abdominal hemorrhage and shock should be treated in a proper, early, timely, and effective manner.
2. *Postoperative Management.*
   (a) Maintenance of the functions of vital organs: ARDS or MODS may occur in severe cases of pancreatic blast injuries, especially in casualties with multiple injuries, or in those who have been in shock for a long time before surgery. If possible, they should receive critical care and treatment in ICU.
   (b) Nutrition support therapy: Because the function of the gastrointestinal tract has not yet recovered in the early stage, especially in patients with associated duodenal blast injury, parenteral nutrition support should be given in the early postoperative period.
   (c) Pancreatic endocrine function monitoring: Blood glucose and urine glucose should be monitored regularly after surgery, and exogenous insulin should be given if necessary.
   (d) Application of drugs that inhibit pancreatic secretion: There is a significant inhibitory effect on the secretion from the gastrointestinal tract and pancreatic juice.
   (e) Smooth, continuous drainage: Negative pressure drainage by double cannula and use of a drip tube to slowly drip sterile isotonic saline or antibiotic-containing liquid for irrigation, or application of a negative pressure closed drainage technique.

4.3.4 Postoperative Complications and Management

The incidence of postoperative complications of pancreatic blast injury can reach 30–40%.

1. Pancreatic fistula. The incidence is 20–30%, mostly in the head of pancreas. Conservative management of pancreatic fistula includes unobstructed drainage, nutrition support, medications to inhibit pancreatic secretion, increase protein intake and application of pancreatic enzyme. Surgery includes fistulectomy, partial resection of pancreas with fistula, and Roux-en-Y anastomosis of pancreatic fistula and jejunum.
2. Peripancreatic infection and abscess. The incidence is about 20%. After the diagnosis is clear, drainage should be carried out as soon as possible, and antibiotics and nutrition support should be given.
3. Pancreatitis. The incidence is about 5%. Fasting, gastrointestinal decompression, inhibition of gastric acid and pancreatic juice secretion, improvement of pancreatic microcirculation, rehydration, and nutrition support should be given.
4. Intraperitoneal hemorrhage. The incidence is 5–10%. When the amount of bleeding is small, nonsurgical treatment can be used; for massive hemorrhage, repeated surgery should be considered for hemostasis.
5. Pancreatic deficiency. The blood and urine glucose should be monitored regularly after the operation. After the injury recovers, the pancreas function can be compensated and recover. Insulin replacement therapy can be used before the recovery. Permanent pancreatic deficiency requires lifelong insulin replacement therapy.
6. Pancreatic pseudocyst. The incidence of pancreatic pseudocyst is 10–30%, which can be removed if it is small and

limited to the pancreatic tail. If the cyst is firmly formed and adheres closely to the posterior wall of the stomach, cyst gastric posterior wall anastomosis is often adopted. Most cysts can be anastomosed with cyst jejunum.

4.4 Spleen

The spleen is soft in texture and rich in blood supply with 150–250 g in weight and 11–12 cm in length. It has a dense capsule and occupies a small area of the abdominal cavity. When it is subjected to a certain external force, it is very easy to cause rupture and bleeding. It is common in peacetime, but the proportion of explosive impact traumatic spleen rupture in wartime is not large.

4.4.1 Clinical Manifestation

The main manifestation is hemorrhagic shock, including decreased blood pressure, rapid and weak pulses, increased respiration rate, anemia, clammy limbs, cyanotic mouth and lips, and changes in consciousness. The degree of shock is related to the severity of splenic injury and the amount of bleeding. The more bleeding, the more severe shock. Signs can be noted such as obvious tenderness, rebound pain, and muscle tension in the abdomen after injury, especially in the left upper abdomen. Abdominal breathing is weakened or disappears after injury. Positive signs include plate abdomen, percussion pain in left costal area in varying degrees, and positive diaphragm stimulation sign. The abdomen swells in varying degrees. When the blood loss is more than 1000 ml, the shifting dullness is positive with bowel sounds weakening or disappearing in auscultation.

4.4.2 Diagnosis of Blast Injury to the Spleen

For patients suspected of splenic blast injury, the followings should be clarified in diagnosis, that is, (a) whether there is splenic blast injury; (b) the severity and scope of splenic blast injury; (c) whether it is combined with other abdominal visceral blast injury or diaphragm rupture; (d) whether it is combined with blast injury of other organs or tissues outside the abdomen; (e) whether there is blast injury requiring emergency treatment, such as craniocerebral injury (brain hernia, increased intracranial pressure), tension or open pneumothorax, cardiac tamponade, airway obstruction (asphyxia), rupture and bleeding of large blood vessel explosive blast injury, etc.

1. Trauma history. All abdominal injuries or closed abdominal injuries or open explosive impact injuries adjacent to the spleen should think of the possibility of splenic blast injury.
2. Clinical manifestations. Same as the above.
3. Abdominal puncture and diagnostic peritoneal lavage. Diagnostic significance lies in dark red noncoagulated blood extracted from the left lower abdomen.
4. X-ray. It can show rib fracture (left 9th to 10th rib), elevated left diaphragm with limited movement, enlarged shadow of spleen, inward displacement of the gastric vesicle, serrated shape of the great curvature of stomach, compressed, descending and displaced colonic splenic curvature, etc.
5. Ultrasonography. It can timely diagnose the severity of splenic blast injury, estimate the amount of intraperitoneal hemorrhage, and judge the existence of splenic blast injury combined with trauma history.
6. CT examination. The sensitivity and accuracy of X-ray and enhanced CT scanning are up to 95%. If the initial CT scanning is negative, close observation and regular CT Reexamination should be made to avoid missing the diagnosis of delayed splenic rupture.
7. MRI examination. MRI findings such as splenic contusion and laceration, rupture, hematoma, and subcapsular hemorrhage are consistent with CT, with different MRI signal intensity of hematoma.

4.4.3 Treatment of Blast Injury to the Spleen

If conditions permit, spleen tissue should be retained as much as possible. Nonsurgical treatment can be conducted to preserve the spleen and spleen tissues as much as possible, if 80–90% of the spleen rupture has been coagulated by blood clot, or the bleeding of spleen rupture causes mild peritoneal stimulation without blast injuries of other organs.

1. Nonsurgical Treatment.
   (a) Nonoperative treatment is applicable to patients younger than 50 years, with clear diagnosis of simple splenic trauma, exclusion of pathological spleen, stable hemodynamics, no expansion of hematoma monitored by B-mode ultrasound and CT, conditions for conversion to surgical treatment at any time, and intensive care unit or corresponding monitoring conditions.
   (b) Nonsurgical treatment includes (a) evaluating the disease progress, treatment effect and prognosis through various monitoring means; (b) general treatment such as strict bed rest and limited activity. Fasting and water deprivation should be applied. Continue gastrointestinal decompression, nutrition support, and balance of water, electrolytes and acid-base for those with obvious abdominal distension. Broad spectrum antibiotics should be used to prevent infection. (c) Application of hemostatic drugs, such as ethylphensulfoamine, aminotoluene acid, hemag-

glutinase, etc., appropriate application of somatostatin. (d) Splenic artery embolization, which can greatly reduce the perfusion of spleen, where the embolization materials include gelatin sponge, silicone rubber, stainless steel ring, tissue adhesive, anhydrous alcohol, self-clotting, etc.

2. Surgery. Before surgery, anti-shock measures should be actively taken, including fluid replacement, blood transfusion, and plasma substitute infusion. For those with comparatively stable hemodynamics, emergency surgery should be performed as soon as possible. For those with unstable hemodynamics, if necessary, rapid arterial blood transfusion can be used to supplement blood volume, and decisive open exploration can be carried out at the same time of antishock. The left rectus abdominis incision or upper abdominal median incision is often used in clinic. According to the findings during the intraoperative exploration, a transverse incision can be added to both sides of the abdomen, or extended upward to the thoracoabdominal symphysis incision, or extended downward to the upper part of the pubic symphysis to deal with the combined injury.
3. Operation method. (a) Fibrin adhesive hemostasis: it is suitable for splenic subcapsular hematoma, capsule tear, or superficial splenic parenchyma laceration. (b) Splenic suture: for those with shallow rupture wound. (c) Splenic reticulum wrapping: it is used for severe splenic rupture, such as one or more lacerations deep into the splenic parenchyma extending to the capsule, lobulated laceration of the spleen, hemorrhage of the splenic parenchyma, etc. (d) Splenic artery ligation: it is used as an auxiliary method for other spleen preserving operations, such as splenic suture and partial splenectomy. (e) Partial splenectomy: it is applicable to severe explosive impact injury at the upper or lower pole of the spleen. The patient's general condition is good, the vital signs are stable, and there is no serious fatal combined injury. (f) Total splenectomy: in order to save the lives of patients with severe splenic blast injury, splenectomy should be carried out decisively to stop bleeding completely and reliably.

After completing the treatment of explosive impact injury to the spleen and effectively controlling the bleeding, the abdominal exploration should be carried out carefully and orderly, attention should be paid to check whether there are combined explosive impact injuries of other parenchymal organs or hollow organs and make timely corresponding treatment according to the findings. The changes of the patient's condition should be closely observed after operation, and the blood pressure, pulse, respiration, body temperature, blood oxygen saturation, and hemodynamics should be monitored. Those with conditions should be monitored in ICU until the patient's condition is stable.

4.4.4 Complications and Management

In case of intraperitoneal hemorrhage, massive stress ulcer bleeding, mesenteric thrombosis, pancreatitis, spleen heat, subphrenic infection, lung infection, etc., correct treatment shall be made in time. After operation, keep the drainage tube unobstructed to prevent slippage, record the drainage flow, observe its shape and color, and measure amylase and hemoglobin if necessary. If the peritoneal drainage volume is less than 20–50 ml per day and the amylase is not high, it is usually removed 48–72 h after operation, and some can be extended to 7 days after operation. Patients with splenectomy have an increased chance of postoperative infection. Broad spectrum antibiotics should be used to prevent infection, especially in children under 2 years of age.

4.5 Kidney

4.6 Clinical Manifestation

Hematuria is an important symptom of renal blast injury, mostly macroscopic hematuria and a few microscopic hematuria. About 5–10% of patients with renal blast injury have no hematuria. The severity of hematuria is sometimes inconsistent with the degree of renal explosion impact injury. Pay attention to inquire about the micturition of the injured after injury, and conduct catheterization if necessary. Renal tissue injury can cause swelling and pain around the kidney. The decrease of blood pressure and even shock is a serious manifestation of renal explosion shock injury and internal bleeding, which can be traumatic shock and hemorrhagic shock. In case of open explosive impact injury, attention should be paid to the direction and depth of injury entrance such as injury site and nearby shrapnel, whether there is urine or blood overflow, whether there is exit and route, so as to estimate the possible scope of combined injury.

4.6.1 Diagnosis

The diagnosis of renal blast injury can be made according to trauma history, clinical manifestations, urine analysis, and X-ray examination. Most of the wounded can make a preliminary diagnosis only based on the trauma history and the symptoms of hematuria and low back pain after injury.

1. Trauma history. The vast majority of patients with renal blast injury have a history of impact, crush injury, or knife and gun wound to the waist or abdomen. Attention should be paid to every detail of the injury process. Detailed investigations are required, including whether there is micturition, hematuria, and state of consciousness after injury, which is of great significance for comprehensive

evaluation of injury and further examination and treatment.
2. Clinical manifestations. It is consistent with the above.
3. Laboratory examinations. Urine analysis is the most important index to evaluate renal blast injury. Dynamic urine analysis is of great significance to evaluate renal blast injury and recovery process. The blood routine test is helpful to evaluate whether it is complicated with urinary tract hemorrhage.
4. X-ray examination. (a) X-ray plain film: Severely combined injury plain film examination plays a key role in diagnosis and treatment. If it is shrapnel injury, whether there are metal foreign bodies and their locations can also be learned. (b) High-dose intravenous pyelogram: It is a diagnostic imaging method for the initial stage of renal blast injury and conducive to the accurate staging of renal blast injuries. (c) Renal arteriography: It can provide more detailed evidence of renal vascular blast injury, while allowing selective embolization to treat active vascular bleeding in the kidney.
5. CT scanning. CT is a common method for the diagnosis of renal blast injury with higher sensitivity and specificity than those of the intravenous urography. It can also provide imaging evidence of renal adjacent tissue and organ injury. Multiplanar reconstruction and three-dimensional imaging technology will provide more abundant and complete information for the diagnosis of renal trauma.

4.6.2 Treatment

1. First aid management. First aid treatment principles include blood transfusion, fluid infusion, anti-shock therapy, and resuscitation as soon as possible, and clarifying whether it is combined with blast injury of other organs. The treatment principle is to preserve renal function to the greatest extent and reduce complications and comorbidity and to make a detailed assessment of the severity of renal injury.
2. Nonsurgical treatment. For mild closed renal blast injury, where surgery is not required, treatment includes strict bed rest for 2 weeks, blood volume supplement, keeping balance of water and electrolytes, and maintaining sufficient urine volume, closely observation of the changes in blood pressure, pulse, body temperature, urine color, and the size of the perirenal mass by B-mode ultrasound regularly. If a perirenal mass is palpable, the size change of the mass should be observed regularly, and B-ultrasound observation should be performed regularly; hemostatic drugs, prophylactic antibiotics, and appropriate painkillers should be applied.
3. Surgery.
   (a) Indications for surgery: (a) Open renal blast injury; (b) patients with low blood pressure after active anti-shock (600 ml blood transfusion); (c) massive urinary extravasation, including severe renal fragmentation and rupture of renal pelvis; (d) renal pedicle injury often needs urgent laparotomy for blood arrest due to serious bleeding; (e) severe blast injury combined with other abdominal organs.
   (b) Surgical methods: First, explore the abdominal visceral organs to control active bleeding. It is emphasized that the renal pedicle should be controlled before opening the Gerota fascia of the kidney to avoid uncontrollable bleeding due to perirenal decompression after opening the Gerota fascia. Cut the retroperitoneum parallel to the inferior mesenteric vein in the middle, and pull the left renal vein upward and forward to expose the beginning of the left and right renal arteries to control the renal arteries. After temporarily controlling the renal artery on the injured side with a vascular clamp or vascular ring, cut the ipsilateral paracolonic sulcus and the Gerota fascia of the kidney, remove the hematoma, preserve the surviving renal tissue as much as possible, closely suture the ruptured collecting system, suture the small blood vessels, suture the ruptured renal tissue, remove the ruptured renal tissue, and drain urinary extravasation.

4.6.3 Complications and Management

Early complications of renal injury include secondary hemorrhage, urinary extravasation, and perirenal abscess. Manifestations such as low back pain, fever, mass, decreased hemoglobin, or increased serum creatinine after injury or operation often represent the occurrence of the above complications. Percutaneous puncture can be used to drain renal urinary cyst and perirenal abscess. Selective angiographic embolization can effectively control secondary bleeding caused by renal segmental vascular blast injury or pseudoaneurysm.

The late complications of renal trauma include Page kidney, hypertension, renal arteriovenous fistula, hydronephrosis, renal pseudocyst, renal calculi, chronic pyelonephritis, and loss of renal function. These complications often occur within 1 year after kidney injury. Renal trauma-induced hypertension mostly disappear within 6 weeks. In case of malignant hypertension, renal vascular repair or nephrectomy is needed. In case of recurrent hematuria, angiography and embolization should be carried out considering the possibility of renal arteriovenous fistula.

4.7 Ureter

4.7.1 Clinical Manifestation

After ureteral blast injury, due to urine extravasation, the injured suffer from low back pain, abdominal pain, fever, nausea, vomiting, abdominal distension, intestinal paralysis, and other symptoms. Most of the wounded can have hematuria.

4.7.2 Diagnosis

The diagnosis was made according to trauma history, clinical manifestations, intravenous pyelography, and retrograde pyelography. Patients with ureteral blast injury have a history of lumbar, abdominal or pelvic injury, often complicated with explosive blast injury of blood vessels and other abdominal organs. If there is an open wound, there is a large amount of fluid leakage through the incision or wound, resulting in ureteral skin or ureterovaginal fistula. Urine extravasation needs to be distinguished from peritoneal exudation. Anuria occurred when bilateral ureters were damaged; some of the wounded had no obvious symptoms in the early stage and had obstructive symptoms such as hydronephrosis due to ureteral stenosis in the later stage.

Intravenous pyelography and retrograde pyelography are the most important diagnostic measures. Intravenous pyelography can find obstruction of contrast medium excretion and hydronephrosis. Sometimes, due to ureteral rupture, the excretion of contrast medium is accelerated. Retrograde pyelography can accurately determine the degree and location of ureteral blast injury. B-mode ultrasound and CT examination can find urine extravasation and abscess formation.

4.7.3 Treatment

The purpose of the treatment of ureteral blast injury is to restore the continuity of ureter, drain urine extravasation, prevent infection, and protect the renal function of the affected side. Priority shall be given to organ explosion and impact injury that threatens the life of the injured. Ureteral blast injury found during operation should be repaired immediately if there is no infection. If the diagnosis of injury exceeds 24 h, nephrostomy can be performed first, and repair operation can be performed 3 months later. Percutaneous drainage of urinary cysts can also be used. Retrograde or anterograde placement of ureteral stents, such as double J-tube internal drainage, can completely heal the ureter. In case of massive urinary extravasation, open surgery is required.

Ureteral repair should follow the following principles: (a) complete debridement of the wound; (b) appropriate tension of ureteral anastomosis; (c) tight suture; (d) indwelling ureteral stent; (e) nephrostomy or indwelling ureteral stent external drainage for complicated ureteral blast injury, wound pollution, or poor tissue vitality; (f) full drainage with a retroperitoneal drainage tube. Ureterocystostomy can be performed directly for lower ureteral injury. If the ureteral defect is large, psoas major muscle bladder suspension or bladder muscle flap plasty can be used to reduce the tension of ureteral anastomosis. For upper and middle ureteral blast injury, ureteral end-to-end anastomosis or ureteropelvic anastomosis should be performed. If the ureteral defect is large, intestinal replacement of ureter or autologous kidney transplantation can be performed.

4.7.4 Complications and Prognosis

Ureteral stricture, ureteral obstruction and hydronephrosis can occur in ureteral blast injury, resulting in damage to renal function. Urine extravasation can lead to retroperitoneal urine accumulation, urinary cyst, abscess formation, repeated urinary tract infection, etc., which needs timely proximal drainage treatment.

5 Blast Injury of Liquid-Contained Organ

5.1 Gallbladder

See Sect. 4 for details.

5.2 Bladder

5.2.1 Clinical Manifestation

1. Intraperitoneal rupture. It is mostly caused by bladder penetrating explosion impact injury and blunt abdominal injury. A large amount of urine enters the abdominal cavity through the bladder hole, resulting in severe urinary peritonitis. Physical examination showed obvious peritoneal irritation signs.
2. Extraperitoneal rupture. The bladder hiatus is located under the peritoneal fold. Due to the integrity of the peritoneum, the scope of urine extravasation is mainly limited to the retropubic space. Patients can have diffuse pain in the lower abdomen, but the pain is often lighter than that of intraperitoneal rupture.
3. Mixed bladder rupture. About 10% of patients with mixed bladder rupture have both intraperitoneal and extraperitoneal bladder rupture. The urine extravasation range is wide and can extravasate to the anterior abdominal wall, penis, scrotum, and perineum. It usually indicates that the fascia boundary of pelvis has been torn, which should be distinguished from combined urethral explosive impact injury.

5.2.2 Diagnosis

The diagnosis of bladder blast injury can be made according to injury history, clinical manifestations, and cystography. The type of rupture is closely related to the filling state of bladder at the time of injury. Intraperitoneal bladder rupture is easy to occur when the bladder is filled, and extraperitoneal bladder rupture is easy to occur when the bladder is empty. Extraperitoneal bladder rupture should be considered if there is swelling, cyanosis, pain, and inability to urinate on the

pubis. Intraperitoneal bladder rupture should be considered for the symptoms of urinary peritonitis such as abdominal distension, abdominal pain, muscle tension, and disappearance of bowel sounds. If there is blood at the urethral orifice, it should be suspected of combined urethral explosion and impact injury.

Hematuria is one of the important symptoms of bladder trauma. Cystography is of decisive significance in the diagnosis of bladder rupture, with an accuracy of 85–100%. Retrograde urethrography should be performed if urethral blast injury is suspected.

CT scanning cystography is an important method for the diagnosis of bladder blast injury. Intravenous pyelography can distinguish whether it is complicated with renal and ureteral blast injury at the same time.

5.2.3 Treatment

1. Bladder contusion. Such injury has only mucosal and muscle damage without interruption of bladder wall continuity, no extravasation of contrast medium, and the bladder wall is complete, which generally needs bi treatment.
2. Interstitial bladder blast injury. Incomplete perforation of bladder wall, no urine extravasation, or although the bladder wall was completely broken, the blood clot or omentum closed the breach during cystography. The rest time of bladder can be prolonged by indwelling urinary catheter, and the catheter can be pulled out after 10 days.
3. Intraperitoneal rupture. The rupture is often located at the top of the bladder, and the fissure is large (5–10 cm), which needs to be repaired by open surgery. The operation generally takes the median abdominal incision to absorb the exuded urine and tissue necrosis, which can be removed. Carefully explore the organs and blood vessels that may be injured by explosion in the abdominal cavity. If the bleeding in the bladder is serious, it is necessary to indwell a suprapubic fistula.
4. Extraperitoneal bladder rupture. It is mostly related to pelvic fracture. Stop bleeding quickly to ensure that there is no blood clot retention, and indwell air bag catheter. If the catheter drainage is not smooth, it needs open surgical repair and regular bladder repair. In case of bladder neck, prostate and vaginal blast injury, repair operation should be carried out immediately.
5. Mixed bladder blast injury. It needs to be repaired by appropriate combination of the above operations.
6. Bladder penetrating injury. All patients with penetrating injury need open exploration to explore all injury channels from inlet to outlet. Explore all organs in the abdominal cavity and explore the interior of the bladder through the incision on the top of the bladder.

5.2.4 Complications and Management

For bladder infection, some patients may have infection due to unconventional use of antibiotics after operation. Antibiotics should be given to purify urine when removing urinary catheter. Some of the wounded suffer from long-term urgent urinary incontinence and need to be treated with anticholinergic drugs. For bladder reflex deficiency, which is often caused by iliac bone fracture, intermittent clean catheterization is needed for a long time.

6 Abdominal Vascular Blast Injury

6.1 Abdominal Great Vessels

6.1.1 Clinical Manifestation

Most of the wounded died at the scene due to rapid bleeding. Massive bleeding, progressive abdominal distension, and severe shock suggest abdominal macrovascular injury. The early wounded were nervous, pale, rapid pulse, decreased blood pressure, and shallow and fast breathing. After coma, the pulse and blood pressure could not be measured, the breathing was weak, the pupils were dilated, and finally the heart stopped and died. Common abdominal aortic injury can appear obvious weakening or disappearance of arterial pulsation of both the lower limbs. The injured side has lower limb pain, pale skin, cold limbs, weak or disappeared arterial pulse, limited movement of the injured limb, and even gangrene of the lower limb due to acute ischemia, suggesting damage to the common iliac artery and external iliac artery.

6.1.2 Diagnosis

Those with hemorrhagic shock in a short time after abdominal trauma without bleeding in other parts are highly suspected of explosive impact injury of abdominal large blood vessels. The wounded should be sent to the emergency room immediately or directly to the operating room. Due to the fierce bleeding and the rapid deterioration of the condition, comprehensive examination is not allowed. It is possible to treat the bleeding only by immediate laparotomy while actively resisting shock.

6.1.3 Treatment

1. Abdominal aortic blast injury. The management of abdominal aortic blast injury depends on good exposure. Usually, the peritoneum on the outside of the right colon and the lower edge of the mesenteric root of the small intestine needs to be cut, and the right colon, together with the duodenum and pancreatic head, shall be turned to the left. If necessary, the peritoneum on the outside of the descending colon shall be cut, separated along the front of the left kidney, and the spleen, pancreas, stomach, and

splenic curvature of the colon shall be turned to the right. Moreover, the incision can be changed to the thoracoabdominal incision for better exposure. The blood flow should be blocked at the proximal and distal ends of the damaged area for repair. The treatment of celiac artery or superior mesenteric artery injury is difficult, but it should be repaired or vascular transplantation is performed; common hepatic artery injury can be ligated, proper hepatic artery injury can be ligated when there is no injury to portal vein, and inferior mesenteric artery can also be ligated. When the renal artery is injured, the blood flow should not be blocked for more than 40 min. After the loss of the left renal artery, the proximal splenic artery can be cut off and anastomosed with the distal splenic artery. The external iliac artery can be transferred and anastomosed with the internal iliac artery.

2. Blast injury of inferior vena cava. The wounded with blast injury of posthepatic inferior vena cava are often in a dying state with coagulation disorder. When Pringle method is used to block the hepatic portal during the operation and still does not alleviate the blood gushing after liver, it indicates that there is a posthepatic vein injury, where Gauze pad shall be used to temporarily fill or press the liver back and up to control the bleeding. If the hepatic vein injury is serious and difficult to repair, the injured hepatic vein can be ligated, but at least half of the main hepatic vein should be repaired. Perihepatic tamponade is an important control method.

   The inferior vena cava below the liver and above the level of renal vein was controlled by inferior vena cava shunt. Insert #34–38 Argyle catheters with side holes at both ends from the right atrial appendage incision to the inferior vena cava, so that the proximal side hole is located in the right atrium, and the distal side hole opening is located under the renal vein. Thread ropes are respectively sleeved at the pericardial end of the inferior vena cava and above the renal vein to block the blood flow of the inferior vena cava. In this way, the blood of the inferior vena cava can flow back to the right atrium through the catheter, so as to avoid sudden and large reduction of the amount of return blood, hemorrhagic shock recovered after treatment.

   The inferior vena cava below the level of renal vein can be ligated below the level of renal vein. The wounded can tolerate it and prevent pulmonary embolism. Bilateral lower limbs can reduce edema, which is superior to lower limb vein reconstruction.

3. Blast injury of portal vein trunk. The mortality of blast injury of portal vein is more than 50%. About 70% of liver blood supply comes from portal vein, which should be repaired immediately after blast injury. The distal injury at the confluence of splenic vein and superior mesenteric vein can be sutured, the proximal end of superior mesenteric vein can be closed, the spleen can be removed, and the splenic vein can be turned to the lower interior for end-to-end anastomosis with the distal broken end of superior mesenteric vein. For large-scale portal vein injury, artificial blood vessels, allogeneic blood vessels, or autologous blood vessels can be used for vascular transplantation to reconstruct the portal vein. If the damage is serious and cannot be repaired, portal vein shunt or ligation can be used.
4. Blast injury of common iliac artery and external iliac artery. Blast injury of common iliac artery and external iliac artery should be repaired or reconstructed to restore the continuity of blood vessels, so as to ensure the blood circulation of lower extremities. According to the situation, methods such as side wall suture, broken end anastomosis of blood vessels, autologous blood vessels, or allogeneic blood vessel transplantation are selected. The injury of common iliac vein and external iliac vein should be repaired as much as possible. If the internal iliac vein is damaged, the broken ends on both sides of the injured vessel can be ligated directly without repair.
5. Presacral vein blast injury. Once presacral hemorrhage occurs in presacral vein blast injury, gauze should be used quickly and effectively to stop bleeding and try to good exposure. According to the vein type of blast injury, suture, gauze packing, blunt instrument smashing sacral foramen, pushpin compression, and other methods are used to stop bleeding. The special hemostatic nail compression can quickly and effectively control hemostasis without special complications.

6.2 Retroperitoneum

6.2.1 Clinical Manifestation

Retroperitoneal hematoma is often accompanied by explosive impact injury of retroperitoneal organs and intermediate organs. The clinical manifestations mainly include abdominal pain, back pain, and tenderness at the corresponding parts, symptoms of paralytic intestinal obstruction such as abdominal distension, weakening or disappearance of bowel sounds, hemorrhagic shock, hematoma caused by severe pelvic fracture. Retroperitoneal hematoma can be presented as lateral abdominal and lumbar ecchymosis, tenderness mass which can be palpable by digital examination of abdomen or rectum. Hematoma which penetrates the retroperitoneum can lead to hemoperitoneum and peritonitis. Abdominal central retroperitoneal hematoma can touch swelling, pulsatile, or gradually increasing masses; retroperitoneal hematoma in renal area and anterior pelvic cavity may have urinary symptoms and signs such as hematuria.

6.2.2 Diagnosis

The possibility of retroperitoneal hematoma should be considered when abdominal pain, low back pain, abdominal distension, rare bowel sounds, and systemic blood loss occur after explosive impact injury, especially pelvic or vertebral fracture. If the vital signs are stable, abdominal X-ray, ultrasonography, CT, and DSA are helpful to make a definite diagnosis.

6.2.3 Treatment

For patients with retroperitoneal hematoma and explosive impact injury of abdominal large blood vessels, 6 h after injury, especially the first hour, is the "golden time" for rescuing such patients. The key to saving lives is to control bleeding rather than just maintain blood flow. The patient should reach the effective treatment institution in the shortest time and control the bleeding before cardiac arrest. Crystal solution, plasma substitute, or whole blood should be quickly input to improve vascular volume. When shock cannot be corrected, laparotomy should be carried out decisively. Small retroperitoneal hematoma or large hematoma without hemodynamic changes can be treated by nonsurgical treatment, but it should be closely observed for 6–8 h. If blood pressure is unstable or decreased, or peritoneal irritation sign occurs, it should be actively treated. If retroperitoneal hematoma is found during operation, the treatment method should be determined according to the cause of injury, instability of hematoma and progressive enlargement of hematoma. Retroperitoneal hematoma caused by large blood vessels should be explored by incision of the retroperitoneum. The steps of surgical treatment are as follows:

1. Preoperative preparation. Preoperative preparation includes indwelling urinary catheter, nasogastric tube, establishing a sufficient number of venous pipelines and application of broad-spectrum antibiotics. For patients with hemodynamic instability, they should be quickly sent to the operating room to complete skin disinfection. The sheet should completely expose the anterior chest and abdominal wall, both sides to the axillary midline.
2. Exploration and bleeding control. Quickly remove the bleeding after entering the abdomen through the midline incision. If the amount of bleeding is large and bright red, the assistant should compress the aorta from the lower edge of the diaphragm. If the operation cannot be completed within 40 min, the abdominal aortic blood flow should be opened every 20 min for at least 10 min; if it is blocked below the level of the renal artery, the time can be extended. The principle of bleeding control before dealing with combined injury should be followed. The hematoma formed by blast injury of abdominal aorta was pulsatile, and the hematoma formed by arteriovenous fistula had continuous tremor. The bleeding of portal vein blast injury can be controlled by compressing the hepatoduodenal ligament. After it is determined that the hepatic artery is not injured by blast injury, the blood flow of hepatic artery should be restored.

   The best way to control the bleeding is to dissect the far and near ends of the blood vessels of explosion impact injury and control them, including after entering the abdomen, the abdominal cavity is full of blood, the abdominal aorta should be blocked from under the diaphragm, and the thoracic aorta above the diaphragm should be blocked in advance when considering the abdominal aorta. Small breach can be pressed, clamped, or filled on the side. In case of defect, transverse or penetrating injury, the two ends of explosion impact injury shall be clamped or blocked by strap. When the massive hemorrhage of retrohepatic inferior vena cava cannot be controlled, the superior or inferior vena cava and hepatoduodenal ligament should be blocked. When it cannot be blocked under the diaphragm, the chest should be opened quickly. The blocking of inferior vena cava may induce arrest due to the sudden decrease of return blood volume.

   After the bleeding is temporarily controlled, the operation should be suspended, and the treatment strategy should be adjusted: prepare enough blood and fluid; establish sufficient rapid heating infusion and blood transfusion devices and channels; prepare fresh frozen plasma and platelets if necessary; contact an experienced surgeon; prepare special sutures, instruments, and instruments.
3. Hemostasis and repair methods: Vascular ligation is the simplest and reliable hemostatic measure. Inferior vena cava and iliac vein below the level of renal vein can be ligated; in extreme cases, portal vein and superior mesenteric vein can also be ligated, but it can cause a large amount of liquid to transfer to the third space and require a large amount of fluid replacement; ligation of common iliac artery and external iliac artery can cause serious limb ischemia, be careful.

   Vascular repair techniques include direct suture repair, vein patch repair, end-to-end anastomosis, autologous vascular transplantation, and artificial vascular transplantation. If the defect is within 2 cm or the intima is intact, end-to-end suture can be used. If the defect is more than 2 cm, multiple injuries of a section of blood vessel and severe contusion, blood vessel transplantation should be carried out. Inferior vena cava, abdominal aorta, portal vein, superior mesenteric vessels, common iliac vessels, and external iliac vessels should be repaired or transplanted.

6.2.4 Postoperative Complications

Strengthen postoperative monitoring, especially pay attention to monitoring vital signs, hourly urine volume and central venous pressure, and supplement whole blood, liquid or plasma to make the blood volume return to normal. Attention should be paid to prevent infection and treat coagulation disorders after massive blood transfusion. It is suggested to prevent and treat acute renal failure, improvement of respiratory function, prevention of pulmonary complications, and prevention of infection.

Part IV

Combined Blast Injury

Burn-Blast Combined Injury

Jihong Zhou and Jun Qiu

1 Overview

1.1 Overview

Burn-blast combined injury (or BBCI) is a combined injury caused by the simultaneous or sequential exposure of personnel to shock waves and heat. BBCI occurs in both wartime and peacetime. Among them, burn injury-dominant BBCI is more common, and blast injury based BBCI is also frequently encountered.

As coal mine gas explosion, inflammable detonation and combustion, and boiler and nuclear reactor facility accidents can induce burn-blast combined injuries, BBCI incidents are occasionally seen among groups in normal times. For instance, at 20:40, June 27, 2015, the dust explosion in a water park called Formosa Fun Coast in New Taipei City injured more than 500 people, of which 498 were sent to the hospital, and 183 to the ICU. Most of the wounded sustained burns, accompanied by varying degrees of blast injuries. Although BBCI patients are accessible to powerful medical resources and timely treatment in normal times, general practitioners are unfamiliar with burn-blast combined injuries and often find it difficult to give the most appropriate treatment and may even use inappropriate treatment strategies or methods, leading to grave consequences.

In wartime, BBCI is a common type of combined injury, especially in conflicts where nuclear weapons and conventional high-tech explosive weapons are used. The explosion of a nuclear weapon can generate intense light radiation and powerful blast waves, inflicting burns on people exposed to optical radiation as well as varying degrees of blast injuries. There are numerous cases of burn-blast combined injuries from the detonation of nuclear weapons. Among them, during a 100,000-ton nuclear explosion, BBCI is the primary type of injuries caused by it; during a less than 100,000-ton nuclear explosion, the range of burns from light radiation is greater than the range of injuries from blast waves; during a more than 100,000-ton nuclear explosion, the range of blast injuries is greater than the range of light radiation induced burns. In modern conventional warfare, explosive weapons have become the most important means of combat that often inflict both blast injuries and flame burns on people. Thus, the occurrence of BBCIs is not uncommon. Especially after massive use of modern high-tech weapons, such as fuel-air explosive (FAE) weapons, the incidence of BBCI surged as a major type of resultant injuries The moment the FAE goes off the temperature rises to about 3000 °C for several milliseconds, which can cause skin burns to those in the cloud-bursting area and its periphery; in the deflagration of the FAE bomb, high temperature can maintain tens of milliseconds, the effect of which will be more intense and can lead to serious burns; with the emergence of third-generation FAE, thermobaric bomb, which is filled with additional combustion supporting compositions like aluminum, the high temperature from explosion lasts and increases substantially, and the resulting burns are more severe. Moreover, FAE bombs tend to ignite surrounding inflammables, which also result in serious burns. So, there is a high incidence of burn-blast combined injuries, and the condition is serious and complex.

1.2 Classification

Burn-blast combined injuries are usually simultaneously or successively caused by shock waves and thermal energy. Some academics refer to burn-dominant BBCI as burn-blast combined injury, and blast injury-dominant BBCI as blast-burn combined injury. Since the BBCI is complex in terms of the site, type, and condition, it is difficult to accurately identify and differentiate, so most academics do not deliberately distinguish between "burn-blast combined injury" and "blast-burn combined injury," which are uniformly called burn-blast combined injury (BBCI).

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The severity of BBCI is usually classified into four levels:

1. *Mild injuries*: Burns and blast injuries are both mild cases.
2. *Moderate injuries*: Either one of the burn and blast injuries is moderate.
3. *Severe injuries*: Either one of the burn and blast injuries is severe, or both are moderate.
4. *Extremely severe injuries*: Either one of the burn and blast injuries is extremely severe, or both are extremely severe.

Regarding injuries above moderate, if accompanied by serious complications like profound shock and dysfunction or failure of vital organs, or serious chronic primary diseases, the condition will be aggravated by one level.

In contrast to single burns and single blast injuries, BBCIs have their own characteristics and needs, both in terms of injury characteristics and pathophysiological processes, as well as the essentials of diagnosis and treatment. The course of a BBCI above the moderate level is similar to that of a burn injury and can be divided into a shock phase, an infection phase, and a recovery phase; but as the main target organs of blast injuries are gas filled structures, the injury is manifested as a light external and heavy internal condition with rapid changes, making it difficult to carry out accurate and prompt diagnosis and treatment. The blast injuries below the moderate level are mainly present as bleeding lung tissues; for above-moderate-level blast injuries combined with burns, in addition to varying degrees of hemorrhage, it may cause varying degrees of pulmonary edema, or damages to other organs, such as capsula fibrosa tears, liver lacerations in multiple places, gastrointestinal hemorrhage, and cysthemorrhagia. There are some differences in the pathophysiological processes and characteristics between burn-dominant and blast injury-dominant BBCIs, and even the pathophysiology of burn injuries followed by blast injuries is quite different from that of those who sustain further burns after blast injuries. There are also many contradictions and conflicts in the treatment of BBCI injuries. For example, the contradiction between fluid therapy for burn shock and the control of hemorrhage and edema from pulmonary and cerebral blast injuries, and the contradiction between sedation and analgesia and respiratory depression. Therefore, it is important to understand the pathophysiological characteristics of burn-blast combined injuries and the contradictions and needs of diagnosis and treatment.

2 Pathophysiological Processes of Burn-Blast Combined Injuries

Depending on the severity of the burn, the differences in the organ and degree of injury caused by the shock wave, and the accompanying injuries (e.g., fragments, impact, crush, and other secondary blast injuries), the pathological changes of burn-blast combined injuries vary, and the development process is more volatile and complicated.

In general, the fundamental pathological process of a moderate BBCI or above is similar to that of a burn and can also be divided into shock, infection, trauma repair, and rehabilitation phases. However, because of being associated with both primary and secondary blast injuries, the pathophysiological changes of BBCI are mainly characterized by the following aspects:

2.1 High Incidence of Shock

When the burn severity is above moderate, it can lead to progressive hypovolemic shock due to the massive and continuous loss of plasma from the burn wound; at the same time, the primary blast injury can result in hemorrhage and edema of the lung tissue, laceration and bleeding of the liver and spleen tissues, hemorrhage and even perforation of the gastrointestinal tract, etc., or different types and degrees of shock; the damage to the cardiac muscle cell and the air embolism of the coronary vessel, however, are factors leading to cardiogenic shock. In addition, the large number of cardiomyocyte inhibitory factors, vasoactive substances, and various inflammatory factors produced by the body after burns and blast injuries may promote and aggravate shock; other important causes of shock are fractures, soft tissue lacerations, fragmentation and projectile injuries, crush and compression injuries, etc. from secondary blast injuries.

Therefore, shock is more common among BBCI patients, which generally occurs earlier, lasts longer, and has a more complex pathophysiological process; the patients with extremely severe BBCI can fall into profound early shock immediately after the injury, particularly when associated with cranio-cerebral injury and hemorrhage of important organs. If they do not receive appropriate and effective treatment in time, the mortality will increase remarkably during the shock phase.

2.2 Serious Infection

The tissue and site of BBCI are conducive to the occurrence and development of systemic infection. Burn wounds, for instance, are an excellent culture medium for the growth and reproduction of various bacteria, which is the main extrinsic pathway of systemic infection; furthermore, the damages to the lung, bronchus, and trachea caused by blast injuries, with extensive avulsion of tissues, hemorrhage and edema, extensive damage to the alveolar epithelium and endothelium, create the conditions for pulmonary and systemic infections; GI tract blast injuries represented as hemorrhage, edema, necrosis, even rupture and perforation, as well as the ischemic and hypoxic state of the intestinal mucosal epithelium during

shock make the intestinal translocation of bacteria early and severe, opening the door for the invasion of endogenous infections. In the meantime, the non-specific immune function of the body is markedly diminished, and the cellular and humoral immune functions are impaired in severe BBCI cases, thus more likely to cause serious traumatic and systemic infections. So, for a severe or extremely severe BBCI, infection occurs earlier, more severely, and lasts a long time, accompanied by a persistent fever and a high incidence of systemic infection, and mostly, a secondary shock.

According to experimental studies, 88.8%, 64.4%, and 58.3% of post-injury blood cultures were positive in dogs with extremely severe, severe, and moderate injuries, respectively. Animals with extremely severe and severe injuries began to show positive blood cultures within 1.8 and 2.2 days of injury. During the infection phase, their incidence of hypothermia was high, with 75% and 50% of animals with severe and extremely severe injuries developing hypothermia in the experiment, and all died.

BBCI also increases the severity of infection. In severe BBCI injuries, all kinds of infections are more serious than in single injuries, such as more serious infections in the skin burn wounds, pneumonia or lung abscesses, infections and perforations in the gastrointestinal contusion sites, and peritonitis. Moreover, it is prone to systemic infections, worsening the condition.

In addition to the factors including severe and extensive multi-site and multi-tissue injuries as well as bountiful cultures of hemorrhage and edema fluid, severe infections after BBCI are also related to a marked decrease in the body's ability to resist infection, which are chiefly manifested as:

1. The extensive destruction of the barrier roles of tissues and organs increases the chances of exogenous infections and the distribution of infection sources. Specifically, burns lead to the destruction of the skin barrier; primary blast injuries destroy the mucosal and tissue barriers of the respiratory tract and alveoli, the gastrointestinal tract, etc.; secondary blast injuries cause damages to the structures and barriers of corresponding tissues.
2. Tissue hemorrhage and edema provide a good culture medium for bacterial growth: heavy local plasmexhidrosis from burns, massive hemorrhage and edema fluids in the respiratory tract and alveoli due to blast injuries, local necrotic tissues caused by secondary blast injuries, hemorrhage and edema fluids, foreign bodies, etc.
3. The immunocompromised body is favorable for the occurrence and development of bacterial infections: the number of peripheral white blood cells (PWB) is reduced after a severe BBCI, and the non-specific immune function and role of the granulocyte are also largely inhibited and weakened; the lymphopoiesis in the spleen and lymph nodes is suppressed, and the number of lymphocytes is lowered. All this will undermine the immune function of the body. As the reticulocytes of the reticuloendothelial system (RES) phagocytize large amounts of red blood cells and cell debris, the body's ability to ingest and kill bacteria is greatly reduced.
4. The dysfunction of some organs also affects the body's ability to resist infections: degraded adrenal function slows down the response to infections, reduced hepatic function weakens detoxification, and renal disorder impedes the excretion of toxic substances from the body. These phenomena can diminish the body's ability to resist infections.

2.3 Serious Lung Injury and Respiratory Disorder

After a burn-blast combined injury, the shock wave causes laceration, hemorrhage, and edema to the lung tissue, and the scorching fume may also cause burns to the respiratory tract, thereby frequently resulting in severe primary lung injury and respiratory dysfunction. At the same time, post-BBCI shock and infection can lead to disturbance of pulmonary circulation and inadequate tissue perfusion, and the lung can develop changes similar to acute respiratory distress syndrome (ARDS), resulting in pulmonary collapse, interstitial and alveolar edema, and hyaline membrane disease among other lesions; cardiac dysfunction can exacerbate pulmonary tissue congestion and edema; the fluid retention in the body due to renal dysfunction can also lead to worsened pulmonary edema; the tissue thromboplastin activated by tissue trauma can boost the formation of disseminated intravascular coagulation (DIC); the lung, as a "blood filter," is exposed to a variety of harmful substances and mediators produced by the body after severe BBCIs, aggravating its pathological damage and dysfunction, such as activation of alveolar macrophages, leukocyte activation and intrapulmonary sequestration, the production and release of vasoactive substances, pro-inflammatory cytokines, and inflammatory factors that can cause secondary damage to lung tissues and notably worsen pulmonary hemorrhage, pulmonary edema, and respiratory disorder.

Pathologically, the main manifestations are different degrees of hemorrhage and edema in the tracheal and bronchial mucosa and submucosa, different extents of laceration, hemorrhage, and edema in the lung tissue, formation of pulmonary bullae, and the development of hemopneumothorax. Of these, the hemorrhage and edema of the lung are progressive in the early stages and manifest as varying degrees of hemorrhage, thrombus, and edematous fluids in the alveoli and bronchi, even filling corresponding alveoli and bronchi.

After the injury, the patients may present with chest tightness, labored breathing, coughing, bloody sputum, accelerated breathing, and dyspnea. On auscultation, there may be different extents of crepitus, moist rales, blistering rales, and increased or absent local breath sounds, and these manifestations are rapidly changing with the course of the disease.

Severe blast lung injury (BLI) is often indicated when severe dyspnea, cyanosis, and hemorrhagic bubble-like fluid from the mouth and nose are present, and there is always a marked decrease in arterial partial pressure of oxygen (PaO_2) and lowered oxygen saturation. X-ray chest examination may consist of only increased bronchovascular shadows in the early stages, followed by consolidation shadows, or even extensive cloudy shadows. Later lung infection and hyaline membrane formation may further exacerbate pulmonary dysfunction, which may include decreased PaO_2 and increased shunt volume.

Inappropriate treatment during the treatment of BBCI injuries will aggravate the pathological lesion and dysfunction of the lung, leading to rapid deterioration of the injury, and even death. The inappropriate therapeutic measures after a burn injury are: excessive, rapid transfusion of blood and fluids. Excess nonmicellar solutions are apt to dilute plasma proteins, causing a decrease in colloid osmotic pressure, and quick aggravation of pulmonary edema, and significantly affecting pulmonary ventilation and ventilation function; prolonged high-concentration oxygen inhalation can lead to "oxygen poisoning" and thus impair pulmonary ventilation.

2.4 Serious Damage to the Heart and Circulatory Function

The pathological changes in the heart after a BBCI injury are mainly manifested by coronary vessel aeremia, subendocardial hemorrhage, interstitial hemorrhage, myocardial fiber degeneration, necrosis, and even rupture. Myocardial fiber breakage can occur as an immediate effect of burns and blast injuries or secondary to myocardial degeneration and necrosis. Ultrastructural observations reveal gelation of myofibrillar proteins and myolysis, dissociation of intercalated discs between myofibers, myocardial interstitial edema, and edematous fluid separating capillaries from myocardial fibers.

Clinically, patients with severe BBCI injuries often immediately present with bradycardia, which gradually returns to normal, then tachycardia, often accompanied by arrhythmia, and myocardial systolic and diastolic dysfunction, a fast decrease in cardiac output, and possibly a marked fall in blood pressure; due to noticeable hypovolemia after the BBCI injury, an obvious drop in blood pressure, or even shock may occur. The electrocardiogram may show ischemic changes in the myocardium, including ST-segment depression and flat or inverse T waves.

In addition to the direct injury to the heart, the above changes may also be closely related to the existence of myocardial inhibitory factors, hemoconcentration, viscosity and rigidity of the erythrocyte membrane (RBCM), and a considerable increase in whole blood viscosity (WBV) after the injury.

In BBCI injuries, the burn wound, lung injury, and respiratory dysfunction are more prominent, and heart injury is thus easily neglected. At the same time, the injury and dysfunction of multiple organs can exacerbate cardiovascular impairment and dysfunction. Profound shock, for example, can worsen cardiovascular microcirculation disorder; pulmonary hemorrhage and edema and changed pulmonary artery pressure will further exacerbate cardiac dysfunction; renal dysfunction increases cardiac stress by causing fluid retention in the body.

2.5 Damage to the Liver and GI Tract

The action of shock waves can lead to varying degrees of laceration and subperitoneal hematoma in the liver of a BBCI case whereby intraperitoneal hemorrhage, delayed subperitoneal hematoma and rupture, acute abdomen, and even hemorrhagic shock occur. Pathologically, changes such as hemorrhage in the hepatic sinusoid, inflammatory cell infiltration, dilatation of the hepatic sinusoids, and cytoplasmic laxity and vacuolization of hepatocytes are seen. There is a marked reciprocal exacerbation of liver damage when the burn is combined with the blast injury. Laboratory tests may show a rise in plasma alanine aminotransferase (ALT) and aspartate aminotransferase (AST), with the increase in AST being more prominent, the extent of which is related to the state of the injury, but the reciprocal exacerbating effect of this abnormality is not obvious when the burn is concurrent with the blast injury.

After a BBCI, there are often clinical symptoms of the GI tract, such as apparent lack of appetite, or even apocleisis, and frequent hematochezia. When there is a gastrointestinal perforation, severe acute abdominal manifestations are detected, i.e., intense abdominal pain, abdominal tenderness, and abdominal rebound pain, and even board-like rigidity; the abdominal X-ray film shows subphrenic pneumatosis. Late-onset gastrointestinal perforations may occur in some patients. The pathological changes of the GI tract are primarily made up of mucosal hyperemia, hemorrhage, erosion, and ulceration; hemorrhage under gastric and intestinal serous membranes, hematoma formation, hemorrhage at the junction of the ansa intestinalis (intestinal loop) and mesentery, and gastrointestinal perforation. The ulcers are comparable to the stress ulcers that form in severely burned patients.

2.6 Renal Dysfunction

After a BBCI, although the shock wave can induce renal contusions, the incidence is relatively low. The determinants of renal dysfunction are mainly related to the size of the burn area, and the disorder is chiefly manifested by oliguria,

hemoglobinuria, and hematuria. The increase of blood urea nitrogen and non-protein nitrogen is closely related to the state of the injury. The pathological basis of renal dysfunction is primarily ischemic damage to the glomerulus, and the lesions and clinical manifestations are similar to those of burns. In the presence of secondary blast injury caused damages, corresponding lesions may occur.

Renal dysfunction in severe BBCIs is usually caused by a number of factors, including:

1. Fall of blood pressure during early post-injury shock or late septic shock, which decreases renal blood flow and lowers or even stops the glomerular filtration rate.
2. Serious burns and other injuries resulting in the destruction of countless red blood cells and blockage of renal tubules by a large amount of hemoglobin, ultimately leading to postrenal renal failure.
3. Serious systemic disturbance of blood circulation and decreased glomerular filtration rate due to the blood stasis of the kidney.
4. Kidney lesions causing a fall in the glomerular filtration rate. Glomerular ischemia reduces or even stops filtration, and heavy glomerular ischemia causes acute renal failure and uremia. The degree of renal ischemia is basically positively correlated with the increase of non-protein and urea nitrogen in the blood.

2.7 Hematopoietic Function and Peripheral Hemogram Changes

Swelling and focal lysis of hematopoietic progenitor cells (HPCs) are seen after a BBCI. The nucleated cells are reduced in number and become sparse within 3 days of injury, with the megakaryocytes being phagocytized. The proliferative state of the bone marrow varies depending on the state of the injury. In milder cases, the bone marrow is in an active proliferative state, constantly releasing mature granulocytes into the peripheral blood, and there is an increase in the number of leucocytes in the circulating blood. In severe cases, the bone marrow is in a state of maturation inhibition, peripheral blood leukocytes are progressively dwindling in quantity, the platelet counts in the peripheral blood are reduced by a large amount, and the phagocytic capacity of the peripheral blood leukocytes declines substantially.

The main manifestations of the peripheral blood in the case of a BBCI injury are:

2.7.1 Leukocytes

The leukocyte counts of the patients with BBCI are mostly increased, especially when accompanied by infection, with neutrophilia being the main cause. In severe cases, however, the trend is different, with most leukocyte counts decreasing rather than increasing. For example, in dogs with severe and extremely severe BBCI injuries, the incidence of decreased leucocyte counts was 75% and 35%, respectively, but the percentage of neutrophils did not fall. The changes in peripheral blood leucocyte counts were correlated with the severity of the injury, and animals that showed a marked decrease or increase in leucocytes had higher mortality and a shorter survival time.

In mild cases, the hemopoietic tissue of the bone marrow is largely hyperplastic, with an increase in nucleated cells, mature granulocytes, and peripheral blood granulocyte counts (PWBC), but no significant change in lymphocytes; this generally falls to a normal range around 15 days of injury. In moderate cases, myeloproliferation and PWBC growth are more pronounced, reaching about 200% of the normal on the first day of injury, which maintains at a high fluctuating level. The PWBC change is more complex in severe cases, and characterized as falling (drop after the injury, remaining below the pre-injury value), rising and falling (ascending in the first day of injury, then descending until below the pre-injury value), rising, falling, and rising again (going up in the first day of injury, down for several days, then up again), and rising (remaining elevated after the injury). The falling pattern and the rising and falling pattern suggest that the body's defensive response is low, the condition is critical, and the prognosis is unfavorable, while the other two types indicate that the body is still responsive and the prognosis is relatively good.

The mechanism for these changes in leukocytes may be related to the following: a sharp rise in leukocyte counts after a BBCI injury is probably associated with a strong stress reaction and the release of profuse corticosteroids. The marked decrease in leucocyte counts after injury may be related to copious leucocyte exudation from the traumatic surface and wound (e.g., large-area burns), a large number of leucocytes sequestered and gathered in the vascular beds of internal organs (more in the lungs, liver, and spleen) with a corresponding reduction in the number of leucocytes in blood circulation, maturation inhibition and delay of bone marrow hematopoiesis, focal necrosis in some bone marrow tissues, etc.

2.7.2 Erythrocytes

In moderate or below BBCI injuries, there is usually no obvious change in peripheral blood erythrocytes. In severe or above injuries, the hemoglobin values are generally elevated during the shock phase, more so than in blast injuries or burns; as the disease progresses, the hemoglobin values continue to fall, and anemia is often present. In extremely severe BBCI injuries, there is remarkable early hemoconcentration and an increase in hemoglobin, followed by profound anemia.

The mechanisms of anemia from BBCI include: (1) heat induced hemolysis during the burn; (2) blood loss due to primary and secondary blast injuries, which can aggravate anemia; (3) further destruction of red blood cells after the burn, often seen with abnormal and fragmented red blood cells, increased plasma hemoglobin contents, and increased urobilinogen excretion; (4) systemic circulatory disturbances during the BBCI, with a large number of red blood cells stagnating in the burn wound and viscera; (5) severe infection also accelerates and aggravates anemia.

2.7.3 Platelets

Severe BBCI injuries can lead to a continuous decline in platelet function and number, which in turn prompts a serious injury and poor prognosis. The decline in platelet number and function may be related to the massive depletion of platelets and the "bone marrow megakaryocyte phagocytosis phenomenon" after the BBCI injury.

2.8 Composition Effect of Burn-Blast Combined Injury

As BBCI is inflicted by both heat and shock waves, the heat effect is either superimposed on or offset by the shock wave effect, which differs partly from the pathophysiological and clinical manifestations of single factor-resulted injuries.

In burn-dominant BBCIs, the burns play a leading role in the overall traumatic condition and the pathophysiological process. The clinical course and outcome of these injuries depend mainly on the severity of the burns and are divided into shock, infection, wound repair, and rehabilitation phases; the clinical manifestations are mostly shock, signs and symptoms of respiratory system damage, and also severe local wound and systemic infection; in severe BBCIs, there is often a profound liver and kidney dysfunction.

In blast injury-dominant BBCIs, the blast injury has a great impact on early pathophysiology, and its pathophysiological process and clinical manifestation are closely associated with the severity of the blast lung injury, the progression of hemorrhage and edema in the lungs, the occurrence of hemopneumothorax, and the dysfunction of the respiratory system; the intraperitoneal hemorrhage, abdominal infection, and hematochezia are closely related to the parenchymatous organ fracture, gastrointestinal hemorrhage, edema, and perforation from blast abdominal injuries. Early deaths are mostly related to the profound hemorrhage and edema of the lung tissue.

As a general rule, the general condition of the BBCI is more severe than that of a single injury, and its severity cannot be explained by what is seen on the body surface. In experimental studies, it has also been observed that the early overall pathology of BBCI injuries is better compared to blast injuries and that the weight gain is more evident in the immediate post-injury period than blast injuries. This is probably related to the water and electrolyte retention in the body during BBCI injuries. It is also found that the sequence of blast injuries and burns has an effect on the degree of injury. For example, those who first get burnt and then inflicted by blast waves have less pulmonary hemorrhagic edema than those who have first sustained a blast injury and then a burn injury.

3 Clinical Features and Main Cause of Death from Burn-Blast Combine Injuries

3.1 Clinical Features

Burn-blast combined injuries have not only the clinical manifestations of burns and but also the basic features of blast injuries, and the pathophysiological processes of burns and blast injuries interact with each other to make their condition and course more complex and serious; the clinical manifestations and outcomes vary greatly with the organs and sites of injury between burns and blast injuries, as well as the severity of the injuries, which has a great impact on the timely and accurate diagnosis and treatment of burn and blast injuries.

The clinical course of most BBCIs is manifested by the course features of the burns, which are divided into four phases: shock, infection, wound repair, and rehabilitation. The clinical features and needs of each phase may be different from those of burns or blast injuries due to the differences in injury conditions.

Apart from the local symptoms and signs of burn wounds and blast organ injuries, the characteristic manifestations are concentrated in the signs and symptoms of shock and the respiratory and circulatory systems. The clinical manifestations often include chest tightness, chest pain, cardiac distress, arrhythmia, cough, frothy sputum, blood tinged sputum, pink frothy sputum, dyspnea, hypoxic cyanosis, cold extremities, and even signs of respiratory failure and cardiac failure; in patients with severe or above BBCI injuries, there is an apparent drop in PO_2 and oxygen saturation and potential liver and kidney dysfunction. The X-ray film may show early increased lung markings or patchy lung consolidation shadows, later widespread consolidation shadows, or even extensive cloudy shadows; some may present signs of hemopneumothorax. The electrocardiogram may reveal an increased heart rate, a tall, peaked P wave, ST-segment depression or elevation, a flat or inverted T wave, low voltage, and the like.

Mild BBCI is a combination of mild burns and mild blast injuries, featuring a small burn area and light lung damage, a

short clinical course and fast recovery, usually heals in 2–3 weeks of injury.

Moderate BBCI is often a combination of moderate burns and mild blast injuries or mild burns in a mild BBCI. The pathological damage is not too heavy, and the conditions have a mutually aggravating effect, but not significant, generally about 1 month to recover.

The majority of severe BBCIs is a combination of severe burns with mild or moderate blast injuries or severe blast injuries with mild or moderate burns; a few cases are moderate burns compounded with moderate blast injuries. Severe BBCIs are often accompanied by varying degrees of shock, serious clinical manifestations, and a striking effect of mutual exacerbation, with complex, rapid changes in the early stage; after aggressive management, most can be recovered in about 2 months of injury.

Extremely severe BBCI is an extremely severe burn concurrent with varying degrees of blast injuries, or an extremely severe blast injury with varying degrees of burns, and a severe burn with a severe blast injury. They are associated with profound shock, severe respiratory impairment, critical condition, quick and great changes in the condition, extremely serious clinical manifestations, and high mortality rates. The prevention of shock as well as pulmonary hemorrhagic edema and the maintenance of respiratory function are key and core to early treatment, while infection control, maintenance of vital organ functions, and coverage and repair of the wound surface are essential in the late stage.

After the burns and blast injuries are combined with each other, the worsening of the condition is manifested by shock, respiratory dysfunction, infection, and complications of the heart, lung, and other viscera. Hematologic changes and critical symptoms (apastia, failure, hypothermia, etc.) occur more frequently, earlier, quickly, and heavily, and the severity of the systemic condition is often difficult to explain by just burns or blast injuries.

3.2 Main Cause of Death

The death rate of burn-blast combined injuries is higher than that of a single injury and has a remarkable correlation with the condition of the injury, the organs involved, and the state of vital visceral functions. When the burn is the primary injury, death is related to the severity of the burn. The larger the burn area, the higher the proportion of deep burns, the greater the mortality, and the shorter the survival time; when the blast injury is the main cause, the mortality is closely related to the degree and progression of pulmonary hemorrhagic edema and the extent of damage to other visceral organs.

The cause of death in burn-blast combined injuries is intricate, which is the result of multiple factors Therefore, through the analysis of the main cause of BBCI death to grasp the key points, it will help focus on the prevention and treatment of each stage and improve the management level.

Burning and punching compound injuries are all caused by explosions, and the main causes of death are impact injuries, shock, infection, and other serious complications.

The BBCI victims who die immediately at the scene of the explosion die primarily of blast injuries. The dead people often have broken limbs, coronary artery aeremia, severe pulmonary hemorrhagic edema, serious hemopneumothorax, liver and spleen rupture, gastrointestinal perforation, facial injury, and foreign body blockage of the oral cavity and respiratory tract, or associated severe fragment wounds. There is no time or way to control these injuries and save lives through emergency medical treatment on the scene of the explosion.

The main cause of early deaths from burn-blast injuries includes severe pulmonary hemorrhagic edema, profound shock, and catastrophic tear of the parenchymatous organ. Some death analysis has shown that about 60% of early deaths are due to pulmonary hemorrhage and edema, about 15% from profound shock, and about 17% as a result of pulmonary hemorrhagic edema complicated by hepatic rupture. It follows that severe pulmonary hemorrhagic edema is one of the most important causes of early death from burn-blast combined injuries. The main reason for the occurrence of severe pulmonary hemorrhagic edema is the direct injurious effect of the shock wave and is also closely related to respiratory tract burns and hypoxia.

Shock is the principal cause of death within 4 days of injury, accounting for 66.1% of the mortality; acute renal failure ranks second, about 8.5%. From 4 to 10 days, the death of various infections accounts for 68.6% of total BBCI cases, with dysfunction or failure of vital organs followed closely behind. From 11 to 15 days, 87.54% of BBCI deaths are caused by infections.

Hence one can see that the main causes of death in the first few hours of a BBCI injury are severe pulmonary hemorrhagic edema, severe parenchymal organ rupture, coronary artery aeremia, and airway obstruction; later on, pulmonary hemorrhagic edema and severe shock; a few days later, infection and vital organ dysfunction or failure.

For this reason, the key to the treatment of burn-blast combined injuries is the maintenance of a patent airway, and the prevention and control of pulmonary hemorrhagic edema in the early stage, and then focus on the prevention and control of shock and infections, in addition to active treatment of burn wounds, maintenance of the functions of vital organs such as the heart, lungs, and kidney, and full mobilization of the body's anti-injury function and repair capacity, and promotion of the repair and healing of the wounds and injured organs.

4 Diagnosis of Burn-Blast Combined Injuries

The burn wound is evident in the diagnosis of burns, except for the early determination of the burn depth that is difficult for general surgeons. The diagnosis of blast injuries and the determination of the condition, however, are the diagnostic difficulties of burn-blast combined injuries. The following aspects should be noted during the diagnosis of burn-blast combined injuries.

4.1 Medical History

BBCI cases all have a history of being injured by explosion shock waves. As a result, in the diagnosis, the first thing to do is to understand the details of the explosion, including the type of explosion (weapon), the size of the blast energy, the distance from the blast center, other people nearby, etc. Take nuclear explosions as an example, the nuclear explosion equivalent and mode, the distance from the burst center, etc. In terms of megaton nuclear explosions, apart from burns due to a long distance, and blast injuries in well-protected conditions, the vast majority of the wounded will sustain burn-blast combined injuries; during 100,000 tons of nuclear explosions, BBCI occurs mainly at a long distance. Among the victims of FAE bombs, those in the vicinity of the cloud-bursting area are often susceptible to BBCI injuries.

4.2 Signs and Symptoms

For burn wash compound injuries must be detailed questioning of their injuries, symptoms and signs, and at the same time to conduct a careful whole-body examination, and when found to have abnormal suspicions should be carefully analyzed and examined to find out the main causes of life-threatening injuries, to give timely treatment and treatment.

For burns, the depth and area should be carefully examined and judged to determine the extent of injury and the countermeasure for treatment. For burn depths, the distinction should be made according to five degrees and five types; the ratio of the burn area to the body surface area can be determined quickly by the Chinese Rule of Nine in conjunction with the palm method. Attention should also be paid to the presence of special burns like respiratory tract burns and chemical burns, and the severity of the injury should be judged as well.

In contrast to body surface burns that are easy to detect, judge, and take seriously, the condition and extent of blast injuries are more difficult to judge, particularly because the symptoms of visceral blast injuries are not easily manifested, but likely to be covered up by the presentation of external injuries such as burns. The signs of the acute abdomen for instance can often be masked by the presentation of abdominal skin burns. Hence, they should be examined and analyzed with special care and attention. Hence, they should be examined and analyzed with special care and attention.

In physical examinations, attention should be paid to whether there are foreign bodies, bleeding, and secretions in the oral and nasal cavities, cyanosis and other signs of hypoxia, airway obstruction, etc.; whether it is associated with head injuries, while paying special attention to the presence of coma and its duration and degree, the pupil size and its reaction to light, changes in limb paralysis and reflexes, and whether there is meningeal irritation; whether there are rib fractures and hemopneumothorax in the chest, abnormalities in thoracic mobility, and changes in pulmonary consolidation and breathing sounds; the diagnosis of abdominal combined injuries focuses on determining whether there are entorrhagia, gastrointestinal perforation, and peritonitis, etc.; whether there are fractures and soft tissue injuries in the extremities.

4.3 Ancillary Examination

1. *Blood gas analysis and pulmonary shunt flow*: In severe BBCI cases, there is often a decrease in blood oxygen and arterial oxygen saturation, which is a reference for observing the development of injury; in blast lung injuries, the pulmonary shunt flow increases notably, and its changes are more sensitive than the partial pressure of oxygen and can reflect the degree of lung injuries in a large part.
2. *Imaging studies*: X-rays are of special value in diagnosing fractures, blast injuries to the chest (e.g., pneumothorax, hemothorax, pulmonary hemorrhage, and pulmonary edema), blast injuries to the abdomen (pneumoperitoneum, etc.), respiratory tract burns, and localization of foreign bodies. However, a CT scan is more sensitive and accurate.
3. *ECG*: ECG changes such as elevated P wave, low voltage, and ST-segment shift and inversion are often seen in burn-blast combined injuries, reflecting cardiac and pulmonary lesions to some extent, but they are non-specific.
4. *Auditory apparatus and hearing examination*: It should be included as a routine examination. The focus should be placed on damage to the tympanic membrane, ossicles, and middle ear, and electroaudiometry should be performed if conditions permit.
5. *Others*: When vascular air embolism is suspected, fundoscopy should be done. Ultrasound examinations are

appropriate for blast lung injury. EEG and cerebral hemogram can provide references. Lumbar puncture to measure cerebral pressure and check cerebrospinal fluid can be performed if necessary. Abdominocentesis has some value in confirming the diagnosis of abdominal injuries.

4.4 Determination of Injury Severity

4.4.1 Determination of the Severity of Burns

Burn Depth

There are a lot of classification approaches. Currently, four degrees and five types are adopted in China, namely I, superficial II, full-thickness II, III, and IV degrees.

1. First-degree burns: Histologically, burns can involve the outermost layer of the epidermis (stratum corneum), hyaline layer (stratum lucidum), granular layer (stratum granulosum), and occasionally the prickly layer (stratum spinosum), but the germinal layer (stratum germinativum) is intact, and active in proliferation and regeneration. Clinically, it is termed combustion erythematosa, locally dry, painful, slightly swollen, red, and free of blisters. After 3–5 days, it partially turns from red to light brown, and heals without scars when the epidermis shrinks, and peels off to reveal a red, tender, and smooth epithelial surface, yet there may be a short period of pigmentation.
2. Second-degree burns.
   (a) Superficial second-degree burns: Tissue damage through burns includes the epidermis and part of the papillary layer. The germinal layer is partially damaged, and epithelial regeneration can depend on the residual germinal layer, and skin appendages, such as the epitheliosis of sweat glands and hair follicles. It is also known clinically as blistering burn, with marked local redness, and vesiculation of varying size, containing yellow (or faint red) plasma-like fluids or protein-coagulated peptones. When the blister bursts, a reddish wound is visible with a soft texture, high temperature, nociceptive sensitivity, acute pain, and numerous dilated and congested capillary networks in a pulsatile or granular pattern. It heals without leaving scars scarring, except for pigmentation.
   (b) Full-thickness second-degree burns: The tissue damage induced by burns involves the area beneath the papillary layer, but some of the reticular layers remain. Due to the residual skin appendages like hair follicles and sweat glands in the dermis, the epithelium can still be regenerated and become an epithelial island for repairing the wound, which recovers spontaneously 3–4 days later. If infection occurs and destroys the skin appendages or epithelial island, the wound will require surgical implants to heal. Clinically, this is characterized by local swelling, a whitish or brownish yellow epidermis, and occasional small blisters. After removal of the necrotic skin, the wound is slightly moist, reddish or red-white, ductile, obtuse, low in temperature, and painful when plucking the hair on it. Red dots the size of pinholes or millet seeds may be observed; the presence of small vessels that are dilated and congested or embolized indicates a full-thickness second-degree burn.
   (c) Third-degree burns: The tissue damage from burns involves the entire skin. Since the skin and its appendages are completely destroyed, there is no source of epithelial regeneration on the wound. Wound repair depends on surgical implants or the ingrowth of the epithelium from the surrounding healthy skin. Clinically, it is known as escharotic burn. Local manifestations are pale, yellowish-brown, or scorched. In severe cases it is scorched or charred, the skin loses elasticity and is hard as leather when touched, the wound is dry, without exudate, cool, and painless to needle prick and hair plucking. A large and thick network of embolized vessels, like branches, can be seen.
   (d) Fourth-degree burns: Burns-caused tissue injuries are deep in the muscles, bones, or internal organs. The clinical presentation is similar to that of a third-degree burn, i.e., a yellowish-brown, charred, or charred wound, loss of consciousness, and limitation of activity.
3. Identification of burn depths (Table 1)
4. Classification of burn severity

   The severity of burns is generally divided into four categories: mild, moderate, severe, and extremely severe
   (a) Mild: Degree II and below, less than 10% of the body surface area.
   (b) Moderate: 11–30% of the body surface area, degree II or below, or 9% or less, degree III or above.
   (c) Severe: 31–50%, degree II or below, or 10–19%, degree III or above, or less than 31%, but with one of the following conditions: (1) serious systemic condition or shock; (2) combined injury (severe trauma, blast injury, radiation injury, chemical poisoning, etc.); (3) moderate or severe respiratory tract burns (involving the area below the larynx).
   (d) Extremely severe: More than 50%, or 20%, above degree III.

Table 1 Clinical identifications of burn degrees

Depth		Injury depth	Appearance features and clinical signs	Sensation	Hair plucking test	Temperature	Healing process
Degree I (erythematous)		The horny layer, hyaline layer, granular layer, etc., with intact basal layer	Apparent local erythema. Mildly red, swollen, heat, painful, blister-free, dry, no infection	Micro-HR, often a burning sensation	Painful	Marginal increase	Symptoms subside in 2–3 days, heal in 3–5 days, flaking, no scarring
II-degree blistering	Superficial degree I	The basal layer and even the papillary layer	Large blisters with a moist, bright red wound, after removal of the epidermis	Acute pain, hyperesthesia	Painful	Increase	If no infection, 1–2 weeks to heal without scarring
	Full-thickness degree I	The reticular layer	Thin fluid deposit under the epidermis, or small blisters, with a slightly moist, whitish wound after removal of the epidermis, sometimes many red dots or tiny vascular branches, and obvious edema	Pain, dysesthesia	Slightly painful	Low local temperature	Usually healed in 3–4 weeks, likely to leave scars
Degree III (escharotic)		Full-thickness injury, even involving subcutaneous fat, muscles, and bones	Pale or charred yellow, dry, leathery wound, with thick and large embolized venous branches visible in most areas	No pain, dysesthesia	Painless and easy to pluck	Localized chills	After 3–4 weeks, the scab will fall off and require a skin graft to heal, leaving a scar or deformity
Degree IV		Muscles, bones, organs	Charred yellow charred, dry, leathery, with thick and large embolized veins visible in most areas	No pain, dysesthesia	Painless and easy to pluck	Localized chills	Black, dry necrosis at 3–4 weeks; amputation (finger) or skin flap repair

4.5 Determination of the Severity of Blast Injuries

Usually, there are probably no obvious clinical signs or only mild chest distress, chest pain, or breath-holding sensations after mild blast injuries. In moderate blast injuries, rapid breathing and coughing may be present, and rales may be heard on auscultation. In severe cases or above, dyspnea, cough, hemoptysis, or blood tinged sputum may appear; the X-ray chest film may show prominent bronchovascular markings or nodular dense shadows with blurred edges, or sometimes faint, cloudy, or ill-defined dense shadows; reduced arterial partial pressure of oxygen, etc. In extremely severe cases, pink frothy fluid may exude from the mouth and nose. In the diagnosis of burn-blast combined injuries, it is necessary to know how the patient was protected and thrown up during the explosion, whether the injury was caused in open ground or a confined space. When the burn is accompanied by chest distress, dyspnea, coughing, hemoptysis, or frothy sputum, the presence of blast injury and its severity should be considered. The order of severity from namely outside worse than inside should always be noted. The tympanic membrane is particularly sensitive to the overpressure generated by the blast. So, if a blast injury is suspected, tympanic membrane perforation and rupture should be checked to provide corroborative evidence for the presence or absence of the blast injury.

During on-spot first-aid and early treatment, it is difficult to perform dynamic chest X-ray or CT examinations, or other more detailed clinical analyses due to the limitation of conditions and examination means. So, it is difficult to quickly and accurately diagnose the conditions and judge the prognosis. The extent and change of blast injuries can be quickly determined clinically by observation of tympanic membrane damage, respiration and cyanosis, chest auscultation, and oral and nasal secretions (Table 2).

Table 2 Quick clinical identification of blast injuries by degree

	Mild	Moderate	Severe	Extremely severe
Tympanic	Rupture	Rupture	Rupture	Rupture
Respiration	–	Rapid	Significant breathing difficulty	Extreme dyspnea
Cyanosis	–	–	Distinct	Severe cyanosis
Chest auscultation	–	Sporadic crepitus Or moist rales	Extensive moist rales	Extensive moist rales Even widespread bubbling sounds
Oral and nasal secretions	–	A little frothy fluids	Large amounts of pink Frothy secretions	Large amounts of pink frothy discharge, even from the mouth and nose

5 Treatment of Burn-Blast Combined Injuries

Generally speaking, patients with mild burn-blast combined injuries (BBCI) do not require hospitalization; patients with moderate BBCI injuries can all recover after treatment; most of the patients with severe BBCI injuries can recover after treatment; only a few patients with extremely severe BBCI injuries can recover after treatment. Because of injury complexity, there are many contradictions and difficulties in the process of treatment. As a result, the needs and characteristics of treatment may vary from case to case; in principle, attention should be paid to the relationship between major and minor injuries, and major and minor contradictions, and effective measures should be taken to improve the treatment effect.

5.1 Therapeutic Principles

1. Based on the principles of burn treatment, the active treatment of burn-blast combined injuries should fully consider the needs and contradictions in the treatment of blast injuries.
2. During early first-aid treatment, attention must be paid to an open airway, good ventilation, and air exchange and smooth oxygen supply.
3. Actively and scientifically prevent shock, appropriately control the infusion speed and the total fluids, try to use more colloidal fluids and less crystalloid solution, protect the cardiopulmonary function, strictly prohibit excessive fluid infusion and too fast infusion, and prevent and control the aggravation of pulmonary edema.
4. Take anti-infection measures earlier and in a targeted fashion while enhancing wound management, nutrition, and organism resistance.
5. Protect vital organ functions such as the heart, lungs, and kidneys.
6. Perform appropriate, timely specific management and treatment, including surgical treatment of blast injuries to the chest, abdomen, and viscera, management and skin grafting of burn wounds, and treatment of traumatic brain injuries, fractures, etc.

5.2 On-Site Treatment Principles

1. Eliminate the source of burn causing further injuries to the body. First, get away from the ignition source quickly. For example, immediately extinguish the flames on the victim using clothes, sheets, or blankets, quickly lie down and roll over to put out the fire, rinse the body continuously with cold water, or jump into a nearby pond. When there is napalm burning on the skin, cover it with a piece of wet cloth to keep out the air and prevent the spread of the sticky gel; when the flames are extinguished, cover the wound with a piece of damp cloth or wet clothes, wash off the fragments of yellow phosphorus, and place it in water to avoid re-ignition with air. Keep rinsing or soaking the burn site with clear, cold water (cold therapy) to stop the heat from progressing deeper, clear the burn wound, wash away harmful substances, and exert the significant pain-relieving effect.
2. Place the injured in a ventilated environment, and keep their airways open. Encourage the coughing and sputum excretion when the patients are conscious; conduct mouth-to-mouth resuscitation other than external cardiac massage for those who have stopped breathing; prevent glossocoma from obstructing the airway; perform tracheotomy when possible for patients with face and neck burns, severe dyspnea, or prolonged coma.
3. Stop bleeding and dress the wound to avoid further contamination and injuries. For those who have wound hemorrhage, bandage as soon as possible to stop bleeding; regarding chest wounds, tightly wrap with thick dressings to prevent the occurrence of pneumothorax; with respect to tension pneumothorax, carry out paracentesis immediately; wrap the burn wound with hydrophilic sterile dressings, or if this is not available, cover with sheets or blankets before sending the patient to a hospital.
4. In cases of hypotension or shock due to blood loss, give oral or intravenous fluids and antibacterial drugs.

5. Administer analgesics orally or by injection to prevent shock. But drugs with significant respiratory depression such as morphine or pethidine should be used with caution.
6. Obtain intravenous access, and infuse electrolyte solutions for those with severe burns or profound shock when conditions permit.
7. Prevent bumps and reduce movement during transportation; keep the patient in a semi-recumbent position during transportation if they cough of pink frothy sputum, have ruptured tympanic membranes, and are bleeding from the mouth and nose.

5.3 Early Treatment Principles

1. The key to the early treatment of BBCI injuries is the maintenance of airway patency and oxygen supply, effective and appropriate fluid therapy, and close observation and treatment of pulmonary hemorrhagic edema along with other reasonable specific treatments. For different types of critical injuries, the treatment process is based on the principle of saving lives and an "upside-down" triage (the most severely injured arrive after the less injured, who bypass EMS triage and go directly to the closest hospitals). Injuries affecting respiratory and circulatory functions and vascular and visceral injuries should be treated with priority.
2. Bed rest: Any patient suspected of a BBCI should stay in bed to reduce the cardiopulmonary burden and prevent aggravation of hemorrhage.
3. Keep the airway open: The patients with respiratory distress should be kept in a semi-recumbent position, and tracheal and bronchial secretions should be aspirated promptly. If there are lots of pink frothy fluids in the trachea, or signs and symptoms of severe upper airway obstruction, or risk of asphyxia, tracheotomy should be performed as soon as possible; when bronchospasm occurs, aminophylline can be given or the cervical vagus nerve closed to relieve the spasm.
4. Oxygen inhalation: For the patients with respiratory distress or a tendency to lower partial pressure of oxygen, oxygen should be uptaken via a facial mask or nasal intubation. It is impossible to correct hypoxemia after oxygen inhalation, assisted mechanical ventilation (AMV) should be employed. Hyperbaric oxygen therapy can be given to patients with air embolism.
5. Careful examination and timely management of closed visceral injuries, and immediate treatment of injuries affecting respiratory and circulatory functions, and life-threatening vascular and visceral injuries. In the presence of serious cranio-cerebral injuries, combined thoracoabdominal injuries, macrovascular injuries, open fractures, etc., an emergency operation may be performed in accordance with the first-aid principle as required by each specialty.
6. AMV: Concerning BBCI evoked hypoxemia, intermittent positive pressure breathing (IPPB) is generally adopted to effectively improve the alveolar tidal volume, increased airway resistance, and baby lung from blast lung injuries, which can serve to effectively promote alveolar ventilation, reduce physiological dead space and pulmonary shunt flow, and improve oxygenation. If IPPB does not result in an arterial oxygen tension of 80 mmHg, continuous positive pressure breathing (CPPB) may be considered instead. However, it should be noted that the use of IPPB and CPPB is generally regarded to be contraindicated or should be discontinued in those with pre-existing air embolism or when air embolism develops during treatment. Some researchers recommend high-frequency ventilation therapy because it provides low tidal volume and airway pressure and cuts down the risk of air embolism formation.
7. Anti-shock therapy: Based on the principles of anti-shock fluid therapy after burns, active and cautious fluid resuscitation therapy is given for hemorrhage and pulmonary hemorrhagic edema of blast injuries and other combined injuries. The fluid therapy must be active and appropriate under the conditions of closely monitoring the pulmonary edema and the changes in brain function. Excessive and rapid infusion of fluids is strictly prohibited. Colloidal solutions are more suitable to prevent the worsening of pulmonary and cerebral edema.
8. Hyperbaric oxygen therapy (HOT): Regarding severe BBCI casualties associated with hypoxemia or air embolism, hyperbaric oxygen therapy is effective in improving hypoxemia, treating air embolism, and improving lung function. High pressure at 6 atm (the oxygen should not exceed 2.5 atmospheres for fear of toxic effects on the lungs and brain) lasts for 2 h, followed by 36 h decompression; the casualty can also be rapidly placed in the air at 6 atm and decompressed according to symptom relief; treated with 100% O_2 instead when reduced to 2.8 atm, and 100% O_2 intermittently during subsequent decompression. To improve tissue oxygenation, and lower the incidence of decompression sickness, the decompression time can be shortened.
9. Prevention and treatment of pulmonary edema and protection of cardiac function. Dehydrants, diuretics, and cardiotonics may be given, and early high dose of corticosteroids is beneficial in alleviating pulmonary edema.
10. Sedation and analgesia to mitigate pain and dysphoria: Sedative and analgesic treatment with pethidine, promethazine, and lytic cocktail can be given to alleviate the intense stimulation of the central nervous system resulting from burn pain. Drugs like morphine with

severe respiratory depression effects should be used with caution.
11. Prevention and treatment of infection: The principles of burn wound management in conjunction with systemic use of antibiotics are recommended.
12. Treatment of burn-blast combined injuries by the principles of specific diagnosis and treatment.

5.4 Fluid Therapy

In the course of fluid therapy for burn-blast combined injury, because the pathophysiology of burn shock is complicated by both severe primary lung injury and hemorrhagic edema from blast injuries, its contradiction in anti-shock therapy must be taken seriously. The principle of treatment for burn shock is active and rapid transfusion of blood and fluids to expand blood volume; however, when there is a moderate or higher blast lung injury often accompanied by pulmonary hemorrhage and edema, excessive fluid transfusion can exacerbate pulmonary edema. Thus, in the anti-shock treatment of burn-blast combined injuries, it is necessary to closely observe the changes in the lung while infusing fluids, but it is unnecessary to refrain from active anti-shock for fear of pulmonary edema, because appropriate and reasonable infusion will not significantly aggravate the occurrence and development of pulmonary edema. The transfusion volume can be increased or decreased as appropriate by referring to the formula for burns, and colloidal solution can be used whenever possible.

The principle of fluid therapy for BBCI should be an individualized fluid infusion plan of "replenishing as much as is lacking." The volume is calculated in line with the area of degree II or above burns and the body weight of the patient; the volume and rate of infusion should be adjusted in the course of treatment according to the patient's basic vital signs and changes in urine output and the development of pulmonary edema,

The basic infusion volume is calculated using "China's universal formula" for burn fluid therapy, i.e., the first 24 h infusion volume: 1.0 ml/kg of 1% BSA^{-1} electrolytes, 0.5 ml/kg of 1% BSA^{-1} colloids, 2000 ml of 5% or 10% sugar solution; the second 24 h: 0.5 ml/kg of 1% BSA^{-1} electrolytes, 0.25 ml/kg of 1% BSA^{-1} colloids, 2000 ml of 5% or 10% sugar solution. Half of the first 24 h infusion volume is given within 8 h of injury, and the remaining half is given in the second 16 h. The proportion of colloidal fluids may be increased if conditions permit.

During field operations, if you are not familiar with the "China's universal formula," you can refer to the "simple fluid replacement formula for adult sustaining burns under field operation conditions" as a guide on rehydration, and then adjust it according to the specific conditions of the casualty. The formula is as follows: total fluid (ml) in the first 24 h = burn area (%) × 100 ml ± 1000 ml (add 1000 ml for overweight, subtract 1000 ml for underweight). Among these fluids, 2000 ml is normal saline (sugar solution), and the rest can be given as 1:1 or 1:2 colloids and electrolytes; in the second 24 h, the amount of colloids and electrolytes is halved, and appropriately added or subtracted.

If there are difficulties with a fluid infusion at the frontline or during the patient evacuation, an early one-time hypertonic saline resuscitation in combination with delayed, continuous fluid infusion can be used: a one-time infusion of a 7.5% NaCl/6% dextran 70 solution according to 3–4 ml/kg body weight (intravenous therapy, 5 min) in the early post-injury period can quickly and effectively restore the blood pressure, enhance cardiac function, resume cardiac output, and improve tissue and organ blood perfusion. The anti-shock effect is fast-acting and can last for 2–5 h; when conditions permit, the formula for fluid infusion after burns can be used for resuscitation, but attention should be paid to whether the patient has traumatic active hemorrhage during treatment to prevent massive blood loss due to post-treatment blood pressure recovery; in addition, because hypertonic solution treatment has a prominent diuretic effect, the amount of rehydration should be appropriately adjusted in accordance with specific conditions at a later stage, and the urine volume maintained at about 1 ml/(h kg).

It needs to be emphasized that the infusion volume obtained using the above computational method is only a guideline, and the specific volume for each casualty should be judged and adjusted according to its anti-shock effect and injury condition (including pulmonary hemorrhagic edema). Clinical indicators to determine the adequacy of the infusion volume for BBCI injuries include the following:

1. *Mental state*: Quiet and cooperative indicates basic recovery of blood volume; irritability is often the evidence of insufficient blood volume or hypoxia. In addition to continued rehydration, it is necessary to exclude airway obstruction or cerebral edema.
2. *Skin*: Normal color, the warmth of limbs, and fast filling response when the nail is pressed signify good peripheral circulation.
3. *Heart rate and blood pressure*: The heart rate should be below 120 beats/min, the systolic pressure above 12 kPa (90 mmHg), and the pulse pressure above 2.7 kPa (20 mmHg).
4. *Urine volume*: The average urine volume per hour for adults should be more than 30 ml, and infusion should be accelerated if it is less than 20 ml; if more than 50 ml, the infusion should be slowed down. Hemoglobinuria requires a high urine volume and urine alkalization.
5. *Central venous pressure (CVP)*: It mirrors the relationship between cardiac output and returned blood volume.

The normal value is 0.49–1.5 kPa (5–15 cmH_2O). Low CVP, blood pressure, and urine output often reflect insufficient blood volume. The infusion should be decelerated if the CVP increases.

6. *Hemoglobin, hematocrit, dielectrics, and acid-base equilibrium*: They should all fluctuate within normal ranges.

5.5 Other Specific Treatment

5.5.1 Early Management of Burn Wounds

Early Debridement

The debridement of burn wounds should be carried out while resuscitating to prevent shock and after the casualty's general condition has stabilized. The choice of debridement methods should be based on the treatment after debridement, i.e., bandaging, exposure, or immediate surgery. For wounds to be bandaged, a more detailed approach should be used; for wounds to be treated with exposure therapy, debridement should tend to be "simple." Cut off the hair around the wound, and trim the finger (toe) nails if the foot and hand are burnt; remove foreign bodies stuck on the wound; if the wound is heavily polluted or has been coated with ointment, first wipe and rinse gently with decontaminant and clean water, then wash with bromogeramine and normal saline, and dry the wound surface with sterile gauze. It is not demanded to remove sand and coal cinders that get caught in the wound, but intracutaneous foreign bodies on the face should be removed as thoroughly as possible for fear of leaving permanent pigmented marks after healing. If the burn blister has ruptured and the vesicle skin is crumpled, it should be cut off; small blisters should be reserved, whereas large blisters should be drained by cutting small openings near the bottom of the blister after disinfection or by aspirating the fluid inside with a syringe; the intact blister skin does not need to be torn off, as it has a good protective effect on the wound that helps reduce water evaporation, relieve pain, avoid the wound from getting deeper because of dryness, protect it from contamination, and lower the probability of bacterial infection. The necrotic epidermal tissue at the site of burn above degree III should be removed; if not, the crust will not dry easily, and the necrotic tissue will be susceptible to infection when in a moist state.

Eschar Incision and Decompression

Because the eschars formed by the ringlike deep burns of the limbs, neck, and trunk are inelastic, and with the swelling of the burn site increasing, the eschar will bind the local formation of annular tension, resulting in a vicious circle of venous return disorder, further capillary exudation, and more prominent swelling, which will mechanically block the arterial blood flow and cause arterial reflex spasm, and may even develop into limb compartment syndrome, finally leading to muscle or limb necrosis. The cricoid eschar in the neck may compress the trachea to cause dyspnea or compress the jugular vein to increase the intracranial pressure and trigger encephaledema.

Therefore, the eschar should, in principle, be removed early once compression symptoms appear. If the burn area is large, the compression symptoms appear early, and it is inconvenient to cut out the scar, escharotomy should be performed locally to release the cricoid eschar, and avoid possible complications.

During the surgery, iodine, alcohol, or povidone-iodine can be applied to disinfect the incision, which should be made through the entire length of the third-degree and fourth-degree burns, to a depth that reaches normal soft tissues, usually to the plane of deep fascia or sarcolemma; if the subfascial tissue is stiff, there is great subfascial tension, or the subfascial intermuscular space pressure exceeds 4 kPa (30 mmHg), the deep fascia should be cut open for decompression.

Treatment of Burn Wounds

Surgical or non-surgical therapy, as well as anti-infection treatment of the wound and body, is carried out as per the wound and injury condition, and by the principles of the burn department.

5.5.2 Treatment of BBCI Accompanied by Soft Tissue Injuries

The associated soft tissue trauma is treated promptly in accordance with the principles of wartime trauma management, including early debridement, wound rinsing with cleaning solution, removal of foreign bodies and necrotic tissues, incision of involved fascia, and excision of necrotic muscle tissues. If there is no obvious tissue necrosis in the wound, it is not necessary to remove all foreign bodies by extending the scope of debridement.

After debridement, if there is no burn wound at the wound, bandaging and delayed suturing can be performed in general; facial tissues with rich blood circulation and strong repairing power can be carefully sutured after debridement and given antibacterial drugs to prevent and control infection.

If the traumatic wound is located within the burn area, it is usually not bandaged as the bandage may aggravate the infection, but can be coated with an effective antibacterial agent. If the wound is large, it can be implanted with a homogeneous skin, and further treated as appropriate.

5.5.3 Treatment of BBCI with Fractures

In the case of BBCI with fractures, if the burn and fracture do not occur at the same site, they should be managed according to the condition of the injury and the order of emergency, in addition to the accentuation effect of the BBCI. If the fracture

and the burn occur at the same site, the treatment is often contradictory and difficult and thus should be handled in the order of priority and urgency according to the condition of the injury.

1. *Debridement*: When the BBCI is accompanied by an open fracture, the burned wound is prone to infection at the fracture site. The necrotic tissue should be removed as much as possible, the dead space eliminated, and the bone surface covered with soft tissues; the wound should remain unobstructed and covered with antibacterial drugs. At the same time, anti-infection measures should be taken to prevent the occurrence of osteomyelitis and systemic infection.
2. *Diaplasis*: The BBCI casualties are less tolerant of early fracture reduction, which can easily induce shock. When the reduction is assessed to have a great impact on the casualty, it can be postponed. If the reduction needs to involve the burned area, the burn wound should be properly and temporarily wrapped to avoid further injuries. When it needs to be cut open, the incision should bypass the burn area in case of infection; if it cannot be avoided, the burn area can be implanted with the homograft to control inflammation before reduction of the fracture, which can otherwise be performed after the burn area heals.
3. *External fixation*: When the fracture area is associated with burns, the use of a small splint for local fixation is good for the early movement of the joint adjacent to the fracture site, fracture union, and functional recovery. It can be used according to the condition of the injury, drainage, or other treatment as needed. Since plaster immobilization is easy to cause infection to the burn wound, it is likely to aggravate tissue necrosis, inconvenient to observe the wound on demand, and increases the difficulty in diagnosis and treatment; apart from this, when the early tissue edema in the burn area is significant, plaster dressing will increase the pressure in the limb and affect blood circulation, easily leading to limb ischemia and even necrosis. So, plaster fixation is usually not recommended, unless there is no other short-cut alternative. Anyhow, plaster fixation is still one of the effective and convenient immobilization methods in wartime. If in use, a longitudinal incision should be made after the plaster is put on, the tightness adjusted at the right time, and a plaster window cut for observation.
4. *Sustained traction*: The merits of skeletal traction are that the fracture reduction is stable, the burn can be managed with exposure therapy, the wound can be observed at any time, and it is conducive to changing fresh dressings and performing procedures such as escharectomy and skin grafting. Thus, it can be considered when the external fixation method is not appropriate. This method also has a number of weaknesses: the traction pin hit from the burn tends to cause pin site infection, osteoporosis, pin position movement, etc. In addition, most cases need to be bedridden for a long time, and the healing of back burns can be affected by long-term compression and infection.
5. *Treatment of exposed bone surface*: When it is associated with an open fracture, bone end exposure is generally not handled for the time being, and the bone surface layer is cut down until bleeding after the recovery of surrounding burns. when it is covered with granulation tissue, the wound is closed by the homograft and autologous skin graft. the surface layer of the bone is usually ablated without perforation. this is because the bone tissue between the holes is prone to necrosis, and the hole can easily lead to the bacterial invasion of the marrow cavity, increasing the probability of complications.

5.5.4 Other Specific Treatment

In cases with ruptured eardrums and tympanum hemorrhage, remove the foreign bodies from the external auditory canal, and keep it dry. Do not place oily drops in the ear, rinse the outer ear, or blow the nose forcefully, except for antibiotics to prevent otitis media.

When the BBCI is accompanied by brain injury, anti-shock fluid therapy and management should be reinforced, and the amount of rehydration should be limited to the extent that it can survive the shock. Head-up tilt is recommended to help venous return and reduce intracranial congestion. For patients with encephaledema accompanied by increased intracranial pressure, hypertonic glucose solution or mannitol can be given for diuresis and decompression according to conditions. When the open brain injury is associated with burns, the dura should be closed earlier, effective antibacterial drugs applied to control the infection, the symptoms of intracranial infection closely observed, and correct specialist treatment given in time.

When associated with abdominal injuries, the patients confirmed with entorrhagia or cavernous viscera damage accompanied by peritonitis should be first given surgical treatment on the basis of anti-shock measures. The surgical incision should be made on skin free of burns. Delayed sutures are recommended to close the incision on the skin and subcutaneous layer. If the defect at the incision is large, a homograft can be implanted. When an abdominal visceral injury is suspected, fasting is required, and if necessary, diagnostic laparotomy should be performed.

Among BBCI cases, if severe hemoglobinuria is caused by major burns, and when there is massive myohemoglobinuria with crush injuries, osmotic diuretics (e.g., mannitol) should be given to maintain the urine output and prevent renal tubular necrosis. After diuretics have been administered, the urine volume should no longer be used as an indication of adequate or inadequate rehydration.

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Projectile-Blast Combined Injury

Jianmin Wang

1 Overview

Various explosive weapons such as bombs, missiles, artillery shells, and landmines are the most commonly used lethal weapons in modern warfare. Fragments and blast waves are the main factors of modern explosive weapons leading to injuries and deaths. According to the statistics of several local wars in modern times, the incidence of fragment-induced injuries was 53–81%. However, with the development of modern weapons and ammunition technology, in addition to the damage caused by fragments of explosive device, blast injuries caused by blast waves often occur simultaneously in the vicinity of the detonation. Sometimes blast waves can even play the main role, so that it is common to see combined injuries caused by fragments and blast waves.

Projectile-blast combined injury (also known as fragment-blast combined injury) refers to the combined injury caused by high-speed projectiles (gunshots, fragments, etc.) and blast waves, common to see in various injuries caused by multiple explosive weapons, since blast waves and fragments of explosive weapons are the main reasons for injuries. Projectile-blast combined injuries are mainly manifested as an enhanced effect of blast waves by fragments, so that severe damage to important target organs such as lungs and brain often occurs combined with open injuries caused by fragments.

2 Pathophysiology of Projectile-Blast Combined Injury

Projectile-blast combined injuries are more severe than a simple blast injury or a simple fragment injury, where the main aggravation part is mainly localized in lungs. However, the aggravation mechanism of projectile-blast combined injury is not yet fully understood. Some experiments have shown that high-speed projectiles could cause damage to not only the tissues and organs impacted directly but those distal to the trajectory caused by fragments, which mostly occur in the heart, lungs, and brain. Therefore, damage of high-speed projectiles to the organs and tissues distal to the impact site may be an important factor for high-speed fragments to aggravate blast injuries. Blast lung injury can be aggravated by pressure waves of high-speed fragments and some harmful substances from the wound tract when the normal structure of lung tissues is destroyed by the blast waves. In addition, in fragment injury and fragment-blast combined injury, aggregation and obstruction of platelets and white blood cells are more often to see in the pulmonary microcirculation, indicating the hyperfunction of platelets and white blood cells during projectile-blast combined injury, which plays a non-negligible role in the lung injury after a fragment injury. In general, the pathophysiological changes of projectile-blast combined injury mainly include three aspects, i.e. biochemistry, hemodynamics, and physical response.

2.1 Biochemical Effects

Thromboxane A2 (TXA_2) and prostacyclin (PGI_2), metabolites of arachidonic acid oxidase, are closely related to lung injury. The functional status of pulmonary vascular endothelial cells can be reflected by 6-keto-PGF1α and TXB_2, the stable metabolites of TXA_2 and PGI_2 in plasma and lung tissues. There was an increase in TXB_2 in plasma and lung tissues during the projectile-blast combined injury, which was higher than simple blast injury, and so was that in fragment injury. Prostacyclin (6-keto-PGF1α) increased after blast injury and fragment injury, but that in plasma decreased after projectile-blast combined injury. The results above indicate that pulmonary vascular endothelial cells may be damaged after fragment injury and blast injury, while the decrease of

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prostacyclin indicates that the projectile-blast combined injury was more serious so that the synthesis of prostacyclin in endothelial tissue was inhibited. Therefore, the damage to pulmonary vascular endothelial cells is an important pathophysiological phenomenon of blast injury, fragment injury, and fragment-blast combined injury.

2.2 Hemodynamic Response

Liu Jiancang et al. have observed the hemodynamic changes of fragment injuries, blast injuries, and fragment-blast combined injuries under high altitude conditions. The results showed that after the injury under plain conditions, the mean arterial pressure (MAP) of pigs increased transiently in the early stage and then tended to decrease, while the central venous pressure (CVP) tended to decrease; after the simple blast injury and simple fragment injury, the right ventricular systolic pressure (RVSP) decreased significantly, while the cardiac output (CO) increased significantly; RVSP and CO after the combined injury were significantly higher than those of the simple blast/projectile injury. Under high altitude conditions, after the injury, RVSP increased significantly, CO decreased significantly, and CVP and MAP tended to increase. Therefore, it is believed that fragment injuries can significantly aggravate blast injuries, while in the hypoxia hypobaric conditions of high regions, blast injuries, fragment injuries, and combined injuries could be more significantly serious than those in the plain conditions, with worsened hemodynamic damage, which were more significant in combined injuries.

2.3 Physical Response to Projectile-Blast Combined Injury

For explosive weapons, fragments and blast waves are the main factors to injury. Due to their different physical characteristics in the air, the range of injury caused by fragments and blast waves may be different. Because the speed of the blast waves which exceeds that of the fragments at the moment of the detonation decays much faster than that of the fragments, the fragments will catch up with the blast wave front after a distance of about 22 times the charge radius. After that, the speed of the blast waves continues to decay rapidly, while the fragments still fly forward at a faster speed. Therefore, the impact range of the fragments always covers that of the blast waves, often accompanied by a combined injury effect of the fragments as well as the blast waves.

The aggravation mechanisms of the projectile-blast combined injury mainly involve two aspects, that is the pathophysiology and the physical mechanics. Firstly, fragments and blast waves, as two injury-causing factors, can cause injury to organs with different sensitivity, thereby aggravating the injury effect. Secondly, simultaneous action of fragments and blast waves on the body can cause serious tearing effects on tissues. Fragments that act on the body tissues can destroy their structure where the strength of the tissue structure is significantly reduced, so is the resistance to blast waves. Therefore, lower blast wave overpressure can lead to severe tearing.

3 Clinical Features of Projectile-Blast Combined Injuries

With the development of explosive weapons and munitions, the damage scope of explosive blast waves has gradually expanded, and the proportion of projectile-blast combined injury has also increased. According to reports, the incidence of explosive fragment injuries was 53–81%, and the incidence of blast injuries was 30.0–50.4%. An Bo et al. who have observed the fragment and blast injuries in sheep during a certain type of grenade static explosion have found that the incidence of fragment and blast injuries were 69.44% and 52.8%, respectively. Under the static explosion of a 30 kg grenade, the average incidence of projectile-blast combined injury was 47.06%.

In general, the projectile-blast combined injury by explosive weapons mostly occurs in the near field. Therefore, it generally has the following characteristics:

1. High overall incidence: In modern warfare, due to the increasingly wide use and enhanced power of explosive weapons, the damage scope of explosive blast waves has expanded, significantly raising the incidence of projectile-blast combined injury. Previous experimental studies have shown that the incidence of projectile-blast combined injury can reach 47.06%.
2. High incidence of multi-site trauma and polytrauma: Because projectile-blast combined injury mostly occurs in the near field, and, more fragments are always distributed within the near field of the effective damage scope of the blast wave, multiple fragment injuries are prone to occur. When analyzing 1155 terrorist-related casualties from the National Israel Trauma Registry (ITR) from October 1, 2000 to September 30, 2002, Peleg et al. have found that there were more injury locations in patients with blast injuries, and 62% had multiple-site trauma.
3. Severe conditions and high mortality: In the study of combined injury caused by grenade bombs, Yang Zhihuan et al. have found that among 16 experimental animals

with combined injuries, except one without lung injury, the other 15 had severe to extremely severe lung injury. Besides, there were 4 cases of lung rupture, 10 cases of stomach perforation, 1 case of small intestine perforation, 10 cases of liver rupture, 4 cases of dismemberment, and 6 cases of body cavity rupture. Regarding the cause of internal organ damage and rupture, the proximal part of the detonation center may be mainly related to the blast wave, while the distal part may be mainly caused by the fragments. Among the 16 animals, except one that survived, all the others died at the scene or within 24 h after injury. Causes of death included severe pulmonary hemorrhage and pulmonary edema, rupture and internal hemorrhage of liver and kidney, gastrointestinal perforation, etc.

4. More common infections, limb defects, and destructive injuries: Since near-field blast injuries are usually combined injuries infected by ground gravel and other pollutants, the incidence of infection is high. Meanwhile, the fragments in the near field are not only dense and fast, but also accompanied by blast waves, so the strong tearing effect on soft tissues could cause more destructive injuries such as limb defects.
5. Prominent treatment contradictions: In the projectile-blast combined injury experiment, in addition to varying degrees of pulmonary edema, the wounded of projectile-blast combined injury also had severe injuries, such as body surface injury, gastrointestinal perforation, and liver rupture, which urgently require immediate resuscitation, while improper resuscitation can aggravate pulmonary edema and promote death. Therefore, the contradiction in treatment is quite prominent.

In terms of individual manifestations, fragments could aggravate the blast injury. Huang Jianzhao et al. have found that lung projectile-blast combined injury was one grade more severe than simple blast lung injury. In the experiment, they used dogs with moderate blast injury combined with high-speed fragment injury in the unilateral hind limb and found that after blast injury, fragment injury, and the combined injury, the partial pressure of oxygen (PaO_2) decreased, while pulmonary artery pressure (PAP) increased, where the change after combined injury was the largest. In addition, the lung body index and lung water content at 24 h after the combined injury were significantly higher than those after the blast injury and the fragment injury. Pathological observations showed that severe lung injury occurred in most of the animals with combined rupture injury, while moderate lung injury in those with blast injury and mild lung injury in those with fragment injury. Therefore, it is believed that high-speed fragmentation can aggravate the moderate blast lung injury.

4 Diagnosis of Projectile-Blast Combined Injuries

The key to the diagnosis of combined impact injuries focuses on the diagnosis of blast injury. Damages caused by fragments or bullets are usually easy to find, while blast injuries are often covered by fragment injuries, which results in missed diagnosis.

4.1 Medical History

The medical history inquiry mainly focuses on the approximate damage scope of the detonation in order to infer the approximate equivalent of the explosive. On this basis, further investigation should cover the location and direction of the wounded, his/her approximate distance from the detonation center, as well as the surrounding objects, terrain, etc. According to all the information above, the severity of the blast injury of the wounded can be estimated.

4.2 Symptoms and Signs

1. Observe the general situation: Since the severe situation of the projectile-blast combined injury and the characteristics of mild external manifestations but severe internal damage of the blast injury, examination of general situations (including consciousness, pain, cyanosis, etc.) and vital signs (including blood pressure, respiration, temperature, etc.) is one of the important ways to understand the overall injury of the wounded.
2. Examine the wound and wound tract: Since the complicated bullet or fragment injuries in the projectile-blast combined injury, focus should be put on the distribution, entrance, and exit of wound tract, and its relative location with the body cavity.
3. Explore thoracic and abdominal physical signs: Through a general physical examination, practitioners should understand whether there is local tenderness, lung consolidation, pneumoperitoneum, abnormal bowel sounds, etc.
4. Examine eyes, ears, and other special organs: By examining eyes and ears, doctors should understand whether there is accompanied ocular/auricular blast injury.

4.3 Hemogram and Biochemical Indicators

1. Complete blood count: Complete blood count can help understand whether there is accompanied serious infections and the blood loss volume.

2. Heart, liver, and kidney function: Myocardial enzyme spectrum and liver/kidney function tests can help understand the severity of damage to the vital organs.
3. Arterial blood gas analysis: In patients with projectile-blast combined injuries, the blood oxygen saturation (SaO_2) usually decreases, the arterial blood oxygen partial pressure (PaO_2) significantly decreases, and the arterial carbon dioxide partial pressure ($PaCO_2$) mostly shows a downward trend, while in patients combined with thoracic wall injury, the $PaCO_2$ often increases.
4. Related cytokines assay: Interleukin-8 (IL-8), interleukin-6 (IL-6), and tumor necrosis factor (TNF) are positively correlated with the severity of lung injury. Thus, the level of IL-8 can reflect the severity of the lung injury. Thomas et al. have found that plasma IL-8 level in severely injured patients was closely related to the occurrence of adult respiratory distress syndrome (ARDS). Yan Jiachuan et al. have also found that the IL-8 level in plasma and lung tissue after fragment injury and blast injury was significantly increased and was related to the severity of the injury. The IL-8 level in the projectile-blast combined injury group, compared with those in the blast injury group and the fragment injury group, was significantly higher, indicating a more serious situation.

4.4 Special Examinations

1. Electrocardiography (ECG): Myocardium can be damaged by the released harmful factors from the fragment injury or the explosion blast wave itself. Therefore, ECG can help to observe the severity of heart damage.
2. X-ray: Through X-ray, situations can be determined including hemopneumothorax, pneumoperitoneum, diaphragm rupture, metal foreign body residues, etc. Moreover, according to the entrance of the projectile and the location of the residual foreign body, the direction and location of the wound tract can be understood to obtain a primary impression of possibly damaged organs.
3. Ultrasonography: Ultrasonography has an important reference value for the judgment of the severity of internal hemorrhagic injury and pulmonary edema. Therefore, ultrasonography could be used for patients suspected of internal organ injury and blast lung injury.

5 Treatment of Projectile-Blast Combined Injuries

The management of projectile-blast combined injury should be carried out based on the rules for the treatment of war injury and combined with the characteristics of fragment injury and blast injury.

5.1 First Aid on Site

The management of projectile-blast combined injury should focus on ventilation, hemostasis, bandaging, fixation, transportation, and basic life support. Keeping the airway unobstructed is the first and foremost, and for the wounded with acute pulmonary edema, severe chest wall injury, and difficult expectoration, tracheotomy should be considered as early as possible. For those with significant active bleeding, effective measures should be taken to stop it. Open wounds should be bandaged with first aid kits. Fractures should be fixed by splints or take other effective measures for temporary fixation according to local conditions. The wounded should be transported after the on-site emergency management above.

5.2 Management Principles of Projectile-Blast Combined Injury

The management principle of projectile-blast combined injury should be based on the treatment of simple fragment and blast injury, and, meanwhile, combined with the characteristics of the combined injury.

1. Damage control surgery for the severely wounded: As there are many severely wounded with projectile-blast combined injury, it is necessary to carry out damage control surgery, mainly including compression hemostasis and bandaging for active bleeding and penetrating gunshot wounds, limited management for gastrointestinal rupture wounds, improving ventilation for blood pneumothorax, etc.
2. Early anti-infection: Explosive wounds have a high incidence of infection due to many pollutants and multiple blind tract wounds (i.e. non-penetrating wounds). Therefore, anti-infection treatment should be given as soon as possible, as well as intensifying the wound treatment, improving the nutritious status, and enhancing the patient's immunity.
3. Protection of the function of heart and lungs: Since blast waves are the main cause of damage to the projectile-blast combined injury, the lung is one of the main target organs. At the same time, due to the serious conditions, the complicated factors, and the multiple and high levels of harmful factors secreted into the body fluid, cardiac and pulmonary functions are more likely to be more severely damaged. Therefore, fluid therapy should be given as soon as possible to protect the functions of vital organs, such as the heart and lungs.
4. Surgical treatment for open wounds: Explosive fragments, another important factor of damage for projectile-blast combined injury, usually cause multiple and

complicated wounds. Proper treatment of open wounds can not only promote the healing but also play an important role in hemostasis and anti-infection.

5.3 Management of Special Injuries

1. Management of soft tissue defects in projectile-blast combined injury.
2. Management of projectile-blast combined injury with fractures.
3. Management of projectile-blast combined injury with craniocerebral injury.

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Combined Radiation-Blast Injury

Yongping Su and Tao Wang

Combined injury is a compound injury that occurs when the body is simultaneously or successively affected by two or more (including two) injury-causing factors of different nature. Among the injuries resulting from modern wars, combined injuries are often a vital feature; among disasters and serious accidents in normal times, the patients with combined injuries are always the important objects of treatment.

The attention drawn to combined injuries is closely related to the application of nuclear weapons in a war and the course of research on their biocidal effects. In 1945, the USA dropped atomic bombs on Hiroshima and Nagasaki, with the number of nuclear explosion casualties accounting for 36.8% and 22.4% of the two cities' population, respectively, among which the presumed incidence of various combined injuries reached 65–85%, leading to tens of thousands of casualties. Subsequent experimental researches on the biocidal effects of nuclear weapons have demonstrated that the burns, blast injuries, and radiation injuries to experimental animals are induced by three instantaneous killing factors, namely optical radiation, shock waves, and early nuclear radiation during the detonation of nuclear weapons, while a high proportion of injuries result from the combination of the aforementioned contributing factors.

Combined radiation-blast injury (CRBI) is a type of compound injury due to the simultaneous or sequential action of two different contributing factors, namely ionizing radiation and shock waves on the body. Ionizing radiation can cause radiation damage, while shock waves can cause blast injuries. The injury-causing effects of shock waves include primary and secondary impact effects. The injury arising from the primary impact effect is a typical blast injury belonging to the category of trauma but different from mechanical trauma and results in the bleeding and ruptures of the acoustic organ, lung, gastrointestinal tract, etc., or the hemorrhage of parenchymal organs such as liver and spleen. The secondary impact effect, however, is that injuries are caused by objects striking the body in the form of secondary projectiles due to the kinetic energy under the action of dynamic pressure, or from some buildings and structures collapsed and fallen due to shock waves, which is called indirect blast injury, mainly including body surface laceration, visceral hemorrhage, tearing, fracture, etc., similar to ordinary trauma. The proportion of combined radiation-blast injuries in the context of a nuclear explosion is high, but there are fewer experimental studies on combined radiation-blast injuries from primary impact effect relative to the radiation-burn combined injuries, burn-blast combined injuries, radiation-burn-blast combined injuries that have received widespread attention, while the combined radiation-blast injuries from secondary impact effect have aroused considerable interest in the form of "radiation-trauma combined injuries."

Of late years, with the heavy use of depleted uranium (DU) weapons on the battlefield and the potential realistic threat of nuclear terror raids typified by radiological dispersal devices (RDD, aka dirty bombs), the combined radiation-blast injuries or blast combined radiation injuries in new manifestation patterns need to be taken seriously. This chapter intends to provide a systematic discussion on the occurrence condition, classification, state, clinical feature, diagnosis and treatment, as well as research progress of combined radiation-blast injuries.

1 Conditions for Occurrence of Radiation Combined Blast Injuries

Combined radiation-blast injury (CRBI) refers to a type of combined injury, primarily a radiation injury, either simultaneously or sequentially caused to human bodies. An environment where both ionizing radiation and shock waves are present as causative factors can produce such injuries. RCBIs can occur in a variety of situations in wartime and peacetime.

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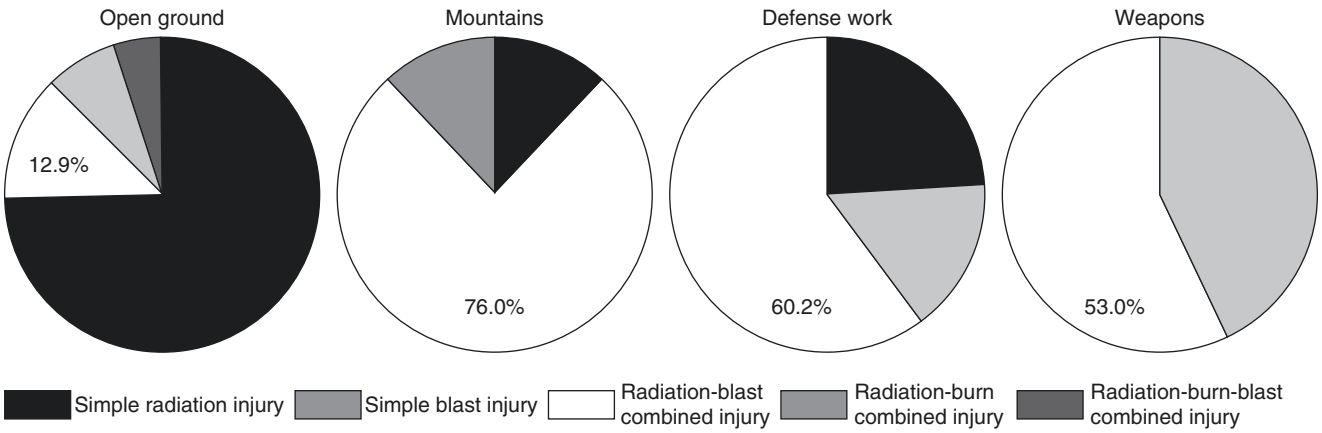

Fig. 1 Injury conditions and types of animals of a 10,000-ton nuclear weapon hitting the ground under different deployment conditions

1.1 Occurrence of Radiation Combined Blast Injuries in Wartime

Modern combat is an exceptionally brutal war based on high-technology weapons under the threat of nuclear weapons; one of its important features is the increasingly destructive and lethal effects of modern weapons. This leads to a variety of dangerous injury-causing factors in the battlefield environment that are prone to combined injuries. In wartime, RCBI injuries mainly occur when a weapon with multiple lethal factors such as ionizing radiation and shock waves is used, either simultaneously or sequentially.

1.1.1 CRBI Through Nuclear Weapons

Nuclear weapon explosion leads to four killing factors (early nuclear radiation, optical radiation, shock wave, and radioactive contamination), combined injuries from which are the main category of nuclear explosion injuries, and also the main object of treatment; among these injuries, CRBI has a high incidence. The statistical information collected after the nuclear attack inflicted on Hiroshima and Nagasaki shows that, if early deaths were included, the total combined injuries were 60–85% of all casualties; if the 20-day survivors were counted, combined injuries accounted for 40% and 42% of all casualties in the above two cities, respectively, of which CRBI had the highest incidence, making up 45.0% (Hiroshima) and 54.8% (Nagasaki) of the total. The animal effects of the surface burst of 10,000-ton nuclear weapons in China showed that of the 432 dogs injured under different conditions, 201 (46.5%) sustained combined injuries, 217 (50.2%) radiation sicknesses, and 14 (3.2%) blast injuries. All combined injuries were predominantly radiological combined injuries, with CRBI being the most frequent, accounting for 39.5% of all injured animals, and 84.8% of all combined injury animals.

The occurrence of CRBI from nuclear explosions depends greatly on the nuclear weapon yield, detonation approach, personnel distribution and protection, etc. Generally speaking, three types of combined injuries, such as radiation-burn-blast combined injuries, burn-blast combined injuries, and burn-radiation-blast combined injuries, occur mainly because of personnel exposure to nuclear explosions; CRBIs are inflicted on those inside fortifications, buildings, or large arms shielding the effects of optical radiation. When Hiroshima and Nagasaki suffered from nuclear attacks, some residents stayed indoors, and others, though outdoors, were still on the back of the building that prevented or mitigated the radiation of direct light the moment when the glaring fireball appeared due to nuclear explosion. On account of building collapse, and the high-velocity impact of glass, bricks and tiles, iron, and other debris, trauma increased to a large extent, so did CRBIs. The aforementioned animal effects of nuclear tests indicate that CRBIs occur frequently, which is the case on open fields (12.9%), and the proportion is even higher when the effects of optical radiation are shielded by mountainous areas (72.0%), fortifications (60.2%), and large arms (53.0%) (Fig. 27.1).

1.1.2 CRBI from Depleted Uranium Weapons

Depleted uranium (DU) is a byproduct of enriching uranium into nuclear fuel, specifically, the uranium with a ^{235}U abundance of less than 0.711%, the main component of which is ^{238}U. DU alloys, characterized by high density (19.3 g/cm^3), hardness, and toughness, are important materials for making depleted uranium bombs and depleted uranium armors. Missiles, bombs, shells, and bullets made of depleted uranium are collectively known as depleted uranium ammunition (DUA). DUA is highly penetrating and mainly used to pierce hard targets such as tanks and underground fortifications.

DUA has multiple killing factors that contribute to multiple types of injuries: shrapnel injuries, burns from spontaneous combustion, high temperature or ignition, α-radiation internal exposure due to radioactive aerosols entering human bodies, and injuries caused by poisonous heavy metals in

uranium. When hitting a tank armor, DUA can not only cause blast injuries, shrapnel injuries, and burns among other injuries to the crew but also makes them inhale a large amount of high-concentration depleted uranium aerosols, resulting in a certain percentage of combined radiation-blast injuries or blast combined radiation injuries.

1.2 Occurrence of Radiation Combined Blast Injuries in Peacetime

At ordinary times, CRBIs occur mainly during nuclear terrorist attacks and serious nuclear accidents.

1.2.1 Nuclear Terrorist Attack

Terrorism is a common problem for countries all over the world, and suicide terrorist attacks by means of suicide bombs, car bombs, etc. are its common forms. Radioactive drugs (radiological warfare agents during wartime) are colorless, odorless, non-stimulating to human senses, and not easily detectable; however, they are one of the preferred tools for terrorist attacks because of their short-term or long-term effects on people, the difficulty in clinical diagnosis and treatment, and particularly, induced mental stress and psychological disorders. A dirty bomb, or radiological dispersion device (RDD), is probably the main means of nuclear terrorist attacks. As a weapon for widespread dissemination of radioactive substances, a dirty bomb detonates conventional explosives such as "yellow dynamite" (picric acid and TNT), whereby a powerful explosive force is produced, and the contained radioactive substances, mainly radioactive particles, are ejected into the air, resulting in contamination from nuclear fallout. The explosion of a dirty bomb is usually considered the main cause of casualties, but the role of radioactive pollution cannot be ignored. Hence one can see that the explosion of dirty bombs is often accompanied by blast injuries, which can induce radiation combined blast injuries or blast combined radiation injuries.

1.2.2 Serious Nuclear Accidents

In the event of serious accidents regarding nuclear facilities, especially in nuclear power plants, nuclear leaks, fires, and explosions result in combined radiation injuries. Especially with the development and utilization of nuclear energy, the combined radiation-blast injuries from nuclear accidents need to be given sufficient attention. For instance, an operator in a nuclear power plant accidentally dropped a container of plutonium and cesium chips into a tetrachloromethane reservoir, and due to the reaction between plutonium and tetrachloromethane, the mixture exploded and generated a blast wave equivalent to 0.6 kg nitroglycerine, tearing and breaking the left hand in the glove box and contaminating it with a large number of radionuclides. During the Chernobyl nuclear disaster, severe radiation sicknesses or above are mostly associated with combined burns, becoming the main cause of aggravated injuries and increased deaths. Although there are no statistics on the occurrence of CRBI injuries in nuclear accidents, it can be assumed that there will be a certain percentage of CRBI injuries if a reactor explodes.

2 Classification and Condition of Radiation Combined Blast Injuries

2.1 Classification

Combined injuries are generally named by listing the primary injury first and the secondary injury second. Combined radiation-blast injuries are classified accordingly. Usually, radiation-dominant injury is termed radiation combined blast injury (or radiation-blast combined injury); if blast injury is the primary damage, it is known as blast combined radiation injury (or blast-radiation combined injury).

Since there are numerous types of blast injuries, and the situation is complicated, further classification of radiation combined blast injuries is generally required according to the sites of blast wave effects, including radiation combined blast chest injuries, radiation combined blast injuries to auditory organs, etc. It is important to note that in serious cases, the injury often involves several sites and organs and may even develop into multiple organ dysfunction syndromes or multiple system organ failures.

2.2 Condition

It is necessary to classify combined radiation-blast injuries to provide timely and effective first aid, diagnosis, medical evacuation, and treatment. According to the severity of the injury, it can be divided into four levels: mild (degree I), moderate (degree II), severe (degree III), and extremely severe (degree IV). Usually, the classification of CRBI is based on the radiation damage, and in combination with the aggravating effect of the injury. Generally, the radiation injury associated with moderate degree blast injuries or above can exacerbate the traumatic condition.

1. Mild RCBI: Mild radiation injury associated with mild blast injury is mild combined radiation-blast injury.
2. Moderate RCBI: Moderate radiation injury associated with moderate blast injury is moderate combined radiation-blast injury.
3. Severe RCBI: Severe radiation injury associated with moderate blast injury, or moderate radiation injury associated with moderate blast injury is generally a severe combined radiation-blast injury.

Table 1 Classification and degree of acute radiation sicknesses

Classification (degree)	Fundamental damage	Dose range (Gy)
Bone marrow	Bone marrow injury	1–10
Mild		1–2
Moderate		2–4
Severe		4–6
Extremely severe		>6
Intestine	Intestinal damage	10–50
Brain	Brain injury	>50

4. Extremely severe RCBI: Extremely severe radiation injury associated with each degree of blast injury, or severe radiation injury associated with moderate or severe impact injury is extremely severe combined radiation-blast injury.

Acute radiation injury often has a clear dose–effect relationship, features a gradual increase in exposure dose, a significantly grown mortality, correspondingly shortened survival time, and remarkable differences in clinical manifestations and prognosis. The classification and degree of acute radiation sicknesses are referred to in Table 27.1.

3 Clinical Features and Pathological Basis of Combined Radiation-Blast Injuries

The comprehensive reactions between different factors and between different causes and the bodies after the body is subjected to two or more different injury contributors are known as "combined effects" of a combined injury. Combined effects are the main feature of combined injuries different from single injury contributors other than single injury factors plus the resultant effects; different factors, injury contributors, and bodies can interact with each other. Usually, radiation injury often plays a dominant role in RCBI, and the basic pathological changes are mainly the lesions of the radiation injury. But in many cases it shows a "mutual aggravation" effect ($1 + 1 > 2$), so that the performance of the original single injury is not exactly the same as the effect of a single injury, and the overall injury becomes more complex and more difficult to treat. The effect can also not be aggravated, or even mitigated ($1 < 1 + 1 < 2$, $1 + 1 < 1$, etc.). The clinical features and pathological basis of RCBI injuries can be described in three aspects: overall effect, cell-tissue-organ effect, and combined effects of important pathophysiological processes.

3.1 Overall Effect

Overall effect mainly reflects the occurrence and development of the injury, course and stage of the disease, mortality, average survival time, dose effect, incidence and extent of main signs, and sequelae and outcome. The overall effect of RCBI is characterized by the following:

3.1.1 Severity of Injuries Depends Largely on Radiation Dose

The severity of lesion and disease course and the prognostic outcome are bound up with the radiation dose, that is, the injury severity and mortality increase while the survival time contracts with the increasing exposure dose. For example, the animal effect of a nuclear test showed that when comparing the animals with acute intestinal radiation sickness accompanied by severe blast injuries to those with mild blast injuries or without injuries, the difference in survival time and major clinical signs was insignificant, reflecting the fast progression of acute intestinal radiation sickness.

3.1.2 Course of Diseases has Radiation Sickness Characteristics

As a rule, there is a staged course of shock (initial phase), pseudo-recovery (latent phase), crisis (critical phase), and convalescence (recovery phase), but the critical phase of RCBI is earlier, longer, and has a shorter latent phase.

3.1.3 Mortality

Mortality is a visual concentration of the overall effect and outcome. Severe CRBI often shows a significant increase in mortality. The studies in mice have revealed that the skin wound making up 15% of the body surface area does not lead to death, and the total body irradiation of Co^{60} γ-ray alone kills 35% of the mice; the combination of the two results in mortality as high as 85%, indicating a noteworthy synergistic exacerbated damage effect. Different wounding sequences however will affect the overall mortality, with the aforementioned pre-injury irradiation model showing an obvious aggravation effect and a remarkable increase in mortality, whereas a study found that the mortality in mice was greatly reduced if skin wound occurred 24 h before irradiation.

But it should be noted that the above model of γ-ray combined skin wound is a strain of combined radiation-blast injuries that responds to the combined effects of the shock wave's secondary effect and the radiation injury. Some investigators have earlier addressed the direct shock wave effects on the lung associated with the injuries caused by different radiation doses are different and found that X-rays at 0.25 Gy, 0.5 Gy, 1.0 Gy, 2.0 Gy, 3.0 Gy, 4.0 Gy, and 5.0 Gy, when associated with the low, medium, and high shock waves, have no significant impact on the body weight and mortality of the animals within 30 days of injury, but the combined radiation-blast injuries of 6.0 Gy irradiation associated with high shock waves show higher animal mortality than that of one-cause injuries, indicating that the overall effect of CRBI

is influenced by the doses of both the radiation injury and the blast wave, i.e., a dose–effect relationship, but the combined effects of mutual aggravation are unapparent as far as mortality is concerned.

3.1.4 Survival Time

Survival time reflects a combination of changes and sequelae in the body's response to injury, including resistance and repair. A certain survival time is necessary to ensure the implementation of life-saving treatment and influenced by both life-saving treatment and the result of the body's response to life-saving treatment. Therefore, survival time can reflect the combined effects more objectively. The survival time of RCBI is generally shorter than that of the injury from a corresponding radiation dose.

3.1.5 Dose Effect

From the comparison of the radiation dose effects of the radiation injury and the combined radiation injury, it can be seen that the radiation dose required to meet the corresponding mortality after the combined radiation injury is lower than that of the radiation injury. The higher the drop, the greater the combined effects. The 30d $LD_{50/30}$ of the mice exposed to radiation is 9.63 Gy, but reduced to 8.20 Gy after combined burns and to 7.61 Gy after combined trauma, demonstrating a significant accentuation effect of both the combined radiation-burn injury and combined radiation-blast injury. The lowered radiation dose caused by the equivalent radiation injuries associated with other damages is the "equivalent dose" resulting from such injury, which is an important index for studying combined effects.

3.2 Cellular, Tissue, and Organ Effects

The combined effects of various tissues and organs after an RCBI vary in performance, most of them showing a mutual aggravation, and the degree of damage is greater than the total of single injuries, some greater than individual injuries, but lesser than the sum of individual injuries. Such differences are related to the response characteristics of specific injury-causing factors in different tissues.

3.2.1 Bone Marrow

Bone marrow is an organ sensitive to ionizing radiation, and the inhibitory effect of radiation injury on hematopoietic function is obvious. The hematopoietic disorder is one of the prominent changes of radiation injury and the first problem of the whole disease course, and the destruction and regeneration of hematopoietic tissues can better reflect the severity of radiation injury. When the radiation injury is associated with different injuries, the performance varies at different times according to different doses of exposure and the severity of the injury. In radiation-trauma combined injuries, the post-injury peripheral white blood cells and platelet levels are 57% and 33% lower than those in the radiation injury group; the DNA damage in the (lineage negative, lin^-) hematopoietic stem cells of the bone marrow is reduced instead (γ-H2AX formation rate is lowered). These studies demonstrate the complex effects on bone marrow hematopoiesis following radiation injuries associated with trauma injuries.

3.2.2 Small Intestine

Intestinal mucosa features quick renewal and high radiosensitivity. In radiation injury conditions, cryptic epithelium apoptosis is obvious, and proliferation inhibition is notable, usually resulting in intestinal epithelial necrosis and shedding, villus shortening and bareness, and seriously impaired gut barrier function. Through nuclear tests, the animal effect data show that the dose required for the occurrence of intestinal radiation sickness from a combined injury is smaller than that of the intestinal radiation sickness alone; for instance, the threshold dose for the occurrence of intestinal radiation injury in dogs with radiation injuries is 8.6 Gy and 6.8 Gy with combined injuries (down by 20% or so). Within the range of crossover doses for intestinal and myeloid types, the majority is myeloid in case of radiation diseases alone and intestinal in case of radiation combined injuries. The studies of radiation-trauma combined injuries suggest that the mice with radiation injuries (9.75 Gy γ-ray irradiation) associated with skin wounds (15% of the body surface area) sustained earlier and more severe pyohemia than those with radiation injuries alone at the same dose, indicating increased permeability and reduced barrier function of the small intestine when accompanied by skin wounds.

3.2.3 Lungs

Shock waves can cause serious damage to the body, especially to fluid- and gas-containing cavities such as the lungs, heart and blood vessels, digestive tract, and ears, due to their implosion and traction effects, making the lungs an important target organ of the shock wave, the injury-causing factor. Shock waves can lead to remarkable pathological changes such as pulmonary hemorrhage, edema, rupture, bulla, atelectasis, and emphysema. It can be inferred that a combined radiation-blast injury manifests as a more severe lung injury.

3.3 Combined Effects of Important Pathological and Physiological Processes

Combined radiation-blast injuries can reflect more intricate combined effects in terms of important pathological and physiological processes such as shock, infection, and tissue repair.

3.3.1 High Incidence of Shock

In the early phase, blast injuries, due to the positive and negative pressure of shock waves, result in pulmonary alveolar rupture and fusion, vascular endothelial damage, and secondary pulmonary edema that affect the gas exchange of the lungs and lead to systemic organ ischemia, hypoxia, and shock; radiation injuries only cause vascular endothelial permeability to increase, and early shock is rarely seen. However, if the radiation injury is associated with the trauma that does not cause shock, shock can occur. Therefore, radiation combined injuries are more likely to complicate shock. The higher the radiation dose, the more serious the injury, the higher the incidence of shock, and the more serious the course of the disease.

Most of the shock in radiation combined injuries occurs in the early post-injury period. This is a combination of severe impairments to the neurological, endocrine, circulatory, and metabolic functions of the body following the intense action of combined killing factors, wherein the reduction of effective circulating blood volume (ECBV) often becomes an important link in the development of shock and constitutes the main cause of death in the initial phase. Therefore, in severe cases, the initial phase of injury is actually the shock phase, clinically characterized by mental excitement, agitation, restlessness, soon shifting to inhibition, slow response, indifferent expression, or even blurred consciousness. At the same time, there are corresponding time-phase changes in respiration, pulse, and blood pressure, as well as fast blood concentration, blood volume reduction, and biochemical metabolism changes.

This type of shock is often characterized by a prolonged excitation period and a shortened inhibition period, which, if left unattended, may affect the timely diagnosis of shock and lead to delayed treatment. Once the inhibition period is entered, the effect of anti-shock measures is significantly reduced. Experimental studies have shown that there are two types of blood pressure changes when shock occurs due to severe combined injuries: one drops after the excitation period, but rises slowly, and can maintain for a period (which is called the compensation stage). At this point, if there is no timely and effective treatment, the blood pressure will fall again (which is known as the exhaustion stage); the other type has no obvious compensation stage after the excitation period, i.e., it enters the exhaustion stage directly after the excitation period, and the blood pressure keeps falling and never rises again. It is worth pointing out that in a few animals with radiation combined injuries, a steep drop in blood pressure can occur with only a very mild trauma after irradiation, i.e., a small amount of trauma can result in decompensated shock.

During wartime, the function of vital organs is generally normal at first in cases of shock from a single injury. However, the function of more than one vital organ may be impaired at the beginning of the radiation combined injury, and the course becomes more pronounced as the injury progresses. This not only allows for great variation in the clinical changes and types of shock, but also has a marked effect on prognosis. Some experimental studies have shown that the mortality of the animals with radiation combined injuries in the shock phase increases significantly.

The shock from combined injuries occurs partly in the critical phase, or after the initial shock phase enters the critical phase again through the latent phase, its process characteristics are different from the initial shock, but similar to the toxic shock. Clinically, it is common that the body temperature and blood pressure drop greatly at the same time; without timely and effective treatment, the consequences are serious, and the mortality is high.

The incidence of shock from RCBI injuries is high, and the cause of its exacerbation is unclear. Some researches suggest that it may be related to the following:

1. The important role of the nervous system in the occurrence and development of shock has long been highlighted. Various parts of the nervous system are damaged to varying degrees during the radiation injury, accompanied by the functional disorders of the cerebral cortex, subcortical layer, brainstem, and autonomic nervous system among all levels of tissue systems, which affect the function of the blood pressure regulation center (autonomic nervous system). As a result, the tolerance to strong stimuli such as trauma, injury, and bleeding is significantly reduced after the radiation injury. The sensitivity to blood loss is much higher than in non-irradiated animals, the changes in blood pressure are more pronounced, the incidence of shock increases, leading to more serious consequences. Thus, the dysfunction of the nervous system, resulting in increased sensitivity to trauma, may be one of the causes for the aggravation of shock in case of RCBI.
2. The reduction in effective blood volume is a major component in the development of shock. In the event of CRBI, the conditions leading to a reduction in circulating blood volume (CBV) increase, which may be an additional cause of shock exacerbation. Trauma, bleeding, and body fluid loss (of the wound) can cause hemoconcentration and hypovolemia. Both radiation injury-induced vomiting and diarrhea can exacerbate fluid loss, electrolyte disturbances, and hypovolemia, thereby promoting the development of shock. In contrast, radiation injury-caused functional and structural changes in the microcirculation, such as microvascular dilatation, extravasated blood, slow-flow, intracellular and intercellular edema, disruption of vascular endothelial barrier function, and increased vascular permeability, lead to increased vascular exudate and vascular bed volume, which in turn reduces the effec-

tive CBV and promotes the development and progression of shock.
3. A variety of toxic substances are produced after trauma due to tissue destruction and protein decomposition, and histamine-like toxic substances also appear in the blood immediately after radiation injuries. Some researches have confirmed that the intensity of gas metabolism is weakened greatly during shock from radiation combined injuries, the glycogen decomposition in the body accelerated, and the lactic acid and inorganic phosphate in the blood piled up, etc., all of which are more obvious than single injury-caused shock. Therefore, the increase in toxic substances may also be among the causes of shock aggravation due to RCBI.
4. The poisoning theory has long been argued in the pathogenesis of shock and radiation sicknesses. Experimental researches show that the susceptibility to bacterial toxins raises largely after radiation injury, and the cause of shock aggravation in the critical phase of RCBI may be related to the effects of bacterial infections and toxins, particularly the effects of Gram-negative bacilli, which cannot be ignored.

3.3.2 High Incidence, Early Onset, and Severity of Infection

Infections are prominent in both radiation sicknesses and blast injuries and occur earlier, more frequently, and more severely in combined radiation-blast injuries.

Why is infection more prominent in RCBI? The analysis can be made in the following ways:

1. Shock is more common and serious in case of CRBI, lowering the systemic resistance to infection. Shock reduces the ability to resist infection, which may be related to the following reasons: (1) hypoxia occurs during shock, and some important organ tissues become degenerative, or even necrotic, leading to functional disturbance, i.e., fewer globulins are synthesized in the liver. (2) Factors such as the damage to capillary endothelial cells due to the extravasated blood and oxygen deficit of the tissue during shock, the effect of decomposition products of damaged tissues such as histamine on the vessel wall, and disruption of mucopolysaccharides in the vascular wall due to radiation injuries increase the permeability of the blood vessel wall. As a result, the intravascular fluid components, even red blood cells, leak out of the blood vessels, and in the meantime, extravascular bacteria easily invade the blood vessels, causing bacteremia and sepsis.
2. The hematopoietic disorder is more serious in case of CRBI, with far fewer neutrophils and lymphocytes, phagocytosis inhibition of the reticuloendothelial system (RES), weakening of specific and non-specific immune functions, and a more significant decrease of serum bactericidal activity, whereby both cellular and humoral factors of the body against infection are greatly undermined.
3. In case of RCBI, there are not only endogenous infections from the gut, oral cavity, and urinary tract, but also exogenous infections from the wound surface and wound, greatly increasing the chances of infection. The bacteria on the wound surface grow more (millionfold to ten million times) in combined injuries than trauma injuries. This is because the number and function of white blood cells drop greatly, resulting in significantly less phagocytized and killed bacteria; the bacterial endotoxin itself inhibits cellular defense; bacteria can easily grow and multiply in the necrotic tissue. Copious toxic substances (enzymes) produced by bacteria facilitate the spread of bacteria on the wound surface, such as lecithinase, which destroys the cell membranes of red blood cells and other cells, causing hemolysis and necrocytosis; hyaluronidase dissolves the dermal and subcutaneous connective tissue matrix; collagenase dissolves collagen fibers. The necrotic tissue of the wound surface is mostly filled with a large number of cenobium, sometimes complicated by fungal infections. The wound surface bacteria can further spread along the lymphatic vessels and blood vessels, sometimes multiplying in quantity in the lumen, and filling the lumen like a tube, which not only blocks the blood vessels (in this case, it also promotes the diffusion of thrombus) and causes further tissue necrosis, but also leads to local lymph gland infections and systemic bloodborne infections.

The common clinical manifestations of RCBI-induced infection are wound infections and focal infections.

The incidence of infections (wound surfaces and wounds) increases, and the course is severe in case of CRBI. Despite that the bacterial species causing such infections are similar to those in general war wound infections, it often tends to be reduced in Gram-positive cocci (especially staphylococcus) and increased in Gram-negative bacilli. Quantitative bacterial examination reveals that although the number of bacteria in individual and combined wound surfaces is similar in the early post-injury period, as the disease progresses, the number of bacteria in combined wound surfaces increases significantly compared to single injuries, enhancing the chance for the spread of wound infections and invasion of the bloodstream. Special infections common in war wounds, such as gas gangrene, tetanus, and fungal infections that occur after extensive use of antibiotics and hormonal agents, have not received sufficient attention in the studies of radiation combined injuries. Some preliminary reports indicate a high bacterial multiplication rate and morbidity among the animals infected with Bacillus tetani after radiation combined inju-

ries, as well as accelerated and more severe clinical signs and disease progression than the animals with single injuries.

Clinically, focal infections from CRBI are most often seen on the body surface, and in the mouth, and throat, including skin and mucous membrane erosions and ulcers, bedsore infections, gingivitis, tonsillitis, and Ludwig's angina. The incidence of lesions in the oral-pharyngeal region grew among the casualties of the nuclear attack in Japan. Most of these focal infections appear before the critical phase and can last for several weeks. Clinically, local pain is perceived, affecting speech and swallowing, and there is foreign body sensation, salivation, and local swelling. Such lesions tend to occur in more concentrated areas of lymphoid tissue, and often at the same site as bleeding. In addition, focal infections can occur in internal organs such as the lungs and intestines.

Wound infections and focal infections occur at a time when the cellular and humoral factors of the body that have anti-infective functions are significantly reduced. Bacteria can readily take advantage of these sites to invade the blood circulation and cause bacteremia, sepsis, or sapremia. Post-injury blood cultures show that the positive rate of blood cultures in animals with radiation combined injuries improves compared to either single traumas or radiation sicknesses. Also, the time of occurrence is earlier. It usually forms right after the wound infection and increases gradually as the disease progresses. In severe cases, there are two peaks in terms of the positive blood culture rate, the first appearing when the wound infection is grave and the necrotic tissue is separated and shed in large pieces, and the second in the critical phase. The first peak is lower and lasts for a shorter period, whereas the second peak is higher and can last longer. The bacteria commonly found in blood cultures are Staphylococcus albus and S. aureus, streptococci, Escherichia coli, and Gram-negative bacilli grow in quantity. There is a trend towards a relatively higher detection rate of streptococci and Gram-negative bacilli, as well as a higher incidence of mixed infections compared to single injuries. It can be seen that the bacterial spectrum of blood cultures and its variations have similarities with these of wound infections. Through detailed analysis, it can also be found that the bacteria detected in blood cultures are often the predominant bacteria present at the site of injury, with only a few being identical to those from other foci of infection. Therefore, it is generally accepted that most of the bacteria present in the blood in case of radiation combined injuries invade the blood circulation via the site of injury.

Clinically, septic symptoms often mark the beginning of the critical phase when the casualty feels depressed, sustain generalized weakness, elevated body temperature, sometimes chills (aversion to cold), along with quickened pulse and accelerated breathing, and exacerbation of the wound infection, followed by metabolic disturbances and other toxic symptoms. Experimental researches show that fever starts earlier, fluctuates widely, lasts longer, and presents more severe toxic and other symptoms from radiation combined injuries than from individual injuries. The trend and degree of fever are closely related to the decrease in white blood cell counts, wound surface ulceration and infection, and blood cultures. Another feature of infection induced by radiation combined injuries is the high incidence of hypothermia. In severe cases, a low fever may occur at the outset, or suddenly after a high fever. The presence of hypothermia is indicative of critical illness and is often the result of a severe infection with Gram-negative bacilli.

3.3.3 Delayed Wound Healing

In case of CRBI, the tissue repair of post-traumatic wounds, wounds, and fractures is difficult, and healing is delayed due to the effects of radiation damage. The degree of this effect depends mainly on the magnitude of the nuclear radiation dose received. If the radiation injury is mild, the local changes of the combined wound are almost the same as those of a single injury; if the radiation injury reaches a moderate degree or above, the effects are obvious, manifesting as weakened inflammatory response, easy complication of infections, increased bleeding tendency, delayed wound healing, and poorer functional recovery. Clinically, the characteristics of delayed wound healing in case of CRBI can be seen from the changes in wound size, the growth of granulation tissues, and the solidity of porosis speed at the fracture site. In the early post-injury period, the early contraction of the wound not only progresses slowly, but also looks paused, and sometimes the wound is even larger than onset. Later on, granulation growth and contraction, epithelial proliferation, and coverage are all delayed than in case of simple trauma. The porosis at the broken end of the fracture is few and slow, and the formation of bone canals is delayed. In general, the more severe the CRBI, the more prominent these characteristics, especially in the critical phase.

The reasons for delayed healing of radiation injuries can be explained in the following aspects:

Weakened Inflammatory Response

In terms of CRBI, it is common to have a small area of the inflammatory response to the wound or injury and a narrow band of inflammatory response around the wound. If a foreign body is present, the response is slight, the formation of an enclosure around the foreign body is delayed, and local bleeding is common at the site of the foreign body in the critical phase. Even when the shallow foreign body is discharged, a fistula that does not heal easily is often left behind.

The body prepares the conditions for tissue regeneration and repair by killing and eliminating bacteria and absorbing and removing necrotic tissues through an inflammatory response. Therefore, inflammation is a prerequisite for repair

and healing after tissue injury: during radiation combined injuries, as the hematopoietic tissue is damaged, the white blood cell count declines, the infiltration of inflammatory cells in the wound surface or wound lessens, and the phagocytic function reduces, making bacteria and necrotic tissues not easily phagocytized and removed. At this point, due to increased vascular permeability, there are often excess local fibrin exudates, but because of neutrophils reduction, the proteolytic enzymes released by them also dwindle in numbers. So, the local fibrin is not easily dissolved, but coagulated with the necrotic tissue, making it difficult to fall off and clear away, which not only affects tissue repair, but also easily leads to bacterial growth, and aggravates the infection. This further hinders tissue repair.

Local Infection and Hemorrhage

Wartime trauma is mostly contaminated by bacteria, but whether it can develop into infection is related to the characteristics of the trauma, the immunity of human bodies, and the situation of bacterial contamination. The systemic and local changes of CRBI are conducive to the occurrence and development of infections. So, the infection is more prominent in case of CRBI, and especially in the critical phase of radiation sickness. Some researches show that during CRBI, the pyogenic infection process of the wound is aggravated, and it is easy to spread and develop cellulitis, osteomyelitis, and septic fistula. The incidence of surgical wound infections is also higher among CRBI patients, which can not only lead to operation failure, but also induce other complications if not treated effectively and promptly. After skin grafting, the already grown and healed skin grafts can become infected. Partially healed wounds can also reopen due to infection. In the event of radiation combined fractures, the overall condition is worsened, in addition to an increased wound infection rate that affects fracture healing, and poor functional recovery of the injured limb.

With regard to bleeding, the injured area bleeds mostly in two periods, namely the early post-injury phase, and around the critical phase. The former occurs as a result of trauma-induced tissue damage and vascular rupture. The bleeding often has some of the following CRBI characteristics: first, reduced tolerance to blood loss and therefore a severe impact of bleeding; second, slower absorption of blood seeping from the injured area, which affects wound healing and creates conditions for local infection; third, after initial irrigation and debridement, the incidence of hematoma within the wound is significantly higher than in a single injury due to an increased propensity for wound hemorrhage.

When systemic hemorrhage occurs due to hematopoietic failure before the critical phase of the CRBI, the bleeding from the wound and wound surface often happens earlier and is more extensive and severe than the bleeding from the body surface skin and mucous membranes. At first, the tendency to bleed from the granulation wound increases, followed by small bleeding spots that soon fuse and expand until the granulation surface is extensively overflowing. At this point, the wound surface infection is also particularly severe. The hemorrhage and infection interact with each other, mutually aggravate, and intertwine with the bleeding spots of the skin around the wound. Then, the wound presents a picture of hemorrhagic necrotic infection, characteristic of a radiation combined injury. If the wound is accompanied by vascular damage and not completely healed, it may then split open and bleed again. The consequences are also more serious because the body's tolerance to blood loss is worse in the critical phase of a radiation combined injury. Bleeding not only causes the loss of blood throughout the body, but also reduces or even cuts off the local blood supply to the trauma and burn and discretizes the tissue. The blood coagulation in the tissue provides conditions for bacterial growth.

During the observation of the CRBI wound surface, such a development process is frequently seen: in the pseudo-healing (latent) phase, the granulation grows well, and looks red, tender, and healthy; after entering the critical phase, the granulation tissue stops growing and becomes pale and stained, prone to infection and bleeding; when entering the recovery phase, the infection and bleeding are controlled and subside, and the granulation tissue and epithelium grow and close up along with the wound surface.

This shows that close prevention and control of infection and hemorrhage are extremely important for the acceleration of wound healing.

Inhibition of Histiocyte Regeneration

Under the effects of nuclear radiation, the regenerative capacity of local tissue cells in the trauma can be directly inhibited, mainly including the regeneration of epithelial cells, fibroblasts, and vascular endothelial cells after skin and soft tissue injuries, division of these cells, collagen synthesis; osteoblast regeneration, and callus formation in case of fractures.

In terms of cutaneous and mucosal wounds, only epidermal cells regenerate and cover the wounds in order to destroy them, stop the growth of granulation tissues and allow them to heal. Therefore, the regeneration of epidermal cells and their functions are extremely important for wound healing. In the process of wound healing, the epidermal cells have functions of division, and proliferation, and migration, and cell division is ongoing throughout the repair process. A basilar membrane is formed when the epidermis and connective tissue come into contact. In case of trauma combined radiation injuries, epidermal cell division and migration can be inhibited or even stopped, thus affecting wound healing.

During wound healing, the capillary endothelial cells around and at the base of the wound swell, divide, form bud breaks, migrate, and connect to each other. These connected

protrusions are initially solid and later gradually hollow out, forming lacunas that allow for blood flow. In case of radiation combined injuries, vascular endothelial cells divide and grow slowly, or stop growing, affecting the formation of granulation tissues. The regeneration of lymphatic vessels is slower than that of blood vessels.

Fibroblasts play an important role in wound healing. They can swell, elongate, protrude, move in a linear direction, and divide and proliferate, release mucopolysaccharides when mature, and form collagen fibers. Only with the formation of intact collagen fibers can the tensile strength increase after wound healing. In case of radiation combined injuries, both fibroblast division and collagen synthesis can be inhibited, whereby the growth and maturation of granulation tissues are affected. Sometimes the tensile strength is quite low after wound healing.

For example, after researchers exposed guinea pigs to 200 C/kg irradiation and rats to 300 C/kg irradiation, suturing was performed immediately after wounds formed on the back, and general histological observations were made on the 7th and 14th days of injury. It was found that there was no significant difference between the animals with combined injuries and single injuries, but the tensile strength of the wound skin was much lower in the animals with combined injuries than with single injuries. The higher the irradiation dose, the more obvious the reduction in skin tightness, and the longer the duration. In rats irradiated with 700 C/kg γ-rays, the collagenation in the wound skin was seen to be 2–3 days later than the control group. if irradiated at a very high dose, the division of fibroblasts, vascular endothelial cells, and epithelial cells stopped, leading to the cease of the healing process.

Medium doses of irradiation can slow down the regeneration of broken tissues, set back porosis, and hold up callus modeling. At higher doses, the callus does not grow, resulting in long-lasting fractures or pseudarthrosis. Nuclear radiation can directly inhibit the activity of osteoblasts, slacken the differentiation of osteoblasts into osteocytes, restrain the activity of alkaline phosphatase, and block the calcification process (calcification requires the participation of alkaline phosphatase). In addition, some researchers irradiated rats with X-rays at 400 C/kg and 800 C/kg and found that porosis after fracture was later than in the control group, and the basophilic mast cells began to increase and were later lower than in the control group until the 3rd week of callus formation. These mast cells are associated with mucopolysaccharide synthesis, which is disturbed after irradiation. This may be related to slowed callus formation. In addition, the disruption of the bone marrow blood circulation will affect the vascularization (organization) of fracture end hematoma and the blood supply in the healing process (the vascular system of bone marrow plays an important role in the nutrition of bone tissue).

These changes affecting fracture union occur mainly during the critical phase of radiation sickness, and fracture union generally proceeds smoothly after entering the recovery phase.

Effects of Radioactive Substances on Wound Surfaces and Wounds

During surface bursts and low airbursts, the casualties in the explosion zone, especially the personnel exposed in the downwind near zone (similarly, the depleted uranium aerosols formed by depleted uranium munitions, and radioactive substances in dirty bombs), open traumatic wounds may be contaminated by radioactive substances, resulting in two adverse effects, namely delayed local healing and absorption into human bodies.

Radioactive substances are adsorbed by local tissues when falling on the wound surface or wound, more so if there is an exudate. The local effects depend mainly on factors such as the intensity of contamination, the time the contaminant remains, and the type of wound. Radioactive contamination locally causes degeneration and progressive necrosis of epithelial cells, connective tissue cells, and muscle cells, and inhibits cell regeneration, especially in the epidermis where the germinal layer cells and fibroblasts are more susceptible to damage. This causes local accumulation of necrotic tissues, delays the process of tissue regeneration, and predisposes to hemorrhage and infection (septic and anaerobic infections). In experiments with domestic rabbits, the incidence of skin-muscle wound suppuration was 11.5%, 22.2% after contamination with radioactive iodine and calcium, and up to 53.8% if the contamination was followed by X-ray exposure.

The absorption of radioactive substances through wound surfaces and wounds depends primarily on the physicochemical properties of these substances (e.g., solubility) and the nature of local injuries. Many radioactive substances are almost completely insoluble in the exudate of wounds, such as oxides of heavy metals, peroxides and carbonates, sulfates, and phosphates, whereas hydrochloric acids and nitrates are readily soluble and therefore highly absorbed. Nuclear explosion fallout has a solubility of 10–20, so there will likely be some absorption into the body through blood vessels and lymphatic vessels, and more than a certain amount will be harmful to the organism. It is generally believed that intact skin does not absorb radioactive substances or absorbs less; burn wound surfaces with unbroken eschars absorb less, while the wounds with abundant blood circulation and those with broken muscles absorb more. As regards exact data, the laboratory findings vary from a few percent to several tens of percent. From a therapeutic point of view, it is important to treat contaminated wound surfaces and wounds as early and quickly as possible. It has been reported that 20% of the radioactive materials can

be removed from the surface of the wound by cleansing within 1 h contamination, 10% within 2 h, and only 3–5% later; 30–70% by surgical methods like debridement within 2 h contamination, but surgical and other decontamination approaches are generally not effective after 6 h contamination.

Other Factors

Neuroendocrine regulation disorders, metabolic disorders, especially systemic changes like protein metabolism disorders, and anemia that appears early in the course of radiation combined injuries are also bound to have adverse effects on local wound healing.

The influence of radiation injuries on wound healing mainly occurs in the critical phase. Therefore, efforts should be made to heal or minimize the wound surface or wound as much as possible before the arrival of the critical phase based on systemic treatment, and firm measures should be taken to prevent and control local infections and hemorrhage in the critical phase, not only making for local wound healing, but also creating good conditions for the overall treatment of combined injuries

3.3.4 Aggravation of Hematopoietic Disorder

The hematopoietic disorder is a prominent change of radiation injury. The severity of radiation injury is well reflected by the destruction and regeneration of hematopoietic tissues. In acute blast injuries, there is always a decrease in peripheral white blood cells (PWBC), but less severe injuries usually show an elevated leukocyte count response. When the radiation injury is complicated by blast injury, it can accelerate and exacerbate the destruction of hematopoietic tissues and lower and set back their regeneration. The aggravation effect of hematopoietic dysfunction from combined radiation-blast injuries is related to the following aspects:

1. Infections become increasingly serious due to combined injuries, and heavy infections can exacerbate the damage to pre-existing hematopoietic tissues. It is often seen that the white blood cell count (WBC) is falling, but after infection with fever, it drops sharply as the body temperature rises. Sometimes, the hematopoietic function has begun to recover, and the PWBC gradually rises, even by several thousand, indicating that the bone marrow has regenerated. But after the complication of sepsis, the WBC drops sharply; the autopsy reveals that the bone marrow is empty again, that is, the original regenerated bone marrow has been subject to another severe damage.
2. The decline in WBC will also be accelerated by the massive depletion of PWBC from traumatic wounds when the hematopoietic tissue is no longer able to produce and release leukocytes as usual
3. Toxic substances produced as a result of tissue necrosis due to the combined severe blast injuries may directly inhibit and damage hematopoietic tissues.
4. On the basis of suppressing hematopoiesis, the hemorrhage from the wound, grown blood exudation and hemolysis due to increased vascular permeability, lysis of red blood cells by lecithase and hemolysin infected with bacteria can accelerate the onset of anemia, but in the early stages, the hemolysis, together with congestion and hemorrhage in the GI tract, can result in further destruction and loss of red blood cells. The lesions of the hematopoietic tissue change accordingly in terms of peripheral hematology.

The lesions of the hematopoietic tissue change accordingly in terms of peripheral hematology:

Changes in Leukocytes

Regarding severe radiation combined injuries, the changes in leukocytes are typical and show obvious time-phase alteration. There is mostly a clear rising peak after injury, which can also be seen in a corresponding single injury. Then, it is followed by a rapid drop in the leukocyte count, which reaches the minimum value earlier than in individual radiation sickness, with a low and long-lasting minimum level. In terms of leukocyte classification, the most drastic change happens in neutrophils, followed by lymphocytes. The trends and characteristics of the leukocyte changes in the total count and classification vary according to different types of combined injuries, which can be used as a reference for differential diagnosis. The response of the leukocyte count in the presence of radiation combined injuries is subject to multiple factors. It is generally accepted that, when complicated by radiation injuries, the higher the radiation dose, the more rapid the leukocyte count decrease; the lower the level, the slower the rebound. Some experiments have also demonstrated that the magnitude of the blast wave overpressure also has an appreciable impact on the change of leukocytes when it is complicated by blast injuries. The studies on the radiation sickness and combined radiation-blast injury to dogs have found that the decrease in leukocytes is more pronounced and the animals died earlier after total body irradiation of 3.4 Gy with blast injuries; the leukocyte counts fell quickly and remained at a lower level (less than 40% of normal leukocyte level) for a longer period in animals subject to 1.5 Gy whole-body irradiation and high overpressure (>0.5 kg/cm^2), whereas the leukocytes of the animals subject to low overpressure (<0.2 kg/cm^2) maintained a relatively higher level.

Changes in Erythrocytes

The changes in erythrocyte-related indicators (erythrocytes, hemoglobin, hematocrit, etc.) during radiation combined

injuries are also significant and have a clear temporal phase. Three phases of initial rise, progressive decrease, and gradual recovery are generally seen. Anemia can occur early after the injury, but is masked by hemoconcentration.

Changes in Platelets

The variation trend in platelet count due to radiation combined injuries is roughly identical to that of the total leukocyte count. The initial rise after injury resembles that of burns alone, while the subsequent decline is similar to that of radiation sickness. However, it sometimes shows a faster and lower decline depending on the type and condition of the combined injury. In the recovery phase, the rise is sometimes greater compared to radiation sickness, often fluctuating up at high levels.

Along with the decrease in platelet count, there is a progressive increase in capillary fragility and coagulation disturbance, as well as clinical bleeding syndromes. These indicators generally return to normal when clinical bleeding stops. Compared to radiation sickness, the bleeding syndromes of combined injuries appear and worsen earlier.

4 Diagnosis of Radiation Combined Blast Injuries

The diagnosis of combined radiaiton-blast injury (CRBI) should take single injuries as the basis, fully consider the characteristics of combined injuries, rely on the type and condition of the injury from explosion to make a group and individual diagnosis. That is, full using the indirect basis of all sources while relying on the direct observation of each casualty. Early classification and clinical diagnosis are made on spot, at early treatment facilities, and in specialized hospitals under different conditions and according to different requirements.

4.1 Early Classification Diagnosis

4.1.1 History of Injuries

Based on the scene and information of nuclear explosion, it is suggested to determine the nuclear weapon yield, the mode of detonation, and the location of the explosion center and project the location and extent of the damage zone, and the type of combined injury that may have occurred. Fully understand the location of the casualty at the time of nuclear burst, the presence or absence of shielding and protection; whether the explosion is seen or heard; whether people are being thrown up, struck, crushed, and buried; the duration and activity in the heavily contaminated area, and the manner of evacuation from the contaminated area. These circumstances will help to indirectly speculate on possible injuries.

Table 2 Comparison of object damage extents, blast wave pressure values, and blast injuries

Extent of object damage	Blast wave pressure value (kPa)	Blast injury
Severe damage to brick and wood homes	15	Mild
Moderate or severe damage to industrial plants	15–25	Mild or moderate
Moderate damage to solid buildings	18	Mild
Severe damage to solid buildings	40	Moderate
Slight or moderate damage to trenches and machine gun emplacements	60–90	Severe or extremely severe
Mild damage to cat ear hole bomb shelters, machine gun emplacements, and light-weight shelters	60–85	Severe or extremely severe
Slight or moderate damage to trucks, infantry vehicles, gun-howitzers	70–120	Moderate, severe, or extremely severe
Slight damage to light-medium tanks, armored personnel carriers	40–50	Moderate, severe, or extremely severe

4.1.2 Surroundings

The overpressure and dynamic pressure of different blast waves can cause different degrees of injury to both people and the surrounding environment. The degree of blast injury to people at the same location and in open areas can be indirectly inferred from the weapon, technical equipment, fortification, and the extent of damage to buildings and structures. The relationship among the extent of damage caused by blast waves, the pressure value of blast waves, and the state of personnel injuries through blast is given in Table 27.2.

4.1.3 Early Signs and Symptoms

Body surface burns and injuries are easy to detect; the difficulty and focus of diagnosis lie in the presence of associated radiation injuries and visceral blast injuries. The following signs and symptoms are conducive to the early diagnosis of combined radiation-blast injuries and the differentiation from other types of combined injuries.

1. Extensive burns without obvious early signs of radiation sickness may be a burn-dominant combined injury.
2. Burns with tinnitus, ear pain, deafness, coughing, or froth-like bloody sputum may be a burn-blast combined injury.
3. Post-injury nausea, vomiting, and diarrhea accompanied by symptoms of both burn and blast injuries may be a radiation-burn-blast combined injury. If there are also central nervous symptoms like ataxia, head shaking, and

convulsion, it may be considered a cerebral form of the radiation combined injury.
4. If the overall injury presentation is more severe than body surface burns or injuries, the possibility of associating with a radiation injury or visceral blast injury should be considered.

4.1.4 Preliminary Estimation of Exposure Doses

If the casualty wears a dosimeter, the exposure dose can be read or monitored. Peripheral blood and classification and absolute reticulocyte and lymphocyte values are recommended to be checked every 12–24 h. If possible, venous blood can be drawn for chromosomal aberration analysis of lymphocytes; urine samples, nasal swabs, and blood preparations are retained for radioactive measurement; items such as watches and pills are collected to estimate the exposure dose.

4.1.5 Peripheral Hemogram Change

In combined injuries with predominantly radiation damage, the WBC falls to varying degrees: the higher the exposure dose, the more rapid and lower the decrease.

4.2 Clinical Diagnosis

4.2.1 Signs and Symptoms

Ask and observe for loss of consciousness, headache, dizziness, tinnitus and dyspnea, chest and abdominal pain, etc. Further determine the condition of the injury based on the clinical presentation of the casualty.

1. Associated auditory organ injuries: There will be tinnitus, ear pain, hearing impairment, or serous or bloodstained fluid coming from the external auditory canal. Tympanic membrane perforation and bleeding are visible on otoscopy.
2. Associated chest injuries: In case of a lung injury, there will be chest pain, cough, hemoptic frothy sputum, and dyspnea. X-ray examination: In case of a lung hemorrhage, there will be a lamellar shadow in the lung field; in case of blood in the chest, there will be a shadow with a curved upper edge in the lower part of the lung field; in case of pneumothorax, there will be air in the chest on the injured side, with the lung being compressed, and the mediastinum biased towards the healthy side. In the presence of cardiac trauma, there is anterior cardiac pain, chest distress, breath-holding sensation, and cold sweats; electrocardiography shows myocardial damage.
3. Associated abdominal injuries: There will be abdominal pain, pressing pain, abdominal muscle tension, diminished or absent borborygmus, and aeroperitoneum. In severe cases, there may be signs of hemorrhagic shock, i.e., irritability, thirst, dry tongue, pallor, tachycardia, and blood pressure drop. Abdominocentesis, lavage of the abdominal cavity, X-ray examination, and B-type ultrasonography are useful in determining the diagnosis.
4. When associated with fractures: There is pain, hemorrhage, swelling, and impaired mobility at the injury site, and an X-ray examination can be performed to clarify the diagnosis.
5. Clinical manifestations of concussion, cerebral contusion, cerebral compression (intracranial hematoma, etc.) when associated with closed craniocerebral injuries.
6. In case of associated limb crush injuries, the injured limb is heavily swollen, consolidated, less elastic, numb or paralyzed, the distal arterial pulses are weakened or absent, and hypovolemic shock and myoglobinuria may occur.
7. In case of associated soft tissue injuries, there may be clinical manifestations of bruises, lacerations, and injuries caused by projectiles and broken slides.
8. In case of associated eye injuries, there may be ocular clinical manifestations.

4.2.2 Laboratory and Function Tests

1. Changes in hemogram: The total number of leukocytes falls dramatically after severe combined radiation-blast injuries, and the lymphocytes can disappear from the peripheral blood, and the chromosomal aberration rate and micronuclei frequency in peripheral blood lymphocytes increase. The myelogram has the characteristics of acute radiation sicknesses.
2. Biochemical criterion: The elevated activities of alanine aminotransferase (ALT) and aspartate transaminase (AST) can be used to diagnose hepatic ruptures; the changes in cardiac enzyme can be used to diagnose myocardial damage.
3. X-ray is aimed at diagnosing blast lung injuries, brain injuries, gastrointestinal ruptures or perforations, and glass fragment injuries.
4. CT and MRI are performed to diagnose thoracic, abdominal, spinal, and cranial blast injuries.
5. Electrocardiogram is used to determine heart and lung injuries and to observe the progression of the disease.
6. EEG and rheoencephalogram are used for diagnosis of brain injuries; if necessary, lumbar puncture is done to determine brain pressure and examine cerebrospinal fluid.
7. Ultrasonography, arterial partial pressure of oxygen, and pulmonary shunt volume are applied to diagnose blast lung injuries.

5 Clinical Classification of Radiation Combined Blast Injuries

The clinical classification of combined radiaiton-blast injury (CRBI) is similar to the course of acute radiation sicknesses because of the radiation injury predominance in RCBI, but the extent of damage is more severe. There is a typical clinical classification according to the range of exposure doses.

1. Bone marrow type acute radiation sickness: After moderate (2–10 Gy) or higher doses of irradiation, the course of disease development is divided into the initial phase, pseudo-healing (latent) phase, critical phase, and recovery phase.

a. Initial phase: The main manifestations are the stress reaction after exposure, and dysfunction of the neuroendocrine system, the symptoms including dizziness, weakness, loss of appetite, nausea and vomiting, conjunctival congestion, etc. PWBC may be transiently increased or mildly reduced.

b. Pseudo-healing phase: Most of the initial symptoms are significantly alleviated or largely disappeared, but the hematopoietic damage is still developing, and the pathological changes are still ongoing.

c. Critical phase: It is the stage when various clinical manifestations of the radiation injury appear clearly, the patient's general condition becomes worse, i.e., obvious fatigue, loss of appetite, nausea, vomiting, and in severe cases, diarrhea, continual hematopoietic disorder, pancytopenia, immune deficiency, frequent local or systemic infections, hemorrhage in different parts of the body (urine, feces, skin mucosa, etc.), water-electrolyte disturbance, acid–base imbalance, etc.

d. Recovery phase: After active treatment, the bone marrow hematopoietic function begins to recover, PWBC and platelet counts gradually increase, and symptoms decrease and disappear.

2. Intestinal form of acute radiation sickness: It is an extremely serious acute radiation sickness with frequent vomiting, severe diarrhea, and watery stool with bleeding among other gastrointestinal tract injuries as the basic damage. The intestinal mucosa is a highly sensitive tissue to irradiation, and serious damage will occur in about one week after 10 Gy irradiation or above, with the pathogenic manifestations that most of the epithelial cells of intestinal mucosa fall off due to necrosis. The intestinal crypt cells are more sensitive to radiation, and after irradiation, quickly become necrotic, decrease in quantity or disappear; the villi epithelium is not repaired after shedding, causing the villi to expose and form a massive wound surface. The damage to the integrity of the intestinal mucosa results in the weakening of the intestinal barrier function, the leakage of body fluids into the intestinal lumen, and the loss of nutrients such as blood cells, plasma (proteins), and water-electrolytes. The bacteria and toxic metabolites spread from the intestinal lumen into the body, leading to infection and toxicity. The disturbance of intestinal motility after high-dose irradiation is also a major cause of vomiting, diarrhea, and intussusception.
3. Cerebral form of acute radiation sickness: It is a critical acute radiation sickness with brain and central nervous system injuries as basic damage, which is more serious than intestinal form; the onset is more rapid, the course of the disease progresses quickly, the clinical stages are not obvious, and most of the patients die within 2–3 days of irradiation. According to the results of clinical observations and animal experiments, the symptoms of cerebral-form acute radiation sickness include ataxia, nystagmus, increased muscle tone, limb tremor, and convulsion. In addition, the onset is rapid, mostly accompanied by vomiting immediately or within a few minutes after irradiation, and diarrhea within 1h, which soon turns into loose stools or fecal incontinence. Flush, large areas of skin erythema, food refusal, and disorientation occur soon after irradiation. With frequent convulsions, the patient's general condition deteriorates fast, and dehydration, shock, coma, and systemic failure may occur. Hematopoietic disorders and intestinal damages are more severe compared to the intestinal form.

On top of radiation injuries, the clinical manifestations of blast injuries in RCBI are the same as previously described, including auditory, thoracic, abdominal, and closed brain damages, fractures, limb crush injuries, soft tissue injuries, etc.

6 First Aid and Treatment of Radiation Combined Blast Injuries

6.1 Treatment Principles

Most RCBI patients require grading treatment, which is generally categorized into three levels: on scene care, early treatment, and follow-up care (subsequent treatment). The principles and experience in the treatment of blast and combat injuries during peacetime and conventional warfare should be fully drawn upon; the treatment principles and experience of a single injury should be fully utilized, and the treatment should be based on the features of the combined injury. The different extents and types of combined injuries should be considered for treatment, the focus of which lies on the major injury other than a mild one. It is important to

emphasize both systemic treatment and proper local treatment, making the two aspects complement each other. It is required to appropriately address the contradictions that arise in treating several injuries that are present at the same time. Attention should be paid to the stages of the disease course, and particular emphasis laid on the treatment at each stage.

Principles of treatment: Serious ones before mild ones, quick rescue and treatment, and close observation. The focus of treatment is on the damage to the heart, lungs, abdomen, and acoustic organ, crush injuries, and glass fragment injuries.

6.2 First Aid on Site and Emergency Treatment

Comprehensive treatments depend on the overall traumatic condition and injured parts. Those with severe visceral injuries should stay in bed for fear of aggravating pulmonary hemorrhage, pulmonary edema, visceral hematoma and ruptures, and heart failure. Due to the high incidence of shock and the prominent infection from RCBI, early anti-shock and anti-infection measures should be emphasized. The main points of rescue and treatment are as follows:

1. First aids are essentially the same as for general combat injuries, including ventilation, hemostasis, dressing, immobilization, transfer, and basic life support.
2. Systemic therapy
   a. Prophylactic injection of tetanus antitoxin (TAT).
   b. Blood and fluid transfusion to prevent and control shock. If there is a lung injury, the amount and rate of transfusion should be controlled to prevent the occurrence or aggravation of pulmonary edema.
   c. Anti-radiation drugs that are effective in acute radiation sicknesses are also largely effective in CRBI injuries and thus should be given as early as possible.
   d. Active control of infections. Oral anti-infective drugs should be administered as early as possible.
   e. One-time high-dose corticosteroids can be applied in the early stages for critically injured patients.
3. In case of suspected serious radionuclide contamination, potassium iodide should be administered as soon as possible, clothing and exposed parts of the body decontaminated locally, and measures to accelerate the discharge of radionuclides taken if necessary; rescuers and casualties should take personal protective measures such as masks and towels.
4. Local treatment
   a. In case of associated auditory organ injuries, plug the outer ear with sterile cotton balls to prevent infection. If an infection has already occurred, wash with 4% warm boracic solution, and place ribbon gauze for drainage. Carry out tympanoplasty if the tympanic membrane is perforated.
   b. In case of associated chest injuries, keep the respiratory tracts of the patients with lung injury open, let them inhale 95% medicinal alcohol-containing drugs via a nebulizer, and give drugs to promote the absorption of edema; for hemothorax, empty the accumulated blood via thoracentesis, and perform closed drainage for hemothorax where the blood cannot be easily pumped; for open pneumothorax, seal and dress the wound firmly and immediately; for tension pneumothorax, perform closed drainage for exhaustion. In case of a cardiac injury, ensure adequate rest, avoid activities, and give symptomatic treatment; in case of heart failure, use Lanatoside C or Strophanthin K
   c. In case of associated abdominal injuries, such as liver and spleen hematoma, and gastrointestinal contusion, ensure absolute bed rest, and give symptomatic treatment; in cases of liver and spleen ruptures and gastrointestinal perforations, stop bleeding and repair by emergency operation.
   d. When complicated by fractures, perform fracture reduction and fixation as early as possible, and make sure the fixation time is generally longer than the fracture time, which depends on clinical manifestations and X-ray examination results.
   e. When it is associated with craniocerebral injuries, sedation, pain relief, and bed rest are required for mild cases (concussions); in severe cases, surgery should be performed as early as possible, such as repair of skin lacerations, diorthosis of skull fractures, and evacuation of intracranial hematomas.
   f. Associated soft tissue injuries with radionuclide contamination should be decontaminated as soon as possible; in case of excessive radionuclides in the body, radionuclide removal should be accelerated; after early irrigation and debridement for soft tissue injuries, initial or delayed suturing is required.
   g. In case of associated limb crush injuries, immobilize the injured limb, avoid unnecessary activities, perform decompression by fasciotomy along the longitudinal axis of the injured limb, and fix it with thick-layered dressings after surgery. Prevent and control acute renal failure.
   h. In case of associated eye injuries, send the patients to the ophthalmologists after first aid.
5. Prevention and control of traumatic asphyxia

   Regarding casualties with severe dyspnea, perform tracheostomy promptly to remove endotracheal secretions, administer oxygen, and keep the airway open. Improve the respiratory function, and encourage the awake casualties to cough up sputum, perform mouth-to-

mouth resuscitation on casualties who have stopped breathing, but do NOT push against the chest.
6. For the seriously wounded with ruptured tympanic membranes, oronasal hemorrhage, and bloodstained frothy sputum, keep the injured head high for medical evacuation, and be sure not to help the casualty walk by the arm.

6.3 Early Treatment

1. Casualties with radionuclide contamination that exceeds the limits of contamination should have blood collected as soon as possible for determination of routine blood indexes and lymphocyte chromosome aberration rates and be decontaminated promptly.
2. Continuous oxygen administration: Administer oxygen under pressure if without rib fractures and pneumothorax, infuse hypertonic glucose and mannitol to reduce pulmonary edema and lower cranial pressure. After the blood pressure becomes stable, give diuretic therapy with furosemide or ethacrynate sodium, and an intravenous therapy of aminophylline to prevent bronchospasm. Do tracheostomy for casualties who are unconscious, have difficulty in sputum excretion, or feel asphyxiated. Cool the head temperature in case of cerebral edema.
3. Promptly perform abdominal laparotomy when abdominal organ injuries are suspected.
4. Pay attention to systemic and local infections: Early application of hematopoietic growth factors (G-CSF, IL-11) is advisable if complicated with moderate hematopoietic radiation sicknesses; broad-spectrum antibiotics for prevention and control of pulmonary infections if associated with disseminated intravascular coagulation (DIC) and hypokalemia following the principles of early, appropriate, and alternate use.
5. Surgical management: Make sure the wounds are closed up before the critical phase, try to fully decontaminate the contaminated wounds, reduce as many wounds as possible, transform open wounds into closed wounds, and convert severe wounds into mild wounds.
   a. Principles of surgery: As surgery may aggravate the condition, it should be well planned and fully prepared, i.e., adequate anesthesia, strict asepsis, skillful operation, and minimal anesthesia and operation time. The debridement should be thorough yet safe for healthy tissues. Hemostasis should be tightly applied, and the wound is generally sutured at a later date. Fracture reduction should be done early, and the fracture fixation time extended appropriately according to the clinical and X-ray examination results.
   b. Timing of surgery: All necessary operations should be performed early in the initial and pseudo-healing phases, and efforts made to allow for the healing of wound surfaces and wounds before the critical phase, during which, surgery is prohibited in principle, except for emergencies (i.e., blood vessel ligation, perforation repair); any surgery that can be delayed should be postponed until the recovery phase.
   c. Choice of anesthesia: Acupuncture, local, and epidural anesthesia can be used in all phases of combined injuries. Ether anesthesia and sodium thiopental anesthesia are suitable in the initial and pseudo-healing phases. For severe blast lung injuries, ether anesthesia is unsuitable, because it may aggravate pulmonary symptoms.

6.4 Specific Treatment

1. For severe body surface contamination, thorough decontamination is required; for casualties with radioactive contamination in the body, targeted pro-discharge drugs are recommended. Table 27.3 describes the methods to block absorption and accelerate the removal of common radionuclides.

2. Aggressive symptomatic treatment for different RCBI injuries
   a. Regarding mild injuries, give symptomatic treatment, strengthen nutrition, and get lots of rest.
   b. Regarding moderate and severe injuries, use antiemetic and sedative drugs in the early stages, and apply anti-releasing drugs as earlier as possible. In the pseudo-healing phase, focus on the prevention of infection and bleeding, and protect hematopoietic function. In the critical phase, further strengthen nutrition (intravenous or nasal feeding) while intensifying anti-infection and anti-hemorrhage treatment, maintain water-electrolyte balance, and give supporting therapy to improve body immunity. In severe cases, transfuse fresh whole blood and composition blood, and if necessary, transplant hematopoietic stem cells early after injury. Strengthen nutrition during the recovery phase to promote rehabilitation.
   c. In extremely severe cases, give early anti-infection and anti-hemorrhage treatment, and correct water-electrolyte disturbances.
3. Perform organ repair and orthopedic surgery after the recovery phase. Do automatic or passive exercises with the help of apparatuses as early as possible, or partial or full body baths, and maintain the function of the injured joint.

Table 3 Ways to accelerate the removal of common radionuclides

Radionuclide	Therapy
^{235}U, ^{238}U	Perfusion of 14‰ $NaHCO_3$ solution; quinamic acid and tiron
^{239}Pu	diethylenetriamine pentaacetic acid (DTPA-Ca) with Glucan or desferrioxamine (DFOA) or tetracycline; 0.5 g ethylene diamine tetraacetic acid (Ca-EDTA) intravenously or by nebulized inhalation
^{226}Ra, ^{228}Ra	EDTA and DTPA IV drips, high calcium diet, decalcification therapy
^{232}Th	EDTA and DTPA IV drips; quinamic acid
Iodine	130 mg potassium iodide (KI) orally
Strontium	Sodium fucoidan, 10 g dissolved in 2 cups of sugar water, orally, twice a day; high calcium diet, decalcification therapy
Cesium	Prussian blue, 1 g dissolved in water, orally 3 times a day, 15 days in total; high potassium diet; stable cesium
^{3}H	Drink plenty of water, apply diuretics such as hydrochlorothiazide
^{99m}Tc	Orally take 100 mg/d stable iodine, and 100 mg potassium perchlorate, 3 times a day; drink plenty of water; apply diuretics
^{60}Co, ^{57}Co	100 mg/day quinamic acid or DTPA, intramuscularly
^{210}Po	Dimercaptopropanol, NA-DMPS, sodium diethyldithiocarbamate trihydrate
^{144}Ce	Same as ^{239}Pu
Transuranium	Same as ^{239}Pu

6.5 Advances of Prophylactic-Therapeutic Research

Radiation injury is the main conflict in radiation-blast/radiation-trauma combined injuries, and its associated blast injury/trauma has complex impacts on the overall effects, cell-tissue-organ effects, and important pathophysiological processes of combined injuries, so the use of anti-radiation drugs to address the main conflict namely radiation injury has become a key part of the treatment of combined radiation-blast injury (CRBI). As mentioned above, although the anti-radiation drugs that are effective in acute radiation sicknesses are also basically effective in radiation combined injuries, some characteristics of their practical use need to be given due attention. The Army-wide Combined Injury Research Institute of the People's Liberation Army Medical University (ACIRI) and the Armed Forces Radiobiology Research Institute (AFRRI) have done a series of researches on the medication of radiation combined injuries.

Based on the important feature that radiation injury is the principal contradiction of radiation combined injuries, the ACIRI conducted an early study on the comparative effects of several anti-radiation drugs (WR_{2721}, cystamine hydrochloride, estriol, "523," and "408") for the prevention and treatment of radiation sicknesses and radiation-burn combined injuries. In terms of improving the 30-day survival rate (8-Gy γ-irradiation complicated with 15% III-degree burns on the body surface area, the 30-day survival rate of the mice in the control group was 8–10%), it increased to 70.8% among radiation injuries and 75.5% among radiation-burn combined injuries through WR_{2721} prophylaxis; after the use of cystamine hydrochloride, it increased to 52.6% among radiation injuries, and 53.7% among radiation-burn combined injuries; estriol treatment did not show any effect (3.7% in terms of radiation injuries and 1.1% in terms of radiation-burn combined injuries); "523" increased the 30-day survival rate of radiation injuries to 57.7%, and that of radiation-burn combined injuries to 62.2%, but the treatment did not show any effect, and the control had some effect; with "408," it increased to 22.7% and 24.9% for the two types of injuries, respectively. The use of these drugs in pairs also had some effect, roughly the same as with the effect of single drugs. Overall, WR_{2721} is good prophylaxis, but has major toxic and side effects. Its application in radiation-burn combined injuries also showed the following characteristics: the effective dose range is reduced (100–500 mg/kg for radiation injuries, and 100–300 mg/kg for radiation-burn combined injuries); the effective time is shortened (2 h to the time immediately after exposure for radiation injuries, and 0.5 h to the time immediately after exposure for radiation-burn combined injuries); the mortality on the day of administration is 28.6% for radiation injuries and 54.8% for radiation-burn combined injuries if 500 mg/kg is taken; 22.2% and 42.4% for radiation injuries and 54.8% for radiation-burn combined injuries if 400 mg/kg; after the administration of 300 mg/kg WR_{2721}, there is no death among radiation injuries, and the mortality of radiation-burn combined injuries is 16.7%. On the whole, although anti-radiation drugs are effective in the prevention and treatment of radiation-burn combined injuries, they reflect some characteristics worth noting. It should be pointed out that the radiation-blast/radiation-trauma combined injuries and the radiation-burn combined injuries have different characteristics, and the above conclusions can be referenced other than indiscriminately copied. At present, no prevention and cure research has been reported concerning the above-mentioned

anti-radiation drugs specific to radiation-blast/radiation-trauma combined injuries, especially since many of these drugs are in the nuclear accident emergency kits of the Chinese army and therefore need to be given sufficient attention.

The AFRRI has also done extensive work on screening for drugs that are effective against radiation combined injuries. The results show that many effective measures for radiation injury control are ineffective for radiation combined injuries, and some even reduce the survival rate of the latter. For example, the PEGylated G-CSF has a good efficacy when applied to radiation injuries and radiation-trauma combined injuries, but is ineffective for the treatment of radiation-burn combined injuries; the captopril, however, is able to improve the survival rate of radiation injuries, but significantly reduces the survival rate of the animals with radiation-burn combined injuries. To date, the institute has identified three drugs that are effective in the control of radiation combined injuries: ciprofloxacin (CIPRO), ghrelin, and vitamin E analogs (tocols).

6.5.1 Ciprofloxacin (CIPRO)

Ciprofloxacin is a quinolone antibiotic proven by clinical researches to have antibacterial and immunomodulatory properties that stimulate hematopoiesis and resist inflammation. Some investigators went on to test the therapeutic effects of ciprofloxacin in mice with radiation injuries (whole-body exposure to 9.25-Gy γ-ray) and radiation-trauma combined injuries (associated with wounds to 15% of the body skin). The results showed that the 30-day survival rate of ciprofloxacin (90 mg/kg by nasogastric tube, daily, 3 weeks) given within 2 h of injury was not statistically different, although it increased from 40% to 50% in terms of radiation injuries; however, the survival rate of radiation-trauma combined injuries enhanced from 35% to 80%, marking a significant difference. After the change of the dosage regimen, namely after 3 days of injury, it was still effective for the radiation-trauma combined injuries. According to mechanism researches, ciprofloxacin has protective effects on bone marrow and intestinal tracts, and anti-inflammatory effects in the model of radiation-trauma combined injuries. It can be seen that ciprofloxacin is an effective drug for the treatment of radiation-blast/radiation-trauma combined injuries.

6.5.2 Ghrelin

Ghrelin is a starvation-induced, predominantly gastric-secreted gastrointestinal hormone that plays the complex paracrine, autocrine, and endocrine roles through specific receptors. Earlier researches have shown that ghrelin has the ability to antagonize sepsis-induced vascular and pulmonary injuries and lower animal death rates. Afterwards, some investigators confirmed in a rat model of sepsis induced by the radiation injury (5 Gy γ-ray whole-body exposure) associated with cecum ligation and perforation (CLP) that ghrelin was effective in attenuating the effects of such combined injury on multiple organs and improved the 10-day survival rate of the animals from 38% to 69%. Mechanism researches demonstrated that ghrelin reduced serum catecholamine levels mainly through activation of the vagus nerve to realize anti-inflammatory action. The effectiveness of ghrelin in the treatment of radiation combined injuries was further confirmed by subsequent typical researches on radiation-burn combined injuries and radiation-trauma combined injuries. The 30-day survival rate increased from 55% to 64% (9.5 Gy γ-ray whole-body exposure) when 113 μg/kg of ghrelin was given in the caudal vein three times consecutively within 24 h, 48 h, and 72 h of radiation injury; the 30-day survival rate was down to 30% after complicated by burns (III-degree burns involving 15% of the body surface area), and up to 73% after the administration of ghrelin; the 30-day survival rate fell to 9% after complicated by trauma (skin wounds involving 15% of the body surface area), then enhanced to 82% after treatment. This shows that ghrelin, despite its limited effects in the treatment of radiation injuries, can improve the survival rates and prognosis of radiation-burn combined injuries and radiation-trauma combined injuries by a large margin.

6.5.3 Vitamin E Analogs

Currently proved four vitamin E analogs with good anti-radiation effects include DT3 (δ-tocotrienol), GT3 (γ-tocotrienol), AT (α-tocopherol), and TS esterification product (α-tocopherol succinate). Among them, TS, AT, and GT3 can be administered 24 h before irradiation to prevent radiation damage; while DT3 can provide relief through prophylactic use 24 h before irradiation and through therapeutic medication 2 h after irradiation. It is initially held that these vitamin E analogs exert their anti-radiation effects by the inherent free radical scavenging, namely the antioxidant effect of vitamin E. However, in-depth studies find that it is realized by inducing the up-regulation of G-CSF in vivo to mobilize the radiation-proof effects of hematopoietic stem cells and hematopoietic progenitor cells. The mouse model of radiation-trauma combined injuries (skin wounds involving 15% of the body surface area associated with 9.2 Gy γ-ray whole-body exposure) finds that TS administered 24 h before irradiation (400 mg/kg) has largely improved the 30-day survival rate (more than 40%), suggesting that TS can be effectively applied for the prevention of radiation-blast/radiation-trauma combined injuries.

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Toxin-Blast Composite Injury

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1 Overview

The so-called toxin-blast composite injury refers to simultaneous and secondary effects of shock wave and toxin on the body. Such injuries are characterized by difficulties in diagnosis and capturing the best treatment opportunity, which is why they are difficult to treat. Toxin-blast composite injuries constituted quite a large portion of the victims in the extra-large aluminum powder explosion in Kunshan on August 2, 2014, and the explosion at a Tianjin hazardous chemical storage warehouse on August 12, 2015, and fatality counts were high. For such instances, rapid emergency response and the proper medical care are vital. In the new century, people have been increasingly concerned about new military revolution spearheaded by advanced science and technology-empowered forces and the various impacts on future warfare arising from this revolution. In particular, people have realized that the widespread adoption of special weapons in modern hi-tech warfare is causing injuries, which constitute a series of new challenges for the development of military medicine across the globe. From the Gulf War and the Kosovo War, to the conflicts in Iraq and Syria, modes of battle such as long-distance strikes, precision-guided strikes, non-contact warfare, and beyond visual range (BVR) combat are continuously evolving. There have been broad applications of weaponry such as missiles, depleted uranium bombs, thermobaric weapons, joint direct attack munitions (JDAM), graphite bombs and fuel-air explosives, and the likes of laser weapons, microwave weapons, infrasonic weapons, weather warfare, electromagnetic pulse (EMP) weapons, new types of nuclear, biological and chemical weapons, binary chemical weapons, and neutron bombs have quietly emerged. Therefore, modern and tech-driven localized conflicts are characterized by high rates of injuries and deaths, extensive weapon effective duration, and complicated injury mechanisms, with increases in new types and forms of injuries that are harder to treat. Modern and tech-driven warfare affects traditional frontline rescue methods, and it is mandatory to renew understandings about certain issues in frontline medical care and engender a shift in philosophy. In future warfare, medical care and equipment must be on the same level as the frontline of battlefield, which would enhance medical treatment quality and reduce deaths, injuries, and subsequent disabilities, while relatively sound medical care support can provide a sort of mental support for combatants, helping stabilize their psyche. The nature of the concept of "injury" is rapidly changing. Under the current circumstances, medicine worldwide, in particular military medicine, the concept of "injury" is changing by leaps and bounds! The future is challenging tradition, and all notions and aspects from military-civilian, peace time–war time, philosophy, and principle to system, model, and academic discipline are all undergoing revolutions! The current international landscape is rather complicated, the world is not truly existing in peace and localized conflicts continue to erupt everywhere. The September 11 attacks shocked the world, and composite blast injuries were the most common among victims. In recent years, gas explosions in mines, explosions from explosives, explosions from car bombs, composite injuries from chemical weapons, poisoning from toxic gas accompanied by crushing injury, composite injuries from modern warfare and weapons, fires in recreational facilities and composite injuries from other accidents (with the majority being toxin-blast composite injury) have been occurring in higher frequency across the globe. At times, medical care personnel, or even those specializing in emergency medicine, have been shown unequipped to deal with these injuries, missing the best opportunity for treatment that might end up being lifelong regrets. At present, the international medical community is increasingly prioritizing studies and subjects related to treatment of toxin-blast composite injury. However, reports have pointed out that there is still a lack of effective

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methods for dealing with toxin-blast composite injury, and there are not any specialized antidotes or medicines for this type of composite injury. This is a great concern since toxin-blast composite injury could happen not merely during times of war but also in times of peace. Thus, toxin-blast composite injury is hard to handle in emergency situations or for clinical physicians, with the crux of the matter being difficult in diagnosis and capturing best opportunity for treatment. Therefore, actively performing basic and clinical care research programs on toxin-blast composite injury is a matter of great urgency.

2 Epidemiological Characteristics

1. Injuries from accidents in normal times usually occur because of explosions at places like chemical factory, weapon factory, and hazardous chemical warehouse.
2. Injuries from suicide terrorist bombing.
3. Injuries from explosion accidents during the course of research, production, and utilization of carrier rockets, missiles, and aerospace flight vehicle.
4. Injuries from explosions caused by weapons such as missiles, fuel-air explosive (FAE), and joint direct attack munitions (JDAM).
5. Injuries from aircraft, vessel, submarine, or other objects being struck by high-energy projectile.
6. Injuries from special types of weaponry.

3 Injuring Characteristics

1. The site of an event that caused toxin-blast composite injury is usually very chaotic, and it is possible that a large number of people are afflicted by toxin-blast composite injury within a short instant. On-site medical and rescue conditions are usually dire and dangerous. Thus, rapid emergency response and proper medical assistance are extremely important.
2. The ratio of toxin-blast composite injury victims is contingent on distance from center of explosion. The closer the distance to the center of explosion, the higher the chances of blast-burn-toxin composite injury, followed by toxin-blast composite injury.
3. Usually there are a large number of victims in need of medical attention concurrently. In many cases, toxin-blast composite injury victims appear in troves, with many suffering from critical wounds that demand emergency rescue and resuscitation. Conventional medical care methods usually will not get the job done.
4. Multiple injuring factors and complicated injuries. Explosion-induced toxin-blast composite injury is usually characterized by two or more injuring causes that mutually worsen each other in depth and/or breadth. Therefore, pathophysiological disorder compounds the severity and complexities in the multiple injuries afflicted to multiple parts of the body. Not only do such injuries affect a wide area and harm multiple body parts and organs, both whole-body and local responses might also be very strong and prolonged.
5. Highly complicated injuring mechanism. With said mechanism possibly associated with direct effects of shock wave and toxic gas, as well as related secondary injuries.
6. Injury to internal organs hidden by external wounds, leading to missed or erroneous diagnosis. When blast injury is compounded by poisoning from toxic gas, injury to internal organs could be overlooked, but how a victim survives or recovers largely hinges on severity of internal organ injury. Lack of knowledge in this area could easily lead to missed or erroneous diagnosis, or missing the best opportunity for medical rescue.
7. Lungs are the main organs injured. The lungs are the organs most susceptible to toxin injury and blast injury, therefore, lung injury is the challenge and focal point in medical rescue of toxin-blast composite injury.
8. Clinical manifestations of toxin-blast composite injury worsen at great pace and medical rescue is difficult. Clinically speaking, toxin-blast composite injuries are usually severe, compounded by various complications that lead to a relatively high rate of injury/fatality. Those suffering from serious toxin-blast composite injuries might die on the spot at the site of explosion.
9. Large destructive power and prolonged duration of effect. Complications are severe during the early stage of toxin-blast composite injuries, and complications become more numerous during the early stage of toxin-blast composite injuries. Such injuries usually affect a large area and many different body parts, thereby increasing the proportion of victims with injuries in multiple parts of the body. Following shock, hemorrhage, loss of consciousness, and other complications, along with increase in blast injury and poisoning, the ratio of severe injuries also rises correspondingly.
10. Simultaneous existence of internal injuries and external wounds. Blast injury afflicted upon the body is often ignored by people, extremely easy to cause missed or erroneous diagnosis. Difficulty and dilemma in treatment: the biggest challenge in treating toxin-blast composite injuries caused by an explosion is how to appropriately deal with the treatment difficulties and dilemmas brought about by the different injury factors. How to properly deal with the dilemma between IV injection and antidotes related to blast lung injury is key to treating toxin-blast composite injuries.

4 Injury Mechanism

The injury mechanism of toxin-blast composite injury is relatively complicated and remains somewhat unclear even today, with more research required to clarify the matter. It has been hypothesized that the injury mechanism of toxin-blast composite injury is related to the direct effects of shock wave and toxic gas, as well as the secondary injuries they induce.

The poisoning mechanism of toxic gas is the result of various kinds of toxic gases produced from the leakage or explosion of special kinds of fuel. For instance, dinitrogen tetroxide (N_2O_4) is currently one of the main liquid propellants for large carrier rockets and missiles used both in China and overseas. When accidental explosion occurs involving dinitrogen tetroxide and unsymmetrical dimethylhydrazine (UDMH) occurs, a litany of nitric oxide would be produced including dinitrogen tetroxide (N_2O_4), nitrous oxide (N_2O), nitrogen monoxide (NO), nitrogen dioxide (NO_2), and dinitrogen pentoxide (N_2O_5), and the mixture of these substances can easily poison those that inhale the gases. The injuring mechanism of nitric oxides chiefly involves the following aspects:

1. People inhale nitric oxides, which enter the body through the respiratory tract, leading to poisoning and injury to the respiratory tract including pulmonary edema and chemical-induced damaging pneumonia.
   a. Nitric oxides inhaled through the respiratory tract dissolve slowly and thus could penetrate deep into the respiratory tract. The gases would dissolve in saturated water vapor or liquids on the surface of pulmonary alveoli, turning into nitrate and nitrite, consequently stinging and corroding alveolar epithelial cells and capillary walls. These developments increase permeability, then a large amount of liquid white blood cells and vessels would leak, resulting in pulmonary edema.
   b. Injuries caused to type 2 epithelial cells of the lungs could reduce the amount of active substances on the surface of the lungs, inducing alveolar collapse and lowering alveolar pressure, leading to capillary hydrostatic pressure increase in response to alveolar pressure. A large amount of liquid would leak from vessels, resulting in pulmonary edema.
   c. Intracellular cyclic adenosine phosphate content would decrease, thereby weakening the function of the biofilm, in turn inducing tissue damage due to lipid peroxidation. If the aforementioned processes are not effectively interrupted, acute respiratory distress syndrome could develop, and in the long run pulmonary fibrosis and obstructive emphysema might occur.
2. Methemoglobinemia: Nitric oxides and nitric acids enter the body through different pathways, turning the body's hemoglobin into methemoglobin, resulting in methemoglobinemia. When methemoglobin content in the body reaches above 15%, cyanosis could occur, which would influence the oxygen-carrying function of red blood cells, further worsening hypoxia of the body and inducing all sorts of internal organ complications.
3. The body's defensive mechanism against viruses and bacteria would weaken: Inhaling nitric oxides over an extensive period could cause epithelial cilia of the bronchial and bronchiolar to fall off, reduction in mucus secretion and decline in alveolar phagocyte function, which would lower the body's defense against viruses and bacteria while significantly raising the chances of respiratory tract infections. Reference materials point out that when certain Muridae were exposed to NO_2 at 4/1,000,000 to 3/100,000 for 4 h, it was discovered that phagocytosis of macrophages and polymorphonuclear neutrophils was inhibited. Epidemiologic studies in humans also indicate that higher chances of respiratory tract infections are linked to outdoor NO_2 level.
4. Other injury mechanisms: Nitric oxides (such as NO_2) are free radicals that can attack cell membrane's unsaturated fatty acids (RH), in turn forming carbon-centered free radicals (R·) and oxygen-centered free radicals (ROO·), which would damage tissues. Some authors have reported that NO and its products can generate diacylglycerol and activate protein kinase C, which would play a role in signal transduction by way of regulating PLA_1 ligand binding in alveolar endothelial cell membrane. Other authors wrote that when human bronchial epithelial cells are exposed to NO_2, granulocyte and granulocyte–macrophage colony-stimulating factor are seen in culture medium, while TNF and IL-8 would elevate, contributing to secondary injuries caused by NO_2.
5. Composite effects cause the injuries to mutually worsen: The toxin-blast composite effects caused by special fuel should not be regarded as the sum of the effects of each individual injuring factor, but rather the classic example of "the whole is greater than the sum of its parts," as the various injuring factors of shock wave and toxin intensify each other's severity. Yue Maoxing et al. observed the effects of severe toxin-blast composite injuries on rats, discovering that pure blast injury resulted in pulmonary hemorrhage as the main lung injury, accompanied by mild pulmonary edema, while pure dinitrogen tetroxide poisoning led to pulmonary edema as the main lung injury, accompanied by some focal bleeding. However, toxin-blast composite injuries were much more severe than pure blast injury or pure dinitrogen tetroxide poisoning, with obvious pulmonary hemorrhage and mild pulmonary edema, and the drop in PaO_2 level two to six

hours after injury, extent of worsening in hemodynamics, and clinical symptoms and signs were all much more significant in toxin-blast composite injuries.

5 Clinical Manifestations

1. Symptoms and signs: The main symptoms and signs include poor general conditions, frequent coughs, breathing difficulty, or even respiratory distress up to 35–40 times/min or more, while tachycardia could reach over 125 bpm, along with other problems such as cyanosis, bloody and frothy fluid from the nose and mouth, chest pain, chest stuffiness, nausea, vomiting, headache, dizziness, and general physical weakness. When the injury is accompanied by UDMH poisoning, there would be significant changes in the nervous system. Other than the symptoms and signs stated above, there could also be problems such as muscle twitching and limb twitching, tightened jaw, breathlessness, exophthalmos, impaired coordination, mydriasis, unconsciousness, or even coma. During auscultation, both lungs would produce low breathing sounds full of dry and moist rales, along with wheezing sounds heard during bronchospasm. When there is traumatic shock, hypovolemic shock might be observed as a clinical manifestation. Blood in stool might be found when blast injury is accompanied by gastrointestinal injury, blood in urine might be found when accompanied by injuries to kidney and bladder, and signs of peritoneal irritation sign might be found when accompanied by ruptures in liver, spleen, or gastrointestinal tract.
2. Laboratory examination
   a. Blood routine examination: It is common to see elevated white blood cell count and increased percentage of neutrophils. After composite injury, if there is reduction in red blood cell, white blood cell, or platelets in complete blood cell, along with drop in body temperature, these are indications of severe injury and unideal recovery.
   b. Chest X-ray: Increased bronchovascular shadows and flaky or foggy shadows might be seen, and issues like subphrenic free air might be observed when gastrointestinal tract is ruptured.
   c. ECG: Tachycardia, low voltage, reduction in ST-T, or even inverted t-wave.
   d. Respiratory function: Arterial blood gas analysis might show clear drop in PaO_2, and Yue Maoxing et al. reported about 19 cases of nitric oxide-induced acute pulmonary edema, in which PaO_2 measurements hovered between only 5.3–7.9 kPa. There could also be imbalance in ratio of ventilation and perfusion, resulting in a rise of pulmonary shunt volume. Zhengguo Wang et al. reported that average pulmonary shunt volume in dogs rose from 4.7% before blast injury to 21.9% 8 h after blast injury. Other changes include lowered lung compliance and obstructive ventilation function disturbance, among others.
   e. Blood methemoglobin test: With nitric oxide poisoning, different degrees of methemoglobin concentration increase might be observed in blood, and when concentration rises above 15%, a clinical manifestation of cyanosis might occur.
   f. Blood enzyme test: With nitric oxide poisoning, increased enzyme activity might be seen in glutathione peroxidase, glutathione reductase, glucose-6-phosphate dehydrogenase, and others, the increase of which depends on concentration of inhaled nitric oxide. When shock wave causes myocardial contusion, rises in SGOT, LDH, and CPK-MB could be observed, and if liver was ruptured, there would also be increases in SGPT and SGOT.
   g. Other supplemental test: B-mode ultrasound and CT scan can show changes in liver, spleen, and kidney ruptures caused by shock wave and help categorize the severity of injuries incurred.

Based on the clinical symptoms and signs mentioned above, as well as relevant laboratory tests, and in combination with the reason(s) behind the explosion event, it would be possible to diagnose toxin-blast composite injury.

6 Rescue Strategies and Measures

1. On-site treatment principles and rescue principles
   a. The medical emergency rescue and treatment of toxin-blast composite injury is a comprehensive system and requires a set of complete, highly efficient, and scientific management methods executed by practical and proficient instructing management personnel. It is necessary to strengthen the organizational command of emergency rescue team, establish adequate commanding organ to take charge of the main command of emergency response and rescue, continually enhance and perfect medical rescue system, and give full play to the technical guidance of frontline treatment and emergency rescue experts group on-site.
   b. Establish a safe, effective, and unobstructed rescue channel: Humans have ushered in the age of information, and therefore, establishing a safe and effective toxin-blast composite injury unobstructed rescue channel is very important, so as to ensure that medical treatment network, communication network, and transportation network can operate in a highly efficient manner.

c. Design a systematic and comprehensive medical treatment plan: In light of the complexity in the medical treatment of toxin-blast composite injury and the abruptness of accidents that cause such injuries, relevant experts should be organized to formulate on-site emergency medical treatment procedures, evacuation procedures and standards, etc. for victims of special injuries. Also include medical emergency plan or rescue plan, etc.
d. Establish large-scale toxin-blast composite injury victim categorization system: Build a high-quality team of emergency rescue team, train a group of self-rescue and mutual rescue key personnel, strengthen on-site treatment, speed up the evacuation of victims, try to minimize the time span between injury and surgery, and underscore that improving basic treatment technique is the number one priority in improving treatment of large groups of toxin-blast composite injury victims.
e. On-site treatment principles: It is crucial to underline the importance of on-site treatment and rescue of toxin-blast composite injury victims in the early stage. It is necessary for medical personnel to rush to the explosion site and effectively provide basic trauma life support (BTLS), then quickly transfer victims to hospitals with relatively sound technical capacity, which would be conducive to substantially raising the chances of successful rescue. Therefore, it is necessary to strengthen the work of on-site first aid, widely promote CPR on-site treatment techniques, and improve the self-rescue and mutual rescue knowledge and abilities of the public. Communication, transportation, and medical treatment meanwhile are the three chief factors before arrival at hospital, and it is necessary to sufficiently make use of the functions and effects of each and every one of them. Underline the importance of the ten minutes and one hour after injury, so that victims can receive the most optimal treatment as soon as possible. It is necessary to persist in scientific treatment principles, including taking into account and simultaneously treating injuring factors of both poisoning and blast injury in victims of toxin-blast composite injuries.
f. On-site treatment principles: Ensure survival before treatment, tend to the severely injured before those mildly injured, rescue first before treatment and administer treatment processes during emergency rescue, leave the accident site as soon as possible, categorize first before evacuation, and medical personnel should focus on treatment, while other personnel stress on emergency rescue so as to maximize opportunities. For severely poisoned victims, the "firstly wear, secondly isolate, thirdly evacuate" emergency rescue protocol should be adopted. The "firstly wear" refers to the fundamental requirement that rescuers should wear proper protective equipment, the "secondly isolate" means that properly equipped rescuers should try to contain the toxic gas and prevent poisoned victims from inhaling in more gas, and "thirdly evacuate" is the evacuation of poisoned victims away from the danger zone upon the basis of "firstly wear, secondly isolate," so as to create better conditions to perform further medical treatment. It would be optimal to allocate two rescuers for each poisoned victims to speed up the rescue and evacuation process.
g. Main contents of on-site treatment:

- Establish unobstructed, safe, and effective emergency rescue channel as soon as possible.
- Quickly cut off the source of injury and it is key to accomplish objectives such as extinguish fire, control explosion, or protect against explosion.
- Control the contaminated zone: Identify the boundary of the contaminated zone, set up clear signs, control traffic in the surrounding area, and prevent unrelated personnel or vehicles from entering the contaminated area.
- Rescue poisoned victims: Evacuate poisoned victims to safety zone for rescue, then send to hospital for emergency treatment.
- Confirm the nature and threat level of dangerous chemicals, so as to better understand the possible scope of toxin dispersion.
- Organize residents in the contaminated zone to protect themselves or evacuate: Instruct dwellers in the contaminated zone to learn how to protect themselves, and if the necessity arises organize them to evacuate.
- Cleaning and decontamination of contaminated zone: Clean and decontaminate the area based on the chemical nature of the dangerous chemicals and degree of contamination.
- Locate and dispose of bodies of dead animals to prevent decomposition from harming the environment.
- Earnestly carry out related tasks in aspects such as meteorology, transportation, communication, material supply, and protection.
- All personnel in rescue team should wear the corresponding protective equipment on site and strictly uphold protection protocols. Since the on-site emergency rescue at the location of dangerous chemical explosion is extraordinarily complicated, emergency rescuers need to understand the physical, chemical, and poisonous properties of the chemical in question, so as to better defend and

protect oneself. Due to the constant and sudden changes on site, relevant personnel need to stay alert and react in a proper and timely fashion.

- Basic principles: Prepare adequately ahead of time, react swiftly, carry out treatment in a multi-dimensional manner, and establish comprehensive system; provide uniform command and collaborate closely; concentrate resources and ensure key tasks are accomplished; and earnestly ensure effective and scientific rescue and treatment.

h. Evacuate from site: Evacuate victims from the site of injury as soon as possible to stop chemical substances from further harming the body, but do not sacrifice preemptive treatment for the sake of hasty evacuation to the hospital. The injuring effect of chemical is closely associated with its concentration and duration of effect. Steps must be taken to understand the physical and chemical properties of the chemical, so as to select the appropriate cleaning and decontamination method. Generally speaking, concentration and duration of effect of the chemical are inversely correlated to harms to the body. Therefore, the first step is to remove clothes contaminated by the chemical, then use a large amount of water to clean the surface of the wound and surrounding skin for the purpose of firstly, diluting the chemical, and secondly, mechanical effects of running water. If there are also burn injuries, washing will also cool down the burns. The requirements are to wash with enough water and wash for an adequately long duration, and regular water (tap water, well water, river water, etc.) is good to use. Sustain the washing and rinsing for more than 1 h, especially when dealing with alkali burns, washing for too short a duration will not produce the needed result. Heat might be generated during washing, but since washing is sustained, heat would be quickly dissipated. Although some hazardous chemicals are not soluble in water, it is still possible to rely on the mechanical effect of running water to wash the surface of the wound clean. When quicklime is involved in the injury, first remove the substance before washing and rinsing with a large volume of water so as to prevent lime from generating heat with water and worsening surface of the wound. The wounded should keep warm if the area of burn injury is large. Therefore, it is necessary to use water with a temperature of around 40°C. Bandage the surface of wounds after sustained washing and rinsing, then quickly transfer to a specialized hospital for further treatment.

Compared with other disasters, it is necessary to plan ahead for chemical disasters, execute rapid local (on-site) decontamination and subsequent whole-body decontamination, and efficiently set up decontamination measures for large groups of victims. However, nations around the world have not yet developed personal washing and decontamination techniques for dangerous chemicals. Accidents involving explosion or body contamination caused by such chemicals occur often, which is why personal washing and decontamination techniques for dangerous chemicals require more in-depth research. Since contamination mechanisms of such chemicals are largely similar to those of chemical and bio weapons, the two could be studied and developed in tandem. New types of solutions produced for the decontamination system or procedure should be abundant and easily obtained. It is also necessary to accelerate the research and production of new types of washing and decontamination equipment, and such washing and decontamination equipment with high washing and decontamination efficiency, speed, smaller consumption of washing and decontamination agent, high mobility and multifunctional properties will surely become the trend in the future. New types of washing and decontamination techniques such as anti-toxic paint, microwave plasma decontamination, auto-oxidation of toxic, laser disinfection, adsorbing decontaminant, hot air disinfection, detergent decontamination are being studied, and the efforts to seek economic, simple, convenient, easily applied and effective decontamination methods and pathways have promoted the growth of washing and decontamination technologies in China.

i. Special anti-toxin: After skin comes into contact with toxin, the key is to wash and decontaminate the affected body parts as soon as possible and apply special anti-toxin drug. The application of special anti-toxin and anti-shock drug is an effective treatment for poisoning and burns from chemicals. In principle, the aim is to reach the effective dosage as quickly as possible, but it is also necessary to pay attention to actual quantity used to prevent adverse effects of the drugs. Clinical treatment and research both indicate that the combination of scopolamine [0.33 mg/(kg day)] and dexamethasone [0.33 mg/(kg day)] shock therapy produces relatively ideal results for the majority of chemical poisoning and burns, thus it is worthy of broad promotion. More troublesome are some toxins for which anti-toxins have not yet been developed. For treating this kind of toxins, increase the urination frequency of the wounded to remove the toxin from inside the body and lower the impact of the toxin on the wounded. Intravenous infusion coupled with diuretic is a common method. Severe methemoglo-

binemia due to poisoning from toxins like cyanide, aniline, or nitrobenzene requires oxygen supply and depending on the situation, injection of fresh blood, to improve anaerobic condition. In the case of methemoglobinemia, intravenously administer 5 ml of 1% methylene blue 5 ml, 2 g of vitamin C, and 20 ml of 0.9% normal saline slowly. It is suggested to apply metacortandracin, hydrocortisone, or dexamethasone at the early stage of dermal toxicity, in order to minimize hemolytic reaction.

j. A new treatment is the application of vitamin B6 with 20AA compound amino acid (Fengnuoan).

- Both clinical treatment research and basic research have proved that the new treatment method of "vitamin B6 with 20AA compound amino acid (Fengnuoan)" solely invented by Yue Maoxing is highly effective in treating toxin-blast composite injury. This is because vitamin B6 is the sole coenzyme in the metabolism of various amino acids and the coenzyme for several dozen enzymes in the liver; therefore, only when a relatively large dosage of vitamin B6 is involved, the body's metabolism could be activated. The start-switch value of enzyme metabolism is 3–5 g, but since vitamin B6 has a short half-life and is quickly excreted from the body, not a single instance of overdose was observed in more than 200,000 cases of clinical application. The aminogram of Fengnuoan is basically identical to that of the human body and provides metabolic substrates and powerful impetus after injection while promoting excretion of harmful substances and ammonia from the body through ornithine metabolism, facilitating the rapid recovery of enzyme metabolism in the liver. The combined usage of the two produces desired effects in promoting enzyme metabolism in the body, urination and detoxification, protecting the functions of the brain and nervous system, enhancing liver functions, raising the body's coagulation function, and improving nutritional condition, thereby fostering a certain degree of restoration to damaged cells. The clever combination of Fengnuoan and vitamin B6 plays a particularly crucial role in metabolism in the body.
- Clinical application method: (1) For patients with severe injury: Fengnuoan 500 ml/day, intravenous drip once per day; 5% normal saline 250 ml + 5 g of vitamin B6 + 2 g of vitamin C, intravenous drip twice per day, continual application until patient condition is stabilized. (2) For patients with moderate injury: Fengnuoan 500 ml/day, intravenous drip once per day; 5% normal saline 250 ml + 5 g of vitamin B6 + 2 g of vitamin C, intravenous drip once per day, continual application until patient condition is stabilized. (3) For patients with minor injury: Fengnuoan 500 ml/day, intravenous drip once per day; 5% normal saline 250 ml + 3 g of vitamin B6 + 2 g of vitamin C, intravenous drip once per day, continual application until patient condition is stabilized.
- Cautions: (1) In emergency room, injury severity should be graded according to Injury Severity Score (ISS), as in score between 9 and 15 is minor, score between 16 and 25 is moderate, and score over 26 is severe. Carry out APACHE scoring of patient upon arrival at hospital. (2) When treating patients with critical or severe toxin-blast composite injury, administer through middle cardiac vein to help control the critical or severe conditions, buy time for emergency operation and subsequent treatment, and significantly reduce mortality rate. No injuries are caused to the major organs; (3) when administering via peripheral veins, there might be swelling and painful sensation around the veins if drip rate is high, so keep the rate low, choose larger veins and hypodermic needles with a bigger gauge to reduce adverse responses. 0.5% to 1% of patients might complain about reactions in the gastrointestinal tract after IV drip, use 10 mg of metoclopramide administered via intramuscular injection to lessen the reactions.

k. Emergency treatment: While treating chemical poisoning, it is mandatory to pay attention to the existence of any life-threatening blast injuries such as cerebral trauma, cardiopulmonary arrest, suffocation, bone fracture, or collapsed lung. If the aforementioned composite injuries exist, apply the necessary emergency treatments in line with first-aid principles for trauma.

l. Protection of surface of wound: Protecting the surface of a toxin-blast composite injury wound from infection is a pretty crucial step. Use clean sheets or clothes to wrap up the wound, with the key being protecting the epidermis while not bursting any blister. For the surface of severe wounds, there is no need to apply any medication to avoid increasing the difficulty of treatment upon arrival at hospital. When washing and rinsing eyes, normal saline (0.9%) may be used and utilize cotton swabs to wipe away foreign substance, then apply antibiotic eye ointment or anti-inflammation eye drops.

m. Sedation, analgesia, and anti-shock: It is common for patient suffering from toxin-blast composite injury to experience different degrees of pain and anxiety, it is

acceptable to administer orally ingested sedatives (i.e. librium, diazepam, etc.). When victim exhibits symptoms such as dehydration or early shock, if the person can still ingest, provide mild saline solution in small and frequent doses, but drinking plain water or sugar water is prohibited. For victims afflicted by serious toxin-blast composite injury, food consumption is prohibited within 24 h after injury because it would be extremely common for vomiting to happen as a result, in addition to abdominal bloating caused by swallowing air. If victim is very thirsty, give a small volume of water to quench the thirst and make sure the person stays warm.

Heightening on-site toxin-blast composite injury rescue capacity and improving treatment capacity during subsequent evacuation process are key steps in enhancing the overall toxin-blast composite injury treatment level. Ambulance needs to be outfitted with resources such as oxygen, painkiller, intravenous fluid, splint, stretcher, medical aspirator, first-aid equipment for trauma, disinfection equipment, long-distance communication device, etc., so as to augment the medical treatment capacity during the course of evacuation. If conditions permit, use emergency medical helicopter to speed up the evacuation.

n. Cut off injuring factors immediately and evacuate from site of explosion as soon as possible: Remove contaminated clothing as quickly as possible, and at the same time disengage from contact with toxin, rapidly and effectively cut off toxin entry pathway, and immediately evacuate victims to an upwind and safe area. For nitric oxide in contact with skin or the eyes, use a substantial amount of water to rinse and apply atropine sulfate and antibiotic solution eye drops to prevent eye infection. For all victims located at site of explosion, keep in mind the possibilities of blast injury and carefully observe any telltale signs.

2. Quickly administer anti-shock and anti-toxin treatment, rectify any cerebral hernia, and at the same time take measures to prevent pulmonary and cerebral edema.
   a. Quickly administer anti-shock and anti-toxin treatment, rectify any cerebral hernia, and at the same time take measures to prevent pulmonary and cerebral edema: Taking active and effective steps to prevent pulmonary and cerebral edema are keys to toxin-blast composite injury prognosis. For the wounded of severe toxin-blast composite injury, main causes of death during the early stage include shock, cerebral hernia, serious burns, poisoning, cardiac arrest after trauma, etc., and taking active and effective steps to prevent pulmonary and cerebral edema early on are crucial to successful rescue. At the same time it is necessary to take measures to prevent pulmonary and cerebral edema. A key anti-shock step is to swiftly establish two or more venous access, then expand capacity, carry out blood transfusion, and ensure sufficient oxygen intake. Moreover, quickly perform surgery while administering active anti-shock measures, and undertake thoracotomy or laparotomy to investigate and rapidly control any localized injuries from deteriorating, which could happen very fast. The main method for cerebral hernia rectification and intracranial pressure reduction early on entails high-rate intravenous drip of 20% mannitol coupled with diuretic. High dosages of dexamethasone and human albumin administered early on could lessen edema of the brain. Yet pre-surgery preparations need to be ready, and surgeries such as clearance of intracranial hematoma, treatment focal contusions, or other decompression surgeries should be performed, which are the fundamental measures for dealing with serious craniocerebral injury and cerebral hernia. However, craniocerebral injury coupled with hemorrhagic shock brings about dilemma in treatment. The basic principle to follow should be: firstly administer anti-shock treatment, then apply dehydrating agent, and use colloidal solution such as whole blood, plasma, or low molecular dextran, which could expand capacity and rectify shock while not worsening cerebral edema. This group was comprised of victims suffering from liquid rocket UDMH and dinitrogen tetroxide poisoning, specifically skin contamination of UDMH and dinitrogen tetroxide. First and foremost, quick and prompt rinsing and decontamination are keys, then apply special anti-toxin with haste. The special anti-toxin drug crucial to treatment of UDMH poisoning is vitamin B6 and acetonyl-acetone, with the general principle being applied early and sufficiently to reach effective treatment dosage as soon as possible while paying attention to any side effects. Scopolamine paired with dexamethasone shock therapy produces relatively ideal effect for dinitrogen tetroxide poisoning.
   b. Rapid, accurate, and thorough diagnosis is required: Usually the procedure involves emergency rescue undertaken in tandem with inspection and medical history inquiry, then continue emergency rescue and inspection to prevent missed diagnosis. If there are any doubts in the diagnosis, when patient condition has been stabilized, use supplementary inspections (i.e. B-mode ultrasound, X-ray, CT scan, etc.) to obtain a thorough diagnosis. Pay special attention to: (1) Whether the wounded of severe craniocerebral injury is also suffering from shock and cervical spine injury; (2) whether severe abdomen crushing injury is coupled with ruptured diaphragm; (3) for pelvis frac-

ture, look for internal organ injury of pelvic cavity and abdominal cavity; (4) whether severe chest injury is coupled with heart injury; (5) for lower chest injury, look for ruptured liver or spleen; (6) especially for toxin-blast composite injury, blast injury to the body is most frequently neglected; and (7) whether there is poisoning from inhaling nitric oxides from asbestos, dust, smoke, or other products generated by the explosion.

c. Sequence of surgery: First and foremost, the first priority is to control any life-threatening traumas, keep this in mind when deciding the sequence of operation. Usually the emergency operation (ruptured heart and great vessels) is followed by acute surgery (ruptured organs of the abdominal cavity, extraperitoneal hematoma, or open fracture) and then selective surgery (closed fracture of the limbs). However, if all injuries require acute surgery, craniocerebral surgery should be a top priority, then surgery for organs of the pelvic, abdominal, or chest cavity, and lastly surgery for limbs, spine, etc. Emergency room surgery is preferred. For victims of serious composite injuries, time is life. For instance, injury to a great vessel of the heart requires surgery as quickly as possible. If the patient is transferred to ward operating room, some patients might die during the transfer process. Surgery needs to be fast and effective, with the absolute priority being saving life, and then preserve bodily functions.
d. Take an active approach to prevent or treat ARDS and MOF after surgery, as ARDS and MOF are major causes of death among toxin-blast composite injury patients in the later stages after trauma. Therefore, early prevention is optimal. Under specific critical situations, combine intravenous therapy of anisodamine (20 mg/8 h) with dexamethasone (40 mg/8 h) shock therapy, and the new therapy of vitamin B6 paired with 20AA compound amino acid (Fengnuoan) to reverse the condition of toxin-blast composite injury patient.
e. Treatment of acute chemical pulmonary edema: The key treatment for dangerous chemical poisoning is the application of special anti-toxin drug, with the general principle being applied early and sufficiently to reach effective treatment dosage as soon as possible while paying attention to any side effects. Main treatment methods for acute chemical pulmonary edema include:

- Bed rest: Patients suspected of suffering from lung injuries should rest in bed to reduce stress on the heart and lungs, which would be favorable to preventing pneumorrhagia from worsening.
- Keep respiratory tract unobstructed: If there are burns in the airway, severe upper airway obstruction, or dangers of suffocation, tracheotomy should be done as early as possible.
- Oxygen therapy includes intermittent high-flow oxygen intake (3–5 L/min), and simultaneously use oxygen bubbled through a 50% alcohol solution for anti-foaming, or use 1% dimethyl silicone oil atomizer for defoaming. How to use: this medicine, readied in breathable form, should be placed 8–10 cm from the patient's nose and mouth to facilitate inhalation. Usually inhalation should continue for 40–60 breaths, then observe patient for 5–10 min, and repeat procedure when necessary.
- Relieve bronchospasm: Use 0.25–0.5% isopropylnoradrenaline, or 0.2% salbutamol or dexamethasone aerosol, inhale several minutes each session. Or, add 0.25–0.50 g of bronchodilator agent aminophylline to 20 ml of 50% normal saline, then inject intravenously at a slow pace. Stop treatment when symptoms subside.
- Mechanically assisted breathing: If oxygen therapy is unable to rectify the excessively low oxygen partial pressure, and if anoxia throughout the body has not improved, mechanically assisted ventilation should be employed. Generally speaking, intermittent positive pressure breathing (IPPB) may be adopted, to raise effective alveolar ventilation while reducing physiologically ineffective cavity and pulmonary shunt volume, thereby improving oxygen utilization. If IPPB is still unable to elevate oxygen partial pressure to 10.7 kPa (80 mmHg), consider switching to continuous positive pressure breathing (CPPB). As the general consensus is that blast injury is usually accompanied by air embolus, so CPPB should be avoided. If aeroembolism occurs during treatment, discontinue immediately. Some have recommended high-frequency ventilation therapy because it offers relatively low tidal volume and airway pressure, making it suitable for patients suffering from air embolus by lowering the risk of aeroembolism.
- Dehydration: Usually 20 mg of furosemide is given once to twice a day for two to three consecutive days, or 250 ml of 20% mannitol administered via intravenous drip to be completed within 30 min.
- Strengthen myocardial contractility: Give 0.2–0.4 mg of cedilanid via intravenous therapy for those with fast heartbeats, and in the case of circulatory failure, dissolve 0.125–0.25 mg of strophanthin K in 20 ml of 25% glucose solution, to be administered via slow intravenous drip.
- New therapy of vitamin B6 paired with 20AA compound amino acid: With proven efficacy for diuresis, detox, anti-oxidation, leakage reduction,

promotion of body enzyme metabolism, and protection of cerebral and nervous systems, it is highly effective in treating chemical-induced pulmonary edema.

f. Pay attention to psychological problems of toxin-blast composite injury patients: It is common for toxin-blast composite injury patients to find themselves unable to mentally cope with the powerful stimuli of the event, often manifested as fear, believing in rumors, etc. This kind of injury inflicts obvious psychological trauma on the patient, so pay attention to psychological problems of toxin-blast composite injury patients and adopt effective countermeasures in a timely manner.
g. Symptomatic treatment and supportive therapy are crucial to treating toxin-blast composite injury, and the basic principles include:

- Pay close attention to changes in injury conditions, in particular arterial gas embolization instigated by blast injury, delayed gastrointestinal perforation, delayed pulmonary edema caused by poisoning, etc.
- Maintain water, electrolyte, and acid-base equilibrium, and rectify hypoxemia promptly.
- Support organ functions and defend against organ function disorders through methods such as sufficient and effective resuscitation, clear and drain the infected areas, and support circulatory and respiratory systems and metabolism.
- Supplement appropriate amount of plasma or albumin when the timing is right.
- Effectively control tic and convulsion, and when administration of vitamin B6 still cannot stop convulsion, apply 0.2 g of phenobarbital sodium via intramuscular injection.
- Apply anti-oxidation agents such as vitamin C, vitamin E, glutathione peptide, fatty acid, or taurine individually or in combination to help reduce lung problems arising from poisoning.
- Regulate immunity by administering ginsenoside, astragalus polysaccharide, artificial recombinant thymosin, etc. to enhance the body's immunity.

3. Beware of delayed chemical-induced pulmonary edema due to toxin-blast composite injury. Beware of delayed chemical-induced pulmonary edema in toxin-blast composite injury victims. This kind of victims is commonly found among youngsters. Clinical manifestations: There will not be obvious symptoms early on and the patient could move about freely. Around 20 h later, patient would experience sudden onset of chest discomfort, breathing difficulty, and pain behind sternum, then more obvious breathing difficulty, followed by coughing up bloody or foamy sputum, along with cyanosis. There would also be tachycardia with heart rate around 125 bpm. ECG would show P-wave in the lungs and multi-lead ST segment depressions. X-ray would show small cloud shadows, and some might be lumped together. Partial arterial oxygen pressure should be around 5.3 kPa when inhaling high-concentration oxygen. Anoxia would continue to worsen, and patient might die of respiratory and circulatory failure. Usually the situation deteriorates rapidly soon after onset of illness, giving rescuers very little time to react, resulting in patient mortality quickly. This is why preventative measures are crucial, and the country's military standard for observation of poisoned victims has been extended from 24 h to 48 h.
4. Cautions about on-site processing and rescue of personnel in contaminated zone
   a. Cautions about evacuation of personnel from contaminated zone

- Ensure adequate protection before evacuation: Prior to evacuation, personnel should put on face mask on one's own or with the help of others, or use wet towel to cover one's nose and mouth. Also, put on safety suit or raincoat to protect exposed skin from harm.
- Quickly determine wind direction: During evacuation, personnel should swiftly determine wind direction using items such as flag, tree branch, or handkerchief.
- Prevent secondary injury: Personnel in contaminated zone should try to use transportation vehicle to evacuate from site.
- Perform first aid in a safe area.

   b. Cautions about on-site first aid

- Correctly performing rinsing, bandaging, restoration, fixation, relocation and other corresponding actions are hugely conducive to lowering disability rate. Reduce pain: Use common and special rescue technique to stabilize patients' feelings and alleviate pain.
- Ensure one's own protection: Delegate tasks, ensure the task allocation, ascertain that everyone is clear about responsibilities, and work together as a team. It is necessary to prepare plans and contingencies for on-site treatment and rescue ahead of time.
- Dealing with contaminants: Pay attention to processing of contaminated clothing in order to prevent secondary injuries.

- Adequately protect eyes of patients: Remember not to neglect inspection and treatment of eyes for possible injuries.
- Medical personnel need to be knowledgeable about protection: On-site first aid for toxin-blast composite injury is a complicated task, and medical personnel need to not only master a certain number of first-aid rescue techniques, but also obtain knowledge about the physical, chemical, and toxic properties of the hazardous chemicals causing the poisoning, and how to protect against them.

Clinically speaking, toxin-blast composite injury develops rapidly and is difficult to treat, resulting in high rate of case fatality. Therefore, comprehensive and effective treatments are crucial, including cardiopulmonary resuscitation (CPR), application of anti-foaming agent, ultrasonic aerosol inhalation, utilization of anti-allergy drug or alkaline neutralizer, elimination of methemoglobinemia, joint application of scopolamine and dexamethasone shock therapy, joint application of vitamin B6 and 20AA compound amino acid, adoption of proper body position and facilitating high-flow oxygen intake to ensure oxygen supply to tissues and cells, maintaining functions of key organs, rectifying imbalance in water, electrolyte, acid, and base. It is suggested to perform surgery when necessary and take an active approach with symptomatic treatment and supportive therapy, to promote the body's recovery and healing, among other goals.

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Part V

Blast Injury in Special Environments

Plateau Blast Injury

Zuoming Yin, Bo Kang, and Hao Yin

An injury caused by the direct or indirect effects of blast wave in a plateau environment is called a plateau blast injury. Historical data on warfare conducted on plateaus indicate that conflicts taking place on plateaus result in more blast injury victims than those carried out on normal plains, and injuries are more severe. Plateau blast injury usually refers to blast injury suffered at an altitude of 2500 m or more above sea level, and the occurrence, development, and treatment of this type of injury share some of the same rules and patterns as those in low-altitude regions, but due to the unique natural and geographical environment of plateaus, such as low-atmospheric pressure and partial oxygen pressure (area 4000 m above sea level only has 61% of that of sea level), arid and cold climate, and strong UV ray. Under such circumstances, the characteristics and treatment of blast injury differ from those in low-altitude regions. Therefore, the correct perception about plateau blast injury is practically meaningful in the treatment of plateau blast injury victims.

1 Injuring Features of Plateau Blast Injury

From a medicine standpoint, a "plateau" usually refers to an area 2500 m above sea level where there are clear biological effects on the human body. The Qinghai-Tibet Plateau of China, dubbed the "Roof of the World," has an average elevation of more than 4000 m and accounts for 1/6 of China's territory. Therefore, the Qinghai-Tibetan Plateau occupies a crucial position in national defense. Due to the unique geological environment of plateaus, blast injuries are usually more severe than those on normal plains. At the same time, the tolerance for shock wave in organisms on plateaus changes drastically compared to low-altitude area. Generally speaking, blast injuries are more severe and complex on plateaus than plains, and fatality rate is also higher. Research report states that blast injuries at elevations of 4000 m and 5000 m above sea level are one to three grades more severe than those on plains, and fatality rates, respectively, climb by 25% and 35%.

1.1 Characteristics of Physical Parameters of Plateau Blast Injury

Since blast wave uses other media to propagate throughout its surroundings, differences in density impart a certain degree of impact on the physical parameters and injuring effects of a shock wave. The higher the density of the medium, the faster the velocity of propagation, and the farther the distance propagated. Compared with areas at low elevation, plateaus have thinner air while atmospheric pressure reduces further and further as altitude increases. When elevation rises above 1000 m, atmospheric pressure reduces by about 10 kPa, oxygen content drops by around 3 kPa, and temperature lowers by roughly 6 °C. The specific mass of air at sea level is 1.2255 kg/m^3, but at an altitude of 4000 m, the figure falls to 0.8020 kg/m^3, only 65.44% of that of sea level, while air density at an elevation of 5500 m is only 50% of that of sea level. Therefore, given thinner air and lower density of mass on plateaus, shock wave propagates slower than at plains and also declines faster and propagates a shorter distance. Therefore, an explosion from the same mass of explosive might produce a lower peak overpressure than that on a plain at the same distance from the center of explosion.

Li Xiaoyan et al. studied how differences in plateau (elevation of 3500 m) and plain (elevation of 380 m) affect the physical parameters and injuring effects of shock wave from detonating 10 kg of TNT. Experiment results indicate that propagation velocity of shock waves from the plateau group does not differ significantly from the plain group; meanwhile, there are some variations in shock wave peak

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overpressure, positive pressure effective duration, and impulse at areas close to and far from the centers of explosions of the two groups, though the figures are more consistent in the zone 6.0–7.5 m from the center of explosion. The general trends include lower peak overpressure for plateau group than plain group, with difference being significant at 6.5 m from center of explosion; positive pressure effective duration for plateau group is longer than that of plain group, with significant statistical differences at 6.0 m and 6.5 m from center of explosion, which might be attributed to slightly longer positive pressure effective duration on plateau compared with plain. In general, the aforesaid results reflect the pattern of reduction in shock wave peak overpressure and extension in positive pressure effective duration in plateau environment. Impulses of shock waves in plateau group were also higher than those of plain group, which would cause more serious injuries to the body. Outcomes from this experiment indicate that when the same mass of TNT is detonated in air, blast injury is more severe on plateaus than that on plains. In particular lung injury, severity is about one grade more severe, and two animals died directly from blast injury.

1.2 Target Organs of Plateau Blast Injury

Liu Xiulai et al. carried out animal testing at a location of 4740 m above sea level on the Qinghai-Tibet Plateau to study the characteristics and patterns of injury from the explosion of shells fired from rocket launchers under alpine highland condition. Ten northern Tibetan sheep weighing between 15 kg and 25 kg were placed inside shelter pits (1.5 m long, 1.0 m wide, 0.8 m deep), each 10 m apart within the bombardment zone. One sheep was placed in each pit, and each animal was tied with rope to a wooden stake driven into the bottom of the pit so that they could not jump outside the pit. 20 min after shells exploded in the bombardment zone, researchers were quickly delivered into the explosion site via armored vehicles, and they discovered that all ten sheep were dead. Of which, nine were struck by shrapnel totaling 24 wounds and the animals averaged 2.67 wounds each, with four wounds being the most. All shrapnel wound openings and passages were heavily contaminated by soil. Six animals were hit in the head by shrapnel (67%), six hit in the neck (67%), four in the chest (44%), five in the abdomen (56%), and four in the limbs (44%). Of those struck in the limbs, there were five bone fractures and all were comminuted fractures, including four in the thigh bone and one in the shin bone. As shown, shrapnel wounds from plateau blast injury have a high rate of occurrence and cause severe injuries and high fatality rate. In terms of the types of wound tract, there were 16 perforating wounds (66.7%), five blindgut wounds (20.8%), and three tangential wounds (12.5%). Upon careful on-site inspection, one sheep was found to be free from

Fig. 1 Experimental site of explosion blast injury on plateau (by Yin Zuoming)

shrapnel wounds. In situ dissection was carried out, discovering hemothorax and numerous blood spots on lungs. A substantial number of experiment outcomes already prove that the fatality rates from plateau blast injury and composite injury are significantly higher than those on normal plains, with air-filled organs such as the lungs and intestines still being the main injured/fatal targets, while injuries to solid organs like liver, kidney, and brain being relatively less frequent (Fig. 29.1).

1.3 Characteristics of Injury Conditions of Plateau Blast Injury

The low pressure and low oxygen content on plateaus could instigate a series of pathological and physiological changes in the body, including reduction in storage function of organs and decrease in the body's tolerance of shock wave, with these differences manifested in more serious and complicated injuries from explosion on plateau compared with those in low-elevation areas. Damon's research results prove that as ambient pressure drops, the body's tolerance for shock wave decreases significantly, demonstrating a significant fall in shock wave overpressure required to cause the same fatality rate in animals. Research outcomes show that when goats under ambient pressure of 103.45 kPa were struck by impact, the reflected pressure needed to cause 50% fatality 1 h later was 392.41 kPa. Meanwhile, when goats under ambient pressure of 48.28 kPa were struck by impact, the reflected pressure needed to cause 50% fatality 1 h later was only 173.79 kPa. Experiment results from Chinese scholars Yang Zhihuan et al. show no difference. Their 2003 study reveals that when the ambient atmosphere pressure decreases, lung injury severity significantly increases, with

this difference manifested as more serious pulmonary hemorrhage and pulmonary edema, and a jump in pulmonary index. Outcomes indicate that as the ambient atmosphere pressure falls, animals become less tolerant of shock wave, with worsening of lung injury and a rise in fatality rate. Coronary or cerebral artery embolism induced by aeroembolism after lung injury, or acute cardiopulmonary insufficiency due to severe pulmonary hemorrhage and pulmonary edema could contribute to the death of animals. Compared with blast injuries on plains, plateau blast injuries are increasingly more severe with the reduction in the ambient pressure because animals are less tolerant of shock wave, leading to higher fatality rates.

1.4 Reduction in the Body's Tolerance of Blast Injury on Plateau

The mechanisms behind the reduction of the body's tolerance of air shock waves on plateau are still not fully understood. Possibilities include lower partial oxygen pressure in the air on plateaus, and this shortage of oxygen results in an increase in the fragility and permeability of pulmonary capillaries. When exposed to a shock wave, pulmonary capillaries are more prone to rupturing and bleeding, and there is more liquid permeation due to the elevated permeability, in turn leading to more serious pulmonary hemorrhage and pulmonary edema compared to blast injuries in areas with low altitude, as well as higher death rate. In addition, due to the lower atmospheric pressure of the plateau environment, the pressure difference effect created by shock wave is more significant compared to areas with low altitude, thereby causing a drop in the body's tolerance for shock wave. At the same time, during resuscitation on plateaus, the lungs have weaker bearing capacity for liquids. Since the lungs are the most susceptible target organs to shock waves, IV injection and infusion volumes and rates have to be adjusted when resuscitation from blast injury coupled with trauma is required. Otherwise, it would be extremely easy to cause pulmonary edema and other serious consequences. When it is necessary to use air transport for evacuation, try to fly at lower altitude to minimize chances for air embolism.

In light of the above, plateau blast injuries may be characterized as exhibiting the following features:

1. Changes in characteristics of physical parameters of blast waves on plateaus: In areas near the center of explosion, plateau blast waves propagate at relatively slower speed compared to those on plains, shock wave peak overpressure is also lower compared to that on plains, but positive pressure effective duration is longer than that on plains. These changes result in higher airblast impulse on plateaus relative to that on plains, thereby intensifying injuries to the body.
2. Target organs of blast injury: The lungs are still the main target organs for blast injuries on plateaus and also the chief cause of death as seen in animal testing. The rates of occurrence of injuries to the gastrointestinal tracts are similar between plateaus and plains, but injuries on plateaus are more severe. This difference might be attributed to increased gastrointestinal gas expansion at areas of high altitude.
3. Injury severity: Relative to plains, injuries on plateaus are usually one to two grades more severe.
4. High rate of fatality: Damon et al. reported that when goats were exposed to shock waves under ambient pressures of 82.8 kPa and 48.3 kPa, the reflected pressure needed to cause 50% fatality 1 h later was 365.01 kPa and 173.88 kPa. In other words, compared with the high ambient pressure of the former, a shock wave under the low ambient pressure of the latter only needs to have 47.64% as powerful an overpressure to cause the same fatality rate. Chinese scholars subjected rats to reflected impacts from overpressure of 190.40 kPa, respectively, under the ambient pressures of 96.60 kPa, 61.33 kPa, and 53.99 kPa, discovering that death rate after 6 h is, respectively, 0%, 25%, and 35%.

2 Clinical Features of Plateau Blast Injury

An injury caused by the direct or indirect effects of blast wave in a plateau environment is called a plateau blast injury. The characteristics of such blast injuries include minor injuries on the outside and severe injuries on the inside (minor wounds on the surface and severe injuries to internal organs), rapid development (rapid deterioration of conditions for moderate or more severe injuries) and usually injuries are suffered by multiple body parts or organs. Therefore, it is necessary to carry out thorough and detailed inspections to prevent any missed diagnosis. Due to the hidden and severe natures of such injuries, victims of such injuries should receive prioritized treatment and attention. Injuries caused by blast waves are often times more severe in air-filled organs. Due to their structural features, the lungs are always the main target organs in blast injuries. The most common manifestation is hemorrhage affecting both lungs, along with hemothorax, pneumothorax, and air embolism. In addition, blast injuries might also cause perforation of intestines (most frequently the lower small intestine and colon) or other organs in the abdominal cavity, along with ruptured bladder, as well as ruptured liver and spleen, and bleeding of organs of the abdominal cavity. Liquid-filled organs such as gall bladder and renal pelvis meanwhile are usually less seriously injured or even unscathed. The degrees of injury to tissues and organs depend upon strength of peak overpressure,

length of positive pressure effective duration, and speed of positive pressure increase. Compared with lung blast injuries on plains, lung blast injuries in high-elevation areas cause more complicated pathological changes in the body, more serious conditions, and higher rate of fatality.

The main clinical features of plateau blast injuries are listed below. Firstly, multiple injuries are usually multi-organ/body part injuries or composite injuries with complicated conditions. Secondly, minor injuries on the outside and severe injuries on the inside, with the possibility of perfectly unscathed surface but obvious symptoms and severe injuries to internal organs. Thirdly, rapid development is manifested with conditions most severe within 6 h or 1–2 days after injury, and when the body's compensation function is maladjusted, conditions could deteriorate quickly to a point of no return.

Main clinical manifestations of plateau blast injuries include chest pain, chest discomfort, sense of suffocation, breathing difficulty, abdominal pain, nausea, vomiting, clear pressure and pain in the abdomen, rebound tenderness and abdominal muscle tension, among others.

2.1 Clinical Manifestations of Blast Lung Injury

The lungs are two of the most susceptible target organs when a shock wave acts on the human body. Blast lung injury is quite often seen in both times of war and accidental explosions during times of peace and is one of the key subjects of research in both military medicine and disaster medicine. After the lungs are injured by a shock wave, a series of pathological and physiological changes to the body would be instigated. Clinically speaking, the symptoms and manifestations of blast lung injury are most frequently covered up by other external wounds, in particular injuries that are more easily diagnosed such as burns or bone fractures. Thus, the most important aspect when it comes to diagnosing this type of injury is to clarify clinical materials. For such patients, the existence of blast lung injury should be fully considered so as to carry out preventative treatments in a prompt manner.

2.1.1 Main Pathological Changes in Blast Lung Injury

The main pathological changes of blast lung injury are pulmonary hemorrhage, pulmonary edema, ruptured lung, formation of pulmonary bullae, pulmonary collapse, and emphysema.

Pulmonary Hemorrhage

Pulmonary hemorrhage is the predominant pathological change in blast lung injury, with severity and scope of bleeding differing drastically due to injuring conditions, from spots of bleeding to diffuse hemorrhage. For those mildly injured, there might just be spots of superficial hemorrhage in the visceral pleura, and for those in more dire situations there might be patches of bleeding on one or more lung lobes, which could affect the deeper parts of the parenchyma. For those severely injured, parallel strips of parenchyma hemorrhage in the intercostal muscles might be seen. It is also common to see the characteristic hemorrhagic indentation in the subpleural tissue adjacent to the chest wall. For blast lung injuries, blood vessel rupture in pulmonary parenchyma may turn into a hematoma, and the patient might even die from suffocation of the body caused by blood clots that block the trachea. Wang Hongya et al. carried out tests of plateau blast injury to the chest using common rabbits at an area with elevation of 3658 m. The researchers performed pathological observations of lung tissues 6 h after the injury.

1. General observations: The plateau injuring group and plain injuring group both showed injuries in the right lungs, with dark red coloring, flushed lungs, and obvious edema. Lung injury is usually manifested as pulmonary hemorrhage and edema, and lightly red edema liquid that oozes out from cross-section. Pulmonary hemorrhage often affects the right middle and right lower lobe, even as deep as the parenchyma, exhibited as irregular patches of bleeding. Of which, patchy hemorrhages are most common (Figs. 29.2 and 29.3).

2. Optical microscope observation: Under optical microscope, diffuse alveolar hemorrhage was found in the injury groups, with edematous fluid filling, alveolar cavity fusion, emphysematous changes, and alveolar wall capillary bed reduction, dilatation, and congestion. The

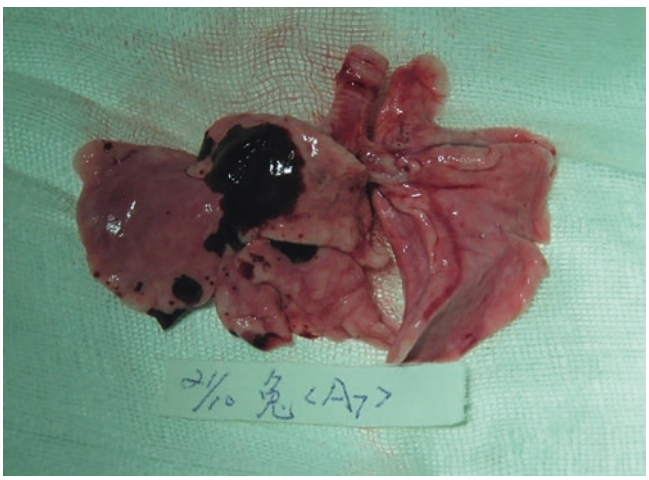

Fig. 2 In the high-altitude blast injury group, there were multiple patchy hemorrhages in the right lungs and scattered patchy hemorrhages in the left lungs (figure by Wang Hongya)

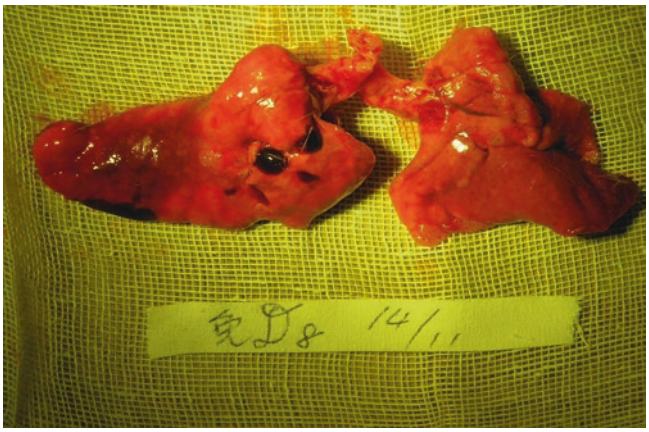

Fig. 3 In the plain injury group, there were patchy and striped hemorrhages and edema in the right lungs (figure by Wang Hongya)

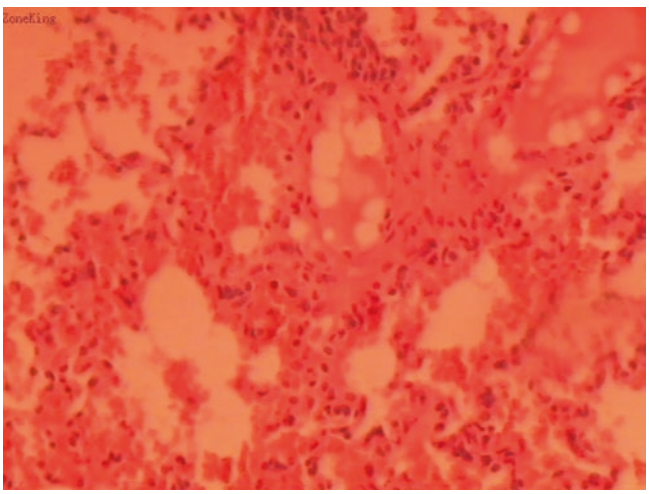

Fig. 4 Pulmonary hemorrhage, edema, and bubbles in the edema fluid in the high-altitude injury group (HE×100) (figure by Wang Hongya)

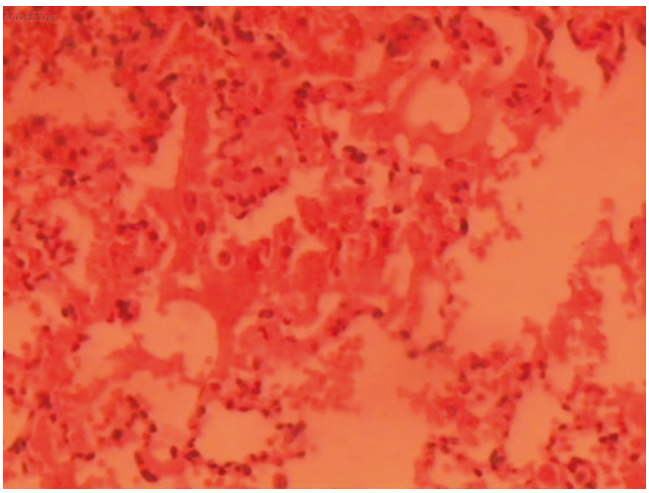

Fig. 5 Pulmonary hemorrhage and edema in the plain injury group (HE ×100) (figure by Wang Hongya)

majority of edemas occur near areas of bleeding, and bubble-like structure consisting of a mix of edematous fluid and gas could be seen. There are significant edema and hemorrhage in the pulmonary interstitium, the alveolar septum and perivascular space have widened to various degrees, and pulmonary infiltration with eosinophilia could be seen. Pulmonary hemorrhage and edema in the plain group were slightly less severe than the plateau group, see Figs. 29.4 and 29.5.

Pulmonary Edema

Pulmonary edema could occur right away after blast injury. For minor injuries, there could be interstitial fluid accumulation or a small amount of fluid in the alveolar cavity. For severe cases, there might be voluminous amount of edematous fluid spilled into the bronchus and trachea, which could be mixed with blood and seen as bloody, foamy fluid. This is usually found near areas of severe bleeding, with the red, foamy mixture consisting of edematous fluid and blood. Under optical microscope, homogeneous reddish fluid with a substantial quantity of red cells and a small amount of air could be seen. Pulmonary edema that occurs during this stage is usually caused by capillary injured directly or indirectly by shock wave. One to two days after injury, light red edema areas with relatively defined boundaries could be found around patchy hemorrhage areas. Edema areas are bloated, full, moist, and glistening, do not lighten in color when pressed, and light red foamy liquid would ooze out from cross-section. Under optical microscope, capillaries pumped with blood could be seen as well as serous fluid and some red blood cells in the alveolar cavity, and some alveolar cavities have hyaline membrane-like structures lining the alveolar walls.

Lung Rupture

Lung ruptures are mainly caused by dynamic pressure. Under the effects of dynamic pressure, the body often collides against a hard object after being thrown or displaced, or wounded after being hit by a secondary projectile. Animal testing observations indicate that rupture openings most frequently appear on the medial side of the lungs, and second most frequently on both sides of the lungs, while openings on the lateral (rib) side of the lungs are rare.

Pulmonary Bullae

Pulmonary bullae is formed when the body collides against a hard object after being thrown or displaced by dynamic pressure, or wounded after being hit by a secondary projectile. Pulmonary bullae is actually tears of superficial lung tissue, while visceral pleura remains intact.

Pulmonary Collapse and Emphysema

Pulmonary hemorrhage and edema could disable the lung's ability to expand. In areas without hemorrhage, sometimes small, irregular patches of collapse could be seen. It is also common to see less air content in alveoli near areas of bleeding. Within areas of diffused bleeding, emphysema and bleeding often coexist, and some alveoli might burst because of over-expansion, resulting in the fusion of several alveolar spaces. Sometimes interstitial emphysema is caused by air entering the interstitial lung through ruptured alveoli or bronchioles. For severe cases, pulmonary bullae containing blood and air would appear in the subpleura, and hemothorax or hemopneumothorax might occur when a lung ruptures.

Acute Respiratory Distress Syndrome

The acute respiratory distress syndrome (ARDS) is a complication that occurs when the lungs are injured by a shock wave, and it is one of the main causes of death in the early stages. Even when emphatic treatment and measures are adopted, fatality rate still stands at 40%. The chief pathological characteristic of ARDS is increased permeability and inflammatory cell infiltration due to pulmonary microvascular endothelial injury, often accompanied by pulmonary interstitial fibrosis. Clinical manifestation is almost incurable hypoxemia that would ultimately develop into respiratory failure.

Important Cellular Inflammatory Response

1. One hour after blast injury, neutrophils from circulating blood migrate to the lung tissue wound, and lung injury would worsen at an accelerated pace because of the release of cytokines, reactive oxygen species, proteolytic enzymes, and cationic polypeptides. Perl et al. verified through blast lung and sepsis composite injury experiments that removal of neutrophils could lessen lung injury severity. In addition, the level of activity of myeloperoxidase (MPO) in injured lung tissues is a key indicator for assessing the extent of neutrophils invasion into lung tissues. Blast lung injury could result in hypoxemia, and this short-term hypoxia not only magnifies the function of neutrophil anticytotoxin, but also prolongs its existence, instigating sustained high inflammatory response, ultimately leading to injury to tissue or organ.
2. Alveolar macrophages are derived from monocytes in the bone marrow, and they could be classified into numerous sub-types based on their position, such as alveolar macrophage (AM), pulmonary interstitial macrophage, pulmonary intravascular macrophage, pleural macrophage, vascular macrophage, dendritic cell, etc. AM is primarily distributed inside alveolar space, accounting for some 80% of resident cells. AM not only possesses phagocytosis function, but also secretion function. AM can induce the neutrophils to stay and accumulate in the pulmonary microvessels, where they would adhere to endothelial cells, release inflammatory mediators and destructive toxic substances, thereby injuring alveolar capillary membrane and inducing permeability pulmonary edema. In blast lung injury experiments, there were increases in inflammatory cells released by AM in both bronchoalveolar lavage fluid and blood.

Apoptosis

Apoptosis is a programmed cell death process intended to maintain normal tissue form and function, and it is a physiological mechanism that plays an essential role in maintaining stability in the body's internal environment. This is usually manifested as the death of a single cell to prevent provocation of inflammatory response. Apoptosis pathways can be generally divided into either caspase-dependent or caspase-independent. The caspase-dependent pathway can be further sub-divided into either death receptor-mediated exogenous apoptosis pathway or mitochondrial-mediated endogenous apoptosis pathway. Caspase occupies a crucial position in apoptosis, and its involvement is a downstream event common in the activation in many different apoptosis pathways. Setiz et al. discovered through injured animal modeling that extracellular signal molecule Fas/FasL stimulated caspase-8 in lung tissues, causing an increase in the expression of caspase-8. This discovery proves extracellularly induced apoptotic mode initiation.

Due to the aforementioned pathological changes, in addition to low pressure, low oxygen, and other disadvantageous environment conditions of plateaus, it is common to see blast lung injury victims to quickly exhibit breathing difficulty and the almost incurable hyoxemia. Blast lung injuries caused by reasons such as explosion of explosives are usually compounded by severe burns, bone fractures, and other problems, creating complex composite injuries that gravely increase difficulty in treatment.

2.1.2 Main Pathological and Physiological Changes in Blast Lung Injury

Wang Hongya et al. carried out tests of plateau blast injury to the chest using common rabbits at an area with elevation of 3658 m, so as to study about features of blast lung injuries to rabbits on plateaus. They recorded breathing rate (BR), heart rate (HR), mean arterial pressure (MAP) and central venous pressure (CVP), measured NO concentration in plasma, and documented changes in arterial blood gas analysis before injury and 5 min, 1, 3, and 6 h after injury. The research indicates:

1. For the plateau injury group, 25% had severe injury, and both minor and moderate injuries stood at 37.5%. Meanwhile, for the plain group, minor injuries accounted for the majority at 62.5%, moderate injuries came in at second with 37.5%, and there were no severe injuries.

Breathing rate increased, peaking at 1 h after injury; blood pressure and heart rate decreased, most obvious 5 min after injury ($P < 0.01$), which then gradually recovered, restoring to pre-injury levels 6 h after injury. Water content in lungs of plateau injury group was higher than that of plain injury group, and pulmonary hemorrhage and pulmonary edema in plain injury group were somewhat milder than those in the plateau injury group. These outcomes show that blast lung injuries in rabbits on plateau were more severe than those on plain.

2. Before injury, plasma NO concentration in plateau group was lower than that of plain group ($P < 0.05$), and after injury the readings in both groups continued to rise, and NO concentrations in both plateau injury group and plain injury group differed significantly related to figures before injury ($P < 0.05$). At the various measurement time slots, changes in NO concentrations in plateau injury group were significantly bigger than those in plain injury group ($P < 0.01$). This shows that NO might be one of the inflammatory media that participates in acute lung injury after blast lung injury. The lack of oxygen on plateau coupled with blast lung injury could result in NO abnormality that further lowers pulmonary perfusion and aggravates ischemic hypoxic injury. This revelation is crucial to early-stage treatment of plateau blast lung injury.
3. The clear drop in various arterial blood gas indices 1 h after injury and persistent metabolic acidosis are the chief characteristic changes in blast lung injury, and these are vital in early-stage treatment.

2.1.3 Clinical Manifestations of Blast Lung Injury

The clinical manifestations of blast lung injury vary depending on severity of injury. Those mildly injured might experience short-term chest pain, chest stuffiness, or sense of suffocation. Those moderately injured might cough, exhibit hemoptysis, or have bloody sputum 1–3 days after injury, while a small number might complain about breathing difficulty, and variably diffuse moist rales or crepitation could be heard through auscultation. Severely injured patients would suffer from breathing difficulty, cyanosis, bloody and foamy sputum, etc., often times accompanied by shock. Hemothorax or pneumothorax might appear in even more serious cases. For some victims, 24–48 h later blast lung injury might develop into acute respiratory distress syndrome (ARDS). Signs: Other than rales in the lungs, signs of pulmonary consolidation and hemopneumothorax might be identified, usually accompanied by symptoms related to injuries to other organs. X-ray inspections would show increased bronchovascular shadows, spotty, or patchy shadows and decreased transmittance in the lungs, as well as larger patches of dense shadows. It is also possible to see pulmonary atelectasis and hemopneumothorax. Chest CT scan: If images show increased density in cloud flocculent shadow that would be indications of pulmonary alveolar and interstitial hemorrhage. Arterial blood gas tests might produce abnormal results to varying degrees of seriousness, with persistent hypoxemia being very common.

In light of blast injury history, clinical manifestations and X-ray inspections, blast lung injuries can be considered quite easy to diagnose, but it is necessary to remember characteristics including minor injury on the outside and severe injury on the inside, rapid deterioration and frequent accompaniment by other injuries, so as to avoid missed or erroneous diagnosis.

2.1.4 Complications of Blast Lung Injury

1. Pneumonia: Pneumonia is the most common complication of blast lung injury and is associated with diffuse capillaries, damaged alveolar membrane, elevated alveolar permeability, and reduction or inactivation of alveolar surfactant after blast lung injury, resulting in lung infections.
2. Acute respiratory distress syndrome (ARDS): Lung changes related to ARDS originate from widespread damages to alveolar capillaries, which raise permeability between endothelial cells, leading to problems such as alveolar hemorrhage and edema. Ultimately, invalid cavity and shunt in the lungs would increase, and pulmonary compliance and oxygenation would deteriorate, resulting in respiratory distress clinically. Generally speaking, pathological changes include three phases: exudation phase, hyperplasia phase, and fibrosis phase. For a patient, the degree of pulmonary fibrosis determines his or her lung performance in the future.
3. Multiple organ dysfunction syndrome (MODS): MODS is the simultaneous or successive failure of two or more injured organs during the course of severe trauma, burns, large abdominal surgery, shock or infection. MODS usually occurs after a patient is resuscitated from the aforesaid problems and his or her condition has stabilized. MODS includes a period of organ injury worsening from minor to severe. For minor cases, there would be abnormal physiological functions of organs, while severe cases would see organ and system failures, which would be termed multiple organ failure. Among blast lung injury victims, it is common to see concurrent existence of trauma, burns, and lung injuries, and shocks and infections are also very frequent. Therefore, MODS complication is a high risk.

2.2 Clinical Features of Abdominal Blast Injuries

Blast injuries to organs in the abdominal cavity usually affect air-filled organs such as gastrointestinal tract. Due to environ-

ment features such as high altitude and low pressure of plateau, relative to plain, gastrointestinal tract would naturally bloat to a certain extent, which would further worsen the severity of plateau blast injury. Pathological changes are mainly manifested as different levels of serosal and submucosal bleeding of the gastrointestinal tract, or even gastrointestinal tract rupture, perforation, with injury of the colon being the most obvious, followed by small intestine and stomach. Multiple intestinal contusions or hematoma of the intestinal wall might be seen in some victims, particularly the colon segment, which is full of air. Under the microscope, blood leakage between the basement membrane of the small intestinal epithelium and the muscularis could be seen, along with fractured muscularis mucosa in multiple places, loss of normal structure of the muscularis, myolysis in certain areas, vacuolization in the muscle nuclei, and incomplete serosal membrane. Furthermore, there might be blood deposits in the capillaries in the submucosa, and exfoliated mucosal epithelium might be observed at times. Pancreas hemorrhage, and liver, kidney, and bladder rupture might occur in a small number of patients, but injuries to these solid organs are relatively rare.

Abdominal blast injuries usually induce hemorrhage inside the abdominal cavity and peritonitis. Hemorrhages are most obvious in injury to blood vessels or the rupture of solid organs such as the liver and spleen. Peritonitis meanwhile is caused by contents that spill out from ruptured hollow organs such as the stomach or intestines.

Based on different injured parts and severity of injury, the following clinical symptoms and signs might be observed: (1) Abdominal pain is the most common symptom and usually starts with the body part injured and then spreads to the rest of the abdomen. Perforation of body parts like stomach, upper bowel, and gallbladder would often induce sharp and diffused pain; with colon perforation, pains are less severe and remain local, but can easily lead to septic shock; (2) nausea and vomiting: nearly half of abdominal blast injury victims would experience short-term or sustained nausea and vomiting, or other digestive tract symptoms; (3) hemorrhagic shock: bleeding from ruptured solid organs inside the abdominal cavity such as spleen and liver could lead to symptoms such as hemorrhagic shock; (4) signs of peritoneal irritation: victims suffering from ruptured hollow abdominal organ could exhibit clear pressing pain, rebound pain, abdominal muscle tension, or other signs of peritoneal irritation during the early stage; (5) other symptoms: injuries to kidney or bladder could result in bloody urine, and fresh and bright red blood from the anus indicates injury to the lower part of the abdomen, possibly the rectum or colon. Perforation of the stomach or intestines could result in symptoms like subphrenic free air, pneumoperitoneum or disappearance of distinguishing dull sound of liver. Injury to pelvic organ might irritate colon, leading to frequent urge to defecate.

Supplemental inspections: (1) X-ray and CT scan: plain abdominal radiograph could confirm whether or not there is perforation in the digestive tract, and if intestine is perforated, X-ray of the abdomen would reveal subphrenic free air. If kidney or ureter injury is suspected, intravenous pyelography may be employed to help with diagnosis. CT scan has higher diagnosis rate for abdominal injuries than X-ray. (2) Diagnostic abdominal paracentesis: If a closed abdominal organ injury is suspected, administer diagnostic paracentesis. The test result is a positive if bloody fluid is extracted. This test could reach positive rate of more than 85% for closed injury. (3) Diagnostic peritoneal lavage: Test result is a positive if peritoneal lavage fluids has a light red color, or if red blood cell count $>0.1 \times 10^{12}$/L, or if peritoneal lavage fluid contains bile, vegetable fiber, feces, or other substances. This test could reach positive rate of more than 97%. (4) Color Doppler ultrasonography: Ultrasonography is convenient, non-invasive and could be done by the bed. It provides real-time and dynamic inspections of changes in organs in the abdominal cavity and offers rather important values in the diagnosis for injuries of solid organs such as the spleen, liver, kidney, and pancreas, as well as retroperitoneal hematoma and abdominal fluid accumulation. (5) Laboratory tests: Serum glutamic pyruvic transaminase (SGPT) activity would raise if liver is ruptured, with figures to surge by four to five folds in 12 h after injury compared with numbers before injury. When the pancreas is injured, elevation of serum amylase and lipase may be observed.

3 Treatment Principles for Plateau Blast Injury

After a blast injury occurred, injuries that are life-threatening should be treated firstly, such as ensuring unobstructed airway, maintaining effective circulation, and controlling obvious bleeding. The emergency treatment principle is identical to the first-aid for general traumas. Based on the characteristics of plateau blast injury, and in accordance with the key factors in the treatment of plateau blast injury and composite injury, pay attention to the lungs because these are the main target organs in blast injury. The lungs are extremely vulnerable to the almost incurable hypoxemia, and severely lung injury is one of the primary reasons for death in the early stage. Therefore, emphasizing the treatment and protection of lung injury are especially important. The number one priority in the treatment of plateau blast injury is to maintain respiratory and circulatory function, including keeping airway unobstructed, providing oxygen, and when necessary adopt measures such as tracheotomy and mechanical ventilator-assisted breathing, as well as blood transfusion, rehydration, and anti-shock treatment.

3.1 Cautions in Treating Plateau Blast Injury

The principles for first-aid, ventilation, hemostasis, wound dressing, shock prevention, and other steps for plateau blast injury are the same as other battlefield traumas. Given the characteristics of plateau blast injury, particular attention should be paid to the following few areas:

1. Swiftly remove victim from the explosion environment, collapsed fortification or building, so that the person is relocated away from the source of explosion. Erratic movement after infliction of blast injury could worsen the situation. If the victim shows respiratory distress, it would be necessary to remove him or her from the battlefield via stretcher. Anyone suspected of suffering from blast lung injury should lie down and rest to lessen stress on the heart and lungs.
2. For victims with breathing difficulties, check to see if his or her mouth and nose are obstructed with foreign obstacles like sand or blood clot and clear out these substances if there are any. For victims exhibiting respiratory distress or coughing up large volume of blood, quickly insert nasopharyngeal airway or oropharyngeal airway, and if necessary, perform tracheal intubation and suck out secretion in order to keep the airway unobstructed.
3. For unconscious victims, pull his or her tongue out from the mouth and tilt head sideway to keep the airway unobstructed. For those with bloody fluid flowing out from nose or mouth, or those with extremely serious breathing difficulty, perform tracheal intubation or tracheotomy, and suck out any liquid inside to keep the airway unobstructed. For victims with breathing difficulty or those with gradually lowering partial oxygen pressure, provide oxygen via nasal cannula or face mask. Use oxygen bubbled through a 50% alcohol solution, administer aminophylline to stop bronchospasm, and when necessary employ mechanical ventilation. For victims showing ARDS during the acute stage, if regular treatments cannot improve his or her condition, apply extracorporeal membrane oxygenation (ECMO).
4. For victims with air embolism, administer pressurized gas at six bar (with oxygen no higher than 2.5 bar) sustained for 2 h, then continue for 36 h at gradually decreasing pressure.
5. When performing resuscitation on the plateau, the lungs cannot bear much liquid, and since the lungs are the chief target organs most sensitive to blast injury, if there is a need to perform resuscitation due to blast composite injury, make appropriate adjustments to the volume and rate of fluid transfusion, prioritize capsules over crystals whenever possible, and monitor hemodynamics carefully, otherwise pulmonary edema and other dire consequences could occur extremely easily.
6. Sedation and analgesia of patient after injury; keep the patient's body temperature normal, prevent pulmonary edema and protect cardiac function; dehydrate, ensure diuresis and strengthen the heart; provide blood and fluid transfusion; and prevent DIC and electrolyte imbalance. Administration of large doses of corticosteroid hormone during the early stage is relatively conducive to defending against interstitial pulmonary edema.
7. Carefully inspect the severity of external wounds and visceral injuries, assess the patient's conditions, rapidly categorize patient injuries, and strictly uphold the treatment principle of dealing with severe injuries before minor injuries. Carry out systematic inspection prior to surgery, and pay particular attention to injuries of internal organs, especially hollow organs such as the lungs and gastrointestinal tracts. At the same time, do not forget inspections of solid organs like the spleen, liver, kidneys, and bladder to avoid missed diagnosis.
8. After patient condition is stabilized, relocate to a low-altitude area for corresponding treatment as soon as possible. If helicopter is used for evacuation, try to lower flight elevation whenever possible to prevent the occurrence of air embolism.

3.2 Treatment Methods for Plateau Blast Lung Injury

3.2.1 Life Support and Regular Symptomatic Treatment

Ensure unobstructed airway and normal circulation and provide blood transfusion, rehydration, and anti-shock treatment. For those with hemopneumothorax, perform closed thoracic drainage as soon as possible. Administer hemostatic drugs to reduce bleeding. Administer sufficient amount of antibiotics to prevent pulmonary infections and lower the chances of complications in the lungs.

3.2.2 Mechanical Ventilation

The key to treating blast lung injury is to maintain respiratory and circulatory functions, including keeping airway unobstructed, providing oxygen, and when necessary adopt measures such as tracheotomy and mechanical ventilator-assisted breathing. When blast lung injury deteriorates into a severe composite injury, the body would initiate strong stress responses, which would easily lead to stress response disorder, including stress ulcer of digestive tract, intestinal infection, hypermetabolism, etc. These could consequently lead to serious pathological harms to organs throughout the body, further complicating patient condition and heightening the difficulty of treatment. Therefore, during the course of treat-

ing blast lung injury coupled with severe burns, personalized treatment methods should be adopted in accordance with the severity of patient condition. After diagnosis is confirmed, perform tracheotomy right away, establish artificial air circulation and keep airway unobstructed. When airway is blocked by substance like phlegm or blood clot, carry out fibrobronchoscopy immediately, clear out obstruction, and rinse the airway clean. Perform electric coagulation for spots of bleeding in the airway. Take active measures to control infection and prevent the occurrence of pulmonary infection. If breathing difficulty does not improve and hypoxemia continues, apply mechanical ventilator-assisted breathing using the high frequency ventilation or positive end pressure ventilation mode, with the goal of reaching $PaO_2 > 80$ mmHg and $SaO_2 > 90\%$. It is suggested to provide ultrasonic atomization inhalation to humidify the airway, promote discharge of phlegm and other liquids, remove stimuli caused by foreign substances, and lessen the effects of various types of inflammatory medium. The utilization of mechanical ventilator should abide by the principle of "early usage, early conclusion, customized application." When the patient's spontaneous ventilation ability has returned, he or she can cough powerfully, and arterial blood gas analysis monitoring shows normal and stable status, consider removing mechanical ventilator as soon as possible to prevent the person from reliance on the breathing machine.

3.2.3 Extracorporeal Membrane Oxygenation (ECMO)

For normal impulse injury to the lungs, mechanical ventilator-assisted breathing is the primary method to help breathing and ensure oxygen supply to the body, including treatment using techniques such as high frequency oscillatory ventilation (HFOV). However, studies in recent years have revealed that although lung-protective ventilation and other treatment measures have been applied, death rate of victims with ARDS complications still exceeds 40%. In addition, lung-protective ventilation has its irreparable shortcomings, such as rise in CO_2 and drop in PH, and the inability to ensure sufficient oxygenation. In particular, regular treatment methods seem inadequate to address refractory hypoxemia in cases of severe pulmonary hemorrhage or even when mechanical ventilator-assisted breathing is adopted. Fatality rate is still rather high when one purely depends on conventional technologies, in particular the onset of refractory hypoxemia renders regular mechanical ventilator-assisted breathing treatment almost fruitless.

In recent years, foreign scholars such as Chairperson Stein of the Israeli Trauma Association and Mackenzie of the Queen Elizabeth University and Royal Hospital of the UK proposed the utilization of the latest technologies such as extracorporeal membrane oxygenation (ECMO) to treat victims of severe blast lung injury, and the results have been encouraging. Said technique can ensure oxygenation while allowing lung tissues to sufficiently "rest," thereby buying time for the cardiac and respiratory functions to recover and minimizing damage to the lungs arising from mechanical ventilation. At present, the new technique of ECMO is primarily used for treating acute reversible heart failure, respiratory failure, or cardiopulmonary failure and is praised for its features such as biocompatibility, little damage caused to blood, and prolonged application duration (average of 4–8 days). The underlying mechanism is the use of bio membrane to provide ventilation in lieu of the human lungs. By draining the venous blood of the patient, the artificial lungs can remove CO_2 and oxygenate blood, then a specially powered pump returns the blood inside the body through vein or artery, while the lungs "rest" during this process. ECMO also provides the necessary hemodynamics support, so that the heart too can "rest" and increase the chances of lungs and/or heart recovery from reversible pathogenic changes. At present, the new technique of ECMO is primarily used for treating acute reversible heart failure, respiratory failure, or cardiopulmonary failure.

Research studies already prove that the adoption of ECMO can provide sufficient oxygenation while imposing no restrictions on ventilation volume of the lungs. Therefore, it is also a great tool for dealing with severe ARDS. ECMO can ensure oxygenation while allowing lung tissues to sufficiently "rest," while minimizing damage to the lungs arising from mechanical ventilation as well as other complications associated with mechanical ventilation. Quite a number of clinical studies have proved that the application of ECMO in treating ARDS patients yields a significantly higher survival rate than subjects in conventional treatment groups using methods like mechanical ventilation. Therefore, if the new technique of ECMO can be applied to treat blast lung injury victims, especially those suffering from serious blast lung injuries, there exists the possibility of helping these seriously injured persons in surviving the most critical phase, winning precious moments essential to pulmonary function recovery, thereby lowering fatality rate and markedly raising blast injury survival rate.

3.2.4 Hyperbaric Oxygen Therapy

Outcomes of experiment by Shan Youan et al. indicate that hyperbaric oxygen therapy offers a certain degree of curative effect for severe blast lung injuries in dogs. Hyperbaric oxygen therapy was shown to reduce the fatality rate in the experimental group, with the control group posting a 40% death rate 24 h after injury, while the death rate of the hyperbaric oxygen therapy group was only 12.5%. Hyperbaric oxygen therapy clearly improved arterial blood gas and hemodynamics indicators in some dogs, raised PaO_2 levels, and significantly lowered the pulmonary index of the animals within the 24 h-period, offering a certain extent of cura-

tive effect for animals suffering from severe blast lung injuries.

3.2.5 Anisodamine and dexamethasone

Outcomes of experiment by Deng Zhilong et al. indicate that administration of hyperbaric oxygen therapy, anisodamine and dexamethasone, and administration of hyperbaric oxygen therapy and joint usage of anisodamine and dexamethasone, both showed relatively ideal curative effects on patients with plateau blast injury compared to subjects in group that did not receive any treatment. The treated subjects exhibited obvious improvement in arterial blood gas, and death rates dropped by 16.7–26.4%. Of the two treated groups, the one with administration of hyperbaric oxygen therapy and joint usage of anisodamine and dexamethasone produced even better treatment results, providing a certain amount of reference for dealing with plateau blast injuries. The mechanism behind the curative effect of hyperbaric oxygen therapy, anisodamine and dexamethasone on plateau blast injuries might be attributed to the following factors: (1) Hyperbaric oxygen can increase plasma physical oxygen capacity and raise partial pressure of oxygen, in turn ameliorating the lung's ventilation function and oxygen supply to tissues. At the same time, hyperbaric oxygen therapy can address air embolism arising from severe lung injury and lessen the danger when air embolism does occur. (2) Anisodamine can allay spasms in blood vessels and small airways, contributing to micro circulation and ventilation functions. (3) Dexamethasone has anti-inflammatory, anti-allergy, anti-free radical properties and can stabilize lysosomes, thereby augmenting the body's stress functions. Research revealed that hyperbaric oxygen therapy, and the use of hyperbaric oxygen therapy in conjunction with anisodamine and dexamethasone, can clearly reduce pulmonary hemorrhage and pulmonary edema compared with the control group. This research result provides beneficial morphological basis for improving arterial blood gas and reducing fatality rate.

3.3 Treatment of Plateau Blast Injury to Abdomen

The principles of treating victims of blast injury to the abdomen are identical to treating regular abdominal organ injuries, with top priority being dealing with the most life-threatening problems, such as ensuring airway stays unobstructed and stopping bleeding. Then swiftly carry out a full-body check to determine whether or not abdominal organ injuries exist, or if there are composite injuries involving other body parts. Patients should lie down and rest, refrain from eating or drinking, and reduce pressure on stomach and intestines. Steps need to be taken to minimize stress on the gastrointestinal tract and carefully observe the color, form, and property of drainage fluid. Insert catheter, record urine volume, observe urine color, and conduct urine analysis when necessary. For patients that might be suffering from visceral injury, draw blood and conduct cross-matching test right away. For patients that have lost a lot of blood or exhibiting hemorrhagic shock symptoms, perform blood transfusion and rehydration treatment in a timely manner, and take other active steps to ameliorate shocks. For those in critical conditions, exploratory laparotomy should be carried out while administering anti-shock treatments. Broad-spectrum antibiotics should be applied in the early stage to actively defend against infection. Active measures should be taken to prevent bleeding or stress ulcer of digestive tract, and sufficient amount of parenteral nutrition should be provided. For patients confirmed with or suspected of visceral injury, undertake exploratory laparotomy, and restore or remove injured organs, then clear out blood or effusion accumulated in the abdominal cavity or substances in the intestines, so as to prevent severe complications such as abdominal cavity infection.

3.4 Initial Surgical Treatment for Extremity Blast Injuries on Plateau in Peacetime

The higher the elevation a particular part of the Earth's surface, the more solar radiation said part receives. Given thin air, less water vapor, and dust, longer daytime, plateaus receive more radiation than plains. Lhasa, for instance, receives 1.68 times more radiation than plains at the same latitude. Ultraviolet ray constitutes a part of radiation from the sun, and the higher the altitude, the stronger the UV ray. For every hundred meter increased in elevation, UV ray intensity increases by 3–4%. UV radiation at Lhasa is 1.7 times more powerful than that measured at the eastern plain in China (Suzhou), and this increase no doubt intensifies the effects of sunburn. The powerful sunshine and UV radiation on plateaus are important in suppressing the survival and reproduction of bacteria in the environment. Therefore, infection of wounds in a blast injury suffered at such high-elevation areas is less severe than those at plains. At the same time, since blast injuries during peacetime usually occur at a single instance, medical care institutions have enough time and human resources to perform relatively thorough debridement, and there are sufficient anti-bacterial medicines sensitive enough to prevent infections. Therefore, if conditions permit, treatment of plateau blast injuries during peacetime should perform primary debridement and suture and primary internal fixation of fractures (Figs. 29.6 and 29.7). Our experimental research and clinical practice both prove this point.

Lei Mingquan et al. used local Tibetan dogs to carry out bullet wound experimental research and performed primary

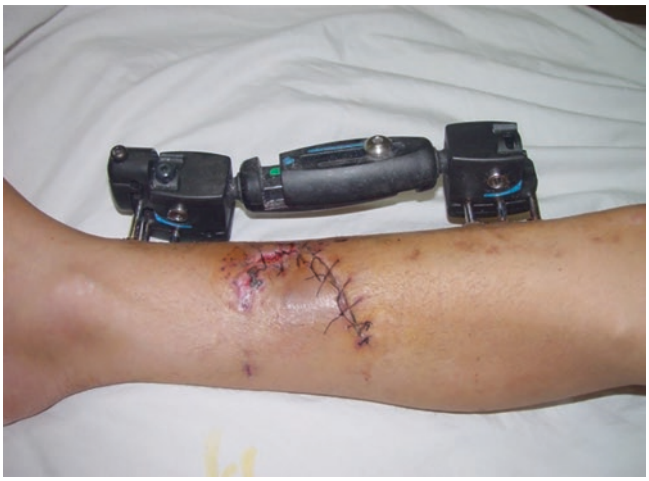

Fig. 6 Primary debridement and suture and primary internal fixation of fractures in high-altitude explosion blast injuries (image by Yin Zuoming)

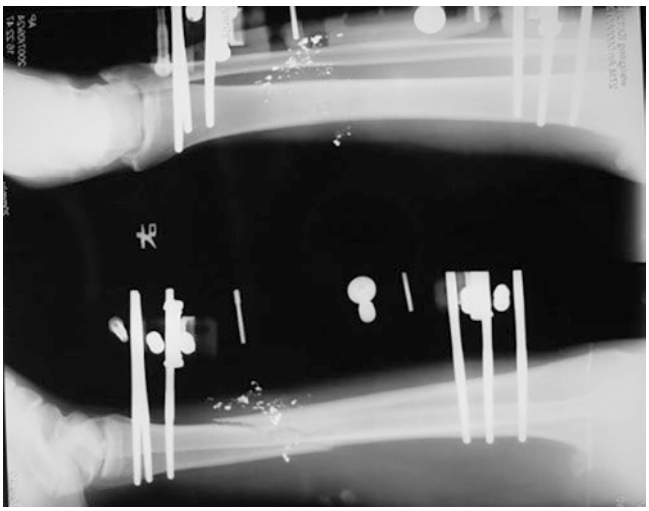

Fig. 7 X-ray scan primary debridement and suture and primary internal fixation of fractures in high-altitude explosion blast injuries (image by Yin Zuoming)

debridement and suture, and primary internal fixation of fractures 12, 24, and 36 h after injury. At the same time, bacterial counts were measured, wound infection conditions were observed, and antibiotics were administered for 5 days as per normal protocols. Primary wound healing was achieved for the 14 dogs, stitches were removed after half a month, and X-ray inspection 2 months later reviewed a moderate amount of bone callus growth, while signs of osteomyelitis were not discovered in any subject. This indicates that if infection was not serious within 12–36 h, anti-bacterial procedures were strictly adopted and wounds were thoroughly cleaned, primary internal fixation of gunshot fractures to the limbs may be conducted. Lei Mingquan et al. undertook primary debridement and suture 24 h after inflicting regular firearm injuries on plateau, and they recorded a grade I healing rate of 97.2%.

Since 1991, Yin Zuoming has performed primary internal fixation of fractures for patients of regular gunshot bone fractures on plateau, and all cases reached clinical healing status. In 2002, Yin Zuoming used small pigs to conduct experiments, proving that treatment of plateau firearm injuries during peacetime yields best results when primary debridement and suture and primary internal fixation of fractures are performed within 6–36 h after injury, with wounds showing no obvious redness, swelling or infection, and no shock, multi-organ failure, or other symptoms in the patient. It was noted that during the process it is necessary to thoroughly clean the wound tract, and since the contusion area and concussion zone of firearm injuries on plateau are wider than those on plains, the wound area to be cleaned would also be bigger. It is also suggested to remove inactivated necrotic tissue while minimize harms to the periosteum, retain residual bone tissue, and perform simple internal fixation. In addition, drainage has to be unobstructed and sustained for an adequate period, and full-body support should be provided after the procedure, along with administration of sufficient amount of effective antibiotics. In 2007, Yin Zuoming performed primary debridement and suture to 47 wounds on 11 victims of blast injuries during peacetime, and no infection occurred. However, three wounds ripped because they were not adequately drained. The wounds had grade I healing rate of 91.5%, and the patients were hospitalized an average of 18.6 days. It is worth mentioning that the aforesaid research was completed on plateau during peacetime, and the derived principles are only applicable to plateau blast injuries during peacetime and are currently not yet suitable for use during wartime.

4 Features of Plateau Burn-Blast Combined Injury

Burn-blast combined injury refers to a situation in which the victim is simultaneously or successively afflicted with burns from heat and blast injury from shock wave. If burns are the main injury, then the term would be burn-blast combined injury, and however, if blast injury is the main problem, the term would be blast-burn injury. However, usually the term burn-blast combined injury is used nowadays for both. When the human body is afflicted with burn-blast combined injury, there would be easily observed burn wounds on the surface, but also blast injury to the internal organs that are difficult to see. The victim would not only show obvious burn shock, wound infection and repair, but also serious visceral injury and functional disorder that are hard to see. The two problems promote each other and together aggravate patient condition, bringing about many dilemmas and challenges for

medical personnel in both diagnosis and treatment. However, the lungs are still the main target organs injured by shock wave. Injury to the respiratory tract is especially serious for victims of large areas of burns and inhalation injury, mainly manifested as airway and lung tissues edema, necrotic shedding of airway mucosa, and gas exchange impairment. Therefore, when blast injury is compounded by large areas of burns and inhalation injury, lung injury would be even more serious and the occurrence of ARDS is almost impossible to prevent. When ARDS occur in victims with blast injury compounded by large areas of burns and inhalation injury, problematic conditions in the lungs would develop quickly. What happen first are changes in one lung segment, and diffused and substantial changes in both lungs within 24 h, along with significant amount of hemorrhagic effusion in the airway. The treatment of this kind of patient is very challenging and the fatality rate is rather high.

When plateau blast injury is accompanied by other injuries (burns, shrapnel wound, etc.), injury conditions would be much more severe. Of which, burn-blast combined injury is most obvious, and the reason might be the full-body response to burns, and the bigger impact imparted on the respiratory system, which is the main target in blast injury. Explosions that can result in burn-blast composite injuries are very destructive, have long time of effect, injure many people, and create complicated injuries that are difficult to treat, among other characteristics. Such explosions could occur during times of peace or wartime. This is why burn-blast combined injury is difficult to treat, with the key problems being difficulty in diagnosis and in capturing the best timing for treatment.

4.1 Basic Features of Plateau Burn-Blast Combined Injury

When burns and blast injury occurring on plateau combine to form burn-blast combined injury, the effects of the two will add to each other's severity, causing the body to generate even stronger stress responses, possibly leading to stress response disorder, stress ulcer of digestive tract, intestinal infection, hypermetabolism, etc. These could consequently lead to serious pathological harms to organs throughout the body, further complicating patient condition and heightening the difficulty of treatment.

The main features of plateau burn-blast combined injury inflicted by explosion include:

1. Unexpectedness of explosion that caused plateau burn-blast composite injuries, making it more difficult to organize and direct rescue efforts.
2. Rate of occurrence of burn-blast combined injury is contingent on a victim's distance from the center of explosion, with the closer the distance from the center of explosion, the higher the chance of burn-blast combined injury.
3. Burn-blast combined injury is clearly characterized by the mutually worsening effects of the injuries, and the occurrence sequence and injury severity of burns and blast injury affect the development of patient conditions.
4. Numerous injuring factors and complicated victim conditions. Shocks are very common, usually occur relatively early at rather severe levels and could worsen quickly. The pathological and physiological disorders of victims of burn-blast combined injury are often afflicted with multiple, severe, and complicated injuries at different parts of the body. The scope of injury is not only broad but affects numerous body parts and various organs, while local and full-body responses are strong and prolonged.
5. Highly complicated injury mechanism: It is projected that injury mechanism depends on the direct effects of shock wave and heat, and the secondary injuries that they cause.
6. External injuries cover up internal injuries, leading to possible missed or erroneous diagnosis. When a blast injury is coupled with burns or other injuries, injuries on the body surface are obvious, while visceral injuries might be overlooked. However, severity of visceral injury is usually the crux that determines how patient conditions develop. Lack of knowledge in this area could easily lead to missed or erroneous diagnosis or missing the best opportunity for medical rescue.
7. Lung injuries are frequently the bottleneck and focal point in treatment of burn-blast combined injury. The lungs are some of the most sensitive organs to injuries caused by shock wave and also the main target organs in burns of respiratory tract. When pulmonary functions are seriously endangered, the lungs' tolerance of fluids also declines. Therefore, lung injuries should be the bottleneck and focal point in treatment of burn-blast combined injury.
8. Infection happened early and in grave severity, with simultaneous existence of serious wound/local infection of the wound and systemic infection.
9. Rapid clinical injury conditions development and extreme difficulty in treatment manifestations include severe injury, numerous complications, and relatively high fatality rate from sickness and/or injury.
10. Simultaneous existence of internal injuries and external wounds that affect and overlap each other, further complicating and aggravating patient conditions.
11. Usually there exist damages to various organ functions: For instance, the cardiac, circulatory systems, the liver,

kidneys, nervous and immunity systems, and other damages and functional disorders.

12. The biggest problem in burn-blast combined injury caused by explosion on plateau is how to deal with treatment difficulties and dilemmas arising from the different injuring factors. The crux of the matter of treating burn-blast combined injury is addressing the dilemma of transfusion, as treatment of burns requires rapid transfusion, which might run contrary to cautious use of transfusion for plateau blast injuries.

4.2 Clinical Expressions of Burn-Blast Combined Injury

Patients of burn-blast combined injury exhibit the various clinical expressions of burns and blast injury at the same time, along with some other general signs and symptoms. These manifestations differ based on the degree of burns and severity of tissues and organs damaged by shock wave.

4.2.1 Clinical Expression of Burns

Different degrees of burns have different clinical expressions. When there are burns of the respiratory tract, there are often severe burns around the mouth and nose, burnt nasal hair, swollen lips, red and swollen oral cavity and oropharynx area, white blisters or mucous membranes, irritable cough, dusts of burnt matters in phlegm, scratchy voice, swallowing difficulty or pain, breathing difficulty and/or wheezing, among others. Subsequently, conditions would rapidly deteriorate into tracheobronchitis, "gong-like" sounds and pain during irritable coughs. Burns on the body surface share similar clinical expressions with burns from regular heat source. “Rule of Three Degrees and Four Levels” should be used to estimate burn area based on Chinese Rule of Nines Method. The severity of burns should be further evaluated in accordance with burn area and depth. Burns severity may be categorized as: minor burns, moderate burns, severe burns, and extremely severe burns.

4.2.2 Clinical Manifestation of Blast Injuries

(1) Lung injuries from the same intensity of impulse would be more severe if burns also exist, and clinical manifestations would also be more serious. Patients of lung injuries might exhibit sustained lack of breathing for 30–120 s after injury, often times accompanied by bradycardia and hypotension. Minor lung injury might only result in short-term chest pain, chest stuffiness, or sense of suffocation. For those more seriously injured, expressions like cough, hemoptysis, and bloody phlegm might be seen, and a small number might complain about breathing difficulty, and diffused, moist rales might be audible during auscultation. For those with severe lung injuries, manifestations include breathing difficulty, cyanosis, large volume of bloody and frothy fluid flowing out from mouth and nose, dull resonance in local areas produced from clinical percussion, weak breathing sounds heard during auscultation, along with widespread moist rales. For patients suffering from pneumothorax or hemothorax, corresponding signs and symptoms might appear. (2) For those with blast injury to the abdomen, the most common clinical expressions are abdominal pain, nausea, vomiting, signs of peritoneal irritation and shock, among others. If internal organ suffers only minor blunt trauma, abdominal pain would usually subside after 3–4 days. If there is ruptured organ, abdominal pain might ameliorate after a short while but would frequently return, accompanied by signs of peritoneal irritation such as tenderness, rebound pain, abdominal muscle rigidity. Hematuria may occur if there is kidney or bladder injury. Rectal bleeding may occur in patients with colon or rectum injury. Perforation of the stomach or intestines could result in symptoms like subphrenic free air, pneumoperitoneum, or disappearance of distinguishing dull sound of liver, and borborygmus might also disappear. (3) When an auditory apparatus blast injury occurs, main signs and symptoms include deafness, tinnitus, vertigo, ear pain, headache, external ear discharge, etc. In a minority of cases, patients might exhibit nausea, vomiting, or vestibular dysfunction, among other signs and symptoms. Inspection might discover eardrum rupture, fracture of auditory ossicles, and temporary or permanent auditory dysfunction. (4) The blast injury to the heart are chiefly manifested as sharp pain in the anterior area of the heart, chest stuffiness, sense of suffocation, cold sweat, and other symptoms of coronary vascular insufficiency, while acute left heart failure may occur in severe cases. Symptoms and signs of acute myocardial infarction may appear in patients with coronary air embolism. In cases where secondary injuries occurred due to displacement, collision or other effects caused by dynamic pressure, the most common problem is internal pericardium hemorrhage. If bleeding is profuse, symptoms and signs of cardiac tamponade may be observed. (5) The main symptoms of a blast-induced traumatic brain injury is the loss of consciousness and could also be simultaneously accompanied by various kinds of mental symptoms and signs such as indifference, anxiety, fear, irritation, sleep loss, dizziness, reduced memory capacity, etc. Serious cases may show ataxia, speech disorder, numbness of the limbs, convulsion, and other symptoms and signs of cerebrovascular air embolism. When there is injury to the brain parenchymal, increased cranial pressure or localized symptoms may appear.

4.2.3 Features of Clinical Expressions of Burn-Blast Combined Injury

While clinical manifestations of burns are obvious, at the same time there would often be auditory capacity loss or auditory apparatus injury, as well as clear clinical expres-

sions of pulmonary injuries, or even respiratory function disorder. It is common to see shocks unlike those associated with burns, localized pains, functional disorders, and clinical manifestations related to functional damage and functional disorders of the respiratory, cardiac, circulatory, nervous systems, and other organs.

4.3 Comprehensive Judgment of Burn-Blast Combined Injury Condition

When both burns and blast injury are minor, or if only one is at moderate level, usually patient conditions would be severe. If both burns and blast injury are moderate or more serious, patient's overall condition should be one level more serious than the more severe of the two injury types. If there are obvious shocks, multiple injuries, or other composite injuries, add another level of severity to the most severe rating of the overall patient condition.

4.4 Treatment Principles for Burn-Blast Combined Injury

Plateau environment could lower the body's tolerance for blast injury, worsen patient condition, and raise fatality rate. Third-degree burns on 40% of the body were inflicted to dogs under simulated plateau environment with elevation of 4000 m (after 24-h acclimatization in low pressure chamber), and the Parkland formula was used to calculate amount of replacement fluid required, then fluid infusion began 1 h after injury. Outcomes indicate that cardiac output (CO) and left ventricle working index (LCWI) of dogs in both plain group and plateau group exhibited obvious drops after injury, with decrease in the plateau group being bigger. Therefore, it is necessary to pay attention to the dilemma between the need to increase fluid volume and the decline in the body's tolerance when performing fluid infusion on plateau. It is suggested to carry out fluid infusion under strict and careful monitoring, pay attention to improvements in cardiac functions and oxygenation in tissues, and refrain from simply limiting fluid volume at the cost of affecting recovery.

The key to treatment of burn-blast combined injury in the early stage is timely and accurate diagnosis and appropriately handling the treatment dilemmas between the injury forms and injury types of composite injuries and multiple injuries. It is also important to carefully observe pulmonary hemorrhage and edema, prioritize the use of capsules when administering anti-shock fluid infusion, strengthen airway care and support treatment for the respiratory tract, heighten defend against pulmonary cerebral and edema, and endeavor to prevent and cure visceral complications such as ARDS, air embolism, and disseminated intravascular coagulation (DIC). Other treatment principles are, respectively, similar to those of pure blast injuries and burns.

5 Features of Plateau Projectile-Blast Composite Injury

Conventional weapons of modern day (i.e. missile, cannon shell, mine, grenade) could generate a substantial amount of dangerous fragments and shock wave upon detonation. Hence, blast injury and projectile injury are the main killing and injuring factors of explosive weaponry. In a projectile-blast composite injury, injury from projectile worsens the severity of blast injury and is usually more dire than plain blast injury. On plateau at an altitude of 2500 m or higher, due to the lack of oxygen and low-atmospheric pressure, the body's physiological compensation increases the stress on tissues and organs while lowering the body's tolerance for external harms, thereby raising damage caused to the body when injured. Due to the low-atmospheric pressure and air resistance on plateaus, projectiles fly faster and create bigger impact upon collision with tissues. Therefore, tissues have to absorb more energy from injuring projectiles and in doing so sustain more damage.

5.1 Injuring Features of Plateau Projectile-Blast Injury

Research by Lai Xinan et al. proves that a projectile's terminal velocity on plateau is faster than that at plain, and comparing subjects in the plateau injury group with subjects in the plain injury group, entry and exit wound areas, wound tract volume, wound tract length ratio, excised necrotic tissue and ratio of broken muscle tissue were all larger in the former. Extents of wound tract muscle fiber ruptures and changes of subjects in the plateau spherical projectile injury group and plateau triangular projectile injury group were both more severe than those in the plain injury group, with the falling activeness in muscle tissue succinate dehydrogenase and ATPase being only 26.86% and 55.77%, respectively, of the plain group. Cavitation in soap shot at plateau is larger than that at plain. Degree of projectile injury on plateau is more serious than that on the plain, this is because, among other reasons, plateaus have less air density, therefore, projectiles have lower air resistance and could fly faster than on plains, creating bigger temporary cavity upon impact with tissues. The aforementioned study also revealed that a projectile's terminal velocity on plateau is faster than that at plain, with a 1.03 g steel ball's velocity on plateau being 112.79% of that on plain, and triangular projectile's velocity on plateau being 115.25% of that on plain. The difference in projectile veloc-

ity between plateau and plain is attributed to difference in atmospheric pressure. Under normal circumstances, for every 100 m rise in elevation, atmospheric pressure falls by 7.45 mmHg, and the atmospheric pressure on a plateau at an altitude of 3658 m is only 64.35% that of sea level. At the same time, air density also drops by 37.35%, as it is closely related to atmospheric pressure. The rate at which a projectile slows down in flight hinges on drag. Since the lower air density on plateau gives less resistance, a projectile decelerates at a slower pace, which is why projectiles on plateau fly at higher velocity than those on plain. The drag area of triangular projectile is larger than that of steel ball, resulting in more obvious velocity changes. Not only do fragments from explosions on plateau cause a larger area of damage, but also more severe injuries to tissues compared with those on plain. A projectile on plateau flies faster than that on plain, and when it hits tissue the rate of deceleration is bigger, releasing more energy through the wound tract. On plains, a bullet's collision velocity against tissue is (660.54 ± 14.22) m/s, while a bullet's collision velocity against tissue on plateau is (701.43 ± 2.98) m/s.

5.2 Pathological and Physiological Features of Plateau Projectile-Blast Injury

Under the effects of a low-oxygen, low-pressure, and low-temperature environment, a series of pathological and physiological changes would take place in the body. The storage functions of key organs decline significantly, and upon this basis, projectile injury would more easily cause damage to internal stability, leading to physiological dysfunction in the various systems, more post-injury complications, and higher rate of death from injury.

5.2.1 Changes in Respiratory System After Plateau Projectile-Blast Injury

The respiratory rhythm and ventilation volume of the human body is regulated through the respiratory center in the nervous system. The remote effect, stress response, pain stimuli, blood loss, and other issues arising from plateau firearm injuries may cause compensatory breathing increase. Research by Yin Zuoming indicates that for the majority of small pigs tested, after being afflicted with plateau projectile wound, they exhibited apnea for several tens of seconds, then tachypnea, eventually stabilizing and gradually turning to deep breaths with respiratory rate obviously slower than usual. This kind of respiratory change could lead to arterial blood gas changes and acid–base disturbance in the body, further intensifying hypoxia and disturbance of internal environment of the body. The minor respiratory alkalosis seen early on in animals after firearm injuries on plain during peacetime is mainly attributed from ventilation. Prior to firearm injuries on plateau during peacetime, the compensatory reductions in $PaCO_2$ and HCO_3^- result from acclimatization to the plateau environment. Thirty minutes to two hours after injury, pH edged higher by a small amount, while both $PaCO_2$ and HCO_3^- dipped slightly compared to pre-injury levels, and respiratory alkalosis was also observed, yet recovery was obvious after 24 h. At the 24-h mark, pH dropped to below 7.35, which is mostly manifested as metabolic acidosis coupled with respiratory alkalosis. This development may possibly be attributed to a state of hyperventilation early after injury, manifested as respiratory alkalosis 24 h later when hyperventilation ameliorated and the body's acidosis worsened, the result was metabolic acidosis coupled with alkalosis. In wartime on plateau, due to the body's rapid entry to the plateau, hypoxia, and wartime factors, obvious metabolic acidosis and respiratory alkalosis in animals were existing obviously before injury. Due to decreased respiratory efficiency caused by the low-oxygen and low-atmospheric pressure of plateau environment, respiratory rate early on after injury is higher than that on plain, and hyperventilation is rather serious and sustains longer, which are manifested as respiratory alkalosis and decompensation. The acid–base disturbance also coexists with obvious hypoxemia in the animal, which is even more pronounced during wartime on plateau.

Research indicates that the respective partial pressure of oxygen during peacetime and wartime on plateau is only 63–73% of those on plain. This difference is primarily attributed to plateau factors. PaO_2 readings 30 min to 6 h after firearm injuries on both plain and plateau during peacetime declined then gradually restored, demonstrating that the body's compensation capacity for firearm injuries during peacetime is relatively sound. PaO_2 readings before firearm injuries on plateau during wartime were significantly lower than that on plateau during peacetime, and the figures continued to fall after injury 24 h after injury, the levels were still significantly lower than the plateau peacetime group and before injury, and hypoxemia was also observed. This difference may possibly be attributed to the following reasons: (1) Excessively fast respiratory rate early on after injury resulted in an increase in ineffective cavity in the airway; (2) accelerated heart rate after injury resulted in accelerated blood flow in alveolar capillaries, leading to ventilation and perfusion ratio imbalance, with some blood flow not fully oxygenated by functional shunt, in turn causing a rise in respiratory rate as a feedback; (3) after firearm injuries, the lungs were injured by a large quantity of inflammatory substance, resulting in alveolar gas diffusion dysfunction; (4) the low-oxygen and low-atmospheric pressure of plateau environment resulted in lower respiratory efficiency; and (5) lung injuries arising from remote effect could also be one of the reasons.

5.2.2 Rise in Endothelin After Injury from Hi-Speed Projectile

Study by Yan Jiachuan et al. discovered that extremity injury on one side of the body caused by hi-speed projectile aggravates moderate blast injury, with the aggravation primarily seen in the lungs. After blast injury, or blast injury coupled with projectile injury, endothelin (ET) content in plasma and lung tissues would rise, taking effect through endothelin receptors. Pulmonary vasoconstriction results in elevated pulmonary artery pressure while simultaneously worsening any existing pulmonary hemorrhage and pulmonary edema. The extent of pulmonary vasoconstriction is contingent on the volume of endothelin, and since ET contents in subjects in composite injury group were markedly higher than other groups, therefore, their pulmonary artery pressures and lung injuries were also more severe than those in other groups. ET contents in plasma and lung tissues in subjects of pure projectile injury group did not change much. Therefore, changes in their pulmonary artery pressure and lung injury were not obvious. Pulmonary capillary endothelial cells were damaged from blast injury, leading to increased seepage, reduction in the capacity of lung tissues in ridding plasma ET and elevated ET release. At the same time, the powerful pressure wave generated by the injuring hi-speed projectile imparts varying degrees of mechanical force on vascular endothelial cells in different parts of the body, and said pressure wave would further cause damage to the pulmonary vascular endothelial cells that are already injured by blast wave. This in turn leads to additional ET release, a surge in plasma ET concentration and heightened pulmonary artery pressure. The rise in ET contents in plasma and lung tissues may possibly be linked to the following reasons: (1) Severe edema and hemorrhage in lung tissues; (2) effects of cytokines; (3) stimuli from injuring hi-speed projectile's pressure wave and hemodynamic disorder; and (4) reduction in the capacity of lung tissues in clearing out ET. Outcomes from said research indicate that after injury plasma ET concentration soared, vasoconstriction intensified, and pulmonary artery pressure elevated, exacerbating pulmonary edema and hemorrhage and worsening pulmonary circulation obstruction, while pulmonary edema and hemorrhage could increase ET synthesis and release. This vicious cycle intensifies pulmonary edema and hemorrhage severity and worsens lung injuries. The mechanism behind how high-speed projectiles worsen blast injury is associated with the increased release of endothelin after injury.

5.2.3 Impacts of Plateau Injury from Hi-Speed Projectile on Hemodynamics

Outcomes from study by Liu Jiancang et al. indicate that right ventricular systolic pressure (RVSP) in subjects in the plateau injury group surged significantly above those in plain injury group, demonstrating that other than cardiac function damage, the aggravation of lung injuries and elevation in pulmonary artery pressure caused a rise in RVSP, leading to heart failure, reduction in cardiac ejection function, and significant drop in ejection fraction that is much more pronounced than that in the plain injury group. At the same time, obstruction of venous return occurred, blood flow was clogged up in the venous system, exacerbating the ischemia and hypoxia in vital organs and tissues. Consequently, CVP readings of subjects in plateau injury group 6 h after injury were significantly higher than subjects in the plain injury group. In this series of changes, the differences in subjects of the projectile-blast composite injury group were most marked. The mean arterial pressure (MAP) readings in the various plateau injury groups all exhibited a rising trend, which may possibly be attributed to the increase of blood flow through initiating contraction of peripheral arterioles, as a way for the body to compensate for the shortage of oxygen in plateau regions. The outcomes above demonstrate that plateau projectile injury could substantially worsen blast injury. Due to the lack of oxygen and low-atmospheric pressure on plateau, blast injury, projectile injury and composite injury are clearly worse than those that occurred on plains. Degree of hemodynamics injury also intensifies, with the difference being most prominent in composite injuries.

5.2.4 Hypercoagulable State Instigated by Plateau Injury from Hi-Speed Projectile

Prostacyclin (PGI_2) imparts a profound effect on vessel dilation and platelet depolymerization, and it is primarily synthesized in vascular endothelial cells. Thromboxane A_2 (TXA_2) is released by platelets, which is not only a potent vasoconstrictor, but also increases platelet aggregation. With ischemia and hypoxia, on the one hand vascular endothelial cells are damaged and PGI_2 generation declines, while, on the other hand, platelets release more TXA_2, leading to PGI_2/TXA_2 imbalance. The outcome is marked rise in vasoconstriction and platelet aggregation, which release more TXA_2, causing formation of thrombus and vascular thrombosis, consequently damaging affected tissues. Research by Yin Zuoming et al. demonstrates that in high-altitude wartime environments, there were increases in plasma TXB_2 before injury, while $PGF_{1\alpha}$ and $PGF_{1\alpha}$/TXB_2 fell, indicating that wartime factors induced $PGF_{1\alpha}$/TXB_2 imbalance in the body prior to injury. Firearm injuries on plain during peacetime, firearm injuries on plateau during peacetime, and firearm injuries on plateau during wartime all showed significant climb in plasma TXB_2, and all peaked at 6 h after injury. This may possibly be associated with ischemia reperfusion damage caused to tissues and organs throughout the animal's body due to strong stress. Varying patterns of firearm injuries during peacetime on plateau and those on plain are basically identical, but the rises in the former were more obvious, demonstrating that the whole body

is in a hypercoagulable state after firearm injuries on plateau during peacetime. This is a state in which disseminated intravascular coagulation (DIC) could easily occur alongside other complications, which would affect perfusion in tissues throughout the body. The body-wide hypercoagulable state may possibly be attributed to the hike in inflammatory agents like TNF-α after injury, which causes damage to vascular endothelial cells. The rise in firearm injuries on plateau during wartime is higher than that during peacetime, with the figure sustaining at a high level even 7–10 days after injury, demonstrating that plateau and wartime factors together wreak havoc on the body in a relatively marked manner. The body remains in a hypercoagulable state for a prolonged period after injury, leading to greater risks of thrombus and DIC. This matter is of key importance when treating firearm injuries on plateau during wartime, namely appropriately dealing with the dilemma between hemostasis after injury, and prevention of thrombus and DIC. Research by Yu Xiyong et al. indicates that after large dosages of vitamin C ingested orally (1.0 g each time), 6-keto-PGF1α level in plasma rose dramatically, while TXB_2 level remained basically constant, hence, $PGF_{1\alpha}/TXB_2$ ratio climbed. Meanwhile, such changes are not seen when small dosages of vitamin C were ingested orally (placebo control group), demonstrating that vitamin C concentration inside the body must reach a certain level to produce the intended effect. Vitamin C is the most effective water-soluble antioxidant in plasma and acts as the first line of defense in the extracellular fluid antioxidant defense system by interrupting lipid peroxidation occurring in plasma. Vitamin C gets rid of free radicals and lipid peroxide that suppress prostacyclin (PGI_2), thereby increasing synthesis of PGI_2. At the same time, vitamin C also stimulates endothelial cells to synthesize PGI_2. Worth noting is that during the antioxidation process, vitamin C both clears away radicals and causes damage through radicals. In other words, a low concentration of vitamin C engenders the production of oxygen radicals, and only when concentration is high enough vitamin C would clear away radicals, which is why the effect of vitamin C hinges on its concentration. Therefore, treatment of firearm injuries on plateau needs to pay attention to strengthening vitamin C administration and other full-body support and treatment.

5.2.5 Impacts of Plateau Injury from Hi-Speed Projectile on Inflammatory Response

Research by Yin Zuoming et al. discovered obvious declines in expressions of inflammatory agents such as TNF-α, IL-1β, and NO in wound tracts of plateau peacetime injury group, and inflammatory response and tissue edema severity levels were clearly less dire than the plain group, and these also occurred later than subjects in the plain group. Wound tract tissue wet/dry ratio and histology results both prove this point. Expressions of anti-inflammatory agents IL-4 and IL-10 in local tissues of the wound tract were markedly lower than those in the plain peacetime group and were expressed at a later time. This also illustrates that local inflammatory response in wound tract after firearm injuries to the limbs on plateau is less severe than that of firearm injuries to the limbs on plain. Possible reasons for these differences between plateau group and plain group might be: (1) Lack of oxygen in plateau environment means less local production of oxygen radicals in wound tract, thereby inducing smaller inflammatory response. (2) The cold environment of plateau delays the onset of and lessens wound tract infection. Due to the reasons mentioned above, critical time of local immunity changes in wound tract of firearm injuries on plateau during peacetime was delayed to the second or third day, hence, debridement deadline could be delayed to the second day after injury. This is conducive to early-stage surgical treatment of firearm injuries wound tract on plateau and cold alpine regions during peacetime, but is unfavorable to the recovery and healing of wound tract on plateau. (3) The expressions of inflammatory agents such as TNF-α, IL-1β, and NO in wound tracts of plateau wartime injury group were obviously higher than plateau peacetime injury group but less than plain peacetime injury group, and time of appearance of anti-inflammatory agent IL-10 was substantially later than plateau peacetime injury group, at a concentration substantially higher than plateau peacetime injury group. This demonstrates that rapid entry into simulated combat environments characterized by features such as plateau, stress, tiredness, hunger and cold temperature, a series of pathological and physiological changes already took place in animals in the plateau wartime injury group before injury. Upon this basis, projectile injury would cause a sort of accumulated trauma, and wound tract inflammatory response after injury was more severe than that of plateau peacetime injury group but less than that of plain peacetime injury group. Meanwhile, critical time of local immunity changes occurs earlier than plateau peacetime injury group, bringing forward to 24 h after injury. Under this type of special environment, understanding the unique pattern of wound tract inflammatory response produced by firearm injuries during wartime is of great meanings to the design of early-stage surgical treatment options for local wound tract of firearm injuries during wartime in said special environment.

5.2.6 Impacts of Plateau Injury from Hi-Speed Projectile on Metabolism

Research by Yin Zuoming indicates that oxygen deficit on plateau itself could affect the body's material metabolism, increasing the decomposition of sugar, protein, and fat, while protein synthesis weakens and creates water, sodium, and potassium metabolism disorders, among other matters. Upon this basis, projectile injury on plateau leads to a bigger stress response than on plain, leading to a series of neuroendocrine

reactions in the hypothalamic-pituitary-adrenal axis and sympathetic nervous system, causing raised secretions of cortical hormone, catecholamine, glucagon, TNF, IL-1, IL-6, and lipid mediators. This in turn instigates increases in energy consumption, metabolic rate, protein consumption, and fat decomposition, with sustained period longer on plateaus than on plains. After plateau projectile injury, glycometabolism is mainly manifested as high decomposition and high glycolysis, and lipometabolism is mainly manifested as high decomposition and high consumption, while protein metabolism is mainly manifested as high decomposition and low synthesis. Plateau projectile injury during wartime is often accompanied by serious hypocalcemia and hypomagnesemia in the early stage. Minor hyponatremia in the later stage, while gradual falls in phosphorus, potassium, and chlorine would start in 12 h after injury. Therefore, when treating victims of plateau projectile injury during wartime, pay attention to supplementations of calcium, magnesium, sodium, and other electrolytes in a prompt manner. At the same time, recovery from plateau trauma during wartime necessitates a large amount of nutrients, and the proper metabolism and nutritional support and adjustment can protect organ functions, augment immunity, protect against infection, and promote healing of wound tract.

5.3 Features of Plateau Projectile-Blast Combined Injury

Outcomes from research by Yang Zhihuan et al. show that hi-speed projectile injury to the limbs aggravates the severity of moderate and severe blast injury, with aggravation mostly occurring in the lungs, limited to the region where the primary blast injury took place. In general, patient condition would be one level more dire than the original injury. Mechanism behind how hi-speed projectile intensifies blast lung injury remains unclear, but it may possibly be linked to the forceful disturbance of blood flow instigated by pressure wave from the impact of hi-speed projectile upon hitting the body, and this blood flow disturbance injures the heart and lungs, which are away from the wound tract. Some literature have reported that when animals were injured by hi-speed projectiles, pressure waves were recorded in aorta, aortic arch, and brain tissues, with readings as high as 300 kPa observed in aortic arch. Evidently, this kind of powerful pressure wave can further damage lung tissues that have already been injured, thus worsening existing injury. In addition, after being severely injured by a hi-speed projectile, the body would secrete excessive amounts of inflammatory agents and cytokines such as thrombin, endothelin, IL-1, IL-6, and TNF-α. These could further cause damage to pulmonary vascular endothelial cells and alveolar epithelial cells, to some extent contributing to secondary pulmonary injuries. Another study proved that pulmonary index rose significantly in animals afflicted with projectile-blast composite injury, revealing the existence of obvious pulmonary edema. This is also a solid evidence. Therefore, secondary injury could be another major reason why hi-speed projectile worsens blast lung injury.

Hi-speed projectile does not seem to exacerbate minor blast lung injury, which could be attributed to very limited area of lung injury, relatively sound integrity of lung tissues and structure, and rather small influence on the lungs imparted by the pressure wave from the hi-speed projectile. Similarly, low-speed projectile does not appear to significantly worsen moderate blast lung injury. The hypothesis is that the relatively weak pressure wave generated by the projectile is associated with the relatively mild secondary lung injuries, but specific mechanism requires further study. Existing research outcomes reveals that when treating blast injury victims, not only it is necessary to treat projectile injuries on the surface, but it is also required to consider possible existence of visceral blast injury, and aggravation of blast lung injury caused by hi-speed projectile. In case of performing fluid resuscitation, if there is relatively severe blast injury, fluid infusion volume and rate should be adjusted to appropriate degrees. Meanwhile, hemodynamics monitoring should be strengthened to prevent injury condition aggravation and other dire consequences due to inappropriate fluid infusion. All in all, plateau blast injuries could be aggravated when coupled with burns or projectile injuries, giving rise to more severe conditions and higher death rate. Aggravation mostly targets the lungs, while the same aggravation does not seem to appear in blast injury to the gastrointestinal tract when existing as burns-blast composite injury or projectile-blast composite injury. Therefore, when treating victims of this kind of injury, it is necessary to pay close attention, make correct judgment about injury, and undertake the corresponding treatments and measures.

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Underwater Blast Injury

Shengxiong Liu and Zhiyong Yin

Underwater blast injury is a type of injury often seen in combats on and around islands and during landing operations. Detonation of depth charges, naval mines, bombs, cannon shells, and other munitions underwater could generate underwater shock wave, which could inflict underwater blast injury upon personnel in water. To deal with such injuries, scores of scholars both in China and abroad have undertaken active studies and explorations.

As early as World War II, several thousand cases of underwater blast injuries occurred, and people noticed that underwater blast injuries could be fatal. During the 1967 Arab–Israeli War, an Egyptian missile struck the Destroyer Eilat of Israel, after which sailors onboard abandoned ship, and another missile detonated nearby when they were in the water. Of the 32 sailors rescued, 27 had been afflicted with blast lung injury, and 24 with blast abdominal injury, of which 22 had been found to have perforated intestines. 19 suffered from both pulmonary and gastrointestinal injuries, and four died. However, experiments pertaining to underwater blast injury were not conducted until the end of the 1960s, after the U.S. military constructed underwater test site at their Kirtland base. Since the "10th Five-Year Plan," the Chinese military has been performing underwater blast injury studies, and a bevy of on-site experiments have produced numerous revelations about features of underwater blast injury.

1 Injuring Features of Underwater Blast Injury

Due to physical properties of water and the difference between pressure in underwater environment and normobaric air, the injuring features of the underwater shock wave clearly differ from the air blast induced injuries under normal atmospheric conditions, especially when the source of a shock wave is located underwater.

1.1 Physical Properties of Underwater Shock Wave

1.1.1 Velocity of Propagation

A shock wave propagates much faster underwater than when in the air. When the peak pressure of an underwater shock wave is relatively high, its propagation velocity is faster than that of sound in water. In contract, when peak pressure is comparatively low, its propagation velocity roughly equals to the speed of sound in water. The speed of sound in water (1437 m/s at 20 °C) is about four times faster than speed of sound in air (344 m/s at 20 °C). Hence, propagation velocity of underwater shock wave is around three to four times faster than air shock wave of same strength.

1.1.2 Distance of Propagation

Since water is denser than air and is relatively incompressible, not only does a shock wave propagate much faster in water, it also propagates much farther.

Take explosion of explosive, for example, when 250 g of explosive is detonated on the ground, peak overpressure at a distance of 2 m from the center of explosion is roughly 1.05 kg/cm^2. When the same amount of explosive is detonated in water, peak overpressure at the same distance soars to approximately 211.30 kg/cm^2. When 100 kg of explosive is detonated on the ground, peak overpressure at a distance of 700 m from the center of explosion is merely 0.0089 kg/cm^2, but when the same amount of explosive is detonated in water, peak overpressure at the same distance is about 1.7600 kg/cm^2. Comparing between the two, there is around 200 time difference in pressure at the same distance.

The same pattern holds true in nuclear explosion. For instance, when a 100-kiloton nuclear weapon is detonated in water, peak overpressure at a distance of 914.4 m from the

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center of explosion is around 190 kg/cm^2, but when the same nuke is detonated in the air, peak overpressure at the same distance does not even reach 1 kg/cm^2.

1.1.3 Mode and Energy of Propagation

As stated previously, when a shock wave propagates and moves in the air, it creates compression zone and rarefaction zone. At first, air particles advance along the shock wave front, but since their velocity continues to slow, eventually they would fall to the back boundary of the compression zone. At this point, air particles have a velocity of zero. Air particles fall into the rarefaction zone after moving with the shock wave front for about 100 m, then start to move toward the source of explosion. Finally, air particles would return to their approximate original positions.

This is not the case when a shock wave propagates in water. Due to water's relative incompressibility and absence of clear rarefaction property, the compression zone and rarefaction zone seen in air shock wave are missing, and water particles also do not exhibit obvious forward and backward movements because of propagation of a shock wave. A shock wave only transmits the energy of its pressure wave through the medium of water, and even water close to the center of explosion does not exhibit violent movements.

When propagating in water, the features of a shock wave's physical parameters include high peak pressure but shorter effective duration (in the realm of several hundred microseconds). This duration is much shorter than the range of several milliseconds to several tens of milliseconds for the air shock wave, or about 1/76.0 to 1/32.8 that of an air shock wave's duration. Although an underwater shock wave has a relatively short positive effective duration, its high peak pressure still translates into a much more powerful impulse than that of air shock wave. In addition, pressure increase duration at the front is extremely short and fast (at the microsecond-level), much faster than the pressure increase duration at the front of the air shock wave.

Moreover, in terms of energy of a shock wave propagating in water, when the same mass of TNT is exploded, underwater blast wave generates an average peak pressure 227.15 to 247.86 times bigger than the air blast wave, while impulse is 8.48 times to 11.80 times higher.

1.1.4 Reflected Wave and Tensile Wave

When a shock wave propagates to the bottom of the water or the surface of other rigid obstructions, it would be reflected, thereby strengthening the effect of the shock wave. However, since it is rare of the incident wave and reflected wave to reach the same spot at the same time, hence the mutually strengthening effect is not that obvious. For instance, when 0.23–3.60 kg of explosives were detonated 3 m underwater in a pool 9 m in depth, results did not show any obvious effects due to reflection from the bottom of the pool.

When an underwater shock wave propagates to the interface between water and air, it would be reflected and create tensile wave in water. Consider point A to be any point underwater. First, the positive pressure of the incident wave imparts its effect, then the reflected tensile wave arrives, and since the direction of this reflected tensile wave is opposite to the incident wave, point A would be subjected to negative pressure. The result is a decline in the effect of the incident wave.

Effects of the tensile wave differ depending on the depths of the point of explosion and point of effect. Consider point A and point B underwater, with point A close to the water surface than point B, and the point of explosion has equal distance from both point A and point B. In this setting, tensile wave reflected from the surface of the water reaches point A first, and the incident wave that arrived at point A would be interrupted at an earlier time by the tensile wave. In other words, although both point A and point B are subjected to the same peak pressure, still the positive pressure effective duration at point A is shorter than that at point B. Or, we could say that the impulse (sum of instantaneous pressure during pressure effective duration) at point A is less than that at point B. Similarly, points of explosion at different depths also have different effects on the same point of effect. When underwater explosive 1 (closer to water surface, i.e. shallower) and explosive 2 (farther from water surface, i.e. deeper) have equal distance away from underwater point A, the tensile wave created from the explosion of the shallower explosive 1 would reach point A sooner, and hence, its incident wave at point A would be interrupted at an earlier time by its tensile wave. Meanwhile, because explosive 2 is located deeper underwater, the tensile wave created from its explosion would reach point A later, and hence, its incident wave at point A would be interrupted at a later time by its tensile wave, or maybe stay uninterrupted, which is why the impulse generated would be higher.

Based on the above, it may be understood that in the event of an underwater explosion and if other conditions remain constant, persons in shallower locations are safer. Therefore, if underwater personnel anticipate an imminent explosion, he or she should swim toward the surface as quickly as possible and try to keep one's body (especially the head) above water.

1.1.5 Calculation of Pressure of Underwater Shock Wave

1. When calculating the pressure of an underwater shock wave, use the Cole's equations:

$$P=\begin{cases}4.41\times10^{7}\times\left(\dfrac{W^{1/3}}{R}\right)^{1.5} & 6\le\dfrac{R}{R_0}<12\\ 5.24\times10^{7}\times\left(\dfrac{W^{1/3}}{R}\right)^{1.13} & 12\le\dfrac{R}{R_0}<240\end{cases}\qquad(30.1)$$

In the above equation, P is peak pressure (Pa), W symbolizes charge quantity (kg), R represents distance between center of explosion and point of observation (m), and R_0 denotes initial radius of explosive charge (m).

Simplification of the above equation yields the following equation:

$$P=13000\sqrt[3]{W/R}\qquad(30.2)$$

In this equation, P is peak pressure and unit is pound/inch^2 (1 pound/inch^2 = 1/14.21 kg/cm^2 = 6.90 kPa); W symbolizes charge weight and unit is pound (1 pound = 0.454 kg); and R represents distance from point of explosion and unit is foot (1 foot = 0.305 m).

2. When influences from boundaries such as bottom and surface of water are not given considerations, the peak pressure from an underwater explosion of TNT spherical charge may be calculated using the equation below:

$$P=465\left(\frac{\sqrt[3]{W}}{R}\right)^{1.1}\qquad(30.3)$$

In this equation, the meanings of P, W, and R are the same as formula (30.2), but measurement units differ, respectively, being kg/cm^2, kg and m.

3. Effects differ depending on the depth at which the explosive is located. If nothing about the underwater explosion could be observed above the surface of water, then the least depth of the explosion may be estimated using:

$$H\ge9.0\sqrt[3]{W}$$

In this equation, H is depth of explosion (m), and W denotes quantity of explosives (kg).

Further, for a TNT spherical charge with the density of 1.6, the overpressure from its shock wave underwater may be calculated using R H Cole's empirical equation:

$$P=533\left(\frac{\sqrt[3]{W}}{R}\right)^{1.13}\qquad(30.4)$$

In this equation, the meanings of P, W, and R are the same as formula (30.3).

1.2 Relationships Between Injury Severities and Physical Parameters of Underwater Shock Wave

As stated previously, under normal atmospheric conditions, the incompressibility and density of water are much greater than that of air. Comparing with an air shock wave, the pressure increase duration and positive pressure effective duration of an underwater shock wave are much shorter. Due to the shorter effective duration, when other conditions remain the same, a person underwater could bear a higher overpressure than when in the air.

1.2.1 Information from Animal Testing

Experiments indicate that in the event of an underwater explosion, injury severities of animals directly correlate to the impulse of the shock wave generated. For example, although animals at different locations endure peak pressure and pressure effective duration that differ rather markedly, as long as they are subjected to similar amount of impulse, their injury severities do not vary much.

For different types of animals, injury severities differ even subjected to underwater shock wave of the same strength. For example, when 1000 tons of explosives were detonated in water, its shock wave could kill all organisms within a range of one nautical mile (1 nautical mile = 1.853 km), but its lethal range enlarges to four nautical miles for fish with swimming bladders (directly lethal peak pressure is 4.93 kg/cm^2, as in 70 pounds/inch^2). This is because although fish with swimming bladders can endure relatively higher peak pressure, they are more susceptible to damage from underpressure (pressure reflected from surface of water).

Experiments also proved that birds are more easily injured than mammals. If an animal is floating on water, its injury would be greatly lessened (Table 30.1). Impulses needed to impart different levels of injuries to humans are relatively close to the ones listed for mammals in Table 30.1.

1.2.2 Information of Persons Injured

Injury severities of persons are not only dependent on physical parameters such as peak overpressure, but also closely related to quantity of explosives. Generally speaking, the smaller the quantity of explosives, the higher the overpressure the human body could tolerate. Table 30.2 lists selected information about relationships between personnel injury severities and overpressure of underwater explosion

In light of all related factors, some have proposed that the standard safe distance for personnel underwater should be 0.3 kg/cm^2 or impulse of less than 0.14 kg/cm^2 ms. The peak pressure needed to kill a person is roughly 17.6 kg/cm^2 (250 pounds/inch^2).

Table 30.1 Relationships between injury severities of different types of animals and impulse of underwater shock wave

Type of animal	Quantity of explosive/kg	Depth of explosive underwater/m	Depth of animal underwater/m	Impulse/kg cm^{-2} ms	Injury severities
Birds (duck)	0.45	3.0	0.6	3.17	50% fatality, severe injury for the rest
	0.45	3.0	0.6	2.52	1% fatality, mostly moderate injury for the rest
	0.45	3.0	0.6	0.70	Light injury
	3.6	10	Above water	9.15–10.55	50% fatality, severe injury for the rest
	3.6	10	Above water	7.04–8.45	1% fatality, mostly moderate injury for the rest
	3.6	10	Above water	2.82–4.23	Light injury
	3.6	10	Above water	2.11	No injury
Mammals (goats, dogs, monkeys)	0.23–3.6	3.0	Head above water, body underwater	2.82	Moderate injury
	0.23–3.6	3.0		1.41	Light injury
	0.23–3.6	3.0		0.35	No injury

Table 30.2 Relationships between personnel injury severities and overpressure from shock wave of underwater explosion

	Minor brain concussion		Gastrointestinal hemorrhage and perforation		Start to death	
Quantity of explosive/kg	Injury distance/m	Overpressure value/kg·cm^{-2}	Injury distance/m	Overpressure value/kg cm^{-2}	Injury distance/m	Overpressure value/kg cm^{-2}
1	100–20	2.93–18.05	20–8	18.05–50.85	8	50.85
3	300–50	1.28–9.70	50–10	9.70–59.76	10	59.76
5	350–100	1.30–5.37	100–25	5.37–25.72	25	25.72
50	–	–	150–75	8.09–17.70	75	17.70
250	–	–	200–100	10.71–23.44	100	23.44
500	–	–	350–50	7.39–10.81	250	10.81

1.3 Injuring Features of Underwater Shock Wave

1.3.1 High Rate of Fatality

Due to the differences in properties of the medium, when the same quantity of explosive explodes underwater, the overpressure generated at the same distance from the center of explosion underwater is much higher than an explosion in the air, and the impulse created is also greater than an air shock wave, therefore, underwater blast injury has a relatively higher rate of fatality. Death rate of air shock wave is mostly under 20%, but that for underwater blast injury could range from 40% to 70%.

1.3.2 Large Fatal Range

Underwater shock wave can kill in an area nine times that of an air shock wave. 0.5 kg of TNT exploded underwater has the same fatal zone boundary as that of 40 kg of TNT exploded in the air.

1.3.3 Lungs Are the Main Target Organs

Lungs are the main target organs in underwater blast injuries. Since the lungs contain a lot of air, a shock wave releases its energy at the interface between tissue and air, thereby causing damage. In addition, when there is severe tearing of lung tissues, air inside alveoli could enter pulmonary veins via tears in the small vessels, creating air embolism, which would move around the whole body through the circulatory system. Death could occur very swiftly if air embolism enters and clogs the main coronary or cerebral artery.

Lung injury severity is closely associated with impulse. When impulse is more than 300 kPa ms, extremely severe lung injuries occur in 91.7% of dead animals. When impulse is below 300 kPa ms, the severe lung injury rate is merely 7.7%.

1.3.4 Much More Severe Damage to Abdomen than Air Blast Injury

Since the abdomen is directly in contact with water whether the person is underwater or floating on his/her stomach, it is more common for organs in the abdomen (mainly the gastrointestinal tract) to sustain injury during an underwater explosion than an air explosion, and the injury is usually more severe. The frequent colon and small intestine injuries, respectively, occur around 50% and 30%, much higher rates than such injuries caused by air shock wave.

Furthermore, when underwater compression wave presses on the abdomen, other injuries could be induced such as diaphragmatic muscle tear, and compression-related ruptures of hollow organs like the intestines and solid organs like the spleen, kidneys, and liver. Compression wave could also propagate inside the anus and cause "explosion" inside the colon.

1.3.5 Severe Injury to Air-filled Organs and Low Rate of Injury to Solid Organs and Liquid-filled Organs

A substantial amount of practice and clinical observations prove that hollow organs filled with air after are often severely damaged under the effects of shock wave underwater. Meanwhile, hollow organs containing liquid such as the gall bladder, urinary bladder, and renal pelvis might be spared from injury. The mildness of injury to this type of organs may possibly be associated with the proximity between their densities and water, which means that a shock wave passes through these organs in a short duration, and therefore these organs only absorb a small amount of the shock wave's energy.

Researchers have conducted the following experiment: Fill animal intestinal cavities with normal saline, then place intestines underwater, which remain largely unscathed after explosion, even when intestines were placed near explosives. However, if there were even tiny amount of bubbles in the intestines, then obvious intestinal wall perforations could be observed immediately after explosion.

1.3.6 Extremely Rare Surface Wounds and Multi-injuries

One clear characteristic of underwater blast injury is the extreme rarity of wounds on the surface of the body. This is because there usually are not many secondary projectiles generated by an underwater explosion, and therefore, personnel will not or are very unlikely to be injured by secondary projectiles. After an explosion, personnel underwater or on water surface may possibly be tossed because of the waves created, and those on the water surface may even be launched into the air, but usually they will not or are very unlikely to land on any hard object; therefore, external wounds on the surface of body are uncommon.

1.3.7 Rare Craniocerebral Injuries

In underwater blast injuries, injuries on the surface of the head are mostly minor, and such injuries might not happen at all. This is because during an underwater explosion, the majority of victims are near the surface and their heads are above water. Animal testing with dogs indicates that if heads are submerged underwater when explosion occurs, eardrum perforation is frequent, and auditory ossicle fracture may even occur in severe cases.

2 Clinical Features of Underwater Blast Injury

Clinical documentations on underwater blast injuries indicate features including body surface external wounds that are extremely rare, head injuries that are usually minor, most injuries inflicted upon organs in the chest and abdominal cavities, severe injuries for organs containing air, mild injuries for organs containing liquid and very minor injuries for solid organs, and more serious injuries on the inside than outside.

2.1 Clinical Pathological Features of Underwater Blast Injury

2.1.1 Lung Injuries are Most Common

Pulmonary hemorrhage is the most common type of lung injury. Usually hemorrhage occurs in both sides of the lungs, from dispersed spots of bleeding to widespread bleeding across the whole lobe. Two parts of the lungs are most susceptible to pulmonary hemorrhage: First is located in areas where alveolar tissues connect with the bronchioles and blood vessels; therefore, tube-shaped bleeding could be seen in areas surrounding the bronchus; second is the surface of the lungs adjacent to body parts such as the heart, diaphragm, ribs, and spine, and streaks or patches of bleeding could be observed. For victims of severe pulmonary edema, substantial hemorrhagic infiltration could cause consolidation of lung lobe, which might conceal the minute changes described above. Bloody and foamy fluid or blood clots could be seen inside the bronchus. In addition, pulmonary edema, bullous interstitial emphysema, pneumomediastinum, and blood and gas accumulation in the pleural cavity arising from tearing of the pleura and lung parenchyma may also be found. Under microscopic inspection, ruptured capillaries and internal bleeding in the alveolar walls may be observed in mild cases, and widespread ruptured alveolar septum with large amount of blood in alveolar space could be seen in severe cases. Interstitial or alveolar edema may be seen nearby the bleeding area, but this is more commonly found in those that have already survived for a couple of hours. Microscopic inspection also frequently reveals injuries around the bronchial tree, characterized by the separation of alveolar tissue from airway and blood vessels, causing the gaps surrounding the bronchus and blood vessels to be filled with blood, edema fluid, lymphatic fluid, and/or gas. When the pulmonary vein ruptures, alveolar venous fistula could occur, which may possibly be the main culprit responsible for air embolism that often causes death in early stage of blast injury victims.

For underwater blast injury victims with lung injuries, severe cases may exhibit anoxia, breathing difficulty, and problems immediately after injury, but body compensation would initiate in the case of most victims; therefore, clinical symptoms of lung injuries could take some time before appearing. Analyzing clinical information on 27 underwater blast injury victims with lung injuries, the most common symptom is hemoptysis, followed by breathing difficulty, some local or even extensive moist rales and dry rales may

Table 30.3 Clinical features and their occurrence rates among 27 cases of underwater blast injury victims with lung injuries

Symptoms and signs	Number of cases	Occurrence rate
Hemoptysis	15	55.6
Breathing difficulty	11	40.7
Dry rales and moist rales	11	40.7
Chest pain	6	22.2
Cyanosis	5	17.5

be heard on auscultation, and chest pain and cyanosis may be observed in a small number of victims (Table 30.3).

2.1.2 Abdominal Injuries Are Relatively Common and Severe

1. Injuries to the digestive tract: Injury could occur from the lower segment of the esophagus to the rectum, but injuries most often happen in the large and small intestines. Hemorrhage and perforation are common injuries. Hemorrhage conditions differ from cases to cases and could be anything from spots of bleeding behind the peritoneum, under the serous membrane or on the mucous membrane, to extensive bleeding.

   Based on the form of bleeding, such hemorrhage may be divided into five types:

   a. Isolated spots of bleeding with the size of a needle, existing independently or in small clusters.
   b. Thin streaks of bleeding or many spots clumped together, sometimes ring-shaped, having extended to areas around the intestinal cavity.
   c. Ring-shaped belt of bleeding with clear boundaries, often accompanied by a small volume of effusion of blood in the intestinal cavity.
   d. Broad bleeding belt comprised of ring-shaped streaks of bleeding, often seen with a substantial amount of blood clots in the intestinal cavity, and found immediately next to the mucous membrane of the area. In some cases, blood clots could be large enough to clog up the intestinal cavity. This type of injury is usually accompanied by perforation.
   e. Net-like bleeding in ring shape or large swath, but this is rather rare. Bleeding usually occurs on the surface of mucous membrane, often seen on direction opposite to the mesentery.

   When gas accumulation in the intestinal cavity remains relatively stationary, acute perforation could easily take place, with perforation ranging from 1 cm to 4 cm in diameter and one to several in number, but some cases might suffer from 20 or so perforations. There is usually bleeding around the perforation. Under the effect of shock wave, if the gas moves to another location, the result could be mucous membrane rupture or hemorrhage, as in the so-called incomplete perforation. Upon this basis, delayed perforation may possibly occur because of necrosis, secondary infection, or posthemorrhagic ulcer. Upon microscopic inspection, this kind of perforation is characterized by obvious rupture in the intestinal wall, coupled with muscle tear and breakage. Pieces of ripped mucous membrane and blood clots may be seen stuck in perforations, indicating that the rupture occurred from the inside out.
2. Injuries to other organs: Hemorrhage could occur in solid organs such as the liver, kidneys, spleen, pancreas, adrenal gland, and testicles. For some cases, acute tearing in liver, kidneys, and spleen may also be discovered.
3. Symptoms and signs: Victims of underwater blast injury to the abdomen frequently complain about sudden and acute abdominal pain after the explosion, as if he or she has been kicked in the belly. Also common are short-term numbness in the lower limbs, nausea, vomiting (sometimes but not always blood seen in vomit), and tenesmus. Some victims complain about sense of electrocution and testicular pain. For victims rescued relatively late or those with severe injuries, minor to moderate shock may occur. For victims without intestinal tract perforation, different degrees of pressing pain and muscular tension in the abdomen may appear, and sometimes rectum bleeding may be found. For victims with intestinal tract perforation, typical symptoms and signs of acute abdominal disease are common, pressing pain of the abdominal wall, muscular tension in the abdomen, board-like rigidity, followed by abdominal distention, weakening or disappearance of borborygmus, and rectum bleeding, among others. In addition, victims of underwater blast injury to the abdomen are often afflicted with temporary and minor paralysis of the lower limbs, which could be attributed to injury of small blood vessels in the spinal cord. Worth mentioning is that if pain killers are used during the evacuation process, some of the signs and symptoms might not express as obviously. Investigations and survey of 28 cases of survivors of underwater blast injury indicate that signs and symptoms in the abdominal region are very common (Table 30.4).

2.2 Typical Cases

Below are three cases of typical underwater blast injuries.

Case 1: Male, 20 years old, soldier, injured because of accidental explosion of explosives during underwater blasting demonstration. During the explosion, the victim's body area below the shoulder was submerged in water and was located merely 5 m from the point of explosion. Right

Table 30.4 Clinical features and their occurrence rates among 28 cases of underwater blast injury victims

Signs and symptoms	Number of cases	Occurrence rate
Right after or soon after explosion		
Sensations of the effects of	24	35.7
underwater shock wave	19	67.9
Sudden abdominal pain	11	39.3
Short and sudden numbing of lower	11	39.3
limb	7	25.0
Nausea or vomiting	5	17.9
Tenesmus	6	21.4
Testicular pain	2	7.1
Sudden chest pain		
Sensation of electrocution		
After admission to hospital (8–11 h after explosion)		
Symptoms of acute abdominal disease	24	85.7

after the explosion, the victim felt a sense of suffocation, followed by sustained and sharp pain in the chest and abdomen. After being evacuated onshore, the victim passed out and immediately coughed up several coughs of blood. Then chest pain subsided, but sustained pain around the navel continued and even worsened. The victim was admitted into the hospital 1 h after injury. After admission to the hospital, the victim excreted dark red bloody feces once, around 100 ml in volume. During inspection, the victim exhibited mental sluggishness, a small amount of moist rales could be heard in the middle and lower lobe, and left and lower lobe of the right lung. There were also mild muscle tension around the navel, obvious pain upon contact, rebound tenderness, no dull sounds of movement, and substantial weakening of borborygmus. Digital rectal examination only found pink, viscous fluid on glove. Chest X-ray revealed patches of shadows in middle and lower lobe, and left and lower lobe of the right lung, indicating pulmonary hemorrhage. Laboratory examination: White blood cells 9×10^9/L, neutrophil granulocyte 81%, urine protein ++, WBC 3-4/HP, Hyal-cast +/LP, Gran-cast 1-3/LP. Diagnosis determined that the victim was afflicted with injury to the chest and abdomen (blast injury). Exploratory laparotomy was carried out after 4 h of treatment and observation. Laparotomy revealed free gas in the abdominal cavity and around 300 ml of old blood fluid. There were also four perforations 0.2×0.2 to 1×0.5 cm in sizes in the lower segment of the ileum. After the laparotomy, the victim went into shock, and balanced electrolyte was quickly administered. This resulted in diffused moist rales in both lungs, and the victim expelled a large volume of foamy and bloody secretion through the tracheal catheter. Diagnosis determined that the victim was afflicted with pulmonary edema. After tracheotomy, and 1 h of oxygen inhalation (use oxygen bubbled through a 95% alcohol solution) and drug treatment [immediate use of intravenous therapy to administer deslanoside (lanatoside C), furosemide, phentolamine, hydrocortisone, etc.], the victim's cyanosis lessened, bloody and foamy phlegm disappeared, and moist rales in both lungs significantly reduced. The victim pretty much recovered fully after 2 weeks in the hospital.

Case 2: Male, 22 years old, soldier, injured at the same place and same time as case 1. When the explosion occurred, the victim's body area below the nipples was submerged in water. At the instant of the explosion, powerful waves of water forcefully struck the side of chest and waist. The victim felt sustained and sharp pain in the xiphisternum and navel area, and the aforesaid symptoms clearly lessened 5 min later. Only sustained, dull pain was felt around the navel area afterward. One hour after injury, the victim excreted bright red bloody feces a total of three times, totaling around 300 ml in volume. The victim was admitted to the emergency room. During inspection, there was mild pressing pain around the navel, and digital rectal examination found pink, viscous fluid on glove. After hospital admission, the victim did not consume food for 1 day and was administered with fluid infusion, injected with carbazochrom to stop bleeding, and orally ingested tetracycline. The victim basically recovered after 3 days.

In the two cases presented above, even though the victims were situated at the same location during the explosion, their conditions differed drastically. Case 1 suffered from perforations in the intestines, but case 2 did not. After analysis, possible reasons include: (1) body position. During the explosion, case 2 faced the explosion from the side, resulting in more minor injuries; (2) volume of air contained in intestines. Perhaps case 1 had more air in the intestines, making them more prone to rupturing.

Case 3: Male, 21 years old, swimmer, body was submerged in water with only head above water, a relatively large firecracker was mistakenly dropped into the water 1.5 m away from the victim and exploded underwater. After the explosion, the swimmer immediately felt sharp pain, coupled with breathing difficulty. Before arriving at the hospital, the victim was first brought to a nearby clinic where a tube was inserted through the mouth. Upon arrival at the hospital, the victim suffered from severe breathing difficulty, with GCS score of 8, blood pressure of 135/91 mmHg, pulse of 116 times/min, breathing rate of 32 times/min, oxygen saturation of 95%, and had to use traditional ventilator (FiO_2, 100%). Upon inspection, breathing sound was obviously weaker on the right side, and external wound was found. Preliminary CT scan revealed hemorrhagic lung contusion involving multiple lobes, accompanied by bilateral lung volume loss and minor hemothorax on the right side. In addition, no abnormalities were discovered in the thoracic and

abdominal cavity. Arterial blood gas analysis indicated severe respiratory acidosis, with pH 7.26, $PaCO_2$ value of 54 mmHg, and PaO_2/FiO_2 value of 222 mmHg. Thereafter, intrathoracic chest tube was inserted on the right side, and victim was transferred to ICU ward. Early treatment objective was to deal with lung contusion, and measures employed included lung debridement, mechanical ventilation, inhaled corticosteroids. On the first morning after hospital admission, X-ray was undertaken, and 4 h later chest X-ray was performed again, and severe opacification was seen in right side of the chest. Neither arterial blood gas analysis nor body check results pointed to any signs of improvement. Bedside bronchoscopy revealed blood and serous secretion. These discoveries, along with worsening clinical process, further confirm the existence of lung contusion. Therefore, in-depth lung debridement and in-depth treatment continued.

On the second morning after hospital admission, as in 10 h after signs of worsening clinical process and adoption of corresponding measures, chest X-ray was once again carried out. Results indicated that ventilation of the right lung began to improve, and body check showed that the victim's mental response had restored, along with better breathing sounds heard from both sides. On the afternoon of the same day, tube was removed from the victim, switching to oxygen mask inhalation of air with 40% oxygen instead. Victim condition maintained its uptrend and was discharged from the hospital on the same afternoon after fully recovering. Telephone follow-up 2 years later, no sequela or chronic effect of injury was found.

3 Treatment Principles for Underwater Blast Injury

Underwater blast injury is a common problem in naval battles, but there are not many articles and reports about how to care for and treat underwater blast injuries. We conducted animal testing by inducing injuries through detonation of TNT underwater, then observed pathophysiological changes of underwater blast injuries and related clinical symptoms and signs, and analyzed treatment principles for underwater blast injury, with the aim of providing corresponding theoretical basis for treatment in the early stage.

3.1 Diagnosis of Underwater Blast Injury

3.1.1 Injury Environment

Personnel underwater are extremely susceptible to injury to explosions from objects like naval mine, torpedo, or bomb, and first and foremost it is necessary to ask the victim about his or her body position and posture when injured and distance from point of explosion.

3.1.2 Symptoms and Signs

When there are lung injuries, severe cases may exhibit anoxia and breathing difficulty, and respiratory distress, bradycardia, and sustained low temperature are indications of severe condition. Therefore, these indications may also be used as warning indicators for assessing severity of underwater blast injury. If the aforesaid changes occur, that would mean severe patient conditions and further inspection and corresponding first-aid measures should be undertaken.

Moreover, hemoptysis often occurs with lung injuries, and blood or bloody and foamy liquid from the nose and mouth are also common. For those afflicted with serious pulmonary hemorrhage and pulmonary edema, signs may appear similar to severe lung contusion, along with dull sounds from percussion, and weakened breathing sounds with widespread dry or moist rales heard through auscultation. B-mode ultrasound, X-ray, CT scan, and MRI may all be used for diagnosis.

3.1.3 Arterial Blood Gas Analysis

Arterial blood gas analysis outcome 30 min after injury shows that lung injury severity is closely associated with drop in arterial partial pressure of oxygen (PaO_2). For those with extremely severe or severe lung injury, PaO_2 figures 30 min and 6 h after injury are both significantly lower than pre-injury level, mostly falling within the range of 8–9.3 kPa (60–70 mmHg). Meanwhile, changes in PaO_2 levels are not obvious in those afflicted with moderate or minor lung injury. PCO_2 30 min after injury in cases of extremely severe lung injury is substantially higher than that before injury, and PCO_2 levels of those with extremely severe or severe lung injury are obviously higher than those afflicted with moderate or minor lung injury.

3.1.4 Exploratory Laparotomy

Research indicates that upon the basis of full resuscitation or simultaneously alongside resuscitation, exploratory laparotomy should be conducted promptly. This is because underwater blast injury often occurs simultaneously with injuries to the abdomen involving multiple organs or body parts, such as multiple intestine perforations. Therefore, laparotomy should not be stopped when one injury has been identified, instead thorough and meticulous inspection shall be performed in order to rule out the possibility of missed diagnosis, which could lead to dire consequences.

3.2 General Principles for Treatment of Underwater Blast Injury

Based on experiment and research outcomes, we are of the opinion that treatment of underwater blast injury should follow the basic principles listed below:

1. Keep airway unobstructed: Immediately clean out any bloody and foamy fluid, blood clots, foreign substance, or other obstructions in the oral and nasopharynx cavities,
2. Protect respiratory function and provide sufficient oxygen: For patients suffering from hemothorax or pneumothorax, large-gauge needle should be used to remove air and blood in the thoracic cavity immediately, and if conditions permit closed thoracic drainage should be undertaken. Provide sufficient oxygen for patient, and when condition permits use oxygen bubbled through a 50% alcohol solution to remove bubbles and facilitate breathing.
3. Rapidly restore body temperature: Excessively low body temperature could seriously suppress cardiac, respiratory, and other functions, which would aggravate underwater blast injury. When treating underwater blast injury patients, pay close attention to changes in body temperature, and for critical and serious cases who's body temperature falls and does not restore, use physical methods, drugs, or warming devices to help the patient warm-up, so as to lessen the impacts of low body temperature on the body.
4. Rest in bed: Minimize activities, and for critical and serious cases strict bed rest should be ordered to reduce pressure on cardiac and respiratory functions, so as to prevent secondary hemorrhage.
5. Prevent gastrointestinal tract perforations from worsening infection in the abdominal cavity: When diagnosis is not clear, avoid orally ingested fluids or foods.
6. Use volume resuscitation: For hemorrhagic shock arising from uncontrolled blood loss, treat patient with limited fluid resuscitation to maintain palpable radial pulse and buy time for evacuation.
7. Evacuate with haste: After patient condition stabilizes, evacuate immediately. For severe cases of lung injury, keep the patient in a head down position to prevent air embolism from entering heart and cerebral vessels, and for those not at risk of air embolism, the semi-reclining position may be adopted. If helicopter evacuation is employed, try to keep flight at a low elevation to avoid potential air embolism risks.
8. Use mechanical ventilation: After first-aid treatment, if full-body anoxia has not improved, consider the use of mechanical ventilation for patients meeting any one of the following circumstances: Respiratory rate > 40 times/min; $PaO_2 < 60$ mmHg; $PaCO_2 > 50$ mmHg; pulmonary shunt >15%; or CAT scan shows scope of lung injury exceeding 28% of total area of lungs. Intermittent positive pressure ventilation (IPPV) or high frequency jet ventilation (HFJV) are the ventilation modes most frequently adopted.
9. Use compressed oxygen: The compressed oxygen treatment method may be employed for patients with air embolism. Quickly place patient within an environment with 6 bar ambient pressure, then decompress based on how symptoms are allaying, and upon reduction to 2.8 bar ambient pressure, immediately switch to using 100% O_2. During the subsequent decompression duration, intermittently use 100% O_2.
10. Deal with pulmonary edema and protect cardiac function: Treatment principles are identical to treating general pulmonary edema and cardiac insufficiency, including the administration of dehydration, diuretic, and cardiotonic drugs. For patients with bradycardia, administer 0.5mg to 1.0mg of atropine via intramuscular injection. Administer cortical hormone in large dosage early on to prevent pulmonary interstitial edema.
11. Use blood transfusion and other infusions to combat shock: Pulmonary edema is frequently seen in patients with lung injuries, whose tolerance for liquid would decline. Therefore, volume of transfusion and/or infusion should not be too high, and pace not too fast, and prioritize the usage of whole blood, plasma, and other capsules while lowering the administration of crystoloid solution. When condition permits, utilize central vein and pulmonary artery intubation monitoring of hemodynamics, so as to gain information conducive to guiding fluid resuscitation.
12. Prevent bleeding and infection: Administer hemostatic drugs and a suitable amount of antibiotics to prevent bleeding and infection in the lungs and gastrointestinal tract.
13. Prevent diffused intravascular coagulation and hypokalemia: For cases of serious lung injuries, diffused intravascular coagulation and hypokalemia could occur. In such instances, make use of transfusion of fresh plasma, frozen blood cell and platelet, coupled with potassium chloride IV and other corresponding treatments.
14. Beware of anesthetics: Victims of blast injuries usually have a low tolerance for anesthetics 24–48 h after injury, so avoid surgery early on if possible. If surgery is required, opt for local anesthesia or regional anesthesia in lieu of general anesthesia. If general anesthesia cannot be avoided, refrain from utilizing ether as anesthetic.
15. Perform surgery: Treatment principles for liver or spleen rupture, gastrointestinal tract perforations and other internal organ injuries are same as those for general trauma, but endeavor to keep it simple and safe.
16. Monitor clinical developments closely: All underwater blast injury victims with or without gastrointestinal injury need to be hospitalized and remain under observation for more than 1 week in case of any delayed intestinal perforation.

3.3 Treatment Principles for Underwater Blast Injury to the Chest or Abdomen

Experiment and research outcomes indicate that the lungs are still the main target organs in underwater blast injury, as well as the primary reason for death in the early stage. Thus, early stage treatment of underwater blast injury victims should consider caring for the lungs to be the key objective in rescue effort. Further, the effects of large impulse could also result in internal bleeding due to gastrointestinal tract perforation or liver rupture, and these should also be focal points.

3.3.1 Treatment Principles for Underwater Blast Injury to the Chest

Since it is very rare to see serious multi-injuries in underwater blast injuries, after ruling out situations such as fractured ribs, pay close attention to injuries to the heart and lungs.

Victims afflicted with minor injuries to the heart and lungs can usually recover after adequate rests and symptomatic treatment, provide mask or nasal tube breathing if condition permits, and make sure patients refrain from arduous activities.

For cases of moderate or more serious lung injuries, consider adopting the following treatments:

1. Strict bed rest
2. Ensuring airway stays unobstructed
3. Artificial respiration: If the treatments stated above do not yield obvious results, and if arterial partial pressure of oxygen remains below 6.6 kPa (50 mmHg), consider using extracorporeal membrane oxygenation (ECMO) device.
4. Compressed oxygen treatment: Compressed oxygen treatment can prolong time of survival and improve recovery rate, and it is also beneficial to patients with arterial air embolism.
5. Prevention of pulmonary edema and protection of cardiac function: Pulmonary hemorrhage and edema are often seen in cases of severe blast lung injury, and dehydration measures such as mannitol and furosemide may be applied. When shock occurs, use hypertonic solutions such as hypertonic glucose, hypertonic saline, or plasma, and utilize recovered tissue edema fluid to make up for insufficient blood volume. In order to protect cardiac function, drugs such as digitalis, lanatoside C, and strophanthin K may be applied.
6. Prevention of blood loss and infection: If patient has been afflicted with serious tears in the lungs and when ordinary non-surgical treatments are ineffective, proceed to surgery.

3.3.2 Treatment Principles for Underwater Blast Injury to the Abdomen

1. Drinking and eating shall be strictly prohibited.
2. For those that go into shock due to injury to solid organ or great vessel, carry out exploratory laparotomy as soon as possible, so as to repair injury and stop bleeding. Examination needs to be done in an orderly fashion, particularly beware of overlooking any issues. Ameliorating shock is a prerequisite for any surgery.
3. Simplicity and safety should be considered paramount for surgery.
4. When there is clear abdominal distension or suspected bowel obstruction, apply continuous gastrointestinal decompression and then if necessary perform surgical exploration.

4 Tiered Treatment for Underwater Blast Injury

Blast injury refers to a biophysical and physical–chemical phenomenon. Specifically, this refers to clinical symptoms and changes in pathological and anatomical aspects that occur because a living body has been exposed to a powerful explosion or shock wave.

At present, blast injury is still a popular and controversial topic of discussion. For medics involved in treating victims of blast injuries in armed conflicts, blast injury is one of their main focal points.

In the past, military medical service support and "time-effect treatment" both prove that the outcome of military medical care hinges on the time of care and measure of treatment adopted, hence the rule of "time-effect." Due to the unique characteristics of underwater blast injury (severe patient condition, high injury death rate, and injury disability rate), studies about its first-aid process and tiered treatment model are important to achieving the best "time-effect" outcomes.

In general, the treatment methods of underwater blast injury are largely similar to those for dealing with air blast injury. For overall management process of blast injury, please see Fig. 30.1, and its specific tiered treatment methods are outlined below.

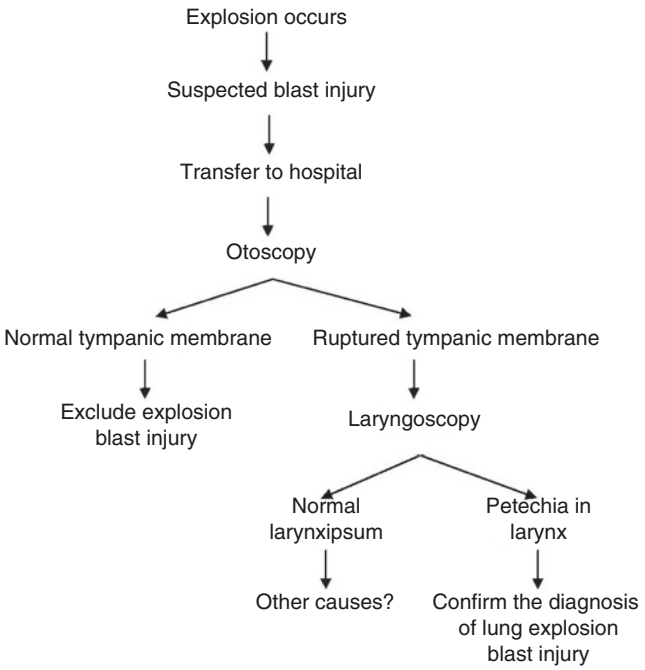

Fig. 30.1 Management process of explosion blast injury

5 Protection Against Underwater Blast Injury

Blast injury is common in modern armed conflicts and is the main type of injury inflicted in future hi-tech warfare. In the last three decades, blast injury has risen as a key subject matter in military medicine study and has gained the attention of military medicine researchers both in China and elsewhere across the globe. Shock wave is the result of energy released from an explosion, which is formed when local air is compressed and moves outward at an increasing speed. The pressure of shock wave soars immediately after the explosion, then weakens exponentially. In the beginning, a certain degree of overpressure is created in the surrounding air, and then a certain degree of negative pressure is generated, with the peak negative pressure being smaller than its peak positive pressure. An explosion in water also generates shock wave, but since water is much denser than air; therefore, underwater shock wave propagates at a much higher speed and farther distance. When the same mass of substance explodes underwater, its range of injury and fatality is three times larger than the injury range and fatality range when exploded in the air. The damage that shock wave imparts on the human body had already been broadly publicized and reported after World War I. In naval battles, troops crossing the sea and landing on beaches, amphibious recon troops, marine troops in charge of underwater demining, obstruction removal, and those that fell into the water were highly vulnerable to underwater blast injury. There were several thousand cases of underwater blast injury during World War II. The injury mechanisms and injury characteristics of underwater blast injury differ from those of air blast injury. Generally speaking, underwater blast injuries are more severe and more difficult to treat.

In the 1970s, we once used plaster and plastic products to create protection against shock wave. These had some degree of effect, but were difficult to actually apply in real-world scenarios. In the 1980s, researchers paired artificial leather with foamed plastic to produce protective suit with the aim of weakening the effects of shock wave. Of which, efforts included bulletproof vest researched and developed by Phillips et al., research on shock wave protection using composite layer consisting of copper foil-covered foam and Kevlar-covered foam conducted by Cooper et al., research on ceramic-Kevlar composite material carried out by Young et al., and research on the performance of foamed plastic in shock wave protection undertaken by Skews. It was discovered that singularly using foamed plastic, Kevlar, copper foil, and bulletproof vest all worsen shock wave injury inflicted on air-filled organs in the human body, while ceramic-Kevlar composite, copper-foamed plastic composite, and Kevlar-foamed plastic composite do not intensify the injury effects of shock wave.

As studies on high polymer materials and composite materials advance forward, researchers have obtained more insights into protection against shock wave. In the 1990s, outcome from research by Yang Zhihuan et al. indicates that nickel foam offers rather solid protection against blast injury, including reductions in blast injury severity and fatality rate in test animals. Nickel foam can substantially lower peak overpressure of a shock wave, with the performance even more prominent when material thickness is increased, but at the cost of extended shock wave positive pressure effective duration. Outcomes of studies on the performance of polyurethane materials in protecting against shock wave indicate that initial shock wave force in the polyurethane material created by shock wave would significantly decline with the increase in size of initial porosity of material. When initial porosity is equivalent to 0.25 mm, polyurethane foam material possesses rather prominent anti-shock and pressure reduction characteristic against the load of an explosion. Perhaps the underlying mechanism is associated with the large number of pores and gaps inside the polyurethane material. First of all, pores in the porous material are eliminated because of compaction of the material under the effect of the shock wave, elastic deformation occurs in the pores and gaps, with some of the impact energy turning into elastic energy, while air gaps compress adiabatically and absorb a

part of the energy. Next, plastic collapse or brittle fracture occurs at the walls of the pores, converting some of the energy into plasticity, while the adiabatic compression process of the air gaps ends. This continues until porous material is compressed to a state similar to compacted material. How the propagation of shock wave declines in porous material is contingent on the energy absorbed and consumed during the compaction process. Foamed plastic also offers some buffering effect under the load of shear stress wave, and at the same time, foamed plastic also exhibits obvious stress wave diffusion under the load of shear stress wave, reflecting the viscoelastic property of polyurethane foam material.

Yu et al. studied the role of closed-cell aluminum foam material in the cracking of lightweight composite armor back plate and developed corresponding three-dimensional finite element model. Ballistics experiment results indicate that aluminum plate foam material has obvious effects in many regards from shock wave shielding and collision energy absorption, to reduction or prevention of back plate crack.

Yang Zhihuan et al. once tested anti-blast injury performance of a composite material comprised of nickel foam, LC4 aluminum alloy, and sponge bars as main components. Outcome from their research indicates that said composite material offers rather ideal protection against blast injury with peak overpressure range of 388.4–399.7 kPa and positive pressure effective duration lasting from 55 to 60 ms. These function as a certain extent of evidence and reference for the research and production of protective equipment. Cfipps et al. used plastic layer and foamed plastic for glass shaping (GRP/PZ) in an experiment with 17 test animals, demonstrating that said material effectively reduced blast lung injury in the test subjects. It offers better protection than foamed plastic covered with shaped lead.

Research by Hattihgh et al. shows that after a shock wave acts on a single-layered material, an intensification wave acts on the wall of the protective material again, which may possibly be related to the movement of the material toward its back wall, and also because of the shock wave passing through the material. On the contrary, this intensification wave does not appear in multi-layered composite material.

This is due to the scattering effect from the surface and microporous structure of multi-layered composite material, which endows said material with a higher shock wave viscosity coefficient. Experiment outcomes prove that the scattering effect significantly slows down the propagation of shock wave in such multi-layered composite material, and the propagation velocity of shock wave in this kind of structure is also much slower than that in other types of structure. The varying ways in which shock wave propagates through different composite materials have captured the attention of scientists, but the majority of knowledge about shock wave propagation in composite materials come from analysis of linear elasticity of ideal composite materials. The applications of composite material layers in protecting against shock wave have made large progress, and such materials offer more advantages than traditional metallic materials in aspects such as resilience, hardness, weight, and convenience. However, there is not a lot of experiment information about the performance of composite materials in terms of defending against impact and collision. Due to its unique properties, polymers have found widespread application in equipment and components that have to bear pulse dynamic load (i.e. collision, explosion, EMP, thermal pulse, etc.), having replaced some metallic materials that have become scarce. Tedesco et al. previously analyzed how layered structures affect the blast wave of conventional weapons, pointing out that a combination of layered materials can weaken blast wave, but stopped short of specifying what kinds of layered material combinations and structures are more effective in attenuating shock waves.

Barker and Oved discovered noticeable decline in a shock wave of certain amplitude when it acted on multi-layered composite material, as well as a certain degree of resonance generated because of multi-layered composite material. Multi-layered composite material structure can support a certain degree of shock wave effects. Due to scattering effect of surface, effective impact viscosity increases as surface impedance and mismatch increase and decreases as surface density increases.

Research by Mouritz indicates that composite material after resin casting possesses higher tensile strength than composite material without such treatment. Composite materials with resin casting have relatively few disadvantages and are strengthened against the destructiveness of shock wave.

If the materials in the various layers remain the same, arranging them in different sequences can directly affect the strength of the shock wave that propagates through and out of them. When a blast wave acts on GRP composite material layer, more layered fracture would be seen, which reduces the compressions, tension, bending, and stress fatigue of the GRP composite material, while adding stitches could lessen the layered fracture of the composite material. Research shows that stitched GRP composite material layer can markedly weaken a blast wave, but conversely offers less protection against bullets.

Research by Woodward et al. on GRP composite material indicates that there are two main destruction processes: First is the dynamic compression before the fracturing of the GRP material, and second is the layered fracture destruction on the surface subjected to impact. Both these processes can effectively stop or reduce the effects of shock wave and fragments.

Research by Shah Khan et al. asserts that polyurethane-resin composite material has higher strength and elastic coefficient than vinyl-resin. Composite material made from

polyurethane and resin performs better than that consisting of vinyl and resin.

MMC or metal matrix composites are comprised of a reinforcing material coupled with a metal basic material. The reinforcer could be continuous fiber or silk made from high-strength material such as carbon, boron or carbide, or short fibers or granules like aluminum oxide, silicon carbide or boron carbide. Compared with base metal without reinforcement, MMC offers unique dynamic and physical properties, hence the material has very promising application potentials in hi-tech and military domains. Some studies have shown that Gr/A1 metal composite is a type of material associated with strain rate, as in the higher the strain rate, the higher the material's tensile strength, buckling strain, and residual strength correspondingly. The material is clearly characterized by the strain rate strengthening effect and dynamic fracture toughness. Other studies have reported that poly carbon fiber-reinforced aluminum composite offers a certain degree of protection from underwater blast wave. Aluminum powder crystal (99.0% purity and 100–200 mesh) is the base material for this kind of composite, while PAN is the basic carbon fiber utilized (7 μm diameter and 200 μm length). The components are pressed to form a hard solid.

In 2004, Xing Shuxing and Yin Zhiyong tested underwater blast injury protection performance of composite materials on rats, and their research outcome indicates that a composite material consisting of PC polycarbonate porous material layer-poly ester material layer-hard sponge layer-fiberglass layer offers clear underwater shock wave reduction effect, with peak positive pressure lowering by 52.55 ± 3.34% and positive impulse decreasing by 46.98 ± 3.38%. This demonstrates that protective measures such as reflecting interfaces created from the conjunction of high-density and low-density media, energy absorption property of porous medium, prestress introduced to and over-expansion restriction applied to the thoracic and abdominal areas of animals, and the utilization of soft, energy-absorbing materials to prolong the pressure increase duration when a shock wave acts on the animal to slow down the acceleration of the underwater shock wave acting on the body are effective.

Water is about 800 times denser than air and is characterized by relative incompressibility and sparsity. Therefore, underwater shock wave is characterized by features such as high speed and long distance of propagation. An air shock wave creates a certain negative pressure, but that is not true for underwater shock waves. Compared with air blast injury, underwater blast injury is characterized by features such as severe injury condition, high fatality rate, and large area of injury.

In light of these characteristics of underwater shock wave, it is necessary to adopt protective measures different from those for air underwater shock wave. In terms of body parts damaged in underwater blast injury, in addition to the lungs being the main target organs, the heart, small intestine, colon, and rectum are all very susceptible to damage. Therefore, other than protecting the chest area to lighten injury on the lungs, underwater blast injury protection should also strengthen defense of the abdominal area to lessen injuries inflicted on organs in the abdomen. Furthermore, underwater blast injury protection has to be practical. The combination of such protective measures into other gears like life vest should be considered or incorporating multiple functions (i.e. impact protection, burns protection, fragment protection, as well as heat preservation and floatation function) into these protective equipment during the research and production process.

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Cabin Blast Injury

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1 Overview

Here, the term "cabin" includes ship, airplane and armoured vehicle cabins, but also generally refers to enclosed or semi-enclosed spaces where personnel may be located. Hence, "cabin blast injury" refers to direct or indirect injuries inflicted on personnel inside a cabin environment due to explosion that occurred inside or outside the cabin and may also be known as "blast injury in enclosed space environment." Cabin blast injuries are frequently seen in wartime when tanks, armored vehicles, above-ground or underground fortifications, or vessels are struck by missiles, cannon shells, land mines, or other high-explosive ammunitions. During peacetime, cabin blast injuries could occur in terrorist bombings, such as detonation of improvised explosive devices inside public buses, train coaches or subway coaches, or car bombs outside buildings. In addition, when explosive substances (i.e. fireworks, firecrackers, pyrotechnics items, etc.) explode indoor during production or storage, or accidental explosions involving high-pressure boilers in factories could also cause cabin blast injury.

In modern warfare, the frequency of cabin blast injury is much higher than open space blast injury. This is because an increasing number of combatants rely on ground surface cabins (tanks, armored vehicles, field fortifications, permanent above-ground fortifications), underground cabins (tunnels), water surface cabins (naval vessels), and underwater cabins (submarines) during armed conflicts. During the Gulf War in 1991, the U.S. military and Iraqi military fielded some 8500 tanks, and even though ground battles totaled only about 100 h, 4200 Iraqi tanks and 2800 armored vehicles were destroyed, along with nearly 70,000 of fatalities and wounded of Iraqi troops. During the Falklands War in 1983, within a month and a half the Argentinian and British navies, respectively, suffered from 11 and 18 sunk or damaged vessels. During the War in Afghanistan that began in 2001 and the Iraq War that commenced in 2003, the U.S. military deployed 40% of armored vehicles in service, and 63–70% of American and coalition troop injuries came from armored vehicle cabins being struck by high-explosive anti-armor rounds. During the Afghan War (1984–1987), the Soviet military lost 1461 tanks and armored personnel carriers, and during the Battle of Grozny in the Second Chechen War (1999–2000), 75% tanks and 85% armored vehicles of the Soviet 131st Motorized Rifle Brigade were destroyed.

Due to the highly destructive power of weapons used to attack cabins and spatial properties of cabin such as enclosed or semi-enclosed environment, cabin blast injuries are characterized by features such as severe injury, high injury death rate, blast injury, and high rate of combined injuries. For instance, fatality rate for casualties in armored vehicle cabin or vessel cabin could exceed 60%, three times more than regular battle death rate, while composite injury rate is three to five times higher than that of regular infantries, and burns rate is 10–20 times more likely to occur than regular infantries.

2 Types of High-Explosive Ammunitions Used to Strike Cabins

There are a plethora of high-explosive ammunitions used against cabins. For instance, based on the type of target, ammunitions may be categorized as anti-armor ammunitions, anti-ship ammunitions, or hard-target penetration bombs; based on the branch of armed forces equipped, ammunitions may be categorized as army, navy, or air force ammunitions; based on the application feature, ammunitions may be categorized as artillery shell, bomb, torpedo, naval mine, missile, rocket, aerial bomb, or demolition device; based on destructiveness, ammunitions may be categorized as explosive ammunitions or kinetic energy penetrator, with the former relying on high-energy explosive in

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the warhead to explode and destroy cabin, while the latter uses purely kinetic energy derived from high velocity generated by high-pressure gas of the propellant to penetrate the cabin or penetrates first and then explodes to destroy the target cabin. To better understand the features of cabin blast injuries due to strikes from weapons, the next section provides explanations about military high-explosive ammunitions used to attack chambers based on the injuring characteristics of ammunitions.

2.1 Explosive Ammunitions

2.1.1 High-Explosive Ammunition and High-Explosive Fragmentation Ammunition

High-explosive ammunition usually has thin casing, and a sizable quantity of high-energy explosives are packed inside the shell, amounting to some 50–80% of the total weight of the ammunition. The direct effects of explosion or impact of explosion are used to destroy cabin targets. Naval mine, land mine, aerial bomb, missile, among others, belong to this type of ammunition. Naval mines and land mines can hold large charges that yield serious destructive power. They are also cheap to produce and easy to deploy, which are why they are the most common kind of ammunition used to attack cabins. Between 1950 and 2001, among the types of weapons used to attack American naval vessels, naval mines ranked first with 77%, followed by aerial bombs in a distant second with 11%. 70% of U.S. tanks destroyed during the Vietnam War in the 1960s and 1970s came from land mines.

High-explosive fragmentation ammunition relies on primary fragments to damage personnel, structures, and material, and charge usually accounts for 20% of the ammunition's weight. This type of ammunition is chiefly used against personnel. However, if the ammunition explodes near a cabin and when fragments carry enough kinetic energy, the lightly protected cabins like those of light armored vehicles may still be penetrated.

2.1.2 High-Explosive Anti-tank Warhead

Also known as shaped charge, these warheads are characterized by a hollow metal liner (often copper or aluminum) which formed in a conical or hemispherical shape, and typically bonded to the explosive fill on the convex side. Upon explosion, the metal liner is rapidly collapsed by the charge, and when the resultant velocity of the metal on the surface of the liner exceeds the rate of collapse, the metal would be turned into a superplastic jet with higher energy density, with the velocity of the jet capable of reaching 7000–14,000 m/s and a temperature of 500 °C. The jet could generate peak pressure of 100–200 GPa on the armor plate, and after weakening the average pressure ranges around 10–20 GPa. The high pressure resulting from the mutual effects between the hot metal jet and target plate causes lateral displacement in the target plate, which would then be penetrated and pierced by the jet. If the resultant velocity of the metal on the surface of the liner does not exceed the rate of collapse, it would create a high-speed projectile also known as self-forging fragment submunition, which would reach a speed of around reaching 1500–3000 m/s and penetrate cabin using its own powerful kinetic energy. Anti-tank rockets, artillery shells, anti-tank missiles, and certain anti-ship missiles use high-explosive anti-tank. The metal jet would create devastating injuries if the jet penetrates the armor and directly strikes personnel inside cabin, causing large losses and damage to tissues and severe burns. Personnel not in the direct path of the jet would be injured mostly by fragments that break off from the cabin.

2.1.3 Enhanced Blast Warheads

These warheads, a new sort of enhanced blast weapons, include fuel air explosive and thermobaric ammunition, and are often seen in aerial bombs, artillery shells, and rocket shells. The first generation of enhanced blast warhead is known as fuel air explosive bombs. Such ammunitions are loaded with liquid hydrocarbons, a type of easily gasified fuel. When the payload explodes in the air above the target, the fuel is dispersed and creates a cloud of mixture with oxygen in the air (thermobaric zone), and then this cloud would be ignited. Pressure in the explosion center in the thermobaric zone could reach 2–3 MPa, and sustained explosion duration is several dozen times longer than those from high-energy explosives such as TNT. Remaining fuel would continue to combust, forming a fireball with temperature upward of 1500–2000 °C. Since overpressure effective duration of the shock wave of the thermobaric zone could last several tens of milliseconds and create high impulse, its explosive power is five to ten times more powerful than TNT of the same mass. This is why fuel air explosive is often considered the current number one explosive when it comes to harm and death to personnel and obliteration of equipment and fortifications. The new generation of fuel air explosive is a thermobaric weapon, which carries solid charge comprised of the explosives with high velocity of detonation and oxidizers such as ammonium nitrate and aluminum nitrate, and powdered flammable metal like aluminum, magnesium, and zirconium. Upon dispersal, the combustion cloud zone consisting of micro solid explosive substance would be detonated, generating higher shock wave overpressure and releasing more heat energy than fuel air weapons of the past.

2.1.4 Improvised Explosive Device

IED broadly refers to bombs built from military ammunition or civil explosives and is used in forms such as suicide bomb, car bomb, and roadside bomb. Improvised explosive device could contain a sizable amount of explosives and is often

detonated with a timer or remote control in close proximity to armored vehicles or buildings; therefore, they can cause serious injury and death for personnel inside these cabins. During the Iraq War and War in Afghanistan, IEDs had become an increasing bane to U.S. soldiers, with troop injury rising from 20% in the beginning to 60% later on.

2.2 Kinetic Energy Ammunition

2.2.1 Armor-Piercing Shell

This type of shell features initial velocity exceeding 900 m/s and high accuracy, relying on its high kinetic energy to penetrate and damage cabin and to injure and kill personnel inside. These ammunitions are commonly seen in tank guns, anti-tank artillery, naval guns, and coastal artillery, but anti-tank missiles and anti-ship missile warheads often adopt this structure as well. Structure of armor-piercing shell primarily falls into two categories. The first category features a solid structure with warhead or core made of dense, rigid, and tough material like tungsten or depleted uranium, and the parts of the shell remaining after piercing through armor plating and fragments broken off from plating are the main injury-causing factors. The second category has a charge chamber instead, which would be loaded with a small amount of explosives and detonated after penetrating into the cabin, causing damage and injury a second time via explosion. Depleted uranium and tungsten alloy armor-piercing warheads generate heavy metal aerosol particles during the penetration process. Depleted uranium aerosol could enter the body through the respiratory tract, digestive tract, skin and mucosae, wound, and other pathways, leading to internal radiation injury and heavy metal poisoning. In addition, depleted uranium warheads could easily heat up and ignite under hi-speed collision and have relatively powerful incendiary effects. Inhaling substantial amount of aerosol tungsten particle could trigger damage to kidney functions.

2.2.2 Deep Penetration Warhead

This kind of munition is used for attacking defensive fortifications buried underground. Its penetrative warhead is capped with high-strength steel or heavy metal alloy material, inside of which would be explosives linked to a delayed fuse, giving the warhead time to penetrate deeper underground before detonation. U.S. Air Forces claimed the destructive power of bunker buster aerial bomb, one type of deep penetrating warhead, is 10–30 times bigger than when the same mass of explosive is detonated on the ground. During the Gulf War in 1991, the U.S. Air Force fired two bunker busters at a civilian underground bomb shelter located in capital Baghdad. The body of the ammunition penetrated reinforced concrete 2.15 m thick and the cover layer before exploding, instantly killing more than 400 people hidden inside.

2.3 Composite Ammunition

This type of ammunition combines two or more destructive effects of the ammunitions introduced above, such as semi-armor piercing high-explosive anti-ship missiles rely on its own kinetic energy to defeat ship armor, then explode inside after penetration, using high-speed projectile, random fragments, and shock wave to damage the target cabin. Artillery shells, rocket bombs, and anti-tank missiles often employ shaped charges that form jets of hot metal to defeat armored targets, while the shrapnels generated from the explosion of the body of the ammunition are effective weapons in wounding and killing personnel. In addition, to enhance damage, composite ammunitions are often loaded with other components such as incendiary, igniting agent, or smoke agent, which would enter the cabin along the explosion to injure personnel via burns or gas inhalation and cause damage to equipment.

3 The Influence of Cabin Structure on Blast Injuries

3.1 Level of Enclosure

Based on the level of enclosure, a cabin may either be classified as enclosed or semi-enclosed. An enclosed cabin refers to an indoor space that is completely enclosed, like those inside vessels, tunnels, tanks, and armored vehicles, and the interior air, temperature, and humidity are all controlled by regulating devices. A semi-enclosed cabin refers to spaces such as public bus, building, and field fortification, where indoor environments that are covered but still connected with the outside via openings like doors, windows, and observation ports. Air inside and outside the cabin flow naturally, and temperature and humidity inside are basically the same as those outside. The cabin enclosure level is closely associated with blast wave propagation inside, as well as the transmission of heat and dissipation of smoke.

3.1.1 Complex Shock Wave

When an explosion occurs inside a cabin, the blast wave acts on the walls of the cabin, leading to reflection and diffraction and creating a complex shock wave comprised of the incident wave and the subsequent series of reflection waves. Unlike the simple shock wave in a free field like that from an explosion in an open space, the peak overpressure of a complex shock wave could be two to eight times more powerful than a free field simple shock wave, while positive pressure effective duration could last anywhere from several milliseconds to more than 10 ms. A simple shock wave surges to its peak pressure at an instant's notice, then exponentially decays to ambient pressure, with positive pressure effective duration mostly staying within a range of several hundred

microseconds to 1 or 2 ms. But since a complex shock wave has higher peak overpressure and longer positive pressure effective duration, it causes more damage to the tissues of personnel inside the cabin, resulting in worst fatality rate. For instance, terrorist bombing on public bus could cause death rate up to 49%, while explosion from the same mass of explosives in an open space would only be 8% or so.

3.1.2 Difficulties in Heat and Smoke Dissipation

The heat released from the explosion and leftover fuel of the projectile could quickly elevate the temperature inside the cabin. If said temperature reaches the ignition point of some of the more flammable items, additional combustions could occur, and the high-temperature and high-pressure state of the cabin also speeds up the rate of combustion of flammable materials. The occurrence rate of combustion inside cabin and burning personnel is many times higher than open space explosion, with larger areas of and more severe burns. During the Fourth Arab–Israeli War (October War) in 1973 in the Middle East, the occurrence rate of burns in tanks and armored vehicles hit by anti-tank weapon was three times higher than those suffered by infantry, and "anti-tank weapon complex" characterized by blast lung injury, blast injury to eardrums, injury from inhalation, and injury to the eyes appeared. During the Falklands War in 1982, of the injured British navy sailors, 34% suffered burns and serious cases had more than 60% body area burned. Due to the enclosed environment of cabins, smokes generated from explosion and combustion will not dissipate as easily, and carbon monoxide, dust, and particulate matters produced by smokes inhaled by personnel in the cabin will irritate and corrode respiratory tract and lung tissue, with chemical pneumonia or pulmonary edema in serious cases. When a fuel air explosive hits a tunnel or fortification, the combustion of the fireball derived from leftover fuel could consume all oxygen inside, and other than the shock wave and high heat inflicting blast injuries and burns to personnel inside, suffocation is also a main cause of death for those in the cabin.

3.2 Cabin Construction Material

Cabins could be built from a wide range of materials from wood and reinforced concrete to steel, aluminum, and soil. The varying physical and chemical properties and differences in ignition point of materials differ drastically, and these variables have a significant impact on the secondary damage deriving from explosion.

3.2.1 Fragments of Cabin

Steel has a density of 7.85 g/cm^3, elasticity modulus under room temperature of 190–220 GPa, and shear modulus of 70–80 GPa. Wood meanwhile has a density of 0.54 g/cm^3, elasticity modulus under room temperature of 9.8–12 GPa, and shear modulus of 0.5 GPa. Since the aforementioned physical and chemical properties of steel and wood differ by two to three orders of magnitude, cabins made out of the two materials differ markedly in standing up to an explosion. Under the effects of a blast wave and high-speed projectiles, a steel cabin has a much smaller chance of being destroyed compared to a wood cabin, but once the cabin has been damaged, the interior of the steel cabin would be filled with cabin fragments that are high in mass and kinetic energy, which would result in more severe injuries to personnel. Interior of wood cabin meanwhile would be the opposite, cabin fragments are relatively lower in mass and kinetic energy, therefore causing less grievous injuries to personnel struck compared with steel debris.

3.2.2 Combustion

Wood has an ignition point of 220–229 °C and therefore could start burning easily. The ignition point of aluminum is merely 550 °C, much lower than steel's ignition point of approximately 1300 °C. Vessel and armored vehicle cabins made from aluminum alloys inflict more serious burns on personnel inside if combustion occurs after being hit. During the Falklands War in 1982, when a torpedo fired by the Argentine Air Force hit the British destroyer HMS *Sheffield*, combustion of the bridge's aluminum alloy materials was a major reason for the severe burns suffered by personnel onboard.

3.2.3 Derivative Shock Waves

Blast wave outside could pass through cabin walls, enter the interior of the cabin and form derived air shock wave. In an experiment, 2.2 g of RDX (charge density of 1.34 g/cm^3) was attached tightly to an enclosed container with 1.0 cm-thick steel wall. Pressure sensor was used to measure pressure inside the container at the instant of the detonation, and high-speed Schlieren photography was used to observe the whole process of shock wave propagation inside the enclosed container. The researchers discovered that the overpressure of the incident wave inside the container could reach 0.030 MPa. The result suggests that when the shock wave from the explosion enters the steel wall, other than the absolute majority being reflected, a small portion of the wave was still projected onto the free surface on the inside wall of the enclosed container. Under the effect of the shock wave, the free surface moved forward, compressed air in front of it and generated derived shock waves.

3.2.4 Impact Shock, and Compressed Wave and Seismic Wave in Soil

When the cabin exterior is in contact with an explosion or when an explosion occurs inside a cabin, some of the energy

of the explosion would couple with the walls and plates of the cabin and propagate toward the surroundings in the form of stress waves, creating impact shock, and compressed wave and seismic wave in soil.

The walls of vessels, tanks, and armored vehicles are mostly constructed from steel plates and other materials that are dense, not very compressible, and do not consume much deformation energy. On the contrary, they can transfer pressure quite well, and when a stress wave propagates along cabin walls and plates, two kinds of impact shock would occur on the walls and plates: First is local acceleration of walls and plates at anywhere from 100 to 1000 g during the instant of the explosion. The acceleration lasts some microseconds, does not cause much displacement, and could increase axial stress on body parts such as limbs and spinal cord in contact with the wall or plate, which might result in bone fracture if stress exceeds fracture threshold. Second is the bending of the wall or plate, as well as the movement of the whole armored vehicle or vessel, and the resulting tossing. At this moment the cabin's acceleration would be around less than 100 g and could last for seconds, throwing personnel in the air or against the cabin walls and plates and causing collision injury.

When underground fortification constructed from soil, rock, and reinforced concrete is hit by an explosive weapon, soil compressed by the shock wave or the contact of the weapon against the ground and exploding would generate compressed wave and seismic wave in soil. When compressed wave and seismic wave in soil act on underground tunnel and fortification, reflections would occur. The overpressure of the incident wave could be several times more powerful than the overpressure of the reflected waves, which would cause serious damage to fortification and injure personnel stationed inside.

4 Injuring Mechanisms

Mechanisms behind damages caused to cabin and injuries inflicted upon personnel inside when cabin is struck by ammunition may be classified into either primary or secondary. Primary mechanism refers to direct effects of the ammunition, such as penetration, blast, fragment, heat, and other damaging and injuring effects. Secondary mechanism meanwhile refers to secondary damaging and injuring effects resulting from the aforesaid mechanism, such as secondary fragments, craters, additional explosions, and combustions.

4.1 Primary Mechanism

4.1.1 Penetrating Effects

Bunker buster and high-explosive anti-tank create hot metal jet flow that penetrates through cabin plate and wall, damaging the cabin and injuring personnel inside. This is called penetrating effect.

Armor-piercing shell, bunker buster, and high-speed projectile can use their high kinetic energy to penetrate into cabin, damaging the cabin and injuring personnel inside. When a warhead collides with cabin plating at high speed, the spot of contact bears immense pressure (30–50 GPa), and when the pressure exceeds the cabin wall material's yield strength, the spot of cabin wall struck by the ammunition would structurally deform, the cabin plate would be plugged or be broken. When the ammunition enters the cabin, if there is enough kinetic energy in the remaining shell or fragments broken off from the cabin wall, they could kill personnel within and detonate or ignite ammunition contained inside. Cabin wall, under the effects of impact load, would generate compression wave, shear wave, or other types of stress waves with high velocity and big amplitude, which could lead to slight displacement and acceleration in cabin plating within several milliseconds, leading to noticeable macro movements like bending or vibration. Body parts directly in touch of cabin plating near the spot struck by ammunition could be injured in forms such as soft tissue contusion, bone fracture, contusion of thoracic and abdominal organs, and impairment of peripheral nerve conduction.

High-explosive anti-tank rounds could generate hot metal jet flow with acceleration of up to or exceeding 7000 m/s at the front, and the mutual effects between the jet flow and cabin wall would create a super high-pressure penetration at the GPa level capable of defeating and piercing cabin walls. The extensiveness of destruction of cabin wall caused by the high-speed jet flow penetration depends on the thickness and strength of the cabin wall material. When a jet flow pierces a reinforced concrete cabin wall, the diameter of the perforation created would be five to seven times the diameter of the jet, and the concrete on the path of the jet flow would burst from the back of the cabin wall (engineering equation for calculating concentrated jet flow penetration of concrete panel). Metal material hit by a jet flow could be damaged in the form of cratering, layered fracture, or perforation. When a metal cabin wall is perforated, the remaining high-temperature metal jet flow containing metal particles and cabin wall fragments would turn into a high-energy fragment cloud that could hit personnel in high density, causing large areas of damage and loss of tissue and burns to deep tissue.

4.1.2 Blast Effects

Static overpressure of shock wave and dynamic pressure of blast wind created by the explosion could damage cabin and injure or kill personnel in what are collectively termed blast effects. Blast effects are most obvious with explosive ammunition, and sometimes kinetic ammunition hitting a cabin at high speed could also produce blast effect.

When high-energy explosives such as RDX, TNT, or plastic explosives (C4 or Semtex) in the charge of an explosive ammunition explodes, a substantial amount of high-temperature and high-pressure gases would be generated at an instant, rapidly compressing gaseous medium surrounding the point of explosion, and drastically and immediately elevating its density, pressure, and temperature. The interface (wave front) between said compressed gas layer and uncompressed gas layer is the shock wave. At the interface, the level of static pressure exceeding atmospheric pressure is the static overpressure, and it decays in the air to the reciprocal of the distance cubed from the explosion center, or put it differently, overpressure at twice the distance would be 1/8 of overpressure at the explosion center. For example, when 1 kg of explosives detonate and create shock wave static overpressure of 500 kPa at the explosion center, static overpressure at a spot three meters from the explosion center would drop to 18.51 kPa. When the shock wave from an explosion inside a cabin, or when shock wave from an explosion outside enters a cabin through channels or cabin wall cracks, due to reflections from cabin walls and concentrations at cabin corners, in addition to the surge in indoor temperature of the cabin caused by the explosion, a shock wave's peak static overpressure could soar by several to several dozen times, while positive pressure effective duration would also extend. As the shock wave front propagates forward, static overpressure behind the front would gradually decline, eventually turning into negative pressure. Cabin subjected to static overpressure could be crushed, deform, collapse, or suffer other structure destruction, and human organs containing air such as eardrum, lungs, and gastrointestinal tracts could be afflicted with contusion, tear, or other injuries under the effects of static overpressure. Compared with the pressure tolerance of buildings, human organs have relatively strong injury resistance against static overpressure. For instance, static overpressure reaching lung injury threshold is around 210–280 kPa, but the same static overpressure is more than enough to damage reinforced concrete structures and buildings.

Due to the pressure difference, air behind the shock front would flow toward explosion center rapidly, creating blast wind or else known as "drag wind," and its impact on a target is known as dynamic pressure. Peak dynamic pressure is related to cube of wind speed, and effective duration is longer than that of static overpressure. The generation of blast wind depends on static overpressure. For instance, at a static overpressure of 7 kPa, blast wind could reach a maximum velocity of 17 m/s, and 73 m/s when static overpressure is 34.5 kPa. When a blast wind is strong enough, it could displace, toss, and strike the human body, with bone fracture, skull fracture, organ bleeding or rupture, and disfiguring amputation in severe cases. Combat injury investigation indicates that under the effect of blast wind, if movement of a 70 kg human body equals to or exceeds 3 m, fatality is likely to occur; when movement is more than 1.5 m but less than 3 m there would be serious injury; and when movement is between 0.3 m and 1.5 m, injury severity hinges on angle of strike against another object and could range from no obvious injury to moderate injury. In addition, blast wind could mobilize sand, rocks, wood splinters, and glass shards nearby, weaponizing them into secondary projectiles that could wound anyone struck.

When a shock wave's static overpressure and blast wind act on the human body, the static overpressure tolerance of the person's organs and tissues clearly declines. For instance, static overpressure that only causes eardrum rupture in 1% of victims when acting alone could be fatal when combined with a blast wind with maximum velocity of 73 m/s (Table 31.1). During a cabin explosion, the majority of personnel inside would be afflicted with both static overpressure and blast wind, which is why injuries are often severe. For example, when an armored vehicle drives over a land mine, shock wave static overpressure concentrates stress locally on longer bones of the limbs of the passengers, and the subsequent blast wind that tosses the crew around would cause bone fractures at those spots of high-stress concentration.

4.1.3 Fragmentation Effects

Fragmentation effects refer to injuries and deaths caused by fragments dispersed from the shell of the ammunition upon explosion.

Ammunition fragments could be categorized as random fragments, preformed fragments, semi-preformed fragments, and high-speed projectiles. Random fragments are naturally formed from the bursting of the shell, and fragments could fly at speeds somewhere between 1500 m/s and 2000 m/s. The shape, size, mass, and number of fragments are random. Preformed fragments are those loaded into the shell by design, while semi-preformed fragments come from a piece of slit material in the shell, which would readily break off into fragments upon being shattered by the detonation of

Table 31.1 Synergistic injury effect of static overpressure of shock wave and dynamic pressure of blast wind

Peak static overpressure (kPa)	Maximum wind speed (m/s)	Human injury effect
7	17	Flash burns
14	31	Fragment injuries (glass shards)
21	46	Serious injury can result in death
34.5	73	Fatal injury occurs frequently
69	131	Fatal most of the time
138	224	100% death

Zipf, K. R. J., & Cashdollar, K. L. (n.d.) Explosions and Refuge Chambers. Aug 14, 2016. www.cdc.gov/niosh/docket/archive/pdfs/NIOSH-125/125-ExplosionsandRefugeChambers.pdf

explosive. Preformed fragments, semi-preformed fragments have more uniform kinetic energy and penetration power and can cause more damage to cabin and those inside compared with natural fragments. High-speed projectiles are formed from explosion of warheads of armor-piercing shells, and properties such as high mass, high speed, and structural shape are conducive to breaching armors (analysis of damage and destruction caused by warhead of semi-armor-piercing explosive anti-ship missile).

Fragments driven by explosives can cause a large area of damage and injury, some ten times more than that of shock wave. Weapon research and production organizations usually consider fragments with 80 J of higher kinetic energy to be capable of injuring, including immediately incapacitating or even fatal wounds. However, from blast injury rescue instances it can be observed that effectively injuring fragments depend on not only kinetic energy, but the relationships between anatomical features of the body part struck and injuring effects are also crucial. When a fragment with kinetic energy below 80 J hits the heart or a large vessel, the wound could be lethal. When a fragment with kinetic energy above 80 J strikes soft tissue of the limbs but does not damage large vessel, such injuries are mild and combatant would not be incapacitated right away.

When a depleted uranium fragment stays in the body for an extensive period, depleted uranium content in the body would rise significantly. The chemical toxicity of and radiation injury of heavy metal are not to be ignored and could cause cancer or mutations. Depleted uranium in the body could result in chronic injury to the liver and kidney and damage to the reproductive system.

4.1.4 Thermal Effects

Damages to cabin and injury or death of personnel caused by heat energy released from the detonation of explosives are called the thermal effects of explosion.

When high-energy explosives such as TNT explode, one-third of chemical energy converts into shock wave and high temperature (2500–5000 K) at the instant of explosion, and the remaining two-thirds are released slowly in the form of combustion. The fireball resulting from this combustion could reach a temperature of 2000–3000 K and last for milliseconds. Enhanced-shock wave ammunitions like thermobaric bomb and fuel air explosive disperse and detonate energetic heterogeneous mixtures to produce a thermal effect. The fireball resulting from such weapons is several times or several dozen times larger in terms of diameter and duration time than those formed by high-energy explosives such as TNT. When an armor-piercing shell or bunker buster penetrates a target, the powerful impact would rapidly raise the temperature of the core or shell. Depleted uranium or zirconium particles in armor-piercing shells that contain such metals might spontaneously combust during the armor-piercing process. When a high-explosive anti-tank explodes, it creates a metal jet flow comprised of microscopic metal fragments that could reach a temperature of around a thousand degrees.

Due to the enclosed environment of a cabin, high temperature from the explosion or leftover fuel of the missile will not dissipate quickly, and this heat inflicts obvious injury upon personnel inside the cabin in the forms of transmission, convection, and radiation. Smoke produced from combustion contains toxic gases such as carbon monoxide, nitrogen monoxide, nitrogen cyanide, hydrogen chloride, and formaldehyde. These toxic gases would stay within the enclosed environment of a cabin for prolonged duration, and personnel inside could easily be poisoned. Moreover, combustion also uses up oxygen and could potentially kill by suffocation.

4.2 Secondary Mechanisms

4.2.1 Secondary Fragmentation Effects

When struck by an explosive weapon, damage of the cabin and equipment inside the cabin, scattered parts of cabin, equipment fragments, glass shards, rocks, and other secondary fragments and splinters could further cause damage and injury. These are known as secondary fragmentation effects. Secondary fragments fly at speed much lower than other fragments and metal jet flow fragments, and transfer energy upon hitting the human body through cutting or crushing. Secondary fragments of high mass could cause destructive injuries to personnel, while secondary frags with smaller mass would usually inflict non-fatal, soft tissue injuries to the torso and limbs.

4.2.2 Cratering Effects

When an explosive ammunition detonates near the ground, upon contact with the ground or after burrowing a certain depth underground, or when a bunker buster explodes after penetrating deep underground, soil and rocks around the point of explosion would rapidly compress due to the effects of products of explosion and shock wave, leaving an explosion cavity and resulting in the cratering effect. For example, if an explosion occurs relatively deep underground, there might not be a bulge or collapse on the ground surface. If an underground explosion occurs relatively closer to the surface, soil on the ground surface would rapidly move upward due to pressure inside the cavity beneath, bulging or throwing media outward, and leaving a crater. Cratering from explosion and the stress wave generated by drastic compression of media surrounding the point of explosion could cause damage or collapse of cabins in underground or above-ground fortifications within a certain area, and personnel inside these cabins would be buried or crushed.

4.2.3 Secondary Blast or Ignition Effects

If a cabin is struck with ammunition that explodes, and such explosion causes ammunitions or flammable items contained inside the cabin to explode or ignite, the resulting cabin damage and personnel injury are called secondary blast or ignition effects. A secondary blast would occur when pressure of the initial blast wave is greater than or equal to the critical initiation pressure of the explosive item. Secondary blasts also happen when explosion product, high-speed projectile or fragment, or hot metal jet flow hits ammunition inside the cabin. Secondary blasts often obliterate a cabin completely, and personnel within usually will not be spared from death or critical injuries. When heat energy released from the initial explosion reaches the ignition point temperature of flammable objects, secondary ignition would take place. 70% of British tanks in World War II ignited after being hit.

5 Features of Cabin Blast Injury

There are two main pathways to gain an understanding about the features of cabin blast injuries. The first is to analyze past cabin blast injury cases, such as cases of blast injuries in armored vehicle cabins or naval vessel cabins, or blast injuries in enclosed environments targeted by terrorist attacks. The second is to conduct animal testing using live rounds to induce destructive effects, and studies based on laboratory simulations. Cabin blast injuries are the most realistic, but they are hard to access. At the same time, there are limitations such as form of combat, environment of explosion, type and equivalent mass of explosion ammunition involved, which make it difficult to gain a thorough understanding about related injuries. Animal testing conducted using live rounds to induce destructive effects and studies based on laboratory simulations can supplement the shortcomings of real-world combat data and provide a more comprehensive comprehension about the features of injuries inflicted upon personnel inside a cabin struck by different kinds of explosive ammunition. Animal tests conducted using live rounds to induce destructive effects are based on actual combat exercises designed based on specific forms of battle. Live rounds are used to strike targets such as tanks, armored vehicles, underground or aboveground fortifications, buildings, or 1:1 models of vessels, armored vehicles, and other cabins, then researchers would perform observations of injury conditions of animals placed inside targets, so as to determine the probability and features of injury if people were housed inside such target cabins. Studies based on laboratory simulations rely on simulated armored vehicle or vessel cabins containing small test animals, then a small equivalent mass of simulated explosion source would be detonated inside or outside the cabin. These studies reveal insights about local or body-wide pathological and physiological changes induced by single or composite injuring factors of the tested weapon or ammunition and serve to verify theories about cabin blast injury mechanisms and treatment of such injuries.

In recent years, the Field Surgery Research Institute (currently Army Medical Center) of the Third Military Medical University of the People's Liberation Army has carried out relatively systematic animal testing using live rounds to induce destructive effects, and studies based on laboratory simulations, having accumulated a rather comprehensive set of test and experiment data about features and injuring mechanisms of cabin blast injuries, and their treatments. In this chapter, cabin blast injury features not only reference past injury cases in battle and terrorist attacks of enclosed environments, but also cabin blast injury research outcomes produced by said research institute.

5.1 Severe Injuries and High Death Rate

Death rate of cabin blast injuries is much higher than average death rate of ground surface battles. In all ground surface battles since World War II, average fatality rate of combatant fluctuates around 18–20%. Analysis of statistics of 769 injured or killed riders in British tanks and armored vehicles during World War II reveals a death rate of 37%, one-third of burned victims were afflicted with third-degree burns, and 45% of limb injuries were open fractures and disfiguring amputations. The majority of these injured British armor troops were caused by armor-piercing shells (51%), followed by high-explosive anti-tanks (37.5%), anti-tank mines (8%), and other weapons (3.5%). Death rate of Soviet tank troops reached as high as 69% and severe injury rate was 22%, while minor injury rate was only 9%. On May 17, 1987, Dassault Mirage F1 fighter, an Iraqi jet aircraft, fired two Exocet missiles at the American frigate USS *Stark*, with death rate as high as 69%.

In terrorist bombings, the death rate, primary blast injury occurrence rate, and area of body burned in bus bombings are all several times higher than blast injuries in open spaces. Statistics of terrorist bombings with 30 or more persons injured that took place in 27 nations between 1991 and 2000 show that occurrence rates of blast lung injuries, blast lung injury complications and pneumothorax in buildings, subway stations, hotels, and other enclosed environments are three times higher than explosions in open spaces. Similarly, eardrum rupture, bone fracture, and burns occurrence rates are, respectively, six to seven times, three times, and 22 times higher than those in open areas (Table 31.2 and Table 31.3).

Table 31.2 Comparison between bus bombing and open space blast

	Open space blast	Bus bombing
Mortality	8%	49%
Occurrence rate of blast injury	34%	78%
Burn area	18%	31%
Average ISS	4 (minor)	18 (moderate/severe)

Leibovici D, Gofrit ON, Stein M, et al. Blast injuries: bus versus open-air bombings: a comparative study of injuries in survivors of open-air versus confined-space explosions. *J Trauma.* 1996;41:1030–1035

Table 31.3 Distribution of injuries caused by terrorist bombings

Injury type	Cabin blast injury (%)	Open space blast injury (%)	Notes
Blast lung injury	21	7	Diagnosed as lung contusion, pneumothorax, mediastinal emphysema, and blast lung injury
Pneumothorax	9	3	Including cases of hemopneumothorax and blast lung injury requiring chest tube insertion
Blast lung injury complications	16	5	Including cases of acute respiratory distress syndrome and blast lung injury requiring mechanical assisted ventilation
Ruptured tympanic membrane	35	5	
Intestinal perforation	3	0	
Fractures	20	6	Including cases of open fractures
Amputation	3	1	Including phalanx
Burns	22	1	

Arnold JL, Halpern P, Tsai MC. Ming-Che, Mass casualty terrorist bombings: a comparison of outcomes by bombing type *Ann Emerg Med.* 2004;43:263–273

Outcomes of animal testing on destructive effects of weapons show that when an anti-ship missile hits a simulated destroyer, test sheep/goats placed in various cabins around the point of impact all died instantaneously due to destructive injuries caused by shock wave and fragments. When high-explosive ammunitions penetrate tank, armored vehicle, or above-ground fortification, half of the test sheep/goats inside these cabins died within 3 h. Using the "new injury severity score" (NISS) to evaluate injury severity of test sheep/goats inside above-ground fortification subjected to explosive weapon strike, the average score for test sheep/goats injury is 20.00 ± 15.76 with a median of 17.5 (9.29) when the fortification is penetrated, and the average score is 12.06 ± 10.88 with a median of 10 (2, 19) when the fortification is hit but not penetrated. These results prove that the respective NISS figures of animal injury in the two different fortification damage conditions stated above differ significantly ($Z = -2.568$, $P < 0.05$). Instantaneous death rate of test sheep/goats inside cabin is related to the type of combat vehicle/structure cabin tested. Armored vehicle cabin has the highest death rate (75%), followed by penetrated tank (67%) and penetrated above-ground fortification (53%).

Outcomes of laboratory simulation studies indicate that when a small equivalent of high-energy explosives detonates in a simulated armored cabin, occurrence rate of abdominal organ injury in test rats inside the cabin was 1.2 times higher than explosion of equivalent explosives in open space, with liver injury occurrence rate 2.13 times higher than explosion in open area (36.7%/11.7%); intestinal endotoxemia occurrence in test rats inside cabin took place 2.5 h earlier than animals subjected to open space explosion, while duration lasted 24 h longer; and positive rate of blood bacteria in mesenteric lymph nodes, liver, portal vein, and periphery in test rats inside cabin was 2.27 times higher than those in open area explosion (35.0%/15.4%). The aforementioned research outcomes reveal that explosions in the enclosed space of a cabin, relative to explosion in open area, result in more severe injuries to solid organ in the abdominal cavity, earlier bacterial translocation from intestine and earlier onset of intestinal endotoxemia.

5.2 Obvious Polarization of Injury Conditions

There is obvious polarization in cabin blast injuries. When the American frigate USS *Stark* was hit by the two Exocet missiles in 1987, of the 17 survivors, other than two that suffered from moderate to severe burns, the other 15 were all afflicted with minor injuries, including three with fragmentation wounds, eight with soft tissue contusion, three with corneal flash burns, and one with dehydration.

Outcomes of animal testing on conventional weapon effects indicate that for tank cabins hit and penetrated by armor-piercing shells, 70% of the tanks would ignite, 67% of test animals within would die, and two-thirds of test sheep/goats had open fractures and disfiguring amputations. Of the test sheep/goats that survived for 10 min or more, 25% had severe injuries, while the remaining 75% suffered from mild injuries.

5.3 Complicated Injuries

Armored cavalry troops and navy troops, which rely on their respective cabin spaces in combat, suffer from much higher rates of blast injury, burns, blunt trauma, and composite injuries than infantries. Statistics of blast injuries in British armored vehicle passengers during World War II indicate that 75% had fragment/shrapnel injuries. Of the tanks hit, 39.7–70% were ignited, and 25% of riders were burned. Blast injury occurrence rate was 1–20%. Of the British tanks that carried burn victims, 66% were hit by armor-piercing shells, 25% by high-explosive anti-tanks, and 9% by anti-tank land mines, with an average of 0.3 person burned per tank struck. During battles in the Sino-Vietnamese War in 1979, 58% of struck Chinese tanks were hit by high-explosive anti-tanks, followed by explosive ammunitions (14.9%), and then anti-tank land mines (11.3%), with a burns occurrence rate of 8.4%. During the Fourth Arab–Israeli War in 1973, most of the Israeli tanks destroyed were hit by high-explosive anti-tanks fired by the Egyptian military. The occurrence rate of burns was 9.3%, and an average of 0.9 Israeli troops was burned for every tank struck.

Of the 12,067 injured troops of the U.S. Navy during World War II, 39.09% were injured from projectiles, 17% from burns, and 12% from blast waves. When the American destroyer USS *Cole* was damaged by suicide bombers in an inflatable boat. 17 sailors near the point of impact were killed, of which 14 died from blunt trauma and three were drowned. In 1987, the American frigate USS *Stark* was hit by two Exocet missiles fired by an Iraqi jet. Of the sailors killed in the incident, 45.9% died from fragmentation injuries, 35.1% from burns, 8.1% from fragmentation-burns composite injuries, 5.4% from inhalation injuries, and 2.7% from suffocation. During the Falklands War between Great Britain and Argentina, the British destroyer HMS *Sheffield* was hit by an Exocet missile. Of those killed onboard, 25% died from suffocation because of inhalation of smoke and dust, and of the 26 injured sailors, 24 had burns and smoke and dust inhalation injuries.

Outcomes of animal testing on destructive effects of weapons show that when new types of armor-piercing shells hit a tank, if tank armor has been penetrated, the cause of death of test sheep/goats was all fragmentation-blast composite injuries or fragmentation-burns composite injuries, and for animals that survived, they suffered from smoke inhalation injuries, fragmentation injuries, blast injuries, and acceleration injuries. If the shell hit but did not defeat the armor, test animals inside would suffer from impact and acceleration injuries, with 62.5% sheep/goats afflicted with varying degrees of dilated meningeal vessels and blood clot resulting from degeneration of nerve cells and mild cerebral edema as observed under microscope. Abnormal EEG waveforms were seen in 50% of the sheep/goats, 12.5% of the animals had left ventricular subintimal hemorrhage, and ECG T-wave abnormalities were seen in 12.5% of the sheep/goats. When a high-explosive weapon strikes an above-ground fortification, 160 test sheep/goats placed inside the cabin were injured or killed. Of which, 110 animals or 68.75% had blast injury, 101 animals or 63.13% had primary or secondary fragmentation injury, 15 animals or 9.38% had collision injury or crushing injury, 12 animals or 10.91% had burns, and 67 animals or 41.88% had two or more different types of blast injuries.

5.4 High Occurrence Rate of Bone Fracture and Visceral Injury

According to statistics on body parts injured among the 608 troops injured in U.S. armored vehicles hit by land mines during the Iraq War and War in Afghanistan, of the 152 casualties there were 2912 wounds, of which 53% were bone fractures, with the largest number of bone fractures inflicted on the head, followed by ribs/sternum, pelvis, and limbs, etc. in that order. Of the 456 survivors there were 1637 wounds, of which 53% were bone fractures, with the largest number of bone fractures inflicted on the feet and ankles, followed by shin bone/fibula, lumbar vertebrae, and upper limbs, in that order.

In 2000, when the American destroyer USS *Cole* was damaged on one side by suicide bombers in an inflatable boat, 92% of the crew killed in the cabins had fracture in long bone, 50% had pelvis fracture, 71% had spine fracture, 86% had skull fracture, and 100% had rib fracture. Of the survivors, 15% had fracture in long bone, none had pelvis fracture, and the number of skull fracture, collar bone fracture, and spine fracture were all 3%, while rib fracture was 8%.

According to statistics on body parts injured among the troops injured in armored vehicles hit by land mines, head injury occurrence rate was highest in casualties, followed by chest, abdomen, and spinal cord in that order, while chest injury was most common among survivors, followed by abdominal and cranial injuries, in that order.

Outcomes of animal testing on conventional weapon effects show that in above-ground fortification struck by high-explosive weapon, injury to the thoracic cavity organs of test sheep/goats had the highest occurrence rate (60.44%), followed by head injury (36.26%) and abdominal cavity injury (30.8%). 20.9% of injured sheep/goats had chest-abdomen composite injuries (Table 31.4).

Table 31.4 The test of injured site of sheep/goats inside aboveground fortification subjected to explosive weapon strike

Injured site	Frequency of various types of animal injury	The occurrence frequency of various injuries in the corresponding injured site
Head injury	36.26%	–
Open brain injury	1.10%	3.03%
Closed brain injury	35.16%	96.97%
Chest trauma	60.44%	–
Open chest injury	5.49%	9.09%
Closed chest injury	54.95%	90.91%
Abdominal injuries	30.77%	–
Open abdominal injury	8.79%	28.57%
Closed abdominal injury	21.98%	71.43%

Note: Since two or more injuries may occur in the same animal simultaneously, the cumulative value of injury frequency of each part is greater than 100%

5.5 Prominent Closed Injuries and High Rate of Traumatic Brain Injury

During the Soviet–Afghan War (1979–1989), of the Soviet armored vehicles hit by land mines, occurrence rate of closed pulmonary contusion in personnel inside vehicle cabins was three times that of explosions in open space. During the Iraq War, of the American military vehicles hit by improvised explosive devices, occurrence rate of blunt impact injury was as high as 96%. Outcomes of animal testing on destructive effects of weapons show that in above-ground fortification struck by high-explosive weapon, test sheep/goats had obvious closed injuries, with occurrence rate three to 32 times more than that in open space injuries.

Traumatic brain injury (TBI) occurred frequently in victims injured during cabin explosions. During the War in Afghanistan, TBI existed in 50% of British military casualties from cabin explosions between November 2007 and August 2010, ranking first in terms of cause of death. During the Iraq War and War in Afghanistan, more than 60% of blast injury troops evacuated back to the U.S. had TBI, with around 80% being minor TBI (mTBI).

In order to understand mTBI injury features in personnel inside cabin due to explosion inside cabin, experiments were carried out by detonating small equivalent of high-energy explosives either inside simulated armored cabin or stuck to the exterior of bottom plate of cabin, then influences of explosion on physiology and cognitive behaviors of test rats inside the cabins were observed. Research results indicate that peak pressure, effective duration, peak impulse, and positive pressure effective duration in explosion inside cabin were all substantially higher than those in an open air explosion, with significant difference ($P < 0.01$). After an in-cabin explosion, cerebral blood flow to the parietal cortex in rats rapidly decreased compared to pre-explosion levels, reaching lowest point 1 h after injury, with a drop of roughly three times more than test rats in the open space explosion group (58.8% vs 18.9%). The levels recovered gradually, but still below pre-injury threshold 24 h after injury. Administering S-Nitrosoglutathione (GSNO) early on after injury could increase cerebral blood flow. Using triphenyltetrazolium chloride (TTC) rapid dyeing to evaluate brain injury, it was discovered that for the rats in the in-cabin explosion group, cerebral ischemia occurred earliest in the part of the brain facing the source of explosion, and cerebral ischemia onset was about 4 h earlier than rats in the open space explosion group. Also, area of cerebral ischemia gradually spread to surrounding tissues, with cerebral ischemia area expanding to largest 8 h after injury, which then gradually decreased but was still obviously larger than open space explosion group 72 h after injury. Water maze and shuttle box were used to observe the impacts of explosions on the cognitive behaviors of test rats. Experiments show that water maze escape latency and average shuttle box reaction time in rats in the in-cabin explosion group were significantly longer than those in the open space explosion group ($P < 0.05$), with the difference most significant 24 h after explosion ($P < 0.01$). The aforementioned results indicate that overpressure from in-cabin explosion is the injuring factor behind the mTBI suffered by personnel inside cabin, with the pathological injury features being impaired cerebral blood perfusion and progressive cerebral ischemia. Compared with explosion in an open environment, cerebral ischemia would occur earlier, last longer, and recover slower. Reversible learning and memory cognitive disorder might exist in injured personnel, being most obvious 24 h after injury.

To understand features of the process of how TBI is inflicted on cabin passengers by the process of high-acceleration changes of the cabin plate when an explosion occurs outside the cabin, a small equivalent of high-energy explosives is placed tightly against the bottom of simulated armored cabin plating, and detonated, then acceleration changes of steel seats in direct contact with cabin bottom and rats positioned in sitting posture on those seats were observed. Experimenters saw that after the explosion there was no obvious deformation of the cabin bottom plating, but acceleration upward of 1000 g was recorded in steel seats in direct contact with cabin bottom that sustained for several milliseconds. After the explosion, rats positioned in sitting posture on those seats had no anatomical damage as far as the eyes can see, but 6 h after injury, some neurons in the spinal cord anterior horn, cerebral cortex, pyramidal cells in

hippocampus, and Purkinje cells in cerebellum exhibited pathological changes of degeneration and necrosis when viewed under optical and electron microscopes. Rats inside the cabin after the explosion were subjected to water maze test, and results indicate that the rats' learning and cognitive abilities declined, most obviously 24 h after the explosion. If the seats were suspended in mid-air to interrupt the effects of the acceleration of cabin plating on the rats, optical microscopy and electron microscopy reveal that cerebral and spinal cord neuron pathological changes, spatial learning and memory dysfunctions and other issues obviously lessened compared with rats positioned in seats in direct contact with cabin plating. The research outcomes stated above demonstrate that when an explosion occurs outside the cabin, physical contact with high-acceleration deformation of cabin plating could induce minor TBI in passengers and transient cognitive dysfunction.

6 Treatment Principles and Techniques for Cabin Blast Injury

To improve the cabin blast injury treatment levels of the Chinese military and standardize cabin blast injury treatment procedures and techniques, in 2017 the Logistics Support Department of the Central Military Commission authorized the issuance of the People's Republic of China national military standard *Cabin Blast Injury Mechanism Standard* (GJB9012-2017). Said standard was compiled by this chapter's author as lead author, with contributions and participations from more than 20 experts and professors with the Field Surgery Research Institute (currently Army Unique Medical Center) of the third Army Medical University Hospital of the People's Liberation Army. During the formulation of said standard, members of the "standard formulation group" carried out in-depth studies on cabin blast injury features. In accordance with the Chinese military wartime treatment system and mission requirements, proven cabin blast injury treatment techniques from both Chinese and foreign militaries were consolidated, and after evaluation by the Chinese Military Trauma Special Commission and application by the military, said technical standard was formed.

Said standard is applicable to organizations and personnel at various levels involved with the battlefield/on-site first-aid, emergency treatment, and early treatment of combat cabin blast injuries in cabins like armored vehicle, vessel, tunnel, and fortification during wartime. The standard also serves as a reference for treatment of blast injuries in enclosed environments such as building, bus, subway, and civilian vessel before and after arrival in hospital during peacetime. Said standard is not applicable for treatment of bodily harms caused by chemical, biological, or radioactive agents or substances released in cabin explosion. The following section outlines the main cabin blast injury treatment principles and techniques stipulated in said standard.

6.1 In-cabin Emergency Treatment

This is a part of the emergency treatment segment in the battlefield/on-site combat injury treatment system of the Chinese military. The interior environment of cabin after explosion is a dangerous place, and victims should be evacuated from the hazardous space as soon as possible. In-cabin emergency treatment focuses on controlling fatal bleeding of limbs using tourniquet.

6.1.1 Searching

Rescuers should gain knowledge about type of in-cabin explosion, cabin structure, and victim distribution before entering the cabin, then assess the damage extent and safety level of cabin, and choose and confirm the appropriate time, method for cabin search and rescue, and personal protection measures. Stay low when entering a smoke-filled cabin for search and rescue. When extinguishing fire on clothes of victims, do not use plastic cloth or chemical fiber and blended fabric to cover the fire. Rescuers should assess victim's vital signs immediately upon discovery, identify life threats rapidly such as major bleeding of the limbs, or suffocation, and adopt protective measures to prevent victims from suffering further injury.

6.1.2 Emergency Treatment

The interior environment of cabin after explosion is a dangerous place, and victims should be evacuated from the hazardous space as soon as possible, so as to avoid emergency treatment procedures that require spending extensive period in the cabin. If the post-explosion cabin environment is relatively safe, early-stage emergency treatment may be performed inside.

Rescuers should use tourniquet to control ruptured large vessel, traumatic amputation, large area of soft tissue avulsion, and other fatal bleeding of the limbs. Tourniquet should be applied at the part of the wound closer to the heart (5–8 cm), and in exigent situations the tourniquet may be applied on the surface of clothing near the wound. When using belt, cloth strap, gauze strap of other items as temporary tourniquet, width of item should be no less than 5 cm. Rescuers should swiftly clean out any blood clot, foreign substance, or secretion blocking the oral and nasal cavities and upper respiratory tract of the victim, and position victim on his or her side, or with his or her head tilted to one side. Rescuers should relocate victim to a safe area outside the cabin by sup-

porting, propping up, carrying on back, carrying on arms, dragging, lifting or carrying on stretcher. When moving victims with head, neck, or back blunt trauma injury, rescuers should stabilize the person's head and neck, keep the his or her head in a straight line with the long axis of the spine so as to minimize movement of the spine.

6.2 Emergency Treatment Outside of Cabin

This is a part of the emergency treatment segment in the battlefield/on-site combat injury treatment system of the Chinese military. Rescuers should perform emergency treatment procedures in a safe area outside of cabin, quickly assess injury level, categorize injuries, prioritize treatment of life-threatening injuries, and pay attention to special treatment requirements for different injuries.

6.2.1 Injury Level Assessment

Rescuers should assess the victim's ventilation, respiration, and circulation statuses, ascertain the victim's cognitive level through yelling, check eye movement and fixation, and responses to pain and stimulation, and quickly identify life-threatening injuries.

Rescuers should use sphygmomanometer or blood pressure gauge to measure central arterial blood pressure. When unable to use sphygmomanometer or blood pressure gauge, make an approximate judgment by touching peripheral artery pulses with hand. If carotid artery, femoral artery, or radial artery pulse can be accessed, systolic blood pressure (SBP) should, respectively, be more than 60–70 mmHg, 70–80 mmHg, and 80–90 mmHg. Determine the injuring factors in the cabin explosion, and categorize the injuries.

6.2.2 Open Up Airways

For unconscious victims, or those already suffering or could suffer from airway obstruction, tilt the person's head up or lift the person's chin to open up his or her airway. Thereafter, if a mouth or nose breathing tube is inserted to help an unconscious victim breathe, lie the person on his or her side (recovery position). For a conscious victim using nose breathing tube, position the person in any position favorable to keeping the airway unobstructed. Do not tilt head and lift chin of victim if he or she is suspected to have neck injury or serious head injury, and do not use nasal breathing tube if victim has maxillofacial trauma or exhibits symptoms of skull fracture such as cerebrospinal fluid rhinorrhea or cerebrospinal fluid otorrhea.

After the treatment procedures outlined above, if airway obstruction symptoms still did not ameliorate, consider the options of cricothyrotomy or tracheostomy, and if conditions permit, use laryngeal mask, trachea and esophagus dual pipe, or trachea pipe.

6.2.3 Maintain Respiration

Rescuers should use airtight wraps to seal any open or sucking wound on the chest, apply pressure with bandage, pectoral girdle, and cotton pad to stabilize thoracic wall of victims with chest injury, so as to mitigate abnormal breathing movement.

If victim with unobstructed airway exhibits progressive respiratory distress, perform needle decompression immediately if tension pneumothorax is suspected. Needle decompression puncture site should be at the space of the second rib at the midclavicular line on the side of injury, or in between the fourth rib and fifth rib at the anterior axillary line. If there is still no improvement after needle decompression, or if hospital evacuation is expected to take a rather long time, insert breathing catheter or set up closed thoracic drainage. When conditions permit, oxygen inhalation should be provided to patients with saturation of pulse oximetry (SpO_2) < 90%, unconsciousness, shock, chest injury, serious head injury, or inhalation injury.

6.2.4 Control Bleeding

Rescuers should check the tourniquet in use, and switch to pressure dressing for non-lethal bleeding. If distal pulse of limb with tourniquet is still accessible, reapply the tourniquet, or add another tourniquet near the existing tourniquet on the end near the heart until the distal pulse disappears. If tourniquet applied to forearm or calf could not control bleeding, switch tourniquet to upper arm or thigh, and remember to record time of tourniquet application. Use of tourniquet should not exceed 2 h, and during the span of application the tourniquet must remain tight. If tourniquet is used for more than 2 h, it may be loosened cautiously to facilitate inspection of blood loss control. If major bleeding occurs again, reapply the tourniquet immediately. Loosening tourniquet on patients with shock should only be undertaken after ascertaining efficacy of fluid resuscitation. Tourniquet that has been applied for 6 h or more should be removed at medical care organization with amputation surgery capability.

Rescuers should use hemostatic agent, hemostatic wound dressing, or pressure bandaging to bandage or fill up and control bleeding in areas like the torso, neck, groin, and armpit, or apply pressure with hand or finger. If the aforesaid methods are futile, try inserting balloon catheter into wound and expanding balloon to apply pressure and stop bleeding.

Dress and stabilize fractured pelvis to reduce bleeding. Using hemostat forceps or ligature to stop bleeding should only be utilized if other measures are ineffective and do not use hemostat forceps without proper consideration. Medical anti-shock trousers may be used to control large soft tissue injury of lower limb bleeding or the pelvis.

6.2.5 Dressing

Rescuers should use sterile dressing to dress wounds. For exposed brain tissue and intestine protruding from the abdominal bowel, do not put them back in the cavity, instead use protective dressing. Airtight dressing should be applied to seal any open or sucking wound on the chest, and breathing status should be closely observed. Rescuers should perform needle decompression immediately if signs of tension pneumothorax are observed. When dressing limbs, keep fingers and toes exposed to facilitate observation, and beware of excessively tight dressing that overly restrict blood flow to limbs. For foreign substances lodged relatively deeply in the body, do not remove them without full consideration, instead keep and stabilize them in their place and then dress the wound.

6.2.6 Fixation

Rescuers may use standard plywood boards, expedient devices, or other materials for over-articular fixation of fractured long bones, injury to major joints, limb crushing injury, and large-area soft tissues injury. Prior to fixation, inspect limb circulation and nervous function status, and fixation should be fastened to a tightness level just enough to feel distal pulse. For those with spine or spinal cord injury, the victim's spine should be stabilized. When applying fixation to a patient in supine position, ensure that his or her cervical spine and lumbar spine remain at natural physiological curvatures. Use objects like triangular bandage or pelvic fixation band for fixation of fractured pelvis.

6.2.7 Cardiopulmonary Resuscitation

For patients with respiratory or cardiac arrest due to suffocation, electric shock, low temperature, poisoning, or other factors, carry out cardiopulmonary resuscitation right away. For patients with blast injury or penetration wound no vital sign, it is not necessary to perform cardiopulmonary resuscitation.

6.2.8 Hemorrhagic Shock Fluid Resuscitation

Fluid replenishment by oral means may be administered for conscious patients that are able to swallow. For patients with the following conditions, it would be advisable to administer intravenous infusion 1 h after injury:

1. No noticeable head wound but exhibits mental status changes and/or weakened or absent pulsation of the radial artery.
2. Systolic blood pressure (SBP) < (80–90) mmHg.

Use 18 G intravenous infusion needle or detaining needle to establish venous access. If venipuncture is difficult, use bone marrow infusion needle to puncture sternum, shin bone, near end of humerus, and other cancellous bone locations to establish intraosseous infusion access. Intraosseous needle should not remain inside body for more than 24 h, and efforts should be made to switch to intravenous infusion as soon as possible. Other than hypertonic fluids, all fluids and medications delivered through intravenous infusion may also be administered via intraosseous infusion.

Delivered fluid should be any crystalline fluid or colloidal fluid available on-site. Colloidal fluids are preferable, such as hydroxyethyl starch, dextran, and gelatin, followed by crystalline fluids like Ringer's lactate solution and other balanced salt compound solution. Intravenous infusion rate should be 500 ml/(15–20 min). If conditions permit, use whole blood or other blood products.

Adopt a low-pressure fluid resuscitation strategy, check patient after every 500 ml of delivered fluid, and stop the infusion process when patient reaches one or more of the following conditions:

1. Patient's consciousness improves (can be awaken or/and lift his or her own head);
2. Apical-radial pulse can be felt;
3. Systolic blood pressure (SBP) 80–90 mmHg;
4. Mean arterial pressure (MAP) 50–60 mmHg.

Low-pressure fluid resuscitation should not be over 90 min, and blood loss should be stopped within this duration. Norepinephrine, dopamine, and other vasoactive drugs, as well as inotropic agents, should only be used when low blood pressure persists under the premise of controlled blood loss and sufficient fluid replenishment.

6.2.9 Analgesia

For victims who have not lost combat capacity, provide orally ingested non-steroidal anti-inflammatory drugs such as meloxicam (mobic) tablets, acetaminophen (paracetamol), etc.

For victims who could no longer reengage in combat and suffer from serious pain but do not exhibit shock or respiratory distress, use opioid analgesics, preferably morphine administered via intravenous or intraosseous infusion, and administer again after 10 min if necessary. Promethazine is synergistic with opioids in terms of pain relief, nausea, and vomit alleviation.

For victims that could no longer reengage in combat and already showed or might suffer from shock or respiratory distress, administer 50 mg of ketamine via intramuscular injection, and administer again after 30 min if necessary. Or administer 50 mg of ketamine via intravenous or intraosseous infusion, and administer again after 20 min if necessary, until pain subsides or when victim exhibits nystagmus.

When applying opioid analgesics or ketamine, pay close attention to victim's mental state and circulatory and respira-

tory conditions, and apply symptomatic treatments should the need arises. Naloxone administered via intravenous or intramuscular infusion can antagonize the side effect of suppressed breathing caused by opioid painkillers.

6.2.10 Application of Prophylactic Antibiotics

For victims that are expected to be delivered to brigade/division-level or equivalent medical care institution within 3 h after injury, antibiotics may be applied.

For victims that are not expected to arrive at medical care institution within 3 h after injury, or if evacuation might be delayed, the following antibiotics are recommended: Oral ingestion of 500 mg of levofloxacin or 400 mg of moxifloxacin for patients with shock or those that cannot ingest orally, administer 1–2 g of ceftriaxone every 24 h or 1–2 g of cefazolin every 6–8 h by way of intravenous therapy or intramuscular injection.

6.2.11 Maintain Body Temperature

It is necessary to minimize exposure of the victim's body, remove any wet clothing, cover victim with thermal reflective blanket, or other insulation material. When conditions permit, heat up IV fluid to 40–42 °C.

6.2.12 Outside Cabin First-Aid for Several Types of Cabin Blast Injuries

Blast Injuries

Victims with the following signs and symptoms may possibly have blast injury: ruptured eardrum, bleeding fluid from the external auditory canal, or bloody foam discharge from the mouth or nose; in shock but without obvious external wound; chest pain, breathing difficulty, hemoptysis, abdominal pain, bloody urine, etc.

Rescuers should keep victim's airways unobstructed; decompression measures should be performed promptly for those showing symptoms of tension pneumothorax; after being provided with hyperbaric oxygen therapy, saturation of pulse oximetry (SpO_2) still below 90% after oxygen inhalation is an indication of severe injury and unfavorable prognosis.

When victim exhibits signs or symptoms of arterial air embolism, position the person in a left decubitus position with head low posture, then provide pure oxygen for inhalation via face mask or airway breathing tube. When conditions permit, swiftly send victim to hyperbaric oxygen chamber for treatment. For victims with abdominal blast injury indicated by signs or symptoms such as abdominal pain, nausea, vomiting, or peritoneal irritation, do not replenish fluids or foods by oral ingestion. When paralytic ileus occurs, use nasogastric tube to relieve pressure in the gastrointestinal tract. Beware of excessive fluid delivery, which could worsen pathological progression of cardiac and pulmonary blast injuries.

Burns and Inhalation Injuries

Use clean and cool water to wash surface of burn wounds, and after rinsing apply burn dressing or dry and clean cloth to cover and protect surface of wounds. Take measures to maintain victim body temperature. For victims with burns covering less than 20% of body surface area, oral ingestion of fluid is acceptable, and burn formula fluid (100 ml containing 0.3 g of sodium chloride, 0.15 g of sodium bicarbonate, 0.005 g of phenobarbital, and a suitable quantity of sugar) is recommended.

For victims with burns covering 20% or more of body surface area, administer crystalloid solution (Ringer's lactate solution or normal saline). For an adult weighing between 40–80 kg, fluid input rate should be (percentage of body surface area burned) × 10 ml/h.

For victims with burns around the face and neck area, sputum containing carbon particles, scratchy or loss of voice, respiratory distress, or wheezing, dry rales, twirls, or other inhalation injury signs and symptoms heard from auscultation of the lungs, pay close attention to his or her ventilation status. If signs and symptoms of airway obstruction appear, quickly undertake cricothyrotomy or endotracheal intubation, and provide hyperbaric oxygen therapy to maintain saturation of pulse oximetry (SpO_2) >92%. Carry out pain relieve measures.

Crush Injuries

Before relieving victim from crush situation, at least 1000 ml of normal saline should be administered via intravenous therapy at rate of 1000–1500 ml/h. If IV is not possible, before relieving victim from crush situation, wrap tourniquet around the crushed limb on the section closer to the heart, not to be removed until completion of fluid resuscitation. Continue intravenous therapy after removing victim from crush situation, and beware not to administer liquid containing potassium or lactate.

Alkalized urine; administer sodium bicarbonate via intravenous infusion, with total volume of 200–300 mmol, equating to 300–500 ml of 5% sodium bicarbonate solution, on day one. Ensure stabilization and fixation of crushed and injured limb and/or pelvis, and avoid using anti-shock trousers for fixation or dressing using elastic bandage with pressure. Administer antibiotics to prevent infection, but refrain from using drugs that are detrimental to kidney functions. Apply pain relieve medications and sedatives.

Combined Injuries

Prioritize the treatment of combined injuries. Choose the appropriate fluid resuscitation strategy in accordance with

the main injuring factors. When hemorrhagic shock coexists with other types of shock, prioritize fluid resuscitation for hemorrhagic shock.

Impact Injuries

Stabilize and fix head and spine when moving victims with head, neck, or back injuries. For fluid resuscitation of victims with impact injuries to the torso, avoid excessive fluid delivery to prevent worsening injuries to internal organs. For fluid resuscitation of victims with neurogenic shock of spinal cord injury, use crystalloid solution. Resuscitation should achieve systolic blood pressure (SBP) ≥110 mmHg or mean arterial pressure (MAP) ≥80 mmHg. When necessary, after delivery of 2000–3000 ml of fluid, apply norepinephrine, dopamine, or other vasoactive agents to adjust vascular tension.

Eye Injuries

Check victim's eyesight, and for those with impaired vision, evacuate to specialized medical institution for treatment as soon as possible. Use hard goggles to protect open wounds of eyeballs, and refrain from applying any pressure on injured eyes. For foreign objects in conjunctiva or cornea, rinse them out or undertake surface anesthesia and then use sterile wet cotton swabs to remove foreign objects.

Traumatic Brain Injury (TBI)

Assess vital signs and cognitive level of victims with head injury, observe the size of his or her pupils, and monitor the person's arterial blood pressure (AP) and saturation of pulse oximetry (SpO_2).

For victims suffering from hemorrhagic shock, it would be ideal to quickly administer 3% or 5% hypertonic saline at a rate of 250 ml/15 min using intravenous therapy, with total volume not to exceed 500 ml. Then use isotonic crystalline or colloid solution. Resuscitation targets are systolic blood pressure (SBP) >90 mmHg and mean arterial pressure (MAP) >60 mmHg. Keep airway unobstructed, and provide hyperbaric oxygen therapy until saturation of pulse oximetry (SpO_2) >92%.

For victims exhibiting uncoordinated movement, fixed or dilated pupil(s) on one side or both sides, accompanied by aggravated cognitive dysfunction and other symptoms of high cranial pressure, it would be advisable to quickly administer hypertonic saline at a rate of 250 ml/15 min using intravenous therapy, and maintain continuous IV at a rate of 50–100 ml/h during the course of evacuation, but total volume should not surpass 500 ml. If hypertonic saline is unavailable, mannitol may also be delivered via intravenous therapy for victims with urine. Tilt victim head at a 30° angle, and adopt measures to sedate victim and lower his/her body temperature. As a preventative measure, use antibiotics to avoid infections.

6.3 Initial Surgical Resuscitation

This is considered an early-stage treatment technique and process in the military medicine system of the Chinese military. Complete life-saving and limb-preserving damage control surgery or emergency surgery at the division- or brigade-level medical institution, carry out resuscitation and rewarming, stabilize the patient's physiological state and prevent the onset of infection to facilitate the safe and smooth evacuation of the victim.

6.3.1 Injury Condition Evaluation

Conduct physical examination and any necessary supplemental testing, evaluate the injury levels and scopes of the victim's heart and circulatory system, chest and respiratory system, abdomen, spine, head, pelvis, limbs, arteries and nerves, monitor his or her physiological functions, identify life-threatening main injuries and those that affect limb functions, and determine the treatment measures needed and application sequence.

6.3.2 Damage Control Surgery

1. For the following types of severely injured victims, employ damage control surgery to control internal organ bleeding, bleeding from injury to large vessels and infections from substances flowing out of ruptured stomach or intestine, rectify low body temperature, acidosis, and coagulopathy, then relocate victim or conduct definitive surgery after his or her condition stabilizes.
   a. Victims with injuries involving multiple body parts, multiple organs, or large vessels, such as those with injuries to multiple organs in the abdomen coupled with large vessel injury; retroperitoneal vascular injury; pancreas and duodenum injury; open pelvic fracture or rupture of pelvic hematoma; major vessel injury to the chest or severe laceration to the lung; multiple traumatic amputations, etc.
   b. Victims whose systolic blood pressure (SBP) remains below 90 mmHg after fluid resuscitation and with unstable hemodynamic status.
   c. Victims with severe injuries at or near one or two of the following indicators: core temperature <34 °C; blood pH <7.25; blood coagulation international normalized ratio (INR) >1.4 or prothrombin time (PT) >1.9 and/or activated partial thromboplastin time (APTT) >60 s.
   d. When there are many victims.

2. Abdominal damage control surgery: After entering the abdomen, control abdominal bleeding by directly applying pressure with hand or dressing. If there is obvious abdominal arterial bleeding, press down on the abdominal aorta at the diaphragmatic foramen with hand, and after temporarily restricting blood flow, control bleeding from ruptured vessel using methods such as ligation, abdominal wall repair, temporary vascular bypass, extravascular balloon compression, etc. Stuffing dressing into abdominal cavity can effectively control bleeding in areas like the liver, pelvis, and retroperitoneum, and stuffed dressing should be removed within 24–48 h. If blood pressure does not clearly restore after stuffing dressing, adopt measures such as suture, excision, and repair to control bleeding from ruptured solid organs. Use techniques and devices like clipping together, ligation, u-shaped staples, repairing, and excision to control contents flowing out from openings in stomach or intestine. For extraperitoneal rectal injury, administer external colon treatment or colostomy. For injuries to bile duct or pancreatic duct, external drainage may be performed. For injuries to distal superior mesenteric artery pancreas, carry out pancreatectomy or drainage. For proximal superior mesenteric artery injury, debridement and closed drainage are feasible. Temporarily closing abdominal cavity with negative pressure dressing is recommended or close wound using materials like polypropylene mesh, pieces of IV bag, or artificial patch, but do not use towel forceps to clamp the wound close, stitching instrument, or continuous stitching technique to close the abdominal cavity.
3. Thoracic damage control surgery: To control major bleeding from lung injury, utilize cutting stapler to perform wedge pneumonectomy, or use long clamps to fasten the two ends of a lung wound and then open up the wound passageway to gain a direct sight for ligation to stop bleeding and control air leakage. For severely injured pulmonary lobe, pulmonary lobectomy may be performed. To control bleeding from ruptured large vessel, utilize temporary vasculature, or Fogarty balloon catheter insertion to block blood flow. If situation is particularly dire, use temporary hilar ligation technique. When the airway is injured, inserting tracheal catheter through the wound is a viable option. For victims with severe bronchus rupture or injury, it would be advisable to conduct pulmonary lobectomy of the involved lobe or pneumonectomy of entire lung on the affected side. For injuries of the esophagus, it would be feasible to perform diversion and drainage, while definitive repair is not suggested. Use continuous stitching to close thoracic wall, do not use towel forceps to clamp the wound close.
4. Other damage control surgeries: For repairable large vessel wound on limb, it would be wise to conduct temporary vessel bypass 3 h after injury, including vascular wound exploration, thrombus resection, restoration of distal blood supply, and limb fasciotomy decompression, among other steps, and damaged vessel repair should be completed within 12 h of injury. If conditions do not permit such a method, temporary ligation may also be employed, and finish damaged vessel repair within 4 h of injury. To control bleeding of fractured pelvis, ideal procedures are extraperitoneal pelvic packing conducted with external fixation or suprapubic incision and utilize vessel embolization when the option is viable. Internal iliac artery ligation should be carried out only when other methods have failed. For victims suffering from traumatic brain injury with continually worsening symptoms of cerebral hernia, it would be advisable to perform cranial decompression or cranial decompression.

6.3.3 Resuscitation and Rewarming

Fluid Resuscitation

When bleeding is still uncontrolled, low-pressure fluid resuscitation should be adopted. After bleeding is controlled, resuscitation should achieve all the following objectives: (1) systolic blood pressure (SBP) >110–120 mmHg, and mean arterial pressure (MAP) >65–70 mmHg; (2) urine volume larger than 0.5 ml/(kg h); and (3) base excess (BE) > −2 mmol/or lactic acid <2 mmol/L. When conditions permit, it would be advisable to carry out transfusion of fresh whole blood or blood components with input ratio of fresh frozen plasma to suspended red blood cells, for victims with hemorrhagic shock and meeting any one of the following circumstances: (4) severely injured patients that have undergone damage control surgery; (5) bleeding has not been put under control for body parts such as the torso, neck, armpit, and groin; hemoglobin (Hb) <60 g/L or hematocrit value (Hct) <25%; or (7) having already delivered 3 L crystalloid solution during the course of low-pressure fluid resuscitation.

For patients intended for blood transfusion, within 3 h of injury it is recommended to administer tranexamic acid 1 g/100 ml normal saline or balanced salt solution via non-transfusion venous access for more than 10 min. When necessary, repeat the step after 8 h. After bleeding is controlled and active fluid resuscitation, if blood pH remains <7.20, it is necessary to apply alkaline drugs such as sodium bicarbonate or tris-hydroxymethyl aminomethane (THAM) to rectify metabolic acidosis. Monitor urine volume and blood electrolytes. When conditions permit examine victim's liver and renal functions and undertake corresponding measures to correct any dysfunctions.

Breathing Support

It would be advisable to provide oxygen inhalation via face mask, with the goal of maintaining arterial oxygen saturation (SaO_2) >92%.

In any one of the following conditions, intubation and mechanical assisted ventilation should be performed: (1) airway obstruction; (2) apnea; (3) excessive breathing work or shortness of breath with signs of respiratory failure; (4) decreased level of consciousness with Glasgow Coma Scale (GCS) ≤ 8; (5) arterial oxygen saturation (SaO_2) < 90% and arterial partial pressure of oxygen in arterial blood (PaO_2) <60 mmHg when the fraction of inspired oxygen (FiO_2) is > 50%; (6) arterial partial pressure of carbon dioxide ($PaCO_2$) > 60 mmHg.

When applying mechanical ventilation to victims with acute lung injuries, adopt a lung protective ventilation mode with low-moisture air of 5–8 ml/kg, restricted peak airway pressure/plateau pressure (≤35 cmH_2O), and best positive end-expiratory pressure of 5–10 cmH_2O.

Rewarming

Rewarm core temperature to higher than 34 °C. When core temperature falls below 32 °C, damage control surgery shall not be performed.

The following rewarming measures can be taken: (1) coverage with quilt/blanket to raise the ambient temperature; (2) inhalation of warmed and humidified oxygen and input of warmed fluid; (3) irrigation of the stomach, colon, and bladder with 37–39 °C warm water; (4) flushing of the chest or abdominal cavity with warm water during surgery, etc.

6.3.4 Emergency Surgery

To keep airway unobstructed, trachea intubation or tracheotomy may be carried out. For hemothorax or pneumothorax, closed thoracic drainage is feasible. For open pneumothorax, close the pleural cavity. For injuries to organ in the chest or abdomen, or vascular injuries to the torso or limbs, adopt exploration, repair, excision, anastomosis, ligation, and other surgical operations. For limbs suffering from compartment syndrome, perform osteo-fasciotomy. For fractured long bone, use external fixator for fixation, or use splint and plaster. For traumatic amputation, carry out bone stump amputation.

6.3.5 Infection Prevention

Debridement should be performed as soon as possible, especially within 6 h after injury and delay stitching the wound. If necrotic tissues are not thoroughly removed, it would be advisable to conduct debridement again 24–48 h after injury. For a wound already infected, keep the wound open and drain sufficiently.

Use plenty of normal saline, sterile water, or drinkable water to rinse wound until the wound is clean.

Non-operative treatment is appropriate for penetrating wounds that meet the following conditions: (1) penetrating wounds that only injure the skin and muscles; (2) the maximum diameter of the exit or entrance is less than 2 cm; (3) no large vessels, nerve damage, or fracture; (4) no injury to the pleura and peritoneum; (5) light contamination.

Non-surgical treatments include disinfection of skin, removing contaminant from wound, rinsing wound, dressing wound after drainage, and bodywide administration of antibiotics. For patients receiving non-surgical treatments, keep a close eye on wounds, and if there are signs of infection, provide corresponding treatment in accordance with infected wound procedures. When conditions permit, select the right kind of antibiotics to treat an infected wound based on bacterial culture of secretions and drug sensitivity test results.

Targeted preventative and treatment measures should be adopted when dealing with tetanus and gas gangrene.

6.3.6 Key Points of Initial Surgical Resuscitation for Several Types of Cabin Blast Injuries

Blast Injuries

For victims suspected of suffering from blast injuries to organs in the chest or abdomen, it is suggested to conduct X-ray scan of injured area, focused assessment with sonography in trauma (FAST), and regular ultrasound examination. Also, monitor arterial blood pressure (AP) and saturation of pulse oximetry (SpO_2), as well as arterial blood gases if the option is available. For those suffering from breathing difficulty, perform trachea intubation or tracheotomy, clean out secretions inside the trachea, provide mechanical ventilation under lung protective ventilation mode, and conduct closed thoracic drainage for hemothorax or pneumothorax. When performing surgery on a blast lung injury victim, it would be advisable to adopt local block or subarachnoid block anesthesia. Low airway pressure should be maintained when applying inhalational anesthetic so as to prevent the occurrence of pneumothorax or air embolism. For victims with ruptured organs from blast injuries to the abdominal area, promptly carry out exploratory laparotomy.

Burns and Inhalation Injuries

Provide hyperbaric oxygen therapy to victims with burns covering 20% or more of body surface area, and/or those with inhalation injuries. When using mechanical ventilation with victims of inhalation injuries, utilize the lung protective ventilation mode.

Volume of fluid delivery during the first 24 h after injury: (Burn area × 100 ml) ± 1000 ml, plus daily physiological

required volume of 2000 ml (5% glucose), colloidal solution (5% albumin liquid, plasma, hydroxyethyl starch, gelatin, etc.), and crystalloid solution (Lactated Ringer Solution or other compound balanced salt solution) with a ratio of 1:2, and alternate between crystalloid solution and colloidal solution. Fifty percentage of total volume of fluid should be delivered within the first 8 h after injury, with the remaining 50% to be completed in the next 16 h. If fluid has already been delivered, subtract the delivered volume from total volume to be delivered. In the second 24 h, the colloidal solution and electrolyte fluid volume is one-half of the actual volume delivered during the first 24 h.

Fluid delivered should be able to maintain urine volume of 30–50 ml/h. If urine volume remains below said figure for one to two consecutive hours, accelerate balanced salt solution input rate by 25%; and conversely, if urine volume surpasses said figure, slow down fluid input rate by 25%. Other indicators of effective fluid resuscitation include cognitive clarity and calmness, warming of limbs, steady breathing, systolic blood pressure (SBP) >100 mmHg, heart rate of 100–130 bpm, and elevated base excess (BE). For circumferential eschar burns to the chest that seriously affect respiratory function, perform escharotomy by incising to the fascia along the midaxillary line and costal margin, then use povidone iodine yarn to fill up the cut. Rinse and disinfect wound surface, drain out vesicle fluid, then use povidone iodine yarn to cover up wound or leave wound exposed, and remember to keep patient warm.

Crush Injuries

For victims removed from crushing situation, fluid delivery should maintain urine volume of more than 100 ml/h until urine turns clear. Deliver sodium bicarbonate and maintain urine pH > 6.5. If the patient's urine volume does not reach 300 ml/h, administer mannitol solution via intravenous infusion at a volume of 1–2 g/(kg day) and at a rate slower than 5 g/h. Maintain water and electrolyte balance, and if serum potassium exceeds 5.5 mmol/L coupled with obvious electrocardiographic abnormality, it would be advisable to take the following steps to reduce serum potassium while maintaining continual serum potassium and ECG monitoring: (1) intravenous therapy of calcium gluconate or calcium chloride; (2) sustained intravenous infusion of sodium bicarbonate and glucose-regular insulin; (3) oral ingestion of cation exchange resin (sodium polystyrene sulfonate); and (4) intravenous therapy of furosemide for patients with urine.

Surgery should remove inactivated muscle tissue, then dissect deep fascia longitudinally for decompression. Amputate limbs that have no chance of survival. Administer antibiotics to defend against infection.

Composite Injuries

When treating burns and perforation wounds, first and foremost ascertain that there are no concurrent blast injuries or collision injuries. If there are concurrent blast injuries, during treatment keep airway unobstructed, maintain victim respiration, avoid excessive fluid delivery, and be circumspect with the use of anti-shock trousers. If there are concurrent collision injuries, during treatment pay attention to the possibilities of closed thoracic and abdominal organ injury or fractured spine and long bone. It would be advisable to utilize external fixator for fixation of open limb fracture with burns.

Collision Injuries

For victims suspected of suffering from traumatic brain injury (TBI), injuries to organs in the chest or abdomen, injuries to spinal cord or spine, and bone fractures, conduct X-ray scan of injured area, focused assessment with sonography in trauma (FAST), regular ultrasound examination, electrocardiogram (ECG), and other supplementary examinations. Also, monitor arterial blood pressure (AP) and saturation of pulse oximetry (SpO_2), as well as arterial blood gases if the option is available.

Victims with contusion myocardiaque should lie in bed and inhale oxygen, while fluid input should be limited. Also, positive inotropic drugs (dopamine or dobutamine) should be administered to augment myocardial contractility. Perform pericardial puncture decompression to deal with cardiac tamponade caused by hemopericardium.

Eye Injuries

For victims with perforation wound to the face or serious loss of vision, eyeball structural damage, ocular proptosis, dyscoria and ocular movement disorder, evacuate to specialized medical institution for treatment as soon as possible, and it would be ideal to complete debridement and other surgical treatments within 6 h after injury. Administer antibiotics to defend against infection.

Traumatic Brain Injury

For head injury victims with perforation wound to the head, open skull fracture and Glasgow Coma Scale (GCS) score of ≤13, evacuate to specialized medical institution for treatment as soon as possible. For victims with Glasgow Coma Scale (GCS) score of 14–15, evacuate depending on the situation. For the physiological parameters of a victim being evacuated, maintain his or her systolic blood pressure (SBP) >90 mmHg, mean arterial pressure (MAP) >60 mmHg, saturation of pulse oximetry (SpO_2) >92%, and arterial partial pressure of oxygen (PaO_2) >80 mmHg. For

victims showing symptoms of cerebral hernia such as dilated pupil, elevated blood pressure, or bradycardia, it would be advisable to administer mannitol via intravenous infusion at a volume of 1 g/kg and a high input rate, and when necessary administer mannitol again after 4 h via intravenous infusion at a volume of 0.25 g/kg and a high input rate. However, do not administer mannitol to victims with hypovolemia, no urine or cardiac failure. For a patient that has unimproved or worsened symptoms of cerebral hernia even after undergoing dehydration treatment, carry out decompressive craniectomy if the option is viable. Keep the patient under monitoring and only relocate after his or her condition has stabilized. Administer antibiotics to protect again infection, and use drugs in the benzodiazepine or barbital category to prevent the onset of epilepsy in victims with cerebral perforation injuries.

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Part VI

Different Types of Blast Injuries

Nuclear Blast Injury

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1 Section One: Nuclear Weapons

1.1 Overview of Nuclear Weapons

The appearance of nuclear weapon completely changed the course and history of development of weaponry in the twentieth century and realized the leap of weapon development from hot weapons to nuclear weapons. The world has never seen any weapon as destructive as nuclear weapon. The course of the research and development of nuclear weapon was filled with twists and turns, interspersed with many tales of intrigue. During the turn of the twentieth century, progress in natural science advanced by leaps and bounds. The discovery of radiation and proposal of theory of relativity in the realm of physics formed the theoretical bedrock for the research and development of the atom bomb. At the same time, various scientists of eminence such as the Curies, Ernest Rutherford, and Enrico Fermi carried out their respective experiments, gradually fleshing out the theories behind atomic reaction. The theory of chain reaction meanwhile was the key that opened the doors to the atom bomb. It would not be long before the first atom bomb was forged.

Starting from the 1930s, nuclear weapon research and production picked up steam in nations such as Germany, Japan, the USA, the UK, the Soviet Union, and France. Germany was the first country to conduct nuclear weapon research and experiment. In December 1938, German scientists Otto Hahn and Friedrich Wilhelm Strassmann identified the phenomenon of nuclear fission in uranium after 6 years of study and grasped the basic method to induce fission of nucleus. In 1938, Otto Hahn succeeded in splitting the nucleus of uranium atom and shocked the global scientific community. In August 1938, Albert Einstein personally wrote to U.S. President Franklin Roosevelt and expounded the importance of the research and development of the atom bomb in details. In 1942, President Roosevelt decided to establish an atomic bomb research institution, codenamed the "Manhattan Project" and headquartered in New York City. This project involved an investment of some USD 2.2 billion and more than 500,000 personnel. The project was under the command of Major General Leslie Groves, University of Chicago, professor Arthur H. Compton was put in charge of preparation for fissile materials, Italy-born American scientist Enrico Fermi was responsible for manufacturing of nuclear reactor, and physicist J. Robert Oppenheimer was appointed the chief designer of the atomic bomb.

Under the directions of Enrico Fermi, the world's first nuclear reactor was completed at the University of Chicago in December 1942. However, it was a challenge to obtain uranium. After countless experiments, Fermi eventually discovered that plutonium is actually a better fissile material than uranium. That's why the Americans built three graphite-moderated, water-cooled reactors, and a reprocessing plant to produce plutonium. By 1945, the Americans had spent more than 2 billion US dollars and produced three atom bombs, respectively, codenamed, "Gadget," "Little Boy," and "Fat Man." At 5:24 a.m. on July 16th, 1945, the first ever nuclear bomb experiment in human history was carried out with a bomb placed on a 30 m-high tower located inside the "Trinity" test site in Alamogordo, New Mexico. "Gadget" was outfitted with a charge of 6.1 kg of plutonium or 22,000 TNT equivalents. During the experiment, the nuclear explosion created temperatures as high as tens of million degrees and pressure in the range of several tens of billion of atmospheres. The 30 m-high tower that held the bomb was melted into gas, and the explosion left a massive crater on the ground. The nuclear explosion created a mushroom cloud that towered over the land like a beacon of terror. Within a radius of 400 m, the desert sand melted into a yellow-greenish glass-like substance, and all animals within a radius of 1600 m were killed. The force of this atom bomb was nearly 20 times more powerful than scientists originally thought. In August 1945, the

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USA deployed two nuclear weapons against Japan at Hiroshima and Nagasaki, which accelerated World War II toward its conclusion. These two instances showcased the tremendous power of nuclear weapon and made nuclear weapon regarded as frightening monsters of terror.

A nuclear weapon uses its own sustainable nuclear fission or fusion–fission reaction to release immense energy instantaneously to generate an explosion powerful enough to cause serious and widespread annihilation. Common nuclear weapons include atom bomb, hydrogen bomb, and other specialized types of nuclear weapons such as neutron bomb, electromagnetic pulse bomb, and reduced residual radioactivity weapon. Nuclear weapon usually refers to the warhead and the shell holding it, sometimes also includes the attitude control system and penetration system. These are together colloquially called nuclear bomb. The nuclear warhead is the main explosive device and may simply be called nuclear device. The nuclear device is comprised of nuclear components, explosive components, fission reaction trigger (neutron generator), and other parts. The nuclear device and detonation control system comprise the nuclear warhead. The deployment or launch system of nuclear weapon is made up of the carrier, launch device, and other auxiliary equipment. Nuclear weapon development is currently in its third generation, with the first being atomic bomb, second is the hydrogen bomb, and the current third generation includes nuclear weapons with special purposes. Special purpose nuclear weapons either enhance or weaken one or some of the destructive elements of traditional nuclear weapon to satisfy different needs based on varying battlefield scenarios or objectives. Some examples include neutron bomb, reduced residual radioactivity weapon, salted bomb, etc. A neutron bomb is also called enhanced radiation weapon (ERW). A tactical nuclear weapon that utilizes nuclear fusion reaction to generate large amount of lethal neutron, but physical power of the blast itself is limited to it does not cause much damage to equipment and facilities. The neutron bomb produces less radioactive contamination than atom bomb, which is why it is regarded as a "clean" nuclear weapon. The neutron bomb is a major type of tactical nuclear weapon.

Nuclear weapons may come in various forms such as nuclear missiles (missiles with nuclear warheads), nuclear bombs, nuclear depth charges, nuclear bunker busters, nuclear torpedoes, nuclear artillery shells, and nuclear land mines, among others. Delivery system for nuclear weapons includes missile, electric motor, artillery, etc. Nuclear land mines do not require a delivery system. Nuclear weapons may be launched from aircraft, land-based vehicle, or vessels on or below the water surface. Nuclear weapons are increasingly miniaturized in order to meet demands in launch site mobility and striking multiple targets at longer distances. Based on combat usage, nuclear weapons may be sub-divided into strategic nuclear weapon or tactical nuclear weapon. Strategic nuclear weapons include ballistic missiles launched from land-based missile silos or nuclear submarines, long-distance airborne missiles, cruise missiles, and nuclear-powered cruise missiles. Tactical nuclear weapons include mid- and short-range missiles, cruise missiles, and nuclear-powered cruise missiles launched from land, sea, or air, as well as nuclear artillery, nuclear land mines, nuclear naval mines, and nuclear torpedoes, among others. The development trends in nuclear weapon include improving flexibility in usage through miniaturization, ammunition diversification, and adjustable yield, increasing destructiveness, raising accuracy, enhancing penetration capacity, development of multi-warhead technologies, and speeding up rapid response. Nuclear weapon, even when not directly deployed in actual warfare, still plays a crucial role as a significant threat in hi-tech and localized armed conflicts.

1.2 Principles of Nuclear Weapons

1.2.1 Nucleus and Changes Within Nucleus

Nucleus

A nucleus is comprised of proton, which has electric charge, and neutron, which does not have electric charge. Proton and neutron are collectively termed nucleon. In a neutral atom, the number of protons in its nucleus is equal to the number of electrons outside the nucleus. This is represented as nuclear charge number or atomic number, denoted by Z. The sum of protons and neutrons inside the nucleus is called the mass number, denoted by A. Thus, the number of neutrons inside the nucleus equals to A minus Z. If X is used to denote a certain element, then ${}^{A}_{Z}\mathrm{X}$ shows the nucleus composition of the element.

1. Element: Atoms with the same charge number in their nuclei are classified into an element.
2. Nuclide: Atoms with the exact same number of protons and neutrons are classified into a nuclide. Radioactive nuclides such as ^{32}P and ^{60}Co are called radionuclides.
3. Isotope: One element could include various nuclides, and they have the same number of protons in their nuclei but different numbers of neutrons. Since the different nuclides of the same element occupy the same position on the periodic table, they are called isotopes. For instance, ^{1}H, ^{2}H, and ^{3}H are different nuclides, but they all belong to the element hydrogen, thus they are isotopes. In addition, there is another important concept related to isotope: Isotope abundance refers to the percentage of atoms in the different isotopes naturally found in an element.
4. Isomer: If two nuclides have the same number of neutrons and same number of protons but different energy states, they are called isomers. For example, ^{99}Tc^{m} and ^{99}Tc are isomers.

5. Mirror nuclei: A pair of nuclei is classified as mirror nuclei if they have the same mass number, nuclear spin, and parity, but opposite numbers of protons and neutrons.

Mass, Conservation of Energy, and Nuclear Binding Energy

Mass Effect

A nucleus is comprised of neutrons and protons, but the mass of a nucleus is not the same as the sum of the mass of protons and mass of neutrons inside. Take $^{4}_{2}\mathrm{He}$ for example:

Mass of a helium atom M ($^{4}_{2}\mathrm{He}$) = 4.002603 u (unit for atomic mass)
Mass of a hydrogen M ($^{1}_{1}\mathrm{H}$) = 1.007825 u
Mass of neutron M (n) = 1.008665 u

The nucleus of helium ($^{4}_{2}\mathrm{He}$) is comprised of two protons and two neutrons, and when the masses of the two protons and two neutrons are added together:

$$2\times M\left(^{1}_{1}\mathrm{H}\right)+2\times M\left(\mathrm{n}\right)=2\times 1.007825\mathrm{u}+2\times 1.008665\mathrm{u}=4.032980\mathrm{u}$$

This does not equal to the atomic weight of helium ($^{4}_{2}\mathrm{He}$), and the difference is:

$$\Delta M=\left[2M\left(^{1}_{1}\mathrm{H}\right)+2M\left(\mathrm{n}\right)\right]-M\left(^{4}_{2}\mathrm{He}\right)=4.032980\mathrm{u}-4.002603\mathrm{u}=0.030377\mathrm{u}$$

The calculation above illustrates that when two neutrons and two protons form a helium nucleus, ΔM would be lost, as in 0.030377 u of mass. For all other nuclei, the above calculation can also be used to prove that the mass of a nucleus does not equal the sum of the mass of neutrons and mass of protons contained within. ΔM is defined as "mass defect."

Nuclear Potential

A nucleus is made up of nucleons (neutrons and protons), and between the nucleons inside a nucleus exists a special type of mutual power called "nuclear potential." Nuclear potential is characterized as a "short-distance" force, whether a nucleus is charged or not does not influence the effect of its nuclear potential, and nuclear potential may be saturated, as a nucleon only mutually affect neighboring nucleons and not all nucleons.

A nucleus is formed by nucleons that are mutually bonded together via nuclear potential. When nucleons form a nucleus, they must exert externally, as in release energy. Nuclear potential is more powerful than Coulomb force, and precisely because it is mostly an attractive force, that's why nuclear potential enables nucleons to overcome Coulomb force to combine into nucleus. Nuclear potential has a very short effective distance, approximately 10^{-15} m. We know that Coulomb force conversely is a long-range force, with its magnitude inversely proportional to the square of distance, while nuclear potential is a short-range force. When the distance between nucleons exceeds a certain very short distance, the force of nuclear potential disappears (effective range less than 3 fm).

In layman's terms, nuclear potential is a short-range but powerful mutual effect, and its main mechanism is attractive force.

Law of Mass and Energy Connection

Mass and energy are properties that simultaneously exist in a substance. Any substance with a certain mass must have a certain association with energy.

Assuming that E denotes energy (J), M denotes mass (kg), and C denotes the speed of light (3×10^{8} m/s), then:

$$E = MC^{2}$$

In any situation when there is a change in energy, there would always be a change in mass as well. Similarly, if there is any change in mass of the substance, correspondingly its energy would also change.

Binding Energy of Nucleus

The energy released when several nucleons bind together to form a nucleus is called binding energy of that nucleus. The higher the binding energy, the larger the energy released when nucleons bind together to form that nucleus, and the

tighter the bond that holds that nucleus together. The ratio between binding energy of a nucleus and its mass (as in the number of nucleons) is known as "average binding energy of nucleus."

For those nuclei with a mass in the middle range, each constituting nucleon has a higher average nuclear binding energy. Conversely, those nuclei with a heavier mass or those with a lighter mass, their constituting nucleons have relatively smaller average nuclear binding energy.

Therefore, when nuclei with heavy mass split into nuclei with medium mass, their nucleons would bind together more tightly in the relatively lighter nuclei, and in doing so could release a substantial amount of energy. When fusion occurs between two relatively light nuclei, the energy released would be even greater.

Changes in the Nucleus

Nuclear Decay

In 1896, while studying phosphorescence of uranium ore, Antoine Henri Becquerel discovered the uranium ore emits invisible rays that can penetrate deeply and sensitize photographic negatives. After conducting further research in magnetic fields to study on this kind of ray property, Becquerel proved that the ray is comprised of three components: Of which, the deflection of one component in a magnetic field is the same as that of a positively charged ion stream; the deflection of another component in a magnetic field is the same as that of a negatively charged ion stream; and deflection does not occur with the last component in a magnetic field. The rays are, respectively, called alpha ray (α), beta ray (β), and gamma ray (γ).

Nuclear decay occurs when the nuclei of some nuclides naturally emit alpha and beta particles and transform into the nuclei of other types of nuclide, or when a nucleus emits photons (gamma rays) while transitioning from its excited state to its ground state.

Types of nuclear decay include:

1. Alpha decay: When the nucleus of a radioactive nuclide (radionuclide) emits alpha particles and changes into the nucleus of another nuclide, this process is called alpha decay. Alpha particles are helium nuclei moving at great speeds. An alpha particle consists of two protons and two neutrons, and so it has a charge of $2e$, and its mass is the same as the mass of a helium nucleus. Usually the nucleus before decay is called parent nucleus, and that after decay is termed daughter nucleus. When the nucleus of a radioactive nuclide undergoes alpha decay, the resulting daughter nucleus has an atomic number or nuclear charge number that is two smaller than its parent nucleus. In other words, it would move two spots forward (to the left) on the periodic table. Mass of the daughter nucleus meanwhile would be four less than its parent nucleus. These are expressed in the following equation:

$$ {}^{A}_{Z}\mathrm{X} \rightarrow {}^{A-4}_{Z-2}\mathrm{Y} + \alpha \qquad (1) $$

$$ \text{For example}: \ {}^{226}_{88}\mathrm{Ra} \rightarrow {}^{222}_{86}\mathrm{Rn} + {}^{4}_{2}\mathrm{He} $$

Alpha decay is a feature of the nucleus of heavy elements. The absolute majority of naturally radioactive nuclides that undergo alpha decay are nuclides with atomic numbers $Z > 82$. The half-life of alpha decay varies drastically depending on the nuclide, with some as short as 10^{-7} s, while others extend to 10^{15} a. The distribution of energy of alpha particles usually falls within the (4–9) MeV range.

Alpha ray is comprised of helium nuclei (alpha particles) moving at great speeds, and hence, their deflection in a magnetic field is the same as that of a positively charged ion stream. The alpha ray has high ionization and low penetration.

2. Beta decay: When the nuclear charge number of a nucleus changes by ±1 while mass stays the same, this kind of decay is called beta decay.

Beta decay refers to the process in which a negative electron e^- is emitted from inside a nucleus. Here, the mass of daughter nucleus and the mass of parent nucleus are the same, except that the resulting daughter nucleus has an additional proton. Therefore, 1 is added to its atomic number and it is moved one spot backward (to the right) on the periodic table. Electrons emitted from this kind of nuclei are called beta particles. This may be expressed in the following equation:

$$ {}^{A}_{Z}\mathrm{X} \rightarrow {}^{A}_{Z+1}\mathrm{Y} + \beta^- + \nu \ \left(\text{neutrino}\right) \qquad (2) $$

$$ \text{For example}: \ {}^{32}_{15}\mathrm{P} \rightarrow {}^{32}_{16}\mathrm{S} + e^- + \nu $$

Beta decay may be regarded as the conversion of a neutron into a proton in the parent nucleus while simultaneously emitting beta particles and neutrinos.

$$ n \rightarrow p + \beta^- + \nu \qquad (3) $$

The half-life of beta decay is chiefly distributed within the range of 10^{-2} s and 10^{18} a. The energy of emitted particles could reach a maximum of several MeV. Beta decay differs from alpha decay in that not only does it occur in heavy nuclei, but nuclides with beta radiation exist throughout the entire periodic table.

Beta ray is a stream of fast-moving electron flow. The beta ray has relatively low ionization and high penetration.

3. Gamma decay: Different kinds of radioactive decays always result in daughter nuclei in excited state, and bombardment by hi-speed particles or absorption of photons could also activate a nucleus into an excited state. Nucleus in an excited state is unstable and may be deactivated directly to its ground state.

   The light-emitting process of when a nucleus transitions from an excited state into a low-energy state or ground state is called gamma decay.

$$\text{For example}: \ {}^{203}_{83}\text{Bi} \xrightarrow{11.8 \text{ hours}} {}^{203}_{82}\text{Pb}^{m} + \beta^{+}$$
$$\downarrow$$
$$ {}^{203}_{82}\text{Pb}^{m} \xrightarrow{6.1 \text{ seconds}} {}^{203}_{82}\text{Pb} + \gamma \qquad (4)$$

   During the process of gamma decay, both the mass and the atomic number of the nucleus remain unchanged, and only the energy state of the nucleus changes. This is why this kind of change is called isomeric transition.

   Gamma ray is an electromagnetic wave with very short wavelength. The gamma ray has low ionization and high penetration.

Law of Radioactive Decay

Temperature, pressure, and humidity of the surrounding environment have no impact on the decay of a radioactive nuclide, which simply follows the law of exponential decay. In other words, the number of atoms decaying every second is proportional to the number of existing radioactive atoms. For example, if a certain radioactive nuclide has N_0 atoms at the beginning, after a time period t, only N atoms would remain, then the relationship between N and N_0 is

$$N = N_0 e^{-\lambda t} \qquad (5)$$

Of which, λ is decay constant and represents the relative rate at which the different nuclides decay, as in the number of nuclides the decay every second is a certain fraction of the original number of radioactive nuclides. Its unit of measurement is reciprocal of units of time (1/s, 1/min, etc.).

Half-Life Time

Half-life time ($T_{1/2}$) is defined as the time it takes for atoms of radioactive nuclide to decay by half. Half-life is associated with decay constant λ in the following manner:

$$T_{1/2} = \frac{0.693}{\lambda} \qquad (6)$$

$T_{1/2}$ for different nuclides varies substantially. For instance, the half-life of ^{232}Th is 1.39×10^{10} years, but that of ^{212}Po is only 3.0×10^{-7} s. The half-life time of several radioactive nuclides with common medical applications is:

^{24}Na has $T_{1/2}$ = 15.6 h; ^{131}I has $T_{1/2}$ = 8.1 days; ^{32}P has $T_{1/2}$ = 14.3 days; ^{59}Fe has $T_{1/2}$ = 47.1 days; ^{60}Co has $T_{1/2}$ = 5.3 years; ^{3}H has $T_{1/2}$ = 12.4 years; and ^{14}C has $T_{1/2}$ = 5720 years.

Nuclear Reaction

The nuclear decay explained earlier is a change that occurs in the nucleus, and the nucleus always changes toward stability, ultimately resulting in a more stable nucleus. If a nucleus undergoes structural change due to external reasons such as bombardment by charged particles, absorption of neutrons or illuminated by high-energy photons, such a change is called nuclear reaction.

Nuclear Fission

This may be divided into either spontaneous fission or induced fission.

Spontaneous fission is akin to radioactive decay in that the nucleus undergoes spontaneous fission on its own without external factors such as particle bombardment. This occurs because a relatively heavy atom has smaller specific binding energy than that of a nucleus with medium mass, and its nuclear fission process emits energy.

Heavy atoms also undergo fission when bombarded by foreign particles, and this kind of fission is known as induced fission. In an induced fission, fission induced by neutron is the most common. Since there is no Coulomb barrier between neutron and target nucleus, neutrons of very low energy can enter and excite the nucleus, resulting in fission. Neutrons are emitted during the fission process and could lead to chain reaction. This is also why neutron-induced fission has gained so much interest.

The atoms of some heavy elements such as ^{233}U, ^{235}U, and ^{239}Pu would split into two new atoms with lighter mass when bombarded by neutrons while at the same time release two to three neutrons and photons. Newly divided nuclei are also called fission fragments, they could be different kinds of isotopes of any elements with atomic number from 30 to 64 and are usually radioactive. The fission process releases an immense amount of energy.

Nuclear Fusion

The reaction when two light atoms combine to form relatively heavier atoms under certain conditions is known as light nuclear fusion reaction. This process also emits neutrons and a massive amount of energy. Since fusion reaction could only occur under extremely high temperature, this kind of reaction is also called "thermal nuclear reaction." For example: Under extremely high temperatures, deuterium and tritium fuse to form helium nuclei and release neutrons and tremendous

amount of energy. Much more energy is released from thermal nuclear reaction than from heavy nuclear fission reactions.

1.2.2 Explosion Principle and Basic Composition of Nuclear Weapons

Atomic Bomb

Explosion Principle

Chain reaction of heavy nuclear fission is the chief principle behind the explosion of an atomic bomb.

Upon bombardment by neutrons, atoms of some heavy elements (i.e. ^{235}U and ^{239}Pu) would split into two new atoms (a.k.a. fission fragments) of similar mass and release two to three neutrons along with around 200 MeV of energy. This process is known as heavy nuclear fission. Take, for example, the reaction equation for ^{235}U is:

$$^{235}\mathrm{U} + {}^{1}_{0}\mathrm{n} \rightarrow \mathrm{X} + \mathrm{Y} + (2 \sim 3)\,{}^{1}_{0}\mathrm{n} + 200\mathrm{MeV} \qquad (7)$$

In this equation, X and Y are new atoms (fission fragments).

This energy does not look like much, just about enough force to make the smallest grain of sand visible to the naked eye to jump. Yet, this only represents the energy released by the fission of a single atom. One mole of ^{235}U contains 6.02×10^{23} atoms, which weigh a total of no more than 235 g. If all these atoms were to undergo fission, the energy would be shocking, roughly equal to energy released from complete combustion of 600 tons of coal. When 1 kg of ^{235}U undergoes complete fission, it would release energy equivalent to approximately 20,000 tons of TNT.

Every fission would release two to three neutrons. If one of these neutrons hits another heavy nucleus and instigates its fission, the same reaction would occur after fission. This would enable heavy nuclear fission to sustain on its own, and this is known as the chain reaction of heavy nuclear fission.

However, not all emitted neutrons might initiate a new fission. For example, some atoms are very small and neutrons might not be able to make contact with uranium atoms. If the uranium core is not big enough, some neutrons would miss and fly pass the uranium without triggering new fission. In addition, other substances in the uranium core would also absorb neutrons, preventing the start of new fission reactions.

The chain reaction of heavy nuclear fission could only occur within a body of or beyond a certain mass. The smallest mass of fissile material capable of sustaining nuclear chain reaction is called critical mass. The size of the material corresponding to critical mass is called critical size. Critical mass is closely associated with the type, purity, density, and geometrical shape of the fissile material chosen. If the wrapping material is neutron-reflective substance, critical mass may be lowered further. To decrease critical mass, fissile materials are often configured into spheres because spheres have the smallest surface area compared with other shapes of the same size, and thus a ball of fissile material minimizes neutrons loss.

Basic Structure

An atomic bomb primarily consists of the nuclear fuel (^{235}U or ^{239}Pu), detonation device, neutron initiator, neutron-reflective layer, and the bomb shell, among other components.

Detonation Process

After the detonation device is triggered, the various explosive modules would detonate simultaneously. The tremendous pressure generated from the explosion would press toward the center, forcing the separate and small pieces of sub-critical fissile materials to instantaneously combine into a sphere. Reaching a supercritical state. Under bombardment of neutrons produced by neutron initiator, heavy fission chain reaction starts and develops increasingly more intense by geometric order, resulting in heavy nuclear fission of a certain amount within an extremely short period, releasing tremendous energy and creating a powerful nuclear explosion. One kilogram of ^{235}U or ^{239}Pu only needs a few millionths of a second to undergo complete fission. The energy released equates to energy from the explosion of 20,000 tons of TNT explosives.

Hydrogen Bomb

Explosion Principle

Hydrogen bomb's explosion principle is light nuclear fusion reaction.

The atoms of some lighter nuclides (such as ${}^{2}_{1}\mathrm{H}$ and ${}^{3}_{1}\mathrm{H}$) would undergo fusion under extremely high temperatures of tens of millions of degrees, and in the process release neutrons and large amount of energy. For example:

$${}^{6}_{3}\mathrm{Li} + {}^{1}_{0}\mathrm{n} \rightarrow {}^{3}_{1}\mathrm{H} + {}^{4}_{2}\mathrm{He}$$

$${}^{2}_{1}\mathrm{H} + {}^{3}_{1}\mathrm{H} \rightarrow {}^{4}_{2}\mathrm{He} + {}^{1}_{0}\mathrm{n} + 17.6\mathrm{MeV} \qquad (8)$$

Since fusion can only happen under extremely high temperature, fusion reaction is also known as thermonuclear reaction, and hydrogen bombs are also called thermonuclear weapons.

Basic Structure

A hydrogen bomb is chiefly comprised of a thermonuclear fuel (usually lithium deuteride), detonation device (a small equivalent atomic bomb), and the bomb shell (usually containing ^{238}U), among other components.

Detonation Process

The atomic bomb is first detonated, lithium deuteride is subjected to the resulting high temperature, high pressure, and neutron bombardment, and the lithium would produce deuterium. Next, the deuterium–tritium would quickly fuse while emitting high-energy neutrons and tremendous energy, resulting in an explosion even more powerful than that of an atomic bomb. Complete fusion of 1 kg of deuterium–tritium mixture would generate three to four times more energy than that released by the complete fission of 1 kg of ^{235}U or ^{239}Pu. Hydrogen bomb is a fission–fusion double-phase bomb. If the shell of hydrogen bomb contains ^{238}U, the high-energy neutrons produced from deuterium–tritium fusion could instigate fission in the ^{238}U, adding to the yield of fission fragments and boosting the force of the explosion. This kind of hydrogen bomb is a fission–fusion–fission triple-phase bomb.

Neutron Bomb

A neutron bomb is a tactical nuclear weapon that utilizes fusion reaction from deuterium–tritium to produce high-energy neutrons for lethal effects. Its structure is similar to that of a hydrogen bomb.

The neutron bomb is characterized by the following features:

1. Large yield of powerful neutrons; neutron bomb relies on the fusion between deuterium and tritium, between deuterium and deuterium, and between tritium and tritium, and 80% of energy released from the fusion comes in the form of neutrons. Compared with an atomic bomb with the same explosive power, a neutron bomb could produce ten times more neutrons, with the average neutron energy reaching 14 Mev or even as high as 17 MeV.
2. Effects of light radiation and shock wave are merely one-tenth of an equivalent atomic bomb, and radioactive contamination is much more limited.
3. Small equivalent, usually around 1000–3000 tons TNT equivalent.

1.2.3 Explosion Methods and Explosion Visual Effects of Nuclear Weapons

The power of a nuclear weapon is usually expressed as TNT equivalent (simplified as equivalent), or in other words, equating to the power of the explosion of an equal amount (in tons) of TNT. The power of nuclear weapon is measured in tons, and common classes are 100-ton, kiloton, 10k-ton, 100k-ton, megaton, and 10-megaton, among others. In general, the equivalent of an atomic bomb is in the 100k-ton range, hydrogen bombs are above the 100k-ton range, and neutron bombs are around the kiloton range.

The damage caused by a nuclear weapon depends not only on the performance of the weapon itself, but also closely related to the method of explosion. The same type and power of nuclear weapon detonated in different media and at different heights would create different levels of damage.

Nuclear explosions may be classified as air burst (air explosion), ground (water) surface burst (surface explosion), or underground (underwater) burst. Air bursts and ground bursts are differentiated based on ratio relationship between actual height of explosion and equivalent. This ratio is called scale height of burst, or SHOB for short. An explosion with SHOB less than 60 is considered a ground burst, and one with SHOB above 60 is deemed an air burst.

$$\text{SHOB} = \frac{\text{Height of burst}}{\sqrt[3]{\text{Equivalent}}} \tag{9}$$

During an air burst or ground burst of a nuke, the three classic visual effects of flash, fireball, and mushroom cloud would appear in that sequence, and the sound of the explosion may be heard within a certain area.

Air Nuclear Burst

During an air burst, the first sight is a brilliant flash, quickly followed by a bright fireball that suspends in mid-air at the site of the air blast. The fireball fiercely bulges outward, compressing the air around it, and rapidly expands outward, forming a shock wave. The fireball swiftly elevates upward while at the same time generating a powerful suction on the ground surface, sucking up a considerable amount of dust and debris, which turn into a column of smoke that rises with the ascending fireball. This smoke column eventually connects with the smoke and vapor around the fireball, forming a mushroom cloud. A nuclear explosion causes a great bang, which could be heard several tens of km or even hundreds of km away from the center of the blast.

Air burst may be categorized as low-altitude air burst ($60 < \text{SHOB} < 120$), mid-altitude ($120 < \text{SHOB} < 200$), high-altitude ($200 < \text{SHOB} < 250$), or super high-altitude. Low-altitude air bursts are mainly utilized to destroy relatively hardened ground surface targets or to targets buried slightly underground without very high compressive strength. Radioactive contamination is usually rather severe at the site of explosion and in downwind areas. High-altitude air bursts are predominantly used to damage large-area targets and kill troops exposed on a large area of ground surface. Since a high-altitude air burst does not draw up as much dust from the ground, it does not generate as much fallout and ground surface radioactive contamination. Super high-altitude nuclear air bursts are those detonated 30 km or higher above the ground, there is a marked increase of light radiation in the energy of the explosion, while energy converted to shock wave significantly decreases. This kind of explosion only generates a fireball, which ultimately cools into smoke and cloud with no dust column that connects to the ground, hence the absence of the mushroom cloud display. The equivalent of a super high-alti-

tude nuclear air burst is very high, that's why it can produce a massive electromagnetic pulse within a huge area. When a 50-megaton nuke is detonated 80 km in the air, it could completely deactivate radio communication for 1 day within a radius of 4000 km. Super high-altitude nuclear air bursts are targeted against satellites, missiles, electronic and communication systems, among others, as they do not have much of an impact on ground surface personnel.

Ground (Water) Surface Nuclear Burst

In a ground burst, since the fireball is in contact with the ground, the fireball is semi-spherical in shape. Surface of the soil at the center of the blast would be burnt, the fireball would suck up a sizable amount of dust and ash, the column of radioactive dust would connect with the relatively dark-colored smoke cloud, a substantial amount of radioactive dust and ash would be generated and eventually fall to the ground, and radioactive contamination in the blast zone and downwind areas would be severe due to the spread and fall of radioactive debris. A water surface burst is similar to a ground surface burst and is capable of producing semi-spherical fireball and mushroom cloud, with high amount of water and vapor in the smoke cloud, and serious radioactive contamination after the fall of debris.

Underground (Underwater) Burst

The visual displays of an underground burst or underwater burst differ depending on the media surrounding the explosion and depth of the blast. Flash and fireball are not seen in an underground blast deep below the ground surface, but fire from the explosion could erupt from the surface in a shallow underground (or underwater) burst, which would launch a huge amount of dust, rock, and other particles from the explosion into the air, create a large and dark smoke cloud, and produce a deep crater, with severe radioactive contamination after the smoke cloud settles.

2 Section Two: Lethal Factors of Nuclear Weapons

There are four main lethal factors of nuclear weapons: light radiation, shock wave, initial nuclear radiation, and radioactive contamination.

The fission or fusion reaction from a nuclear explosion instantaneously generates high temperature upward of tens of millions of degrees and at the same time produces a powerful flash of light. Vaporized substances of the bomb shell and the rapidly heated air surrounding the center of explosion together form a bright fireball in the air. The light and heat emitted from the flash and fireball are known as light radiation.

The rapidly bulging fireball drastically compresses air around it, creating a nuclear blast wave.

The large amount of neutrons produced during the course of a nuclear explosion would be absorbed by the hydrogen atoms in the air and release gamma rays. Nuclear fission products would also generate a great deal of gamma rays within the first dozen seconds or so after the explosion. The gamma rays from these neutrons and other sources are called initial nuclear radiation.

Nuclear fission products, unfissiled nuclear fuel, and element activated by neutrons would all condense from gaseous state to dust particles then settle on the ground surface, creating radioactive contamination on the ground and in the air. The gamma rays and beta rays emitted by these particles are known as residual radiation.

In addition, the substantial amount of X-rays and gamma rays generated during the explosion process mutually affect the molecules and atoms in the surrounding region, producing a great deal of charged particles. These charged particles move at high speeds, creating a very powerful transient electromagnetic field around the center of the blast. This electromagnetic field expands in all directions in the form of a wave known as electromagnetic pulse (EMP). A nuclear electromagnetic pulse has very high field strength, very wide spectrum, and high speed of propagation, and its area of effect is much bigger than light radiation, shock wave, or initial nuclear radiation. However, since a nuclear electromagnetic pulse does not cause much damage to people, it is usually not listed as one of the primary lethal factors of nuclear weapons.

Light radiation, shock wave, or initial nuclear radiations only produce their effects in the several tens of seconds after the blast, and these are collectively termed "instantaneous lethal factors." Radioactive contamination meanwhile could continually cause injuries and deaths for months or even longer.

The lethal and destructive effects of a nuclear blast are also related to the power of explosion and height of burst. An air burst of a megaton or bigger nuclear bomb causes destruction primarily through light radiation and shock wave, with the former being particularly widespread in area of effect, and capable of causing big fires in urban regions. Low-yield air busts of nuclear weapons in the 10-kiloton range usually causes the widespread injuries and deaths through initial nuclear radiation, followed by shock wave and then light radiation. Neutron bombs are usually less than kiloton, and their main lethal effect is initial nuclear radiation from neutrons and gamma rays.

2.1 Light Radiation

2.1.1 Formation of Light Radiation

Comprised of the light and heat from the nuclear explosion fireball burning at tens of millions of degrees, light radiation radiates to the surrounding and is also known as thermal radiation. Light radiation usually releases about 35% of the total energy of the nuclear blast.

2.1.2 Main Properties of Light Radiation

Release of Energy

The energy released from light radiation comes in two pulses.

The first pulse is the flash, which lasts an extremely short period, and the energy released only accounts for about 1–2% of total light radiation energy, predominantly in the form of UV rays. This stage would not cause skin damage but might lead to injury to the eyes.

The second pulse is the fireball, which could last anywhere from several seconds to tens of seconds, and the energy released by the fireball makes up around 98–99% of total light radiation energy, chiefly in the form of infrared rays and visible light. Burns suffered by personnel mostly come from this stage.

Radiant Exposure

Radiant exposure is the main parameter in assessing the damage and destruction caused by light radiation. Radiant exposure refers to the energy projected by the fireball, during its own duration of illumination, onto a unit area perpendicular to the propagation direction of the light radiation. The unit of measurement is joule per square meter (J/m^2) or joule per square centimeter (J/cm^2).

Propagation of Light Radiation

Light radiation has the properties of normal light and propagates rectilinearly at the speed of light within the atmosphere (3×10^8 m/s). During its course of propagation, light radiation is weakened due to the reflection, scattering, and absorption of various media in the atmosphere, but still imparts its effect through transparent objects.

2.2 Shock Wave

2.2.1 Formation of Shock Wave

The high-temperature, high-pressure fireball produced by a nuclear explosion expands outward fiercely, compressing air layers in its surrounding and creating a spherical compression zone with extremely high air density. As the compression zone speedily moves outward, a spherical rarefaction zone with pressure lower than standard atmospheric pressure is formed behind it. The two zones closely adjoin one another and swiftly propagate in the surrounding media, forming the nuclear blast wave. Usually such a blast wave accounts for roughly 50% of the total energy released in the nuclear explosion and constitutes the main force of destruction in a nuclear blast that demands the most attention.

2.2.2 Main Properties of Shock Wave

Pressure of the Shock Wave

Pressures of the shock wave come in three forms, namely overpressure, dynamic pressure, and underpressure (negative pressure). The pressure above standard atmospheric pressure within the compression zone is known as overpressure, and force from hi-speed air movement is called dynamic pressure. Overpressure and dynamic pressure on the shock wave front are at the highest, and these are known as peak overpressure and peak dynamic pressure. They are expressed as pressure borne by a unit area, with measurement unit being Pascal (Pa), with 1 Pa = 1 N/m^2, 1 kPa = 7.501 mmHg. The pressure below standard atmospheric pressure within the rarefaction zone is known as negative pressure. The destructive force of a shock wave mostly originates from overpressure and dynamic pressure, negative pressure meanwhile has a relatively smaller effect.

Propagation of Shock Wave

A shock wave propagates under the same pattern as a sound wave. The higher the pressure, the faster the velocity of propagation, and the initial velocity could reach several km/s. Propagation velocity drops following farther distance propagated and decline in pressure. When pressure falls to atmospheric pressure, the shock wave turns to a sound wave and disappears.

Effective Duration of Shock Wave

The time it takes for a shock wave to reach a certain distance is known as the shock wave's arrival time. When a shock wave arrives at a certain spot, the time it takes for the pressure to rise to its peak is called pressure increase duration. The time that peak pressure stays in effect is known as positive pressure effective duration. The shorter the pressure increase duration and the longer the positive pressure effective duration, the greater the destructive power and vice versa.

2.3 Initial Nuclear Radiation

2.3.1 Formation of Initial Nuclear Radiation

A lethal factor unique to nuclear blast, initial nuclear radiation is also known as penetrating radiation and is comprised of gamma rays and neutron streams within the first dozen seconds or so right after the explosion. Energy released from

initial nuclear radiation makes up about 5% of total energy released from a nuclear explosion.

2.3.2 Main Properties of Initial Nuclear Radiation

High Propagation Velocity

Gamma rays travel at the speed of light, while the propagation velocity of neutrons depends on their energy, with the highest velocity close to the speed of light.

Short Effective Duration

Neutrons from nuclear fission and fusion, and gamma rays generated from neutrons captured by hydrogen, have an effective duration of less than half a second. Initial nuclear radiation's effective duration on targets on the ground surface usually stays within 20 s regardless of equivalent of the nuclear weapon. This is because gamma rays released from fissile fragments are mostly nuclides with short half-life that decay quickly, and they rise farther from the ground with the ascending fireball and smoke cloud.

Scattering

Initial nuclear radiation basically propagates rectilinearly, but could scatter upon collision with other media during the propagation process, and travels to target objects in random movement directions.

Strong Penetration That May Be Weakened by Media

Initial nuclear radiation has high penetration, but is weakened to varying extents after being absorbed when passing through different media. The ability for a medium to weaken initial nuclear radiation is usually expressed as the medium's half value layer. A half value layer of a medium refers to the thickness required to weaken initial nuclear radiation by half. Table 1 lists the half value layer of some media for initial nuclear radiation. As shown, a soil layer 14 cm in thickness can weaken initial nuclear radiation by 50%. Furthermore, different media have different weakening effects on different types of rays.

Generation of Induced Radioactivity

The atoms of certain stable nuclides in the soil, weaponry, foods that contain salt and pharmaceuticals could form radioactive nuclides from capturing neutrons of a nuclear explosion. This type of radioactive nuclide is known as induced or artificial radioactive nuclide, and the resulting radioactivity is thus called induced or artificial radioactivity.

Initial nuclear radiation is usually expressed as radiation dose, measured in the unit gray (Gy). The amount of neutrons is usually expressed as neutron flux, which is measured as the number of neutrons in a unit area (m^{-2} or cm^{-2}).

Table 1 List of the half value layer of some media for initial nuclear radiation

	Half value layer/cm				
Radiation	Soil	Concrete	Wood	Water	Iron
Gamma rays	14.0	10.0	30.0	20.0	3.2
Neutrons	13.8	10.3	11.7	5.5	4.7

2.4 Radioactive Contamination

2.4.1 Formation of Radioactive Contamination

A huge amount of radioactive nuclides are created and vaporized during a nuclear blast, which would then be dispersed within the fireball. When the fireball cools into smoky cloud, the particles in the cloud and dirt rising from the ground would congeal into radioactive particles. Under the pull of gravity, these radioactive particles would descend back to the ground in what is known as radioactive fallout. The resulting contamination of the air, ground surface, water source, various objects, and human bodies is collectively referred to as radioactive contamination.

2.4.2 Main Properties of Radioactive Contamination

Composition

Radioactive fallout is mainly comprised of the three components of nuclear fission products, induced radioactive nuclides, and unfissiled nuclear fuel. Radioactive fallout primarily emits beta and gamma rays.

Physical and Chemical Properties

1. Form: Fallen particles are mostly spherical or elliptical grains, and within each is an even distribution of radioactive substances. Color of particle is linked to soil in the blast zone and could be black, gray, or other hues. Particle size is related to explosion method. Particles are larger from a ground burst, from several micrometers to a few millimeters, while particles in an air burst are relatively smaller, usually between several micrometers to several dozen micrometers.
2. Solubility: Solubility is connected to particle size, chemical composition of radioactive substances, and pH of solvent. Solubility is relatively lower in water, at around the 10% range. Solubility is relatively higher in acidic solvent, for instance, 35–60% solubility in 0.1 mmol/L hydrochloric acid.
3. Specific activity: The specific activity of fallout decreases with the increase in particle size. In terms of fallout 1 h after the explosion, specific activity of a ground burst is 10^7 to 10^{10} Bq/g, whereas that of an air burst is 10^8 to 10^{13} Bq/g.

Pattern of Decay of Fallout

Experiments indicate that within the first to 5000 h after the explosion, decay of radioactive dose (as in dose rate) on ground surface may be approximated using the "Sixfold Law," meaning that radiation dose rate is reduced by a factor of 10 for every sixfold increase in time. For example, when the radiation dose is 80 cGy/h 1 h after the blast, dose rate would drop to 8 cGy/h six hours after the blast, and then further down 0.8 cGy/h to 36 hours after the blast.

Radioactive Contamination Volume

1. Ground surface contamination: This is measured as radiation dose at a height of 0.7–1 m above the ground and expressed in the unit gray (or centigray) per hour (respectively, Gy/h and cGy/h).

   Usually a region with 0.5 cGy/h is classified as bordering contamination. Ground surface contamination severity is divided into four levels: Radiation dose rate of 0.5–10 cGy/h indicates a mildly contaminated area, 10–50 cGy/h is a moderately contaminated area, 50–100 cGy/h is a severely contaminated area, and dose rate above 100 cGy/h means a very is a moderately contaminated area.
2. Human body or object surface contamination: This is measured as radioactivity on a unit area and expressed in becquerels per square meter or becquerels per square centimeter (respectively, Bq/m^2 and Bq/cm^2).
3. Substance contamination: This is measured as specific activity and expressed in becquerels per kilogram or becquerels per gram (respectively, Bq/kg and Bq/g).
4. Air or liquid contamination: This is measured as radioactive concentration and expressed in becquerels per liter or becquerels milliliter (respectively, Bq/L and Bq/mL).

3 Section Three: Lethal Factors of Nuclear Weapons

The lethal factors of nuclear weapons are primarily expressed in the types of injuries caused and the range of injury and death. This section looks into the injuring effects of the four lethal factors, injury types and severity caused by nuclear weapons, range of injury and death of nuclear weapons, and influences of the lethal effects of nuclear weapons.

3.1 Injuring Effects of Four Main Lethal Factors

3.1.1 Injuring Effects of Light Radiation

Light radiation may burn skin and mucous membrane on the body surface, which may be called direct burn or light radiation burn. Under the effects of light radiation, structures, fortifications, clothing, and other objects may be ignited, and burns from the resulting fire are known as indirect burns or fire burns. The injuring effects of light radiation are largely dependent on the amount of light impulse.

Light radiation burns are mainly characterized by the following features:

Direction of Body Area Burned

The rectilinear propagation of light radiation means that burns are usually induced on the side facing the source of the blast. Boundaries of the burn area are relatively clear.

Superficiality of Depth of Burns

Since light radiation has a short effective period, burns are usually superficial instead of deep. Other than large areas of deep burns inflicted on victims close to the explosion, most such injuries are second-degree burns. Even if there are third-degree burns, usually the subcutaneous layer stays unaffected. Depth of wound is often even across the whole wound.

High Occurrence Rate of Burns in Special Parts of the Body

1. Exposed parts of the body such as the face, ears, neck, and hands are most susceptible to burns.
2. Respiratory tract burn injury: Respiratory tract burn injury is a type of indirect burn, arising from the inhalation of hot air, dust, sand, smoke, or even flame in a combustion environment.
3. Eye burn injury: Light radiation could burn the eyelid, cornea, and fundus Burn injury of the fundus is also called retinal burn and is a special type of injury caused by light radiation. If a person stares directly at the fireball, the focus of the eyeball would magnify light impulse of the incident light by 10^3 to 10^4 times, forming a fireball image on the cornea and causing burn. Cornea burn boundary is three to four times larger than boundary of skin burns.

Flash Blindness

After the powerful light of a nuclear explosion stings the eye, rhodopsin, is "bleached and decomposed," leading to temporary vision impairment known as flash blindness. When a person suffers from flash blindness, he or she would instantly suffer from visual impairment, darkened vision, abnormal color vision, bulging pain, etc. In serious cases, there may be headache, dizziness, nausea, vomiting, and other symptoms of autonomic nerve dysfunctions. However, such symptoms normally will not last long and could subside without sequela within 3–4 h after the explosion even when no treatment has been administered. Flash blindness could be inflicted much further away than fundus burns and imparts a rather signifi-

cant impact on directing personnel, pilots, drivers, and observational personnel.

3.1.2 Injuring Effects of Initial Nuclear Radiation

Initial nuclear radiation is a lethal factor of nuclear weapons. After the human body takes in a certain dosage of nuclear radiation, acute radiation sickness may possibly occur, as well as the biological effects of low dose external irradiation.

3.1.3 Injuring Effects of Radioactive Contamination

Radioactive contamination damages personnel in three ways:

1. **External exposure injury**. When a person stays in a seriously contaminated zone for a prolonged period and is exposed to gamma rays at a dosage >1 Gy, acute radiation sickness from external exposure may possibly take place. This is the main form of injury induced by nuclear fallout.
2. **Internal radiation injury**. When radioactive particles of the fallout enters the body through respiration, food or wound, radioactive nuclides would accumulate in the body and cause internal radiation injury after building up to a certain level.
3. **Beta ray skin injury**. Radioactive particles of the fallout in direct contact with skin could cause direct beta ray burns when dosage >5 Gy.

Unprotected personnel exposed for an extended period in a contaminated zone may suffer from composite radiation injuries involving all three of the above.

3.2 Injury Types and Severity Caused by Nuclear Weapons

3.2.1 Injury Types

The four lethal factors of nuclear weapon could individually, simultaneously, or consecutively act on the human body. Victims could suffer from different kinds of injuries, which are collectively known as nuclear weapon injuries. A single lethal factor would cause a singular injury, and when two or more lethal factors act on a person simultaneously or consecutively, they suffer composite injury. Nuclear weapon injuries are complicated in terms of categorization and severity. Related composite injuries will be discussed in a later portion of the book.

3.2.2 Injury Severity

The various singular or composite injuries may be classified as minor, moderate, severe, or extremely severe based on injury severity (if only classified as minor, moderate, and severe in a three-tier scheme, then the extremely severe level should be included as a part of severe level).

1. **Victims with minor injuries**. Usually victims will not lose the capacity to participate in combat, and there is no need for hospitalization, but need to undergo the necessary medical treatment and care.
2. **Victims with moderate injuries**. Usually victims would lose the capacity to participate in combat, most require hospitalization but in general prognosis will be ideal.
3. **Victims with severe injuries**. Usually victims would immediately or quickly lose the capacity to participate in combat. If active medical care is provided, in general prognosis will be relatively ideal and the majority would heal.
4. **Victims with extremely severe injuries**. Victims would immediately lose the capacity to participate in combat. Based on present day medical care levels, if emphatic efforts are used, the victim may recuperate from some of the injuries.

Whether a victim loses combat capacity, and the immediacy of such loss, varies depending on the type of injury and body part(s) damaged. In the majority of radiation injuries, victims will not lose the capacity to fight. Burns and blast injuries meanwhile, especially those inflicted on special parts of the body, may quickly render the victim unable to participate in combat. For example, although eye burn injuries do not really affect other parts of the body, the victim would not be able to see properly, let alone aim or conduct reconnaissance.

3.3 Range of Injury and Death of Nuclear Weapon

The range of injury and death of nuclear weapon is expressed as injury and death boundary, injury and death radius, and injury and death zone. When a nuclear weapon detonates, there are three instantaneous lethal factors that could kill or injure persons within a certain area, and this area is called the injury and death zone. The distance between ground zero of a ground burst (or surface zero of an air burst) and the spot at which people will not be injured or killed is called injury and death radius, while said spot marks the injury and death boundary. Actual area of injury and death zone may be calculated using the injury and death radius. As such, individual ranges of injury and death for light radiation, shock wave, or initial nuclear radiation may be calculated, as well as their combined range of injury and death. From the center of explosion outward, the severity of personnel injury would decrease from the near to far. Usually the area in which people are killed or injured may be divided into the four injury

and death zones classified as extremely severe, severe, moderate, and minor. The boundary of the minor injury and death zone is the boundary of the entire injury and death zone. The farthest distance at which second-degree burns of the skin can be inflicted by the burst of a 10 kiloton or bigger nuclear weapon would be the boundary of its injury and death zone. The farthest distance at which mild radiation sickness (>1.0 Gy) can be inflicted by the burst of a nuclear weapon below 10 kiloton would be the boundary of its injury and death zone.

The respective individual injury and death radius of the three instantaneous lethal factors, as well as the radius of the three factors combined, on personnel exposed on open ground is measured as the distance at which 50% of the personnel would be injured. The area of the injury and death zone is an approximate number. Kiloton-level nuclear bombs have injury and death zones ranging from less than 1 km^2 to several km^2. Ten kiloton-level nuclear bombs have injury and death zones ranging from more than 10 km^2 to several dozen km^2. 10^2 kiloton-level nuclear bombs have injury and death zones ranging from 100 km^2 to several hundred km^2. Megaton-level nuclear bombs have injury and death zones ranging from several hundred km^2 to 1000 km^2. It should be emphasized that nuclear weapons have massive injury and death zones, but moderate and minor injury and death zones could account for 40–70% of total area. In other words, when people inside the injury and death zone are relatively evenly distributed, a rather large portion of injuries caused by nuclear weapon detonation would be moderate and minor injuries.

3.4 Main Factors Affecting Destructive Power of Nuclear Weapons

The destructive power of nuclear weapons could be affected by many factors, mainly categorized in the following three aspects.

3.4.1 TNT Equivalent and Explosion Method of Nuclear Weapon

TNT Equivalent of Nuclear Weapon

When the equivalent is different, the singular or combined injury and death range of each or all three instantaneous lethal factors differ, and the types and severity of injuries caused also vary drastically. With the rise in equivalent, although the total injury and death range expand, the singular or combined injury and death range of each or all three instantaneous lethal factors do not expand proportionately. Of which, light radiation injury and death range enlarge the most, followed by shock wave and then initial nuclear radiation.

For the detonation of nuclear weapons below the 10 kiloton-level, the biggest injury and death radius is attributed to initial nuclear radiation, followed by shock wave and then light radiation. Therefore, pure radiation sickness and composite radiation injury are the most common types of damage inflicted upon personnel exposed to such a detonation on open ground. The occurrence rate of composite injury is 20–80% for a ground burst and 30–100% for an air burst.

For the detonation of nuclear weapons above the 20 kiloton-level, the biggest injury and death radius is attributed to light radiation, followed by shock wave and then initial nuclear radiation. In addition, injury and death radii of light radiation and shock wave rapidly expand with the rise in equivalent, while that of initial nuclear radiation does not enlarge much, whose range usually stays within 4 km. For the main types of injuries suffered by personnel exposed to such a detonation on open ground, the number of pure burns, burn-radiation-blast composite injuries, and burn-blast composite injuries increase with the rise in equivalent.

For the detonation of nuclear weapons above the 500 kiloton-level, since the on-site kill zone is larger than the injury and death range of initial nuclear radiation, basically all injuries are either pure burns or burn-blast composite injuries. The occurrence rate of composite injury is 60–90% for a ground burst and 30–50% for an air burst.

Explosion Method of Nuclear Weapon

Generally speaking, when two nuclear bombs have the same TNT equivalent, air bursts have bigger overall injury and death range than ground bursts. However, the injury and death ranges of the four lethal factors are not the same. Air bursts create bigger ranges of burns and blast injuries than ground bursts, but within a certain distance close to the center of explosion, ground bursts induce more severe injuries than air bursts. Ground bursts create a bigger range of initial nuclear radiation injury and death than air bursts. When it comes to radioactive contamination, ground bursts contaminate a smaller area but in worse severity, while air bursts contaminate a bigger area at less severity. The higher the scale height of burst (SHOB), the lesser the contamination.

3.4.2 Population Density and State of Protection

When an area with high population or a concentration of deployed troops is attacked by nuclear weapons, the resulting injuries and casualties would no doubt be serious. Within the injury and death range, if there is a high concentration of

people near the center of the blast, then the ratios of composite injuries and severe injuries will definitely increase.

During a nuclear attack, if people are sufficiently prepared and effective protective measures have been adopted, then injury and death range would be reduced compared to unprotected personnel exposed on open ground. Since the effects of a single or several lethal factors have been nullified or lessened, the ratio of singular injuries would rise, while the ratio of composite injuries would in turn decrease, and injury severity would also drop.

3.4.3 Natural Conditions

Climate Conditions

1. Low visibility can shorten the injury and death radii of light radiation and initial nuclear radiation.
2. Ice and snow reflect light and thus would strengthen the effects of light radiation.
3. When a nuclear bomb is detonated at a high altitude, clouds would absorb and weaken the effects of light radiation and initial nuclear radiation on the ground. Meanwhile, when a nuke is detonated below the clouds, the clouds would intensify the effects of light radiation on the ground.
4. Rain and snow can accelerate fallout, reduce air contamination but worsen localized contamination of the fallout. Radiation dose from ground contamination may decrease when contaminants are washed away by substantial rainfall, or when covered up by snow.
5. Direction of high-altitude wind can alter shape of cloud area and contaminant distribution along transverse direction, while wind speed can alter and contaminant distribution along longitudinal direction. The stronger the wind, the bigger the area contaminated and the more even the contamination, with contamination severity in nearby regions generally reduced while severity in faraway regions relatively increased.
6. Cold weather means higher atmospheric density, which could shorten the injury and death radius of initial nuclear radiation. People tend to wear more and thicker clothes in cold weather, with less body area exposed, and thus light radiation burns would substantially decrease.

Geography and Landform

1. The frontal slopes of hills, hilly terrains, structures, and other objects or landforms would reflect and strengthen a shock wave, the rear slopes meanwhile can nullify or reduce the effects of the three types of instantaneous lethal factors.
2. Pits, explosion craters, culvert, ditches, and other structures below ground level can diminish the effects of the three types of instantaneous lethal factors.

4 Section Four: Occurrence of Nuclear Blast Injuries

Shock wave is a mechanical wave and transmits energy through vibration in the propagation medium. A nuclear blast wave has the same property as a blast wave generated from conventional explosives. In a nuclear explosion, the injuries induced by the shock wave which acts on the human body are known as nuclear blast injuries.

4.1 Casualty Range of Nuclear Blast Injuries

During a nuclear explosion, there are three instantaneous lethal factors, and the injury and death range of blast injury is usually ranked second among the trio. Whether ground burst or air burst, when a nuclear weapon of small equivalent is detonated, as in a weapon below the 10 kiloton-level, the injury and death range of shock wave is smaller than initial nuclear radiation but larger than light radiation. Conversely, when a bigger nuclear weapon is detonated, as in a weapon above the 10 kiloton-level, the injury and death range of shock wave is smaller than light radiation but larger than initial nuclear radiation.

Generally speaking, injury and death range is closely associated with method of explosion. In the majority of cases, the injury and death range of shock wave in an air burst is bigger than that in a ground burst. There are several reasons behind this difference. First and foremost, quite a portion of a ground burst's shock wave is transmitted into the ground and then compresses the soil, creating an underground (in-soil) compression wave that propagates below the ground surface; second of all, if the nuclear weapon detonates upon hitting the ground, a certain portion of the shock wave's energy would be consumed during the process of the formation of the bomb crater, which may possibly and consequently create seismic wave that directly transmits into the ground; and thirdly, the dust and sand immediately drawn up after a ground burst also weaken the propagation of the shock wave to a certain degree.

In addition, there is also a close connection between the TNT equivalent of the weapon and the shock wave's injury and death range. Injury and death range increases correspondingly with the rise in equivalent. However, the expansion in injury and death range is not proportionate to the rise in equivalent. The book *Trauma War Wound Pathology* mentions that when TNT equivalent rises from 10 kiloton to 5 megaton, injury and death radius would only expand by 15.5–16.1 times, while injury and death zone would enlarge by 151–168 times. See data in Table 2. This difference is the outcome of the spherical propagation of the shock wave in all directions, and the shock wave's energy is being con-

Table 2 Relative values of injury and death range of the shock wave from explosions of nuclear weapons with different equivalent

TNT equivalent (kiloton)	1		10		50		100		500		1000		5000	
Explosion method	Ground burst	Air burst	Ground burst	Air burst	Ground burst	Air burst	Ground burst	Air burst	Ground burst	Air burst	Ground burst	Air burst	Ground burst	Air burst
Injury and death radius	0.54	0.54	1	1	2.6	2.5	3.4	3.3	6.5	6.5	8.8	8.5	17.1	16.5
Injury and death zone	0.17	0.16	1	1	3.9	3.6	6.6	6.1	24.6	23.5	44.4	39.8	169	152

Note: SHOB of ground burst is 0, and SHOB of air burst is 120
Injury and death radius refers to the distance between ground zero of a ground burst (or surface zero of an air burst) and the spot at which people are injured to different extents or killed
The table uses the injury and death radius and injury and death zone of the ground burst and air burst of a 10 kiloton nuclear weapon as reference

Table 3 Proportions of blast injury and death zone in total injury and death zone under different explosion methods and TNT equivalent conditions

TNT equivalent/kt	Explosion method	Total injury and death zone/km^2	Blast injury and death zone/km^2	Blast injury and death zone	
				As percentage of total injury and death zone	Reference values for air bursts and ground bursts
1	Ground burst	3.02	0.72	23.8	0.85
	Air burst	3.02	0.85	28.2	
10	Ground burst	4.45	2.49	55.9	0.83
	Air burst	4.37	2.95	67.5	
50	Ground burst	5.47	4.30	78.6	0.81
	Air burst	5.47	5.31	97.1	
100	Ground burst	47.8	28.3	59.2	1.79
	Air burst	98.5	32.6	33.1	
500	Ground burst	152	106	69.4	1.8
	Air burst	324	125	38.5	
1000	Ground burst	254	191	75.1	1.92
	Air burst	539	211	39.2	
5000	Ground burst	794	726	91.4	1.95
	Air burst	1720	804	46.8	

Note: SHOB of ground burst is 0, and SHOB of air burst is 120
Scaled height of burst, or SHOB for short, is the ratio of cube roots of the height of the explosion (meter, m) and equivalent of the explosion (kiloton, kt). SHOB of ground burst is 0–60, and SHOB of air burst is 120–250

sumed by the entire space of propagation, which is why the increase in the injury and death zone is much more obvious than that of the injury and death radius.

Due to different explosion methods and equivalent, the percentage of total injury and death zone occupied by blast injury and death zone varies hugely. The book *Trauma War Wound Pathology* mentions that when TNT equivalent is below 10 kiloton, blast injury and death zone usually account for a relatively small proportion of total injury and death zone in a ground burst at less than 30%, while that figure may expand to 30–50% for an air burst. However, when TNT equivalent rises above 10 kilotons, blast injury and death zone usually account for 60–90% of total injury and death zone in a ground burst, while that figure may increase to as high as approximately 97.08% for an air burst (for details please, see Table 3).

4.2 Radii and Areas of Various Injury and Death Zones of Nuclear Blast Wave

The radii and areas of various injury and death zones of nuclear blast wave need to be calculated using explosion method and equivalents, the radii and areas of various injury and death zones, as well as their proportion of total areas, differ. Injury and death area may be calculated using injury and death radius, and therefore, the injury and death range of shock wave may also be defined.

Injury severity of personnel lessens if the farther they are from the center of the blast. In general, the state of the injury in injury and death zones may be categorized as extremely severe, severe, moderate, or minor based on severity of injury induced. In actual application, usually the following ratios are used to determine area of injury and death zones:

Extremely severe injury and death zone accounts for roughly 15% of total injury and death, severe injury and death zone around 10% of total, moderate injury and death zone around 25% of total, and minor injury and death zone around 50% of total. The reason why extremely severe and severe injury and death zones occupy relatively small proportion, while moderate and minor injury and death zones account for a relatively larger share, is because the pressure necessary to cause blast injuries attenuates much more rapidly at close distances and declines slower at farther distances.

4.3 Ratios of Different Injury Severity Levels in Different Injury and Death Zones of Nuclear Explosion

Speaking of ratios of injury severity levels, first and foremost it is necessary to consider the demarcation of the various shock wave injury and death zones. Demarcations are decided by which mainly based on comprehensive judgment in accordance with pressures actually measured and the farthest distance where a certain level of injury occurred. It is commonly accepted that the area with peak overpressure exceeding 1 kg/cm^2 is roughly equal to the shock wave's extremely severe injury and death zone, area with measurement between 0.6 and 1 kg/cm^2 would be severe injury and death zone, area with measurement between 0.4 and 0.6 kg/cm^2 would be moderate injury and death zone, and area with measurement between 0.2 and 0.4 kg/cm^2 would be minor injury and death zone. However, experiment data indicate that within the same injury and death zone, there are marked variations in blast injury severity. For example, when calculations are made using peak overpressure, there are different proportions of injury severity levels within an area in the same peak overpressure range, as shown in Table 4.

5 Section Five: Clinical Manifestations of Nuclear Blast Injury

When a nuclear weapon is detonated, its nuclear energy is chiefly transmitted to the surrounding zone in three methods: shock wave, thermal radiation, and nuclear radiation, with energy transmitted by shock wave making up approximately 50% of total energy. Therefore, the shock wave accounts for the majority of injuries caused in a nuclear explosion.

5.1 Pattern of Nuclear Blast Injury Severity

5.1.1 High Number of and High Severity Level of Primary Blast Injuries

The injuring mechanism of blast wave means that the majority of injuries caused by the overpressure and negative pressure of nuclear blast wave are closed injuries (injury to internal organs), hence the high occurrence of primary blast injury. During a nuclear explosion, the shock wave overpressure generated is high, positive pressure effective duration is long (several tens of milliseconds to a hundred milliseconds) and area of effect is vast, therefore producing a higher number and bigger ratio of severe primary blast injuries than non-nuclear explosion. Some features of primary blast injuries include minor external wounds with serious internal injuries. Usually there are not many wound openings on the surface of the body or even the lack thereof, but very severe injuries involving multiple internal organs including hemorrhage and edema of body parts such as the lungs, heart, auditory apparatuses, and organs inside the abdominal area.

On-site nuclear explosion experiment outcomes indicate and verify these characteristics. During an experiment involving the ground burst of a 10 kiloton nuclear weapon, there were 23 dogs distributed across the open ground that

Table 4 Different proportions of injury severity levels within different areas with the same peak overpressure range

Overpressure value (kg/cm)	No injury	Mild	Moderate	Severe	Extremely severe
<0.2	45	35	15	5	–
0.2–0.4	10	50	25	15	–
0.4–0.6	5	40	25	25	5
0.6–1.0	5	25	25	25	20
>1.0	–	5	5	35	55

suffered blast injuries, mostly primary blast injuries in the form of closed internal organ injuries. In three experiments featuring megaton nuclear weapons, of the 169 dogs distributed across the open ground that suffered blast injuries, 143 had closed injuries (primary blast injuries), and only 26 dogs showed body surface or soft tissue open wounds after being hit by sand grains and rocks.

5.1.2 High Occurrence Rate of Secondary Blast Injuries

Nuclear blast waves can cause secondary blast injuries at much farther distances than primary blast injuries. Dynamic pressure from a nuclear blast wave may cause people to fall or be tossed, leading to collision injuries, bone fractures, dislocations, tears, and other injuries. Dynamic pressure could also launch glass shards, rocks, bricks, wood splinters, and other objects that may inflict projectile injuries on people. Buildings and fortifications that fell by the dynamic pressure could hit and crush victims, and there may also be other secondary blast injuries in forms such as burns and radiation injuries. Information about post-atomic bomb explosion status in Japan during World War II indicate that among the open wounds, contusions and joint dislocations tallied, those that can be confirmed as secondary (indirect) blast injuries, respectively, accounted for 80%, 68%, and 55% of total, while those categorized as direct blast injuries, respectively, made up only 1%, 5%, and 3% of total.

5.1.3 High Occurrence of Multiple Injuries and Multi-Part Injuries

Due to the large range of effect of a nuclear blast wave, it can create a "plane" of lethal effects because the shock wave forms a "plane" that "wraps" up the body from head to toe and inflicts identical or similar injuring effects on different parts and organs of the body. Therefore, in a nuclear explosion, it is common for victims to suffer from multiple injuries affecting multiple body parts.

In the nuclear explosion experiment involving 169 dogs, on average each animal suffered injuries to 2.5 body parts or organs, and the number was higher for animals with extremely severe injuries, averaging 5.43 of body parts or organs injured. For dogs with severe, moderate, and minor injuries, the average numbers of injured body parts or organs were, respectively, 2.63, 1.60, and 1.18.

5.1.4 Many Combined Injuries

There are numerous biological injuring factors in a nuclear explosion, including shock wave, light radiation, and nuclear radiation, among others. Compared with other types of blast injuries, nuclear blast injuries produce relatively higher ratio of combined injuries, including some that are more unique.

Due to the radioactive contamination from a nuclear explosion, there is a very high ratio of blast injury victims also afflicted with unique radiation injuries. Moreover, since the heat released at the center of the explosion of a nuclear weapon is much higher than those generated by other weapons or explosives, it is also very common to see burns and burn-blast combined injuries from a nuclear explosion.

Data on victims that survived 20 days after the detonation of atomic bombs over Japan during World War II indicate that radiation-blast composite injuries, radiation-burn-blast combined injuries and burn-blast composite injuries, respectively, accounted for 45.0%, 12.5%, and 15.0% of injuries from the bombing of Hiroshima, while the radiation-blast combined injuries, radiation-burn-blast composite injuries, and burn-blast combined injuries, respectively, accounted for 54.8%, 11.9%, and 9.5% of injuries from the bombing of Nagasaki.

6 Section Six: Treatment Principles for Nuclear Blast Injuries

During a nuclear explosion, other than the shock wave's direct kill radius, there would be a larger area of injury and death outside, with bodily injuries arising as a result of the shock wave such as destruction of buildings on the ground surface, remnants of walls, glass shards, and protruding reinforced concrete. In terms of treatment of nuclear blast injuries, the first priority is to look for the victims themselves, especially those buried under collapsed fortifications and buildings. After rescuing victims, take steps to avoid suffocation, and for those with difficulty breathing, check to see if their mouth or nose is plugged up by dirt, clean promptly if so. For unconscious victims, pull his or her tongue out of the mouth and lay the person down in a lateral position. For victims with large volumes of bloody foamy fluid discharges from the nose or mouth, or extremely severe breathing difficulty, promptly perform trachea intubation or tracheotomy, and clean out secretions inside to ensure unobstructed airway. Administer the appropriate treatments to those with external wounds. If there is external bleeding, use whatever device or material available to stop bleeding and dress wound in a timely manner. Apply temporary fixation to fractured bones, apply simple dressing for open wounds, and provide pain killers to those with external wounds or other injuries

that cause unbearable pain. After confirming the injured body part(s) of a victim, provide specific treatment based on the part(s) afflicted with blast injury, in manners similar to treating regular trauma. For victims with plain eardrum rupture but no infection, and if surface area injured is less than 80%, dry treatment may be adopted. Refrain from applying medicine to or rinsing the external auditory canal without thorough considerations to avoid infection. The injuries in the majority of these victims would heal on their own, but eardrum repair may be required in others. For victims with injury in the abdominal area, it is necessary to identify the specific part(s) injured, and refrain from using pain killers to avoid covering up possible problems. Other than emergency surgeries, operations involving general anesthesia using inhalation anesthetics should be delayed until 24–48 h after injury.

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Injuries from Conventional Explosive Weapons

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1 Section One: Types of Conventional Explosive Weapons

Explosion is an extremely quick process of physical or chemical energy release. In other words, it is the transformation of energy from one form into one or more other forms within a relatively short span of time and within a certain space, and this conversion process is accompanied by powerful mechanical effects. The explosion of the common explosive is the conversion of chemical energy into mechanical energy, while nuclear explosion is the conversion of nuclear reaction energy into mechanical energy. Explosive weapons that utilize common explosives are called conventional explosive weapons, while explosive weapons that use nuclear reaction energy are called nuclear weapons. In terms of conventional explosive weapons, based on method of effect and intended targets, one system of categorizing such explosive weapons include lethal and blast weapons, anti-armor and anti-structure weapons, thermobaric weapons, and simple explosive weapons.

1.1 Lethal and Blast Weapons

The so-called lethal and blast weapon category refers to weapons that include both lethal and blasting functions, able to destroy structures using shock wave, and at the same time capable of injuring or killing living targets through the production of vast amount of fragments. It is a type of weapon with broad application scope. Lethal and blast weapons are divided into the lethal subcategory and blast subcategory munitions. The so-called lethal munition is a kind of "thick casting, small filler" munition, with the aim to produce a large amount of metal fragments upon explosion to maximize lethality against living targets. Blast munition meanwhile is a kind of "thin casting, big filler" munition, with the purpose of generating a powerful shock wave upon explosion to destroy enemy fortifications and structures.

Lethal and blast weapons could be something as big as missile warhead, cruise missile, rocket and artillery shell, or as small as a hand-thrown grenade. Common lethal and blast weapons include grenades, shells for artillery and cannons of various calibers, rockets, cruise missiles, and so on. The key to the technical performance of lethal and blast weapon is to simultaneously enhance both lethality and blast power. The former chiefly relies on improving performance of fragments. The earliest fragmentation weapons had metal casings that shattered into a small number of fragments in different sizes. Later, grooved metal casings were invented to produce even-sized fragments upon explosion. An even more complex munition would be loaded with tiny metal balls, and this composition struck and effective balance between density and lethality in terms of the quantity, scattering direction, and velocity of "fragments." In other words, the number of fragments was maximized upon the premise of ensuring that each fragment had just enough power to kill or injure a target.

Therefore, lethal and blast weapons mainly kill or injure through both fragments and shock wave.

1.2 Anti-Armor and Anti-Structure Weapons

Anti-armor and anti-structure weapons are subdivided into weapons specializing against armor and designed for fortifications and structures. The two subcategories both rely on kinetic energy, but they differ in terms of the mechanisms behind their respective penetration and their intended targets.

Anti-armor weapons may be further divided into either armor-piercing munition or high-energy anti-tank munition based on their armor penetration principle.

Armor-piercing munition is the typical kinetic energy munition, and primarily relies on the munition's massive

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kinetic energy (including the munition's strength, weight, velocity, etc.) to force its way through and defeat the target's armor plating. Armor-piercing round is characterized by high initial velocity, long direct distance and high precision, and it is the main type of munition in tank guns and anti-tank guns. Modern armor-piercing rounds have very sharp tips and long and narrow bodies made from high-strength materials such as steel alloy or depleted uranium alloy.

Armor-piercing munition is the product of the dilemma between armor and armor-piercing weapon, and first emerged on battlefields as early as the nineteenth century. At first it was designed to defeat armored warships but application was limited. With the advent of the tank during World War I, armor-piercing munitions were used more extensively, and with wide usage came performance improvements. Armor-piercing munition during this period were "suitable caliber armor-piercing rounds," meaning that diameter of the penetrator and the caliber of the shell were the same. This type of armor-piercing round is also known as "common armor-piercing round." Different common armor-piercing munitions with different tips have different names, such as sharp-tipped armor-piercing round, blunt-tipped armor-piercing round and capped armor-piercing round. The first two types were mainly deployed against homogeneous armors, while the latter, outfitted with ballistic cap and regular cap, has greater penetration and may be used against heterogeneous armors that had undergone surface hardening enhancement.

In the 1990s, as tank gun technology advanced by leaps and bounds, various types of armor-piercing shells fabricated from tungsten alloys and depleted uranium were used in real battles, and these armor-piercing rounds showed much-improved penetration capacity. One hundred and five millimeter special alloy armor-piercing shells could defeat 580 mm-thick homogeneous steel armor plating from a distance of 2000 m; 125 mm tungsten alloy fin-stabilizing discarding sabot armor-piercing shells could defeat 850 mm-thick homogeneous steel armor plating from a distance of 2000 m, and from the same distance 125 mm special alloy armor-piercing shells could defeat 960 mm-thick homogeneous steel armor plating; tungsten alloy armor-piercing shells could defeat 800 mm-thick homogeneous steel armor plating, and special alloy armor-piercing shells could defeat 900 mm-thick homogeneous steel armor plating.

After an armor-piercing round perforates the target vehicle's armor, it could cause severe injury to passengers inside through kinetic energy of remains of the round, secondary fragments created during the armor-penetration process, and burns from vehicle's fuel ignited by the high-speed kinetic energy.

High-energy anti-tank (HEAT) munitions are also known as "explosive core anti-tank munitions," and they rely on superplastic jet of hot metal formed by the explosion of the shaped charge at the core to perforate armor plating. It is otherwise called "shaped charge anti-tank munition" and is also one of the main types of anti-tank munition. High-energy anti-tank round is a type of anti-armor munition based on chemical energy and the Munroe effect. Specifically, the shaped charge (often times semi-spherical, funnel-shaped, among other configurations) inside the conical shell is detonated a certain distance away from armor plating, forming a focused superplastic jet of hot metal that can penetrate vehicle armor and cause damage to passengers and equipment inside. Thus, this type of charge also has numerous other names like conical charge, shaped charge, hollow charge, or energy-focusing charge. Through proper designs in charge shape and detonation distance, and the addition of a metal liner inside, the modern HEAT round has static armor penetration depth at more than five times the diameter of the metal liner. Penetration depth increases with the increase in metal liner diameter, but penetration depth improvement diminishes once metal liner diameter exceeds 150 mm. Some modern anti-tank missiles capable of producing penetration depth more than 1000 mm rely on warheads in a tandem configuration, and they perform better against explosive-reactive armors.

The invention of the HEAT munition came about with the nineteenth century discovery of the focus of blast energy created when an explosive is hollowed. During the early phase of World War II, it was discovered that when the void in the explosive is lined by a thin metal cap, the charge's armor penetration capacity enhanced dramatically, and this realization greatly expanded the application of shaped charge. In the Spanish Civil War fought between 1936 and 1939, the intervening German military became the first to adopt widespread usage of HEAT munition.

When a HEAT round detonates, the compressed metal liner would focus together, and is violently forced outward from the center of the conical bottom. Due to the tremendous pressure created by the blast wave, usually upward of 100 atmospheric pressure, the hot jet formed from the charge's metal liner would shoot out at a speed of 8000–9000 m/s.

After defeating the target vehicle's armor protection, the main injury factors of the munition include the superplastic metal jet, blast wave, secondary fragments created during the armor-penetration process, and burns from vehicle's fuel ignited by the high-speed kinetic energy.

Anti-structure weapons primarily refer to the type of munition designed to strike against fortified structures, as exemplified by the bunker buster. Bunker buster is a type of hardened penetration munition mainly deployed to strike reinforced surface targets or underground facilities. The main feature of the bunker buster is that it will not explode right away upon hitting the ground, instead it continues to burrow beneath the surface, and then detonates after pene-

trating a certain depth, so as to achieve the goal of destroying targets buried and protected underground. Bunker busters could obliterate shelters or labs hidden underground, and cause widespread injury and destruction to personnel and equipment within these subterranean facilities. On May 8, 1999, the Joint Direct Attack Munition (JDAM) that blew up the Embassy of China in Yugoslavia was carrying a bunker buster.

A bunker buster usually consists of two parts, the body and the penetrator. Based on specific functions, bunker busters may be subdivided into the categories of anti-runway munition, anti-surface installation munition, and anti-underground fortification munition, and based on principle of explosion, bunker busters may be subdivided as nuclear bunker buster or conventional bunker buster. Conventional bunker busters are most widely used at present.

After a bunker buster penetrates into the target subterranean building, it inflicts injury and damage through a complicated mix of factors including powerful shock wave, debris and munition fragments, powerful shock vibration and burns, among other causes.

1.3 Thermobaric Weapons

Thermobaric weapon is a new type of weaponry that uses both air and explosives to create high temperature and pressure, hence the term "thermobaric" derived from Greek words for "heat" and "pressure." Thermobaric weapon is derived from aerosol bomb (fuel-air explosive), and could be considered an augmented development of the aerosol bomb.

Development for thermobaric weapons commenced in the 1980s, designed to deliver tactical strikes against cave-like fortified installations underground. This kind of munition can penetrate through relatively thin layer of rocks above the installation or enter cave entrance exposed to the outside, allowing the warhead to penetrate into the interior of underground facilities before exploding. Such a setup enables the bomb to annihilate the entire underground bunker system, maximize casualties of personnel within, and completely wipe out weapons and equipment stored inside.

The US military began the research and development of thermobaric weapons in the early 1980s, and the most iconic large air-fuel bomb is the massive ordnance air blast bomb, shortened as MOAB and thus colloquially known as the "Mother of All Bombs," that weighs 21,000 lb or 9450 kg. At 4 pm on February 13th, 1991, the Amiriyah shelter in Baghdad was penetrated by a GBU-28, and 1100 Iraqi hidden inside were all killed. Russia also secretly developed large vacuum bombs, colloquially referred to as "Father of All Bombs." Said thermobaric device weighs a total of 7100 kg and has a yield equivalent of 44 tons of TNT.

Thermobaric munitions combine the features of both high-energy explosive and fuel-air weapon, and may be considered a high-energy explosive loaded with fuel. Its velocity of detonation usually fluctuates around 3–4 km/s, so a lot slower than that of conventional high-energy explosives (8 km/s). At the same time, before detonated it sucks up a large amount of oxygen from its surrounding, creating an anaerobic environment. A key element of thermobaric munition is the addition of powdered form of substances like aluminum, boron, silicon, titanium, magnesium and zirconium in the explosive. These powders would ignite and release a vast amount of energy when heated, considerably augmenting the incendiary effects and pressure of the thermobaric explosive.

When thermobaric explosives blow up, the process mainly involves the following reactions:

1. Anaerobic explosion reaction: This type of reaction does not require sucking in oxygen from the surrounding air, and the process lasts one millionth of a second or so. It is primarily a redox reaction in molecular form. This step releases a certain portion of energy and generates a great amount of products rich in fuel.
2. Anaerobic combustion reaction: This type of reaction also does not require sucking in oxygen from the surrounding air, and the process lasts one ten thousandth of a second or so. It is mostly the combustion of fuel particles.
3. Post-explosion aerobic combustion reaction: This step requires sucking in oxygen from the surrounding air, and the process lasts one thousandth of a second or so. It is chiefly the combustion of fuel-rich products with the surrounding air. This process releases a tremendous amount of energy, extends the effective duration of high-pressure shock wave, and continually enlarges the fireball. The three aforesaid reactions constitute the basic function of the thermobaric weapon: The initial anaerobic explosion reaction establishes the munition's high-pressure effect and destructive force against objects. The post-explosion anaerobic combustion reaction stabilizes its high pressure and establishes the munition's penetration against objects, while the post-explosion aerobic combustion reaction establishes its high-pressure impact and incineration effects, which translate into injury and damage against personnel and objects. Through adjusting the ratios of the compositions in the charge of a thermobaric weapon, the three aforementioned reactions may be altered so that the munition would perform specifically as intended.

Based on the three reactions stated above, a thermobaric weapon causes harm to personnel through the three following factors: First of all is the blast wave. This is the predomi-

nant factor that injures and kills people. Second of all is burns from high temperature and heat radiation. This factor may worsen the injuries inflicted on people by the shock wave, and could also burn people on its own. Third of all is the lack of oxygen or suffocation. In the limited and enclosed space of structures such as caves, this effect is particularly obvious.

1.4 Improvised Explosive Device

An improvised explosive device, or IED, usually refers to an explosive device made from equipment and materials easy to obtain such as destructive, lethal, toxic, smoke-producing and incendiary substances, made to achieve aims such as causing destruction, injuring people, and creating chaos and suffering. IEDs are characterized by properties such as easy to obtain, simple to produce, and clandestine deployment and usage, among others. An IED is an asymmetric warfare weapon, and it has become one of the terrorizing and dangerous weapons among terrorists and extremists as they threaten both civilians and soldiers anywhere on the planet. Commercial, military, home-made materials and equipment, and munitions or munition parts may be utilized to make an improvised explosive device. Low cost, easy concealment and material availability are some of the main reasons why IEDs are so popular. Improvised explosive devices may be detonated through more than 90 ways, and most of the time they functions like traps and mines, making them hard to detect and allowing them to strike unprepared victims. Since the War in Iraq, armed groups in Iraqi territory have used IEDs to inflict great harms to US military troops deployed and stationed there. According to statistics from the US Department of Defense, during the War on Terror in Iraq, more than 3100 American troops have been killed by improvised explosive devices (accounting for 69% of the 4471 total casualties), and the number of soldiers injured by IEDs even surpassed the 33,000 mark.

The first time that by improvised explosive devices were deployed in large scale was World War II, during the railway war in Belarus between the Belarussian resistance and Nazi Germany. Between 1943 and 1944, the Belarussians IEDs equipped with remote detonation and delayed fuse to destroy more than a thousand Germany trains. During the Vietnam War, the Viet Cong used IEDs to attack enemy vehicles, vessels and personnel on land and water. In many cases, the Viet Cong fashioned IEDs from American military equipment and materials that did not detonate or fire. Thirty-three percent US military injuries and 28% casualties in Vietnam resulted from mines. These numbers also include both mines made from improvised explosive devices and manufactured by factories.

Improvised explosive devices could be made from any type of materials and detonator. This sort of "homemade" device intends to use explosive, sometimes with the addition of toxic chemicals, bio toxins or radioactive substances, to injure and kill. IEDs may be produced based on functions, containers and delivery methods, so they come in a diverse range of forms. IEDs can be armed with commercial or military explosives, homemade explosives or military equipment or equipment components.

IEDs are unique because they are made by the user with whatever resources available at hand. IEDs are increasingly difficult to discover and defend against because production techniques are advancing and belligerents have learned to customize their devices to attack specific targets or types of targets. IEDs are mainly divided into three categories, namely assembled IED, car-borne IED and suicide IED.

Cars and people are the primary targets in IED attacks. IED attacks are characterized by the following features:

1. High rate of injury: The injury/death ratio caused by IEDs was 2:1 during World War II, 2.6:1 during the Korean War, 3:1 in the Vietnam War and up to 9:1 during the War in Iraq.
2. Tactical surprise: On the battlefield, the rumbles of armored vehicles or the sounds created by falling artillery shells would alert people to the upcoming/incoming strike. However, the sudden explosion of an unexpected IED is more like a tactical surprise in the sense that victims will not even know what hit them, or where the enemies are.
3. Strategic influence: Terrorists often film the entire IED attack, and broadcast the recording online or through electronic media to terrorize American military and citizens from a psychological perspective and destabilize the will and determination of the American War on Terror.
4. Low cost and ease of concealment of improvised explosive devices make them hard to detect during the course of production and attack.

2 Section Two: Main Injuring Factors of Conventional Explosive Weapons and Their Injuring Mechanisms

The main injuring factors of explosive weapons against people include primary shock wave, secondary fragments, dynamic pressure, impact vibration, burns, inhalation injury and radiation injury. The first five are the main injuring factors of conventional explosive weapons, as radiation injury is usually inflicted by nuclear weapons. Blast injury predominantly comes from overpressure of the primary shock wave, and the most susceptible organs are eardrums, lungs, stom-

ach, intestine and other organs containing air or with cavity. Secondary fragments include fragments from the munition itself, and other projectiles launched by the explosion such as rocks. Any part of the body may be injured, and severity depends on kinetic energy of the fragment. Dynamic pressure mostly throws and tosses people, then inflict collision injury when the body collides against another object. Impact vibration injury is closely related to the acceleration of vibration and the conditions of the platform on which the body was located. In addition, flames and smokes started by the explosion could burn people, including inhalation injury caused by breathing in hot air.

2.1 Injury and Death Mechanisms of Fragments

Fragments injure organisms mostly because of the fragment's kinetic energy, as kinetic energy drives the fragment into tissue of the organism. When a fragment pierces the tissue of an organism, there are two forces in action. One is the forward momentum that causes the fragment to advance forward along its flight path, directly damaging tissue, resulting in penetrating wound and/or perforating wound, and leaving behind a permanent wound passage. This is the main injuring factor of direct tearing injury. Furthermore, the moment a high-speed fragment collides with the surface of the body, under the force of forward momentum, a massive shock wave may be created and worsen injury to body tissues. Second of all is the lateral force, which acts perpendicular to the wound passage and mainly propagates around the wound passage in the form of stress wave. Under the powerful stress wave of the lateral force, pulsating transient cavity would form in a process that may possible injure surrounding soft tissues and bone tissues.

2.1.1 Effects of Direct Tearing Injuries from Fragments

When a fragment hits body tissue, when the stress acting on the local tissue exceeds the tissue's tolerance, the tissue would be injured because of being ripped and torn.

2.1.2 Effects of Hydrodynamics and Accelerated Particles

French scholar Hugier asserted in 1848 that the "explosion" effect of a projectile on body tissue is the effect of water particle dispersion. When the fragment acts on the body, its kinetic energy may also be passed to the liquid particles in the surrounding tissues, accelerating them. These accelerated particles would move like secondary fragments and quickly leave the wound passage, creating an "explosion" effect affecting the surrounding, leading to widespread injury in the neighboring tissues.

2.1.3 Transient Cavity Effect

The formation of transient cavity is an important characteristic of injuries caused by high-speed fragments. When a fragment passes through body tissues at high speed, the soft tissues around the wound passage would expand outward due to the effects of pressure (upward of 10 MPa), and could form a cavity 10–30 times bigger than the projected fragment itself, but this cavity would only exist for several milliseconds. Transient cavity is the physical phenomenon of rapid change that occurs inside tissues after a high-speed projectile passes through body tissues, and also one of the primary causes of severe tissue and organ injury.

A transient cavity is not only much larger than the projectile itself in terms of size but also has a pulsating period marked by a rapid expansion and then contraction. Pressure within the transient cavity is highest during its initial phase of formation, and when the transient cavity bulges to its greatest size, pressure drops to the lowest value and creates negative pressure. The periodic expansion and contraction of a transient cavity not only produces a powerful stress wave that could propagate within the body and injure adjacent and nearby tissues and organs but also causes severe infection because of the suction generated by the negative pressure within the cavity.

The size of a transient cavity depends on the energy passed from the projectile to the tissues, as well as the physical and mechanical properties of the tissues themselves. When a fragment flies at velocity below 340 m/s, basically it will not be able to create a transient cavity. Since different tissues in the human body are structured differently with varying mechanical properties, cavity effects created by fragments also differ. The cavity effect is relatively more obvious in tissues such as muscle, liver, and the brain. In muscle tissues, for 1 J of energy a transient cavity roughly 0.7 mL in volume would be created. However, in tissues with lower density (such as the lungs), or those with higher density (i.e., bones), transient cavity effects are not as obvious.

2.1.4 Effects of Shock Wave

The moment a high-speed fragment hits the surface of the body, it may generate a shock wave with peak pressure of around 10 atm. A strong shock wave is unique in that pressure at the wave front declines exponentially, and creates a negative pressure zone with pressure lower than atmospheric pressure. A shock wave propagates around the body at a speed of roughly 1450 m/s, but lasts for an extremely short duration, declining by half within 30 μs. Hence, it is generally believed that such type of shock wave does not cause much harm. Yet outcomes from experiments in recent years indicate that the overpressure of shock wave, particle acceleration and displacement can cause obvious body tissue injury. Especially the effects of overpressure, even when tis-

sue displacement can be avoided, overpressure of shock wave can still inflict obvious and serious internal injuries to cells.

The severity of injury caused by a fragment largely hinges on two factors: First of all is the injuring capacity of the fragment itself, and second of all is the anatomical physiological, and biomechanical properties of the affected tissues and organs. The former includes the fragment's own kinetic energy, structure, shape, flight stability, and other physical factors, while the latter includes density, elasticity, toughness, and viscosity of the tissue/organ; how much gas or liquid they contain; location of injury; and so on.

Kinetic Energy of Fragment

A fragment can injure the body because the fragment itself possesses kinetic energy. When a fragment collides with the body, it directly damages body tissues, and accelerates body tissues in contact with the fragment, in turn pulling or shaking nearby tissues, further adding to contusion and squeezing pressure. Therefore, kinetic energy is the prerequisite condition for causing damage to the body, and how much energy is transmitted to body tissues determine severity of injury. Kinetic energy equation is: $E_k = \frac{1}{2}mv^2$, and in this equation E_k E is kinetic energy, m is mass, and V is velocity. From the equation above, it can be seen that kinetic energy of the fragment mostly depends on its velocity and mass.

The velocity of the fragment includes its initial velocity, impact velocity, and residual velocity. The initial velocity of the fragment refers to the velocity at which the munition travels upon leaving the muzzle of the gun or cannon, or the maximum velocity given to the fragment by the explosion product after the munition explodes. Impact velocity refers to the velocity at the moment the fragment hits the target. Residual velocity refers to the velocity at the moment the fragment pierces through the target. An injury with a residual velocity of zero usually only has an entrance wound but not an exit wound, otherwise known as a penetrating wound.

Explosive fragments may travel at initial velocity anywhere from several meters per second to several km per second. Research outcomes from the past few decades indicate that fragments with small mass but high velocity are very dangerous. When fragment mass is the same, the higher the impact velocity, the bigger the impact kinetic energy, the larger the wound cavity created, the greater the amount of devitalized tissue to be removed, and the more severe the injury.

Steel pellet is the most common type of pre-formed fragments loaded into munitions. Firing 6.35 mm steel pellets at the hind legs of dogs to cause penetration wounds reveal that different impact velocities produce wounds that vary clearly in severity. At an impact velocity of 1300 m/s, naked eye observation could see wound passage filled with shards and blood clots, muscle tissues with a dark purple hue, large amount of tissue loss, extensive bleeding between fascicles, some hematomas that reached 80 mm in diameter, and contusion zones as wide as 15 mm. At an impact velocity of 450 m/s, muscle tissues inside the wound passage exhibit a bright red hue, fragmented tissues are relatively fewer, contusion zones are harder to distinguish clearly, and submuscular bleeding is rare. There may possibly be minor bleeding, and the amount of devitalized tissue is rather little.

Kinetic energy of a fragment is directly proportional to fragment mass. Due to inertia, the heavier the fragment, the farther it may travel in flight, and the more serious the injury it causes. Mass of the fragment also affects its energy transfer efficiency. The lower the mass of the fragment, the quicker the decline of its velocity. Thus, with the same impact energy, a fragment of a lower mass has a higher energy transfer efficiency. For example, a low-mass steel pellet with impact velocity of 1450 m/s would slow down to a speed of 40% upon piercing into tissues. When this kind of small fragment suddenly decelerates in tissue, a large amount of energy is released into an area of relatively short distance near the entrance wound, resulting in a shallow but wide wound passage.

Stability of Fragment

Pillar-shaped, spherical and triangular fragments are more stable in flight, but those that are obviously longer or wider along the length or width such as pillar-shaped fragments could easily lose stability in mid-flight due to drag, resulting in motions like yaw or spin. Yaw refers to fragment motion that deviates from the vertical axis of flight, and spin refers to the fragment spinning along its central axis while flying in the opposite direction.

An irregular fragment may possibly hit the surface of the body from different angles, leading to different injury severity and wound passage shape. When a fragment enters the tissue with a small contact surface area against the tissue, energy transmitted to the tissue would decreased, resulting in less severe injury. On the other hand, when contact surface area against tissue is large, more energy is transmitted and a more serious injury is inflicted.

Structural Features of Fragment

Usually irregularly shaped fragments have bigger drags and slow down quicker. When a triangular or square pierces the body, skin would be torn irregularly and an upside-down trumpet-shaped wound passage that is shallow and wide would be created, and penetrating wound occurs more than 80% of the time. Steel pellets usually create entry wounds that are circular with smooth edges and slightly larger than the diameter of the pellet. When such a projectile moves at velocity above 1000 m/s, entry wound surface area could be

ten times or more than that of the exit wound. Since steel pellets usually have smooth surface and high sectional density (cross-sectional area/mass), they often move along curved trajectories after entering the body, leading to multi-organ injuries.

Spherical fragments have relatively steady flight path and low ballistic coefficient (capacity to overcome air resistance in flight), meaning that they usually have larger drags and decelerate quickly. Thus, they are not very penetrative against tissue, mostly resulting in rather shallow wound passage and comparatively small transient cavity. Triangular fragments also do not fly very steadily and have big drag, but they are rather penetrative against tissues and cause deeper wounds. Experiment outcomes indicate that a 0.15 g steel pellet fired at the chest of pig at a velocity of 1100 m/s could not pierce through the pig's thoracic wall. However, an irregular fragment weighing a mere 0.07 g flying at a velocity of only 600 m/s or so can perforate the thoracic wall, lungs, and thoracic aortic wall of a test sheep/goat, resulting in serious, fatal bleeding of the test animal. This illustrates that structural feature of a fragment has a huge influence on the fragment's lethality.

Features of the Injured Tissue

When fragments have the same kinetic energy, consequences are more dire when vital organs such as the heart or brain are struck compared to non-vital organs like the limbs. Tissue thickness (wound passage length) has a great influence on injury severity. For example, spherical fragments usually open up rather shallow wounds, and thus, they usually do not cause as much damage to vital organs. Irregular fragments meanwhile are relatively penetrative and may cause much more harm to vital organs.

The injuring effects of a fragment is directly affiliated with tissue density, water content, elasticity, and other factors. The denser the tissue, the higher the water content, the lower the elasticity, the more serious the injury. Bone tissues are dense and inelastic, and fractures often occur when bones are struck by fragments. Comminuted fractures often occur with long bones, while holes with radial cracks are frequently punched through skulls, ribs, and long bone epiphyses. Muscles and brain tissues have high water content, which easily absorb energy, leading to serious injury. Liver, kidneys, and other solid organs have high density and low elasticity, and would often rupture when hit. Tissue loss amount would be identical to transient cavity, surrounded by radial cracks and fissures. Empty organs like stomach and intestines are filled with gas or liquid. The formation of transient cavity may possibly transmit energy through gas expansion or liquid conduction that could end up perforating a faraway body part, or injuring inner membrane. Blood vessels are elastic, and unless they are hit directly, usually they will not rupture, but inner membrane injury could be sustained due to being pulled and stretched, resulting in thrombus. Lung tissues have low density and high elasticity, and contain large amount of air. Thus, upon being hit by fragment, resulting transient cavity would be small and injury mild. Skin is highly elastic and consumes much energy of the fragment, which is why skin resistance against fragment penetration is approximately 40% greater than that of muscle.

2.2 Combined Injuring Mechanism of Shock Wave

2.2.1 Composite Shock Wave-Heat Injuring Mechanism

Thermal radiation is one of the major injuring mechanisms in an explosion, and hot air or heat energy in the air could be transmitted to respiratory mucosa or lung tissues and cause injury to the corresponding body parts. Airway mucosa may be injured when temperature of inhaled gas reaches 150 °C, and temperature at the site of explosion could rise up to a 1000 °C. Dry and hot air quickly cools down upon inhalation and may just injure upper respiratory tract mucosa; moist and hot air on the other hand transmits much more energy than dry air and cools down a lot slower, so it may cause severe airway, bronchus, and lung injuries.

Thermal radiation generated during the moment of explosion mostly targets the respiratory tract and lungs, and at the same time such air-containing organs are also susceptible to blast injuries. Therefore, when a shock wave is coupled with thermal radiation, lung injury may significantly worsen. The main injuring mechanisms are as outlined below.

Airway Obstruction

The upper respiratory tract is the first part to be injured by thermal radiation Throat tissues are loose and would rapidly swell after injury, possibly resulting in complete blockage of the upper respiratory tract within just 1 h. The lower respiratory tract may also become partially or wholly obstructed. Obstruction of the upper and lower respiratory tracts would clearly raise airway resistance, resulting in breathing difficulty, hypoxia, and carbon dioxide retention. The main reasons for airway obstruction include (1) hyperemia, edema, bleeding, necrosis, or increased seepage of airway mucosa, and necrotic and exfoliated mucosal fragments and inhaled dust particles could easily obstruct small airways; (2) bronchospasm; and (3) further constriction of the small airways due to interstitial pulmonary edema.

Decline in Lung Compliance

Lung surfactant is a phospholipid protein complex, which is secreted by alveolar type II epithelial cells. The surfactant, which has half-life of around 43–45 h, is evenly distributed on alveolar surface to maintain alveolar surface tension.

Excessive heat in the airway could damage alveolar type II epithelial cells, leading to drop in lung surfactant, shortening of surfactant half-life and increase in inactivation, thereby leading to decline in lung compliance. In addition, after airway is burned, interstitial pulmonary edema and alveolar edema may also cause a fall in lung compliance.

Rise in Lung Water Content

This is typical pathological expression of pulmonary injury arising from pulmonary edema. At the same time, a larger number of animal testing and clinical practice also prove that pulmonary edema may occur after inhalation injury. Generally speaking, interalveolar pressure and plasma colloid osmotic pressure serve to counter the entry of intravascular fluid into interstitium and alveoli. However, pulmonary collapse due to in lung surfactant reduction after inhalation injury of airway would lead to increased pulmonary capillary permeability and decreased plasma colloid osmotic pressure, among other issues that conduce pulmonary edema.

Pulmonary Ventilation/Blood Flow Imbalance and Increased Intrapulmonary Shunt

Pulmonary shunt in a normal person does not exceed 5%, but inhalation injury of airway always result in serious ventilation/blood flow imbalance, due to the main reasons listed here: (1) Increased physiological dead cavities. Hypoxia, stress and other factors raise peripheral vascular resistance and elevate pulmonary vascular resistance as well, decreasing pulmonary hypoperfusion and creating local invalid cavity-like ventilation. (2) Increased lung surface tension, bronchospasm and other issues lead to reduction in lung compliance and elevated airway resistance, resulting in decreased alveolar ventilation. (3) Atelectasis and pulmonary edema also raises intrapulmonary shunt, worsening hypoxemia.

Serious inhalation injury of the airway causes degeneration or necrosis of airway mucosa cells, cilia damage, hypopnea, and weak coughs, greatly lowering the ability of the airway to expel sputum and remove bacterial and foreign substances. Large amount of exfoliated mucosa and exudated substances form ideal culture medium for bacteria. Moreover, inhalation injury of the airway causes obvious decline in bodywide immunity system, and hence pulmonary infection is basically inevitable.

Studies in recent years show that some cells such as neutrophil polymorphonuclear granulocyte (PMN), alveolar macrophage, and monocyte, and their secreted inflammatory mediators play crucial roles in the pathogenesis mechanism of airway inhalation. After inhalation injury of the airway, a large amount of PMN accumulate in, infiltrate and degranulate the lungs, releasing oxygen radicals, myeloperoxidase (MPO), elastase (Ela), leukotriene B4, platelet-activating factor (PAF), thromboxane (TXA2), and other substances that damage tissues. For example, an excessive amount of oxygen radicals may damage vascular endothelial cells and basement membrane while increasing capillary permeability; destruction of lung interstitium and inhibition of protease activity would intensify the damage of protease to the lungs; damages to alveolar type II epithelial cells affect production of lung surfactants; induced membrane peroxidation not only causes damage but also speeds up the metabolism of arachidonic acid, with the two working together to injure pulmonary vessels. Through animal testing and prospective clinical study, the Army Medical University (formerly Third Military Medical University) discovered that TXA2 and prostacyclin (PGI2) also participate to the onset and growth of burn pulmonary edema. The underlying mechanism may be formation of microthrombus due to platelet accumulation in pulmonary microvessels, blocking microcirculation and releasing medium such as ADP, histamine, 5-hydroxytryptamine, and bradykinin, leading to raised pulmonary vascular permeability and pulmonary edema.

2.2.2 Composite Shock Wave-Hypoxia Injuring Mechanism

Wang Jianmin and Chen Jing

Following the development of modern high and new technology weapons, a new variety of weaponry that generates explosion and oxygen shortage such as fuel-air bomb and thermobaric munition are being gradually deployed in actual battles, resulting in the increasing frequency of composite shock wave-hypoxia injuries. The main target organ of composite shock wave-hypoxia injury is the lungs because on the one hand, since the lungs are the main targets of the effects of shock wave, and on the other hand, lungs are also the most susceptible to hypoxia.

A large number of studies indicate that high-plateau, low-oxygen environment may significantly lower the injuring or fatal threshold of shock wave. Damon et al. reported that under regular pressure condition (82.8 kPa) at near sea-level, when dogs and goats were subjected to shock wave, reflected overpressure required to cause 50% death after 1 h of injury was respectively 370.53 kPa and 365.01 kPa. However, when ambient pressure is lowered to 48.30 kPa, when dogs and goats were subjected to shock wave, reflected overpressure required to cause 50% death after 1 h of injury was respectively 215.97 kPa and 173.88 kPa, indicating that in low-oxygen conditions animals are clearly less tolerant of shock wave. Bowen et al. estimated people's tolerance of air shock wave based on experiment data. During the long effective duration of the instantaneous rise in the shock front, the aver-

age overpressure required to kill a person at sea level is 424.35 kPa, but when ambient pressure lowers to 34.5 kPa, on average it only requires an overpressure of 153.8 kPa to produce lethal effect. Yang Zhihuan et al. carried out blast injury experiment on 90 rats under various ambient pressures, and research outcomes indicate that as ambient pressure decreases, shock wave fatality rate significantly increases. At shock wave incident overpressure of 190.4 kPa and overpressure effective duration of 10 ms, there was not a single case of animal death in the normal atmospheric pressure group (96.60 kPa). However, the ambient pressure is decreased to 53.99 kPa (equivalent of a plateau at altitude of 5000 m) and 61.33 kPa (equivalent of a plateau at altitude of 4000 m), the same shock wave overpressure strength and duration would respectively produce animal fatality rates of 35.0% and 25.0%. It was also discovered at the same time that the lower the ambient pressure, the more severe lung injuries, as seen in worsened levels of pulmonary hemorrhage and pulmonary edema, and elevated lung-body index.

The mechanism behind the aggravation of composite shock wave-hypoxia injury may be associated with a multitude of factors. First and foremost, oxygen shortage may raise pulmonary capillary brittleness and permeability, and upon exposure to shock wave, pulmonary capillaries are more prone to rupturing and bleeding, while higher permeability also increase fluid seepage, in turn leading to pulmonary hemorrhage and pulmonary edema more severe than normal oxygen supply conditions. Second of all, a low-oxygen environment is also a low-ambient pressure environment in most cases. Shock waves in such low ambient pressure conditions produce more obvious pressure difference than normal oxygen supply conditions, leading to lowered shock wave tolerance in organisms.

The mechanism behind the hypoxemia's aggravation of blast injury may also be related to multiple neurohumoral factors.

Oxidative stress response induced by hypoxia may worsen pulmonary injury. Oxidative stress response refers to excessive production of highly active molecules such as reactive oxygen species (ROS) and reactive nitrogen species (RNS) in the body when the subject is stimulated by various types of harmful stimuli. When oxidation exceeds oxide removal, the outcome is imbalance in the oxidation/anti-oxidation system, leading to the pathogenic process that injures body tissues. A huge number of experiment and clinical research outcomes indicate that hypoxia-induced oxidative stress response is closely linked to hypoxia-induced pulmonary injuries.

Higher expressions of nitric oxide synthase and hypoxic inducible factor promoted by oxygen deficit also intensify pulmonary injuries. Nitric oxide synthases (NOS) are crucial to modulating vascular tension, of which, endothelial nitric oxide synthase (eNOS) and inducible nitric oxide synthase (iNOS) modulate the synthesis of nitric oxide (NO), which is a key factor participating in hypoxia-induced oxidative stress response. Therefore, NOS could worsen pulmonary injuries caused by shock wave through induced oxidative stress response.

Similarly, inflammatory response induced by hypoxia also aggravates pulmonary injuries. Inflammation is a pathological process frequently seen in clinical diseases, and may occur in any part of the body and in different organs. Usually when the body is subjected to an inflammatory agent, degeneration and necrosis of local tissues and cells may occur on the one hand, and on the other hand the body's resistance to diseases would be induced to increase in order to remove the inflammatory agent, restore damaged tissues and reachieve balance in the body's internal environment, and balance between the body's internal environment and the external environment. Other than oxidative stress response, studies at the molecular, cellular, and clinical levels have shown that oxygen shortage and inflammatory response are closely related to each other.

In summary, lack of oxygen can induce oxidative stress response and inflammatory response in the body, and these two reactions are crucial steps that occur relatively early during the hypoxic lung injury process. ROS and NOS, two key substances in oxidative stress response, may induce more oxidative stress response and bodily harms, while the rise in inflammatory agents may hasten the occurrence of hypoxic disorder.

3 Section Three: Epidemiological Features of Normal Blast Injuries

Since countries around the world have tightened measures to control nuclear weapon development, the deployment of nuclear weapons in conventional warfare is rigorously restricted. At the same time, civil nuclear energy industrial facilities are soundly protected and monitored, which is why blast injuries resulting from nuclear explosions are rare.

In the development of modern conventional weaponry, explosive weapons is the most marked and has been advancing at an extremely rapid pace. From regular artillery shell, bomb and land mine, to high-explosive squash-head (HESH) munition, shaped charge, shock wave-enhanced weapon (i.e., fuel-air bomb, etc.) and others, the blast waves they produce are increasingly powerful, and have become one of the determining factors in the destruction of equipment and structures, and injury and casualty of personnel. In other words, conventional explosive weaponry has become one of the main and important sources of injuries in modern and future warfare.

3.1 Characteristics of Shock Wave of Conventional Explosive Weapons

The most common injuring factors of conventional explosive weapons are shock wave, fragments, high temperature and superplastic jet, among others, but different types of explosive weapons are characterized by different injuring factors and efficiency. Take for example land mine and artillery shell, their chief injuring factor is fragment, and at the same time shock wave also plays a rather significant role in killing and wounding targets. Lethal and explosive munitions increase the quantity and velocity of fragments, resulting in a higher proportion of fragmentation injuries, meaning that fragments are its absolute number one injuring factor, but of course they could still inflict blast injuries. Shock wave-enhanced weapons such as fuel-air bomb are specially designed to augment the power of shock wave, so that shock wave is the primary injuring factor, so much so that analysis of US fuel-air bomb injury data during the Vietnam War reveals 50.4% of wounds created by such weapons may be attributed to blast injury. Thermobaric munitions meanwhile increased both the strength of shock wave and quantity of fragments, raising both the numbers of blast injuries and fragmentation injuries, and worsening their severity levels; therefore, composite injuries and multiple injuries also occur more frequently and to more serious degrees.

The detonation of a fuel-air bomb includes basically all the main injuring factors of conventional explosive weapons; therefore, in the next segment fuel-air bomb is used as the central subject in the analysis of explosion injuring factors and shock wave characteristics.

Fuel-air bombs are similar to other explosive weapons that employ explosives such as TNT in that they are in essence conventional chemical explosives. However, its method and process of explosion differ, and its explosion generates shock wave with obviously different characteristics. Therefore, their injuring characteristics and outcomes also vary.

Traditional conventional explosive weapons are loaded with high-energy explosives (such as TNT) in high and concentrated payload. During the explosion process of this type of explosives, its own oxidizer and reducing agent are utilized to achieve explosion on its own. The explosion process is fast, peak pressure at the center of the blast during the explosion is very high, peak overpressure of the shock wave generated is also very high, but shock wave's positive pressure effective duration is rather short.

When a fuel-air bomb explodes, its fuel-air explosive needs to mix with air from the surrounding environment, and its own reducing agent works with oxygen in the environment to instigate redox reaction, and then explosion. Compared with the explosion of TNT, the explosion process of fuel-air bomb is relatively slower, and shock wave pressure at the center of the blast is lower than that of TNT, with peak value fluctuating around 3000–5000 kPa. However, positive pressure effective duration of its shock wave lasts for a much longer period, ranging from a few milliseconds to more than 10 ms. The impulse (pressure × effective duration) created by a fuel-air bomb is huge, and when its shock wave propagates in all directions, its rate of attenuation is much slower than that of the shock wave from a TNT explosion. Shock waves of fuel-air bomb also travels farther; therefore, their destructive range and level also clearly outperform TNT explosions.

When a fuel-air bomb detonates, shock wave overpressure is the main factor in the its vapor blast zone, with the overpressure peak value maintaining at a relatively steady level and acting on all directions within the vapor blast zone, while dynamic pressure is pretty small, or even zero (no dynamic pressure). During this period, blast injuries are mostly primary blast injuries, mainly inflicted on internal organ. Outside the vapor blast zone, its explosion resembles that of TNT, with both shock wave overpressure and dynamic pressure working together, but they act directionally, specifically they spread to all directions from the center of explosion. Pressure declines exponentially as distance increases away from the center of the blast, but pressure from the shock wave of fuel-air bomb is much higher than that from TNT, and attenuates slower. Precisely because of these features, the fuel-air weapon causes much more widespread and powerful destruction than other traditional conventional explosive weapons. As fuel-air bomb technology advances, such as the advent of solid fuel and single-action detonation technique, overpressure in vapor blast zone is raised increasingly higher, and powerful dynamic pressure can also be generated within the zone, further adding to the complexity of the destructive effects of the weapon.

Fuel-air bombs injure and kill with mechanisms other than shock waves such as lack of oxygen, high temperature and fragments, causing serious composite injuries. Blast-burn composite injuries, blast-fragment composite injuries and blast-burn-fragment composite injuries are common, and could be accompanied by oxygen shortage and other harms. Fuel-air bombs could create very complicated and varying injury situations.

When a blast wave acts on the interior enclosed fortification, chamber or room, the complicated reflections and superposition of blast pressure of the shock wave, coupled with secondary blast injuries like severe hypoxia, burns and collision injuries, together complicate injury situations that make treatments more difficult and circumstance more dire.

3.2 Features of Blast Injuries of Conventional Explosive Weapons

Zhou Jihong, Qiu Jun, and Yao Yuan

Injuries caused by conventional explosive weapons are related to their injuring factors and mechanisms. The main injuring factors of conventional explosive weapons include projected fragments, shock wave and high temperature. The injury types and features can be summed up as various open wounds caused by fragments, with scope of injury covering any body part or organ of the side of the victim facing the center of the blast; different sorts of burn injuries inflicted by high temperature; and blast injuries originating from shock wave. However, the majority of traditional conventional explosive weapons produce shock waves with relatively short overpressure effective duration, high rate of attenuation, and effective radius smaller than the injury and death radius of fragments, which is why traditional conventional explosive weapons mostly inflict fragmentation injuries and burns, while blast injuries occur at relatively less frequency and severity, especially with munitions of comparatively small equivalent.

When a fuel-air bomb blows up, blast wave is the main injuring factor, with shock wave's overpressure and negative pressure inflicting mainly blast injuries, while shock wave's dynamic pressure can cause different secondary blast injuries by launching objects as projectiles, tossing and displacing people, collapsing buildings, and so on. At the same time, the explosion of a fuel-air bomb also produces other dangerous injuring factors such as fragments, high temperature and oxygen shortage, hence blast injuries from fuel-air munitions are frequently accompanied by fragmentation injuries, burns and suffocation.

When the fuel aerosol and vapor of a fuel-air bomb is detonated, inside the vapor blast zone a powerful and even shock wave is instantaneously created, with peak overpressure ranging around 3000–5000 kPa and positive pressure effective duration lasting around 5–15 ms. The main force of the shock wave inside the vapor blast zone is overpressure, which is why the majority of casualties inside the vapor blast zone died from instantaneous death caused by primary blast injury without obvious displacement. Injuries commonly seen in casualties include severe pulmonary hemorrhage, pulmonary edema, pulmonary rupture, coronary artery air embolism, hepatic rupture, splenic rupture, hemoperitoneum in the chest, serious auditory apparatus injury, and sometimes accompanied by fragmentation injuries, skin burns, and so on. Direct causes of death within the vapor blast zone are mostly acute respiratory and circulatory failures. Outside the vapor blast zone, the farther the distance from the center of the blast, the more the peak overpressure attenuates, but the rate of attenuation is slower than TNT explosion. Starting from the perimeter of the vapor blast zone, dynamic pressure gradually becomes more important as injuring factor. Dynamic pressure radiates from the center of the explosion, and just like overpressure, it gradually weakens the further away from the center of the blast. People within 2–3 m of the edge of the vapor blast zone may be thrown a certain distance, be afflicted with relatively serious internal organ and body surface soft tissue injury, or even instantaneous death. Some frequently seen injuries include hepatic rupture, splenic rupture, pulmonary hemorrhage, pulmonary edema, myocardial hemorrhage, gastrointestinal hemorrhage, limb fractures, and so on. People more than 4–5 m away from the edge of the vapor blast zone may not be obviously displaced and internal organ injuries are usually relatively minor because shock wave dynamic pressure would have declined markedly. However, clear auditory apparatus injuries may occur within a range of 4–50 m away from the edge of the vapor blast zone. When a fuel-air bomb of a relatively large equivalent is detonated, people several dozen meters away from the vapor blast zone may suffer from temporary threshold shift and eardrum hyperemia, or even ruptured eardrum in some cases.

4 Section Four: Evaluation of Injuries from Conventional Explosive Weapons

4.1 Evaluation Models for Injuries from Conventional Explosive Weapons

Studies on the injuring effects of shock waves are inseparable from physical models of injuring effects. Blast injuries in organisms mostly depend on the rate of propagation of shock wave, peak pressure, pressure increase duration, pressure effective duration, and the type of organism, as well as ambient pressure. In most cases, the higher the peak pressure, the more severe the injury; the longer the positive pressure effective duration, the more severe the injury; and the shorter the pressure increase duration, the more severe the injury. Bowen curves, Axelsson effective work model and Stuhmiller thoracic wall motion model are the most common non-auditory apparatus injury mechanisms and analysis techniques.

In 1968, Bowen proposed three types of curves for assessing lung injury and death threshold based on the cephalocaudal axis of the human body and direction of shock wave. The first type represents a situation in which the body's cephalocaudal axis is parallel to the shock wave's direction (as in the person is lying down with face or back toward the center of explosion), the second type represents a situation in which the person is standing with face, side or back toward the center of explosion, and the third type represents a situation in which the person is located near a reflection surface. The shock wave injury threshold (pressure of incident shock

wave) near a reflection surface is usually lower than shock wave injury threshold for a person standing in a free field. Death threshold refers to whether or not the subject is alive 24 h after injury. The main limitation in the Bowen curve is that the model was built based on an ideal shock wave, making it not very suitable for complex shock waves in an enclosed chamber. The Stuhmiller model and Axelsson model are more suitable for studying complex shock waves.

The Stuhmiller model is regarded as using the lungs as sensitive model to describe blast lung injuries. Said model is based on shock wave load applied on the thoracic wall. Motion of the thorax generates compression wave in the thoracic cavity, and the compressed lungs produce intrapulmonary pressure. Since the lungs are compressible, Stuhmiller et al. firstly established mathematical model of piston movement and compressible gas pressure in the lungs, as shown below.

$$p(t) = p_0\left[1+\frac{1}{2}(\lambda-1)\frac{v}{c_0}\right]^{\frac{2\lambda}{\lambda-1}} \tag{1}$$

where $p(t)$ are pressures, p_0 is intrapulmonary pressure, c_0 is the speed of sound, v is speed of piston, and λ is specific heat.

The normalized work is further derived according to said formula. Said "normalized work" is defined as total power of shock wave divided by lung volume and ambient pressure. The formula is as listed below.

$$W^* = \frac{W}{P_0 V} = \frac{1}{P_0 L}\int_0^{\infty}\rho_0 c_0 v^2 \mathrm{d}t \tag{2}$$

where W^* is normalized work, P_0 is ambient pressure, V is lung volume, c_0 is the speed of sound, v is thoracic wall movement speed, and ρ_0 is lung density.

Although lung injury is caused by local stress that exceeds the threshold of tissue and not the situation described in said model, the pathological situation of the whole lung injury is related to the average energy (normalized work) distribution of the lung tissues. In other words, work done to lung by thoracic wall movement is closely associated with the animal's pathology conditions and lethality. Said model is applicable to lung injuries arising from both complex shock waves and shock wave in free field.

The Axelsson model is also used to assess injury to the human body when subjected to complex shock waves. Axelsson developed the model based on test results involving 255 goats/sheep and cylindrical test chamber.

In Fig. 1, A is effective surface area, M is effective mass, V is initial lung volume, x is displacement distance, C is damping coefficient, K is elastic coefficient, P_0 is ambient pressure, and $p(t)$ is overpressure at moment t.

The formula for said model is as follows.

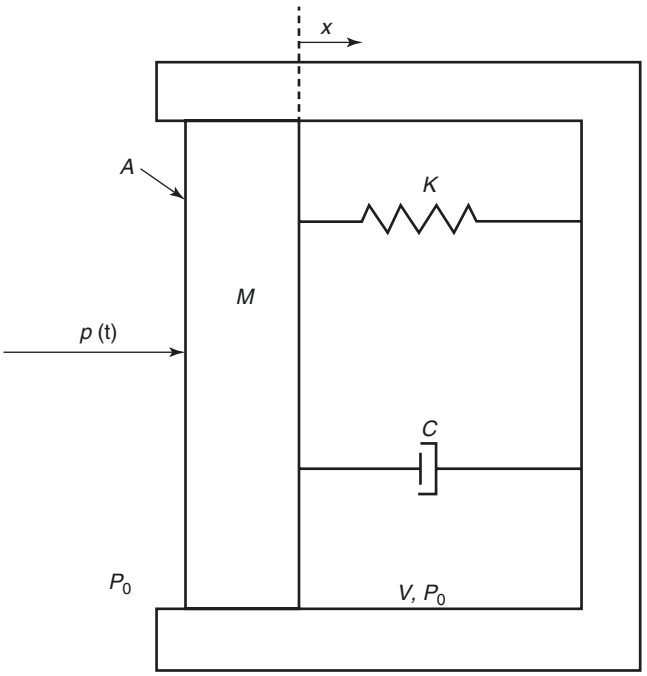

Fig. 1 Single degree of freedom model for single compartment single lung (Axelsson, 1996)

$$M\cdot\frac{\mathrm{d}^2x}{\mathrm{d}t^2}+C\cdot\frac{\mathrm{d}x}{\mathrm{d}t}+K\cdot x = A\cdot\left[p(t)+P_0-\left(\frac{V}{V-A\cdot x}\right)^{\gamma}\cdot P_0\right] \tag{3}$$

The input of the model is shock wave overpressure measured from four directions of the cylinder, and based on overpressure values, thoracic wall movement speed v is calculated. The Adjusted Injury of Severity Index (ASII) may be derived from v.

4.2 Criteria for the Assessment of Injuries from Explosive Weapon

Criteria for the assessment of injury effects from explosive weapon mainly involve fragment and shock wave. In terms of specific criteria, pathological standards function as the basis.

4.2.1 Classification and Fundamental Basis of Severity of Biological Injuries (Table 1)

4.2.2 Assessment Methods for Severity of Fragmentation Injuries

For specific contents in rating of fragmentation injury severity based on injured body part and injury grade, please see Table 2.

Table 1 Classification and fundamental basis of injury severity

Injury severity classification	Score	Fundamental basis
No injury	0–0.1	No anatomic structure or functional damage; or if injury is very mild, as in injury undetectable by existing medical methods and techniques (as in no injury, or the subject does not perceive injury)
Minor injury	0.2–0.3	Body surface injury with little impact on body function; or injury that imparts relatively little impact on body function, that does not impart obvious influence on the subject's cognitive and behavioral functions, and that may heal on its own without medical treatment (as in injury that the subject can perceive but does not require medical treatment in the near term)
Moderate	0.4–0.6	Injury that affects bones, muscles, blood vessels, and nerves in tissues and organs, that clearly affects the subject's cognitive and behavioral functions, but that does not constitute a life-threatening situation, and that the subject may perform certain combat duties after simple medical treatment (as in injury that requires a certain degree of medical treatment but does not require evacuation from the battlefield)
Severe	0.7–0.8	Injury that affects tissues and organs in the head, brain, chest cavity and abdominal cavity, and with injured area amounting to 60% or higher, or that is severe enough to cause the loss of 60% or more tissue or organ function. Or if the limb has one or more damaging injury (as in injury that the subject may possibly survived from after medical treatment but would render the subject basically unable to continue combat)
Extremely severe	0.9–1.0	One or more injury that seriously affects important tissues and organs in the head, brain, chest cavity and abdominal cavity, or damaging injuries to two or more limbs, and injuries that render the subject unable to survive for more than 2 h without medical treatment (as in injury that is relatively difficult for subject to survive even after medical treatment and that renders the subject completely unable to continue combat)

4.2.3 Assessment Methods for Severity of Blast Injuries (Including Burns)

Formulated based on the injury score (IS) system, scores are given according to the body part injured, grade of injury and severity of injury of test animals. Specific injury score (IS) is calculated as per the following formula.

$$\mathrm{IS} = \left(E + G + \mathrm{ST}\right)\left(\mathrm{SD}\right) \tag{4}$$

where E is score for extent of injury, G is score for grade of injury, ST is score for severity type, and SD is score for severity degree. In Formula (4), details regarding the various scores are shown respectively in Tables 3, 4, 5, and 6, where E is related to extent of injury, and its value changes according to the contents in the lists, so for example, if an external injury includes injuries to the head, neck, forelimb, chest, abdomen, and rear limb, a score of 6 would be assigned. G is related to surface area of injury, number of bone fractures, percentage of body surface area injured and percentage of organs injured, with score fluctuating between 1 and 5. Severity score S is related to severity type ST and severity degree SD, so for example, lung injury with only pulmonary ecchymosis would be given SD = 1, while that with ruptured lung or penetrating injury would be given SD = 5; lung injury with only pleural surface bleeding would be given ST = 1, while that with diffuse hepatization would be given ST = 4.

As shown in Table 7, the largest injury score obtained from each formula ranges between 24 and 64. Use Excel to analyze the probability of appearance of each value, then add all the probabilities together to obtain total probability, then add incidence factors to the figure and multiply the sum by 1 or 2 (lethal factor) to obtain SII value. Incidence factors include whether or not pneumothorax, hemothorax, hemoperitoneum, coronary air embolism and cerebrovascular air embolism will occur, and the extent of their occurrence. For atypical cases, middle ear injury score may be adjusted to obtain ASII value (Table 8).

During the course of explosion, the relationships between injury score for different organs, including solid organs in the abdomen, and injury level are shown in Table 9.

Lung Injury Grades

Negative: No injury

Slight injury: Scattered petechiae or relatively small ecchymosis but less than 10% of whole lung

Minor injury: Relatively obvious ecchymosis or scattered pulmonary consolidation but less than 10% of whole lung

Moderate injury: Scattered parenchymal contusions or pulmonary consolidation, but bleeding area is less than 30% of whole lung

Severe injury: Parenchymal contusions or diffuse pulmonary consolidation, with area equal to or more than 30% of whole lung

Throat, Windpipe Injury Grades

Negative: No injury

Slight injury: Scattered petechiae or solitary ecchymosis, with depth less than one layer and area less than 10%

Minor injury: Scattered petechiae or diffuse contusions, with depth between one layer to two layers layer and area less than 30%

Table 2 Rating sheet for grading of fragmentation injury to different body parts

Body part	Tissue or organ	Description of grade of injury	Injury severity rating
Head and neck	Brain stem	Superficial hemorrhage, no damage to parenchyma	0.3
		Minor contusion, superficial, punctate	0.4
		Small area of contusion, diameter <1 cm	0.5
		Large area of contusion, diameter >1 cm	0.7
		Laceration, penetrating wound, transection	1.0
	Cerebellum	Superficial hemorrhage, no damage to parenchyma	0.4
		Minor contusion, superficial, diameter <1 cm	0.6
		Small area of contusion, diameter <3 cm	0.7
		Large area of contusion, diameter >3 cm	0.8
		Penetrating wound, depth ≤2 cm	0.9
		Penetrating wound, depth >2 cm	1.0
	Cerebrum	Superficial hemorrhage, no damage to parenchyma	0.3
		Minor contusion of parenchyma, diameter <1 cm	0.4
		Small area of contusion, diameter 1–4 cm	0.5
		Large area of contusion, diameter >4 cm	0.7
		Penetrating wound, depth ≤2 cm	0.8
		Penetrating wound, depth >2 cm	1.0
	Skull	Closed simple fracture, crack, linear, no displacement	0.4
		Comminuted fracture but intact dura, depression ≤2 cm	0.6
		Complex, open, exposure or loss of brain tissue	0.8
		Extensive fracture, depression >2 cm	0.9
	Scalp	Abrasion, contusion, minor laceration	0.3
		Severe laceration, length >20 cm and extending to subcutaneous area	0.5
	Ears	Eardrum perforation area <25%, ossicular chain intact	0.3
		Eardrum perforation area 25–50%, ossicular chain intact	0.5
		Eardrum perforation area 25–50%, ossicular chain intact	0.7
		Eardrum perforation area >75%, ossicular chain dislocated	0.9
	Eyes	Individual foreign object in the conjunctiva sac, or irritation from smoke, fire or light	0.2
		Foreign objects on corneal surface or in conjunctival sac, but quantity limited and countable	0.3
		Laceration of eyelid and lacrimal tubule, or epithelia corneal erosion	0.4
		Eyeball penetrating injury, ocular rupture	0.7
	Oral cavity	Minor superficial laceration, no internal organ damage	0.2
		Deep laceration, mild injury of oral tissues and organs	0.3
		Deep laceration, serious injury of oral tissues and organs	0.5
		Mandible fracture, maxilla fracture	0.7
	Throat	Contusion, hematoma	0.3
		Laceration, no perforation, incomplete layer	0.5
		Perforation, whole layer	0.7
		Transection, extensive damage	0.9
	Windpipe	Contusion, hematoma	0.3
		Laceration, no perforation, incomplete layer	0.5
		Perforation, whole layer	0.7
		Rupture, transection, extensive damage	0.9
	Facial skin	Facial soft tissue contusion, laceration	0.2
		Severe facial soft tissue laceration, length >20 cm and extending to subcutaneous area	0.5
	Nose	Nasal mucosa rupture	0.3
		Nasal bone fracture	0.5
	Cheekbone	Cheekbone fracture	0.4
	Nuchal soft tissue	Nuchal soft tissue contusion, laceration	0.2
		Severe nuchal soft tissue laceration, length >20 cm and extending to subcutaneous area	0.4
		Phrenic nerve injury	0.5
		Bilateral phrenic nerve injury	0.7
	Cervical vertebra	Spinal cord contusion with transient neurological signs (abnormal sensation)	0.3
		Spinal cord contusion with incomplete spinal cord injury syndrome (with partial sensory or motor functions remaining)	0.5
		Spinal cord contusion with complete spinal cord injury syndrome at C4 or below vertebrae (quadriplegia or paraplegia, and with no sense of function)	0.6
		Spinal cord contusion with complete spinal cord injury syndrome at C3 or above vertebrae (quadriplegia or paraplegia, and with no sense of function)	0.7
	Esophagus	Esophagus laceration, no perforation, incomplete layer	0.3
		Esophagus perforation, whole layer	0.5
		Esophagus rupture, transection, extensive damage	0.8

Table 2 (continued)

Body part	Tissue or organ	Description of grade of injury	Injury severity rating
Chest	Diaphragm	Contusion, hematoma	0.3
		Laceration, ≤10 cm	0.5
		Laceration, >10 cm, with obvious tissue loss	0.7
		Rupture, with diaphragmatic hernia formed	0.8
	Pleural space	Pleural laceration	0.5
		Hemothorax/pneumothorax/hemopneumothorax	0.7
		Open (sucking) chest wound, hemothorax/pneumothorax/hemopneumothorax at least on one side >1000 mL	0.8
		Tension pneumothorax	0.8
	Lungs	Contusion, punctate pulmonary hemorrhage seen on lung surface, diameter <2 cm	0.3
		Contusion, punctate pulmonary hemorrhage seen on lung surface, diameter <5 cm	0.5
		Penetrating wound, pulmonary hemorrhage around the wound passage only affects one lobe	0.6
		Penetrating wound, pulmonary hemorrhage around the wound passage affects more than one lobe	0.7
		Penetrating wound to both lungs, pulmonary hemorrhage around the wound passage affects more than one lobe	0.8
	Ribs	Fracture of one rib	0.4
		Fracture of two ribs	0.6
		Fracture of three to five ribs, with unilateral flail chest	0.7
		Fracture of more than five ribs, with unilateral flail chest	0.8
		Bilateral flail chest	0.9
	Heart	Minor contusion of the endocardial lining, diameter <2 cm	0.3
		Contusion of the endocardial lining, diameter >2 cm	0.4
		Heart laceration, no perforation, no atrium or ventricle affected	0.5
		Perforation of atrium or ventricle; rupture of atrium	0.9
	Pericardium	Pericardium laceration, pericardial effusion	0.7
		Hemopericardium, but without cardiac tamponade or cardiac trauma	0.8
		Hemopericardium, with cardiac tamponade, but without cardiac trauma	0.9
		Pericardial hernia	0.9
	Aortic arch/thoracic aorta/abdominal aorta	Intima tear, vessel not ruptured	0.4
		Minor laceration, superficial, circumference of vessel wall not completely affected	0.5
		Severe laceration, complete transection, segmental loss	1.0
	Pulmonary artery/pulmonary vein	Intima tear, vessel not ruptured	0.4
		Minor laceration, superficial, circumference of vessel wall not completely affected	0.5
		Severe laceration, complete transection, segmental loss	0.9
		Severe laceration on both sides, complete transection, segmental loss	1.0
	Subclavian artery/brachiocephalic artery	Intima tear, vessel not ruptured	0.3
		Minor laceration, superficial, circumference of vessel wall not completely affected	0.4
		Severe laceration, complete transection, segmental loss	0.8
	Thoracic vertebra	Spinal cord contusion with transient neurological signs (abnormal sensation)	0.4
		Spinal cord contusion with incomplete spinal cord injury syndrome (with partial sensory or motor functions remaining)	0.6
		Spinal cord contusion with complete spinal cord injury syndrome (paraplegia, and with no sense of function)	0.7
	Brachiocephalic vein/subclavian vein/superior vena cava/inferior vena cava	Intima tear, vessel not ruptured	0.3
		Minor laceration, superficial, circumference of vessel wall not completely affected	0.4
		Severe laceration, complete transection, segmental loss	0.8

(continued)

Table 2 (continued)

Body part	Tissue or organ	Description of grade of injury	Injury severity rating
Abdomen	Lumbar vertebra	Spinal cord contusion with transient neurological signs (abnormal sensation)	0.3
		Spinal cord contusion with incomplete spinal cord injury syndrome (with partial sensory or motor functions remaining)	0.5
		Spinal cord contusion with complete spinal cord injury syndrome (paraplegia, and with no sense of function)	0.7
	Solid organs	Minor contusion and laceration, area beneath capsule <50%, kidney laceration depth <1 cm, liver or spleen laceration depth <3 cm	0.4
		Moderate contusion and laceration, area beneath capsule >50%, kidney laceration depth >1 cm, liver or spleen laceration depth >3 cm	0.6
		Kidney laceration extends to cortex and medulla, liver laceration affects <50% of parenchyma, segmental rupture of spleen	0.7
		Whole kidney damaged, loss of big parts of liver or spleen	0.9
		Liver degloving injury, all veins of the liver torn and ruptured	0.9
	Superior mesenteric artery	Intima tear, vessel not ruptured	0.3
		Minor laceration, superficial, circumference of vessel wall not completely affected	0.5
		Severe laceration, complete transection, segmental loss	0.7
	Hollow organs	Contusion or hematoma of stomach/small intestine/large intestine/gall bladder	0.4
		No perforation of stomach/small intestine/large intestine/gall bladder, incomplete layer	0.5
		Perforation of whole layer of stomach/small intestine/large intestine/gall bladder	0.7
		Extensive degloving of stomach/small intestine/large intestine/gall bladder, complicated tissue loss, transection	0.8
Pelvis and limbs	Upper limb blood vessel	Intima tear, vessel not ruptured	0.3
		Upper limb blood vessel minor laceration, superficial, circumference of vessel wall not completely affected	0.4
		Upper limb blood vessel severe laceration, complete transection, segmental loss	0.7
	Upper limb nerve	Median/radial/ulnar nerve contusion	0.4
		Median/radial/ulnar nerve laceration	0.5
		Incomplete brachial plexus injury	0.6
		Complete brachial plexus injury	0.7
		Bilateral brachial plexus injury	0.8
	Upper limb bone/joint	Sprain	0.2
		Simple or oblique fracture; extra-articular fracture, some parts of joint affected	0.4
		Simple fracture, whole joint affected	0.6
		Complicated, comminuted, fragmentary fracture	0.7
	Upper limb soft tissue	Muscle/tendon/ligament bruise, contusion, hematoma	0.4
		Muscle/tendon/ligament partial tear	0.5
		Muscle/tendon/ligament complete tear	0.6
	Lower limb blood vessel	Intima tear, vessel not ruptured	0.3
		Lower limb blood vessel minor laceration, superficial, circumference of vessel wall not completely affected	0.4
		Lower limb blood vessel severe laceration, complete transection, segmental loss	0.7
	Lower limb nerve	Sciatic nerve contusion	0.3
		Sciatic nerve laceration	0.5
		Femoral/tibial nerve contusion	0.4
		Femoral/tibial nerve laceration	0.6
	Lower limb bone/joint	Sprain	0.3
		Simple or oblique fracture; extra-articular fracture, some parts of joint affected	0.5
		Simple fracture, whole joint affected	0.6
		Complicated, comminuted, fragmentary fracture	0.7
	Lower limb soft tissue	Muscle/tendon/ligament bruise, contusion, hematoma	0.4
		Muscle/tendon/ligament partial tear	0.5
		Muscle/tendon/ligament complete tear	0.6
	Cauda equina	Cauda equina contusion with transient neurologic signs (abnormal sensation)	0.3
		Cauda equina contusion with incomplete cauda equina injury syndrome	0.5
		Cauda equina contusion with complete cauda equina injury syndrome	0.6
	Pelvis	Pelvic ring fracture, posterior pelvic ring not damaged; isolated fracture, with no damage to integrity of the pelvic ring	0.5
		Open fracture; pelvic ring fracture, damage to parts of the posterior pelvic ring	0.6
		Open fracture, blood loss ≤20%	0.7
		Open fracture, blood loss >20	0.8

Table 3 Injury score rating details—scoring for extent of injury (*E*)

Item	Extent of injury							
1	Head	Neck	Fore limb	Chest	Abdomen	Rear limb		
2	Skull	Vertebra	Ribs	Fore limb	Rear limb			
3	Head	Neck	Fore limb	Chest	Abdomen	Rear limb		
4	Throat	Epiglottis	Arytenoid cartilage	Laryngeal ventricle				
5	Front	Moderate	Back					
6	Apex of right lung	Apex of left lung	Right heart	Left heart	Right diaphragm	Left diaphragm	Central diaphragm	
7	Right atrium	Left atrium	Right ventricle	Left ventricle				
8	Stomach	Small intestine	Large colon	Microcolon	Cecum	Rectum	Gall bladder	Urinary bladder
9	Liver	Spleen	Pancreas	Adrenal gland	Kidney			
10 and 11	Tympanic	Malleus	Incus	Stapes	Fossa ovalis	Round window		

Note: (1) external injury; (2) bone fracture; (3) burn; (4) throat; (5) windpipe; (6) lungs; (7) heart; (8) hollow organ in abdominal cavity; (9) solid organ in abdominal cavity; (10) right ear; (11) left ear

Table 4 Injury score rating details—scoring for grade of injury (*G*)

Item	Parameter	1	2	3	4	5
1	Area of injury (cm^2)	0–1	2–10	11–20	21–30	>30
2	Number of bone fractures	1	2	3	≥4	
3	Percentage of surface area	≤10	11–25	26–50	>50	
4	Percentage of organs	≤10	11–30	31–60	>60	
5	Percentage of organs	≤10	11–30	31–60	>60	
6	Percentage of organs	≤10	11–30	31–60	>60	
7	Percentage of organs	≤10	11–30	31–60	>60	
8	Area of injury (cm^2)	0–1	2–10	11–20	21–30	>30
9	Percentage of organs	≤10	11–30	31–60	>60	
10 and 11	Evaluation	Bleeding only	Minor avulsion of eardrum	Middle ear damage ≤50%	Middle ear damage ≥50%	

Note: (1) external injury; (2) bone fracture; (3) burn; (4) throat; (5) windpipe; (6) lungs; (7) heart; (8) hollow organ in abdominal cavity; (9) solid organ in abdominal cavity; (10) right ear; (11) left ear

Table 5 Injury score rating details—scoring for severity type (ST)

Item	1	2	3	4	5
1	Abrasion	Contusion	Avulsion or penetration		
2	Incomplete	Complete	Composite		
3	Minor burn	Moderate/browned	Severe/carbonized		
4	One to five petechiae	More than six petechiae	Ecchymosis	Diffuse contusion	
5	One to five petechiae	More than six petechiae	Ecchymosis	Diffuse contusion	
6	Petechiae	Ecchymosis or bulla	Isolated bleeding	Diffuse bleeding	Penetration/rupture
7	Petechiae	Ecchymosis or bulla	Isolated bleeding	Diffuse bleeding	Penetration/rupture
8	Serous membrane or mucous membrane	Two layers	Whole layer		
9	Intracapsular contusion	Intracapsular hematoma			

Note: (1) external injury; (2) bone fracture; (3) burn; (4) throat; (5) windpipe; (6) lungs; (7) heart; (8) hollow organ in abdominal cavity; (9) solid organ in abdominal cavity; (10) right ear; (11) left ear

Table 6 Injury score rating details—scoring for severity degree (SD)

Item	1	2	3	4	5
1	Superficial	Depth	Penetration of body wall	Perforating injury	
2	Close wound	Open wound			
3	Singeing	First degree	Second degree	Third degree	
4	One layer	Two layers	Whole layer	Reduced diameter of hematoma	Rupture/avulsion
5	One layer	Two layers	Whole layer	Reduced diameter of hematoma	Rupture/avulsion
6	Pleura	Solid organ	Scattered hepatization	Diffuse hepatization	
7	One layer	Two layers	Whole layer	Rupture/avulsion	
8	Mucous membrane intact	Mucosal ulcer	Rupture/avulsion		
9	Bladder intact	Avulsion of surface	Rupture/avulsion		
10 and 11	Auditory ossicles intact	Malleus fracture/ loss	Ossicular chain fracture	Round/oval window fracture	

Note: (1) external injury; (2) bone fracture; (3) burn; (4) throat; (5) windpipe; (6) lungs; (7) heart; (8) hollow organ in abdominal cavity; (9) solid organ in abdominal cavity; (10) right ear; (11) left ear

Table 7 Injury scoring method

Item	Content	Injury parameter				Injury formula	Max score
				Severity			
		Extent (*E*)	Grade (*G*)	Severity type (ST)	Severity degree (SD)		
1	External wound	0–6	0–3	0–3	1–3	(*E* + *G* + ST)(SD)=	36
2	Fracture	0–5	0–4	0–3	1–2	(*E* + *G* + ST)(SD)=	24
3	Burn	0–6	0–4	0–3	1–4	(*E* + *G* + ST)(SD)=	52
4	Throat	0–4	0–4	0–4	1–5	(*E* + *G* + ST)(SD)=	60
5	Windpipe	0–3	0–4	0–4	1–5	(*E* + *G* + ST)(SD)=	55
6	Lungs	0–7	0–4	0–5	1–4	(*E* + *G* + ST)(SD)=	64
7	Heart	0–4	0–4	0–4	1–4	(*E* + *G* + ST)(SD)=	48
8	Abdominal cavity hollow organ	0–8	0–5	0–3	1–3	(*E* + *G* + ST)(SD)=	48
9	Abdominal cavity solid organ	0–5	0–4	0–2	1–4	(*E* + *G* + ST)(SD)=	44
10	Right ear	0–6	0–4		1–4	(*E* + *G*)(SD)=	40
11	Left ear	0–6	0–4		1–4	(*E* + *G*)(SD)=	40

Table 8 SII scoring method

Injury	Value range	Max value	Probability	Incidence factors	Incidence factor probability	Score calculation formula	Score variation range
External	0–56	56	0–1.0	Pneumothorax, hemothorax, hemoperitoneum, coronary air embolism and cerebrovascular air embolism	0, 1, 2	Score = total probability + total incidence factors × incidence multiplier (1 for survival, 2 for death)	0–10.00
Fracture	0–24	24	0–1.0				
Burn	0–52	52	0–1.0				
Throat	0–60	60	0–1.0				
Windpipe	0–55	55	0–1.0				
Lungs	0–64	64	0–1.0				
Heart	0–48	48	0–1.0				
Abdominal cavity hollow organ	0–48	48	0–1.0				
Abdominal cavity solid organ	0–44	44	0–1.0				
Right ear	0–40	40	0–1.0				

Table 9 Relationships between the injury score and injury level of different organs

			Injury score		
Injury level	Lungs	Throat	Windpipe	Abdominal cavity hollow organ	Abdominal cavity solid organ
Negative	0–0.03	0–0.04	0–0.04	0–0.05	0–0.05
Slight injury	0.04–0.06	0.05–0.067	0.05–0.07	0.06–0.08	0.06–0.09
Minor injury	0.07–0.30	0.068–0.267	0.08–0.33	0.09–0.38	0.10–0.41
Moderate injury	0.31–0.56	0.268–0.367	0.34–0.51	0.39–0.58	0.42–0.64
Severe injury	0.57–1.0	0.368–1.0	0.52–1.0	0.59–1.0	0.65–1.0

Moderate injury: Petechiae, ecchymosis, or diffuse contusions, with depth of two layers and area less than 60%

Severe injury: Diffuse contusions with depth exceeding two layers and area more than 60%

For certain cases of upper respiratory tract injury, bleeding or edema may shrink the diameter of the airway and cause breathing difficulty. In certain cases of severe pulmonary hemorrhage, diffuse parenchymal hepatoid lesion with hemorrhagic fluid flowing into bronchus may occur.

Abdominal Cavity Hollow Organ Injury Grades

1. Negative: No injury
2. Slight injury: Small intramucosal contusions with depth less than two layers, coupled with contusions to two organs but affecting an area less than 10 cm^2.
3. Minor injury: Diffuse contusions with depth between one layer and two layers and affecting an area less than 30 cm^2, or coupled with mucosal ulcer.
4. Moderate injury: Full-layer contusions or mucosal ulcer affecting an area between 21 and 30 cm^2.
5. Severe injury: Full-layer contusions with mucosal ulcer, or one or more perforations, affecting an area larger than 30 cm^2.

Abdominal Cavity Solid Organ Injury Grades

Negative: No injury

Slight injury: Small cysts or hematomas in one or two organs, but with area less than 10% of organ

Minor injury: Cysts or hematomas with area less than 30% of organ, and coupled with parenchymal maceration of slightly lacerated organs

Moderate injury: Deep lacerations or parenchymal maceration with area more than 60% of organ

Severe injury: Deep lacerations or parenchymal maceration with area more than 60% of two or more organs

4.2.4 Assessment for Composite and Overall Injury Severity

When an injury involves multiple body parts and multiple systems and organs, it is necessary to make correct assessment of the victim's overall injury status. Refrain from simple evaluation by adding or averaging the individual assessment scores of the different injured body parts, because the relationships between the assessment scores of the different body parts and the IS scores of the various systems and organs are not linear. Since injury severity and death rate are related to the quadratic sum of IS, which remains valid even in patients with multiple injuries, the quadratic sum of IS may be used to estimate overall injury status.

Said evaluation method is called injury severity score (ISS). Take the higher IS values of the three most seriously injured areas of the body to calculate the quadratic sum as evaluation value.

$$\mathrm{ISS} = {}_{\max}\mathrm{IS}^2 + {}_{2\mathrm{nd}}\mathrm{IS}^2 + {}_{3\mathrm{rd}}\mathrm{IS}^2 \tag{5}$$

For the evaluation of composite injuries, use the following formula.

$$P(I/H)_{\mathrm{total}} = 1-(1-P_1)(1-P_2)(1-P_3)\cdots(1-P_n) \tag{6}$$

Note: In the formula, P_n is injury score for a single body part.

Overall ISS evaluation method uses the IS values for a single body part as basis. In evaluation of overall injury status, injury scores for individual single body parts are also used as basis.

4.3 Computer Simulation Evaluation of Injuries from Explosive Weapons

Evaluation of injuries from explosive weapons through computer simulation relies on theories and techniques in disciplines such as medicine, ballistics, biomechanics, mathematics, and computer science to develop models for weapon's injuring and lethal elements, target, and the mutual effects between the two. In other words, mathematical formulas are used to express their physical properties, then computation is utilized to solve models, and obtain mechanical response and damage effect of anatomical structure of target under the action of a weapon's injuring and lethal elements, so as to analyze, predict, and evaluate damage sustained by the target.

4.3.1 Status Quo and Development

Weapon damage effect evaluation in the 1950s and 1960s primarily relied on animal testing, whereby researchers observed features of injuries inflicted on organisms by different weapons and munitions, after which damage determination basis of a weapon's injuring and lethal elements would be developed. Starting from the 1970s, dummy targets were used to examine dynamic mechanical response of a target under the actions of a weapon's injuring and lethal elements. Physical parameters related to injury were obtained, based upon which injuring effects were determined. With advancements in computer technology, computer models and simulation analysis rose to become main pathways for weapon damage effect evaluation in many countries.

ComputerMan

In the 1970s, the Ballistic Research Laboratory (BRL) of the USA developed the ComputerMan, which was the first human body computer model used for weapons research. Since injury determinations on humans were predominantly based on anatomic injury assessments of test animals, there were many conflicts and arguments about how to accurately convert animal-based injury effects for human application. Researchers attempted to use computer modeling and simulation of human bodies and injury processes to solve this problem. The form and structure of the ComputerMan were based on the male body, and raw data were sourced from a set of images of the horizontal transverse plane of the human body. The 108 images were divided into five groups based on human body composition: head and neck (1–18); chest, abdomen, and pelvis (19–44); left arm (50–75); left leg and foot (76–113). Layers 45–49 are cross-sectional images of the female pelvic organs. Distance between anatomical layers of the head and neck is 1.2 cm, and that of the chest, abdomen, and pelvis is 2.6 cm. The 108 horizontal transverse plane images were registered based on reference points, then stacked layer upon layer and converted into a 3D model (Fig. 2). ComputerMan included 181 human anatomical tissues, and each group of tissue has a corresponding tissue code. Therefore, the ComputerMan was essentially the aggregation of a series of tissue codes for the human body when it is penetrated and perforated by projectiles that flew along ballistic tracks.

Since incapacitation and casualty are the most basic injury types on a battlefield, the ComputerMan was mostly used to predict the probability of incapacitation and fatal effects caused by bullets, fragments, and other projectiles. Incapacitation evaluation mainly focused on two kinds of combat roles and three combat times, as follows.

1. Immediate combat, immediate defense
2. Combat <30 s, defense <30 s
3. Combat <5 m, defense <5 m

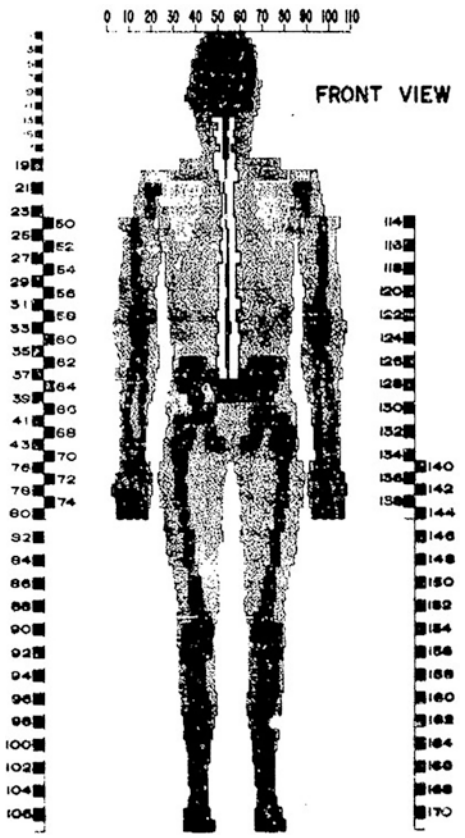

Fig. 2 Computed front view

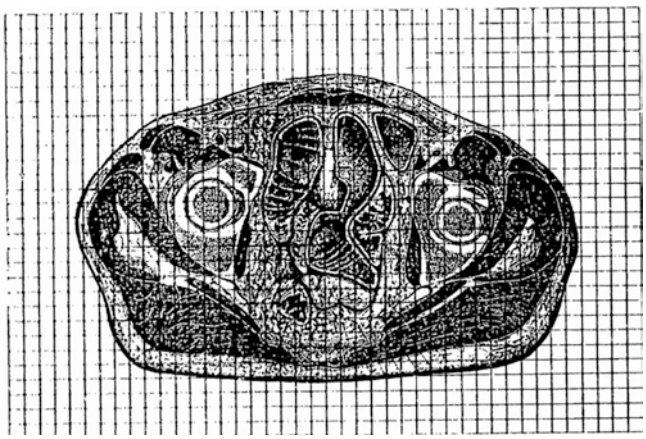

Fig. 3 Grid division of cross-sectional image

Surgeons provided quantitative assessed values for level of incapacitation for each group of tissue when penetrated by projectile, expressed as a number between 0 and 100. About one-third of tissues were given a non-zero value. Each layer of horizontal transverse plane image that comprised the ComputerMan was divided into 0.5 cm × 0.5 cm grids (Fig. 3), and the corresponding tissue code (Fig. 4) and inca-

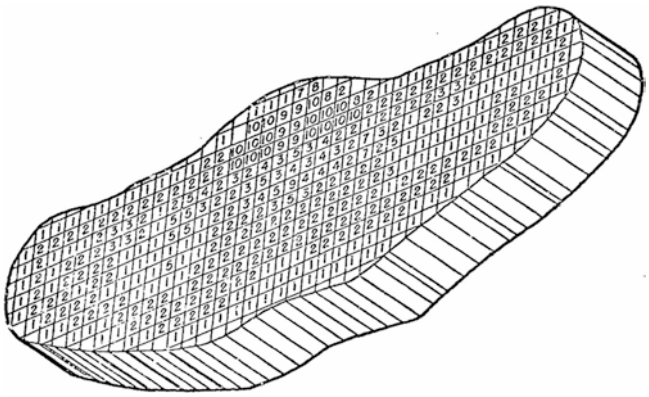

Fig. 4 Organization code of cross-sectional image grid

pacitation assessed value of each grid were stored on a computer as a three-dimensional array.

Casualty evaluation focused on four timeframes between injury and obtainment of medical treatment, namely, 30 min, 1 h, 6 h, and no treatment. A group of experts conduct estimation of possibility of death when a certain unit of human body tissue is lost, with the result given a number between 1 and 10. Each layer of horizontal transverse plane image that comprised the ComputerMan was divided into 1.0 cm × 1.0 cm grids, and the corresponding tissue code and casualty assessed value of each grid were stored on a computer as a three-dimensional array.

Operational Requirement-Based Casualty Assessment Software System (ORCA)

In the 1990s, the Joint Technical Coordinating Group for Munitions Effectiveness (JTCG/ME) of the US Department of Defense and the Air Crew Survivability Evaluation Committee jointly developed the Operational Requirement-Based Casualty Assessment Software System (ORCA), which was a drastic improvement over ComputerMan. ORCA's injury evaluation extent includes all conventional injuries and war wounds, but evaluations are made based on certain assumptions and limitations: (1) Only applies to injuries related to military duties; (2) no medical treatment is administered; and (3) no secondary effect is generated. ORCA is an automated and interactive system. Based on injury parameters input (i.e., fragment, shock wave, acceleration, heat, chemical gases, and laser) and the military duty requirements assigned to the personnel, the ORCA program can output a general description of the anatomic injuries of the injured person, details of the injury process (such as bleeding), injury score based on simple trauma grading criteria, status of the 24 functional units of the human body, and remaining combat capacity in various timeframes after injury (immediate, 30 s, 5 min, 1 h, 24 h, and 72 h). The evaluation process is illustrated in Fig. 5.

ORCA allows users to input detailed descriptions of certain combat duties, or to choose from a mission's duty requirements from 18 military roles in the database, as well as detailed and accurate military missions, or pre-set plans and outlines. Users may also use one's own mission database to formulate personalized duty requirements. Aside from simulation evaluation for the injuring effects of a single injury factor, ORCA also enables grouped processing of multiple injury factors and prediction of composite effect under the actions of multiple injury factors. Therefore, it has found application in assessment of weapon effect, protection requirement, medical treatment, and combat planning.

4.3.2 Computer Simulation Method and Cases

Computer Analysis of Ballistic Injury

The theory behind computer analysis of ballistic injury is based on digitized human body modeling, and uses values and calculations to obtain the trajectory of projectile after entering the body, and types and quantity of tissues on the path of the projectile trajectory. Then the level of capacity loss and injury of the injured person arising from the loss of affected tissues are evaluated in accordance with pre-set capacity loss determination criteria and casualty criteria. Method and steps in computer analysis of ballistic injury are described below:

1. Construction of digital anatomical model and finite element model of human body: Relying on layer-by-layer CT/MRI scan of the human body, or freeze cutting of human specimens, a collection of two-dimensional cross-sectional images of the human body is acquired. Each image is digitized and stored to create an original data set of two-dimensional cross-sectional images of the human body. Organize, segmentize and code each image in the dataset, register the corresponding spatial relationships of each layer of image, and 2D images are imported into image reconstruction software to generate a 3D model after registration.

   Utilize finite element pre-processing software to divide digital anatomical model of the human body into mesh, then define the material properties of each organ (tissue) in finite element analysis software.
2. Construction of projectile digital geometric model and finite element model: With the help of CAD software such as Proe or Solidwork, develop the geometry of a projectile according to the dimensions of its constituent parts, then synthesize the three dimensional geometry model of said projectile by assembling the constituents.

   Use finite element pre-processing software to divide the projectile digital geometric model into mesh, and define the material models of the different parts of the projectile in finite element software.

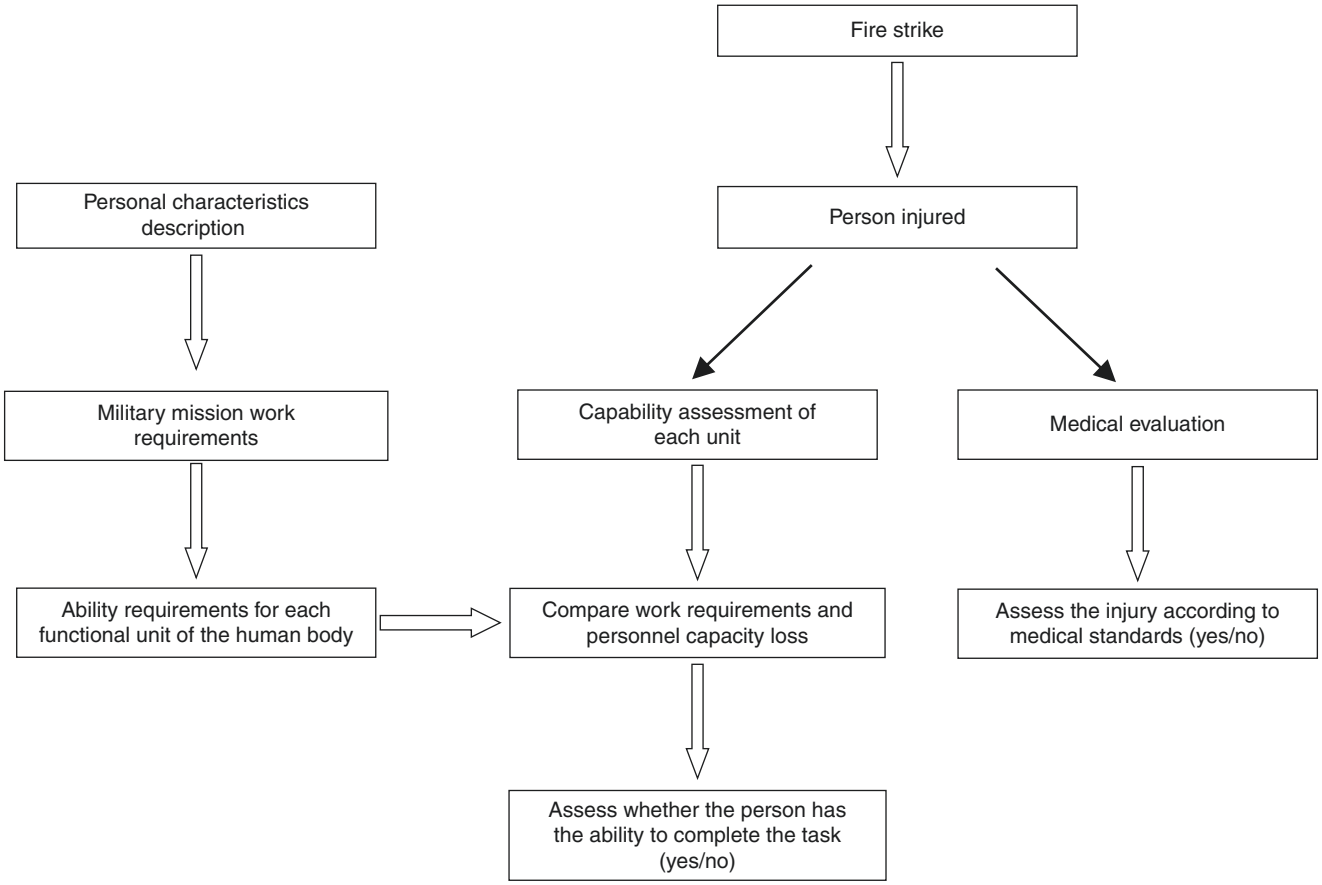

Fig. 5 Flowchart of the assessment of the combat capability of the wounded

3. Calculation of the bodily penetration ballistic trajectory of the projectile: Within finite element software, the finite element model of projectile and the finite element model of human body are positioned and assembled according to certain positional relationships, then define the type of contact between the projectile and body tissue, and material failure criteria, and allow the software to automatically complete numerical solution.
4. Extraction and statistical processing of calculation results: Make use of finite element processing software to visualize calculation outcomes in the previous step, including trajectory of the projectile, shape of the wound passage, and so on. Confirm the types of tissues on the path of trajectory of the projectile, calculate the total volume of the wound passage and the volumes of the different types of tissues of the wound passage, and obtain data such as speed and energy of projectile, and pressure applied to tissues surrounding the projectile trajectory.
5. Evaluation of injury condition: In terms of judging criteria of injuries caused by high-speed projectiles such as ammunition and fragment, early on, injury status judging criteria included kinetic energy, conditional casualty probability as standard of injury status judging criterion, energy transfer in gelatin as evaluation standard, and incapacitation and casualty evaluation principles based on scores given by experts. Computer simulation analysis of ballistic injury specifically requires the appropriate judging criteria applied in combination with tasks shouldered by combatant, then overall evaluation is formulated based on computation results.

Method and Steps of Ballistic Injury Simulation Analysis Illustrated Via Handgun Penetrating Wound of the Lower Limb

Construction of Three Dimensional Anatomical Model and Finite Element Model of Lower Limbs

Materials from the "Chinese Visualization Human Data Set" (Fig. 6a) of the Third Military Medical University Hospital are used as two dimensional section image of human lower limbs. Each image in the data set is categorized as muscle, compact bone, or spongy bone, respectively, colored in green, white, or red (Fig. 6b). Images of

each layer are configured through spatial registration, then imported into the Mimics software to produce 3D model (Fig. 6c), which would then be exported in the .stl format. Import the .stl document into finite element pre-processing software Hypermesh to divide three dimensional model of lower limb into mesh (Fig. 6d), which would then be exported in the .k format. Import the .k document into explicit transient nonlinear finite element analysis software LS-DYNA to define the properties of the materials of the lower limb. Muscle tissue are assigned viscoelastic material properties, while bone tissue are given linear elastic material properties.

Construction of Projectile Digital Geometric Model and Finite Element Model

Pistol bullet parameters are based on the 9 mm Makarov cartridge. When constructing geometric model, simplify its structure into the two components of case and lead core, then Solidwork software is used to complete the drawing of geometric shapes and create 3D models, which would then be exported as .stl files. Import .stl documents into finite element pre-processing software Hypermesh to divide the bullet into mesh, which would then be exported as .k files. Import .stl documents into explicit transient nonlinear finite element analysis software LS-DYNA to define the material properties

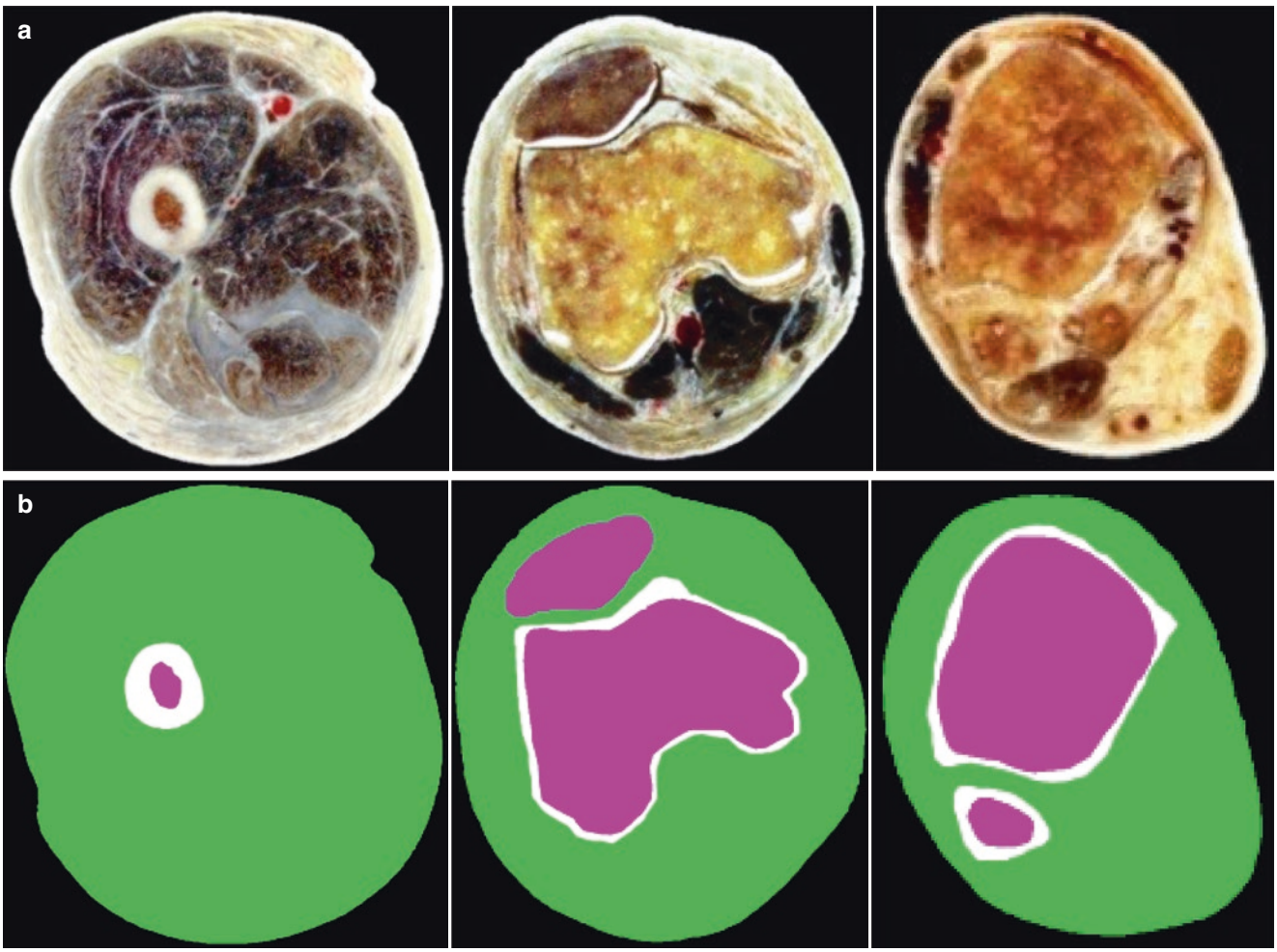

Fig. 6 Schematic diagram of the construction of the three-dimensional model and finite element model of the anatomical structure of the lower extremities. (**a**) Original image of 2D section of human lower limb, showing thigh, knee, and calf from left to right. (**b**) Two-dimensional image processed by image segmentation that correspond to image (**a**), with the zones colored in green, white, and red, respectively, corresponding to muscle, compact bone, and spongy bone. (c) Three-dimensional image of the lower limb, with the left image showing the surface, and the right image showing the interior. (**d**) Finite element mesh modeling of the lower limb, with the left image showing the surface, and the right image showing cross sections

Fig. 6 (continued)

of the bullet. Both the case and core are given the plastic material property (using the Johnson-Cook plastic material model) (Fig. 7).

Calculation of Numerical Simulation of Lower Limb Penetrated by Bullet

In finite element analysis software LS-DYNA, configure the 9 mm pistol bullet to enter the fleshy muscle part of the thigh horizontally from the outside (Fig. 8). Pistol bullet speed is standard, as in 360 m/s. The type of contact between bullet and lower limb muscle is set to penetrating contact, the type of contact between muscle and bone is set to automatic surface-to-surface contact, and the constraint boundary condition of the model is set to restrict the degree of freedom of the lower limb model in the vertical direction. Said calculation model works within the dynamic environment of LS-DYNA, and the solutions and results are exported as . d3plot files.

Extraction and Statistical Processing of Calculation Results

Import .d3plot files into finite element processing software LS-PREPOST to achieve visualization and data analysis of results of simulation of lower limb penetrated by bullet. The trajectory of the bullet in lower extremity tissue, wound passage formation process (Fig. 9a) and changes in form of wound passage after bullet passes through tissue (Fig. 9b) are illustrated by way of the longitudinal section of the central axis of the bullet. It can be seen that the wound passage is a perforating injury, and the diameter of the entry wound and exit wound of the passage created when the bullet just passed through the lower limb muscle tissues are respectively 2.98 cm and 1.66 cm, with the entry wound being clearly larger than the exit wound, and a transient cavity volume of roughly 58.58 cm^3. Thereafter, due to pulsation in the transient cavity, the form of the wound passage changed, and at 1000 μs, diameter of the entry wound and exit wound are respectively 3.74 cm and 4.49 cm, showing an obvious

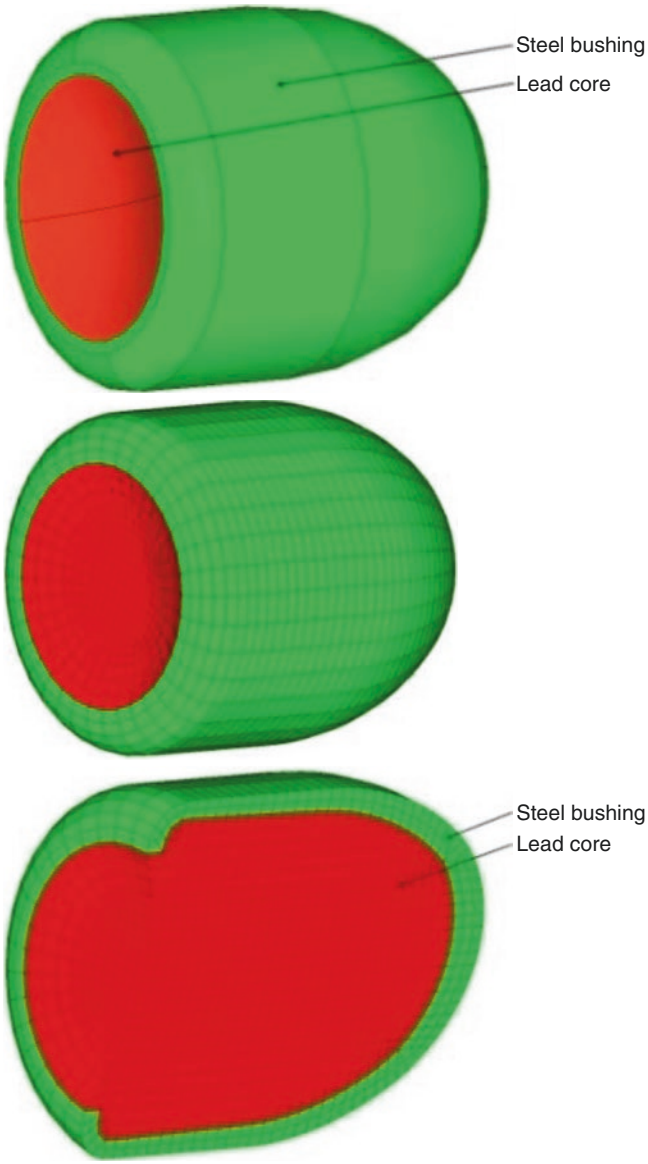

Fig. 7 Schematic diagram of geometric model and finite element model of 9 mm pistol bullet

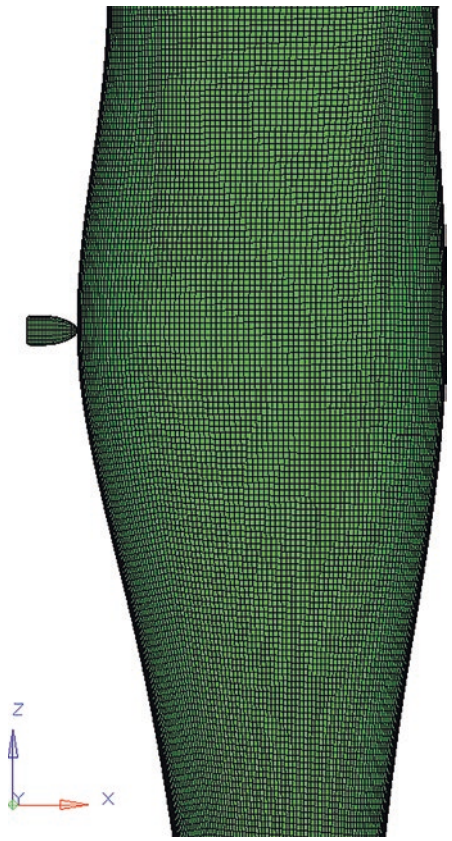

Fig. 8 Calculation model of pistol penetration into lower limbs

enlargement of the exit wound to the point of slightly surpassing the entry wound in size, and transient cavity volume was approximately 180.39 cm^3. The velocity and energy curve of bullet (Fig. 10) indicate that the bullet's initial velocity was 360 m/s, and residual velocity upon perforating and leaving the lower extremity was 307 m/s; initial energy was 413 J and residual energy was 308 J, equating to 105 J of energy dissipated in the tissues of the lower limb (energy absorbed by the tissues). Stress generated when the bullet penetrated the muscle propagated to the tissues surrounding the wound passage following the pulsation of the transient cavity (Fig. 11). At 1000 μs, equivalent stress was 228 kPa, which attenuated to 0 at the 4.58 cm mark of the wound passage.

Overall Evaluation of Injury Status

1. Based on kinetic energy as standard, a projectile possessing kinetic energy of 78 J would be deemed capable of injuring or killing, and a person would be deemed injured or killed when struck by one such projectile. When a projectile possesses kinetic energy ≥78 J, the injury or death probability $P(I/H)$ of a person is 1; and when a projectile possesses kinetic energy of <78 J, the injury or death probability $P(I/H)$ of a person is 0. In the case illustrated here, the initial kinetic energy of the bullet was 413 J, and it may be determined that injury or death probability of person hit by this bullet would be 1.
2. The method of applying conditional casualty probability as standard of injury status judging criterion holds that for the same kind of projectile with mass m and velocity v, the severity of trauma created is a function of mv^{β}.

$$P_{(I/H)} = 1 - e^{-a\left(mv^{\beta} - b\right)^{n}} \tag{7}$$

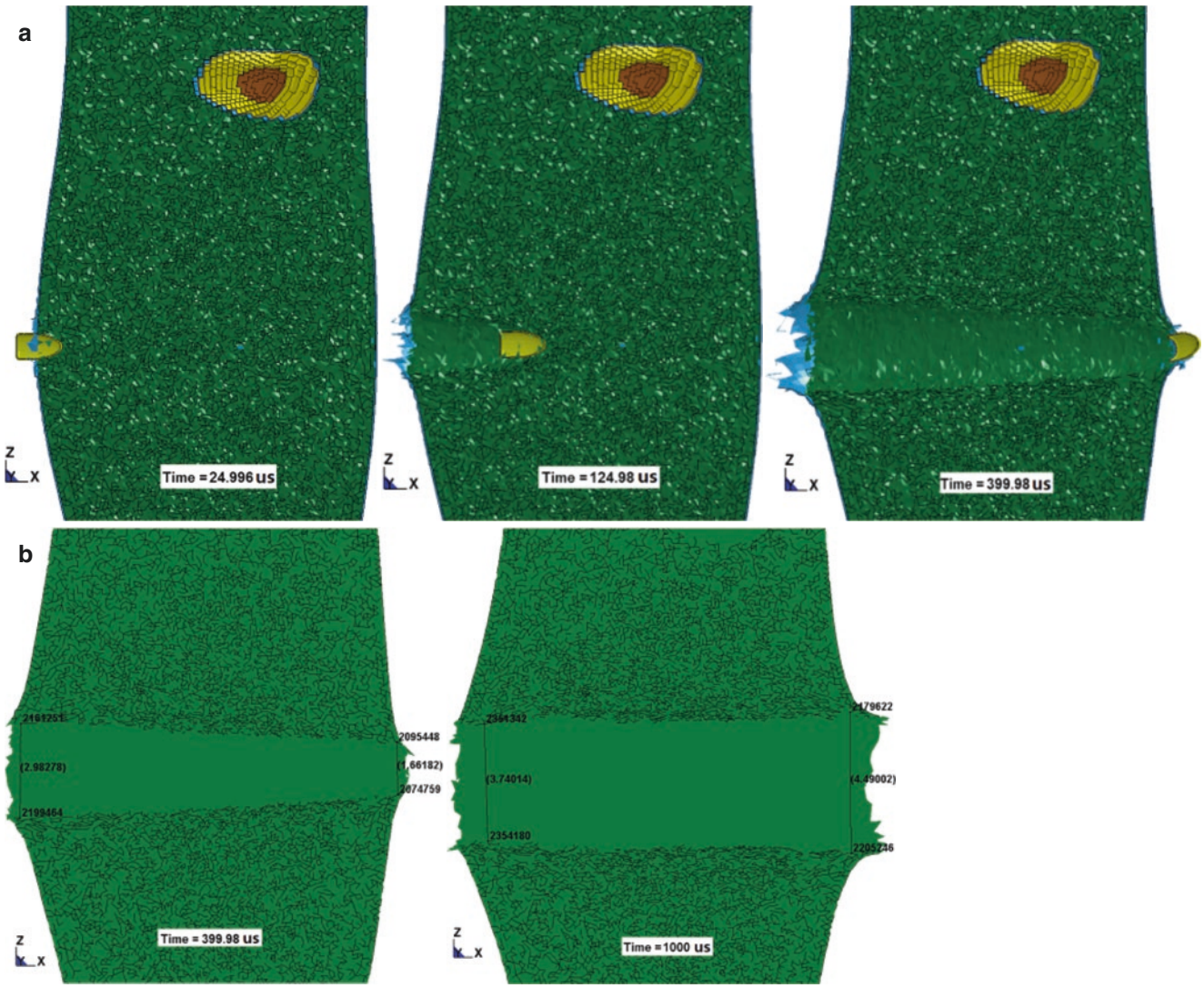

Fig. 9 Visual display of simulation results of warhead penetration into lower limbs. (a) The trajectory of bullet in the lower extremity. The left, middle, and right pictures respectively show the bullet just entering the muscle, in the muscle tissue, and just passing through the muscle. (b) Shape of wound after the bullet penetrates the tissue. The left image shows the wound shape when the bullet just penetrates the muscle; the right shows the shape 600 μs after the bullet penetrates the muscle

The probability β that a soldier on a given mission would lose combat capacity during a specified period of time after a random hit is the value determined by experiment results ($\beta = 1.5$), and a, b, and n are constant numbers determined by the type of projectile, mission assigned to said soldier and time of loss of combat ability. For the attack 30 s and defend 30 s situations, see values for a, b, and n in Table 10.

In the case illustrated here, the bullet has mass 6.1 g and initial velocity 360 m/s, so based on calculation using Formula (6), it may be predicted that the probability of a soldier losing combat ability would be 1 under the two specific conditions of attack 30 s and defend 30 s.

3. According to the method of using energy transfer in gelatin as evaluation standard, the incapacitation probability of a soldier hit by a random projectile hits a soldier and incapacitated him may be calculated according to the following formula.

$$P_{(I/H)} = \frac{1}{1 - e^{-(a+bx)}} \tag{8}$$

In the formula, x is penetration depth, a and b are constant numbers determined by mission assigned to said soldier and time of loss of combat ability, with the values identical to those in Table 1. In the case illustrated here, the bullet perforated the body part struck, with a penetra-

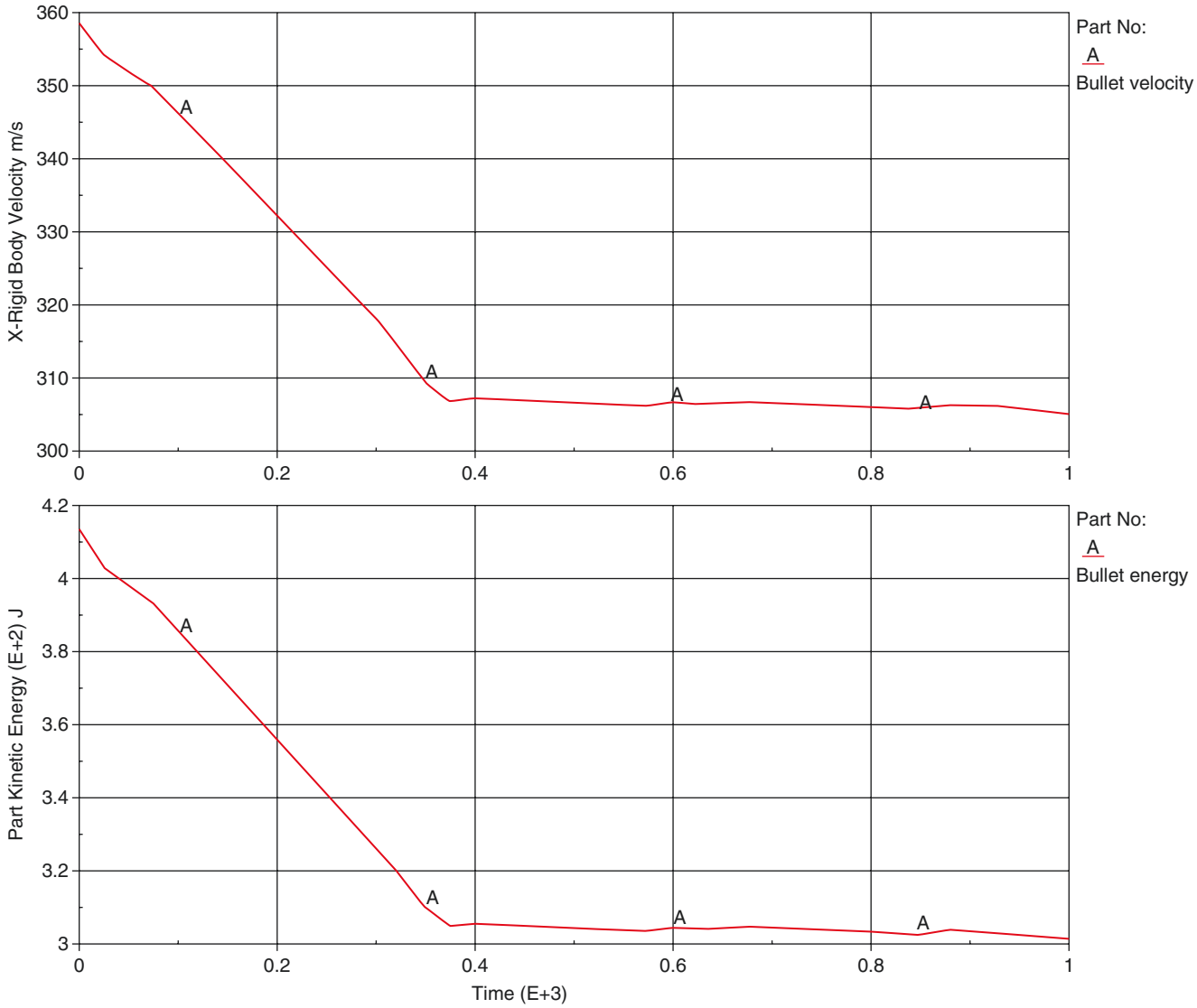

Fig. 10 Velocity and energy curve of bullet

tion depth of 13.5 cm; thus, based on Formula (4), it may be predicted that the probability of a soldier losing combat ability would be 1 under the two specific conditions of attack 30 s and defend 30 s.

Computer Analysis of Blast Injury

The theory behind computer analysis of blast injury is based upon digitized model of the human body, and mechanical response data such as stress, strain and acceleration caused by shock wave overpressure in human organs and tissues are obtained by numerical calculation. According to the injury thresholds (injury judgment criteria) of different tissues, injury level of targeted organ is predicted and evaluated. Method and steps of computer analysis of blast injury are detailed below.

1. Construction of digital anatomical model and finite element model of human body: With two-dimensional cross-sectional images of the human body, CT or MRI continuous scan images as raw data set, organize, segmentize, and code each image in the dataset, register the corresponding spatial relationships of each layer of image, and import images into image reconstruction software to generate a 3D model.

   Use finite element pre-processing software and finite element analysis software to divide digital anatomical model of human body into mesh, then assign the appropriate material models to different human body tissues based on their respective material properties.
2. Construction of shock wave load model: There are two common methods for using finite element analysis soft-

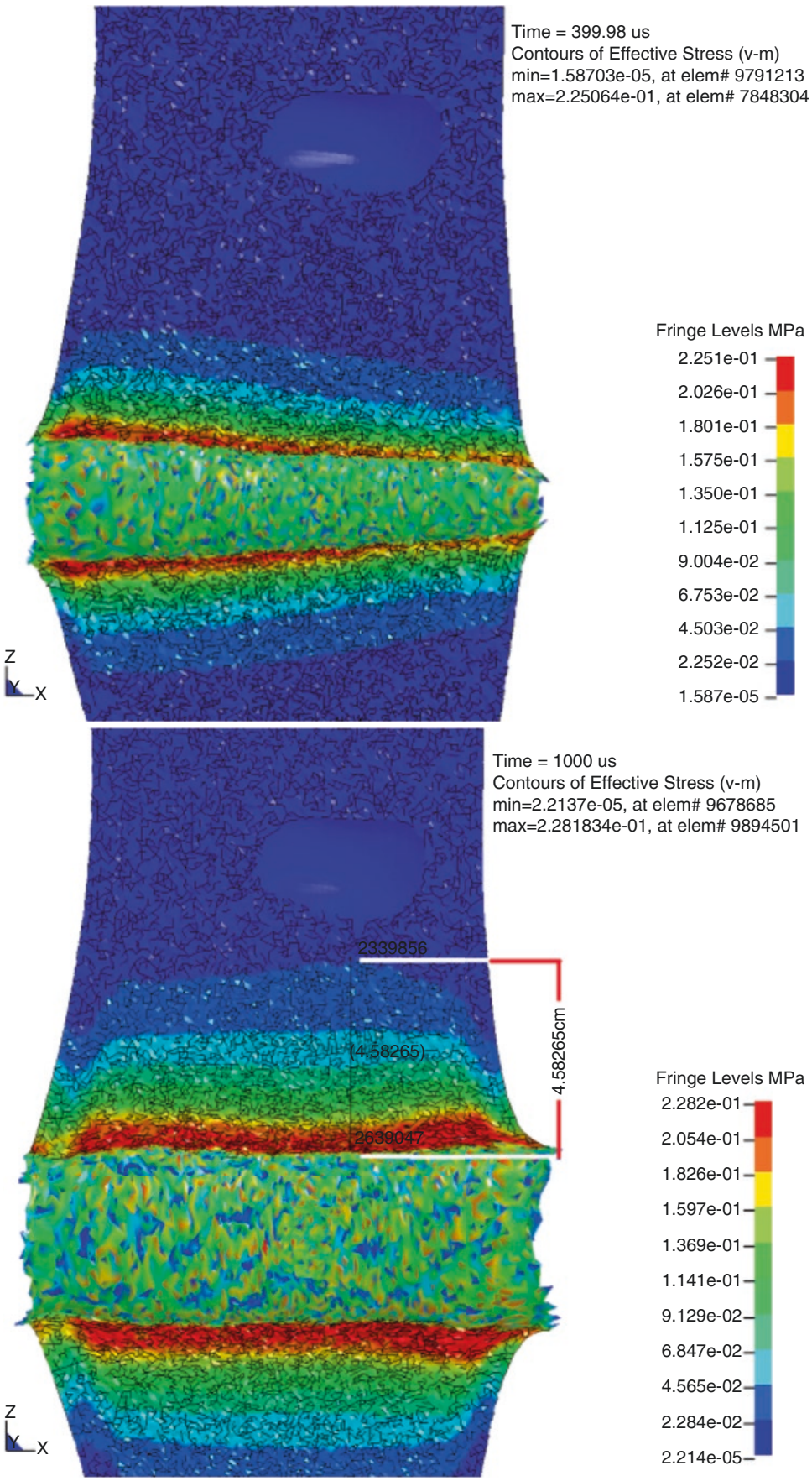

Fig. 11 Stress distribution diagram of wound tissue

Table 10 *a*, *b*, and *n* values for attack 30 s and defend 30 s

Mission and time of loss of combat ability	*a*	*b*	*n*
Attack 30 s	0.76442	31,000	0.49570
Defend 30 s	0.88771	31,400	0.45106

ware to build shock wave load model. One is to establish a pre-set pressure–time curve, and the other is to utilize simulated explosive detonation to generate pressure pulse. Utilizing explosive detonation to generate pressure pulse in air grid would restrict parameters such as intensity of pressure pulse and effective duration.

3. Numerical solution of shock wave loading on human body: Define pressure boundary, contact model and calculation method in finite element analysis software, then calculate values of shock wave loading on human body.
4. Extraction and statistical processing of calculation results: Use finite element post-processing software to achieve visualization and data analysis of results of numerical calculation outcomes, extract mechanical response data of targeted organ, and carry out statistical analysis of stress distribution and acceleration changes of the targeted organ.
5. Injury status evaluation: Bowen model, Stuhmiller model, and Axelsson model are the main nonauditory apparatus blast injury evaluation standards. The Bowen model determines injury level based on the shock wave's peak overpressure and positive pressure effective duration. It provides the peak overpressure necessary to cause 1%, 50%, 90%, and 99% fatality 1 h injury on a person weighing 70 kg under different positive pressure effective duration. In injury status evaluation, the survival rate of a person weighing 70 kg subjected to blast injury may be projected based on shock wave's maximum peak pressure and positive pressure effective duration as measured. The Bowen model is applicable to evaluation of blast injuries sustained in a free field. The Stuhmiller model relies on shock wave load in front of, behind, to the left and right of the thoracic cavity to predict lung injury probability and lung injury severity. Said model is applicable to lung injuries arising from both complex shock waves and shock wave in free field. The Axelsson model predicts overall injury score of lungs, upper respiratory tract, gastrointestinal tract, and solid organs in the abdomen based on inward velocity of the thoracic wall. This model is mainly used for evaluating injuries caused by complex shock waves. Blast injury computer simulation analysis specifically requires information about load environment in which a combatant is situated, then utilize the appropriate judging criteria and values to calculate results for overall evaluation.

Let Us Use a Human Chest Subjected to Complex Shock Wave with Overpressure of 100 kPa as Example to Illustrate the Method and Steps in Blast Injury Simulation Analysis

Construction of Digital Anatomical Model and Finite Element Model of Human Chest

CT scan imagery data of a normal Chinese adult male is used as raw data of two-dimensional section image of the human chest, which was obtained from 64-slice CT system manufactured by GE. CT scan data are grayscale images in Dicom format and has resolution of 512 bit × 512 bit × 8 bit, scanned parallel to the orbitomeatal line and each scan layer has a thickness of 0.625 mm. Different anatomical structures are recognized and segmented based on CT images (Fig. 12a), including muscle tissues (including skin), bone tissues (sternum, ribs, vertebrae, collar bones, shoulder blades, and humeri), and internal organs (heart and lungs), with the different organs and tissues coded in different colors. To lower modeling difficulty, everything in the torso except for bone tissues and internal organs are considered the same type of tissue, and muscle tissue organ material properties were applied during the modeling process. The segmented images are imported into the Mimics software to produce 3D model (Fig. 12b), which would then be exported in the .stl format. Import .stl documents into finite element preprocessing software Hypermesh to divide the chest's 3D model into mesh and create the chest's three-dimensional finite element model (Fig. 13), which would then be exported as .k files. Import the .k document into explicit transient nonlinear finite element analysis software LS-DYNA to respectively describe bones and organs using elastic–plastic constitution and viscous–elastic constitutive model.

Construction of Shock Wave Load Model

To simplify the calculation process and minimize calculation quantity, direct loading of stress wave is adopted to simulate the impact of shock wave inflicted upon the human body. Figure 14 is stress wave peak value–time curve. Space domains are described by void material *MAT_NULL and linear polynomial equation of state *EOS_LINEAR_POLYNOMIAL.

Numerical Solution of Shock Wave Loading on Human Body

Firstly, use Define LS-DYNA to construct simulated complex shock wave environment. The method entails building a cubic air grid, then inserting the chest model and a flat steel panel into the air grid, and complex shock wave is configured by adjusting the difference between the chest and the steel plate's reflection surface. Complex shock wave configured this way is relatively simpler and also easier to understand. See Fig. 15 for specific calculation of working condition.

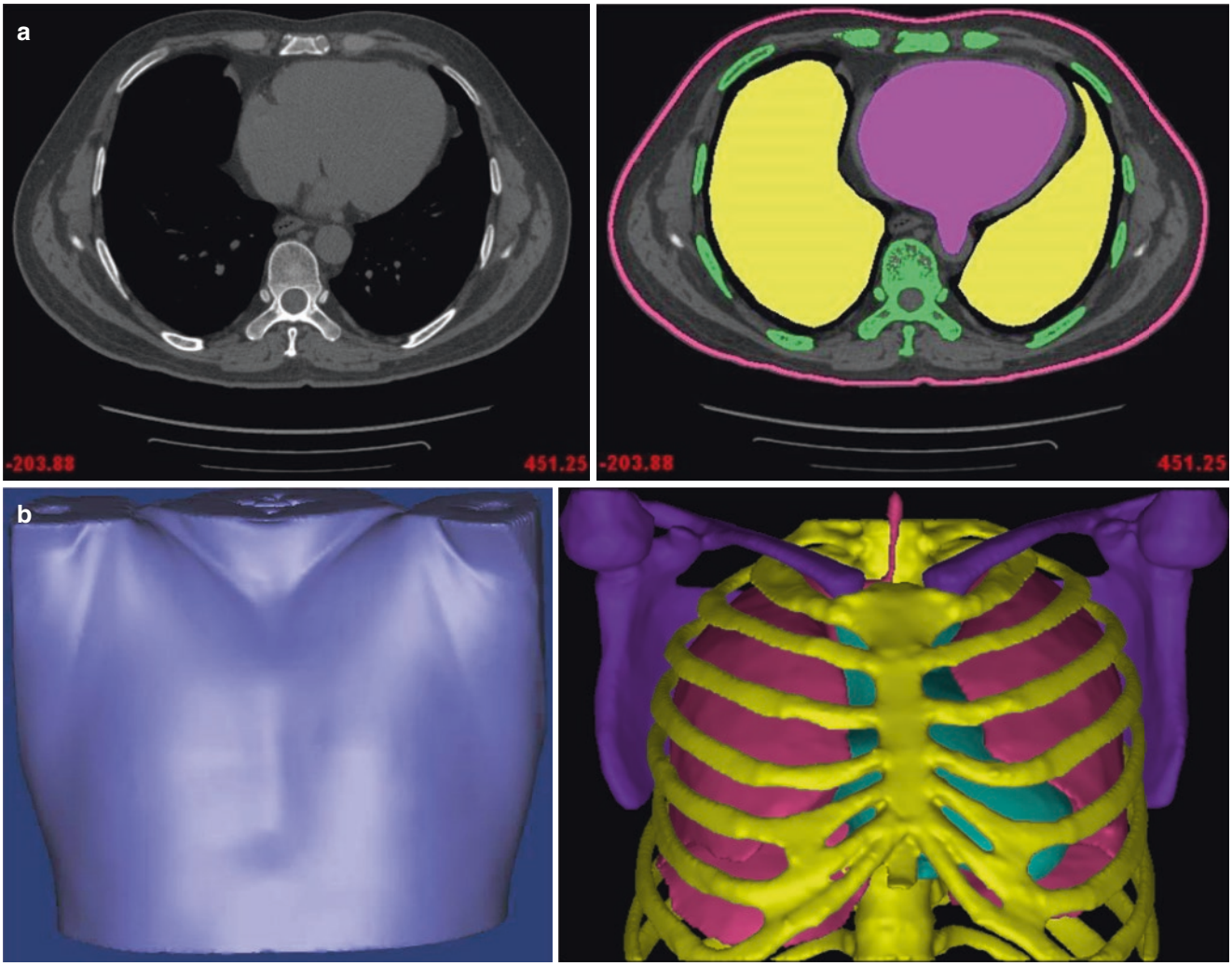

Fig. 12 Schematic diagram of three-dimensional model of human chest. (**a**) Left: Original cross-sectional image of the human chest CT scan. Right: Cross-sectional image after image segmentation. (**b**) Three-dimensional model of the anatomical structure of human chest. Left: body surface; right: thorax and internal organs

The chest model is placed inside the air grid at a distance of 25 cm from flat steel panel, then loaded with stress from a 200 kPa shock wave. The blast wave inflicts impact through fluid substance-air against solid substance-human body and steel plate. The process is an interaction between fluid substance and solid substance, so the complete fluid-solid coupling algorithm was employed. Automatic single-surface contact (*CONTACT_AUTOMATIC _SINGLE_ SURFACE_ID) is used as contact type. Said calculation model operates in LS–DYNA environment, and the solutions are exported as .d3plot files.

Extraction and Statistical Processing of Calculation Results

Import .d3plot files into finite element processing software LS-PREPOST to achieve visualization and data analysis of numerical simulations, mainly including pressure field distribution in human chest cavity after shock wave loading, stress wave propagation in the thoracic bones, rib movement speed and changes in pulmonary pressure.

1. Pressure field distribution in chest cavity: At 0.179 ms (Fig. 16a), a shock wave with stable front and propagating forward was formed. At 0.419 ms (Fig. 16b), the shock wave arrived at the surface of the thoracic cavity, and due to difference in the body's resistance, a part of the shock wave entered the body while another portion was reflected, which then coupled with a shock wave that arrived subsequently to form an even more powerful load. When the shock wave continued to propagate, the reflection overpressure at the front rapidly declined to the overpressure when there was no reflection. At 0.599 ms

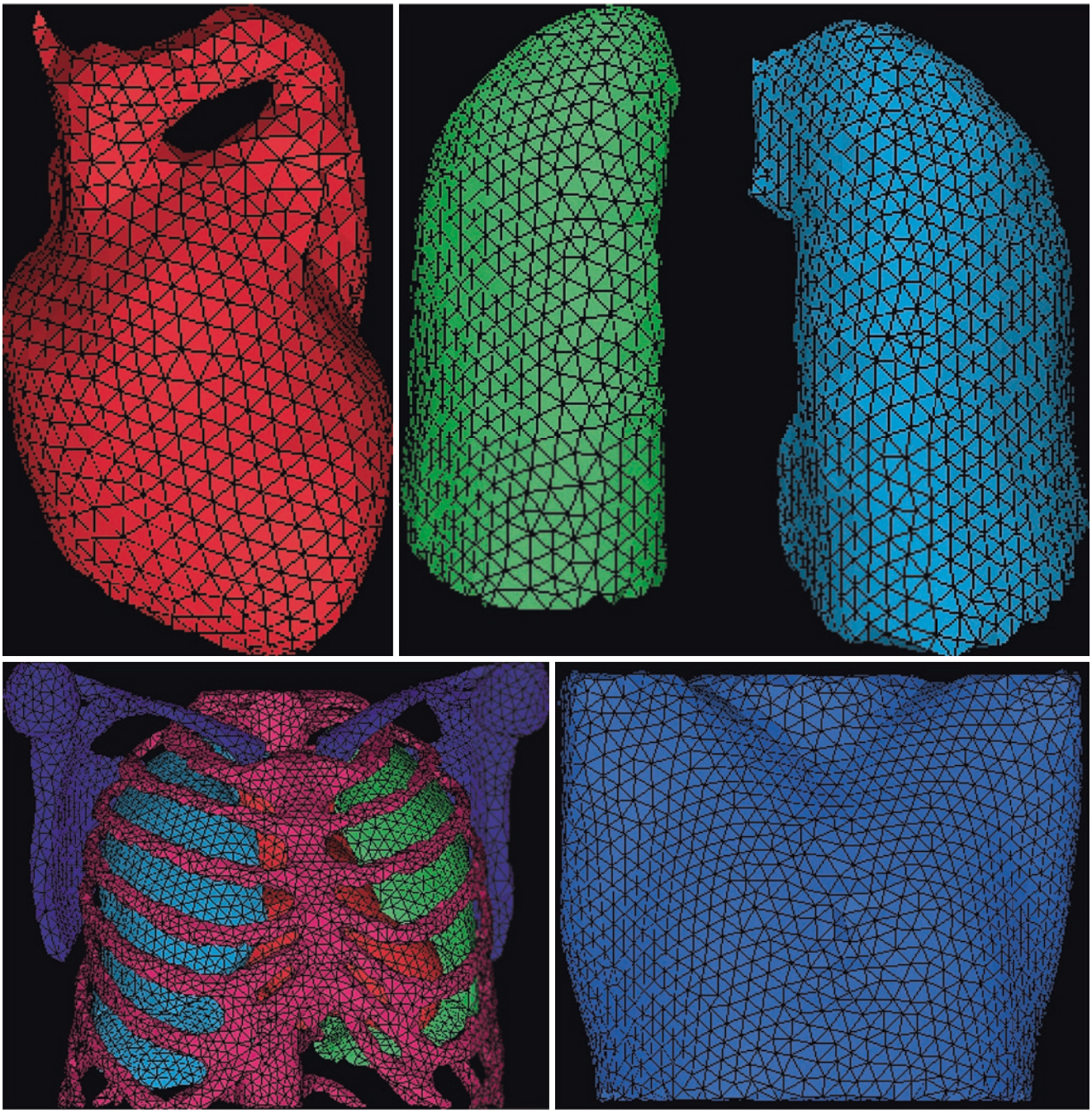

Fig. 13 Finite element model of heart. Finite element model of lungs. Finite element model of thoracic vertebrae. Finite element model of chest skin. Finite element model of chest

(Fig. 16c), the shock wave continued moving forward, and because of the negative pressure of the shock wave, negative pressure was generated on the surface of the body, right after which diffraction occurred around the chest cavity, with roughly similar pressures forming on both sides. At 1.079 ms (Fig. 16d), pressure quickly moved from the two sides of the body toward the back, and the whole chest cavity was surrounded by a pressure field. At 1.639 ms (Fig. 16e), the shock wave left the body and continued propagating forward until being reflected upon encountering the steel plate. At 2.199 ms (Fig. 16f), pressure reflected by the steel plate arrived at the back of the body, and on the one hand overlapping and scattering occurred because the reflected pressure encountered the earlier incident pressure, while on the other hand the body was once again enshrouded by a pressure field

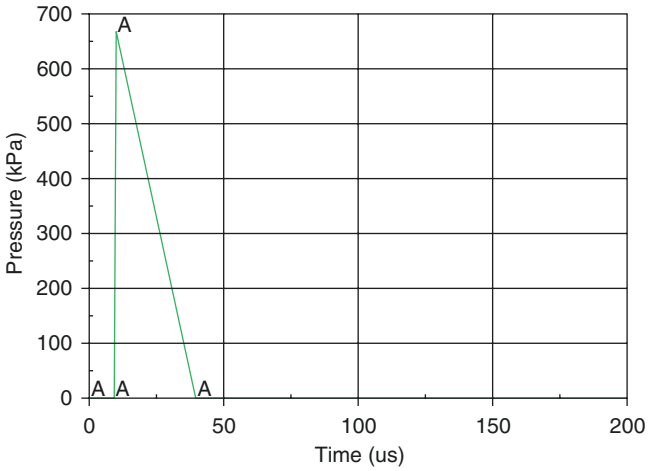

Fig. 14 Load shock wave curve

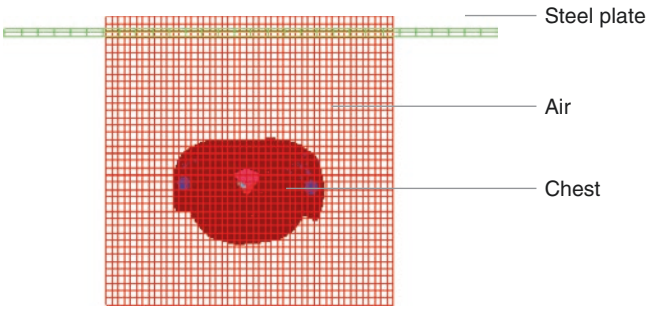

Fig. 15 Complex environment of blast injury composed of chest finite element model, air and steel plate

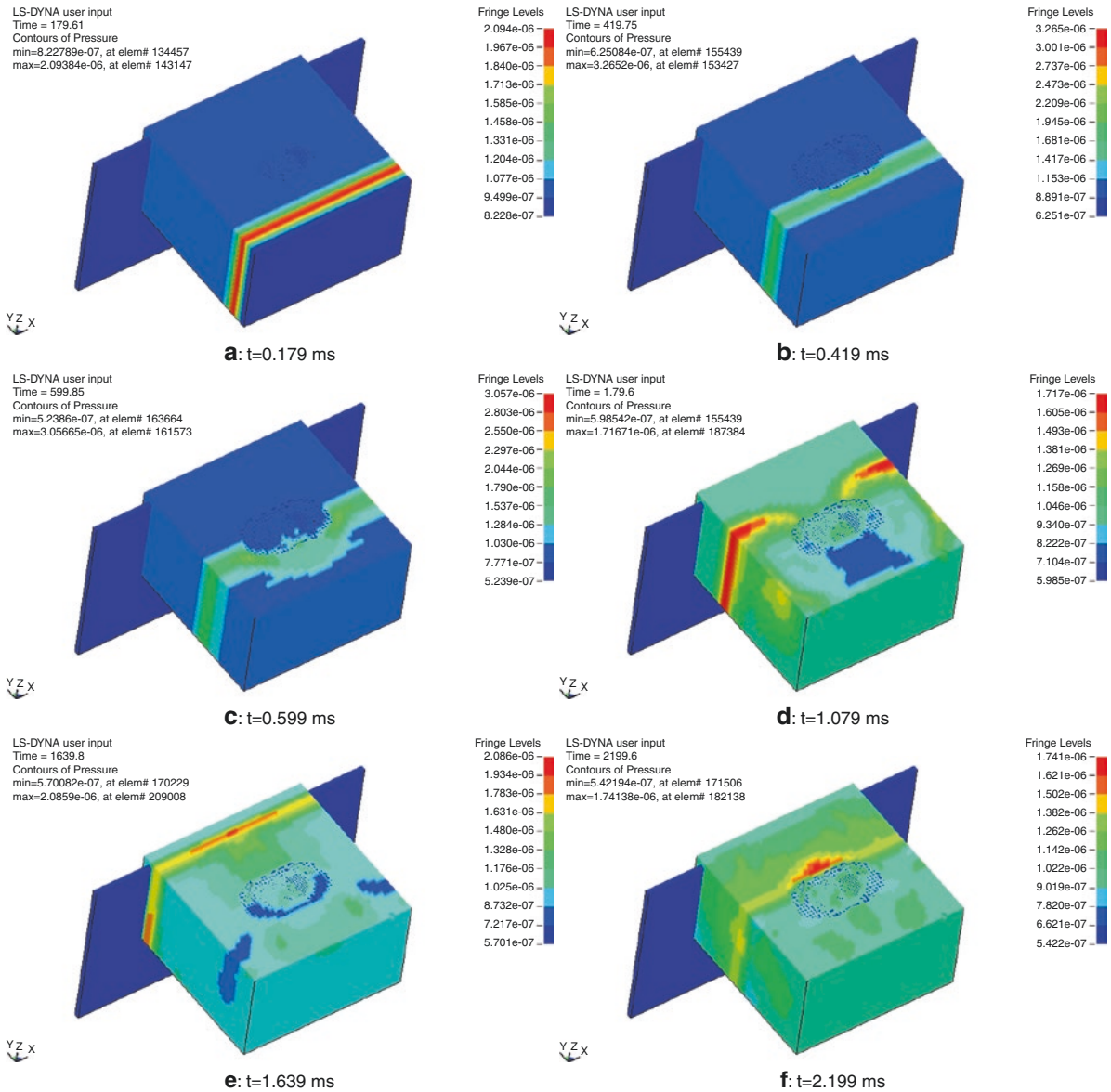

Fig. 16 (**a**–**f**) Simulated pressure field after 100 kPa complex shock wave loading

because the reflected pressure continued propagating forward. This process was repeated to complete the entire course of complex shock wave loading.

Then analysis is performed on the shock wave pressure curves at the front, back, left and right of thoracic cavity (Fig. 17). It was discovered that positions 2 and 3 on the left and right sides of the body had basically identical curves, with similar peak pressure of around 55–80 kPa. Shock wave peak pressures at four spots in the front of the body surface were highest, reaching 175 kPa. The one spot of pressure curve measurement at the back of the body firstly recorded a 70 kPa shock wave resulting from diffraction of shock wave, and then a 130 kPa reflection pressure that came from shock wave reflected from steel plate. Waveforms of the shock waves at the four positions were all overlapped with reflection waves and exhibit features of complex shock waves.

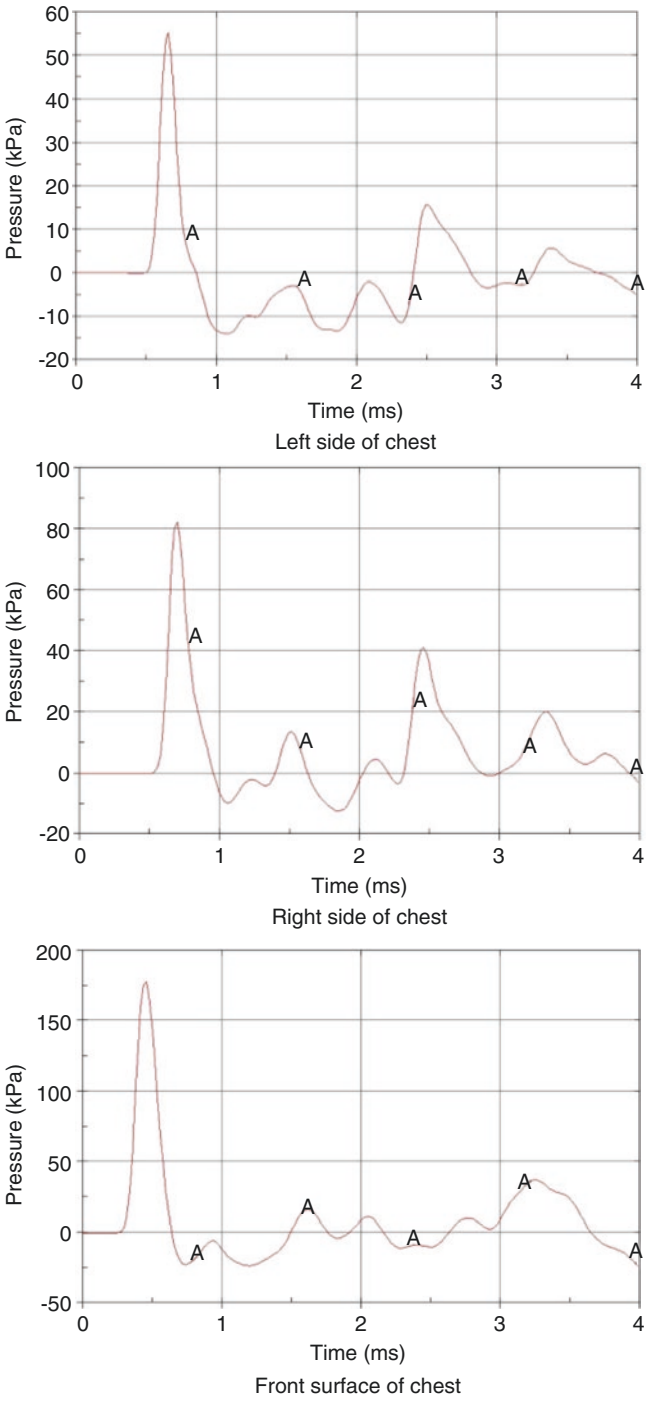

Fig. 17 Pressure curves of the front, back, left, and right positions of chest after 100 kPa complex shock wave loading

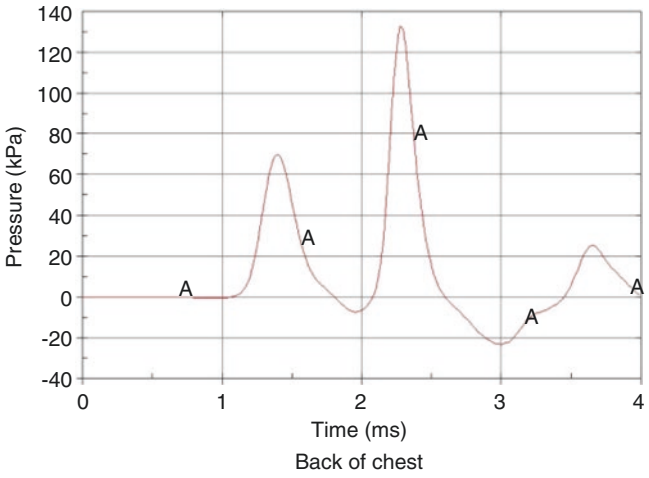

Fig. 17 (continued)

2. Propagation of stress wave on thoracic bones: After the thoracic cavity was subjected to loading of complex shock wave, soft tissues of the chest deformed inward due to the applied pressure, thoracic vertebrae moved inward also because of the applied pressure, and the ribs deformed and compressed inward in line with the direction of the action of shock wave, which then made contact, rubbed and collided against the lungs, causing the contacted parts of the lung tissues to deform.

At 19.607 μs (Fig. 18a), when the stress wave entered the body, the manubrium, sternum body, and xiphoid process showed the darkest colors. At 299.59 μs (Fig. 18b), the fourth, fifth, sixth, and seventh costal cartilages began to darken in color. Between 339 μs (Fig. 18c) and 379 μs (Fig. 18d), the sternum body and ribs began to darken in color, in particular the fifth, sixth, and seventh ribs. At 439 μs 339 μs (Fig. 18e), other ribs started to darken, and at 2299 μs (Fig. 18f), all ribs and collar bones turned darker.

3. Movement speed of ribs: Choose the surface of the fifth ribs at the chest as movement position of ribs to obtain the highest inward velocity of ribs, which was 1.6 m/s. Curves of the changes in rib velocity are shown in Fig. 19.
4. Distribution of stress field on the lungs: After the chest was subjected to loading of shock wave pressure, the pres-

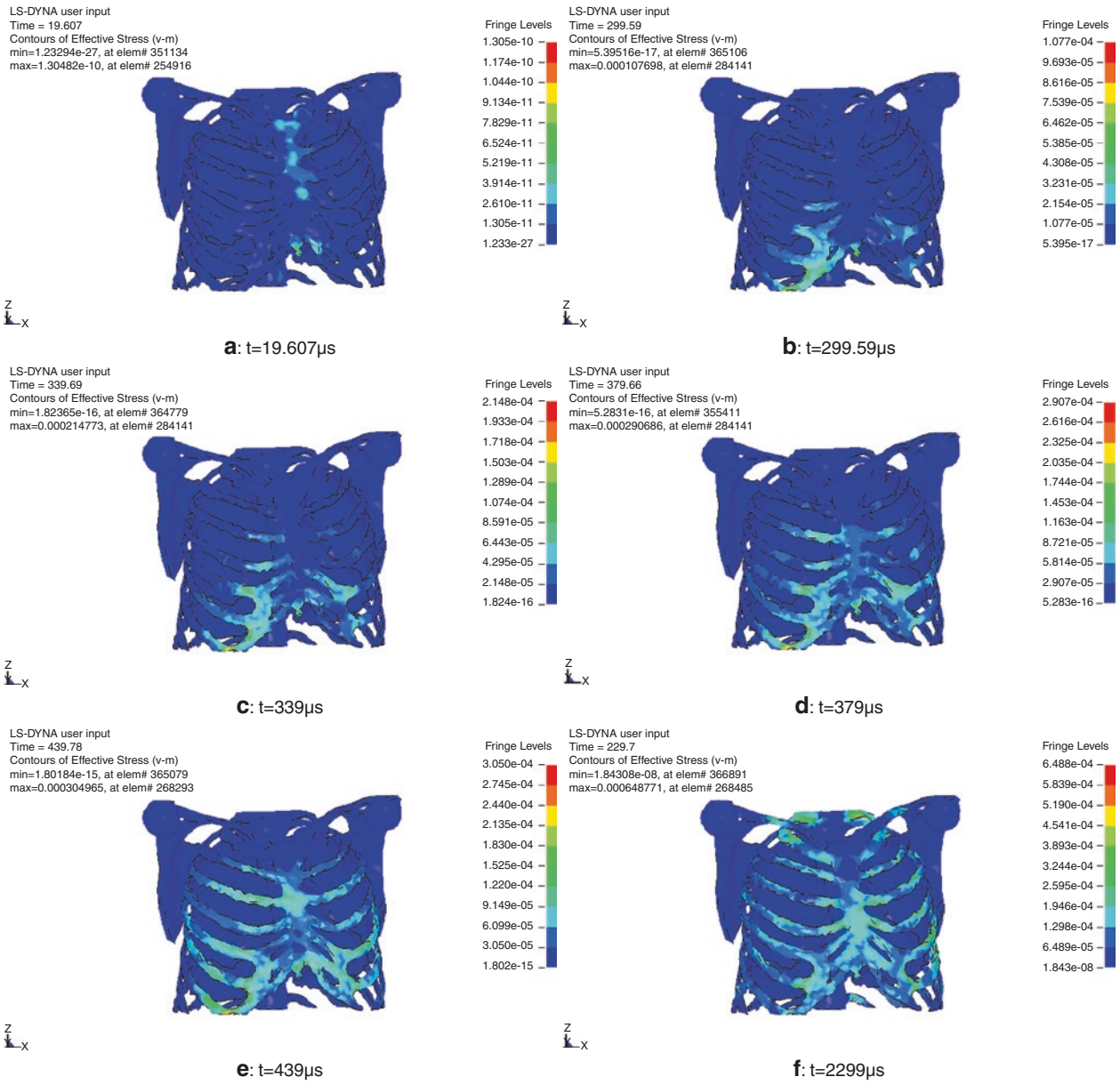

Fig. 18 (**a**–**f**) Stress wave propagation in thoracic bones after 100 kPa complex shock wave loading

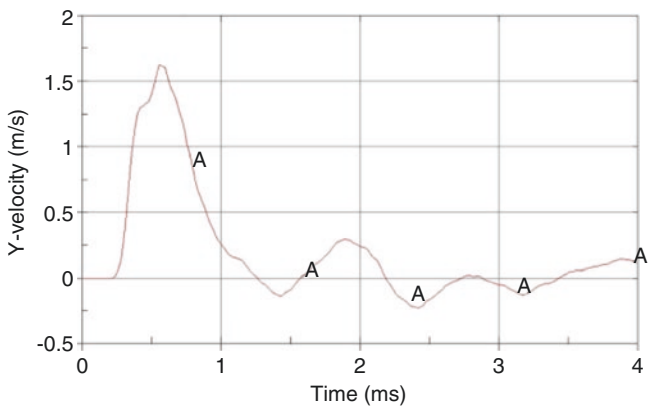

Fig. 19 Velocity curve of rib movement after 100 kPa complex shock wave loading

sure was variously transmitted to the lungs and heart via muscle soft tissues, thoracic bones, and ribs. After being subjected to pressure, the lungs collided, pressed and deformed against the heart, ribs, and thoracic bones, with stress forming on surface of lungs due to deformation.

At 399.79 μs (Fig. 20a), the lower lobes of the lungs showed the darkest color. At 519.59 μs (Fig. 20b), the color might spread to all directions. At 819 μs (Fig. 20c), the upper lobes of the lungs started to gradually darken, while the bottom of the lower lobes were the darkest. At 1820 μs (Fig. 20d), the entirety of the lungs turned dark. From the back of the lungs, it may be seen that the bottom of the lower lobes were the darkest at 1499 μs (Fig. 20e) and 1820 μs (Fig. 20f).

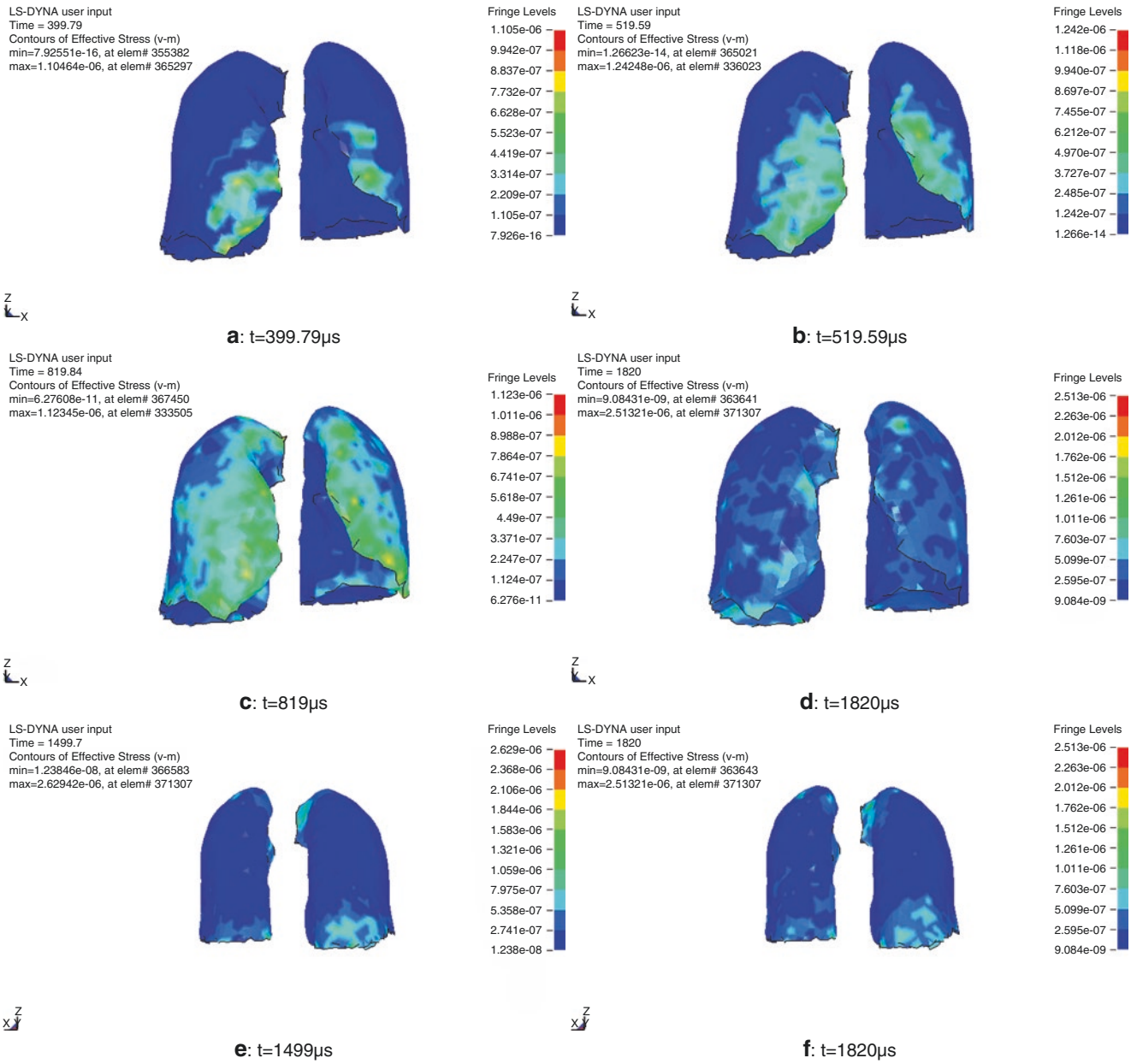

Fig. 20 (**a**–**f**) Lung stress wave propagation after 100 kPa complex shock wave loading

5. Changes in internal pressure of the lungs: After the chest was subjected to loading of 100 kPa complex shock wave, the fifth rib at the chest was used as position for cross section of the lungs. All measurement spots of the left and right lungs were selected, then post-processing procedure was undertaken to obtain the average pressure curve inside the lungs (Fig. 21). It may be observed that the greatest average pressure in the left lung was 38 kPa, and that in the right lung was 34 kPa.

Injury Status Evaluation

Greatest rib velocity after the loading of 200 kPa complex shock wave was 1.6 m/s. According to thoracic wall velocity-based injury status evaluation standard (Axelsson model), velocity less than 3.6 m/s indicates no injury, and therefore, it is predicted that the loading of 100 kPa complex shock wave would not cause injury to the human body.

After the loading of 100 kPa pressure, the average pressure in the left lung was 38 kPa, and that in the right lung was

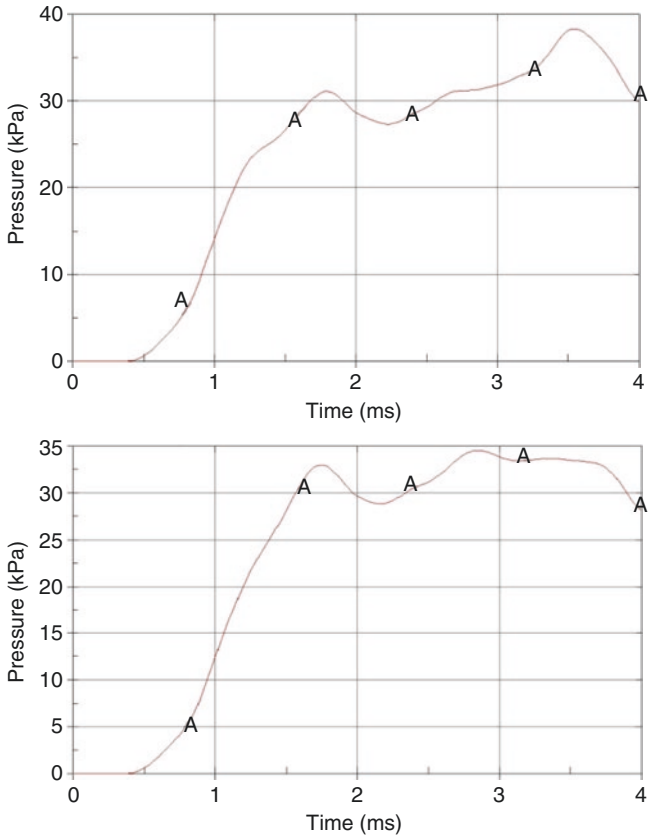

Fig. 21 Average pressure of left and right lungs after 100 kPa complex shock wave loading

34 kPa. In accordance with the pulmonary internal pressure injury standard proposed by Greer, pressure less than 60 kPa indicates no injury, pressure between 60 and 100 kPa indicates slight injury, and that ranging between 100 and 140 kPa indicates minor injury. Thus, based on numerical calculation outcomes, it may be projected that the loading of 100 kPa complex shock wave would not cause injury to the human body.

After the loading of 100 kPa pressure, pressure equivalents at the front, back and two sides of chest were respectively 175 kPa, 130 kPa and 80 kPa. Based on calculation using the Stuhmiller model, outcomes indicates that probability of absence of lung injury is 100%.

5 Section Five: Protection Against and Treatment of Injuries from Conventional Explosive Weapons

5.1 Protection Against Explosive Fragmentation Injuries

In modern combat scenarios, probabilities of fragmentation injuries arising from artillery, grenades, rockets, hand grenades and land mines continue to rise, and such injuries account for 67–75% of all injuries and casualties. Of these, deaths due to combat injuries to the head, chest, or abdomen make up 82–87% of total, far higher than death rates associated with injuries to the limbs. Statistical analysis of combat injuries indicate that protecting the head, chest and abdomen alone, which merely comprise 39% of the surface area of the human body, can immensely reduce death rate, and bulletproof vests and bulletproof helmets play key roles in protecting these vital parts of the body. The USA has claimed that during World War II, infantry protective equipment saved the lives of at least 70,000 American soldiers. Relevant research reports from the UK state that bulletproof vests can lower injury rate by 27% and reduce death rate by 40%. Therefore, developments in bulletproof vests and protective helmets have progressed by leaps and bounds ever since World War II.

The advancements in infantry protective equipment is first and foremost manifested in progress in protective materials. The research and development advancements in protective materials are pivotal, evolving through stages such as carbon steel, manganese steel, titanium alloy, aluminum alloy, nylon fiber, glass fiber and Kevlar aramid fiber, as well as thermosetting resin composite. These developments continue to enhance the protective functionality of infantry protective gears. 1984 was a milestone year, the successful research and production of the PASGT by the US military marked a momentous point in the development of modern military helmet, and since then, helmets fabricated from nonmetal composite bulletproof materials have risen as the mainstream in modern military helmets.

Following continual enhancement in the mechanical performance of high-strength and high-modulus fiber such as aramid fiber and ultra-high molecular weight polyethylene fiber, the bullet resistance capability of nonmetal bulletproof helmets also improved in tandem, while weights were reduced. However, while continually rising bullet resistance capability and protection requirements meant that the helmets were increasingly more successful in stopping harms from penetration of high-speed projectiles, the nonpenetrating transient deformation and permanent deformation at the side opposite from projectile contact also increased. The reasons is because composite helmets utilize bullet-resistant mechanism wholly different from metal helmets. Early metal bulletproof helmets rely on their own hardness to dull high-speed fragments, and resist bullet penetration by creating a bigger compression area and dissipating collision energy. Common nonmetal bulletproof helmets, however, rely on tensile deformation of the fibers of bulletproof materials to absorb the kinetic energy of high-speed fragments to stop bullets, and the resulting bulging and deformation mechanism is a necessary condition for bulletproof functionality.

In addition, impact energy of a high-speed projectile is proportional to the square of its velocity. For example, when

a bullet moves at 400 m/s and another moves at 600 m/s, the impact energy between the two increased by more than one-fold. The compression zone and deformation zone created in a bulletproof helmet may possibly cause the inside of the helmet to collide against the head, leading to blunt head and brain injury.

Bulletproof vests are mainly used to protect key organs in the chest and abdomen areas from the injury and lethal effects of bullets and fragments. As munition technology advances, bullets and fragments become more penetrative, and especially starting from the middle of the twentieth century, continual advancements in high-energy ammunition render bulletproof vests made from protection materials as represented by nylon largely useless in effectively defending against even small-caliber bullets and high-speed fragments. Under this circumstance, American company DuPont developed the aromatic polyamide fiber (as in "Kevlar"). Kevlar vests are noted for features such as softness, light weight, heat tolerance, thermal insulation, low flammability, and outstanding bulletproof performance. Its bulletproof performance is twice that of alumina ceramic material, thrice that of soft materials like nylon, and five times better than steel-based materials. Yet its mass is 45% lighter than glass fiber, and relative density only 1/5 of steel. Modern bulletproof vests may be classified as either soft body armor or hard body armor. The former is made from high-strength fiber materials that can defend against knife attack and low-speed projectile, while the latter can protect the wearer from high-speed fragment and rifle bullets of calibers such as 5.8 mm and 7.62 mm.

5.2 Protection Against Blast Injuries

Protecting against blast injuries involve many different factors such as location of the person, infantry gear and equipment, armored chamber, and defensive fortification.

5.2.1 Protection Through Simple Actions

Estimating based on the Bowen curve, for a person in an explosion that occurred in a wide open space, threshold for 1% fatality when the person is standing straight is 190 kPa, but when the person lies down parallel to the direction of blast wave propagation, threshold for 1% fatality would rise to 280 kPa. Therefore, during the instant of explosion, lie down parallel to the direction of blast wave propagation can effectively reduce the injury effects of the blast wave.

Pressure difference is one of the main injury mechanisms of shock wave, and therefore, keeping one's month opened at the instant of explosion can effectively lessen the pressure difference between the inner eardrum and the outside world, thereby reducing injury effects the shock wave inflict upon the auditory apparatus.

5.2.2 Protection Through Landform

Terrains and landforms could both shelter or strengthen the effects of a blast wave. Generally speaking, when a person is hidden behind a terrain feature or landform, or inside a ditch or trench, injury effects of shock wave would be substantially reduced. In an on-site explosion evaluation experiment involving large equivalent explosive, two groups of five test sheep/goats each were placed at equal distances from the center of the blast. One group was situated in an open space and positioned in natural standing posture, while the other group was located in a trench 1.5 m deep and also positioned in natural standing posture. Animals in the open space group had a death rate of 100%, while no sheep/goat died in the trench. Thus, it can be seen that terrain feature or landform offer very significant advantages in protecting against shock wave.

Yet choosing the wrong terrain feature or landform would not only provide zero protection but may even worsen the injury effects. Estimating based on the Bowen curve, for a person in an explosion that occurred in a wide open space, threshold for 1% fatality when the person is standing straight is 190 kPa, but when the person is located inside a chamber environment where reflection surfaces exist, threshold for 1% fatality would drop to merely 100 kPa.

Infantry Personal Protective Equipment

Protective gear that defend infantry from shock waves usually comprise of components for three body parts, namely the auditory apparatus, head and chest. In terms of protective equipment for the auditory apparatus, the main function is to protect the auditory apparatus from being injured by shock wave. Stuffing the ears with earplugs, cotton balls or other foam materials can effectively mitigate the injury effects the shock wave has on the auditory apparatus, with injury effect lessened by as much as 90%. Although auditory apparatus protective devices such as earplugs offer relatively solid performance against shock wave, but at the same time they impair oral communication between users, thereby lessening the effectiveness of personnel. Therefore, new auditory apparatus protective equipment emphasize filtering out the effects of high-frequency sound waves generated from an explosion while maximally retaining the transmission of low-frequency sound waves used in verbal communication.

In terms of protecting the head and chest areas from shock wave, at present there is not any specialized protective equipment designed specifically for the head or chest areas, as current head and chest protective equipment are mainly intended for defense against bullets and fragments. The lack of shock wave protective equipment for the head and chest may be attributed to two reasons. On the one hand is general difficulty in defending against shock waves, and on the other hand, range of moderate blast injury threshold is usually within the range of 50% fragmentation fatality threshold,

which is because propagation range of shock wave from TNT explosive is usually much smaller than the injury and lethal range of fragments from an explosion. However, with progress in new types of munition, especially developments of thermobaric weaponry, injury threshold of blast wave has declined significantly while injury range has enlarged substantially. Furthermore, the number of personnel stations inside tunnels, fortifications, and other enclosed chamber spaces is becoming increasingly larger in modern warfare. In such enclosed environments, the occurrence rate of fragmentation injury is relatively low, while blast injuries are more common. Thus, defending against blast waves from new types of munition is crucial in infantry personal protective gear in modern warfare.

Armored Chamber

Tanks and armored vehicles constitute primary land battle platforms in modern combat, and protecting passengers inside these vehicles is another vital facet of soldier personal protection. Generally speaking, tanks are relatively enclosed and sealed off from the outside, which is why blast waves cannot easily enter the inside of tanks. After studying more than 50 cases of efficacy evaluation on anti-tank missile strikes against armored chambers, the author discovered that when an anti-tank missile does not penetrate the armored chamber, shock wave overpressure inside the chamber remains below 10 kPa. Thus, when there is not non-penetration wound inflicted on personnel inside chamber environment, the possibility of injury arising from shock wave overpressure is relatively low. However, powerful shock wave dynamic pressure may cause the armor to accelerate due to impact, and vibration acceleration could in turn cause human body to accelerate, resulting in acceleration injury. Injuries due to acceleration arising from impact and vibration are not inflicted only at the parts where the body make contact with solid object, but the force may also propagate through bones and solid tissues in the body, thereby injuring other body parts. Researches indicate that the bone system is the body part most sensitive to acceleration injury. Hirsch established an ankle and foot acceleration injury tolerance level and standard system for standing person, which states that the level of human tolerance to short bursts of impact is less than 3 m/s in peak speed changes (less than 10 ms in duration of acceleration to max speed). Vertebrae are slightly more tolerant, and the level of human tolerance to short bursts of impact is less than 4.5 m/s in peak speed changes (less than 20 ms in duration of acceleration to max speed).

Other than the bone system, the nervous system is also very susceptible to acceleration injury. Studies indicate that vibration acceleration can cause degeneration and necrosis of the peripheral nerves, and injuries such as brain hemorrhage and edema. The underlying injury mechanism may possibly be associated with the inertia arising from vibration acceleration. Of these, injury caused by rotational acceleration is more severe than damage arising from linear acceleration.

Powerful impact vibration may also injure or displace other internal organs, leading to various biological and chemical changes of different types and in various tissues. Particularly when impact vibration frequency causes resonance in human tissues and organs, injuries to internal organs and tissues would be even more severe. After studying efficacy evaluation research on anti-tank missile strikes against armored chambers, the author found out that when an anti-tank missile does not penetrate the armored chamber, even though shock wave overpressure inside the chamber remains very low (less than 10 kPa), relatively severe extensive endocardial hemorrhage was still discovered in some test animals.

Defensive Fortification

Various types of defensive fortification offer rather good protection from blast waves, for example the aforesaid trench provide pretty good protection from blast wave. Therefore, the trench is one of the simplest defensive fortification in this regard.

Other than trenches, various kinds of civil fortifications and permanent fortifications are also important defensive fortification to protect personnel from blast waves. Civil fortifications are mostly underground fortifications constructed in urban areas, and offer rather excellent protection from blast waves and other injury and lethal factors. However, if a civil fortification were to be struck with a sufficiently powerful force at the entrance or a vulnerable spot, casualty inside could be grave. That is because the injury and lethal effects of shock waves are more deadly inside an enclosed space, and at the same time, tunnel and other structural collapse could result in suffocation and other secondary injuries. The bombing of Chongqing and the "June 5th Great Tunnel Disaster" during the Second Sino-Japanese War both occurred because the entrances of the bomb shelters buckled, leading to poor ventilation and the death of thousands.

5.3 Principles for Treatment of Fragmentation Injuries

Explosive fragmentation injury is a kind of firearm injury, and generally speaking, firearm injuries are prone to infection, thus requiring debridement. Early and thorough debridement is the best method to protect against infection. However, since wound passage in fragmentation injury is usually uneven, "thorough" debridement may result in excessive removal of muscles and other soft tissues, leading to delayed wound healing. In recent years, some Chinese and

foreign scholars have advocating focusing on more comprehensive decompression drainage during the early stages of surgical treatment of modern firearm injuries, instead of simply stress on "thorough" debridement. Experiments and studies indicate that wound passage infection occurrence rates in "thorough debridement" group and "incision and drainage" group do not differ much, but wound passages in the "incision and drainage" group healed significantly faster than the other group.

Principles for debridement of primary wound: (1) Carry out debridement as soon as possible after injury, and try to complete debridement within the shortest possible timeframe before the onset of infection, and apply antibiotics as quickly as possible; (2) strict abide by sterile operation requirements. During wartime, surgical conditions may be poor, but adequate conditions must be created to strive to achieve sterile operation; (3) enlarge wound to sufficiently expose wound passage, which would be beneficial to exploring injury status of tissues deep inside, so as to minimize chances of missed or erroneous diagnosis; (4) endeavor to remove foreign objects like sand, fragments and cloth, cut away necrotic and damaged tissue, and properly deal with bone fragments, nerves, tendons, and vessels; (5) carry out debridement early but delay suture. Injury effects of firearms could cause injury to tissues far away from the ballistic path, and whether or not such injury would degenerate into necrosis is hard to tell early on. Therefore, in principle, the wound should stay open instead of performing suture. However, for wounds that are recent, relatively clean and shallow, primary wound closure may be considered after adequate debridement. In addition, primary suture should be administered to the head, face, reproductive organs, and joint capsule, and the same holds true for wounds to the chest and abdominal cavities.

Under the premise of applying proper debridement principles, it is also necessary to perform debridement at the right time. Germs inside the wound passage of a firearm wound would only turn into infection after a certain duration of multiplication (incubation period). The shorter the time between injury and debridement, the lower the chance of infection. In the majority of cases, a wound remains within the contamination stage 6–8 h after injury, with infection yet to form. Thus, it is general consensus that the window of 6–8 h after injury is the golden period for debridement. However, this timeframe is not absolute. Infection formation in wound is affected by a multitude of factors. In general, the length of the incubation period of germs is influenced by environment temperature and humidity. Germs multiply quickly and infection occurs earlier under higher temperature humidity, and in the opposite case, infection occurs slower. Moreover, the length of the incubation period is also dependent on the nature of the wound, type, quantity and toxicity of germs, as well as the local and bodywide immunity of the victim, and whether or not antibiotics are used.

If wound contamination is serious, and the victim's bodywide condition is poor, particularly if tissue hypoperfusion or local circulation disturbance exists, wound infection could take place as soon as 3–4 h after injury. If wound contamination is mild, and the victim's bodywide condition is sound, while local blood circulation is ideal, wound infection onset could delay to 12 h after injury or later. In cold and arid areas, where environment temperature is relatively low, and if antibiotics are applied, sometimes obvious infection would not occur even 12–24 h after injury, but operative indications for debridement still exist.

For those with clear wound infections, and wounds that have existed for more than 24 h, debridement should be avoided. At this juncture, debridement may possibly destroy formed granulation tissue barrier and promote the spread of infection. To deal with this kind of wound, the requirement is to remove clearly necrotic tissues and foreign objects, then keep the wounds open and continuously apply medication.

Injuries from high-speed small fragments are usually characterized by many wounds and wounded areas, small wounds and numerous penetrating wounds. To treat such injuries, abide by normal rules for treating firearm injuries on the one hand, and at the same time it is also important to pay attention to the characteristics of small fragmentation wounds themselves.

For small fragmentation injuries, surgical debridement is still a primary choice. However, debridement principles differ somewhat from traditional debridement procedures. Traditional debridement requires complete opening of wound passage to flush out and remove various kinds of foreign substances, then cut away deactivated or necrotic muscle tissue due to injury, and maintain open drainage for 3–5 days before delayed suture. The theoretical basis for treatment principle here is that transient cavity pulsation not only causes massive tears and contusions to wound tissues, leading to serious contamination inside wound passage, but also severely ruin micro circulation of surrounding tissues, resulting in tissue ischemic necrosis, which is why most tissues inside wound passage need to be removed. Main judgment technique for tissue necrosis is the "4C" method, as in color (dark red), consistence (soft and mushy), contractility (lack of contraction upon being squeezed) and capillary (no bleeding after incision). Another reason that supports large scale of tissue removal is the severity of infection of tissue inside the passage of firearm wound. Although projectile, fragments and visible bits of cloth are not difficult to remove, and contaminants such as dirt may be rinsed out through adequate flushing, but some smaller foreign objects basically invisible to the naked eye could be spread around in the deeper parts of wound passage, which could turn into focus

of infection. Cutting away active but severely contaminated tissue is certainly favorable to putting infection under control, but this kind of method, which was established a century ago when antibiotics were not common, has been increasingly questioned. Removal of excessive amount of muscle or wound edge will result in relatively large quantity of tissue loss, requiring transfer flap or skin graft to cover wound, and affecting functional recovery later on.

For the treatment of small fragmentation injury, Bowyer previously conducted a relatively detailed research. He struck the hind legs of pigs with 0.2 g steel rods at a speed of 500 m/s. The boundaries of deactivated tissues all reached close to 1 mm at different times, but there were not obvious clinical differences between the different times. There were no clear changes in the types and quantity of bacteria on skin surface after injury compared with those before injury. Bacteriological examinations of wound passages indicate that only two out of the 12 wound passage samples showed positive bacterial growth, of which, one was sampled 1 h after injury, with a small quantity of nonpathogenic *staphylococcus* and *streptococcus faecalis* having grown near the surface and deep inside the wound of the animal. The other one was sampled 6 h after injury, and a small quantity of nonpathogenic *staphylococci* with mixed growth was found on the wound surface of the animal. All anaerobic culture samples returned negative results. The aforementioned experiment results indicate that deactivated tissue region around passage of small fragmentation wound is rather restricted, and in general there is no need for surgical treatment. At the same time, infection probability of such wounds is also relatively low; therefore, most fragmentation wounds do not require surgical procedure if there is no infection as they can recover on their own. Thus, the majority of small fragmentation injuries in modern warfare are suited for conservative treatment.

5.4 Principles of Treatment for Blast Injuries

The treatment of blast injury hinges on nature of the injury. Explosive weapons are powerful can causes injuries through a variety of complicated factors. Therefore, features of blast injuries differ clearly from other types of injuries, chiefly including the ones listed below.

5.4.1 Unexpectedness of Blast Injuries and Difficulty in Organizing Rescue Effort

Blast injuries during wartime or those from terrorist attacks are characterized by sudden occurrence and unpredictability of the explosion. In addition, the destructive effect of the explosion and tremendous injury and lethal effects on the ground would causes severe damage to equipment and facilities. Thus, usually injuries and casualties are more serious than other types of injuries. Medical rescue resources may not be mobilized and react in time, adding to difficulties in timely medical treatment and on-site first-aid organization and command.

5.4.2 Numerous Injury Effects and Complicated Injury Conditions

The injury effects of blast injuries are usually the combination of two or more injury factors which mutually intensify or expand upon each other. Therefore, pathological and physiological disorders frequently compound and complicate composite injuries afflicting multiple body parts. There are many kinds of explosive weapons, and so injury methods are just as diverse, inflicting different degrees of injuries to different body parts. Not only do explosive weapons produce a large injury range, but they create injuries affecting multiple body parts, numerous organs, and various kinds of tissues, with intense and prolonged reactions throughout the body and locally.

5.4.3 Severe Injury Conditions, Multiple Complications, and High Death Rate

Some explosive weapons are quite powerful and effective for a long duration, which is why complications during early stage of injury can be very deadly. Victims of severe composite blast injuries often die on-site, and later deaths are frequently attributed to multiple organ failures and severe complications. Large injury area and multi-body part injuries mean a higher ratio of victims with injuries in numerous body parts. Following the rise in shock, hemorrhage, coma and other complications, and increase blast injuries, multi-body part injuries and burns, the ratio of severely injured victims also climb correspondingly.

5.4.4 Simultaneous Existence of Internal and External Injuries That Can Easily Lead to Missed or Erroneous Diagnosis

Blast injuries are often characterized by diversity of injury conditions and complicatedness in changes. In particular, burn-blast composite injuries or mechanical trauma-blast composite injuries are dangerous because burns on body surface caused by heat or tissue damage caused by mechanical forces (i.e., bone fracture, major hemorrhage, etc.) are readily observable, yet coexisting blast injuries to the body may in turn be overlooked, resulting in missed or erroneous diagnosis.

5.4.5 Severe Loss of Soft Tissue

When a pressure wave propagates in media of different densities, various effects such as spallation, implosion, acceleration, deceleration and pressure difference may occur, causing tears and blast injuries to tissues. Soft tissues are often

destroyed, exposing deep tissues beneath. In particular, suicide bombings in recent years have brought about new changes in blast injuries. Often times various kinds of foreign objects are added to the explosives, causing an increase in body parts injured, worsening the severity of damage to soft tissues, and leaving behind a greater amount of foreign objects in the bodies of victims.

5.4.6 Complicated Infections and Great Treatment Difficulties

Given the serious loss of soft tissue resulting from blast injury, the existence of foreign substances like dirt, fragments and hair around the wound passage and in deep tissues may worsen wound surface contamination, heighten rate of infection and speed up injury development. Immunity response is suppressed when various kinds of tissues are severely damaged, easily leading to infections and other complications.

5.4.7 High Rate of Destructive Limb Injuries, Severe Impact On and Difficulty in Repair of Limbs

Blast injuries often have destructive consequences, including widespread loss of soft tissues and bone tissues, damage to vessels and nerves deep beneath the surface and around the injured areas, and exposure of joints, bones and tendons, all of which could severely affect functionality and complicate surgery repair, with some patients requiring amputation. Some failures in limb salvage may be attributed to injuries existing in other organs coupled with life-threatening blood loss and sepsis.

5.4.8 Untimely Evacuation and Missing Best Opportunity for Treatment

Armed conflicts may produce a huge number of victims with complicated injury conditions, while medical treatments are hampered by restrictions in medical environment, instrument, human resource and various other factors. Delayed evacuation may result in missing best opportunity for treatment, leading to vast growth of germs on wound surface and increased rate of infection in victims. Necrosis or inflammation in the tissue of some reversible injuries may possibly occur, leading to worsened injury condition and tissue necrosis. Some victims may not receive first-aid resuscitation in time, resulting in death.

5.4.9 Treatment Difficulties and Dilemmas

The greatest challenge in treating blast injury is how to deal with difficulties and dilemmas associated with the different injury factors. For instance, how to balance between dilemmas, such as rapid transfusion to treat burns versus careful transfusion necessitated in treating blast lung injuries, is vital to properly dealing with burn-blast composite injuries.

Based on the features of blast injuries described above, first and foremost abide by the basic principles of combat injury treatment when treating blast injuries, as in stopping blood loss, bandaging, fixation, pain relief and prevention of suffocation. Hemostasis mainly involves stopping bleeding in obvious active wound through compression and bandaging. For arterial bleeding of the extremities, use tourniquet for hemostasis and mark the patient properly for prioritized evacuation. For those with obvious wounds, bandage wounds in a prompt manner. Chest wounds in particular require bandaging with thick dressing. In the case of tension pneumothorax, pleural puncture and aspiration is needed. For bone fractures and joint injuries, utilize wood boards, branches or other materials on-site to perform simple stabilization and fixation. For victims suffering from intense pain, provide effective pain relief medication. At the same time, pay attention to prevent traumatic asphyxia and strengthen respiration function.

For blast injuries to different parts of the body, symptomatic treatment should be performed correspondingly. The key to dealing with blast injuries to the ears is to prevent infection and promote eardrum healing. Blast wave may directly impact the eyes and create eye contusion, but it is more common for the eyes to be afflicted with indirect injuries when substances such as dirt and sand kicked up on-site at the location of explosion, or particles from the detonation, act on the eyes. Lungs are the central target organs subjected to the effects of shock wave, and main pathologies include pulmonary hemorrhage, pulmonary edema, ruptured lungs and pulmonary bulla. Lung collapse and emphysema are also quite common. Victims of minor blast lung injuries may recover in a few days after adequate rests and symptomatic treatment. Victims of relatively more serious blast lung injuries, or when compounded by other injuries, may require proactive comprehensive treatment. When subjected to the direct action of a powerful shock wave, a person may also be afflicted with serious heart injuries. Main pathologies include heart wall bleeding, and rupture or necrosis of myocardial fibers. When the heart is injured, usually the lungs would already be suffering even worse damage, which is why treatment of heart and lung injuries should focus on protecting cardiac and pulmonary functions, such as the administration of cardiac agents, diuretics, dehydrants, and so on.

Among blast injuries to the abdomen caused by shock wave, injuries to the liver and spleen are the most common. During underwater explosion, it is easier for the intestines and other hollow organs filled with gas to sustain injury. For those with paralytic ileus, before restoration of effective peristalsis, it is necessary to continuously apply gastrointestinal decompression. For those that did not sustain any perforation, if symptoms quickly subside after conservative treatment, such patient should still be further observed for another week to ascertain that there is no delayed perfora-

tion. Ruptured hollow organs are liable for spilling contents into the abdominal cavity. In such cases, if there is peritoneal irritation and subsequent shock, endeavor to elevate systolic pressure to 90 mmHg and pulse pressure higher than 20 mmHg before undertaking surgery. Due to the large number of organs in the abdominal cavity, surgical exploration must be carried out in a detailed and thorough manner so as to avoid missing any crucial discoveries and problems.

Craniocerebral blast injury has risen as a focal point in recent years, and treatment in the early phase is generally identical to that for craniocerebral trauma.

The limbs and spine are often injured because of strikes from secondary projectiles or collisions against hard objects when person is displaced. Treatment for these injuries are identical to treatment for normal limb and spine injuries.

5.5 Principles of Treatment for Combined Injuries Caused by Explosive Weapons

Injury from explosive weapon is a process comprised of overall effects of multiple factors, among which, fragment-shock wave composite injury is the most common type. Its treatment is predominantly based on treatment principles of fragmentation injury and treatment principles of blast injury.

Injuries caused by new types of modern, high-energy explosive weapons are even more complicated. For example, new types of thermobaric weapons not only result in fragment-shock wave composite injuries but usually coupled with damage from thermal radiation and lack of oxygen. Not only would thermal radiation burn body surface, but it may also lead to inhalation injury. Under certain special conditions, oxygen deficit may worsen lung injuries. Therefore, the treatments of composite injuries caused by new types of high-energy explosive weapons require actions to ensure unobstructed airway and protection of cardiac and pulmonary functions on top of other, conventional on-site first-aid procedures.

In the event of a nuclear explosion, radiation composite injury is another important type of composite injury. The treatment of radiation-blast composite injury requires more thorough debridement procedure on top of local treatment, and usually the scope of treatment is larger than that for conventional combat injuries. In terms of full body treatment, pay attention to the following points.

1. Protect against shock. The probability of shock may increase in radiation-blast composite injury, that's why it would be ideal to take preventative and curative measures in a timely manner.
2. Protect against infection. The body is vulnerable to more infection pathways in case of radiation-blast composite injury. Aside from wounds, probability of endogenous infection in areas such as the stomach and intestines also increase significantly. Therefore, overall infection rate rises, and effective anti-infection measures should be adopted as early as possible.
3. Protect against bleeding. Radiation-blast composite injury is more prone to bleeding than simple bleeding; thus, employ hemostatic drugs and hemostatic measures in a timely fashion to prevent the onset of secondary bleeding.
4. Stimulate hematopoiesis. Radiation-blast composite injury may hinder hematopoietic function. When conditions permit, consider blood transfusion and bone marrow infusion to produce positive effects in stimulating regeneration of hematopoietic tissues.

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Land Mine Blast Injury

Bingcang Li

Land mine is a type of explosive weaponry buried just beneath and deployed on the ground surface. Land mines may produce fragments and shock waves that create various types of injuries to different parts of the body.

The development of land mine is closely associated with development of explosive. China invented explosive, and that is why land mine was also first produced and put to use in China. Literatures indicate that a device made from iron, loaded with explosive, armed with fuse and known as "sky-shaking thunder" appeared as early as sometime between the twelfth century and thirteenth century. Other than being launched using catapults, they could also be suspended from ropes or deployed on the ground for destructive and lethal effects. The Song Dynasty military used this device to effectively defend themselves against the sieging Jin army in 1130. In 1580, renowned Ming Dynasty general Qi Jiguang (1528–1588) had a type of land mine called "fire-producing wheel of steel" produced when he was stationed at Jizhou. When an enemy steps on the contraption, the steel wheel would spin and produce spark via friction to ignite and detonate the mine.

The form, production and features of more than ten types of land mines were documented in the Ming Dynasty military book *Wubei Zhi* or *Treatise on Armament Technology*, including schematics of the devices. Land mines in those days were primarily made out of stone, ceramic or iron. They would be buried underground, and explode to lethal effects when stepped upon, tripped, pulled, ignited or detonated by other means. Early land mines were mostly circular or square, and made from stone. Deep hole would be drilled in the center, and the charge would be loaded and packed inside. A little crevice would be reserved for a thin bamboo pipe or reed carrying the fuse. Some sort of mud or clay would be used to seal the hole. Land mines would be buried where enemies travel, to be ignited and detonated when targets approached the device. This kind of stone land mine is also called "explosive rock mine." They were broadly used in warfare because production was simple and required materials were readily available. However, they neither a big charge nor produced powerful explosions, so further improvements were made. The later forms of land mines, in particular detonation mechanisms, were continuously improved, enlarging the effective injuring and lethal area of the weapon.

Zhai Rushuo, a jinshi scholar of 1601 who held the post of tixue qianshi in Huguang, recorded in the *Bing Lue Zuan Wen*: "Zeng Xian, the deputy minister of defense in 1546 or the 25th year of the reign of Jiajing Emperor of Ming Dynasty, produced land mine by digging a trench about 10 ft long, inside of which charge would be placed, to be covered by rocks and then sand. The ground surface would be smoothed evenly, fuse would be buried underneath, ignition mechanism would be placed on the ground surface, and upon detonation, rocks would be launched and enemies frightened." *Exploitation of the Works of Nature* by scientist Song Yingxing (1594–c. 1666) and *Wu Bei Zhi* (Treatise on Armament Technology) by militarist Mao Yuanyi (1587–c. 1644) both recorded about land mines and their production methods, including detailed land mine schematics and their shape upon explosion. In ancient times, land mines were mostly circular or square, and made from stone. Deep hole would be drilled in the center, and the charge would be loaded and packed inside. A little crevice would be reserved for a thin bamboo pipe or reed carrying the fuse. Some sort of mud or clay would be used to seal the hole. Land mines would be buried where enemies travel, to be ignited and detonated when targets approached the device. Of course, these early land mines were easy to make because materials were readily available, but they were not very powerful because they held only small charges.

After the middle of the nineteenth century, following the advents of various kinds of powerful explosives and advanced detonation technologies, land mines became more formulated

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and diversified, laying the groundwork for modern land mines. The first iteration of the modern antipersonnel land mine was produced by Russia in 1903, and antitank land mine was researched and developed by Germany in 1918. During the Russo-Japanese War fought between 1904 and 1905, the Russian military deployed antipersonnel land mines during a battle at Lvshunkou on Chinese territory. During World War I between 1914 and 1918, since tanks played a main role on the battlefield, antitank land mines were consequently developed and deployed in actual combat. After World War I, countries began to prioritize the research and manufacturing of antitank land mines. By 1935, the Soviet Union, the UK, the USA and France all researched and produced a wide array of antitank land mines capable to defeating armored vehicles. During World War II between 1939 and 1945, the types, power and quality of land mines all reached new heights, including the advent of scatterable land mines. However, since land mines in this period were large, heavy, made with iron casing and loaded with a big charge, they were easy to detect and remove while difficult to carry and transport. Thereafter, plastic land mines that were harder to detect and lighter to carry around were researched and developed. After the 1960s, the types, power and quality of land mines once again increased. Mines were smaller, lighter, more powerful and could be delivered from a long distance away or in the air by way of rocket, artillery, aircraft or other dispensing methods. These advancements enabled quick deployment of land mines over an extensive area, a revolutionary progress compared with previous land mine deployment techniques. After the 1980s, following advancements in electronic technology, sensor technology, information processing technology, charge loading technique and mine deployment equipment, old mines were replaced by new iterations in quick succession, developed toward more intelligence-driven orientation such as artificial intelligence capable of better detection, identification, tracking and positioning, as well as other functions like more accurate detonation timing, antielectronic inference and antidetection capability. Smart land mines not only produce better results in attacking their targets, but can also integrate into battlefield information system, thereby considerably augmenting the combative efficacy of the mine field, and transforming land mine from a weapon deployed purely for defensive purposes into one that possesses both offensive and defensive potentials. In addition, the effective area of mine is also expanding into the air. Without a doubt, land mine warfare will become even more diverse in format in the future.

1 Section One: Types of Land Mines

There are hundreds of types of land mines, and may be divided into the following few categories according to intended target.

1.1 Antipersonnel Land Mine

These mines are designed to kill and injure people, and come in a myriad of forms from columns and squares to rectangles and other irregular shapes. They could be laid in mine fields or mine clusters to kill and injure nonmechanized forces, or to threaten the enemy mentally to slow down the movement of infantry. The burst height of antipersonnel land mines usually range between 0.5 and 2 m, and often times realized by way of delayed charge, standing rope and electronic timer. Modern antipersonnel land mines are sometimes required to self-destruct or render themselves inert after a certain period. In other words, after a mine has been laid and activated, if it is not triggered by a target after a specific period, it would self-destruct by exploding on its own, or render itself inert through the intended failure of some key component(s). Antipersonnel land mines usually consist of casing, charge and fuse. Casing is mainly made from metal, plastic or other nonmetallic materials, and plays a huge role in the detectability of the mine. Current standard requires that each land mine be equipped with magnetic signal equal to 8 g of iron for ease of detection and demining. The charge of the mine is directly associated with its power, and usually high-energy explosives like TNT, RDX or plastic explosive is utilized. Mine fuse is largely divided into two categories; one is mechanical fuse such as pressure trigger (detonated by weight), tripwire (detonated by action) or pressure-release mechanism (detonated by weight and then removal of weight).

1. According to injuring and lethal factors, antipersonnel land mines may be classified as follows: (1) explosive land mine, usually armed with pressure trigger to injure or kill person that stepped on the mine using blast wave; or (2) fragmentation land mine, usually injures or kills people within its effective radius using preformed or semi-preformed fragments and blast wave, and are frequently armed with fuse like tripwire or pulled fuse, pressure trigger or pressure-release mechanism and electronic firing mechanism.
2. According to control method, antipersonnel land mines may be classified as follows: (1) commandable land mine or (2) uncommandable land mine.
3. According to dispersal method, antipersonnel land mines may be classified as follows: (1) scatterable land mine or (2) buriable land mine.

1.2 Antitank Land Mine

These mines are designed to destroy tanks, armored vehicles, artillery and other weapons and vehicles, or to render them incapable of movement or combat.

1. According to targeted area of attack, antitank land mines may be classified as follows: (1) continuous track land mine, (2) vehicle bottom land mine, (3) full-width (dual-use) land mine, (4) side armor land mine or (5) top armor land mine.
2. According to method of action, antitank land mines may be classified as: (1) Contact land mine, which may be further subdivided into pressure-trigger land mine or tilt-road land mine; (2) noncontact land mine, which may be further subdivided into magnetic fuse land mine, vibration fuse land mine, optoelectronic fuse land mine, sonar fuse land mine or composite fuse land mine; (3) active attack land mine or (4) smart land mine.
3. According to dispersal method, antitank land mines may be classified as (1) scatterable land mine or (2) buriable land mine.

1.3 Antiair Land Mine

Based on the type of aerial target, antiair land mines may be classified as follows: (1) antihelicopter land mine that possesses active detection, tracking, identification and positioning functions, and can attack helicopters flying at low altitude; (2) antiparatrooper land mine, which are usually deployed on grounds that need to deny access by enemy parachutists, and designed to injure or kill descending paratroopers by exploding toward the air after detonation, or launching fragments into the air after exploding on the ground; (3) floating mine, which are suspended in mid-air by way of mechanisms such as balloon to destroy aircraft flying at low altitude or descending paratroopers.

1.4 Special Purpose Land Mine

Based on the designed purpose, special purpose land mine may be classified as follows: (1) signal land mine, usually laid around an area that requires warning, and they emit light or sound alarm signals upon detonation; (2) illumination land mine, which lights up using illuminate or launches illumination flair into the air upon detonation for illumination purpose; (3) smoke land mine, which produces smoke screen using smoking agent upon detonation to confuse the enemy or cover the movement of friendlies and (4) incendiary land mine, which burns profusely using incendiary agent upon detonation to burn down target or create a fiery obstruction to slow down any enemy movement.

2 Section Two: Land Mine Contamination

The vast quantity of land mines deployed is a result of their ease of production, cheap production costs and simplicity in dispersal. Most land mines remain active and lethal 50 years or even a century after deployment.

2.1 Distribution of Remaining Land Mines

There are roughly 110 million land mines scattered in 62 countries around the globe, with more than half distributed in the Middle East and North Africa, followed by Southeast Asia and sub-Saharan Africa, as well as Central and Eastern Europe. Countries with the most laid land mines in the world are in order: (1) Egypt, 23 million land mines; (2) Iran, 16 million land mines; (3) Angola, 10–20 million land mines; (4) Afghanistan, ten million land mines; (5) Iraq, ten million land mines; (6) Cambodia, 8–10 million land mines; (7) Kuwait, five million land mines; (8) Bosnia-Herzegovina, three million land mines; (9) Mozambique, three million land mines and (10) Somalia, one million land mines. The number of active land mines in Egypt account for about one-fifth of world total. The chief reason behind this situation can be traced to World War II, when Egypt was one of the main sites of conflict in North Africa. The British military and Afrika Korps of Nazi Germany were the main belligerents in this theater of war. In addition, during the Third Arab–Israeli War and the Fourth Arab–Israeli War, the Egyptian military engaged in fierce battles on the Sinai Peninsula, which littered the area with a massive number of land mines. Bordering the western edge of China, Afghanistan has been torn by warfare and chaos for more than four decades, resulting in more than ten million land mines scattered across this country that is just over 650,000 km^2 in size. Cambodia has a territory of over 180,000 km^2, which is home to a population of some 15 million but also the world's six highest number of land mines that remain from the Anti-French Resistance War, Cambodian Civil War and Cambodian–Vietnamese War. It is unknown how many mines have been left on the border between China and Vietnam, but there are more than 560 mine fields totaling over 4 million km^2 resulting from the border skirmish. Between 1992 and 1999, the Chinese military extracted more than 2.8 million mines alone in the border areas, and destroyed over 400 tons of other explosives [1].

2.2 Harms of Remaining Land Mines

Among the remnants of war, land mines are the hardest to remove. They threaten human societies in numerous ways, principally manifested in the following aspects.

2.2.1 Bodily Injury

Land mines are indiscriminate, so they not only kill and injure soldiers during wartime, but they also cause tremendous casualties and injuries among civilians long after a conflict has ended. It is impossible to obtain an exact tally as to how many civilians around the world are killed or injured by land mines each year. Existing casualty and injury data are mostly statistics from different hospitals, but those that never arrived at the hospital would always remain unknown. Reports indicate that a person somewhere on this planet is killed or injured by a land mine every 20 min, and each year there are about 15,000–28,000 land mine victims, with women and children being particularly at risk. According to statistics by the International Committee of the Red Cross, women account for about 10–15% of land mine victims. This ratio is associated with their roles in societies, with the proportion even higher in societies where women play a larger role. Usually women suffer worse consequences than men after being maimed by a land mine explosion. They are often subjected to discrimination, and abandoned, or they would lose their social status.

The International Campaign to Ban Landmines (ICBL) was jointly founded by the six nongovernmental organizations Handicap International, Human Rights Watch, Medico International, Mine Advisory Group, Physicians for Human Rights and Vietnam Veterans of America Foundation in October 1992. The group is dedicated to promoting the thoroughly implementation of the *Mine Ban Treaty*, and regularly publishes the periodical *Landmine Monitor*. In the 2006 edition of the *Landmine Monitor*, ICBL pointed out that a total of 11,000 land mines were tripped in Colombia during the year, 875 in Cambodia, and 363 in both Iraq and Palestine.

There are about 2 million tons of land mines and other explosives remaining in Laos. Between 1964 and 2008, a total of 50,136 triggered land mines in Laos, and among them there were 20,726 survivors (41.34%). Between January 1997 and September 2002, a total of 6114 persons were harmed by land mines in Afghanistan, of which there were ten times more male victims than females. In terms of age, 15 to 19-year-olds accounted for 14%, 10 to 14-year-olds accounted for 29%, and 5 to 9-year-olds accounted for 13%, meaning that 56% of land mine victims were youngsters and children below the age of 19. Statistics indicate that there are some 1.5 million land mine victims in Afghanistan, and on average a hundred persons were killed by land mines each month. Warfare has continued on and off in the past four decades in Iraq, which is another country tormented by land mines, particularly the Kurdistan Region in northern Iraq that shares border with Iran. Between July 1998 and July 2007, emergency clinics in the Kurdistan Region capital of Erbil alone received 285 land mine victims. These victims had an average age of 26.5 ± 13.2 years old (ranging from 6 to 71 years old), and 50% of victims fell between the range of 19–35 years old. Males made up 95.1% of total, 96.8% were civilians, and amputation rate was 72% (58.6% lower limbs, 13.3% upper limbs). According to statistics, neighboring Iran is yet another state plagued by land mines. Between 1988 and 2005, provinces in the western part of Iran, namely East Azerbaijan, West Azerbaijan, Kermanshah, Khuzestan, Ilam and Kurdistan, had 3708 land mine victims, including 252 females or 6.8% of total.

2.2.2 Harms to Animals

The number of wild animals and livestock injured by land mines, and their types of injuries, lack official statistics, but there have been news reports about wild elephants being killed or wounded by land mines. It would not be far-fetched to assume that other relatively large wild animals have been harmed by land mines as well. Land mines might threaten the natural food chain of wild animals, and also threaten ecological environment, while harming livestock and thus weighing on farmers and herders economically.

2.2.3 Hindrance to Agriculture

Land mines left on arable lands not only pose a serious health threat but also economic threat to farmers as such fields cannot be safely cultivated, bringing detriments to both the income of farmers and development of agriculture. Reports indicate that 45% of arable land in Afghanistan have been rendered unusable due to land mines, exacerbating the already dire situations among Afghan farmers.

2.2.4 Obstruction to Transportation

During wartime, land mines were intentionally placed on roads, as well as railroads, bridges and airports, to impede enemy movement and attack. Not only do land mines in these areas inhibit traffic, but they also threaten anyone that traverse in such zones, further restricting local economic progress.

2.2.5 Increase of Social Burden

The existence of land mines add to social burdens in two main aspects. First of all, people maimed by land mines intensify social burden. Although the actual costs associated differ between economies, land mines are responsible for massive waste of human resource and fiscal expenditure. Second of all is the costs related to sweeping land mine. According to statistics compiled by the UN, a land mine produced at the cost of USD 10 is extracted at a cost of

around USD 300–1000. In 2008, land mine sweeping expenditures of 33 countries exceeded USD 400 million. The land mine removal cost in Cambodia alone may require several billion US dollars [2]. Current progress of demining is about 100,000 devices per year, and at this rate, we would have to wait 3100 years before the last land mine is extracted. Unfortunately, another two to five million new land mines are laid each year.

3 Section Three: Features of Land Mine Explosions

3.1 High Death Rate Before Arrival at Hospital

Fatality rate of land mine before hospital arrival could reach as high as 75%, while death rate in hospital is merely 2.1%. Eighty percent of casualties occur at the site of land mine explosion, and 9% occur during the process of evacuation. Based on evacuation conditions and different regional characteristics, it would take anywhere from 5 to 6 h to 9 h for evacuation to hospital after injury.

3.2 High Rate of Shock

Massive bleeding and tremendous pain are two chief reasons why shock is common in victims injured by land mine explosion. Rate of shock as stated in past reports vary drastically from 35% to 95.4% due to incomplete statistics and different injury conditions.

3.3 High Rate of Limb Injury

The body part injured by land mine explosion is closely linked to the type of mine trigger, and body posture when the mine was triggered. In terms of antipersonnel land mine, limbs have the highest chance of injury. When victims of such a land mine explosion arrive at the hospital, one-third would have lost a limb already, of which, the upper limbs of children are more easily injured, because often children would mistake a land mine for a toy and interact with the device using their hands.

3.4 Mop-Like Foot and Calf Injury

When a lower limb triggers a land mine, the foot is often severely injured or lost, edges of the wound by the bottom end of the calf would be covered in burnt, blackened and hardened skin, remaining muscles and tendons hang loose like bundles of yarns of a mop. This is the mop-like calf injury typically associated with land mine explosions. Such muscles and tendons would retract, shorten and become brittle. Lacerated nerves and vessels may retract a length of 5 cm or more, and nerves may enlarge by 0.2–0.3 cm due to tissue edema. The remaining long bones are often pointy with slanted sides.

3.5 Severe Wound Contamination and High Rate of Infection

Virtually every land mine explosion wound is chock-full of sand, stone, mud and tatters, which is why the infection rate of land mine wounds is far higher than that of gunshot wounds (11%). The infection rate of land mine injury amputations is 27.7%, and the infection rate for injuries without amputation is 51.2%, mostly involving anaerobic infections, but other issues such as gas gangrene, blood poisoning and posttraumatic osteomyelitis are also frequent.

3.6 High Rate of Amputation

An Iranian scholar reported that between 1988 and 2005, provinces in the western part of Iran, namely, East Azerbaijan, West Azerbaijan, Kermanshah, Khuzestan, Ilam and Kurdistan (all these areas share borders with Iraq and Turkey) received a total of 3713 victims of land mine explosions. Of which, 1499 had amputations, a rate of 40.37%. Below-the-knee amputation is most common. Victims averaged an age of 23 years old, and 92% of victims were male. Compared with adults, amputation rate in children is even higher, up to 82.6%.

3.7 Neuroma After Amputation

Neuroma is a tumor-like structure at the nerve stump in the scar area after amputation. Neuroma is very sensitive to stimuli, and even the lightest of touch could generate immense pain. It is hard to distinguish painful neuroma from phantom limb pain. Clinically speaking, it is difficult to find specificity in pain after lower limb amputation, but MRI examinations are particularly helpful in early phase diagnosis. Traditional radiography can help determine the bony origin of pain such as elevated bone crest, heterotopic ossification and osteomyelitis. Seventy-five cases of neuroma after lower extremity amputation were treated between 2000 and 2006. It takes anywhere from 1 month to 12 months before the onset of painful neuroma after amputation. On average, it takes 9.6 months for painful neuroma symptoms to appear after starting the use of artificial limb, and it takes

an average of 7.8 months between the appearance of painful neuroma symptoms and surgical excision. After an average of 2.8 years in follow-up period, all patients reported to no longer suffer from such pains. If neuroma is less than 1 cm in diameter, it is difficult for radiography to detect.

3.8 High Rate of Multiple Injuries Affecting Multiple Body Parts

Land mines injure with a combination of shock wave, fragments and secondary projectiles, which is why victims are often afflicted with multiple injuries to multiple anatomical body parts, or multiple injuries to a single body part. Of which, head injuries caused by land mines are most dangerous, with fatality rate ranging from 23% to 92%. Land mine fragments can inflict both perforating wound and penetrating wound to the head, as well as depressed fracture of skull, epidural hematoma and subarachnoid hemorrhage. The eyeball injury occurrence rate is 21.7%, usually resulting in blindness. If the spine is injured during a land mine explosion, burst fractures of vertebral bodies have a high chance of occurrence, pressing on the spine or causing severe injury to the spine.

3.9 Serious Psychological Harm

Land mine victims often develop severe mental disorders because of their handicap or after being abandoned by society. Not only are they prone to anxiety, depression and other neuropsychiatric symptoms, but they also tend to abuse drugs and even have suicidal tendencies.

4 Section Four: Injury Factors of Land Mine Explosions

As a small type of explosive weapon, the main injury mechanisms of land mines are shock wave, fragments and heat. Since land mines are designed to attack different targets, they also vary in quantity of charge, size and number of fragments, and other additional fillers. In other words, different mines produce different shock wave overpressure, fragment velocity, heat and other main injury and lethal factors. With regard to antipersonnel land mine, usually the charge would not weigh more than 200 g, and often times in the proximity of 50 g. Such mines are mostly built from metal or plastic casing, and loaded with or without preformed fragments. Take for example the type-66 antipersonnel land mine of the Chinese military (formerly the M18A1 Claymore is a directional antipersonnel mine produced for the US military) has a plastic shell, but the top is loaded with 700 steel pellets weighing in at a total of 650 g, capable of injuring, killing and damaging living targets and unarmored vehicles within a distance of 50–100 m.

4.1 Injury from Shock Wave

As most people know, a blast wave propagates faster in medium with higher density medium than in medium with lower density. The density of water is 1 kg/L, while the density of air is 1.295 g/L, meaning that the former is roughly 772 times denser than the latter. Therefore, blast wave propagates much faster in water than in the air, and also propagates further. A lower-limb land mine explosion injury experiment involved placing test rabbits on the ground, in water at mid-thigh depth, and in water at xiphoid depth. When 600 mg of RDX charges were detonated to injure the feet of the rabbits, peak pressure at a distance of 8 cm from the source of explosion varied in the different environments, with an average of 0.76 MPa on the ground, 62.62 MPa at mid-thigh depth and 72.67 MPa at xiphoid depth. Correspondingly, greater peak pressure yielded higher tibia and femur fracture occurrence rates, as well as a larger scope of sciatic nerve injury. It is thus easy to understand why, when a shock wave acts on the human body, relative displacement would occur due to different propagation speed of shock wave been bone tissues with higher density and muscle tissues with lower density, leading to tissue contusion or tissue separation. For example, if a land mine was triggered by a lower limb, there would be bone loss below the calf, while soft tissues like skin and muscle may remain, creating the mop-like calf injury typical of land mine explosions.

4.2 Fragmentation Injury

Based on stats compiled from 1500 cases of land mine explosion injury, occurrence rate of land mine fragmentation injury is as high as 88.3% (1324 cases). The degree of injury caused depends on the shape and velocity of the fragment. When a triangular or square fragment hits the body, its velocity decelerates rapidly due to a high rate of energy transfer, thereby creating a passage with large entry wound and small exit wound, or an exit-less penetrating wound. The entry wounds created by these fragments are usually irregular in shape, and wound passages are shallow and wide like an upside-down trumpet. Spherical fragments have smooth surfaces and low drag in movement, so they decelerate at a slower pace and penetrate deeper into tissue but transfer less energy. A spherical fragment usually creates a hole-like entry wound with even and smooth edges, and diameter slightly bigger than that of the projectile. When a spherical fragment flies at speed exceeding 1000 m/s, usually the diameter of the

entry wound would also be quite large. In addition, smooth spherical fragments are prone to change trajectory upon encountering tissues of different densities inside the body, creating curved and complicated wound passages that may possibly injure multiple organs. The energy transfer rate and wound passage of cylindrical fragments are midway between triangular fragments and spherical fragments. The general consensus is that fragments have to fly at a velocity of at least 100 m/s to injure or kill, and a velocity less than 50 m/s only creates skin contusion in most cases. When fragments move at a velocity above 200 m/s, all sorts of typical injuries may be inflicted. When fragments fly at a velocity above 340 m/s, the formation of temporary cavity may possibly create larger scopes of serious injuries.

4.3 Heat Injury

Heat such as flame, boiling water, hot oil, heated metal and high-temperature gas could injure human tissues and organs, as evident in daily life. Based on animal testing studies, 90 °C/5 s heat injury conditions could cause I^0 burns on the skin of common rabbits. Ninety degree Celsius for 8–20 s heat injury conditions could cause superficial II^0 burns on the skin of common rabbits, and 90 °C/22–30 s heat injury conditions could cause deep partial-thickness II^0 burns on the skin of the rabbits, while 95 °C/25–50 s heat injury conditions would cause III^0 burns on the skin of the rabbits. Thus, it may be seen that heat injury extent is not only associated with temperature but also how long a body part has been subjected to such temperature. Previous literature states that the explosion of a type-72 antipersonnel land mine, which weighs a total of 125 g, generates temperature upward of 2737 °C, sufficient to burn and blacken the skin of people in vicinity, and turn their muscles and tendons brittle.

It should be noted that since land mines are a type of explosive weapon, the injuries resulting from its explosion would not be the outcome of a single factor, but rather a combination of the numerous factors explained above. However, compared with larger explosive weaponry, land mines create shock wave overpressures that are not as powerful, do not produce as many fragments that weigh lighter and do not generate temperatures as high that cover a much smaller distance. Therefore, comparatively speaking land mines do not cause severe overall injuries, as they mostly inflict serious injury to specific parts of the body. Of course, it should be pointed out that since multiple factors are involved in a land mine injury, it is likely that a composite injury would result. Fragments have a high chance of injuring multiple body parts and organs, creating multiple injuries. Combined injuries and multiple injuries are much more difficult to treat and recover from than simple injuries. Besides, when shock waves and fragments act on a body at the same time, remote injury effects may possibly be produced (as in injuring organs and tissues far away from the part that triggered the land mine). Remote injury effects from weapons may be hard to detect, so it is necessary for medical care personnel to pay extra attention.

When a blast wave tears apart the clothing of the victim, it also kicks up sand and rocks, which is why the wounds from a land mine explosion are often seriously contaminated by these foreign substances. A blast wave could also produce a sort of "throwing effect" by tossing and displacing a victim, and causing him or her to collide against the ground or other hard objects.

5 Section Five: Treatment of Injuries from Explosions of Land Mines

5.1 Take Active Steps to Protect Against Shock

It is suggested to carry out blood transfusion and fluid transfusion quickly, and restore effective circulation as soon as possible. Whole blood is the most ideal colloidal resuscitation fluid, but if that is not available, dextran or plasma surrogate will suffice.

5.2 Carry Out Debridement Early and Delay Suture

This primary wound surgical treatment principle is deduced from the valuable experiences of past wars. For land mine explosion injuries, which usually result in extensively contaminated wound passages, this primary wound surgical treatment principle is even more crucial. It would be best to perform debridement 6–8 hrs after injury, but before the onset of infection. However, for victims with shock or close to death, debridement should only be undertaken after shock has been mitigated and his or her overall conditions stabilized. Prior to debridement, use a considerable volume of normal saline to flush wound passages, then utilize a mixture of hydrogen peroxide and benzalkonium bromide at a ratio of 1:1000 for disinfection. During debridement, gradually clean out all foreign substances and projectile fragments, then cut out necrotic or deactivated tissue and completely stop bleeding. It is acceptable to slightly enlarge the wound so as not to leave any dead space remaining. The limb(s) injured by the explosion of a land mine would swell very obviously and quickly. For injured limbs with pale white skin, low temperature, severe swelling and hardened texture, dissect deep fascia to decompress and to alleviate or prevent the

onset of fascial space compartment syndrome. Keep the wound open after debridement, ensure adequate and unobstructed drainage and refrain from stitching up the primary wound.

5.3 Change of Medication for Wound

Since debrided land mine explosion wound should stay open, usually they emit considerable volume of fluid. Refrain from bandaging the wound at this juncture; instead, just cover the wound surface with two to three layers of gauze and moisten the cover with antibiotic solution routinely. Flush with hydrogen peroxide for wounds suspected of gas gangrene. This method helps prevent gas gangrene and enables localized application of antibiotics, while at the same time minimizes soaking in seeped fluid, reduces pain of patient and facilitates care.

5.4 Treatment of Bone Fracture

When carrying out debridement, remove any dissociated small shards of bone, but keep larger dissociated bone pieces in place to avoid loss of bone and to use them as support for bone regeneration. Any bone pieces connected to soft tissue or periosteum should be preserved in place as much as possible. In principle, do not perform internal fixation for firearm-related bone fracture. In the past, emphasis is placed on external fixation after reduction of fracture, but nowadays many scholars opine that if infection in injured extremity is not too bad, internal fixation may be undertaken for firearm-related bone fracture upon the premise of thorough debridement and effective protection against infection, since this method is favorable to the stability of the ends of fractures and healing of fractures.

5.5 Dealing with Serious Loss of Soft Tissues

For victims suffering from serious loss of soft tissues, make repairs using vascularized muscle flap transposition 5 days after thorough debridement, but only if wound surface is clean, granulation tissues are fresh, wound edges are free from redness and swelling and there is no sensation of pressing pain. This method is not only beneficial to correction of limb deformity but also creates favorable conditions for rebuilding limb functionality later on, while also bolstering defense against infection. It is common for land mine explosion victims to undergo amputation. If soft tissues of the amputated limb are relatively healthy, it may also be utilized for repairing soft tissue loss.

5.6 Amputation

Amputation brings about immense emotional suffering upon victims, and it may even be considered brutal, but is oftentimes the only measure that can save life. For victims in persistently poor conditions even after multiple injuries, sometimes amputation is an unavoidable cost to save his or her life, and in some cases multiple amputations are needed. Open amputation is a common amputation method, but closed amputation may also be performed in certain situations.

Bone fractures near the site of injury should not be removed during amputation without careful consideration. Instead, carry out an appropriate form of fixation. Living tissue and muscle further away from the level of amputation should also be preserved as much as possible, and even if these tissues are irregularly shaped, do not abandon them at will because they may be used as flaps to close residual wounds, repair tissue defects, extend the length of the amputated limb and promote tissue recovery.

5.7 Rehabilitation After Amputation

Proper recuperation therapy offers tremendous benefits for both the daily life and psychological health of patients. However, according to statistics by the World Health Organization, only 5% of land mine injury amputation patients receive adequate recuperation therapy, and the majority of these are located in big cities.

Functional training of the amputated limb and of the whole body after amputation can promote both local and bodywide blood circulation, which is conducive to reducing swelling in the amputated limb, lessening muscle apraxia atrophy, preventing synarthrophysis and hastening the restoration of movement function. Limb-strengthening exercises may be performed on bed 1 day after surgery, and patients may actively move the amputated limb 3–4 days later. Prosthetics are usually installed after the wound has healed. At present, the application of new materials and computer control technologies provides a relatively comprehensive and systematic prosthesis treatment. Those with prosthetics installed may stand and walk almost the same as before if systematic recuperation training is undertaken.

In recent years, the emotional health of land mine injury survivor has attracted increasing attention. Ideal mental therapy not only stabilizes the emotions of victims and helps

alleviate their pains but may also encourage them to adopt a more positive outlook about the future.

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Blast Injuries from Mining Gas

Mingxiao Wang

Compared with coal mine conditions in other coal-producing countries around the world, all coal mines in China are gaseous, with 44% classified as highly gaseous. Gas explosions result in a special type of blast injury. The shock waves generated by such explosions not only injure miners directly, with injury extent intensified due to the enclosed space of mines, but the high temperature and toxic gases produced instantaneously also inflict miners with burns and chemical gas poisoning. Gas explosions in mines are extremely detrimental to both financial loss and the lives of miners. According to relevant materials and statistics, miner death rate of downhole mining gas explosions may exceed 90%. Take for examples gas explosions in the mines of Zhengzhou Coal Industry of Henan, Pingmei Shenma Group and Shaanxi Tongchuan Coal Industry in 2004, each explosion could kill a hundred or more miners. The frequency of these disasters have shocked both the domestic and international communities. It would not be an understatement to claim that gas explosion is the number one threat to the lives of coal miner workers. To better serve the public and protect the lives of miners in a responsible manner, governments ought to organize experts to carry out exhaustive and earnest studies into mining gas blast injuries, including aspects from prevention of explosion on site to treatment, safe evacuation, hospital rescue and subsequent recuperation therapies for victims of such blast injuries. At the same time, it is urgent and important to establish a mine medical care support system at the national level. This section of the book is dedicated to explorations and studies with regard to the subject at hand.

1 Section One: Overview

Flammable mining gases commonly found in mines include methane, ethane, ethylene and hydrogen sulfide, among many others. Coal mining gas is released from coal and surrounding rocks, and primarily composed of methane. Also colloquially known as swamp gas, marsh gas and so on, such gas is produced by the decomposition of organic matter in an anoxic environment when ancient plants turn into coal. Such mining gases usually exist in coal in adsorption state or free state, and under certain circumstances, free and adsorbed mining gases exist in a dynamic equilibrium state. However, when such conditions change, this equilibrium is broken. For example, when pressure drops or temperature rises, some mining gases would switch from an adsorbed state to the free state. If such conditions persist, the scope of the broken dynamic balance of the mining gases would continue to expand, and larger areas will be saturated with freed mining gases. When the concentration of freed gases reach a certain level, it could turn dangerous (permitted safe gas concentration downhole is usually no more than 1%, there is danger of explosion at 5%, and explosion is most powerful at concentration range of 8–10%). If the environment has enough oxygen and there happens to be a fire source at a sufficiently high temperature, mining gas explosion would occur. In essence, mining gas explosion is a type of chemical explosion, the outcome of a violent chemical reaction when a mixture of mining gas, chiefly CH_4, and air is ignited. Mining gas explosion is a powerful and complicated oxidation reaction, and also a free radical chain reaction process consisting of steps such as chain initiation, chain propagation, chain branching and chain termination. When the various components of gas mixture reach explosive concentration levels, and if a source of fire exists, the chain reaction process would be initiated. Chain propagation and chain branching subsequently occur in quick successions, the speed of reaction dramatically accelerates, heat released from the reaction swiftly raises temperature, and volume expands rapidly, resulting in an explosion. This is known as a thermal chain reaction, among other terms. Although the duration of explosion is not long (usually shorter than 1 s), it is nevertheless

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extremely dangerous and destructive: Instantaneous temperature exceeding 1850 °C, powerful shock wave (explosive gas speed could reach several hundred meters or even a thousand meters per second) and the generation of toxic gases (CO, CO_2, NO, etc.) could inflict on-site miners with a multitude of injuries. Oftentimes, such catastrophes kill hundreds of people, such as those at Daping mine in Zhengzhou, Tongchuan mine in Shaanxi and Jixi mine in Heilongjiang, more than a hundred casualties were recorded in each of these events. Of which, the Jixi mine gas explosion killed more than 200. Most of the casualties died on site, but even among the lucky few survivors, after being evacuated from the mine and transported to the hospital, death rate is still very high due to the multiple injuries inflicted. The author has worked at mine hospital for nearly four decades since after graduation, and has participated or commanded various mining gas explosion rescue efforts. The destructiveness and mayhem of mining gas explosions, the severe wound surface of the injured and the painful expressions of the victims are all unforgettable. While writing this section, I would often recall the painful wails of the victims and the equally painful cries of their families, and I could still remember vividly the rows of bodies. These are heart-wrenching memories, and I hope that this book can help medical professionals by providing some effective mining gas explosion treatment experiences.

There are three necessary conditions behind every mining gas explosion. First of all is that mining gas must reach a certain level of concentration, and with concentrations of 5% or 6% to 14% or 16%, the gas mixture has explosive potential. Second of all is the existence of a high-temperature fire, the general consensus is that 650–750 °C is needed. Third is sufficient oxygen, specifically oxygen concentration in the gaseous mixture needs to be more than 12%. All three factors are necessary. Yet mining gas is a flammable gas with some utilities in society. How to appropriately make use of mining gas, and how to eradicate conditions that make mining gas explosive in various steps, so as to effectively prevent mining gas explosion and suitably develop and utilize mining gas are key issues in the field of coal mine industrial production safety. China has accumulated rich experiences in preventing such accidents, so this section of the book mainly delves into focused discussions and studies from the point of view of medical care.

2 Section Two: Characteristics of Mining Gas Blast Injuries and Pathological and Physiological Changes in the Human Body

Mining gas explosions produce typical composite injuries in people.

2.1 Characteristics of Mining Gas Blast Injuries

During mining gas explosion, substantial energy is released, and because of the enclosed downhole space of mines, the tremendous energy can rapidly form high pressure through the air, with pressure reaching more than ten times atmospheric pressure. The resulting shock wave is powerful and propagates in all directions at velocity up to 2000 m/s. This kind of shock wave can move around obstacles like sounds waves do, inflict blast injuries on people including ruptured eardrum and minor bleeding in internal organs. In serious cases, death is common. Mining gas explosion is the most dangerous of all causes of injuries and deaths in mines.

Negative pressure appears immediately after the shock wave, and this too can damage tissues and organs. After reflecting off of mine shaft walls, coal beds and other downhole surfaces, the secondary reflected wave coming after the initial shock wave could also cause injury. The closer a person is to the center of the blast, the higher the peak pressure of the shock wave, and the more dangerous the shock wave. Such blast waves are always inflicted upon multiple body parts and systems, both internally and externally. This type of blast injuries are severe, complicated and worsen quickly. Many victims die immediately on site, and the small number of survivors would often die within a matter of days after being evacuated from the mine. This is why mining gas explosion injury is dangerous and has a high death rate. Mining gas blast injury may be divided into three types based on the biological effects.

1. When shock wave moves on the interface between dense connective tissues and loose connective tissues, organs containing gas and fluid would suffer the worst injuries. The auditory apparatus, lungs, stomach and intestines are most susceptible to blast injuries. The nervous system is also vulnerable to blast injury. Damage to auditory apparatus is mainly in the middle ear, and eardrum may rupture and bleed, while ossicle chains could be severed. Alveoli may rupture and bleed, even the lungs could rupture. Organs inside the abdomen may also be torn or ruptured due to impact from the shock wave. Shock wave may also transmit through the skull and into the head to inflict damage to brain tissue. At the onset, a blast injury victims might feel fine, but then respiratory failure would gradually occur within 24 h, primarily attributed to suffocation arising from burns and edema in the airway. The author has participated in the rescue of various mining gas explosions, and has seen many survivors died soon after removal from explosion environment, evacuation from mine and admission into hospital because of this.

2. Mechanical injury inflicted upon the human body due to movement of debris, rocks, coals, support structures, equipment and other objects caused by the shock wave.
3. Falling injury caused after a person is lifted off the ground by primary and secondary shock wave.

In summary, blast injury could lead to multiple injuries in the human body.

2.2 Burns and Inhalation Injuries from Mining Gas Explosion

Temperature created by a mining gas explosion could reach somewhere between 2150 and 2650 °C. Most downhole victims inside mines would suffer different degrees of burns during a mining gas explosion, and the worst cases are those nearer the center of the blast. In actual cases, high temperature generated by mining gas explosion continues to damage the human body in an enclosed environment, which is why many burn victims suffer from severe burns. Severe burns are often accompanied by large quantity of seepage, resulting in serious loss of body fluid. The rapid decline in blood volume may result in shock due to hemodynamic changes, with a high death rate.

At the same time, under the special downhole environments of mines, coal mining gas explosion would produce high temperature gases, as well as hot carbon particles, that inflict inhalation injuries to the respiratory tracts of miners. Chemical components in carbon particles would inflict chemical burns on the respiratory tract. Based on records on respiratory tract burn victims in mining gas explosions, many would die within 48 h of lifting out of mine and admission into hospital, but there are also some that die after 1 week or so.

1. A mining gas explosion quickly consumes a substantial amount of oxygen, and the restrictive condition does not allow prompt resupply of oxygen, thereby leading to a drop in oxygen level in the mine. The longer a person stays in such an environment, the more he or she inhales smoke and low-oxygen air, leading to hypoxemia and tissue hypoxia that could be fatal very quickly.
2. During a mining gas explosion, a considerable amount of toxic gas is generated, such as CO, CO_2, NO, NO_2, SiO_2 and Hsi, which are seriously detrimental to normal human body metabolism.

After carbon monoxide and harm gases enter the human body, they would swiftly spread into the brain, and diffuse into the cerebral interstitial space, resulting in swelling of brain cells and heightened brain pressure. These would result in a series of mental and nervous symptoms such as convulsion and unconsciousness. Nervous symptoms are typical. Victims would exhibit high cerebrospinal fluid pressure and muscle tremor, which may be attributed to peripheral nervous disorder after nervous system poisoning, and some survivors may remain in a vegetative state years after the initial event. Others show mild ataxia and symptoms of Parkinson's disease. Blood pressure would be elevated in victims in the early stage, but would gradually return to normalcy later.

Acute carbon monoxide poisoning is a serious composite injury common in mining gas explosion. Sometimes burn and blast injury might not be too dire, but symptoms of carbon monoxide poisoning would be obvious, mainly manifested as the following.

1. Cerebral edema: Brain edema is mostly associated with blast injury due to mining gas explosion, but causes are complicated. With carbon monoxide poisoning, carbon monoxide binds to hemoglobin and tissue cytochrome oxidase in the blood, causing hemoglobin to lose its ability to carry oxygen, resulting in inhibited tissue cell respiration that may make the victim delirious. If the situation persists, the outcomes would be vascular endothelial cell injury, increased brain barrier permeability and metabolic dysfunction of brain cells, consequently leading to a string of changes such as cerebral edema and elevated cranial pressure.
2. Pulmonary edema: This is linked with blast injuries and burns resulting from mining gas explosion, but the resulting carbon monoxide poisoning not only induce basal ganglia injury, the thalamus would also be damaged. The outcome is autonomic nerve dysfunction, leading to hemodynamic changes, and blood clots and edema of the lungs. In addition, carbon monoxide may also work directly on pulmonary capillary and lymphatic vessel, causing them to constrict and convulse. Both could cause vascular permeability changes and increased seepage, leading to pulmonary edema. A rise in excretion in the respiratory tract would lead to different levels of obstruction, causing hypoxia in endothelial cells such as lung tissues while increasing permeability, and so on, consequently worsening pulmonary edema.
3. Shock: Shock is one of the serious complications of carbon monoxide poisoning. Bodywide anoxia and damages to small vessels and capillaries due to carbon monoxide poisoning would elevate vascular permeability, even paralytic expansion, plasma seepage, blood concentration and formation of DIC. Blood volume and circulation disorders would bring about hypovolemic shock. When coupled with injuries sustained during the blast and large volume of seepage, the majority of victims would experience different levels of shock after on-site treatment and hospital admission. Sometimes shock would persist and are difficult to cure, often leading to death.

Among the oxynitrides produced after a mining gas explosion, nitrogen dioxide is the most toxic. Other nitrogen

oxides (N_2O, NO, NO_2, NO_3, N_2O_4 and N_2O_5) are not stable and may potentially convert into NO_2. NO_2 is a kind of strong oxidant that may induce lipid peroxidation in lungs and form highly destructive oxygen radicals. NO_2 could also undergo hydrolysis and turn into nitric acid or nitrite, both of which could raise permeability of alveolar capillary membrane, degrade collagen and inhibit pulmonary surfactant system function. In addition, acting on the neurohumoral reflex may also cause pulmonary lymphatic spasm, backflow obstruction, leakage of fluids from blood vessels and lymphatic vessels, resulting in pulmonary edema.

Aldehydes produced after mining gas explosion are highly toxic constituents in the smoke. There are numerous types of aldehydes, and they are among the most important fat soluble gases. Among the various sorts of aldehyde compounds, arolein is one of the most important and has a stronger simulating effect than formaldehyde. Incomplete incineration of flammable downhole materials like wood and cloth inside a mine could produce a considerable amount of aldehydes, which are highly corrosive in a local setting. Inhalation of high concentration of aldehydes may cause pulmonary edema. The mechanisms behind aldehydes damage include the reduction of cilia movement, degeneration of bronchial and tracheal membrane proteins that induce inflammatory response, lessening of alveolar macrophage activity, and injuring alveolar capillary membrane that leads to increased permeability, and in turn pulmonary edema.

In light of the above, it is clear that mining gas explosion could cause very serious harms to people. However, it should be pointed out that analysis of this subject seen in various textbooks and tool books in China tend to analyze the related matters separately, but in reality there are numerous factors involved and acting together instead of singularly. Precisely because the issue involves various factors, medical treatment professionals need to see the whole picture, instead of focusing solely on one problem at the expense of other problems, which may worsen victim condition and increase fatality rate.

It should also be emphasized that, unlike other forms of blast injuries, the majority of mining gas explosion victims exhibit a drop in total white blood cell count 24 h after injury, which progressive trend lower. In most cases, other than making judgments based on characteristics of bone marrow and peripheral blood, it may be deduced that toxic gases may temporarily suppress myeloid hematopoietic system, or temporarily reduced the body's immunity function. When organizing and participating in an emergency rescue effort for 200 miners trapped in a mine at Wangjialing in Shanxi that spanned 8 days and nights, the author carried out laboratory examinations on a group of more than rescued 150 miners, discovering that many of them showed decreased instead of increased total white blood cell count. The underlying mechanism remains unknown and requires further research and investigation.

3 Section Three: Clinical Manifestations and Treatments of Blast Injuries from Mining Gas

3.1 Clinical Manifestations

The majority of mining gas explosion victims would die on site, but for survivors the situation remains critical:

1. Polydipsia. This symptom usually appears early, and polydipsia becomes more pronounced when patient is in shock.
2. Dysphoria. Insufficient blood volume compounded by blast injury and respiratory tract burn worsen oxygen deficit.
3. Cognitive dysfunction. Mining gas explosion not only causes the multitude of injuries stated above, but is also prone to producing long-lasting cognitive dysfunction.
4. Poor flowage in blood vessels. The poor flowage in blood vessels complicate establishment of venous access, and in some cases vein incision is required to establish infusion channel.
5. Little or no urination. There may be little urination and insufficient blood volume early on, and renal failure may develop later on.
6. High pulse rate Pain, insufficient blood volume and oxygen deficiency all contribute to accelerated heartbeat.
7. Breathing difficulty. This is the outcome of direct lung injury or central cerebral edema, oftentimes a conjunction of both.
8. Intense pain. Victims usually suffer from unbearable pain.

The issues outlined above are early manifestations of shock. If treatment is untimely or inappropriate, other clinical expressions such as cold limbs, cyanosis, paleness, accelerated breathing and unconsciousness may occur.

Since most mining gas blast injuries are composite injuries, corresponding symptoms attributed to other problems like multiple bone fractures, respiratory tract burns and chemical gas poisoning may also exist. Diverse clinical manifestations, complicated injury conditions, and severe development are a clinical feature of mining gas blast injury.

3.2 Treatment

Since mining gas explosions always occur at an instant, and usually inside the confined downhole space inside the mine, it is common for large groups of people to be killed and injured. The author has participated in several such disasters where more than a hundred people died and scenes were gruesome.

The best course of action is prevention. Preventing mining gas explosion is fundamental, and with current downhole technology, mining gas explosion is totally preventable. At present, the nation has unveiled corresponding downhole mining gas explosion prevention standards. As long as work is carried out in strict accordance with rules and regulations, mining gas explosion can be effectively prevented.

If a mining gas explosion does occur, first and foremost flee the site, and strive for every second to perform rescue or be saved. If conditions allow, endangered personnel should try to save oneself and help save other victims by putting on self-rescuer. Quickly leave the toxic site and out of the mineshaft for better treatment. Check the disaster area in earnest, search for and promptly extinguish the source of fire. Cut off power supply to prevent a second explosion, search for victims, remove any obstructions, and improve ventilation to rid toxic gases. Swiftly move victims to location where fresh air is abundant, quickly remove victims from source of heat, and during movement remember to protect wound surfaces. For those suffocating or without heartbeat, administer cardiopulmonary resuscitation immediately.

At present, there is not a uniform nationwide standard for the treatment of mining gas blast injury. But mine medical personnel have summarized the following procedure based on practice.

3.2.1 On-Site Treatment

1. Keep respiratory tract unobstructed. After a victim has been evacuated from the site and relocated to a place where air flow is sufficient, if his or her airway is obstructed, a make-shift technique is to use several thick needles to puncture holes beneath the cricoid cartilage to always symptoms of obstruction. In case of tension pneumothorax, use needles with glove flap, puncture hole at the second intercostal space along the midclavicular line. For those with abnormal breathing, stabilize temporarily using cloth strap or bandage. For those with foreign objects in oral cavity or nasal cavity, remove them immediately to facilitate breathing. For those with burns or edema in respiratory tract, carry out tracheotomy as soon as possible, or else respiratory tract edema may cause lethal suffocation. Take note that tracheotomy should be conducted as early as possible, or else suffocation may occur for a variety of reasons, rendering previous rescue efforts a waste of time.
2. Rectify central hypoxia. When a victim exhibits agitation, cyanosis or cognitive dysfunction, provide oxygen immediately, and preferably pressurized oxygen supply if available.
3. Hemostasis and symptomatic treatment. If there are problems like open wounds, bone fractures and ruptured blood vessels, stop bleeding with compression bandage or manual compression, and administer an appropriate degree of fixation. For bleeding limbs, tourniquet may be utilized. Be sure to remember when tourniquet was applied, so as to avoid tissue ischemic necrosis due to excessive prolonged restriction and compression.
4. Pain relief. Whether mining gas blast injury victims suffer from burns or composite injuries, most would experience intense pain, so provide pain reliefs correspondingly. Use different of painkiller based on different injury conditions, but beware not to accidentally cover up symptoms of primary injury in order to avoid erroneous or missed diagnosis.
5. Adequately protect wound surface.

3.2.2 Hospitalized Treatment

En route to the hospital, make use of every second to perform treatment. If conditions permit, establish venous access in the ambulance, provide oxygen supply and maintain vital signs. Based on practical experience, the following measures should be taken upon admission into hospital.

Reception of Victim

1. Principles for receiving victim: First of all carry out evaluation of those with life-threatening conditions. The focus of physician should be placed on identifying such life-threatening problems, then conduct further examination before eliminating life-threatening potentials, followed by prompt, rapid and highly effective triage at emergency room of hospital.

   After the occurrence of a coal mine gas explosion, usually many people would be injured or killed. The sudden influx of a large number of victims is very similar to aftermath of disasters or modern wartime events. Unlike general traumas, refrain from conventional diagnosis as each second should be valued. Actions must be taken immediately, and triage carried out with precision and efficiency.
2. Special requirements for receiving victims of coal mine gas explosion: After a sudden influx of groups of mining gas blast injury victims, the emergency department would turn into “battlefield” for saving mining gas blast injury victims at the blink of an eye. The hospital needs to initiate emergency protocol, all clinical physicians should be put on alert and readied. Once victims arrive at the hospital, carry out a fierce “joint battle” in accordance with injury condition prediction “battle plan” and the characteristics of mining gas blast injuries.

   The emergency department is the frontline of the hospital, a crucial site for receiving mining gas blast injury victims, and the forefront of orderly medical treatment. Only with the correct categorization and organization would victims receive the proper treatment in a timely manner, maximizing the works and efforts carried out by medical personnel in a tense but orderly fashion.

Therefore, rapidly conduct screening and diagnosis, then categorize the victims. This is a crucial link in the chain, and only with this step completed adequately would subsequent medical treatment be properly oriented. Therefore, rapidly conduct screening and diagnosis, then categorize the victims. This is a crucial link in the chain, and only with this step completed adequately would subsequent medical treatment be properly oriented.

During emergency rescue of mining gas blast injury victims, the "rescue, diagnosis and treatment" principle must be uphold. In other words, ensure timely resuscitation of critical victims and maximize saving lives. This order of priority is of extreme importance, and absolutely do not perform diagnosis first before treatment as per usual protocol. The top priority is to save lives, then carry out diagnosis, or perform diagnosis while conducting life-saving rescue.

Mining gas explosions create widespread and serious damages. Not only are there surface wounds, but, oftentimes, internal injuries are even more severe, involving multiple body parts and numerous organs. Injury conditions are complicated, and it is mandatory to first identify the most critical injury in every patient, and swiftly gain the upper hand against those life-threatening risks, so as to avoid missing the most ideal opportunity to deal with such gravest dangers.

Reception time is short, victims are numerous and mission is difficult, which is why hospitals ought to consider this a top priority. Hospital director and executive deputy directors have to take charge in person. Seasoned technical experts with extensive clinical experience should be assigned to take command at the frontline, and partake in reception (triage). Team members involved in prehospitalization treatment should also collaborate proactively. All medical personnel need to stay calm and composed, so as to work in a tense but orderly manner. Tend to severely injured patients with proper care, while not neglecting those suffering from moderate and minor injuries.

3. Due to attention from various levels of the government in recent years, the standards of coordinated first-aid efforts between different organs and departments have continuously improved. Commanding organs should collectively, scientifically and uniformly coordinate the movement of victims, so as to maximally avoid large groups of victims rushed to the same hospital all at once.

Examination Upon Reception

Carry out preliminary evaluation of victim conditions (including mental conditions) as quickly as possible; establish and maintain unobstructed respiratory tract, perform tracheotomy promptly; ensure effective ventilation (stabilize flail chest in case of occlude open pneumothorax); maintain or restore effective circulation volume; conduct orderly physical examination throughout the body; use splint to stabilize obvious or suspected bone fractures, and refrain from bending patients suspected of spine injury; abstain from moving patients unless necessary, and when necessary move patients as little as possible (as in rotating between emergency room–radiology room–ward–operation room); conduct appropriate medical consultation for patients suffering from multiple injuries; physician in charge should optimize sequence of treatment and arrangement of timeframe of treatment performed by specialists.

It must be stressed that injury condition evaluation of mining gas blast injury victims, their resuscitation, and determination of sequence of treatment constitute a complicated matter and real-world problem crucial to life and death. In-depth understanding and correct solution are necessary, as are saving lives and minimizing deaths. Prevention of vital organ failure caused by cell hypoxia and posttraumatic complication occurrence are also necessary. In terms of injury condition evaluation during reception of victim, if coal mine gas explosion prehospitalization evaluation (RPMB) was conducted during emergency treatment at hospital, the said evaluation score may function as important reference (Table 1). Then conduct triage based on body parts injured.

Avoid Common Mistake During Patient Reception

When receiving mining gas blast injury victims, it is necessary to quickly determine urgency and severity. Due to the large number of victims, there will not be much time, and in such situations it is easy to make mistakes or inadequate considerations. However, the first few minutes when a patient arrives at the hospital are not only decisive in the quality and speed of early stage treatment, but also constitute a crucial phase that affects prognosis. Medical personnel needs to stay highly alert during patient reception and avoid making common mistakes.

1. Focusing only on superficial issues while overlooking real dangers would result in erroneous diagnosis and judgments, and delay treatment opportunities. Most mining gas explosion inflict injuries on multiple body parts and numerous organs. The symptoms and signs of the various body part and organ injuries early on may not necessarily match clinical severity. For example, the head's susceptibility to edema because of its ample blood supply, profuse bleeding from wounds, superficial changes to victim appearance, bloody and messy abrasion wounds, deformed limbs because of bone fracture and dislocation, and other instances are all injuries that scream for attention. On the contrary, massive bleeding in body cavity, severe cardiopulmonary contusion, and especially craniocerebral trauma arising from mining gas blast injury, lung blast injury due to mining gas explo-

Table 1 Coal mine gas explosion prehospitalization evaluation standard (RPMB)

Score	R (respiratory rate)	P (pulse)	M (movement and response)	B (total burn area or area of third-degree burn, or inhalation injury extent)	
4 points	15–24 times/min	<100 times/min	Able to obey verbal commands, responses normal	No burn	No inhalation injury
3 points	25–35 times/min	100–120 times/min	Attempt to cringe from painful stimulation	Total burn area <10%, or zero area of third-degree burn	Suspected inhalation injury surface, burn wound on neck and chest, or burn around nose
2 points	>35 times/min	121–140 times/min	Buckle or curl up in response to painful stimulation	Total burn area 31–50%, or third-degree burn <10%	Minor inhalation injury (pathology at nose, mouth or throat), singed nose hair, or dried, painful or red nose and throat
	10–14 times/min				
1 point	<10 times/min	>140 times/min	Stretch in response to painful stimulation	Total burn area 31–50%, or third-degree burn 10–20%	Moderate inhalation injury (pathology at throat or airway), raspy voice, wheezing, or obstruction in upper respiratory tract
0 point	No breathing	No pulse	No response to painful stimulation	Total burn area >50%, or third-degree burn >20%	Severe inhalation injury (pathology at bronchus or lungs), oxygen deficiency, bronchus, dry or wet rales on both lungs

Note: (1) Subtract 1 point if victim has head, neck, chest or abdomen injury or poisoning; (2) subtract 1–4 points if victim has inhalation injury; (3) first-aid rescuers on-site may reference this traumatic score for preliminary judgment of injury conditions, with maximum score of 16 points and minimum score of 0 point. Minor: 16 points; moderate: 15–14 points; severe; 13–11 points; critical 10–7 points; dangerously critical: 6–1 point; death: 0 point

sion, injury to organs in the abdominal cavity and severe shock may not be apparent due to inconspicuous external wounds, but these are life-threatening dangers that lurk beneath the surface. If attention is focused on the former, it would be easy to overlook the latter, with dire consequences.

Receiving physicians should develop the habit to first check vital signs of victim regardless of injury type. After respiratory circulation stabilizes, meticulously check all parts of the body, and for potential injuries that cannot be ruled out right away, maintain strict observation. Injury to organs in the three main cavities of cranial cavity, thoracic cavity and abdominal cavity may not exhibit typical symptoms and/or signs early on, which is why multiple examination techniques should be employed.

2. Reversal of the sequence of diagnosis and treatment: In medical procedure during normal times, it is diagnosis first, treatment second. The medical treatment process for mining gas blast injuries must be rescue-diagnosis-treatment. Of course, the rescue process will also include some diagnosis, but further diagnosis should be undertaken only after patients have been saved from life-threatening risks. For example, patients with breathing difficulty should be supplied with oxygen as soon as possible, and administer endotracheal intubation or tracheotomy if the necessity arises For those with low blood pressure, stop major bleeding and gain venous access as quickly as possible so as to enable blood transfusion. The reasons behind breathing difficulty and low blood pressure may be checked and analyzed during the rescue process, or after respiratory circulation has stabilized. Physicians accustomed to regular work routine often violate the rule of rescue before diagnosis. It is necessary to act more flexibly and change stubborn adherence to the pattern of diagnosis before treatment. Based on actual clinical circumstances, either rescue and diagnose at the same time, or rescue first before diagnose.
3. Complacent with the discovery of injuries to one or two body parts or organs: The majority of mining gas blast injuries are composite injuries or multiple injuries, and there is no doubt that complacency with the discovery of injuries to one or two body parts organs and forgoing continual discovery of other potential problems will ultimately harm the patient. There have been a myriad of clinical deaths resulting from lack of timely measures due to missed diagnosis.
4. Wasting too much time on inspections that could be delayed: Traditional physical checks usually suffice in providing preliminary insights about the injury, while thorough and accurate diagnosis always requires a variety of supplemental examinations. Unless extremely necessary, these supplemental tests do not need to be performed soon after injury, especially those that requires moving and lifting the patient, or those that requires relocating to special examination facilities. Actually there have been clinical deaths in patients due to untimely rescue from circulatory failure or asphyxia in X-ray room and CT room. Furthermore, tests also delay post-injury treatment time, and the length of this time is decisive in survival rate, infection prevention and preservation of bodily function.

For the various supplemental tests that require moving and lifting patient, principally speaking these should be conducted after his or her vital signs have stabilized. In

recent years, it is unclear the injured organ and injury severity of serious multiple injuries. The method of undertaking CT scan quickly under the accompaniment of physicians, then sent to surgery room for rescue may be an option for facilities with high-performance CT, but choose this option carefully and in full consideration of the hospital's actual situations. If hospital conditions permit, move equipment to facilitate bedside examinations. For injuries with clear diagnosis there is not much need to supplement with additional checks. For problems or procedures such as abdominal wall tension after injury, tenderness, rebound pain, lack of intestinal peristalsis, and verified abdominal cavity organ injury after performing abdominocentesis, peritoneal lavage, be decisive and carry out diagnosis and treatment simultaneously by way of abdominal probe.

5. Insufficient attention for complications during early phase: Some complications may occur soon after injury, such as fat embolism after bone fracture, acute renal failure after compression, osteofascial compartment syndrome due to use of tourniquet during first-aid rescue, brain herniation after craniocerebral injury, upper body paraplegia after spinal injury, and acute cardiac tamponade due to intracardiac hemorrhage. These complications could lead to patient disability or death. It is very dangerous and detrimental if secondary complications are overlooked because attention is trained only on primary injuries.
6. Overlooking potential risks in some tests: Some examinations may induce major or even life-threatening dangers. For example, when a patient with high cervical spine injury undergoes motor examination without fixed traction on the cervical spine may result in fractured or dislocated cervical spine compressing the cervical spinal cord and causing respiratory arrest. Pain from abnormal movement examinations of bone fractures may worsen shock, fracture ends may injure adjacent vessels and nerves, or cause fat embolism among other problems. These tests should be done with utmost care, and it would be best to avoid them altogether unless absolutely necessary. Even if these tests must be done, most mine hospitals are outfitted with devices that may be moved to facilitate bedside examinations.

All in all, when receiving patients, focus resources and energy, carry out correct triage, and swiftly send patients to the correct and corresponding department. Timely treatment is key so make haste, but beware of simple and low-level mistakes when working under pressure.

Examinations of Injuries to Different Parts of the Body

Preliminary Diagnosis When patients arrived at different departments, ensure the provision of life support for those in critical conditions. After their conditions have stabilized, earnestly analyze the formation, existence and development process of the injuries of each victim based on the features of mining gas blast injuries and in close conjunction with clinical expressions. Refer to various test results when carrying out preliminary diagnosis.

Examination and Diagnosis of Different Parts of the Body Based on the features of mining gas blast injuries, concentrate on key areas and the individual characteristics of each victim when carrying out preliminary diagnosis. Again, it should be underscored that missed diagnosis on injured body part of those with multiple injuries must be avoided during this phase. It is also necessary to confirm treatment measures in accordance with thorough examination. Second of all, adopt a critical mindset when identifying and treating specific injuries. After mining gas blast injury victims stabilize, strictly adhere to clinical diagnostic procedures, gain in-depth understanding about patient's external wound history, and seriously conduct bodywide checks, including specialized examinations and selected supplemental tests.

External Wound History

The main goal is to understand the real injury mechanism or process.

The majority of mining gas blast injuries are severe, while rescuing try to collect info about medical history within the shortest amount of time possible, and then predict body part(s) injured, injury level and injury condition changes based on medical history. Detailed injury history may be obtained later when victim respiration has stabilized.

It would be best if external wound history is provided by the victim himself/herself, but someone else on-site would suffice if the victim is in critical condition or unconscious. If no one is available to provide info about the injury process, and the victim is unconscious, physicians should plan for the worst, and make plans for problems like craniocerebral injury, cervical spine fracture, major bleeding, and injuries to organs in the thoracic or abdominal cavities. This task should be undertaken by an experts group comprised of those with extensive clinical experience, theoretical knowledge and practical abilities.

1. Time of injury: The span between time of injury and time of treatment is one of the key factors that determine the life or death of patient. Some reversible injuries eventually become irreversible as treatment is delayed. Take for example shock, which may be easily recovered from if sufficient blood volume is replenished early on. Otherwise, as time passes, bad changes in homeostasis, hemodynamics and cells would eventually deteriorate

into organ failure from which recovery is nearly impossible. Severe burns resulting from mining gas explosion would cause the body to lose fluid rapidly, with the rate of decline peaking at around 5–8 h after injury. There may be different judgment about state of shock when diagnosis is conducted at different time. Adopt effective and targeted measures. Clinical experiences indicate that accurate judgment and emphatic measures can substantially raise rescue success rate, while the opposite would increase death rate.

2. Reason of injury: If possible, explanation by the victim himself/herself should provide important clues about injury. Physician knowledge about injury mechanism is foundation for correctly oriented tests, especially since mining blast injuries are usually a combination of various factors. What are the main causes of injury conditions of victims? Is it caused by one or various factors? What's the extent or injury? Search in earnest to maximize chance of obtaining valuable diagnostic information. For example, a patient might say that he suddenly heard a massive boom while at work, felt a heat wave rushed toward him, smoke blurred all vision, and then perceived difficulty breathing. Then he lost consciousness. At this stage, physician may arrive at the preliminary conclusion that cerebral hypoxia or poisoning from inhalation of toxic gases are possible.
3. Environment of injury: The work environment of coal mine shaft is rather unique because of the combination of properties such as enclosure, darkness, wetness, smokiness and noise. Mining gas explosion worsens the environment, and potential dangers to the human body are much more dire than the same situation on ground surface. Due to the lack of in-depth understanding, it is difficult to accurately describe the issue. However, the location of the victim at the time of injury should provide very helpful information for diagnosis, and when necessary refer to the mine's downhole map.
4. Appearance of symptoms and signs: In most cases, the appearance and changes of symptoms after injury may be utilized as clues for diagnosis and directions for treatment. For example, a victim with head injury may stay consciousness for a short duration, then gradually lose awareness and turn unconscious. It is possible that the victim is not suffering from cerebral contusion and laceration, but rather epidural hematoma. A lot of mining gas blast injury victims display agitation, and in such cases first and foremost remember blast injuries and external craniocerebral injuries. However, inhalation injury or poisoning may also be the culprit, or even oxygen deficit, or secondary brain injury due to blast lung injury, as well as insufficient circulation volume. Carry out physical examination in earnest, make use of chemical tests, appropriately utilize imaging checks and instruments like CT, MRI and B-mode ultrasound to further ascertain diagnosis.

   Severe chest injuries can usually be diagnosed within several minutes upon admission to hospital, for instance flailing chest, abnormal breathing, and forced breathing with mechanical ventilation assistance. Wounds visible to the naked eye or subcutaneous emphysema on the chest wall or neck perceivable through palpation. Chest X-ray can further help diagnosis, and for severe shocks that cannot be explained by injuries in other body parts, seriously consider dire injuries to organs in the chest cavity, such as myocardial damage, cardiac tamponade and massive hemothorax.

   In terms of symptoms and signs of injuries to the abdomen, surgeons should be familiar with these. However, avoidable deaths still occur on occasion, so refrain from complacency and in particular look for signs in the abdomen. Clinically experienced physicians are actually prone to erroneous judgments, so heed these words in earnest.
5. Treatment prior to arrival at hospital: Prehospital admission treatment is important. Earnestly collect information about whether or not a victim has been given first-aid, and if so who provided the prehospital arrival first-aid such as basic-level personnel, or professional teams. Report information such as first-aid rescue process, situation regarding use of medication such as painkiller or vasopressor agent, quantity and type of fluid utilized in transfusion, whether or not tourniquet was used, airway openness status and prehospital arrival injury score. These information should all be obtained in earnest.

Full-Body Check

After vital signs have stabilized, promptly carry out full-body examination. Make use of conventional visual inspection, palpation, percussion, auscultation and other physical methods to swiftly establish preliminary but accurate judgments. Mining gas blast injuries not only inflict serious damage to the surface of the victim's body but, oftentimes, there are closed injury concealed. Check and identify these hidden dangers as early as possible by undertaking the necessary tests. Usually preliminary inspections are carried out based on the following criteria.

If there is no visible major bleeding but blood pressure is relatively low, consider the possibility of hemorrhage in the pleural cavity, peritoneal cavity or retroperitoneal space. Consciousness dysfunction is usually an indicator of craniocerebral injury. Use GCS score to record extent of unconsciousness, then undertake nervous system checks, and pay attention to signs such as size of pupil and reaction to light. Check the cranial nerves to determine if there is damage to the brain stem, and unequal size of pupils is a dangerous signal. Pain or tenderness in the cervical spine is a common

symptom indicating cervical fracture or dislocation. Take lateral X-ray bedside as soon as possible. For fracture of lumbar vertebra, also perform X-ray imaging quickly to produce a clear diagnosis. Those who can speak loudly are basically free of serious injury to the chest, and percussion and auscultation can determine pneumatosis or effusion in the pleural cavity, while X-ray imaging can help understand whether or not the lungs and mediastinum have been injured.

It is suggested to pay attention and look for any signs of peritoneal irritation when performing abdominal examination, namely, abdominal tenderness, rebounding pain, abdominal muscle tension or weakened or disappeared peristaltic sounds. These can help determine whether abdominal laparotomy is required. In acute injuries, it may be hard to pinpoint the specific organ injured. When it is difficult to eliminate the possibility of injury to organs in the abdomen, carry out abdominocentesis and/or peritoneal lavage. If thrombectomy sucks out noncoagulated blood, that is a positive result; but negative result from thrombectomy is not sufficient to eliminate possibility. Relatively speaking peritoneal lavage is more valuable, and if conditions permit carry out bedside B-mode ultrasound, or if allowed it would be even better to conduct CT scan.

Deformed limbs and those that cannot move at will means bone fracture or dislocation. Slight percussion to base of wrist or heel may induce bone fracture pain in the upper or lower extremity. Based on the result, specific bone fracture location may be identified. For an unconscious patient, tap on the person's processus styloideus radii or malleolus medialis, while simultaneously performing auscultation at the manubrium sternum or pubis. The side with bone fracture would produce a lower tone and volume, based on which judgment may be made about whether or not there are fractures in the upper or lower limbs. Of course, bedside X-ray scan would be even more helpful. If pulse at radial artery or dorsal artery of foot are strong, possibility of major blood vessel damage in the upper and lower limbs may be eliminated. If fingers and toes can move at will, it's basically a confirmation that peripheral nerves have not been damaged much. Pay extra attention to victims with multiple injuries, and during the early stage concentrate efforts of saving lives, though do not leave nonfatal injuries unattended.

Specialized Examinations

These are very important and may sometimes provide crucial basis for diagnostic judgment. Carry out thorough and earnest inspections in accordance with physical examination standards and protocols. Look for unique symptoms and signs indicative of various types of injuries. These include both important positive and negative signs. Conduct detailed specialized checks for common injuries inflicted by mining gas explosion such as burn, inhalation injury, poisoning, craniocerebral injury, chest injury and abdominal injury. Take for example external head wound, use composite cognitive score (CCS), head examination, leakage of cerebrospinal fluid, signs in the eyes, fundus status, motor dysfunctions, perception dysfunctions, cerebellar signs and other checks.

Supplemental Examinations

Take advantage of opportune timeframes in carrying out examinations and tests that are necessary or possibly necessary for mining gas blast injury, then make appropriate choices in supplemental examinations. Supplemental examinations can quickly and accurately provide objective indicators about the victims, but usually conduct such tests bedside to prevent causing secondary injuries during the course of examination.

1. X-ray: This can provide diagnostic basis for mining gas blast injury, but it should be underlined that X-ray must be not undertaken at the cost of delaying rescue or endangering victim safety. Use bedside X-ray machine, but this requires the operating physicians to be knowledgeable enough about radiology. If severe and multiple injury victim conditions permit, obtain X-ray imaging of head, cervical vertebra, chest, pelvis and suspected or confirmed bone fractures during first time. Chest X-ray is more valuable when taken with patient standing up instead of lying down, but victim injury condition should still take precedence, and images may also be taken from an inclined position or multiple angles. Digital radiography (DR) technology has been widely adopted today because it offers dramatically clearer imaging, which greatly improves diagnosis accuracy, so make good use of it if available.
2. B-mode ultrasound. This is particularly valuable for injuries in the chest and abdomen, use bedside if such equipment is available. B-mode ultrasound is mostly utilized in checking organs within the thoracic and abdominal cavities for problems like hemorrhage, effusion or ruptured organ, but beware that hollow organs in the abdominal cavity may possibly affect accuracy.
3. CT scan: This offers relatively high-resolution imaging and tissue contrast, with CT image cleared of tissue structure overlapping images. CT scan can reveal abnormal changes and is chiefly used for injuries to the head, brain, chest, abdomen, spine and major joints. In recent years, high row helical CT has found successful application in severe multiple injuries. Due to its short scanning duration and high diagnostic value, it has been very useful in emergency surgeries and praised by emergency rescuers.
4. MRI: This can provide highly valuable diagnostic evidences for mining gas blast injuries, especially those without external wounds. MRI is primarily used for the brain, spinal bone marrow, craniocervical junction, internal organs and deep soft tissues. MRI can take advantage

of water imaging principles to provide images of the biliary tract and urinary system, but may also be used selectively. Changes in MRI images offer special diagnostic value for brain tissues of mining gas blast victims.

3.3 Injury Severity Scoring

3.3.1 Injury Severity Score: Abbreviated Injury Scale (AIS-ISS)

The majority of mining gas blast injuries are multiple injuries and composite injuries with complicated circumstances. Injury score is most capable of reflecting severity level of these injuries. For mining gas blast injuries AIS-ISS is the top choice for scoring inside hospital as it can accurately reflect a patient's injury status. At present, the Abbreviated Injury Scale Standards 2005 (AIS2005-ISS) is the latest edition available, and it may assist clinical physician in making judgment about victim injury status by providing guidance on correct diagnosis. What follows below is a brief explanation about Injury Severity Score—Abbreviated Injury Scale (AIS-ISS).

AIS is a rating standard based on anatomical indicators. Since inception in 1971, the AIS is renewed once every 5 years, and the latest edition is that from 2005. Here, the AIS-85 is used as example to explain the subject: The AIS-ISS rating system uses diagnostic codes from the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM). AIS-85 comes in the two forms of coding manual for microcomputer use and abbreviated table for clinical application. For clinical application abbreviated table, please refer to the Abbreviated Injury Scale 1985 revision. AAAM. Arlington Heights, IL, 1985. The said standard divides the body into seven areas, namely, head, neck, shoulder, chest (including thoracic vertebrae), hip (including lumbar vertebrae), limbs (including pelvis) and body surface. Injury severity is rated based on diagnosis as either minor, moderate, serious (not life-threatening), severe (life-threatening but survivable), critical (sometimes survivable) and maximal (untreatable). These are respectively given a score of 1 point, 2 points, 3 points, 4 points, 5 points or 6 points. ISS is based on the AIS rating, with a minimum score of 1 and maximum score of 75. The ISS scoring method is based upon the first five levels of the AIS-85, and specific ISS value is calculated as square of the most severe injury score of the three most severely injured body parts. When AIS reaches level 6, an ISS value of 75 may be given directly.

Below Is an ISS Calculation Example

If a person is hit by a car, his/her injury diagnosis is: (1) Fracture at the second and third ribs on the right; (2) tension pneumothorax on the right side; (3) perforated stomach; (4) comminuted fracture of the pelvis; and (5) brachial plexus nerve contusion. Using the Baker method to assess (1) chest, (2) abdomen and (3) limbs, then check AIS-85 (blunt trauma abbreviated table) to obtain AIS values of respectively 4 points, 4 points and 3 points. In turn, ISS = $4^2 + 4^2 + 3^2 = 41$. Literatures and documents indicate that for victims with injuries to only a single body part, it would be best to express injury severity using AIS value, but for multiple injuries or injuries to multiple parts, scoring for composite injury victim must use ISS. The AIS-ISS scoring method can reflect to a certain extent the severity of injury, which is why the system has been broadly employed. In general, it is held that those with a score of ISS < 16 are mildly injured, those with score ≥16 are seriously injured, and those at the ≥25 level are critically injured. It has also been proven that there is a close relationship between a victim's age and his/her death rate, with the connection particularly prominent in those suffering from moderate injuries. For example: At the ISS cumulative score range between 10 and 19, a victim 70 years of age or older is eight times more likely to die than one who is younger than 50. But in case of ISS cumulative score >50, death rates are nearly identical. Among the dead, the higher the cumulative score, the shorter the time of survival, and it is extremely rare for those with cumulative score of 10 or below to die. When ISS based on AIS-85 as applied to blunt trauma or perforation wound victim below the age of 50 years old, if cumulative score is 40, death rate hovers around 50%, when cumulative score is 25, death rate hovers around 25%; and if cumulative score is 18, death rate hovers around 10%. Modern medicine has a lot of potential and possibility in raising the survival rate of victims with cumulative score in the range between 10 and 50 points, and even when cumulative score passes 50, as long as equipment are advanced enough and rescue timely enough, survival possibility exists. Yet researches indicate that there are still quite a few shortcomings in utilizing ISS for evaluating multiple injuries. For example, (1) ISS evaluation only includes the most severe values in the three body parts out of the total six. If the fourth body part has less severe injury, it would not be included, thus the system does not thoroughly reflect the victim's injury condition. (2) In ISS evaluation matrix, only the most severe score from each body part is chosen. If two equally serious injuries exist, one would be eliminated. For example: Liver laceration has an AIS score of 5 points and is a life-threatening injury. Posterior hepatic vena cava tear meanwhile is another dangerous injury but would be ignored by the system. (3) Victims with the same ISS have significantly different rate of survival. For example: Two victims both have ISS score of 25, with one suffering from severe head injury with at least one injury given AIS value of 5 points ($5^2 = 25$), while the other has a complicated urinary tract tear injury with AIS value of 4 points in addition to an open radius fracture with AIS value of 3 points ($4^2 + 3^2 = 25$).

Obviously there is a difference in survival rate between these two cases. (4) ISS system assigns the same weight to AIS values of identical injury severity in different body parts. For example, full-layer stomach perforation and brain laceration both give an AIS of 4 points, and the resulting ISS values are both 16 points. Yet it does not reflect the injury condition differences between the two. Therefore, it can be seen that although the ISS method has seen broad adoption in the evaluation of multiple injuries, it is nevertheless flawed in some ways. To better reflect injury status, Boyd CR et al. created Trauma Injury Severity Score (TRISS). The said system combines ISS evaluation, RIS evaluation, nature of injury, age of victim and other elements, then calculate the patient's probability of survival (PS) to reflect the person's injury level.

To explain the specifics in the calculation of PS value, please refer to this example here. A 56-year-old victim has been afflicted with perforation wound, and anatomical indicators are as follows: (1) Fracture at the second and third ribs on the right; (2) tension pneumothorax on the right side; (3) perforated stomach (external wound); (4) comminuted fracture of the pelvis; and (5) brachial plexus nerve contusion; using the Baker method to assess injuries to the (1) chest, (2) abdomen and (3) limbs, then check AIS-85 (blunt trauma abbreviated table) to obtain AIS values of respectively 4 points, 4 points and 3 points, and therefore, $ISS = 4^2 + 4^2 + 3^2 = 41$. Physiological indicators: Known indicators include GCS = 13 points, SBP = 9 kPa and RR = 9 times/min, then check table to obtain quantified values of respectively 4, 3 and 2, then check corresponding values on Table 2 to obtain their respective weights of 3.7472, 2.1978 and 0.5816. The sum of the latter three is RTS value, as in RTS = 3.7472 + 2.1978 + 0.5816 = 6.5266. Choose the appropriate PS value table in accordance with clinical information, and in this example the correct PS table is the one for perforation wound for ≥55 years old. From the *y*-axis locate the spot for RTS value of 6.5266, and from the *x*-axis look for the coordinates for 41, and the value of the two scores on the table is the PS value being sought. The said example has a PS of 0.49, and since the said victim's PS < 0.50, he or she has a rather high chance of death.

3.3.2 A Severity Characterization of Trauma (ASCOT)

Like the TRISS method, A Severity Characterization of Trauma (ASCOT) is also a quantified injury severity evaluation method that comprehensively predicts injury level by taking into full account physiological indicators, anatomical indicators, age factors and injury types. Yet the former's physiological indicator weighing system is simple but rational. In its composition, anatomical indicators adopt an AP scoring system whereby any scores of AIS ≥ 3 are not ignored, thereby overcoming the relevant disadvantage in the ISS method. Its age brackets are more appropriate than that of the latter (Table 3), while the weights of injury types (blunt trauma and perforation injury) are also more sensible. Therefore, the ASCOT method is currently considered the most comprehensive, multifactor injury prediction technique. Contents of the said method are briefly outlined below.

Table 2 Weighted age groups in ASCOT method

Weighted group	Age group
0	0–54
1	55–64
2	65–74
3	75–84
4	≥85

Table 3 Weighted table of clinical information for the ASCOT method

Clinical information	Blunt trauma	Perforation wound
Constant	−1.1570 (K_1)	−1.1350 (K_1)
Glasgow Coma Scale (G)	0.7705 (K_2)	1.0626 (K_2)
Systolic pressure (SBP)	0.6583 (K_3)	0.3638 (K_3)
Respiratory rate (RR)	0.2810 (K_4)	0.3332 (K_4)
AP		
A	−0.3002 (K_5)	−0.3702 (K_5)
B	−0.1961 (K_6)	−0.2058 (K_6)
C	−0.2086 (K_7)	−0.3188 (K_7)
Age	−0.6335 (K_8)	−0.8365 (K_8)

The ASCOT method consists of two steps, namely, the collection of clinical information, and the numerical model calculation for PS (probability of survival).

Collection of Clinical Information

1. Physiological indicators: These include the Glasgow Coma Scale (GCS), systolic pressure (SBP) and respiratory rate (RR). These three factors can all acutely reflect the severity of victim injury condition, and they are reliable when measured right after the victim has been delivered to the emergency department. Check table to see what their respective values indicate.
2. Anatomical indicators: Categories are based on ICD-9-CM injury diagnosis with reference to AIS-85 classification standards, then the aforesaid AP scoring system is used to comprehensively evaluate anatomical injury severity, resulting in the *A*, *B* and *C* value.
3. Age factor: Champion is of the opinion that victims with age ≥55 years old have significantly higher chance of death compared with victims with age <55 years old suffering from the same injury condition. For the former, death rate increases significantly for every increment of 10 years of age. Author added weights to the various age brackets (Table 2) to overcome the over-simplicity of age categorization in the TRISS method, and effectively enhanced injury condition prediction accuracy in the ASCOT method based on age.

4. Injury type: The so-called injury type here refers to whether the injury inflicted in blunt trauma or perforation wound. Since treatment reasons differ, their death rates also vary significantly. The author applied a weighted system for processing, and the details are listed in Table 3.

Numerical Model Calculation for PS Value

The numerical model in the ASCOT method is PS = $1/(1 + e^{-k} \ldots e = 2.718282$; and $K = K_1 + K_2G + K_3S + K_4R + K_5A + K_6B + K_7C + K_8$ge. In the equation, $K_1 \ldots K_8$ should use the weights for blunt trauma or perforation wound as provided in able 3 based on the type of injury sustained by the victim. Weights for age come from Table 3. Then input the A, B and C values of *G*, SBP and RR scores and AP scores into the numerical model to calculate the PS value. Physicians may determine victim injury condition quantitatively and qualitatively based on the PS value, make judgment of treatments to be administered and predict prognosis.

The Feicheng Mining Affairs Bureau Central Hospital (now Feicheng Mining Central Hospital) designed an ASCOT application software for microcomputer usage. Information collection menu indicates that numerical model calculation is done by the microcomputer. AIS-90 has been coded into the software; thus, evaluation and quantification of anatomical injuries are much more scientific. The application of said software not only enables rapid and accurate acquisition of PS value, and also serves as a storage of first-hand material for injury score data. The said software has been used on laptop microcomputer to carry out prospective validation of 250 relatively severe cases. The software has an accuracy of 83.3% and a false positive rate of 17.7%. Its specificity is 95% and false positive rate is 5%.

3.3.3 Mining Gas Blast Injury Bodywide Complication Diagnosis with Injury Severity Score, Abbreviated Injury Scale (AIS-ISS)

Mining gas blast injuries are predominantly composite injuries and multiple injuries, with victim condition severe and complicated. Bodywide complications are also common. These complications may occur right away after injury (i.e. shock), but usually take place a short while after injury. Bodywide complications are always life-threatening, no less dangerous than the severe injury itself.

The onset of mining gas blast injury complication is dangerous. If the harm can be thought of during the early stage, symptoms and signs are noted as they appear, and corresponding checks are conducted based on appropriate plans, then it would not be hard to discover them promptly. For example, during early phase of serious burn, in particular cases in which resuscitation was delayed, shock is inevitable. Most cases may be clearly diagnosed based on clinical processes and changes, meticulous observations and examinations, and responses to treatments administered.

However, for crushing of the victim's body created by the mining gas explosion, acute renal failure complication is unique in its own way. (1) Victim falls unconscious due to the accumulation of unique effects of oxygen deficit and large amounts of toxic and harmful substances inside mine generated from mining gas explosion, then stays in the anoxic and toxic environment; (2) limbs or torso of victim are not compressed by external, heavy objects for extensive period, but instead compressed by his or her limb in a fixed position. (3) Unknown external wound history of victims. It is very easy to overlook considerations for crush syndrome; (4) during the early stage, there are no obvious abnormalities, no signs of wounds on skin, no clear swelling of limbs and victim can even move around, which is why the problem is often missed in diagnosis. The hospital where the author worked once received two mining gas blast injury victims. They passed out for extensive periods after breathing in large amount of low-oxygen air and toxic and harmful gases. Crush syndrome arising from compression by their own weights in fixed position resulted in acute renal failure complication early on.

Complications common in mining gas blast injuries include traumatic shock, wound infection, electrolyte disorder and acid–base imbalance, blood coagulation disorder, stress ulcer, acute respiratory distress syndrome, acute renal failure, multiple organ dysfunction syndrome, and so on.

4 Section Four: Treatment of Mining Gas Blast Injuries in Hospital

4.1 Rectify Injury: Hemorrhagic Shock

Hemorrhagic shock happens when the body has been subjected to the stimulation and tissue damage of serious trauma. It is a form of acute circulatory insufficiency characterized by micro circulation dysfunction induced by "blood vessel-nerve" reaction, as well as the resulting tissue and organ blood flow insufficiency, oxygen deficiency and general damage to organs.

Hemorrhagic shock is shock arising from massive loss of blood and is commonly seen in external wound bleeding, peptic ulcer bleeding, esophageal variceal rupture, obstetrical and gynecological diseases, and so on. Whether or not shock occurs after bleeding does not hinge on the volume of blood loss, but rather the rate at which blood was lost. Shock always happen when large amount (more than 30–35% of total blood volume) is lost at a rapid pace and without prompt replenishment.

Shock caused by damaging factors such as serious trauma suffered by the body includes many reasons such as major

blood loss, but also intense pain, the release and absorption of products from decomposition of necrotic tissues, and harmful elements from problems such as trauma or infection. These lead to dysfunctions in the body's normal physiological capacity, which is why such shock is often called trauma-hemorrhagic shock.

Coal mine gas explosion produces severe composite injury in the very typical sense. The resulting shock may arise from trauma, blood loss, burn-induced fluid loss, and so on, which is why this type of shock is also called trauma-dehydrating shock.

Thus, the first priority in treating mining gas blast injury victims at the hospital is to deal with shock.

4.1.1 Replenish Blood Volume

Fluid transfusion volume for the first 24-h after injury may be calculated based on the following equation:

$$\text{Burn area} \times 100 + 1000 = \text{Total fluid transfusion volume}$$

At least half of the total volume needs to be delivered within the first 8 h.

In the selection of fluid for transfusion, pay careful attention to ratio between crystalloid solution and colloid solution, usually the range between 1:1 and 1:1.5 is ideal.

Transfusion of whole blood: Fresh blood would be optimal, and in emergency situations deliver 300–600 mL via arterial line, then replenish subsequently in accordance with actual patient conditions.

Plasma: Fresh plasma, frozen or dried plasma, or CS-706 plasma may all be used.

Low molecular dextran.

In emergency situations, administer 100–150 mL of 5% glucose solution by way of intravenous therapy, and crystalloid solutions such as sodium lactate, recurrent sodium chloride or normal saline are all acceptable.

Volume and rate of transfusion shall be determined according to actual victim conditions. But rate of transfusion should be faster during the early stage.

If shock persists even after sufficient blood and fluid transfusions and replenishment, it would be necessary to consider the possibilities of factors such as hemorrhage, metabolic acidosis, DIC, infection or cardiopulmonary insufficiency.

4.1.2 Stop Blood Loss

For external bleeding, apply dressing, and use pressure dressing when necessary, or surgical suture. For internal bleeding, carry out surgical exploration and hemostasis when necessary.

4.1.3 Relieve Pain

Strong and effective pain relief is very important. Pethidine and morphine are two viable medications.

4.1.4 Administer Vasoactive Agent

It is suggested to administer vasoactive agents and inotropic drugs scientifically based on patient conditions.

Applicable symptoms include rise in CVP after volume increase and persistent shock; appearance of serious neurological signs (pale skin, cold limbs, inadequate filling of capillaries, etc.); concurrent pulmonary hypertension or left heart failure; refractory shock, elevated peripheral resistance, and so on.

4.1.5 Rectify Acidosis

It is suggested to follow conventional treatment procedures.

4.1.6 Protect Cardiac, Pulmonary and Renal Functions

It is suggested to protect cardiac, pulmonary and renal functions by adopting targeted measures based on changes in patient conditions, following conventional treatment procedures will suffice.

4.1.7 Protect Against Infection

Use broad-spectrum antibiotics simultaneously because downhole environment of coal mine is contaminated. Coupled with increase in wound surface contamination possibilities during a victim's evacuation process, it would be key to administer antibiotics to protect the life of the victim. Clinically speaking, broad-spectrum antibiotics are most often used.

4.1.8 Make Appropriate Use of Traditional Chinese Medicine

Traumatic shocks are categorized as "syncope" and "loss of body compositions".

Etiological Mechanism

(1) Blood loss and fluid loss, *qi* is lost along with blood, and trauma damages the veins. The large volume of blood lost is coupled with loss of *qi*, then *yang* energy dissipates along with *yin* energy. Or vomiting after injury and excessive diarrhea damage the spleen and stomach, resulting in abnormal rises and falls of body compositions that are discharged in partly clear, partly murky forms. The excessive consumption of *yin* fluid results in *yang* energy dissipating along with *yin* energy; (2) too much heat toxins consumes *qi* and *yin* energy, leading to damage, and after injury detrimental toxins invade the body, heat toxins coagulate and turn into fire, which consumes *qi* and *yin* energy, leading to "loss of body compositions"; intense pain injury after intense pain, causing *qi* to reverse its cause, leading to abnormal flow of *qi* and blood, thereby affecting venous function and the heart and mind, then in turn leading to "syncope."

Sub-Divisions

(1) "Syncope" is further divided into cold syncope and hot syncope; (2) "loss of body compositions" is further divided into loss of *yin* blood, loss of *yang qi* or loss of both *yin* and *yang*; clinically speaking, syncope is mainly characterized by sudden fainting and unconsciousness, and cold hands and feet; loss of body compositions could be expressed as syncope, but also perspiring huge beads of sweat, opened mouth with closed eyes, inability to clench fist, uncontrolled excretion and pulse weakened to the point of disappearance, among other signs. Early on, it is common to see pale faces, cold limbs, palpitation, excessive sweating, shortness of breath, lethargy, reduced urine volume, mental tension, irrigation, weak pulse and dropped blood pressure. Serious cases may exhibit indifference, delirium, cyanosis of lips and end of limbs, shallow and rapid breathing, and even wheezing due to phlegm in throat, and lack of urine. Early pulse condition is usually slow and weak, and for serious cases pulse may be weakened to the point of disappearance, or pulse cannot be found, and even blood pressure reading cannot be obtained. This is an acute problem of internal medicine, but in external injury, in particular mining gas blast injury, it appears differently, possibly in more urgent and serious manner, which is why it is necessary to administer treatment based on the problem.

Treatment

Emergency Treatment

Strengthen qi, rescue yin and fortify against loss: Use 10% shengmai injection, or 10–30 mL shengmai injection, then add 30 mL of 50% glucose solution and administer via intravenous drip. Administer once every 15–30 min for three to five consecutive times. After blood pressure rebounds or stabilizes, mix 50–100 mL with 250–500 mL of 50% glucose solution and administer via intravenous drip, until patient is recovers from syncope state.

Pharmacological studies have proven that shengmai injection has the following effects: benefits the heart, raises blood pressure, and improves micro circulation; resists gram-negative bacterial endotoxin; promotes phagocytic function of reticuloendothelial system; excites the pituitary–adrenal cortex; reduces cyclic phosphonucleotide levels in plasma of endotoxin shock animals; and promotes sedation.

Restore yang energy, rescue reversal, fortify body against loss: Use shenfu injection or shenfu danshen injection 10–20 mL, then add 40 mL of 50% glucose solution and administer via intravenous drip once or twice. Next, mix 40–80 mL with 250–500 mL of 10% glucose solution and administer via intravenous drip twice a day.

Pharmacological studies have proven this type of formula benefits the heart and improves micro circulation. Animal testing prove that it can clearly raise blood pressure in dogs suffering from acute hemorrhagic shock and increase contractile force of heart of anesthetized rabbit.

Restore both yin and yang to fortify body against loss: Choose between shenfu injection or shenfu danshen injection, plus shengmai injection or shengmai injection, based on actual circumstances and administer in method and dosage as above.

Detoxify, discharge heat and open up closure: This is principally applicable to hot syncope. Use Xingnaojing Zhusheye or Qingkailing Zhusheye 20–40 mL, mix with 250–500 mL of 10% glucose solution and administer via intravenous drip once or twice a day.

Pharmacological studies have proven this type of formula has an obvious heat dissipation effect, and can improve intestinal blood flow, reduce ischemia, lessen endotoxin's inhibition of the reticuloendothelial system and offer anti-inflammation and sterilization benefits.

Improve blood flow, disperse blood stasis and open up closure: This is chiefly applicable to those with shock coupled with DIC. Use compound danshen injection and Angelica sinensis injection 4–10 mL each, then add 40 mL of 50% glucose solution and administer via intravenous drip once every 30 min. After two consecutive sessions, switch to 20–30 mL mixed with 100–250 mL of 10% glucose solution and administer via intravenous drip.

Pharmacological studies have proven this type of formula improves micro circulation, promotes blood perfusion in tissues and strengthens blood supply to key organs, which is why it plays a crucial role in helping patients recover from shock.

Treatment Based on Syndrome Differentiation

Loss of Body Compositions

(1) Loss of yin blood: Blood loss due to trauma, major damage to yin blood and depletion of body fluid. Delirium with profuse sweating, pale face, parched lips and tongue, palpitation, irritation, few and yellow urine, cold limbs and weak but rapid pulse. It would be best to quickly replenish blood, strengthen the mind, strengthen qi, fortify the body against loss of body composition, and consume shengmai san. Prescription: 15 g ginseng, 15 g *Ophiopogon japonicus* and 6 g *Schisandra chinensis*. (2) Weak qi and loss of yang. Blood loss from trauma, depletion of yin liquid, loss of yang, yin too weak for yang, yang energy leak, extension of yin damage to yang, decline of yang energy, and then loss and depletion. Symptoms include indifference, closed eyes but opened mouth, cold hands and feet, lack of heat, fear of coldness, icy cold body, few but frequent urination, lack of color and luster on face, lack of color on lips, cold sweat and pulse weakened to the point of disappearance. It would be best to quickly strengthen *qi*, restore deficiency, restore *yang* energy, rescue reversal, and consume shenfu soup or ginseng sini soup. Prescription: 15–30 g ginseng radix rubra, 30 g zhifupian, 30 g dried ginger, 9 g radix glycyrrhizae preparata and 9 g cortex cinnamomi. (3) Loss of both yin and yang. Blood

loss from trauma, yang energy leak, and loss of yin and yang energy to point of disappearance. Symptoms include delirium, sedated eye movement and opened mouth, dilated pupils, wheezing due to phlegm in throat, lack of breath and tiring breathing, profuse and oily sweat, tongue capsule shrinkage, icy cold body and limbs, lack of control of excretion and pulse weakened to the point of disappearance. It would be best to quickly restore yang energy and rescue yin energy, and consume sini soup coupled with shengmai san. Prescription: 15 g zhifupian, 12 g dried ginger, 10 g radix glycyrrhizae preparata, 9 g cinnamon (consume later), 15–70 g ginseng radix rubra, 15 g *Ophiopogon japonicus* and 9 g *Schisandra chinensis*.

Syncope

(1) Blood stasis and qi stagnation: Blood loss from trauma and damage to yin energy, injury to blood and damage to qi, blood stasis and qi stagnation, closure and obstruction or meridians and vessels, and disorders of qi. Symptoms include unconsciousness, swelling at site of injury, bluish and purplish bruises, lack of luster on face, clenched jaw, bruises on tongue and rapid and powerful pulse. It would be best to promote blood circulation in order to remove blood stasis, and to relieve pain while enlivening the spirit. Have the patient consume fuyuan huoxue tang and zhuyu huxinsan (empirical formula). Prescription: 3 g cinnabar (mix w/ water), 9 g amber powder (mix w/ water), 9 g each of dried resin of *Commiphora myrrha* Engl. and resin of *Boswellia carterii* Birdw. or *Boswellia bhaurdajiana* Birdw., 3 g notoginseng root powder (mix w/ water), 3 g Moschus (mix w/ water), 6 g Chinese thorowax root, 12 g *Angelica sinensis*, 15 g *Carthamus tinctorius*, 10 g *Zanthoxylum schinifolium*, 12 g Chinese rhubarb, 9 g peach seed, 10 g *Acorus calamus* and 6 g licorice root. (2) Phlegm, wetness and internal blockage: Untainted body with excessive phlegm, blood loss due to trauma, blood injury damages qi, qi reversal and wet phlegm and internal blockage. Symptoms include unconsciousness, inability to recognize people, quietness and lack of irritation, sounds of phlegm in the throat, occasional nausea, clenched jaw, eyes shut, clenched fists and alternating rapid and slow pulse. It would be best to disperse phlegm and clear away phlegm, open up and invigorate the mind, and consume ditang soup. Prescription: 15 g ginseng, 12 g *Arisaema heterophyllum* Blume, 12 g *Pinellia ternata* (Thunb.) Breit., 9 g dried fruit of *Citrus aurantium* L. or *Citrus sinensis Osbeck*, 20 g *Poria cocos* (Schw.) Wolf, 15 g dried tangerine or orange peel, 15 g *Acorus calamus*, 10 g dried middle layer of stem of *Bambusa tuldoides* Munro, *Sinocalamus beecheyanus (Munro) McClure* var. *pubescens P.F. Li* or *Sinocalamus beecheyanus (Munro) McClure* var. *pubescens P.F. Li*, 6 g licorice root and 3 g raw ginger. (3) Phlegm heat rising attack: Trauma and blunt trauma, chaotic *qi*, dispersed blood, mutual combination of phlegm heat, rise of toxic heat, closed eyes and clear mind. Symptoms include unconsciousness, irritation and unease, rapid breathing, wheezing due to phlegm in throat, clenched jaw, reddened, swollen and painful injury site, long pulse and red tongue with yellow coating. It would be best to clear heat, disperse phlegm, open up and invigorate the mind, and consume longdan xiehan soup with phlegm soup. Prescription: 15 g dried root of *Gastrodia elata* Bl., 30 g *Uncaria rhynchophylla* (Miq.) Miq.ex Havil, *Uncaria macrophylla* Wall., *Uncaria hirsuta* Havil., *Uncaria sinensis* (Oliv.) Havil. or *Uncaria sessilifructus* Roxb. (consume later), 20 g *Concha haliotidis*, 10 g dried immature fruits of *Citrus aurantium* L., 15 g *Radix scutellariae*, 10 g Chinese thorowax root, 30 g dried mature seeds of *Plantago asiatica* L. or Plantagodepressa Willd. (boiled within its cloth bag packaging), 12 g *Pinellia ternata* (Thunb.) Breit., 6 g dried fruit peel of *Citrus reticulata* Blanco, 12 g *Arisaema heterophyllum* Blume, 15 g *Acorus calamus*, 12 g *Angelica sinensis* and 6 g *Radix glycyrrhizae preparata.*

Acupuncture Treatment

Insert needles into acu-points such as Renzhong, Baihui, Quchi, Sanyinjiao, Qihai and Dadun.

In light of the above, treating and rectifying shock is a key step. Rescue practice shows that many victims die in the early stage due to shock. Traditional Chinese medicine is no doubt highly effective in dealing with various types of serious clinical symptoms arising from mining gas blast injuries, but TCM techniques and medications have not been widely adopted in the past, and still remains so in present day. The information above is stated with the hope of providing a clinical reference for another set of effective methods in treating mining gas blast injuries.

4.2 Treatment of CO Poisoning

1. Provide hyperbaric oxygen therapy, or supplement with fresh blood transfusion based on necessity of patient conditions.
2. Dehydration and diuretic therapy, hibernation and temperature reduction, and sedative therapy. Use 2% mannitol to induce dehydration, delivered through either direct injection or rapid drip in batches. Administer 250 mL or up to 400 mL in first dose, and at least 500 mL and as much as 1000 mL has to be delivered in the first 24 h. An additional option is to administer 25–50% glucose mixed with vitamins, delivered through IV drip between treatment sessions. However, pay attention to possibility of electrolyte imbalance, since dehydration to lower brain edema may lead to massive loss of electrolyte, so remember to maintain electrolyte balance. Hibernation and application of sedative drugs may also be employed to reduce oxygen consumption in the brain.

3. Hyperbaric oxygen chamber treatment.
4. Supplemental treatment. Make appropriate use of hormones to lessen the body's emergency response, administer brain metabolism drugs to protect brain function, as well as zhongyao xingnaojing, angong niuhuang pills, and so on.

4.3 Treatment of Injuries of Respiratory Tract

In terms of practical experiences in rescue, respiratory tract injury is one of the chief causes of death, and tracheotomy and vaporized medicine administration should be carried out promptly, and ensure timely dilution and elimination of secretions, so as to keep the respiratory tract unobstructed and uninfected.

4.4 Treatment of Skin Burn

1. Debridement. Under painless condition, shave away hair around the surface of burn, clean the skin of debris, use soap water to rinse the wound surface and surrounding region, remove any grease and oil, then use sterilized normal saline to rinse and scientifically disinfect the wound surface. Keep blisters from second-degree burns exposed, for larger blisters first disinfect, then puncture them and drain them by positioning them lower. Also, cut away shrunken epidermis and exfoliated necrotic tissue.
2. Dressed treatment
3. Exposed treatment. These two types of treatment have their respective pros and cons, so choose based on actual patient conditions. Select conventional treatments and therapies for burns.

4.5 Treatment of Combined Injuries

Most mining gas blast injuries are composite injuries, and after promptly and accurately judging injury condition, carry out corresponding treatments for multiple injuries such as craniocerebral injury, injuries to organs in the chest and abdomen, and various types of bone fractures. Categorize patients into different tiers based on injury severity, focus on main treatment dilemmas, select most opportune windows for treatment, and do not miss out on best time to save patient lives.

According to statistics, under the joint efforts of professional medical personnel in China's coal mine hospitals of various levels, present rate successful rescue in coal mine accidents and incidents stands above 98%, a figure among the top echelon in the global medical care community.

5 Section Five: Establishment of Coal Mine Medical Emergency and Rescue System in China

5.1 Background of the Chinese Mining Industry

Mining industry is one of the foundations of the Chinese economy, a cornerstone for sustainable development of society, and an integral part of a harmonious society. China is a major mineral resource producer, and at present the mining industry is undergoing a boom. As of the end of 2014, there is a total of 12,000 coal mines that employ a force of more than six million and produce over 3.87 billion tons of coal, a surge of nearly 2.7-fold compared with the 1.4 billion tons in 2002. There are nearly 100,000 non-coal mines that provide employment for over four million. Mineral ore total output approaches the 10 billion ton-level, and the figure is expanding by more than 10% on an annual basis. In the short term, the mining industry is anticipated to maintain its growth in its proportion of the Chinese industrial structure. The expedited growth of the industry and its inherently hazardous nature bring about tremendous pressure on safe production, and mine disasters and accidents are not uncommon.

The CPC Central Committee and State Council of China consider safe product a top priority, and have continued to strengthen work safety management including the issuance of safe production laws, closure of small coal mines and the implementation of a series of other measures, resulting in continual decline in mining disasters. According to statistic information of the State Administration of Work Safety, ten extra-major mining accidents with one hundred or more casualties each occurred between 2003 and 2008, resulting in 1663 total casualties and many more injured. Sixteen extra-major mining accidents with one hundred or more deaths each occurred between 2009 and 2014, and in 2014 coal mine disaster casualty fell below the thousand persons-mark for the first time, a plunge of 85.7% compared with 2002.

5.2 History and Status Quo of Mine Medical and Rescue System in China

Looking back, the mines of the old China were controlled by capitalists and foreign imperial powers, and the lives of miners were not considered within the scope of work safety. Disease, workplace injury, occupational illness and mining accident led to the deaths of many miners. The "ten thousand persons-craters" in Hebei, Liaoning, Heilongjiang and other areas are sufficient evidences of the dangerous situations that miners had to deal with back then. Mines in those days did

not have hospitals in their surrounding regions, and the few clinics that did exist existed to serve capitalism instead of serving endangered mine workers.

After the founding of New China, economy progressed from a focus on agriculture to an emphasis on modernized industry. Industrial development far exceeded social development, and as such, mines came before society. In other words, many mines appeared from square one, and production came before social life. While mines were developed, social institutions were established, with enterprises taking the lead in building and running hospitals and schools. That was the construction and development orientation at the time. Medical care at mines during the time fell within the scope of corporate mine planning and construction, resulting in rather technically capable mine hospitals founded to serve employees, to serve local folks and to serve households in the mining areas. At the time, work productivity was low, work safety assurance were not sound, and workplace accidents occurred frequently. In light of such problems, China established a mining accident medical care model led by the former Ministry of Coal Industry, founded upon enterprises, and implemented jointly by mine rescue teams and mine hospitals, which yielded rather impressive outcomes. The absolute majority of mines in China at the time complied with requirements of setting up health stations at the entry point of mine shafts, which form the basic unit in miner health care and emergency rescue. In terms of work safety and emergency rescue, gradual success was achieved in exploring and implementing a mining accident medical response system based on coal mining company hospitals. This system reaches further into mines, mine shaft entry point and even accident sites downhole. Such gradual development facilitated timely, on-site and immediate treatment of mining accident victims, crucial to buying more time for subsequent care in hospital. This system ended after the Ministry of Coal Industry was shut down in 1998. These changes are mostly the outcomes of reforms of coal mining company hospitals being removed from their social functions.

China's recognition of work safety deepened in 2002, as manifested in the founding of the State Administration of Work Safety, and the restoration of past efforts in developing emergency rescue system. Under this new direction, mine hospital and rescue inherited traditional methods while establishing new system. Strengthening and improving mine medical assistance system became a key part of mine rescue system development. In accordance with the principles of tiered responsibility system, consolidated resources and combination of peacetime and wartime methods, and by relying on the former Coal General Hospital (Beijing), the national "Mine Hospital Rescue Center" was founded. Based on the distribution of national coal mining companies, coal mine corporate medical care institutions, mineshaft entry medical station and local medical care institutions, and by relying on large-scale coal company hospitals, national- and provincial-level mine rescue bases and set up by some local hospitals, provincial-level medical care backbone teams, and corporate medical rescue teams, a mining disaster rescue network was established, which has achieved a certain degree of success in rescue endeavors.

The Emergency Response Command Center of the State Administration of Work Safety took the lead and partnered with local governments to jointly found 21 mine rescue teams. At present, there are seven national-level mine rescue teams and 14 regional-level mine rescue teams, and each team follows a model in which four teams forms a group, among other mechanisms, to create medical care squadrons. They have so far rescued more than 10,000 persons. Such efforts connect together mine medical care institutions throughout China, and this joining of forces has been pivotal in providing rescue and medical care in extra-major mining accidents and disasters, especially those involving large groups of casualties and injured personnel.

5.3 Chinese National-Level Efforts in Building Mine Medical Assistance System

Since the Chinese economic reform and opening-up, the economy of China has expanded by a rapid pace, and also rising in tandem is demand for energy. Coal constitutes 70% of energy consumed in China, and demands for this resource skyrocketed. Statistics show that China is the undisputed number one coal producer in the world, and output level has far exceeded that of the world's number two producer, the United States. Yet, as Chinese coal product speedily increases, mining accidents also occurred in high frequency in China due to a multitude of factors such as predominance of downhole mining, and insufficiency in management level, worker professionalism and safety equipment. Since 2004, mining accidents involving more than a hundred casualties, such as disasters in Zhengzhou of Henan Province, Tongchuan of Shaanxi Province and Fuxin of Liaoning Province, have not been uncommon. These shocking catastrophes even prompted the International Labor Organization to speak out against importing "blood-stained coal" of China. Aiming for the strategic goal of safe development, harmonious development and fully coordinated and sustainable development, the CPC Central Committee and State Council of China elevated the priority of work safety at coal mines, and mandated different levels of the Party Committee and government to employ substantive and effective measures in different phases to reduce mining accidents, and strengthen construction of mining accident emergency rescue system. Looking at both domestic and international practices, coal mine accident emergency rescue includes two major aspects. One aspect is emergency response and rescue on-site at the mining accident, and the other is urgency response and rescue at hospital. Therefore, in 2002

the Mine Hospital Rescue Center of the State Administration of Work Safety was founded to take charge of medical assistance of coal mine accidents. The center is headquartered at the Coal General Hospital (present day Emergency General Hospital), under which are 42 provincial-level sub-centers. After 6 years of operation, it has been decided at the state-level to establish and develop the Chinese mine medical assistance system from the three aspects of organization system, objective system and management system.

5.3.1 Development of Organization System

According to state regulations, the functions of Chinese coal mine medical assistance centers are to consolidate coal mine hospital and health-care resources, make use of organizational and other support and funding from the government, and achieve the functions of coal mine medical assistance functions as stipulated by the national government. After the founding of New China, the CPC and government have taken a genuine interest in the physical and emotional health of coal mine workers, and subsequently established various levels and various types of coal mine medical assistance institutions. According to statistics, there are some one thousand medical care organizations within the Chinese coal sector, and they employ around 150,000 personnel. For many years, they have been providing high-quality medical care services to the 5.5 million coal mine workers of China. During their extensive periods of coal mine medical assistance endeavors, they have accumulated advanced experiences in the matter, contributed tremendously to rescuing Chinese coal mine workers in mining disasters, and gained widespread commendations for playing a crucial role in fortifying the well-being of miners. Therefore, based on the actual circumstances of coal mines in China, certain resources have been rationally consolidated, and scientifically and meticulously organized. The national government has allotted appropriate supports for emergency rescue equipment and budget in the form of specialized funds. This is going to be the most scientific, most economical and most effective method. According to the status quo of coal mine medical care institutions in China at present, from the point of view of organization system development, it has been proposed that the key here is to strengthen the development of a pair of three-tiered models.

1. State Administration of Work Safety Mine Hospital Rescue Center—provincial-level mine medical assistance sub-centers—bureaus of mining general hospitals. The State Administration of Work Safety Mine Hospital Rescue Center is headquartered at the Coal General Hospital, and both these bodies are administered as one organization with two names. The entity's main function is to take charge of specific organization and command of coal mine accident medical emergency response and rescue tasks under the leadership of the State Administration of Work Safety. The provincial-level mine medical assistance sub-centers are respectively headquartered at local coal mine medical care institutions with excellent medical technical capacity, scale and compliant facilities, they are administered by the State Administration of Work Safety Mine Hospital Rescue Center, and are responsible for specific organization of coal mine medical and rescue tasks within their province. Based on mining enterprises distribution in China, all bureaus of mining have been outfitted with general hospitals or central hospitals, and these are put in charge of medical emergency response and rescue tasks within the territory of jurisdiction of their mining bureaus, and also accept directions and commands from provincial-level mine medical assistance sub-centers. The State Administration of Work Safety Mine Hospital Rescue Center—provincial-level mine medical assistance sub-centers—bureaus of mining general hospitals type of linear model is the main organizational structure of China's mine medical rescue system.
2. Bureaus of mining general hospitals—mine hospitals—mineshaft health stations. This model was established when bureaus of mining existed. The function of this model is to take charge of basic health-care tasks of miners, and to take charge of medical emergency response and rescue tasks during mining accidents. The bureaus of mining general hospitals—mine hospitals—mineshaft health stations model has become the main organizational structure for mine medical assistance at the basic level. As long as we focus on strengthening the development of these two models, the development of coal mine medical assistance organization system is assured. For this purpose, China has carried out broad-spectrum exchanges with developed countries such as the USA, and foreign parties have expressed emphatic praises about the effectiveness of this type of organizational structure in mining accident rescues in China.

5.3.2 Development of Objective System

From the perspective of management science, all organizations have objectives, and any organization without objective is ineffective. The objective of China's coal mine hospitals is constituted by two main sets of indices:

1. Emergency response index. Accidents in which 30 or more persons are injured or killed are classified as grade I emergency response, and medical emergency response and rescue efforts are undertaken with nationwide coal mine medical care resources as mobilized by the State Administration of Work Safety Mine Hospital Rescue Center (relevant medical care resources in other systems to be mobilized when necessary). Coal mine accidents in which 10–29 persons are injured or killed are classified as grade II emergency response, and emergency response and rescue efforts are undertaken with provincial coal

mine medical care resources as mobilized by provincial-level mine medical assistance sub-centers (relevant medical care resources in other systems in the province to be mobilized when necessary). Coal mine accidents in which less than nine persons are injured or killed are classified as grade III emergency response, and emergency response and rescue efforts are undertaken by general hospital of the competent mining bureau using medical resources within the jurisdiction of the responsible mining bureau. As per the principle of "exception to every rule" in management science, there are exceptions to emergency response as well. Certain special mining accidents with relatively large impact that exceed the conventional medical emergency scope are not restricted by protocols of the three-tier emergency response system.

2. Rescue success rate index. Since the majority of downhole coal miners possess basically sound health status, as long as a mining accident is not deadly, on-site treatments are appropriate, scientific treatment and evacuation upon lifting out of mine shaft are effective, usually lives may be preserved. In terms of mining accident rescue efforts elsewhere around the world, a representative approach is that of the USA, which emphasizes emergency rescue after admission to hospital. France meanwhile stresses on emergency rescue before hospital admission. In accordance with actual situations in China, both prehospital and in-hospital treatments are important, both aspects should be underscored, and the transition between the two phases should also be a point of focus. Looking at Chinese coal mine medical institution rescue statistics and indices for mining accident victims since the inception of New China, pegging rescue success rate at 95% is befitting for actual conditions. This standard is far higher than rescue success rate for critically injured or ill patients in regular medical institutions (70%). Coal mine medical organizations at various levels should be assessed against this benchmark each year, and incentive/punitive scheme should be employed in accordance with this assessment, so as to incrementally raise rescue success rate each year. Statistics show that in 2007 a total of 4081 miners were injured in coal mine accidents in China. Of which, 4001 survived, equating to a rescue success rate of 98%. Real-world evidence points out that establishing a set of scientific mine medical rescue objectives grounded in actual circumstances, evaluating the accomplishment of these objectives on an annual basis, and conducting closed evaluation supplemented with an incentive/punitive scheme, can gradually improve mining accident rescue success rate.

5.3.3 Development of Management System

Establishments of organization system and objective system only serve as framework for coal mine medical rescue system development, because the key to maximize this framework is management, which should be carried out according to the aspects outlined below:

1. Establish legal system. Formulate corresponding laws, regulations and rules at the state level, and ensure at the legal level that coal mine medical rescue efforts can be executed smoothly.
2. Establish mining accident rescue rules and regulations for specific coal mine medical care institutions. The goal is to ensure that each step in the coal mine accident medical rescue process is scientific and effective, and supervision and inspection for earnest implementation should be carried out frequently.
3. Establish authoritative experts groups and formulate and develop corresponding coal mine accident rescue preemptive plan. It is suggested to host different types of training, establish specific medical rescue teams and adopt an active approach in drilling and competition so as to earnestly enhance coal mine accident rescue performance standards; national-level centers should host nationwide symposiums once per year to facilitate exchanges and communications about coal mine accident medical rescue work, learn from each other, supplement each other's weaknesses, and augment everyone's capacity in a concerted manner; various provincial-level coal mine medical rescue sub-centers, as well as mining bureau general hospitals, mine hospitals, mineshaft health stations and coal mine rescue teams also need to frequently organize discussions and meetings about experiences and lessons learned from coal mine accident medical rescue works. In terms of mining medical rescue efforts elsewhere around the globe, the USA, Australia, Japan and Europe have accumulated some advanced experiences, while other nations with production capacity similar to China, such as Russia and India, have also developed a certain level of standards in coal mine medical rescue. Learning from their experiences and lessons can effectively enhance coal mine accident rescue success rate in China and reduce rate of disability in earnest.
4. Foster coal mine medical rescue and safety culture, and ensure success in the final and crucial step of mining accident rescue. Focusing on the concept of "ensuring safety of miners and safeguarding the final step," foster work safety culture in every aspect, and ensure that such culture pervades thoroughly within every part of relevant medical institutions.

In terms of coal mine medical rescue in China, organization development serves as the foundation, objective development serves as the direction, and management development serves as the key. Together these three aspects consist the coal mine medical rescue system of China.

Blast Injuries from Explosion of Chemicals

Aimin Luo

1 Section One: Chemicals and Hazardous Chemicals

1.1 Definition of Chemicals

The term "chemicals" here refer to various elements (as in chemical elements) and chemical mixtures comprised of elements, and "chemicals" here include those both natural and artificial.

Statistics in American chemicals periodicals indicate that there are some seven million kinds of chemicals in the world, of which more than 100,000 are sold on the market as merchandise, over 70,000 are used in daily life, and around 1000 new chemicals appear around the world each year. In the 2012 edition of *Global Chemicals Outlook*, it was pointed out that there are approximately 140,000 types of chemicals in the market at the time.

1.2 Hazardous Types of Chemicals

Chemicals in China are classified as per GHS, the *Globally Harmonized System of Classification and Labeling of Chemicals* of the United Nations. Establishing and implementing a chemical hazard class, labeling and safety data sheet system in line with the international community can facilitate chemical import and export convenience, and reduce trading costs in China. At the same time it helps prevent and lessen dangers of chemicals to people and the environment, and conduct innocuous treatment of chemicals and wastes.

The classification of chemicals is mostly based on GB 13690-2009 *General Rule for Classification and Hazard Communication of Chemicals* [1], GB20576-GB20599, GB20601 and GB20602 *Safety Rules for Classification, Precautionary Labeling and Precautionary Statements of Chemicals* [2], GB/T 17519 *Guidance on the Compilation of Safety Data Sheet for Chemical Products* and other national standards [3]. These standards contain technical contents identical to those in the *Globally Harmonized System of Classification and Labeling of Chemicals* (GHS) of the United Nations.

Hazard class of a chemical refers to the properties of its physical, chemical, health or environment hazards, such as flammable solid, carcinogenicity or acute oral toxicity.

1.2.1 Physical Hazards of Chemicals

There are 16 types of physical hazards of chemicals in total. A chemical's form of problem such as fire or explosion is related to its physical hazard characteristics.

1. **Explosives**. Chemicals that are explosives include the following.
   (a) Explosive substance (or mixture);
   (b) Explosive item, but not including the following devices: The explosive substance or mixture contained within, due to its quantity or nature, will not burst out, light up, smoke, heat up or generate large sounds or generate other effects outside of the device after accidental or random ignition or detonation.
   (c) Substance, mixture or item manufactured to produce an actual explosion or firecracker effect not mentioned in (1) or (2) above.

   An explosive substance (or compound) is a solid or liquid substance (or compound of such substances) that can produce air through chemical reaction, and the temperature, pressure and speed of the produced gas can cause destruction to the surrounding environment. These include pyrophoric substance even if they do not produce gas.

   A pyrophoric substance (or pyrophoric mixture) is a substance (or a mixture of such substances) that generate heat, light, sound, gas, smoke or any combinations of these effects through spontaneous chemical reaction and combustion without exploding.

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An explosive item is an item that contains one or more explosive substance or mixture.

Pyrophoric item is an item that contains one or more pyrophoric substance or pyrophoric mixture.

2. **Flammable gas.** A flammable gas is a gas that may be flammable with the air at the temperature of 20 °C and standard atmospheric pressure of 101.3 kPa.

   A chemically unstable gas is a combustible gas that may undergo explosive reaction in condition whereby there is a lack of air or oxygen.
3. **Flammable aerosol.** A flammable aerosol refers to a substance contained in an aerosol can, as in any container that may not be refilled. Such container may be made from metal, glass or plastic, and holds forcefully compressed, liquefied or dissolved gas that may or may not include liquid, paste or powder. Such container is also outfitted with a release mechanism that allows the gas to spray out as solid or liquid particles suspended in a gas, or as foam, paste or powder, or in a liquid or gaseous state.
4. **Oxidizing gas.** An oxidizing gas is a gas that, compared to air, allows other substance to burn more easily by means such as providing oxygen in most cases.
5. **Compressed gas.** A compressed gas refers to a gas injected into a storage container at a pressure equal to or greater than 200 kPa (gauge pressure), or liquefied gas or frozen gas. Compressed gases include gas under pressure, liquefied gas, dissolved liquid and frozen gas.
6. **Flammable liquid.** A flammable liquid refers to a liquid with flash point below 93 °C.
7. **Flammable solid.** A flammable solid is a solid that is combustible, or that may combust or support combustion easily or through friction.

   Flammable solids could take form as powder, particle or paste, and they may ignite readily upon short contact with fire source such as a lit match. After the fire spread quickly, such solids could create immense dangers and very dire consequences.
8. **Self-reactive substance.** A self-reactive substance is a thermally unstable liquid or solid substance, or mixture, prone to violent exothermic decomposition even without oxygen (air). Based on uniform classification system, this definition divides substances into explosive substance, organic peroxide or oxidizing substance, or mixtures of such substances.

   If a self-reactive substance or its components can easily combust, deflagrates in laboratory experiment, or shows violent effects when heated in closed condition, said substance should be deemed to possess explosive property.
9. **Pyrophoric liquid.** A pyrophoric liquid is a liquid that can spontaneously ignite even in small quantity within 5 min after contact with air.
10. **Pyrophoric solid.** A pyrophoric solid is a solid that can spontaneously ignite even in small quantity within 5 min after contact with air.
11. **Self-heating substance.** A self-heating substance is a solid or liquid substance, or compounds of such substances, other than pyrophoric liquid or pyrophoric solid, that can react with air and heat up on its own without any other source of energy. This kind of substances or compounds differ from pyrophoric liquid or pyrophoric solid because they only combust after an extensive period (several hours or several days) and only when the mass is great (kg-level).

    It should be noted that spontaneous combustion resulting from the self-heating property of such substances or mixtures is the outcome of reaction between air and the substance or mixture, and the produced heat is not transmitted to an external domain sufficiently quickly. When the rate of heat production exceeds the rate of heat consumption and reaches spontaneous combustion temperature, spontaneous combustion would occur.
12. **Substance that releases flammable gases when in contact with water.** A substance that releases flammable gases when in contact with water is a solid or liquid substance, or mixtures of such substances, that can easily release spontaneous combustible gases or a dangerous quantity of flammable gases upon reacting with water.
13. **Oxidizing liquid.** An oxidizing liquid is a liquid that may not be combustible on its own, but could ignite or promote other substances to combust by releasing oxygen.
14. **Oxidizing solid.** An oxidizing solid is a solid that may not be combustible on its own, but could ignite or promote other substances to combust by releasing oxygen.
15. **Organic peroxide.** An organic peroxide is an organic substance in liquid or solid form that contains bivalent -O-O- structure, and may be considered a hydrogen peroxide derivative in which one or two hydrogen atoms have been replaced by an organic group. This jargon also includes organic peroxide formulas (mixtures). Organic peroxides are thermally unstable substances or mixtures that may easily undergo exothermic self-accelerating decomposition. In addition, they may possess one or more of the following properties: (1) being prone to explosive decomposition, (2) rapidly combustible, (3) being sensitive to collision or friction and (4) being prone to react dangerously with other substances.

    If organic peroxide's component can easily explode, rapidly deflagrates, or shows violent effects when heated in closed condition in laboratory experiment, said substance should be deemed to possess explosive property.

16. **Mordant.** A mordant is a substance or mixture that uses chemical effects to obviously damage or destroy metal substance or mixture.

1.2.2 Health Hazards

There are ten types of health risks associated with chemicals.

1. **Acute toxicity.** Acute toxicity refers to a substance that may produce harmful effects within a single dose, or within 24 h after oral ingestion or skin contact with multiple doses, or 4 h after inhalation.
2. **Dermal corrosion/irritation.** Dermal corrosion refers to irreversible damage of the skin, as in observable epidermis and dermis necrosis within 4 h after application of the said test substance.
3. **Severe eye damage/irritation.** Severe eye damage refers to incompletely reversible tissue damage within 21 days after application of said test substance to the surface of the eye, or severe physical visual ability decline.
4. **Respiratory or skin allergies.** Respiratory allergy refers to the ability of a substance to cause allergic reaction in the respiratory tract upon inhalation. Skin allergy refers to the ability of a substance to cause allergic reaction on the skin upon contact.
5. **Germ cell mutagenicity.** Germ cell mutagenicity refers to the ability of a substance to potentially cause mutations in human germ cells that may be passed on to offspring.
6. **Carcinogenicity.** Carcinogenicity refers to the ability of a chemical substance or chemical mixture to cause cancer or induce an increased probability of cancer occurrence.
7. **Reproductive toxicity.** Reproductive toxicity refers to harmful effects on sexual function and fertility in adult males and females, as well as developmental toxicity in offspring.
8. **Specific target organ systemic toxicity: single exposure.** A substance with this property can produce specific, nonfatal toxicity of target organs from a single exposure.
9. **Specific target organ systemic toxicity: repeated exposure.** Substances categorized under this category can produce toxicity of target organs upon repeated exposure. All health impacts including those that may harm bodily functions, reversible or irreversible, instantaneous or delayed, are included.
10. **Inhalation hazard.** "Inhalation" here refers to a liquid or solid chemical substance that may directly enter the body through the oral cavity or nasal cavity, or that may enter the airway and lower respiratory tract during vomiting. Inhalation toxicity includes chemical pneumonia, different degrees of pulmonary damage (or death upon inhalation) and other severe and acute effects.

1.2.3 Environment Hazards

There are two types of environment hazards in chemicals.

1. **Hazard to aquatic environment.** Acute water toxicity refers to a property of a substance that can cause harm to organism exposed to said substance within a short time span.
2. **Chronic water toxicity.** Chronic water toxicity data are not as easily obtained than those for acute water toxicity, and experiment processes and scopes have not yet been standardized.

1.3 Hazardous Chemicals

1.3.1 Definition of Hazardous Chemical

As of March 2003, some 3823 items were listed as hazardous chemicals in accordance with the *Hazardous Chemicals Safety Administration Regulation* (State Council decree No. 344) and the former State Administration of Work Safety's *List of Hazardous Chemicals* (2002 edition) (former State Administration of Work Safety 2003 public notification No. 1). In June 2003, *List of Highly Toxic Chemicals* (2002 edition) (former State Administration of Work Safety 2003 public notification No. 2 jointly issued by eight organs) included 335 highly toxic chemicals. Based on the *Globally Harmonized System of Classification and Labeling of Chemicals* (GHS) of the United Nations, China formulated rules and standards for classifying and labeling chemicals according to hazard class, and devised a chemical hazardous level system with 28 categories. Since the *List of Hazardous Chemicals* (2002 edition) is mainly a dangerous goods labeling and classification system for items like explosive, flammable liquid and corrosive substance, it differs drastically with the current hazardous chemicals classification system featuring 28 categories. The current *Hazardous Chemicals Safety Administration Regulation* (State Council 2011 decree No. 591, State Council 2013 decree amendment No. 645) amended the definition of hazardous chemical, stipulating that "a hazardous chemical refers to highly toxic chemicals and other chemicals that are toxic, corrosive, explosive, flammable, combustion-supporting or characterized by other dangerous properties, and may harm people, facilities and the environment." At the same time, the *List of Highly Toxic Chemicals* (2002 edition) includes many varieties and do not really fit actual applications of the administration of highly toxic chemicals, requiring amendments.

1.3.2 List of Hazardous Chemicals

Chinese administration of hazardous chemicals adopts a list-based administration system, and any hazardous chemical named on the *List of Hazardous Chemicals* shall be administered through measures such as administrative permits in accordance with relevant laws and regulations of the country.

According to Article 3 of the *Hazardous Chemicals Safety Administration Regulation* (State Council decree No. 344): "The list of hazardous chemicals is confirmed, published, and amended in a timely manner, by the State Council's work safety supervision department in collaboration with the State Council's competent departments in charge of industry and informatization, public security, environment protection, health and sanitation, quality supervision, inspection and quarantine, transportation, railway, civil aviation and agriculture."

Based on the national standards on chemical classification and labeling, 81 of the more hazard classes out of the 91 hazard classes in the 28 chemical categories are used as principles for determination of hazardous chemicals (Table 1), which in turn forms China's *List of Hazardous Chemicals* (2015 edition). The *List of Hazardous Chemicals* (2015 edition) was jointly published by ten departments including the former State Administration of Work Safety (2015 No. 15). On this list are 2828 items.

Toxic chemicals are chemicals with potent, highly toxic harms, and include artificially synthesized chemicals and their mixtures, and natural toxins, as well as chemicals with acute toxicity that may easily cause harm to public safety.

Table 1 Hazardous chemical determination principles

Hazard and hazard class		Categories						
Physical hazard	Explosive	Unstable explosive	1.1	1.2	1.3	1.4	1.5	1.6
	Flammable gas	1	2	A (Chemically unstable gas)	B (Chemically unstable gas)			
	Aerosol	1	2	3				
	Oxidizing gas	1						
	Pressured gas	Compressed gas	Liquefied gas	Frozen gas	Dissolved gas			
	Flammable liquid	1	2	3	4			
	Flammable solid	1	2					
	Self-reacting substance and mixture	A	B	C	D	E	F	G
	Self-heating substance and mixture	1	2					
	Pyrophoric liquid	1						
	Pyrophoric solid	1						
	Substance or mixture that releases flammable gases when in contact with water	1	2	3				
	Metal corrosive	1						
	Oxidizing liquid	1	2	3				
	Oxidizing solid	1	2	3				
	Organic peroxide	A	B	C	D	E	F	G
Health hazard	Acute toxicity	1	2	3	4	5		
	Dermal corrosion/irritation	1A	1B	1C	2	3		
	Severe eye damage/irritation	1	2A	2B				
	Respiratory or skin allergy	Respiratory allergen 1A	Respiratory allergen 1B	Skin allergen 1A	Skin allergen 1B			
	Germ cell mutagenicity	1A	1B	2				
	Carcinogenicity	1A	1B	2				
	Reproductive toxicity	1A	1B	2	Additional type (breast-feeding effect)			
	Specific target organ systemic toxicity - single exposure	1	2	3				
	Specific target organ systemic toxicity - repeated exposure	1	2					
	Inhalation hazard	1	2					
Environment hazard	Aquatic environment hazard	Acute 1	Acute 2	Acute 3	Chronic 1	Chronic 2	Chronic 3	Chronic 4
	Ozone hazard	1						

Those with dark backgrounds are categories used as determination principle for hazardous chemicals

Table 2 Comparison of changes in bounds of determination of high toxicity

Item	List of highly toxic chemicals (2015 edition)	List of highly toxic chemicals (2002 edition)
Peroral	$LD_{50} \leq 5$ mg/kg	$LD_{50} \leq 50$ mg/kg
Dermal	$LD_{50} \leq 50$ mg/kg	$LD_{50} \leq 200$ mg/kg
Inhaled	(4 h) $LC_{50} \leq 100$ mL/m^3 (gas), or 0.5 mg/L (steam), or 0.05 mg/L (dust or mist)	(4 h) $LC_{50} \leq 500$ mL/m^3 (gas), or 2 mg/L (steam), or 0.5 mg/L (dust or mist)
Corresponding hazard categories	Acute toxicity, type 1	Acute toxicity, type 1 and type 2

Compared with the *List of Highly Toxic Chemicals* (2002 edition), bounds of definition of high toxicity varies quite significantly (Table 2).

As new chemicals continue to appear, and as people become more aware of the dangers of chemicals, as per relevant regulations in Article 3 of the *Hazardous Chemicals Safety Administration Regulation* (State Council 2011 decree No. 591), a chemical not listed on the *List of Hazardous Chemicals* (2015 edition) does not mean that it does not fit the determination principle of a hazardous chemical.

To maintain a comprehensive grasp of the hazards of hazardous chemicals in the nation, China is implementing a hazardous chemical registration system, and identification and categorization system for mixtures and hazardous chemicals not listed on the *List of Hazardous Chemicals* (2015 edition). Enterprises shall identify and categorize chemicals in accordance with the *Chemical Hazard Identification and Categorization Administration Regulation* (State Administration of Work Safety decree No. 60) and other relevant regulations. If a chemical has been identified as a category of hazardous chemical, the *Hazardous Chemical Registration Administration Regulation* (State Administration of Work Safety decree No. 53) shall be followed, said hazardous chemical shall be properly registered, and said chemical's hazard shall be understood from the source, so as to ensure the safe usage of said hazardous chemical. Full coverage of safe administration of hazardous chemical is achieved through a combination of list administration, and identification and categorization administration methods.

1.4 Other Relevant Flammable and Explosive Items in China

Article 112 of the *Work Safety Law* (2014 chairperson decree No. 13) stipulates the definition of hazardous good: A hazardous good refers to flammable good, explosive item good, hazardous chemical, radioactive good and other items that may threaten health and property. Clearly, within the scope of the *Work Safety Law*, flammable good, explosive item good and hazardous chemical overlap in certain ways.

Chemicals in China are classified as per GHS, the *Globally Harmonized System of Classification and Labeling of Chemicals* of the United Nations, and a chemical hazard class, labeling and safety data sheet system in line with the international community has been established and continues to be implemented, while chemicals categorization is mostly based on national standards like the GB 13690-2009 *General Rule For Classification And Hazard Communication Of Chemical*. For example, types of explosives include explosive substance (or mixture), explosive good, and substance, mixture or item manufactured to produce an actual explosion or firecracker effect (as in pyrophoric item). However, in relevant regulations and standards system regarding hazardous goods in China, fireworks, firecrackers, explosives for civil use and other flammables and explosives have not yet been included in the ranks of hazardous chemicals, but rather independently categorized and administered.

1.4.1 Explosives for Civil Use

To strengthen safety and administration of explosives for civil use, prevent the occurrence of accidental explosions, and safeguard the lives, property of the people and public safety, the 134th executive meeting of the State Council held on April 26th, 2006, deliberated on and passed the *Administration Regulation on Safety of Explosives for Civil Use* (State Council decree No. 466), effective starting from September 1st, 2006. The 54th executive meeting of the State Council held on July 29th, 2014, deliberated on and passed the *Decision to Amend Certain Administration Regulations* (State Council decree No. 653), which amended the aforesaid regulation.

In Article 2 of this regulation, explosives for civil use are defined as any type of gunpowder, explosive and their product, as well as detonator, fuses and other ignition and detonation device for nonmilitary use and named on the list of explosives for civil use. The list of explosives for civil use was compiled and published jointly by the State Council's organs in charge of the explosive goods industry and public security. Obviously, the administration of explosives for civil use in China adopts a list-based mode of administration.

The *List of Explosives for Civil Use* (Commission on Science, Technology and Industry for National Defense of the People's Republic of China and Ministry of Public Security of the People's Republic of China 2006 public notification No. 1), shows a total of 59 types of explosives for civil use, including 27 types of industrial explosives, ten types of industrial detonators, five types of industrial cord-type explosive devices, five types of other explosives for civil use, and 12 types of raw materials (Table 3).

Table 3 List of explosives for civil use

No.	Name	English name	Note
I.	Industrial explosives		
1	Nitroglycerine	Nitroglycerine, NG	Glyceryl trinitrate explosive mixture
2	Ammonite	Ammonite	Explosive containing ammonite
3	Porous granular ANFO explosive		
4	Modified ANFO		
5	Expanded ammonium nitrate explosive	Expanded AN explosive	
6	Other types of ANFO explosives		Powdery ANFO, ammonium slackwax, ammonium nitrate–asphalt–paraffin explosives, and so on
7	Water gel explosive	Water gel explosive	
8	Emulsion	Emulsion	
9	Powdery emulsive	Powdery emulsive	
10	Emulsified granular ANFO explosive		Heavy ANFO explosive
11	Viscous explosive		
12	Explosive containing decommissioned gun powder		Emulsified, pasty or powdery explosive containing decommissioned gun powder
13	Other industrial explosive		
14	Seismic charge	Seismic charge	
15	Seismic explosive		
16	Artificial weather modification explosives		Includes artillery rounds, rockets, and so on, and restrictions on production, purchase, sales and transportation administration
17	Rock crushing equipment		
18	Primer	Primer	
19	Explosive processing equipment		
20	Primers for oil and gas wells		
21	Perforating charge	Perforating charge	
22	Perforator	Perforator	
23	Shaped cutting charge		
24	High-energy gas fracturing charge		
25	Ignition pack		
26	Other oil and gas well blasting equipment		
27	Other explosive goods		
II	Industrial detonator		
28	Flash detonator	Flash detonator	
29	Electric detonator	Electric detonator	Includes regular electric blasting cap, and electric blasting cap permitted for coal mine
30	Detonator with shock-conducting tube	Detonator with shock-conducting tube	
31	Semiconductor bridge electric detonator		
32	Electron-delay detonator	Electron-delay detonator	
33	Magnetoelectric detonator	Magnetoelectric detonator	
34	Electric detonator for oil and gas well		
35	Electric detonator for seismology		
36	Detonating relay		
37	Other industrial detonators		
III.	Industrial cord-type explosive devices		
38	Industrial blasting fuse	Industrial blasting fuse	
39	Industrial detonating fuse	Industrial Detonating fuse	
40	Linear shaped charge	Linear shaped charge	
41	Shock-conducting tube	Shock-conducting tube	

Table 3 (continued)

No.	Name	English name	Note
42	Firing cable		
IV.	Other explosives for civil use		
43	Ignition gear for airbag		
44	Other ignition devices for special purposes		
45	Pyrotechnic items for special purposes		
46	Other ignition devices		
47	Signal flares for maritime rescue		
V.	Raw materials		
48	Trinitrotoluene, TNT	Trinitrotoluene, TNT	Restrictions on purchase, sales and transportation administration
49	Hexogen, RDX	Hexogen, RDX	Restrictions on purchase, sales and transportation administration
50	Picric acid	Picric acid	Restrictions on purchase, sales and transportation administration
51	Propellant for civil use		Restrictions on purchase, sales and transportation administration
52	Pentaerythritol tetranitrate, PETN	Pentaerythritol tetranitrate, PETN	Restrictions on purchase, sales and transportation administration
53	Octogen, HMX	Octogen, HMX	Restrictions on purchase, sales and transportation administration
54	Explosive compound	Explosive compound	Restrictions on purchase, sales and transportation administration
55	Black powder	Black powder	Except for black powder used in production of fireworks and firecrackers, and restrictions on purchase, sales and transportation administration
56	Initiating explosive	Initiating explosive	
57	Delay device		
58	Ammonium nitrate, AN	Ammonium nitrate, AN	Restrictions on purchase and sales approval administration
59	Other explosives for civil use deemed to require administration by the Commission on Science, Technology and Industry for National and the Ministry of Public Security.		

1.4.2 Fireworks and Firecrackers

To strengthen safety and administration of fireworks and firecrackers, prevent the occurrence of accidental explosions, and safeguard the lives, property of the people and public safety, the 121st executive meeting of the State Council held on January 11th, 2006, deliberated on and passed the Administration Regulation on Safety of Fireworks and Firecrackers (State Council decree No. 455).

Article 2 of this regulation, fireworks and firecrackers are defined as firework and firecracker products, and black powder, smoke and fire agents, firing cables and other civil-use items for the production of fireworks and firecrackers. The types, categories, grades, specifications, quality, packaging and labeling of fireworks and firecrackers can be found in *Fireworks and Firecracker Safety and Quality* (GB10631) and the regulations of other national standards and industrial standards.

1.4.3 Easily Produced Explosive and Hazardous Chemicals

According to the *Administration Regulation on Safety of Hazardous Chemicals* (State Council decree No. 591). The Ministry of Public Security compiled the *List of Easily Produced Explosive and Hazardous Chemicals* (2011 edition), and then in 2017 the Ministry of Public Security released a new version of the *List of Easily Produced Explosive and Hazardous Chemicals* (2017 edition). Easily produced explosive and hazardous chemicals are administered using a list-based administration method, and the list includes 74 types in nine categories. Please see Table 4 for details.

Table 4 List of easily produced explosive and hazardous chemicals (2017 edition)

No.	Item name	Alias	CAS number	Main flammable and explosive hazard categories
1. Acids				
1.1	Nitric acid		7697-37-2	Oxidizing liquid, type 3
1.2	Fuming nitric acid		52583-42-3	Oxidizing liquid, type 1
1.3	Perchloric acid [concentration > 72%]	Perchloric acid	7601-90-3	Oxidizing liquid, type 1
	Perchloric acid [concentration 50–72%]			Oxidizing liquid, type 1
	Perchloric acid [concentration ≤50%]			Oxidizing liquid, type 2
2 Nitrates				
2.1	Sodium nitrate		7631-99-4	Oxidizing solid, type 3
2.2	Potassium nitrate		7757-79-1	Oxidizing solid, type 3
2.3	Cesium nitrate		7789-18-6	Oxidizing solid, type 3
2.4	Magnesium nitrate		10377-60-3	Oxidizing solid, type 3
2.5	Calcium nitrate		10124-37-5	Oxidizing solid, type 3
2.6	Strontium nitrate		10042-76-9	Oxidizing solid, type 3
2.7	Barium nitrate		10022-31-8	Oxidizing solid, type 2
2.8	Nickel nitrate	Nickel (II) nitrate	13138-45-9	Oxidizing solid, type 2
2.9	Silver nitrate		7761-88-8	Oxidizing solid, type 2
2.10	Zinc nitrate		7779-88-6	Oxidizing solid, type 2
2.11	Lead nitrate		10099-74-8	Oxidizing solid, type 2
3 Chlorates				
3.1	Sodium chlorate		7775-09-9	Oxidizing solid, type 1
	Potassium chlorate solution			Oxidizing liquid, type 3
3.2	Sodium hypochlorite solution		3811-04-9	Oxidizing solid, type 1
	Potassium chlorate solution			Oxidizing liquid, type 3
3.3	Ammonium chlorate		10192-29-7	Explosive and unstable explosive
4 Perchlorates				
4.1	Lithium perchlorate	Lithium perchlorate	7791-03-9	Oxidizing solid, type 2
4.2	Sodium perchlorate	Sodium perchlorate	7601-89-0	Oxidizing solid, type 1
4.3	Potassium perchlorate	Potassium perchlorate	7778-74-7	Oxidizing solid, type 1
4.4	Ammonium perchlorate	Ammonium perchlorate	7790-98-9	Explosives, item 1.1
				Oxidizing solid, type 1
5 Dichromates				
5.1	Lithium dichromate		13843-81-7	Oxidizing solid, type 2
5.2	Sodium dichromate	Sodium bichromate	10588-01-9	Oxidizing solid, type 2
5.3	Potassium dichromate	Potassium dichromate	7778-50-9	Oxidizing solid, type 2
5.4	Ammonium dichromate	Dichromi	7789-09-5	Oxidizing solid, type 2
6 Peroxides and superoxides				
6.1	Hydrogen peroxide solution [content > 8%]	Hydrogen peroxide	7722-84-1	(1) content ≥60%
				Oxidizing liquid, type 1
				(2) 20% ≤ content < 60%
				Oxidizing liquid, type 2
				(3) 8% < content < 20%
				Oxidizing liquid, type 3
6.2	Lithium peroxide	Lithium oxide	12031-80-0	Oxidizing solid, type 2
6.3	Sodium peroxide	Sodium peroxide; sodium dioxide	1313-60-6	Oxidizing solid, type 1
6.4	Potassium peroxide	Potassium dioxide	17014-71-0	Oxidizing solid, type 1
6.5	Magnesium peroxide	Magnesium dioxide	1335-26-8	Oxidizing liquid, type 2
6.6	Calcium peroxide	Calcium dioxide	1305-79-9	Oxidizing solid, type 2
6.7	Strontium peroxide	Strontium dioxide	1314-18-7	Oxidizing solid, type 2
6.8	Barium peroxide	Barium dioxide	1304-29-6	Oxidizing solid, type 2
6.9	Zinc peroxide	Zinc dioxide	1314-22-3	Oxidizing solid, type 2
6.10	Carbamide peroxide	Urea hydrogen peroxide; hydrogen peroxide carbamide	124-43-6	Oxidizing solid, type 3

Table 4 (continued)

No.	Item name	Alias	CAS number	Main flammable and explosive hazard categories
6.11	Peracetic acid [content ≤16%, water content ≥39%, peracetic acid content ≥15%, hydrogen peroxide content ≤24%, contains stabilizer]	Peracetic acid; peracetic acid; acetyl hydrogen peroxide	79-21-0	Organic peroxide, type F
	Peracetic acid [content ≤43%, water content ≥5%, peracetic acid content ≥35%, hydrogen peroxide content ≤6%, contains stabilizer]			Flammable liquids, type 3
				Organic peroxide, type D
6.12	Dicumyl peroxide [52%<content ≤100%]	Dioxyl peroxide; vulcanizing agent DCP	80-43-3	Organic peroxide, type F
6.13	Benzoyl hydrogen peroxide	Perbenzoic acid	93-59-4	Organic peroxide, type C
6.14	Sodium superoxide		12034-12-7	Oxidizing solid, type 1
6.15	Potassium superoxide		12030-88-5	Oxidizing solid, type 1
7 Flammable reducing agent				
7.1	Lithium	Lithium metal	7439-93-2	Substances and mixtures that release flammable gases in contact with water, type 1
7.2	Sodium	Sodium metal	7440-23-5	Substances and mixtures that release flammable gases in contact with water, type 1
7.3	Potassium	Potassium metal	7440-09-7	Substances and mixtures that release flammable gases in contact with water, type 1
7.4	Magnesium		7439-95-4	(1) Powder: Self-heating substances and mixtures, type 1
				Substances and mixtures that release flammable gases in contact with water, type 2
				(2) Pellets, chips or ribbons
				Flammable solids, type 2
7.5	Magnesium–aluminum powder	Magnesium–aluminum alloy powder		Substances and mixtures that release flammable gases in contact with water, type 2
				Self-heating substances and mixtures, type 1
7.6	Aluminium powder		7429-90-5	(1) With coating: Flammable solids, type 1
				(2) Without coating: Substances and mixtures that release flammable gases in contact with water, type 2
7.7	Alumina silica		57485-31-1	Substances and mixtures that release flammable gases in contact with water, type 3
	Alumina silica powder			
7.8	Sulfur	Sulfur	7704-34-9	Flammable solids, type 2
7.9	Zinc dust		7440-66-6	Self-heating substances and mixtures, type 1; substances and mixtures that release flammable gases in contact with water, type 1
	Zinc powder			Self-heating substances and mixtures, type 1; substances and mixtures that release flammable gases in contact with water, type 1
	Zinc ash			Substances and mixtures that release flammable gases in contact with water, type 3
7.10	Zirconium metal		7440-67-7	Flammable solids, type 2
	Zirconium metal powder	Zirconium powder		Flammable solids, type 1; substances and mixtures that release flammable gases in contact with water, type 1

(continued)

Table 4 (continued)

No.	Item name	Alias	CAS number	Main flammable and explosive hazard categories
7.11	Hexamine	Hexamethylenetetramine; urotropine	100-97-0	Flammable solids, type 2
7.12	1,2-ethylenediamine	1,2-diaminoethane; ethylene diamine	107-15-3	Flammable liquids, type 3
7.13	Monomethylamine [anhydrous]	Aminomethane; methylamine	74-89-5	Flammable gases, type 1
	Monomethylamine solution	Aminomethane solution; methylamine solution		Flammable liquids, type 1
7.14	Lithium borohydride	Lithium borohydride	16949-15-8	Substances and mixtures that release flammable gases in contact with water, type 1
7.15	Sodium borohydride	Sodium borohydride	16940-66-2	Substances and mixtures that release flammable gases in contact with water, type 1
7.16	Potassium borohydride	Potassium borohydride	13762-51-1	Substances and mixtures that release flammable gases in contact with water, type 1
8 Nitro compound				
8.1	Nitromethane		75-52-5	Flammable liquids, type 3
8.2	Nitroethane		79-24-3	Flammable liquids, type 3
8.3	2,4-dinitrotoluene		121-14-2	
8.4	2,6-dinitrotoluene		606-20-2	
8.5	1,5-dinitronaphthalene		605-71-0	Flammable solids, type 1
8.6	1,8-dinitronaphthalene		602-38-0	Flammable solids, type 1
8.7	Dinitrophenol [dry or water content<15%]		25550-58-7	Explosives, item 1.1
	Dinitrophenol solution			
8.8	2,4-dinitrophenol [water content ≥15%]	1-hydroxy-2,4-dinitrobenzene	51-28-5	Flammable solids, type 1
8.9	2,5-dinitrophenol [water content ≥15%]		329-71-5	Flammable solids, type 1
8.10	2,6-dinitrophenol [water content ≥15%]		573-56-8	Flammable solids, type 1
8.11	2,4-sodium dinitrophenol		1011-73-0	Explosives, item 1.3
9 Others				
9.1	Nitrocellulose [dry or water (or ethyl alcohol) content<25%]	Nitro-cotton	9004-70-0	Explosives, item 1.1
	Nitrocellulose [nitrogen content ≤12.6%, ethyl alcohol content ≥25%]			Flammable solids, type 1
	Nitrocellulose [nitrogen content ≤12.6%]			Flammable solids, type 1
	Nitrocellulose [water content ≥25%]			Flammable solids, type 1
	Nitrocellulose [ethyl alcohol content ≥25%]			Explosives, item 1.3
	Nitrocellulose [unmodified, or plasticizer content<18% if plasticized]			Explosives, item 1.1
	Nitrocellulose solution [nitrogen content ≤12.6%, nitrocellulose content ≤55%]	Nitrocellulose		Flammable liquids, type 2
9.2	4,6-binitro-2-sodium aminophenol	Sodium picramate	831-52-7	Explosives, item 1.3
9.3	Potassium permanganate	Potassium permanganate; potassium permanganate	7722-64-7	Oxidizing solid, type 2
9.4	Sodium permanganate	Sodium permanganate	10101-50-5	Oxidizing solid, type 2
9.5	Guanidine nitrate	guanidine nitrate	506-93-4	Oxidizing solid, type 3
9.6	Hydrazine hydrate	Hydrazine hydrate	10217-52-4	
9.7	2,2-bi(hydroxymethyl) 1,3-propanediol	Pentaerythritol, pentaerythrite	115-77-5	

1.5 Categories of Common Explosive Chemicals

Other than explosives, under certain circumstances, some chemicals including flammable gases, flammable aerosols, certain compressed gases, volatile matters of flammable liquids (i.e., gasoline, benzene, etc.), flammable solid dust or powder (i.e., aluminum powder, pulverized sulfur, etc.), self-reacting substances (benzenesulfonyl hydrazide, etc.), self-heating substances (self-heating metal powder, sodium sulfhydrate, etc.), pyrophoric liquids (pentaborane, aluminum alkyl, etc.), pyrophoric solids (white phosphorus or yellow phosphorus, magnesium powder, anhydrous sodium sulfide, etc.), substances that release flammable gases in contact with water (potassium, silicon aluminum powder without coating, etc.), oxidizing liquids (iodine pentafluoride, hydrogen peroxide, etc.), oxidizing solids (potassium peroxide, silver nitrate, etc.), organic peroxides (di-tert-butyl peroxide, etc.) and others may reach or satisfy explosive conditions and result in explosion.

1.5.1 Categories of Explosives

Based on the hazardous property, explosives may be divided into six categories:

1. Item 1.1: A substance, compound or product with the hazard of total explosion (total explosion refers to ignition of almost the totality of the charge at an instant).
2. Item 1.2: A substance, mixture or product with the hazard of ejection but not total explosion.
3. Item 1.3: A substance, mixture or product with the hazard of combustion and relatively small hazard of detonation, or relatively small hazard of ejection, or both hazards, but not the hazard of total explosion. (1) Combustion that generates obvious thermal radiation; (2) combustion one after another, while at the same time generating relatively small detonation, or relatively small hazard of ejection, or both.
4. Item 1.4: A substance, mixture or product with no obvious hazard of explosion. These substances, mixtures or products, if they were to be ignited or detonated, would only pose a small risk and the requirement is that they are maximally controlled within their packaging. At the same time, it shall be ensured that no fragments visible to the naked eye shall be ejected, and external flame generated by the explosion shall not cause other substances inside the packaging to undergo total explosion.
5. Item 1.5: A substance or mixture with hazard of total explosion but itself is not very sensitive. These substances or mixtures are at risk of total explosion but they are extremely insensitive, and thus it is highly unlikely they would convert from detonation or combustion into explosion under normal circumstances.
6. Item 1.6: A product that is extremely insensitive and with no hazard of explosion. These products only contain extremely insensitive explosive substance or mixture, and other products that have been proven to have almost zero chance of accidental initiation.

1.5.2 Categories of Flammable Gases

Flammable gases are divided into two categories, please see Table 5 for details.

1.5.3 Categories of Flammable Aerosols

If an aerosol contains any flammable component as per GHS classification (for flammable gases, see GB20577; for flammable liquids, see GB20581; and for flammable solids, see GB20582), said aerosol should be considered to be flammable. Flammable component does not include self-heating substances, pyrophoric substances or substances that release flammable gases in contact with water.

Based on the heat of chemical combustion of its components, a flammable aerosol may be classified either as category 1 or no category, if applicable, according to the foam test of is component (for foam aerosol), as well as ignition distance test and enclosed space test (for spray aerosol).

1.5.4 Categories of Compressed Gases

Based on the physical state of the packaging, compressed gases may be divided into four categories (Table 6): (1) compressed gas, (2) liquefied gas, (3) frozen gas or (4) dissolved gas (1 and 2).

1.5.5 Categories of Flammable Liquids

Flammable liquids may be divided into four categories, please see Table 7 for details.

Table 5 Categories of flammable gases

Categories	Categories
1	A gas under conditions of 20 °C and 101.3 kPa:
	(a) A gas that may ignite when it accounts for 13% or lesser volume in the air mixture;
	(b) A gas that can combust at a flammability range of at least 12 percentage points when mixed with air no matter the minimum flammability.
2	A gas, except for those in category 1, that possesses flammability range under conditions of 20 °C and 101.3 kPa:

Note: (1) For some administration purposes, ammonia and methyl bromide may be considered special cases; (2) please see GB25078 for categorization of smoking agents

Table 6 Categories of compressed gases

Categories	Categories
Compressed gas	A gas that remains in complete gaseous state when contained under high pressure and at temperature of −50 °C; including all gases with critical temperature ≤−50 °C (such as compressed natural gas CNG)
Liquefied gas	A gas that exists partially in a liquid state when packaged under high pressure and at temperature above −50 °C. This is further sub-divided into: (a) high pressure liquefied gas: critical temperature between −50 °C and +65 °C; low pressure liquefied gas: critical temperature higher than +65 °C (such as liquefied petroleum gas)
Frozen gas	A gas that exists partially in a liquid state due to low temperature during packaging (such as liquefied natural gas CNG)
Dissolved gas	A gas that is dissolved in a solution in liquid state when packaged under high pressure (such as dissolved acetylene)

Table 7 Categories of flammable liquids

Categories	Categories
1	Flash point below 23 °C and initial boiling point no higher than 35 °C
2	Flash point below 23 °C and initial boiling point higher than 35 °C
3	Flash point no less than 23 °C and initial boiling point no higher than 60 °C
4	Flash point higher than 60 °C and flash point no higher than 60 °C

Table 8 Categories of flammable solids

Categories	Categories
1	Combustion rate test:
	Substances or mixtures other than metal powder:
	(a) Damp portions cannot retard spread of flame
	(b) Combustion time <45 s or combustion rate >2.2 mm/s
	Metal powder: combustion time ≤5 min
2	Combustion rate test:
	Substances or mixtures other than metal powder:
	(a) Damp portions can retard spread of flame by at least 4 min
	(b) Combustion time <45 s or combustion rate >2.2 mm/s
	Metal powder: combustion time <5 min and ≤10 min

1.5.6 Categories of Flammable Solids

Flammable solids may be divided into four categories, please see Table 8 for details.

1.5.7 Categories of Self-Reacting Substances

Self-reacting substances may be divided into the seven categories of A, B, C, D, E, F, or G.

1. Any self-reacting substance or mixture that may explode or rapidly deflagrate in its packaging is classified as type A.
2. Any self-reacting substance or mixture that possesses explosive properties, and neither explode nor rapidly deflagrate, but is prone to explode when heated in its packaging is classified as type B.
3. Any self-reacting substance or mixture that possesses explosive properties, and will not explode, rapidly deflagrate, or explode when heated in its packaging is classified as type C.
4. Any self-reacting substance or mixture that exhibit any of the following status in laboratory experiment is classified as type D: (a) When heated in limited conditions, partially deflagrate, does not rapidly deflagrate, and does not exhibit violent reactions; or (b) when heated in limited conditions, does not explode at all, combusts slowly, and does not exhibit violent reactions; or (c) when heated in limited conditions, does not explode or deflagrate at all, and exhibits moderate reactions.
5. Any self-reacting substance or mixture that does not explode or deflagrate at all, and exhibits minor reactions or no reaction when heated in limited conditions in laboratory experiment is classified as type E.
6. Any self-reacting substance or mixture that neither explodes in cavitation state nor completely deflagrates, exhibits minor reactions or no reaction, and has very little explosive energy or no explosive energy when heated in limited conditions in laboratory experiment is classified as type F.
7. Any self-reacting substance or mixture that neither explodes in cavitation state nor completely deflagrates, does not exhibit reaction, does not have any explosive energy when heated in limited conditions in laboratory experiment, and as long as it is thermally stable (self-accelerating decomposition temperature or 60–75 °C for a 50 kg package). Any liquid mixture that has reduced sensitivity against any thinner with boiling point no less than 150 °C is classified as type G, and any liquid mixture that has reduced sensitivity against any thinner with boiling point less than 150 °C is classified as type F.

1.5.8 Categories of Pyrophoric Liquids

A pyrophoric liquid would be classified into pyrophoric liquid category 1 if said liquid combusts within 3 min after being placed on inert carrier and exposed to the air or if it combusts or carbonizes filter paper within 5 min after making contact with air.

1.5.9 Categories of Pyrophoric Solids

A pyrophoric solid is a solid that is easily flammable within 5 min after making contact with air even in a small quantity. Said type of solid would combust within 5 min after making contact with air, and if so it would be classified into pyrophoric solid category 1.

1.5.10 Categories of Self-Heating Substances

Self-heating substances may be divided into two categories. For example, if a 25 mm × 25 mm × 25 mm sample of the substance returns positive result after testing under temperature of 140 °C, it would be classified into self-heating substances category 1.

1.5.11 Categories of Substances That Release Flammable Gases in Contact with Water

Substances that release flammable gases in contact with water may be divided into three categories.

Category 1: Gas generated from reaction of contact with water is pyrophoric, or if flammable gas is released from reaction of contact with water at maximum rate ≥10 L/(kg min).

Category 2: If flammable gas is released from reaction of contact with water at maximum rate ≥20 L/(kg min), and does not meet criteria of substances that release flammable gases in contact with water in category 1.

Category 3: If flammable gas is released from reaction of contact with water at maximum rate ≥1 L/(kg min), and does not meet criteria of substances that release flammable gases in contact with water in category 1 or category 2.

1.5.12 Categories of Organic Peroxides

Organic peroxides are divided into seven categories.

1. Any organic peroxide that may detonate or rapidly deflagrate within packaging is classified as type A organic peroxide.
2. Any explosive organic peroxide that neither detonates nor rapidly deflagrates within packaging, but is prone to decrepitate within packaging, is classified as type B organic peroxide.
3. Any explosive organic peroxide that cannot detonate, or rapidly deflagrate, or decrepitate, within packaging is classified as type C organic peroxide.
4. Any explosive organic peroxide that exhibit any of the following status in laboratory experiment is classified as type D organic peroxide: (a) Partially detonates, does not rapidly deflagrate, and does not exhibit violent reactions when heated in enclosed condition; (b) does not detonate at all, gradually deflagrates, and does not exhibit violent reactions when heated in enclosed condition; or (c) does not detonate or deflagrate at all, and does not exhibit violent reactions when heated in enclosed condition.
5. Any organic peroxide that definitely does not detonate or deflagrate when tested in laboratory, and only exhibits minor effects or no effect when heated under enclosed condition, is classified as type E organic peroxide.
6. Any organic peroxide that definitely does not detonate or deflagrate in cavitation state when tested in laboratory, only exhibits minor effects or no effect when heated under enclosed condition, and has only weak or no explosive power, is classified as type F organic peroxide.
7. Any organic peroxide that definitely does not detonate or deflagrate in cavitation state when tested in laboratory, exhibits no effect when heated under enclosed condition, and has no explosive power, is classified as type G organic peroxide.

2 Section Two: Explosions in Industrial Activities

Explosion is a rapid physical or chemical change that releases energy at an instant, coupled with movement and energy release. Whether or not substance participating in this change itself actually changes or not, the essence of this change is the instantaneousness and concentration of the energy release. Energy may be released in forms such as light energy, thermal energy and mechanical energy.

2.1 Explosions and Categories

2.1.1 Definition of Explosion

An explosion is the course of extremely quick physical or chemical energy release or conversion in the substance system, and the rapid release or conversion of tremendous amount of energy contained within or generated by the system within limited space and an extremely short time. During this sort of energy release or conversion process, the system's energy would convert into mechanical energy, thermal energy, light energy and so on.

Generally speaking, an explosion may be characterized by the following features:

1. The course of an explosion occurs and develops very fast.
2. Pressure near the site of explosion drastically rises, and the major of explosion would be accompanied by rapid surge in temperature.
3. An explosion usually causes noise loud or quiet.
4. An explosion may cause surrounding medium to vibrate, or damage of nearby objects.

In essence, the characteristic of the explosion itself is the instantaneousness and concentration of the energy release, and the rapid rise in pressure at the site of the blast and surrounding area.

2.1.2 Categories of Explosions

There many different ways to categorize an explosion. Based on source of explosion energy, an explosion may be classified as a nuclear explosion, physical explosion or chemical explosion; based on phase of explosion reaction, an explosion may be classified as a gas-phase explosion, liquid-phase explosion or solid-phase explosion; based on rate of explosion, an explosion may be classified as a deflagration, explosion or detonation; based on reason of explosion, an explosion may be classified as an explosion requiring source of fire or an explosion that does not require source of fire, and so on.

The next section will mostly analyze explosion categorization based on explosion energy. Nuclear leakage has occurred in human industrial activities, but industrial nuclear explosions have never been reported to the public. For example, the Chernobyl nuclear plant disaster was an accident in which human mistakes resulted in steam thermal energy explosion of the nuclear reactor. The magnitude 9.0 earthquake near Fukushima instigated a tsunami, which cut off external power supply to the nuclear plant in Fukushima. The power station's cooling system stopped operating because of loss of electricity, the nuclear reactor continued to rise in temperature, which boosted steam pressure beyond limit, resulting in explosion.

Physical Explosion

Physical explosion is the extremely rapid course of physical energy released due to loss of control. During this process, substance in the system releases energy contained within the system at an extremely fast pace, converting such energy into forms such as mechanical energy and thermal energy. This process is purely physical in nature, and there is only a change in state but no chemical reaction. For example, when liquefied natural gas explodes upon encountering steam, metal vaporizing and exploding due to wire overloaded with current, a tire bursting, boiler drum exploding, balloon bursting after being filled with too much air, molten steel or slag exploding upon encountering steam of water and so on are all instances of physical explosion. Physical explosions are rather common in industrial activities. In 2016, a crack in a nozzle on the high-pressure steam main of a boiler of Madian Ganshi Power Supply Co., Ltd. in Dangyang City of Hubei caused the rupture of high-pressure main steam pipe, generating high-pressure and high-temperature steam (temperature and pressure 530 °C, 9.5 MPa) and instantaneously impacted the centralized control room, resulting in 21 dead, five injured (including three severely injured). This was a typical physical explosion.

Just like the explosion of a boiler drum, the container explosion of Boiling Liquid Expanding Vapor Explosion (BLEVE) is also a type of physical explosion. On November 19th, 1984, a rupture in a pipe at the liquefied natural gas (LNG) terminal of Petróleos Mexicanos (Pemex) in San Juan of Mexico City released a huge amount of liquefied petroleum gas, which formed a vapor cloud and exploded upon contact with a fire source. The resulting fireballs expanded to 360 m in diameter, and four spherical tanks along with 44 horizontal tanks were all destroyed, as well as basically all facilities inside the terminal. The explosion and combustion waves impacted structures within 1200 m, destroyed more than 1400 residential buildings, killed around 650, injured some 6000 and turned 31,000 homeless. When the disaster occurred, two spherical tanks with a capacity of 2400 m^3 were 90% full, and four other spherical tanks with a capacity of 1600 m^3 were also half filled. BLEVE of the four LPG spherical tanks inside the fire took place within 5–6 min. A seismograph at the National Autonomous University of Mexico 25 km away from San Juan recorded the explosion vibration at a magnitude of 0.5.

Chemical Explosion

When high-speed exothermic chemical reaction (mostly oxidation reaction and decomposition reaction) occurs with a substance, and at the same time large amount of gas is generated, which then rapidly expands and impact externally, an explosion would take place. Explosion of explosive is a common form of chemical explosion. Chemical explosions in industrial activities can generally be grouped into two categories:

1. An explosion of explosive chemical compound refers to an explosion in which combustion of substances like organic peroxide, nitro compound or nitrate causes an explosion, or the decomposition reaction of some chemical compounds. For instance, the explosion of butanone peroxide, trinitrotoluene, nitroglycerin, and so on, or the explosion of copper acetylide, or the decomposition explosion of acetylene, ethene, vinyl chloride, and so on. On February 28th, 2012, a major explosion took place at Hebei Ke'er Chemicals Co., Ltd. in Shijiazhuang, with 25 dead, four missing, 46 injured and RMB 44.59 million in destruction. A reaction kettle containing reaction product guanidine nitrate involved in the accident was operating at illegal and excessive temperature (maximum outlet temperature of heat conduction oil heater of reaction kettle allowed is 215 °C, but the temperature was raised to 255 °C, which elevated temperature of materials inside the reaction kettle to near the 270 °C detonation point of guanidine nitrate). Connection of heat conduction oil hose at ball valve at the bottom of the reactor leaked and caused a fire, and inappropriate response by on-duty personnel allowed the external fire to elevate the temperature of the bottom of the reaction kettle. Local accumulation of heat energy reached the detonation point of guanidine

nitrate, leading to the rapid decomposition explosion of guanidine nitrate inside the kettle and unreacted ammonium nitrate. Guanidine nitrate and unreacted ammonium nitrate involved in the accident are both explosive compounds.

Generally speaking, explosion of explosives for civilian use belong to this type of chemical explosion. On May 20th, 2013, an explosion occurred at the emulsified seismic charge column production workshop of Baoli Industrial Explosives Jinan Science and Technology Co., Ltd. in Zhangqiu City of Shandong Province, resulting in 33 dead, 19 injured and direct economic loss of over RMB 66 million. Degree of danger was escalated when waste explosives in seismic charge involved with the accident were mixed with PETN, the detonation explosive in the accident, during the recycling and reuse process. PETN was subjected to forceful friction, compression and collision within the charge loading machine, then exploded instantaneously and detonated all the emulsified explosives inside the machine, and led to sympathetic detonation of all other explosives around the workshop. During this disaster, the initial explosion was that of explosive compound PETN, then the explosion of industrial explosives such as emulsified explosives and seismic charge columns.

2. An explosion of hazardous substance mixture refers to explosion caused by the mixture of oxidizing and reducing substances or other substances. For example, nitric acid and grease, liquid oxygen and coal powder, anhydrous maleic acid and caustic soda are all combinations that could explode when mixed together. Generally speaking, explosion of fireworks and firecrackers also belong to said type of chemical explosion.

Of which, when things like mine gas, town gas, hydrogen, acetylene and other flammable gases, steam of flammable liquids such as gasoline and benzene, flammable liquids sprayed into mist, metal powder and dust, coal powder and dust, flour and other flammable powders and dusts, mix with air in certain proportions and under certain restricting conditions, explosions could occur. These are deemed chemical explosions.

On December 31st, 2014, a major explosion took place inside the axle assembly workshop of Guangdong Fuwa Engineering Group Co., Ltd. in Foshan City, resulting in 18 dead, 32 injured and RMB 37.86 million in direct economic loss. The cause for the explosion was an explosive mixture composed of air and flammable gases generated from evaporation of reducing agent (inspection indicates mixture has flash point −26 °C and explosion limit 0.9–7.5%, and mainly consisting of 33.3% methylal, 17.5% trimethyl-benzene, 12.94% methyl alcohol, 10.9% 1-methoxy-2-propanol, 8.3% butyl acetate, etc.) that flowed into the ditch beneath the main assembly line of the axle assembly workshop, which exploded when lit by spark created from on-site electric soldering. Said accident was an explosion of steam of flammable liquid inside the workshop.

On August 2nd, 2014, an aluminum powder explosion occurred at Zhongrong Metal Manufacturing Co., Ltd. in Suzhou City of Jiangsu Province, resulting in 97 dead, 163 injured (later reports indicate that 49 died from severe injury, and 95 had stabilized and were still being treated), and RMB 351 million in direct economic loss. The dust removal system in the workshop involved had not been cleaned for a long time as per regulation and protocol, aluminum dust had accumulated excessively, aluminum dust in the dust collection bin was moistened, underwent redox exothermic reaction, reached the ignition temperature of the powder and dust cloud, then set off a series of explosions including the workshop's dust removal system.

2.1.3 Destructive Effects of Explosion

The destructive effect of an explosion refers to the total outcome of effects from the explosion itself and effects from products of the explosion. These are chiefly manifested as shock waves, explosion fragments, seismic waves, fires, toxic gases, and so on.

1. Shock wave. A high-pressure and high-temperature gaseous product with high energy density is formed during the instant of an explosion, and it expands into the surrounding area at an extremely fast speed, forcefully compresses the air around and causing the surrounding air to quickly soar in temperature, pressure and density. The outcome is a kind of supersonic shock wave, which propagates through the air to reach targets such as structures and organisms nearby, and affecting them with an extremely powerful shock wave effect. On August 12th, 2015, the dangerous goods warehouse of Rui Hai International Logistics Co., Ltd. at the Port of Tianjin was the site of a major fire and explosion, with the two main explosions respectively forming a crescent-shaped crater 15 m in diameter and 1.1 m in depth, and a massive, round crater 97 m in diameter and 2.7 m in depth. Areas affected by the blast wave were divided into either severe impact zone or moderate impact zone. Within the severe impact zone, the farthest distances away from the center of the blast in the various directions are: 3000 m to the east (AsiaTrak (Tianjin) Ltd.), 3600 km to the west (office building of China Unicom), 2500 m to the south (Zhenhua Logistics Group) and 2800 km to the north (Tianjin Toyota Tongshang Steel Company). Within the moderate impact zone, the farthest distances away from the center of the blast in the various directions are: 3430 m to the

east (International Logistics Quarantine and Inspection Center Section Two) 5400 km to the west (office building of China Certification & Inspection (Group) Co., Ltd.), 5000 m to the south (logistics building of the Port of Tianjin) and 5400 km to the north (Tianjin Maritime College).

2. Fragments impact. Mechanical damage effect of an explosion would cause fragments from containers, equipment, devices, construction materials, and so on to disperse and inflict damage in a vast area. Fragments could fly in different directions, usually up to distances of several tens of meters or even hundreds of meters. Take for example the November 13th explosion of the benzene factory of the Jilin branch of China National Petroleum Corporation (CNPC) involved a loose preheater flange in the air intake system that instigated the explosion of equipment including the T101 nitrobenzene primary distillation column. High-kinetic energy fragments produced by the explosion impacted tank zone No. 55 approximately 165 m away from the initial explosion zone, causing subsequent leaks, fires and explosions in a nitrobenzene storage tank (a volume of 1500 m^3) and two benzene storage tanks tank (a volume of 2000 m^3 each).
3. Seismic effects. When explosions take place, especially those that cause substantial impact on the ground surface, oftentimes induce temporary seismic waves. During the major fire and explosion involving the dangerous goods warehouse of Rui Hai International Logistics Co., Ltd. at the Port of Tianjin, the first explosion took place at 23:34:06 on August 12th, creating an earthquake roughly equal to magnitude 2.3 or the equivalent of 3 tons of TNT. At 23:34:37 on August 12th, the second, more powerful explosion occurred, with intensity on par with 21 tons of TNT. The official Weibo of the China Earthquake Networks Center (CENC) issued information saying that "based on feedback from online users, seisesthesia was noticed in Tanggu and Binhai in Tianjin, as well as Hejian, Suning, Jinzhou and Gaocheng in Hebei." The major fire and explosion involving the dangerous goods warehouse of Rui Hai International Logistics Co., Ltd. at the Port of Tianjin produced shock waves that impacted some structures outside the port area, and while some were not subjected to direct effects of the blast wave, the seismic vibration caused by the explosion damaged some doors and windows near the ground. This kind of damages were seen as far as 8500 m to the east (Dongjiang Port Hotel), 8300 m to the west (Zhengdeli residential building), 8000 m to the south (Heliyuan residential community) and 1330 m to the north (Yongding Xinhe toll booth on Haibin Avenue).
4. Secondary incidents. When an explosion occurs, a fire could be started subsequently if there are flammable items on site. In places where flammable dust is abundant, even the slightest blast wave would kick up the fine particles covering the ground, and create a secondary explosion that is even larger in area. If containers holding toxic substances are ruptured, then toxic leaks could lead to poisoning in nearby personnel.
5. Toxic gases. An explosion could produce a certain amount of toxic gases such as CO, CO_2, NO_X, H_2S and SO_2; these are especially fatal during explosions that take place in a confined space as the high concentration of toxic gas could easily poison and even kill people.

2.2 Analysis of Common Factory Explosions

Explosions commonly seen in factories often are not a single type of explosion, which might involve physical explosions such as those of containers, and then chemical explosions of gases leaked from these containers or those of vapor clouds, as well as chemical explosions from explosive substances. In recent years, as the Chinese petroleum and chemical industry advances by leaps and bounds, explosions due to loss of control of chemical reactions have also occurred in relatively high frequency.

For industries where fires and explosions occur often, such as petroleum and chemical, fires are the most common, followed by explosions and poisoning from the release of toxic substances. In terms of deaths and injuries, toxic leaks are usually the most dangerous, even though production machinery basically cannot be damaged through poisoning. In terms of economic loss, explosions are the most destructive. Please see Table 9 for the three types of chemical industry accidents.

Delving into the subject further, the majority of form of explosions at petroleum and chemical factories is the explosion of unconfined vapor cloud, as in the release of a substantial amount of volatile and flammable vapor clouds that pervade through the different parts of a factory, then ignited and a vapor cloud explosion would occur. For example, in the Pemex liquefied natural gas (LNG) terminal accident mentioned above, ruptured pipeline released a vast amount of LPG, which turned into a vapor cloud that ignited and exploded upon encountering a fire source. Relevant articles show ratios of losses from fires and explosions in hydrocarbon chemical plants around the world. Table 10 includes losses from natural disasters such as floods and hurricanes [4].

Table 9 Three types of chemical industry accidents

Accident type	Possibility of occurrence	Potential casualties	Potential economic loss
Fire	High	Low	Moderate
Explosion	Moderate	Moderate	High
Toxic release	Low	High	Low

Table 10 Ratios of losses from fires and explosions in hydrocarbon chemical plants

Accident type	Fire	Vapor cloud explosion	Explosion	Others
Ratio/%	31	36	30	3

2.2.1 Explosion of Flammable Gas

In industrial production, flammable gas explosions are divided into two situations. The first is explosion of decomposed explosive gas, and the second is explosion of flammable gas mixture. These two differ in criteria for explosion.

Explosion of Decomposed Explosive Gas

Some gases such as acetylene, ethene and ethylene oxide could explode even without air or oxygen, and without a source of fire. In essence, this is decomposition explosion. Other than the gases mentioned above, some other decomposed explosive gases are ozone, hydrazine, allene, methyl acetylene, vinyl acetylene, nitric oxide, nitrogen dioxide, hydrogen cyanide, tetrafluoroethylene and so on.

When a decomposable explosive gas undergo decomposition under certain temperature and pressure conditions, substantial level of decomposition heat would be generated, providing the energy necessary for an explosion. Generally speaking, gases with decomposition heat above 80 kJ/mol may explode under certain conditions (temperature and pressure). Decomposition heat is the internal reason for such type of gas explosion, while certain temperature and pressure are external reasons. For example, when acetylene is heated or compressed, it may undergo reactions like polymerization, addition, substitution or explosive decomposition. When temperature reaches 200–300 °C, acetylene molecules would start to undergo polymerization exothermic reaction, forming relatively complex compounds (like benzene) and release heat. The released heat would in turn elevate the temperature of acetylene, and when temperature rises to 700 °C, the unpolymerized acetylene would undergo explosive decomposition. If acetylene decomposition takes place in a sealed container (i.e., acetylene storage tank, acetylene generator, acetylene cylinder, etc.), the climb in temperature would rapidly increase internal pressure by 10–13 times, resulting an explosion. Besides, explosion from acetylene decomposition is also related to pressure. The critical pressure of acetylene is 142 kPa, and common international practice sets 140 kPa as limiting pressure of acetylene generator in welding and cutting operations, while some countries are even more stringent, allowing only 130 kPa as limiting pressure of acetylene generator. Meanwhile, the critical pressure of decomposition explosion of ethylene oxide is 40 kPa, which is why its production, storage and transportation require extra care.

Explosion of Flammable Gas Mixture

It is hard to differentiate between flammable gas mixture and explosive gas mixture, and explosions may occur under certain conditions while combustion may take place under other conditions, which is why within certain premises the two are interchangeable. The difference in combustion and chemical explosion is the rate of combustion reaction (oxidation reaction).

Combustion reaction is usually divided into three stages: (1) dispersal stage, (2) inductive stage and (3) chemical reaction stage. Flammable gas mixture first undergoes the dispersal stage, during which molecules of the flammable gas spread and make contact with oxygen molecules in the air, composing a flammable gas mixture that is the principle criterion that precipitates combustion or explosion. In general, the flame from the combustion of dispersing flammable gas mixture spreads at a rate of 5–30 m/s and a very low overpressure. The flame from the deflagration stage of flammable gas mixture spreads at a rate of 30–500 m/s, and overpressure could reach 2–3 mbar, and when flame spreads at a rate of 500–1000 m/s, overpressure could reach 1 bar. The flame from the explosion (or detonation) stage of flammable gas mixture spreads at a rate of around 2200 m/s, and overpressure could soar to 20 bar.

Explosion Limit

A key parameter to express the explosive hazard of a flammable gas mixture (or vapor, or flammable dust) is explosive limit. The explosive limit of a flammable gas mixture or steam may be expressed as concentration (%) of that flammable gas mixture or vapor in the air, while that of a flammable dust would be expressed as mass concentration (g/m^3).

Volume fraction and mass concentration of a flammable gas mixture or vapor at 20 °C may be converted per the following formula.

$$Y = \frac{L}{100} \times \frac{1000M}{22.4} \times \frac{273}{273+20} = L \times \frac{M}{2.4} \quad (1)$$

In the equation:

L: Volume fraction, %;
Y: Mass concentration, g/m^3;
M: Relative molecular weight of flammable gas mixture or vapor.

The maximum concentration of a gas or vapor able to generate an explosion is called the upper explosive limit (UEL), and the minimum concentration of a gas or vapor able to generate an explosion is called the lower explosive limit (LEL), A standard practice is to use the ratio of the UEL/LEL difference and LEL, to express hazard level H, as in the following.

$$H = \left(L_{up} - L_{down}\right) / L_{down} \quad \text{or} \quad H = \left(Y_{up} - Y_{down}\right) / Y_{down} \quad (2)$$

In most cases, the higher the value of H, the wider the range of explosive limit of that flammable mixture, the greater the risk of explosion.

Explosive limit is not a physical constant because it changes as conditions change. When judging the explosive hazard of a substance under certain processing or technical conditions, it is necessary to take into full account the conditions surrounding the substance to determine the correct explosive limit. Explosive limit is affected by numerous factors such as the substance system's temperature, pressure, retardant medium (type, content, etc.), explosive container (material, size, etc.), source of ignition (type, activation energy, etc.), among other variables.

Evaluation of Consequence of Explosion

The result of the explosion of a flammable gas mixture depends on at least the following several aspects of physical factors: Type of gas, stoichiometric ratio of combustion of the gas, location and type of source of ignition, confined space and ventilation (volume and location), initial turbulence, blocking ratio, size, shape and location of obstructions, number of obstructions (relative to given blocking ratio), etc. At present, there are three main types of methods for simulating or predicting consequence of explosion of a flammable gas mixture, each of which possesses its own application scope, pros and cons.

Empirical Models

Empirical models encompass various methods, such as conversion of certain portion of energy from a flammable gas mixture explosion into air shock wave as expressed in TNT equivalent (TNT Method), the TNO method from the Netherlands, the explosion intensity-based Multienergy Method, the flame speed-based Baker–Strehlow Method (B-S Method), the Congestion Assessment Method, the Sedgwick Loss Assessment Method and so on. Empirical models describe correlations between physical quantities, but rare take into account rules or models of physics. Empirical models are the simplest for estimating overpressure from deflagration. Of these, the TNT Method is a simple method in which flammable materials with known energy are converted into TNT equivalent. TNT equivalent mass may be estimated using the formula below.

$$m_{TNT} = \frac{\eta m \Delta H_c}{E_{TNT}} \quad (3)$$

In the formula,

m_{TNT}: TNT equivalent mass (kg);
E_{TNT}: TNT explosion energy (4686 kJ/kg);
η: Empirical explosion efficiency (nondimensional);
m: Hydrocarbon mass (kg);
ΔH_c: Flammable gas explosion energy (kJ/kg). For flammable gas, heat of combustion may be used as substitute for energy of explosion.

Empirical explosion efficiency η is the biggest issue in the method as it is used to adjust the predictions of various factors. For examples, the level of mixture between flammable substance and thee air, and the rate of conversion from heat energy to mechanical energy. The majority of papers and articles are of the opinion that most flammable vapor clouds range η between 1% and 10%, while flammable vapor clouds of propane, diethyl ether and acetylene η are respectively at 5%, 10% and 15%. Explosion efficiency may also be utilized for solid explosive substances such as ammonium nitrate and rock blasting explosive.

Phenomenological Models

Phenomenological models express the main physical processes during the course of an explosion, two representatives here are the SCOPE model and CLICHE model. The SCOPE (Shell Code for Over-pressure Prediction in Gas Explosions) model was initially applied in simulating explosion of modules in offshore oil rigs. At present SCOPE 3 is used, which can process various types of objects, explosion product's rear venting and so on. The CLICHE (Confined LInked CHamber Explosion) model is used to study explosions in confined spaces of buildings, but its application has been expanded to the simulation explosions of land-based factories and offshore platforms.

Computational Fluid Dynamics (CFD)

There are eight main types of computational fluid dynamics (CFD) models, namely EXSIM, FLACS, AutoReaGas, the even more sophisticated CFX-4, COBRA, ICRC (Imperial College Research Code), NEWT and REACFLOW. The main features of each model are listed on Table 11 [5]. CFD can show changes in factors such as shock wave overpressure, shock wave overpressure arrival time and sustained time and temperature at any specific location within the explosion space and nearby area during the course of the explosion.

In industrial production and daily life, many explosion accidents arose from explosive mixture comprised of flammable gases and air. Explosive mixtures are formed when flammable gases escape into the air from processing machinery, equipment piping, storage tank or other "containers," or when they exist in confined space, or when air seeps into equipment or pipeline holding flammable gases. Such mixtures would explode upon contact with any source of ignition. This type of accident is the focus of industrial explosion prevention.

Table 11 Summary of flammable gas mixture numerical models

Name	Type	Grid	Accuracy	Physical model
EXSIM	Three-dimensional CFD finite element	Structuralized, Cartesian coordinates, PDR processing of objects smaller than grid size	First order time sequence, second order space	Vortex breakage model
FLACS	Three-dimensional CFD finite element	Structuralized, Cartesian coordinates, PDR processing of objects smaller than grid size	First order time sequence, process variable is second order	Empirical correlation
AutoReaGas	Three-dimensional CFD finite element	Structuralized, Cartesian coordinates, PDR processing of objects smaller than grid size	First order time sequence and space	Empirical correlation
CFX-4	Two-dimensional or three-dimensional CFD finite element	Structuralized, body-fitted grid	Higher order time sequence and space	Vortex breakage model and light flame model
COBRA	Two-dimensional or three-dimensional CFD finite element	非Nonstructuralized, Cartesian coordinates, cylindrical polar coordinates, hexahedral coordinates, self-adaption, PDR processing of objects smaller than grid size	Second order time sequence and space	Empirical correlation
ICRC	Two-dimensional CFD finite element	Nonstructuralized, self-adaption	Implicit time, second order (TVD) space	Laminar flamelet and PDF transport equation model
NEWT	Three-dimensional CFD finite element	Nonstructuralized, self-adaption	Higher order time sequence and second order space	Laminar flamelet and vortex breakage model
REACFLOW	Two-dimensional or three-dimensional CFD finite element	Nonstructuralized, self-adaption	First order or second order time sequence and space	Vortex breakage model

2.2.2 Explosion of Flammable Dust

Fires and explosions caused by dust most frequently occur at coal mines, flour factories, sugar factories, textile factories, feed, plastic or metal processing plants, grain depots and other such types of buildings, mines and enterprises. Dust explosion itself is a special kind of combustion phenomenon, and it too requires the three requisites of flammable substance, comburent and ignition source.

1. **The dust here refers to flammable dust.** Flammable dust may be categorized as either organic dust or inorganic dust. Organic dust such as flour, wood dust and chemical fiber dust are basically flammable dust. Meanwhile, inorganic dust like metal dust and the dust of some minerals (such as coal, sulfur, etc.) are also flammable dust. Sand and dirt are also fine particles, but since they themselves cannot burn, thus they are not considered explosive or dangerous.
2. **For an explosion, flammable dust must be suspended in a comburent gas (such as air), and concentration needs to reach the dust's explosive limit.** Suspension of flammable dust in the air may be caused by various mechanical effects or actions such as pulverization, grinding, transportation and ventilation. Dust with large particle size usually settles into deposited dust that is only able to combust, and only dust with small particle size could suspend in the comburent gas. Similarly, the hazard of explosive dust is also expressed using lower explosive limit (LEL). Generally speaking the limit is between 20 and 60 g/m^3, as combustion is difficult to sustain at any lower concentration, let alone explosion.
3. **Explosion also requires source of ignition sufficient to ignite.** Such dusts have relatively low spontaneous ignition point and minimum ignition energy, and when an external energy source exceeds minimum ignition energy (generally between 10 and 100 mJ) or when temperature is higher than spontaneous ignition point (usually between 400 and 500 °C), an explosion would happen.

When the three requisites above are met, dust explosion and fire could take place. It ought to be noted that dust could very likely lead to a second explosion that is even more devastating. When flammable dust suspends in an environment with enough oxygen to sustain combustion, and when an appropriate ignition source is present, the initial explosion may occur. The blast would cause disturbance throughout the surrounding environment, and kick up dust that previously deposited on the ground and on surfaces of equipment, spreading into dust cloud that may be ignited into an even more destructive second explosion.

Laboratory measurements of dust explosion parameters are closely related to factors such as instruments used, test conditions, criteria and definitions. Dust explosion parameters like ignition temperature, lower explosive limit, mini-

mum ignition energy, explosion pressure, pressure increase rate and other parameters are not the basic properties of the substance itself. For example, pressure increase rate depends on the height of the space of the test container when other conditions remain constant. If the container's volume is larger than 0.04 m^3, pressure increase rate $\frac{dp}{dt}$ and container volume V follows the "cubic law" as shown in Formula (4).

$$\left(\frac{dp}{dt}\right)_{max} V^{1/3} = K_{st} \tag{4}$$

The explosion index of dust K_{st} rises as explosion power increases.

2.2.3 Explosion of Explosives

According to hazard level of explosives in the GHS system, explosives are mainly comprised of explosives, their mixtures and explosive items, as well as these two types of substances, mixtures or products that are not mentioned but in reality do generate explosions and flames. Some representative substances in the above, such as 2,4,6-trinitrotoluene (TNT), cyclotrimethylene trinitramine, trinitrotoluene mixed with aluminum (compound explosive of hexogen, TNT and aluminum powder); blasting detonator, incendiary ammunition, smoke ammunition, ammonium nitrate asphalt paraffin explosives; and black powder, firework and fire cracker products. Features of explosive item:

Explosiveness

The explosiveness of an explosive hinges on the material's own composition and properties. The difficulty of explosion meanwhile depends on the sensitivity of the energetic material (substance) itself. Generally speaking, the higher the sensitivity, the easier the substance explodes. An explosive may blow up when it is affected by external forces and factors such as heating, collision, friction, flame and changes in acidity or basicity.

Sympathetic Detonation

When an explosive blows up, it may cause other explosives within a certain distance to explode as well, in what is called sympathetic detonation. This is one of the unique properties of explosives. The occurrence of sympathetic detonation is the result of the propagation of shock wave, which is more powerful at closer distances.

Let us use explosive as an example and analyze its chemical reaction characteristics.

An explosive is an unstable chemical system, but it remains stable in normal environment. Prior to receiving a certain amount of external energy, usually an explosive would not just blow up on its own. Under certain environment conditions, an explosive would undergo chemical changes in one of the following four forms: decomposition (thermal decomposition), combustion, explosion or detonation.

1. Decomposition: One characteristic of decomposition is the slowness. This reaction occurs within the whole explosive and not concentrated in certain region. The reaction speed is contingent on ambient temperature. The rate at which an explosive decomposes accelerates exponentially with the rise in temperature. In spaces where ventilation and heat dissipation is poor, heat generated from decomposition cannot readily dissipate, which could cause the explosive to rise in temperature, leading to the combustion or even explosion of the explosive.
2. Combustion: One characteristic of combustion is that it could occur in the anoxic environment. Combustion starts at certain part of the explosive, turns into combustion reaction, then spreads and expands. This is a type of oxidation reaction, and produces light and heat. The rate at which the combustion reaction area expands is called burning linear velocity (burning velocity), and usually stays below several meters per second. When burning velocity reaches several hundred meters per second, the term is deflagration. The burning velocity of an explosive does not exceed the speed of sound.
3. Explosion: One characteristic of explosion is that it occurs within a certain area of the explosive, creates an explosion reaction zone, then rapidly propagates within the explosive (propagation velocity of the explosion reaction zone is explosive velocity). Blast wave generated by the explosion reaction zone affects neighboring explosive and surrounding medium in the form of high pressure. Violent oxidation reaction, powerful light, high temperature and high pressure are produced. Once an explosion reaction starts, it is not affected by ambient temperature. In general, explosive velocity of normal explosives may reach several thousand meters per second, which is faster than the speed of sound.
4. Detonation: This is an explosion that propagates per the largest steady speed (velocity of detonation wave propagation) within the explosive. This is the highest form of explosion.

Sensitivity of explosive may be divided into sensitivity to heat, sensitivity to flame, sensitivity to friction, sensitivity to collision, sensitivity to blast energy, and sensitivity to static electricity, among others. On the one hand, the sensitivity of an explosive is related to the explosive's structure, physical and chemical properties such as composition, molecular structure, bond energy between atoms in molecule, heat of formation and activation energy. On the other hand, sensitivity is also related to the explosive's physical form and conditions of the charge such as explosive's temperature, physical

form and crystal form of the explosive, particle size of explosive, charge density and additives. The detonation of an explosive may occur in due to four factors: (1) thermal energy (spark, flame or heat), (2) mechanical energy (collision or friction), (3) electricity or (4) blast impact.

The majority of explosions that occurred in chemical factories are considered to have happened on the ground. The explosion's sideway overpressure p_0 is usually described by the following empirical Formula (2).

$$\frac{p_0}{p_a} = \frac{1616\left[1+\left(\frac{z}{4.5}\right)^2\right]}{\sqrt{1+\left(\frac{z}{0.048}\right)^2}\sqrt{1+\left(\frac{z}{0.32}\right)^2}\sqrt{1+\left(\frac{z}{1.35}\right)^2}} \quad (5)$$

In the formula, p_a is ambient air pressure (kPa); $z = \frac{r}{m^{1/3}}$. r is distance (m) from center point of the blast; m is mass of TNT (kg). Usually it is necessary to establish thermal dynamics model to calculate explosion energy, and convert that into corresponding TNT mass [4, 6].

2.2.4 Explosion Caused by Accumulation of Heat from Chemical Reaction

Spontaneous heating is the accumulation of reaction heat from chemical reaction within the whole system, leading to rise in temperature. Generally speaking, if reaction heat dissipates externally as it is generated, the temperature of the whole system would not rise. But if the rate of generation of reaction heat is higher than the rate of external dissipation, the temperature of the whole system would climb. This rise in temperature provides more activation energy for chemical reaction in the reactant, speeding up the reaction process, including hastening the generation of reaction heat, and the heat generation-external dissipation balance in the system is increasingly lopsided. At first, temperature of the whole system hikes at a gradual pace, but as time elapses, the rate of temperature increase would pick up speed, and ultimately the whole system would heat up rapidly.

This rapid heating of the system would end in two situations:

1. Spontaneous ignition explosion: When the temperature reaches the system substance's ignition temperature, the reactant would spontaneously ignite. The reactant would ignite and start combusting in the air, but if the spontaneously ignited reactant is held within an enclosed container, the container's internal pressure would surge, possibly leading to an explosion. Worth noting is that the ignition source here is fire from the spontaneously ignition from chemical reaction of the reactant itself, which is fundamentally different from ordinary external ignition source that has nothing to do with the reactant.
2. Uncontrolled reaction explosion: The surging temperature causes the system substance's vapor pressure to soar. If the reacting system is held inside an enclosed container, the reaction heat may cause the vapor pressure of liquids such as reactant consisting of liquid with low boiling point, or organic solvent, to rise quickly. When vapor pressure exceeds the container's pressure limit, the sealed container would blow up.

Explosions caused by build-up of chemical reaction heat have occurred quite frequently among petrochemical and other industries in China in recent years. On August 2nd, 2014, an aluminum powder explosion occurred at Zhongrong Metal Manufacturing Co., Ltd. in Suzhou of Jiangsu Province. Aluminum dust that had excessively accumulated in the dust collection bin of the dust removal system was moistened, underwent redox exothermic reaction, reached the ignition temperature of the powder and dust cloud, then set off a series of explosions including the workshop's dust removal system. On August 12th, 2015, the dangerous goods warehouse of Rui Hai International Logistics Co., Ltd. at the Port of Tianjin was the site of a major fire and explosion. Some nitrocotton, stored inside a container at the southern side of the arrival zone of the dangerous goods warehouse, dried up due to loss of wetting agent, and exothermic decomposition occurred at an accelerated pace due to factors such as hot weather, resulting in accumulation of heat that led to self-ignition. The fire instigated large-scale combustion of other nitrocotton and hazardous chemicals in nearby containers, causing hazardous chemicals like ammonium nitrate stockpiled in the arrival zone to explode. On March 21st, 2019, a major explosion occurred at Tian Jia Yi Chemicals Co., Ltd. in Yancheng of Jiangsu Province [7]. The company illegally stockpiled denitrifying wastes inside its solid waste warehouse over an extensive period, heat continued to accumulate and caused the wastes to spontaneously ignite, which then started to burn and eventually exploded. The ignition sources in the three accidents mentioned above are all directly associated with accumulation of chemical reaction heat, and are classified as spontaneous ignition explosion.

Issued by the former State Administration of Work Safety, the *List of First Group of Hazardous Chemical Techniques Under Key Supervision* and the *Safety Regulation Requirements, Key Supervision Parameters and Recommended Control Proposals for First Group of Hazardous Chemical Techniques Under Key Supervision* (State Administration of Work Safety 3rd Office (2009) No. 116) stipulated 15 chemical processes, namely, phosgene and phosgenation process, electrolytic process (chlorine-alkali), chlorination process, nitrification process, ammonia synthesis process, cracking (pyrolysis) process, fluoride process, hydrotreating process diazotization process, oxidation process, peroxide process, amination process, sulfonation

process, polymerization process and alkylation process. In addition, the *Notification about Issuance of List of Second Group of Hazardous Chemical Techniques Under Key Supervision and Amendments to Some Typical Techniques in First Group of Hazardous Chemical Techniques Under Key Supervision* (State Administration of Work Safety 3rd Office (2013) No. 3) stipulated three new types of chemical processes including coal chemical process, calcium carbide production process and diazotizing process. The absolute majority of these hazardous chemical processes under key supervision involve exothermic reaction, and they are at risks of explosion due to uncontrolled reaction, hence subjected to strict supervision and regulation. At the same time, *Guiding Opinions about Strengthening Safety Risk Evaluation of Fine Chemical Reactions* (State Administration of Work Safety 3rd Office (2013) No. 3) issued by the former State Administration of Work Safety also mandated the performance of fine chemical reaction safety risk evaluation in order to determine and confirm risk level, adopt the proper regulatory measures, and prevent harms to the public due to fire, explosion or poisoning arising from uncontrolled reaction in fine chemicals production.

3 Section Three: Application of Blast Injury Principles

Air shock wave generated by explosion is the main form of external output of explosion energy. For example, when a high-pressure container explodes, energy in the fragments launched by the explosion and residual deformation energy of the container account for roughly 3–15% of total explosion energy only, as the majority of energy converts to air shock wave.

Shock wave effect principles include overpressure principle, impulse principle and overpressure–impulse principle. The overpressure principle states that when shock wave overpressure reaches a certain level, it would cause a certain degree of injury or damage to the target. The next section will look at application of blast wave overpressure related to China's safety evaluation, the petrochemical industry, civil explosives, fireworks and blasting industries, and blasting works.

3.1 Recommended Overpressure Principle in Safety Evaluation

Safety evaluation is also known variously as hazard assessment, risk assessment and so on. The goal is to achieve safety in a system by applying safety system engineering principles and methods in order to identify and analyze risk factors and harmful factors existing in the system, The identification of hazard source, evaluation of risks and controlling such risks constitute the basics of safety system engineering. When explosion risks are inherent in the system, it is necessary to carry out quantitative evaluation of blast wave, its scope and degree of impact, so as to provide references for accident prediction and prevention, rational safety investment and corporate safety management improvement. *Safety Evaluation* (former State Administration of Work Safety, China Coal Industry Publishing House, 2002) provides bodily injury and structural damage thresholds for shock wave overpressures as seen in China's safety evaluation works. Please see Tables 12 and 13 for details [8].

Different countries adopt slightly different approaches in measuring air shock wave from explosion.

The CPR18E "Qualitative Risk Assessment Guide" (first edition, 1999) submitted by the National Institute for Public Health and the Environment (RIVM) states that 0.03 bar (G) of overpressure is sufficient to cause breakage in the majority of glass on the side subjected to pressure, 0.1 bar (G) of overpressure is enough to damage 10% of houses with 2.5% probability of death among personnel inside, while 0.3 bar (G) of overpressure would raise probability of death among personnel (inside and outside) to 100%.

The Risk Based Inspection (API 581) of the USA has been converted to the domestic industrial standard SY/T 6714-2018; blast overpressure for equipment damage and personnel death are both determined to be 5 psi (g), as in 34.475 kPa.

Table 12 Bodily harm caused by shock wave overpressure

Overpressure ΔP (MPa)	Harmful effect
0.02–0.03	Minor injury
0.03–0.05	Auditory apparatus injury or fracture
0.05–0.10	Severe internal organ injury or death
Greater than 0.10	Death among majority of personnel

Table 13 Structural damage caused by shock wave overpressure

Overpressure ΔP (MPa)	Damage effect
0.005–0.006	Some doors, windows and glass broken
0.006–0.015	Majority of doors, windows and glass broken on the side subjected to pressure
0.015–0.02	Damaged window frame
0.02–0.03	Wall cracked
0.04–0.05	Wall cracked significantly, roof tiles fell off
0.06–0.07	Toppled wooden structure, loosened building frame
0.07–0.10	Toppled brick wall
0.10–0.20	Damaged shock-proof reinforced concrete, toppled small building
0.20–0.30	Damaged large steel structure

3.2 External Safety Distance Between Production Equipment and Storage Facilities of Hazardous Chemicals

External safety distance is set to prevent or reduce impact of production equipment and storage facility building accidents (fire, explosion, poisoning, etc.) on subjects of protection outside the building. In other words, a distance or risk control line is established between the subjects of protection and these equipment or facilities at risk of accidents. The *External Safety Distance between Production Equipment and Storage Facilities of Hazardous Chemicals Determination Method* (GB/T 37243-2019) mandates the determination method for external safety distance between production equipment and storage facilities for dangerous chemicals, to be used for site selection for such hazardous chemical production equipment and storage facilities, and planning of usage of surrounding lands [9]. Article 4.2 in the standard stipulates that hazardous chemical production equipment and storage facilities involving explosives shall adopt "accident outcome method" to determine external safety distance. Obviously, the "explosives" here have clear definitions, referring to those defined in the *List of Hazardous Chemicals* (2015 edition).

External safety distance is calculated using Formula (6) based on worst-case scenario and air shock wave overpressure threshold values given in Table 14:

$$\Delta P = 14\frac{Q}{R^3} + 4.3\frac{Q^{2/3}}{R^2} + 1.1\frac{Q^{1/3}}{R} \tag{6}$$

Table 14 Air shock wave overpressure threshold values for different types of subjects of protection

Subject of protection (categorized based on GB36894)	Air shock wave overpressure threshold value (Pa)[a]
Highly sensitive subject of protection or key subject of protection	2000
Type I subject of protection in regular subjects of protection	
Type II subject of protection in regular subjects of protection	5000
Type III subject of protection in regular subjects of protection	9000

[a] 2000 Pa threshold is the upper limit at which structures would basically remain undamaged; 5000 Pa threshold causes semiminor damage to structures (2000–9000 Pa) but toward the mild end of the spectrum, and may result in shattering of all glass, minor movement of tiles on roof, and falling of small quantity of wall plastering; 9000 Pa threshold is the upper limit in causing semiminor damage to structures (2000–9000 Pa), and may result in hosing structure partially damaged to the point of rendering the building inhospitable, minor deformation of steel structure, but no damage to reinforced concrete pillars. The thresholds stated above virtually are not sufficient to directly cause death to personnel outside

In the equation:

ΔP: Air shock wave overpressure value, expressed in 10^5 pascal (Pa);
Q: TNT equivalent of one explosion, expressed in kilogram (kg);
R: Distance between point of explosion and subject of protection, expressed in meter (m).

3.3 External Distance in Engineering Design for Explosives for Civil Use, Fireworks and Firecrackers

The external distance between a hazardous building/structure associated with explosives for civil use and surrounding residential and business structures, public transportation routes, high-voltage cables, urban area boundaries and so on should be determined based on the hazard level of said hazardous building/structure and the related explosive quantity. The explosive quantity is the largest quantity of dangerous goods inside or outside the building/structure that may possibly explode or combust at the same time The *Safety Code for Design of Engineering of Civil Explosives Materials* (GB50089-2018) stipulates the hazard levels of such buildings/structures [10]. For example, at level 1.1, there is a table that specifies the external distance between a single building with a certain explosive quantity (kg) and the boundaries of subjects of protection such as residences with 50 or less people, or ten or less households. The determination principle for said external distance is essentially the relationship between values of shock wave overpressure and building/structure damage level seen in civil explosive material engineering and design. Please see Table 15 for details.

The *Safety Code for Design of Engineering of Fireworks and Firecracker* (GB50161-2009) stipulates determination principle for minimum allowable external distance in buildings/structures related to engineering of fireworks and firecrackers [11]. At present, the main reference used is the relationship between values of shock wave overpressure and building/structure damage level seen in civil explosive material engineering and design.

3.4 Allowable Safety Distance in Blasting Works

The *Blasting Safety Regulations* (GB6722-2014) stipulates safety and technique requirements in the purchasing, transportation, storage, usage, processing, inspection and destruction/decommissioning of blasting equipment undertaken in

Table 15 Building damage level

Damage grade	Damage extent	Damage description									Note
		Glass	Wooden door and window	Brick outer wall	Wooden roof	Reinforced concrete roof	Tile roof	Canopy roof	Inner wall	Reinforced concrete pillar	Overpressure ΔP ($\times 10^5$ Pa)
I	Basically no damage	Occasional damage	No damage	No damage	No damage	No damage	No damage	No damage	No damage	No damage	$\Delta P < 0.02$
II	Semiminor damage	Small parts or large parts of strips or pieces of damage	Small amount of damage to window panes	No damage	No damage	No damage	Small degree of movement	Small quantity of plastering fell	Small quantity of batten wall plastering fell	No damage	$\Delta P = 0.09$–0.02
III	Minor damage	Large parts of pieces of damage or pulverization	Large amount of damage to window panes, and damage to window and door frames	Appearance of small cracks with largest width ≤5 mm and slight slanting	Deformation of wooden building panels, and occasional cracks	No damage	Large degree of movement	Large quantity of plastering fell	Large quantity of batten wall plastering fell	No damage	$\Delta P = 0.25$–0.09
IV	Moderate damage	Pulverization	Window pane fell or toppled internally, and large amount of damage to window and door frames	Appearance of rather large cracks with largest width between 5 and 50 mm, obvious slanting, and appearance relatively small cracks in brick pillars	Cracks in wooden building panels and wooden building purlins, and loosened wooden building frames	Appearance of very small cracks with largest width ≤1 mm	Large degree of movement to the point of all having fallen	Partial damage or bending of wood joist	Appearance of small cracks on brick inner wall	No damage	$\Delta P = 0.40$–0.25
V	Semisevere Damage	–	Destruction of door and window panels, and fell window frames	Appearance of significant cracks with largest width >50 mm, obvious slanting, and appearance relatively large cracks in brick pillars	Breaking of wooden building purlins, occasional cracks in wooden building frame components, and dislocation of support	Appearance of obvious cracks with largest width between 1 and 2 mm, but may continue to be used after repair	–	Collapse	Appearance of relatively large cracks on brick inner wall	No damage	$\Delta P = 0.55$–0.40
VI	Severe Damage	–	–	Partial collapse	Partial collapse	Appearance of rather large cracks with largest width >2 mm	–	–	Appearance of significant cracks on brick inner wall to the point of partial collapse	Slanting	$\Delta P = 0.76$–0.55

blasting or performed by blasting organizations. This standard is applicable to various types of civil blasting works, and blasting engineering with nonmilitary purpose performed by the People's Liberation Army or People's Armed Police.

3.4.1 Allowable Safety Distance Between Blasting Site and People or Other Subjects of Protection

The allowable safety distance between blasting site and people or other subjects of protection should be determined based on the largest extents of the various harmful effects of blasting (seismic wave, shock wave, individual projectile, etc.) as considered and verified separately. When determining blasting safety distance, impacts of secondary damages possibly induced by blasting such as landslide, rock-fall, avalanche and movement of blasted muck pile should be taken into account, and an appropriate extension of safety distance should be added, or an extra hazard buffer zone demarcated in accordance with actual circumstances. Of these, allowable safety distance of air shock wave in blasting is divided into the following two situations.

Open-Air Ground Surface Blasting

When explosive quantity for a single blast during an open-air ground surface blasting does not exceed 25 kg, allowable safety distance of air shock wave for blasting personnel inside shelter should be determined using Formula (7).

$$R_k = 25Q^{1/3} \tag{7}$$

In the equation:

R_k: Shortest allowable distance of air shock wave for personnel inside shelter, m;
Q: TNT equivalent for a single blast, second-delay blasting is the maximum charge quantity of a single segment, millisecond-delay blasting is the total charge quantity, kg;

Blasting Processing or Large-Equivalent Ground Surface Blasting

During blasting processing or special engineering that requires large-equivalent ground surface blasting, air shock wave overpressure to be borne by different subjects of protection should be calculated and verified, then determine the corresponding allowable safety distance. When blasting is conducted in flat terrain, overpressure may be calculated using Formula (6). Formula (8) is the same as Formula (6).

$$\Delta P = 14\frac{Q}{R^3} + 4.3\frac{Q^{2/3}}{R^2} + 1.1\frac{Q^{1/3}}{R}\Delta \tag{8}$$

In the equation:

ΔP: Air shock wave overpressure, 10^5 Pa;
Q: TNT equivalent for a single blast, second-delay blasting is the maximum charge quantity of a single segment, millisecond-delay blasting is the total charge quantity, kg;
R: Distance between source of explosion and subject of protection, m.

Standard of safety allowance of air shock wave overpressure: 0.02×10^5 Pa for unprotected, nonworking personnel, and 0.1×10^5 Pa for working personnel inside shelter. Please refer to Table 11 for relationship between structural damage level and shock wave overpressure.

3.4.2 Storage Quantity and Distance for Blasting Equipment

Blasting equipment shall be stored inside blasting equipment depot, and illegally storing blasting equipment by any individual is strictly prohibited.

Storage quantity and storage method in a single depot shall abide by regulations stipulated in GB50089. The total volume of the main depot shall not violate the following regulations: (1) quantity of explosives equal to the organization's need for half a year and (2) quantity of blasting equipment equals to the organization's need for a full year. When different types of blasting equipment are stored in the same depot, the largest quantity of explosives housed in a single depot shall abide by regulations stipulated in GB50089.

Store a single type of blasting equipment in specialized depot. If conditions do not permit and it is necessary to store different types of blasting equipment in the same depot, storage shall abide by the following regulations: (1) explosives, penetrating charges, fuse and detonating tube may be stored together in the same depot; (2) blasting equipment such as blasting cap shall be stored in individual depots; (3) black powder shall be stored in individual depots; and (4) ammonium nitrate shall not be stored alongside any other material. When different types of blasting equipment are stored in the same depot, the largest quantity of explosives housed in a single depot shall abide by regulations stipulated in GB50089.

Maximum storage capacity of small-scale blasting equipment depots shall abide by standards stipulated in GA838 [12].

Site selection, external distance and general layout of mobile blasting equipment depots abide by standards stipulated in GB50089 and other relevant regulations.

Inside underground mine shafts, only small, sub-depots are allowed, and capacity shall not exceed 3 days of production usage for explosives, or 10 days of production usage for priming materials.

Therefore, it can be seen that the *Safety Code for Design of Engineering of Civil Explosives Materials* (GB50089) is fundamental to determination of site and external distance of blasting equipment depots, as well as other aspects such as storage capacity and method of blasting equipment.

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Terrorist Blast Injuries

Min Yu, Jiashu Li, and Xianghong Zhang

1 Section One: Trends in Changes in Terrorist Bombing

Since the 1990s, terrorist attacks around the world have increased drastically, with frequency of terrorist bombings surging exponentially. Forty-four major terrorist attacks with hundred or more injuries and casualties took place in 2015, soaring by a staggering 266.67% compared with two decades ago (1996), and a whopping rise of 51.72% relative to 10 years ago (2006), as shown in Fig. 1. There are many daunting challenges in international security.

In this new age, major terrorist bombings are developing and trending toward new directions: diversification of bombing attack motivations, expansion of scope of attack, broadening of targets attacked, diversification of methods of attack, advancements of weapons used, young age and localization of terrorists, and so on. After the September 11 attacks, internationalism became the top priority in the national security of the USA. Thereafter, counterterrorism and international terrorists began to lock horns.

It can be seen from Fig. 2 that terrorist bombings had been on the rise since 1970. There was a minor drop between 1990 and 2000, but the numbers grew sharply the September 11 attacks in 2001.

1.1 Diverse Motives of Terrorist Bombings

All human actions are driven by root causes. Between the 1970s and 1990s, terrorist organizations were motivated by political and religious reasons to carry out terrorist bombings. But in this new age, terrorist bombings are increasingly driven by more diverse range of motives such as ethnic conflicts, colonialism, racism and hegemonism, making the matter more complicated and varied. As globalization advances, social conflicts become more prominent. Territorial disputes, political stalemates and changes in both domestic and international arenas could all become reasons that spur terrorist attacks. World security has to deal with more serious challenges.

1.2 Expanded Areas of Terrorist Bombings

As the global political landscape shifts, the actions and movements of terrorist organizations also shift. First of all, terrorist bombings had grown exponentially, and areas of attacks continue to expand. In 1990, there were more than 1700 terrorist bombings in 66 countries and regions around the globe. By 2015, the number of terror-

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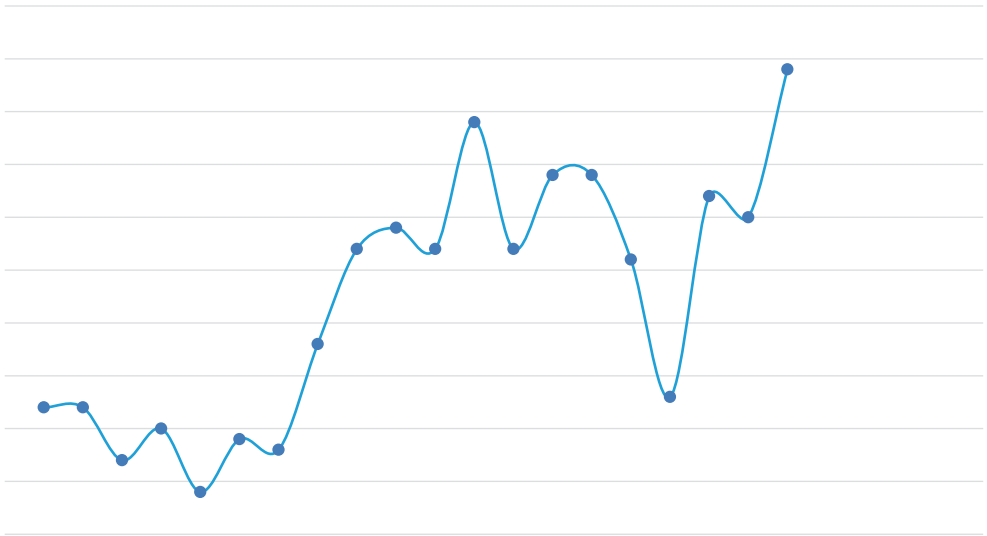

Fig. 1 The number of major terrorist bombings with more than 100 casualties from 1996 to 2015

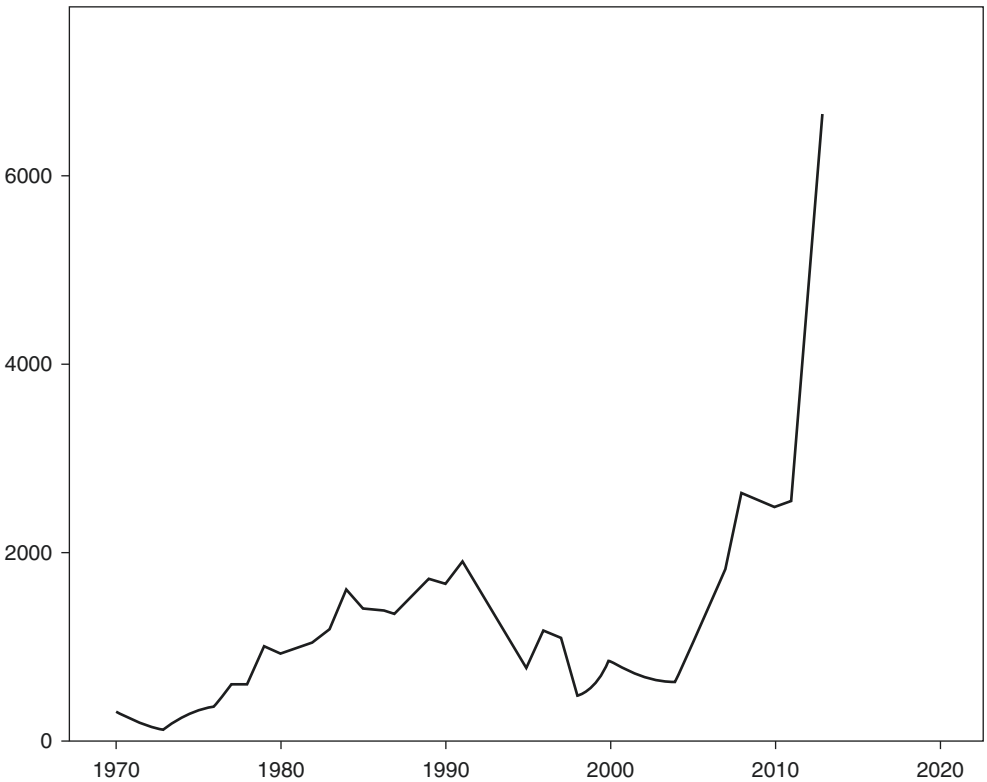

Fig. 2 The number of terrorist bombings worldwide year by year since 1970

ist bombings soared by 3.7 times compared with 1990 to over 7600 cases, while attacked areas climbed by 19.7% compared to 1990 to 79 states and regions. What's more, countries and regions with high frequency of bombings were also changing. In 1990, the majority of terrorist bombings took place in South America (28.9%), sub-Saharan Africa (16.6%) and Western Europe (13.1%), and nations most frequently subjected to bombings were Peru (12.4%), Angola (10.9%), the Philippines (8.1%), Chile (7.6%) and Chile (7.6%). But in 2015, geographical pattern in terrorist bombings had shifted. Attacks most frequently occurred in Middle East and North Africa (48.9%), South Asia (28.1%) and sub-Saharan Africa (7.8%), while countries most often impacted were Iraq (28.9%), Afghanistan (9.7%), Pakistan (8.2%), Ukraine (5.8%) and India (5.2%).

1.3 Spreading Targets of Terrorist Bombings

Generally speaking, regular bombings usually target specific individuals, but terrorist bombings often attack targets with symbolic significance. Looking back at the 1970s, terrorist bombings were clearly driven by political motivations, and main targets were military facilities, government buildings, soldiers and police officers, government officials and other "hard targets" with military and political symbolism. As terrorist attack motivations diversify, attacked targets became more random and obscure, from soldiers, police officers and government officials, to businesspersons and civilians, from military and political facilities to public infrastructures and areas (i.e., train station, park, big shopping mall, etc.), all these could become targets of terrorist attacks. Data between 2000 and 2009 show that civilians have become the most preferred target (27.8%) in bombings perpetrated by terrorist organizations.

Looking back at the major terrorist bombings in recent years, targets of terrorist organizations have broadened from "hard targets" such as military, police and political figures to "soft targets" with weaker defenses. Terrorists are more inclined to choose targets with major political, economic or cultural significance, or areas at times with large gatherings. For example, civil facilities with weak security measures such as luxury hotels, bustling business districts and train stations. On the morning of July 17th, 2009, the Ritz-Carlton and Marriott hotels in the southern part of the Indonesian capital Jakarta, as well as surrounding commercial areas, were the targets of terrorist attacks that killed nine and injured more than 50.

1.4 Networked Propaganda of Terrorist Organizations

The internet is a coin with two sides. The twenty-first century is the Information Age, and the internet has penetrated into every facet of society, bringing convenience but also innumerable hidden dangers. The twenty-first century is the Information Age, and the internet has penetrated into every facet of society, bringing convenience but also innumerable hidden dangers.

In particular, some radicals that are not strict adherents to Islam use online platforms to voluntarily form relatively independent and loose terrorist groups of small sizes. They use the internet to learn ways and techniques to carry out terrorist attacks, communicate with each other to find those of the same ilk, then coordinate and execute terrorist attacks. This makes it harder for law enforcement to notice suspicious activities before an attack is perpetrated or to obtain information about this new generation of increasingly dangerous internet terrorists.

In addition, after a terrorist attack concludes in success, terrorist groups have the penchant for spreading the news through the internet, so as to amplify the terror that already resonates across society.

1.5 Recruitment of Local Youth into Terrorism

One major headache facing European and North American countries in counterterrorism is the increasingly young and localized terrorists. In a terrorist attack perpetrated on July 21st, 2005, that has yet to be concluded, suspects caught were all immigrants from Africa. However, suspects apprehended in counterterrorism actions in Europe in recent years hail from different nations, including those born in Europe. For instance, the 7 July 2005 London bombings that shocked the world were conducted by local British citizens. Investigations indicate that local terrorists are mostly small groups that work independently. These voluntarily formed organizations espouse Islamic extremism, they are highly mobile and extremely dangerous.

It is worth noting that European universities have turned into hotbeds for cultivating terrorists. On the one hand, Islamic extremists are active in universities, and they often use political gatherings and religious study groups to search for potential candidates as conduits to develop local terrorists. On the other hand, some foreign youths take advantage of universities by applying for study abroad openings as a way to sneak into Europe. Many of these applicants are Al-Qaeda members or come from Pakistan and other areas where the Taliban is active. Furthermore, the use of the internet by terrorist organizations to recruit the new generation of European and North American youths is also in an uptrend.

1.6 Expansion of Terrorist Bombing Operations from "Al-Qaeda" to "Lone Wolf"

On the afternoon of July 22nd, 2011, bombing and mass shooting in downtown of Norwegian capital Oslo and on Utøya island resulted in 77 dead. This is a typical "lone wolf" terrorist attack. Suspect Anders Behring Breivik is not affiliated with any international terrorist organizations, and is opposed to the diversification in Europe and Islamic immigrants.

The term "lone wolf" terrorist was first seen in the 1990s. In 1993, white supremacists Alex Curtis and Tom Metzger called on other white supremacists to act alone or carry out small-scale underground activities by imagining oneself as a lone knight or squad, and clandestinely striking against the government or other targets each day. Such a "knight that acts alone" is called "lone wolf." Al-Qaeda incorporates this

concept into its global jihad, and has developed a system of lone wolf terrorist attack strategies.

The counterterrorism document issued by the US Department of Defense in December 2010 states that lone wolf terrorists refer to those that act completely on their own without the instruction of any terrorist organization leader, and at the same time, they did not make contact with other any terrorist organizations while carrying out their political cause or perpetrating terrorist attack.

Compared with organized terrorist attacks, the lone wolf model has various distinguishing characteristics. First of all, there are many reasons that could instigate a lone wolf operation. Some are compelled by Islamic extremist or terrorist ideals, some are driven by economic, emotional or psychological factors, then lone wolves train themselves to execute bombings. Secondly, lone wolves are less picky in their targets, meaning that their strikes are harder to predict. Third of all, lone wolf operations are much more sudden, and lone wolves usually attack "soft targets" such as large gatherings of civilians, making such strikes more dangerous. Fourthly, lone wolves do not belong to monitored terrorist organizations, they maintain fewer contacts with the outside world, so they are more clandestine and more difficult to be identified by law enforcement agencies.

Former US President Barack Obama once said that it is unlikely for the USA to suffer another catastrophic terrorist strike from Al-Qaeda, but the bigger threat is actually lone wolf operations like the aforesaid bombing and mass shooting in Norway.

1.7 Diversified and Integrated Means of Attack

Today, terrorists carry out attacks in more diverse forms. Terrorists may place suspicious items or hoax devices to lure their targets, then pick an opportune time to detonate a powerful explosive to perpetrate a highly targeted terrorist attack.

An alarming fact is that terrorists are no longer satisfied with a single mode of terrorist attack, as an increasing number of perpetrators choose to combine bombing with one or more other forms of attack, thereby maximizing damage, injury, casualty and instilling fear into society.

On the morning of December 5th, 2013, an attacker drove a car laden with explosives into the Yemeni Ministry of Defence and blew up the gate to the compound. Then gunners in a second vehicle shot at those inside the courtyard, followed by an intrusion of armed personnel who engaged in a fierce fire fight with the security stationed inside. This attack resulted in 68 dead and 215 injured. It was clearly a well-organized operation that led to serious consequences. Local media reported that the attack had the marks of an Al-Qaeda operation.

1.8 High-Tech Explosive Weapons

Terrorist organizations continue to recruit technologists and skillful personnel to participate in the development, research and production of new types of explosive devices. As science and technology advances at a breakneck pace, terrorists have also learned to deploy high-tech explosive devices. Take for example the electromagnetic bomb, it is small and easy to carry, not to mention low cost of production and conveniently available materials. It has become a top tool of terror for perpetrators.

To circumvent anti-terrorism surveillance, terrorists spare no efforts in continuously improving the production techniques and detonation methods of bombs, giving rise to weapons such as plastic bomb and liquid bomb. On December 25th, 2009, Nigerian extremist Umar Farouk Abdulmutallab bypassed security check for liquid explosives by hiding powder explosives inside his underpants, then obtained water from lavatory onboard a flight, and used a syringe to inject liquid and detonate the explosives.

2 Section Two: Main Methods and Types of Terrorist Bombings

Based on data tallied between 2012 and 2015, more than 50% of terrorist attacks each year were bombings. Obviously, bombing is the preferred form of attack among terrorists. According to investigation and statistics, there were more than 1800 terrorist bombings in 2007, accounting for 57.5% of terrorist attacks recorded that year. The number of terrorist attacks in 2007 rose by nearly 4% compared with 2006, while the number of people injured from bomb explosions grew by roughly 20% relative to a year ago, and casualties soared by around 30% (Fig. 3, Table 1).

Terrorist bombings may also be executed in a variety of forms, and some of the more common methods and techniques include regular bombs, car bombs, suicide bombs, mail bombs, fixed bag bombs, inflatable boat bombs and so on.

2.1 Bombing

Terrorist bombings refer to bombs directly placed inside or outside buildings or other targets by terrorists, concealed from view, then detonated using delay fuse or remote control in order to kill and injure people, or destroy structures. This kind of sudden bombing is a relatively common form of attack as it is simple for terrorists to implement, and since large equivalent of explosive may be used, the attack could cause substantial damage to target building while killing and wounding many people. Improvised explosive devices (IED)

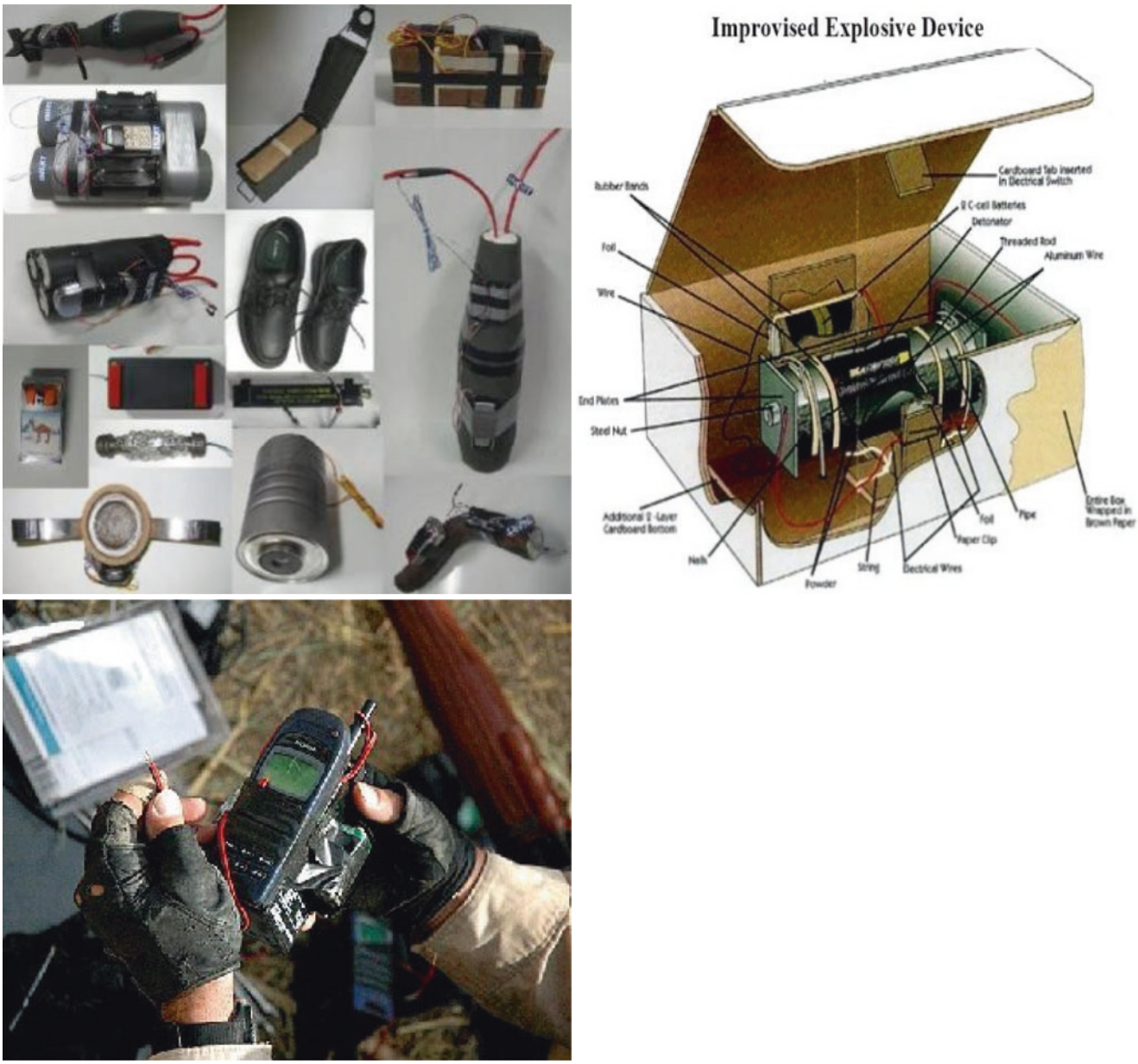

Fig. 3 Various improvised explosive devices

Table 1 Types of terrorist attacks worldwide between 2012 and 2015

	Type of terrorist attack						
Year	Explosion	Armed attack	Kidnapping	Infrastructure facility attack	Assassination	Unarmed attack	Hijacking
2012	62%	25%	4%	4%	5%	23 times	–
2013	57%	23%	6%	6%	8%	Ten chemical or biological attacks	13 times
2014	54%	23%	10%	7%	6%	39 times	42 times
2015	52%	23%	9%	8%	8%	101 times	37 times

are often hidden roadside in places like Iraq and Afghanistan to attack vehicles that pass by and kill and wound people.

2.2 Car Bomb

Car bombs may be divided into moving car bomb and stationary car bomb. A moving car bomb refers to a car laden with bomb (explosive) and driven by terrorist toward a predetermined building or other target, then detonating the bomb (explosive) in a suicide attack. A stationary car bomb refers to a car hidden with bomb (explosive) and parked near a building or other target, then detonated using delay fuse or remote control. Characteristics of car bombs include high equivalent of explosives used, high rate of success, serious damage and severe harms to large number of innocent people.

2.3 Suicide Bomb

A suicide bomb refers to terrorist secretly carrying a bomb on oneself, then detonating the carried bomb at an opportune time once he or she arrives at or approaches the target, with the perpetrator also dying in the process, hence the term suicide bombing. Suicide bombers work clandestinely and are hard to detect. Explosive devices carried by suicide bombers usually have small equivalent, detonation devices are relatively simple and most fuses are triggered either by pulling or electric ignition, while remote control detonation or high-tech detonation gadgets are rare. To minimize sacrifice while maximizing political goal, terrorists often use suicide bombing. Suicide bombing incidents are characterized by features such as extremely high rate of success, massive damage, high rate of casualty and difficult to defend against (Table 2).

Suicide bombing refers to a bomb or other explosive device carried in-person by a terrorist, then detonated to attack a specific target location. In such an attack, the explosive is carried by an actual terrorist on oneself, he or she would approach the target and then detonate the bomb. Such an attack method is extremely random and uncertain, very secretive and thus hard to protect against. Usually the goal is to create large number of casualties and injuries among the target group of people at the cost of very small number of terrorist death. On August 15th, 2017, a group of three female members of the Nigerian terrorist group Boko Haram strapped bombs around their waists and detonated them near the entrance to a refugee camp in Mandarari, Nigeria, killing 28 and wounding 82.

There were 141 suicide bombing incidents around the world in 2010. Though this number only made up 5.6% of total number of terrorist bombings that year, these suicide bombers killed 48.1% and injured 35.6% of total bombing victims.

Table 2 Average wound and death toll in worldwide terrorist bombings between 1970 and 2014

Type of terrorist bombing	Average killed persons/time	Average injured persons/time	Death to injury count ratio/time
Nonsuicide bombing	1.14	3.45	1:3
Suicide bombing	10.16	24.16	1:2.4

2.4 Mail (Package) Bomb

A mail bombs refer to terrorists using letter or parcel to deliver bombs or incendiary devices to predetermined targets, which would explode or ignite and combust when the package is opened, leading to explosion or fire. Mail or parcel bomb usually are not very destructive, and its main objective is to injure people and cause psychological insecurity.

2.5 Fixed Bag Bomb

Fixed bag bomb refers to bombs made by hand using plastic explosives or TNT. Terrorists would hide a bomb in a case or package, then place the item inside a building or near the target building, and use a delay detonator or remote control to set off the explosive so as to kill and injure people, or destroy properties within the area. This kind of sudden bombing is a relatively common form of terrorist attack, particularly with small-scale bombing incidents.

2.6 Inflatable Boat Bomb

This refers to loading an inflatable boat with explosives, then steering the boat toward a vessel or other target in the water. When the inflatable boat arrives in the vicinity of the vessel or target, explosive onboard is detonated to cause damage. The inflatable boat bomb is a novel type of suicide terrorist attack aimed at targets in water. In October 2000, the USS Cole moored in Yemen was the subject of an inflatable boat bombing attack, resulting in serious damage to the hull and the injuries and casualties of a large number of sailors.

With the appearance of new types of explosives and continual advancements in explosive technologies, there are more complicated and diverse options at the disposal of terrorists to carry out clandestine attacks that cause even greater harms to society. Take for example the September 11 attack, Al-Qaeda operative hijacked passenger jetliners and flew them into major targets on the ground, resulting in a tremendous number of casualties and injuries.

3 Section Three: Main Hazards of Terrorist Explosion

Terrorist bombings often rely on extremely asymmetrical method characterized by "low cost for big returns" to bring immense dangers to various aspects of society.

3.1 Threat to Public Safety

Terrorist organizations do not concern themselves with any morals or ethics of humankind, nor do they care about restrictions imposed upon them, and they endeavor to use the most shocking and bloody methods in their bombings so as to maximize the terror and psychological impact on the public.

The numbers of injuries and deaths arising from terrorist attacks have been rising each year, seriously threatening the physical well-being of the public, and depriving many innocent people their right to life and health. Explosions severely damage nearby buildings and infrastructure facilities, seriously harm public and personal properties, and bring about dire and negative impacts on social stability.

3.2 Impact on Domestic and International Political Landscape

To achieve their political goals and intentions, terrorists often choose special times to perpetrate their bombings and attacks, so as to induce fear into the public, foster an atmosphere of unease in society, and in turn disrupt domestic political order. As globalization continues to progress at a rapid pace, countries around the globe have become more dependent on each other, but terrorist bombings directly impact international political landscape. On August 29th, 2003, senior Shiite cleric Mohammed Saeed Al Hakim was killed in a terrorist bombing, plunging the already tumultuous Iraq into further chaos. Chasms in the Shiite sect deepened, the power grab between the Shiite and Sunni sects intensified. The purpose of the 2004 Madrid train attacks in March that year intended to punish Spain for its close relationship with the Bush administration of the USA and allies. The bombings affected the subsequent election in Spain and altered the country's political landscape. In July of the same year, Spain withdrew its troops from Iraq.

3.3 Restriction to Social and Economic Development

Terrorist bombing activities seriously hamper socioeconomic development. First of all, terrorists strike against targets through bombings, creating large-scale casualties, injuries and destruction of infrastructure facilities, thereby causing direct losses to individuals and society. Secondly, governments have to invest substantial amount of funding and human resources to defend against and crack down on terrorist bombings, which account for a sizable chunk of public security costs, in turn lowering other socioeconomic benefits. Third of all, terrorist organizations use illegal methods to acquire capital to fund their activities, resulting in tremendous loss of capital, great disruption to normal economic order and severe harm to national economic benefits; and fourthly, terrorist bombings gravely affect local economic growth, in particular countries and regions highly dependent on tourism.

3.4 Casualty

Explosions mainly cause harm to people through air shock waves and fragments. For people really close to the point of explosion, due to the extremely high overpressure of air shock wave surrounding the area of explosion, usually victims would die right away. Meanwhile, for those situated a certain distance away, air pressure may still have enough power to cause injuries to body parts such as the lungs and eardrums. In addition, after a bomb explodes, surrounding buildings are damaged, creating brick chips, rocks, glass shards, wood splinters and other projectiles that fly at incredible speed. These fragments are very dangerous against humans.

4 Section Four: Analysis of the Characteristics of Terrorist Explosion

Unlike shooting, stabbing or vehicle-ramming attack, the last of which has risen in frequency in recent years, terrorist bombing is very secretive but influential and hard to contain. The organization of a terrorist bombing incident is marked by some unique characteristics.

4.1 Myriad Types of Bombs with High-Energy Explosives Becoming Dominant

As explosive manufacturing technology continues to develop, terrorist bombings have increasingly utilized more diverse and powerful explosives, such as TNT, hexogen, hexolite (composition B explosives), composition C explosives, nitroglycerine gelatin explosives, chlorate explosives, nitric acid explosives and acid explosives. In the past few years, terrorists often use composition C plastic explosives made in the USA to carry out their bombing attacks. For example, the bombings that occurred in Bali in October 2002 were performed using composition C plastic explosives.

4.2 Diversification, High-Tech Orientation and Intelligentization of Explosive Devices

As the battle between terrorist bombings and counterterrorism heats up, terrorists continuously strive to increase the covertness, suddenness, success rate and danger of their attacks by constantly developing new types of explosion techniques. They also use advanced technologies and new products to produce explosive devices, which have become increasingly small, smart, powerful, remote controllable and mobile, among other features.

4.3 Diverse Means That Are Sudden and Difficult to Guard Against

As counterterrorism technologies advance constantly, conventional terrorist bombing methods have become less successful, therefore, terrorists have turned to time bomb, remote controlled bomb, car bomb, suicide bomb, inflatable boat bomb and such other bombing methods and techniques that are more sudden, harder to defend against and more likely to succeed.

4.4 More Extensive Attack Targets and More Serious Damage

Looking back at recent terrorist bombing incidents, a noticeable trend is the increasingly wider scope of targets and higher level of danger. After the September 11 attacks, several large-scale international terrorist bombing incidents show adherence to the trend of wide area of distribution, broad scope of subjects attacked, flexible explosion methods and breakthrough against weak points. Other than targeting symbolic buildings and military facilities, terrorists have begun to strike against well-known tourist attractions, cultural and recreational sites, commercial complexes and other venues with large concentration of people so as to create the most shocking impact.

5 Section Five: Destruction Factors and Damage Types of Terrorist Bombing Injuries

5.1 Factors of Destruction and Destruction Caused by Terrorist Explosion

The direct injuring factors of all explosions include shock wave, high-temperature fireball and projectiles (or fragments from the casing or packaging of the explosive). These include direct blast injuries or indirect blast injuries caused by the shock wave (the latter also includes crushing injury due to toppled buildings, suffocation from being buried, injuries from rocks, glass shards, wooden sticks, metal objects and other items that fly at high speed, etc.), direct burns from the high-temperature fireball and/or burns from flammable objects ignited by the fireball, and perforating wound and/or penetrating wound directly inflicted by high-speed projectiles.

These injuring and killing factors could cause injury on their own, but could also injure a person at the same time, producing composite injury. In most cases, especially during a powerful explosion, composite injuries are the most common. This is why "blast injuries" in terrorist bombing incidents are usually complicated injuries that worsen each other, resulting in severe situations that exacerbate quickly, which are hard to treat, and result in high death rate including death on site (Fig. 4).

5.2 Main Types of Terrorist Explosion Injuries

5.2.1 Direct Impact Injury (Blast Injury)

Direct impact injury here is also known as blast injury, and is serious injury caused directly by the dynamic pressure and overpressure, or even negative pressure, of the blast wave. These could be severe and critical, deadly acute massive blood loss due to rupturing of liver, spleen or major blood vessel; pulmonary hemorrhage and edema or breathing difficulty due to obstructed respiratory tract; cardiac failure due to rupture of myocardial fibers and hemorrhage; severe single or multiple fractures resulting in paraplegia or punctured blood vessels and so on. An explosion may also cause moderate or lighter injuries, with the former possibly turning into life-threatening problem very quickly, demanding on-site first aid, while the latter could be delayed to a certain extent.

Direct impact injury from an explosion is usually caused by dynamic pressure and overpressure, although negative pressure is also dangerous but to a lesser extent.

Impact and Displacement by Dynamic Pressure

Dynamic pressure in the blast wave of an explosion exists in the form of a powerful blast wind that may directly impact the body, or cause bone fracture, dislocated joint, ruptured liver and spleen, craniocerebral injury, internal organ bleeding, body surface and soft tissue tears, or even ruptured body cavities and severed limbs by displacing the body, with injury occurring when the body is thrown against some object or upon landing. At the same time, dynamic pressure could also act on the body indirectly via high-speed rocks and sands, causing perforating wounds to walls and cavities of the body, or obstructions in respiratory tract. These are called indirect injuries.

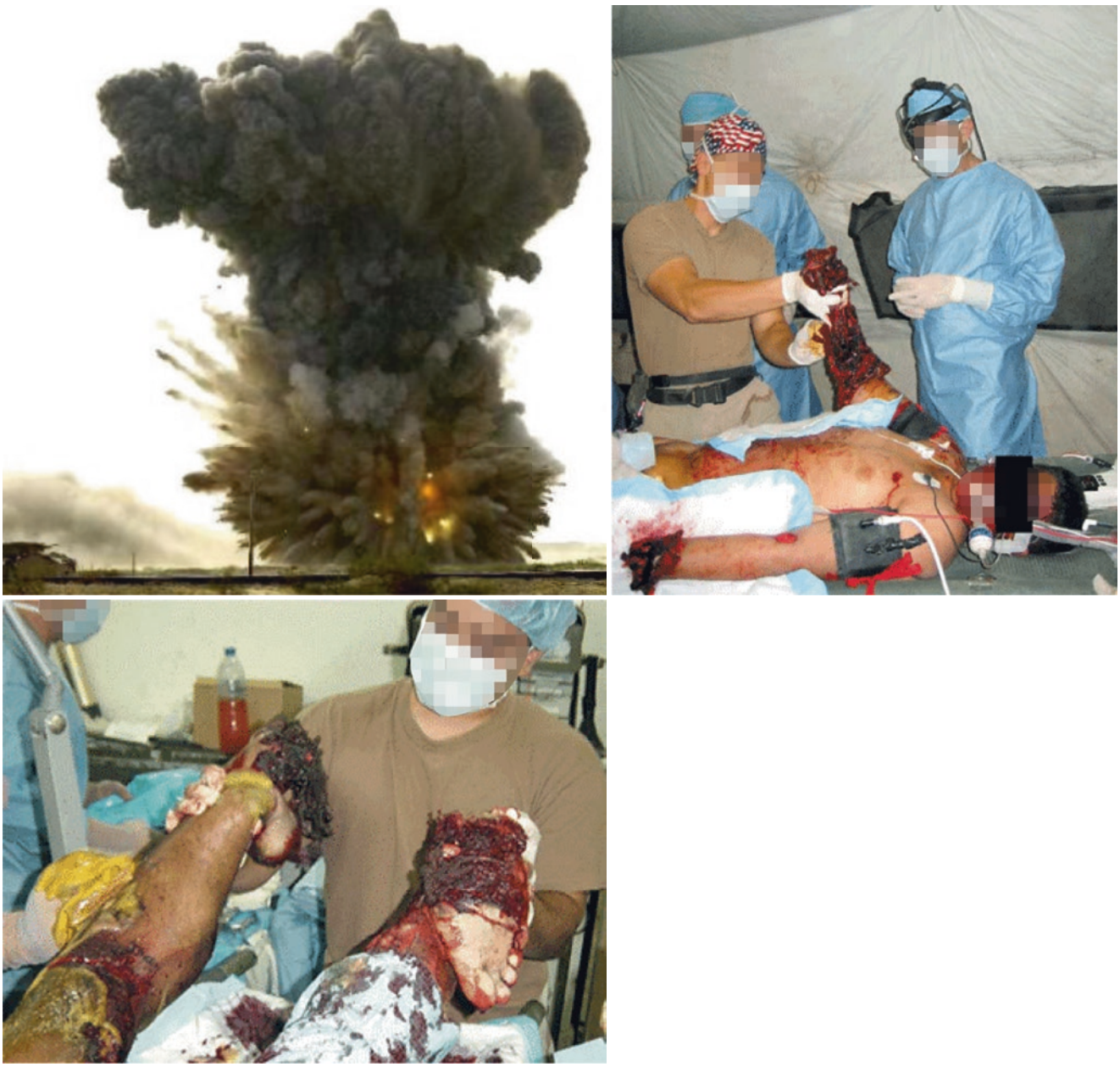

Fig. 4 Roadside bombs and the injuries caused

Rapid Squeezing Effect Directly Caused by Overpressure

Overpressure in the blast wave of an explosion refers to the part of pressure that exceeds normal atmospheric pressure, which causes injury by directly squeezing the body from different sides. Although this is similar to being squeezed by water when diving deep underwater, but the pressure is applied suddenly, with peak pressure, which is often very high, reaching maximum level almost immediately. Since organs containing gas or liquid such as the auditory apparatus, heart, lungs, stomach, intestines and bladder are very sensitive to sudden changes in atmospheric pressure, thus overpressure mainly inflicts harms upon these organs, with auditory apparatus and lungs being the most susceptible to injury (often accompanied by damage to the heart as well).

1. Direct effects: When overpressure enters the external auditory canal, pressure is directly applied on the eardrums, causing pressure difference inside (tympanum) and outside the eardrum (external auditory canal), and leading to eardrum rupture, bleeding and auditory ossicle fracture.
2. Hemodynamics effects: When overpressure acts on the abdominal wall and thoracic wall, abdominal pressure

would rise, pushing the diaphragm upward and suddenly forcing excessive volume of abdominal venous blood to rush into the heart. This leads to sudden jump in cardiopulmonary volume, abruptly decreasing chest volume, in turn resulting in rapid surge in internal pressure of the chest. Right after being subjected to overpressure, the effect of negative pressure expands the thoracic cavity. This sudden compression and expansion together cause a series of hemodynamic changes within the thoracic cavity, leading to injury to cardiac and pulmonary vessels, or disorders in cardiac and pulmonary cycles.

3. Implosion effect: When a shock wave's overpressure acts directly on the body, more gaseous components in the body are compressed, while less liquid components are compressed. Then right after the effect of overpressure is the action of negative pressure, and at this stage the previously compressed gaseous components swiftly expand as they transmit energy to surrounding tissues in a radial manner. This sort of "explosion" injury is called implosion effect. For example, gaseous components in alveoli are first compressed then expanded like many little "explosion sources" that would cause tear or bleeding of alveolar wall.
4. Fracture effect: When the stress wave of a shock wave passes from relatively dense media (such as solid parts of organs) to relatively loose media (such as liquids and gases), reflection would occur at the contact interface between the two, and injuring the dense medium due to an abrupt rise in surface pressure. For example, subendocardial hemorrhage, hemorrhagic edema in the alveolar wall and hemorrhage in filled bladder and gastrointestinal tract belong to this category.
5. Inertia effect: The velocity at which a shock wave propagates in different tissues varies due to inertial differences. A shock wave propagates faster in less dense tissues, but slower in denser tissues, and the contact point between two tissues of different density is susceptible to injuries like tear and bleeding. Hemorrhage at the junction of the mesentery and intestine is an example in this category.

Relatively Light Injuring Effects of Negative Pressure

The negative pressure of a shock wave refers to the part of the pressure below regular atmospheric pressure at the rarefaction zone. Negative pressure causes air to move in the opposite direction. The current general consensus is that negative pressure has similar injuring mechanism as overpressure, and could also cause similar impact injuries, but its peak value and injuring effects are much more moderate than those of overpressure, thus only able to inflict some minor blood vessel injuries.

Direct impact injuries are always multiple organ injuries with numerous pathogeneses occurring at the same time. Most injuries are closed injuries and composite injuries.

5.2.2 Indirect Impact Injury

A shock wave could also injure a person indirectly through the following two methods: First of all, the powerful dynamic pressure of a shock wave could launch some objects (i.e., sand, rock, glass shard, etc.) that would cause body injury as if shot by a projectile. Second of all, structures (such as residential buildings), fortification, equipment and other objects may be damaged or toppled, and injuring people by crushing or collision in forms such as crushing injury, craniocerebral injury, bone fracture, internal organ rupture, bleeding and so on.

Furthermore, high-temperature and high-speed sand and dust kicked up by a shock wave could enter the oral cavity and respiratory tract, inducing airway obstruction. The scorching hot dust may also cause burns in the respiratory tract and oral mucosa, which could lead to very severe consequences. When different types of explosive weapons blow up in urban areas, the area in which indirect impact injury is inflicted, and the number of people afflicted with such type of injury, could be much more and much larger than those of direct impact injury, in particular areas with high density of buildings. Among the blast injury victims in the March 11 Madrid attacks, and July 7 bombings of the London Underground and public bus, more than 80% were indoors, with the absolute majority suffering from this kind of indirect impact injury.

5.2.3 Fragment Injury

Fragment injury or shrapnel injury is a serious injury caused directly by metal or nonmetal shrapnel (as in fragment) that fly at high speed after the explosion of an explosive. These projectiles could strike important body parts (such as the head, face, neck, chest, abdomen and limbs), major organs (brain, heart, lungs, liver, spleen, kidneys, bone marrow, major blood vessels, etc.) and cause one or multiple or even critical perforating injuries (or deep penetrating injuries). Such injuries could become life-threatening very quickly, and requires immediate on-site treatment. They could also result in moderate or milder penetrating wounds beneath the skin surface or in noncritical organs, and in such situations delayed treatment may be considered. Statistics show that among injuries caused by modern terrorist attacks, the injuring range of fragment injury (shrapnel injury) and the ratio of victims of this type of injury are much larger and higher than impact injury. Furthermore, this type of injury is usually the main culprit behind on-site or early deaths.

5.2.4 Burn

A high-temperature fireball is formed at the moment of explosion of an explosive. This fireball could have a high-temperature range of around 15–40 m, with temperature rising to some 3000 °C within several meters of center of the blast in a time of 23 ms after explosion, and temperature

reverting to normal at around 3 s. This temperature level at such duration is sufficient to cause direct burns to skin and special parts of the body (head, face, respiratory tract, perineum, etc.). However, indirect burns or secondary burns caused by flames from the ignition of various flammable items are even more common. Of which, severe or more severe burns (second-degree burns covering more than 20% of body surface, or third-degree burns covering more than 5% of body surface) would often worsen to shock very quickly, requiring on-site emergency rescue. In particular, antishock measures and ensuring the respiratory tract stays free of obstruction are crucial to keeping a victim alive, while burns of moderate or lesser extents may receive delayed treatment. In the event of a terrorist attack, pure burns are actually rather rare, as the majority of injuries are coupled with impact injuries and/or fragment injuries (as in burn–blast combined injury or blast–burn composite injury, burn–fragment composite injury or fragment–burn composite injury, or even burn–fragment–blast composite injury or blast–fragment–burn composite injury). The clinical expression, pathological process, local pathogenesis of burn on skin and pathological changes of internal organs, as well as their treatment measures, of an explosive burn are basically similar to regular burns.

5.2.5 Other Injuries

If a terrorist bombing is accompanied by nuclear (radiation), chemical (i.e., vesicant agent, choking agent, toxic agent, etc.) or bioterrorist attack (i.e., bacteria, virus, rickettsia, etc.), their corresponding injuries will also occur. Due to the compounding effect of composite injury, victims are prone to quickly go into life-threatening shock, thus requiring on-site emergency rescue, while injuries of moderate or lesser extents may receive delayed treatment.

Among the direct injuries inflicted by any of the injuring factors from the explosion of a high-energy explosive, in terms of the scope of injury, fragment injuries are usually more frequent in wide-open areas, followed by impact injuries, and then burns. In terms of the extent of burns, due to the short sustained duration of the high-temperature fireball (usually just several seconds, or even shorter), thus most burns are inflicted on the surface, often times resulting in second-degree burns or less. Burn area is relatively small, and most are moderate or milder burns. However, if the explosion occurred indoor in an urban area, or in a cave, or if there are flammable items that caught on fire, burns would be particularly severe especially when there is a dense concentration of people. Some victims may even be burned to the point of charred throughout the whole body. Generally speaking, when a highly powerful explosive or some sort of special explosive blows up, burns may become one of the most common forms of injury. When equivalent increases, the proportion of burn injuries also increase.

5.3 Different Levels and Types of Terrorist Bombings

Injuries from an explosion may be categorized as one of the four levels below:

Primary blast injuries are caused by direct impact from a sudden surge in ambient pressure. The amplitude, increase duration and sustained duration of ambient pressure all affect the biological impacts of primary blast injuries. Such impact injuries appear to be mild on the surface, but organ contusions are severe. Blast lung injury is one of the main reasons of death on the scene.

Secondary blast injuries are also known as projectile wounds, which come from fragments or other objects that are propelled forward with gas moved by the release of powerful energy from the explosion. Such projectiles could cause dismemberment, abdominal wall tear, and substantial injuries to soft tissues. A rising number of terrorists have learned to wrap bombs in metal objects in order to augment penetration and worsen traumas inflicted.

Tertiary blast injuries partially arise from people being launched by the force of the blast wave and then colliding with some other object, resulting in injuries to bones and penetrating traumas. Some other injuries are caused by the tremendous heat generated from the explosion. In general, the tremendous heat would affect body parts exposed, such as the face, neck, hands and calves of victims.

Quaternary blast injuries are also called combination blast injuries. These mostly come from secondary incidents, such as inhalation of toxic gas, collapse of building, fallen electric cables, secondary explosions of vehicles or other factors that lead to composite injury.

Based on literature and articles, the average numbers of victims of different types of injuries and their proportions of total in a terrorist attack are shown in Table 3.

Table 3 Average numbers of victims of different types of injuries and their proportions of total in a terrorist attack

	Injury type	Average number (standard deviation)	Proportion of total victims (%)
Primary blast injury	Blast lung injury	17.70 (±29.01)	6.77
	Eardrum injury	38.63 (±56.17)	14.77
Secondary blast injury	Gastrointestinal injury	1.25 (±1.64)	0.47
	Chest injury	21.7 (±27.82)	8.30
	Abdominal injury	13.92 (±23.34)	5.32
	Head and neck injury	80.36 (±205.19)	17.45
	Hand and foot injury	49.59 (±70.07)	18.96
Tertiary blast injury	Fracture	26.66 (±25.47)	10.19
	Amputation	6.63 (±8.54)	2.53
Quaternary blast injury	Burn	39.74 (±53.98)	15.19

6 Section Six Main Features of Terrorist Bombing Injuries

Terrorist bombings occur suddenly and creates large numbers of injuries and deaths within a short time, bringing about severe impacts to both the physiological and psychological states of people.

6.1 Multiple Causes, Complicated Conditions and Serious Damage

There are many factors that could cause injuries in a terrorist bombing. One category is direct injuring factors, principally referring to shock wave, heat, toxic gas, projectiles and other factors directly generated by the explosion that injure victims. Another category is indirect injuring factors, referring to the collapse of nearby buildings, combustion of materials, stampede caused by people chaotically and other factors induced by the explosion that injure victims. In a terrorist bombing incident, most victims suffer from composite injuries arising from multiple injuring factors that act on and even intensify and expand upon each other (i.e., impact injury, burn, inhalation injury, projectile wound, crushing injury, collision injury, etc.). Terrorist bombing and attack victims usually have complicated injury conditions, with both internal and external problems, often times at severe levels. Other than external wounds, most victims would suffer from different degrees of injuries in various internal organs. Of which, the lungs are especially sensitive to shock wave and are most likely to suffer from the worst injuries.

6.2 Clear Direction

Generally speaking, the amplitude of pressure generated by the explosion is inversely proportional to the square of the radius from the center of the blast. The severity level of blast injury is closely related to factors such as type and strength of explosive, range of effect from the center of the blast, and environment in which the explosion takes place (Table 4).

6.3 Sudden Occurrence with Many Wounded

Terrorist attacks are often covert before they occur and there are not any signs beforehand. In addition, explosions are often powerful, while at the same time they take place in areas of high population and building density. Therefore, the outcome of a terrorist attack is usually a huge number of wounded (as little as a few or a dozen or so, but up to hundreds or even thousands in major incidents such as the September 11 attacks in the USA), bringing about great difficulties for on-site rescue efforts.

Table 4 Impact of explosion environment on injury condition

	Explosion environment	Average number	Standard deviation	*p*-value
Casualties	Enclosed environment	14.20	17.828	0.842
	Open environment	15.63	14.071	
Injuries	Enclosed environment	179.22	252.509	0.840
	Open environment	192.70	141.147	
Blast lung injury	Enclosed environment	13.53	16.230	0.728
	Open environment	10.67	16.657	
Eardrum injury	Enclosed environment	47.69	62.557	0.024[a]
	Open environment	2.80	2.775	
Chest injury	Enclosed environment	12.85	18.556	0.432
	Open environment	28.00	32.762	
Abdominal injury	Enclosed environment	7.09	9.203	0.219
	Open environment	3.33	1.966	
Head and neck injury	Enclosed environment	33.56	46.345	0.172
	Open environment	14.40	15.437	
Hand and foot injury	Enclosed environment	22.57	26.972	0.209
	Open environment	38.71	25.960	
Limb fractures	Enclosed environment	20.56	20.957	0.567
	Open environment	35.50	47.149	
Amputation	Enclosed environment	3.25	2.006	0.018[a]
	Open environment	8.00	4.243	
Burn	Enclosed environment	36.53	50.122	0.907
	Open environment	41.00	76.870	

[a] Statistical significance $p < 0.05$

6.4 Multiple Complications and Rapid Deterioration

Conditions of blast injury victims deteriorate very quickly, usually peaking within 6 h after injury, and sometimes at around 1–2 days. Once the body's compensation capability loses control, conditions could spiral and render treatments almost useless.

6.5 Heavy Casualties with a High Rate of Mortality

Terrorist bombings take place abruptly, and explosions are powerful, thereby causing relatively large numbers of direct casualties on-site. In addition, the scenes of such bombings are usually very chaotic, which further complicate the situation for rescuers. Many severely injured victims do not have many chances at surgical operation, treatments in later stages are difficult, and ultimately these circumstances result in the rise in the number of deaths. Statistics indicate that a total of 5080 terrorist bombings occurred in 2012, of which, 2665 resulted in injuries and deaths, with an average fatality rate of 20.0% (the highest fatality rate being 92.9%).

6.6 Severe Psychological Trauma

Victims of a terrorist attacks usually exhibit very strong stress response, including fear, nervousness, incoherence, fidgetiness, dysfunction of autonomic nerve, irregular heartbeat, shortness of breath, sweating, trembling, inarticulate speech, confused logic and other symptoms. Some may even find it difficult to get out of bed, or experience nervous breakdown within a certain duration. In most cases, fear and anxiety are the most common psychological features, and the majority of victims show emotion reactions such as avoiding being attacked or trying to escape. These situations not only occur in regular people, but may affect rescuers as well.

7 Section Seven: Medical Aid of Injuries Caused by Terrorist Bombing

Terrorist bombings take place abruptly, causing a large number of injuries and casualties, and medical aids are difficult in both organizational and technical aspects. In particular, if the terrorists responsible aim to maximize the terrorizing effect of the incident, they may initiate a second attack as rescuers and first-responders arrive on scene. Thus, terrorist bombing rescue ought to follow the principles below:

7.1 Rescue Principles

7.1.1 Safety First Principle

Terrorist bombings take place suddenly, bombs are often very powerful, main buildings on-site may have been badly damaged, and do not forget potential safety risks in a possible second explosion or subsequent problem (such as collapse of building). Rescuers must abide by the safety first principle, especially in prioritizing one's own safety and that of victims.

7.1.2 Joint Self-Rescue and Mutual Rescue Principle

When an emergency situation like a terrorist bombing takes place, situation at the scene is chaotic and everyone is basically out of control because of overwhelming fear. Before external medical resources arrive at the scene, on-site self-rescue and mutual rescue are crucial. Victims should try to swiftly and safely relocate themselves away from the site of explosion, and take active measures to help themselves and others.

7.1.3 Save Lives Before Treatment Principle

When preparing for a terrorist bombing, terrorists often include considerable amount of nails and other sharp objects in the explosive to maximize injuring and lethal effects, and worsen harms inflicted to people. When a terrorist bombing incident occurs, a huge number of victims would appear on-site at the location of the blast within an extremely short time span. These victims would suffer from a diverse range of complicated injuries of varying extents.

Rescuers and first-responders need to adopt the save life before treatment principle, as in saving the lives of those critically injured victims first and carrying out necessary on-site treatment to prevent or treat shock and relieving suffocation problem, then evacuate them to medical care institutions for further treatment. This principle can maximally minimize on-site death rate.

7.1.4 Unified Command Principle

Organizational command with unified coordination is key to maximizing success in rescue effort for a terrorist bombing incident. Rapidly and effectively establishing on-site first aid command structure is extremely vital. Different levels of medical care institutions should formulate corresponding prearranged plans, and proactively establish on-site command organization composed of management personnel and medical experts. In the case of a terrorist bombing incident, quickly initiate the prearranged plan and realize unified organization and highly effective command.

7.1.5 Categorized Evacuation Principle

The immense number of victims on-site makes it difficult to organize emergency treatment and evacuation. To make the most out of limited emergency rescue and evacuation resources, it is necessary to effectively organize on-site emergency rescue and evacuation by way of appropriate categorization. Prioritize those more severely wounded over less seriously wounded victims, strictly abide by evacuation indications, and for severely injured victims, it is imperative to first carry out any necessary on-site treatment, then select suitable transportation method for evacuation, and ensure safety along the route before performing the actual evacuation procedure.

7.2 Organization of Medical Aid

Appearance of large number of victims within a short period, and victim injury conditions are complicated and severe. Organization of medical effort, whether on-site or at hospital, is particularly crucial.

7.2.1 Organization of On-Site First Aid

Organization of on-site first aid includes the inspection and categorization of victims at the scene, and organizing the implementation of on-site first aid.

Basic Requirements

The following tasks must be undertaken in earnest: (1) Organs and institutions at different levels must make prearranged plans, appoint personnel to take charge of this task in a part-time manner, and ensure coordination between military and local resources. (2) In the prearranged plans, other than medical rescuers, those from firefighting, defense, disease prevention and control, and other organizations should also be included. (3) Organization structure of medical rescue should be divided into command group, categorized search group, on-site emergency rescue group, on-site temporary treatment group, evacuation group, assistance group, etc. The command group is in charge of leadership, command and coordination tasks; the categorized search group categorize victims discovered and send them respectively to on-site emergency rescue group, on-site temporary treatment group or evacuation group; the on-site emergency rescue group should be composed of capable anesthetist, internal medicine personnel and surgeons, as well as experienced nurses, and they are responsible for on-site resuscitation and emergency rescue of critically wounded victims; the on-site temporary treatment group is in charge of dressing wounds, stopping bleeding and fixating bone fractures; the evacuation group is tasked with sending victims, both those that have received temporary treatments and those that do not require treatments, to nearby hospitals or designated medical care institutions; the assistance group is responsible for the normal necessities of the other rescuer, as well as medical device, equipment, medicine, etc.

Main Tasks

Task 1: the inspection and categorization of victims on-site. Due to the wide range of explosive weapons or devices that may be used for a terrorist bombing, injuring factors may be complex, resulting in many different injury types, injury forms and injury conditions, with a high proportion of severely wounded victims. It is suggested to carry out treatment and evacuation of victims in a quick and orderly manner, first and foremost it is imperative to stick to the injury categorization principle. Thorough injury inspection should be conducted at the scene, to determine the injury type and injury level of each victim, and undertake on-site emergency medical treatment in accordance with specific situations, thereby reducing the chances of missed diagnosis or erroneous diagnosis. Victims should be classified into four categories of mild, moderate, severe or critical injury. When performing categorization, it is necessary to make accurate judgments on injury type, injured body part(s), injury development trend and injury condition, so as to facilitate the performance of effective treatment measures in a prompt manner. **Task 2: the emergency rescue of victims at the scene.** Emergency rescue of victims at the scene of a terrorist attack is basically similar to emergency rescue contents of those wounded on the battlefield. Principal contents include administering emergency treatments for five types of potential life-threatening or gravely consequential injuries (as in the "five major procedures" of stopping bleeding, fixating bone fracture, dressing wound, preventing shock and keeping respiratory tract free of obstruction), along with other emergency rescue procedures. When administering emergency treatments to victims at the scene, pay attention and follow the principle of treating those severely wounded first before tending to those more mildly injured, prioritize treating victims with life-threatening injuries, organize the public to help themselves and help others, so as to minimize on-site fatality rate. After taking active measures to prevent or treat shock and relieving suffocation problem, victim should be evacuated to emergency rescue station, specialized hospital or emergency medical center as soon as possible for further treatment.

The Search and Rescue of Victims Buried at the Scene of the Blast

For the search for victims that may have been buried on-site, other than relying on people and police dogs, it is even more important to take advantage of new types of noncontact detection technology and detection devices so as to make sure that critically injured victims with weak breathing and heartbeat, and even the dead, may be found in time.

When digging up victims buried at the scene of the bombing, carry out excavation or demolition by organizing human resources and advanced machinery and equipment, and make sure that buried personnel do not subject to secondary injury during this process.

The basic principle of on-site emergency rescue of buried victims is to maintain vital sign, and the most important measures are to stop bleeding and keep respiratory tract free of obstruction. At the same time, remove buried victims from the site of incident as soon as possible. Pay special attention to on-site emergency rescue of victims suffering from crushing injury. Actual on-site emergency rescue efforts conducted after the Wenchuan earthquake and Yushu earthquake show that victims afflicted with crushing injury remain conscious before being discovered, but within just several minutes after being found, or during the subsequent lifting and evacuation process, they would quickly or suddenly die.

The Medical Evacuation of Victims On-Site

The medical evacuation of victims refers to the process of transporting victims from the site of the explosion after a terrorist attack to a specialized hospital or emergency medical center. The medical evacuation of victim is not a simple process of transportation, but also the "continual medical care" during this course of transportation. Therefore, the medical evacuation of victim is an important content and one of the key steps in the on-site rescue of victims after a terrorist attack, as well as a crucial feature in the treatment of various types of trauma resulting from a terrorist incident. The medical evacuation of victim after a terrorist attack may be conducted via different transportation methods and modes. A multidimensional medical evacuation system with stretchers carried by personnel and vehicles complementing each other, and land, air and maritime routes working together. Specialized transportation vehicles should function as the main force, and combine land, sea and air capacities, so as to ensure that victims receive subsequent treatment in a timely manner.

During the medical evacuation process, pay attention to the following points: (1) Strictly monitor evacuation indicators that may reveal potential risks in unconsciousness or suffocation, as well as other possible dangers during the course of evacuation. It is necessary to first properly perform on-site emergency medical treatment before the start of evacuation, including preparations for the evacuation such as continual blood transfusion, fluid transfusion, oxygen supply and other continual monitoring and uninterrupted treatments. Dedicated medical personnel should also be arranged to accompany the evacuating victims. (2) Strive to choose and use the most appropriate mode of transportation. It is necessary to place victim in a suitable position for the evacuation, so as to avoid injuring the victim again during the course of evacuation (i.e., shaking of the car en-route and worsening conditions by causing dislocation of fractured bone, or blood vessel rupture).

7.2.2 Organization of Treatment in Hospital

Many victims that have arrived at hospital after receiving on-site emergency treatment need to continue further treatment. Injured people victimized by a terrorist bombing always arrive at a hospital in droves, thereby requiring that hospitals arrange subsequent treatments based on highly efficient organization of specialized treatment.

Swiftly Initiate Prearranged Emergency Plan

After a terrorist bombing takes place, relevant rescue management authority needs to notify relevant hospitals to prepare for arrival of victims based on actual circumstances and needs. Hospitals should initiate prearranged emergency plan swiftly, make all necessary preparations, open up green channels for treating large groups of victims, and involve the whole hospital to implement grade I rescue action for major incidents. It is necessary to quickly establish rescue command group and experts group, and notify backup medical personnel of the emergency department and members of the hospital's emergency response group.

In accordance with notifications from superior organs and protocols in prearranged, generally speaking, personnel are divided into the command and contact group, categorization group, emergency rescue group, debridement and suture group, observation group, transfer group, and coordination and documentation group. (1) The command and contact group should be made up of hospital leaders, emergency department directors and head nurses, and this group is responsible for instructing on-site emergency rescue efforts. They need to notify all examination departments, medical imaging departments, the pharmacy, disinfection supply department, medical device department, economic management, department and other related departments of the hospital to get ready, so as to ensure the treatment process stays smooth, and required material support are provided in a timely manner. (2) The categorization group should be made up of experienced physicians and nurses from the emergency department. Their job is to tag victims with numbered wristbands in the proper order, and quickly categorize victims based on injury inflicted, then swiftly and reasonably send them to the right destination. (3) The emergency rescue group should be made up of physicians and nurses seasoned in emergency rescue and care. Members of this group should adhere to the first diagnosis responsibility system, with each member taking responsibility for the patients in beds specifically assigned to them. At the same time, members in this group are also charged with monitoring and caring for specific patients as they depart for examinations and during the course of such movements and transfers. (4) The debridement and suture group is in charge of the debridement and

suture procedures of victims, including other treatments and processes such as dressing and fracture fixation. (5) The observation group is in charge of technical measures and other issues related to blast injury treatment of victims of terrorist bombing with moderate level of injuries. (6) The transfer group is in charge of accompanying mildly injured victims during their examinations, assistance with distribution and transfer, collecting samples to be examined, and prompt retrieval of examination results. (7) The coordination and documentation group is in charge of filling out "emergency incident victim summary table" and converting the information into electronic format. Info should include a victim's name, gender, age, personal ID number, diagnosis, injury condition, whereabouts, physician responsible for first diagnosis, nurse, home address and contact phone number. Such information should be amended in a timely fashion, and this group's members shall report to the command and contact group every half an hour regarding dynamic changes among the victims.

Prepare Emergency Drugs and Medical Devices

Based on the features of blast injuries, it is necessary to keep in contact with the hospital's disinfection supply department so as to maintain timely replenishment of debridement and suture instrument set, tracheotomy instrument set, various dressings, elastic bandages and so on. Also, the pharmacy should be contacted to provide the necessary tetanus antitoxin, hemostatic drugs, antibiotics, normal saline and so on, and have them ensure the supply of medicines. Set up emergency beds at the emergency rescue zone, inspect beds in debridement room, make room for the treatment of mildly injured victims in the fluid transfusion hall, and coordinate an adequate number of beds for patients to be put under observation. Appoint specific personnel to make regular preoperative preparations in emergency surgery rooms.

Rapid Triage

Emergency rescue room physicians should make quick triage of a victim based on five aspects, namely his or her injured body part(s), injury type, circulatory signs (blood pressure and pulse), breathing and consciousness, then categorize each victim for the kind of treatments to be administered. Categorize victims into either minor, moderate or severe injuries, then admit them to the corresponding treatment zone. After patient zoning and administering treatments for 2 h, treatment expert group should reevaluate all patients. Patients with severe injuries should be admitted into the red zone (emergency rescue zone) through green channel, then delivered to emergency ward. Those with moderate injuries should be observed further, while victims with minor injuries should undergo debridement and suture treatment, then sent to transfusion room for further treatment and observation.

Organize and Carry Out Effective Treatment

The multiple injuries arising from an explosion require, first and foremost, treatment measures including keeping respiratory tract free of obstruction, controlling bleeding, maintaining adequate perfusion, and when necessary perform emergency surgery. After receiving a victim, quickly undertake injury condition assessment, first deal with life-threatening injuries before handling soft tissue injuries, and conduct debridement and suture in a timely manner. After injury, administer antibiotic drugs right away to prevent infection. At the same time, inject tetanus antitoxin to prevent tetanus infection, and examine the patient by way of means such as X-ray, CT scan and B-mode ultrasound. According to patient categorization, organize relevant departments to carry out specialized treatments.

Earnestly Conduct Psychological Counseling for Victims and Their Families

In most cases, victims of a terrorist bombing would also suffer from different degrees of anxiety and fear, expressed as worries about prognosis, fear of injury and disability, and urge to receive the best treatment and nursing care, among other manifestations. During the whole treatment process, medical personnel should try to understand the negative emotions that victims exhibit. Arrange psychological counselors to conduct psychological counseling for victims and their families, while at the same time making proper food and rest arrangements for victims and their families.

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Explosion Incidents

Liang Zhang

Explosion incidents and blast injuries are not uncommon in everyday life, and they are often reported in the news, including various types of terrorist attacks as shown in international news including suicide bombers and car bombs. Domestically speaking, the absolute majority of explosion incidents are accidents such as those related to coal mine gas, chemical factory, firework and firecracker production, town gas or propane tank. This chapter is a collection of case analyses of different reasons of explosions, and provides an overview about various types of explosion incidents.

1 Section One: Terrorist Attack and Bombing

Terrorism refers to the intention and action of utilizing means such as violence, destruction and threat to generate fear in society, cause harm to public safety, encroach on personal property, or to coerce national organs and international organizations to realize the political and ideological purposes of the perpetrators. The United Nations regard terrorism as "war crime during time of peace."

Relative to weapons of mass destruction like biological, chemical, nuclear devices or dirty bombs, regular bombs are economically more tolerable, materials are easy to acquire (the cheapest materials such as gasoline are basically obtainable without much difficulty or expenditure), simple to produce, diverse in their detonating mechanism, and simple to make and use after brief training, among other features. Therefore, among all terrorist organization actions, bombing is the most popular and common form of attack. Between 1969 and 1983, some 200 terrorist bombing incidents took place around the globe, and in the following decade 3163 bombings occurred. According to data from the Global Terrorism Database (GTD), between 2001 and 2008 there were a total of 8928 terrorist bombing incidents, accounting for 53.7% of total terrorist attacks during that period. Among terrorist bombings, suicide bombing is the most drastic as they produce bigger psychological and political impacts. A study by the Institute for National Security Studies (INSS) of Tel Aviv University states that 735 attackers perpetrated 735 suicide bombing incidents in 2015, with 4330 dead and 8800 injured. 2016 saw more deaths from terrorist attacks than any other year, as a total of 800 terrorists perpetrated 469 suicide bombing incidents in 28 countries around the globe, with 5650 dead and 9480 injured.

1.1 Typical Cases of Terrorist Bombing

In the past three decades, there have been numerous terrorist bombing incidents that caused serious deaths and injuries among civilians, as listed below:

1. Oklahoma City bombing. On April 19th, 1995, 27 year-old American Timothy McVeigh parked a truck laden with 7000 lb of mixture composed of ammonium nitrate and fuel (power equivalent to 2 tons of TNT) by a nine-story federal government building in Oklahoma City. McVeigh then detonated the truck, causing an explosion that resulted in the death of 168 people, including 19 children, while injuring more than 500.
2. On August 7th, 1998, terrorist organization Al-Qaeda used bombs to strike American diplomatic buildings in the Kenyan capital of Nairobi and the Tanzanian port city of Dar es Salaam, killing 253 people.
3. On September 13th, 1999, a powerful bomb destroyed much of a residential district in the Russian capital of Moscow, claiming the lives of 118 people.
4. The 2002 Bali bombings. On October 12th, 2002, a car bomb and other bombs were detonated at venues including two night clubs on the island of Bali, a popular tourist destination in Indonesian. The attacks killed 202 people, including 88 Australians and 38 Indonesians. On October 1st, 2005, another series of bombings shook the island of

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Bali. Three bombs were detonated at the same time at the Jimbaran Beach Resort and Kuta tourist area, killing 22 people including the three suicide bombers.
5. Madrid train bombings. On the morning of March 11th, 2004, more than ten bombs previously planted on commuter trains used by Madrid office-goers exploded at around the same time. The trains were carrying droves of people on their way to work and school, resulting in the deaths of 190 people and over 1500 were injured.
6. London 7/7 bombings. On July 7th, 2005, multiple subway and bus bombings took place in London. This coordinated attack has been confirmed to have been conducted by suicide bombers, and the explosions killed 56 people
7. 2017 Saint Petersburg Metro bombing. On April 3rd, 2017, two bombings targeted the Saint Petersburg Metro system, and the attack left 15 people dead and more than 50 wounded.

There have been a small number of terrorist bombings in China perpetrated by religious extremists, along with a few other criminal explosion incidents, but relatively speaking these are very minor in explosion power and consequent damage.

1. Urumqi terrorist attack. At approximately 7:50 am on the morning of May 22nd, 2014, two vehicles without license plates rammed into pedestrians on North Gongyuan Street in Sayibak District in Urumqi of the Xinjiang Autonomous Region, then the two cars blew up and caught fire. The incident claimed the lives of 39 innocent bystanders, and additionally injured 94 others.
2. Urumqi Railway Station terrorist attack. At around 7 pm on the evening of April 30th, 2014, terrorists attacked people with knives at an exit of the Urumqi South Railway Station, and they also detonated explosive devices. The attack left three people dead (including one innocent bystander and two assailants) and 79 injured.
3. On the morning of March 15th, 2017, a suicide bomber attacked the office building of Long Peng Group in the Qitaihe City of Heilongjiang Province, which left three people dead (including the suspect) and ten others wounded.

1.2 Features of Injuries from Terrorist Bombing

Terrorist bombing incidents could take place in an open environment (i.e., in front of a building, near a plaza, etc.) or within an enclosed space (i.e., subway station, subway coach or inside a building). After an explosion takes place, surrounding structures and environment would be damaged, and the powerful shock wave, collapsed building, launched fragments, pieces of projectiles created from unfixed or easily damaged objects could all cause substantial injuries and deaths in people in the vicinity, especially when there is a large and dense concentration of people. To increase the destructiveness of explosion, perpetrators would also add steel pellets, nails or other objects to the explosive device.

1. Impact injury. It is actually rather difficult to find serious explosion incident survivor of typical blast lung injury (less than 1%), as blast lung injury has a relatively high death rate (>11%). However, in the Madrid train bombings, 17% of victims suffered from blast lung injury, and a reasonable explanation here is that evacuations were comparatively efficient, and many such victims received timely treatment in hospital before the onset of serious organ failure. Kluger analyzed blast lung injury data related to suicide bombing attacks in Israel, and opines that when overpressure reaches 35 psi, probability of death from blast lung injury is merely 1%, but when overpressure rises to 65 psi, fatality rate could climb as high as 99%. When overpressure ranges between 15 and 50 psi (pounds per square inch), probability of perforation of eardrum is 50%, and when overpressure ranges between 50 and 100 psi, probabilities of lung injury and lung rupture are 50%. During the Madrid bombings, it was discovered that a rather high ratio of victims simultaneously suffered from both lung injury and eardrum perforation. Therefore, eardrum perforation is a telltale sign of possibility of lung injury, and is indicative of corresponding medical check and support.
2. Multiple injuries and composite injuries. A multiple injury refer to a number of complicated injuries inflicted upon multiple parts of the body, and these make up a rather high ratio of injuries created in explosion incidents. For example, blunt trauma and perforating wounds produced by projectiles launched by the explosion, blast lung injury and rupturing of intestine, or burns coupled with chemical or radiation injury, among other combinations. The worst type of multiple injury is an injury that involves multiple body parts, multiple organs and multiple systems.

Other than impact injury, when people are thrown in the air by the force of the explosion and then lands or collides with wall or other hard objects could also lead to serious injuries including closed craniocerebral injury, blunt trauma to the abdomen or chest, distal fracture, etc.

A composite injury refers to other situations arising from the explosion that cause a combination various injuries. For example, burn caused by high temperature of the explosion, inhalation of smoke and dust, or chemical or radioactive contamination from dirty bombs. Reports have even pointed out cases of cross-contamination between organisms of the same species due the fragment

from an explosion, and in other cases, victims were found to have been hit by terrorist weapon fragments that tested positive in hepatitis or HIV. At least one report indicates that a suicide bombing incident in Israel resulted in a victim being infected with hepatitis B from a perforating wound inflicted by the bone fragment of the suicide bomber.

3. Features of organization of rescuers for terrorist bombing incident. Since it is unlikely to encounter a large group of victims suffering from severe multiple injuries and composite injuries during peacetime, once a serious explosion incident takes place, medical personnel would be hard pressed to properly carry out victim categorization and treatment. The challenge in victim triage is to quickly identify the extremely small number of survivors that require emergency medical treatment. When performing rapid clinical assessment, it is necessary to take into account that some serious internal injuries have yet to manifest themselves in some victims.

Although medical imaging diagnosis is accurate, but such technologies are not very applicable in real life scenario when blast injury victims arrive in droves. This is because medical imaging diagnosis takes too much time, and may even delay treatment when time is spent on diagnostic radiography, inadvertently increasing death rate. A more practical approach is to use abdominal B-mode ultrasound, as it is fast but also accurate enough to meet the majority of needs in the assessment of victims. However, B-mode ultrasound is only able to identify internal bleeding, but not other situations pertaining to internal organ damage or internal organ rupture. Thus, the best course of action would be performing full-body CT scan or enhanced CT scan promptly after the peak of arrival of victims.

2 Section Two: Explosion of Hazardous Chemicals

As scientific and technological development progresses, humans have become increasingly dependent on chemical products, and everything from daily life of regular folks to many aspects of the economy rely on chemicals. According to data about chemical enterprise safety incidents between 2005 and 2015 released by the former State Work Safety Administration, the number of chemical enterprise safety incidents and the number of casualties associated both showed a rising trend during this period, with the exception of 2008–2009. The most dangerous is the explosion of hazardous chemicals.

Hazardous chemicals refer to toxic or other chemicals with poisonous, corrosive, explosive, flammable, combustion-supporting or other properties that can cause serious harms to people, facilities and the environment. In accordance with different chemical properties, there are varying requirements in their storage, transportation, fire safety and other aspects. Explosion incidents may easily occur due to negligence or accidents in aspects such as their storage or transportation. Once such an explosion takes place, it is hard to deal with and may very well lead to tremendous injuries, deaths and property losses.

2.1 Typical Cases in Explosion of Hazardous Articles

2.1.1 Explosion from Improper Storage of Hazardous Articles

1. On the morning of April 16th, 1947, a fire started onboard the vessel *SS* Grandcamp docked at the Port of Texas City, which subsequently detonated her cargo of 2300 tons of ammonium nitrate. The initial explosion also set off a chain reaction, causing the nearby chemical factory to blow up. Considered one of the most severe industrial explosion in the United States, this incident killed around 600 and injured more than 3500.
2. Shenzhen Qingshuihe explosion. On August 5th, 1993, a fire started adjacent to the Qingshuihe fuel depot inside a dangerous goods warehouse that was storing an illegally large stock of chemical materials, which caused a massive chain explosion. There were a warehouse with 240 tons of hydrogen peroxide solution 30 m south of the center of the blast, a Shenzhen Gas liquefied gas depot with eight large storage tanks and 41 horizontal tanks located less than 300 m south of the center of the explosion, and a Sinopec gas station around 300 m to the west. If the chain explosion were to continue, nearly a hundred km^2 in the surrounding area would have been leveled, and even put Shenzhen downtown at risk. Fortunately, fire fighters and first-responders tackled the situation properly, and the explosion did not end up reaching the hydrogen peroxide solution warehouse or the fuel depot, otherwise the consequence would have been catastrophic. Nevertheless, the explosion left 15 dead and more than 800 injured, while 39,000 m^2 of buildings were destroyed, with direct economic losses amounting to RMB 250 million.
3. Tianjin Binhai New Area explosion incident. At around 23:30 on August 12th, 2015, a fire and subsequent explosion took place at the dangerous articles warehouse of Rui Hai logistics company located at the Port of Tianjin in Tianjin's Binhai New Area. The disaster shocked the world as it left 165 dead (including 24 fire fighters on active duty with Public Security organ, 75 Port of Tianjin fire fighters, 11 Public Security officers that participated in the rescue, and 55 employees of the involved and

surrounding companies and nearby residents), eight missing and 798 injured, along with RMB 6.866 billion in direct economic losses.

2.1.2 Explosion of Chemical Transport Pipeline

As urbanization advances at an accelerated pace, underground cable and pipeline networks become increasingly dense and complicated, as well as hidden dangers. Some industrial pipelines are located close to residential areas, and there are many potential hazards.

1. Nanjing propene leak and explosion. On the morning of July 28th, 2010, at the Plastic Factory No. 4 removal and deconstruction site in Qixia District, a propene pipe was ruptured by workers, leading to a propene that resulted in an explosion. At least 22 people died, and 120 people were hospitalized.
2. Qingdao oil pipeline explosion. At 3 am on November 22nd, 2013, a Sinopec oil pipeline in Huangdao District in the city of Qingdao ruptured, then it was ignited and detonated at around 10:30 am, leaving 62 people dead, 136 people injured and RMB 750 million in direct economic losses.
3. Taiwan propene leak and explosion. On the evening of July 31st, 2014, an underground propene pipeline in Kaohsiung of Taiwan ruptured and leaked a substantial volume of propene, which encountered flame and resulted in a chain explosion that left 32 people dead and 321 people injured. Among the dead, there were five police firefighters and two volunteered firefighters.

2.1.3 Explosion in Fireworks and Firecracker Production and Manufacturing

Fireworks and firecrackers have long existed in China, and they are often used under various circumstances to enhance festive and celebratory ambiance, while also generating immense economic benefits for related industries and markets. Due to past economic constraints, lots of fireworks and firecrackers were manufactured by hand in small workshops, and there had been various explosion accidents that claimed the lives of many. As China continues to improve safe production supervision of the firework and firecracker industry and raise the requirements of safety and protection standards, the frequency of such explosion accidents plunged, as did the number of related deaths and injuries. That said, accidents do still occur.

1. At 9:47 am on August 16th, 2010, a major firework and firecracker explosion accident took place in the city of Yichun in Heilongjiang Province, which left 34 people dead, three missing, 152 people injured, and direct economic losses totaling RMB 68.18 million.
2. At approximately 15:20 on September 22nd, 2014, a gunpowder explosion accident took place at an export firework factory in Pukou Town of Liling of Hunan Province. Numerous workshops were destroyed, 14 people were killed and 33 people injured.
3. At about 16:30 on February 5th, 2016, an explosion accident occurred when leftover materials from a firework and firecracker factory were being destroyed by the Gui'an New Area Administration of Work Safety in Guizhou Province. The accident left 12 people dead, five people missing and 16 people injured.

2.2 Features of Explosion Incidents of Hazardous Articles

1. On-site situation. Explosions of hazardous articles usually result from some sort of fire beforehand, and several explosion would take place after on another. For example, the Qingshui Bay explosions in Shenzhen first took place at 13:26, and then at 14:27. Another example is the accident in Binhai New Area of Tianjin started with a fire in the warehouse discovered at 22:51, and then at 23:34 the first explosion happened with a force producing seismic energy roughly equal to ML 2.3 or equivalent of 3 tons of TNT, and then 30 s later a second explosion took place with a force producing seismic energy roughly equal to ML 2.9 or equivalent of 21 tons of TNT (other articles claim that explosion equivalent is roughly 600–800 tons of TNT if measurement is based on the extent of structural damage). After a fire starts, first-responders like firefighters and medical personnel would rush to the scene, and if an explosion takes place during this period, these on-site like firefighters and medical personnel would be put in grave danger. Therefore, it is necessary to obtain accurate knowledge about the types, quantity, location of any dangerous chemicals, as well as their chemical and physical properties, so as to choose the proper timing and effective protective measures for first-responders that will enter the site for firefighting and emergency rescue tasks. Otherwise, additional explosions or inhalation of toxic gases could very well worsen the situation by injuring or killing these firefighters and medical personnel. For example, during the Tianjin accident 165 people were killed, including 99 firefighters of different types and 11 police officers, and there were also five firefighters among the eight people missing. Of the 32 people killed in the propene leak and explosion that occurred in Kaohsiung of Taiwan, there were five police firefighters and two nominal firefighters.
2. Injury features. (a) Victims near the center of an explosion would usually suffer from multiple and complicated

injuries such as impact injury, burn, chemical poisoning and bone fracture. The closer a victims is to the center of a blast, the more serious the injury. (b) Buildings within a range of several km may be damaged by the explosion's shock wave (Table 37.3), mainly seen as shattered glass, displacement of items inside the buildings, and some structures closer to the center of a blast may even have damaged windows and doors. In most cases, people inside these buildings will not suffer from impact injury, but they may sustain external wounds caused by glass shards and other sharp objects, blunt trauma caused by being struck by blunt objects, or bone fracture caused by being thrown by dynamic pressure of the explosion's shock wave, as well as other accidents such as tripping and falling while trying to escape outside a building. Teda Hospital is the closest medical institution to the site of the Tianjin explosion, as of 6 am on the morning of the next day after the nighttime explosion, the hospital had received 431 victims, with the majority being nearby residents who sustained blast injuries, burns, cuts from sharp objects and injuries from being thrown. Of these, more than 60 people were hospitalized, and critically injured patients were afflicted with multiple injuries or composite injuries including external wounds to the head and brain, external wounds to the chest and abdomen, and external wounds to the limbs, with the orthopedics department being the busiest part of the hospital. (c) Explosions of hazardous chemicals could also be accompanied by the release toxic substances that may lead to environment contamination, and problems with the respiratory tract, skin and other body parts. For instance, substances such as potassium chlorates, sulfur, charcoal, silver powder and nitrate are major constituents in firework gunpowder. These substances themselves, or their chemical products after being ignited, could cause bodywide poisoning. Potassium chlorates in the body may cause methemoglobinemia and damage kidneys (Table 1).

3. Hazardous chemical explosion rescue principles. (1) The golden rule in emergency rescue at the scene of a hazardous chemical explosion is to stop the worsening of burns and poisoning, while effectively maintaining a victim's respiratory and circulatory functions. (2) Cleanse chemical contamination on skin, eyes and other parts of the body in a timely manner. (3) Do not apply ointment, mercurochrome or merbromin on surface of fresh wounds, and do not dress wounds with dirty bandage. (4) When victim is poisoned due to ingestion of toxin, if toxin is not a corrosive substance, try to induce vomiting to expel the toxin. When victim accidentally ingested highly acidic or basic substance, have him/her ingest substances like milk, egg white, soy milk or starchy paste, but refrain from gastric lavage for the time being. (5) After carrying out simple emergency medical procedures on-site, evacuate victim to hospital in a prompt fashion. Accompanying persons should provide hospital with reasons for burn or poisoning, and name of involved chemical. If involved chemical is unknown, sample of said chemical substance or victim vomit containing such substance needs to be brought to the hospital for testing. (6) First-responder that participates in medical rescue on-site should beware of one's own safety. If time to be spent at the scene is not long, use wet towel to cover one's mouth and nose for simple protection against water-soluble poisons, or put on protective gears such as respiratory masks if such equipment are available. (7) While saving victims, also try to remove source of poison in order to prevent the toxic hazard from spreading.

Generally speaking, the diagnosis and treatment of acute poisoning must include detailed inquiry about medical history, meticulous physical examination, prompt collection of sample and earnest analysis of toxin. Only by carrying out such actions in earnest would medical personnel be able to quickly make accurate diagnosis, formulate and prepare

Table 1 Damages of buildings around site of Tianjin explosion

No.	Name of building	Distance (m)	Building damage	Damage extent
1	Port of Tianjin Public Security Bureau	400	Only framework remained of this five-story office building	Severe Damage
2	Haigangcheng residential community	800	Glass shattered at an instant, and damages to door frames and anti-theft doors	Minor damage
3	Tianbin Apartment	1000	All window panes shattered, and large sections of external parts of ceiling fell	Minor damage
4	Teda Football Field	2000	Damage of external steel structure, glass, doors and windows	Extremely minor damage
5	CAERI Tianjin branch	4000	Minor damage of building	Extremely minor damage
6	MOTECH	5000	Minor damage of office area	Extremely minor damage

highly effective treatment measures, so as to lower the chances of death or disability of victim due to poisoning.

3 Section Three: Explosion of Coal Mine Gas

The main constituent of coal mine gas is methane (CH_4), which is created from decomposition of cellulose and organic matters by anaerobic bacteria during the early stage of the formation of coal, a process involving the accumulation and decay of ancient plant matter. While coal is being formed in high-temperature and high-pressure environments, physical and chemical processes would also produce coal gas. When such gas encounters flame, it would combust, leading to coal gas explosions that directly threaten the safety of coal miners.

3.1 Typical Cases of Coal Mine Gas Explosion

1. On February 22nd, 2009, a coal mine gas explosion took place at the Tunlan mine belonging to the Shanxi Coking Coal Group, which claimed the lives of 78 miners.
2. On October 29th, 2011, a coal mine gas explosion erupted at the Xialiuchong coal mine within the territory of Changjiang Town in Hengshan County of Hengyang City in Hunan Province. Official figures report that 35 miners headed down the shaft during that shift. Of which, 29 miners lost their lives, the six remaining survived after being rescued and hospitalized.
3. On November 27th, 2014, a major coal mine gas explosion shook Songlin coal mine at Songhe Township in Pan County of Liupanshui City, which also killed 11 miners, injured eight others, and caused direct economic loss totaling RMB 30.032 million.
4. On October 31st, 2016, a coal mine gas explosion happened in Jinshangou coal mine in Yongchuan District of Chongqing, which left 33 miners dead and one person injured.
5. On the afternoon of December 3rd, 2016, a coal mine gas explosion occurred in Chifeng of Inner Mongolia. When the explosion took place, 181 people were working, and after the incident, 149 people were safely evacuated out of the shaft, but 32 miners were killed.
6. On February 14th, 2017, a relatively big coal mine gas explosion took place at Zubao coal mine in Lianyuan City, which resulted in the death of ten miners and three others injured.

3.2 Features of Blast Injury from Coal Mine Gas Explosion

1. Impact injury. The enclosed environment of mine shaft means that shock wave as powerful as ten times that of atmospheric pressure could be generated during a coal mine gas explosion. Such a powerful shock wave could inflict serious harm and damage upon miners and mining facilities. The closer a person is to the center of the explosion, the higher the probability of death, and survival rate of people working near the center of the blast is basically zero. Impact injury is mainly manifested as blast lung injury, as well as other problems such as perforated eardrum, broken or dislocated ossicle, and fractured rib.
2. Burn. Burn injury is one of the most common forms of injury after a coal mine gas explosion. After explosion, air circulating within mine shaft could reach up to 1850 °C, or even 2650 °C in enclosed environment, and such air would move at a speed of 2000 m/s. However, the majority of such hot air moves by just once and makes contact with the human body over a very short duration. Thus, most burn victims suffer from burns in exposed body parts, and usually the area of burns range from 2% to 97% of total body surface area, with second-degree and third-degree burns being most common. Since coal mine gas is lighter than air, victims are usually burned in exposed upper body parts such as the head, face and upper limbs, and deep burns in the lower extremities are less common. However, ignited clothing could still cause large areas of deep burns.
3. Inhalation injury. A coal mine gas explosion not only causes the rapid movement of hot air, but also produces various toxic gases such as CO, CO_2, NO_2, C_2H_4, CH_4 and H_2S. The enclosed environment of a mine shaft is also unfavorable to the dissipation of toxic and high-temperature gases, making victims highly susceptible to physical, chemical and toxic inhalation injuries to the respiratory tract. Around 70% to 100% of burn victims also suffer from inhalation injuries to the respiratory tract, and when coupled with blast lung injury, the occurrence rate of fatal acute respiratory distress syndrome (ARDS) is rather high.
4. Poisoning from toxic gas and damage from cerebral anoxia. CO, CO_2, NO_2 and other gases produced after a coal mine gas explosion could poison the body. CO_2 is heavier than air and would usually settle toward the ground. If a victim is lying on the ground, he or she could inhale too much CO_2, leading to hypoxia, asphyxia and poisoning. CO is lighter than air and would usually accumulate toward the ceiling, with concentration reaching up

to 0.6%, which is 400 times more than allowable concentration. The affinity between CO and Hb is 240 times higher than O_2, while dissociation rate is only 1/3600 that of oxyhemoglobin. CO may bind to myosin, inhibit cytochrome oxidase, leading to lack of oxygen in tissues and cells, inducing cerebral edema and cerebral circulatory dysfunction, and resulting in central nervous system symptoms such as disorientation, hypophrenia and mental disorders. Some reports indicate that among victims of inhalation injury, 17% exhibit CO poisoning including symptoms such as dizziness, nausea, vomiting, irritation, agitation, depressed mood or lack of speech, with such symptoms lasting 3 ± 2 days or even longer.

5. Composite injury. The majority of victims of coal mine gas explosion sustain multiple injuries from multiple sources throughout the body. For example, one victim may suffer from impact injury and burn, coupled with internal bleeding from trauma to the head, chest or abdomen caused by collapsed structure, or crushing injury or bone fracture in the limbs.
6. Traumatic tattoo. After a coal mine gas explosion, coal particles may be imbedded into the body, and after the body heals, some traumatic tattoos from external wounds may remain on the body, particularly prominent around the face, chest and back.
7. Features of medical aid. The sudden nature of coal mine gas explosion results in a large number of victims with complicated injuries. Properly organization is one of the most important steps in a rescue effort. The crux of the matter is to "locate victims quickly, rescue victims quickly, evacuate victims quickly, identify injuries and categorize victims quickly, and deal with critically injured victims quickly." Other than regular triage and sorting procedures, also pay attention to other features of coal mine gas explosion injuries: (1) Coal mine gas explosion usually results in serious contamination, and coal dust residue on victim body is hard to remove, making it difficult to judge conditions such as peripheral circulation and open wound. Thus, missed diagnosis is very possible, which is why repeated examination should be conducted after early stage treatment in order to avoid missed diagnosis. For victims of composite injuries that do not go into shock, consider utilizing the immersion bath method. Immerse victim for half an hour before thorough debridement. (2) When carrying out triage and sorting, stay concentrated in the task at hand and do not be distracted by cries or moans of lightly injured victims. Instead, focus on identifying severely injured victims to avoid prioritizing the wrong victims. (3) Ensure the swift transfer of victims, especially those with severe burns. Delivery victims to medical care institutions with the proper treatment capability. Following socioeconomic development in China, roadway transportation and hospital treatment levels have improved by leaps and bounds, consequently the survival rate of coal mine victims has also risen significantly. (4) Various types of oxygen supply and hyperbaric oxygen chamber methods can yield rather positive and obvious effects in treating oxygen deficiency and toxic gas poisoning. (5) Do not neglect psychological stress response in victims, and provide emotional intervention in a timely manner.

4 Section Four: Explosion of Dust

Dust explosion refers to an explosion arising from flammable dust within explosion limits that encounter a heat source (flame or high temperature). The resulting flame would immediately propagate throughout the entire space in which the dust mixture has spread. The rate of chemical reaction is extremely fast, and a tremendous amount of heat is released, producing very high temperature and very high pressure. The system's energy converts into mechanical energy, as well as light and heat radiation that combine into dangerously destructive power.

In factories and mine, more than 70% of dust created during the production process is flammable. A common reason for the occurrence of a dust explosion is the existence of relatively strong reducing agent (such as C, H, N, S and other elements) in the dust particles themselves, or coated or surface of dust particles. When such reducing agents exist at the same time within the same space as peroxide and explosive dust, redox reaction would take place. During this chemical process, a large volume of gas is generated, and even though the volume of gas may be relatively small at times, a tremendous amount of combustion heat energy would still be released within a short span. Dust explosions usually happen in production sites where there are substantial amounts of aluminum powder, zinc powder, aluminum material processing powder, various types of plastic powder, intermediaries of organic synthetic drug, flour, sugar, saw dust, dye, Bakelite ash, baby formula, tea powder, tobacco powder, coal dust, plant fiber dust, and so on.

Dust explosion is a very complicated process that is affected by a myriad of physical factors, and the underlying explosion mechanism is not yet entirely clear. The general consensus is that whether a dust explosion is easy or difficult to occur closely depends on the physical and chemical properties of the dust, environment conditions, and so on: (1) Speed of oxidation. Since dust explosion is caused by reaction between dust surface particles and oxidizer, it is widely believed that dust particles with fast rate of oxidation (i.e., dye, magnesium powder, iron oxide, etc.) are more prone to exploding. (2) Heat of combustion. When combination reaction takes place in the mixture of dust particles and oxidizer, a massive amount of heat of combustion would be released,

and the larger the heat of combustion of the substance (i.e., carbon, coal dust, sulfur, etc.), the greater the chance of exploding. (3) Charge characteristic. In general, dusts that are easily charged (i.e., starch, synthetic resin powder, dust and powder of fiber, etc.) are prone to exploding, and even substances with poor conductivity may also generate static electricity during continual friction with machine or air. When electric charge build up to a certain level, electricity would be released in the form of spark, which may become the source of fire that initiates an explosion.

One of the most notable features of dust explosion is the likelihood of multiple explosions. Powder and dust that have settled on the ground and covering the surface of equipment would be kicked up by the first explosion and float in the air, during which a negative pressure zone would also be created around the center of the blast. When fresh air rushes into the explosion center, the floating dust would once again mix with air, setting the stage for a second explosion. The concentration of the dust mixture is far higher than that of the first explosion, and thus it is much more powerful and destructive as well.

4.1 Typical Cases of Dust Explosion

1. On February 24th, 2010, a corn starch explosion took place in Qinhuangdao of Hebei Province, which killed 19 people and injured 49 others.
2. On May 20th, 2011, a flammable dust explosion accident occurred in the polishing workshop of the Foxconn complex in Chengdu of Sichuan Province, which left three people dead and 16 people injured.
3. On the afternoon of August 5th, 2012, an aluminum dust explosion incident happened inside a building in Ouhai District of the city of Wenzhou, which caused the building to collapse and started a fire. The explosion claimed the lives of 13 people, and also injured 15 others.
4. On April 16th, 2014, a stearic acid dust explosion incident took place in Dongchen Town of Rugao City, which is under the administration of Nantong City of Jiangsu Province. The explosion killed nine people and wounded eight others.
5. On August 2nd, 2014, an aluminum dust explosion in the polishing workshop of a factory shook Kunshan of Jiangsu Province, and left 146 people dead and 91 injured.
6. On the evening of June 27th, 2015, dyed corn starch was being scattered in the air to enhance the festive atmosphere at a theme park in Xinbei City of Taiwan, but staffs were unaware of the existence of combustion conditions. A subsequent dust explosion caused the death of 12 people and injury of 498 others.
7. On September 9th, 2016, a flour explosion at an incineration site in the Republic of Benin killed almost a hundred people.

4.2 Features of Blast Injury from Dust Explosion

1. Burn. The most obvious injury condition from a dust explosion is severe burn. Due to the tremendous amount of heat released during the instant of the explosion, the energy generated by chemical reaction could raise temperature to some 2000 or 3000 °C. In addition, the high probability of multiple explosions may instantaneously char and carbonize the skin of victims within a certain range of the blast waves. In the Kunshan dust explosion incident, 73.5% of the victims sustained burns that covered more than 90% of body surface area (Table 2), and the majority of them had deep burns.
2. Impact injury. Dust explosions also cause impact injuries, with blast lung injury being the most common, and medical imaging taken within 24 h of injury may show (1) changes in lung ground glass density: pathology is lessening of density, showing slightly raised cloud shapes, and overlapping vascular shadows may be seen in pathology, with light and uniform density; (2) patchy consolidation: expressed as segments of lung or lobules are large lamellar, lobed or segmented, and appear to be flaky blurred shadows or dense shadows, with blurred edges and uneven density; (3) diffuse consolidation: expressed as diffuse patchy blur of varying sizes in one or both lung parenchyma, integration may be observed, and density is uneven, with victims exhibiting the most severe clinical manifestations including breathing difficulty, or even hemoptysis. None of the chest imaging changes in patients would appear alone, usually multiple symptoms and problems exist at the same time. Medical imaging reexaminations in 2–5 days may reveal blurred dots or blurred dot patches in patients that previously did not show any obvious abnormalities.

Table 2 Distribution of burn body surface areas of 185 victims of the Kunshan dust explosion

Total body surface area (TBSA)	Number of cases (*n*)	Percentage (%)
99%	35	18.9
90–99%	101	54.6
80–89%	18	9.7
70–79%	10	5.4
50–69%	8	4.3
<50%	13	7.1

Note: TBSA means total body surface area

3. Inhalation injury. Dust explosion victims would suffer from different degrees of inhalation injury. Dust inhaled would accumulate in airway, and thus fiberoptic bronchoscopy and irrigation should be performed as per conventional protocol so as to protect the respiratory tract.
4. Composite injury. The shock wave created by a dust explosion is immensely powerful, sometimes even powerful enough to launch heavy machinery and equipment into the air and propel them through walls. People within the blast zone would be thrown by the powerful air current, and sustain injuries such as bone fracture after colliding with something. Such injuries would combine with other injuries such as burns and impact injuries, and worsen overall situation.

5 Section Five: Explosion of Town Gas in Residential Area

The most common form of explosion in daily life are those related to natural gas pipeline or propane tank, usually the results of accidents that arise from hidden risks such as pipeline aging or incompliant installation.

5.1 Typical Cases of Natural Gas Explosion

1. On the afternoon of March 25th, 2017, the natural gas pipeline in a residential building of a community in the city of Baotou in the Inner Mongolia Autonomous Region exploded, which caused the center of the entire building to collapse, leaving three people dead and 25 people injured.
2. On the evening of August 9th, 2016, a natural gas explosion took place within a residential building in Zhengzhou of Henan Province. Doors and windows were blasted off their hinges, and two people were lightly wounded.
3. On the morning of April 6th, 2017, a gas explosion occurred inside the cafeteria in a bank in Yiwu of Zhejiang Province, killing one person and injuring four others.
4. On the morning of November 14th, 2011, a major propane tank leak and explosion incident took place in a dining establishment located at a business area in Xi'an of Shaanxi Province, which claimed the lives of ten people and injured 36 others.
5. On October 10th, 2015, an explosion shook Wuhu of Anhui Province. The pressure relief valve on a propane tank in a small eatery fell off, causing a substantial amount of liquefied gas to spew, which immediately caught fire and exploded, killing 17 people in the residential area where the shop was located.

5.2 Features of Blast Injury from Natural Gas Explosion

Explosions related to natural gas pipeline or propane tank in residential areas usually do not generate much energy, and only affect a small area within an immediate vicinity. In addition, there are often barriers such as building walls to obstruct the explosion, so usually such explosions do not cause major damages, and most injuries are either external wounds caused by fragments, or burns. In business areas, restaurants or other venues with high concentration of people, the fire that starts after the explosion is a main reason for fatalities and injuries, as is disorderly fleeing of scared people.

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