Injury Principles and Mechanisms of Shock Wave

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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.

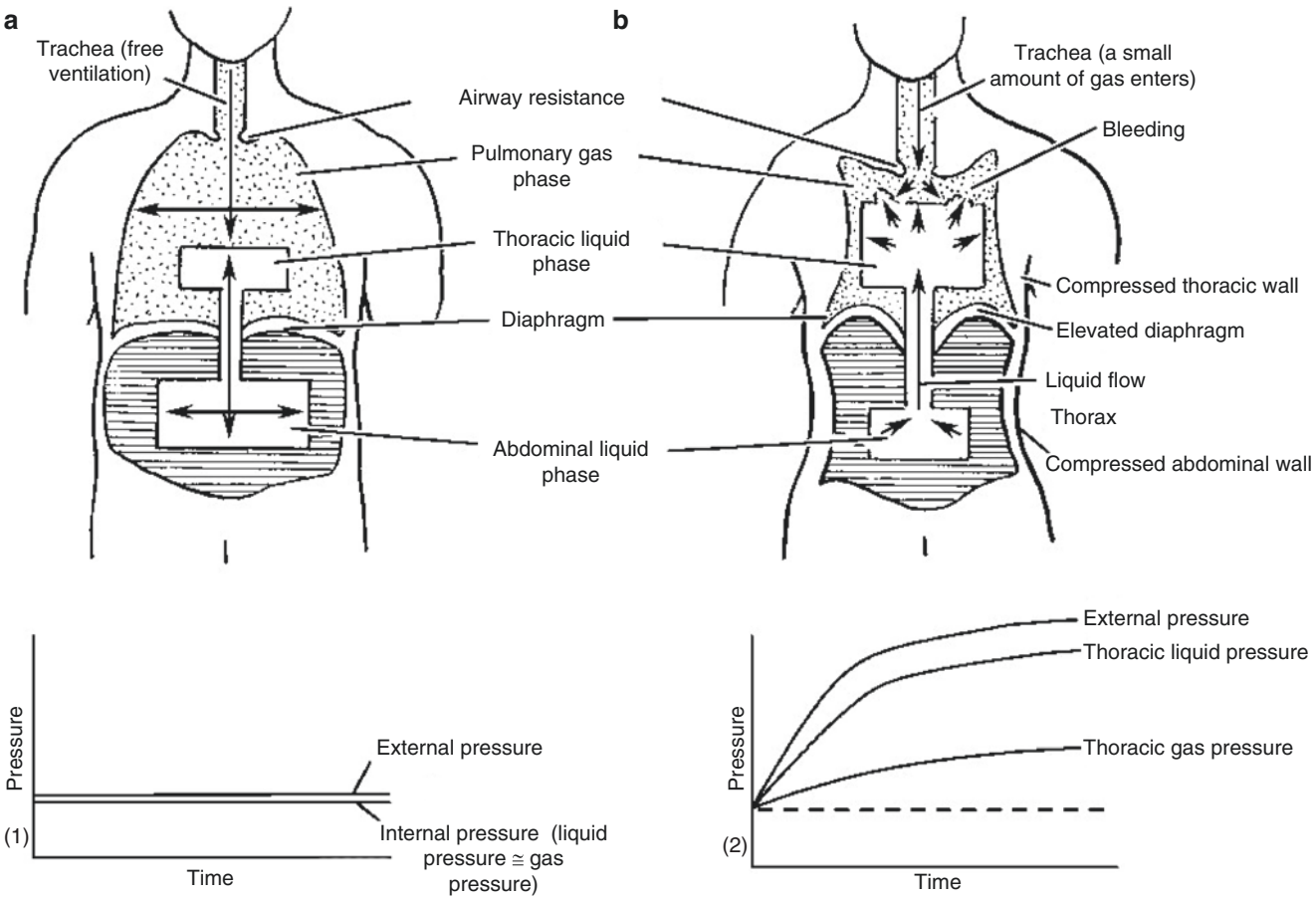

Fig. 1 Hemodynamic changes in blast injury. (**a**) Normal hemodynamics; (**b**) Hemodynamics after overpressure

(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.

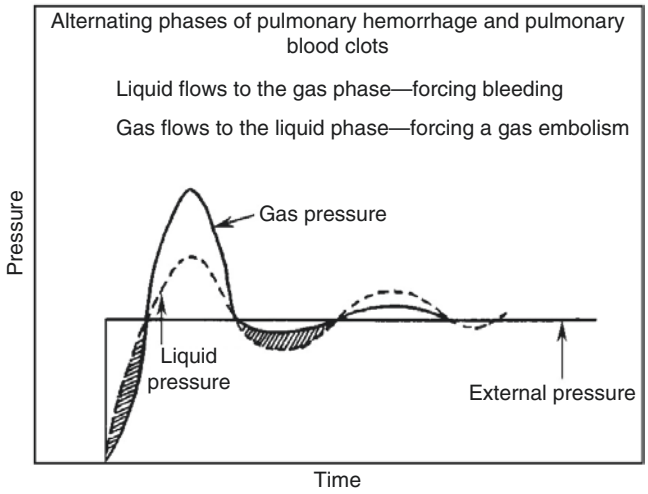

Fig. 2 Injury mechanism of pressure difference caused by shock wave

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.

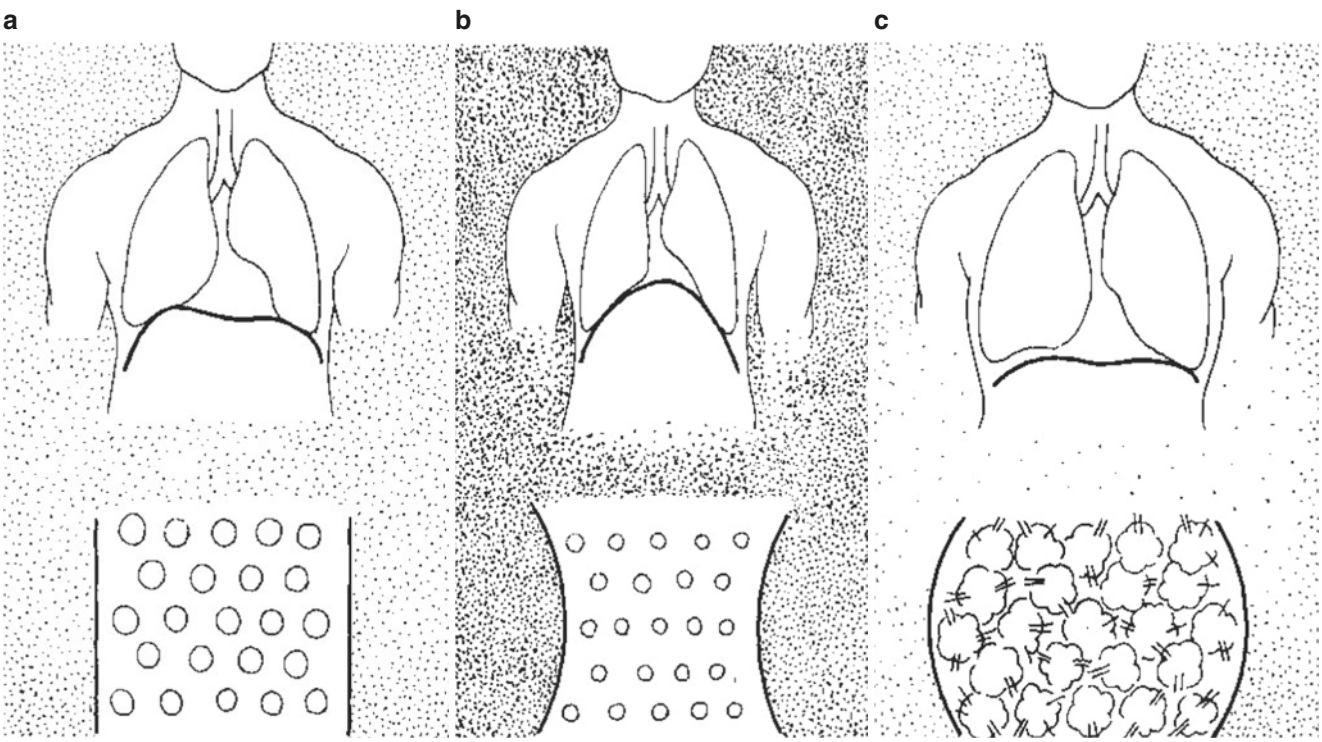

Fig. 3 Schematic diagram of implosion effect. (**a**) Normal atmospheric pressure; (**b**) Overpressure action; (**c**) After action

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

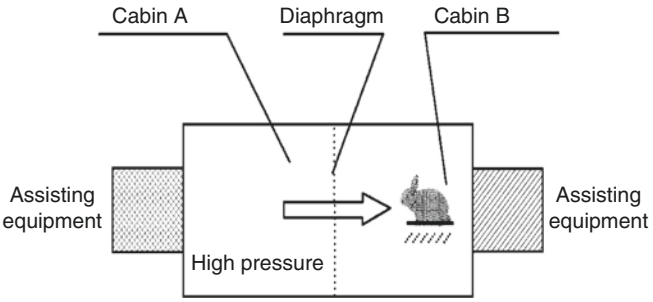

Fig. 4 Segmented simulation cabin of shock wave

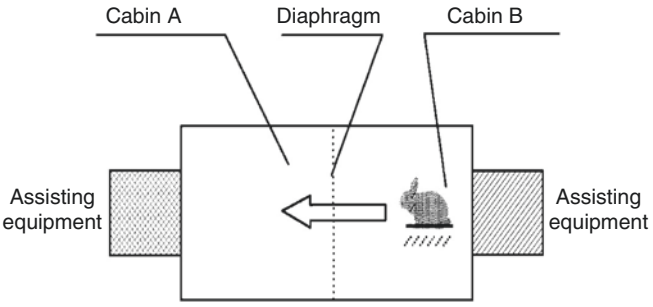

Fig. 5 Simulation cabin in compression section of shock wave

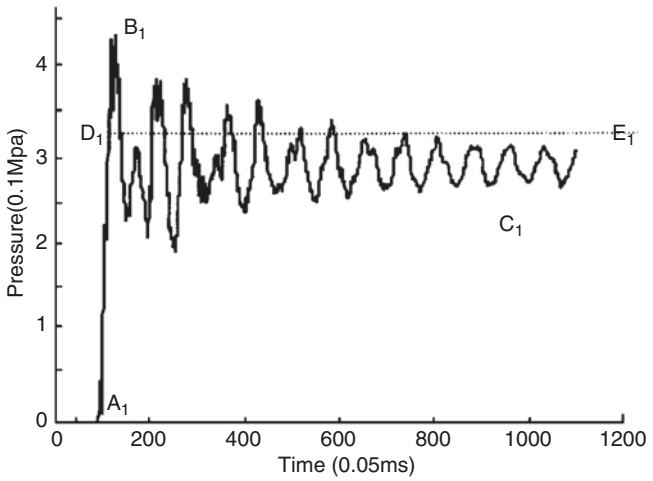

Fig. 6 Compression wave in the compression section of the simulated shock wave. (A_1) Starting point of compression wave; (B_1) Pressure peak value of compression wave; (C_1) Steady-state pressure value of compression wave; (D_1–E_1) Parallel line corresponding to the pressure value of 0.32 MPa

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.

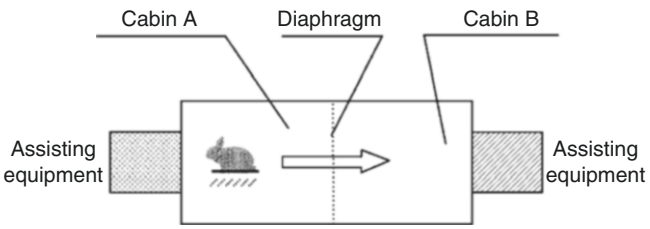

Fig. 7 Simulation cabin for decompression phase of shock wave

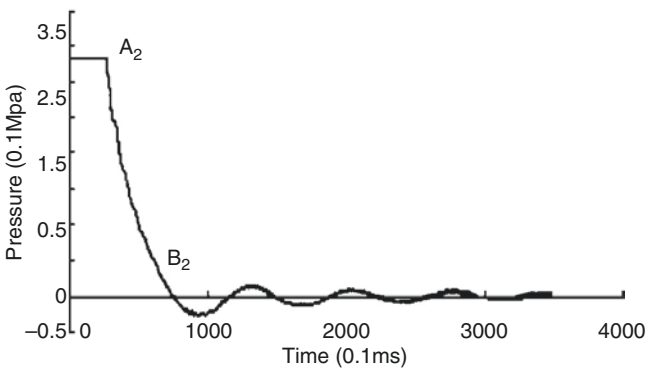

Fig. 8 Decompression wave in the decompression section of the simulated shock wave. (A_2–B_2) Decompression wave section

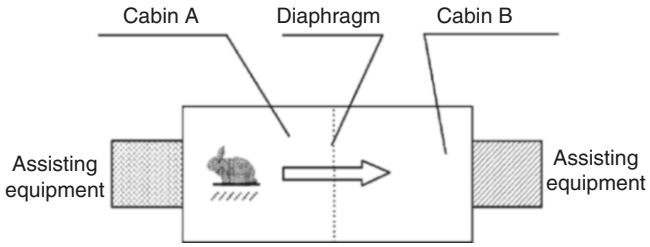

Fig. 9 Simulation cabin for negative pressure phase of shock wave

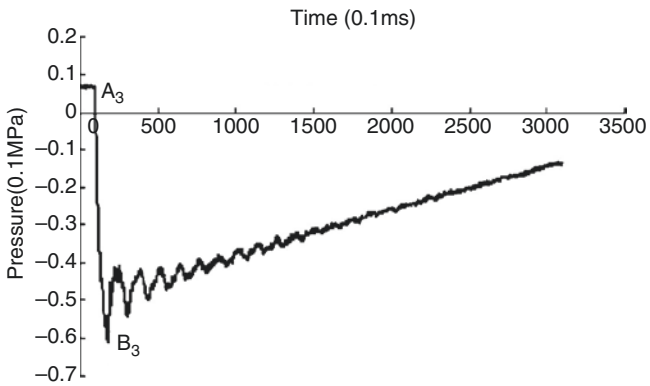

Fig. 10 The negative pressure wave in the negative pressure section of the simulated shock wave. (A_3–B_3) Negative pressure wave section

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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