Damage Control Management in the Polytrauma Patient

Second Edition

Hans-Christoph Pape
Andrew B. Peitzman
Michael F. Rotondo
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Foreword by C.W. Schwab

> The value of damage control surgical approach will only continue to increase in importance within the context of casualty care as the complexity of intentional injury continues to escalate in both the civilian and military environments.
>
> Brian Eastridge MD, FACS
>
> Out of the Crucible.[1]

Throughout the history of medicine, physicians and surgeons have been forced to react with unconventional approaches to massive anatomic destruction and rapid physiologic depletion. To prevent death, they invented, innovated, and forged solutions. This "forced empiricism" is a unique characteristic of the surgery for trauma and, over time, has improved man's ability to survive physical trauma.[2]

The benefits of damage control in both the military and civilian sectors have been validated over the last 15 years. The term "damage control" was initially adapted to surgery to describe a three-staged approach to exsanguinating abdominal injury by using a truncated laparotomy for bleeding and contamination control. Its utility in response to the American urban gun violence epidemic of the 1990s resulted in improved survival. At the same time, it resulted in serious complications like abdominal compartment syndrome and the prolonged "open abdomen." These, in turn, precipitated novel management schemes and innovations in critical care and forged groundbreaking surgical techniques. During this same period, many young surgeons training in our busy intercity trauma centers learned the principles of damage control surgery and later, as military surgeons, applied the concepts to critically wounded soldiers in Iraq. The DC approach was expanded to orthopedic, vascular, thoracic, and neurologic surgery with the development of techniques to swiftly control bleeding, relieve compartment pressure, and reestablish profusion and afford skeletal stabilization. On these same battlefields, an improved understanding of resuscitation and the need to reverse coagulopathy led to the use of early whole blood, component therapy, and procoagulants as an effective prototype of promoting hemostasis and oxygen

[1] Out of the Crucible, Capt. Eric Elster and Dr. Arthur L. Kellermann (editors), published by The Borden Institute, Washington, DC (in press).

[2] A national trauma care system: integrating military and civilian trauma systems to achieve zero preventable deaths after injury. Washington, DC: The National Academies Press doi: 1.0.17226/23511.

delivery – damage control resuscitation. By combining damage control surgery and damage control resuscitation, the allied military medical teams reported unprecedented survival with wounding patterns that historically had been mortal.

Several other important concepts emerged from Iraq and Afghanistan. Damage control applied in mass casualty events in these austere environments was verified as an approach to match limited human and material resources with the critical needs of a maximum number of wounded soldiers.[3] Damage control management was adapted to the worldwide military trauma system where minimal acceptable care delivered at intervals across geographically separated medical units was established and ingrained in the fabric of military medicine. Individual patient care was supplemented with video feedback between forward surgeons, critical care transport medical teams, and reconstructive surgeons across the globe and led to standardized approaches and improved outcomes. As the formal war period was winding down, these broader concepts of military damage control translated to the civilian sector and proved their value in Boston, San Bernardino, and Orlando.

Frequent mass casualty events from active shooters and explosive devices are increasing and appear almost as daily events in our world. Suddenly, our emergency systems and hospitals provide the safety net for dozens of patients with wounds more commonly seen in combat than in civilian life. Thus, there is an *imperative* that all medical personnel be expert in the use of damage control for an individual patient and as an effective mass casualty and disaster management process.

The second edition of *Damage Control Management in the Polytrauma Patient* benefits from editors who are experts in the concepts and techniques of damage control. The flow of knowledge between the disciplines of trauma orthopedics and surgery and integration of the military and civilian experiences provide critical information that is new, uniquely broad, and rare to find in a single compendium. The selected topics are contemporary, relevant, and contributed by military and civilian authors who have applied and verified the uses of damage control in orthopedics, trauma, and emergency surgery. Thus, in my opinion, this edition will be *required reading* for all who provide the medical readiness to protect human life.

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[3]Remick KN, Shackelford SA, Oh JS, Seery J, Grabo D, Chovanes J, et al. Adapting essential military surgical lessons for the home front. AmJDisaster Medicine (in press).

Preface by P.M. Rommens on behalf of the European Society for Trauma and Emergency Surgery (ESTES)

The second edition of the book *Damage Control Management in the Polytrauma Patient*, edited by Pape, Peitzman, Rotondo, and Giannoudis, is a milestone publication for the European surgical community. It answers to a real need for modern, high-quality trauma care in most European countries.

Thanks to many new member countries, the European Union (EU) has grown rapidly during the last decades. The unification process is a complex task and will go on for several more generations. Harmonization of medical care – for us of special interest is the care of the emergency surgical patient – is one of many topics on the to-do list of the EU. Looking at incidence of accidents, organization of trauma care and mortality after trauma in the member countries of the EU, we are confronted with most diverse facts and figures. Quality of road infrastructure, of motorcycles and motorcars, and density of population are very different from country to country. Prevention of accidents by limit of speed, obligation to wear a helmet or security belt, restriction of alcohol consumption, and the implementation of these regulations by intense control is also very variable. Due to continuing industrial and social development, the number of motorcycles and motorcars has grown quickly. The consequence is that the incidence of heavy traffic accidents is still raising and the *polytrauma patient* continues to be sad and daily reality.

In contrast with this, regional organization of trauma care, establishment of trauma centers, basic and postgraduate training of medical and paramedical staff involved in trauma care have not evolved parallel with the increasing challenge.

The European Society for Trauma and Emergency Surgery (ESTES) is an umbrella organization of national societies of trauma and/or emergency surgery. Thirty-two European societies are institutional members and more than 500 surgeons are individual members. The vision of ESTES is enhancing and harmonizing the care of the critical ill surgical patient. Different sections have been founded to realize these goals: skeletal trauma and sports medicine, visceral trauma, disaster and military surgery, emergency surgery, and polytrauma. The last section is a compilation of the most important European guidelines on primary and secondary care of the severely injured.

This publication is an important instrument for all medical and paramedical care providers, who are involved in the management of the polytrauma patient. It gives a comprehensive overview of modern organization and evidence-based principles of care of the severely injured. Several eminent ESTES members have contributed as chapter editors. We therefore are very

happy to endorse this publication with our logo and recommend it to a European readership of emergency physicians, anesthetists, general surgeons, (orthopedic) trauma surgeons, and rehabilitation staff. We very much hope that this work will be accepted as a guide for treatment in the different settings of trauma care all over Europe. With these different realities, we should not overlook our common and unique goals of treatment: the polytrauma patient should survive, independent of the country, the place and the time of his/her accident, he or she should suffer the least morbidity, and have the best rehabilitation and recovery possible. This book gives theoretical background as well as practical evidence for good polytrauma care. We congratulate the editors to this initiative, also, ESTES is grateful for being involved in sharing their knowledge and wish the second edition of the book *Damage Control Management in the Polytrauma Patient* good acceptance and distribution.

Prof. Dr. Dr. h. c. Pol M. Rommens
ESTES Secretary-General

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Part I

Introduction: Pathophysiology

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Robert J. Winchell

1.1 Introduction

By any measure, injury is a serious public health problem. Worldwide, road traffic injuries are the leading cause of death among the young (aged 15–29 years), responsible for over a million deaths per year [1]. In the United States, unintentional injury is the leading cause of death for persons under the age of 45 years and is among the top ten causes of death for all decades of life [2], a pattern that has not changed significantly in decades (Fig. 1.1). Overall, injury is responsible for almost a third of all years of potential life lost. Moreover, it is a substantial economic burden [3]. In real numbers, highway transportation-related events in the United States were responsible for about 2.2 million injuries and 33,000 deaths in 2010 [4]. And yet, there is no focused public health policy at the federal level to address the problem of injury in a systematic fashion. Moreover, state and regional approaches are nonuniform, ranging from the very robust to the nonexistent. This lack of policy-level response is not universal. By comparison, the 2014 West African Ebola outbreak drew tremendous headlines and produced a massive public health response worldwide, despite accounting for only about 23,000 identified cases and about 9,800 deaths worldwide in its first year, according to CDC statistics [5] (Fig. 1.2). Over 50 years after *Accidental Death and Disability: The Neglected Disease of Modern Society* [6] was published by the National Academy of Science, injury remains the "neglected epidemic [6]" of modern society.

When *Accidental Death and Disability* was published in 1966, the field of injury care consisted of largely disconnected elements: ambulance services, emergency departments, intensive care units, and trauma research units. The report established the foundational and seminal elements of what has come to be recognized as a trauma system, recommending measures to address the entire spectrum of injury including epidemiology, prehospital care, definitive care, rehabilitation, research, and injury prevention. Significant progress has been made in these individual areas, including the evolution of the Emergency Medical Services (EMS) system, establishment of national standards for trauma centers, dramatic improvements in automobile safety, as well as a greatly expanded base of scientific knowledge in the areas of injury, shock, and resuscitation. The Injury Prevention and Control Center was established within the

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	Age Groups										
Rank	<1	1-4	5-9	10-14	15-24	25-34	35-44	45-54	55-64	65+	All Ages
1	Congenital Anomalies 4,758	Unintentional Injury 1,316	Unintentional Injury 746	Unintentional Injury 775	Unintentional Injury 11,619	Unintentional Injury 16,209	Unintentional Injury 15,354	Malignant Neoplasms 46,185	Malignant Neoplasms 113,324	Heart Disease 488,156	Heart Disease 611,105
2	Short Cestation 4,202	Congenital Anomalies 476	Malignant Neoplasms 447	Malignant Neoplasms 448	Suicida 4,878	Suicida 6,348	Malignant Neoplasms 11,349	Heart Disease 35,167	Heart Disease 72,568	Malignant Neoplasms 407,558	Malignant Neoplasms 584,881
3	Maternal Pregnancy Comp. 1,595	Homicida 337	Congenital Anomalies 179	Suicida 386	Homicide 4,329	Homicide 4,236	Heart Disease 10,341	Unintentional Injury 20,357	Unintentional Injury 17,057	Chronic Low-Respiratory Disease 127,194	Chronic Low-Respiratory Disease 149,205
4	SIDS 1,563	Malignant Neoplasms 328	Homicida 125	Congenital Anomalies 161	Malignant Neoplasms 1,496	Malignant Neoplasms 3,673	Suicide 6,551	Liver Disease 8,785	Chronic Low-Respiratory Disease 15,942	Cerebro-vascular 109,602	Unintentional Injury 130,557
5	Unintentional Injury 1,156	Heart Disease 169	Chronic Low-Respiratory Disease 75	Homicida 152	Heart Disease 941	Heart Disease 3,258	Homicide 2,581	Suicide 8,621	Diabetes Mellitus 13,061	Alzheimer's Disease 83,786	Cerebro-vascular 128,978
6	Placenta Cord Membranes 953	Influenza & Pneumonia 102	Heart Disease 73	Heart Disease 100	Congenital Anomalies 362	Diabetes Mellitus 684	Liver Disease 2,491	Diabetes Mellitus 5,899	Liver Disease 11,951	Diabetes Mellitus 53,751	Alzheimer's Disease 84,767
7	Bacterial Sepsis 578	Chronic Low-Respiratory Disease 64	Influenza & Pneumonia 67	Chronic Low-Respiratory Disease 80	Influenza & Pneumonia 197	Liver Disease 676	Diabetes Mellitus 1,952	Cerebro-vascular 5,425	Cerebro-vascular 11,364	Influenza & Pneumonia 48,031	Diabetes Mellitus 75,578
8	Respiratory Distress 522	Septicemia 53	Cerebro-vascular 41	Influenza & Pneumonia 61	Diabetes Mellitus 193	HIV 631	Cerebro-vascular 1,687	Chronic Low-Respiratory Disease 4,619	Suicide 7,135	Unintentional Injury 45,942	Influenza & Pneumonia 56,979
9	Circulatory System Disease 458	Benign Neoplasms 47	Septicemia 35	Cerebro-vascular 48	Complicated Pregnancy 178	Cerebro-vascular 508	HIV 1,246	Septicemia 2,445	Septicemia 5,345	Nephritis 39,080	Nephritis 47,112
10	Neonatal Hemorrhage 389	Perinatal Period 45	Benign Neoplasms 34	Benign Neoplasms 31	Chronic Low-Respiratory Disease 155	Influenza & Pneumonia 449	Influenza & Pneumonia 881	HIV 2,378	Nephritis 4,947	Septicemia 28,815	Suicide 41,149

Fig. 1.1 Leading causes of death in the United States, 2013 (Source: CDC WISQARS. http://webappa.cdc.gov/sasweb/ncipc/leadcaus10_us.html)

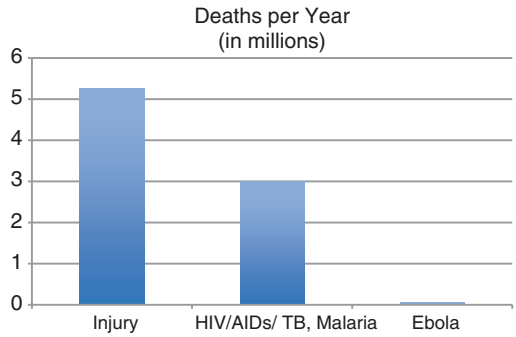

Fig. 1.2 The relative impact of injury [5, 46]

Centers for Disease Control and Prevention in 1985. These advances have been associated with substantially lower death rates from injury over the last 30 years (Fig. 1.3). Though there is significant evidence to demonstrate that coordination of these individual elements into a comprehensive system of trauma care leads to improved outcomes after injury [7–15], these data have not led to a broad implementation of trauma systems across the country [16, 17]. At the present time, systems for the provision of injury care remain a patchwork, dependent upon the degree to which state and local government has been inspired to address the problem and the extent to which volunteer efforts by engaged system stakeholders can drive improvement.

The sections that follow trace the historical evolution of the approach to injury care within the context of changing socioeconomic circumstances, the emergence of a set of essential elements that define a trauma system, and the integration of these elements into a functional design. The legislative and structural challenges to effective implementation of trauma systems will be discussed, and strategies of successful models will be explored with the objective of framing future efforts to expand the coverage of trauma systems throughout the nation.

1.2 The Process of Evolution

In the current age of highly technical and institution-based medical care, it is worth looking back to realize that it has not always been this way. At the start of the twentieth century, only a bit more than 100 years ago, the majority of

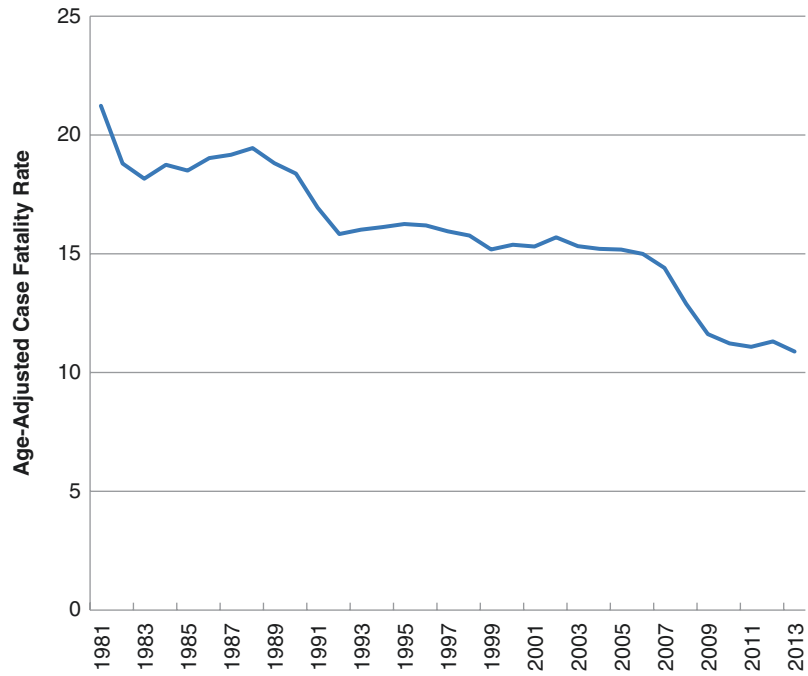

Fig. 1.3 Age-adjusted case fatality rate from motor vehicle incidents (Source: CDC WISQARS. http://www.cdc.gov/injury/wisqars/fatal_injury_reports.html)

health care was delivered in the home. Large hospitals existed primarily for the care of the indigent or casualties of war, a state of affairs that dated back to antiquity. Outside of the military, these hospitals were usually established and operated as private charities or as elements of social infrastructure funded through local government to care for the poor.[1] There was often little differentiation between the care of the sick and the housing of the poor and indigent, and many hospital facilities served both purposes. In either case, these facilities were places to be avoided by those with the means to do so. The sick and injured were cared for at home by their family, and "no gentleman…would have found himself in a hospital unless stricken by insanity or felled by epidemic or accident in a strange city" [18]. Based upon the care available and the associated high mortality, this conception of hospital care was well founded. Prior to the late nineteenth century, medicine had little to offer by way of curative therapy, and the hospital environment itself often carried a great risk of death through infectious complications. Only with the rise of the Lister's antisepsis, the bacterial theory of disease, and the expansion of surgical capabilities that followed did the balance shift. Hospitals began to take their current form as places that offer highly technical therapies well beyond what could be accomplished in the home and as places that offered the potential to cure.

Even though the development of the hospital was well under way in the early decades of the twentieth century, the concept of the modern trauma center is much newer. Through the 1950s, hospitals and hospital care were defined by the conquering of infectious diseases, the evolution of modern surgical techniques, and the progressive interdependence of the hospital and the academy of medicine, including training and research. In both the historical and the practical literature on hospital development from the mid-twentieth century, the care of the injured is a passing comment, if it is mentioned at all. Only since the late 1980s, with the development of national standards and the Trauma Center Verification program of the American College of Surgeons Committee on Trauma, has the idea of the modern trauma center become firmly

[1] In fact, prior to the advent of Social Security in the 1930s, the provision of aid to needy populations was felt to be completely outside the Constitutional mandate of the Federal government.

established as a highly sophisticated institution that is focused on improving injury care through clinical excellence, teaching, and research.

The first hospitals to embody the concept that care of the injured should be an area of specialty and focus, recognizing the importance of this idea in improving outcomes, arose in the 1960s. Their roots were firmly in the traditional past of the public hospital. These hospitals were a refuge for the indigent sick by intent and the injured by necessity. They were also at the heart of medical education and research. Two hospitals among the first with organized trauma services were the Cook County Hospital [19] and San Francisco General Hospital, but the public hospitals in many US cities also functioned increasingly as centers for the care of the injured. Over the next decade, an expanding number of such facilities became known for their hard-earned trauma expertise. Initially the hospitals that could claim the title of "trauma center" were almost exclusively located in large urban areas with high rates of poverty and violence. Outside the sphere of influence of these centers, injury care remained haphazard, undertaken in the facility that happened to be the closest and by the practitioner who happened to be available. Injury care was largely a matter of chance, a situation that persisted well into the 1970s, and, it might be argued, persists to the present day. These chances could be improved if the patient, or those bringing the patient to the hospital, had the knowledge of the receiving hospital's capabilities and the ability to choose their destination. The principle that injured patients fared better at a hospital experienced in trauma care was initially based upon the experience of the providers of direct patient care but has subsequently been upheld by objective data [20–22]. The fact that all hospitals are not created equal in terms of care of the injured has been well established, and it follows that one key element of establishing a system to improve care after injury is the ability to identify hospitals with the commitment to care for the injured and to verify their capabilities.

The next logical step in the process is to establish a way to ensure that injured patients are treated at the appropriate trauma center. Historically, patients literally applied for admission to a hospital. The decision to admit was often made by the hospital board and weighed many factors beyond need for medical care [18]. Not all types of illness were admitted for treatment, nor were all socioeconomic groups. Even into the middle of the twentieth century, the mission of the hospital to care for certain diseases, including contagion and mental illness, was a topic of debate [23]. Patients presented themselves to the hospital either under their own power or with the assistance of friends and family. The first hospital-based ambulance system in the United States, providing a vehicle and a trained attendant to be summoned to transport patients to the hospital for care, was established by Bellevue Hospital, in New York City, in 1869. The system began with two horse-drawn vehicles, which were to be kept "in good order and fit for service at all times," and presaged modern regulations regarding ambulance equipment lists by requiring that a box be kept beneath the driver's seat containing among other things a quart flask of brandy and two tourniquets [24]. Ambulance systems soon appeared in other major cities, transitioning from horse to internal combustion engine in the early twentieth century. These systems focused primarily on getting the patient to the hospital rather than initiating care and did not evolve far beyond the provision of the most basic prehospital care until much later.

For the next 100 years, through the 1950s and 1960s, there was also little evolution in the standards regarding ambulance equipment or training of attendants. Because the focus of ambulances remained primarily that of transporting bodies, vehicles were designed for use interchangeably as ambulances and hearses, and mortuaries often functioned as ambulance agencies due to the interoperability of the vehicles. The principle that a modern network of Emergency Medical Services (EMS), with the expectation that properly equipped vehicles, manned by medically trained staff, would respond within minutes of a call as an essential public service, arose from recommendations made in the 1966 white paper *Accidental Death and Disability* and from the provisions of the Highway Safety Act of 1966 [25], which was enacted later the same year. This

act included provisions for funding as well as requirements that states take action or face penalties. Implementation was further accelerated by the passage of the 1973 Emergency Medical Services Systems Act [26], which established guidelines and provided funding for regional EMS development. With this stimulus, EMS systems rapidly developed and matured during the next 25 years of the twentieth century, coincident with the differentiation of hospitals into trauma centers. Combined with the recent wartime experience in Korea and Vietnam, which clearly demonstrated the advantages of rapid evacuation and early definitive treatment of casualties [27], it became increasingly apparent that coordination of field treatment and transportation to ensure that injured patients arrived at a capable trauma care facility was of critical importance, and the conception of a trauma system, as opposed to an isolated trauma center, began to evolve.

Initially, the concept of a trauma system was centered upon the large urban trauma centers, which established standards and protocols within their region, promulgated through their own EMS systems or through their relationship with EMS providers in the region. For hospitals with their own large EMS, or those with clearly pre-existing roles in the community, the destination hospital was preordained. It was common knowledge that serious injured patients were best cared for at the trauma center, even though there were no established rules or regulations directing the flow of ambulance traffic. The growing knowledge that outcomes for seriously injured patients were better in hospitals that had the experience and resources to care for them, combined with an increasing perception of the element of chance involved in unregulated choice of ambulance destination, led to the first efforts to coordinate the prehospital system to transport injured patients with the dedicated facilities that provided definitive care.

Drawing on the experience at the Cook County Hospital, the State of Illinois passed legislation establishing a statewide coordinated network of trauma centers in 1971 [28]. This statewide plan identified many of the terminologies and concepts that would come to be considered key elements of trauma system design, including the concept of an administrative lead agency to govern system operations, the identification of different levels of trauma hospital capability, the integration of EMS, and the role of process improvement. The first operational statewide trauma system was created with the establishment of the Maryland Institute for Emergency Medicine in 1973. The small size of the state of Maryland allowed for implementation of a system in which all severely injured patients within the state were transported by air to a single dedicated trauma facility. In the years that followed, similar efforts were made to establish cooperative networks of trauma centers that were connected by a coordinated EMS system and linked by shared quality improvement processes. These efforts were driven both by the Vietnam experience and by the finding that a large proportion of deaths after injury in non-trauma hospitals were due to injuries that could potentially have been better managed and controlled [29]. The implementation of such systems led to dramatic decreases in what was perceived to be "preventable death," [7] as well as overall improvements in post-injury outcomes that were duplicated in widely varying geographic settings. Following the models established in Illinois and Maryland, these regional systems were founded upon the premise that all critically injured patients should be transported to a trauma center and that other acute care facilities within a region would be bypassed. Based upon the "exclusion" of non-designated hospitals from the care of the severely injured, this approach is frequently referred to as the exclusive model of trauma system design.

The exclusive model works well in urban and suburban settings, where there are sufficient trauma centers to provide access and to care for the expected number of injuries. Though often described as a regional system, an exclusive system functions as a funnel, not a network, and it does not utilize, let alone maximize, the resources of other health-care facilities within the region. This approach has the advantage of focusing patient volume and experience at the high-level centers and the disadvantage of leading to attenuation of skills in non-designated centers, with

resultant loss of flexibility and surge capacity. Paradoxically, because of the attrition of local resources, the model may serve to decrease access to competent care in larger geographical areas and in low-resource areas. In such circumstances, transport times to the trauma center may be very long, especially in periods of inclement weather when aeromedical transport cannot be used. Moreover, the volume of injuries seen may overtax the resources of the few available trauma centers, and the number and length of interfacility transfers may place a severe burden on EMS resources. The only way to increase the depth of coverage within an exclusive system is to recruit or build additional trauma centers, which can be both expensive and politically difficult, given the complex set of drivers that lead a hospital to undertake the significant commitment to being a trauma center.

The limitations of the exclusive model, and the difficulties in deploying the model on a large scale, were experienced throughout the 1990s [30]. Despite evidence of the benefit of trauma systems, very few states and regions were able to establish a system as a matter of governmental policy, and fewer still were able to fulfill a set of eight criteria that had been proposed as cornerstones of exclusive system design [31]. This stagnation in system development arose in part due to the difficulties inherent in extending the exclusive system model and from a lack of public support for system finance and governing policy. In a broader sense, exclusive systems often lacked a truly integrative overarching structure that could tie together and build upon the significant gains achieved by the individual components in the trauma system universe. The 1999 Institute of Medicine report *Reducing the Burden of Injury: Advancing Prevention and Treatment* [32] helped to open the aperture through which injury care was perceived and identified five broad areas of focus for future development: improving coordination and collaboration between individual programs, strengthening capacity for research and practice, integrating the full spectrum the injury field, nurturing public awareness and support, and promoting informed policy making. In this context, the thrust of trauma system development embraced a different paradigm: the inclusive model of trauma system design.

As the name suggests, the inclusive model involves the design of a system in which all health-care facilities within a region are involved with the care of injured patients, at a level commensurate with their commitment, capabilities, and resources. Ideally, through its regulations, rules, and interactions with EMS, the system functions to efficiently match an individual patient's needs with the most appropriate facility, based upon resources and proximity. Under this paradigm, the most severely injured would be immediately recognized and either transported directly or expeditiously transferred to the top-level trauma care facilities. At the same time, there would be sufficient local resources and expertise to facilitate the optimal management of the less severely injured, avoiding the risks and resource utilization incurred for transportation to a high-level facility whose resources were not truly needed. The basic concepts of the inclusive model were described in the 1992 *Model Trauma System Care Plan* [33] and refined in the 2006 *Model Trauma Systems Planning and Evaluation* [34] document, both published by the Health Resources and Services Administration of the US Department of Health and Human Services. Consistent with the findings of the 1999 IOM report, the 2006 *Model Trauma Systems Planning and Evaluation* document places the previously identified elements of trauma system function within an overarching public health framework, emphasizing the need to integrate the entire spectrum of injury care, from prevention through rehabilitation. The document also highlights the importance of coalition building at the grassroots level and of policy development and implementation at the governance level. The inclusive system model has been the primary guiding framework for systems development for the last 10 years.

Despite its relatively universal acceptance at the theoretical level, the inclusive model is often misconstrued and misapplied in practice, not as a system with global involvement of all facilities but as a *voluntary* system in which any hospital that wishes to participate is *included* at whatever level of participation they choose.

This approach fails to fulfill the primary goals of an inclusive trauma system that all resources in the region are involved and to ensure that the needs of the patient are the primary driver of resource utilization. An inclusive system does mean that all hospitals must participate in the system and be prepared to care for injured patients at a level commensurate with their resources and capacity, but it does not mean that hospitals are free to determine their level of participation based upon their own perceived best interest. Objectively assessed, the needs of the patient population served are the parameters that should determine the apportionment and utilization of system resources, including the level and geographic distribution of trauma centers within the system. When this maxim is forgotten, system function suffers, and problems of either inadequate access or oversupply can develop.

The implementation of a scheme for the distribution of system resources that is based solely on the needs of the patient population served can be fraught with conflict. While the potential for such conflict exists at all levels of resource allocation, it is often most prominent involving decisions around trauma center designation because these decisions often carry heavy political and financial consequences that extend well beyond matters of patient care. To heighten the challenge, these difficult policy implementation decisions are generally the responsibility of the lead agency governing the system, which is most often a relatively underfunded state or regional agency. In addition to lacking staff and resources, these agencies are most often led by political appointees and government employees who do not stay in a particular job for more than a few years at a time. These factors have the combined effect of limiting both the stability of institutional direction and the ability of agency leaders to take a strong stand in the face of opposition from large and well-funded health-care organizations. These challenges have proven the Achilles' heel of inclusive system development and have hampered their broad implementation, which continues to fall far short of a nationwide system for the care of the injured.

1.3 The New Era

The period of time from the 1980s through the early years of the twenty-first century saw the rise of the trauma center as a center of excellence, a place where injured patients had better care with demonstrably better outcomes, and the concomitant understanding that with such differences in care, all injured patients deserved to be treated at a trauma center. At the same time, major changes in health-care finance in the age of managed care and managed competition increased the financial pressure on hospitals, especially those caring for the most severely ill patients and those patients with insufficient funding. At a time when the system-based approaches to trauma care were trying to grow, and there was a need for more high-level trauma centers, the economic pressures on hospitals made trauma care an undesirable mission, one that could lead to financial ruin. The 1990s saw a rising tide of trauma center closings and contractions, even involving some of the iconic public hospitals and foundational trauma centers. One study reported that while 60 trauma centers closed between 1981 and 1991, over 300 closed between 1990 and 2005 [35]. The same study identified that, not surprisingly, financial pressures were one of the chief risk factors for trauma center closure.

The crisis atmosphere engendered by the real and threatened loss of trauma care capacity led many regions to develop methods of funding support to assist trauma centers in their care mission. In the first years of the twenty-first century, the decline of managed care and managed competition, a nationwide decrease in levels of interpersonal violence, and other changes in the climate of health-care finance combined to create a sea change in the financial attractiveness of providing care for the injured in many geographic regions. Hospitals began to look upon the care of the injured population not as a burden to be shifted but as a potentially profitable line of business to be actively sought after.

Somewhere in the first 5 years of the twenty-first century, the balance of forces shifted to the point that the number of hospitals claiming trauma center status was larger than the number

of traditional trauma centers threatened with closure. According to self-designation data collected annually by the American Hospital Association [36], in the year 2000, 258 (6.1%) of hospitals reported having a level I trauma center. In 2010 the number had risen to 387 (9.4%), and in 2013 there were 416 hospitals claiming level I trauma center status. This data is concordant with that collected by the Trauma Center Verification Review and Consultation program of the American College of Surgeons Committee on Trauma, which reports a similar increase in verified trauma centers, rising from 208 in 2005 to 430 in 2014, with level I centers rising from 81 to 120 over the same period. This new economic climate, while having the benefit of increasing the number of participants in the inclusive trauma system, has done so at the cost of a major redistribution in the way resources and patients are deployed. Not surprisingly, the largest number of new trauma centers arose not in the major urban areas, which remain relatively poor and where trauma center closings remain a concern, but in more affluent suburban areas. These new centers can encircle the pre-existing centers and decrease their patient volumes, as well as cutting into government incentives intended to stabilize those pre-existing centers. Increasingly, providing care for the population of patients suffering injuries as a result of motor vehicle crashes, falls, or other accidents could be a profitable undertaking, especially if the patients with the highest level of acuity could still be transferred to larger traditional centers, avoiding the financial risks associated with handling complex cases, with associated high resource utilization, under current strategies implemented to control health-care costs. As a result, established centers find themselves once again facing an unpredictable economic future, and decisions around the designation of new trauma centers in many geographical areas have become increasingly contentious. This trend may well result in decreased access to trauma center resources for highly vulnerable populations [37], despite larger overall numbers of centers.

This new era, in which the concern has shifted from trauma center closings to trauma center "propagation," carries with it a new set of challenges. These challenges cut to the very core of many unique elements that drive the social and political philosophy of the United States and have clearly placed the determination of need for a new trauma center as much in the political arena as the scientific one. In the political climate of the second decade of the twenty-first century, governments have been generally unwilling to regulate free markets, including health care. Yet Adam Smith's "invisible hand" of the marketplace is not likely to provide wise guidance for the development of a sustainable network for the care of the injured, which is arguably a central feature of the social structure. Recent history has demonstrated that changes in economic factors can easily lead hospitals to exit the trauma "market" regardless of the burden of injury within the population and that the commoditization of trauma care has the potential to disrupt stable access to trauma care across the board. The current debate over "trauma deserts" [38] identified in the city of Chicago, the most prominent of which has at its center a large and capable hospital that closed its trauma center in the late 1980s, brings the issue of the potential conflict between public service and economic performance into sharp relief.

Ultimately, the model of the inclusive trauma system has been well developed, and there is substantial evidence to show the efficacy of these systems in improving outcomes after injury, but it is undeniable that inclusive systems are difficult to develop, finance, operate, and sustain. The system has a scale and function that undeniably places it in the realm of an essential element of the public service sector, yet it operates primarily within the private sector, the largely market-driven world of health-care delivery. In most areas, the public health elements of the trauma system are not well recognized and not well funded within the governmental bureaucracy of the state or region. This infrastructure has been increasingly challenged to find funding for many other critical social elements, including the overall provision of health care itself. If trauma system development is to proceed, these barriers must be identified and overcome.

1.3.1 Optimal Design Elements

The functional development of trauma systems has paralleled an evolving understanding of the problem of injury. Trauma centers and the coordinated EMS systems that bring patients to them began as a reactive element; trauma was seen as a sporadic event, an "accident" that could not be prevented or predicted. The best a system of care could do was to be very efficient in delivering care to those affected. Early system advances were logically driven by the frontline care providers, whether in the field or the hospital, and resulted in substantial improvements in outcome for the injured. Increasing sophistication in the acute care of the injured also highlighted the fact that most injury mortality occurs at the scene, prior to any intervention, and can never be addressed by post-injury treatment, no matter how well optimized. This realization, as well as a deepening body of research on the causes and mechanisms of injury, illustrated the need to focus efforts on prevention of injury if further progress was to be made.

This evolution of understanding is analogous to the course of historical progress made in the treatment of epidemic diseases and the development of modern public health systems, an observation not lost on those involved with trauma system development. If injury is viewed not as a sporadic event but as an *epidemic*, it is a logical next step to apply well-proven principles of public health that have been so successful in the management of infectious epidemic diseases to the broader problem of injury. This concept was at the heart of the 2006 *Model Trauma Systems Planning and Evaluation* document, which adopted the public health framework developed by the CDC.

The CDC framework builds upon the 1988 Institute of Medicine report *The Future of Public Health* [39], which proposed that there were three core functions of public health agencies: assessment, policy development, and assurance. The report placed the primary responsibility for public health on the state. It recommended that the federal government function to establish nationwide objectives and provide technical assistance and funding to strengthen state capacity while at the same time assuring "actions and services that are in the public interest of the entire nation [39]." This basic framework was further expanded by the 1994 Core Public Health Steering Committee into ten essential services, represented graphically in relation to the three IOM core functions in Fig. 1.4. This framework was applied, utilizing functional elements felt to be critical from experience in trauma systems, to create an injury-specific diagram of essential services that was put forward in the *Model Trauma Systems Planning and Evaluation* document (shown in Fig. 1.5). The public health structure is a good model to use for setting the structure and outlining what we need from a trauma system from a high-level strategic perspective. It is based upon broad principles that are global in application and as a result provides a working framework that is largely independent of specific circumstances.

The challenge of the public health model for trauma system development, like the larger public health model from which it is derived, is that it offers no tactical advice as to how the specific goals are to be achieved. Further, the high degree of variability in geography, resource availability, and political climate across the country requires that any such implementation be context dependent and thus tailored to specific local circumstances. There is no global approach or proven framework to assist in pulling the elements of a trauma system together. Further, the federal government has not taken up the role outlined by the Institute of Medicine of assuring "actions and services that are in the public interest of the entire nation."

In this evolution to an expanded public health model, the approach to trauma care has grown far beyond the frontline providers of emergency care and into a complex and interconnected entity that touches on a large group of people distributed across many professions, some far removed from direct health care. This multidisciplinary and integrative process brings together groups who approach the problem of injury in fundamentally different ways, melding the epidemiologists, the statisticians, and the regulators, all of whom

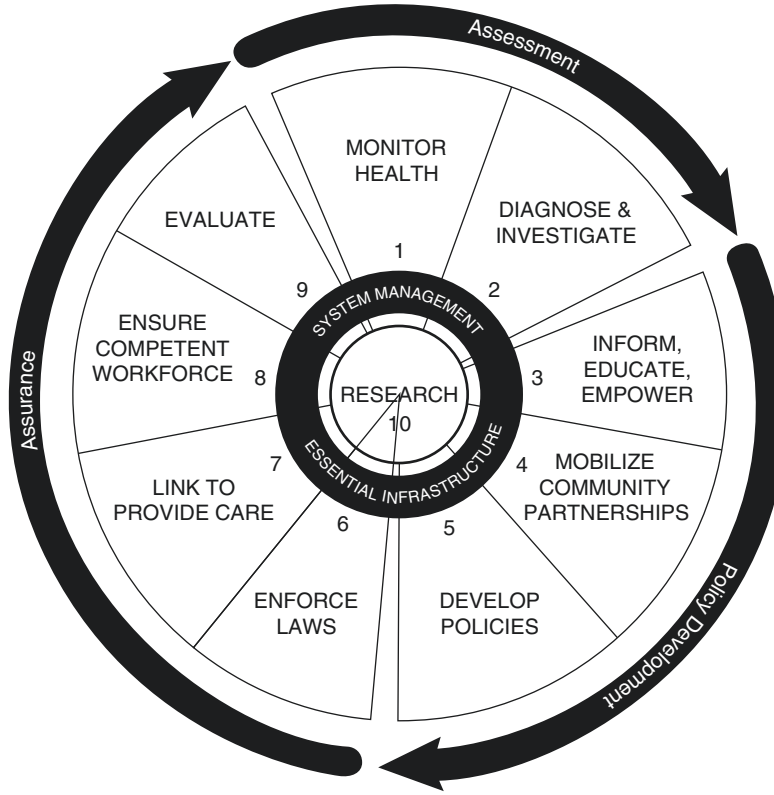

Fig. 1.4 The CDC model of public health: the ten essential functions and their relationship to the three goals defined by the IOM [33]

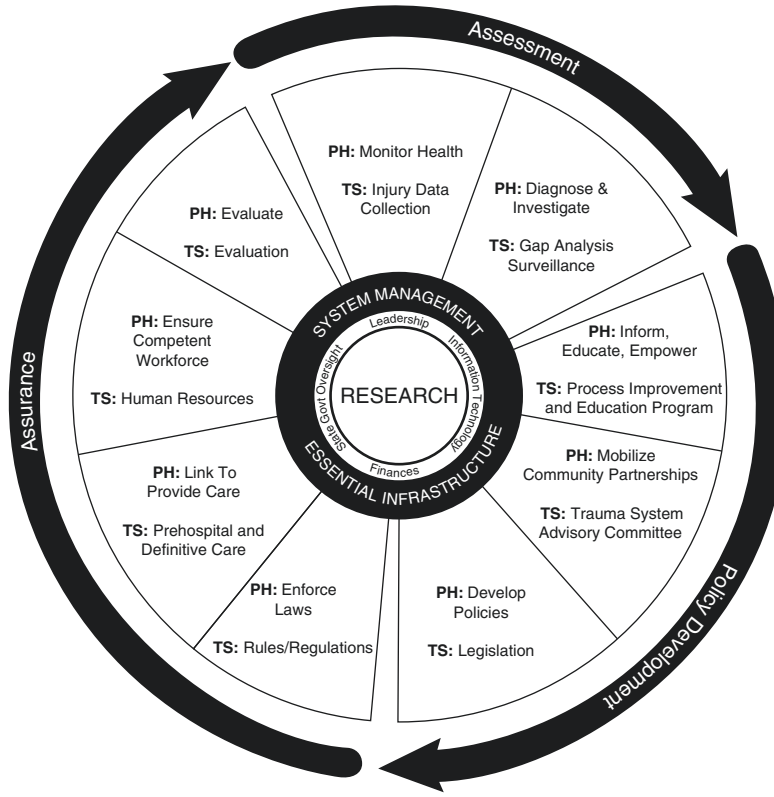

Fig. 1.5 The public health model of trauma system design [33]

manage problems at a broad population level, with the clinicians and other providers in acute care and rehabilitation, who approach the problem from the level of the individual patient. The development and maintenance of such a coalition is a considerable project, as is the governance and leadership necessary to ensure its longevity, sustainability, and success. These factors alone would be sufficient to make trauma system development a long and arduous process.

The funding mechanisms for trauma systems and trauma care are equally complex and in many cases work to make the problem of cooperative system development event more difficult. Elements of the trauma system that address the larger population-based issues of epidemiology, prevention, and governance have most frequently arisen from governmental agencies already working in more traditional public health arenas. These agencies are often relatively poorly funded and have been even more vulnerable in difficult economic times. As a result, resources are chronically scarce, and efforts are often divided across a number of different project areas. Further, the time frame for progress is on a longer scale and considers data and trends longitudinally. Those collecting and analyzing the data are often far removed from the front lines of patient care and the individual patient perspective. Thus, the problem of injury has not often been uniformly enough of a priority within the public heath bureaucracy.

In contrast, direct patient care after injury is funded through the health-care delivery system and has been subject to the variations in the health-care market, payer reform, and efforts toward cost containment. The expenditures are generally orders of magnitude larger than those seen for the more population-based functions and typically funded through a combination of government benefits and private insurance. The work in direct patient care follows a much shorter time frame, as the episodes of care typically extend over days and weeks, rather than years. Frontline providers work almost exclusively at the level of the individual patient and rarely see the problem of injury framed in the context of population health or overall health-care costs. This relatively narrow focus often limits the degree to which frontline care providers become involved in the broader area of policy development and implementation around the problem of injury across its full spectrum.

This differential in both funding and focus, between the public health and direct patient care sectors, is a major challenge to the cohesion of trauma systems. While trauma centers and EMS agencies deal in millions of dollars, most of which are external to the governance of a trauma system, the necessary elements of infrastructure that form the essential glue binding the system together are often sacrificed to lean governmental budgets at the state level. Thus, in many systems, the medical elements are fairly well developed at the level of the individual center or small cooperative network, while development has stalled at the level of system integration, large-scale prevention, and quality assurance because there are insufficient resources to carry out these large-scale system tasks on a daily basis. The situation is perpetuated by a lack of public understanding of the need and resultant inability to mobilize legislation that produces structural change. This is the impasse that most regional trauma systems face in the second decade of the twenty-first century. There are a few systems that have been able to allocate and preserve the critical infrastructure needed to administer the system and continue to grow, but most have not.

1.4 Barriers to Implementation

Given a general acceptance of the primary elements that make up a trauma system, and the previously cited evidence of their effectiveness in improving the care of the injured, it is perplexing that trauma system development remains so haphazard and inconsistent, raising the question of why trauma systems have not really caught on. A significant component is undoubtedly the nature and perception of injury among the general population and, even more importantly for trauma system development, the role of post-injury care in modulating that risk. From a psychological perspective, studies suggest that individual estimates of risk are inaccurate, tending to overestimate more sensational and

dramatic causes [40], leading individuals to rate the risks of injury lower than what they actually are. Moreover, media coverage is highly influential on societal perception of risk [41] and hence supports for policies to reduce that risk [42, 43]. Media coverage that addresses injury is generally focused on the event and its immediate aftermath, with relatively little coverage of the availability and impact of post-injury care or celebration of trauma survivors, in direct contrast to disease entities such as cancer, in which the opposite is generally true. These elements combine to lessen public awareness of the personal risks of injury and impact of trauma care. Data show that over half of those surveyed did not know that injury was the leading cause of death in the first decades of life and that though the public general supports the concept of trauma care, most believe it to be already in place [44].

Another significant element in the complexity of trauma system development and implementation lies in the multifaceted nature of trauma systems. By their very nature, trauma systems involve a large number of people and agencies, each with their own focus and expertise and each with unique and sometimes divergent culture, objective, and focus. This reality puts the design and operation of a trauma system beyond the purview of one single professional group or single sector of the trauma care spectrum; an effective solution requires the creation and maintenance of a broad coalition. It further establishes the need for a neutral governance process that can balance competing priorities while keeping the needs of the population served as the guiding principle. Finally, it can be argued that a system of care for the injured, for that matter, a system of health care in general, is part of the essential network of public services provided by government. These considerations place a large portion of trauma system development firmly in the governmental, and hence the political and legislative, arena. This is especially true with decisions mediating complex issues of resource allocation, financial support, and governance. In this arena, scientific arguments alone are insufficient to make the case, and the problem of injury has rarely held public attention in a way that has engendered decisive political action. In the complex interplay of the political process, market forces, and patient needs, it has proven impossible for most regions to achieve public policy support and significant stable funding. Without these elements, systems struggle to make consistent and lasting progress in trauma system implementation beyond a level that can be sustained by the largely volunteer efforts of system stakeholders who share the mission.

The nature of health care, and particularly the nature of injury care, presents a particularly difficult challenge within the context of the socioeconomic structure of the United States. A majority of US citizens will endorse the concept of emergency care for the sick and injured as a fundamental human necessity, and that the provision of such care is a vital function of society. This concept dates back to the founding principles for the original public hospitals and gained increasing prominence in US policy through the mid-twentieth century, reaching a peak with the establishment of the Medicare and Medicaid programs in the 1960s. The momentum was focused on the problem of road traffic accidents during the Kennedy administration and carried over to the care of the injured with the publication of *Accidental Death and Disability* and the subsequent burst of progress in EMS and trauma system development that it enabled.

The era of strong public support, and thus of federal support, came to an end in the last decades of the twentieth century, as policy turned more toward deregulation, limited government, and the culture of individual financial responsibility that characterized the 1980s and 1990s. In this setting, the model of the large publically financed hospital providing care for those in need became largely untenable, and most city and county governments have divested themselves of this responsibility or operate their health-care facilities on increasingly austere budgets. Private hospitals have been forced to assume an increasing share of the care for patients with little or no funding, while insurers have exerted intense downward pressure on payments. Health-care facilities have become increasingly competitive for patients with a funding source and face increasing risk in providing care for those who do

not. Injury care can be either a catastrophic loss or a significant profit, depending upon the population treated and the severity of injury encountered.

As a result, health care today is characterized by intense competition between health-care systems and health-care providers, driven by economic forces that create a focus on individual patient encounters rather than the broad provision of emergency care to the population as a whole. The result is an atmosphere that is highly disruptive to the coalition building and cooperation necessary in a public health-based trauma system model. The implementation of the ACA contains financial incentives for health-care systems to think more broadly in terms of "population health," but these incentives may not have a beneficial effect on trauma care, as the populations referred to are actually small diagnosis or disease-based groups rather than the entire population at risk of injury. Moreover, the uncertainty created by widely variant projections of the true financial impact of the ACA has led to further cost-cutting measures on the part of health-care systems and increased economic pressures that work to make trauma system development more difficult.

This situation is compounded by the national trend toward decreased social services and minimization of government intervention that leaves most state legislatures and state bureaucracies unwilling to take a strong position in establishing standards and regulations governing emergency medical care. With no stabilizing authority to intervene, health-care facilities may engage injury care in areas where it is profitable, while abandoning injury care in others that are less so. The result is a maldistribution of resources, leading either to lack of access as described above or to an oversupply with duplication of efforts, resources, and increased cost to the region as a whole.

1.5 The Road Ahead

The challenges to trauma system development are substantial, but not insurmountable. Several regions, usually of smaller geographic scale, have created successful and sustainable trauma systems, despite economic and political challenges. The single most important factor in these regions has been the crystallization of a focused political effort resulting in strong governing policy, both in the establishment of authority for operations and in the financing of critical system infrastructure. The difficulty in generalizing these successful models lies in the inherently unique local factors that tipped the political scale to the side of decisive action. In some circumstances, the impetus has arisen from successful grassroots efforts to raise public interest and awareness, which drive legislative action; while in others, the progress has been driven because of a focused interest in the executive branch. In either circumstance, the essential turning point has been in finding the political will to create policy that provides some objective authority over the resources necessary for the care of the injured and for that matter all patients with emergency or "unscheduled" illness, in order to ensure availability and access, but without a degree of governmental control that some factions with current society find unacceptable. The other key element of successful solutions has been in the ability to find stable funding for essential trauma system infrastructure, in order to support system oversight, quality improvement, and day-to-day operations. Efforts that focus solely on supporting trauma centers for underfunded care often result in adverse incentives for overall system development.

The Affordable Care Act (ACA) will certainly change the balance of forces and dynamics affecting trauma systems. However, it is difficult to predict in which direction, as some elements within provisions of the Act stand to increase funding available for trauma care, while others remove existing funding streams; and both of these elements occur in a setting intended to decrease overall expenditures for health care. Although as yet there has been no grassroots support or political agenda sufficient to drive a policy-level solution to the nationwide problem of uniform systems for trauma care, the ACA and the tools being used in its implementation do

contain some elements that may prove to be useful in this arena. In a recent perspective paper, Sylvia Burwell, the US Secretary of Health and Human Services, outlined three strategies that will be used to guide reform of the health-care payment system under the ACA [45]. The first strategic area centers on creating incentives to provide value-based care that center on alternate payment models, including the potential for shared responsibility for a particular patient group, both among providers and among health-care facilities. The second focuses on the integration of facilities and coordination of health-care efforts with an emphasis on population health. Both of these areas have the potential to provide financial incentives and a financial basis for the development of truly sustainable and effective systems of care. This is a stark contrast to the current fee-for-services models, which are most often a strong disincentive to cooperative regional systems.

The primary challenge to these options lies in the way the word *population* is generally understood in the world of accountable care organizations and bundled payment, where the concept of *population* referred to is in fact a subgroup of patients with a specific disease process (e.g., diabetics or patients with heart disease), rather the entire population of the region, who are all at risk for injury. Payment reforms have potential to provide a strong impetus to drive regionalization of emergency care, if they are implemented in a way that either coalesces the health-care market to large integrated systems with such broad coverage that there is a financial incentive to provide efficient injury care for the entire regional population or if similar pressures create an environment in which the major health-care providers within a region have a financial incentive to cooperate and to decrease duplication of expensive efforts. Any change in the pattern of health-care funding, away from current competitive fee-for-service structures that focus on individual patient encounters toward mechanisms that incentivize a population-based approach, will greatly aid the normative commitment to progress toward public health centered trauma systems.

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The Concept of Damage Control

2

Claudia E. Goettler, Peter V. Giannoudis, and Michael F. Rotondo

2.1 Historical Management of Injury

> Trauma surgery is just general surgery, but faster and under blood. – Anonymous

As the majority of trauma resuscitation and operation was historically performed by general surgeons, the practice of trauma and surgical critical care developed slowly as a general surgical subspecialty by those with special interest in this patient population. Surgical procedures for injury care, therefore, have been based entirely on elective general surgical procedures. Hence, injury to the stomach would receive an operative approach similar to that of a perforated ulcer. This was gradually modified by war experiences. Patients from the war zone generally had massive destructive wounds, and there was also delay to definitive care. This resulted in the development of novel operative techniques for trauma, such as pyloric exclusion and distal rectal washout, some of which have stood the test of time and some of which have not.

2.2 Failure of a General Surgical Approach in Trauma

> The operation was a success but the patient died anyway. – Anonymous

Since general surgeons have long been trained to identify and repair operatively any diagnosed injury or disease, prolonged operative procedures for definitive repair were the norm. Patients who bleed during elective operative procedures either have control maneuvers instituted prior to the vascular incision as in vascular surgery or rapid pressure or clamp control of inadvertent vascular injury during a case. Additionally, hemorrhage nearly always occurs only moments before control is achieved when the patient is already in an operating suite, draped, and in many cases already open.

This is radically different from the physiologic pattern in trauma patients who are injured minutes to hours prior to arriving in the operating room and hence have been bleeding for an extended period of time prior to instituting surgical control. Additionally, this bleeding results in

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difficulty in obtaining rapid surgical control by obscuring the operative field and tissue planes. Similarly, intestinal contamination, ongoing prior to operative control, results in an increased degree of contamination by virtue of both the length of contamination time and the high energy of intestinal content distribution.

Finally, elective general surgeons usually have time and information, such as imaging and history, to allow planning for operative procedures, even if only during the brief initial workup. In contrast, surgeons faced with a trauma patient do not know what disease process they will find on opening, even when guided by a CT scan, resulting in further delay in control while determining injuries. Moreover, these patients are more likely to be unstable and/or unresponsive, resulting in less information and time for operative planning.

Taken in their entirety, these factors – delay in operative presentation, unknown pathology at the start of operation, difficult and delayed control of hemorrhage, and contamination – result in a major difference between general surgical and trauma patients. This is the concept of physiologic exhaustion that is found commonly in traumatized patients and occasionally in emergency general surgical patients. While elective general surgical patients should be fully evaluated and optimized before surgery, and emergency general surgical patients should be briefly "tuned-up" prior to surgery with fluid boluses, blood, and/or antibiotics, many trauma patients cannot wait even minutes for operative intervention due to extreme instability. These patients do not have any physiologic reserve and arrive in the OR in extremis. They may not tolerate the time under anesthesia needed to complete a full operative exploration and repair. Hence, using traditional approaches, these patients died either on the table during the course of their operation or shortly thereafter, due to ongoing nonmechanical bleeding, usually from coagulopathy or from subsequent multisystem organ failure. The underpinning for damage control is that the patient is incapable of undergoing a traditional operative approach due to physiological exhaustion and thus needs an abbreviated initial operation controlling only hemorrhage and contamination to expedite the aggressive resuscitation in the intensive care unit.

2.3 The Development of the Abbreviated Laparotomy

> He who fights and runs away, may live to fight another day. – JA Aulls, 1876

Gradually, changes in the operative approach toward this group of extremely ill trauma patients began to be discussed and published in the literature. Stone and colleagues were the first to describe aborting a laparotomy by the use of abdominal packing when intraoperative coagulopathy developed [1]. This report was published in 1983. Several subsequent reports of this technique, specifically for hepatic injury, and then a large series showing survival advantage by Burch and colleagues followed [2]. Unfortunately, adoption of this technique was slow and in some cases was deemed a failure to finish operating or an attempt to shift work to another time.

The next iteration of this technique by Rotondo and colleagues resulted in renaming this care pattern "Damage Control" [3]. It should be noted that despite the name, derived from the navy ship damage management, this was a civilian trauma development rather than military. The "Damage Control" sequence was defined. Since then, and with a new name, the technique has become increasingly accepted and has resulted in undoubted decreases in mortality.

2.4 Basic Tenants of Damage Control

2.4.1 Damage Control Part 0: Rapid Transport to Definitive Care

A crucial part of salvage in the selected extremely unstable trauma patients is the rapid transportation to a center capable of providing definitive care. The most direct method of transportation with the fewest delays in transitional facilities is necessary to maximize survival [4]. During this period, judicious resuscitation should be under

way. The traditional approach of normalizing vital signs in patients with prolonged transport times is inadvisable. Permissive hypotension, resuscitating patients to goal systolic pressure of approximately 90 mmHg with concomitant signs of end-organ perfusion, allows for adequate tissue perfusion while decreasing the potential for clot disruption from increased hydrostatic pressures. For patients who present with injury complexes generally leading to hemodynamic instability or those who exhibit instability, this approach should be extended in the trauma resuscitation area at the trauma center where Damage Control Resuscitation (blood-plasma-platelets), limiting crystalloids and utilizing goal-directed hemostatic resuscitation, is preferred. Damage Control Resuscitation, the details of which are covered in subsequent chapters of this text, compliments the Damage Control Surgery Concept, and when utilized together, mortality can be significantly reduced.

2.4.2 Damage Control Part 1: Rapid Control of Hemorrhage and Contamination

Operative intervention is focused on full exposure and rapid hemorrhage control. For major hepatic injury, packing is optimal, though multiple other more time-consuming methods may be necessary. Major vascular injury that cannot be safely treated by ligation can be considered for vascular shunting. However, ongoing arterial bleeding, whether in a viscera or cavity, will not be controlled by packing alone – surgical control is required. Intestinal contamination should be controlled by whipstitch, intestinal ligation, or stapling. No attempts at formal resection are undertaken, and the intestine is left discontinuous. Details of management of specific organs are found in further chapters. The abdomen is closed by one of many quick temporary methods. The entire operative intervention should take about 1 h and certainly no longer than 90 min. Effective utilization of Damage Control Resuscitation may indeed extend this window of operative intervention.

2.4.3 Damage Control Part 2: Resuscitation

Once out of the operating room, attention is turned to full resuscitation in the intensive care unit. Coagulopathy, anemia, acidosis, electrolyte abnormalities, and hypothermia should be aggressively corrected. Normalization of physiology is an indication to return for definitive operative care and is usually accomplished in 24–36 h. With the advent and effective use of Damage Control Resuscitation techniques, the frequency and degree of physiologic perturbation is decreasing, and the time to normalization is reduced.

However, patients who fail to improve or have subsequent worsening of parameters must be considered as having either ongoing bleeding or a missed injury. These patients are returned to the operating room as an emergency for another look, which should be thought of as a return to Damage Control Part 1, with limited goals of hemorrhage control, identification of injury, and prevention of ongoing contamination. In some patients, several cycles through Damage Control Parts 1 and 2 may be necessary.

2.4.4 Damage Control Part 3: Return for Completion of Operative Repairs

When fully resuscitated and physiologically normalized, patients will tolerate a second surgical insult and longer operative times. They are then returned to the operating room for unpacking, second look, and definitive management of injuries. During this operation, all injuries should be clearly identified and repaired, including recreation of intestinal continuity. The luxury of the second look as well as potential difficulties with abdominal wall closure has led to an increase in primary anastomosis for colonic injuries, with good results. Feeding access should be considered in all of these patients. About half of this selected population will be able to tolerate primary fascial closure during this operation. The remainder is managed with sequential closure methods, primary allograft closure, or granulation and skin grafting (Figs. 2.1 and 2.2).

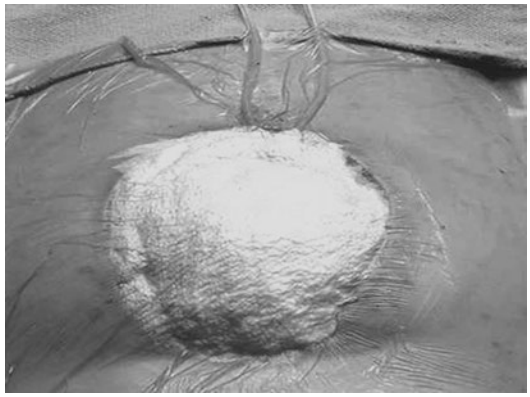

Fig. 2.1 As edema resolves, the defect becomes smaller and may be able to be closed primarily. The vacuum dressing is easily and inexpensively created with plastic sheeting against the bowels, gauze, drains, and an adhesive dressing

Fig. 2.2 Abdominal defects that cannot be closed primarily are allowed to granulate, usually via absorbable mesh, and then are skin grafted

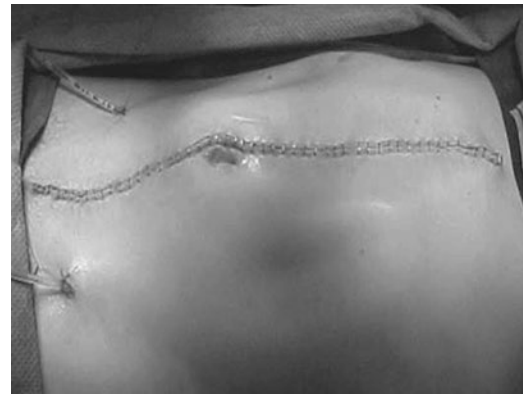

Fig. 2.3 Once the skin graft can be separated from the underlying intestines, the patient can undergo component separation and reconstruction of the abdominal wall

2.4.5 Damage Control Part 4: Definitive Abdominal Closure

A section of patients managed with Damage Control cannot be safely closed at the completion of Damage Control Part 3, either due to high intra-abdominal pressures or contamination requiring repeated washouts. Some can be closed subsequently during their hospital course. Historically 50–60 % of Damage Control patients were discharged with definitive abdominal closure but with the application of Damage Control Resuscitation and concomitant goal-directed hemostatic techniques – visceral edema is more limited and definitive closure rates are now steadily climbing [5].

The remaining patients are treated with a temporizing method, such as vicryl mesh and skin grafting, until they have completely recovered from their metabolic insult. Typically, these patients will be at home for 6–9 months, recovering mobility and optimal nutritional condition during which time the skin graft separates from the underlying intestines. At this time, an elective return to the operating room is undertaken for abdominal closure, with component separation and/or mesh or allograft, as well as stoma reversal if needed (Fig. 2.3). Long-term outcomes in these patients have been shown to be quite good.

2.5 Indications for Damage Control

2.5.1 Early Decision Making

In order for patients to benefit from a Damage Control sequence, the decision to abort operative intervention must be made early. It should be considered even prior to the arrival of the patient if there is hypotension in transport or in the trauma resuscitation area. While hypotension may well resolve with resuscitation, it is an early indicator that the patient is not prepared to tolerate a pro-

longed operation. Elevated lactate and base deficit are also early warning signs of physiologic derangement. While neither alone is an indicator for abbreviated laparotomy, they should induce the thought process. Absolute indicators will be discussed below; however, it cannot be stressed enough that a Damage Control operation should take only 60–90 min, and hence the decision to abort should be made early in the operation. Waiting to abort until the patient has reached physiologic exhaustion makes salvage extremely unlikely and results in almost certain death.

2.5.2 Triad of Death

There is extensive evidence that coagulopathy, acidosis, and hypothermia all interact to worsen each other in a vicious spiral that eventually results in ongoing hemorrhage and death. Early recognition of any of these findings is an indicator for Damage Control Resuscitation as well as a Damage Control abbreviated laparotomy. While many studies indicate varying absolute numbers, temperature less than 34, pH less than 7.2 (or base excess greater than 8 in a patient with a corrected pH due to hyperventilation), and/or laboratory or clinical evidence of coagulopathy should result in initiation of the Damage Control Approach [6]. Continued interaction with the anesthesia team is necessary to maintain awareness of these factors while operating. There is growing evidence of improved outcomes with layering damage resuscitation into damage control laparotomy. Clearly as our understanding of resuscitation has evolved over the last 15 years and refinement of Damage Control Surgery has ensued, survival rates continue to improve [7, 8].

2.5.3 Associated Injuries

Other injuries may contribute to the decision to interrupt laparotomy. Patients with multiple intra-abdominal injuries should be considered for abbreviated laparotomy at each stage of repair, as the time necessary for complete repair becomes rapidly prohibitive. This is seen in patients with multiple widely spaced intestinal injuries or combined vascular and intestinal injuries. Other sources of blood loss also contribute, though they are of lesser immediate concern, such as extremity fractures and lacerations; but they cause concern as the loss of blood from these is often underestimated when hidden either by the skin or the drapes. Similarly orthopedic injuries can and should be temporized in these patients [9].

Multi-compartment injuries also call for Damage Control, such as management of hemorrhage of the abdomen and the chest. Clearly, full management of abdominal injuries and closure would compromise a patient who also requires thoracic exploration. Hence, rapid termination and temporization within one compartment followed rapid control and temporization within another compartment cuts the total operative time, blood loss, and heat and evaporative losses. This will rarely result in patients with Damage Control dressings on both abdominal and thoracic incisions or on combined abdominal and sternotomy incisions (Fig. 2.4).

Any other potentially life-threatening extra-abdominal injury that requires timely intervention is an indicator to stop operating after hemorrhage and contamination control and provide a temporary closure. This allows for more rapid evaluation of these associated injuries such as severe intracranial injury or aortic transection, as well as early and aggressive correction of coagulopathy, which could contribute to mortality in these injuries. This is also the most efficient way to get patients with liver or pelvic injuries to angiogram if indicated.

Fig. 2.4 Damage control of combined sternotomy and laparotomy. Note massive abdominal distention

Lastly, the variability of the physiologic reserve should be assessed for the patient. Older patients and/or those with comorbidities are likely to be intolerant of long operative times and should have frequent reassessment of the need for abortion of the procedure.

2.5.4 Predicted or Present Abdominal Compartment Syndrome

While abdominal compartment syndrome was a pervasive problem 20 years ago, it is encountered far less frequently now with the use of Damage Control Resuscitation. Nonetheless, prediction of patients who are likely to develop abdominal compartment syndrome, and therefore selectively leaving these patients open with a temporary abdominal closure rather than closing fascia still remains an important adjunct to Damage Control Surgery. This is done even in patients with definitive completion of their operation to prevent the cascade of physiologic injury occurring with abdominal compartment syndrome. Patients at risk for developing massive visceral edema are those who have received more than 10–15 units of blood products and/or more than 5 L of crystalloid [10]. Additionally, any patient with increasing peak ventilatory pressures of more than 10 points at fascial approximation is at extremely high risk.

2.5.5 Planned Reoperation

Finally, temporary abdominal closure can be done in any patient who requires further evaluation prior to completion of repair of injuries, such as planned second look or serial washouts or debridement.

2.6 Expansion of Damage Control Principles

With the success of the Damage Control sequence in visceral trauma and its general adoption by the trauma community, it is increasingly utilized in other traumatic injuries [11, 12]. Vascular and now orthopedic injuries are treated by Damage Control techniques, which is the focus of this text. The utilization of this technique can be expected to improve the limb salvage, though data from large studies are not yet available. Additionally, the concept of damage control and the lethal triad has also spilled over into general surgery and is likely resulting in improved outcomes in this population as well.

2.7 Summary and Conclusion

The evolution of the abbreviated laparotomy or "Damage Control" for trauma has improved patient survival by decreasing the operative stress on patients in physiologic exhaustion. This technique requires rapid control of bleeding and contamination, temporary abdominal closure, and then intensive care resuscitation of physiology with return to the operating room for eventual definitive operative repair. This sequence should be utilized in patients with coagulopathy, acidosis, and hypothermia. While mortality in a subset of critically ill trauma patients has decreased with this modality, these patients have a very high incidence of morbidity and frequently require prolonged hospitalization and multiple operative procedures. The addition of Damage Control Resuscitation has not only decreased mortality further but also reduced morbidity. The success of Damage Control in management of abdominal pathology has led to the expansion of the concept into orthopedic and vascular trauma and into all aspects of surgical care.

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Changing Epidemiology of Polytrauma

3

Fiona Lecky, Omar Bouamra, and Maralyn Woodford

Epidemiology is the study of health and disease in populations, the scientific approach typifying public health medicine. The paradigms are somewhat different to the reductionist approach of much clinical science, which seeks to understand disease processes at an "omic" level. The rationale that underpins epidemiology suggests effective disease control must begin and end by understanding the impact of a disease (and its prevention/management strategies) at a population level, globally, nationally and locally, including the identification of vulnerable groups, aetiological factors and societal costs.

An epidemiological perspective on polytrauma – significant injuries affecting more than one body region – and its management must draw from the significant "injury control" literature. The latter has only just started to distinguish between polytrauma and major injury to a single body system. However, it still sets an important context for more detailed descriptions of polytrauma found in trauma registries. This chapter will first update on the global injury burden prior to a polytrauma focus.

3.1 Global Burden of Injury

Trauma fulfils the disease classification criteria for a global pandemic, this being a recurrent and significant cause of morbidity and mortality over time and across continents despite efforts to control its impact. Worldwide over 10,000 people die every day as a result of an injury [1]. Injury accounts for 10% of all disability-adjusted years of life globally lost. The injury control literature identifies road traffic collisions (RTC), falls and intentional violence (including self-harm) as the major vectors of traumatic injury, and this has been the case since 1990 [2]. Undoubtedly the major burden of injury is increasingly occurring in middle- and low-income countries as they industrialise and adopt motorised transportation. Despite a lower population incidence, injury remains the commonest cause of death and disability in children and young adults in the developed world [2].

Annual incidence and trends over time vary across the developed world. Data is obtained from national statistics which use International Classification of Disease codes, a taxonomy

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with limited descriptions of injury severity. The Abbreviated Injury Scale (AIS) dictionary has a greater level of detail (over 2000 injury codes) and allocates to every injury a severity score between 1 (mild) and 6 (maximal) [3]. These can be summated into the Injury Severity Score (ISS) [4] as a global reflection of the anatomical severity of injury suffered by each individual patient. Severe injury is defined as ISS >15. Within Europe, most hospital admissions with injury have much lower ISS values (range 4–8), being due to single isolated limb fractures in children or the elderly (falls) and isolated mild head injury (blunt assault) in young adults.

3.2 Defining Polytrauma and Data Sources for Study

As precise descriptions of injury severity are required to diagnose polytrauma (Box 3.1), databases using AIS as opposed to ICD injury classifications are needed. The definition of polytrauma has been renewed recently in an international panel and the following parameters were calculated on the basis of a nationwide registry:

Box 3.1

Polytrauma can be defined as significant injury[a] in at least two out of the following six body regions:

Head, neck and cervical spine
Face
Chest and thoracic spine
Abdomen and lumbar spine
Limbs and bony pelvis
External (skin)

[a]Significant injury = abbreviated injury score of ≥3 out of 6

An abbreviated injury score of greater than 2 points in at least two body regions in conjunction with a pathological value of another kind. Five different parameters were identified as follows: systolic blood pressure smaller or equal 90 mmHg, Glasgow Coma scale equal or smaller than 8 points, base excess smaller or equal 6.0, partial thromboplastin time greater or equal 40 seconds and age greater or equal 70 years [5]. We have previously identified that national trauma registries are best placed in this regard and utilised the Trauma Audit and Research Network (TARN), the largest European trauma registry to this end [6, 7].

It is important to note that with the Box 3.1 definition, the limbs and bony pelvis constitute one body region; therefore multiple limb fractures or a limb and pelvic fracture will not constitute polytrauma without injuries to either head/abdomen/thorax/face/external areas.

3.3 Changing Epidemiology of Polytrauma

In 2007 we characterised polytrauma epidemiology over the period 1989–2003; we have repeated this analysis utilising a 2008–2013 data set which enables us to study the changing epidemiology of polytrauma [6, 7].

In this recent study of over 180,000 patients submitted to TARN, it can be seen from Table 3.1 that most patients eligible for TARN have not experienced polytrauma.

For there to be significant trauma to more than one body regions, the ISS should be >18, and the median ISS for all age groupings is 9 in the trauma registry sample. The mortality rates increase significantly after age 65 when RTC causes a much lower proportion of injuries. Within this age group, there is also a reversal of male preponderance and an association of higher mortality with male gender which is probably

Table 3.1. Trauma Audit and Research Network data

Age groups	0–15 *n* = 12,647	16–65 *n* = 110,646	>65 *n* = 60,548
Age (years) (median)	7.30	44.30	81.30
ISS	9	9	9
% male	68.6%	70.7%	42.3%
% injured by RTC	30.2%	32.0%	9.5%
Mortality %	2.4%	3.7%	10.8%

due to comorbidity and has been described elsewhere [8]. The major changes from our previous epidemiological characterisation are:

(i) The increase in the proportion of all major trauma sustained by older adults from 21% in 1989–2003 to 33% in this series ($p < 0.001$), reflecting perhaps the ageing of the population and also a cohort of frail people – vulnerable to falls – who now survive to beyond 65. The latter may be due to improved lifestyles, better primary care detection/management of risk factors for early deaths and better secondary care.
(ii) The reduction in the proportion of all major trauma that is caused by RTCs in children (from 46% to 30%), adults (from 40% to 32%) and most notably in older people (from 19% to 9%) which is consistent with the hypothesis expressed in (i). Other mechanisms such as falls in older people and sport in the young now predominate.
(iii) Within each age/gender cohort, a halving of mortality in acute care despite no change in the median ISS (mortality rates were 5.5, 7.9 and 24.7% for male children, younger adult males and older adult males in 1989–2003, respectively). This may reflect improved care or increasing health in the population, hence improved resilience to major injury.

The gender split within each age cohort is unchanged from 1989 to 2003, but the median age for all TARN patients has increased from 33 to 41 years in younger adults and 75–79 years in older adults, whereas in children it has fallen from 11 to 7 years [6].

3.4 Polytrauma Demography, Causes, Incidence Within Trauma Registry and Outcome

It can be seen from Table 3.2 that 12% (22,032/183,841) of trauma registry cases have polytrauma, that is, significant injuries in more than one body region (given that the limbs and pelvis constitute one body region). This is a slight increase from our previous report of 10% prevalence and is accounted for by a relative increase of 30% (11.5–15.0) in the proportion of all major trauma cases in younger adults resulting in polytrauma. This finding may seem a little counterintuitive with a fall in proportion of all major trauma being caused by RTCs and may reflect better detection of polytrauma with the increased use of 3D imaging. Similar to our previous analysis, the risk of polytrauma is lowest in older adults (7.1%). The median ages for polytrauma do now (in contrast to our previous report) differ significantly from that from other trauma registry cases for children (10.2 versus 7 years) and younger adults (36.5 versus 45 years), less so for older adults (78.1 versus 81 years).

The proportion of polytrauma cases caused by road traffic collisions (RTC) approximately doubles when compared to proportion of all major

Table 3.2. Trauma Audit and Research Network

Polytrauma cases by age groups, 2008–2013					
Age groups		0–15	16–65	>65	Total
Polytrauma		1198	16,547	4287	22,032
Proportion of all trauma cases %		9.5%	15.0%	7.1%	12.0%
Age (years) (median)		10.20	36.50	78.10	41.90
ISS (median)		29	29	26	29
Cause of injuries	RTC	841	10,410	1561	12,812
	%	70.2%	62.9%	36.4%	58.2%
Gender	Male	789	12,811	2297	15,897
	%	65.9%	77.4%	53.6%	72.2%
	Female	409	3736	1990	6135
	%	34.1%	22.6%	46.4%	27.8%
Mortality at 30 days %	n	120	1685	1050	2855
	%	10.0%	10.2%	24.5%	13.0%

injuries caused by RTCs. The RTC proportion quadruples in older adults (36 versus 9%). However, the proportion of polytrauma cases caused by RTCs across all age groups has fallen to 58% overall – from 73% in 1989–2003. This fall in has occurred most dramatically in older adults where polytrauma is now caused by RTCs in only 36% of cases compared to 69% in 1989–2003, whereas the proportion has only reduced by 10% in absolute terms in the other age groups [6]. Penetrating trauma is responsible for 3.9% (n = 848) of all polytrauma cases over this timeframe, an increase on previous of 2.1%. Male preponderance decreases somewhat in children (66% vs. 68%, a change in direction from 1989 to 2003) and increases in younger and older adults by 7% and 10%, respectively.

The ISS scores of polytrauma patients are treble that of patients with major isolated injury (29 vs 9) but are slightly higher than the median of 25 previously reported; despite this, mortality associated with polytrauma has halved in nearly all age gender cohorts from 20% to 10% in those <65 and from 40% to 20% in those >65 years). However, mortality still increases 2.5–4-fold across the age groups when compared to isolated injury to approximately 10% in the young and 24% in those over 65 years. In this cohort, 62% (versus 75% in 1989–2003) of polytrauma deaths occur in children and people of working age [6].

3.5 Patterns of Injury as Markers for Polytrauma and Changes over Time

Of the 22,032 polytrauma cases detailed here, 50% (11,122/22,032) have at least one limb+/or pelvic fracture – this is a marked reduction from our previous polytrauma series where the 73% of cases had these extremity injuries. As per our previous analysis, however, most (47,581/58,703) = 81%) extremity injuries occur in isolation. Half of all polytrauma cases have a traumatic brain injury (TBI) and two thirds (as opposed to 50% in our previous report) a thoracic injury, but in contrast to thoracic injury, significant TBI injury occurs in isolation in three quarters of cases. Significant thoracic trauma is more likely to occur in the context of polytrauma (15,288/33,705 = 45%) as is significant abdominal trauma (3741/6880 = 54%). Significant abdominal trauma however is present in only 17% of polytrauma cases (3741/22,032). Injuries to the face and external (skin) body regions are rare in the context of polytrauma. The increasing prevalence of thoracic trauma and reduction in prevalence of extremity injury is the main observed change in polytrauma injury pattern when compared to previous study.

3.6 Polytrauma Mortality, Impact of Age and Body Area and Changes over Time

Table 3.3 indicates that polytrauma with TBI has generally the highest mortality rate across all three age groups (12.5%, 16%, 29.9%). In children, polytrauma with thoracic trauma confers the highest risk of mortality (12.6%), whereas in older adults it is polytrauma with abdominal injury (31%). This is somewhat changed from our previous analysis where abdominal polytrauma was the most lethal across all age groups. For isolated injuries and polytrauma, mortality does not appear significantly different between children and adults of working age, but there is a sharp rise in mortality for all patterns of injury after 65 years; this was true in our earlier study. Interestingly the relative increase in mortality with age being greater for cases of isolated injury in our previous analysis still applies.

In terms of overall mortality burden, it is first interesting to note that the impact of polytrauma is greater than the sum of its parts for children and young adults. For example, if the rates of mortality for isolated injuries of the limb/pelvis, brain, thorax and abdomen are summated for children from Table 3.3, this comes to 6.1% which is less than the polytrauma mortality rates

Table 3.3 Patterns of injury and mortality in polytrauma and isolated injury, by age group

Injury type for polytrauma						
	Frequency	Percent				
Blunt	21,183	96.1				
Penetrating	849	3.9				
Total	22,032	100.0				
Patterns of injuries and mortality in polytrauma and isolated injury, by age groups, 2008–2013						
			0–15 years mortality %,	16–65 years mortality %,	>65 years mortality %,	Overall mortality %,
			No. of deaths	No. of deaths	No. of deaths	No. of deaths
Limb AIS 3+	Isolated (*n*)	47,581	0.1% (4)	0.8% (232)	5.2% (721)	2% (957)
	Polytrauma (*n*)	11,122	6.5% (36)	7.5% (639)	19.6% (401)	9.7% (1076)
TBI AIS 3+	Isolated (*n*)	39,745	2.3% (75)	6.6% (1388)	19.3% (2973)	11.2% (4436)
	Polytrauma (*n*)	11,302	12.5% (103)	16% (1298)	29.9% (713)	18.7% (2114)
Thoracic AIS 3+	Isolated (*n*)	18,417	2.7% (11)	2.2% (266)	9.7% (583)	4.7% (860)
	Polytrauma (*n*)	15,288	12.6% (102)	12.4% (1474)	28.4% (738)	15.1% (2314)
Abdominal AIS 3+	Isolated (*n*)	3139	1% (6)	2.3% (52)	7.6% (18)	2.4% (76)
	Polytrauma (*n*)	3741	11.2% (26)	14.9% (463)	31.9% (127)	16.5% (616)

for children with TBI/chest or abdominal injuries. Although polytrauma accounted for only 12% of cases in this trauma registry sample, it accounted for almost a third (2860/9189 = 31%) of all deaths, the remainder occurring mainly in the context of isolated significant head injury and limb fractures in the elderly. This analysis has not dealt in detail with the disability consequences of polytrauma; inevitably they are considerable, but large series are rare due to the challenges of follow-up [9]. It appears that a major improvement in care for polytrauma groups of all ages has occurred as evidenced in the large relative decreases in in mortality for all age groups regardless of body region injured compared to 1989–2003. The biggest gains have been in the management of polytrauma in children (Fig. 3.1). This now means that polytrauma accounts for a third rather than a half of all in hospital trauma deaths.

3.7 Summary

Injury is a global pandemic and the second most costly disease worldwide with the burden set to increase. Within the largest European trauma registry, true polytrauma (ISS>17 with at least two AIS 3+ injuries) using abbreviated injury scale criteria occurs, in only 12% (2008–2013) of cases but causes up to one third of all deaths in patients reaching hospital alive, mainly in adults <65 years. Road traffic collisions remain the predominant cause, but falls are catching up. The thorax and head are now the most frequently injured body area in polytrauma as opposed to limb/pelvic injury in previous reports. However, thoracic and abdominal traumas remain specific markers for the presence of polytrauma. Traumatic brain injury in the context of polytrauma carries the greatest mortality risk. Polytrauma is less frequent in those over 65 years, but the increasing silver trauma phenomenon with the associated higher mortality means this generation accounts for one third of recent polytrauma deaths. Major improvements in mortality (50% in relative terms) are observed since this registry reported on a 1989–2003 cohort. However, within the younger age groups, the mortality associated with polytrauma is greater than the sum of its parts suggesting a role for further targeted improvements in care across the generations.

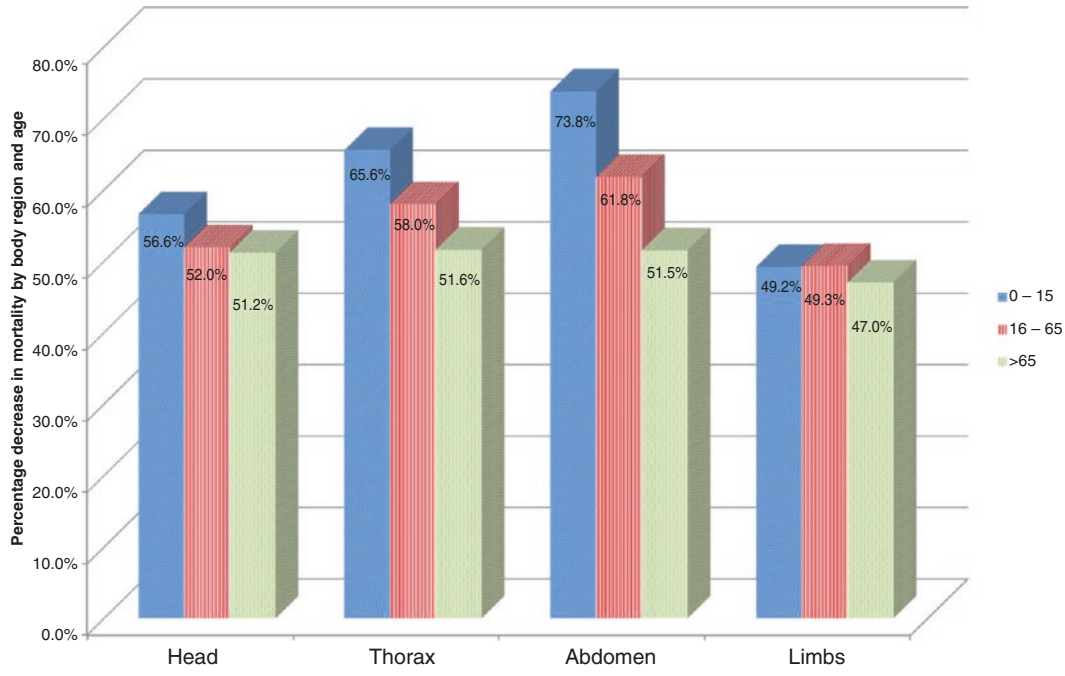

Fig. 3.1 Relative % decrease in polytrauma mortality by body region and age $\left(\frac{\text{Mortality rate}\,2008-2013}{\text{Mortality rate}\,1989-2003}\right)\times 100\%$

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Response to Major Injury

4

Todd W. Costantini and Raul Coimbra

4.1 Systemic Inflammatory Response Syndrome

The systemic inflammatory response syndrome (SIRS) after injury occurs as a result of a complex set of interactions which result in a substantial host immune cell response and activation of both the complement and coagulation cascades. SIRS is defined as two or more of the following: (1) temperature >38 °C or <36 °C, (2) respiratory rate >20 breaths per minute or pCO_2 <32 mmHg, (3) heart rate >90 beats per minute, and (4) white blood cell count >12,000 or <4000 or >10% bands (Table 4.1). This SIRS response occurs within minutes of injury as a result of tissue hypoperfusion and tissue injury. While an adequate host inflammatory response is an essential component of the injury response, there is also the potential to cause significant damage to host tissues. A sustained inflammatory response to injury has been correlated with increased morbidity and mortality in trauma patients [1].

Table 4.1 Systemic inflammatory response syndrome (SIRS) criteria

Two or more of the following:
Temperature >38 °C or <36 °C
Respiratory rate >20 breaths per minute or pCO_2 <32 mmHg
Heart rate >90 beats per minute
White blood cell count >12,000 or <4000 or >10% bands

4.2 Immune Response to Injury

While the SIRS response in sepsis is mediated by invading pathogens, major trauma incites a sterile inflammatory response (Fig. 4.1). In trauma patients, hemorrhage can result in a period of ischemia that results in tissue hypoxia. The restoration of adequate volume and tissue perfusion after control of hemorrhage and resuscitation improves blood flow and causes reperfusion injury to injured cells. This ischemia-reperfusion injury causes oxidative stress on the injured cells and results in the release of free radicals and pro-inflammatory cytokines. If tissue ischemia is prolonged or ischemia-reperfusion injury severe enough, cells may undergo apoptosis or necrosis and further drive inflammation. Major trauma also elicits an immune response caused by tissue injury related to tissue damage itself. Both ischemia-reperfusion injury and tissue damage cause an

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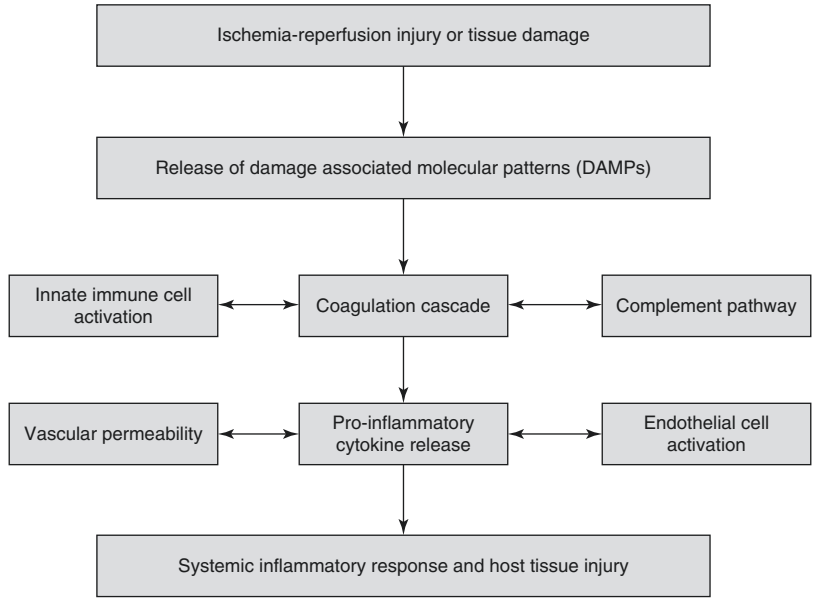

Fig. 4.1 Severe injury causes a systemic inflammatory response. Tissue ischemia-reperfusion after hemorrhage or tissue damage from trauma can elicit a sterile inflammatory response caused by release of damage-associated molecular patterns (DAMPs). This injury results in activation of the innate immune system, coagulation pathway, and complement cascade leading to a systemic inflammatory response (SIRS) and host tissue injury

innate immune response. The innate immune system consists of peripheral blood mononuclear cells (PBMC) and polymorphonuclear leukocytes (PMN). Major injury results in the release of damage-associated molecular patterns (DAMPs) which are released by damaged or dying host tissues and secreted by activated immune cells. The innate immune system initiates an inflammatory response when these DAMPs, also known as alarmins, bind to pattern recognition receptors on host inflammatory cells or activate the complement cascade. DAMPs are either proteins released from injured cells or products of abnormal cellular metabolism. Common protein DAMPs include heat shock proteins and high-mobility group box 1 protein (HMGB-1), while DNA, mitochondrial DNA, and microRNA are common nonprotein DAMPs released from injured cells [2]. DAMPs released after injury bind to pattern recognition receptors including toll-like receptors (TLRs) and nod-like receptors that propagate the injury response through activation of immune cells.

The TLR family is present on endothelial and immune cells and is a key mediator of the inflammatory response. Binding of a DAMP to the TLR activates numerous intracellular signaling pathways which upregulate the production of pro-inflammatory transcriptional factors. These transcriptional factors, including nuclear factor-kappa B (NF-KB), activator protein-1 (AP-1), and phosphoinositide 3-kinase (PI3K)/Akt, propagate the inflammatory response through the production of cytokines and acute-phase proteins. Pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin (IL)-1β exert several downstream effects which further propagate the inflammatory response. TNF-α modulates the immune response by stimulating the production of other pro-inflammatory mediators such as leukotrienes and prostaglandins which together mediate the inflammatory response after injury by increasing capillary leak, causing damage to epithelial and endothelial barriers, and increasing adhesion molecules on both inflammatory cells and vascular endothelial cells.

Recent studies from the Inflammation and the Host Response to Injury program ("Glue

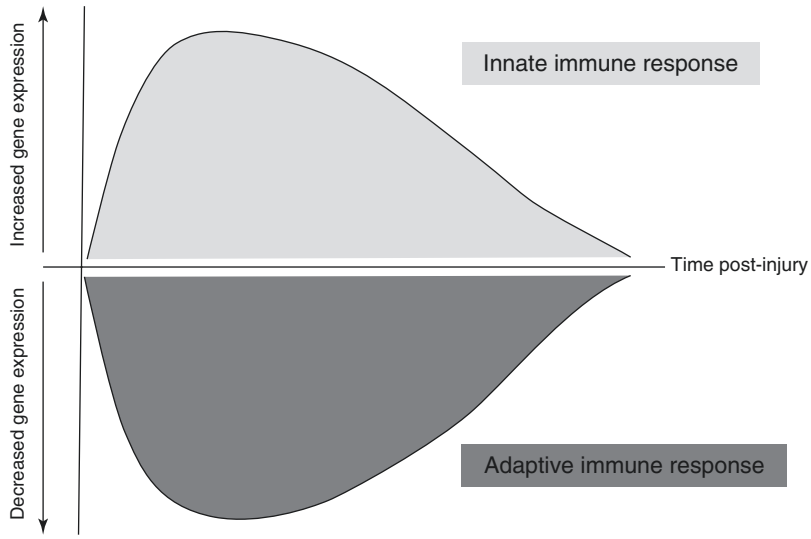

Fig. 4.2 Severe injury alters leukocyte gene expression. Injury causes wide-ranging changes in leukocyte gene expression after blunt trauma. Leukocyte gene analysis has demonstrated increased expression of genes associated with the innate immune response with a simultaneous suppression of adaptive immunity genes [1]. This suggests a systemic inflammatory response from innate immune cells with a simultaneous compensatory anti-inflammatory response (CARS) from adaptive immune cells (Modified from Xiao et al. [1])

Grant") have given important insights into the immune response to injury [3]. Prior dogma proposed a model describing the inflammatory response as an initial period of SIRS followed by a compensatory anti-inflammatory response (CARS) that was associated with immunosuppression [4]. Analysis of the leukocyte genomic response from severe blunt trauma patients enrolled in the Glue Grant demonstrated increased expression of genes associated with systemic inflammation and innate immune response with a simultaneous suppression of adaptive immunity genes consistent with the CARS. They found that over 80% of cellular functions and signaling pathways after severe blunt trauma were altered, a finding the authors termed a "genomic storm" [1]. While there was no difference in the genomic changes in patients that suffered a complicated recovery from injury, the genomic changes were increased and lasted longer than patients with an uncomplicated recovery. These findings have led to a new model describing the immune response to injury that describes a simultaneous dysregulation of both the innate and adaptive immune responses (Fig. 4.2) [1, 3].

Table 4.2 Components of the innate immune system

Immune cells	*Function*
Neutrophils	Most abundant phagocytic cell, produce reactive oxygen species that can damage host tissues
Monocytes	Recruited to sites of tissue injury, mature into macrophages
Macrophages	Phagocytosis of cellular debris and necrotic cells
Natural killer cells	Secrete cytokines, recruit other innate immune cells
Dendritic cells	Antigen presentation, activate T cells of adaptive immune system
Physical barriers	*Function*
Skin	Physical barrier to external environment
Mucosal epithelial cells	Physical, chemical, and mechanical barrier to prevent pathogens from accessing underlying tissue and immune cells

4.3 Innate Immune Cells

The innate immune system gives nonspecific immunity that provides the first line of defense against injury and infection (Table 4.2). These cells play a critical role in eradicating infection

by identifying foreign pathogens (i.e., bacteria) through pathogen-associated molecular patterns (PAMPs), preventing spread of infection, and destroying pathogens through phagocytosis and cytotoxicity. In the case of major injury, DAMPs activate the innate immune system to respond to injured cells/tissue through a coordinated response that results in cytokine expression, cytotoxicity, and ultimately activates cells of the adaptive immune system.

4.3.1 Neutrophils

Neutrophils are the predominant cell type of the innate immune system. Activated neutrophils are the primary effector cell in the acute inflammatory response. These circulating cells respond to injury signals by binding to adhesion molecules expressed on endothelial cells and enter injured tissue by chemotaxis in response to cytokines such as interleukin-8 (IL-8). After migration to the extracellular matrix in sites of tissue injury, neutrophils respond through degranulation and oxidative burst which is cytotoxic to surrounding cells and is designed to control local tissue injury. While PMN activation and degranulation plays a vital role in responding to injured cells and tissue, the release of reactive oxygen species and proteases during oxidative burst can be deleterious to the host tissues resulting in organ injury. Further, recruited neutrophils secrete several pro-inflammatory cytokines including TNF-α and interferon gamma (IFN-γ) that further drive the inflammatory response.

4.3.2 Monocytes/Macrophages

Monocytes make up a small portion of circulating cells at baseline, representing approximately 5–10% of leukocytes in the peripheral blood. The spleen acts as a large reservoir of monocytes that are poised to respond to injury or infection by release into the circulation and trafficking to sites of injury. Once recruited to sites of tissue injury, monocytes leave the circulation and mature into macrophages where they play a critical role in phagocytosing cellular debris and necrotic cells. Macrophages further propagate the inflammatory response by releasing the pro-inflammatory cytokines TNF-α, IL-1, and IL-12. In addition, tissue macrophages play an important role in the innate immune system as antigen-presenting cells that cause activation of T cells of the adaptive immune system.

4.3.3 Dendritic Cells

Dendritic cells (DCs) are the link between innate and adaptive immunity. DCs are antigen-presenting cells that serve as strong activators of the systemic inflammatory response. There are several subpopulations of DCs that have different tissue distribution and various roles in the inflammatory response. DCs are derived from hematopoietic stem cells in the bone marrow and can be classified as myeloid DCs or plasmacytoid DCs based on lineage and tissue environment. Myeloid DCs are derived from circulating monocytes, are rapidly recruited to sites of injury after major trauma, and secrete cytokines including IL-12 that primes T cell activation.

4.4 Gut Barrier Failure and the SIRS Response

The gut barrier performs a vital function in the innate immune system in preventing toxic luminal factors from accessing protected layers of the intestine [5]. It is well recognized that the loss of intestinal barrier integrity is the central culprit in the development of SIRS following injury. Several studies have demonstrated that severe injury results in compromised intestinal barrier integrity that is marked by structural and histological changes within the intestinal epithelium [6–9]. In the gut, a subset of myeloid DCs and local cytokines control the equilibrium between regulatory (Treg) and effector T cells (Th17) and coordinate the balance between tolerance to inflammation during homeostasis and an innate immune response that is required to response to injury [10, 11]. In homeostasis, resident gut DCs

drive the differentiation of naïve $CD4^+$ T cells to Tregs, which maintain tolerance through release of TGF-β and subsequent release of tolerant $CD103^+$ DCs into the mesenteric lymph [12]. After gut barrier failure following injury, pro-inflammatory stimuli activate resident gut DCs to produce the pro-inflammatory cytokine IL-6 that drives the development of pro-inflammatory effector Th17 cells [13]. These Th17 cells respond to gut injury through release of IL-17, with increased neutrophil recruitment to the gut and further release of pro-inflammatory mediators. These gut-derived pro-inflammatory factors are carried through the mesenteric lymph where they propagate the SIRS response by causing endothelial and red blood cell dysfunction, neutrophil activation, cytokine release, and potentially distant organ injury [14]. Therefore, the gut has been termed the "motor" of the SIRS response after injury with numerous preclinical studies aimed at limiting gut inflammation as a means of limiting distant organ injury after major trauma [15, 16].

4.5 Coagulation Cascade

Acute coagulopathy occurs early after severe injury, with clinically significant coagulopathy noted in approximately 25% of patients upon arrival to the emergency department [17, 18]. In a multicenter study, an elevated international normalized ratio (INR) greater than 1.5 at admission was associated with an increased risk of multiple organ failure and death [19]. Cross-talk between inflammation and the coagulation cascade can result in a self-perpetuating cycle that drives the SIRS response. Platelets become activated by trauma and serve as a source of pro-inflammatory mediators that activate innate immune cells and drive the SIRS response. Similarly, tissue injury and DAMP release after trauma cause activation of endothelial cells and immune cells which results in platelet activity and further drives the coagulation cascade. Acute coagulopathy after trauma is also associated with activation of the protein C pathway [20]. Activated protein C is a known mediator of inflammation and may be another link between coagulation and inflammation in the SIRS response to injury.

4.6 Complement Activation

The complement cascade is a part of the innate immune system which responds rapidly after injury [21]. Activation of the complement system also alters the coagulation cascade, linking these two pathways in the response to injury [22]. Levels of circulating activated complement proteins correlate with injury severity, contribute to multiple organ failure, and are related to outcomes [23, 24]. The complement system is comprised of circulating plasma proteins which are normally in their inactive state. Once activated by a stimulus such as the presence of foreign pathogens or tissue trauma, proteins of the complement system become activated. These activated complement proteins are responsible for driving the SIRS response through opsonization and phagocytosis and further cause the recruitment of other inflammatory cells to the site of tissue injury.

The complement cascade can be activated through three different pathways. In the classic pathway, complement proteins are activated by immunoglobulin G (IgG) or IgM bound to an antigen. The classic pathway is also activated by acute-phase proteins which are produced in response to injury. The alternate pathway of complement activation occurs when complement binds directly to the PAMPs displayed by bacteria, viruses, and fungi and does not rely on antibodies binding to the pathogen. The mannose-binding lectin pathway is similar to the classic pathway of complement activation. Mannose-binding lectin is produced by the liver in response to injury and infection and binds to the surface of invading pathogens, initiating the complement cascade. This system is highly regulated, as activation of the complement cascade can be damaging to host tissues. Some evidence suggests that activation of the mannose-binding lectin pathway may lead to MOF in patients with sterile inflammation [25].

4.7 Multiple Organ Failure

The exaggerated host response to the severe injury can lead to significant tissue injury, end organ dysfunction, and ultimately multiple organ failure (MOF). MOF is defined as progressive, potentially reversible dysfunction of two or more organ systems including the lung, liver, gastrointestinal tract, renal, and hematologic systems. Post-injury MOF is the most significant cause of late deaths in trauma patients [26–28]. Risk for MOF is related to the extent of injury and can be predicted early, where early cytokine production following injury may predict patients at risk for developing MOF [29]. There are several factors that can identify patients at risk of developing MOF. Scoring systems performed in the emergency department at the time of admission have the ability to predict the development of MOF within 7 days of hospitalization after injury [30]. Organ failure scores, such as the Sequential Organ Failure Assessment score (SOFA score) or the Denver MOF score, can be utilized to describe organ failure in critically ill patients and can predict mortality with mortality rates directly related to the number of organ systems which have failed [31]. ICU patients that develop MOF have a 20-fold increased mortality compared to ICU patients that do not develop MOF, as well as a significantly increased ICU and hospital length of stay [32].

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Defining the Lethal Triad

5

Mitchell Dyer and Matthew D. Neal

5.1 Introduction

Worldwide, trauma leads to millions of deaths and severe injuries each year. Analysis of the leading causes of death in trauma patients reveals that hemorrhage is one of the most common and preventable etiologies in the acute period [1]. For those that survive the initial insult, the cause of both morbidity and late mortality after trauma is, in part, excessive inflammation and a vicious cascade of coagulation abnormalities. Thus, traumatic hemorrhage can be broadly categorized into two groups: (1) early surgical bleeding and (2) coagulopathy. Surgical bleeding is secondary to the injury (e.g., splenic laceration, femur fracture), and treatment often involves mechanical control through surgical or interventional procedures. Coagulopathy following trauma presents in two distinct forms. It is now widely recognized that severe injury is characterized by a unique, endogenous coagulopathy, referred to as the acute traumatic coagulopathy (ATC) that may be present on admission in as many as 30% of injured patients [2]. This devastating condition presents a major obstacle in the care of trauma patients and is an evolving area of active research. The second form of coagulopathy following trauma is a consequence of iatrogenic factors and resuscitation. Previous resuscitation strategies involving large volumes of crystalloid and/or packed red blood cells in isolation (without blood component therapy) led to a dilutional coagulopathy. Although this remains a clinically important challenge, modern resuscitation strategies that limit crystalloid and focus on 1:1:1 ratio-based transfusion have limited this component substantially [3, 4]. In this chapter, we will focus on the previously named "lethal triad" which links coagulopathy with hypothermia and acidosis as major contributors to the ongoing hemorrhage despite control of surgical bleeding.

5.2 History

Coagulation abnormalities have long been described in both shock and trauma. A biphasic response of initial hypercoagulability followed by hypocoagulability was reported in experimental animal studies and humans in hemorrhagic shock [5–7]. Simmons and McNamara both reported clinical series of coagulopathy following massive trauma in Vietnam soldiers [8, 9]. As well, coagulation abnormalities have been reported with isolated traumatic head injuries [10].

In 1982, Kashuk et al. coined the phrase "bloody vicious cycle." They reported that in patients that suffered a major abdominal vascular

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injury, most mortalities were attributed to hemorrhage; however, a significant percent of those patients had control of the surgical bleeding yet continued to exsanguinate seemingly secondary to an uncontrollable coagulopathy [11]. This uncontrollable coagulopathy, along with synergistic effects from hypothermia and acidosis, constitutes the "bloody vicious cycle" or other times called the lethal triad (Fig. 5.1). It is important to recognize, however, that our understanding of the clinical problem has evolved substantially and that although hypothermia and acidosis likely contribute to ongoing coagulopathy, there is very clearly a unique endogenous coagulopathy that occurs in a subset of trauma patients. As such, the bloody vicious cycle may still exist for some patients; however, it is clear that some unfortunate, severely injured patients move straight to a coagulopathy that independent of other factors. In this chapter we will review the components of the lethal triad with a focus of the coagulopathy component, its underlying mechanism, identification of coagulopathy in the trauma patient, and treatment options.

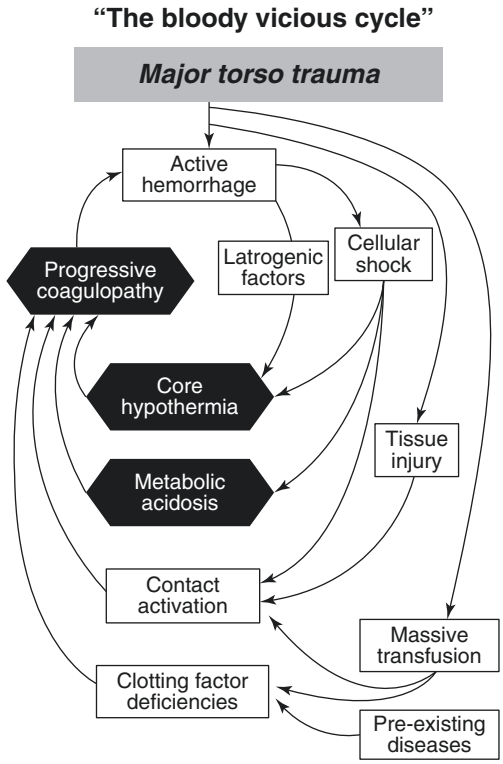

Fig. 5.1 "The bloody vicious cycle" is hypothesized to result from multiple factors that ultimately lead to acidosis, hypothermia, and coagulopathy which in turn results in ongoing hemorrhage despite surgical control of bleeding from organ injury (Reprinted with permission from Moore [12])

5.3 Hypothermia

Hypothermia in trauma patients can result from a multitude of reasons including alcohol intoxication, central nervous system injuries resulting in deranged thermoregulation, fluid resuscitation, and likely most importantly skin and body cavity exposure to the surrounding environment. In one series 66% of severely injured trauma patients were hypothermic defined as a core temperature <36 °C, on admission to the emergency department [13]. Furthermore the authors found the lowest recorded temperature was significantly lower in patients who died compared to those who lived (32.9 °C vs. 35 °C). As well, hypothermia was associated with increased time spent in the field, increased injury severity score (ISS), and increased volume of blood transfusions. Jurkovich et al. demonstrated that hypothermia was independently associated with mortality, and in patients whose core temperature fell to below 32 °C, there was not a single survivor [14]. While it is clear that patients who are severely hypothermic do not do well, it is unclear whether the hypothermia results from the injury or the hypothermia leads to poorer outcomes.

It has been postulated that the hypothermic effect on coagulopathy results from processes such as depression of enzyme function and platelet dysregulation [15–17]. In vitro studies that have analyzed the coagulation cascade found that enzyme function, platelet adhesion, and aggregation were impaired below 33 °C [18]. A series of trauma patient blood samples were analyzed with thromboelastography (TEG) that was adjusted to patient core temperature to identify the effects of hypothermia on coagulation [19]. Interestingly, TEG results demonstrated that most patients, regardless of core temperature, were hypercoagulable on presentation, although once

clot was formed, clot propagation was found to be slower in the most hypothermic (33.0–33.9 °C) patients. Platelet function during the clot polymerization phase and overall clot strength were also found to be less in patients with lowest core temperatures. While coagulation abnormalities can be shown in severe hypothermia both in vitro and in vivo, it is not clear whether the effects of hypothermia on coagulation are significantly relevant to most trauma patients. Still, rewarming measures should be taken in all hypothermic trauma patients to counter whatever contribution hypothermia adds to coagulopathy.

5.4 Acidosis

Trauma patients most often become acidotic secondary to hypoperfusion resulting from massive hemorrhage. This hypoperfusion leads to a switch from aerobic to anaerobic metabolism at a cellular level ending in a buildup of excess lactate. In 1964, Broder et al. demonstrated that an excess lactate level was associated with an increased risk of death and was a marker of progression of shock [20]. In a series of trauma patients admitted to the ICU, it was found that inability to clear lactate at 48 h was associated with a significant risk of death [21]. Acidosis is associated with poor outcomes in trauma patients, but the role in the triad is less defined.

Acidosis has been implicated in worsening the coagulopathy seen in severely injured trauma patients. In fact, acidosis has been found to be an independent risk factor in developing life-threatening coagulopathy in massively transfused trauma patients [22]. Dunn et al. demonstrated impaired hemostasis when blood pH dropped to less than 7.2 in an animal model [23]. TEG analysis of blood samples with altered pH levels demonstrated abnormal polymerization of clots and decreased time to clot development at severely depressed levels (pH = 6.8) [24]. Individual clotting factor functions can be directly impaired by acidosis. Factor VII is crucial in the initiation of clotting and formation of the platelet plug and recombinant forms have previously been used to treat coagulopathy in trauma patients. Meng et al. reported that at a pH of 7.0 compared to 7.4, factor VIIa activity was significantly decreased in an in vitro model [25]. In a pig model, acidosis was found to decrease plasma fibrinogen levels and decrease thrombin generation; however, in this same model when the acidosis was corrected with bicarbonate, the coagulopathy was not corrected as well [26, 27]. This suggests the effect of acidosis on coagulation may not be reversible or that acidosis and coagulopathy may be present but not directly linked. As with hypothermia the clinical contribution of acidosis to the net coagulopathy observed in trauma is difficult to fully assess. While it appears that the effect is likely mild until the acidosis is severe (<7.2), it cannot be ignored when treating the critically injured trauma patient.

5.5 Coagulopathy

The third component of the lethal triad is coagulopathy or the "nonsurgical" bleeding in critically ill trauma patients. Traditional thought had been coagulopathy developed secondary to coagulation factor loss in hemorrhage, dilutional effects from crystalloid resuscitation, and the contributing effects of hypothermia and acidosis as previously discussed [28]. This concept came under scrutiny due to observational reports of coagulopathy existing early after the sustained injury and prior to aggressive resuscitative efforts. In 2003, the term *acute traumatic coagulopathy* (*ATC*) was coined [29]. Defined by elevations in prothrombin time (PT), activated partial thromboplastin time (aPTT), and thrombin time (TT), it was discovered that nearly 25% of severely injured trauma patients demonstrated coagulopathy on arrival to the emergency department. Importantly, these patients received minimal prehospital fluid administration, and, on univariate analysis, there was no correlation between the amount of fluid given and development of coagulopathy. Around the same time, MacLeod et al. found a similar incidence of coagulopathy in trauma patients on arrival to the emergency department [30]. Further analysis of this patient population revealed coagulopathy,

defined by elevated PT or aPTT, was an independent predictor of all-cause mortality with increased adjusted odds of mortality by 35% and 326%, respectively. Together these two landmark studies redefined the thought process of coagulopathy in trauma. Because these coagulation abnormalities were identified on arrival to the hospital and without significant resuscitation prior to lab draws, it appears that there is an endogenous coagulopathy associated with severe trauma. This inherent coagulopathy has been referred to by different names including *acute coagulopathy of trauma* (*ACOT*), *acute coagulopathy of trauma-shock* (*ACOTS*), *early trauma-induced coagulopathy* (*ETIC*), and *trauma-induced coagulopathy* (*TIC*) in addition to ATC [31–34]. Going forward we will refer to this endogenous coagulopathy as *ATC*.

While there has been a shift in the understanding of *ATC*, the underlying mechanism has yet to be elucidated. Initial work in shock and observations of coagulopathy in military trauma victims attributed the hypocoagulable state to onset of disseminated intravascular coagulation (DIC) [5, 8, 35, 36]. Others have argued that while DIC and *ATC* share similar changes, they are, in fact, distinct entities as *ATC* does not truly meet DIC criteria, nor do most trauma patients exhibit disseminated clot burden [37–39]. Two current leading hypotheses are (1) the activated protein C (aPC) and (2) fibrinolysis pathways (Fig. 5.2).

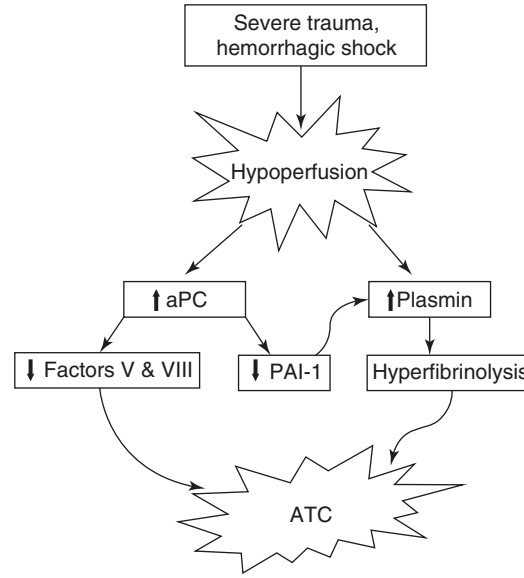

Fig. 5.2 Mechanisms of acute traumatic coagulopathy (ATC): The aPC and hyperfibrinolysis pathways are leading hypothesis for the underlying mechanism of ATC. As shown here there is cross talk between pathways, and there is likely multiple pathways contributing to development of ATC. As well, hypoperfusion is a key component to development of ATC; when absent in many studies, ATC does not develop. *aPC* activated protein C, *PAI-1* plasminogen activator inhibitor 1, *ATC* acute traumatic coagulopathy

5.5.1 Activated Protein C

Protein C is a protease that plays an important role in normal hemostasis. It is activated by a complex reaction that is dependent on thrombin, thrombomodulin, and the endothelial protein C receptor [40]. Activation of protein C (aPC) with cofactor protein S leads to inactivation of factors V and VII in the coagulation cascade. Another anticoagulant effect of aPC is inhibition of plasminogen activator inhibitor 1 (PAI-1), which then results in increased active plasmin and increased fibrinolysis (Fig. 5.2).

The aPC hypothesis proposes that severe injury and shock leads to tissue hypoperfusion, activation of protein C, and bleeding resulting from the downstream effects of the anticoagulant properties exerted by aPC. Brohi et al. found in trauma patients with tissue hypoperfusion, defined by an elevated base deficit, that low protein C levels were associated with prolonged PT and aPTT values [41]. Accordingly they also found in the same group of patients high levels of thrombomodulin, low PAI-1, and high D-dimer levels that all suggest activation of protein C. In this study, low protein C levels were associated with increased mortality. These observations in trauma patients were taken to the laboratory, and in a mouse model of trauma and shock, the development of coagulopathy was associated with increased aPC levels [42]. Interestingly, when mice were pretreated with an antibody that selectively inhibits the anticoagulant properties of aPC, the coagulopathy was reversed; although when an antibody that completely blocks the

function of aPC was used, there was 100% mortality in the mice. Taken together, these data suggest that aPC has multiple roles in the severely injured trauma patients, some of which are protective. Later work by Cohen et al. that directly measured aPC levels in human trauma patients found that increased levels of aPC were associated with increased mortality, transfusion requirements, and organ injury [43]. Central to all of the findings discussed above is tissue hypoperfusion. Neither in humans nor mice was coagulopathy noted in those that did not exhibit evidence of hypoperfusion. The link between hypoperfusion and activation of protein C is yet to be elucidated.

5.5.2 Fibrinolysis

Fibrinolysis is a normal part of the homeostasis in coagulation; however, excess breakdown of normal clot production can lead to or exacerbate ongoing hemorrhage. It is hypothesized in *ATC* that severe injury leads to tissue hypoperfusion, activation of plasmin, and hyperfibrinolysis [44]. Using TEG analysis, fibrinolysis was identified in 34% of patients requiring massive transfusion [45]. Fibrinolysis was associated with tissue hypoperfusion (elevated lactate levels) and increased transfusion requirements, and logistic regression analysis revealed fibrinolysis as a predictor of hemorrhage-related death in the same group of patients. In a series of nearly 2,000 trauma patients, hyperfibrinolysis was only found in 2% of victims but was associated with mortality of 76% compared to 10% in patients who did not have hyperfibrinolysis [46]. Further work with TEG analysis in severely injured trauma victims found a fibrinolysis level of 3% or greater to be significantly associated with a higher risk or hemorrhage-related death [47]. The CRASH-2 study demonstrated that tranexamic acid, a drug designed to inhibit fibrinolysis, was associated with decreased mortality, lending further hypothetical support to the important role of hyperfibrinolysis. Despite the positive findings in terms of reduced mortality, it is unclear that the benefit of tranexamic acid in CRASH-2 was due to reduced fibrinolysis and/or a reduction in bleeding – this will be discussed further in the treatment section of this chapter. As with the aPC pathway, tissue hypoperfusion was found to be a critical component in patients with hyperfibrinolysis. On the other hand, recent work from Moore et al. stratified three fibrinolytic phenotypes (>3% hyperfibrinolytic, 0.81–2.9% physiologic, <0.08% fibrinolytic shutdown) and found increased mortality with both hyperfibrinolysis and fibrinolysis shutdown [48]. Patients with hyperfibrinolysis were dying from hemorrhage, whereas mortality associated with fibrinolysis shutdown was secondary multisystem organ failure. The mechanism underlying this remains to be elucidated, but the ability to identify and ultimately treat these different phenotypes in trauma patients will be a key future area of research. While there is evidence mounting for many different hypotheses of the mechanism of ATC, it is more likely that each hypothesis contributes to the overall pathophysiology. This can be easily seen between the aPC and hyperfibrinolysis pathways. As described earlier aPC has been shown to inhibit PAI-1 and therefore creating more available plasmin and, in theory, increased fibrinolysis. It is likely that there is cross talk between multiple pathways that contribute to ATC. What is clear is the importance of tissue hypoperfusion to the development of ATC regardless of the pathway that leads to it. It is also important to note the multiple functions of many of these pathways as evidenced by the 100% mortality rate in mice when there was complete blockade of aPC. From the available literature, it appears that while the understanding of ATC is progressing, there is still a large gap in knowledge and that the answer is likely to come from multiple pathways rather than a single one.

5.5.3 Traumatic Brain Injury-Associated Coagulopathy

The association between traumatic brain injury (TBI) and coagulopathy has been well documented, and the pathophysiology is believed to be secondary to release of tissue

factor into systemic circulation after breakdown of the blood-brain barrier, a period of microvascular thrombosis leading to coagulant factor consumption, and then coagulopathy, much like a DIC picture [49]. In 2008, a systematic review, a prevalence of ~33% for coagulopathy following TBI, was identified, and this was independently associated with increased mortality [50]. A recent systematic review of isolated TBI and coagulopathy found a prevalence of ~35% [51]. However, these reviews are difficult to interpret as the definitions for coagulopathy were heterogeneous in both reviews, and they include many studies that predate the studies by Brohi and MacLeod that defined *ATC*. Therefore, TBI certainly appears to be associated with coagulopathy, but its contribution, if any to the pathophysiology of *ATC*, remains uncertain. As well, a recent review of TBI did not find a greater degree of coagulopathy after isolated TBI compared to injury to other body regions [52].

5.6 ATC Identification

As *ATC* is associated with increased mortality in the trauma patient, the ability to correctly identify and therefore treat has become an increasing important topic of research. In the massive transfusion literature, which affects only approximately 5% of all trauma patients, there have been retrospective studies performed in attempts to develop scoring systems to predict patients who will require massive transfusion [53–55]. On the other hand, there is a paucity of standardized tests or algorithms for the "identification" of *ATC*. The initial studies by Brohi and MacLeod that defined *ATC* used elevated PT and aPTT values to define coagulopathy [29, 30]. However, multiple studies have shown PT/INR to be a poor predictor of coagulopathy in addition to the fact that elevation of PT or aPTT can be associated with a *pro*-thrombotic state [56–58]. As well, most standard coagulation tests only evaluate either a single factor or one side of the coagulation pathway, whereas trauma patients suffering from hemorrhage likely have multiple abnormalities, and therefore standard PT and aPTT are poor predictors of bleeding [59]. Others raise issue that standard coagulation tests do not account for fibrinolysis or platelet function and therefore ignore an important part of the hemostatic mechanism which limits the usefulness in guiding treatment [60]. Finally, these standard tests are plasma-based assays and lack the ability to account for other critical components of whole blood to coagulation.

A growing area of research in trauma coagulopathy and resuscitation is the use of thromboelastography (TEG) and rotational thromboelastometry (ROTEM) to assess coagulation status (Table 5.1 and Fig. 5.3). These similar, but different, machines use whole blood and analyze the viscoelastic properties as the blood goes through a clot formation and dissolution (fibrinolysis) [62]. The popularity of TEG/ROTEM is that functional information about clot development and strength can be obtained, whereas traditional coagulation tests only are able to provide information on levels in circulation. While TEG/ROTEM have been in clinical use for 60 years in cardiac and liver transplant surgery and have been shown to decrease blood product transfusions, its use in trauma is in its relative infancy [63–65].

Table 5.1 Thromboelastography values: typical readout from a TEG will provide many parameters of the coagulation status of a patient and allows the clinician to determine what component therapy to administer to the patient based on the individual parameter results

Value	Description	Normal range
Reaction time (R)	Time to thrombin generation	4–9 min
Kinetics (K)	Measure of time to reach 20 mm amplitude	1–4 min
Alpha angle (α)	Rate of clot formation due to fibrin cross-linking	47–74°
Maximum amplitude (MA)	Clot strength (80–80% platelets; 10–20% fibrin)	55–73 mm
LY 30	Fibrinolysis	0–7.5%

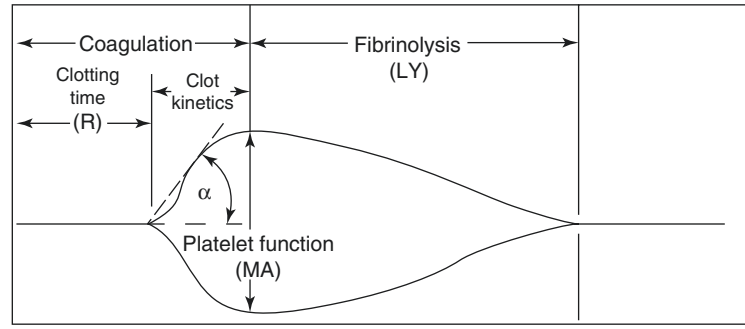

Fig. 5.3 Example of a normal TEG tracing. Values correspond to Table 5.1 (Image adapted from Nystrup et al. [61])

Early animal work suggested that TEG was better at identifying clinically relevant coagulopathies than PT, aPTT, or activated clotting time (ACT). Pigs subjected to hypothermia, hemorrhage, or both had baseline and post-intervention PT, aPTT, ACT, and TEG analysis performed, and it was found that PT, aPTT, and ACT were not sensitive in detecting the coagulation abnormalities that TEG analysis was able to identify [66]. The ability to use TEG as a point of care test has been investigated as well. Cotton et al. prospectively performed rapid-TEG (r-TEG) on a series of trauma patients and found that r-TEG values not only correlated with traditional coagulation lab tests but all results from the r-TEG were available within 15 min compared to 48 min for PT, INR, and aPTT [67]. Linear regression analysis also found that among these patients, r-TEG results were predictive of transfusion requirement. In a series of nearly 2,000 trauma patients, admission r-TEG data was obtained and compared to conventional coagulation tests (CCT) including PT, aPTT, INR, platelet count, and fibrinogen levels and found to correlate well with the CCTs. However, r-TEG data was superior in predicting the need for red blood cell, plasma, and platelet transfusions [68].

The evidence to support TEG and r-TEG is encouraging as a new avenue to rapidly identify patients with *ATC*. However, almost all the literature is retrospective in nature, and importantly while r-TEG values have been found to be predictive of transfusion, there are no standardized algorithms or data to guide how much to transfuse based on cutoff levels. As well, there is no current evidence that has utilized TEG to follow response to resuscitative efforts in trauma patients. A Cochrane review of the literature for TEG and ROTEM was not able to identify any evidence to support the accuracy of TEG and little evidence for ROTEM and suggested these tests be used only on a research basis, although one of the authors of this chapter (MDN) published a rebuttal as an addendum to the Cochrane review highlighting the deficits in the analysis [69]. On the other hand, a recent consensus conference held to determine guidelines for the use of viscoelastic testing in trauma patients supported the use of viscoelastic testing in the early phases of resuscitation given the strong association between abnormal results and increased mortality and need for massive transfusion [70]. As well, it was recommended viscoelastic testing be used to determine administration of antifibrinolytic therapy as TEG and ROTEM are currently the only practical options of detecting hyperfibrinolysis rapidly early in the trauma resuscitation process. Holcomb and colleagues have suggested that TEG replaces the use of admission INR in trauma patients [68].

At this time viscoelastic testing either in the form of TEG or ROTEM appears to be a promising new tool in the armamentarium of the trauma surgeon despite the lack of general consensus on how best to utilize it. The American College of Surgeons Resources for Optimal Care of the Injured Patient 2014 (sixth edition) recommends that thromboelastography should be available at level I and II trauma centers. Further research, specifically prospective trials, are needed to clearly identify threshold levels for transfusion, how much to transfuse, and how best to use viscoelastic testing to follow the resuscitation process and adjust accordingly.

5.7 Treatment

Severely injured trauma patients suffering from hemorrhage and coagulopathy are at an increased risk of mortality, and therefore prompt resuscitation and treatment are of upmost importance. There has been a recent shift in focus in the resuscitation of these severely injured patients from initial IV fluids to blood component therapy [3, 71, 72]. In addition to the focus of early blood component utilization, there has been literature published to suggest the ratio in which these products administered affect outcomes in these patients, namely, higher ratios of platelets, plasma, and cryoprecipitate to PRBC that are associated with improved mortality [73, 74]. The prospective, observational, multicenter, major trauma transfusion (PROMMTT) study followed the transfusion practices and tracked patient outcomes in ten level 1 trauma centers across the United States and found that higher plasma/PRBC and higher platelet/PRBC ratios were associated with decreased mortality in the first 6 h following admission [75]. Even after the results from the PROMMTT trial, the question of the ideal ratio existed as others had reported different optimal transfusion ratios [76, 77]. This was investigated with a large, multicenter, randomized clinical trial, the pragmatic, randomized optimal platelet and plasma ratios (PROPPR) study, that compared 1:1:1–1:1:2 (plasma/platelet/PRBC) and found that the 1:1:1 group had more patients achieve hemostasis and less die from hemorrhage without significant transfusion-related events [4]. While adopted at many centers prior to publication, results of the PROPPR study confirm and provide the basis for adoption of massive transfusion protocols (MTP) to treat trauma patients suffering from massive hemorrhage. However, it is important to recognize that the PROPPR trial failed to show a significant difference in the primary endpoints of 24 h and 30-day mortality comparing 1:1:1–1:1:2 ratios. As such, the "ideal" ratio is unknown, although a clear preponderance of the evidence suggests improved outcomes with higher ratios of plasma and platelets.

A growing interest in the literature is "goal-directed resuscitation" where rapid viscoelastic testing is used to guide the treatment and resuscitation of coagulopathic and massively bleeding patients rather than empiric, fixed ratios of blood products. Observational data have demonstrated that MTP did not lead to correction of coagulopathy based on serial viscoelastic test results nor correction of hypoperfusion as measured by lactate levels [78]. One institution reviewed their pre- and post-MTP mortality rates, where pre-MTP treatment of severely injured trauma patients had been guided by TEG analysis. They found that in patients requiring 6 U PRBC and in blunt injury, TEG and MTP were equivalent. In penetrating injury or patients requiring more than 10 U PRBC, mortality rates were lower with TEG-driven treatment compared to MTP [79]. The use of TEG to guide fibrinogen replacement and administration of prothrombin concentrate complex has been associated with lower mortality rates observed than what would be predicted based on the trauma injury severity score (TRISS) and the revised injury severity classification (RISC) [80]. Proponents of "goal-directed resuscitation" argue that, as opposed to MTP, TEG-driven resuscitation is data driven, corrects underlying coagulopathy, addresses *ATC*, and does not follow a "one-size-fits-all" approach [81, 82]. As appealing as "goal-directed resuscitation" appears, it is important to note that there is limited data to this approach, algorithms are institution dependent, and further studies need to be performed before full recommendations can be given on this approach [83]. The authors' institution has recently implemented a TEG-based MTP to guide resuscitation for massively bleeding patients at the University of Pittsburgh Medical Center (UPMC) (Fig. 5.4).

A final active area of investigation is the use of antifibrinolytic agents in severely injured trauma patients. As discussed earlier, hyperfibrinolysis has been implicated in *ATC* and is able to be identified via viscoelastic testing. Tranexamic acid (TXA), an antifibrinolytic agent, is a synthetic analog to the amino acid lysine that binds and inhibits the actions of plasminogen. It has proven transfusion benefits and a proven safety profile [84]. The effects of tranexamic acid in

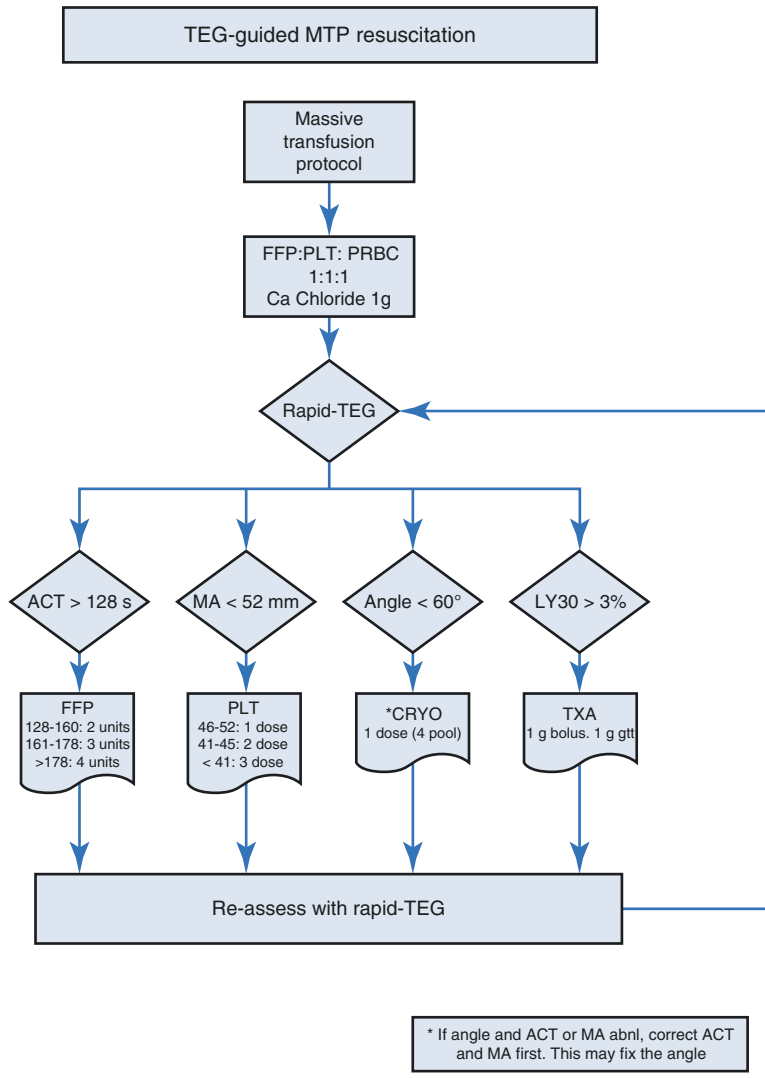

Fig. 5.4 Goal-directed transfusion algorithm that demonstrates the different approach to resuscitation of a trauma patient in hemorrhage shock compared to 1:1:1 in massive transfusion protocols. *ACT* activated clotting time, *MA* maximum amplitude, *FFP* fresh frozen plasma, *Plt* platelets, *Cryo* cryoprecipitate, *TXA* tranexamic acid, *PRBC* packed red blood cells (Image used with permission from University of Pittsburgh Medical Center Department of Surgery)

bleeding trauma patients or patients deemed high risk of bleeding were examined in the CRASH-2 study, a randomized, placebo-controlled trial [85]. It was found that early administration of TXA was associated with decreased risk of all-cause mortality and death from bleeding, while no increased risks of vascular occlusive events were found. TXA was then studied in the military population in the MATTERs study, a retrospective study in the administration of TXA in severe combat injuries [86]. As with the CRASH-2 study, a survival benefit was identified in those who received TXA, and subgroup analysis demonstrated an even larger benefit in patients requiring massive transfusion. The MATTERs trial also noted decreased coagulopathy in patients who received TXA, but there was a higher rate of vascular occlusive events in the TXA patients. TXA appears to have a survival benefit to critically injured trauma patients; however, it is important to note the data was obtained with the administration of TXA left to clinical judgment, not based on identification of hyperfibrinolysis. Therefore, there may be other benefits of TXA such as anti-inflammatory properties, or there may be an even greater benefit in patients identified via TEG/ROTEM to be hyperfibrinolytic. As well, TXA may not be appropriate in all patients as argued by Moore and colleagues after they identified the importance of fibrinolysis shutdown [48].

Conclusion

The "lethal triad" of trauma consists of hypothermia, acidosis, and coagulopathy and portends a poor outcome in patients who suffer from all three. While each component is important, the focus of much recent research has been on the coagulopathy component, specifically *ATC*. While the exact pathophysiology remains unknown, there are likely many pathways that overall contribute to tip the balance from procoagulant to anticoagulant. The ability to determine coagulopathy in a severely injured trauma patient is evolving as well with the increasing utilization of viscoelastic testing. There is great promise in this testing to provide a more individualized and targeted approach to resuscitation of the hemorrhaging trauma patient. Treatment of the bleeding trauma patient has shifted recently, with a movement away from crystalloid resuscitation to early blood components. As well, the use of adjunct medications such as TXA is promising avenues to correct coagulopathy. Advances in treatment of *ATC* will likely parallel advances in the use of viscoelastic testing given its ability to analyze whole blood coagulation. Overall, while there remains much to be elucidated, it is currently an exciting and promising time in traumatic coagulopathy research and the resuscitation of massively bleeding patients.

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Part II

General Treatment Principles

Damage Control Resuscitation

6

Eric J. Voiglio, Bertrand Prunet, Nicolas Prat, and Jean-Stéphane David

6.1 Introduction

Damage control resuscitation (DCR) for trauma, initially described to address the entire lethal triad immediately upon admission to a combat hospital before damage control surgery (DCS) [1], is now accepted as part of an integrated approach DCR-DCS from point of wounding to definitive treatment [2]. Therefore, DCR can be divided in two steps: while bleeding is ongoing and once bleeding has been stopped.

6.1.1 Physiological Bleeding Control

When bleeding occurs, the baroreceptors located in the aortic arch and carotid sinus detect the drop in arterial pressure. This information is transmitted to the brain stem, which immediately increases sympathetic tone [3]. This increased sympathetic tone causes tachycardia (oxygen transportation is ensured by less blood that circulates faster) and vasoconstriction which favours the blood circulation of the heart and brain at the expense of all other organs and tissues (gut, kidney, muscle and skin). Vasoconstriction at the bleeding site decreases bleeding flow and allows platelets and the activated coagulation factors to seal the leak by a vascular clot [4] (Fig. 6.1). Fibrinolysis regulates coagulation [5] and prevents vascular occlusion. In favourable cases, the bleeding has stopped or slowed. In unfavourable cases, because the vascular breach is too large or the bleeding sites are multiple, the trauma patient is in a situation where the coagulation factors have been consumed, fibrinolysis is activated [6], a large volume of blood has been lost, tachycardia and vasoconstriction are not sufficient to compensate for blood loss and therefore the

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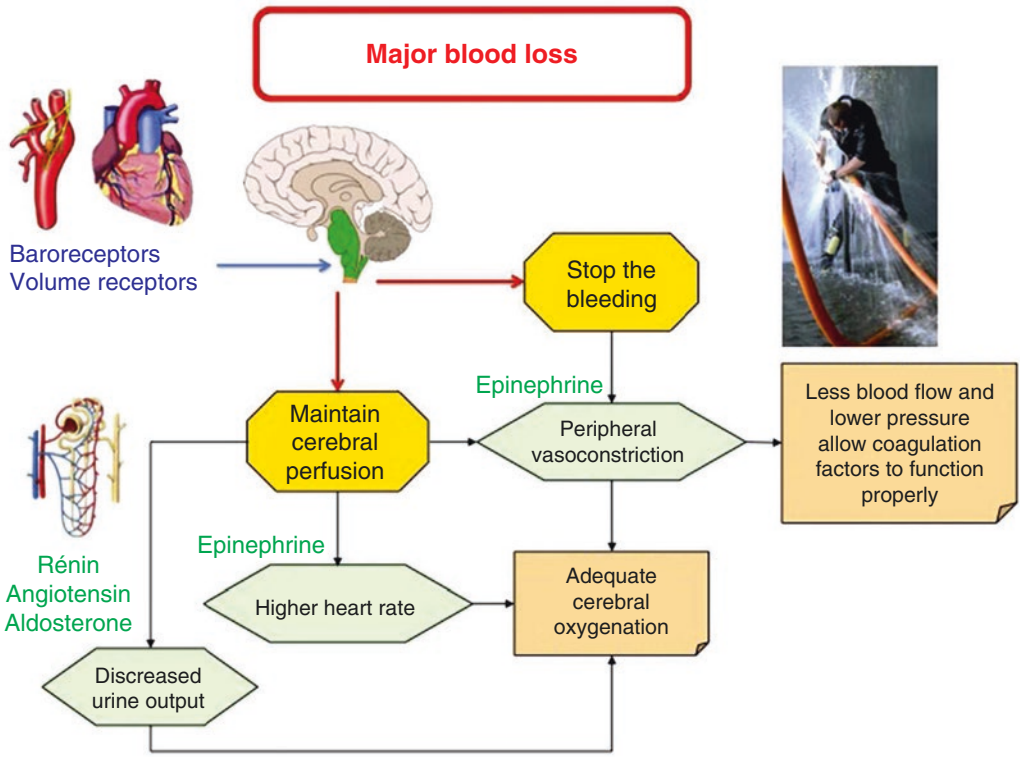

Fig. 6.1 Simplified pathophysiology of bleeding. Bleeding induces hypovolaemia and low blood pressure that trigger volume and baroreceptors which, in turn, transmit the information to the central nervous system [3]. This results in increased sympathetic vascular tone with the double role of maintaining cerebral blood flow and stopping the bleeding. Vasoconstriction in the entire organism (except the heart and brain) deviates the blood supply to the brain, while decreasing blood loss by decreasing the flow and pressure at the level of the vascular injury leaves time for clot formation [4]. Increased heart rate allows partial compensation of the loss of oxygen transportation by increasing the rapidity of red blood cell circulation. At the same time, urine output is decreased by activation of the renin-angiotensin-aldosterone complex with the goal of compensating hypovolemia (E. Voiglio et al. *J Visc Surg*. 2016,153,13–24)

oxygen carrying capacity continues to decrease while the bleeding goes on.

6.1.2 The Lethal Triad: Hypothermia, Acidosis and Coagulopathy

Blood loss causes hypothermia, as the blood plays, among others, the role of a heat transfer liquid. In the cells of a bleeding trauma patient, because of oxygen deficiency, glycolysis stops at the step of pyruvate, which, instead of being consumed by the Krebs cycle, feeds lactate production [7]. Therefore bleeding trauma patient develops lactic acidosis. Coagulation proteins are enzymes that function at 37 °C and pH greater than 7.2. Under hypothermic and acidotic conditions, the coagulation factors have decreased activity [8]. Being the blood hypocoagulable, the bleeding continues later exacerbating hypothermia and acidosis which themselves exacerbate coagulopathy: the haemorrhagic vicious circle [9] is constituted which leads to the death of the trauma patient by exsanguination (Fig. 6.2).

While it is very difficult to take out a trauma patient from this vicious circle, it is very easy to drive him there. It is sufficient to delay the time of haemostasis by a superfluous ‘equipment’ and unnecessary imaging investigations (further

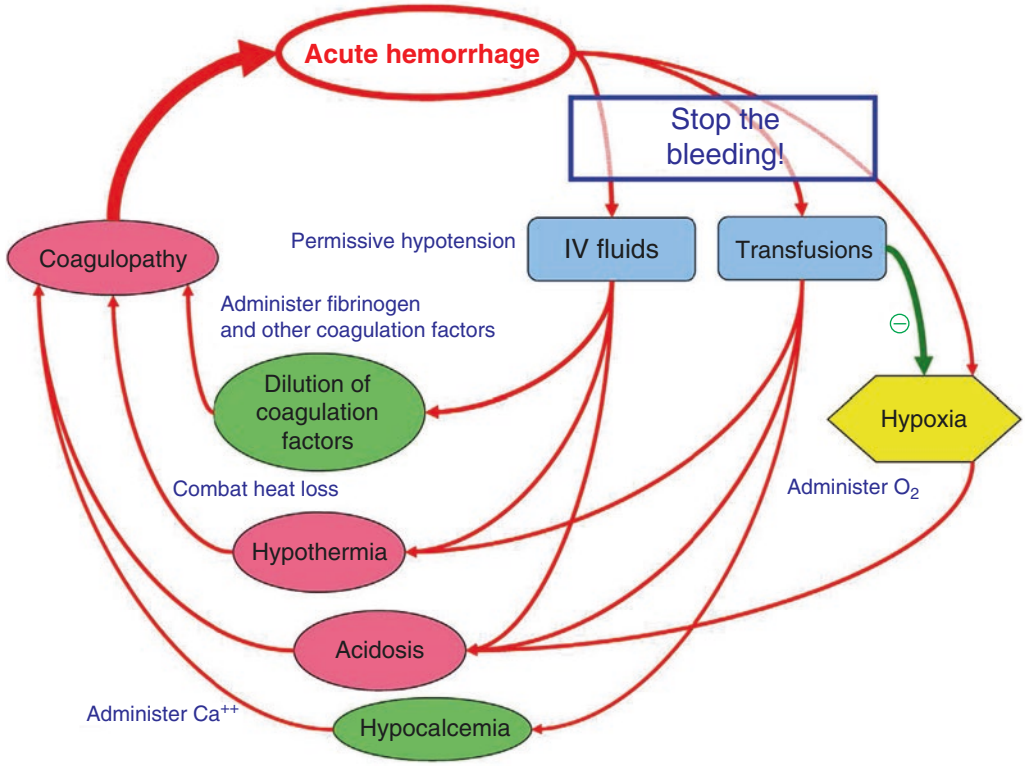

Fig. 6.2 Simplified bloody vicious circle. Acute bleeding triggers cellular hypoxia resulting in metabolic acidosis (lactic acid) [7] and hypothermia (decreased metabolism, loss of heat transport by hypovolaemia). Hypothermia and acidosis lead to coagulopathy because the coagulation factors are enzymes that do not work efficiently below 34 °C and or pH <7.25 [8]. Coagulopathy exacerbates bleeding. Crystalloid volume resuscitation results in dilution of the coagulation factors, cooling and induction of acidosis by dilution and hyperchloraemia. Transfusions add to the deleterious effect of perfusions via the citrate anticoagulants added to PRBC (acidosis and hypocalcaemia) [10]; conversely, transfusions can decrease cell hypoxia by improving oxygen transportation. The only way to interrupt the vicious circle is to stop the bleeding [11]. Administration of oxygen, and limiting IV fluid volume, the strategy of permissive hypotension, combating hypothermia, early transfusion of packed red cells, correction of coagulation disorders by supplying the necessary factors and correction of hypocalcaemia can slow down the vicious circle and buy the time necessary to obtain haemostasis (E. Voiglio et al. *J Visc Surg*. 2016,153,13–24)

haemorrhage), to dilute his/her blood with perfusions (hypothermia, dilution acidosis, anaemia, dilution of coagulation factors, hypocoagulability induced by hydroxyethyl starch [12]) and to rely on a misleading 'haemodynamic stability' artificially achieved by administration of vasopressors (lactic acidosis from visceral and peripheral ischaemia). It has been demonstrated for patients with severe injury of the abdomen and hypotensive at admission that the probability of death increases by 1% every 3 min spent in the shock room [13]. Medico-surgical procrastination is a great provider of haemorrhagic vicious circle.

6.2 Damage Control Resuscitation Before Bleeding Is Stopped

6.2.1 Initial Assessment: Advanced Trauma Life Support (ATLS) Protocol

The treatment of bleeding is to stop the bleeding [11]. *Damage control resuscitation* is a management strategy of which goal is to enable survival of the trauma patient until bleeding is controlled while keeping the risk of iatrogenicity to a minimum. *Damage con-*

trol resuscitation is part of ATLS ABCDE protocol [14] that ensures oxygenation of the cells:

- Airway: airway is most often secured by orotracheal intubation. When orotracheal intubation is impossible and airway has to be secured, cricothyroidotomy is a DC procedure [15]. C-spine is protected by a cervical collar.
- Breathing: trauma patient is given 100% O_2. The SaO_2 is monitored. If a pleural effusion (pneumo- and/or haemothorax) is present, a chest tube is placed. A sucking thoracic wound is treated by a vented chest seal [16].
- Circulation: control of bleeding is initially achieved, depending on situations, by direct pressure eventually enhanced by haemostatic dressings [17], by tourniquet placement [18] or by placement of a pelvic sling [19]. ECG and blood pressure are monitored non-invasively. Two large-bore intravenous lines or one intraosseous line is placed. Crystalloid perfusion is started. In case of haemorrhagic shock, permissive hypotension and transfusion of red blood cell unit (RBC) (O Rh- then type specific) and early plasma administration are recommended [14]. A FAST echography is performed to look for intraperitoneal bleeding and cardiac tamponade [20].
- Disability: GCS score is calculated, pupillary reactivity and symmetry are checked, and focal neurological deficits are searched.
- Exposure: patient's dresses are removed, and a logroll is performed to allow complete examination including the back. Body temperature is monitored.

Whenever a patient presents haemorrhagic shock by an active bleeding that cannot be controlled by external manoeuvres, *damage control* resuscitation is indicated as long as haemostasis has not been achieved most often by surgery, sometimes by interventional radiology.

6.2.2 Targeted Blood Pressure with Permissive Hypotension and Restrictive Fluid Administration

Traditional fluid resuscitation in the polytrauma patient involved rapid infusion of large volumes of clear fluids in an attempt to rapidly restore circulating blood volume and blood pressure. It has become apparent that this approach has several potentially detrimental consequences. The premise of permissive hypotension is to keep the blood pressure low enough to avoid exacerbating haemorrhage by hydrostatic clot disruption while maintaining adequate end-organ perfusion [21]. The concept of damage control resuscitation aims to achieve a lower than normal blood pressure, also called 'permissive hypotension', and thereby avoid the adverse effects of early aggressive resuscitation using high doses of fluids while there is a potential risk of tissue hypoperfusion during short periods [22]. Permissive hypotension and restrictive fluid administration are therefore reciprocal components of this approach; initial fluid administration is delayed or minimized, and less aggressive resuscitative end points are used. A targeted systolic blood pressure (SBP) of 80–90 mmHg is recommended until major bleeding has been stopped in the initial phase following trauma without brain injury [23]. In patients with severe traumatic brain injury (GCS $\leq$8), maintenance of a mean arterial pressure $\geq$80 mmHg is recommended [23]. This approximately equates to aiming for the restoration of a palpable radial pulse. A restrictive fluid administration strategy is recommended to achieve target blood pressure until bleeding can be controlled [23]. Such an approach decreases both the severity and incidence of dilutional coagulopathy and as such complements a strategy of haemostatic resuscitation. Second, this reduces fluctuations in, and elevation of, systolic blood pressure which may disrupt the premature blood clot forming in areas of injury causing further bleeding. Therefore, it would appear that restricting initial IV fluid administration in the severely injured should have advantages, and the

infusion of large volumes of crystalloid is no longer appropriate. In specific situations, permissive hypotension may also be of benefit, particularly in patients with severe haemorrhage from an arterial source. Great caution should be taken in those with concomitant head injury, and further work is required to clearly delineate which patients might benefit the most from this approach [24].

6.2.3 Vasopressor Agents

Vasopressors may be required transiently to sustain life and maintain tissue perfusion in the presence of life-threatening hypotension, even when fluid expansion is in progress and hypovolaemia has not yet been corrected [23]. If used, it is essential to respect the recommended objectives for SBP (80–90 mmHg) in patients without traumatic brain injury [23]. Norepinephrine is the agent of choice to restore and maintain target arterial pressure in haemorrhagic shock. Although it has some β-adrenergic effects, it acts predominantly as a vasoconstrictor. Arterial α-adrenergic stimulation increases arterial resistance and may increase cardiac afterload; norepinephrine exerts both arterial and venous α-adrenergic stimulation. Indeed, in addition to its arterial vasoconstrictor effect, norepinephrine induces venoconstriction at the level of the splanchnic circulation in particular, which increases the pressure in capacitance vessels and actively shifts splanchnic blood volume to the systemic circulation [25]. This venous adrenergic stimulation may recruit some blood from the venous unstressed volume. Moreover, stimulation of β2-adrenergic receptors decreases venous resistance and increases venous return [25]. Animal studies that investigated uncontrolled haemorrhage have suggested that norepinephrine infusion reduces the amount of fluid resuscitation required to achieve a given arterial pressure target, is associated with lower blood loss and significantly improved survival [26, 27].

Furthermore, because vasopressors may increase cardiac afterload if the infusion rate is excessive or left ventricular function is already impaired, an assessment of cardiac function during the initial ultrasound examination is essential. Cardiac dysfunction could be altered in the trauma patient following cardiac contusion, pericardial effusion or secondary to brain injury with intracranial hypertension. The presence of myocardial dysfunction requires treatment with an inotropic agent such as dobutamine or epinephrine. In the absence of an evaluation of cardiac function or cardiac output monitoring, cardiac dysfunction must be suspected in the presence of a poor response to fluid expansion and vasopressor.

6.2.4 Red Blood Cell Transfusion

Blood's main duty is to carry and deliver oxygen to tissues. During bleeding, this capacity is degraded due to two principal phenomena: drop in local blood flow and loss of oxygen carrier, haemoglobin. As seen in the previous section, local blood flow can be restored at least temporarily by fluid infusion and vasopressors use. This fluid infusion, combined with the physiological response to blood loss leading to fluid transfers from cellular and interstitial compartments to the vascular bed, causes the dilution of the haemoglobin and the drop in haemoglobin level (Hb). However, because the relationship between Hb and adverse outcomes in patient with haemorrhagic shock has not been assessed yet [28], it is not possible to determine with certitude the optimal Hb in trauma patients.

Because no artificial oxygen carrier is available so far, the only way to restore the capability of blood to carry oxygen to the tissues is to transfuse RBCs. RBCs are available as packed RBCs (PRBCs) from blood banks. The shelf storage time is limited to about 40 days at 4 °C, but the longer the storage, the more lysed RBCs release intracellular toxic content as potassium or free haemoglobin. This is why a LIFO (last in, first out) procedure for PRBCs release from blood banks needs to be implemented for severely injured patients [29].

In the *European* guideline, the Hb threshold for PRBCs transfusion is set to 7–9 g/dL [23] where in US guideline Hb is set to 7 g/dL [30]. These recommendations are based on studies showing that PRBCs transfusions can be associated with increased mortality, lung injury, increased infection rate and renal failure in injured patients and mainly on the *Transfusion Requirements in Critical Care* (TRICC) study demonstrating no efficacy of liberal approach (Hb threshold of 10–12 g/dL) versus restricted approach (7–9 g/dL) on mortality [31]. For patients with concomitant haemorrhagic shock and traumatic brain injury, recent studies demonstrate no beneficial effect of a higher Hb threshold for RBCs transfusion on mortality or neurological outcomes but a higher risk of thromboembolic events [32, 33], even if a higher Hb improves local cerebral oxygenation [34].

RBCs play also a major role in haemostasis. Circulating RBCs marginate the platelets close to the endothelium, enhancing their adhesion capabilities [35], and support thrombin generation providing interactions with coagulation factors on their cellular surfaces [36].

6.2.5 Fibrinolysis Prevention

Fibrinolysis is a key component of the physiological haemostasis system. It mainly involves the tissue plasminogen activator (tPA) and its inhibitors, the plasminogen activator inhibitors (PAI1 and 2) to regulate the activation of the plasminogen into plasmin, responsible for fibrin binding and degradation. However, a huge stimulation of the coagulation system after severe trauma and activated protein C (aPC) system activation by tissue hypoperfusion [37] can lead to an exacerbation of the fibrinolysis. This hyperfibrinolysis is an essential part of the acute coagulopathy of trauma (ACoT) and is associated with a mortality rate of nearly 90% [38].

The best way to assess hyperfibrinolysis in trauma patients is to use viscoelastic tests. However, the low sensitivity of this method does not allow to detect low increases in fibrinolytic activity, still accountable for ACoT [39].

Hyperfibrinolysis contribution to ACoT can be lower by the use of an antifibrinolytic agent. The CRASH-2 study [40] assessed the systematic injection of tranexamic acid (TXA) in trauma patients with or at risk of severe bleeding. The competitive binding of the plasminogen/plasmin site on the fibrin allows the TXA to inhibit the fibrinolysis. The injection of a loading dose of 1 g of TXA over 10 min followed by the infusion of 1 g over 8 h led to a significant reduction in mortality from bleeding without an increase in thromboembolic events rate. From that same trial, a deeper analysis showed that TXA lowers the risk of death by bleeding by 2.5% if given less than 1 h after trauma and by 1.3% if given between 1 and 3 h after trauma. However, the risk is increased by 1.3% if the TXA is given more than 3 h after trauma [41]. The MATTERs study conducted in military setting later consolidated these conclusions [42]. Based on these results, the *European* guideline recommends the systematic injection of TXA (1 g/10 min, 1 g/8 h) as soon as possible, within the 3 h after the injury [23].

6.2.6 Plasma and Platelet Transfusion in Haemostatic Resuscitation

Coagulation factors and platelets can be shed, consumed, diluted or inactivated in severe trauma patients. Even if they play only a partial role in the ACoT, their replacement is crucial to restore the haemostasis. Standard available fresh frozen plasma (FFP) contains all the major coagulation factors in proportions close to the physiological levels and seems to have anti-inflammatory property while lessening the endothelial hyperpermeability after haemorrhagic shock [43]. Its transfusion should be initiated as soon as possible to avoid iatrogenic or physiological dilutional coagulopathy during a balanced resuscitation with PRBCs. However, the optimal ratio of FFP to PRBCs remains of debate. Some studies showed a potential benefit of an FFP-PRBCs ratio close to 1:1 [44, 45]. However, these results were discussed and potentially flawed by survival bias (i.e. less severe patients survive longer

enough to get more plasma, thawed plasma being available later than PRBCs) [46]. The recent PROPPR randomized clinical trial [47] compared 1:1:1 FFP-PLT-PRBCs ratio to 1:1:2 in severe trauma patients without survival bias. Unfortunately, the results showed a nonstatistically significant reduction in mortality for the 1:1:1 ratio group, letting the question open. The *European* guideline proposes to transfuse 1 FFP every two PRBCs during the initial management of patients with expected massive haemorrhage, continued with goal-directed therapy based on standard laboratory (PT or aPTT inferior to 1.5 times the normal controls) and/or viscoelastic tests [23]. To resolve the delay in availability of the FFP, plasma can be stored as thawed plasma or liquid (fresh nonfrozen) plasma. But in this form, labile coagulation factors like FVIII can be depleted [48]. Lyophilized plasma provided by the *French* military is a nice option. Available in 10 min, stable at room temperature and universal, it offers a great alternative to FFP [49].

Fibrinogen, a key component in the coagulation cascade, is the first and most depleted factor in haemorrhagic trauma patients [50]. However, FFP concentration in fibrinogen is not high enough to restore fibrinogen levels with only FFP transfusion [51], and it may be required to administered fibrinogen through cryoprecipitate or fibrinogen concentrate.

Platelet depletion or dysfunction [52] in trauma patients needs to be addressed by platelet transfusion. Platelets are available as platelet concentrate (PLT) or apheresis platelets (aPLT) containing approximately six times more platelets and plasma. The *European* guideline [44] proposes to transfuse platelets if platelet count is less than 50.10^9/L in trauma patients or less than 100.10^9/L in case of ongoing bleeding or traumatic brain injury.

The best way to replace shed whole blood after or during haemorrhage would be to use whole blood, in replacement for component therapy. Even if used and authorized in remote military setting when blood products are lacking and needs for transfusion surge [53, 54], this technique has not reached the routine clinical practices because of some misconceptions (necessity for whole blood to be ABO specific, impossibility to obtain leucoreduced whole blood while maintaining platelets and loss of platelet function caused by cold storage) [55].

6.2.7 Viscoelastic Techniques and Administration of Concentrated Factors

Standard coagulation tests are of little use for haemorrhagic shock management because they generally require more than an hour, and urgent corrective action may not be delayed that long. To adapt the treatment of haemostasis after the initial phase, viscoelastic techniques (VETs) may be very useful. VETs have been developed for several years and represent a comprehensive assessment of clot formation based on the mechanisms originating coagulopathy, including, in a second stage, inflammatory phenomena [56, 57]. It is possible to obtain a faster and more accurate evaluation of haemostasis through the use of activator or inhibitor which allows to distinguish phenomena occurring during ongoing bleeding such as fibrinogen deficit and hyperfibrinolysis. Identifying deficits makes possible to intervene specifically with clotting factor concentrates, avoiding the use of labile blood products (LBP), and, although this remains to be demonstrated formally, reduce morbidity related to the use of the LBP (multiple organ failure, infection, ARDS, TRALI and TACO) [58–60]. According to the latest *European* guideline, VETs are accepted as alternative to standard coagulation tests to guide the treatment of posttraumatic coagulopathy (grade 1C) [23].

6.2.7.1 Principles of Clot Viscoelastic Property Studies

Clot formation is assessed with ROTEM® (Tem GMBH, Munich, Germany) or with TEG® (Haemoscope Corporation, Niles, Illinois, USA). These tools explore dynamics of clot development, stabilization and dissolution (fibrinolysis) [60–64]. The measured parameters are time (s), amplitude (mm) or angles. The measurements are

made on whole blood collected in a citrated tube. The recalcified blood is then placed in a cuvette heated to 37 °C (or to temperature of the patient), in which a pin is plunged. The speed of rotation thereof will depend on the viscosity of blood. According to the technique, it is either the cuvette which rotates (TEG®) or the pin (ROTEM®). In the latest version of TEG®, measures are made by an electro-optical technique. To accelerate the technical and differentiating phenomena involved in haemostasis disorders, activators are added. They depend on the type of techniques used [60].

ROTEM® analyser uses routinely four channels: INTEM (intrinsic contact activation pathway explored by adding ellagic acid), EXTEM (extrinsic pathway explored by adding tissue factor), FIBTEM (addition of cytochalasin D which blocks the platelets to explore fibrinogen function) and APTEM (addition of aprotinin for inhibiting and therefore exploring fibrinolysis). Two other channels are used in specific circumstances: HEPTEM (INTEM + heparinase to assess heparin effect) and ECATEM (addition of ecarin to detect thrombin inhibitors). In trauma, most useful channels are EXTEM and FIBTEM. Thus, a deficit in prothrombin and in fibrinogen and a low platelet count can be discriminated. As an example, an EXTEM with a short clotting time (thrombin formation correct) and with a diminished maximal clot firmness will suggest low platelet activity if maximal clot firmness is normal with FIBTEM.

TEG® analyser uses generally one single channel after activation by kaolin (equivalent to INTEM). However, it has been shown that platelet and fibrinogen contributions to maximal amplitude could not be differentiated [65]. Therefore TEG® can now be performed with addition of both tissue factor and kaolin (rapid-TEG) to explore the extrinsic pathway, and with addition of abciximab, a potent platelet inhibitor, to explore fibrinogen function [66].

6.2.7.2 Coagulopathy Diagnosis by VETs

At admission, the results of the standard biology are correlated to some ROTEM® parameters, e.g. clotting time (CT) (EXTEM) and PT (prothrombin time) or maximal clot firmness (MCF) (FIBTEM) and level of fibrinogen [67, 68]. Similarly, TEG® R parameter (equivalent to CT) is correlated to PT; correlations were observed between the parameter R (equivalent to CT) and PT [69, 70]. However, this good correlation between standard and viscoelastic techniques at admission may vary during the management [57]. Thus, a CT EXTEM is less correlated to PT after attempt to correct coagulopathy and/or depending to pathophysiological criteria as acidosis and hypothermia [57]. The standard test that estimates the concentration of clotting factors does not take into account the effect of inflammation that develops in the hours following the trauma and activates coagulation. Thus, only VETs that take into account all parameters can provide a fair image of coagulation status [57]. The possibility to predict the need for massive transfusion has been reported with ROTEM® [68, 71, 72] as well as with TEG® (rapid-TEG) [73]. Many algorithms have been proposed to treat bleeding disorders. However these algorithms are specific to either technique and non-interchangeable.

6.2.7.3 VETs and Coagulation Factor Concentrates

Post-traumatic coagulopathy is complex and includes phenomena of coagulation factor loss, dilution, thrombocytopenia, platelet disorders, consumption and fibrinolysis [74]. Fibrinogen deficiency is the most observed among factor deficiencies. The massive release of tissue factor which activates haemostasis and increases thrombin generation is important to consider. Thus, in trauma patients, thrombin generation remains increased as long as factor levels remain >30% [75]. This increase in thrombin generation associated with the frequently observed fibrinogen deficiency suggests the order of administration of haemostatic products. It is thus likely that fibrinogen concentrates have to be administered first, followed in a second phase (ideally according to standard coagulation tests or VETs) by the FFP and the prothrombin complex concentrate (PCC) except, of course, situations of severe haemorrhagic shock when fibrinogen FFP and PCC are administered simultaneously. This method is

only valid when fibrinogen concentrates are available (fibrinogen concentrates and/or cryoprecipitate).

ROTEM® was evaluated in trauma through retrospective or prospective observational studies. The level of proof thus remains relatively low. Schöchl et al. suggested in a first study that ROTEM®-guided administration of coagulation factors improved patient survival when compared to a predictive mortality score (TRISS) [76]. The same group showed that when comparing patients treated with factor concentrates guided by ROTEM® with patients receiving labile blood components (LBC) guided by the standard biology, they could reduce significantly the use of LBC but also the incidence of multiple organ failure without affecting survival [77]. In a recent study, an Italian team confirmed the reduction of the use of LBC reducing costs significantly by more than 23% but still with no change in survival [78]. The issue with all these studies (however this could also be considered an advantage) is that ROTEM® use is combined with that of factor concentrates making it difficult to know what ultimately is most important [79]. A European randomized study should start soon to compare standard biology and ROTEM® using LBP in the same initial ratio (iTACTIC Study, NCT02593877, trial.gouv).

As regards TEG®, a retrospective study involving 1974 patients showed that TEG® could perfectly replace the standard biological tests [80]. In a recent randomized work, it has been shown that the use of TEG® in comparison with the standard biology could improve patient survival at 28 days without association with a modification of LBP consumption in the first 24 h except for cryoprecipitate (paradoxically greater in the group standard biology). A higher consumption of FFP and PCC was observed in the group standard biology in the early hours [81]. According to the authors, this result was related primarily to a decreased mortality from bleeding and a decreased early mortality by earlier diagnosis of coagulopathy and appropriate action. A reduction of ICU stay length with an increased number of ventilator-free days was also observed.

Finally, in severe trauma, situations of hyperfibrinolysis whose prognosis is catastrophic can be observed. TEG® and ROTEM® allow a rapid and accurate diagnosis of hyperfibrinolyses [82] but will lack sensitivity to assess the intensity of fibrinolysis especially if minor or moderate [39]. Usually thresholds of 3% maximum fibrinolysis (maximum lysis) on TEG® and 15% on ROTEM® are applied to diagnose hyperfibrinolysis. If in Europe, tranexamic acid is widely used since the CRASH-2 trial in severe trauma [40], in North America, the practice is rather to administer tranexamic acid to patients with hyperfibrinolysis documented by VETs [82].

6.3 Damage Control Resuscitation once Bleeding Has Been Stopped

Further resuscitation once haemostasis has been achieved is the intensive care unit resuscitative phase where physiological and biochemical stabilization is achieved and a thorough tertiary examination is performed to identify all injuries (Fig. 6.3) [83]. This step is devoted to reverse the sequelae of hypotension-related metabolic failure and support physiological and biochemical restoration. Simultaneous treatment of all physiological abnormalities is essential, and as a result, the first several hours in the ICU are extremely labour intensive and often require the collaborative efforts of multiple critical care physicians, nurses and ancillary staff [84]. Efforts to warm during surgery, shorten the shock and improve coagulation are pursued. An aggressive approach to correction of coagulopathy is paramount, and procoagulant objectives remain the same. Assessment of visceral dysfunction is achieved (in particular the lung, kidney and liver).

One of the keys to physiological restoration is the establishment of adequate oxygen delivery to body tissues. Haemodynamic optimization in this step of major post-shock inflammation often requires a significant fluid volume expansion due to vasodilation. The needs of vasopressors can also be very consequent. Objectives of blood pressure change and aim to restore adequate per-

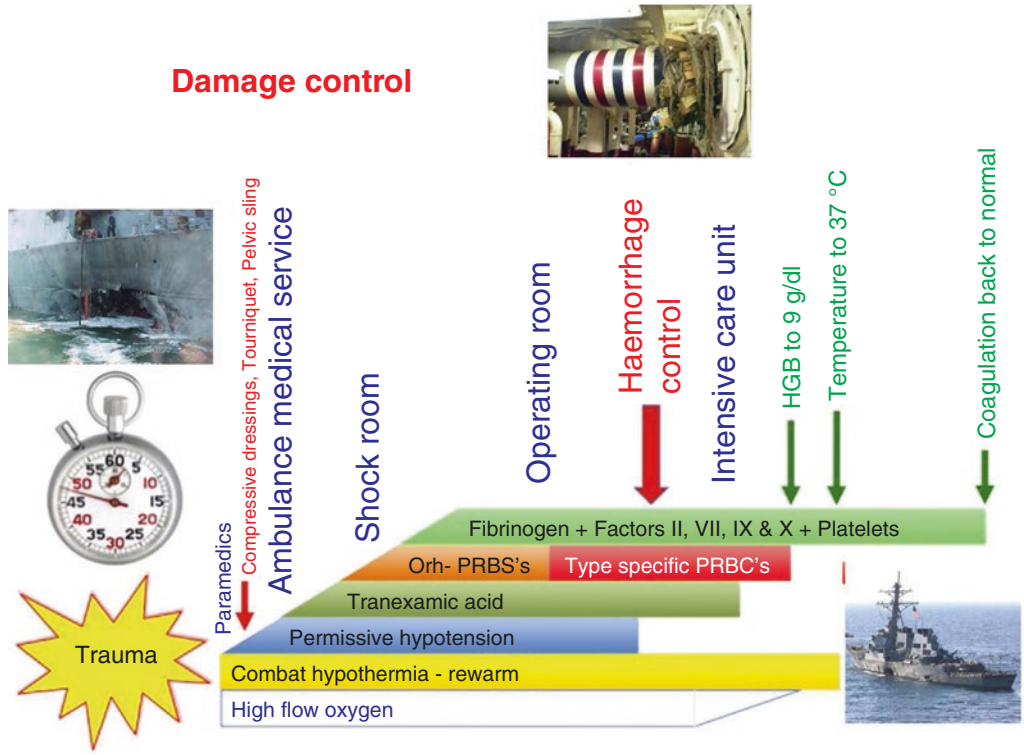

Fig. 6.3 Increasing power of damage control. Damage control should be started in the field by the paramedics who are trained to stop bleeding with local pressure or tourniquets, administer oxygen and combat hypothermia. The race against the clock starts. The emergency team in the field should strive for only minimal vascular filling, the objective being to obtain a systolic blood pressure of 90 mmHg [23]; tranexamic acid should be administered [42]. O-negative and then type-specific PRBC transfusions are started with the objective of obtaining haemoglobin of 9 g/dL (according to European guideline [23]); coagulation disorders are corrected by administration of fibrinogen [23],coagulation factors [77] and platelet concentrates [23]. The patient is transferred rapidly to the operating room (or angiography suite, as necessary). When bleeding has been arrested, blood pressure should return to normal. Damage control resuscitation should be pursued until preset objectives of haemoglobin, temperature and coagulation parameters are attained. The comparison with naval damage control can be made in that not only should the water inflow be stopped, but the vital functions of the vessel must be restored as well (electricity, communications, propulsion, rudder) (E. Voiglio et al. *J Visc Surg*. 2016,153,13–24)

fusion of all organs (MAP = 65 mmHg). Invasive monitoring devices are generally used to guide fluid administration and normalize haemodynamics. Abramson and colleagues did show that serum lactate clearance correlates well with patient survival and that the ability to clear lactate to normal levels within 24 h was paramount to ensuing patient survival [85]. Immediate and aggressive core rewarming not only improves perfusion but also helps reverse coagulopathy. All of the warming manoeuvres initiated in the trauma bay and operating theatre should be duplicated in the intensive care unit. Gentilello showed that failure to correct a patient's hypothermia after a damage control operation is a marker of inadequate resuscitation or irreversible shock [84]. A complete physical examination or 'tertiary survey' of the patient should occur. This should include relevant imaging studies where appropriate, and the patient should also proceed to CT scan to detect occult injuries if stable enough. In cases of blunt trauma, completion of the spinal survey is imperative. Finally, the scheduled revision surgery is the last step of the DC strategy and occurs after 12–48 h (sometimes 72 h) of stabilization.

The consensual approach is to consider the second look when lethal triad is under control. It has two objectives: the final repair organs (pack-

ings removal, intestinal anastomoses and definitive vascular repair) and the permanent closure of the abdomen.

One should keep in mind that if a patient does not normalize haemodynamically or lactic acid or base deficit fail to improve, the patient should be taken back to the operating theatre earlier for re-exploration. Generally, two subgroups of patients are seen in this step that require 'unplanned' re-operation before physiological restoration. The first is the group of patients who have ongoing transfusion requirements or persistent acidosis despite normalized clotting and core temperature. Monitoring of the clinical (blood pressure, tachycardia, suction drains, dressings) and biological parameters (haemoglobin, lactate level) can lead to the decision to further surgery and/or angiography. These patients are usually found to have ongoing surgical bleeding or a missed visceral injury that was not treated adequately during the initial damage control operation and have a very high mortality rate [86]. The second group requiring unplanned return to the operating theatre have developed abdominal compartment syndrome defined as sustained or repeated intravesical pressure above 20 mmHg in the presence of new single or multiple organ system failure [87]. This could be the consequence of abdominal trauma which is accompanied by a visceral oedema and haematomas but also the use of intra-abdominal packing.

Conclusion

The treatment of bleeding remains to stop the bleeding. DCR is together with DCS part of a global DC strategy. DCR is a potent tool to hinder and even reverse the lethal triad. Delaying bleeding control under the pretext that DCR is available and effective is a fallacious conduct that results in increased morbidity and mortality.

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Head Injury

7

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The management of a head-injured patient with multiple other injuries presents one of the most challenging and difficult clinical scenarios in trauma critical care. This is due in part to the fact that the treatment of other injuries, such as orthopedic, spine, and craniofacial fractures, has the potential for worsening the neurologic outcome. This potential worsening is not necessarily directly related to the primary repair or the timing of surgery, but more to the fact that additional surgery with potential blood loss and possible resultant hypotension or hypoxia can adversely affect an injured brain. It has been shown that a single episode of hypotension or hypoxia can adversely affect outcome of all severities of head injury [1–6].

Management of polytrauma patients with head injuries requires strict adherence to ACLS principles, close coordination, and communication between all involved surgical specialties, including simultaneous procedures when appropriate. Decision making is all about assessing relative risk with priority initially given to life-threatening and neurologic injuries, but with a simultaneous evaluation and possible management of orthopedic, spine, and craniofacial injuries. This often requires flexibility on the part of the involved services as surgical decisions should not be based on convenience. This approach provides an opportunity for imaginative and innovative surgical management. Definitive repair procedures are not always possible or appropriate because of the length of the case, inability to appropriately provide neurologic monitoring, and the potential for significant blood loss and massive fluid administration. Other options include placement of external fixation, choosing a surgical approach based on reduced operative time and reduced blood loss, or staged procedures.

The trauma surgery team often serves as the coordinator of care for multiple injured patients, as each involved service can be somewhat myopic in their approach to the patient. However, once the life-threatening hemodynamic injuries are stabilized, the neurosurgery consultant feels the obligation to protect the brain and spinal cord at all costs. While the neurosurgeons are usually very involved with the simultaneous management of spine injuries, they also have to be well informed about the effects of delayed treatment of orthopedic or craniofacial injuries. Again, this very often comes down to an assessment of relative risk. This is because ultimately, while patients with lost or suboptimally functioning

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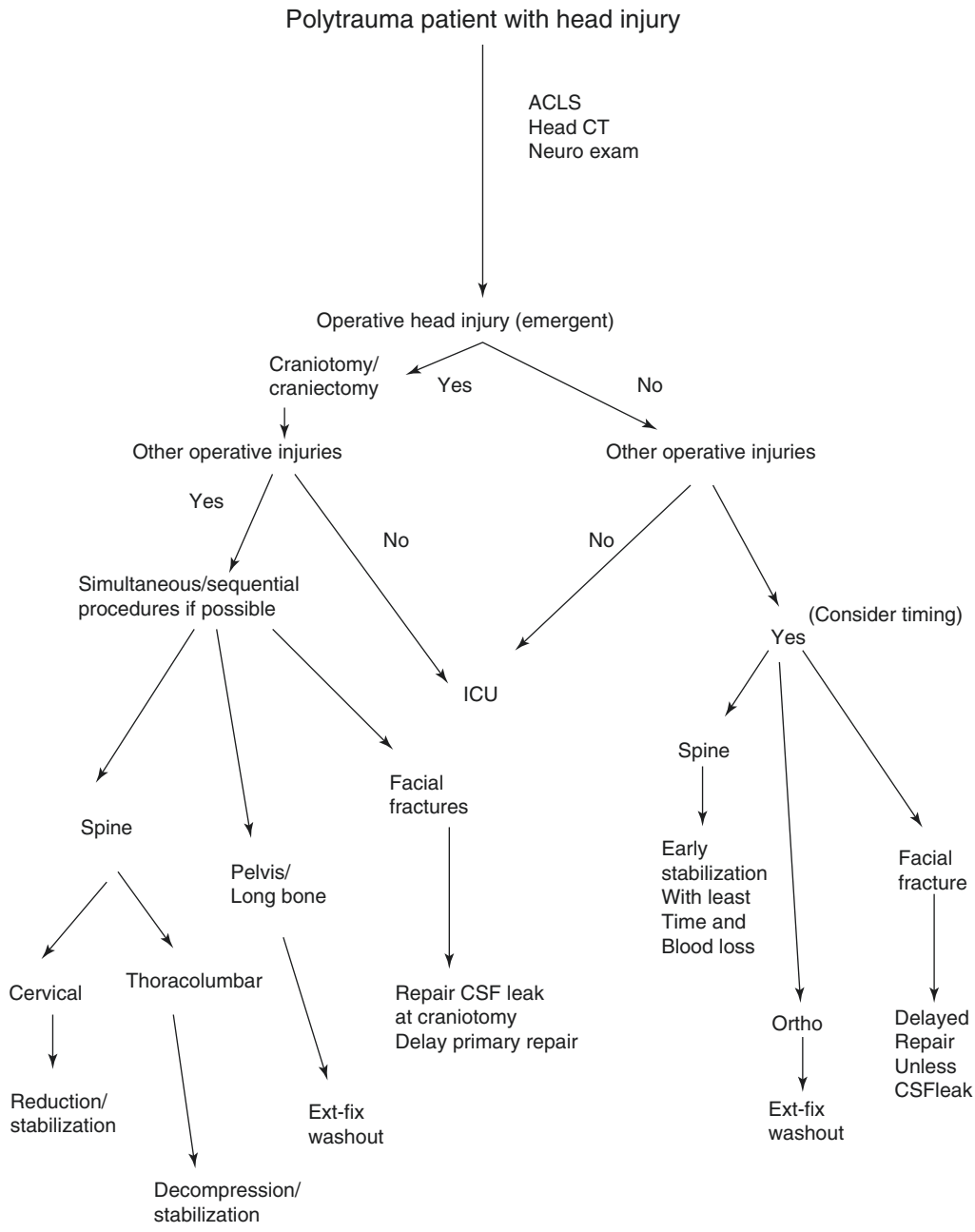

Fig. 7.1 Decision-making flow sheet for polytrauma patient with associated head injury

limbs or craniofacial defects can return to independent functioning, this is less often the case in patients with significant brain injuries. Again an overall concern for avoiding secondary injury must be paramount. The intent of this chapter is to provide a template for rapidly managing head injuries in polytrauma patients including an initial assessment paradigm (Fig. 7.1) to aid in making decisions about the need for emergent neurosurgical intervention, as well as a system for prioritizing and coordinating the treatment of other injuries.

7.1 Initial Assessment

The overall approach to a head-injured patient should be aggressive and timely treatment of the primary injury and avoidance of secondary injury. The basic underlying questions that need to be addressed early in the assessment are as follows: "Does this problem need immediate surgical intervention? Is the injury likely to require intervention in the near future? Or is this an injury unlikely to need surgical intervention?" This decision is based on a clinical and radiographic assessment of the patient. Injuries such as epidural hematomas (EDH), subdural hematomas (SDH), intraparenchymal hemorrhages, contusions, or penetrating injuries with altered neurologic status (especially pupillary abnormalities and lateralizing motor finding) often require emergent surgical intervention. The question of salvageability often arises especially in patients with fixed, dilated pupils and Glasgow Coma Score of 3 [7, 8]. While the decision should be individualized, the survivability of such injuries is debatable. However, it is imperative that the clinical assessment is not altered by pharmacologic muscle paralysis and/or injuries/medications that can alter pupillary function. There is also a danger of being too reassured by a patient's initial clinical assessment such as in patients with the "talk and die syndrome." This is generally seen in a young patient with bifrontal and/or subfrontal injuries (often contusions) who initially may be awake and talking (Fig. 7.2). Because of basal-frontal swelling near the brainstem, any perturbation such as a seizure or hypoxemia can set in motion a rapidly progressive clinical spiral leading to fixed dilated pupils which then requires emergent surgery to avoid poor outcome [9].

Once the decision is made that a patient needs an operation, the question then arises: What operation? The operative plan must take into account the presence of intracranial hematomas, overall brain swelling, intracranial foreign bodies, involvement of major vascular structures, and the involvement of air sinuses that need to be addressed to avoid CSF leak. The general approach is to evacuate hematomas, debride devitalized tissue, remove accessible foreign bodies, and repair CSF leaks. The other decision is whether the patient is likely to have significant problems with raised intracranial pressure postoperatively. Concerns for postoperative management often lead the decision to perform a craniectomy with storage of the bone subcutaneously in the abdomen or in the freezer until which time the brain swelling has subsided and the bone can be replaced. This procedure also requires dural augmentation with pericranium, or a dural substitute to allow swelling of the brain out of the bone defect, thus decompressing the brain. The procedure can be performed unilaterally, bifrontally, or bihemispheric depending on the pattern of injury [10–14] (Fig. 7.2b,c). The removal of bone must be adequate to ensure that as the brain swells out

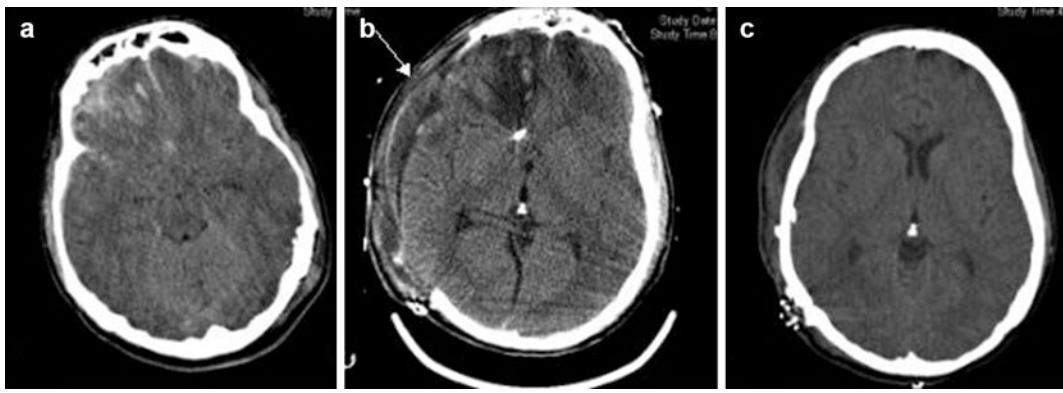

Fig. 7.2 Patient at risk for "talk and die syndrome." (**a**) CT scan showing frontal contusions. (**b**) CT scan showing right hemicraniectomy (*arrow*) after progressive neurologic decline. (**c**) CT showing replacement of bone at 8 weeks after patient made excellent neurologic recovery

of the defect, it is not lacerated or contused against a bone edge. Craniectomy, if performed adequately, is the most effective and durable way to reduce intracranial pressure. While ultimate outcome is still heavily tied to the severity of the initial injury and neurologic status, there is evidence that hemicraniectomy can reduce intracranial pressure and improve brain oxygenation as measured by invasive monitoring in head-injured patients [15, 16]. The other advantage of hemicraniectomy and the subsequent improvement in ICP and brain oxygenation is it often makes postoperative management less physiologically stressful to the patients as they are less likely to require osmotic diuretics, blood pressure support, and heavy sedation. In patients with less severe injuries but labile intracranial pressure, the placement of a ventriculostomy for CSF diversion can be an extremely effective and less physiologically stressful way of treating raised intracranial pressure.

After aggressive surgical treatment of the primary intracranial injury, the introduction of aggressive neurocritical care has been shown to positively impact brain-injured patients [17–19]. Using monitoring to measure brain oxygenation and intracranial pressure, the use of continuous EEG to detect seizures and ischemia, and protocol-driven approaches to glycemic control and hyperthermia are now routinely implemented [20–22]. Early tracheostomy and enteral access are now initiated to facilitate patient care [23, 24].

Finally, consulting services such as orthopedics often enquire about when a patient is stable enough to allow further surgery. This decision needs to be individualized for each patient based on injury severity, clinical exam, neurophysiologic stability based on monitoring, the perceived urgency of the proposed surgery, and the potential physiologic stress of the surgery including length, risk of large blood loss, or large fluid volume administration. Physiologic monitoring is also recommended during the case (ICP, brain oxygenation) which may affect positioning and the potential of aborting the procedure if a problem arises. Essentially, management in the OR must be an extension of the management in the ICU with the same vigilance with regard to monitoring and proactive intervention [25].

As can be seen from the above discussion, there is an apparent paradox in the treatment approach to a patient with a head injury and other associated injuries. We aggressively and definitively treat the primary CNS injury to help avoid secondary injury while treating other associated injuries in a damage control fashion. This is due in part to the fragile nature of CNS structures with little reparative capability, little function reserve, and a narrow timing window with regard to salvageability after injury. We will discuss this approach in the subsequent sections. Finally, by definition, surgical decision making involves relative risk assessment, and therefore it is imperative that patients and families understand the complexity of these interactions and have realistic expectations for outcome and recovery.

7.2 Head Injury Associated with Major Chest/Abdominal Injury

Stabilizing a patient's cardiopulmonary status must always be the highest priority in dealing with a trauma patient. However, there are clinical scenarios in which a patient is taken emergently to the operating room to deal with life-threatening thoracoabdominal trauma with a suspected brain injury and no confirmatory neuroimaging. There is traditional support for placing burr holes on the side of a "blown" pupil in the ED or in the OR during thoracoabdominal surgery. While if performed correctly there is the potential to relieve pressure from an evolving EDH or SDH, this may not be the definitive procedure and a craniotomy may also be required [26, 27]. This in theory could follow after cardiopulmonary stabilization. In the case of bilateral "blown" pupils, practitioners must keep in mind that hypoxia/hypotension can cause pupillary abnormalities that do not indicate an intracranial space-occupying mass [28]. As neurosurgeons, we are sometimes asked to place intracranial monitors in patients undergoing "crash" thoracotomies or exploratory laparotomies with suspected

intracranial pathology. However, elevated ICP is difficult to interpret in a patient lying flat or head down and receiving massive fluid resuscitation. Additionally, therapeutic interventions such as mannitol would not be indicated in a hypotensive patient. The key is early cranial imaging. A rapid helical CT scan preoperatively if possible, or utilization of portable CT scanners interoperatively, would obviate this problem.

7.3 Head Injury Associated with Cranial Facial Injury

Head injuries from blunt force trauma or penetrating injuries have the potential for involvement of the air sinuses such as the frontal sinus as well as cranial facial structures [29, 30]. While not absolute indications for surgical intervention, the presence of pneumocephalus and/or a CSF leak must be closely monitored. Increasing pneumocephalus or rhinorrhea/otorrhea that does not resolve spontaneously may require surgical intervention. The goal of surgery in a patient with significant intracranial pathology (hematoma, contusion) associated with pneumocephalus/CSF leak (for instance, from an anterior skull base fracture/frontal sinus fracture) is to adequately deal with the intracranial pathology and stop the CSF leak: essentially isolating the brain from the nose [31, 32]. There is often input from multiple services including neurosurgery and ENT, OMFS, or plastics, but it is generally in the patient's better interest to delay the definitive repair of facial fractures because of the overall extension of the operative time [33]. If CSF diversion is required to protect the repair, a ventriculostomy is preferred to a lumbar drain in a patient with suspected intracranial swelling. Compression of the optic nerve could also be addressed by this approach if indicated [34].

Skull base fractures can also cause damage to carotid arteries. There should be a high index of suspicion with these types of fractures. Treatment options include medical management, or more invasive surgical or endovascular therapy. Therapy depends on the risk-benefit ratio of each option considering the natural history of each injury type including mild intimal irregularities, intimal flaps, pseudoaneurysms, fistulas, and occlusions. The need for treatment is determined in part by the collateral circulation to the brain and the degree to which the lesion is thrombogenic [35]. It is beyond the scope of this chapter to discuss the management of traumatic carotid injury, but the risk of anticoagulation would have to be considered in a patient with significant intracranial injury [36].

7.4 Head Injury with Spine Injury

There is a high incidence of associated spine fracture with head injury especially with falls and motor vehicle accidents [37]. It is often difficult to clinically clear the cervical spine in a patient with a significant head injury even without obvious radiographic abnormalities. But the more pressing difficulty occurs with the combination of an operative head injury and an operative spine injury. In the case of spine fracture/dislocation with a spinal cord injury such as with bilateral jumped facets and burst fractures, early decompression and stabilization is optimal [38, 39]. While these injuries can be decompressed in traction, this delays mobilization which has been shown to negatively impact outcome secondary to pulmonary issues [40–43]. With simultaneous operative injuries, the cranial portion could be completed, and the cervical spine reduced and stabilized through an anterior approach if possible. In critically ill patients with unstable thoracolumbar fractures, a posterior approach can be utilized in a time-efficient and less physiologically stressful manner for decompression/stabilization even if an anterior approach will subsequently be required. This staged stabilization/decompression allows mobilization and thus helps avoid life-threatening pulmonary complications [42, 43]. These stabilization procedures can sometimes be performed in a percutaneous/minimally invasive method, significantly reducing blood loss [44].

Blunt vertebral artery injury is associated with complex cervical spine fractures involving sub-

luxation, extension into the foramen transversarium, or upper C1 to C3 fractures [45]. The posterior circulation stroke rate has been reported as high as 12–24% after traumatic vertebral artery injury [46, 47]. Treatment of vertebral artery injuries such as dissections, pseudoaneurysms, fistulas, and occlusions must be individualized with regard to the use of anticoagulation and/or endovascular treatment based on the patients' clinical status, risk of ischemic/thrombotic potential for the individual lesions, and the suitability of the patient for anticoagulation based on their intracranial pathology and other injuries [46–48].

7.5 Head Injury with Orthopedic Injury

The combination of head injuries and significant orthopedic injuries are relatively common with motor vehicle accidents and falls. Major orthopedic trauma can be both life threatening (pelvic fractures with vascular injuries) and limb threatening especially with open and potentially contaminated extremity injuries. Ideally, head injuries and orthopedic injuries can be assessed and if possible treated simultaneously. If the patient is going to the OR for an operative head injury, every effort should be made to treat orthopedic injuries in a damage control fashion. Any open fracture or other critical musculoskeletal injury that can realistically be splinted or casted and treated definitively in a delayed fashion should be. Any injury that can be treated with external fixation or washed out and closed when the patient initially goes to the OR for a head injury should be treated simultaneously. This damage control method has been shown to be both safe and effective [49–55]. The problem often arises when these injuries are not dealt with simultaneously and early. Once the patient is in the ICU, there is generally resistance to allow the patient to go to the OR if they have labile vital signs especially brain oxygenation and intracranial pressure difficulties. In this case, these decisions need to be made in a coordinated fashion between the involved services. This often results in a compromise in which the patient is allowed to go to the OR for a less time-consuming and less physiologically stressful procedure. While this is an area of discussion, there is growing evidence to support this approach [51–53, 56].

7.6 Summary

The management of a polytrauma patient with a significant head injury represents one of the most complicated clinical scenarios encountered by the trauma team. However, it provides an opportunity to implement innovative and imaginative management strategies that require communication, coordination, and flexibility among the involved subspecialties. Primary intracranial pathologies need to be treated aggressively and secondary injuries avoided at all costs. Head injuries, spine injuries, orthopedic injuries, and craniofacial injuries with CSF leaks need to be assessed and treated simultaneously if possible. The definitive treatment of the noncranial injuries can often be delayed with the initial therapies designed to stabilize spine, pelvis, and long bone injuries as well as repairing CSF leaks associated with cranial facial injuries. This facilitates management in the ICU and helps prevent pulmonary complications, which are a major source of morbidity and mortality. Additionally, neurocritical care, provided in the ICU, needs to be extended to the OR to avoid secondary brain injury. Subsequently, with stabilization and improvement of the head injury, the noncranial injuries can be readdressed.

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8 Chest Trauma: Classification and Influence on the General Management

Frank Hildebrand, Hagen Andruszkow, and Hans-Christoph Pape

8.1 Introduction

Most thoracic injuries are caused by blunt trauma, whereas penetrating injuries only account for around 10% of these cases. Isolated blunt chest trauma usually occurs after a minor traumatic impact and is characterized by mild injuries (e.g., thoracic bruises, rib fractures) that can be treated conservatively in the vast majority of cases. In young adults with isolated chest trauma, a mortality rate of 0–5% is described, whereas an increase to 10–15% is found in the elderly population [1–3]. In geriatric patients aged ≥85 years, initial blood pressure <90 mmHg and specific injuries (hemothorax, pneumothorax, serial rib fracture, pulmonary contusion) have been identified as risk factors for posttraumatic complications and adverse outcome [4].

In 80–90% of cases, severe chest trauma is associated with concomitant injuries [5]. Therefore, thoracic injuries represent one of the most common diagnoses in severely injured patients [6]. In addition to the significant clinical implications in the acute posttraumatic phase, severe chest trauma also has a major impact on the later clinical course. In this context, a significant increase in ventilation time and prolonged intensive care treatment has been observed [6]. Furthermore, chest trauma has been shown to result in higher incidences of acute respiratory distress syndrome (ARDS), systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome (MODS), and infectious complications (pneumonia) compared to severely injured patients without severe chest trauma [3, 7–9]. Accordingly, thoracic injuries have been associated with increased posttraumatic mortality. Particularly in patients with combined chest trauma and severe traumatic brain injury (TBI), mortality rates of more than 70% have been described [1–3, 7, 10, 11]. Furthermore, 50–75% of deceased polytraumatized patients had a thoracic injury. This chest trauma-related increase in posttraumatic complications might partly be explained by an enhanced inflammatory response that has especially been described after pulmonary contusions [1–3, 7, 10–12].

8.2 Injuries After Chest Trauma

Thoracic injuries can affect the chest wall and the intrathoracic organs, including the pleura, diaphragm, lungs, mediastinum, and the great blood vessels.

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8.2.1 Chest Wall Injuries

Rib fractures account for the majority of thoracic injuries and are found in around 50% of patients with blunt chest trauma. Rib fractures can result in pleural or pulmonary lacerations with development of pulmonary hematoma, hemothorax, and pneumothorax. Typically, ribs IV–X are affected. In the case of fractures of the first two ribs, a particularly severe traumatic impact has to be assumed. As these two ribs are anatomically close to vital structures, lesions of the brachial plexus and different vessels (e.g., subclavian artery and vein) may occur. Furthermore, lung contusions are likely. Fractures of the lower ribs are mainly caused by a direct local impact and can be associated with injuries to abdominal organs, such as the liver, spleen, and kidneys.

In the younger trauma population, rib fractures are usually caused by a severe traumatic impact and are frequently associated with pulmonary contusions. In elderly patients, however, minor trauma also regularly results in rib fractures due to decreased bone elasticity and osteoporosis. In these patients, the fracture-related pain often results in a reduction of breathing depth with subsequent fluid retention and associated pulmonary complications, such as pneumonia and atelectasis. In older patients, each additional rib fracture increases the probability of death by 19% and the incidence of pneumonia by 27% [13, 14].

In cases of *serial rib fractures,* at least three ribs of one or both thorax cavities are concerned. In addition to the risks of single rib fractures, increasing numbers of fractured ribs are associated with a reduced stability of the chest wall, which might lead to a flail chest.

A *flail chest* is found in about 15% of patients with blunt chest trauma [15]. It is defined as at least two fractures per rib in at least two ribs. This results in a segment of the chest wall that is separated from the rest of the thoracic cage. A separated segment of the chest wall is not able to contribute to lung expansion and is associated with paradoxical breathing (inward motion during inspiration and outward motion during expiration). Posterior flail segments are stabilized by overlying muscles as well as the scapula and therefore may not cause severe complications. In contrast, anterior and lateral flail segments are mobile and can seriously impair respiratory function. Additionally, a flail chest is frequently associated with lung contusions [12].

Sternal fractures are seen in about 5% of patients with chest trauma [16]. Most fractures involve the upper- or mid-part of the sternum. Sternal fractures are frequently accompanied by pulmonary and myocardial injuries, as well as fractures of the thoracic spine.

Sternoclavicular dislocations may occur either in the anterior or posterior direction. Posterior dislocations are more severe, as they can result in injuries of mediastinal blood vessels, as well as in tracheal or esophageal damage [17]. In general, the more common anterior dislocations can be treated conservatively, whereas posterior dislocations usually require closed or surgical reduction.

Due to the close anatomical relationship, chest trauma is frequently accompanied by *clavicular fractures.* In particular, a high coincidence of clavicular fractures and specific thoracic injuries (hemothorax, pneumothorax, lung contusions) has been described. Furthermore, fractures of the clavicula have been associated with injuries to the upper extremities and the cervical spine, as well as a higher overall injury severity in multiple-trauma patients. Therefore, clinical and radiologic diagnostics are recommended to specifically focus on these body regions in the case of a clavicular fracture [18, 19].

With a prevalence of approximately 4%, *fractures of the scapula* are quite uncommon in severely injured patients [20]. Similar to clavicular fractures, scapular lesions are frequently associated with other injuries, such as pneumothorax, hemothorax, and pulmonary and spinal injuries [21]. Most fractures affect the body and neck of the scapula and can be treated conservatively. In contrast, displaced intra-articular glenoid fractures and displaced juxta-articular fractures require a surgical intervention [21, 22].

8.2.2 Injuries of the Intrathoracic Organs

8.2.2.1 Pleural Injuries

A *pneumothorax* occurs in 15–40% of patients with chest trauma [23–25] and is defined as a collection of air in the chest or pleural space that might result in a partial or entire collapse of the lung. Leading symptoms are a unilateral breath sound and tissue emphysema, which might either be caused by pleural laceration due to fractured ribs (closed pneumothorax) after blunt trauma or by penetrating injuries (open pneumothorax) [16]. Lesions of the tracheobronchial tree might also result in a pneumothorax. The most frequent complication of a pneumothorax is the development of a tension pneumothorax.

In cases of a pneumothorax in which air enters the thoracic cavity and is captured during the process of exhalation, a *tension pneumothorax* can develop. This results in a collapse of the ipsilateral lung with subsequent compression of the mediastinum and the contralateral lung. A suspected tension pneumothorax (distension of jugular veins, unilateral breath sound, tissue emphysema) has to be decompressed immediately by needle thoracostomy or a chest tube. A *hemothorax* is found in about 20–40% of patients with blunt thoracic trauma. It results from vascular lesions after blunt or penetrating trauma. In this context, a hemothorax can be caused by diverse bleeding sources, such as intercostal arteries, internal mammary arteries, lung parenchyma, and the heart, as well as hilar and great vessels. The therapy of a hemothorax is the placement of a chest tube. An undrained hemothorax can lead to a *tension hemothorax* with ipsilateral lung compression and subsequent displacement of the mediastinum [16]. A chronic hemothorax can be complicated by pleural empyema or a fibrothorax that might result in a restrictive pulmonary disease [12]. A lesion of the thoracic duct can result in the development of a *chylothorax*. A rupture of the upper part of the thoracic duct is associated with a left-sided chylothorax. Crossing the midline lesions of the lower parts of the thoracic duct results in a right-sided chylothorax.

8.2.2.2 Diaphragm Injuries

A diaphragmatic rupture can be caused by blunt or penetrating injuries. It occurs in 0.2–5% of patients with blunt chest trauma [26, 27]. Ruptures on the left side are three to four times more common than lesions on the right side. In 5–10% of cases with diaphragmatic injuries, a bilateral rupture is found [12]. A high proportion of diaphragmatic ruptures are primarily not diagnosed [12], and the mortality of missed diaphragm ruptures has been reported to be as high as 30% [28].

8.2.2.3 Lung Injuries

Parenchymal lung injuries appear as pulmonary contusions and lacerations. *Pulmonary contusions* are one of the most frequent injuries in thoracic trauma patients [12]. Pulmonary contusions are either caused by direct trauma to the lung parenchyma or by indirect mechanisms, such as deceleration and shear forces. Lesions usually occur in peripheral lung sections that are adjacent to bony structures [28]. Pulmonary contusions regularly appear 3–6 h after trauma and generally resolve within 5–7 days [28, 29]. Histopathologically, these injuries are characterized by an extravasation of blood and edema into the interstitial and alveolar space. Especially in younger patients, pulmonary contusions can also be found without accompanying osseous lesions [12, 16, 30, 31]. However, serial rib fractures and a flail chest are commonly associated with pulmonary contusions [32]. *Pulmonary lacerations* are characterized by a disruption of the parenchymal architecture. With the exception of stab wounds, lung lacerations are always accompanied by pulmonary contusions [33]. Pulmonary contusions and lacerations can be complicated by the development of *ARDS*, which is the consequence of a systemic inflammatory response after chest or general trauma. Pathophysiologically, ARDS is caused by the damage of the alveolar-capillary barrier by activated neutrophils resulting in an extravasation of fluid into the alveolar space [34, 35]. This systemic inflammatory response can also affect primarily uninjured pulmonary sections [31, 36]. Radiographically, ARDS manifests as a diffuse

bilateral pulmonary infiltration [29]. Diverse trauma-related predictive models have been suggested for early prediction of ARDS [37–39]. In this context, age, Acute Physiology and Chronic Health Evaluation (APACHE) II Score, injury severity, blunt trauma mechanism, pulmonary contusion, massive transfusion, and flail chest have been associated with an increased risk of ARDS [37].

8.2.2.4 Injuries to the Mediastinum

A *pneumomediastinum (mediastinal emphysema)* may occur after pharyngeal, tracheobronchial, or esophageal lesions after either penetrating or blunt trauma. Besides chest radiography, diagnostics should include esophageal and tracheobronchial endoscopy. A *mediastinal hematoma* results from vascular injuries and might result in an enlargement of the mediastinum. Mediastinal widening is diagnosed in cases with a diameter of >8 cm and a mediastinum-to-chest ratio of >0.25. *Tracheobronchial injuries* occur in 0.2–8% of patients with blunt chest trauma and are frequently accompanied by pulmonary or vascular injuries [40, 41]. Tracheal lesions usually appear as transverse tears between the cartilaginous tracheal rings or longitudinal tears in the posterior tracheal membrane. Tracheal injuries require surgical repair to ensure airway continuity. *Esophageal injuries* after blunt chest trauma are rare. The majority of esophageal lesions are located in the cervical and upper thoracic sections. Depending on their location, esophageal injuries can result in right- or left-sided pleural effusion. In order to avoid subsequent complications, such as edema and infection (mediastinitis), surgical repair is required.

Pericardial injuries (e.g., organ and vascular ruptures) can result in air entrapment (pneumopericardium) or hemorrhagic influx (hemopericardium) into the pericardial cavity. A hemopericardium may be complicated by the development of pericardial tamponade with increased pericardial pressure and subsequent hemodynamic instability. In particular, lesions of the intrapericardial aorta and left cardiac ventricle endanger the patients, whereas bleeding of the atrium or right ventricle may not cause noticeable symptoms. The majority of *pericardial tamponades* occur after penetrating trauma, but also appear in about 1% of blunt chest trauma patients [12, 16]. Immediate pericardiocentesis is indicated for restoration of normal cardiovascular function [42, 43].

Cardiac injuries are observed in 15–25% of patients with chest trauma [44]. The incidence increases to 75% in cases of sternal fractures, parasternal rib fractures, and ruptures of the diaphragm [45]. *Myocardial contusions* occur due to the rupture of the intramyocardial vessels and can result in structural injuries and functional changes. Structural injuries include the perforation of cardiac muscles or the ventricular septum, as well as disruption of the papillary muscles and valves [12]. Furthermore, arrhythmias may arise as a functional complication following myocardial contusion. *Cardiac aneurysms* are focal evaginations of the septal or ventricular walls. True aneurysms frequently appear in the left ventricular anterior wall or at the apex after severe blunt trauma. Cardiac pseudoaneurysms typically occur after penetrating trauma and are usually located in the left ventricular posterolateral wall. *Cardiac ruptures* frequently affect the right ventricle due to its thin wall and the anatomical position in the thoracic cavity. Severe torsion forces can also cause a cardiac avulsion with separation of the heart from the great vessels.

Traumatic aortic injuries include a spectrum of lesions due to severe deceleration trauma and most frequently affect the aortic isthmus, followed by the aortic root and the diaphragmatic aorta. Traumatic aortic ruptures occur in 2% of patients with blunt chest trauma. In approximately 90% of cases, complete aortic ruptures are fatal at the scene of the accident. They can only be survived in cases of pseudoaneurysm formation with containment of active bleeding by the aortic adventitia, a thrombus, or mediastinal structures. Traumatic aortic dissections are characterized by intimomedial tears. Type B (descending aorta) dissections can be treated conservatively, whereas Type A (ascending aorta) dissections require surgical repair due to the risk of pericardial bleeding, coronary artery

laceration, and aortic valve rupture. Traumatic aortic aneurysms are focal dilatations that include the entire aortic wall, and these aneurysms are at risk of rupture. Open surgery should be performed in aneurysms of the ascending aorta that are symptomatic, rapidly expanding, or greater than 5.0–5.5 cm in diameter. In aneurysms of the descending aorta exceeding 6.0 cm, repair by endovascular stent grafting is recommended.

Injuries of the great intrathoracic vessels are only found in 1% of blunt chest trauma patients [12, 16], but in more than 90% of patients with penetrating trauma. After blunt trauma, injuries are mainly caused by deceleration mechanisms and predominantly affect aortic branch vessels and the superior or inferior vena cava, as well as pulmonary veins.

8.2.3 The Deadly Dozen

Among the aforementioned thoracic injuries, 12 typical life-threatening injuries have been identified that have been called the "deadly dozen." These 12 injuries are divided into the lethal six and the hidden six. The *lethal six* (acute airway obstruction, tension pneumothorax, pericardial tamponade, open pneumothorax, massive hemothorax, flail chest) are immediately life-threatening injuries and have to be identified, and treatment has to be initiated during the primary survey. The *hidden six* (aortic rupture, myocardial contusion, tracheobronchial injuries, diaphragmatic rupture, esophageal rupture, pulmonary contusion) represent injuries that are easily missed. They have to be identified, and treatment has to be initiated during the secondary phase.

8.3 Diagnostics

The correct diagnosis of thoracic injuries, as well as prompt assessment of the severity of chest trauma, is crucial for the further clinical course and outcome. Different diagnostic tools are available in clinical routine.

8.3.1 Plain Chest X-Ray

Plain chest X-ray is a frequently used diagnostic tool to detect thoracic injuries [46]. In the clinical routine, chest X-rays are performed in the anterior-posterior (ap) and lateral directions of the upright sitting patient in full inspiration. However, in multiple-trauma patients, plain chest radiography has to be obtained in the supine position and, therefore, only in the ap direction. As a lateral view is not possible under these circumstances, the superimposition of different structures and organs in the ap plane exacerbates the interpretation, thereby limiting the diagnostic value [47]. In this context, the incidence of missed pneumothoraces (occult pneumothoraces) on plain chest X-ray is thought to be 2–15% [48–51]. Additionally, pulmonary contusions are frequently either missed or underestimated by chest radiography, especially in the early posttraumatic phase at admission [52–57].

8.3.2 Computed Tomography

The deficits of plain chest X-ray for the diagnosis of thoracic injuries can be compensated by a computed tomography (CT) scan, which represents the most important examination method in chest trauma patients [47]. A thoracic CT scan is superior to conventional chest radiography for the assessment of pneumo- and hemothorax, pulmonary parenchymal lesions, and bony injuries [23, 56, 58]. However, there is an ongoing discussion about whether this additional information changes the treatment strategy [59, 60]. Marts et al. reported a change in clinical management in only 6.5% of patients with chest trauma [56]. In another study, CT has been credited with changing the treatment in up to 20% of chest trauma patients [61]. Further studies found therapeutic changes in 30–70% of the cases due to a CT-related amendment of diagnostic information [62–64]. In general, a chest CT scan (pro re nata as part of a whole body CT) is recommended in all trauma patients with multiple injuries, suspected chest trauma, abnormal findings in the

initial chest X-ray, and in cases of respiratory insufficiency [64–68].

8.3.3 Thoracic Ultrasonography

Ultrasound examinations represent a noninvasive diagnostic tool that offers several advantages, including general availability, low procedural time/effort, and the possibility of repeated examinations [69, 70]. With a sensitivity of 81% for the detection of intrathoracic fluid [71], ultrasonography represents a reliable tool for diagnosis of hemothorax [72]. Furthermore, with a sensitivity of 92–100%, thoracic ultrasound examinations also seem to be suitable for the detection of pneumothoraces [69, 73–75]. As a potential disadvantage, subcutaneous emphysema aggravates an accurate diagnosis by ultrasound [71]. Additionally, evaluation of bony lesions, as well as tube and line malpositioning, remains the domain of conventional radiography. Therefore, ultrasound examination cannot be used as an exclusive diagnostic procedure. However, it has a supplementary role in the diagnostics of chest trauma.

8.3.4 Bronchoscopy

Bronchoscopy represents both a diagnostic and therapeutic tool. It is of particular value for the diagnosis of tracheobronchial lesions, supraglottic injuries, aspiration, bleeding, and lung contusions [76]. In addition, early diagnosis and assessment of severity of lung contusions are supposed to be more reliable by bronchoscopy than by conventional chest radiography [77]. Besides its diagnostic use, bronchoscopy is also a therapeutic tool (e.g., clearance of the respiratory tract, prevention of atelectasis formation, bleeding control). Despite these advantages, indications for bronchoscopy in the acute phase after trauma are rare (e.g., severe bleeding and tracheobronchial ruptures). As bronchoscopy also has the potential to enhance respiratory insufficiency [78], it cannot be considered as a routinely used tool in primary diagnostics of multiple-trauma patients.

8.4 Classification

The evaluation of injury severity and the prediction of outcome are important functions of scoring systems. Early assessment of severe chest trauma is decisive for the clinical course of multiple-trauma patients, such as timing and kind of surgical interventions (early total care vs. damage control), and early and adequate therapy of the chest trauma itself is crucial to avoid posttraumatic complications [1, 3, 12].

Several scoring systems for the classification of blunt chest trauma have been developed. Most of these scores are based on pathological-anatomical changes. One of the most commonly used scoring systems is the thoracic Abbreviated Injury Scale (AIS_{chest}). Other anatomic scoring systems include the Wagner Score [79] and the Pulmonary Contusion Score (PCS) by Tyburski [80]. Some scoring systems, such as the Thoracic Trauma Severity Score, also include physiological parameters [81].

8.4.1 Abbreviated Injury Scale

The Abbreviated Injury Scale (AIS), first described in 1969 by John D. States and revised in 1998, is an anatomical scoring system allocating a severity score to every injury of different body regions (head, face, neck, thorax, abdomen, spine, upper extremity, lower extremity, external and other trauma). The score value ranges from 0 to 6, and high severity scores are associated with a lower probability of survival. The AIS represents the basis for calculation of the Injury Severity Score (ISS). In general, the AIS correlates with mortality [82, 83] and the AIS_{chest} has been demonstrated to be an independent predictor for prolonged hospitalization [84, 85], duration of mechanical ventilation [86], and a risk factor for the development of posttraumatic MODS [87].

8.4.2 Pulmonary Contusion Score

The Pulmonary Contusion Score (PCS) was developed in 1999 by Tyburski and colleagues [80].

This score is based on a plain chest X-ray at the time of admission and 24 hours after trauma. After separation of the lung into an upper, middle, and lower third, pulmonary contusions in each third are assessed by a value of 1–3. The sum of these values represents the PCS. A PCS value of 1–2 is classified as mild, a value of 3–9 as moderate, and a value of 10–18 as severe pulmonary contusion (Table 8.1). An increased severity of lung contusion over the first 24 h has been associated with a higher mortality rate and a prolonged duration of mechanical ventilation [80]. The PCS has been criticized due to the weaknesses of the assessment of pulmonary contusions by plain chest X-ray.

Table 8.1 Pulmonary contusion score by Tyburski [80]

Calculation of the pulmonary contusion score (PCS)		
Dividing the lung fields into upper, middle and lower third		
Assigning a score of 1–3 to each region on the basis of the amount of radiologic parenchymal changes		
Mild pulmonary contusion	Moderate pulmonary contusion	Severe pulmonary contusion
PCS 1–2	PCS 3–9	PCS 10–18

8.4.3 CT-Dependent Wagner Score

Wagner and Jamieson developed a chest trauma score based on a CT scan [79]. In this score, the severity of chest trauma is classified according to the volume of pulmonary lesions (Fig. 8.1). Pulmonary lesions of ≥28% of total air space are

Fig. 8.1 CT-dependent score according to Wagner and Jamieson [79]

Table 8.2 Thoracic trauma severity score by Pape et al. [81]

Grade	PO_2/FiO_2	Rib fractures	Pulmonary contusion	Pleural lesion	Age (years)	Points
0	>400	0	none	None	<30	0
I	300–400	1–3 unilateral	1 lobe unilateral	Pneumothorax	30–40	1
II	200–300	4–6 unilateral	1 lobe bilateral or 2 lobes unilateral	Hemothorax/Hemopneumothorax unilateral	41–54	2
III	150–200	>3 bilateral	<2 lobes bilateral	Hemothorax/Hemopneumothorax bilateral	55–70	3
IV	<150	Flail chest	≥2 lobes bilateral	Tension pneumothorax	>70	5

classified as grade 1, 19–27% as grade 2, and <19% as grade 3. The authors showed an association between the size and type of parenchymal injuries and the need for mechanical ventilation [79].

8.4.4 Thoracic Trauma Severity Score

The Thoracic Trauma Severity Score (TTS) is a CT-independent scoring system that is based on five anatomical and physiological parameters at the time of admission: extent of pulmonary contusion, number of rib fractures, pleural lesions, age, and PaO_2/FiO_2 (Horowitz) ratio [81]. Each parameter is assigned a value of 0–5 (Table 8.2), and the TTS score ranges from 0 to 25.

The sensitivity and specificity of the different scoring systems for predicting posttraumatic complications and outcome have not been fully elucidated. In general, CT-dependent scores are thought to be more reliable for the assessment of trauma severity and susceptibility to posttraumatic complications (e.g., ARDS). In contrast, CT-independent scoring systems might be helpful for early evaluation of the risk profile at admission after chest trauma. However, it has been suggested that these scores should be based on anatomical and physiological parameters due to the limited diagnostic value of conventional chest X-ray. Recently, the predictive value of the PCS, the Wagner score, and the TTS score was analyzed [88], and it was shown that the TTS score best predicts ARDS, MODS, and in-hospital mortality in multiple-trauma patients [88].

8.5 Treatment

Severe chest trauma represents the second most common diagnosis in multiple-trauma patients [5, 6]. Treatment of thoracic injuries has to be performed according to established guidelines (e.g., ABCDE-algorithm according to the Advanced Trauma Life Support®, ATLS®). In this context, chest trauma is sufficiently treated by airway control (e.g., intubation) and treatment of breathing and ventilation problems (e.g., mechanical ventilation, placement of chest tube) in >80% of the cases. Operative interventions are required in 2–16% of patients with chest trauma. Indications for emergency surgery are penetrating or open chest trauma, hemothorax with an initial chest tube output of 1,000–15,000 ml, pericardial tamponade, or cardiac injury, as well as tracheobronchial or major vessel injuries. Indications for urgent or elective interventions include a blood loss over the chest tube of >200 ml/h over 5 h or >400 ml/h over 2 h, diaphragmatic or esophageal lesions, persistent bronchopleural leakage, pneumato- or hematoceles, valvular damage, non-drainable clots, and flail chest. In an emergency situation, anterolateral thoracotomy represents the standard approach, whereas the posterolateral approach is favored in the hemodynamically stable patient.

There is also a high coincidence of thoracic injuries and extremity trauma (e.g., femoral fractures). The timing and type of fracture stabilization have the potential to substantially influence pulmonary function as well as the development of posttraumatic complications in multiple-trauma patients with chest trauma. Therefore, this chapter focuses on both the general aspects

of the treatment of chest trauma and the significance of adequate treatment strategies for fracture stabilization in cases of concomitant chest trauma.

8.5.1 Airway Management

Usually, oral intubation is already performed at the scene of the accident or in the emergency department. If not, it has to be considered in the initial posttraumatic period as early intubation has been shown to reduce morbidity and mortality in patients with severe chest trauma. Indications for intubation include TBI (Glasgow Coma Scale <9), chest trauma with respiratory insufficiency (e.g., SaO_2 <90%, breathing rate <10/min or >30/min), hemorrhagic shock, and cardiopulmonary reanimation. In cases of a suspected ventilation time of more than 7–10 days, tracheotomy has been recommended. Tracheotomy seems to be favorable due to improvements in respiratory mechanics and the reduction of infectious complications. However, the effect of tracheotomy on total ventilation time and the duration of intensive care treatment have been controversially discussed.

8.5.2 Mechanical Ventilation

In the anesthetized, mechanically ventilated patient, a reduction in pulmonary functional residual capacity due to supine positioning has been observed. Furthermore, a posttraumatic reduction in the compliance results in hypoventilated areas, particularly in dorsobasal lung sections, with an increased risk for atelectasis formation. As these lung sections show the best pulmonary perfusion, a ventilation/perfusion mismatch with increased intrapulmonary shunting is also observed. Additionally, the increased intrathoracic pressure due to mechanical ventilation exerts hemodynamic effects, such as decreased cardiac output. Besides trauma-related pulmonary injuries, mechanical ventilation with high inspiratory pressure also has the potential to result in additional damage to the lung parenchyma. Therefore, lung protective ventilation with low tidal volumes (5–6 ml/kg), high positive end-expiratory pressure (PEEP), and limited inspiratory peak pressure (<35 cm H_20) should be used in case of severe chest trauma.

8.5.3 Positioning Therapy

Positioning therapy has been applied in patients with chest trauma to prevent and treat pulmonary functional disorders. There are a variety of positioning procedures including the semi-recumbent position, the lateral position, the prone position, and continuous axial rotational therapy. Mechanically ventilated patients should always be positioned in a *semi-recumbent position* (45°) in order to avoid pulmonary aspiration and ventilator-associated pneumonia. In patients with unilateral lung injuries, *lateral positioning* of almost 90° ("good lung down") has been recommended.

A complete *prone position* is the 180° contrast to the supine position, meaning that the patient lies with the chest down and back up. An incomplete prone position is a transfer between 130° and <180°. Prone positioning is applied in patients that suffer from severe ARDS with life-threatening hypoxemia (PaO_2/FiO_2 <100). Contraindications for prone positioning include an open abdomen, unstable spine injuries, TBI with increased intracerebral pressure, severe arrhythmia, acute shock, and substantial facial trauma [89–91]. Prone positioning is recommended for at least 12 h. It results in an increase in pulmonary gas exchange due to an improved ventilation/perfusion ratio [92–94] and recruitment of alveolar space with reduced atelectasis formation [95–99]. These effects occur either immediately (≤30 min) or up to 12 h after retransfer into a supine position [100–102]. Incomplete prone positioning is less effective [103]. Compared to continuous axial rotational therapy, prone positioning seems to be associated with stronger and faster therapeutic effects. However, no differences are evident between these positioning procedures after 72 h [104].

Overall, it seems noteworthy that, if applied correctly, prone positioning represents a relatively safe procedure that does not result in a significant increase in intra-abdominal pressure in patients without abdominal injuries [105, 106]. However, it can be complicated by facial edema (20–30%), pressure ulcers (20%), noncompliance of the patient (20%), and arrhythmia (5%), as well as by tube and catheter dislocation (1–2%) [107]. Despite improved arterial oxygenation, prone positioning does not result in a significant reduction of morbidity, ventilation time, or length of stay in the intensive care unit in patients with ARDS [107, 108]. In contrast, a decrease in ventilator-associated pneumonia after prone positioning has been described [107, 108].

Continuous axial rotational therapy is characterized by continuous rotation of the patient about the longitudinal axis in a self-rotating bed. Depending on the different bed systems, a rotation of up to 62° to each side can be achieved. Potential indications are the prevention of pulmonary complications (e.g., atelectasis, pneumonia) in patients with chest trauma [109–111]. Furthermore, it is used for the treatment of ARDS if, for example, prone positioning is contraindicated. Kinetic therapy is recommended for at least 3–5 days [109–111]. The best effects of axial rotational therapy [110–115] are described for a rotation of >40° to each side. Contraindications are unstable spine injuries, acute shock, and adiposity (≥160 kg). Complications associated with kinetic therapy include pressure ulcers, hemodynamic instability, kinetosis, and catheter dislocation.

The potential benefits of continuous axial rotational therapy have been discussed controversially [116]. Besides the positive effects observed in some studies, other trials failed to show a significant effect on morbidity, ventilation time, and length of stay in the intensive care unit [117–122]. Furthermore, recent studies did not find a beneficial effect of mechanical ventilation with prophylactic kinetic therapy compared to early extubation and aggressive weaning in patients with severe thoracic trauma [123, 124]. Due to the small and inhomogeneous study populations, a generalization of these results to the treatment of severe blunt chest trauma patients is questionable. Nevertheless, the role of kinetic therapy and its prognostic relevance should be clarified in further studies. Furthermore, reliable parameters for the indication of kinetic therapy should be validated.

8.5.4 Fracture Treatment in Multiple-Trauma Patients with Thoracic Trauma

In patients with severe abdominal injuries and hemodynamic instability, initial management should avoid complex operative procedures. Such interventions in the acute phase have to be performed rapidly and should not add a further significant burden to the patient. The primary focus under these conditions is hemorrhage control and the performance of other life-saving procedures. Complex reconstructive work is delayed until the patient is able to withstand the additional surgical trauma. This “damage control” approach has been adapted to patients with extremity trauma. In specific subgroups of these patients, extensive surgical procedures for fracture stabilization have been associated with an increased incidence of complications, such as SIRS and MODS.

In general, there are two treatment strategies for fracture care in multiple-trauma patients. The aforementioned concept of “damage control orthopedics” (DCO) is characterized by temporary external fracture fixation in the primary phase with secondary conversion to definitive osteosynthesis after stabilization of the patient’s physiological and immunological status in the intensive care unit. In contrast, primary definitive fracture fixation is performed within the concept of “early total care” (ETC) [125–130]. Although early fracture fixation has been described to be essential to avoid pulmonary complications after multiple traumas [131, 132], the optimal treatment strategy (ETC versus DCO) for fracture care remains the focus of intensive research [125–133]. This is particularly true for multiple-trauma patients with severe chest trauma [133]. Several investigations

have demonstrated a decreased risk for infection and pulmonary dysfunction after ETC treatment in these patients [125, 129, 134, 135], whereas other studies have reported an increased rate of pulmonary failure after ETC. Accordingly, an inconsistent use of ETC and DCO has been shown in an analysis of the trauma registry of the German Trauma Society in patients with chest trauma [128, 133, 136, 137].

In the majority of studies, it is accepted that stable patients benefit from the ETC concept, whereas unstable patients and patients "in extremis" might benefit from DCO. In a prospective randomized study, Pape et al. [138] introduced an additional group of patients that were in an unclear condition ("borderline" patients; Table 8.3). These patients were distinguished from stable, unstable, and "in extremis" patients (Fig. 8.2). In this study, borderline patients were shown to have a significantly higher incidence of acute lung injury (ALI) after ETC treatment compared to fracture stabilization according to the DCO concept [128]. For the identification of these patients, the severity of thoracic trauma and physiological pulmonary parameters are of central importance. This emphasizes the significance of chest trauma for the development of posttraumatic complications after fracture stabilization in multiple-trauma patients. The timing of secondary definitive osteosynthesis within the DCO concept seems to not be advantageous before 5 days after the trauma [128, 139]. Giannoudis recommended secondary fracture fixation based on defined parameters (Table 8.4) [139].

In conclusion, early definitive fracture stabilization seems to increase the risk of adverse outcome in multiple-trauma patients with severe chest trauma. However, further prospective randomized studies are needed to increase the sensitivity and specificity of parameters to identify those patients who might benefit from the DCO concept of fracture care. Recently, further treatment strategies for fracture stabilization in multiple-trauma patients, including "early appropriate care" (EAC) and "safe definitive surgery" (SDS), have been developed [140–144]. The

Table 8.3 Borderline patients according to Pape et al. [138]

ISS >40
Hypothermia <35 °C
Multiple trauma with ISS >20 and AISchest >2
Multiple trauma with abdominal/pelvic injury (AIS > 2) and shock (RRsystol <90 mmHg)
Bilateral lung contusion in chest radiography or CT
Pulmonary artery pressure (PAP) >24 mmHg
Increase of PAP >6 mmHg during femoral nailing

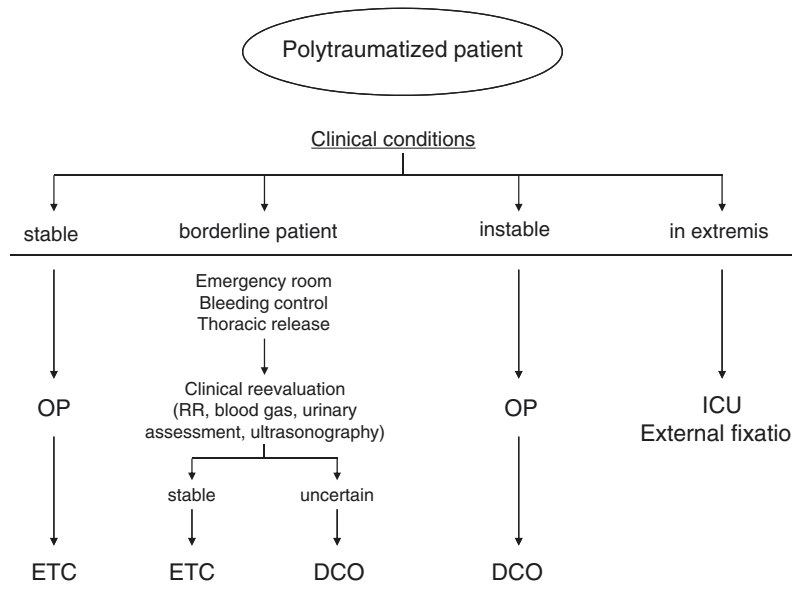

Fig. 8.2 Treatment algorithm according to Pape et al. [138]

Table 8.4 Signs of stabilization according to Giannoudis [139]

Hemodynamic stability
Stable arterial oxygenation
Lactate <2 mmol/l
Absence of coagulopathy
Normothermia
Urine production >1 ml/kg/h
No need for catecholamines

EAC protocol aims to determine the optimal timing of definitive fracture fixation by the presence and severity of metabolic acidosis [37, 141, 143, 145]. In contrast to the dichotomous EAC protocol, the SDS represents a dynamic concept for surgical decision-making in multiple-trauma patients [140, 146]. Within the SDS concept, continuous reevaluation of the clinical situation and physiological parameters allows decision-making according to the current clinical course and physiological status. In consequence, benefits of both the treatment strategies of ETC and DCO can be guaranteed depending on the individual situation [140, 146].

8.5.5 Surgical Chest Wall Stabilization

Possible indications for an operative stabilization of the chest include flail chest, reduction of pain and disability, severe chest wall deformity/defect (impression >5 cm), thoracotomy for other indications, open fractures, and symptomatic non-unions [147]. Among these indications, the flail chest is considered to represent the best indication for early stabilization.

Although many patients with a flail chest can be treated conservatively by sufficient pain relief, internal pneumatic stabilization by mechanical ventilation, and tracheobronchial toilet [147, 148], operative fixation has been associated with a reduced duration of mechanical ventilation, a decrease in ventilation-associated complications, and a reduced mortality rate, as well as improved long-term results (e.g., pain, respiratory dysfunction). The best results of rib osteosynthesis have been observed when the stabilization was performed early (24–72 hours after injury), and no additional lung contusions or severe TBI was present [147, 148].

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Abdominal Compartment Syndrome

9

Rao R. Ivatury

9.1 Introduction

Abdominal compartment syndrome (ACS) has tremendous relevance in the care of critically ill or injured patients, because of the effects of elevated pressure within the confined space of the abdomen on multiple organ systems. Our knowledge of this lethal syndrome has evolved over the past 15 years. Of note, we now recognize that ACS should be viewed as the end result of a progressive, unchecked rise in IAP, called intra-abdominal hypertension (IAH), as illustrated in Fig. 9.1. We also have learnt that the adverse effects of elevated IAP occur at lower levels than previously thought [1].

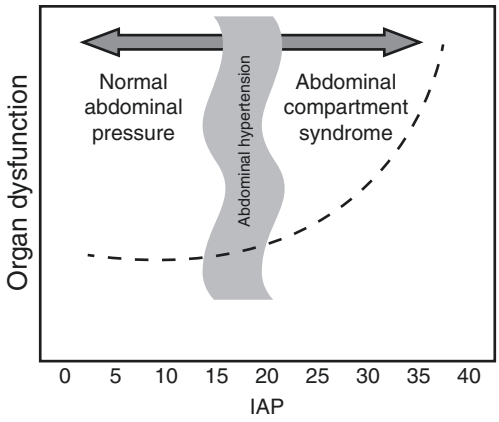

Fig. 9.1 Progression of IAP (intra-abdominal pressure) from normal to IAH (intra-abdominal hypertension) to abdominal compartment syndrome with organ failures

9.2 Historical Background

The pathophysiology of IAH has been known since late 1800s [1]. Marey (1863), Braune (1865), Schatz (1872), Wendt of Germany (1873), Oderbrecht of Germany (1875), Quinke of Germany (1878), Mosso and Pellacani of Italy (1882), and Heinricius of Germany (1890) all described the ill effects of IAH. In the next century, Emerson (1911), Bradley (1947), Gross (1948), Olerud (1953), Kashtan (1981), Harman (1982), Richards (1983), and Kron, Harman, and Nolan (1984) were the greatest contributors in the field. Fietsam et al. from William Beaumont Hospital, Royal Oak, Michigan, were the architects of the term intra-abdominal compartment syndrome [2]. They described it: "In four patients with ruptured abdominal aortic aneurysms….manifested by increased ventilatory pressure, increased central venous pressure, and decreased urinary output associated with massive abdominal distension not due to bleeding. This set of findings constitutes an intra-abdominal compartment syndrome … Opening the abdominal incision was associated with dramatic improvements." As with many advances in

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medicine, this precious knowledge was forgotten, rediscovered, and forgotten again. Many proponents of the syndrome faced skepticism and ridicule till the clinical syndrome was rediscovered in patients with life-threatening abdominal injuries undergoing "damage-control" surgery. In this group of patients, IAH became a prime cause of avoidable morbidity and mortality [3–11].

Our knowledge of IAH and ACS was spurred by the shared experiences of trauma centers dealing with the nightly horrors of "America's uncivil war" (CW Schwab). The phenomena were codified by trauma surgeons who popularized the clinical practice of such advances as IAP monitoring by bladder pressure and non-closure of fascia after laparotomy ("open abdomen"). The consequent results were nothing short of dramatic [3, 4, 8]. Further advances were also realized through the efforts of a remarkable group of clinical researchers interested in the subject. After a preliminary meeting in 2001 in Sydney, Sugrue and associates formally established the World Society of Abdominal Compartment Syndrome (WSACS) in 2004 in Noosa in Australia. This society, though a small group of motivated intensivists, redefined the current concepts of IAH and ACS through multinational clinical trials, literature review and analysis, multiple publications including a monograph on the subject [1] and guideline and consensus development [12–15].

With this historic background, this chapter will summarize the WSAC consensus definitions, a brief review of pathophysiology, and WSACS recommendations and algorithms. It will then evaluate their impact on the current status of IAH in critically injured or ill patients and offer some projections for the future. The issues of "open abdomen" approach for prevention of IAH and ACS, while highly relevant, are beyond the scope of this chapter, however.

9.3 Current Definitions of IAH and ACS [12, 13]

IAP Intra-abdominal pressure (IAP) is the pressure concealed within the abdominal cavity. IAP should be expressed in mmHg and measured at end expiration in the complete supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line. The reference standard for intermittent IAP measurement is via the bladder with a maximal instillation volume of 25 mL of sterile saline. Normal IAP is approximately 5–7 mmHg in critically ill adults.

IAH Intra-abdominal hypertension is defined by a sustained or repeated pathologic elevation of IAP ≥12 mmHg. It is graded as follows: grade I, IAP 12–15 mmHg; grade II, IAP 16–20 mmHg; grade III, IAP 21–25 mmHg; and grade IV, IAP >25 mmHg.

ACS Abdominal compartment syndrome (ACS) is defined as a sustained IAP >20 mmHg (with or without an APP [MAP-IAP] <60 mmHg) that is associated with new organ dysfunction/failure. It should be noted that this definition moves ACS to a much earlier point in the trajectory of clinical course than the traditional fully manifested syndrome with multiorgan failure.

Primary ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention.

Secondary ACS refers to conditions that do not originate from the abdominopelvic region. Recurrent ACS refers to the condition in which ACS redevelops following previous surgical or medical treatment of primary or secondary ACS.

Risk Factors for IAH/ACS [12–15] ACS can develop in both nonsurgical and surgical patients. These factors include diminished abdominal wall compliance (abdominal surgery, major trauma, major burns, prone positioning), increased intraluminal contents (e.g., acute pancreatitis, hemoperitoneum/pneumoperitoneum or intraperitoneal fluid collections, intra-abdominal infection/abscess, intra-abdominal or retroperitoneal tumors, liver dysfunction/cirrhosis with ascites, capillary leak/fluid resuscitation from massive fluid resuscitation or positive fluid balance), and damage-control laparotomy. Other miscellaneous causes include bacteremia, coagulopathy, massive incisional hernia repair, obesity or increased body mass index, PEEP, peritonitis, and sepsis.

9.4 Pathophysiology of ACS

IAH affects multiple organ systems in a graded fashion.

9.4.1 Cardiovascular Effects

Elevation in IAP leads to a reduction in cardiac output (CO) [9–11], most consistently seen at an IAP >20 mmHg. This is due to a combination of decreased inferior vena caval flow and an increased thoracic pressure (which decreases both inferior and superior vena caval flow). Other contributory factors include cardiac compression, decreased ventricular end-diastolic volumes, and marked increase in systemic afterload. This may lead to spuriously elevated central venous pressure, pulmonary artery pressure, and pulmonary artery occlusion ("wedge") pressure. Combined with a reduced CO, this may erroneously suggest a state of hypervolemia or biventricular failure [1, 9–11]. Improvement in CO after a saline fluid bolus may be therapeutic and clarify the diagnostic conundrum.

9.4.2 Pulmonary Dysfunction

With an acute elevation in IAP, respiratory failure characterized by high ventilatory pressures, hypoxia, and hypercarbia eventually develops [9–11]. Diaphragmatic elevation leads to a reduction in static and dynamic pulmonary compliance. The increase in IAP also reduces total lung capacity, functional residual capacity, and residual volume [9–11]. These lead to ventilation-perfusion abnormalities and hypoventilation producing hypoxia and hypercarbia, respectively. A porcine model by Simon et al. demonstrated that prior hemorrhage and resuscitation (ischemia-reperfusion injury) exacerbate the cardiopulmonary sequelae of IAH [16]. Chronic elevation of IAP, as in central obesity, also produces these derangements in the form of obesity hypoventilation syndrome (OHS) [11]. Abdominal decompression improves the acute respiratory failure almost immediately [9–11].

9.4.3 Renal Sequelae

Oliguria progressing to anuria and prerenal azotemia unresponsive to volume expansion characterize the renal dysfunction of ACS [1, 9–18]. Oliguria can be seen at IAP of 15–20 mmHg, while increases to 30 mmHg or above lead to anuria. Volume expansion to a normal CO and the use of dopaminergic agonists or loop diuretics may be ineffective in these patients. However, decompression and reduction in IAP promptly reverses oliguria, usually inducing a vigorous diuresis [17, 18]. The mechanisms of renal derangements with IAH involve reduced absolute and proportional renal arterial flow, increased renal vascular resistance with changes in intrarenal regional blood flow, reduced glomerular filtration, and increased tubular sodium and water retention [1, 17, 18].

9.4.4 Abdominal Visceral Abnormalities

Mesenteric arterial, hepatic arterial, intestinal mucosal, hepatic microcirculatory, and portal venous blood flow all have been shown to be reduced with IAH in animal models [19, 20]. Clinically, many investigators demonstrated that gut mucosal acidosis, demonstrable by intramucosal pH (pHi) measured with gastric tonometry, is a sensitive change after ACS [1, 4, 21]. Further increases in IAP may lead to intestinal infarction, often present in the ileum and right colon without arterial thrombosis. Prolonged low-grade elevation of IAP may be associated with bacterial translocation in rat and murine models [22]. Thus, despite normal systemic hemodynamics, profound splanchnic ischemia can be ongoing with IAH. It has been suggested that such ischemia is associated with an increased incidence of multisystem organ failure, sepsis, and increased mortality [1, 4, 9–11]. Furthermore, laboratory evidence suggests that prior hemorrhage and resuscitation actually lowers the critical levels of IAP at which mesenteric ischemia begins [23]. Many investigators note a relationship between IAH, sepsis, multisystem organ failure, and the need for reoperation and mortality. These are

some of the strongest arguments for the routine measurement of *IAP in critically ill patients.*

9.4.5 Abdominal Wall Abnormalities

Increased IAP has been shown to reduce abdominal wall blood flow by the direct, compressive effects of IAH under conditions of stable systemic perfusion, leading to local ischemia and edema [24]. This can decrease abdominal compliance (defined as a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm and expressed as the change in intra-abdominal volume per change in IAP) and exacerbate IAH. Abdominal wall muscle and fascial ischemia may contribute to such wound complications as dehiscence, herniation, and necrotizing fasciitis.

9.4.6 Intracranial Derangements

Elevated intracranial pressure (ICP) and reduced cerebral perfusion pressure (CPP) have been described with acute changes in IAP in animal models and in human studies [25]. In animal models, the changes in ICP and CPP are independent of changes in pulmonary or cardiovascular function and appear to be the direct result of elevated intrathoracic and central venous pressures with impairment of cerebral venous outflow. Reduction in IAP by surgical decompression reverses this derangement. Furthermore, chronic elevation in IAP has been implicated as an important etiologic factor in the development of benign intracranial hypertension, or pseudotumor cerebri, in the morbidly obese [11].

9.4.7 Polycompartment Syndrome [25]

A polycompartment syndrome, where two or more anatomical compartments have elevated compartmental pressures, is a potential companion of IAH, e.g., intra-abdominal leading to intrathoracic and consequent intracranial hypertension. IAH helps to explain the severe pathophysiological condition occurring in patients with cardiorenal, hepatopulmonary, and hepatorenal syndromes. When more than one compartment is affected, an exponential detrimental effect on end-organ function in both immediate and distant organs may occur. The compliance of each compartment is the key to determining the transmission of a given compartmental pressure from one compartment to another. In high-risk patients, these interactions must be considered for optimal management [25].

9.5 Recommendations in Management

The following are the recommendations from WSACS [13] in the clinical pursuit of IAH and ACS based on the GRADE methodology (grading, assessment, development, and evaluation). Quality of evidence is graded from high (A) to very low (D). Recommendations range from strong recommendations to weaker suggestions.

The *recommendations* include use of protocolized monitoring and management of IAP [GRADE 1C], efforts and/or protocols to avoid sustained IAH among critically ill or injured patients [GRADE 1C], decompressive laparotomy [27] in cases of overt ACS [GRADE 1D], conscious and/or protocolized efforts be made among ICU patients with open abdominal wounds to obtain an early or at least same-hospital-stay abdominal fascial closure [GRADE 1D], and among critically ill/injured patients with open abdominal wounds, use of strategies utilizing negative pressure wound therapy [GRADE 1C].

The *suggestions* include the following: clinicians ensuring that critically ill or injured patients receive optimal pain and anxiety relief [GRADE 2D]; brief trials of neuromuscular blockade as a temporizing measure in the treatment of IAH/ACS [GRADE 2D]; considering the potential contribution of body position to elevated IAP among patients with, or at risk of, IAH or ACS

[GRADE 2D]; liberal use of enteral decompression with nasogastric or rectal tubes when the stomach or colon is dilated in the presence of IAH/ACS [GRADE 1D]; neostigmine be used for the treatment of established colonic ileus not responding to other simple measures and associated with IAH [GRADE 2D]; using a protocol to try and avoid a positive cumulative fluid balance in the critically ill or injured patient with, or at risk of, IAH/ACS after the acute resuscitation has been completed [GRADE 2C]; use of an enhanced ratio of plasma/packed red blood cells for resuscitation of massive hemorrhage versus low or no attention to plasma/packed red blood cell ratios [GRADE 2D]; use of percutaneous decompression to remove fluid (in the setting of obvious intraperitoneal fluid) in those with IAH/ACS as, when this is technically possible [GRADE 2C], this may also alleviate the need for decompressive laparotomy [GRADE 2D]; trauma patients with physiologic exhaustion undergoing abbreviated laparotomy be treated with the prophylactic use of the open abdomen and expectant IAP management [GRADE 2D]; not routinely utilizing the open abdomen for patients with severe intraperitoneal contamination undergoing emergency laparotomy for intra-abdominal sepsis unless IAH is a specific concern [GRADE 2B]; and avoiding the routine use of bioprosthetic meshes in the early closure of the open abdomen compared to alternative strategies [GRADE 2D].

The WSACS noted that the evidence did not support any recommendations about the use of abdominal perfusion pressure (MAP-IAP) in the resuscitation or management of the critically ill or injured and the use of diuretics, albumin, or renal replacement therapy to mobilize fluids in hemodynamically stable patients with IAH after the acute resuscitation has been completed.

9.6 Management of IAH and ACS [13]

The most effective approach in the management of IAH and ACS is best summarized by the algorithms recommended by WSACS (Figs. 9.2 and 9.3).

9.7 Current Status of IAH and ACS

As noted earlier, the efforts of WSACS made a profound impact on our understanding of the disease and our clinical approach. Anticipation of the complication, measures of prophylaxis, earlier recognition, and intervention: all of these translated into fewer organ failures and better survival. The complications of the open abdomen may be offsetting some of these benefits. More advanced care of "laparotomy," however, is minimizing this weakness.

In a prospective, observational study, Cheatham and Safcsak [28] studied 478 consecutive patients who were treated with open abdomen for IAH and ACS according to "a continually revised management algorithm" and noted a significantly increased patient survival to hospital discharge from 50% to 72% ($p = 0.015$) and an increase in same-admission primary fascial closure from 59% to 81% over the period of the study, one of the first clinical series showing better outcomes with a management focus on IAP that did not increase resource utilization. Balogh and associates [29] prospectively analyzed 81 consecutive severely injured shock/trauma patients (mean ISS 29) admitted to the intensive care unit. They had a protocol of two-hourly intra-abdominal pressure (IAP) monitoring. No patient developed ACS, even though 61 (75%) had IAH. One patient with IAH and one without died. Multiorgan failure occurred in one patient without IAH (5%) vs. 4 with IAH (7%). The authors commented that monitoring and intervening for a less serious IAH and the avoidance of the deadly ACS are remarkable successes of critical care in the last decade. Recent evidence concluded that the current practice of restricted fluid resuscitation and liberal use of damage-control strategies among trauma patients along with monitoring for IAH has lowered the prevalence of ACS [30]. While established trauma centers and academic institutions were eliminating ACS by aggressive application of the concepts narrated in this chapter, these paradigms apparently have yet to be promulgated widely.

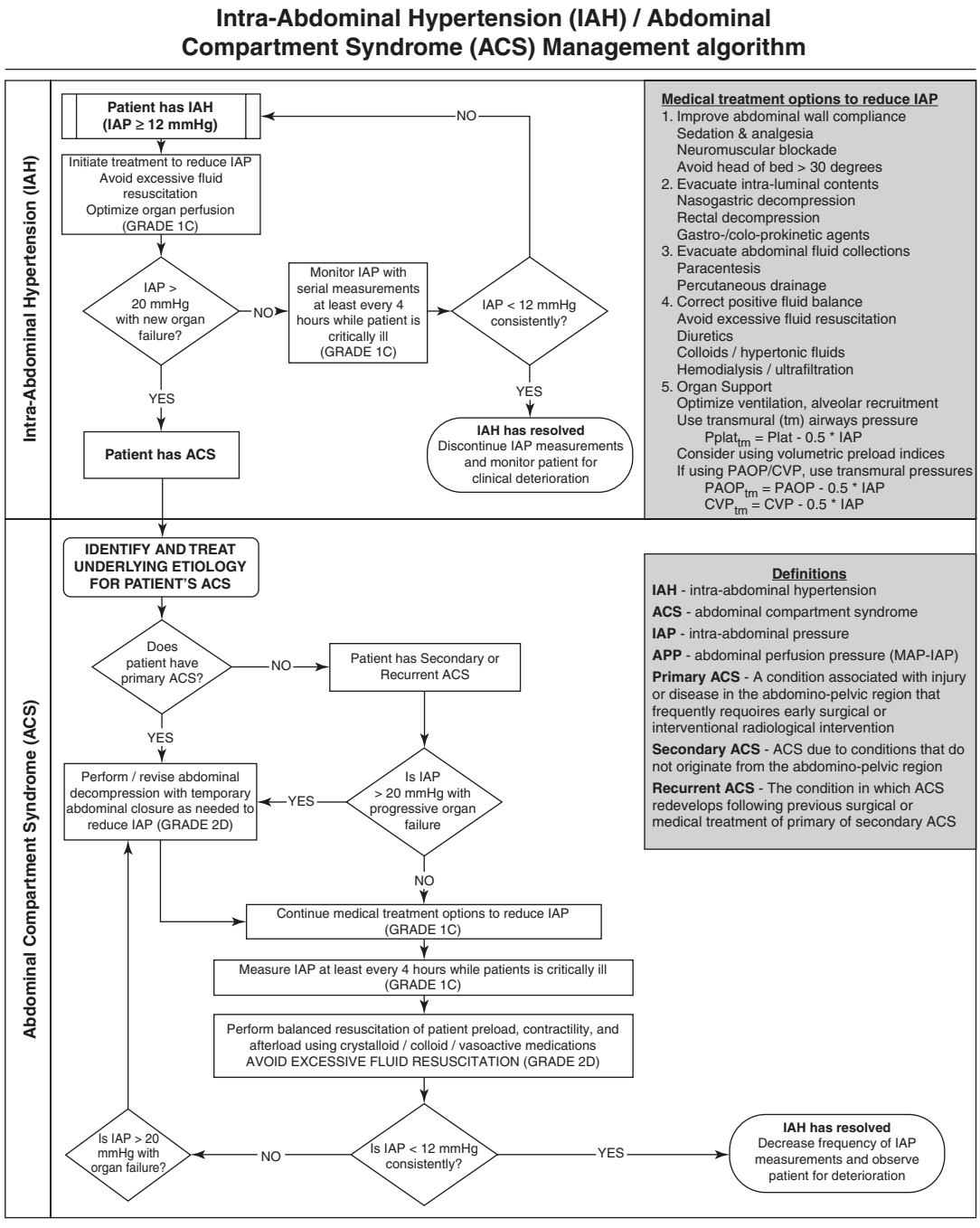

Fig. 9.2 Management algorithm for IAH and ACS (Reproduced from Kirkpatrick et al. [13])

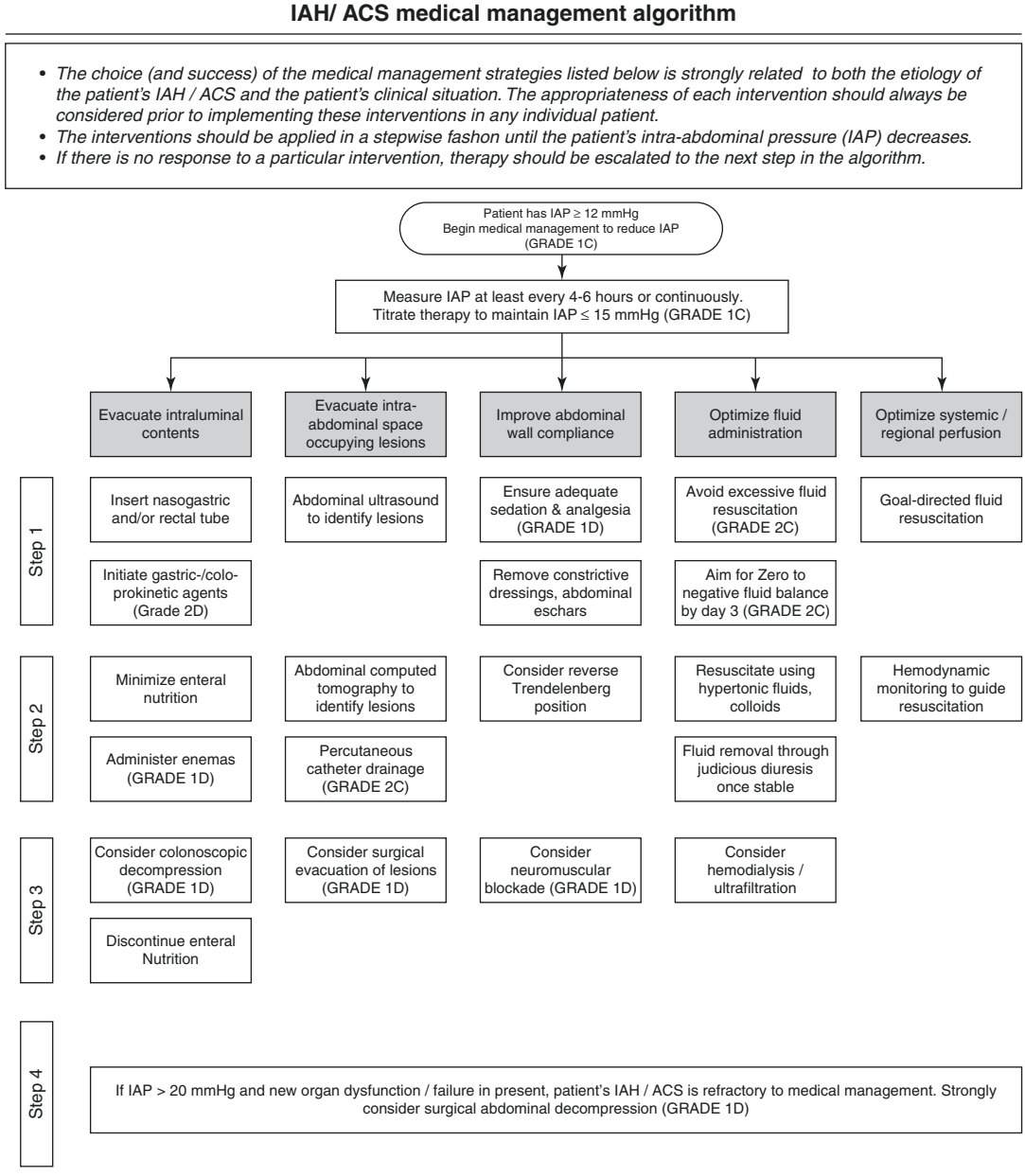

Fig. 9.3 Medical management of IAH and ACS (Reproduced from Kirkpatrick et al. [13])

9.8 Awareness and Appreciation of IAH and ACS

In 2013 the WSACS distributed a survey of 13 questions to 10,000 members of the WSACS, the European Society of Intensive Care Medicine (ESICM), and the Society of Critical Care Medicine (SCCM). A total of 2244 clinicians responded (response rate, 22.4%), a majority from North America. The majority of responders (85%) were familiar with IAP/IAH/ACS, but only 28% were aware of the WSACS consensus definitions. Overall knowledge scores were low (43 ± 15%). Respondents that were aware of the WSACS had a better score compared to those who were not (49.6% vs. 38.6%, $P < 0.001$), suggesting ignorance of established consensus definitions and guidelines [31]. Another study [32] surveyed Dutch surgeons with a literature-based and expert consensus survey. Sixty of 87 (69%) invited surgeons completed the questionnaire. Many of these surgeons exhibited a good knowledge of IAH and ACS, but only 27% used this in their daily practice. Another survey tried to clarify the current understanding and clinical management of intra-abdominal hypertension (IAH)/abdominal compartment syndrome (ACS) among Chinese intensive care physicians in tertiary hospitals [33]. The study concluded that urgent systematic education is absolutely necessary for most intensive care physicians in China to help to establish clear diagnostic criteria and appropriate management. A similar lack of application of definitions and guidelines was reported among German pediatric intensivists [34] and Australian critical care nurses [35].

In summary, IAH and ACS are common complications in the care of the critically ill or injured patients, medical or surgical, young or old. They can cause profound morbidity and mortality, if unanticipated, unrecognized, and uncontrolled. Appropriate monitoring and early intervention, based on the precepts of WSACS, can minimize organ failures, morbidity, and mortality. It appears, however, that the dissemination of the current knowledge of IAH and ACS is yet incomplete. It is definitely time to promulgate the pathophysiology of increased pressure in rigid compartments [36].

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10 Orthopaedic Surgery Approach to Damage Control: Decision-Making and Indications

Marius Keel and Hans-Christoph Pape

10.1 Introduction

Decision-making may include several processes, such as assessment of the patient, assessment of the surgeries and the resources required, and the perspective of management. It requires the vision, the knowledge and the skills of an experienced trauma surgeon or of a trained team of specialized surgeons according to the injury pattern. In this line, the concept of the borderline trauma patient respects the fact that the patient status can change over time and may affect the decision-making process. The widely used concept relies on the triad of death and the factor of organ and soft tissue injuries, which may include head, abdominal or chest trauma as well as severe extremity injuries and complex spinal and pelvic trauma. It is interesting to note that the only *evidence-based definition of polytrauma* summarizes a number of parameters rather than just a single one: cofactors were calculated on the basis of a nationwide registry. Five independent physiologic variables were identified as follows: hypotension, level of consciousness, acidosis, coagulopathy and age [1] (Table 10.1 and Fig. 10.1).

Table 10.1 Evidence-based definition of polytrauma

AIS >2 points and at least one of the following covariables
Hypotension (systolic blood pressure <90 mm Hg)
Level of consciousness (Glasgow Coma Scale [GCS] score <8)
Acidosis (base excess ≥6.0)
Coagulopathy (international normalized ratio 1.4/ partial thromboplastin time >40 s)
Age (>70 years)

The measurement of inflammatory markers that highlight patients at risk is helpful in deciding which patients are best served by damage control surgery. The use of a single parameter is inappropriate, as multiple factors can influence the course in multiple ways.

These are best described for the triad of death (shock, hypothermia, coagulopathy):

1. Hypothermia is known to affect coagulation and does not address the clinical situation completely, if addressed alone [2]. It has to be viewed and treated within the general context.
2. Coagulopathy affects several other pathways, such as the cellular energy turnover, the cardiac effects induced by hypothermia. This may not allow for safe definitive surgery (see below) [3].
3. Shock belongs to the triad of death as well, and the parameter used the most is serum lactate. However, care should be taken not to rely on

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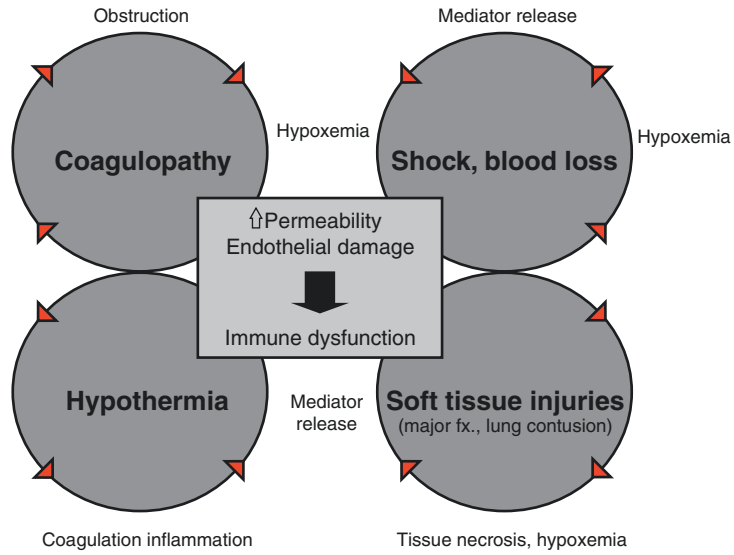

Fig. 10.1 Four vicious cycles

lactate alone: Various metabolites may affect the measurement of metabolic acidosis [4]. Elderly with chronic diseases – such as renal failure – may demonstrate pathological lactate values [5]. These factors contribute to the general inflammatory response after trauma.

4. Soft tissue, respectively, organ injuries of the head, abdomen, chest, spine, pelvis and the extremities are parameters of the first trauma hit that put the patient at risk as summarized below [6–8].

It has also been suggested that patients at risk of adverse outcome, such as those with head injury, bilateral lung contusions, multiple long bone injuries, coagulopathy, hypothermia, or estimated operation time of >6 h, should be considered for sequential staged surgical management [9] (Table 10.2). Markers of the adequacy of shock reversal, such as serum lactate, are measured routinely in trauma centres. It is therefore easy to envisage the routine use of markers of pro- and anti-inflammatory systems such as IL-6, IL-10 and procalcitonin to aid in the decision to carry out damage control surgery. Delaying definitive surgery until the shock state is fully reversed would appear to be not only beneficial but imperative [12].

The Polytrauma Grading Score (PTGS) [9] has reconfirmed that multiple parameters are important, and even in a prospective database analysis, acid-base abnormalities [11] and coagulopathy, the number of pBRCs administered and the injury severity score have been proven to be of value.

Table 10.2 Recommendations to consider damage control within the safe definitive surgery concept

Parameter/clinical diagnosis	Recommendation
Head injury	Degree unclear in the literature, no recommendation possible
Bilateral lung contusions	TTS score [10]
Estimated operation time of >6 h	Includes visceral operations, followed by orthopaedics [11]
Multiple long bone injuries	>2 of the lower extremity
Hypothermia or coagulopathy	Unresponsiveness to resuscitation

Therefore, the decision-making according to the ‘four pathophysiological cascades of polytrauma’ appears to be valuable. The parameters of these four cascades to be remembered could be summarized in the following phrase:

> Soft tissue injuries (major extremity fractures, severe pelvic fractures, spinal injuries especially with spinal cord injury, organ injuries such as head injuries AIS>2 or lung contusions AIS>2), coagulopathy (platelets <90,000) and shock (systolic BP<90 mmHg, requirement of vasopressors) contribute to hypothermia (core temp. <33 °C) and systemic inflammation, and are dangerous. [5]

Ich verstehen das mit dem soft tissue injury nicht, Organverletzungen wie Leber oder Head sind doch auch grosse Risikofaktoren?

10.2 Steps of Decision-Making: Safe Definitive Surgery Concept to Include Damage Control and Early Definitive Care

After the initial assessment is completed using ATLS principles, the treating physician usually gets a fairly good impression about whether the patient is at risk for acute haemorrhage that may lead to lethal outcome [11].

The *safe definitive surgery* concept encompasses both components from early definitive surgery and damage control since the clinical scenario can change rapidly that may require a change in the management [12, 13].

These stages apply for the surgical approach. It is understood that nonsurgical causes of instability have to be addressed in a parallel fashion: these usually imply issues of coagulopathy, hypothermia or any combination of the four pathological cascades.

The patient's condition may range from clinically stable to a state named 'in extremis', where there is imminent danger of death. Fortunately, the majority of patients belongs to the group classified as 'stable' or to the 'borderline' patient group (grade I or II (if stable after resuscitation)) that can be safely stabilized during the course of the emergency treatment.

Stable patients have the physiological reserve to withstand prolonged operative intervention where this is appropriate and can be managed using an early total care approach, with reconstruction of complex injuries.

For the *borderline patient,* primary stabilization strategies may be used but should be undertaken with caution and forethought given to operative strategy should the patient require a rapid change of treatment rationale. Additional invasive monitoring should be instituted and provision made for intensive care unit admission.

To reduce the surgical burden, an unreamed nail may be considered for the femur if possible, and the surgeon should be alert to the possibility of having to convert to the damage control pathway at any time throughout the procedure if the clinical condition of the patient deteriorates, called 'bail-out' procedure.

Treatment in *unstable patients* has evolved to utilize a 'damage control' approach as preemptive intervention. This entails rapid life-saving surgery only as absolutely necessitated and timely transfer to the intensive care unit for further stabilization and monitoring. Temporary stabilization of fractures using external fixation, haemorrhage control and exteriorisation of gastrointestinal injuries where possible is advocated. Complex reconstructive procedures should be delayed until stability is achieved and the acute immunoinflammatory response to injury has subsided. This rationale is intended to reduce in magnitude the 'second hit' of operative intervention or at least delay it until the patient is physiologically equipped to cope.

Conclusion

Decision-making should be performed rapidly and may be subject to revision before, during or after the first surgical phase. Some trigger factors are known that require damage control or abbreviated surgeries. Among these are severe head and chest trauma, multiple fractures if the patient is unstable, or uncontrollable exsanguination due to severe abdominal or pelvic trauma. Damage control orthopaedics is recommended for an unstable patient or a patient in extremis, and it has some utility for the borderline patient as well. Specific injury combinations for which damage control orthopaedics should be considered are femoral fractures, if bilateral, pelvic ring injuries with profound haemorrhage and multiple injuries in elderly patients.

This process of decision-making may be defined as 'injury patient tailored' for damage control orthopaedics, e.g. safe definitive surgery. Regarding this strategy, it continues to be essential to validate prognostic criteria, as achieved in the Polytrauma Grading Score.

Further studies should be fulfilled to better understand the role of damage control orthopaedics in the treatment of patients that sustained a combination of orthopaedic trauma and concomitant injuries to the chest and head.

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General Surgery Approach to DC: Decision Making and Indications

11

Molly Deane and Jose J. Diaz Jr

11.1 Introduction

The process by which to select the appropriate patients to undergo damage control surgery is the fundamental beginning to decision making. Classically, the indications to truncate an operation rather than proceed with primary definitive surgical care are acidosis, hypothermia, and coagulopathy (the so-called lethal triad) [1]. Rotondo and colleagues coined the term "damage control" laparotomy for exsanguinating penetrating injuries (with transfusion of greater than 10 units packed red blood cells) where a survival benefit was noted for a subset of maximally injured patients, those with major vascular and two or more visceral injuries [2]. The process of damage control surgery is divided into phases. The surgeon must maneuver the patient through these phases and constantly reevaluate the overall status.

11.2 Indications

In the severely injured patient, time is of the essence, and the primary goals of damage control are to control hemorrhage and stop contamination. This is followed by a period of time for resuscitation and restoring physiologic reserve. Patient selection comes from recognizing the severity of the mechanism of injury, the complexity of the injury pattern, and/or the presence of physiologic derangements in light of recognized patient comorbidities. It is usually a combination of multiple factors that end up necessitating the use of damage control surgery. The surgeon's attention to subtle clinical findings at the index operation may be the first sign a patient would benefit from the damage control approach. Noting that the patient feels cold, that there is no presence of clot in the surgical field, or that the bowels are becoming boggy and edematous are key intraoperative findings.

It should be noted that overuse of damage control exposes patients to the risks of multiple operations, open abdomen management, and prolonged intensive care stay thereby negating the potential benefit of the concept [3]. It is estimated that fewer than 30% of civilian trauma laparotomies benefit from the damage control approach [3]. Its overuse has been demonstrated to cause potential harm and can result in long-term morbidity [4]. The most frequent complications following an open abdomen include gastrointestinal fistulas, intra-abdominal abscesses, and ventral hernias [5]. Careful identification of the appropriate patient and refraining from the overuse of damage control surgery can avoid unnecessary complication.

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11.3 Patient Factors

Early recognition of the patient's existing comorbidities, prior surgical history, and whether they are taking any anticoagulants or antiplatelet function medications can allow for early selection of patients for damage control surgery. In addition, some patients may have low functional reserve and benefit from a truncated operation and ushering to the ICU.

11.4 Mechanism/Injury Pattern

The type of mechanism of injury can be crucial to selection of patients for damage control, and those selected will typically have blunt polytrauma, multiple penetrating torso trauma, severe contamination, and major bleeding sources in other regions. These warrant consideration for damage control surgery as they require prioritization of injuries and the severity of injury is likely to result in the lethal triad.

Early into the operative case, it is possible to identify patients who are more likely to benefit from damage control due to the degree of the injuries. Patients who require packing of complex liver injury are best served by damage control. These patients will potentially require angio-embolization prior to their next operation. Patients with multiple bowel injuries such as combined small bowel and colon injuries with segmental resection will require "planned second look." These patients may require additional bowel resection with multiple anastomoses. Similarly, patients with multiple combined and complex injuries within the abdomen such as gastric and renal or pancreas and spleen will need a damage control laparotomy. Abbreviated control of multiple complex injuries with packing or stapling and adequate drainage can be utilized to control the abdominal injuries and facilitate stabilization in order to be able to address other areas of injury.

When considering the mechanism of injury in a patient with multiple cavity or extremity injuries, it is important to keep in mind the possibility of involving multiple surgical teams to treat injuries simultaneously. Examples may include one team performing a laparotomy and packing the liver and resecting bowel, while another team obtains vascular control with a shunt in an extremity or places multiple extremity external fixation devices for complex crush injury associated with fractures. Not only considering that the patient require a damage control surgery for management of their general surgical injuries, but placing these in context with the other injuries allows for efficient management and expedited transition to the ICU for ongoing resuscitation. In these challenging cases, it is critical for the surgeon to triage the critical procedures and involve the appropriate teams.

11.5 Physiologic Derangements

Patients are actively resuscitated upon arrival, and the process extends into the operating room and intensive care unit with the goal of avoiding the lethal triad prior to its start to avoid failure of correction. Early recognition of any of the following should indicate the potential need for damage control surgery [6].

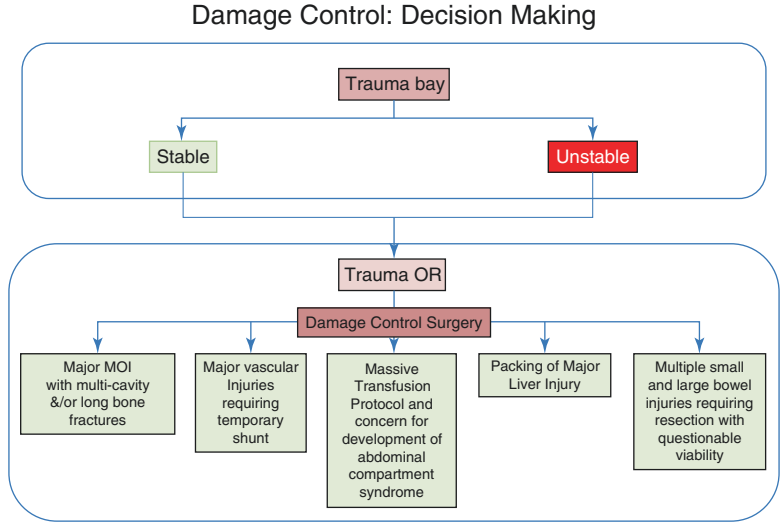
Significant bleeding requiring >10 units PRBC
Severe metabolic acidosis pH <7.20
Hypothermia temp <35.0 °C
Operative time >90 min
Coagulopathy as seen by either laboratory result or "nonsurgical" bleeding
Lactate >5 mmol/L

11.5.1 Decision Making: Considerations at the Index Operation

Trauma patients undergoing laparotomy for truncal injuries should be prepped from the chin to both thigh and bilateral posterior axillary lines to have full access to all body regions and maximize efficiency of the operation.

"Time is of the essence." Once the decision has been made to proceed with damage control surgery, the surgeon's goal should be to complete

the initial procedures within 90 min or less. The clear objectives are (1) to control hemorrhage and (2) stop or control contamination. The abdomen should be left open and a temporary abdominal closure barrier placed. These patients undergoing damage control surgery typically require a massive transfusion and are at risk for abdominal compartment syndrome. In addition, rapid return into the abdomen is occasionally required.



In addition, it is not uncommon to start a trauma laparotomy and find oneself in the midst of a very unexpected injury pattern which quickly goes from bad to worse. What may have started out as a semi-elective laparotomy turns into a fight for the patient's survival. Once must be able to quickly shift gears and move to a "damage control" mode.

11.6 Hemorrhage Control

The first step in the trauma laparotomy is performing a generous midline incision as it provides exposure to all four quadrants in the abdomen. The next step is deciding on exposure, commonly a self-retaining surgical retractor should be ready to place and retract the abdominal wall.

Hemorrhage control initially starts by exposing the bleeding. This is best done by efficiently packing the abdomen with laparotomy pads. This has the dual effect of "mopping up" and tamponading the bleeding. This maneuver allows the anesthesia team time to begin to catch up with their resuscitation. The importance of packing cannot be underestimated, as it allows for time for both the surgical team and the anesthesia team to strategize and determine if this is a patient who can now have their injuries formally addressed or dealt with in a damage control manner.

While the initial maneuver of hemorrhage control is packing, pattern recognition will guide the surgeon to the potential source of bleeding. If the patient remains profoundly hypotensive following packing, a significant source of arterial hemorrhage is likely. A well-packed liver injury that continues to bleed may have a retrocaval injury. If bleeding is coming from the mid abdomen after packing of the gutters, bleeding may be coming from the root of the mesentery. If an enlarging retroperitoneal hematoma is identified in any of the zones, a surgical bleeding is likely to be encountered. Suspicion of this type of injury may require temporary aortic occlusion with either a cross-clamp or intra-aortic balloon both of which require additional areas to be prepped and additional equipment.

Major vascular injuries should be initially controlled with Allis clamps for lateral injuries or

cross-clamps for transections. For major arterial or venous injuries, initial management may be either vessel ligation or the placement of temporary intravascular shunts in critical arteries [7]. In the context of uncontrolled hemorrhage, the subclavian vein, iliac vein, and inferior vena cava can all be ligated at the risk of development of severe limb edema. The external carotid can be ligated without consequence and the internal carotid with the risk of neurologic deficit. Ligation of the femoral artery can result in critical limb ischemia, slightly higher than the risk of ligating the subclavian artery, however both can be shunted [8].

For patients with severe liver injuries, the initial goal should be control of bleeding, as it is the uncontrollable hemorrhage which is the cause of early death and the related requirement of massive transfusion which contributes to late fatal complications [9]. A complex liver injury can be managed with a Pringle maneuver followed by supra and infrahepatic packing. The falciform ligament should be divided as not to injure the liver during packing. Pattern recognition should continue to be appreciated when the packed liver packing does not stop bleeding and appears. An arterial injury should be suspected and suture ligated must be performed if possible. A necessary consideration is the role of angiography as an adjunct for therapeutic intervention. While another option is going to angio-embolization after packing of a major liver injury, when planning on taking a patient with significant abdominal trauma for operation, placing the patient on a radiographically compatible table or in a hybrid suite can facilitate performing angiography for control of visceral or pelvic hemorrhage after the abdomen has been packed or externally fixated.

11.7 Contamination

In some circumstances the mechanism of the diffuse extent of injury can require a "second look" in order to evaluate the evolution of injuries. In high-energy injuries the extent of bowel wall injury is often not apparent at the initial operation, and a second evaluation is crucial as these injuries can result in delayed ischemia or perforation, which can threaten anastomoses and stomas. Knowing the patient will require a "second look" based on the injuries allows a switch to proceeding with damage control and foregoing a longer more definitive operation. Those necessitating "second look" consist of boggy or edematous bowel, dusky with poor perfusion without frank ischemia, and in areas of mesenteric hematomas without bowel compromise.

After resecting affected sections of bowel, the next decision point is when to perform the anastomosis and in what fashion. With regard to deciding between stapled versus handsewn, numerous studies have attempted to evaluate if one is superior to another and have yet to identify a striking difference [10]; however in difficult cases with edematous bowel, many surgeons will tend toward performing a handsewn anastomosis. A potential reason to perform a handsewn anastomosis is when there is bowel edema. There are comments in the literature stating that there still may be a difference in outcomes between the two, which has simply failed to be demonstrated by existing literature. It has been reported that handsewn anastomoses do consume significantly more time and this should be taken into account [11].

For colonic injuries, these can be primarily repaired or resected and placed back into continuity with an anastomosis. The average leak rate is 16% and from the largest study 18%. There is the suggestion that repair and anastomosis is preferable in a patient in whom there is an open abdomen. The rationale is such that while this is a "high-risk" anastomosis, with the abdomen open, the bowel can be inspected for potential complications prior to abdominal closure, and this eliminates the potential morbidity of an ostomy [12]. Another suggestion is to wait to decide whether to perform an anastomosis versus ostomy at the second operation after determining whether the patient is able to be closed [13]. The reasoning is that there is an increase in complications including anastomotic leaks when patients are not closed within 5 days. All of these factors address thinking of damage control surgery with care of bowel in isolation. This should be taken in

the context of potential confounding factors such as suspicion of tenuous blood supply or other associated injuries, such as pancreatic injury, which may place a repair or anastomosis at additional risk.

11.8 Management of the Open Abdomen

After patients have undergone correction of their physiologic derangements, they should return to surgery for definitive repair. Ideally, this first trip back to the operating room should occur within 36 h. This return to the operating room can be done in either an on-demand or scheduled fashion; however, on-demand laparotomy is associated with a reduction in re-laparotomies and negative laparotomies and may result in cost savings. In a normothermic patient, they should undergo re-laparotomy for ongoing transfusion requirement of 2 units of RBC/hour [14]. There is the potential that during this first return to the operating room, they may not be able to achieve definitive repair of their injuries or the edema is such that it is not possible to close the abdomen. Significant risk of development of abdominal compartment syndrome is a contraindication to abdominal closure, as is recurrence of physiologic derangement or ongoing contamination.

The concept remains that there must be a continual reevaluation of the status of the patient and their injuries in order to determine whether or not the patient is ready to undergo their definitive surgical therapy. When evaluating which patients were unlikely to achieve primary fascial closure during their initial hospitalization, it is patients who had higher numbers of explorations and developed intra-abdominal abscess/sepsis and blood stream infections and those who develop acute renal failure or enteric fistulas and ISS >15 [15].

As soon as physiologically possible, patients do better with abdominal closure. In keeping this in mind, it is possible to reduce the morbidity associated with damage control laparotomy. For patients closed at first take back, the overall complication rate was reduced to 47%, significantly lower than the reported 63% for all patients managed with damage control with an average of 1.66 complications per patient [16]. In addition, patients closed within 7 days of their index operation were found to have less daily pain, higher rate of return to work after injury, and higher quality of life [17].

Conclusions

When considering damage control management, it is important to continually reassess the patient's clinical status and prioritize this along with their injuries to perform focused operative interventions with the main goal of resuscitation. At each point one must ask whether the patient has ongoing physiologic derangements or are they able to move on to the next phase. It is important to recognize that this is a dynamic process, and at each branch point, the patient can return to a state of dysfunction and require ongoing damage control management.

One approach to conflicting priorities is to involve multiple teams simultaneously with each addressing a separate injury/region in order to minimize operative time/stress and more quickly address hemorrhage and contamination.

Careful consideration of the long-term outcomes aids in operative management in order to give critically injured patients the highest chance of the best possible outcome given their particular circumstance and injuries. The goal is to do what is vitally necessary up front, move to the ICU for resuscitation, then stage the definitive repairs. Minimizing the number of patients selected for damage control to only those necessary and then minimizing the number of interventions, time to anastomosis, and time to closure have improved outcomes.

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New Technologies for Vascular Injuries and Hemorrhage Control

12

Megan L. Brenner and Thomas M. Scalea

12.1 Introduction

The rapid control of hemorrhage is at the cornerstone of all injury care. In most cases, direct surgical exploration and control of hemorrhage suffices even with large volume blood loss. However, some new advances have given us additional tools that can be quite helpful in selected cases. Binders can reduce the bony elements of the pelvis, reducing pelvic fracture hemorrhage. A number of commercially available hemostatic agents provide direct pressure onto blood vessels and promote clotting. Newer resuscitation strategies stress the use of plasma and platelets early. This balanced resuscitation likely prevents the coagulopathy of trauma instead of treating it.

In addition, endovascular techniques have revolutionized care for badly injured patients. Angiography allows for precise definition of bleeding arteries without surgical exploration. This minimally invasive technique does not risk the disruption of the existing hematoma and does not increase the blood loss or heat loss associated with open surgical procedures. In order to be effective, all components of this resource must be immediately available at all times.

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Obtaining proximal control of the aorta at the diaphragm is a standard maneuver to reduce intra-abdominal blood loss. It requires a laparotomy or, in some cases, entering the left chest. The standard intraoperative techniques of inflow control within the abdomen include direct clamping, manual compression, sponge-stick compression, and the archaic "aortic occlude." Transfemoral balloon occlusion of the aorta has been utilized for decades in cases of exsanguinating hemorrhage below the diaphragm for numerous non-trauma conditions. Recently, the technique has been adapted for use in trauma patients and is currently performed by acute care surgeons in the USA. Patients who may benefit from this include those in shock with intra-abdominal or pelvic bleeding.

It is mandatory that surgeons interested in the care of badly injured patients "lead the charge" in these new areas. While other disciplines certainly have advanced skills and much too often in areas such as endovascular techniques, only the surgeons understand the care of the entire patient. Regardless of how care is organized in an individual center, the trauma surgeon must remain "captain of the ship." Patient selection is key to the successful utilization of these innovative techniques, again stressing that trauma surgeons need to be involved. It is imperative to serially assess patient stability and change course if the patient's status changes. These patients are usually quite dynamic, and it requires minute-to-minute and even second-to-second decision-making to utilize these innovative strategies safely.

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Fig. 12.1 Hybrid operating room at RA Cowley Shock Trauma Center

12.1.1 Endovascular Damage Control

Damage control, described in 1993, represented a paradigm shift in trauma care using damage control. All operative care was no longer accomplished at the time of presentation. Instead, only life-threatening hemorrhage was addressed at the index operation. Nonsurgical hemorrhage was controlled with temporary hemostatic packing, gastrointestinal contamination temporarily controlled, and the abdomen left open. The patient was then brought to the intensive care unit. After physiologic stabilization, the patient is returned to the operating room for completion of definitive care.

Initially, damage control was accomplished with direct surgical maneuvers only. However, it soon became clear that endovascular procedures could serve as a valuable adjunct to a traditional surgical approach. Angiographic embolization, which was then being introduced for trauma hemostasis all over the body, became part of damage control. Typically, patients underwent their initial surgical procedure and were immediately taken to the angiography suite for catheter hemostasis. As experience accumulated, trauma practitioners recognized catheter hemostasis as a powerful tool to control hemorrhage in areas that were difficult to approach surgically, such as bleeding deep in the right lobe of the liver or in the pelvis.

As damage control has evolved and access and expertise to endovascular techniques have increased, the use of endovascular hemostasis as part of damage control has become more frequent. These endovascular techniques are now sometimes used as definitive therapy with operative care, but still are often used in combination with operative hemostasis. The advent of hybrid operating rooms has greatly increased the number of possibilities. These novel ORs now allow all care to be delivered in a single location, eliminating the need for multiple transfers (Fig. 12.1).

12.1.1.1 Angiography and Embolization

The management of pelvic and renal hemorrhage using catheter-based techniques was published in small numbers almost 50 years ago [1, 2]. Shortly thereafter, the first report of intercostal injury controlled by embolization was published in 1977 [3]. Angiography for pelvic fractures was first described in 1972. Later case reports suggested angioembolization alone or in combination with surgical therapy for highly lethal pelvic hemorrhage was both feasible and effective [4–6]. At the

same time, the group at Kings County Hospital in Brooklyn pioneered endovascular therapy for trauma and reported good outcomes in several series treating both splenic and pelvic hemorrhage [7–9]. Endovascular control of hemorrhage became routine. Roudsari et al. recently reported the use of angioembolization for pelvic and liver injuries increased from 30–50% in 1996 to 100% in 2010 at a designated level 1 trauma center [10].

Early on, operative exploration was thought to be necessary for all abdominal injuries. Early diagnostics like physical exam and diagnostic peritoneal lavage could identify patients with injuries, but could not identify the injured organ or the severity of the organ. CT scanning allowed for organ-specific diagnosis and preoperative injury grading and allowed clinicians to manage some injuries without surgery. Catheter hemostasis then became attractive as an adjunct to observation. Patients with isolated solid organ injury could be managed without the need for open exploration. When effective, this reduced morbidity and hospital length of stay. Soon, patients with multiple injuries such as a liver and splenic injury started to be managed with catheter hemostasis alone. At that time, patients had to be stable to use these techniques.

Perhaps the greatest limitation in the use of angioembolization is universal availability. Even at major trauma centers with advanced capabilities, there often are discrepancies in availability of this adjunct during hours of peak trauma intake, nights and weekends [11]. One novel solution being explored is to expand the skillset of initial trauma responders, who are most commonly present at the bedside when victims of significant injury arrive, often in the middle of the night and on weekends [12]. Certainly, basic endovascular skills, such as the emergent placement of transfemoral balloons, should be in the armamentarium of the acute care surgeons who routinely insert large-bore lines percutaneously. However, additional expertise as needed can be gained in well-designed courses [13]. Additional study is required to determine the extent of training required to safely incorporate more advanced skills such as embolization into the skillset of the trauma/acute care surgeon and how to maintain competence over time as these procedures are used relatively rarely, even in busy centers.

Interventionalists in Japan, where acute care surgeons are not as readily available as in other countries, have extended the use of catheter-based hemorrhage in the treatment of severe solid organ and pelvic injuries, including hemodynamically unstable patients [14–16]. In Japan, where waiting for a surgeon may not always be feasible, by virtue of necessity, trauma practitioners have heavily adopted interventional methods with success using a technique they have termed damage control interventional radiology (DCIR), a non-operative technique analogous to damage control surgery in poly-injured patients. In Japan, these interventionalists utilize "prompt and rapid endovascular strategies in trauma occasions" (PRESTO), referring to the aggressive and time-conscious use of endovascular therapy in trauma. They have adapted their environment to be able to use this safely. The admission area in these facilities has the ability to perform X-ray, CT, fluoroscopy, and resuscitation in the same room [17]. Because some admitting emergency physicians are also trained interventionalists, the usual hurdles to rapid angioembolization are essentially nonexistent.

Angiography may also be used for diagnostic purposes, particularly in the multi-trauma patient whose body habitus or hemodynamic instability prevents travel to the CT scanner. Injuries identified with diagnostic angiography can be treated at the same time. This includes endovascular control of splenic, hepatic, pelvic, carotid, lumbar, and intercostal artery injuries. Technology has advanced from simple nitinol coils to Gelfoam and plug devices which can be used to rapidly stop hemorrhage in these locations. N-Butyl cyanoacrylate (NBCA) is used with success as it acts quickly and permanently occludes vessels [18, 19], as opposed to some agents such as Gelfoam "slurries," coils, or plugs, which can sometimes take many minutes to provide complete occlusion. This makes endovascular techniques attractive, even in patients with substantial ongoing hemorrhage.

The appropriate level of angioembolization can be controversial. Embolization at the level of an

origin artery can be accomplished quickly and, in many vascular beds, effectively controls bleeding. However, continued bleeding via collateral circulation can occur. The regional sparing of organ tissue that can be achieved with more distal "selective" or "super-selective" embolization makes it attractive. The complexity and time required that may be associated with achieving this pinpoint hemostasis of a bleeding pelvic vessel or solid organ injury site must be balanced against concerns for ongoing bleeding and/or other pressing needs in a multi-injured trauma patient. This is particularly pertinent when being used as part of damage control. Defining the optimal approach is challenging, as the variety of variables that must be considered makes focused study of this issue problematic.

While the use of angioembolization technique is currently well established, with a proven track record in the treatment of pelvic and solid organ bleeding after injury [15, 16, 20–23], patient selection continues to be an active area of discussion. Patient physiology plays a significant role in the utilization at most centers. Associated radiographic findings, including significant hematoma and active contrast extravasation or "blush," also likely guide the wise employment of this modality. Despite extensive study on the topic, ideal selection criteria remain a matter of disagreement. Several association guidelines have been developed for the management of severe solid organ and pelvic hemorrhage such as the Western Trauma Association, the Eastern Association for the Surgery of Trauma, and the American Association for the Surgery of Trauma.

Embolization can often be used as definitive hemostasis. This technique seems ideally suited for stable patients with high-grade splenic injuries. In such cases, observation may fail between 25% and 50% of the time. Mandatory use of coil embolization in the proximal splenic artery in high-grade splenic injuries, regardless of whether a contrast blush is present, has been shown to result in splenic salvage over 95% of the time (Fig. 12.2).

Embolization can also be combined with surgery and is an integral part of total patient care when using damage control techniques. It is perhaps most commonly combined with operative hemostasis in patients with high-grade liver injuries. In such cases, patients are typically in shock and are explored initially. Large vessel bleeding is controlled surgically. However, injured vessels deep in the parenchyma, particularly in the right lobe, are difficult to approach via laparotomy. In these cases, after surgical bleeding is controlled, the liver is packed for temporary hemostasis. Angiography then identifies these injured blood vessels, and they can be occluded using a variety of endovascular techniques. The patient can then be unpacked and the liver reexamined when the patient is fully resuscitated.

Hepatic embolization, while effective, can be morbid. Dabbs et al. described a 40% incidence of major hepatic necrosis after embolization for liver injuries [24]. This often becomes symptomatic 2–5 days after embolization. While the symptoms are nonspecific, they often include elevations in liver function test, leukocytosis, and fever. CT scan shows air in areas of nonviable liver. While several treatment options exist, our approach is early hepatic lobectomy, which can be accomplished safely [25]. This is simply a more modern application of damage control.

Embolization can also be very effective to arrest pelvic hemorrhage either as a primary modality or combined with extraperitoneal pelvic packing. Patients often present in profound shock. Extraperitoneal pelvic packing is performed via either a low midline or Pfannenstiel incision. Care must be taken to avoid entering into the

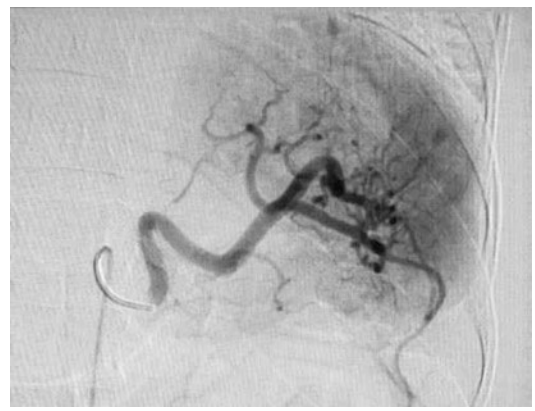

Fig. 12.2 Grade 3 splenic injury with pseudoaneurysms requiring proximal splenic artery embolization

abdominal cavity as the intact peritoneum is essential for effective pelvic packing. Packing can be followed by immediate angiographic embolization. The pelvis can then be unpacked and closed relatively early.

Angiographic embolization is also effective deep in the retroperitoneum following laparotomy for penetrating injury. The patient is generally explored first and injuries within the abdomen controlled. Controlling vessels deep in the pelvis, particularly in the hypogastric distribution, can be extremely difficult. Likewise, controlling distal branches of the lumbar arteries can be difficult. In these cases, the patient can be packed and those blood vessels visualized with angiography and occluded with embolization.

While endovascular damage control is most often used on the abdomen, it can be helpful in the chest as well. Direct surgical control of proximal intercostal arteries can be difficult. The narrow angle between the ribs and the spine makes exposing and controlling bleeding from the intercostal vessels in that location problematic. This is particularly true in the lower intercostal arteries, which are located deep in the sulcus behind the diaphragm. When these are discovered at the time of chest exploration, they can be temporarily controlled with packing and then definitively controlled with angiographic embolization.

12.1.2 Stent Grafting

Endovascular stent grafting as damage control can also be used as definitive therapy or even as a bridge to open repair. Stent grafts may be used temporarily or permanently to cover partial- or full-thickness injuries, particularly in major vascular injury that cannot be easily or rapidly accessed with surgical exposure. Placing a stent across a full-thickness injury may be extremely helpful in the physiologically devastated patient by rapidly decreasing blood loss and preventing coagulopathy.

The signature application of endovascular stent grafts is perhaps their use in the treatment of blunt thoracic aortic injury (BTAI). In the past, standard therapy for any patient with a diagnosed BTAI involved an emergent open treatment, usually via left posterior thoracotomy. The recognition that repair for these injuries can be deferred safely using anti-impulse therapy has allowed severely injured patients with BTAI to be treated using damage control principles, as these injuries no longer require emergency care. Thus, patients can be stabilized in the ICU and the aortic injury treated with a minimally invasive approach 1–2 days later.

The advent of thoracic endovascular aortic repair (TEVAR) utilizing endovascular stent grafts as definitive therapy has resulted in both improved morbidity and mortality among patients who survive to reach care after BTAI. Although appropriate patient selection remains paramount to success, the clear success of TEVAR has dramatically altered the standard of care for BTAI patients throughout the world.

In their landmark 2008 report, Demetriades and colleagues of the AAST BTAI study group [26] documented significant improvements in BTAI care associated with the transition from open to endovascular repair. In 193 patients with BTAI, TEVAR was associated with significantly decreased transfusion requirements and lower mortality compared to open repair. A more recent multi-institutional study is consistent in these original findings demonstrating a significantly lower PRBC requirement (mean 5.9 vs. 3.1 units, $p < 0.002$) in the first 24 h and a lower overall mortality (8.6% vs. 19.7%, $p = 0.021$) and aortic-related mortality (13.1% vs. 2.5%, $p = 0.003$) among patients treated with TEVAR compared to open repair [27].

The 2008 report of the AAST BTAI study group demonstrated a significant rate of TEVAR-related complications [26], as 18.4% patients had some form of stent graft-specific complication, most notably endoleak in 13.6%. The continued advancement of endovascular technologies since this report, including stents specific for aortic trauma, as well as operator experience, has decreased these adverse events following TEVAR [28] (Fig. 12.3). Paralysis, stroke, and left upper extremity ischemia with left subclavian artery coverage have significantly decreased over time [29–32], and grading systems to help delineate suitability for endovascular versus open approach as well as nonoperative management have evolved [33–35].

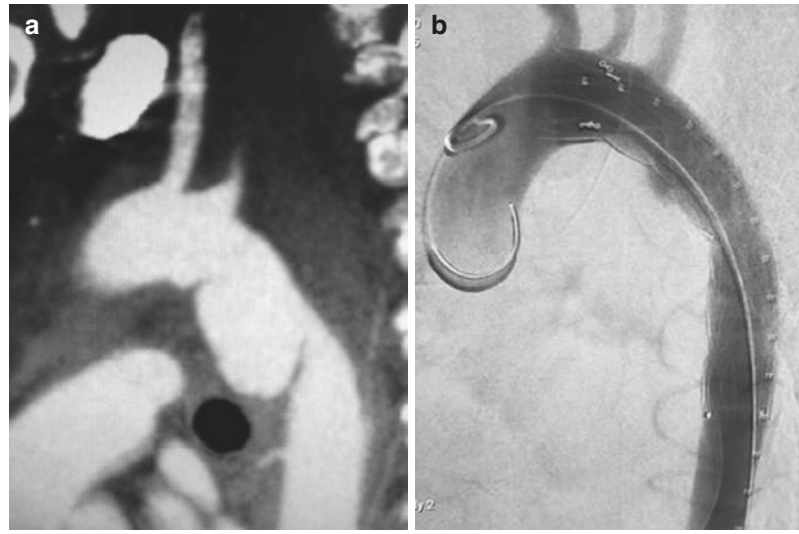

Fig. 12.3 Grade 3 blunt thoracic aortic injury treated with stent graft

The success of TEVAR for BTAI has paved the way for collaborative efforts between trauma surgeons and endovascular providers in exploring other anatomical sites where endovascular stent grafts may provide more ideal solutions than more traditional means of open vascular control and repair. In one recent study by Branco and colleagues [36], investigators reviewed the recorded rate of endovascular stent graft use among trauma patients from the American College of Surgeons National Trauma Databank. These researchers found that, compared to the 0.3% rate of utilization in 2002, there was a significant increase in endovascular technology utilization for trauma by 2010 (to 9.0%, $p < 0.001$). The most dramatic changes in utilization occurred among injured vessels located at sites associated with anatomically challenging exposures – including iliac and axillo-subclavian locations. When outcomes were compared between matched patients who underwent endovascular and open procedures, patients undergoing endovascular procedures had significantly lower in-hospital mortality (12.9% vs. 22.4%, $p < 0.001$) and decreased rates of sepsis after intervention (7.5% vs. 5.4%, $p = 0.025$). A variety of similar investigations have demonstrated similar findings reflective of increased utilization of endovascular approaches to hemorrhage control and blood vessel injury – associated with improved outcomes over historical controls [37]. As stent grafting is likely much quicker than a difficult open repair, this becomes attractive as part of damage control.

The vast majority of patients with free aortic rupture die at the scene. Most that survive to reach the hospital have ruptures that are at least partially contained. In poly-traumatized patients, other injuries are often more immediately life-threatening. In those patients, repair of the aorta must be deferred as they require operative therapy for life-threatening injuries in the cranium, abdomen, or pelvis. Some may even be too unstable to have imaging preoperatively.

In those cases, the diagnosis of BTAI can be made by intraoperative TEE. As the patient stabilizes, the aortic injury can be temporized by the use of anti-impulse therapy. The patient can then have their BTAI stented, ideally before they leave the operating room.

12.1.3 Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA)

The most common cause of each death after trauma remains hemorrhagic shock which can be compounded by ongoing coagulopathy. Early proximal control of the aorta can be a lifesaving

maneuver by reducing ongoing bleeding until the injury can be approached. An intra-aortic occlusive balloon placed through an open approach was first described for controlling major aortic hemorrhage in the Korean War [38]. Reports of use for control of bleeding during pelvic surgery [39, 40], hepatobiliary surgery [41], orthopedic surgery [42], postpartum hemorrhage [43], and repair of ruptured AAA [44–46] strongly support the use of the REBOA in serious intra-abdominal and/or retroperitoneal bleeding. Physiologic parameters such as serum lactate, pH, pCO2, and central, cerebral, and coronary perfusion in animal models of hemorrhagic shock have been shown to improve with REBOA use [47–50]. Early descriptions of its use for trauma were rare [51, 52] until recently, but as experience increased, indications began to include control of noncompressible torso hemorrhage after trauma in the abdomen and pelvis.

The use of REBOA to obtain proximal control at the level of the diaphragm prior to entering the abdomen may have a role in early control of traumatic intra-abdominal hemorrhage. Currently, indications for REBOA include persistent hypotension with hemorrhage below the diaphragm, including severe pelvic hemorrhage. Patients with abdominal injury, which arrive in extremis or in cardiac arrest, have traditionally been treated with left thoracotomy for aortic control and to facilitate open cardiac massage. However, in patients arriving in arrest from blunt mechanisms without evidence of severe great vessel injury, ED thoracotomy with open cardiac massage has been largely replaced in some centers by REBOA with closed chest compression, particularly in light of data suggesting that open chest compressions and closed chest compressions result in similar EtCO2 [53].

The appropriate location of balloon placement and inflation is determined by source of hemorrhage. The initial evaluation process, including chest and pelvic X-rays and FAST exam, helps localize potential site(s) of hemorrhage. For intra-abdominal hemorrhage, the balloon is inserted and inflated at the level of the diaphragm (Zone 1) (Fig. 12.4), while for pelvic hemorrhage with a negative chest X-ray and abdominal FAST exam, the balloon is inserted and inflated at the distal abdominal aorta (Zone 3) (Fig. 12.5). Contraindications for REBOA include suspected major cardiac, pulmonary, or vascular injury above the diaphragm.

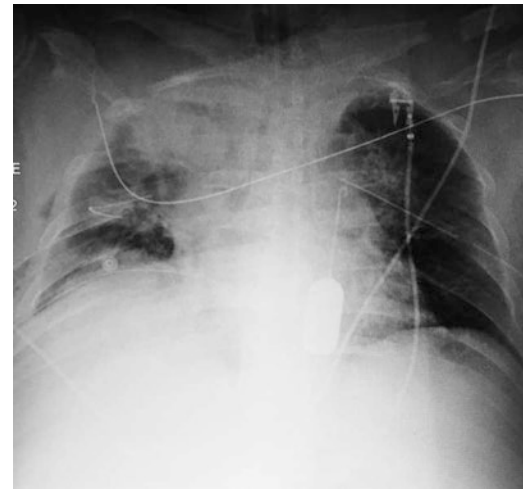

Fig. 12.4 ER-REBOA balloon occlusion in Zone 1

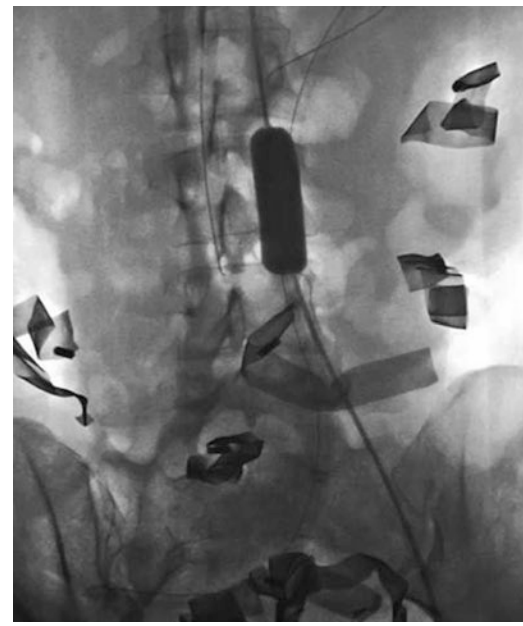

Fig. 12.5 ER-REBOA (Prytime Medical Inc.) balloon occlusion in Zone 3. The device was inserted in Zone 1 in the resuscitation area for severe intra-abdominal and pelvic hemorrhage, and the patient was taken to the hybrid OR for abdominal exploration and packing followed by angioembolization of the left hypogastric artery. The balloon was deflated and repositioned in Zone 3 after abdominal exploration, hemostasis, and packing

Placement can be performed in the resuscitation area using portable or digital X-ray or in the operating room under fluoroscopy. An immediate increase in SBP should occur if the technique is successful.

Several case reports and case series have documented significant increases in systolic blood pressure with REBOA [14, 54, 55]. Transportation to definitive treatment, or first to the CT scanner (Fig. 12.6), can then occur with the REBOA in place and the balloon inflated. The duration of aortic occlusion that can be safely tolerated varies considerably and may be impacted by patient physiology. Recent evidence suggests that patients may survive Zone 1 occlusion for up to 150 min [14], and our clinical experience suggests the REBOA can be left inflated safely even longer in Zone 3. Concern for visceral and extremity ischemia is warranted. As clear guidelines for this do not exist, the current recommendation is to deflate and remove the devices as rapidly as possible.

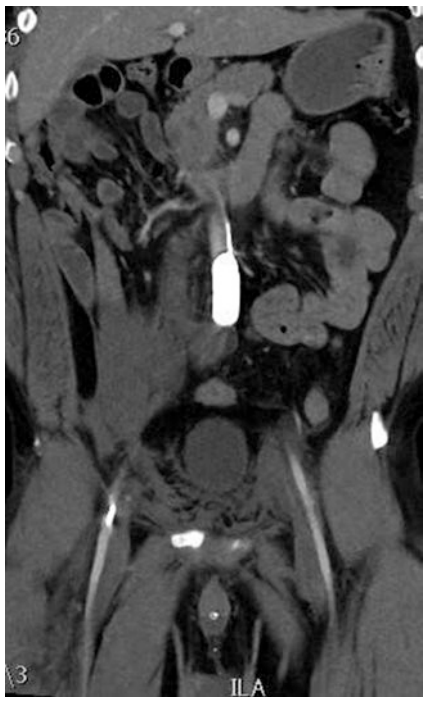

Fig. 12.6 ER-REBOA balloon occlusion in Zone 3. Contrast is visualized in the common femoral arteries due to collateral circulation, and the 7 French sheath visualized in the right common femoral artery

Access complications can be potentially limb-threatening, and a small case series in Japan describes a limb loss of 21% [56]. It is unclear whether the irreversible extremity ischemia in these patients was related to the procedure, initial injury, or both. These studies describe the experience with REBOA abroad; thus, it is not known whether these results can be applied in the USA where different technologies, protocols, indications, and providers' experiences and skills may apply.

Our earliest experience with REBOA in two high-volume trauma centers demonstrates REBOA to be a potentially lifesaving technique [55]. The devices used were standard "off-the-shelf" aortic occlusion balloons used for years by vascular surgeons to temporize hemorrhage from ruptured abdominal aneurysms. The procedure was modified for use in the resuscitation area using portable X-ray for device confirmation and taught to ACS in a 1-day formal training course. In this series, REBOA was performed rapidly by those ACS with no procedure-related complications and no deaths from hemorrhage.

A later and more comprehensive series from the same two institutions compared REBOA to ED thoracotomy (EDT) [57]. Survival and other outcomes were improved in the REBOA group, patients were less likely to die in the ED with REBOA, and death from hemorrhage only occurred in patients who were treated with EDT. The 18-month span of the study demonstrated a clear paradigm shift in treatment of patients arriving in arrest, with more REBOAs rather than EDT performed for the same indication. Most importantly, the study demonstrated no access complications, limb ischemia, or amputations.

Data from the American Association for Surgery in Trauma (AAST) Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) demonstrated no difference in aortic occlusion times between EDT and REBOA, no difference in survival, and no groin access or limb ischemia complications [58]. Arterial access is emerging as a rate-limiting step of REBOA: open groin cutdown is required in 50% of cases. Data from our institution also suggests that time

to aortic occlusion with REBOA is faster than with EDT only in cases where rapid percutaneous cannulation is achieved [59]. Improvements in technology will undoubtedly decrease time to aortic occlusion as users become more experienced and proficient at CFA access and REBOA placement. Recently, a new REBOA balloon catheter compatible with a seven-French introducer sheath has been FDA-approved (ER-REBOA, Prytime Medical Inc., San Antonio, TX). Arguably, this smaller size sheath may result in fewer access site complications.

REBOA can potentially revolutionize care when using damage control by rapidly controlling bleeding. Surgeons could potentially then safely spend more time providing definitive care during the index operation. In addition, the hemodynamic stability gained by the use of REBOA may allow the use of an expanded set of endovascular hemostatic options for hemorrhage control, particularly within difficult areas to access surgically. One could easily envision a patient with a high-grade liver injury being treated with REBOA as a bridge to definitive therapy with embolization, as opposed to an initial laparotomy followed by embolization.

12.1.3.1 Training and Credentialing

Privileges to perform REBOA are institution-dependent but, in our opinion, at minimum should require completion of a formal training course unless endovascular skills have previously been acquired. For ACS who have basic wire and catheter skills, REBOA requires a few more steps beyond simple arterial or venous line placement. These additional skills can be learned by brief, focused training [13, 60]. Gaining these skills has translated to safe and effective performance of REBOA in the clinical setting [55, 57, 58]. ACS are the only providers at the bedside initially caring for a patient who is exsanguinating, making them the only real option for these patients, rather than waiting for an interventionalist to come from other areas of the hospital or outside. This is simply too late to use REBOA for most patients.

12.1.3.2 REBOA in the ED and Pre-hospital Environments

The use of REBOA in the pre-hospital environment is occurring in the United Kingdom [61] and Japan [62]. Emergency physicians and medics as part of highly skilled mobile units are performing the procedure on the roadside for patients in extremis from noncompressible torso hemorrhage. REBOA may also have a role in the pre-hospital setting particularly in military or austere environments where delay of definitive hemostasis is unavoidable. Significant impediments to early use of REBOA include choosing the appropriate candidate and cannulating the common femoral artery (CFA). The former is even more challenging without traditional means of initial trauma evaluation such as abdominal ultrasound and pelvic X-rays. The latter is dependent on a variety of patient factors and notably the skillset of the provider, which must include the ability to cut down on the CFA when percutaneous cannulation is not possible, which currently occurs about half the time [58]. Regardless of the size of introducer sheath, access will remain a barrier to rapid aortic occlusion in those patients where percutaneous access is not quickly attainable. Prerequisite training for REBOA should include acquisition of ultrasound-guided cannulation skills if this is not within a provider's skillset.

12.1.3.3 Hybrid Trauma OR

A trauma hybrid operating and angiography suite expands the options for innovate care and can supplement traditional damage control technology. The table must perform fluoroscopy and all needed equipment, devices, and experienced staff must be available within minutes. The hybrid OR should ideally be located only a few feet from the trauma admitting area. This allows the team to perform multiple procedures almost simultaneously – for example, a laparotomy and an extremity angiogram – without change in location or time delay.

A fluoroscopy table, C-arm, monitors, lights, and anesthesia, surgical, and endovascular supplies are the basic materials needed to use the hybrid trauma suite. Additional instruments such as a TEE, rapid infuser, and intravascular

ultrasound, as well as cardiopulmonary bypass and dialysis pumps, may be needed. Advancements in C-arm technology have led to the production of a CT/C-arm hybrid machine, the Artis Zeego by Siemens, which combines high-resolution angiography with CT fluoroscopy. This "CT" is not as sensitive as the 64-slice helical CT but can give excellent quality three-dimensional views to supplement two-dimensional angiography. This allows the team to obtain CT images without travel and additional contrast use and acquire sensitive information such as endoleaks which can be poorly visualized on angiography. In addition, it can produce a head CT of adequate quality to rule out severe intracranial hemorrhage, particularly mass lesions that require evacuation. This may play a role in decisions made in the treatment algorithm of a multi-trauma patient (Fig. 12.7).

The technology for a hybrid OR is costly, approximately $3–9 million total for the room and 1.5–5 million for just the fluoroscopic equipment. In addition, the OR staff need to be comfortable with the acquisition, preparation, use, and billing of endovascular devices. A competent radiation technologist is essential to help provide images critical to decision-making and treatment. Radiation safety should be practiced with vigilance.

The use of the hybrid OR extends the use of damage control principles. Using damage control often requires multiple transports between the operating room, the ICU, and the angiography suite. The hybrid operating room allows all of these to happen in a single location when managing a poly-traumatized patient such as one with a pelvic fracture, aortic injury, and severe liver injury.

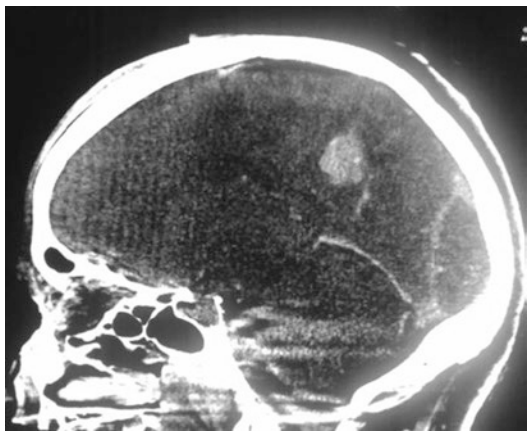

Fig. 12.7 CT scan of the head performed in the hybrid room with the Artis Zeego (Siemens)

12.1.4 Novel Hemostatic Tools

New techniques for hemorrhage control continue to be developed not only for hospital use but also in the pre-hospital setting. Direct pressure suffices to temporize hemorrhage from distal extremities. This is much more difficult if the bleeding is deep in the extremity or in a location that is difficult to compress. Recent experience has demonstrated the potential value of early tourniquet utilization following extremity injury in both military and civilian settings [63, 64].

One problem area that has received focus for the military has been "junctional hemorrhage," or bleeding from sites at the junction of the extremity or neck and the torso that are not amenable to traditional extremity tourniquet utilization. In order to provide a solution for temporary control at these locations, a variety of novel devices have been developed and studied [65]. One such device, the Combat Ready Clamp (CRoC), has been shown to provide for effective prolonged control of junctional hemorrhage sites in large animal models [66]. The device is presently approved for utilization in combat settings by the Department of Defense, but data on human utilization is lacking. Other devices such as the abdominal tourniquet, SAM junctional tourniquet, and Jett tourniquet have been utilized in military settings with anecdotal success.

Local hemostatic agents can also be quite helpful when hemorrhage cannot be controlled by direct pressure alone. These agents are divided into three categories. Synthetic sealant agents and agents that work by mechanical action are considered medical devices. Hemostatic agents that produce local clotting are considered drugs [67].

Sealant agents polymerize in an aqueous environment producing a mechanical seal. This is independent from the coagulation process. These

agents are useful in a number of settings, including sealing vascular anastomoses. Complications include systemic embolization, anastomotic stenosis if used in vascular surgery, and growth problems when used in children.

Agents that work by mechanical action require platelet activation to be useful. These agents create a three-dimensional structure that allows clot formulation. A number of these agents swell. Intraoperative blood salvage cannot be used when using any of these agents, and they may become adherent to neurologic structures causing neuropathic pain and/or neurologic dysfunction.

A number of these mechanical action agents were popularized during recent military conflicts. Several were manufactured utilizing crustacean shells. Unfortunately, the use of these requires removal which can be difficult. Exothermic reactions which can be severe occur with at least one product. In addition, antigenic reactions can occur. For the most part, these agents are now utilized only in the pre-hospital setting or the emergency department and are applied externally. Some trauma centers, however, continue to utilize these agents internally as part of packing when using damage control techniques. It is imperative to remember that these must be removed when the patient is reexplored. Some of the manufacturers are now making these agents radiopaque to be sure they are not inadvertently left in a patient.

Adhesive agents enhance the coagulation cascade, producing a fibrin-rich clot at the site of hemorrhage. The composition of these agents varies most utilizing fibrin with or without fibrinogen. These agents may be loaded onto a sponge to facilitate delivery of the hemostatic agent. The liquid fibrin glues are generally effective when treating mild hemorrhage or diffuse ooze. The fibrinogen/thrombin patches are used for more severe hemorrhage, particularly with active discrete points of hemorrhage, as applying the patch with some pressure prevents it being washed away. These agents can be utilized to seal leaks from structures such as the lung.

The skilled clinician will utilize these local hemostatic agents for their individual properties in order to achieve the greatest efficacy. Figure 12.1 depicts an algorithm for their use.

12.1.4.1 Intra-abdominal Foam

Another novel technique for control of noncompressible hemorrhage that has been proposed has been the use of self-expanding foam. This polyurethane-based expandable agent can be introduced into the abdominal cavity via a percutaneous delivery system of a patient suffering significant intra-abdominal bleeding. On the battlefield, where distance from surgical care may be quite large and pre-hospital times extensive, this could be quite helpful. If delivered effectively into the abdominal cavity of a bleeding patient, this self-expanding foam can assist in the tamponade of bleeding sources and keep an injured soldier alive until such time as they can be delivered to a facility capable of providing subsequent laparotomy. The foam can then be removed upon entry into the abdominal cavity and the operative control of bleeding sources accomplished.

Several groups have provided preclinical data that support the potential of intra-abdominal polyurethane foam as an adjunct for pre-hospital treatment of abdominal bleeding [68–71]. While this animal and cadaver data appears to demonstrate the potential of this adjunct, there has been no clinical utilization to date, and this intervention has only been proposed for the most austere of military settings. Significant additional study is required to define the optimal patient selection and utilization of this potential therapy.

Additional agents such as ClotFoam, a fibrin-based hemostatic foam, and XStat, a nonabsorbable, expandable hemostatic sponge, show promise in early investigation through limited clinical trials.

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Part III

Phases of Damage Control

Prehospital Damage Control

13

Eileen M. Bulger

13.1 Recognition of Hemorrhagic Shock

Patients who have sustained blunt or penetrating torso trauma are at risk for significant bleeding and should be assessed for signs and symptoms consistent with hemorrhagic shock. Early signs of shock can be very subtle and may include diaphoresis, pallor, altered mental status, and tachycardia. While traditional triage rules have focused on a systolic blood pressure of ≤90 mmHg as evidence of shock, hypotension is generally a late sign and suggestive of severe hemorrhage.

13.1.1 Shock Index

Several authors have assessed the utility of using the shock index (SI), which is defined as the ratio of heart rate divided by systolic blood pressure, as a predictor of hemorrhagic shock. A shock index ≥0.9 after injury has been associated with hemorrhagic shock. A recent meta-analysis assessed the ability of the SI to predict critical bleeding which was defined as at least 4 units of red blood cells in the first 24 h [1]. This paper reviewed five studies, one of which was in the prehospital setting and the others early after hospital arrival. All demonstrated a relationship between an increase in shock index and the need for transfusion. The prehospital study reported that an SI ≥1.0 after at least 1 l of fluid had a specificity of 90.5% and a sensitivity of 47.9% for predicting the need for >5u blood in the first 4 h [2].

A limitation of the shock index is that it relies on normal physiologic response to hemorrhage and thus may be impacted by medications such as beta-blockers that will suppress tachycardia. In addition, elderly patients who have a baseline hypertension may not manifest as significant a decline in blood pressure as a younger patient. Some authors have suggested that a systolic blood pressure <110 mmHg may be more predictive in an elderly trauma population [3]. One author suggested using Age x SI [4]. When applied to all patients, this score did not perform well, but when restricted to those over age 55 years, the area under the receiver operating curve increased significantly. A recent study from the National Trauma Databank review the relationship between shock index and outcome for patients over age 65 years [5]. This study demonstrated that patients with an SI ≥1.0 were significantly more likely to require a blood transfusion, require an exploratory laparotomy, and develop in-hospital complications. SI ≥1.0 was also a significant predictor of in-hospital mortality.

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13.1.2 Prehospital Lactate

Due to the limitations of traditional vital signs, several authors have assessed point of care lactate testing as a marker of shock in the prehospital setting [6–9]. Lactate is a marker of metabolic acidosis, which develops with inadequate tissue perfusion. A number of studies in the hospital have demonstrated an association with elevated lactate or a high base deficit and hemorrhagic shock [10–13]. A recent multicenter trial demonstrated that among a trauma population with a systolic blood pressure between 70 and 100 mmHG, a prehospital lactate >2.5 mmol/L was a better predictor of the need for resuscitative care than SBP or SI [7]. Resuscitative care was defined as any of the following within 6 h of emergency department arrival: blood transfusion of 5u or greater, operative or angiographic intervention for hemorrhage control, or death.

13.1.3 Prehospital ABC Score

The ABC score is a scoring system that has been used in the emergency department to predict the need for massive transfusion in trauma patients (Table 13.1) [14]. The score assigned points based on SBP<90 mmHg, HR>120 beats/min, evidence of penetrating torso trauma, and free fluid seen on a focused abdominal sonogram for trauma (FAST). A score of 2 or greater on ED arrival was associated with a 40% rate of massive transfusion. Efforts to allow this score to be used in the prehospital setting have focused on education of aeromedical crews to perform ultrasound to assess for intra-abdominal fluid. A recent study demonstrated that the sensitivity and specificity for hemoperitoneum in this setting were 46% and 94% [15]. The ABC score has been used in some helicopter EMS services as an indication for prehospital blood transfusion. As the technology evolves and training paradigms are established, this approach may become more feasible.

Table 13.1 ABC score to predict massive transfusion

Variable	Points
Penetrating mechanism	1
ED SBP of 90 mmHg or less	1
ED HR 120beats/min or greater	1
Positive FAST exam	1

FAST focused abdominal sonogram for trauma

13.2 Damage Control Management

13.2.1 Hemorrhage Control Procedures

Traditional management of the severely injured patients has focused on an ABC approach where airway, breathing, and circulation are assessed and managed in that order. While this is still the best approach for the majority of patients, it has been recognized that in certain circumstances, where there is obvious external bleeding, a CAB approach may be preferred. This approach has been endorsed by the US military in the combat casualty care guidelines in response to the significant number of extremity injuries associated with improvised explosive devices. This approach has also been supported by the Tactical Emergency Care Committee for civilian events where there still may be an imminent threat and placement of a tourniquet can be accomplished much more quickly than airway management.

The American College of Surgeons Committee on Trauma recently published an evidence-based guideline for management of external hemorrhage for civilian EMS providers [16]. For extremity hemorrhage, this document supports direct pressure with a hemostatic dressing (if available) as the first line of therapy with placement of a tourniquet whenever direct pressure is either ineffective or not practical. For junctional hemorrhage, wound packing with a hemostatic dressing and direct pressure are advocated. There are a number of junctional tourniquets now on the market, but there is insufficient evidence to promote their use.

In the civilian community, it is important to remember that the majority of life-threatening bleeding in trauma patients is internal bleeding in the chest, abdomen, or pelvis, and thus rapid

triage and transport to a trauma center capable of achieving hemorrhage control in the operating room and/or angiography suite is vital. For patient with unstable pelvic fracture, there is data to support use of a sheet or pelvic binder to help close the volume of the pelvis and thus potentially reduce venous bleeding. Many patients with unstable pelvic fractures also have arterial bleeding that will not be controlled with pelvic binding alone. Thus, these patients need rapid transport to the highest-level trauma center available.

13.2.2 Airway Management

Patients with hemorrhagic shock and intact airway who are not at risk for imminent decline should be given supplemental oxygen and transported without delay. Patients with airway obstruction, significant airway edema, or depressed level of consciousness that suggest they cannot protect their airway need to be supported with bag mask ventilation and ideally placement of a definitive airway based on the skill of the EMS provider. Prehospital intubation has been controversial in the literature with wide variation in intubation success rates across the USA [17]. This is in large part due to variability in the training and skill maintenance approaches among prehospital agencies. The ability to use neuromuscular blocking agents in the field also significantly improves intubation success rates [18, 19]. There are several rescue devices also available on the market for patients with difficult airway management.

Previous studies of prehospital intubation have focused on patients with suspected traumatic brain injury based on a prehospital Glasgow Coma Score (GCS) ≤8. A study by Davis et al. of rapid sequence intubation in the prehospital setting suggested worse outcome compared to historical controls [20]. This study was limited in that the cohorts could not be matched based on prehospital GCS and so Head AIS score was used instead. Subsequent studies by this research group demonstrated that intubation itself may not have been the problem, but rather high rates of hyperventilation and preintubation hypoxia may have impacted outcome [21, 22]. A subsequent review of patients intubated by aeromedical services who had lower rate of hyperventilation demonstrated improved outcome [23]. A randomized controlled trial of prehospital intubation for suspected TBI with low GCS score also demonstrated improved neurologic outcome in the intubated group [24].

Thus, for patients with low GCS or those requiring emergent airway protection, prehospital intubation is recommended provided that EMS providers have appropriate training and ongoing skill maintenance [25]. For the suspected TBI patient, patients should be preoxygenated if possible to avoid hypoxia, and ventilation should be controlled to prevent hypocarbia, which may cause cerebral vasoconstriction. End tidal CO_2 monitoring has been promoted as a method to monitor appropriate ventilation in the prehospital setting; however, studies have shown that end tidal CO_2 is not a reliable marker of $PaCO_2$ for patients with hemorrhagic shock due to decreased pulmonary perfusion [26]. A low end tidal CO_2 in this setting is actually more predictive of compensated shock than of excessive ventilation. Excessive ventilation which can increase intrathoracic pressure has also been shown to be detrimental in animal models of severe hemorrhagic shock due to the reduction in venous return [27, 28].

13.2.3 Needle Thoracentesis

Needle thoracentesis is indicated for patients with suspected tension pneumothorax and hemodynamic compromise. This may occur in the setting of penetrating or blunt chest trauma. Needle thoracentesis can be accomplished at the second intercostal space midclaviclular line or fifth intercostal space, midaxillary line. Longer needles may be needed for obese patients to ensure penetration into the chest cavity. There have been few studies of the practical application of needle thoracentesis. One study of its use in an urban EMS system did not identify any major safety

concerns and reported four patients with unexpected survival that appeared to be at least in part related to rapid chest decompression [29].

13.2.4 Fluid Resuscitation

It has been recognized for many years that artificially increasing the blood pressure before hemorrhage control has been achieved can result in increased bleeding. A landmark study by Bickel et al. randomized patients with penetrating torso injury in an urban EMS system to no fluid administration prior to hemorrhage control vs. wide open crystalloid resuscitation [30]. In this study, the delayed resuscitation group had a reduction in 24 h mortality. This led to the concept of hypotensive resuscitation, which suggests that for patients with penetrating trauma, a SBP of 80 mmHg or a palpable radial pulse is adequate and crystalloid resuscitation should be limited until hemorrhage control is achieved. For patients with an SBP, <80 mmHg or absence of a radial pulse than moderate fluid resuscitation should be administered with a goal of 80 mmHg. Extension of this approach to blunt trauma patients is controversial as these patients frequently have an associated TBI and prehospital hypotension has been associated with worse outcome for TBI patients [31].

A recent pilot study of hypotensive resuscitation in blunt trauma patients did not show impaired outcome, but importantly excluded any patient with suspected severe TBI [32]. Thus, for blunt trauma patients, we advocate a controlled fluid resuscitation strategy which targets a blood pressure that will not put them at risk for impaired cerebral perfusion, but avoids excessive resuscitation when hypertensive. The other population of patients for whom traditional blood pressure goals may not be adequate is the geriatric patient with baseline hypertension.

The next question to address is what fluid is ideal for prehosptial resuscitation. Numerous studies have failed to show any benefit of colloids over crystalloids, and two recent multicenter, randomized controlled trials of prehospital hypertonic saline also failed to demonstrate any benefit for trauma patients with hypovolemic shock or severe TBI [33, 34].

In the emergency department, we have focused on more rapid shift from crystalloid to blood product resuscitation. A balanced blood product resuscitation with early administration of platelets and plasma in conjunction with red blood cells has been associated with improved outcome in a number of studies to address the early development of trauma-induced coagulopathy [35–37].

These data have led investigators to consider options for blood and plasma resuscitation to be initiated in the prehospital setting. Several systems have deployed packed red blood cells and thawed plasma on aeromedical services, and there is a clinical trial that is currently enrolling patients in an urban ground EMS system with a plasma resuscitation strategy. A recent report by Holcomb et al. demonstrated that patients receiving prehospital blood products had evidence of improved acidosis on arrival and a reduction in subsequent blood product usage, and while there was no difference in 24 h mortality, there was a reduction in 6 h mortality among the most critically injured patients [38]. There are ongoing efforts in the laboratory to develop freeze-dried and lyophilized plasma products that will be logistically more feasible for use in the prehospital environment [39, 40].

13.2.5 Tranexamic Acid

Tranexamic acid (TXA) is an antifibrinolytic drug, which blocks the conversion of plasminogen to plasmin and thus inhibits the breakdown of clot. Fibrinolysis is one component of the early coagulopathy that can develop in severely injured patients with active hemorrhage. The CRASH-2 study was a multicenter trial that randomized injured patients with concern for bleeding to receive TXA vs. placebo within 8 h of injury [41]. This study enrolled over 20,000 patients and demonstrated difference in survival at 14.5% for the TXA group vs. 16% for placebo. A subgroup analysis suggested better outcome if the

drug was given within 3 h after injury and no benefit beyond 3 h [42].

This finding has led many to suggest that perhaps earlier is better and thus the drug should be started in the prehospital setting. The primary concern with this approach is establishing the appropriate patient selection criteria to avoid exposure of the drug to patients who are not likely to benefit. While there were no major safety concerns raised in the CRASH-2 study, several authors have raised concern about potential safety issues that may not have yet been adequately identified [43]. There are several prehospital clinical trials currently enrolling patients that will address these issues. For agencies that have decided to implement TXA protocols, a guidance document emphasizing the importance of coordination with the regional trauma system has been published by the American College of Surgeons Committee on Trauma in conjunction with the National Association of EMS Physicians and the American College of Emergency Physicians [44].

13.2.6 Avoiding Hypothermia

Hypothermia in injured patients has been associated with increased mortality [45]. This is due in part to the effects of hypothermia on the exacerbation of coagulopathy. Studies have shown that the majority of trauma patients that arrive in the ED are hypothermic despite normal ambient temperatures [46]. Patients are often exposed to the elements, clothing has been removed to provide medical care, and shock which leads to peripheral vasoconstriction can lead to additional heat loss. It is important to recognize this risk and take steps to avoid additional heat loss by removal of wet clothing, covering the patient with blankets, and administering warm IV fluids whenever possible. Blood products in particular are generally stored at 4C and thus need to be warmed when administered. For prehospital administration, there are in-line warmers that can be attached to the IV tubing.

13.2.7 Triage and Rapid Transport

Current triage recommendations are based on the CDC Field Triage Guidelines which were last updated in 2011 [47] (Fig. 13.1). These guidelines recommend transport to the highest-level trauma center available for patients at greatest risk of needing immediate intervention. These include patients with hemodynamic instability (SBP <90 mmg), altered mental status (GCS<14), or respiratory distress (RR <10 or >29 breaths per minute) and patients with signs of significant anatomic injury. These patients are at risk for internal hemorrhage and thus should be considered as potential damage control patients. Prehospital procedures should be limited to the lifesaving interventions as discussed above with a focus on minimizing scene time. Recent data has demonstrated that the CDC field triage criteria do not perform as well when assessing elderly trauma patients. Thus, elderly patients require a high index of suspicion for occult hypoperfusion, even with what appears to be a normal blood pressure, and significant injury despite a minor mechanism event.

In order to minimize scene times, recent emphasis has been placed on improved coordination between EMS and law enforcement to optimize EMS access to the scene for major shooting events with rapid patient evacuation. A series of conferences known as the Hartford consensus conferences have laid out strategies to enhance hemorrhage control education for law enforcement, encourage joint training between EMS and law enforcement, and body armor protection for EMS providers to facilitate access to a warm zone more rapidly [48–51].

There is controversy regarding the optimal use of aeromedical transport to facilitate triage to a higher-level trauma facility, while not extending scene time [52]. In an urban setting when transport time to major centers is short, then delaying ground transport to await helicopter arrival is likely not beneficial. However, there are a number of studies that have demonstrated worse outcome for patients injured in a rural setting, and delays in access to high-level trauma center may play a role. Two recent studies support the use of aeromedical transport to facilitate access to major

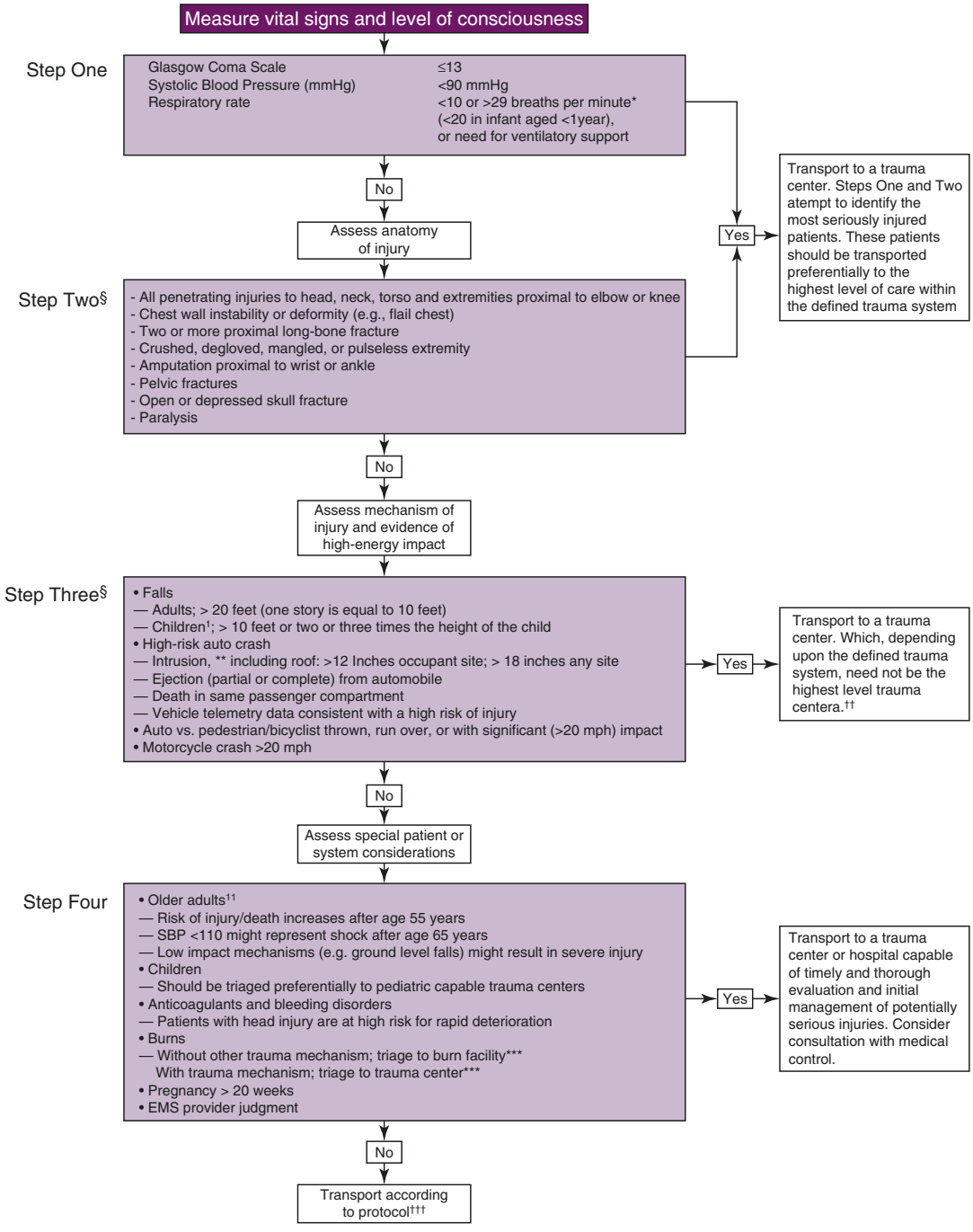

Fig. 13.1 CDC field trauma triage algorithm: 2011 (From CDC web site: www.cdc.gov/mmwr/preview/mmwrhtml/rr6101a1.htm)

trauma centers. Brown et al. utilized the National Trauma Databank to compare patients transported by helicopter vs. ground transport. While patients transported by helicopter were more severely injured, had longer transport times, and required more hospital resources, they were more likely to survive and be discharged home after their injury [53]. Another study focused on patients with evidence of shock or traumatic brain injury in the prehospital setting and again demonstrated higher injury severity, longer transport times, and more prehospital procedures among those transported by air but no difference in survival based on mode of transport [54].

13.3 Summary

The principles of damage control management and resuscitation should extend into the prehospital setting and thus influence decision-making regarding lifesaving interventions, fluid and blood product resuscitation, and rapid triage and transport to the most appropriate trauma center. Early identification of the patient in hemorrhagic shock remains a challenge, and thus a high index of suspicion remains a priority. Further study is needed to provide tools to aid in identification of high-risk patients.

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Phase I: Abbreviated Surgery (General Surgery)

14

Brian P. Smith and Patrick M. Reilly

A routine approach to accessing cavities, packing the operative field, and establishing adequate retraction and exposure is paramount to setting the tone of the operation and optimizing chances of success. Furthermore, the team must remain mindful that multiple injuries will evolve in parallel and frequent re-triage, and prioritization of intervention is necessary for patient survival. On occasion, multiple teams of surgeons will need to operate in different cavities simultaneously. The importance of efficient and cooperative teamwork cannot be understated.

14.1 Head

Injuries in the head can manifest in several different ways. They range from innocuous injuries with no outward signs of trauma to devastating traumatic brain injury (TBI). Among all injured patients, TBI remains the leading cause of death [1–3]. And among patients with many specific injury complexes such as pelvic fractures or solid organ injuries, TBI remains a significant risk factor for death [4, 5]. Because of the relative frequency with which they occur in combination with other injuries, the trauma surgeon must be able to recognize and at times temporize head injuries that threaten each patient's life.

14.1.1 Traumatic Brain Injury

Detailed management of TBI can be found elsewhere within this text. However, the trauma surgeon must be able to recognize basic signs of injuries that are associated with TBI. Loss of consciousness, depressed mental status, and neurologic deficits (particularly lateralizing signs) are common indicators of TBI. If time and physiology permit, computed tomography of the brain is an excellent test for the detection of TBI. For patients with severe injuries that necessitate immediate operation, the surgeon is faced with challenging decisions. Options range from foregoing central nervous system imaging until definitive management of other life-threatening injuries is obtained to prioritizing medical imaging even in the face of hemodynamic instability [6, 7]. The latter approach has gained some favor as CT scanning technology evolves, and images can be obtained faster and with better proximity to trauma bays and operating theaters.

Alternatively, the brain can be triaged during emergency surgery of another cavity with

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portable CT technology if available or insertion of an intracranial pressure monitor. Empiric decompressive craniectomy during or temporally related to other surgeries has also been described when lateralizing symptoms are observed. We recommend involvement of a neurosurgical specialist if available to help with this decision-making.

14.1.2 Scalp and Soft Tissue Injuries

Lacerations of the face and skull can prove problematic for the trauma surgeon who is trying to care for other injuries because of the large volume of blood loss that can occur from these sites. This is particularly true for patients who are anticoagulated. The first line of therapy should be gently direct pressure over the source of bleeding with a gloved hand. Basic absorptive dressings should be used and can be supplemented with hemostatic dressings (such as any kaolin- or chitosan-based product). If basic maneuvers fail to stop bleeding, temporizing sutures (so-called whip stitches) can be utilized. Raney clips also provide rapid and effective hemorrhage control for some lacerations. In such cases, we prefer an automated reloading clip applier rather than the traditional manual loading Raney clip forceps. Regardless of the hemorrhage control technique, the surgeon must remember to revisit the wound and perform any necessary debridement and irrigation prior to definitive closure once other sources of hemorrhage have been addressed.

14.1.3 Nasopharyngeal and Oropharyngeal Bleeding

The primary maneuver for dealing with bleeding from the mouth or nose is the protection of the airway. Once the lungs are protected with an endotracheal tube or surgical airway, the source of bleeding can be addressed. Numerous techniques have been described for emergency control of pharyngeal bleeding. Most of these involve tamponade of bleeding spaces. The nares can be packed with gauze or commercially available nasal tampons. Posterior packing can also be achieved with inflatable nasal balloon catheters. In the absence of that device, a large Foley urinary catheter can be inserted into a nostril, the balloon expanded, and gentle traction applied to hold the balloon in place. Balloons can also be directly inserted into injury tracts to obliterate hemorrhagic spaces. Mortality rates of patients that undergo emergency control of facial hemorrhage are high (approaching 90%) due largely to massive hemorrhage and concomitant injuries [8]. Catheter angiography and embolization are useful adjuncts for definitive control of facial and pharyngeal bleeding [9, 10].

14.2 Neck

The confines of the neck, combined with the large number of organ systems that exist in and pass through it, make it a challenging field in which to operate. A slight shoulder role will help to gently extend the neck, and the head should not be placed too much to either side as injuries generally traverse from one side to the other.

14.2.1 Vascular

Injuries to the carotid and vertebral vessels account for 26.7% of all injured blood vessels in patients with vascular injuries [11]. The optimal incision for hemorrhage control in the neck is unknown as injuries can cross multiple zone and sides. However, the standard longitudinal incision along the anterior border of the sternocleidomastoid does provide access to a majority of the length of most vessels. Additionally, it can be extended onto the sternum should sternotomy be necessary.

We favor mandatory exploration of all carotid sheath hematomas in the absence of preoperative imaging. Bleeding that arises from the carotid arteries is controlled with proximal and distal occlusion as with any other vascular surgery. Patients presenting with isolated carotid injuries and stable hemodynamics should undergo pri-

mary repair of the injury [12]. If there are other compelling sources of hemorrhage, the carotid should be shunted with an appropriately sized conduit until such time that repair is possible. For isolated carotid arterial injuries, temporary shunting does not appear to offer an advantage [13]. We recommend irrigation with heparinized saline, passage of Fogarty balloon catheters distally, as well as forward and back bleeding prior to insertion of shunts and definitive repairs. In rare cases the carotid vessels can be ligated in order to expedite surgery in a patient who is dying from other injuries. The risk of persistent neurologic defect after ligation is hard to estimate because of frequently associated injuries; however, many small series report a stroke rate around 50% [13, 14].

Bleeding that persists despite control and exposure of the carotid system most likely arises from the vertebral artery. These exposures can be particularly challenging because of the deep location of the vertebrals in the neck and their confinements in the osseous canal. This makes proximal and distal control particularly challenging. These wounds should be packed with bone wax in the area of injury. Definitive proximal control can be obtained by dividing the manubrial origins of the sternocleidomastoid and sternohyoid muscles and isolating the vertebrals after they arise from the subclavian arteries. Catheter angiography and embolization of these injuries are also a useful approach [15].

Damage control surgery in the neck leaves little room for repair of venous injuries. These wounds should be ligated to facilitate repair of other life-threatening problems.

14.2.2 Trachea

As with oral and pharyngeal injuries, tracheal injuries mandate protection of the airway with an endotracheal tube of a surgically created cricothyroidotomy or tracheostomy. Occasionally, the traumatic injury itself will create an opening through which the airway can be accessed. Generally the airway should be secured before the patient leaves the resuscitation area. In that regards, tracheal trauma will rarely impact the surgeon's decision to abbreviate the operation in a damage control fashion. Tracheal injuries encountered during neck exploration should be repaired if the patient's physiology permits. Primary anastomosis with 4-0 oiled vicryl or absorbable monofilament suture is preferred [16]. Repairs should be protected with vascularized tissue such as strap muscle. There is little data to support or refute the drainage of tracheal repairs in the neck. In the context of an abbreviated surgery for trauma, temporary drainage should be strongly considered.

14.2.3 Esophagus

Injuries to the esophagus are rare, and slightly less than half of all esophageal injuries occur in the area of the cervical esophagus [17]. Several algorithms have been described regarding the evaluation and management of suspected cervical esophageal injuries [17–21]. Clinical indicators of injury, or imaging that demonstrates trajectory involving or adjacent to the esophagus, generally mandate surgical exploration.

Patients brought directly to the operating room because of instability or associated injuries should be formally explored through an anterior sternocleidomastoid, collar incision, or bilateral sternocleidomastoid incision if necessary. The area of the esophagus in question should be fully mobilized and explored. Care must be taken to avoid injury to the recurrent laryngeal nerves. If the surgeon is unable to identify perforation in areas suspicious for injury, adjuncts such as esophagoscopy or instillation of methylene blue might be helpful. If patient physiology precludes prolonged exploration, the area should be well drained, and additional testing (such endoscopy or contrast imaging) should be conducted as soon as other life-threatening problems have been resolved.

Nondestructive injuries should be debrided to healthy tissue and closed. There are no

randomized controlled trials comparing one- to two-layered closure in trauma. We do recommend that closed tissue should be healthy and tension-free [22]. Injuries that result in obvious perforation should be closed in the same fashion. Special consideration should be given to perforations in order to exclude a second adjacent injury resulting from a "through-and-through" trajectory. The closure of esophageal injuries over a nasoenteric tube for gastric drainage/feeding and protection of the repair with vascularized tissue, such as an infrahyoid muscle, are generally recommended [17].

Destructive injuries such as complete transection can be more challenging to manage. This is particularly true if the distal esophagus has retracted into the mediastinum or is difficult to find because of concomitant soft tissue injury. If the surgeon is unable to identify the distal esophagus, we recommend laparotomy, gastrostomy, and retrograde cannulation of the esophagus. This aids identification of the distal cervical esophagus, which can be closed as a stump. The stomach opening can then be converted to a gastrostomy tube for drainage and/or feeding. The proximal esophagus should be matured as an end cervical esophagostomy.

14.3 Thorax

Thoracic surgery for the trauma patient generally takes the form of thoracotomy or sternotomy, and deciding which incision to make is sometimes challenging (Fig. 14.1). The trauma surgeon should have a good understanding of which exposures each incision has to offer, as well as slight modifications that can be used to facilitate certain procedures. For instance, the so-called taxi hailing position (supine with single hemithorax 30° elevation and ipsilateral arm overhead adduction) allows adequate exposure for thoracotomy while preserving the abdomen for laparotomy and the contralateral hemithorax for bilateral thoracotomy if needed.

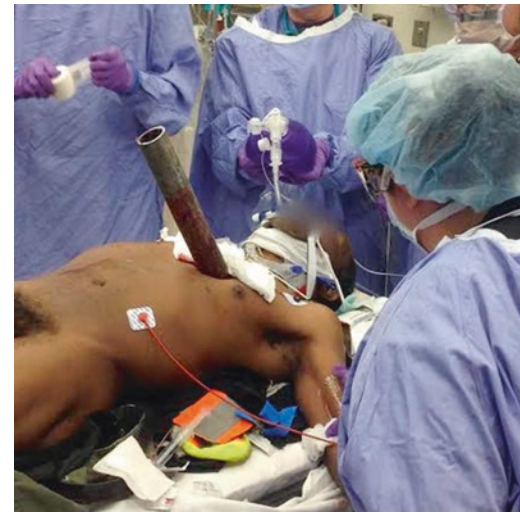

Fig. 14.1 The surgeon must know the advantages and limitations of various thoracic surgical exposures

14.3.1 Emergency Thoracotomy

Emergency department (ED) thoracotomy has indications for injuries beyond isolated thoracic wounds. However, it is most commonly performed on patients with thoracic injuries. Absolute indications and contraindications to the procedure are not well agreed upon [23–26]. Adjuncts to the history and physical exam, such as cardiac ultrasound, have been suggested as tools to aid clinical decision-making [27]. Once the decision is made, the surgeon should proceed with speed and proficiency. We recommend absolution of the surgeon from team leader responsibilities regarding the medical part of the resuscitation (if possible) when the surgeon participates in the thoracotomy. ED thoracotomy should not delay other lifesaving maneuvers such as decompression of the right chest with thoracotomy, control airway for ventilation, and intravenous or intraosseous access for volume resuscitation.

The procedure starts with a rapid prep of the chest wall. The left arm should be raised above the head. The skin is incised at the inframammary cleft in the area of the fourth or fifth interspace. The incision should extend from the sternum through serratus anterior to latissimus dorsi. The

incision should then be deepened to the intercostals. Next, the intercostals should be divided. We prefer a large set of curved Mayo scissors to ride the top edge of the rib anteriorly and posteriorly. A self-retaining rib spreader (such as a Finochietto retractor) should be placed with the spreading bar (or "U bar") toward the stretcher. This technique preserves access to the sternum in the case that the procedure be converted to a bilateral anterior thoracotomy.

At this time, it is often helpful to direct the airway team to hold ventilation. This affords the surgeon better access to the pericardium and descending thoracic aorta. The pericardium should be incised anterior to the left phrenic nerve from the apex to the proximal aorta. Hemopericardium should be evacuated and the heart delivered. A quick inspection of the heart will reveal any injuries. Once the source of bleeding has been identified, bag ventilation should be resumed. We advise primary control of cardiac or proximal major vascular injuries (aortic root, cava, pulmonary artery, etc.), prior to clamping the descending thoracic aorta as the increased afterload will compound bleeding from these vessels. Oftentimes, major hemorrhage can be controlled with a gloved finger as supplies and instruments are gathered for more definitive control. A surgical skin stapler is a rapid and effective way to decrease hemorrhage from cardiac injuries. This technique is usually supplemented with pledgeted suture repair. We favor leaving the original staples in place once the repair is complete. Small vascular clamps are also effective in controlling atrial injuries prior to definitive closure. Special consideration should be given to injuries in proximity to coronary arteries. These wounds should be closed with pledgeted horizontal mattress sutures outside of the adjacent coronary artery [28].

Patients remaining in extremis after central bleeding has been temporized should undergo cross-clamping of the thoracic aorta. This is done by retracting the left lung cephalad. The aorta can be palpated and manually occluded against the vertebral bodies, and a straight vascular clamp is obtained. Next the parietal pleura is opened anteriorly and posteriorly in a perpendicular fashion at the level of an intervertebral disk. This avoids avulsion of the spinal arteries originating from the mid vertebral level. The vascular clamp is then applied with care, taken to avoid injuring the esophagus. Timing of aortic cross-clamping should be noted, and efforts to remove or replace the clamp in a more favorable position should be expedited.

Life-threatened hemorrhage from the lung can be temporized with manual direct pressure. This can be supplanted with a Duval lung clamp. We have not had success with more advanced lung procedures in the resuscitation bay and believe that suture ligation of lung bleeding or tractotomy should be performed in the operating room with better lighting, exposure, and instrumentation. Bleeding that does not stop with direct pressure should be temporized with pulmonary hilar control. This is generally easier to perform from the opposite side, with one hand passing cephalad and one passing caudal to the lung hilum. The nondominant hand can manually occlude the hilum and guide the insertion of a clamp. Alternatively, the technique preferred by this group is manual occlusion and placement of a Rumel tourniquet using umbilical tape. A large angled or curved Debakey clamp can be used to pass the tape posterior while manual occlusion is maintained. Once in place, this technique allows the surgeon to operate with both hands, affords a lower profile than a large clamp for better visualization, and might generate less hilum trauma than a mechanical clamp.

Finally, it is important to mention that in the absence of definitive knowledge of a normal right pleural space, the right chest should be triaged in some fashion. Finger or tube thoracotomy is a commonly used technique. Alternatively, the surgeon can also open the right pleural space through the left chest by dissecting under the sternum across the anterior precordial mediastinum. If no assistant is present, the surgeon should give strong consideration to bilateral anterior thoracotomy. Some surgeons prefer this incision for ED thoracotomy regardless of mechanism or assistant availability [29]. Regardless of incision,

the surgeon should be quick to move the patient to a more controlled operating space once hemorrhage is temporized.

14.3.2 Heart

Traumatic injuries in the heart are rarely managed with abbreviated surgery. Temporizing maneuvers might be employed to control hemorrhage until the patient can be moved to a more favorable operating environment (as mentioned previously), better instrumentation is obtained, or expanded surgical expertise is consulted. Generally speaking, however, most traumatic cardiac injuries are definitively repaired at the index operation. Most cardiac injuries are more easily approached through a median sternotomy. If patient physiology permits and the suspicion for posterior mediastinal injuries is low, median sternotomy is the incision of choice [29]. If there is concern for pericardial tamponade, we recommend prepping and draping the patient awake and gowning and surgical preparation prior to the induction of anesthesia. This allows the surgical team to rapidly enter the chest if tamponade physiology develops. The patient in extremis should be approached through a left anterolateral (or bilateral) thoracotomy.

Surgical cardiac injuries most commonly result from penetrating mechanisms [30]. Frequently, these patients have active bleeding or signs of shock or death that necessitate surgical intervention. Many variables have been associated with patient outcomes, particularly the mechanism of injury, the presence of signs of life during evaluation, and the number of injuries to the heart and great vessels [31–34]. It is common for penetrating injuries (particularly gunshot wounds) to cause multiple holes; therefore diligent inspection (including the intraventricular septum) is required. Early utilization extra corporeal membrane oxygenation and transesophageal echocardiography might aid in diagnosis and repair of near lethal injuries. Currently, the mortality of patients with multiple cardiac and great vessel injuries remains extremely high [34].

As mentioned previously, definitive repair of cardiac wounds should be performed with a 3-0 or 4-0 permanent monofilament suture. Large needles are commonly employed to span the defects, and pledgets or large felt buttresses can be used to minimize cardiac muscle damage. After hemorrhage is controlled, and injuries to other structures have been ruled out, the patient can be discharged to the ICU for resuscitation and monitoring. The pleural spaces and pericardial spaces should be drained. There are no randomized controlled studies evaluating the role of pericardial closure after cardiac surgery for trauma. Data from the elective cardiac surgery literature is mixed [35].

14.3.3 Great Vessels (Cava, Pulmonary Arteries, Pulmonary Veins, and Aorta)

Data regarding the management of traumatic injuries to these vessels is sparse, and they are usually grouped into a common category. Many injuries to these vessels are more easily and successfully managed by way of endovascular approaches. However, the selection bias of those reports is critical as many of those patients fall outside the purview of traditional damage control surgery. Those patients who do require open surgical intervention are best served with direct suture repair of the injured vessel.

Injuries to the superior vena cava can be controlled with vessel loops above and below the level of injury. Side-biting vascular clamps or end-biting (such as Allis) clamps placed adjacent to one another along the length of the injury can eliminate or slow bleeding enough to repair the vessel. Care should be taken to minimize narrowing of the vessel. More destructive injuries might require vein patch to preserve patency. Data on the utility of antiplatelet therapies and anticoagulation after repair of caval injuries is lacking. Venous air embolism is a potential complication of large caval injuries. The treatment for this problem involves steep Trendelenberg positioning to trap the gas in the right ventricular apex, preserving blood flow through the ventricular

outflow tract. The ventricle can also be aspirated through the cardiac wall, or by way of an endovascular approach. Reports of this technique are limited to case studies.

Pulmonary artery (PA) injuries should be primarily repaired. Like many cardiac wounds, pledgets can be used to minimize further tissue damage and distribute the suture tension across the wound. Posterior and distal PA injuries might necessitate the use of cardiopulmonary bypass to adequately expose and control the injury.

Pulmonary venous injuries that survive to operation are uncommon. Descriptions in the literature consist mostly of case reports. They present in two varieties: intrapericardial rupture that occasionally involves the left atrium and presents as hemopericardium or intrapleural rupture presenting as massive hemothorax. Some authors have described repair of intrapericardial wounds without the assistance of cardiopulmonary bypass [36–38]. However, the elevation of the heart to expose these vessels typically results in decreased preload followed by hemodynamic instability. Therefore, the surgeon should be prepared to initiate bypass if it is necessary for adequate exposure and repair [39]. Extrapericardial wounds care can generally be controlled manually or with the assistance of a balloon catheter. If ligation of the pulmonary vein is required, the corresponding lobe of the lung should be considered for resection. However, the optimal timing of this procedure is not known.

Injuries to the thoracic aorta that are encountered during open surgery are primarily repaired with sutures. As with some other complex great vessel injuries, wounds that involve posterior surfaces or the transverse arch, where the origins of the right brachiocephalic and the left carotid and/or subclavian arteries originate, might benefit from cardiopulmonary bypass to reconstruct. The wounds can be balloon occluded temporarily if necessary. To our knowledge, shunting of the arch vessels as a maneuver to abbreviate the index operation has not been described.

Stable patients, in whom an aortic injury is diagnosed before bringing the patient to the operating room (this sample of patients is, overwhelmingly, people injured by blunt mechanisms and diagnosed using cross sectional imaging), should be repaired by way of endovascular stenting [40–43]. There is little role for open repair as the morbidity and mortality of this approach remains high. There is probably a role for technology, such as Rapid Endovascular Balloon Occlusion of the Aorta (REBOA), even in open cases using a hybrid open/endovascular approach. To date, however, this role is poorly understood.

14.3.4 Trachea and Bronchi

Most injuries of the distal large airways result from blunt mechanisms and are suspected because of large, ongoing air leak after pleural evacuation of pneumothorax [44]. Diagnosis should be confirmed with bronchoscopy, and these injuries should be primarily repaired and buttressed with healthy tissue in a semi-urgent fashion. In this regard, there is little that needs to be done at index operation if a tracheal or bronchial injury is encountered and the surgeon is eager to abbreviate the operation for other reasons. Simple injures can be primarily closed. In the absence of respiratory failure resulting from air leak, more complex injuries should be managed with wide pleural drainage and evacuation of pneumothorax until such time that definitive repair is possible.

14.3.5 Lungs

As mentioned previously, massive pulmonary hemorrhage can be controlled with direct pressure or segmental/hilar vascular control. Peripheral injuries can be wedge resected using surgical stapling devices. The use of tractotomy procedures is well described and accepted to control hemorrhage emanating from within and lung parenchymal injury. Staples with 2.5–3.5 mm leg lengths generally work for this procedure. Bleeding vessels in the now open tract should be controlled with 3-0 or 4-0 mattressed sutures. Topical hemostatic agents can be used to supplement the sutures in the event of minor bleeding. Low volume air leaks can also be managed with

pleural drainage and pressure-controlled ventilation in order to abbreviate surgery if necessary.

Emergency lung resection, although rare (incidence of 0.08–1.3%), is associated with high morbidity and mortality, especially when a pneumonectomy is required [45, 46]. The majority of studies have been limited to small single-center reports with great variability. The contribution of fulminant right heart failure to mortality in the setting of emergency pneumonectomy is an often mentioned but poorly understood phenomenon. As prolonged hilar occlusion and deferred major pulmonary resections have been described as treatment strategies for refractory pulmonary bleeding, the surgeon should give strong consideration to damage control strategies in these cases [47]. Inhaled prostacyclin should be considered for patients with evidence of right ventricular failure awaiting definitive management of major pulmonary resection [48, 49].

14.3.6 Esophagus

Blunt traumatic thoracic esophageal injuries are very rare, and most wounds to this region of the esophagus are caused by gunshot injuries [50, 51]. Wounds should be closed primarily with careful attention to approximation of the mucosal layer. Like all esophageal perforations, we recommend wide drainage of the affected mediastinal and pleural spaces. Flap protection of the closure can be delayed if damage control surgery is being conducted, during such time, the lumen of esophagus/stomach should be aggressively drained.

Exposure of the injured segment can be challenging, particularly in the area below the tracheal bifurcation and behind the heart. We have successfully treated a gunshot wound in this region complicated by a through-and-through-type injury of the proximal pulmonary artery using a covered esophageal stent. Esophagoscopy was used intraoperatively to diagnose the injury (based on trajectory), and the stent was deployed in the ICU as the patient required ongoing extracorporeal membranous oxygenation as a sequel of the PA injury. To our knowledge, the treatment of gunshot thoracic esophageal injuries with covered stents is undescribed otherwise.

Authors have also described T-tube drainage of penetrating esophageal injuries in patients who cannot undergo official repair due to other reasons [17, 52]. This provides a rapid means of drainage and preserves tissue for delayed reconstruction after resuscitation is complete.

14.3.7 Temporary Thoracic Closure

Patients requiring damage control surgeries can undergo temporary chest closure in order to abbreviate operating time. Several techniques have been described, and there is no scientific evidence to support one approach over another. We prefer to drain all entered spaces. Pleural spaces can be controlled with standard chest tubes. Generally, we use one tube on each side, brought out through the skin in a standard position. We prefer large Blake drains for the pericardium and mediastinum. These can be brought out through the incisions to preserve skin and drain locations for the planned washout and closure.

We create a visceral covering by sandwiching a 6 in. Esmarch tourniquet between two pieces of Ioban with the adhesive side facing inwards. The Esmarch is cut slightly longer than the incision, and the Ioban should overlap the Esmarch on every side. In the absence of Esmarch tapes, surgical towels or any other device to add integrity to the Ioban can be substituted. This bandage is then placed in the wound, under the chest wall to cover the visceral pleura. The incision is then gently reduced manually. Surgical towels or sponges are then laid into the wound, and a large adhesive dressing is applied to the entire incision. Care should be taken to seal any drains that are brought out through the dressing.

The surgeon should also note the airway pressures that are present before and after the dressing is applied to reduce the risk of generating thoracic compartment syndrome. Nonsurgical bleeding resulting from trauma-induced coagulopathy can be controlled with packing. In these cases, monitoring airway pressures, as well as gas exchange, is critical.

14.4 Abdomen

Of the various body regions in which a trauma surgeon might operate, the abdomen is generally regarded as the most familiar. And while injury patterns are often seen from one patient to the next, it is important to remember that no two patients are identical. Systematic exploration and frequent reassessment of operative progress, patient physiology, and evolving plans ensure adequate and timely surgery.

14.4.1 Laparotomy

A midline laparotomy extending from the xiphoid process to the pubis symphysis is a typical incision for torso hemorrhage control. The process should be performed quickly, and the exploration should occur in a systematic fashion to inspect for bleeding. The surgeon should consider a bilateral subcostal incision for patients who have undergone previous laparotomy. This should be lower than a standard chevron, allowing better access to the pelvis.

Once the abdomen is open, hemoperitoneum is temporized with packing. This should allow sufficient time to establish a self-retaining retractor. The surgeons should then examine all zones of the retroperitoneum, solid organs, deep pelvis, and hollow viscera for a sign of bleeding. Autologous transfusion of shed blood is a useful strategy. Although not ideal, shed blood can be used in the face of hollow visceral injury [53, 54].

There has been recent interest in the use of endovascular balloon catheters to help control hemorrhage as surgical procedures are initiated. Studies have attempted to compare outcomes relative to resuscitative ED thoracotomy and quantify blood loss during the time leading up to definitive surgery [55–61]. The use of endovascular aortic balloon occlusion in a highly lethal swine model was associated with higher blood pressures and lower mortality compared to animals bled without the device [57]. Demonstration of such outcomes in humans, however, remains limited.

14.4.2 Liver

Restoration of normal liver anatomy is a simple maneuver that helps slow bleeding from the injured surface. The shape can then be maintained and buttressed by the addition of dry surgical laparotomy pads, and it can generally be performed without any formal mobilization of the suspensory ligaments. If the bleeding continues, the surgeon should arrest vascular inflow by application of a Pringle maneuver [62, 63]. This is performed by manual occlusion of the porta hepatis and division of the pars flaccida. We prefer vessel loop occlusion of the porta as the low profile of the loop (as opposed to a vascular clamp) facilitates easier operating in the right upper quadrant. If the bleeding stops with the application of a Pringle, the course is most likely portal venous or hepatic arterial in nature. The injury should be gently extended and explored. Bleeding vessels can be ligated, clipped, or occasionally controlled with linear stapling devices. Care should be taken to avoid exacerbation of the injury. Hepatic arterial bleeding is generally amenable to catheter embolization (Fig. 14.2).

Ongoing liver bleeding that persists despite application of a Pringle maneuver suggests hepatic venous injury. These injuries and those limited to the retrohepatic vena cava carry high mortality rates. If the bleeding arises from a branch or segmental branch of the hepatic vein, the injury should be suture ligated. Exposure of

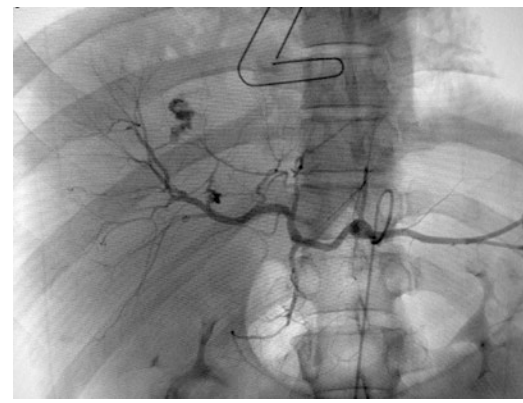

Fig. 14.2 Catheter angiogram demonstrating hepatic arterial bleeding

the hepatic veins is performed directly through large wounds, or by mobilizing the right or left lobes of the liver based on the branch suspected to contain the injury.

Other advanced surgical techniques for refractory liver bleeding include the use of an atriocaval shunt or venovenous bypass after total vascular occlusion of the liver with a Pringle maneuver and suprahepatic (or intrapericardial) and suprarenal inferior vena cava occlusion. Mounting evidence suggests, however, that injuries to the retrohepatic vena cava or deep intraparenchymal liver might be served better with extensive perihepatic packing to restore anatomy and complimentary angioembolization to rule out arterial injury [63–66].

14.4.3 Spleen

The spleen is a commonly injured organ, and it can be the source of life-threatening hemorrhage. And while nonoperative management of spleen injuries has become commonplace, there remain times when those injuries themselves, or the combination of spleen and other injures, preclude nonoperative management. In such cases, the surgeon must consider the risks and benefits of splenic salvage. For surgeons who need to abbreviate the operation, we recommend splenectomy. This reduces the likelihood of postoperative bleeding complications from splenic sources. The surgeon must also ensure vaccination against *Streptococcus pneumonia*, *Haemophilus influenza* type B, and *Neisseria meningitides* during the postoperative period.

14.4.4 Stomach, Small Intestine, and Colon

The key concept for managing of hollow visceral injuries is control of spillage to minimize contamination. The surgeon should also make efforts to preserve as much bowel length as possible for definitive reconstruction. Enteral spillage can be controlled in several different ways. Temporary suture repair is a fine, as a low profile temporizing solution. However, it might be time-consuming if several injuries need to be addressed. Surgical clamps such as Allis and Babcock clamps provide speed but can be cumbersome as the bowel is examined and manipulated. We often used umbilical tape passed through small defects at the mesenteric border and gently ties around the hollow visceral injuries. This provides a type a "proximal and distal" hollow visceral control and allows the surgeon to reassess which injuries can be repaired primarily and which should be formally resected during a more favorable part of the operation. It is important to note the gastrointestinal continuity can be deferred to a later operation during cases when the surgeon is trying to abbreviate emergency laparotomy. However, consideration should be given to proximal injuries (duodenal and jejunal), particularly if there are upstream wounds. Managing these injuries with "blind ends" for prolonged periods of time might compromise upstream repairs.

14.4.5 Common Bile Duct, Duodenum, and Pancreas

Biliopancreatic injuries can be challenging to diagnose and manage. Surgeons should maintain a high index of suspicion for these types of injuries when wounds are in proximity to the head of the pancreas or adjacent structures. Mobilization of the duodenum, ascending colon, and root of the small bowel mesentery toward the left upper quadrant (Cattell-Braasch maneuver) will expose the duodenum and uncinate process of the pancreas. The distal common bile duct can be examined with intraoperative cholangiography performed through the gall bladder or distal cystic duct. On occasion, the pancreatic duct can be enhanced during antegrade cholangiography by applying gentle pressure to the duodenum during the study (Fig. 14.3). Open cannulation of the ampulla of Vater with retrograde imaging has also been described; however, the procedure can be challenging when anatomy has been distorted by adjacent trauma. We do not advocate the creation of a duodenotomy for the sole purpose of

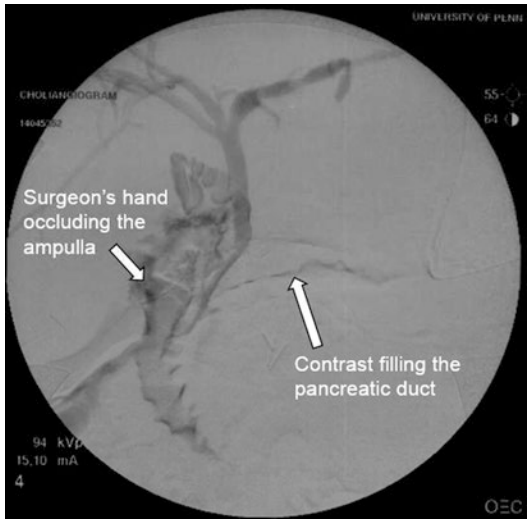

Fig. 14.3 The surgeon's hand is used to apply pressure against the duodenum to occlude the ampulla of Vater and reflux contrast into the pancreatic duct during antegrade cholangiopancreatography

imaging. These cases are better managed with wide drainage and subsequent imaging with MRCP prior to definitive closure. If an injury to the common bile duct is identified, T-tube drainage is a fast and reasonable procure to minimize contamination [67]. Extraluminal tube drainage should also be applied.

Suspected pancreatic injuries, in the absence of other compelling reasons to proceed with laparotomy, should be evaluated with CT scan, as many can be managed without operation [68]. The management of pancreatic injuries found during laparotomy varies by location and grade. Many injuries can be safely treated with hemostasis and aggressive drainage. Injuries distal to the neck of the pancreas can be treated with distal pancreatectomy. The timing of this procedure, however, remains at the discretion of the operating surgeon. Wide drainage with packing might be a reasonable option for patients who require abbreviated surgery. Similarly, emergent division of the pancreas to access superior mesenteric venous or portal bleeding with planned reconstruction or completion pancreatectomy might be required in extreme cases [68].

Optimal management of duodenal injuries remains a controversial topic. Recently, the literature has favored primary repair with a focus on intraluminal (rather than extraluminal) drainage, particularly for low-grade injuries [69]. Definitive management might involve techniques such as triple tube drainage (draining gastrostomy, draining retrograde duodenostomy, and feeding jejunostomy) or pyloric exclusion [70–72]. Suture closure of the injury with nasogastric and perhaps nasoduodenal tube drainage is sufficient to move the patient back to the ICU for resuscitation prior to committing to a particular reconstruction.

14.4.6 Aorta, Cava, and Other Abdominal Major Vascular Injuries

Zone 1 retroperitoneal injuries are best explored based on their location relative to the transverse mesocolon. Lesions above the mesocolon should be approached by mobilizing the descending colon and splenic flexure toward the midline. This will expose the celiac axis to the superior mesenteric artery (SMA). Celiac arterial injuries are uncommon but carry a nearly 50% mortality rate [73, 74]. The trifurcated nature of the vascular complex makes shunting these injuries nearly impossible. Therefore, patients in extremis should undergo primary repair or ligation of the celiac axis. Splenectomy and reimplantation of the splenic artery to the aorta for retrograde perfusion of a ligated celiac axis have been performed with unsatisfactory results.

The morbidity and mortality of SMA injuries is correlated with Fullen zone classification and Organ Injury Scale grade. Wounds to Fullen zone 3 or 4 that are not amenable to repair can be ligated with anticipation of some segmental bowel ischemia [75]. Injuries that involve Fullen zone 1 or 2 are better managed with temporary shunting as these patients generally have other injuries that need to be addressed prior to mesenteric vascular reconstruction [76].

Hematomas arising from below the transverse mesocolon are best exposed with a Cattell-Braasch maneuver. This includes suspected

injuries to the iliac vessels. Shunting of abdominal aortic injuries has been described, oftentimes, requiring large conduits such as chest tubes. Generally, however, abdominal aortic injuries are more easily managed with definitive primary repair or patch angioplasty (with synthetic or biosynthetic materials). Temporary intravascular balloon occlusion is also a useful adjunct for hemorrhage control during the initial exposure.

The abdominal inferior vena cava is usually exposed by way of a Cattell-Braasch maneuver. Direct occlusion of the cava against the vertebral bodies/retroperitoneum can be achieved manually or with the help of sponge sticks or formal aortic occlusion devices. Lateral venorraphy is the repair of choice for major abdominal/pelvic venous vascular injuries. The surgeon should be sure not to narrow the vessel to great degrees. The surgeon should also consider the possibility of through-and-through-type injuries for larger vessels. On occasion, when mobilization of the back wall is impossible, anterior injuries will need to be extended to provide intraluminal exposure and repair of posterior wounds (Fig. 14.4). Many major abdominal venous injuries can also be ligated for patients in extremis. Special consideration must be given to the portal and superior mesenteric veins, however. As occlusion of hepatic flow, more importantly, small bowel venous drainage can result in devastating consequences.

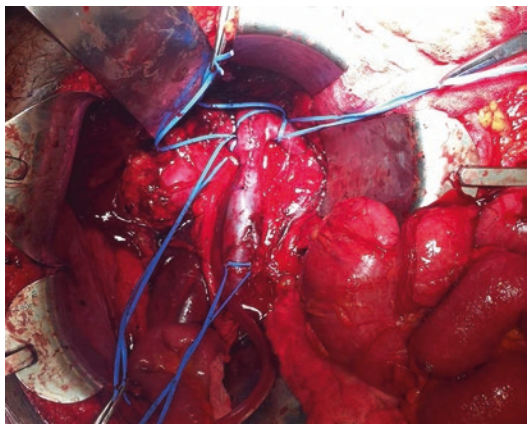

Fig. 14.4 Extension of an anterior cava injury for internal closure of the posterior wound

14.4.7 Genitourinary System

The kidney is the most commonly injured part of the GU system, with most resulting from blunt mechanisms. Many of these, however, will never come to surgical exploration. Among patients with penetrating mechanisms, however, laparotomy is more common. Bleeding injuries can be controlled with vascular control of the renal hilum (Fig. 14.5). Whether to obtain vascular control by taking down Gerota's fascia laterally and reflecting the kidney toward the midline or open the retroperitoneum medially adjacent to the aorta is at the discretion of the operating surgeon. Some authors have described increased kidney salvage rates with vascular control prior to delivery of the kidney; however, these findings are hard to substantiate [77–79].

Among patients with penetrating injuries, nephrectomy occurs in 25–50% of cases. This seems more tightly correlated with the degree of injury rather than surgical approach [80–83]. Although many renal wounds do not necessitate

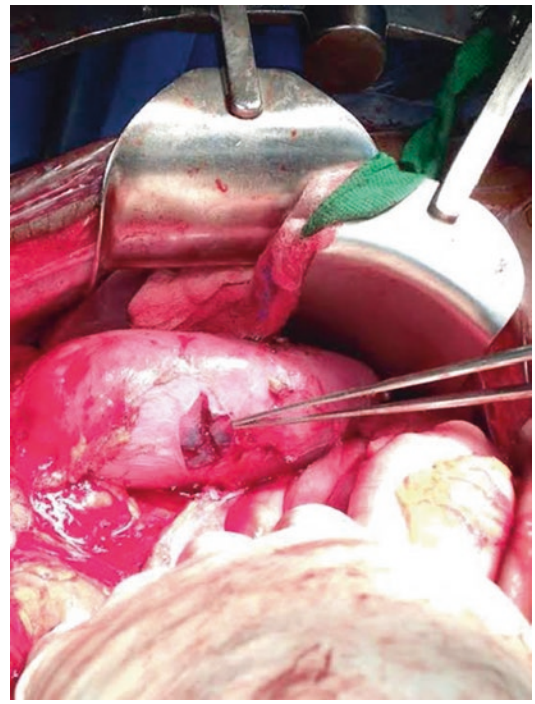

Fig. 14.5 Penetrating kidney injury that is hemostatic with vascular control

nephrectomy, it might be the best surgical choice for patients who have other sever injuries driving a damage control approach. In these cases, the surgeon should inspect the contralateral side for the presence of another kidney. Traditional teaching recommends performing an on-table single-shot intravenous pyelogram to confirm functionality of the other kidney; however, this should not delay lifesaving surgery in the presence of competing priorities.

Ureteral injures are not life-threatening and can be quickly temporized during abbreviated laparotomy. Partial wound should be widely drained and definitely repaired (preferably over a stent) during the take-back operation. If the primary surgeon is able to stent the injury during the first operation, that is also favorable. Ideally, the stent should extend proximally into the renal pelvis [84]. Complete injuries should be ligated at injury site. Again, definitive management can be addressed during subsequent operation. If reconstruction is delayed, the collecting system should be drained by way of a percutaneous nephrostomy tube [84].

The bladder is also a commonly injured part of the GU system. Fortunately, most of these injuries can be managed with transurethral urinary drainage. Injuries that are open to the peritoneal system should be closed with an absorbable suture. We prefer a two-layered closure. However, the surgeon is not compelled to perform formal repair if other organs require interventions, or patient physiology mandates more aggressive resuscitation. As with other GU injuries, aggressive, wide drainage, even if only temporary, is advised.

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Abbreviated Surgery: Orthopaedic Surgery

15

Roman Pfeifer, Kai Sprengel,
and Hans-Christoph Pape

15.1 Introduction

The stepwise approach to the orthopaedic stabilization of the severely injured patient foresees manoeuvres to support resuscitation. This is achieved by i.v. infusions, coagulatory support, vasopressors if required and providing temporary stabilization and by minimizing the load of surgery. The idea behind it is that the biological reserve of the patient is maintained, and the pathogenetic changes induced by trauma are not exaggerated through inadequate surgeries. It is along the concept of the adage *primum non nocere* or "do no further harm".

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15.2 Value of Early Orthopaedic Fracture Fixation

The value of fracture fixation has been emphasized since the 1970s. Orthopaedic fracture stabilization belongs to the most important tasks in the general treatment. It shortens the duration of the mechanisms addressed in Chap. 15, such as endocrinologic, humoral and inflammatory changes [1, 2]. Therefore, as a prerequisite, it is important that whenever a patient is in a good condition, he should undergo any required surgery:

> *Chiefly before trauma care can be improved, the reasons for the delay in fracture fixation need to be answered. One can speculate that the patients were in shock, were undergoing other more urgent procedures, or had other injuries being monitored. However, one can also speculate many patients had their fracture fixations delayed due to surgeon or operating room unavailability. Some patients may have required "operative clearance" by the system at their institution and this may have provoked a delay as well.* [3]

In contrast, if an improper selection of the duration of surgery is performed, the benefits of fracture fixation can be counteracted [4, 5]. These patients can lose too much blood, experience a drop in body temperature and suffer the changes associated with these issues, such as exaggerated inflammatory reactions and postoperative complications [6] Table 15.1.

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Table 15.1 Principles of abbreviated surgical management in the OR

A: General considerations:
Spend less than 2 h in the OR after haemorrhage control for fixation of fractures
Spend as little time as possible if haemorrhage control cannot be achieved and the patient is in the OR for other reasons
B: Intra-op principles:
Core temp.: if dropping, try to correct or terminate surgery
Coagulation: if worsening, try to correct or terminate surgery
Ventilation: increased airway pressure, try to find a cause (pneumothorax)
Need of vasopressors increasing: terminate surgery and look for a cause
Urinary output decreasing:

15.3 Calculations on the Incidence of Abbreviated Orthopaedic Surgeries: The Polytrauma Grading Score (PTGS)

So far, the incidence of abbreviated surgeries has been based on assumptions of the borderline trauma patient and it was assumed to be around 15–20% [5, 7].

Our group recently developed a new scoring system named the PolyTrauma Grading Score (PTGS) [6]. It was developed based upon a multivariate analysis. It reconfirmed that multiple prognostic parameters are important. Among them are the admission blood pressure, acid base abnormalities [7], coagulopathy, the number of pBRCs administered and the injury severity score (Table 15.2). We differentiated certain target mortality rates based upon the new definition of polytrauma (15%, 30%, 45%). Based on the initial definition of target mortality rates for low (mortality, 5%)-, intermediate (mortality, 15%)- and high-risk groups (40%), stable patients were defined by a score value of up to 5 points. Patients with a score value between 6 and 11 points were therefore found to be in a borderline condition; more than 11 points were associated with an unstable condition. Under these prerequisites, the following distribution of patients was observed: 80.6% in stable condition, 14.6% borderline patients and 4.8% in unstable condition. Based upon these calculations, the abbreviated surgery would apply to about 15% of patients. It has to be considered that patients in extremies had been excluded by definition.

In general, the initial polytrauma patient assessment can be structured according to the "four pathophysiological cascades of polytrauma" described above [5]. The parameters of these four cascades to be remembered could be summarized in the following phrase: Soft tissue injuries (major extremity fractures, crush injuries, severe pelvic fractures, lung contusions AIS > 2), coagulopathy (platelets <90,000) and shock (systolic BP < 90 mmHg, requirement of vasopressors) contribute to hypothermia (core temp. <33 °C) and are dangerous.

15.4 Selection of Patients for Early Abbreviated Surgery: Safe Definitive Surgery Concept

Several methods have described regarding a selection process to determine which patient should undergo a primary definitive procedure versus a temporary stabilization followed by definitive fixation at a later stage [7]. The safe definitive surgery concept encompasses both components from early definitive surgery and damage control. Its prerequisite is to go to the operating room, if required by the injury pattern, and perform the appropriate type of surgery according to the criteria listed in Table 15.2. Safe definitive surgery implies the four cascades of polytrauma and puts special emphasis on respecting lung function, coagulation, temperature and acid base changes (Fig. 15.1).

Specific prognostic criteria have been developed to minimize the systemic risks depending on the current physiologic/immunologic state of the individual patient [8].

Table 15.2 Logistic regression model to evaluate the impact of risk factors to develop a scoring system (n = 5,988 patients)

Predictor	Regression coefficient	Odds ratio (OR)	95% confidence interval (95%-CI)	p-value
BP 76–90 mmHg	0.249	1.283	0.962–1.712	0.09
BP ≤ 75 mmHg	0.642	1.889	1.398–2.580	<0.001
Base deficit 8–10	0.474	1.606	1.119–2.304	0.01
Base deficit >10	1.251	3.371	2.553–4.453	<0.001
INR 1.4–2.0	0.160	1.174	0.897–1.536	0.24
INR > 2.0	0.899	2.457	1.754–3.442	<0.001
NISS 35–49	0.900	2.460	1.916–3.157	<0.001
NISS 50–75	1.188	3.279	2.487–4.324	<0.001
pRBCs 3–14	0.671	1.957	1.514–2.529	<0.001
pRBCs ≥ 15	1.692	5.430	3.910–7.539	<0.001
Platelets <150,000	0.555	1.743	1.383–2.196	<0.001
constant	−3.867	–	–	<0.001

From Hildebrand et al. [26]
Parameters graduated as low risk (Table 2) were set as categorical reference group

Knowledge of treatment algorithms is of paramount importance to avoid distraction from occult problems. A pertinent example would be neglect of intra-abdominal exsanguination while attempting to deal with severe extremity injuries. The trauma team has to quickly ascertain the extent of the injury as well as assess the pulmonary and haemodynamic status of the patient. Standardized diagnostic and operative tactics should be applied and coordinated to avoid mistakes that could impact negatively on the patient's prognosis. During the course of treatment, the clinical scenario can change rapidly and management plans must able to adapt accordingly. Therefore, the clinical judgement of the patient's condition is crucial.

The use of markers of occult tissue hypoperfusion, such as serum lactate, to demonstrate adequacy of resuscitation has become a commonplace, and the measurement of inflammatory markers that highlight patients at risk is helpful in deciding which patients are best served by damage control surgery. Most recently, orthopaedic surgeons from Cleveland, (Ohio) proposed to use solely parameters associated with the acid base system only universally for virtually all patients with fractures [9]. They discuss that normalization of base excess and lactate values allows patients to be cleared for major orthopaedic operations, without assessing any other parameters. We feel that this is a dangerous approach since lactate by itself can be influenced by many other parameters, and it does not allow for safe definitive surgery [10, 11]. However, there is a contribution of various metabolites to the "unmeasured" anions in critically ill patients with metabolic acidosis [12]. Also, patients that do not have altered lactate values may demonstrate severe lung contusions or may be on medications that cause elevaled lactate levels. Moreover, along with an increasing number of patients that undergo acute injuries in their later stages of life, chronic diseases, such as renal failure, can occur and may be the reason for seemingly patholigical lactate values [13]. Finally, many elderly patients are on Coumadin for medical reasons. In these, it would also be unwise to only look at lactate levels for medical clearance.

In this line, an international group of authors used the similar principles to develop an evidence-based definition of polytrauma. In addition to a minimum of two significant (AIS 2 or greater) injuries, several cofactors were calculated on the basis of the registry. These were associated with a raise in the mortality rates from about 12–15% (ISS > 16) to about 40%. Five independent physiologic variables were

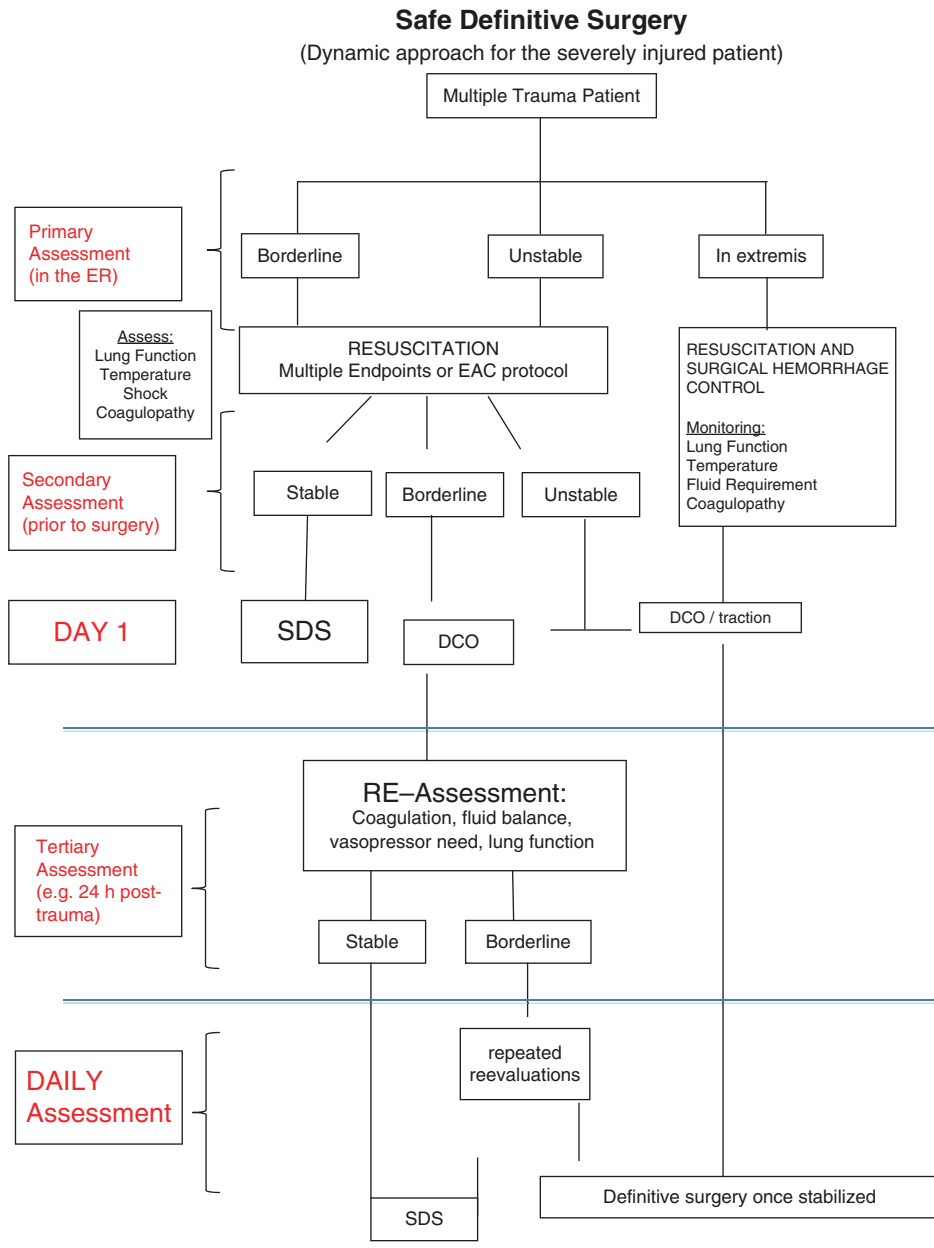

Fig. 15.1 Inclusive protocol of tapered early surgery for orthopaedic injuries in polytrauma patients. It is of note that all *stable* patients are excluded as their treatment is considered to be early fracture stabilization. This concept is designed to combine the positive effects of early fracture stabilization and early temporary surgery, depending on the patient condition [22]

identified, and their individual cutoff values were calculated based on a set mortality rate of 30%: hypotension (systolic blood pressure <90 mm Hg), level of consciousness (Glasgow Coma Scale [GCS] score <8), acidosis (base excess ≥6.0), coagulopathy (international normalized ratio 1.4/partial thromboplastin time >40 s) and age (>70 years). This study supports the idea of multiple factors to be required for the assessment of a multiply injured patient [14].

It has also been suggested that patients at risk of adverse outcome, such as those with head injury, bilateral lung contusions, multiple long bone injuries, coagulopathy, hypothermia or estimated operation time of >6 h, should be considered for sequential staged surgical management [15]. Markers of the adequacy of shock reversal, such as serum lactate, are measured routinely in trauma centers. It is therefore easy to envisage the routine use of markers of pro- and anti-inflammatory systems such as IL-6, IL-10 and procalcitonin to aid in the decision to carry out damage control surgery. Delaying definitive surgery until the shock state is fully reversed would appear to be not only beneficial but imperative [8].

Many trauma scoring systems (e.g. the abbreviated injury scale [16] injury severity score [17], revised trauma score [18, 19] and Glasgow Coma Scale [20] have been developed in an attempt to describe the overall condition of the trauma patient. Bosse et al. [21] noted that "there is no score that assists in decision-making during the acute resuscitation phase". This idea supports the notion that one should not rely exclusively on a single parameter, but rather a combination of parameters that should cover several pathophysiological pathways [5]. Most of the components are scores that have been routinely used in the past and are widely accepted. For screening purposes, the following threshold levels have been used:

- Pulmonary dysfunction (PaO_2/FiO_2 <250)
- Platelet count (<95,000)
- Hypotension unresponsive to therapy
- >10 blood units transfused per 6 h
- Requirement for vasopressors

These recommendations are underlined by the PolyTrauma Grading Score (PTGS), as the score is the result of deductive calculations from a nationwide database [8].

In this score, the following factors are crucial:

- Severity of the injuries sustained
- Presence or absence of a criterion indicating borderline status and associated factors associated with a high risk of adverse outcomes, systolic blood pressure 76–90 mmHg, INR 1.4–2, thrombocytes, base deficit 8–10, NISS 35–39 and pBRC 3–14

These appear to be among the factors governing which line of treatment should be used in patients with polytrauma.

Several guides to treatment have been developed especially for the management of fractures of the long bones and pelvis [22]. Additional clinical criteria that we have been used as a basis for shifting to damage control orthopaedics include a pH of <7.24, a temperature of <35 °C, operative times of more than 90 min, coagulopathy and transfusion of more than ten units of packed red blood cells. Furthermore, certain specific orthopaedic injury complexes appear to be more amenable to damage control orthopaedics; these include, for example, femoral fractures in a multiply injured patient, pelvic ring injuries with exsanguinating haemorrhage and polytrauma in a geriatric patient.

15.5 Strategies and Technical Aspects During Abbreviated Surgery

15.5.1 Absence of Chest Injuries and Long Bone Fractures

The following scenario, depending on the severity of chest injury for patients with long bone fractures (femur and tibia), has been advocated:

In patients without thoracic trauma that respond adequately to resuscitation, all major fractures are stabilized, as long as the patient is stable during the surgeries. A new assessment of parameters applies intraoperatively. In patients with thoracic trauma with an AIS of 2–4 points ("borderline patient"), unreamed nailing is performed to minimize the potential risk of acute fat embolization. In patients in a critical condition (haemodynamically unstable) or with associated severe thoracic trauma (AIS >4 points), intramedullary stabilization is avoided, and a temporary external fixateur is applied until the pulmonary situation has stabilized. Additional

clinical criteria as a basis for shifting to damage control orthopaedics include coagulopathy, a pH of <7.24, pathological lactate, a temperature of <35 °C, planned operative times of more than 2 h and transfusion of more than ten units of packed red blood cells.

15.5.2 Presence of Chest Injury for the Indication of Abbreviated Surgery

The assessment of pulmonary function requires special attention: The progressive nature of a pulmonary contusion can be problematic and is frequently underestimated. Early after injury, the blood gas parameters can still be within normal limits, and the chest radiograph may be negative.

Signs of deterioration of pulmonary function include:

- Presence of a lung contusion on the initial chest radiograph or CT scan
- Worsening oxygenation reflected in an increased FiO_2 >40% or PaO_2/FiO_2 <250
- Increased airway pressures (e.g. >25–30 cm H_2O)

Chest trauma is an independent parameter of outcome [23]. Along with the improvement in diagnostics, more sophisticated diagnoses of parenchymal pulmonary changes were an option. Therefore, scores for quantification of the degree of chest injury were developed [24, 25]. It became evident that the use of combined parameters for the assessment of trauma patients is superior to using just one. For thoracic trauma, it was the oxygenation, number of rib fractures, degree of parenchymal contusion, presence of pleural involvement and patient age. ROC analysis clearly indicated that the combination, as used in the TTS, was superior to ISS and AIS chest alone [3].

15.5.3 Ipsilateral Femoral and Tibial Shaft Fractures

In stable patients, all fractures are stabilized during the same initial operation (Table 15.3). If the patient is in questionable state and improves, one long bone fracture is stabilized definitely according to the surgeon's preference or the degree of soft tissue injury. If the patient then deteriorates, the second and following fractures can be stabilized by external fixation or traction.

15.5.4 Pelvic Trauma and Packing

Pelvic fracture is often seen in conjunction with multisystem trauma and can lead to rapid occult haemorrhage. Treatment should be conceived as part of the resuscitative effort and early intervention can be life-saving. Bleeding is commonly from multiple small sites rather than injured major vessels and, due to the large volume of the retroperitoneum, in severe cases spontaneous arrest is unlikely. Furthermore, it is common for the retroperitoneum to be breached during the injury further decreasing barriers to the ongoing

Table 15.3 Staged management of ipsilateral femoral and tibial fractures

Admission condition	Stable	Borderline		Critical	Comments
Resuscitation response		+++ (responder)	−−−responder	Spanning ex.fix.	
Day 1 surgery	Femur retrograde tibia antegrade	Femur retrograde	Femur ex.fix		
		Tibia antegr./ex.fix.	Tibia ex.fix.		
Secondary conversion		Femur retrograde			

haematoma expansion. Treatment with the pneumatic antishock garment or pelvic belt-straps can give some temporary stabilization, but results are inconclusive, and severe complications have been reported in relation to their use.

There has been increasing interest in the use of selective angiography in these cases to embolize bleeding vessels. However, this intervention requires a full set-up of a level I trauma center, and even in these, it is often time consuming. Patients must be relatively stable and careful selection is crucial. Embolization is an adjunct where continued arterial haemorrhage is suspected. In severe injuries with profound haemodynamic instability, external fixation, pelvic C-clamp and open tamponade by packing are performed in many European centers and in some in the USA. In these, it is often combined using a C-clamp. In vertical pelvic instability (C-type injury), the lower extremity of the respective should be accessible for better ease of reduction.

Following application of external fixation devices, if there is prior evidence of free intraperitoneal fluid, a midline laparotomy should be performed and the intra-abdominal organs examined for bleeding following standard management protocols for blunt abdominal trauma. In the absence of intra-abdominal injury, the attention should be directed to the retroperitoneum. Following skin incision, ruptured pelvic soft tissues are usually readily visible. After evacuation of haematoma, the packing is performed using lap sponges in meander technique.

Conclusions

The polytrauma trauma patient who requires massive transfusion incurs the greatest risk for the multifactorial interactions between acidosis, hypothermia, and coagulopathy and soft tissue injuries. There continues to be an ongoing challenge to identify better predictors of outcome, improved means of resuscitation, greater understanding of physiologic derangements and better timing to institute damage control. There also remains a need to better understand the cellular and subcellular mechanisms triggered by profound shock, exsanguination, acidosis, hypothermia and coagulopathy. Delays in the decision to perform damage control contribute to a higher morbidity and mortality. Therefore, damage control is a vital part of the management of the multiply injured patient and should be performed before metabolic exhaustion.

Damage control orthopaedics is ideal for an unstable patient or a patient in extremis, and it has some utility for the borderline patient as well. Specific injury complexes for which damage control orthopaedics should be considered are femoral fractures (especially bilateral fractures), pelvic ring injuries with profound haemorrhage and multiple injuries in elderly patients. Specific subgroups of multiply injured orthopaedic patients who may benefit from damage control orthopaedics are those with a head injury, chest trauma or a mangled limb. A limited form of damage control orthopaedics (limb damage control orthopaedics) is a rational alternative for the treatment of isolated, complex limb injuries.

Clinical data and emerging discoveries in molecular medicine may continue to provide answers to the question of when orthopaedic surgeons should use a damage control orthopaedics approach.

Whatever approach is taken, it is clear that the management of patients with multiple injuries must pay close attention to the systemic effects of the respective procedure and timing. This process of decision-making may be defined as “injury-patient tailored” for damage control orthopaedics. However, with regard to this strategy, it remains necessary to prospectively define and validate prognostic criteria performing studies similar to the Lower Extremity Assessment Project which should be fulfilled to better understand the role of damage control orthopaedics in the treatment of patients who have sustained orthopaedic trauma, especially those with concomitant injuries to the chest and head (Table 15.4).

Table 15.4 Case example for abbreviated initial surgery

Injury	Day 1	Day 3	Day 7	Day 15	Day 20	Day 34
Blunt chest trauma TTS score:	Vent	Vent	Vent	Vent	Vent	
Pelvic hemorrhage	Laparotomy, packing	Second look		ORIF		
Large bowel laceration	Resection, Hartman					
C type pelvic fx.	Ex.fix			ORIF plating		Rehab
L fem shaft frx (open)	Ex fix, vac	amputation	Vac	Vac	Vac	
R fem shaft fx.	EX. Fix.		IMN			
R patella frx (open)	Partial resection	-	-	-	-	
R prox tibia Frx (open)	Ex fix, Vac	Vac	Vac	Vac	IMN, local flap	
Duration of surgery: all Duration of surgery: Ortho	2.5 h 40 min.	2h	45 min	4h	2h	9h
Immunologic markers						
IL-6 (ng/L)	3,210	1,507	60.4	22.3	Normal range < 5,4	
IL-8 (ng/L)	2,900	64,4	22.4	< 5	Normal range <62	

IL-1 beta interleukin-1 beta, *IL-6* interleukin-6, *IL-8* interleukin-8, *Amp* amputation, *DFN* distal femoral nail, *DHS* dynamic hip screw, *dist* distal, *ex fix* external fixation, *extub* extubation, *fem* femoral, *flap* myocutaneous skin flap, *fx* fracture, *hum* humerus, *ORIF* open reduction and internal fixation, *prox* proximal, *reconstr* reconstruction, *reosteosynth* reosteosynthesis, *tib* tibial, *vac* wound vac, *vent* ventilation, *UTN* unreamed tibial nail

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ICU Care Following Damage Control Surgery

16

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16.1 Introduction

Damage control surgery (DCS) has radically changed our surgical approach to critically ill patients who arrive with severe bleeding. The primary goals of DCS are to expedite operative interventions, to minimize ongoing physiologic derangements, and to prevent early deaths from exsanguination and refractory shock. The purpose of this chapter is to discuss the ICU care after DCS that is needed to reverse physiologic derangements and prepare the patient for a second operation where definitive operative treatment will be rendered.

16.2 Mechanical Ventilation

Typically DCS patients arrive to the ICU on a mechanical ventilator under the effects of general anesthesia. Sufficient analgesia and sedation should be instituted so the patients are not discordant with the ventilator as they recover from anesthesia. Ventilator asynchrony increases intrathoracic pressure and can decrease venous return in hypovolemic patients and thereby compromise cardiac function at a time when optimizing oxygen delivery (DO_2) is paramount to reversing shock. Furthermore, increased work of breathing can substantially increase oxygen consumption (VO_2), exacerbating the difference between DO_2 and VO_2. The optimal mode of mechanical ventilation for DCS patients continues to be debated. Initially we provide full ventilator support with the assist control mode using a low inspiratory time/expiratory time (I/E) ratio to minimize the auto-PEEP that can occur as the patient awakens. In addition, it increases this ventilator mode which allows increased respiratory rates to expire the increased CO_2 production that occurs with effective resuscitation. After initial volume resuscitation, low levels of PEEP are prophylactically applied to maintain functional residual capacity. Higher-level PEEP can be used to therapeutically increase mean airway pressure to promote oxygenation in patients with high FIO_2 requirements [1]. In patients with ongoing hypoxia despite high PEEP, we convert to a pressure control mode utilizing protective low tidal volumes, moderate PEEP levels, and increased I/E ratio to allow optimized higher mean airway pressures while limiting peak airway pressures. Neuromuscular blockade is initiated in refractory hypoxia to further limit ventilator asynchrony, protect the lung, and prevent acute respiratory

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distress syndrome (ARDS). Early use of cisatracurium in severe ARDS has been demonstrated in a multicenter prospective randomized controlled trial (PRCT) to improve survival and ventilator liberation without increasing muscle weakness [2]. Interestingly, earlier PRCTs testing cisatracurium in ARDS demonstrated that it also reduced systemic pro-inflammatory cytokine levels, suggesting that it can reduce biotrauma-induced remote organ injury [3]. While early prone positioning in patients with severe ARDS has been shown in a recent multicenter PRCT to decrease mortality, we rarely utilize it early after DCS because severe ARDS usually occurs later in the clinical course [4, 5].

16.3 Resuscitation

16.3.1 End Points

Intensive care unit (ICU) resuscitation is a key intervention after DCS, and the primary goal of this resuscitation is to increase DO_2 to reverse shock. In the 1980s, Shoemaker and colleagues observed that survivors after shock became hyperdynamic increasing their DO_2 to >600 mL/min/m^2, cardiac indexes (CIs) to >4.5 L/min/m^2, and VO_2 to >150 mL/min/m^2 [6]. They proposed that these "supranormal" values be the resuscitation goals for high-risk patients to reverse shock and early PRCTs supported this practice. In the late 1980s, with the widespread availability of pulmonary artery catheters (PACs) capable of continuously measuring mixed venous hemoglobin saturation (SvO_2), many surgical intensivists presumptively placed PACs in high-risk patients to monitor oxygen transport variables and maximized DO_2 in an attempt to eliminate unrecognized flow-dependent impaired VO_2. By the early 1990s, supranormal DO_2 resuscitation became standard of care. However, as the 1990s evolved, PRCTs testing supranormal DO_2 resuscitation could not confirm this to be beneficial, and some studies showed it to be harmful [7]. During this time period, abdominal compartment syndrome (ACS) emerged as an epidemic after DCS. In the mid-1990s, surgical intensivists at the University of Texas-Houston (UT-Houston) developed a computerized clinical decision support (CCDS) protocol that utilized PAC-derived data to direct ICU resuscitation in trauma patients after severe bleeding. The protocol goal was to maintain DO_2 to >600 mL/min/m^2 for 24 h using five sequential algorithms for interventions: (1) transfuse packed red blood cells (PRBCs) if the patient's hemoglobin [Hgb] <10 g/dL; (2) volume loading with Lactated Ringer's (LR) if the patient's pulmonary capillary wedge pressure (PCWP) <15 mm Hg; (3) bolus with normal saline (NS) to optimize CI-PCWP (i.e., create a "Starling curve") if the patient's Hgb ≥10, PCWP ≥15, and DO2I <600; (4) infuse dobutamine if the patient's [Hgb] ≥10, PCWP ≥15, and DO2I <600; and (5) infuse norepinephrine if the patient's mean arterial pressure (MAP) <65 mm Hg. It was observed that most patients responded quite favorably to the initial interventions of PRBC transfusion and volume loading and easily met the DO_2 goal. However, roughly 15% of patients were nonresponders. Some had ongoing bleeding that required a hemorrhage control intervention, but most nonresponders had persistent low CIs and high systemic vascular resistances. With ongoing volume loading proceeding through the "Starling curve" intervention, these nonresponders developed problematic tissue edema in the lungs (i.e., fulminant acute lung injury) and the gut (i.e., ACS). This information prompted a decrease in the DO_2 goal in the CCDS logic to therapeutic, rather supratherapeutic, levels (i.e., 450 ml/mL/min/m^2). Subsequently, it was observed that these patients had significantly less ACS, multiple organ failure (MOF), and deaths as compared to those resuscitated to a supranormal DO_2 goal. As a result, by the mid-2000s, the use of PACs decreased dramatically, and a variety of alternative monitors were promoted to replace PACs. This fueled an ongoing debate over optimal end points of resuscitation that has yet to be resolved. In the UT-Houston, CCDS logic has been updated and currently uses central venous pressure (CVP) as the initial indicator of volume

status. The early goals of resuscitation are (1) CVP of 10 to 15 cm H_20, (2) mean arterial pressure (MAP) of ≥65 mm Hg, (3) [Hgb] ≥8 g/dL, (4) urine output of ≥0.5 ml/kg/h, and (5) central venous oxygen saturation ($ScvO_2$) of ≥70%. If the patient is resuscitated into the CVP range and there is need for ongoing interventions, bedside echocardiography is performed to assess volume status and ventricular function with the goal of optimizing cardiac performance [8]. Transthoracic and transesophageal echocardiography is being utilized in the ICU setting to determine a patient's cardiac index and volume status as well as follow a patient's response to resuscitation [9, 10]. The cardiac index and central venous pressure estimates obtained with ultrasound have been demonstrated to correlate well with estimates from a pulmonary artery catheter [11]. Recent studies suggest that lactate clearance is a superior end point for resuscitation compared to the above described goal-directed therapy [12]. An elevated lactate level occurs when there is a shift to anaerobic metabolism and lactate levels decrease with effective resuscitation. However, it has repeatedly been observed that a persistent high lactate despite aggressive resuscitation is a strong predictor of MOF and mortality [13]. This may not be due to inadequate DO_2 but rather due to dysfunctional mitochondria, which is referred to as tissue dysoxia [14]. Unfortunately, we currently do not have any therapies to reverse mitochondrial dysfunction [15]. Other monitored variables that have been proposed as resuscitation end points include gastric mucosal interstitial PCO_2 ($PgCO_2$), as measured by gastric tonometry, and tissue hemoglobin oxygen saturation (StO_2), as measured by near-infrared spectroscopy. These variables have been extensively studied in shock resuscitation, and persistent abnormal values are predictive of MOF and death. However, correction of these abnormalities has not been demonstrated to improve patient outcomes. Given the added expense of these monitors, they have not been adopted for this indication [13]. In summary, resuscitation is a key intervention, but the optimal end points remain unclear. A resuscitation protocol, however, is important in identifying the nonresponder who needs alternative interventions and will be harmed by ongoing unbridled resuscitation efforts.

16.3.2 Crystalloid Versus Colloid Debate

Based on convincing experimental data in the 1960s, isotonic crystalloids became standard of care in resuscitation during the Vietnam conflict [16, 17]. For various reasons, early mortality decreased, but a new entity called "shock lung" emerged as a major cause of late deaths. This complication was soon described in civilian publications as ARDS and was illustrated to be the major source of morbidity and mortality in the ICU. Since the pathogenesis was not understood, controversy emerged over whether ARDS was an iatrogenic complication of isotonic crystalloids. Over the ensuing years, numerous PRCTs comparing crystalloids to colloid use in resuscitation have displayed no consistent differences in the incidence of ARDS or mortality. These results are supported in numerous meta-analyses and by the recently published SAFE (Saline vs. Albumin Fluid Evaluation) and ALBIOS (Albumin Italian Outcome Sepsis) PRCTs [18, 19]. Given the markedly increased costs of colloids, it is difficult to justify their routine use. However, a series of studies looking at the clinical presentation of ACS after shock resuscitation indicated that early excessive crystalloids play a key pathogenic role. Large-volume crystalloid resuscitation can limit the diffusion of oxygen and nutrients across an edematous interstitial space [20]. In addition, it can cause intracellular edema with reduction in intracellular efficiency and poor glucose regulation [21], as well as causing the release of inflammatory cytokines and increasing organ dysfunction [22]. The endothelial glycocalyx is a complex of plasma proteins, glycosaminoglycans, and proteoglycans that is necessary for maintaining endothelial osmotic integrity. Both hemorrhagic shock and crystalloid resuscitation can result in thinning of the endothelial glycocalyx, which leads to intravascular volume depletion, and the need for continued resuscitation [23,

24]. Crystalloid resuscitation can induce pulmonary edema and acute lung injury [23, 25, 26] and has been associated with arrhythmias and a lower than expected cardiac output [27]. Aggressive crystalloid resuscitation can result in bowel wall edema, causing relative tissue ischemia secondary to a longer oxygen diffusion distance, and increased risk of abdominal compartment syndrome [28, 29]. Combined, all of this evidence provides the rationale for the use of colloids in DCS patients who have received a large volume of crystalloids. In our ICU resuscitation, we use albumin boluses after 6 l of crystalloid administration. An interesting footnote in the crystalloid vs. colloid debate is the use of hydroxyethyl starches (HES). In 2011, PRCTs by Joachim Boldt, MD, testing HES were retracted because of research misconduct. A more recent systematic review (excluding these studies) convincingly showed that HES is associated with increased AKI and mortality. As a result HES should not be used in shock resuscitation. It should also be known that HES has a US Food and Drug Administration (FDA) black box warning.

16.3.3 Type of Crystalloids

LR solution is preferable to NS because it provides a better buffer for metabolic acidosis, and large-volume NS causes a hyperchloremic acidosis, but to date clinical experience has not documented any differences in mortality between LR- and NS-based resuscitation. Recent studies have suggested that NS induces hyperchloremia that causes AKI, but a recent PRCT failed to prove this supposition [30]. However, NS is preferred in patients with obvious traumatic brain injury because it has a higher osmolarity and thus reduces cerebral edema. Additionally, NS is used when blood is being transfused because theoretically the calcium in LR could exceed the chelating capabilities of the citrate in the stored blood, resulting in the formation of clots that could enter the circulation and compromise the microcirculation. Plasmalyte is an alternative isotonic crystalloid that contains magnesium instead of calcium and has what some consider more "physiologic" electrolyte concentrations [31]. Limited experimental data indicate that LR and Plasmalyte restored volume and correct electrolyte and acid-base deficits equally well [32]. However, there is the concern that the magnesium may counteract the vasoconstrictive response to hypovolemia and thereby increase mortality in severe hemorrhagic shock [31].

16.3.4 Blood Transfusions

In the past, a [Hgb] of 10 g/dL was believed to be the optimal level in critically ill patients. However, PRBC transfusions have been shown to be a strong independent risk factor for adverse outcomes (including infections, MOF, and deaths) across a broad spectrum of high-risk hospitalized patients [33]. Additionally, there are a number of plausible mechanisms to explain these adverse outcomes including suppression of cell-mediated immunity, elaboration of pro-inflammatory mediators that provoke neutrophil cytotoxicity, and decreased RBC deformity that impairs microvascular perfusion. As a result of concerns over potential harm, consensus panels lowered recommended level to a [Hgb] of >7 g/dL. This recommendation is supported by a recent PRCT where ICU patients randomized to restrictive blood transfusions (transfuse if [Hgb] <7 g/dL and maintain between 7 and 9 g/dL) did as well and possibly better than patients who were liberally transfused (transfuse if <10 g/dL and maintain between 10 and 12 g/dL). The choice of transfusion trigger is dependent on the clinical scenario. During active shock resuscitation with ongoing bleeding, maintaining for a higher [Hgb] is preferred. Blood is a good volume expander and will thus limit crystalloid infusions, and the higher [Hgb] provides a margin of safety, and higher [Hgb] levels may promote hemostasis. By our CCDS logic, we initially maintain [Hgb] >8 g/dL, and once bleeding is controlled, the transfusion trigger is lowered to 7 g/dL unless there is evidence of high oxygen extraction (i.e., $ScvO_2$<70%) in which case additional transfusion is warranted.

16.4 Correction of Coagulopathy

In the early 1980s, it was recognized that a substantial number of trauma patients with severe bleeding often died despite operative repair/control from ongoing coagulopathic bleeding caused by the "bloody vicious cycle" of acidosis, hypothermia, and coagulopathy. This recognition coincided with the seminal report by Harlan Stone in 1982 in which he first described the principles of DCS in patients found to have coagulopathic bleeding during emergency laparotomies. These reports spurred intense investigation into understanding and treating trauma-induced coagulopathy (TIC). Unfortunately, the exact pathogenesis of the TIC remains elusive and is beyond the scope of this discussion. However, by the late 1990s, it was recognized that critically injured patients arrive coagulopathic at the trauma center (TC) and that most of the deaths from exsanguination occur within the first 6 h. These data provided the rationale for starting massive transfusion protocols (MTPs) very soon after TC arrival.

Trauma-induced or trauma-associated coagulopathy may be seen in 25–41% of trauma patients on arrival to the hospital [34–36]. Trauma-induced coagulopathy may be caused by endothelial cell injury, a platelet defect, depletion of platelets and coagulation factors (depletion of factors I, II, V, VII, VIII, IX, and X), and an expenditure of platelets and factors in hyperfibrinolysis or disseminated intravascular coagulation [34, 37–39]. TIC has been associated with a poor prognosis, including increased incidence of multiple organ failure and death [35, 40]. A bleeding trauma patient may have the immediate trauma-induced coagulopathy compounded by the development of a consumptive coagulopathy. Porcine trauma models have demonstrated that hemorrhage impairs clot strength [41]. The development of an immediate coagulopathy has also been associated with traumatic brain injury [42].

Hypothermia negatively affects the coagulation system. Factors XI and XII have been demonstrated to be less effective at 35 °C [43]. Hypothermia has been demonstrated to both induce and decrease platelet activation [44–46]. Valeri and associates demonstrated that hypothermia affects a platelet's ability to produce thromboxane B2, causing a prolongation of bleeding time [47]. The liver and spleen sequester platelets in the setting of hypothermia [48]. Trauma porcine models have demonstrated that hypothermia does inhibit clot formation time and clotting time, but does not impair maximum clot firmness [41, 49]. Coagulopathy will persist despite transfusion to an adequate platelet count if the hypothermia remains uncorrected [50]. Coagulopathy associated with hypothermia will not manifest as prolongation of prothrombin time (PT) and activated partial thromboplastin time (APTT) because these laboratory tests are performed with the sample warmed to 37 °C and so do not demonstrate coagulopathy in a hypothermic patient [38, 51]. Experimental work and subsequent clinical observations indicated that ratios of FFP to RBC <1:2 were warranted to avoid factor deficiency, but platelets were not justified with initial PRBC transfusion. Recently, the military has promulgated a preemptive platelet to FFP to RBC ratio of 1:1:1 based on the argument that this represents the composition of shed whole blood. The debate over the optimal ratio of components in empiric MTPs is still ongoing and is not pertinent to this discussion because in the ICU after DCS blood components should be guided by laboratory determinations. This emphasizes the need for a rapid comprehensive assessment of coagulation which thromboelastography (TEG) provides. This technology of real-time viscoelastic analysis of blood clotting assesses clot strength from the time of initial fibrin formation to clot retraction, ending in fibrinolysis. In contrast, the standard battery of coagulation studies (PT, PTT, and bleeding time) is based on isolated, static end points. Rapid TEG is further advantageous as the addition of tissue factor to the whole blood specimen stimulates a rapid reaction. The resulting coagulation tracing can be transmitted to the ICU as a functional profile graphic within 5 min of blood sampling. This point-of-care monitoring offers near real-time determination of the impact that specific component therapy is having on coagulation. TEG goal-directed hemostatic therapy has become the standard of care in MTP resuscitation in many

trauma centers, and clinical studies have shown that it can significantly reduce unnecessary blood component therapy [52].

16.4.1 Recombinant Factor VIIa (rFVIIa)

Recombinant factor VIIa (rFVIIa) is currently licensed for treatment of patients with congenital factor VII deficiency and patients with hemophilia and inhibitory autoantibodies. However, it has been used off-label for the prevention and treatment of bleeding in patients without congenital factor VII deficiency or hemophilia. A recent Cochrane review assessed randomized controlled trials comparing rFVIIa to placebo or comparing different doses of rFVIIa, including trials that utilized rFVIIa as a prophylactic agent or as a therapeutic agent. The final assessment was that data was not sufficient to prove the effectiveness of rFVIIa as a general hemostasis drug [53].

16.4.2 Tranexamic Acid (TXA)

Hyperfibrinolysis has been associated with increased mortality [54]. Hyperfibrinolysis has been defined by a LY-30 equal to or greater than 3% on thromboelastography (TEG) [55, 56]. The CRASH-2 trial incited interest in the preemptive administration of TXA to injured patients; however the study methodology made it difficult to apply broadly. At this time a rational approach based on current data and expert opinion would be to limit the use of TXA to trauma patients with severe bleeding with fibrinolysis (i.e., TEG with a LY-30 equal to or greater than 3%). TXA should be administered within 3 h of the injury as it has been shown that the use of TXA after 3 h of injury resulted in increased mortality [57].

16.5 Hypothermia

Hypothermia is an important component of the "bloody vicious cycle" of death because it impairs coagulation, decreases cardiac output, and causes cardiac dysrhythmias. Additionally, it is a strong predictor for adverse outcomes including MOF, prolonged ICU stays, and death. Unfortunately, reversing hypothermia during active shock of a DCS patient is quite difficult; therefore, the focus needs to be on prevention. This starts in the emergency department with placement of a urinary catheter that can monitor temperature (T). After initial trauma evaluation is complete, the exposed body is covered with warmed blankets, and fluids and blood products are infused via fluid warming devices (e.g., Level I fluid infuser). Initiation of warming strategies that begin preoperatively is more effective [58, 59]. In the operating room, the room is warmed, forced warm air blankets are applied, the head is covered with a heat reflecting cap, and mechanical ventilation is provided with warm (38 °C), humidified air. Patients who received warmed intravenous fluid maintained their core temperature approximately 0.5 °C higher than patients' given room temperature intravenous fluids [60]. Forced-air warming maintains core temperature approximately 0.5–1 °C higher than extra thermal insulation [61]. However, if the forced-air warming blanket becomes wet, it will inadvertently cool the patient [62]. Humidifying and warming the inspired gases in the ventilator circuit may not prevent hypothermia, but it has been demonstrated to slow the decrease in temperature [63]. Adjuvant measures include warming the ambient temperature of the operating room, warming the irrigation fluids, and wrapping the patient's head. If the patient becomes hypothermic despite the aforementioned preemptive measures to prevent hypothermia, then strategies like active intravascular rewarming have been shown to be successful [64]. Cardiopulmonary bypass has also been used successfully [65]. While lavage of the open abdomen and irrigation of chest tubes with warm fluid were commonly used in the past, they are relatively ineffective in transferring heat and rarely used in short-duration DCS. Once the patient arrives in the ICU, the same measures are applied. In our experience utilizing the above measures in 95 DCS patients requiring a MTP hypothermia was not a significant problem. At ICU admission, mean T was 35.4 ± 0.1 °C, 27 (28%) had a T

between 32 and 35 °C, and only one (1%) had a T <32 °C. Additionally, mean T increased rapidly to >37 °C during the first 4 h of resuscitation and remained normal thereafter.

16.6 Acidosis

Acidosis is an important component of the "bloody vicious cycle" of death because it impairs coagulation, decreases myocardial contractility, promotes cardiac dysrhythmias, and inhibits oxidative metabolism and desensitized adrenergic receptors. Acidosis is a strong independent predictor of adverse outcomes including MOF, prolonged ICU stays, and death. Acidosis is caused primarily by a rise in lactic acid production secondary to tissue hypoxia and usually resolves when the volume deficit is corrected. Administration of sodium bicarbonate may cause a leftward shift in the oxyhemoglobin dissociation curve, reducing tissue oxygen extraction, and it may worsen intracellular acidosis caused by carbon dioxide production. Bicarbonate infusion, therefore, is limited to persons in protracted severe shock. In our experience utilizing the above measures in 95 DCS patients requiring a MTP, severe acidosis was common at the start of ICU resuscitation, with 26 (27%) patients having a pH between 7.0 and 7.2 but only three patients (2%) with a pH <7.0. With effective resuscitation and no sodium bicarbonate administration, the pH increased into the normal range within 8 h.

Conclusion

Damage control surgery (DCS) has changed the surgical and ICU approach to critically ill patients who arrive with severe bleeding. Following surgical interventions to prevent early death from exsanguination, the ICU goals are to reverse physiologic derangements and prepare the patient for a second operation where definitive operative treatment will be rendered (Table 16.1).

Table 16.1 Approach to intensive care following damage control surgery

	Approach	Goal(s)
Mechanical ventilation	Provide adequate analgesia and sedation	Assure patient-ventilator synchrony
Resuscitation end points	Implement a resuscitation protocol	Optimal end points have not been identified. A resuscitation protocol will assist in identifying nonresponders who need alternative interventions
Crystalloid vs. colloid	Initial resuscitation with crystalloid. Albumin used following administration of 6 l of crystalloid. Hydroxyethyl starches are not used in shock resuscitation.	Judicious use of crystalloid, knowing that there are negative consequences of large-volume crystalloid resuscitation. Safe use of colloid to limit crystalloid volume
Blood transfusions	Hgb is maintained >8 g/dL during active shock resuscitation with active bleeding. Once bleeding is controlled, the Hgb is maintained >7 g/dL	During active hemorrhagic shock resuscitation, a higher Hgb provides a margin of safety, and blood will provide volume and limit crystalloid resuscitation. Otherwise, a restrictive approach to blood transfusion has been demonstrated to be safe and may be beneficial
Correction of coagulopathy	TEG goal-directed hemostatic therapy	Significant reduction in unnecessary blood component transfusion
Hypothermia	Prevention of hypothermia	Reversing hypothermia in the setting of hemorrhagic shock is very difficult. The goal is to prevent hypothermia from occurring
Acidosis	Focus on resuscitation	Limit bicarbonate infusions to patients in protracted severe shock and acidosis

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17 Damage Control Phase III: Repair of All Injuries, General Surgery

Babak Sarani and Patrick Maluso

17.1 Goals and Timing of Damage Control Phase III

The primary goals of the third phase of damage control surgery (DC III) are to achieve definitive repair of organ injuries and to close the fascia over surgical wounds where possible. Although the optimal timing is variable and dependent on numerous patient factors, DC III is typically undertaken 24–36 h after the initial surgery. This time is needed for appropriate resuscitation, allowing the patient to reestablish proper homeostasis. The patient will thereby tolerate the longer operative time and more extensive intervention(s) that may be necessary to definitively repair the injuries sustained. Specifically, the decision to proceed with DC III should not be undertaken until the patient's coagulopathy has been corrected and he/she is normothermic and has a normal acid-base balance. Additional considerations such as vasopressor requirements also impact on the timing and probability of success of DC III. Ideally, patients should be weaned off of vasopressors entirely or, at a minimum, their pressor requirements should be decreasing. Ongoing physiologic instability or hypothermia despite appropriate medical therapy should raise concern for a missed injury. Definitive repair should be delayed but early operative re-exploration to evaluate for missed injury may be warranted. On-demand repeat laparotomy in these cases can decrease patient mortality [1].

Some notable scenarios exist wherein earlier timing of definitive repair is potentially favorable. For example, in peripheral vascular injury, thrombosis of a temporary shunt and subsequent potential for tissue loss may motivate earlier initiation of DC III. There are no studies assessing the maximal time that bowel segments can be left in discontinuity, but most trauma surgeons recommend creation of anastomoses or stoma to decompress isolated segments of the intestine no later than 96 h following DC I. It is logical to assume that earlier anastomosis or creation of a stoma is preferred, as long as the patient has been appropriately resuscitated and normal physiologic milieu reestablished.

17.2 Repair of Injuries: General Considerations

Once in the operating room, the patient should be positioned, prepped, and draped to ensure adequate exposure of all injuries to be addressed. This may include extremity exposure sufficient to allow proximal and distal control of vascular injuries and to allow for autologous vein harvest. In cases where a single position to undertake all

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necessary repairs is not possible, phases of the operation should be planned preoperatively. The surgeon can also elect to stage the procedures based on the patient's physiologic reserve and status. Communication and coordination with the anesthesia team both before and during the operation are vital in making these decisions, and the surgeon must be prepared to adjust the operative plan if the patient shows signs of instability.

17.3 Repair of Vascular Injury

Vascular repair in DC III involves removal of shunts placed in DC I and placement of interposition grafts. Although vascular surgery can be consulted to assist with definitive repair, excellent outcomes have been reported following vascular repair by experienced trauma surgeons [2]. As with elective vascular operations, proximal and distal control is obtained using atraumatic clamps. Next, the vessel wall should be inspected for damage to the intima or muscular layers and should be debrided as necessary. The portion of the vessel that was used to tie and fix the shunt in place should be resected. If the ends of the vessel are not long enough to support a tension-free end-to-end anastomosis, an interposition graft should be fashioned. Prior to completion of the anastomosis, care should be taken to remove any intraluminal thrombus. The proximal vessel is allowed to bleed for several pulses, while the distal vessel may be allowed to backbleed in order to remove thrombus. A Fogarty balloon catheter should be used to remove clot. The full technique for performing a vascular anastomosis is standardized, and full details are beyond the scope of this chapter. Use of extraanatomic bypass may be necessary when autologous sources are unavailable and contamination is severe.

The choice of conduit is an important consideration in planning repair of vascular injuries. The choice between autologous conduit (commonly a reversed saphenous vein graft) and synthetic alternatives such as polytetrafluoroethylene (PTFE) remains controversial. Data regarding where synthetic grafts can be safely used following damage control surgery are conflicting, and there are no definitive, well-designed trials upon which to base strategy. In a review of surgical experiences surrounding the Korean and Vietnam conflicts, synthetic grafts were found to have a 77% complication rate and a higher incidence of amputation than vein grafts (31% vs 18%) [3]. The small numbers of prosthetic grafts placed and high energy wounding patterns limited this review. A study of 206 patients with vascular trauma demonstrated that PTFE had lower rates of long-term patency than autologous grafts, but that infectious complications only occurred in the setting of exposed graft and concomitant osteomyelitis [4]. Similarly, a small study using a canine model and a small number of trauma patients found that the use of vein conduits in contaminated wounds was associated with a greater incidence of vascular disruption than PTFE; however, the small numbers involved in the study limit its generalizability [5]. In a subsequent retrospective review, Mitchell and Thal concluded that fears of anastomotic dehiscence following infection of vein autograft were overstated and found vein to be a safe conduit in vascular trauma [6].

In the absence of definitive data comparing synthetic with autologous grafts in trauma patients, the choice of conduit should be informed by careful consideration of patient factors, which include the caliber of the damaged vessel, location (extremities versus trunk), and amount of contamination present within the wound. Large vessels can be treated with either prosthetic graft or cadaveric homografts [7, 8]. While use of synthetic grafts allows for speedier operation and obviates the need to create more wounds, use of autologous material or homograft may be associated with lower infection and therefore anastomotic dehiscence rates. Regardless of the choice of conduit, every attempt should be made to cover the anastomosis in order to prevent complications such as pseudoaneurysm formation or actual free disruption and life-threatening hemorrhage.

17.4 Repair of Injuries: Solid Organ Abdominal Injuries

Following removal of the temporary abdominal closure device, the abdominal contents should be carefully inspected with particular attention paid to

any repairs made during the index operation and for identification of possible missed injuries. Special consideration is required in patients following ballistic injuries as the zone of injury can extend beyond the direct path of the projectile, resulting in a delayed manifestation of the true extent of injured tissue. This may necessitate further bowel resection to allow for safe anastomosis.

Similarly, the severity of pancreatic injury may be better assessed during this phase because small volume leak from the pancreatic substance will manifest as saponification. This finding will frequently lead the trauma surgeon to drain the pancreatic bed or consider pancreatic resection, depending on the nature of the injury and timing since injury [9].

Severe hepatic injury is often initially managed with packing and embolization or ligation of bleeding vessels. At the time of DC III, depending on the location and severity of the injury, resection of devitalized segments of the liver may be necessary. Whereas most surgeons avoid hepatic resection as much as possible, one study found that an aggressive debridement strategy resulted in a significant decrease in the overall number of procedures as well as complications. However, this approach was also associated with a significant risk of intraoperative hemorrhage [10]. At the least, the presence of devitalized hepatic tissue or deep laceration should raise concern for a postoperative bile leak or abscess, and the region should be drained preemptively.

The right hemidiaphragm should be inspected in any injury pattern that includes significant hepatic injury. Diaphragmatic defects should be repaired as best as possible in order to decrease the risk of biliopleural fistula [11]. Although rare, formation of a biliopleural fistula is associated with the need for prolonged tube thoracostomy and possible respiratory failure due to inflammation of the lung [12].

17.5 Repair of Gastrointestinal Injury

The repair of gastrointestinal injuries during DC III is a complex issue. Although creation of a small bowel anastomosis is generally considered to be safe and appropriate in DC III, creation of an anastomosis involving the large intestine requires careful consideration. Overall, the literature favors primary repair and the creation of delayed anastomosis after damage control laparotomy but with due consideration of known risk factors for anastomotic leak. In one meta-analysis of randomized controlled trials, primary repair had lower morbidity and procedure-related cost than diversion with no difference in mortality [13]. A similar multicenter prospective randomized study of 297 patients found no difference in abdominal complications between the ostomy and primary anastomosis groups, irrespective of associated risk factors [14]. A single-institution review of patients with colonic injury following penetrating trauma found that 81% were successfully treated with delayed anastomosis of their injuries following damage control laparotomy but found that persistent metabolic acidosis or intraabdominal contamination were risk factors for leak [15]. Other smaller studies also support this finding, demonstrating that delayed anastomosis and immediate anastomosis have similar complication rates [16]. Recent military data also have shown no difference in complications between delayed anastomosis and diversion [17]; however, one study found lower complication rates in patients treated with an immediate ostomy than those who underwent damage control [18]. Conversely, other studies have found that delayed colonic anastomosis has higher complication rates than seen with anastomoses created in a single laparotomy, suggesting that the open abdomen is particularly deleterious to the viability of colonic anastomoses [19–21]. Although there is no clear consensus, overall these studies suggest that delayed anastomosis is a viable and safe management strategy for colonic injury within the damage control sequence.

Following creation of an anastomosis, studies suggest a significant risk of enteral leak if the abdominal wall is not closed. While a perceived inability to definitively close the abdomen should not rule out anastomosis during DC III operations, the risk of leak and fistula formation with a persistent open abdomen must be taken into consideration. If the abdomen is left open following

definitive repair of bowel injury, conventional teaching involves attempting to minimize these risks by covering anastomoses with omentum or bowel. Studies supporting this intervention involve esophageal anastomoses [22]; there are no studies upon which to base this strategy following damage control operation.

Potential for abdominal closure is not the only important determinant of optimal surgical management of bowel injuries. The decision between creation of an anastomosis during DC III and formation of an ostomy should be undertaken after consideration of the patient-specific risk factors. Factors such as the presence of significant medical comorbidities, tobacco use, and malnutrition present well-known risks for anastomotic leaks [23–25]. Other risk factors that are especially relevant to trauma patients and to patients undergoing damage control laparotomy include ongoing malperfusion due to sepsis, cardiopulmonary failure, or any other cause as well as perioperative blood transfusions [26]. Unfortunately, there are few studies evaluating risk factors specifically due to delayed anastomosis following injury. Demetriades et al. found that anastomotic leak was increased in penetrating trauma patients with severe fecal contamination, transfusion of four or more units of blood within the first 24 h, and in those who received single-agent antibiotic prophylaxis perioperatively [27]. Another study of patients undergoing colon resection for cancer found that the probability of leak was independently associated with the American Society of Anesthesiologists grade and need for emergency operation [28]. Similarly, perioperative vasopressor requirements are associated with a more than fourfold increase in anastomotic leak rates [29]. These findings underscore the importance of appropriate timing of DC III operations. If patient risks cannot be adequately managed in a timely fashion, bowel anastomosis should not be undertaken at the time of DC III operations, and instead an ostomy should be formed with closure of the abdomen. Alternatively, the timing of DC III should be delayed.

Patients with traumatic brain injury rely on maintenance of normal cerebral perfusion pressure, and in these patients any septic insult and subsequent depression of blood pressure can be especially deleterious. Because of their inability to tolerate the possible septic complications of an anastomotic leak, patients with TBI and risk factors for leak should be managed with an ostomy rather than attempt at anastomosis during DC III operations. Similarly, elderly or chronically ill patients with little physiologic reserve are less likely to survive the septic complications of an anastomotic leak, and preference should be given to formation of an ostomy in these cohorts.

The anatomic location of the injured bowel is important in determining whether to attempt anastomosis. Proximal small bowel injuries should be managed during DC III with anastomosis whenever possible in order to maximize the amount of bowel available for absorption and to reduce complications related to malnutrition and electrolyte shifts during recovery. Although not specific to trauma, the colorectal surgical literature demonstrates higher leak rates for low colonic and rectal anastomoses, suggesting that these injuries should be treated with creation of an ostomy or at least protected initially by a proximal diverting stoma [26]. Similarly, left-sided anastomoses present a risk factor for anastomotic leak [19].

Creation of a stapled versus hand-sewn anastomosis during DC III remains controversial. One multicenter retrospective study showed a slight increase in leak rates for stapled anastomoses in trauma patients [30], and another study showed a more than twofold increase in risk for anastomotic leak in stapled anastomoses in emergency general surgery patients [31]. The possible reason to account for this involves bowel edema which may preclude adequate sealing of the two loops of the intestine using a stapler. However, a prospective multicenter study of trauma patients showed no difference in outcomes between the two techniques [27]. Furthermore, while not specific to trauma patients, one randomized prospective study of 652 patients found no significant difference in leak rates between stapled and hand-sewn anastomoses [32], and a larger prospective study of 1,417 patients with anastomoses above the peritoneal reflection also found no difference in leak rates [28]. Ultimately, the

surgeon's technical proficiency with each technique should determine the operative approach to anastomosis with the understanding that the operative strategy must be tailored to the patient and the condition of the tissues.

If creation of an anastomosis during DC III is ruled out based on any of the above factors, stoma formation should be undertaken. When creating an ostomy, the site should be chosen in order to minimize potential for fecal contamination of the midline laparotomy wound and subsequent necrosis of the abdominal fascia. Choice of an ostomy site 3–4 cm lateral to the lateral edge of the rectus abdominis muscle in order to reduce proximity to the midline laparotomy wound may help to minimize risks associated with spillage of intestinal contents into the surgical wound and will facilitate application of stoma appliances without interfering with midline wound dressings. Furthermore, such lateral placement of stomas will facilitate component separation closure of the abdominal wall should the patient develop a hernia.

Lastly, consideration for placement of feeding tubes or tubes to decompress the alimentary tract is appropriate during this phase of damage control operation. The overall care plan must now also include how to allow the patient to convalesce. Durable enteral feeding access should be considered prior to abdominal closure as gastrostomy or jejunostomy tube placement may be risk prohibitive after DC III. When placed, the tubes should exit the abdominal wall well away from the lateral border of the rectus abdominis muscles so as to minimize risk that a leak will contaminate the midline wound. In addition, as discussed above, leaving the medial border of the external oblique muscle unviolated facilitates a later component separation operation.

17.6 Abdominal Wall Closure

Once all necessary operative goals are met, definitive abdominal closure should be attempted as quickly as possible to minimize the deleterious effects of an open abdomen. The length of time the abdomen is left open correlates directly with the incidence of complications and is inversely related to the probability of primary fascial closure [33]. If repeated attempts at fascial closure are unsuccessful, the abdominal wall should be closed using an inlay mesh or via intentional creation of a ventral hernia with skin-only closure. Planned future ventral herniorrhaphy may be necessary with either technique.

Primary fascial closure is the optimal method for closing the abdomen. This technique involves the direct approximation of the fascial edges and has the lowest incidence of hernia and enterocutaneous fistula formation following damage control laparotomy. Although complications are less likely with primary fascial closure, care must be taken to avoid excessive tension on the abdominal wall, as this can precipitate failure of the closure or may predispose patients to development of abdominal compartment syndrome [34]. Although it is the preferred method for abdominal wall closure, a ventral hernia will develop in up to 30% of these patients [35]. Although not studied well, primary fascial closure can be augmented with mesh reinforcement in an attempt to lower this risk [36, 37]. Permanent, synthetic meshes are relatively contraindicated in patients with risk factors for mesh infection such as wound soilage, and many authors recommend use of biologic mesh in these instances. More advanced techniques of fascial closure such as a separation of abdominal wall components laterally to allow for direct apposition of the fascia at the midline may be used, but a detailed discussion of these methods is beyond the scope of this chapter [38, 39].

If primary fascial closure is not possible, an alternative is functional closure by placing a mesh inlay as a bridge between the edges of the fascia. Most commonly, a biologic mesh is used due to concern about infection. The mesh acts as a scaffolding for ingrowth of native fascial tissue [40]. Once the mesh is placed, the skin is closed and drains can be placed to prevent seroma accumulation as needed. Unfortunately, the natural evolution of an inlay placement of biologic mesh is development of a "neo-hernia" due to stretching of the mesh over time. This can lead to patient dissatisfaction and need for repeat operative

repair. While the late incidence of ventral hernia formation after functional closure is not well established, one study using acellular dermal matrix found an 80% incidence over a mean follow-up of 21.4 months despite skin closure over the mesh [41].

If skin closure is not possible, inlay placement of biologic mesh should be avoided as exposed mesh will undergo degradation and dissolution until it is replaced by granulation tissue over the viscera. Rather, a less expensive dissolving mesh, such as Vicryl™, can be used. This process usually occurs over 3–6 weeks. The resultant granulation tissue will require skin grafting and will ultimately lead to a ventral hernia which can be repaired in 8–12 months when the inflammatory process in the abdomen has resolved and the viscera are again mobile [42]. This delayed ventral herniorrhaphy is frequently referred to as DC IV. The granulation phase, prior to skin grafting, is associated with up to 20% risk of developing an entero-atmospheric fistula formation. As noted above, this risk is highest in patients with an exposed anastomosis [43].

Conclusion

In conclusion, DC III refers to the phase of damage control related to definitive repair of injuries and closure of surgical incisions and wounds. The timing and method of repair need to be customized to each patient by taking into account patient- and injury-specific factors that impact on the probability of success.

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18 Phase III: Second Operation: Repair of All Injuries, Orthopedic Surgery

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18.1 Introduction

Definitive fixation of major fractures may occur within 24 h in stable patients or in borderline ones that respond adequately to resuscitation, as mentioned in Chap. 10. In those patients submitted to damage control procedures within the resuscitation period, such as external fixation or traction, the second stage of fixation can be planned thoroughly according to the patient status.

In this light, it is well described that a patient's competence to endure a surgical procedure is determined by the interplay between the time after injury, the magnitude of the operation, and the patient's individual physiological response. It is a prerequisite to respect local tissue and wound factors such as swelling, skin condition, or lesions located close to the planned surgical incision.

Among the factors responsible for influencing the physiological response, cardiovascular, respiratory, and immunologic factors are known to play a role. The magnitude of surgery – such as secondary osteosyntheses of the femur, pelvis, and spinal column – can represent an additional burden. Major surgeries have been described to be associated with systemic complications, such as SIRS and organ failure, if performed at the wrong time, or in a patient that is not yet ready to tolerate the surgical impact. In a clinical study, a new onset of organ failure or deterioration of existing SIRS occurred in almost half of patients undergoing major surgeries, while only 32% demonstrated complications after minor surgeries [1].

Interestingly, the clinical deterioration did not entirely correlate with the duration of the surgery or the degree of intraoperative hemorrhage. In contrast, some patients appeared to have present with a hyper inflammatory state prior to the procedure, which was discussed to play a role in the development of their complications.

The principles described below have to be viewed with respect to the limitations of clinical studies: The separation of the effects of surgery from other factors in polytrauma patients represents a challenge. Well-defined patient populations are required, and inclusion of adequate patient numbers appears to be difficult to achieve.

18.1.1 Effects of Surgery: Time and Magnitude

Historically, the postsurgical response is well described: An inflammatory and hypermetabolic

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state occurs after every major surgery. Early descriptions by Billroth demonstrated delays in wound healing and an increased risk of infection. Following these descriptions of the metabolic answer, it became clear that more complex mechanisms occur [2, 3].

Similar mechanisms occur after surgical or accidental trauma: The nervous system activates the hypothalamus – pituitary axis, which or releases hormones such as cortisol, glucagon, catecholamines, and numerous inflammatory cytokines [4].

According to our current understanding, the surgical stress response has three key components [5]:

1. Sympathetic nervous system activation
2. Endocrine response with pituitary hormone secretion and insulin resistance
3. Immunologic and hematologic changes including cytokine production, acute phase reaction, neutrophil leukocytosis, and lymphocyte proliferation

18.1.1.1 Sympathetic Nervous System

The sympathoadrenal response directly modifies the function of numerous organs, including the liver, pancreas, and kidney. Gluconeogenesis is increased, glucagon production is stimulated, and water is retained to maintain fluid volume and cardiovascular homeostasis.

18.1.1.2 Endocrine Response

During the endocrine response, increased catabolism mobilizes substrate to provide energy and retention of salt and water to maintain fluid volume and cardiovascular homeostasis. Specifically, corticotrophin stimulates cortisol secretion from the adrenal cortex resulting in increased blood glucose levels, arginine vasopressin stimulates the kidney to retain water, and insulin secretion by the pancreas is often diminished [5].

18.1.1.3 Inflammation

Inflammation appears to be the most important parameter in terms of changes induced by secondary surgeries. A hyperinflammatory state is known to occur in every polytrauma patient. Subsequently, depending on the general condition and the severity of the injury, a compensatory phase (CARS) has been discussed. It may be followed by a period of anergy. Whether or not the anergic phase develops and whether there are any clinical consequences are difficult to foresee, and the determining factors are discussed below. Recently, the "Inflammation and the Host Response to Injury Collaborative Research Program" has gathered a large prospective data set on early patient assessment in patients with blunt multiple trauma. The information collected by this group clearly shows the relevance of numerous clinical parameters and biomarkers for prediction of clinical complications, such as pulmonary dysfunction. Moreover, the data underline the special meaning of the trauma-induced inflammatory response as an independent risk factor for adverse outcome. The authors discuss that many changes are set at the time of injury [6].

18.1.2 Clinical Studies on the Relevance of Biochemical Markers

Inflammatory markers appear to be relatively stable indicators for the assessment of the clinical course. Roumen's classical publication compared several inflammatory mediators and highlighted that Il-6 is most specific for trauma patients, while TNF-α and Il 1-ß demonstrated a greater accuracy in patients with hemorrhage and in non-survivors after ARDS and MOF [7]. Our group demonstrated that early elevated Il-6 levels were able to discriminate trauma patients who later develop MOF [8]. In patients undergoing orthopedic operations, interleukin-6 serum levels have been demonstrated to be closely related with the magnitude of the injury (burden of trauma) and with the operative procedure (second hit) [9]. Il-6 concentrations vary according to the type of orthopedic surgery, as the inflammatory response induced by femoral nailing is biochemically comparable to that induced by uncemented total

hip arthroplasty. In contrast with Il-6 and 8, the clinical significance of other cytokines is controversial. While Smith et al. described an Il-10 increase after major surgery [10], Hensler depicted noticeably defect of its secretion [11]. Therefore, Il-10 may not be an adequate parameter to monitor the severity of trauma. Likewise, tumor necrosis factor alpha is unable to quantify the burden of blunt trauma or of surgical procedures [12].

We previously looked at the timing of major secondary surgery and investigated whether the inflammatory response is different when early (days 2–4 after injury) or late (after day 4 following injury) surgery is performed. In 128 polytraumatized patients, *s*econdary surgery on days 2–4 was associated with a higher incidence of postoperative organ dysfunction (46.5%) than secondary surgery on days 5–8. An association between the combination of initial Il-6 values > 500 pg/dl surgery on day 2–4 and the development of MOF ($r = 0.96$, $p < 0.001$) was also observed. In contrast, no correlation between the initial Il-6-values > 500 pg/dl and surgery on days 5–8 ($r = 0.57$, $p < 0.07$) was discernable. Therefore, it was concluded that no distinct clinical advantage in carrying out early (day 2–4 days) secondary definitive fracture fixation could be determined. In contrast, in patients who demonstrated initial Il-6 values above 500 pg/dl, it was evident that it may be advantageous to delay the interval between primary temporary fracture stabilization and secondary definitive fracture fixation for more than 4 days. The main conclusion was that in patients with blunt multiple injuries undergoing primary temporary fixation of major fractures, the timing of secondary definitive surgery should be carefully selected, because it may act as a second-hit phenomenon and cause a deterioration of the clinical status [13]. This assumption is supported by numerous studies:

Tanaka et al. reported that superoxide production by neutrophils (PMNLs) stimulated with concanavalin A and cytochrome d was elevated in trauma patients at day 3 after trauma and returned to normal by day 7 [14]. Botha et al. demonstrated an earlier window of PMNL priming. The in vitro oxygen radical release by unstimulated PMNLs was *resolved by 72 h*. The authors concluded to "postpone avoidable second hits" [15].

Ogura et al. demonstrated a significant priming of PMNLs in trauma patients between days 2 and 13. This reaction was most sustained at days *2–5 after the injury* and was thought to be related to the length of the inflammatory response. The authors also investigated the effect of a second hit on inflammatory parameters in trauma patients. Their measured IL-6 levels demonstrated a 2.5-fold elevation after a second hit and a significant increase in the FMLP response in PMNLs after a second hit [16]. The manifold interactions between different inflammatory cells and their secreted mediators are well described. Johnson et al. reported that IL-6 augments the cytotoxic potential of neutrophils via selective enhancement of elastase release [17].

Recently, the importance of both, biochemical and clinical markers has been stressed. The EPOFF study group looked at these issues and reconfirmed that multiple clinical parameters are important. Among these are lung function, coagulation, parameters of hemorrhage, and soft tissue injuries [18]. Others have investigated clinical predictors of complications and compared them with biochemical markers (Acute Physiology And Chronic Health Evaluation III [APACHE III], organ failures, age, underlying cause, alveolar-arterial oxygen gradient, plateau pressure) predicted mortality with an area under the ROC curve (AUC) of 0.82; a combination of eight biomarkers and the clinical predictors had an AUC of 0.85.

Their best biomarkers were the neutrophil chemotactic factor, IL-8, and SP-D, a product of alveolar type 2 cells. The authors focused particularly on pulmonary complications. It is therefore not surprising that they suggest acute inflammation and alveolar epithelial injury as important pathogenic pathways in human ALI/ARDS [19]. Moreover, they conclude that a combination of biomarkers and clinical predictors is superior to clinical predictors or biomarkers alone for predicting mortality and may

be useful for stratifying patients in clinical trials.

In summary, the numerous clinical and experimental investigations regarding the inflammatory response support the theory that the timing of major secondary surgery does affect the clinical course in patients with blunt trauma.

18.1.3 Clinical Studies on the Effects of Secondary Surgery

The EPOFF study groups have gathered a lot of information on the timing of primary and secondary surgeries. They combined the search for clinical and biochemical parameters [20, 21].

Some of their findings were based upon a review of 4,314 polytrauma patients, where patients with blunt trauma and multiple surgeries were differentiated. Those patients that were submitted to major secondary surgery at days 2–4 after the initial injury demonstrated a higher incidence of organ dysfunction than those operated between days 5 and 8 [22]. Similar observations were made in a prospectively documented cohort study [1]. The authors investigated a well-defined cohort of 133 multiply injured patients that had a well-selected high Injury Severity Score. In these patients, the authors describe a deterioration of the clinical status in as many as 38% of cases. One of the major common findings in these patient groups was that the deterioration of organ function parameters began within a period of 48 h after surgery and after inflammatory parameters had become abnormal. [23].

18.1.3.1 Clinical Parameters for Decision Making

Classical indicators for the surgical risk such as old age, previous cardiac or chronic respiratory illnesses are less important in the decision making for the operability for secondary surgery in polytrauma. Their prevalence is low within the usually young population of the polytraumatized. The classification of the "American Society of Anesthesiologists" (ASA), is equally low in suitability for this purpose since its variability is too high to be able to judge the surgical risk on a daily basis. There are no investigations in terms of the reliability of the clinical prognosis of an experienced surgeon.

When the clinical course is uneventful, signs of posttraumatic inflammation diminish usually within 48-72 hours. In patients who have a high risk for organ failure, the hyperinflammatory state can last longer and is clinically determined by three major factors. If these factors are present, patients are at high risk for secondary organ failure, and prolonged surgical interventions should be avoided (Tables 18.1 and 18.2).

Favorably, most patients do recover to a certain stage from their injuries and have a negative fluid ratio, normal chest x-rays, and normal cardiovascular status. In these patients, we have made the clinical observation that the fourth postinjury day appears to be earliest at which a prolonged non-lifesaving surgery can safely be performed.

Other authors focused mainly on the aspect of pulmonary dysfunction. This might be reasonable since ARDS used to be a major cause of death before the syndrome of multiple organ failure had been described. Certainly lung func-

Table 18.1 Time course of changes of biochemical markers

Author, year	Time after injury	Comment/explanation
Tanaka 1991	Days 3–7	PMNL stimulation elevated
Waydhas, 1996	First week	Inflammatory parameters react to surgery
Botha, 1995	Day 3	PMNL unstimulated, respond by hyperactivity
Ogura, 1999	Days 2–5	Priming PMNL increased, elastase release high
Giannoudis, 1999	First week	Il-6, TNF, proinflammatory response increased
Pape, 1999	First week	Il-6, TNF, RES increased
Hensler, 1997	First week	Il-10
Pape, 2000	Days 2–4 critical	Il-6 and Il-8
Pape, 2007	Day 1	Hyperstimulation

Table 18.2 Criteria to allow for secondary surgical fixation

PaO_2/FiO_2 ratio >250
PEEP <8 cmH_2O, each time for at least 24 h preoperatively
No additional infiltration in chest plain radiographs
Platelet count >95,000/μl
Leukocyte count >2000/μl or <12,000/μl
Balanced or negative fluid ratio for 24 h minimum before surgery
Intra-cranial pressure <15 cmH_2O, no signs of raised ICP in head CT
Serum creatinine <2 mg/dl
Serum bilirubin <3 mg/dl

tion (often assessed via the pO2/FiO2 ratio) does play an important role. Patients with close to normal lung function (pO2/FiO2 quotient >280 mmHg) withstood a standard procedure in 89% of the cases, while those with a worse respiratory function developed a post-organ failure in 70%.

A pO2/FiO2 ratio of <280 mmHg, a C-reactive protein of >11, and a platelet count of <180,000/μl were accompanied with a clearly increased incidence of a post-operational organ failure. As a further indicator, an increased level of the preoperative inflammatory response was indicated, shown by an increased elastase concentration in the serum (>85 ng/ml).

18.2 Secondary Orthopedic Stabilization: Technical Aspects

One of the major questions that orthopedic surgeons have to address after polytrauma is whether the techniques of fracture fixation should be the same as for isolated fractures. Fortunately these aspects appear to be similar regardless the severity of injury, provided that the soft tissue aspects and the general patient's condition are respected.

Some considerations however may be different. These are related to the principles of early patient's mobilization. To achieve this, some authors have recommended that fractures of the humerus shaft should be stabilized by an intramedullary nail in polytrauma. The rationale to do so is that mobilization with crutches is much more frequently obtained when surgical fixation is performed. Usually, all other orthopedic fixation techniques remain the same, provided that all soft tissue issues have been addressed (Table 18.3).

Regarding the conversion of an external fixation for the definite stabilization of major fractures, infection is of time concern. Numerous studies have shown that conversion should be performed within a 14-day time interval, in order to minimize the infection-related complications. The hospital-acquired infections can be extraordinarily severe.

Therefore, a conversion should be managed as if a superficial infection is present. We have studied this issue and developed a standardized protocol for secondary conversion of an external fixator [24]. We have also demonstrated that inclusion of this protocol leads to a reduction of infectious complications in these patients (Fig. 18.1).

Briefly, the steps involve:

- Preoperative holding area/OR: thorough cleaning with alcohol
- Removal of external fixation
- Prepping/draping
- Excision of pin sites by one surgeon/overdrilling of pin holes
- Thorough irrigation
- Change of gloves, equipment used for excision (drapes included)
- Definitive procedure

Conclusion

The pathophysiological sequences after accidental trauma show a biphasic process of the immunomodulatory response. The main peak of the inflammatory response usually occurs during the first 24–48 h (Fig. 18.2). Within the following

Table 18.3 Fixation principles for orthopedic injuries in polytrauma patients

Anatomic area	Technical aspects	Similar to isolated trauma condition	Different to isolated trauma condition
Proximal humerus	Plate osteosynthesis avoid hemiarthroplasty	+	
Humerus shaft	Intramedullary nail		+
Distal humerus	Locked plate (as in isolated fractures)	+	
Elbow	Cautious timing, beware of formation of heterotopic ossifications	+	
Forearm	May be delayed	+	
Wrist/hand	May be delayed	+	
Proximal femur	May be delayed, avoid THA or hemiarthroplasty (patients usually young)		+
Femur shaft	As early as patient condition permits, consider undreamed, plating, or ex fix		+
Distal femur	Delayed, technically comparable with isolated trauma	+	
Proximal tibia/ tibial plateau	Delayed, technically comparable with isolated trauma	+	
Tibia shaft	Early, technically comparable with isolated trauma	+	
Distal tibia	Delayed, technically comparable with isolated trauma	+	
Ankle/foot	Delayed, technically comparable	+	
Pelvis	Pelvic binder/C-clamp, consider least amount of blood loss		+
Acetabulum	Delayed, high infectious risk in ICU patients		+
C-spine	Early if neurodeficit	+	
Thoracic spine	Delayed unless neurodeficit		+
Lumbar Spine	Early, if neurodeficit		+
Sacrum	Delayed, high infection risk for ICU patients	+	

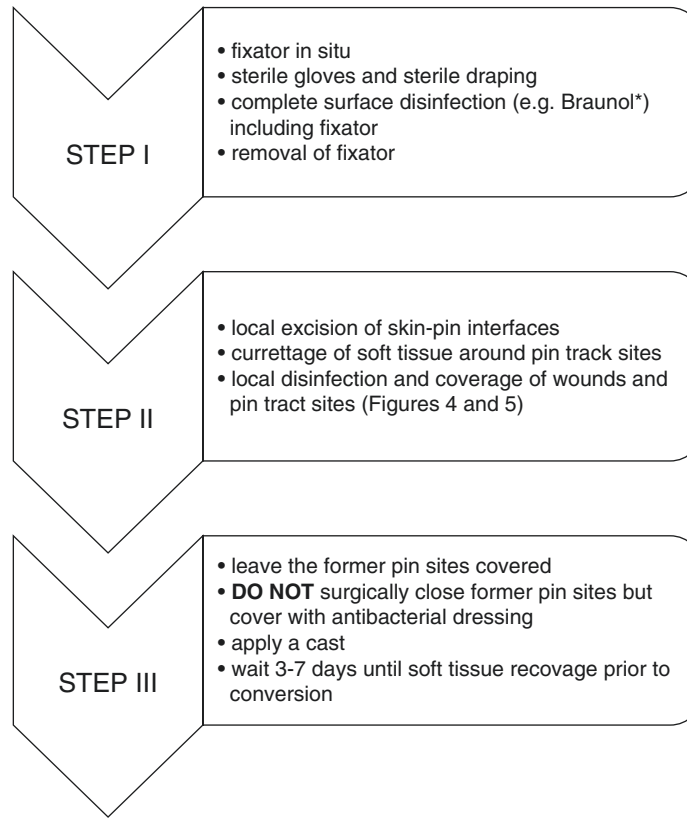

Fig. 18.1 Staged conversion of external fixator

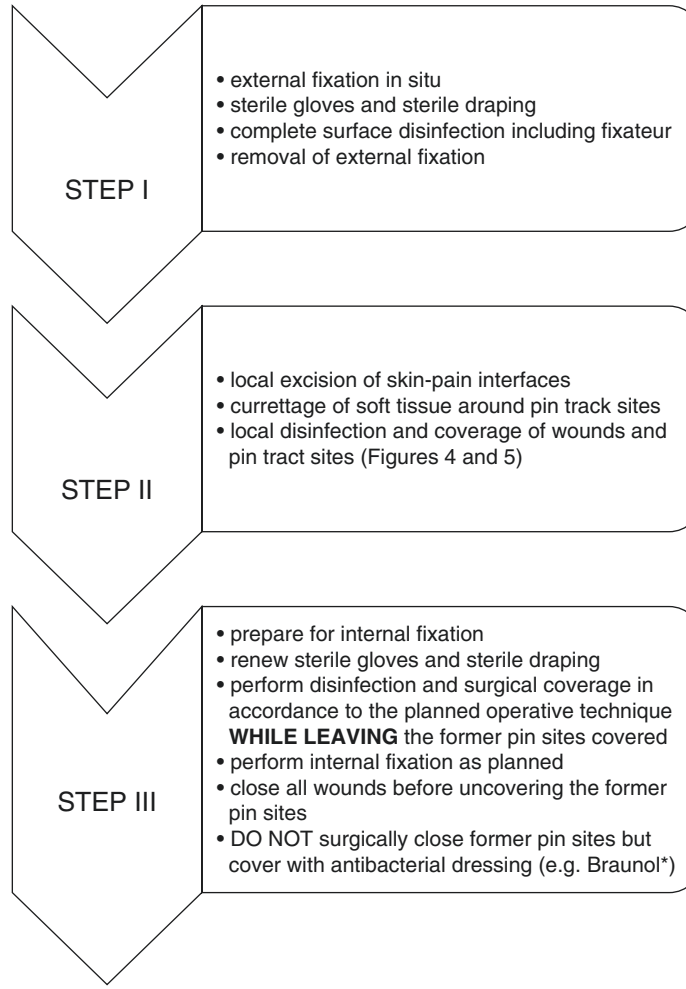

Fig. 18.2 Acute conversion of external fixator

week, surgical trauma through a secondary operation is shown to represent an additional burden. The parameters to examine are pulmonary function, normalization of hemostasis, a negative fluid balance, and absence of clinical signs of infection. The favorable days for major surgeries appear to be the days after the hyperinflammatory phase is over, i.e., after postinjury day 4 (Fig. 18.2).

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Phase IV: Late Reconstruction, Plastic Surgery for Orthopedics

19

LCDR Scott M. Tintle and L. Scott Levin

19.1 Orthoplastics

In 1919, Sir Harold Gillies wrote a pioneering textbook that has proven pivotal in the modern development of plastic surgery. The preface to his text was written by Sir W. Arbuthnot Lane, an orthopedic surgeon, thus beginning the publicized modern era of "orthoplastic surgery" [27]. Orthoplastic surgery is not just a term that should be casually used anytime a plastic and orthopedic surgeon work on the same patient for it does not define the true collaboration between orthopedic and plastic surgeons who are capable of seeing that bone, muscle, tendon, and skin are best treated compositely. One team does not fix the bone and the other "cover" it. Orthoplastic surgery incorporates early involvement of both orthopedics and plastic surgery with plans developed and followed by the team for the ideal treatment of the limb. Timing of fracture fixation and soft tissue coverage are optimized for the betterment of the composite limb and not for the schedule of the individual teams. When the focus is diverted erroneously to one tissue, frequently the result can be a perfused, possibly united, and covered but poorly functioning limb. When this team functions properly, a synergistic effect occurs, and all parties, most importantly, the patient, benefit.

19.2 Brief History of Microsurgery in Orthopedics

The collaboration of orthopedic and plastic surgery can be largely summarized by the history of microsurgery. The concept of triangulation of a blood vessel was introduced by Alexis Carrel in 1902 which allowed for successful end-to-end vascular anastomosis. The fields of vascular surgery, plastic surgery, and microsurgery were certain to develop following this profound discovery, for which Carrel would be awarded the Nobel Prize in Physiology or Medicine [39, 40]. With the development of the operating microscope and instruments and suture small enough in caliber to suture the 1 mm blood vessel, a revolution in every surgical discipline including orthopedic surgery followed.

The first *thumb* replantation was performed in Japan by Tamai in 1965, following *the first arm replantation* by Malt and McKhann in Boston in 1962 [40]. The 1960s ushered the era of Harry Buncke and his numerous microvascular experiments and multiple advances to the field of microsurgery which would eventually lead to the title of the "father of microsurgery." At this time, digital and limb replantation were largely the impetus driving the research and surgical efforts in the development of microsurgery. The concept

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of tissue coverage and pedicled flaps in which large masses of tissue were transferred on an axial artery and vein pedicle was introduced by McGregor in 1972 when he described the groin flap. Shortly thereafter, the free tissue transfer was reported by Taylor and Daniel, and the era of free tissue transfer was born [44].

The past 30 years has seen the evolution of microsurgery from the simple concept of replantation to chimeric flaps, functional muscle transfers, perforator, and even freestyle flaps. The advancements in microsurgery and the collaboration of orthoplastics have allowed for the concept of limb replantation and the fields of solid organ transplantation to come together in the field of vascularized composite allotransplantation in the hopes of complete restoration of function following major extremity amputations.

19.3 Orthopedic Trauma Surgery

Trauma remains the most common reason for the necessity of plastic surgery involvement in orthopedics. The orthoplastic approach is imperative for routinely successful outcomes in complex open fractures. The end-state goal is to obtain a non-infected union, healed soft tissues, and optimal functional outcomes. For any plastic surgeon working closely with orthopedic surgeons, it is imperative to understand the four basic surgical principles that the Arbeitsgemeinschaft fur Osteosynthesefragen (AO) group developed for the surgical treatment of fractures back in 1950. These principles remain paramount:

1. Anatomic reduction of fractures
2. Stable internal fixation
3. Preservation of blood supply with careful attention to soft tissue handling
4. Functional rehabilitation of the injured limb

It is not coincidental that two of the four principles, namely, the preservation of the blood supply and the rehabilitation of the injured limb, involve the soft tissues. These principles highlight the importance of preserving soft tissues in order to achieve fracture union and optimal outcomes [44].

19.4 Reconstructive Algorithm Principles

While initial treatment considerations have largely been discussed in previous chapters, it is imperative to highlight three paramount principles that we feel are important to long-term success in the treatment of these combined orthoplastic injuries:

1. Appropriate antibiotics are always started and continued until all wounds are closed or it is deemed appropriate to stop by the combined team.
2. Aggressive wound debridement is performed in the operating room every 48–72 hours until definitive wound closure is achieved. Early wound closure or flap reconstruction is generally performed as soon as is feasible.
3. In most cases, definitive wound coverage is performed at the time or immediately following the definitive internal fixation of fractures. Indications for internal fixation remotely prior to definitive wound coverage are rare. Flap coverage with external fixation and a delayed definitive fixation is also a strong consideration in select cases.

While presentation of all options for soft tissue coverage of the extremities is outside the scope of this chapter, the following briefly describes our treatment considerations and unique aspects of upper and lower extremity reconstruction.

19.5 Ladder of Reconstruction

The classic soft tissue ladder of reconstruction recommends using the simplest, least morbid technique to achieve soft tissue coverage. In our experience treating over a decade of war trauma and our combined many decades treating trauma at level 1 trauma centers, we have observed a shift toward using free flaps for a number of reasons when pedicled flaps may have been previously recommended [24, 47, 49]. The potential benefits of free tissue transfer are well described, and the soft tissue elevator has rapidly become

the more utilized concept for soft tissue reconstruction [7, 49]. This elevator concept is a simple change in thought process and suggests that the optimal method of coverage for the best functional outcome be utilized immediately even when bypassing more simple measures which may achieve closure. The elevator considers more carefully the continued reconstruction necessary, the end-state and rehabilitative goals. Clearly, more skill is needed as one rises up the reconstructive elevator, and this needs to be considered in the treatment planning. A unique consideration in the elevator concept is the use of biologic advancements that may simplify wound coverage techniques. These biologics can be used exclusively or more commonly as a combined approach to wound management along with other more traditional techniques [4].

We have found the use of bioartificial dermal regeneration templates to be very useful in the reconstructive efforts for patients with large wounds. These dermal matrices have an important role in the management of large, complex soft tissue injuries. These dermal regeneration templates provide initial wound coverage and help establish a well-vascularized wound bed suitable for definitive soft tissue coverage. These dermal substitutes have been used extensively in the treatment of burns, and more recently papers chronicling our war trauma experience have begun to highlight the success of these regenerative matrices [12, 37]. The use of a dermal substitute has multiple benefits in the treatment of large wounds, including: coverage of exposed tendons, nerves, blood vessels, and/or bone. The use of this matrix may make a wound smaller and may altogether eliminate the need for a tissue transfer. Additionally, the use of the substitute helps develop a well-vascularized bed. We have utilized the matrix to lessen the need for shortening of amputated extremities by providing more stable soft tissue coverage and durability [12, 17, 37]. The decision to utilize a dermal substitute in place of a tissue transfer is based on many factors. Clearly, the patient's disease state, comorbidities, available flap donor sites, other extremity injuries, and, realistically, cost all go into this decision. While the use of a dermal substitute beneath a split-thickness skin graft does improve the ability to raise a viable skin flap upon reoperation for secondary procedures (i.e., bone grafting, tendon reconstruction, nerve grafting), we frequently would proceed up the reconstructive elevator to a free tissue transfer when we anticipate these procedures (Figs. 19.1a, b and 19.2a–c).

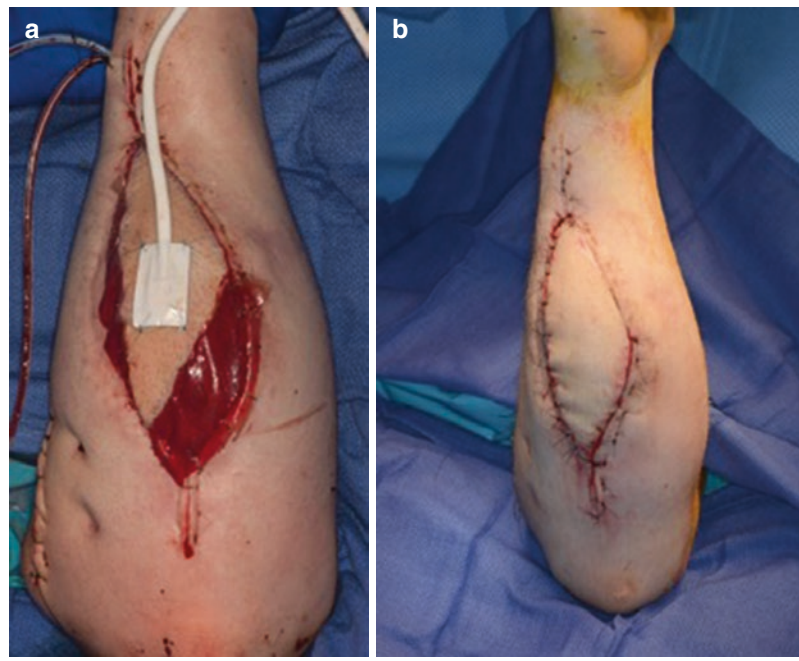

Fig. 19.1 (**a**, **b**) Osteocutaneous free fibula performed for large segmental defects of the radius and ulna. Muscle coverage achieved temporarily with integra dermal matrix followed by excision and closure of wound at 2 weeks from the original flap procedure

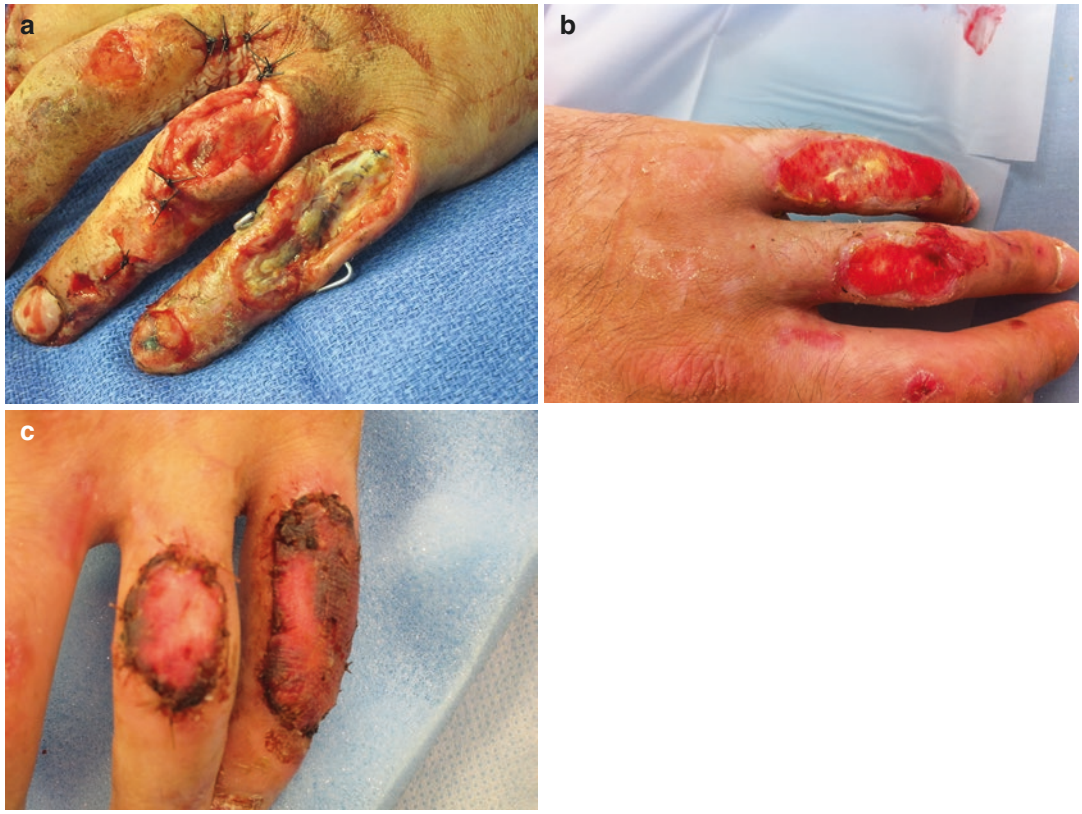

Fig. 19.2 (**a**) Open small and ring finger fractures with small finger extensor tendon repair covered with integra (picture pre-integra placement). (**b**) Granulation tissue after 3 weeks of integra placement. (**c**) Five days following full thickness skin graft

19.6 Timing of Soft Tissue Coverage

In 1986, Godina posthumously published his landmark paper on the soft tissue coverage of 532 patients with open wounds. This manuscript advanced microsurgery substantially and established the important soft tissue principles of early aggressive wound debridement, early free tissue transfer, and aggressive rehabilitation in order to optimize the functional outcomes [6]. While the concept of the emergency free flap was introduced, upon closer look at the work, the manuscript demonstrated no difference in the infection rates between the flaps that were performed at the first operative setting (emergency free flaps) versus those that were performed within the first 3 days of the injury. The paper did, however, demonstrate that the longer the flap was delayed, the longer the patients spent in the hospital and the higher the infection rates.

While the need for early radical debridement and soft tissue coverage with vascularized tissue is well established in civilian literature [1, 5, 6, 9, 10, 13, 23, 51, 53], the days of Marco Godina's team in Ljubljana are different from what exists in most healthcare systems today. The tireless team of dedicated microsurgeons who worked around the clock to provide early definitive emergency soft tissue coverage of open wounds is a rarity in today's healthcare environment.

While all efforts should be made in modern practice to get wounds covered rapidly, the military experience and that of other civilian providers has demonstrated acceptable outcomes with wound coverage in the subacute time period [15,

24–26, 50]. While the variables that lead to successful soft tissue coverage in the subacute time period remain ill defined, adherence to the established principles of radical debridement to create a clean wound bed and the use of healthy vascularized tissue have led to successful wound coverage with low flap failure rates and low rates of infection [47].

The literature is clear that successful results can be obtained in the subacute time period, but significant shortcomings in the reporting of open fracture data are apparent highlighting the inconsistencies of treatment algorithms. While most authors of these soft tissue coverage papers endorse a multidisciplinary approach (orthoplastic) to the management of these injuries, the data is often published in a specialty journal (orthopedics or plastic surgery) without adequately addressing the composite nature of the limb salvage protocol. It is not uncommon that the timing of and the type of initial and definitive fracture stabilization are not apparent from the data presented. This unfortunately makes it very difficult to comprehend the implications of the overall limb salvage protocols presented.

19.7 Flap Reconstruction

In our experience, two types of flaps are most useful in extremity reconstruction: fasciocutaneous and muscle. The debate over the optimal flap for open fractures is common and will not be easily answered. Many studies have shown the utility of muscle flaps in bone healing, but fasciocutaneous/perforator flaps have also reported high success rates, and both appear to be viable options for extremity coverage [2, 11, 32–35, 47, 48, 52, 53]. Fasciocutaneous flaps are utilized preferentially over muscle flaps in our practice frequently because they spare core strength which is essential to functional recovery of severely injured patients. Additionally the relative ease of flap elevation for secondary procedures such as bone grafting, tenolysis and/or tendon repairs, and delayed nerve grafting compared to muscle flaps is strongly desirable.

19.7.1 Unique Lower Extremity Considerations

In the lower extremity the reconstructive efforts are imperative in order to return patients to even primitive weight bearing and ambulation. Coverage of open tibia fractures is the prototypical case in which a plastic surgeon is involved in the care of an orthopedic fracture patient. This case specifically highlights the importance of a well-thought-out orthoplastic treatment algorithm. A recent review of IIIB tibia fractures at a level 1 trauma center revealed an overall infection rate of 36% when flap coverage was provided in the first 7 days. When coverage was provided later than 7 days after injury, the infection rate soared to 57%. The treatment protocol included the use of a wound vacuum device application following initial debridement and fracture stabilization frequently with an intramedullary nail, and then repeat debridement and irrigation every 48 h until definitive soft tissue coverage. What is not clear from this study, however, is the timing or method of definitive fracture fixation. The high rates of infection, even in the group that underwent flap coverage within 7 days, raise concern about the treatment protocol, and the long delay to flap treatment suggests poor orthopedic and plastic surgery collaboration. In our experience, successful salvage of lower extremities with severe open tibia fractures is possible only with close orthoplastic relationships [21]. All fracture stabilization and soft tissue procedures should be thoroughly coordinated by both the orthopedic and plastic surgeon with the best overall outcome in mind—as opposed to the usual focus on either fracture union or flap viability.

The foot and ankle have a highly specialized bony architecture and tissue properties that make coverage very challenging. It is important to consider the various regions of tissue coverage in the foot and ankle, and we frequently divide the tissues into dorsum, posterior, and plantar coverage. All have distinct properties and pose challenges for differing reasons. The dorsum of the foot is very thin and frequently requires free tissue transfer for coverage. This region requires

thin pliable tissue to cover the exposed bones, joints, and tendons that are devoid of paratenon. Fasciocutaneous flaps are frequently utilized for this coverage as this allows the replacement of thin, esthetic, "like with like" tissue that will hopefully allow for optimal shoe wear. The posterior aspect of the foot and ankle is considered a unique region because of the Achilles tendon and its important role to ambulation. It is common that the tendon has been compromised either by infection or trauma. This region is also a common region for wound dehiscence [22]. In situations where small flaps are necessary, the reverse sural is a viable option, but for larger flaps or in situations where the Achilles tendon has been compromised, we have utilized the anterolateral thigh flap and reconstructed the Achilles in a one-stage procedure as highlighted by Duhamel et al. [3] (Fig. 19.3a–d). One of the most specialized skin/adipofascial layers in the body is the plantar heel pad and the reconstruction of this region is very difficult. While various fasciocutaneous, muscle, and myocutaneous flaps have been described and utilized for this coverage, we have found it critical to have a muscle component of a flap for heel coverage. We consider the following all viable options for coverage of the heel: latissimus myocutaneous flap, rectus abdominis, vastus lateralis, or an anterolateral thigh flap with vastus lateralis muscle. It is imperative with any heel reconstruction that a patient critically protect the reconstruction. This is best accomplished through education and the proper selection or modification of a patient's shoe [46].

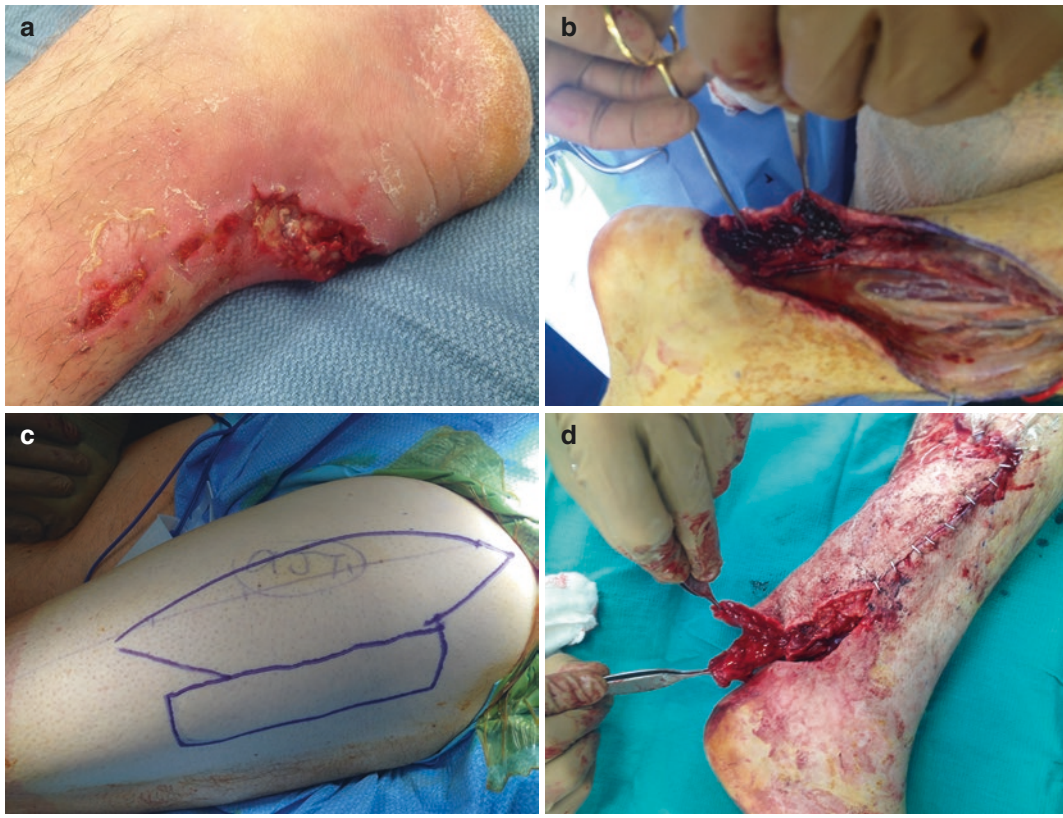

Fig. 19.3 (**a**) Achilles tendon wound dehiscence and infection with debridement of Achilles tendon. (**b**) Status post debridement with loss of Achilles tendon. (**c**) Design of an anterolateral thigh flap with tensor fascia lata tissue for Achilles reconstruction. (**d**) At final inset, note the tensor fascia lata is rolled up and sutured into the calcaneous with suture anchors

19.7.2 Unique Upper Extremity Considerations

The plastic surgeon or an orthoplastic approach to the upper extremity is critical to the complete care of upper extremity pathology. Hand surgery is a subspecialty of both plastic and orthopedic surgery and was originally conceived by Sterling Bunnell to be a specialty capable of total composite care of the upper extremity in a true orthoplastic fashion. Unfortunately, despite the fact that microsurgery is critical to the total care of the upper extremity, subspecialization within the field of hand surgery has diluted the training of hand surgeons, and microsurgical skill is declining among hand surgeons [29]. For this reason, the plastic surgeon is frequently vital to the combined orthoplastic reconstruction of upper extremity defects.

While in the lower extremity the difficulty in reconstruction is frequently the need to wear a shoe and to bear weight, the upper extremity is complicated by the necessary motion in order to be an effective extremity. Motion is often compromised by the underlying musculoskeletal trauma or condition. This is very common at the elbow and the hand and these factors need to be considered in the reconstructive effort. When deciding upon the definitive soft tissue reconstruction, the need for further tendon, nerve, or bone grafting needs to be considered. Sometimes a skin graft will achieve coverage, but it will make further reconstructive efforts very challenging and a free tissue transfer should be considered (Fig. 19.4a–c).

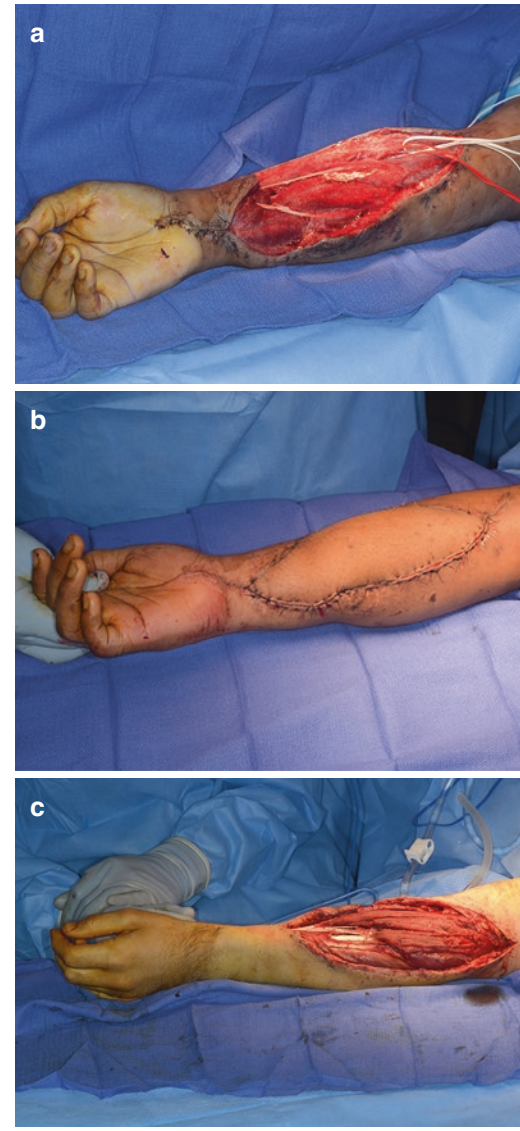

Fig. 19.4 (**a**) Large forearm fasciocutaneous loss with median nerve deficit requiring grafting. (**b**) Forearm reconstruction with anterolateral thigh flap to facilitate ease of flap elevation for definitive nerve grafting. (**c**) Similar forearm wound with no further reconstruction necessary treated with integra followed by split thickness skin grafting

19.7.3 Vascularized Bone Grafting

Segmental bone loss and the eventual need for bone grafting are common in severely injured patients. It is important for an orthoplastic team to have an armamentarium of options with regard to the creation of bone. Large segmental bone gaps are challenging to reconstruct. Multiple options to include conventional nonvascularized grafting, bone transport, and Masquelet techniques are all available and have a role in the treatment of bone gaps. The use of microsurgery has proven revolutionary in the creation of bone. Ian Taylor described the vascularized fibula in 1975, and it has been widely utilized since due to its size, acceptable donor morbidity, and direct

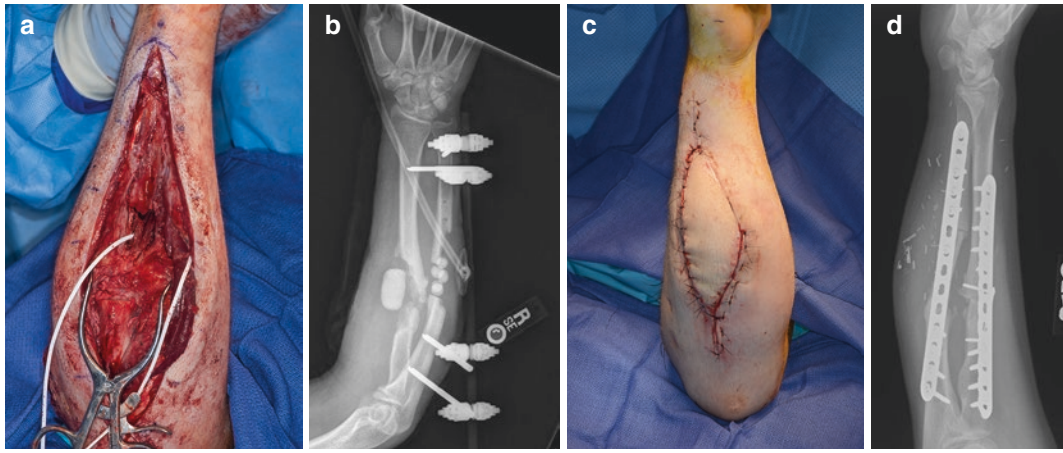

Fig. 19.5 (**a**) Both bone forearm fracture nonunion status post internal fixation and multiple infections. (**b**) Infected nonunion with external fixation and antibiotic spacers in place. (**c**) Osteocutaneous free fibula for the combined segmental ulna defect and soft tissue defect. The radius was treated with a portion of the fibula in a nonvascularized fashion. (**d**) Healed both bone forearm fracture

dissection [41–43]. These grafts have been utilized extensively in the upper and lower extremities for segmental defects as well as for osteonecrosis of the hip. The flap is versatile as it can be taken as bone only or as an osteomyocutaneous flap if desired due to its consistent peroneal artery perforators (Fig. 19.5a–d).

19.7.4 Medial Femoral Condyle

While the fibula has historically been the most frequently transferred vascularized bone, the medial femoral condyle continues to grow in popularity and is a necessary flap for smaller segmental bone defects in the extremities. Its use continues to grow for carpal bones and long bone nonunions [18–20, 31]. It is additionally very useful to stimulate the union of a failed joint fusion [8]. The use of the medial femoral condyle has even been suggested for the use in intermediate to large osseous defects [16, 28]. Additionally, the graft can be taken with cartilage from the knee to provide an osteoarticular graft for reconstruction of joint surfaces [14].

Yamamoto et al. recently described the vascular anatomy of the graft [16]. The team found that the nutrient vessels of the medial femoral condyle were consistently supplied by the descending genicular artery, the superomedial genicular artery, or both. They found that the

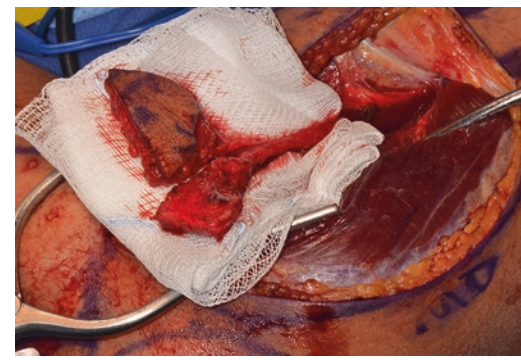

Fig. 19.6 Medial femoral condyle vascularized bone graft with skin island

descending genicular artery arose from the superficial femoral artery just proximal to the adductor hiatus and then divided into two to three branches: the osteoarticular branch, the muscular branch, and the saphenous branch. The descending genicular artery was present in 89% of their specimens. It branched off of the superficial femoral artery approximately 13.7 cm above the knee joint. The superomedial genicular artery was present 100% of the time and is the alternative supplying vessel in cases where a patient does not have a descending genicular artery. This dissection, however, is more challenging, and the average pedicle is only 5.2 cm in length and is on average 0.78 mm in diameter [16] (Fig. 19.6).

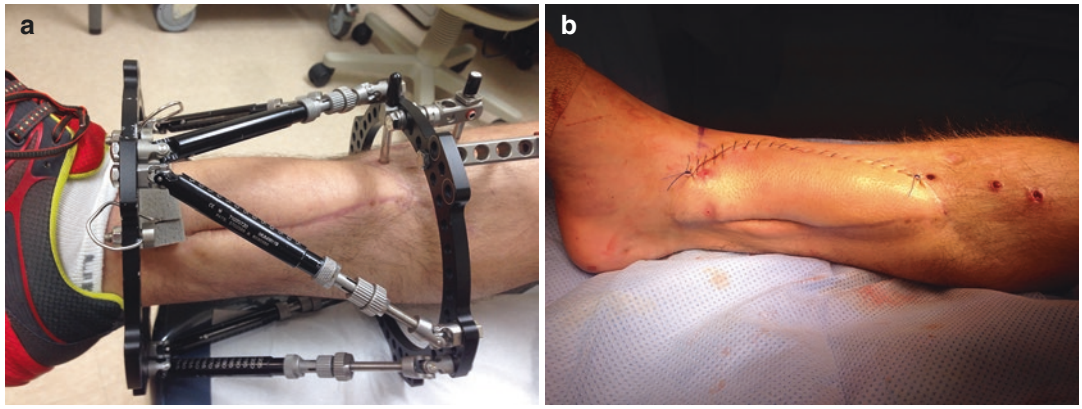

Fig. 19.7 (**a**) Open distal tibia fracture with original plate fixation complicated by wound breakdown and hardware removal. Treated with anterolateral thigh flap and ringed external fixator. Note the ability to wear shoe. Patient was weight bearing as tolerated. (**b**) Flap following frame removal bone grafting and delayed open reduction and internal fixation

19.8 Postoperative Care

The postoperative care of orthoplastic patients is critical to the overall outcome. After a flap reconstruction, we use splints and are liberal with the use of an external fixator to ensure that there is no pressure on a flap and to ensure no tension on the vascular anastomosis or pedicle. The flap is left visible and a petroleum gauze bandage is utilized to cover the suture lines. Internal Doppler devices are commonly utilized and/or surface monitoring if available. A surface perforator is often identified in order to do handheld Doppler checks postoperatively. Should any signs of venous congestion or flap compromise be noted, then we return to the operating room urgently. Standard deep vein thrombosis prophylaxis is utilized and patients are placed on a full-dose aspirin for 1 month. We believe in utilizing a staged dangle approach that progresses to full freedom at 6 weeks. Inpatient consultations for occupational therapy, physical therapy, rehabilitation medicine, and prosthetics are obtained the second day of admission. After wound reconstruction, patients undergo extremity rehabilitation limited only by the particular flap utilized. Range of motion is started as soon as feasible and early ambulation when the flap is ready is usually the goal. The use of state-of-the-art multiplanar spatial frames frequently facilitates weight bearing within 2 weeks and ambulation within 6 weeks of reconstruction when treating lower extremity fractures [46] (Figs. 19.7a–b and 19.8).

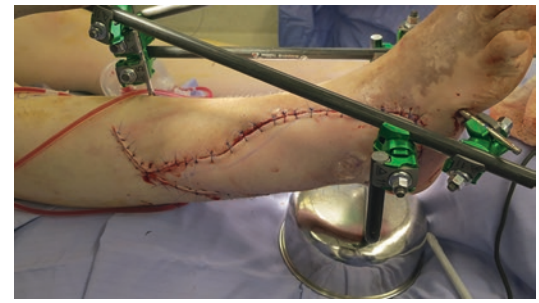

Fig. 19.8 External fixation in place for tibia fracture and anterolateral thigh flap. This external fixator was modified to prevent any pressure from being placed on the flap. This external fixator was converted to a ringed external fixator 3 weeks following the flap procedure

19.9 Vascularized Composite Allotransplantation

The pinnacle of the reconstructive ladder/elevator leads to vascularized composite allotransplantation. This is the culmination of true restoration. VCA has been fully realized and numerous hand transplants have taken place to date [36, 45]. The main current barriers to more widespread utilization remain the refinement of immunosuppression

with a move toward immunomodulation [30, 38]. The long-term outcomes or routine hand transplantation still needs to be further researched. Additionally, the early successes and failures with VCA highlight the psychological influence on the outcomes that need to be further elucidated. Hopefully, in the near future, a refinement in the indications, the ethics, and ultimately the finances of VCA will occur in order to sustain its continued growth.

Conclusions

For more than 50 years, advances have been made in extremity salvage and reconstruction using the operating microscope. Since 1991, the orthoplastic approach has served as the ideal approach to the traumatized limb, applying principles and practices of both specialties to clinical problems simultaneously. The results of this approach optimize patient recovery, decrease costs, and improve functional outcomes.

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Phase IV: Late Reconstruction – Abdominal/Chest Wall Closure

20

Whitney M. Guerrero and Timothy C. Fabian

20.1 Damage Control: Background for Laparotomy

Damage control laparotomy is a mainstay of trauma surgery. The physiologic consequences of multiple devastating injuries often preclude definitive management of every injury at the index operation. The threat of intra-abdominal hypertension and the possibility of abdominal compartment syndrome in the context of an abdomen closed under tension make primary abdominal closure at initial laparotomy untenable for some patients [1–5]. The most desirable outcome for the patient would be primary or delayed primary closure during the index hospitalization.

Different centers have published approaches to the open abdomen. These techniques include serial laparotomy followed by primary fascial closure, bridged mesh closure, acute component separation, or staged closure [2, 3, 6–8]. The primary goal across all approaches is to close the abdomen as soon as is safe, keeping intra-abdominal pressure in mind.

Some institutions have reported fascial closure rates after damage control laparotomy with temporary abdominal closure of up to 90%, while in our experience, a much lower percentage of fascial approximation occurs following temporary abdominal closure. We attribute much of this difference in closure rates to selection bias. If all patients are managed with damage control laparotomy and temporary abdominal closure, surely most will ultimately allow for fascial closure because most could have been managed without a temporary closure. If temporary abdominal closure is limited to the most severely injured patients, it follows that fewer will be amenable to fascial closure during the same hospital stay. Promotion of primary closure, of course, is the best way to avoid having to manage a difficult open abdomen.

Negative pressure is widely used to promote primary closure. Both the Barker vacuum pack technique (BVPT) and commercial negative pressure wound therapy (NPWT) devices may be used to this effect. In theory, vacuum mobilization of the intraperitoneal fluid reduces retraction of the fascia and prevents loss of abdominal domain.

Another approach to increase the proportion of patients who may be primarily closed is the use of hypertonic saline (HTS), which in animal models has been shown to decrease visceral edema. A study conducted by Harvin and colleagues showed better rates of early primary fascial closure (closure on or before post-injury day 7) when HTS was administered. At the primary surgeon's discretion, patients with open abdomens

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received 30 ml/h of 3% saline as maintenance fluids and were compared to a control cohort who received 125 ml/h of 0.9% saline. The HTS cohort had a 96% primary closure rate compared to an 80% primary closure rate for the normal saline group ($p < 0.001$) [9]. There were limitations to the study: it may be debated whether total fluid load may have had an effect, or whether perhaps sicker patients were excluded from the HTS group at the discretion of the treating surgeon. Both infusion of greater than 5 L of crystalloid and transfusion of packed red blood cells in excess of 10 units within the first 24 hours of trauma are independent risk factors for abdominal compartment syndrome [10]. Fluid restriction in a more general sense therefore can help reduce the risk for intra-abdominal hypertension.

When primary or delayed primary closure is impossible, it becomes necessary to consider the management of the giant ventral hernia. At Presley Regional Trauma Center in Memphis, the staged closure or planned ventral hernia approach is favored [11]. DiCocco et al. reported, with excellent follow-up, a very low recurrence rate (5% for PVH compared with 14% for all other methods) [12]. We will address the approach to the staged closure in the remainder of this chapter.

20.1.1 Staged Management of Abdominal Wall Defects

In the following sections, we will describe the staged approach to the management of the complex open abdomen. This approach is employed in Memphis and was developed by Dr. Fabian and colleagues. When it is determined that fascial edges will not be approximated without undue tension and the abdomen may not be closed in a traditional manner, our three-stage approach is used.

The goal of Stage One is coverage: a biosynthetic replacement is inserted in the open abdominal wall. Stage Two is the creation of a "planned ventral hernia" (PVH), and Stage Three is the definitive takedown and reconstruction of the PVH. Each stage, as well as the complications encountered therein, will be discussed in detail. Major overall complications include incidence of mortality, fistula formation, and hernia recurrence.

20.1.1.1 Stage One: Techniques for Temporary Coverage of the Open Abdomen

The goal of the first stage of our approach is coverage of the abdominal viscera. At our institution, the majority of our experience with coverage has been with prosthetic materials. In the past, polytetrafluoroethylene (PTFE) and polypropylene sheets have been used, but these have long since fallen out of favor. PTFE is quite costly when used for temporary fascial closure. Polypropylene sheets had been commonly used, but showed an unfortunate propensity for the development of intestinal fistulae [13].

Absorbable woven polyglactin 910 mesh is our prosthetic of choice. The mesh is applied as a bridge between fascial edges covering the abdominal viscera. Once this has been in place for approximately 1 week, an attempt at secondary fascial closure is routinely made: the prosthesis may be pleated, sutured, and incised to reapproximate the fascial edges. This is not possible in a substantial subset of patients; for these individuals, the open wound is allowed to granulate and is covered with a split-thickness skin graft. Most patients have a fully granulated wound after 2.5–3.5 weeks, and the mesh can be removed without danger to the bowel. If the mesh is left in place for a prolonged time period, there is a significantly increased risk for intestinal fistulae. Split-thickness skin grafts are therefore placed as soon as an adequate granulation bed exists.

20.1.1.2 Stage Two: Building the Planned Ventral Hernia

If pleating of the mesh is unsuccessful (as is the case for 75% of patients with temporary mesh closure), the open abdomen will require granulation and skin grafting, which are the basis of the formation of a planned ventral hernia.

Intestinal fistulae are a dreaded complication in the management of the open abdomen and are

most likely to occur during this stage. Approximately three quarters of intestinal fistulae occur in the small bowel. They may be associated with a failed suture line, but more often result from erosion into the bowel by the mesh itself or at a site of breakdown in the granulation tissue bed. Our experience with utilization of absorbable mesh followed by split-thickness skin grafting has resulted in an 8% fistula rate in those patients who survive the acute trauma. Analysis of those data allowed for the addressing of issues of management techniques that might reduce the fistula rate [14].

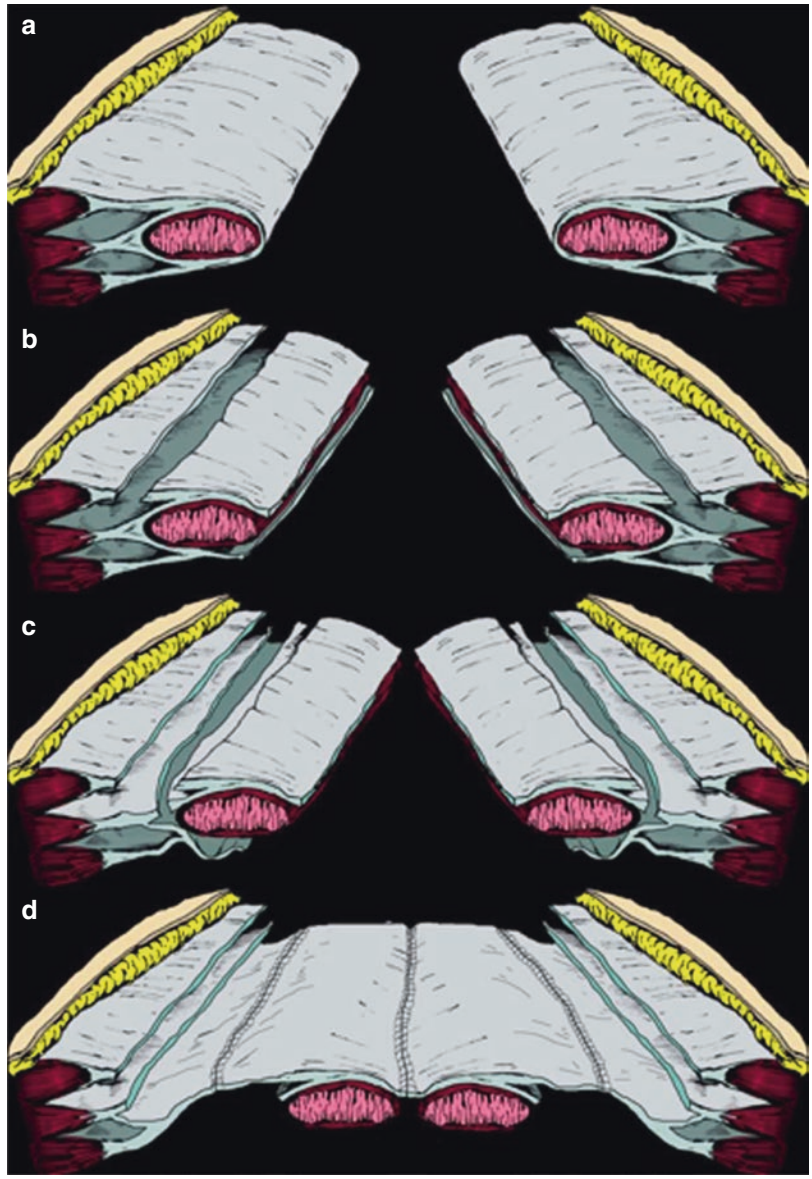
The risk of fistulae may be reduced by (1) avoiding nonabsorbable mesh, (2) approximating the fascia by pleating the absorbable mesh, and (3) covering the granulation bed with a split-thickness skin graft in a timely manner. We have discussed the first two points at length. To the third point, an analysis of fistula development and time to wound closure revealed that patients who developed fistulae on average had their mesh in place for 4 weeks, compared to 2.5 weeks for patients who were successfully managed without the development of a fistula. Thus, for patients whose wounds cannot be closed secondarily, split-thickness skin grafting should be undertaken as soon as possible. Once the bowel has adhered to the edges of the wound, the graft should be applied, and while fistulae can never be avoided completely, careful adherence to these three principles can keep the fistula rate below 5%.

20.1.1.3 Stage Three: Definitive Reconstruction

The single most important factor in successful reconstruction of a planned ventral hernia is timing. Extensive adhesions present a significant obstacle to the completion of this operation. In our experience, it takes a full 6–12 months before these adhesions resolve to allow safe definitive reconstruction. Patients must be followed long term, and the resolution of adhesions may be assessed clinically. Soon after PVH formation, adhesions between the viscera and the graft are dense. The two planes cannot be separated on physical exam. An attempt to reconstruct at this phase would be extremely difficult at best, resulting in inadvertent enterotomies and the accompanying increased risk of wound infection and reconstruction failure. Over time, however, these dense adhesions will resolve. On clinical examination, the skin graft can be pinched away from the underlying viscera with the fingers. When the two planes separate easily, reconstructions may be undertaken. There is a danger in waiting too long, as this can result in loss of abdominal domain and higher rates of recurrence.

Abdominal wall reconstruction can be approached in two ways. The more popular of the two involves the use of permanent prosthetic materials. The appeal of this approach lies in its simplicity. However, infections of permanent prosthetic materials pose a mammoth challenge, the treatment for which invariably involves removal of the mesh. Follow-up care may span months or years. Another approach, autologous tissue transfer circumvents this problem, but may pose another. If inadequate tissue exists for coverage of the defect, hernia recurrence is more likely. Careful planning and meticulous technique can overcome the challenge of adequate tissue coverage and most hernia recurrence, while even the best technique cannot prevent every prosthetic infection. For these reasons, at our institution we favor autologous tissue transfer.

The component separation technique was devised by Ramirez and colleagues to repair large abdominal wall hernias by tissue transfer [15]. In their description, the rectus abdominis musculofascial unit is mobilized medially to provide autologous continuity. Approximately 5–8 cm can be mobilized with each musculofascial unit, but patients must be closely monitored for abdominal compartment syndrome for 24–48 h following repair. Standard component separation may not offer enough tissue mobilization to adequately cover the large defects that may result from modern damage control laparotomy, necessitating the use of mesh. A technique was subsequently developed at our institution, which provides for closure of larger defects, which cannot be closed by the standard components separation. This reconstructive procedure has been termed modified components separation [11].



At our institution, approximately 50 patients per year are managed with temporary abdominal closure after damage control laparotomy. In a 15-year review by DiCocco and colleagues in 2010, more than 5000 trauma and more than 2500 non-trauma laparotomies were performed during the study period. Follow-up ranged from 9 months to nearly 15 years. The majority of patients had a penetrating mechanism and the vast majority were men. Most abdomens were left open due to visceral edema, and the defect sizes ranged from 20 to 1800 cm^2. Patients managed traditionally had a hernia recurrence rate approaching 20%, while those managed with our staged approach with modified component separation had a hernia recurrence rate of only 5% [12].

20.1.2 Operative Approach

In the operating room, the skin graft is pinched away from the underlying viscera somewhere

near the midportion of the wound. The graft is sharply incised, and, with a combination of sharp and blunt dissection, it is dissected from the underlying viscera. This excision generally takes approximately 1 h. The most dense adhesions are usually over the liver as well as at the native myofascial edge of the wound. It is imperative to gently dissect the adhesions from the liver, with attention to avoiding dissection beneath Glissen's capsule, which results in significant troublesome oozing. The myofascial edge is virtually always more densely adherent than the adhesions to the omentum or intestines. However, occasionally, small areas of dense adhesions to small bowel are encountered. Injury to the intestine may require short areas of resection. Avoidance of permanent mesh for reconstruction is desirable due to the risk of infection of the foreign body.

The anatomy of the abdominal wall must be considered in understanding the mobilization and tissue transfer involved with modified components separation. Both the anterior rectus fascia and the posterior rectus fascia comprise two lamellae. The anterior rectus fascia is composed of an extension of the external oblique fascia combined with a component of the internal oblique fascia. Thus, a fusion of the external oblique and a lamellum of the internal oblique fascia produces the anterior rectus sheath. The posterior rectus fascia comprises the medial fascia of the transverse abdominus muscle and the posterior lamella of the internal oblique fascia. Thus, the internal oblique fascia splits to form a component of both anterior rectus fascia and posterior rectus fascia above the accurate line.

Performance of the modified components separation technique begins with a division of the external oblique component of the anterior rectus sheath. The lateral plane where this division occurs is located by grasping the rectus abdominus muscle between the thumb anteriorly and the fingers posteriorly and squeezing this tissue; this allows for identification of the lateral portion of the anterior rectus fascia. This is approximately 1 cm lateral to the lateral board of the rectus and is where the incision of the external oblique fascia is begun. This incision is extended 6–8 cm above the costal margin superiorly and down to the pubis inferiorly. Following division of the external oblique component of the anterior rectus sheath, blunt dissection is carried out between the external oblique fascia and the internal oblique fascia, bilaterally out to the area of the anterior axillary line to allow for mobilization of the rectus musculofascial component. Following division of the external oblique fascia, the posterior rectus fascia is sharply dissected from the rectus abdominus muscles bilaterally. Care is taken to avoid injury to the inferior epigastric vessels, which provide blood supply to the rectus abdominus muscles. For moderate-size defects, that may be all of the mobilization required to allow for fascial closure. However, most of the large abdominal wall defects will require further separation of abdominal wall components. The modification of the components' separation description of Ramirez and colleagues that the author and colleagues have added is to next divide the internal oblique component of the anterior rectus sheath. The location of the internal oblique component is readily seen after the external oblique component of the anterior sheath has been divided. The internal oblique component is divided superiorly up over the lower costal margin, but it is very important to not divide it inferiorly below the linea semilunaris, because there is no posterior rectus sheath below that point. If the internal oblique component of the anterior rectus sheath were divided lower than the semilunar line, then a large hernia defect would be produced. The complete mobilization of these abdominal wall components provides approximately 10 cm of medial advancement in the epigastium, 20 cm in the mid-abdomen, and 8 cm in the lower abdomen. The epigastric region is always the tightest and is the most common location for the occasional need for adjunctive mesh. Following these steps, the abdominal wall reconstruction is completed by approximating the medial edge of the posterior rectus sheath to the lateral portion of the anterior rectus sheath with polypropylene suture. The repair is completed by reapproximating the medial edges of the anterior rectus sheaths in the midline.

Analysis of 73 patients who had definitive abdominal wall reconstruction using the modified

components separation technique found that recurrent hernia developed in four patients (5%). The mean follow-up of patients undergoing reconstruction was 24 months [12]. The author and colleagues have discovered that waiting too long for abdominal wall reconstruction produces inferior results. When need for adjunctive mesh was grouped with hernia recurrence and they were considered as complications, the complication rate was 7.6%. Patients with those complications were reconstructed at 20 months following discharge compared with 10 months in those without complications. This is probably secondary to progressive loss of abdominal domain and consequent closure under tension. Ideally, reconstruction should take place when the skin graft can be pinched from the intestines; this is usually the case within 6–12 months from initial hospital discharge.

20.1.3 Summary and Conclusion

The staged management of patients with giant abdominal wall hernias with autologous tissue transfer using the Memphis modification of the component separation technique provides a safe and effective approach for initial management and definitive reconstruction. The rate of fistula formation can be controlled using absorbable mesh and by covering granulating wounds early. Reconstruction should be undertaken as soon as resolution of dense adhesions allows. From the experience at the Presley Regional Trauma Center, the modified component separation technique is the procedure of choice for definitive abdominal wall reconstruction.

20.2 Damage Control: Background for Thoracotomy

In contrast with damage control laparotomy, damage control thoracotomy for severe thoracic injury is performed infrequently with reported overall mortality of 20–40% [16, 17]. One review of 840 thoracotomies performed for trauma over 5 years found that only 31 patients (4%) required damage control thoracotomy with a mortality rate of 24% [18].

Thoracic compartment syndrome after thoracotomy is rare [19], but temporary closure of the chest wall may be undertaken. Methods of temporary closure include closure of the skin only, either suture or with towel clips [20], or the use of a Bogota Bag as employed in temporary abdominal closure [21]. In addition, the thorax may be packed to control hemorrhage [18], though there is some controversy surrounding infection and ventilatory restriction from intrathoracic packs [22]. Vacuum-assisted closure devices may be employed to avoid thoracic compartment syndrome [17].

Concern for intrathoracic infection and ventilator challenges from retained packing has led to some reluctance to perform damage control operations analogous to those performed for the abdomen. In one study, 61 patients who required emergent thoracotomy for trauma either received temporary chest closure with or without intrathoracic packing (TCCP) or traditional definitive chest wall closure (DEF). Concerns for infection and elevated peak airway pressures were not borne out; TCCP showed no difference in infection rate and actually lead to lower peak pressures on arrival to the intensive care unit. There was no survival benefit observed for TCCP [23].

20.2.1 A Note on Resuscitative Thoracotomy

While early experience with battlefield thoracotomies led to reluctance to perform the procedure for fear of overwhelming infection [24], resuscitative thoracotomy (RT) has become an important part of the management algorithm for penetrating trauma patients in extremis [25]. Though only 1–2% of blunt trauma patients survive RT, 15% of patients with penetrating thoracic wounds and 35% of patients with penetrating cardiac wounds survive [26]. The chest wall is routinely closed in the operating room, though if concern for thoracic compartment syndrome arose, temporary closure techniques could certainly be used.

20.2.2 Chest Wall Reconstruction

Whether from destructive penetrating injury or massive blunt insult, trauma is a major cause of severe chest wall loss. Reconstruction may be required in the form of soft tissue coverage or fixation with soft and rigid materials [27]. Elements of both soft and rigid fixation may need to be employed to restore the best measure of functionality to the chest wall after severe trauma.

20.2.3 Soft Tissue Injury

Acellular dermal matrices elicit a local inflammatory response and may be an appropriate choice for reconstruction. These can be stretched to span the defect and sutured directly to the rib to provide a degree of rigidity. Primary or flap closure over the prosthesis is essential [27]. A number of pedicled flaps have been described in the context of surgical oncology, though these strategies may be reasonably applied to the coverage of a traumatic soft tissue defect.

A pedicled latissimus flap developed for chest wall reconstruction after mastectomy was described in 1906 [28]. This has been widely employed for traumatic soft tissue injuries. A latissimus flap allows coverage of most chest wall defects; there is a large potential arc of rotation on a pedicle [29], and it is the most reliable choice for coverage of the posterolateral chest and midback [30]. If necessary, one may use preoperative tissue expansion to improve coverage [29, 31].

The pectoralis flap is the approach of choice for superior anterior chest, sternal and cervical regions, and intrathoracic coverage. Rectus abdominis flaps, either free or pedicled, may be used for anterolateral and anterior chest wall defects. For back, posterior neck, and shoulder defects, the simple muscular or myocutaneous trapezius flaps are frequently the simplest and most cosmetic option [29].

Occasionally a chest wall defect will not be amenable to coverage with an easily accessible flap. The defect may be too large or may be surrounded by damaged tissue. In this case, an omental flap may be used in conjunction with a skin graft. The omental flap is pedicled on the gastroepiploic artery and may be fed through a subcutaneous tunnel or via a transdiaphragmatic route – transdiaphragmatic is generally preferred as the subcutaneous tunnel results in a 21% incidence of abdominal wall hernias [32]. The use of an omental flap necessitates a laparotomy, and the size and viability of a patient's omentum is variable [29].

20.2.4 Rigid Fixation

The goal of rigid fixation of the chest wall should be to restore structural integrity while preventing paradoxical motion during respiration. This is especially important for anterior chest wall defects [33]. To this end, a combination of layered dermal substitute, defect-spanning flexible plates, and muscle flap coverage has been described in the plastic surgery literature [34].

Surgical rib fixation is an acceptable way to increase pulmonary function while decreasing thoracic deformity, ventilator dependence, and length of hospital stay [35–37]. Standard rib plates are curved metal alloy. Some allow for intraoperative customization to better fit the individual rib curvature. These come in multiple sizes and are fixed to the rib either by intraosseous screws or by clips that surround the rib. Multidirectional plates are available for more complex defects. These allow for longitudinal and vertical stabilization of the rib cage if necessary [38]. Surgical rib stabilization is not without its complications. Infection is always a concern, and incidences of nerve injury have been reported [39].

There is not at this time a widely accepted set of indications for rib plating (save for flail chest as described above), and no defined algorithm for the timing of the procedure currently exists. Its use is at the discretion of the surgeon.

20.2.5 Summary and Conclusion

Thoracic compartment syndrome after resuscitative or damage control thoracotomy is by no means as frequently encountered as abdominal

compartment syndrome, but it is a real entity that can have serious consequences for patients. Once the patient can tolerate it, there are multiple techniques and approaches available for chest wall reconstruction; a touch of ingenuity on the part of the surgeon may be required for maximal restoration of chest wall structure and function.

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Part IV

Special Circumstances and Outcomes

Principles of Damage Control for Pelvic Ring Injuries

21

P.V. Giannoudis and Hans-Christoph Pape

21.1 Introduction

Pelvic fractures account for up to 8% of all skeletal fractures [1] and usually affect the adult population. They present after high-energy trauma with car and motorcycle accidents being the most common mechanism of injury. Pelvic fractures in the elderly population are secondary to low-energy trauma and in a sense represent a different entity. For this reason, their management will not be covered in this chapter.

While the establishment of national and regional organized trauma systems has contributed to a more unified management of these severe injured patients, pelvic ring disruptions continue to be a substantial source of morbidity and mortality [2]. In the emergency setting, the management of patients with pelvic fractures remains perplexing requiring the input of different specialties and leadership skills to take promptly difficult decisions. The presence of associated injuries to the head, chest, and abdomen may create competing actions between clinicians leading to unnecessary delays which can harm the patient. Appropriate assessment and treatment of all the injuries therefore is crucial and can lead in fewer deaths and less long-term disability.

The diversity of pelvic ring disruptions led to the development of different classification systems over the years based on fracture location, pelvic stability, injury mechanism, and direction of injury force applied. Nowadays, the Young and Burgess classification system, which classifies the fractures according to the direction of the force applied during the course of the accident (anterior-posterior compression (APC), lateral compression (LC), vertical shear (VS), and combined mechanical injury (CMI)), continues to be used extensively in the clinical setting [3]. This can be attributed to the fact that it allows detection of the posterior ring injury and predicts local and distant associated injuries, resuscitation needs, and expected rates of mortality. The most severe patterns of injuries within the subgroups created APC III, LC III, vertical shear (VS), and combined mechanical injuries are suggestive of major ligament disruption. These most severe injury patterns, for instance, the APIII, require the most blood replacement, followed by VS, followed by CM, followed by LC III injury patterns [3].

Patients with pelvic fractures could be divided into two distinct subgroups. In the first group of patients, usually stable pelvic fractures with most

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of the injury confined to the ligamentous tissues are present. The management approach in these circumstances is focused on the restoration of the osteo-ligamentous structures on a more semi-elected basis. In the second group, displaced pelvic ring fracture is a common finding necessitating emergency hemorrhage control and a multidisciplinary team approach for the other organ system injuries. Noteworthy, the prevalence of pelvic fractures presenting with hemodynamic instability has been described to range from as low as 2% up to 20% [2, 4]. Early recognition and appropriate clinician decisions in this group of patients can hence provide significant improvements in outcome.

The management of this cohort of patients possessing the highest risk of early and late complications and mortality has advanced over the years to what is known today as "damage control orthopedics."

21.2 Impact of Pelvic Injuries on the Hospital Course

21.2.1 Influence of Pelvic Hemorrhage

In patients with high-energy injuries, quick identification of hemorrhage related to the pelvic injury in the hemodynamically unstable patient is both critical and time-dependent.

Arterial bleeding (iliac vessels and their branches to the inferior abdominal viscera and pelvic organs) is a major contributor to hemorrhagic shock in pelvic fractures. Other sources of bleeding include the venous plexus and fractured cancellous bone surfaces. These are usually low-pressure bleedings and they leak into the retroperitoneal space. The retroperitoneum can contain several liters of blood and bleeding may continue even after that. Thereby, uncontrolled hemorrhage may develop, as the retroperitoneal space does not allow for self-tamponade [5]. This can lead to constant further hemorrhage creeping proximally around and above the psoas muscle or along the gluteal muscles. This phenomenon has been named the "chimney effect" [6]. Exsanguination and/or pelvic and abdominal compartment syndrome may ensue. According to Heetveld et al. every 3 min of hemodynamic instability without hemorrhage control increases the mortality by 1% [7].

In general terms, the principles of physiological state classification applied for patients with multiple injuries also are valid for those with pelvic fractures. Four different categories have been identified as follows [8]:

(a) *Stable* (no clinical signs of shock, no chest trauma)
(b) *Borderline* (systolic blood pressure 80–100 mmHg, chest AIS 2 or more, blood transfusion 2–8/2 h, and other criteria)
(c) *Unstable* (dropping or undulating systolic blood pressure of >90 mmHg, pulse of <100 beats/min, chest AIS 2 or more, CVP >5 cmH2O, urine output of >30 ml/h despite adequate fluid resuscitation, and blood transfusion over a period of 2 h)
(d) *In extremis* (patients with either absent vital signs or presence of severe shock due to uncontrollable hemorrhage needing mechanical resuscitation or repeatedly catecholamine infusion, despite complete blood volume replacement within 120 min (>12 blood transfusions/2 h))

As a rule of thumb, the initial management focuses on hemorrhage control. Thus, complex reconstructions are delayed until the patient is hemodynamically stable and in a better physiological condition to withstand the additional surgical burden. Avoidance of coagulation disturbances, systemic inflammatory response, adult respiratory distress syndrome, and multiple organ dysfunction syndrome is of paramount importance for reduced mortality rates.

Hemorrhage may occur from external or internal bleeding: For external bleeding, the main maneuver consists of application of direct pressure and rapid surgery. In internal bleedings, it has to be decided whether angiography and embolization is required or whether packing of the extraperitoneal space is favored [9].

21.2.2 Initial Assessment

The first step in the treatment of restoring hemodynamic stability includes the administration of intravenous crystalloid fluids and whole blood. When replacement of fluid and blood does not stabilize the patient's vital signs, additional steps must be taken. Any subsequent interventions should be rapid and minimally traumatic focusing on hemorrhage control and other lifesaving measures.

In hemodynamically unstable patients with no obvious site of hemorrhage, careful clinical examination of the pelvis is mandatory even when radiographs look normal or a pelvic image demonstrates a stable fracture configuration. Physical examination of the pelvis should include thorough inspection of the flanks, lower abdomen, groin, perineum, and buttocks to detect any wounds or bruises. Injuries to cervix, uterus, and ovaries are rare [8]. Inspection of the external genitalia is the first action to take, and then, a more meticulous digital examination can reveal: lacerations, blood at the external urethral meatus, a high-riding prostate, perineal hematoma, hematuria, and vaginal bleeding (Fig. 21.1). Any of these findings or the inability to urinate in association to an anterior pelvic ring fracture should be an indication for retrograde urethrogram [10]; a cystogram should follow through a suprapubic catheter, if a urethral injury has been diagnosed. In this way, we can assess both the urethral and bladder integrity (Fig. 21.2).

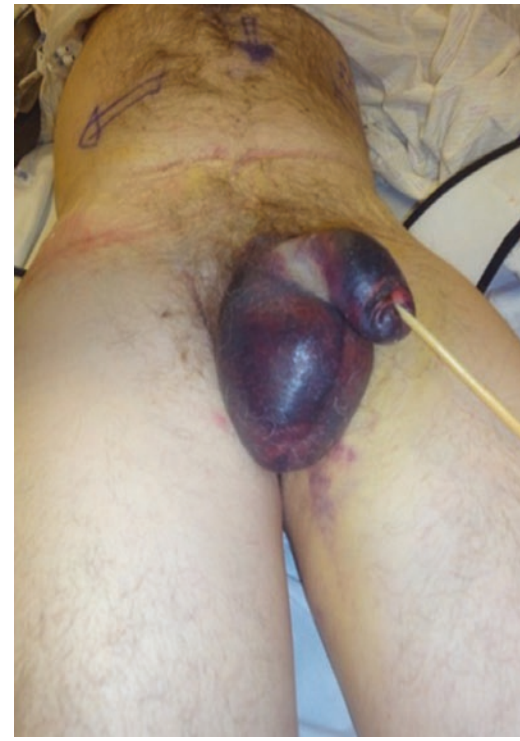


Fig. 21.1 Scrotal swelling following pelvic trauma

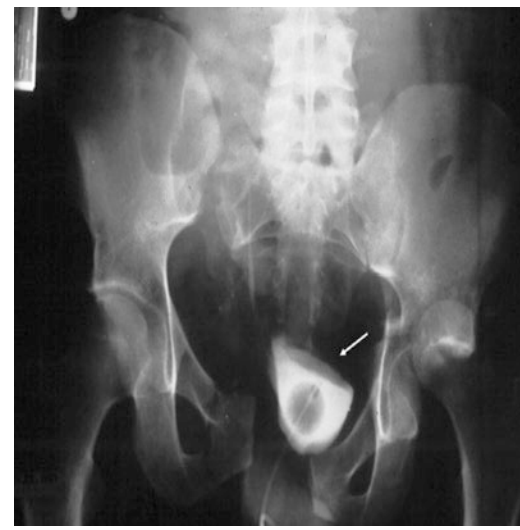


Fig. 21.2 AP pelvic radiograph (complex pelvic and acetabular fracture) illustrating the acquisition of a retrograde cystogram (*arrow*)

21.2.3 Radiographic Assessment

The basic radiographic examination includes a pelvic X-ray in most institutions. While in previous recommendations, inlet (beam directed caudad at 60°), outlet (beam directed cephalad at 45°),[83] and Judet (iliac and obturator oblique) pelvic radiographs have been advocated, this is no longer the rule in level I trauma centers, where a CT scan is done anyway and a 3D reconstruction can be obtained through free software options [11]. If there is no CT option, and if the pelvic plain film shows a pelvic fracture, there is a 50% probability of retroperitoneal bleeding.

It is of note that in stable patients the usefulness of a pelvic X-ray (PXR) has been questioned, as it was discussed to detect only

66–87% of pelvic fractures [11]. Moreover, the X-ray may miss non-bony pelvic injuries. However, for the unstable blunt trauma patient, the pelvic radiograph is necessary because these patients can neither provide a reliable physical examination nor undergo immediate CT scan. As a result, PXR provides a useful gross estimate of pelvic injury. Moreover, as would be expected, in unstable pelvic fracture patterns, sensitivities of the plain film are improved over minor pelvic fractures, 75–91% [11].

The fracture pattern on the initial anterior-posterior pelvic X-ray is the best initial guide to determine the probability of pelvic arterial bleeding. As mentioned above, the Young and Burgess classification system has been utilized more frequently than the Tile system when comparing the potential association between pelvic fracture type and retroperitoneal hemorrhage [12].

A significant correlation has been reported between greater blood product requirement and unstable fracture patterns. However, a limitation of predicting hemorrhage based on classifying the initial pelvic X-ray is that major posterior element disruption may not be detected in 9–22% of fractures when compared with CT.

21.3 Control of Mechanical Pelvic Instability and Hemorrhage

The nature of the pelvic fracture, whether closed or open, and the hemodynamic status of the patient will determine the treatment plan. In unstable patients, apart from the fluid resuscitation and administration of appropriate pharmacotherapy targeting the clotting cascade (i.e., administration of tranexamic acid) [13], there is a need to enhance the tamponade effect in the pelvis in order to increase the intrapelvic pressure and to facilitate hemostasis. This can be achieved by either invasive or noninvasive measures.

21.3.1 Noninvasive Measures

The noninvasive techniques are usually applied on scene or in the emergency room as soon as a diagnosis is available. They include application of a circumferential sheet, pelvic binder, internal rotation of both legs, traction of the lower limbs, and application of military antishock trousers (MAST).

Direct pelvic compression can be applied using a bedsheet, a pelvic sling (Fig. 21.3), or a

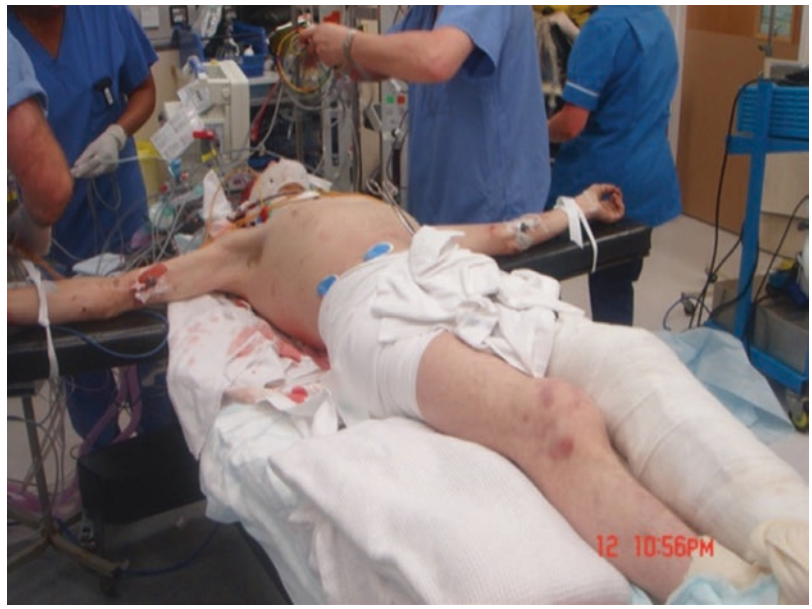


Fig. 21.3 Application of pelvic sling in a patient with multiple injuries

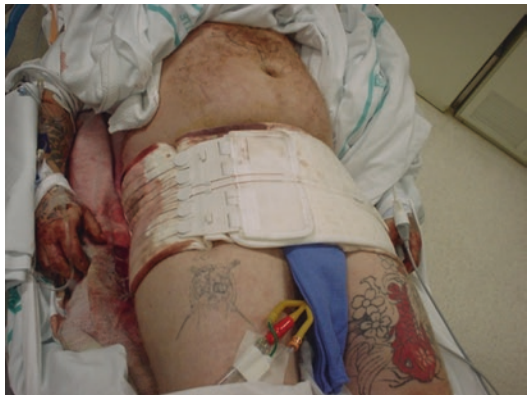


Fig. 21.4 Application of pelvic binder

commercially available pelvic belt (Fig. 21.4). By each of them, satisfactory pelvic compression appears to be achievable without limiting access to the patient [14–16].

Vermeulen et al. first illustrated the prehospital use of an external pelvic compression belt (Geneva belt) in a series of 19 patients in 1999 [17]. Their device was applied by paramedics at the accident scene as soon as there was a clinical suspicion of unstable pelvic fractures. The area of application is usually at the level of greater trochanters/symphysis pubis directly on to the patient's skin. It has been suggested that the force required to reduce unstable open book pelvic fractures is around 180 N based on cadaveric models. This led to the development of new commercial splints (SAMsling, SAM Medical Products TM, Oregon, USA), which use controlled and consistent stabilization with an autostop buckle to reduce the risk of overcompression in internal rotation injuries [18]. It has been shown that simple application of this sling increases pelvic stability by 61% in response to rotational stress and 55%, flexion–extension [19, 20].

Clinical judgment and reassessment are important in using these techniques. Potential complications include skin necrosis if left in place too long or applied too tightly. In lateral compression injuries with transforaminal sacral fractures, possible visceral or neural injury may occur if applied too vigorously.

21.3.2 Invasive Measures

21.3.2.1 Arterial Inflow Arrest: Arterial Occlusion and Cross Clamping

In cases that rapid exsanguination of the patient is imminent, occlusion of the aorta can be used as a temporary measure to control the hemorrhage [21]. This can be performed directly by open cross clamping [22] or via percutaneous or open balloon catheter techniques [23]. Other authors have reported satisfactory control of arterial bleeding with ligation of the hypogastric artery attributing this to the remarkable collateral supply within the pelvis [24].

21.3.2.2 External Fixation

The anterior fixator has been shown to contribute to hemostasis by maintaining a reduced pelvic volume, allowing tamponade, and by decreasing bony motion at the fracture site, thus allowing clots to stabilize (Fig. 21.5) [8].

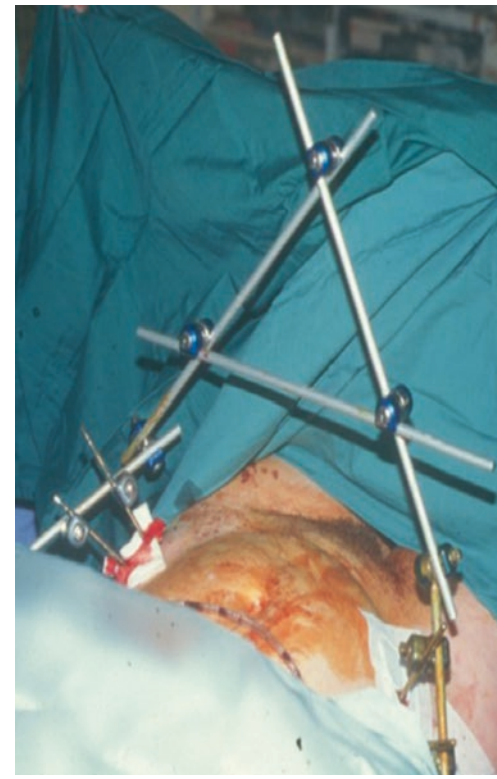


Fig. 21.5 Application of anterior external fixator – A-frame configuration prior to laparotomy

In the emergency situation, external stabilization of the pelvis indeed becomes the first priority. In many instances, external fixation replaces the stability achieved by a pelvic sling or a pelvic binder.

It has been discussed that optimal results are achieved in rotationally unstable LC fracture types II and III and APC types II and III. Stabilization can be achieved in 64–83% of these fractures [25]. With additional vertical instability (VS or CM type injury), stabilization can be achieved in only 27% of cases, and supplementary ipsilateral skeletal traction is needed [26, 27].

The fixation can either be achieved using the anterior superior iliac crest or the supraacetabular region. Both localizations have advantages and disadvantages, as listed in Table 21.1.

Briefly, the anterior superior pelvic crest is easily accessible and allows for quick fixation. However, it may be subject to loosening because of the thin layer of bony attachment. Recently, a percutaneous technique has become available that might be an alternative. At this stage, however, no data is available in larger series, and it is used in certain institutions only [28].

In type C or open book fractures, external fixator constructs appear to demonstrate a high rate of secondary displacement. In these fracture types, additional posterior fixation is required in order to provide a stable construct. Ganz et al. developed a large external fixation device that connects to the pelvis in the area adjacent to the sacrum and is thought to reduce the iliac wing toward the sacrum [29] (Fig. 21.6). As it applies sustained forces, it is able to provide a counteracting force for pelvic packing. Also, it can be left in place for several days and does not cause soft tissue issues as reported with pelvic slings. These issues are listed in Table 21.2.

These clamps have been applied in hemodynamically unstable patients and prophylactically in stable patients with unstable pelvic ring disruptions. Their use is, however, limited to a specific set of indications, for example, they are not applicable in fractures of the ilium and trans-iliac fracture dislocations. Complications include potential injury to gluteal neurovascular structures and overcompression with the risk of secondary nerve injury in sacral fractures [30].

Although potentially lifesaving, these devices should be applied by an experienced surgeon and considered only in cases of posteriorly unstable pelvic fractures accompanied by hemodynamic instability.

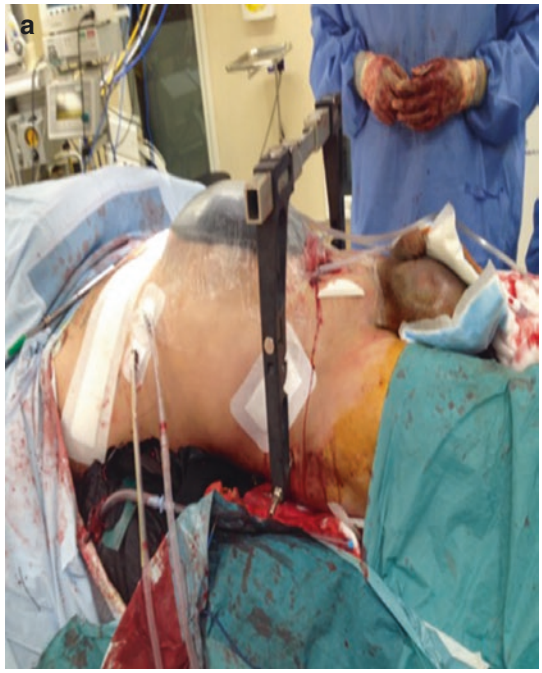
21.3.2.3 Acute Fracture Fixation

Provisional fixation of unstable pelvic ring disruptions with a pelvic clamp or an external frame with a supracondylar pin has proven markedly beneficial in the resuscitative phase of management [31]. If, however, the patient undergoes a laparotomy to deal with visceral injuries, symphyseal disruption and medial ramus fractures should be plated at the same time. Because neither blood loss nor operative time is greatly increased, combining these repairs decreases the risk of complications in a patient who is already compromised [32].

A role has also been suggested for percutaneous fixation; however, only surgeons appropriately trained should use this technique. Percutaneous pelvic fixation techniques allow for acute and

Table 21.1 Advantages and disadvantages of stabilization of the pelvic ring with anterior pelvic fixator using either the anterior superior iliac crest or the supraacetabulum area

	Anterior superior pelvic crest	Supraacetabular external	Percutaneous
Advantages	Easily accessible	Good purchase, allows for one Schanz pin only	Excellent fixation, no soft tissue problems
Disadvantages	May be subject to early loosening Requires multiple pins	Trend toward more radiological assessment Soft tissue issues in obese patients	Time to fixation might be increased



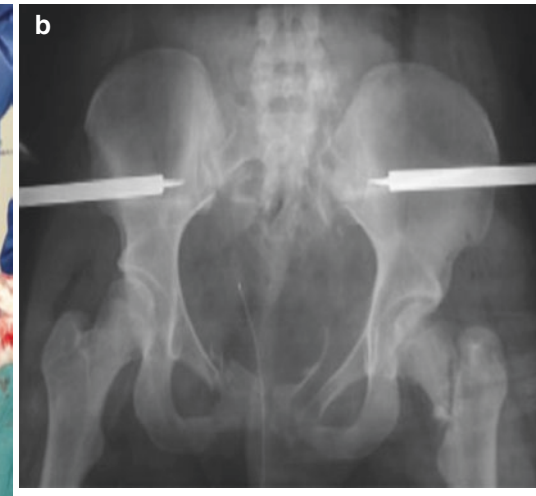


Fig. 21.6 (**a**) Application of C-clamp. (**b**) AP pelvic radiograph demonstrating stabilization of the posterior ring with the C-clamp

Table 21.2 Advantages and disadvantages between pelvic C-clamp and pelvic sling (binder) for temporary stabilization of the posterior pelvic elements

	Pelvic sling	Pelvic C-clamp
Advantages	Allows for prehospital placement Does not require immediate X-ray control	Provides excellent stability Allows for pelvic packing
Disadvantages	Soft tissue necrosis in prolonged use Frequent malplacement by paramedics May cause overcompression in case of sacral crush injuries	Pin tract issues in concomitant acetabular fractures requiring posterior approaches Displacement and overcompression issues if used without proper radiologic control

definitive treatment of anterior and posterior pelvic ring injuries, without extensive dissection [33].

Fixation can be performed acutely, even as a component of the patient's resuscitation. Operative blood loss is minimal and wound complications are unusual. Minimally invasive anterior ring fixation includes external fixation with retrograde or anterograde screws in the medulla of the superior ramus. Closed reduction and fixation with percutaneous sacroiliac screws offers definitive stable fixation for many posterior pelvic ring injuries, such as fracture/dislocation of the sacroiliac joint or sacral fractures, with the advantage of minimal dissection and a reduction in wound complications.

Nevertheless, in the acute setting and especially in the "extremis" clinical condition of the patient, such an approach is not advocated as it is time consuming, and often extensile approaches are necessary predisposing the patient to uncontrollable hemorrhage, coagulation disturbances, and early mortality.

21.3.2.4 Pelvic Angiography and Embolization

Information about the rate of arterial injury in pelvic trauma has primarily been derived from angiographic studies, with reported rates ranging from 0.01% to 2.3% for all pelvic

trauma and from 9% to 80% in unstable pelvic injuries [5, 34].

Timely identification and control of pelvic hemorrhage is pivotal to decrease pelvic fracture-related mortality. The main controversy regarding the treatment of patients with profuse, exsanguinating hemorrhage relates to the role of angiography and embolization. This technique is time consuming and can be performed in only approximately 10% of the cases [35].

Simultaneous treatment of other injuries cannot be performed during this procedure, and mortality of up to 50% has already been reported, despite effective bleeding control [36]. Angiography requires skilled radiologist and technical staff as well as transportation of a critically ill patient to the angio-suite knowing that there have been series where 20% of these patients had cardiorespiratory arrest during the procedure [36].

However, in a hospital where interventional radiology is available, angiography is both diagnostic and therapeutic for pelvic hemorrhage [37, 38].

Indeed, early angiography and embolization has proven to be one of the most important interventions to control arterial pelvic hemorrhage [5, 37, 38]. In recent and prospective series, early diagnostic angiography has revealed arterial injury or bleeding following pelvic fractures in 44–76% of hemodynamically unstable pelvic fracture patients [39–41].

Extravasation of contrast, false aneurysms, and occlusion due to thrombus or vasospasm are all signs of arterial injury, which may require embolization. Overall, embolization is safe and effective and achieves up to 90% success rates [39–41].

Nevertheless, there are several aspects of the current management strategy of early angioembolization for hemodynamically unstable patients with pelvic fractures that are concerning.

Predicting the patient who does or does not require embolization remains a challenge. Secondly, although angioembolization may be effective in controlling pelvic arterial bleeding, it has not been shown to decrease the necessity for blood product resuscitation. Third, there are a number of institutions that do not have angiographic capabilities, hence necessitating transfer of any such patient – not an ideal option in the already hemodynamically unstable patient.

Current consensus is that before angiography, aggressive resuscitation needs to be initiated; other sources of bleeding (chest/abdominal) need to be ruled out, and provisional pelvic stabilization with either a sheet or external fixator should be performed. If the patient remains hypotensive, angiography is indicated [5].

21.3.2.5 Pelvic Packing

Where ongoing hemodynamic instability is encountered, pelvic packing can complement the external fixation (Fig. 21.7). It is effectuated through a lower abdominal laparotomy, adjusted to the pelvic wound. Packs have to be inserted in the prevesical and presacral spaces and have to be removed or changed within 48 h [42]. Abdominal injuries are simultaneously assessed and treated.

In some European centers, external fixation of the pelvic fracture and surgical packing of the retroperitoneum is performed in favor of angiography [5].

Pelvic packing (PP) eliminates the often arduous decision by the trauma surgeon: OR versus IR? All patients can be rapidly transported to the operating room, and PP can be accomplished in less than 30 min. It may be also ideally suited for austere conditions and in settings where angiography is unavailable or

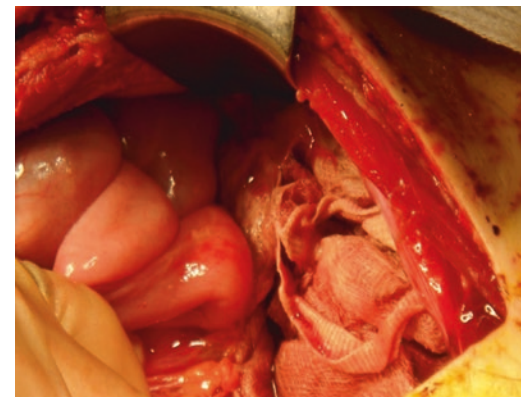


Fig. 21.7 Changing of pelvic packs 48 h after laparotomy where packing was applied

unable to be done expeditiously. Emergent retroperitoneal packing appears to be a safe procedure that has a role in damage control of critically injured patients. It can be done immediately and with ease in conjunction with external fixation of the pelvis and other surgical procedures to stabilize the patient.

21.4 Damage Control Orthopedics for Pelvic Fractures with Hemodynamic Instability

In patients with pelvic fractures being in an "unstable" or "extremis" clinical condition, prolonged operative interventions could initiate a series of reactions at the molecular level predisposing the patient to an adverse outcome. Any surgical intervention here must be considered immediately lifesaving and should therefore be simple, quick, and well performed. Rigid rules relating to timing should be avoided to prevent unnecessary delay – time is usually critical to survival of the patient [43].

Protocols designed to reduce mortality should stop bleeding, detect and control associated injuries, and restore hemodynamics. A staged diagnostic and therapeutic approach is required. The severity of bleeding is a crucial hallmark for survival during the early period after injury.

Numerous clinical pathways were developed and published during the last 20 years for management of hemodynamically unstable patients. All of them consist of abdominal diagnostics, pelvic binding and/or external fixation, pelvic retroperitoneal packing, angioembolization, and orthopedic fixation of the pelvis.

Because of the disastrous sequelae of uncontrolled hemorrhage in young patients, only external devices that are easy to apply can be used effectively. These devices, by external compression, reduce the intrapelvic volume and create a tamponade effect against ongoing bleeding. Pelvic packing should be considered in cases where, despite the application of the external fixator, ongoing bleeding is encountered.

Angiographic embolization is not usually indicated in this patient population. However, in cases where hemodynamic stability with volume replacement can be achieved but ongoing pelvic hemorrhage is suspected (expanding hematoma), then angiography could be considered as an adjunct to the treatment protocol.

In general terms the criteria to apply the DCO principle are shown in Table 21.3. DCO in practice consists of different stages, including: resuscitation, hemorrhage control, decompression, decontamination, fracture splintage, and prompt transfer to the intensive care unit where close monitoring of all the vital organs can take place and resuscitation can continue until physiological normality has been achieved [8].

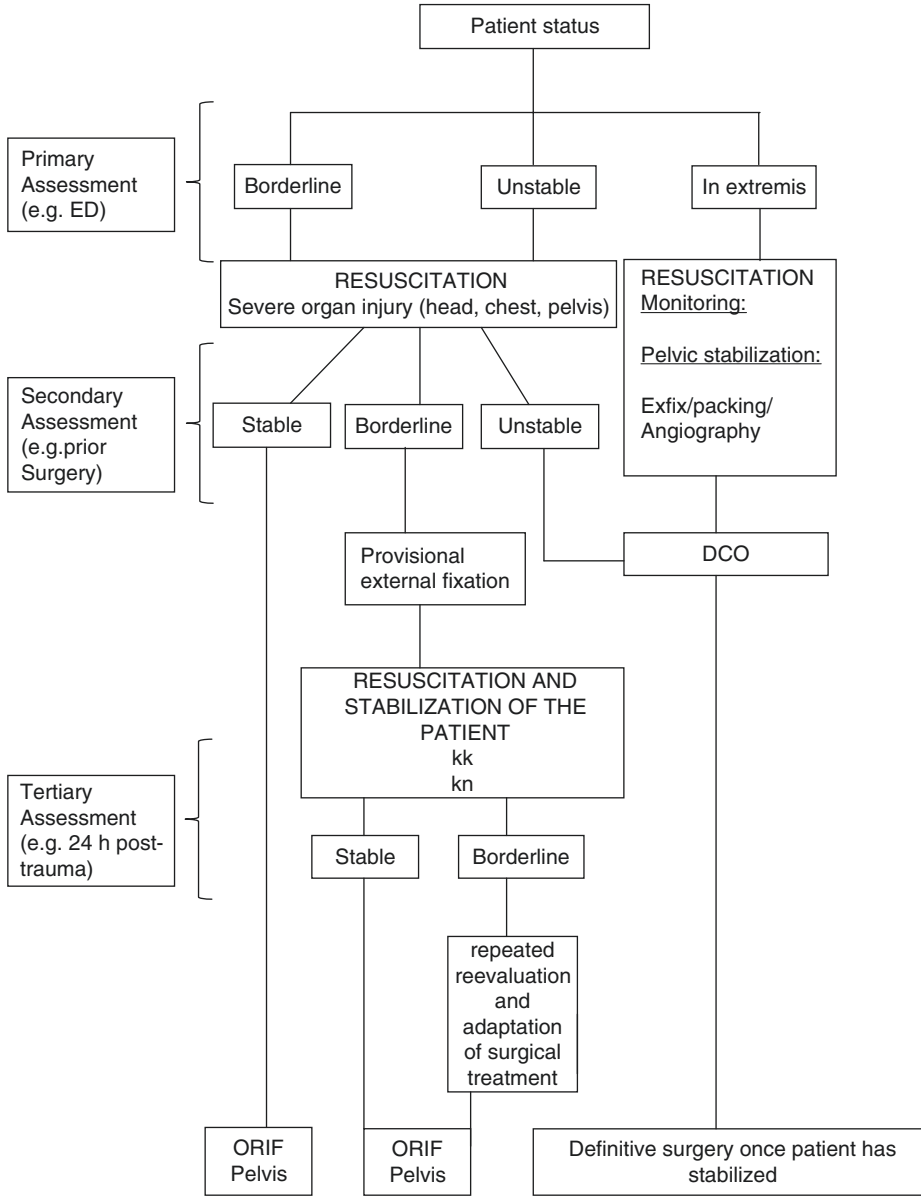
These principles are adapted to the pelvis as shown in Table 21.4.

During the resuscitation phase, it is essential to appreciate that in the patient with significant ongoing blood loss, ratios of blood products similar to whole blood will be required to reach simultaneously acceptable levels of hematocrit, clotting factor concentration, and platelet count. The need of having in-house massive transfusion protocols is therefore of paramount importance in order to be able to deal efficiently with increased resuscitation requirements. Nunez TC et al. developed a score to assist clinicians to predict the need for activation of a massive blood transfusion protocol. The authors identified four parameters (systolic blood pressure (<90 mmHg), heart rate (120 bpm), penetrating mechanism, and positive fluid on abdominal

Table 21.3 DCO criteria for application in pelvic ring injuriwa

Criteria for application of DCO for pelvic fractures
Hypothermia < 34 °C
Acidosis pH <7.2, serum
Lactate >5 mmol/L
Coagulopathy
Blood pressure <70 mmHg
Transfusion approaching 15 units
Injury severity score >36

Table 21.4 Integration of the DC concept into the management of pelvic ring injuries



ultrasound) and assigned a score of 1 to each one if present. They reported that a score of 2 predicts 38%; a score of 3, 45%; and a score of 4, 100% the need of massive transfusion [44].

For the hemorrhage control, different options to stop the bleeding from the pelvic ring were previously discussed. Moreover, prompt identification of other sources of bleeding should also take place particularly from the abdominal and chest cavity. Any additional interventions (decompression of cavities, i.e., intracranial hemorrhage) and decontamination (irrigation and debridement of open wounds) should be performed as quickly and efficiently as possible trying to avoid time-consuming procedures. Associated fractures to the extremities can be managed promptly by the use of external fixators for temporarily stabilization followed by delayed

definitive reconstruction when the patient has overcome the initial physiological crisis phase and can tolerate well the surgical stress of prolonged reconstruction procedures.

21.4.1 Ongoing Treatment

If definitive stabilization is needed, it should be effectuated according to the principles of "damage control orthopedics" [8]. Definitive stabilization in closed pelvic fractures with internal fixation is recommended between the third and seventh day post-injury [8]. In open fractures, the timing is not adequately covered and fixation techniques are controversial. Traditionally, only external fixation has been used, but there are authors publishing good results after internal fixation [45] or suggesting internal fixation when there is no gross contamination of the fracture site [46]. In comminuted iliac wing fractures, early open internal fixation is preferred since external fixation cannot be applied [47]. Combination of internal and external fixation has been described by Leenen et al. [48], and percutaneous internal fixation has been used for open fractures with less complications [49].

When wounds are associated to the pelvic fractures, their treatment includes extensive irrigation, debridement (up to healthy tissue with capillary bleeding) and removal of foreign bodies and bony fragments (Fig. 21.8) [50]. For the washout, either free flow or pulsed lavage techniques can be used. The wounds can either be left open or vacuum-sealed dressings can be used in order to drain them. A second look, with or without closure, should be effectuated after 48–72 h [51, 52, 53].

The possibility of a compartment syndrome associated to the abovementioned injuries shouldn't be neglected. The major pelvic compartments are the iliopsoas, the gluteus maximus, and the gluteus medius/minimus. Measurement of their pressure is mandatory. Plastic surgical techniques can be undertaken in order to treat these wounds and eliminate dead spaces. Split- or full-thickness skin grafts are used as well as suction drains, vacuum-sealed drainage, or free flaps (Fig. 21.9).

The management of the open pelvic fracture should follow the same guidelines and principles as for any open fracture of the extremities. The perineal wounds must be judged (because of their location) for a potential contamination of the fracture site and/or of the retroperitoneal hematoma. Perineal wounds involving or not the rectum require fecal diversion, early sphincter repair (when injured), and local wound management [54]. When placing the stoma, we should bear in mind the eventual location of any orthopedic incision, suprapubic catheter, and external fixator pins. The restoration of the continuity has an

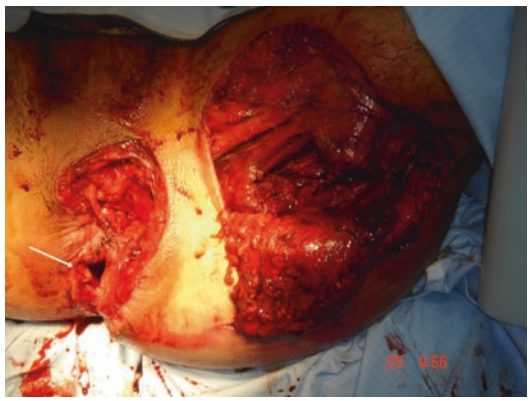


Fig. 21.8 Pelvic fracture with perianal laceration (*arrow*) and laceration to left buttock region

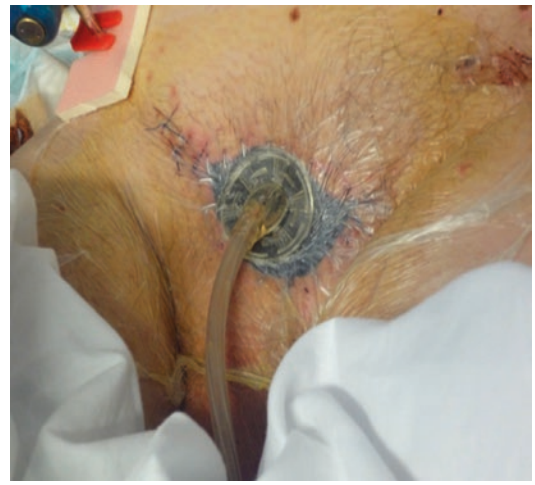


Fig. 21.9 Application of vac pack to open wound – pelvic fracture stabilized with anterior external fixator

important rate of complications, and therefore awareness of the patient with regard to this issue is of paramount importance [54].

In "complex" pelvic and acetabular fractures, perineal soft tissue swelling and "butterfly" hematoma is frequently present. This is the result of the extravasation of the retroperitoneal hematoma through the superficial perineal fascia of Colles. It can be managed with a scrotal sling for 5–7 days and then with a triangular sponge wedge (usually after surgical management of the lesions) [55]. Surgical insult to the perineum, local venous thrombosis, the pudendal post during skeletal traction, transient hypoalbuminemic state, and scrotal skin breakdown with sloughing and local infection can contribute to the expansion of the swelling.

21.5 Special Situations

21.5.1 Hemipelvectomy

A particular type of pelvic injury is that of the hemipelvectomy. This can be defined as an unstable ligamentous or osseous hemipelvic injury with rupture of the pelvic neurovascular bundle. Usually it is characterized by wide separation of the pubic symphysis and the sacroiliac joint, with various degrees of soft tissue and neurovascular disruption and stretching. The nonviable limb may still be attached to the trunk. Simultaneous and not sequential care is mandatory. Only in stable patients we can proceed to pelvic X-ray, USS, DPL, and retrograde urethro-cystogram. Direct pressure or obvious arterial clamping should be immediate.

It must be noted that a partial hemipelvectomy should be preferably completed. This is a lifesaving procedure as bleeding cannot be controlled when the limb remains partially attached [56]. Partial vessel injuries do not allow closure of the vessel lumen by muscular contraction. The remaining pelvis can be fixed internally, and the hemipelvectomy can be converted to a hip disarticulation if there is no massive wound contamination. Ultimate closure requires a spectrum of plastic surgical techniques [57]. Some complications seen with this type of injury include problems with wound healing, soft tissue and skin flap necrosis, iliopsoas necrosis from avulsion of its blood supply, local infection leading to lethal systemic sepsis, and meningitis probably secondary to ascending infection along the avulsed lumbar and sacral roots [58].

Conclusion

Pelvic fractures in high-energy trauma are usually associated with hemorrhagic shock. Early hemodynamic stabilization is of capital importance and should be taken into account starting from the prehospital care of the injured. Patients need to be managed in a synchronous fashion by a multidisciplinary team upon their admission to the trauma center. When a pelvic fracture is suspected, temporarily stabilization should be attempted before admission. The hemodynamic and pelvic stability, primarily, and the associated injuries will determine the sequence of management. The early adequate treatment of the pelvic fractures, starting from the hemodynamic parameters, decreases the mortality and improves the outcome. The concept of damage control orthopedics for pelvic fractures should be considered in borderline patients that do not respond to resuscitation. It should be applied in patients in an unstable and extremis physiological state (Table 21.4) [59].

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Principles of Damage Control for Pediatric Trauma

22

Christine M. Leeper, Andrew Peitzman, and Barbara A. Gaines

22.1 Introduction

Pediatric general surgery has embraced the concept of "damage control" operations as the standard of care for decades, though not necessarily by name. Temporary abdominal wall closure has been utilized by pediatric surgeons in the management of congenital abdominal wall defects since first described by Schuster in 1967 [1]. The use of prosthetic material for coverage of abdominal contents allowed for second-look procedures, avoidance of abdominal compartment syndrome, and delayed closure after resuscitation and stabilization. The effect of this strategy was to improve survival and decrease morbidity in critically ill neonates with a range of pathology (gastroschisis, necrotizing enterocolitis, midgut volvulus, etc.). The use of damage control principles for the management of trauma followed later as this practice increased in popularity for the management of injured adults.

The main tenets of damage control are the same in pediatric patients as in adult patients: minimize iatrogenic injury (hypothermia, hemodilution), address shock and coagulopathy with appropriate resuscitation, and achieve rapid surgical control of bleeding and fecal stream with delayed definitive closure if necessary. However, children have important anatomic and physiologic differences that require adaptation of or occasional divergence from adult damage control practices.

22.2 Epidemiology of Trauma in Children

Trauma is the leading cause of death for children and adolescents in the United States [2], as well as a source of tremendous morbidity and cost to patients, families, and society [3]. More

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than 90% of pediatric trauma is attributed to blunt force trauma, whereby the most frequent mechanism of injury in the children is falls; however, the most common cause of death is motor vehicle crash (MVC). The remaining 10% of pediatric injuries are attributed to penetrating trauma, of which firearm injuries are most lethal. Penetrating injuries are associated with a threefold higher mortality rate as compared to blunt trauma, with up to 70% of deaths occurring prior to hospital arrival [4]. Hemorrhagic injuries are much less prominent in pediatric patients, with traumatic brain injury being the number one source of morbidity and mortality in injured children [5].

22.3 Special Considerations: Pediatric Anatomy and Physiology

Clinicians often extrapolate best practice from the adult trauma management guidelines; however, this can be problematic as a still-developing child's anatomy and physiology can vary dramatically from that of an adult. These differences are important to identify in the setting of an injured child.

22.3.1 Anatomic Predisposition to Injuries

Regarding head and brain development, children have a greater head-to-body ratio, incomplete brain myelination, and thinner cranial bones which can result in increased head injury severity from a comparable blow. The smaller shape and size of the pediatric patient's body predisposes them to multiple injuries and increased severity of injuries, as the energy from traumatic incident will generate a much larger force compared to the adult patient. Children have decreased musculature and subcutaneous tissue as compared to adults. As a result, the liver and spleen are located more anteriorly in the abdomen, and the kidney is more mobile, which places these organs at greater risk of injury from abdominal trauma [6].

22.3.2 Hemodynamics

Unlike adults, children are usually healthy at the time of trauma and are rarely on medications that affect hemostasis or hemodynamics [7]. They have outstanding early compensatory mechanisms that allow for the loss of up to 45% of their circulating blood volume before becoming hypotensive [8]. Hypotension is therefore a late sign of hemorrhagic shock, and at this stage children are less able to recover and compensate for the hemodynamic changes. Therefore, one should have a high index of suspicion for hypovolemic shock in the presence of other supporting symptoms such as tachycardia, delayed capillary refill, and poor perfusion. Normal vital signs vary with the age group, and therefore a good basic familiarity with what is the normal range for each age group is essential using age-adjusted criteria (Table 22.1) [4, 8–10].

Table 22.1 Age-adjusted vital signs for pediatric patients [4, 8–10]

Age group	Respiratory rate (breaths/min)	Heart rate (beats/min)	Systolic blood pressure (mm Hg)	Urinary output (mL/kg/h)
Infant: 0–12 months	<60	<160	>60	2.0
Toddler: 1–2 years	<40	<150	>70	1.5
Preschool: 3–5 years	<35	<140	>76	1.0
School age: 6–9 years	<30	<140	>80	1.0
Preadolescent/adolescent: 10–18 years	<30	<100	>90	0.5

22.3.3 Hypothermia

The counterpart to exposure is consideration of the environment and the potential for hypothermia in children. Though smaller in size overall, infants and children have a proportionately large body surface area relative to their weight that predisposes them to greater amounts of heat loss and resulting hypothermia. Maintenance of normothermia is critical in preventing coagulopathy, cardiorespiratory compromise, and unnecessary increase in metabolic demands [11]. In addition to increased room temperature, additional methods such as warmed blankets, head wraps, warmed humidified oxygen, warmed fluids and blood, and radiant heat sources should be utilized promptly [6].

22.4 Damage Control Resuscitation

22.4.1 Acute Traumatic Coagulopathy

Acute traumatic coagulopathy is a multifactorial dysregulation in the hemostatic and inflammatory systems in response to an injury event. While exogenous contributors to coagulopathy may potentiate this process, namely, the "vicious triad" of dilution, acidosis, and hypothermia, ATC is a unique entity that encompasses the body's endogenous maladaptive response to injury. ATC as defined by admission INR $\geq$ 1.3 is common in critically injured children, with incidence rates that range from 20% to 40% [12–15]. ATC is clinically relevant as it predisposes patients to poor outcomes. In patients with traumatic brain injury, admission ATC is associated with worse outcomes on the Glasgow Outcome Scale, a measure of functional disability [16–19]. In patients with polytrauma, retrospective studies have found an association between admission coagulopathy and mortality [12, 14, 15, 20, 21]. This association is even more pronounced in patients with abusive head trauma [22], a mechanism of injury that is unique to the pediatric population.

Our understanding of the drivers and mechanism behind ATC in adults is advancing; endothelial activation and hyperpermeability leading to protein C activation, unregulated hyperfibrinolysis, platelet dysfunction Weibel-Palade body degradation, glycocalyx shedding, and inflammatory mediators [23–29] have all been implicated in the adult basic science literature. Literature regarding the pathophysiology of ATC in children is lacking. Children have a distinct hemostatic response to trauma and injury profile as compared to adults; however, the impact of this phenomenon on bleeding and coagulopathy has not been fully elucidated. Hemorrhagic shock is less common, while traumatic brain injury is the number one cause of morbidity and mortality in children [5]. Further, overall mortality is much lower [30], and late death after injury [31] and death due to organ failure or sepsis is a rarity in pediatric trauma patients [32]. The factors responsible for the disparate response to injury are largely unknown. Regarding hemostasis, the level and activity of clotting factors are incredibly varied across infancy, childhood, and adolescence [33]. It is clear that adult "normal" ranges and pathways cannot be assumed equivalent in children, and further research is indicated to clarify the etiology of ATC in a pediatric cohort.

22.4.2 Massive Transfusion in Pediatric Trauma

22.4.2.1 Defining Massive Transfusion in Pediatric Patients

Pediatric trauma encompasses the care of injured patients aged 0–18+, necessitating weight- and volume-based definitions of massive transfusion to standardize practice. Unfortunately, centers employ diverse definitions of massive transfusion that may include blood loss of greater than 40 cc/kg [34, 35], greater than 70 cc/kg [36–38], greater than 50% total blood volume lost in first 24 h [39], and greater than 50% total blood volume lost in first 3 h or 100% total blood volume lost in first 24 h [40]. Further complicating the development of a standardized definition is the fact that total

blood volume varies by age: infants less than 3 months have 90–100 cc/kg, children older than 3 months have 70–80 cc/kg, and obese children or adult-sized adolescents have 60–65 cc/kg.

In a 2015 study, using data from the Afghanistan and Iraq conflicts, Neff et al. addressed these inconsistencies to propose the use of 40 cc/kg of all blood products given at any time in the first 24 h to define pediatric massive transfusion. This definition identified children at increased risk of both 24-h and in-hospital death and distinguished a population that was more severely injured and more often in shock, hypothermic, and coagulopathic. Using this definition in future research, studies and protocols will allow for consistent and valid comparisons across cohorts undergoing massive transfusion.

22.4.2.2 Massive Transfusion Practices in Pediatric Centers

A recent survey of pediatric centers reported that 97% had a massive transfusion protocol in place for trauma patients [41]. Local definitions for what defined MTP were utilized and not expressly stated. All centers utilized trauma surgeon judgment to trigger MTP (100%), followed by other physician judgment (75%), transfusion of uncrossmatched packed red blood cells (56.1%), and hypotension (56.1%) as the next most common activation criteria. Out of 131 centers, 57% target 1:1 plasma to RBC ratio and 87.8% target ≥ 1:2 plasma to RBC ratios with the initial round of resuscitative products. Regarding platelets, 72.5% target 1:1 platelet to RBC ratio and 78.6% target ≥ 1:2 platelet to RBC ratios with the initial round of resuscitative products. Regarding the use of other adjuncts, 49% incorporate cryoprecipitate and 50% include antifibrinolytics (TXA or aminocaproic acid) in their MTP policies.

22.4.2.3 Component Blood Therapy Ratios

Massive transfusion protocols (MTP) have been shown to decrease morbidity and mortality in adult trauma patients by expediting time to transfusion and replacing red blood cells, plasma, and platelets in a fixed ratio (1:1:1) to minimize coagulopathy, hypothermia, and acidosis [42, 43]. In pediatric patients, the evidence regarding optimal ratios is lacking. Challenges to answering this clinical query are twofold: first, both the incidence of massive transfusion for hemorrhagic shock and the outcome of mortality are rare occurrences in pediatric patients. The vast majority of studies are inadequately powered to make a conclusion regarding mortality as an outcome and rely on retrospective data as prospective single-center studies are typically futile given the rarity of death. Second, most studies do not have an adequate experimental group, as many centers report failure to achieve 1:1:1 component ratio resuscitation in many cases despite the utilization of a massive transfusion protocol. Nosanov et al. reviewed 105 massively transfused trauma patients and concluded that higher plasma/PRBC and platelet/PRBC ratios were not associated with increased survival in children. However, only 19 died, 34% achieved 1:1 plasma/PRBC ratio, and 12% achieved 1:1 platelet ratios [39]. All deaths were due to traumatic brain injury. Chidester compared patients in whom MTP was activated with patients who received uncrossmatched packed cells but no formal activation. The plasma/PRBC ratio in both groups was 1:3, although the MTP target was 1:1. Mortality was equivalent between groups, though interestingly, DVT incidence was less in non-MTP group [44]. Hendrickson reported a pre-/post-MTP analysis that improved plasma/PRBC ratios (1:1.8 post vs. 1:3.6 pre) but again failed to achieve the target of 1:1. There was no difference in mortality [37]. Lastly, Edwards described a military cohort of injured patients in combat hospitals in the Middle East; 1:1 ratio was not associated with improved survival in massively transfused patients and in fact worsened survival in patients who were transfused but did not require MTP [36]. Prospective, multicenter trials are required to determine the effect of balanced ratio resuscitation in a critically injured pediatric population.

22.4.3 Directed Resuscitation/ Thromboelastography

Many adult centers are incorporating guided resuscitation strategies using viscoelastic studies (Fig. 22.1) as an adjunct or replacement

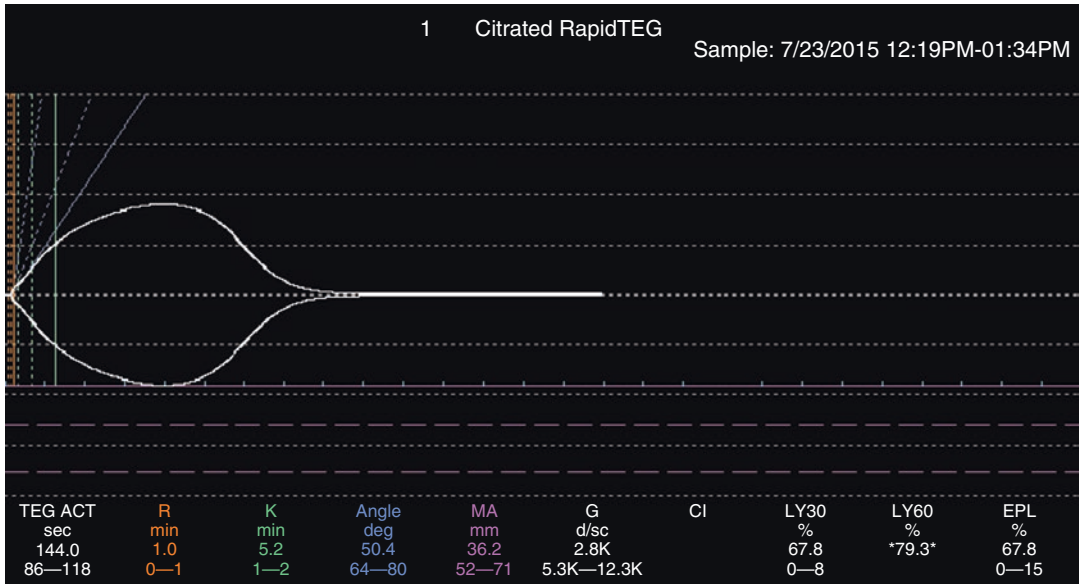


Fig. 22.1 Rapid thromboelastograph in a 7-year-old female with arterial laceration who presented in hemorrhagic shock. Tracing demonstrates inadequate clotting factors, fibrinogen, and platelets as well as primary hyperfibrinolysis

for empiric fixed-ratio transfusion. These studies, which include thromboelastography (Haemonetics© TEG©) or thromboelastometry (TEM, © TEM Systems Inc.), differ from conventional tests of coagulation like prothrombin time (PT) and partial thromboplastin time (PTT) in that they can provide detailed information regarding clot formation, kinetics, strength, and breakdown in a matter of minutes. Research in both adult and pediatric patients demonstrates that TEG derangements correlate with conventional tests, are better predictors of poor outcome and need for transfusion [20, 45, 46], and can be used to target patient's specific coagulation deficiencies during a resuscitation [47, 48].

22.4.4 Permissive Hypotension

Permissive hypotension is a resuscitative strategy in bleeding trauma patients that calls for judicious fluid administration while still maintaining end-organ perfusion. This approach is based on the premise that increased hydrostatic pressure and can inhibit or disrupt clot formation [49], large volume crystalloid can lead to dilution and hypothermia [50], and fluids may cause direct cellular damage through the activation of inflammatory mediators [27, 51]. There is evidence in the adult trauma literature and animal studies to suggest that permissive hypotension may decrease mortality in adults with hemorrhagic shock [52, 53]. There is no evidence in the pediatric literature to support or reject this strategy. However, hypotension is a late sign of hemorrhagic shock in children and often portends impending cardiovascular collapse. It is typically not well tolerated by pediatric patients, and therefore this strategy is not routinely utilized for pediatric trauma patients.

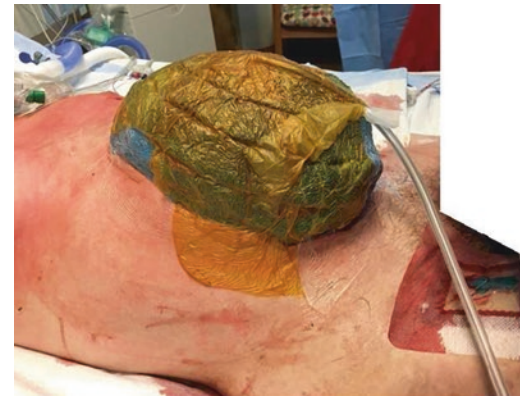
Further, hypotension in pediatric patients may be associated with isolated head injury as opposed to hemorrhagic shock. Permissive hypotension is not appropriate in patients with traumatic brain injury as even a single measure of systolic blood pressure <90 mmHg in the course of prehospital and initial hospital resuscitation results in increased mortality and disability [54–56]. A recent study of the National Trauma Databank revealed that hypotension occurred after isolated head injury in children of all ages and the risk of hypotension after head injury is as great as after hemorrhagic injuries in children aged 0–4 years [57]. While hypotension is considered to be

secondary to hemorrhagic shock until proven otherwise, the source of hypotension in pediatric patients is not always clearly defined. This high proportion of pediatric patients with traumatic brain injury is also a deterrent to wide adaptation of this strategy in children.

22.5 Damage Control Surgery for the Pediatric Trauma Patient

Damage control surgery for the pediatric patient achieves the same goals as in adult patients: early control of surgical bleeding, control of intra-abdominal contamination, abdominal packing for control of coagulopathic bleeding, temporary abdominal closure and delayed definitive procedures and closure to allow for second-look operations, resuscitation in the setting of coagulopathy, hypothermia and acidosis, and avoidance of abdominal compartment syndrome. As fewer injured children present in hemorrhagic shock, the relative infrequency of this strategy in the pediatric trauma patient precludes large clinical trials to investigate optimal timing of operations, various closure devices, resuscitation strategies, etc. However, there are published case reports and many anecdotal experiences to demonstrate the successes of damage control surgery in children [58–61].

As in adults, a rapid transfusion device and cell saver should be available in the event of massive blood loss. The patient is prepped from the neck to knees to allow for entrance into either the chest or abdomen and to permit access to the femoral vessels. Upon entrance to the abdomen, the four quadrants are packed to tamponade the bleeding, and the abdomen is then explored in a systematic fashion after allowing time for the anesthesiology team to resuscitate and transfuse as needed. Surgical bleeding and fecal contamination should be addressed expeditiously, and temporary abdominal closure should performed using any number of methods, including negative pressure wound dressings, temporary patch abdominoplasty, sterile bag closure (Bogota bag, bowel bag, Steri-Drape, or silo), and skin-only closure (Fig. 22.1).



15 year old patient who sustained liver and bowel injury due to blunt abdominal trauma. A negative pressure wound dressing is in place after damage control procedure (Courtesy: Children's Hospital of Pittsburgh of UPMC)

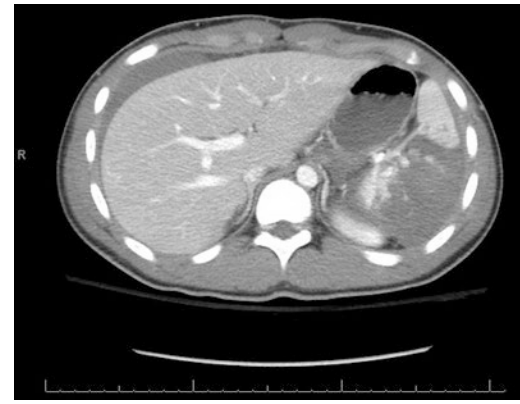


Fig. 22.2 Grade 4 splenic laceration with a large intraparenchymal hematoma and active extravasation, managed nonoperatively

Solid organ injury is common is pediatric trauma patients. The spleen is the most commonly injured solid organ (25–39%), followed by the liver (15–37%), kidney (19–25%), and pancreas (7%) [62]. Management and outcomes of the pediatric patient with solid organ injury differs dramatically from management of the adult patient with similar grade of injury. In contrast to adults, splenic injury and hepatic injury in children are typically successfully managed nonoperatively in the vast majority of cases even with high-grade injuries [63] (Figs. 22.2 and 22.3). Therefore, a trial of resuscitation and observation is indicated for most solid organ abdominal injuries. Failure of nonoperative

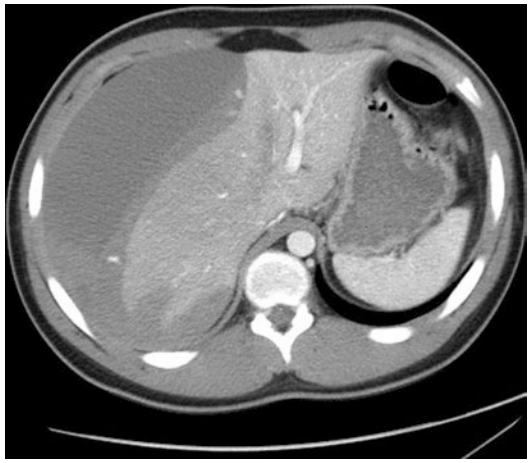


Fig. 22.3 Grade 4 liver laceration with large subcapsular hematoma, managed nonoperatively

management in patients attempting conservative management is signified by worsening abdominal exam, ongoing transfusion requirements, and hemodynamic instability (particularly hypotension) [64]. Immediate operative intervention is indicated in patients with hemodynamic instability, peritonitis or suspected hollow viscus injury, and major pancreatic ductal disruption.

22.5.1 Splenic Injury

Splenectomy easily controls bleeding from a massively injured spleen and is indicated in the hemodynamically unstable patient. Children with splenic injuries who have ongoing bleeding, but are not in shock, are potential candidates for splenic sparing operations such as partial splenectomy and mesh splenorrhaphy; however, these techniques can be time-consuming and are not appropriate in the setting of hemorrhagic shock [7]. The use of splenic artery embolization has increased the success of nonoperative management in the adult population, while the role of interventional radiology is less clear in the pediatric population where splenic preservation is already the rule rather than the exception. However, limited data suggests that splenic artery embolization is feasible and safe, may increase the rate of splenic salvage in children [65–67], and results in preserved splenic function [68], particularly those with high-grade injuries or evidence of splenic vascular injury.

Overwhelming postsplenectomy infection (OPSI) is a rare consequence of splenectomy with an incidence of 0.23% per year that is increased in children less than 2 years of age. Vaccinations against encapsulated bacteria, including pneumococcus, *Haemophilus influenzae* type B, and meningococcus, should be administered after splenectomy [69]. Children who receive antibiotic prophylaxis have a decreased incidence of OPSI; therefore, it is recommended for all pediatric patients after splenectomy for trauma. Oral penicillin V twice daily or amoxicillin is first-line therapy, with erythromycin or Bactrim as alternates for patients with penicillin allergy. The duration of antibiotic prophylaxis is controversial and evidence-based guidelines are lacking. Most experts recommend that prophylaxis be continued until age 5 or, for patients who become asplenic after age 5, for at least 1–2-year duration. For high-risk patients, prophylaxis is typically continued into adulthood. Long-term use of antibiotics carries the increased risk of antimicrobial resistance; therefore, the decision to continue antibiotics should be based on the clinical circumstances of each individual patient [70–72].

22.5.2 Liver Injury

A major hepatic injury is more difficult to control in the operating room; high-grade injury, hepatic vascular injury, and the need for operative intervention are associated with high rates of mortality in both adult and pediatric patients [73]. The likelihood of successful intervention is increased with adequate operative exposure, an experienced co-surgeon, good anesthesia support, and supradiaphragmatic intravenous access. Operative technique may include parenchymal compression, the use of the Pringle maneuver with intermittent clamping of the porta hepatis, suture ligation of bleeding vessels, and the avoidance of deep liver sutures [74].

Goals of the definitive operation are to ensure adequate hemostasis, control bile leak, debride

nonviable tissue, and adequately drain the resection bed. Large parenchymal fractures are best treated with anatomic or nonanatomic resection, assuming sufficient residual liver remains [7]. Resection can be efficiently performed using mechanical staplers. Adjuvant interventions may include interventional radiology procedures such as angioembolization or endoscopic retrograde cholangiopancreatography with stenting. These procedures are performed less commonly in pediatric populations [75]; however, the experience of multiple high-volume trauma centers over the past decades demonstrates both feasibility and safety in children and suggests diagnostic and therapeutic benefits when utilized in appropriate pediatric patients [65, 66, 76]. These patients may include those with evidence of ongoing bleeding and hemodynamic stability and those with concern for ongoing bleeding after damage control procedure or later in their hospital course. Follow-up imaging for patients with high-grade injury may be warranted and should be pursued based on clinical presentation and postoperative condition.

22.5.3 Kidney and Pancreas Injury

Operative intervention for kidney injury is indicated in patients with hemodynamic instability, expanding retroperitoneal hematoma, vascular pedicle injury, or other major renal vascular injury [77]. In an initial damage control operation, nephrectomy can be performed for massive parenchymal destruction and ongoing bleeding, and the ureter may be ligated with plans for future urinary reconstruction once the patient has stabilized.

Pancreatic injury is uncommon in children and typically does not require surgical intervention at the initial damage control surgery. Regarding the definitive operation, optimal management is controversial and frequently debated among experts. For both penetrating and blunt pancreatic injuries, the presence of main pancreatic ductal injury and the location of injury (proximal vs. distal) are the major factors guiding management decisions. For main pancreatic ductal injury (Grades 3–5), resection is the preferred approach with distal pancreatectomy for most distal duct injuries and conservative interventions (closed suction drainage and endoscopic stenting) for proximal ductal injuries involving the head of the pancreas [78]. Preservation of the spleen, particularly in pediatric patients, should be prioritized if possible. Radical procedures such as pancreaticoduodenectomy or the creation of pancreatic-enteric anastomoses are not widely recommended.

22.5.4 Hollow Viscus Injury

Hollow viscus injuries in pediatric patients are typically the result of a direct blow to the abdomen, a deceleration mechanism (e.g., belted patient in MVC), handlebar injury, or associated lumbar spine injury [79] and involve the jejunum, duodenum, colon, and stomach in decreasing order of frequency [62]. The diagnosis of intestinal injury can be challenging as imaging is less sensitive than for solid organ injuries and a patient's symptoms may be mild, delayed, and nonspecific. Clinicians should have a high index of suspicion in patients with tachycardia, abdominal wall bruising, or seat belt sign [80, 81]. Symptoms may include abdominal pain, peritonitis, and bilious emesis. Radiologic indicators may include free fluid, but often imaging is normal. Laboratory findings may include elevated lipase, white blood cell count, or lactate.

Immediate operative intervention is indicated in patients with hemodynamic instability, peritonitis, or in patients with a high degree of suspicion for injury based on the presence of above factors. The injuries resulting from hollow viscus trauma can include frank bowel perforation or tearing, bowel wall hematoma, and mesenteric tears that may devascularize bowel and result in ischemia or necrosis. The first priority of the damage control procedure is control of intestinal content spillage and removal of devitalized tissue

with delayed restoration of bowel continuity or diversion at subsequent operations when the patient is hemodynamically stable.

22.5.5 Child Abuse

Abusive head trauma (AHT) is an injury mechanism unique to the pediatric population that preferentially affects the youngest and most vulnerable patients. The incidence of AHT is estimated to be 20–30 cases per 100,000 children [49] with a case fatality rate that ranges from 15% to 35%. Presentation to care for abused patients peaks around 2–3 months of age, and the majority of children are 2 years of age or younger [49, 56]. Challenges in caring for this cohort include frequent delays in diagnosis as young patients cannot report their abuse history and guardians may not be forthcoming about or aware of the abuse [57–59], as well as the fact that patients likely have suffered multiple discrete events over a period spanning days to months [60] and identification of "time of injury" may be impossible.

The abusive head injury population differs clinically from the accidental head injury population in that they have higher mortality rates [49–51] and worse neurologic outcomes that persist decades after injury [49, 52–55]. The reasons for this are multifactorial; from a mechanistic standpoint, evidence suggests that the pathophysiology of brain damage in abusive head trauma does not result from a direct traumatic mechanism, but instead from a hypoxic-ischemic insult that occurs after trauma-induced apnea [61–64]. Hyperextension injury to the neck due to violent shaking has been implicated in damaging the central pattern generator of respiration in the brainstem [61].

Evaluation of patients with suspected child abuse should start with a thorough medical history and physical exam with careful photographic and written documentation of all findings. Patients with high suspicion of abuse should also have a complete skeletal survey, head CT if facial/scalp bruising, neurologic symptoms or other concurrent injuries of abuse, and CT abdomen/pelvis if signs of abdominal trauma or elevated transaminases. Consultation to ophthalmology for a dilated eye exam, neurosurgery for intracranial hemorrhage and neurology for seizures may be indicated. Social work involvement with notification of the appropriate authorities and plans for safety at discharge are imperative.

Damage control procedures in victims of child abuse follow the same principles of damage control surgery in accidental trauma patients. One consideration that complicates diagnosis and management of these patients is that clinical presentation of injuries may be unusual. For instance, a patient with massive abdominal hemorrhage requiring immediate laparotomy may present with cardiac arrest of unknown etiology, chief complaint of "found down," "fussy" "irritable" or nonspecific nausea, vomiting, or mental status changes. Maintaining a high index of suspicion will facilitate early intervention and appropriate resuscitative and operative strategies to address the severe injuries that a badly abused child has sustained. Another note for the trauma surgeon is the importance of recognizing injuries consistent with the history versus those consistent with a diagnosis of child abuse (e.g., a patient found to have duodenal transection where the history is a fall from 4 ft while playing). Unless a patient has a mechanism to support the injury (motor vehicle collision deceleration injury), this is diagnostic of child abuse and should be stated unequivocally as such.

22.6 Damage Control Principles in Pediatric General Surgery

22.6.1 Abdominal Compartment Syndrome (ACS)

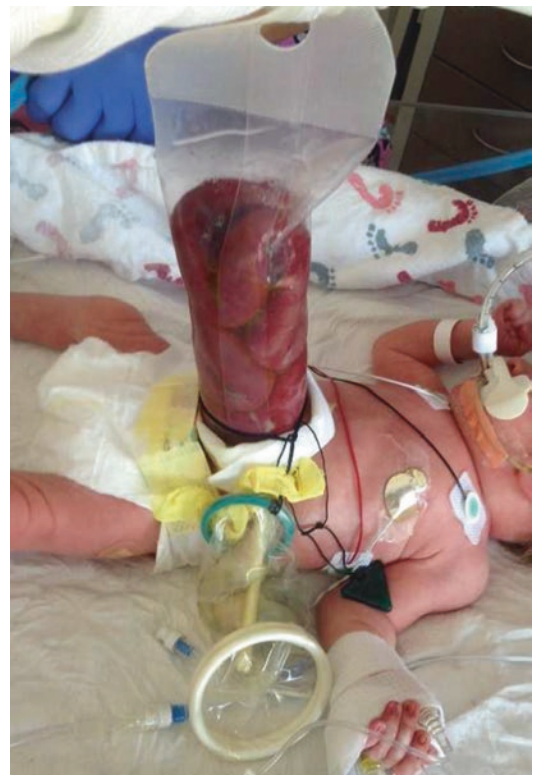
Intra-abdominal hypertension is defined as an intra-abdominal pressure (IAP) greater than or equal to 12 mmHg, while ACS is defined as an IAP above 20 mmHg with the development of

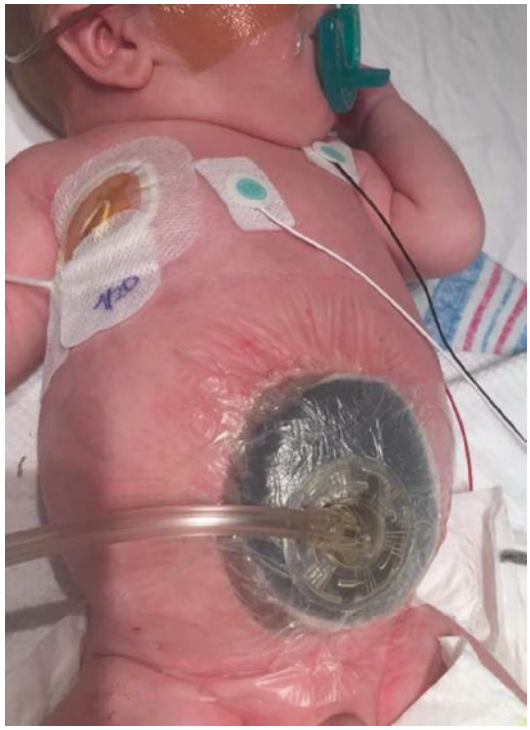
new or worsening organ dysfunction/failure [82, 83]. It is often diagnosed in a delayed fashion in pediatric patients perhaps due to lack of familiarity or recognition of this phenomenon and is associated with high mortality rates between 40% and 60% in children [84–86]. While the most common cause of ACS in children is abdominal trauma with hemorrhage or visceral edema, other causes include intraperitoneal sepsis, massive fluid resuscitation, massive ascites, rapid tumor growth as in stage IV-S neuroblastoma, and severe constipation [87, 88].

ACS can be diagnosed and monitored using bladder pressure measurements. Initial medical management includes adequate sedation and paralysis, evacuation of intralumenal intestinal contents, evacuation of large abdominal fluid collections, optimization of fluid administration by goal-directed therapies, and correcting positive fluid balance. Surgical management includes decompressive laparotomy with the goals of decreasing the elevated IAP to stop organ dysfunction, allowing room for expansion of the viscera during ongoing resuscitation, providing temporary abdominal viscera coverage, preventing excessive fascial retraction, and allowing a means for continued evacuation of fluid from the peritoneal cavity [7, 89]. Coverage can be achieved through multiple methods including application of a negative pressure wound dressing (e.g., vac-pac), temporary patch abdominoplasty, or silo. Staged abdominal closure is performed when ACS has resolved.

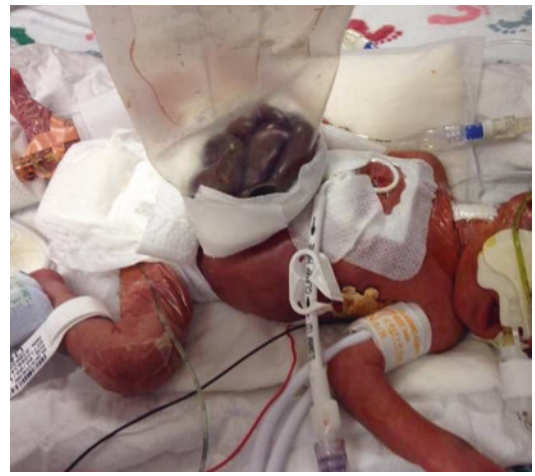
22.6.2 Abdominal Wall Defects: Gastroschisis

Gastroschisis is a congenital abdominal wall defect in which the intestines fail to assume their position in the peritoneal cavity in utero and develop outside the abdomen. Postnatal management requires early coverage of abdominal contents followed by definitive closure of the abdominal wall defect. Historically, immediate primary repair was routine practice as patients did not survive without closure. Schuster first described the use of prosthetic material for temporary closure in infants in 1967 [1]. In patients at risk of developing abdominal compartment syndrome, the use of a silo allowed for gradual intestinal reductions through serial procedures either in the operating room or at the bedside, followed by delayed abdominal wall closure (Fig. 22.3). Patients who undergo silo placement as compared to primary repair have similar complications and mortality but longer hospital stays and costs. About 50% of patients with gastroschisis will develop subsequent ventral hernia that may require later repair [90–92]. Sutureless closure of the abdominal wall defect with negative pressure dressing has also been utilized in many institutions and can be performed at the bedside [93] (Fig. 22.3).





This strategy allows for abdominal decompression as well as periodic evaluation of bowel viability in cases where the initial operation revealed marginal segments of the intestine or concern for ongoing ischemia.

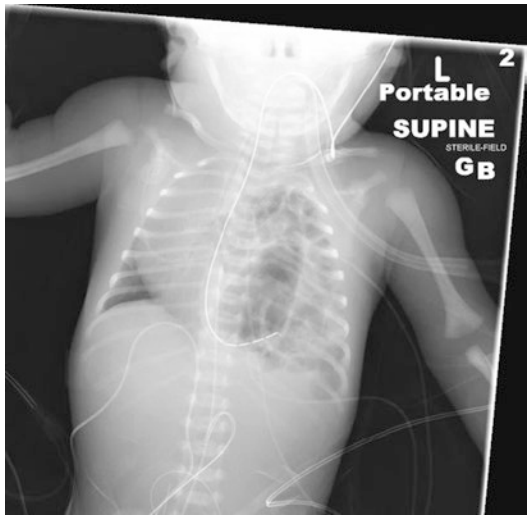


22.6.3 NEC

Necrotizing enterocolitis (NEC) is a multifactorial disease that leads to disruption of intestinal integrity followed ultimately by bowel necrosis and bacterial translocation. Birth weight is inversely associated with incidence (1:1,000) and mortality (15–30%). Younger gestational age is also a predictor of poor outcome. The clinical presentation of NEC includes feeding intolerance, abdominal distention, and often bloody stool [94]. Pneumatosis intestinalis is the hallmark radiographic finding, and operative indications include worsening hemodynamics or physical exam, free intraperitoneal air, or clinical concern for bowel necrosis. Initial abdominal exploration accomplishes removal of devitalized intestine and diversion versus primary anastomosis depending on bowel character and a patient's clinical status. Fascial closure may be accomplished at that time; however, TAC may be indicated in some populations.

22.6.4 Congential Diaphragmatic Hernia

Temporary abdominal closure is also useful in the reduction and repair of a large congenital diaphragmatic hernia (CDH). The most common type of CDH is a Bochdalek hernia, or defect in the posterolateral diaphragm, the majority of which occur on the left side. Clinical presentation includes respiratory distress in a newborn caused by pulmonary hypoplasia and pulmonary hypertension. Radiographic findings demonstrate passage of abdominal contents into the chest cavity. Reduction of these contents into the abdomen during operative repair can result in intra-abdominal hypertension. Further, a recent study noted a reduction in cerebral blood flow in infants who underwent repair of a large CDH with primary abdominal closure [95]. The use of TAC devices has been shown to be feasible and effective in reducing both IAH and the resultant decrease in cardiac output [88, 96–98].



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Principles of Damage Control in the Elderly

23

Pol Maria Rommens and Sebastian Kuhn

23.1 Introduction

Demographic development in high-income countries leads to an absolute and relative increase of elderly people. Whereas the longevity has steadily grown, birth rate decreases to a value below the population replacement rate. In *The World FactBook 2011*, Germany ranks second in the statistics of median age of the country's population. Only Japan has an older population with a median age of population of 46.7 years. Germany has a median age of 46.0 years. On the places three to six rank Canada, Great Britain, Poland and the USA with mean ages of their population of 42.1, 41.1, 40.3 and 38.2 years respectively [1]. In comparison with 2016, the total population of the EU will have decreased with 5% in 2050, whereas the rate of persons above 60 years will have increased with 40% [2].

Among the elderly population, many are healthy, mobile, and sportive and have high demands on quality of life and mobility. Conduct of an active lifestyle leads to participation in "dangerous" activities such as being pedestrian within dense traffic, using motor vehicles or motorcycles, hiking and climbing, or working at a height (e.g., on a ladder). All this leads to an increasing part of elderly persons among polytraumatized patients. Broos et al. calculated in 1988 a percentage of 12% of persons above the age of 65 years (n = 49) among 416 multiple injured patients [3]. More than three decades later, the percentage of polytraumatized patients of 65 and older has risen up to 30% in the USA [4]. The German TraumaRegister DGU® documents an increase in the average patient age from 39.0 years in 1999 to 50.9 years in 2014. Currently patients of ≥60 years of age account for 37% of all polytraumatized patients [5]. Whereas the percentage of severely injured patients in adolescents and young adults is strongly in favor of males and the percentage of low-energy trauma in elderly is strongly in favor of females, the incidence of severely injured in the elderly is equal among females and males [6]. Blunt trauma mechanisms account for almost all injuries in the multiple injured older persons [7]. Motor vehicle accidents and falls from height as well as pedestrians being hit by a motor vehicle are the most frequent causes of trauma [8, 9].

There is a rapidly increasing interest in the older polytraumatized patient. In recent literature, these patients have been identified as a specific group of trauma patients, which require

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special attention and adapted treatment protocols. In the large majority of these recent papers, elderly persons are classified as being 65 years and older.

23.2 Age-Related Physiologic Alterations in Adults

Aging is characterized by a continuous decline in physiological reserves. Moreover, aging is connected with the appearance of diseases. In a recent US study on 32,440 patients of 55 years of age and older, who suffered hip fractures, more than 95% had at least one comorbidity. Hypertension, deficiency anemia, and electrolyte disorders were the most common [10]. Loss of physiological reserves and comorbidities in the elderly are both considered as independently influencing factors in regard to morbidity and mortality after trauma [11].

The most important declines of organ function concern the heart, lungs, kidneys, liver, and bone. A reduced cardiac output and a low physiologic reserve of the lungs limit the ability to respond to hemodynamic instability due to blood loss. As a result of reduced functions of kidneys and liver, clearance of metabolic and toxic agents and drugs is delayed or hindered. Bone mineral density is reduced in elderly persons. A similar trauma mechanism may lead to more and more complex injuries than in the younger adult. These factors negatively influence morbidity and mortality [12, 13].

One or several comorbidities are present in the vast majority of the elderly. Hypertension and heart disease are very common. There is a reduced ability to respond to hypovolemia and shock. Younger adults react to hypovolemia with centralization of intravascular volume leading to increased heart rate and temporary increased blood pressure. The elderly are not able to develop the same response. Moreover, this physiological response may be hindered due to medications, especially beta blockers and steroids, which are often prescribed in the elderly. A normal heart rate or a normal blood pressure may therefore give the false impression of the patient being less severely injured than he/she really is or appearing to respond to resuscitation [14]. An increasing number of elderly persons receive anticoagulants for prophylactic (e.g., atrial fibrillation, after vascular stent implantation) or therapeutic (after thromboembolism) reasons. These drugs, especially coumadin, phenprocoumon, low-molecular-weight heparin, and factor X blockers, may prolong duration and volume of blood loss after trauma [15]. Oral anticoagulant drug therapy should be interrupted and replaced by low molecular or unfragmented heparins given as a "bridge treatment."

Chronic obstructive pulmonary disease (COPD) is a common disease of the elderly and is often connected with congestive heart failure. COPD leads to a reduced vital capacity and pulmonary function, which increases the risk of pulmonary complications during and after surgery [16]. Steroids, which are given for COPD may alter the physiological response to trauma, including a reduced immunologic reaction and delayed wound healing.

Anemia is frequently detected in the elderly. The blood transfusion regimen after severe geriatric trauma should therefore not be too aggressive, with hemoglobin values of 10 or lower generally being acceptable as endpoint of resuscitation [17].

Also diabetes is often diagnosed in persons older than 65. This disease is responsible for an important number of consecutive complications such as peripheral microangiopathy, peripheral neuropathy, heart and renal failure, retinopathy with reduced eyesight, and wound healing disturbances. Glucose levels should be kept in normal ranges throughout the whole length of intensive care and in-hospital stay [18, 19].

23.3 Mechanisms and Patterns of Injury

Low-energy injuries, especially domestic falls, remain the predominant cause for fractures in the elderly. The risk of suffering a fracture after a fall steadily increases with age. Comorbidities such as cardiac arrhythmia, decreased vision, peripheral neuropathy, and a longer reaction time make the elderly more prone to a fall. Decreased bone

mineral density due to osteoporosis is another factor which dramatically enhances the risk of suffering a fracture after a fall [20].

Blunt trauma is also the leading cause of high-energy injuries in the elderly. Motor vehicle accidents, either as the driver or as a pedestrian hit by a car and falls from height, are the number one and two mechanisms of polytrauma [8, 9]. Different studies show high percentages of *traumatic brain injury* and of *musculoskeletal injuries*, whereas thoracic and abdominal injuries are less common [9, 21]. In a retrospective analysis of the National Trauma Data Bank of USA (2002–2006), Siram et al. found that elderly patients (above 75 years of age) had significantly higher rates of *pelvic, upper*, *and lower extremity fractures* as well as intracranial injuries than younger adults [22]. Sullivan et al. performed an epidemiologic study using data from 1993 to 2010 of the Nationwide Inpatient Sample (NIS) looking at more than 600 million Medicare-paid hospital discharges of patients above the age of 65. Whereas traditional hip fractures declined by 25.7%, geriatric *acetabular fractures* increased by 67% and *pelvic fractures* by 24% [23]. Although most of the NIS patients suffered a low-energy trauma, this data supports our observation that the incidence of pelvic and acetabular fractures is rapidly increasing in the old patient population. In a retrospective Canadian study on 276 multiple injured patients of age 65 and older between 2004 and 2006, *spine injuries* were present in 17% [7]. The type of treatment of spine injuries is relevant for outcome. In a retrospective study on 154 severely injured patients with a mean age of 76 years and an ISS of 23, nonoperative treatment of spine injuries was associated with a higher mortality [24]. Due to the high incidence of osteoporosis, *blunt chest trauma* in the elderly is commonly combined with rib fractures. There is a higher mortality than in younger adults and children [25]. With the number of rib fractures, mortality and morbidity (number of ventilator days, number of intensive care unit (ICU) days, pneumonia) increase significantly [26]. *Abdominal trauma* is less frequent than traumatic brain injury or musculoskeletal injuries. It is usually associated with motor vehicle accidents and associated with a higher mortality. In an older Swedish retrospective study over a 30-year period, mortality after abdominal trauma in patients older than 60 years was 27.6%, whereas mortality in the younger patient population was 9.1% [27]. In a more recent retrospective study performed over a 6-year period on 90 patients with age over 55 years, mortality after trauma laparotomy was 23.3% and progressively increased with age [28].

23.4 Principles of Management of the Polytraumatized Elderly

23.4.1 Triage

The principles of advanced trauma life support (ATLS) should also apply for the elderly. Airway, breathing, and circulation have the highest priority in posttraumatic resuscitation. Life comes before limb. The main challenge of the prehospital phase is the correct estimation of the severity of the injuries and their impact on the physiologic status of the patient. Underestimation is a common problem and has different reasons: inadequate training, unfamiliarity with protocol, and age bias [29]. In a retrospective study performed at the Maryland Ambulance Information System over a 10-year period, under triage rate was significantly higher in patients aged 65 years or older: 49.9% versus 17.8%. As stated above, the response to trauma is reduced in elderly victims due to a limited physiologic reserve, comorbidities, and medication. This may lead to the false impression that the "first hit" was not that severe. Underestimation leads to undertreatment, which enhances the risk of mortality. Emergency medical service providers and emergency physicians, who are not really familiar with treatment protocols of elderly trauma victims, transported their patients to non- or low level trauma centers, where adequate treatment was not initiated or delayed. A third problem may be age bias. Some care providers may feel that it is not worthwhile starting aggressive resuscitation in the severely injured elderly, as the chances for a good outcome are regarded as bad or non-existing [29]. A retrospective cohort study conducted in Ohio,

USA, on 455 trauma patients of 80 years and above showed that patients, who were sent directly to a trauma center, had significant better outcomes than those directed to an acute care hospital [30]. Other publications suggest that existing triage protocols are inadequate for assessing the severity of injury in the elderly trauma patient [31–33]. When age and comorbidities are integrated in the decision on where to transport the patient, the rate of under triage is considerably diminished [34]. The statement *bring the right patient at the right time to the right hospital* is more valid than ever for elderly trauma patients. In doubt, such a patient should be transported to a trauma center.

23.4.2 Resuscitation at Admission

In-hospital treatment should be guided under the goal not to harm the patient. Too aggressive resuscitation or prolonged and extensive surgeries may bring the patient more harm than benefit [35]. Even though the "second hit" may not be excluded completely, it should be minimized as much as possible. A basic rule is to avoid the lethal triad of hypothermia, coagulopathy, and acidosis [36].

Hypothermia, as frequently observed during primary survey and initial resuscitation, has been recognized as an independent factor, which increases morbidity and mortality after severe trauma [37]. Hypothermia reduces cellular activity and metabolism, such as the activity of the factors of the coagulation pathway [38]. Noninvasive and invasive measures to warm-up the patient must be used as soon as possible in the emergency room to prevent or treat hypothermia: higher room temperature, thermal blanket, and warmed-up intravenous infusions [39]. Already at the stage of pre-ICU resuscitation, massive hemorrhage protocols should be started to compensate blood loss. It is recommended to combine packed red blood cells (RPBC) with fresh frozen plasma in a 1:1–1:2 ratio for simultaneous treatment of blood loss and coagulopathy [40, 41]. Platelets should be administered to maintain a platelet count above 50×10^9/l in all trauma patients and above 100×10^9/l in patients with ongoing bleeding and/or traumatic brain injury. Alternatively, a concentrate-based goal-directed strategy has proven to be effective. An early use of prothrombin complex concentrate is recommended for the emergency reversal of vitamin K-dependent oral anticoagulants [17]. Coagulation monitoring to measure coagulation treatment should be implemented as early as possible and used to guide further hemostatic therapy. Monitoring should include rotary thromboelastomer [14, 17].

Base deficit with values of −6 mmol/L or lower has been shown to be a sign of severe injury and enhanced mortality in all, but especially in elderly patients [42]. Base deficit and serum lactate are markers for peripheral tissue perfusion and oxygenation. Consequently, high base deficit (−6 mmol/L of lower) and high serum lactate levels are specific for bad peripheral perfusion and low oxygen consumption. In a retrospective cohort study, Callaway et al. looked at the relation between lactate levels and in-hospital mortality in blunt trauma elderly patients. There was a 4.2 increased odd ratio of dead in patients with a severely increased lactate level. There was a 4.1 increased odds ratio of dead in patients with a markedly increased base deficit [43]. This data clearly supports early and continuous invasive monitoring and intensive care therapy in this group of patients. Scalea et al. showed already in 1990 that "emergent" invasive monitoring could reduce mortality in severely injured elderly patients from 95% to 47% [44]. This was confirmed by the data of Demetriades et al. who performed a comparative study on 336 patients of age 70 years or more. Mortality with early invasive monitoring could be reduced from 53.8% to 34.2% [45].

23.4.3 Primary Operative Phase

In this early operative phase, the surgeon should be guided by two directives: *treat first what kills first* and *do not harm*.

Most patients with a pneumo- and hemothorax after blunt thoracic trauma are successfully treated with a chest tube [25, 26]. Besides aiming for a better oxygenation, inserting a chest

tube is part of the monitoring of ongoing blood loss or air leakage, which may enforce thoracotomy in a small minority of patients. There must be a high index of suspicion in patients with multiple rib fractures. In a retrospective cohort study on 171 patients, one third of patients older than 45 years of age with more than four broken ribs had pulmonary contusions and half had hemopneumothorax. This population suffered a higher morbidity (ventilator days and hospital length of stay) than their younger counterparts [26, 46].

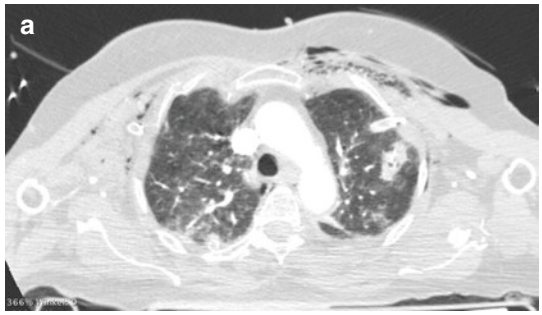
Damage control laparotomy is useful, also in elderly patients. In a retrospective study performed at a level I trauma center, more than half of the patients of 55 years of age or older survived, despite higher mortality than in younger patients [47]. The main goals are to stop bleeding and to interrupt contamination. The surgery is abbreviated; long surgery is avoided. Complete and definitive repair of the injuries is postponed to later operative phases, when physiologic disturbances (lethal triad) have been corrected by intensive care therapy. Life-threatening injuries such as liver bleeding or splenic rupture are treated by liver packing and splenectomy. Ruptured or ischemic bowel is treated with partial resection without anastomosis. The abdomen is left open, which avoids intra-abdominal compartment syndrome, shortens operation time, and enforces second-look surgery [48].

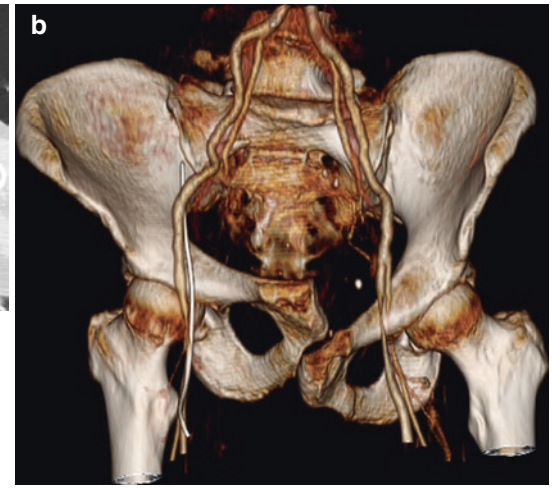
Musculoskeletal injuries are the most frequent lesions in elderly severely injured persons. Not all fractures have to be stabilized in emergency. The main goals of emergent treatment of fractures are to control major instability, stop contamination, and reduce pain. The techniques used should be less aggressive, limited in time consumption, and ensure high enough stability for easy nursing in an intensive care unit.

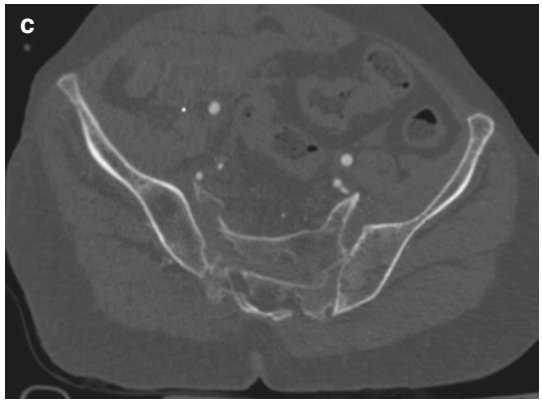
High-energy pelvic trauma requiring surgical treatment belongs to these skeletal injuries and is associated with high mortality rates in elderly patients [24, 49]. Osteo-ligamentar instability leads to life-threatening hemodynamic instability due to blood loss in the small pelvis. Several provisional measures can be taken to reduce pelvic ring instability. In patients with suspected pelvic instability with signs of hemodynamic instability, pelvic binders should already be applied at the site of accident prior to transport to a trauma center. At the latest, they should be applied in the emergency room [50]. The benefit of the binder has been proven in several studies [50, 51]. To avoid pressure sores, the binder should not be retained for longer than a few hours. Especially elderly patients with a frail and damaged soft tissue envelope are at high risk of developing pressure sores or open wounds. Pelvic clamping and external fixation both restore stability of the broken pelvic ring and reduce blood loss [52, 53]. In elderly, special attention should be paid preventing penetration of the screws through the weaker bony structures. We therefore prefer the use of the supra-acetabular external fixator [54]. In case of combination of pelvic with abdominal trauma, which necessitates laparotomy, it is of utmost importance applying the external fixator first. Laparotomy in patients with unstable pelvic trauma without previous external fixation is connected with very high mortality [55]. The external fixator can be left in place for definitive treatment of anterior pelvic ring instabilities. With meticulous care of the pin tracks, infection rate is low [52]. If present, posterior pelvic ring instability must be fixed in a second, scheduled surgery.

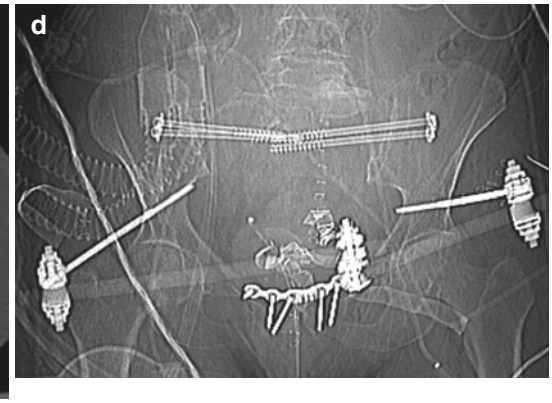
Pelvic packing is a damage control procedure, which is only used in patients, who do not respond well to external compression despite aggressive resuscitation. Overall mortality is as high as 28% in adults [56]; survival of elderly patients after pelvic packing is not known but may be lower than in younger adults. Angiography and selective embolization is a well-known and effective damage control procedure for intrapelvic arterial bleeding. Technical success ratio is published to be near 100%, clinical efficacy 85% [57]. The challenge is identifying the patients with arterial bleeding and adequate timing of the intervention. Patients with a high pelvic AIS and a low base excess level on admission as well as patients with a blood transfusion need of more than 0.5 units/h were found to be at high risk of active arterial bleeding [58]. When an emergency total body CT scan with contrast, ideally including an

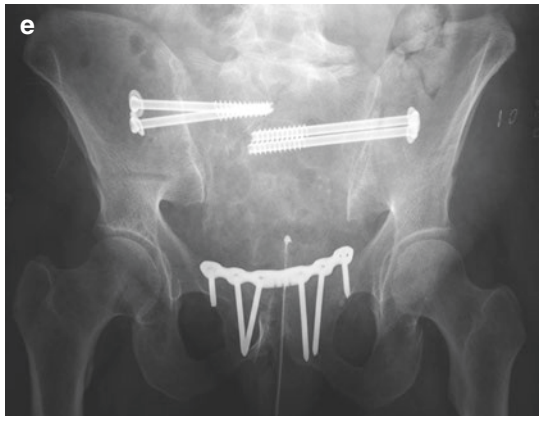
arterial phase, is performed very early after admission, presence and location of intrapelvic arterial blush can be detected. These patients are transferred to the angiography suite immediately thereafter and angiography with embolization performed subsequently. The procedure can be started within 1 h after admission [59]. Some authors favor using "antishock" iliosacral screws as a resuscitation tool [60–62]. Prerequisite is a thorough knowledge of sacral anatomy and experience with iliosacral screw osteosynthesis in a nonemergency setting (Fig. 23.1a–e). Due to the emergency environment, the chance of screw malpositioning is higher; a replacement of the screw(s) during second-look surgery may be necessary [61].







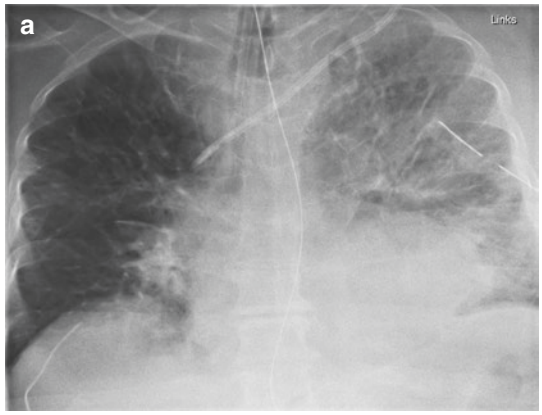




Skeletal injuries of the lower extremity also ask for urgent stabilization. Proximal fractures near to the trunk have higher priority than distal lesions. Proximal femur fractures and femoral shaft fractures are connected with high instability, important blood loss, heavy pain, and difficult nursing. Depending on the localization in the proximal femur (intracapsular, intertrochanteric, subtrochanteric), hip arthroplasty or hip preserving surgery will be performed. The surgery should be done as soon as possible and be the definitive solution (Fig. 23.2a–i). Also femoral shaft fractures need urgent stabilization. The type of treatment should be less invasive and operation time short. External fixation is the first choice; minimal invasive plate osteosynthesis (MIPO) and intramedullary nailing are less attractive alternatives. The choice of primary treatment must depend on the physiologic condition of the patient before surgery. Borderline patients profit more from external fixation than from nailing as primary procedure [63]. Dislocations and fracture dislocations at the knee joint as well as fractures of the lower leg can easily be stabilized with a (joint-bridging) external fixator. The procedure creates stability and allows soft tissues to recover from trauma. Open fractures, which are most frequent at the lower leg, should be treated in emergency with soft tissue debridement, open wound care, or vacuum-assisted closure if appropriate and external fixation. External fixation will not be the definitive treatment. Mostly, the fixator will be removed and intramedullary nailing for shaft fractures, open reduction, and internal fixation for (juxta) articular fractures performed.

At the upper extremity, operative stabilization is rarely needed in emergency. Only open fractures, contaminated wounds, and fracture dislocations need surgical revision resp. reduction and stabilization. Reduction of septic load, prevention of secondary damage, and optimization of nursing at the intensive care unit are the main

Fig. 23.1 (**a**–**e**) This 84-year-old female pedestrian is hit by a car. She suffered a severe polytrauma but could be rescued very early after accident. At the site of accident, her initial vital signs were the following: (A) Unprotected and obstructed airway. (B) Insufficient breathing efforts with no SpO_2 reading. (C) Pulse 40/min, RR_{sys} 50 mm Hg. (D) GCS 3, pupils dilated with little response to light. (E) Multiple contusions. The pre-hospital treatment of the emergency physician included intubation, volume therapy, vasopressors, and mechanical resuscitation. The patient arrived in the emergency room in severe shock under continued medical and intermittent mechanical resuscitation. Initial base excess was −21.3 mmol/l, hemoglobin 2.3 g/dl, and core body temperature 33.6 °C. After initial radiographic examination, the patient was diagnosed with the following injuries: bilateral unstable thorax (AIS 5) with bilateral hemopneumothorax (AIS 4) (**a**). Fracture of the left clavicle and scapula (AIS 2). Splenic lesion, grade 2 (AIS 2). Fractures of the transverse process of the 1st, 3rd, and 5th lumbar vertebra (AIS 2). Vertically unstable AO-type C3 pelvic ring injury (right-sided sacral transforaminal fracture, left-sided iliosacral dislocation, pubic symphysis rupture, and left-sided pubic bone fracture) (AIS 5) (**b**, **c**). Ankle fracture, type Weber B (AIS 2). The calculated ISS was 54, NISS 66, TASH 22 points (77% risk for mass transfusion), and a RISC II prognostic survival score of 0.5%. Emergency surgical procedure included insertion of chest tubes left and right (pre-CT). Application of a supra-acetabular external fixator, packing of the small pelvis, plate osteosynthesis of pubic symphysis, and anterior pelvic ring injury and bilateral "antishock" iliosacral screws (**d**). During the ER and primary surgical phase, the patient received 18 PRBC, 12 FFPs, and 4 TC. The patient survived the primary operation phase and was admitted in the intensive care unit for further hemodynamic situation and improvement of physiologic situation. Secondary surgical treatment included depacking of the small pelvis and osteosynthesis of the clavicle on day 2. Revision of the left-sided iliosacral screws on day 20 (**e**). The patient was discharged after 22 days to an intensive care nursing facility. However, her Glasgow Outcome Scale (GOS) at discharge was 2 points, equivalent to a vegetative state. (**a**) Axial CT cut through the upper thorax showing bilateral lung contusions and left-sided chest tube. (**b**) 3D reconstruction of the pelvic ring calculated from the total-body CT taken during the resuscitation phase. It shows a disruption of the pubic symphysis with severe displacement and a left-sided superior and inferior pubic rami fracture. There is a widening of the left iliosacral joint. (**c**) Axial CT cut through the level of S1, showing a sacral transforaminal fracture on the right and an iliosacral dislocation on the left. (**d**) Scout of total-body CT taken after the primary operative procedure. It shows an anterior pelvic external fixator, a symphyseal plate and two iliosacral screws in S1 bilaterally. (**e**) Pelvic a.p. overview taken after the second operative procedure shows the new position of the left-sided iliosacral screws



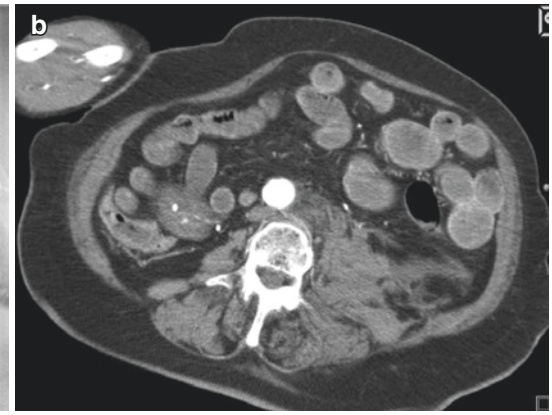


Fig. 23.2 (**a**–**i**) This 79-year-old female suffered a fall from 5 to 6 m height. Initial assessment at the site of accident gave the following data: (A) Unprotected airway. (B) Respiratory rate 10/min, insufficient oxygenation, SpO2 90%. (C) Pulse 50/min, RRsys 152 mm Hg. (D) GCS 5, pupils symmetric with no response to light. (E) Multiple contusions. Prehospital treatment of the emergency physician included intubation, volume therapy of 1,000 ml crystalloid and 500 ml of colloids, and intermittent use of vasopressors. On arrival to the emergency room, the patient was in severe shock with a base excess of −19.5 mmol/l, hemoglobin of 7.2 g/dl, and a core body temperature of 34.6 °C. Initial radiographic examination revealed the following injuries: serial rib fractures (AIS 3) with pneumothorax (AIS 2) and diaphragmatic rupture (AIS 4) (**a**). Fracture of the 1st lumbar vertebra AO-spine type B1 (AIS 3). Multiple mesenteric vascular lesions of the distal ileum (AIS 4) (**b**). Vertically unstable. AO-type C1 pelvic ring injury (AIS 4). Left-sided transforaminal sacral fracture and bilateral pubic rami fractures (**c**). Intertrochanteric femoral fracture with long lesser trochanter fragment (AIS 3) (**d**). The calculated ISS was 48, NISS 48, TASH 14 points (23% risk), and a RISC II prognostic survival score of 7.6%. Emergency interventional and surgical procedures included chest tube insertion on the left side, closed reduction and left-sided insertion of two "antishock" iliosacral screws in S1, and closed reduction and bilateral insertion of retrograde transpubic screws. Laparotomy, removal of abdominal hematoma, reduction of intestinal content out of the left thorax, closure of diaphragmatic rupture, and ligation of the mesenteric vascular lesions of the distal ileum. Resection of ilium was not necessary. Fasciotomy of the left lower leg due for compartment syndrome. During the ER and primary surgical phase, the patient received 26 PRBC, 16 FFPs, and 4 TC. After these emergency procedures, the patient remained hemodynamically unstable despite ongoing resuscitation. It was decided to perform an angiography and, in case of an active arterial bleeding in the small pelvis, to embolize the bleeding artery. Angiography showed an active bleeding of a left vesical artery. A selective embolization with histoacryl was performed (**e**–**f**). The patient thereafter became hemodynamically stable and was transported to the intensive care unit for further stabilization. Secondary surgical procedures included osteosynthesis of the subtrochanteric femoral fracture with a dynamic hip screw, cerclage, and long trochanteric plate (day 3) (**g**–**i**). Debridement, VacuSeal application, secondary closure with skin graft of the lower leg (multiple surgeries between day 8 and 31). The patient survived the accident and was discharged after 54 days in hospital with a Glasgow Outcome Scale (GOS) of 5 (good recovery). (**a**) A.p. overview of the thorax after chest tube insertion on the left. It shows a severe left-sided lung contusion and an intrathoracic shadow above the left diaphragm, which is compatible with a left-sided diaphragmatic rupture and intrathoracic localization of intestines. (**b**) Axial CT cut through the abdomen showing intraperitoneal free fluid. (**c**) Axial CT cut through the level of S1, showing a sacral transforaminal fracture on the left with severe displacement. (**d**) Coronal CT reconstruction of the pelvis and lower extremities showing the unstable intertrochanteric fracture on the left with a long lesser trochanter fragment. (**e**) Intraoperative view of angiography showing an active bleeding of a left vesical artery. (**f**) Intraoperative view of angiography after selective embolization shows no arterial bleeding anymore. (**g**) Pelvic a.p. overview taken after the secondary operative procedure shows two iliosacral screws in S1 on the left side and bilateral retrograde transpubic screws. (**h**) A.p. overview of the left proximal femur after osteosynthesis of the intertrochanteric fracture with a dynamic hip screw (DHS) with long femoral plate, cerclage wires, and a buttress plate. (**i**) Lateral view of the proximal femur after osteosynthesis with DHS, cerclage wires, and buttress plate

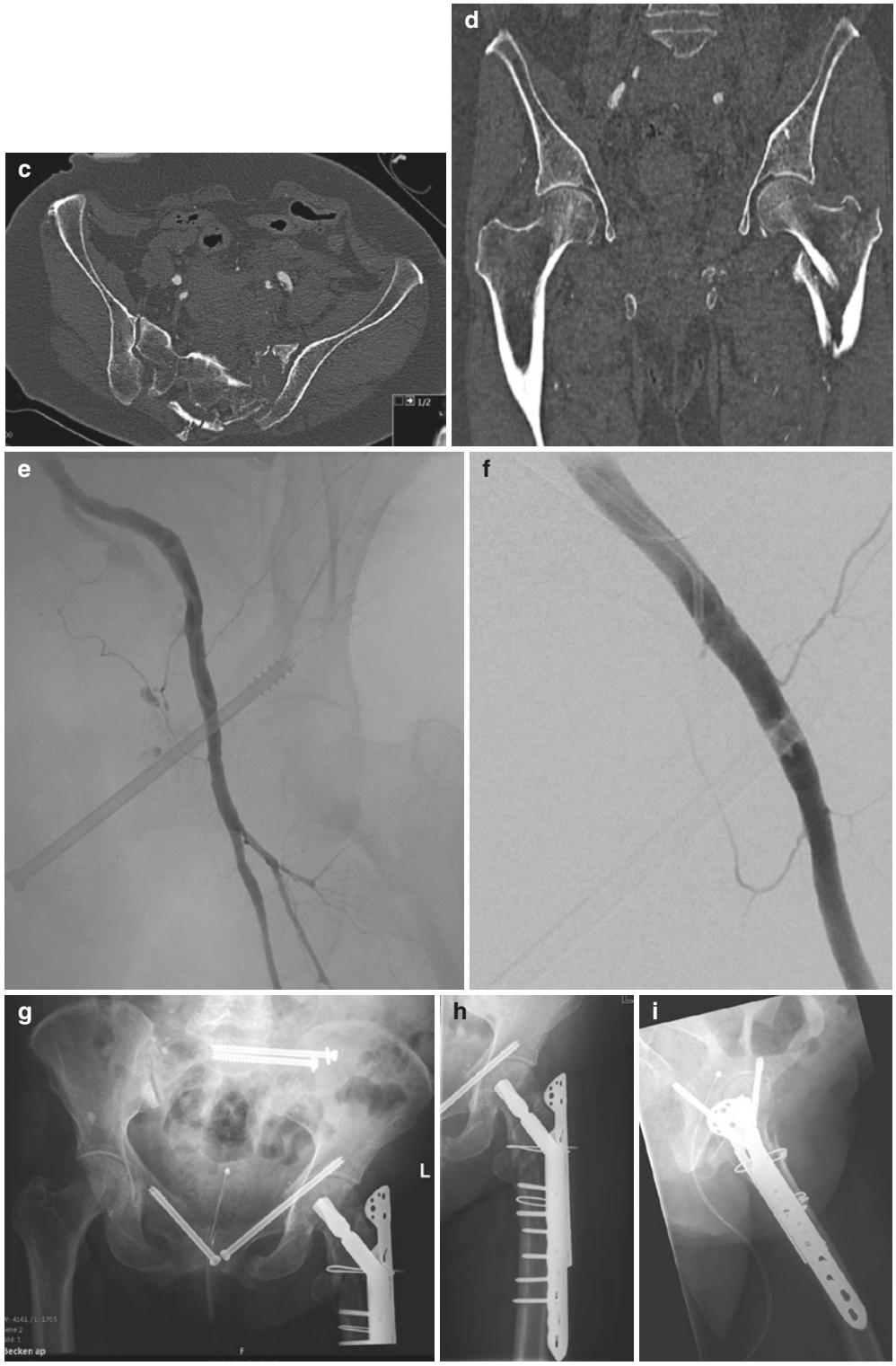


Fig. 23.2 (continued)

reasons for early surgery. Noncomplicated fractures (e.g., proximal humerus, distal radius) can be treated secondarily during later operative phases. Splints or plaster casts are used before surgery. Careful padding of the soft tissue envelope prevents pressure sores or neurovascular damage.

Injuries of the spine are not subject of emergency and damage control operations. The only exception is a partial and progressive paresis due to a posttraumatic instability of the vertebral column. But even then, life has a higher priority than paresis. Long-lasting spine surgery should not be done in a borderline patient. Due to low bone mineral density, it may be difficult to discover posttraumatic anomalies in the bony structures of the spine. It is recommended to perform a total body CT scan, which enables an analysis of the whole vertebral column, to discover such lesions [64]. An MRI gives specific information on damage of the ligaments and degree of stenosis at the spinal canal. The last examination is not available in the emergency setting. It should only be performed at a later stage for answering specific questions such as degree of disco-ligamentous instability, myelin contusion, and intraspinal hemorrhage. Operative fixation is advantageous in patients with an unstable spine. Nonoperative treatment was associated with a higher mortality in a retrospective study on 154 severely injured patients with a mean age of 76 years [24]. Timing of operation is depending on the physiologic condition of the patient. It must be possible to place the patient for several hours in the prone position on the operation table [65].

23.4.4 Secondary Operative Phases

After primary surgery, which is kept as less aggressive and less time-consuming as possible, the patient is sent to the intensive care unit, where his/her physiologic condition will be optimized. Second-look surgery and reconstructive procedures must be scheduled in intense cooperation with the intensivist. Timing of further surgical procedures will follow the same recommendations as for primary surgery: do not harm [65, 66]. This means that necessary surgical interventions will better be divided into consecutive operative phases instead of one longer procedure. Between these interventions, the patient will return to the intensive care unit and be prepared for further surgery. The sequence of interventions will be guided by the urgency of the procedures. Second-look procedures after damage control laparotomy or pelvic packing have the highest priority together with soft tissue debridement of open and contaminated wounds. Exchange of external fixation to intramedullary nailing of shaft fractures of the femur or tibia has lower priority, but needs to be done at an early stage. Early nailing after external fixation can be done as a one-step procedure while avoiding deep (intramedullary) infections due to pin track infections [67].

Operative stabilization of injuries of the spine or posterior pelvic ring needs thorough preoperative planning. The patient must be in such a good condition that he/she endures a surgical procedure of several hours with a moderate amount of blood loss and being in the prone position [68]. The patients benefit when the surgery is done as early as possible [69].

Open reduction and internal fixation of (juxta) articular fractures and of fractures of the upper extremity have a lower priority than the above-mentioned procedures. Nevertheless, these operations should be done within 3 weeks after trauma at the latest. Due to scar tissue formation, mobilization of fracture fragments becomes more difficult, and exact reduction is less easily achieved [70].

Conclusion

Due to their limited physiologic reserves, morbidity and mortality of elderly patients after high-energy trauma are higher than in younger adults. Less severe trauma can lead to more and more severe complications than in younger patients. It is of great importance to recognize the threat trauma has on the elderly person, making a correct initial triage essential. Specific triage criteria must be applied for the elderly trauma patient.

Elderly persons suffering several injuries should be sent to a trauma center immediately. All elements of management must be guided by the recommendations "save life before limb" and "do not harm." The importance of tight and continuous communication with the intensivist cannot be underlined enough. Principles of damage control are also valid for the older patient. Resuscitation must be aggressive, and early invasive monitoring is necessary to avoid the lethal triad of hypothermia, acidosis, and coagulopathy. Primary surgical procedures are merely done to stop bleeding, control contamination, and fix major instabilities. Also secondary procedures should be kept short; required interventions should be done consecutively with a period of intensive care between them. The surgeries should be scheduled as early as the physiologic condition of the patient allows further operative treatment, ideally within 3 weeks after trauma. Although the majority of elderly patients survive damage control measures, outcome of management shows higher mortality levels. The surviving patients experience in-hospital morbidity measures, which are comparable to younger patients. This data provides support for damage control procedures in severely injured elderly patients [12, 71].

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Damage Control in Vascular Injury

24

L.P.H. Leenen

The highest goal in damage control surgery is to stop the bleeding. Major injuries to the vessels therefore pose the major challenge in the damage control approach. Vascular injuries of the torso are an immediate threat to the life of the victim, whereas vascular injuries to the extremity threat the preservation of the limb.

Early adequate diagnostics by CT and/or angio CT even in the hemorrhagic-threatened patient that seems to be feasible in treatment early on is possible in the same location, not losing much time by transportation between locations within the hospital. This has major implications for the patient with vascular, profound bleeding patients.

Early pinpointing of the vascular injury and readily treatment with catheter-guided embolization and balloon control of the lesion will further expedite control and treatment of these injuries and opens a new era in damage control of vascular injuries.

Of course adequate selection of these patients is of the essence.

Over the past years, major changes have taken place in the management of vascular injuries.

The invention of hybrid rooms, where both operative and catheter-guided interventions can be performed, and the invention of resuscitative endovascular balloon occlusion of the aorta (REBOA) have revolutionized the treatment of the patient with vascular injuries.

The hemodynamically unstable patient is nowadays preferably received in a hybrid operating room with diagnostic CT capabilities. However still not the standard, the current knowledge calls for such a facility to optimize quick and precise diagnostics by whole-body CT and immediate possibilities for catheter-guided treatment of compelling bleeding.

However diagnostic and treatment protocols are to be adjusted as they are considerably different from the standard ATLS protocols and trauma team approach [1]. Only if the circumstances are accommodated to the infrastructure we can enter the new paradigm shift.

24.1 REBOA

For many years uncontrollable hemorrhage in the abdomen and the pelvis was thought to be best treated with left anterolateral thoracotomy and cross clamping the thoracic aorta and subsequently increasing blood flow to the brain and heart. Nevertheless in this heroic procedure, the results remained dismal. Already in the 1950s, balloon occlusion of the aorta was performed [2]. Only in the recent years when endovascular treatment of aortic aneurysms was popularized and a

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wide variety of vascular diseases were treated endoluminally, new interest was pointed to the use of large balloon catheters to control noncompressible hemorrhage. Already in non-heartbeating donor procedures, catheters have been used to occlude the aorta above and below the renal arteries to perfuse the kidneys for preservation with a total blockage of the aorta, even without the need of fluoroscopic control.

After a series of preclinical experiments [3, 4], it was noted that this procedure had physiologic benefits for abdominal and pelvic hemorrhage and shock. Thereafter it was used increasingly in humans [5, 6].

The REBOA was further revolutionized by a group of physicians in several trauma centers throughout the United States and in Japan. Recent case reports and multi-institutional trials have demonstrated safe and effective control of hemorrhage using REBOA in patients with life-threatening hemorrhage below the diaphragm [7, 8].

24.1.1 Indications

Indications and contraindications for REBOA are indicated in Fig. 24.1. In short any of the indications for an emergency room thoracotomy are also an indication for this procedure, added with severe pelvic trauma with severe hemorrhage. A protocol is depicted in Fig. 24.1.

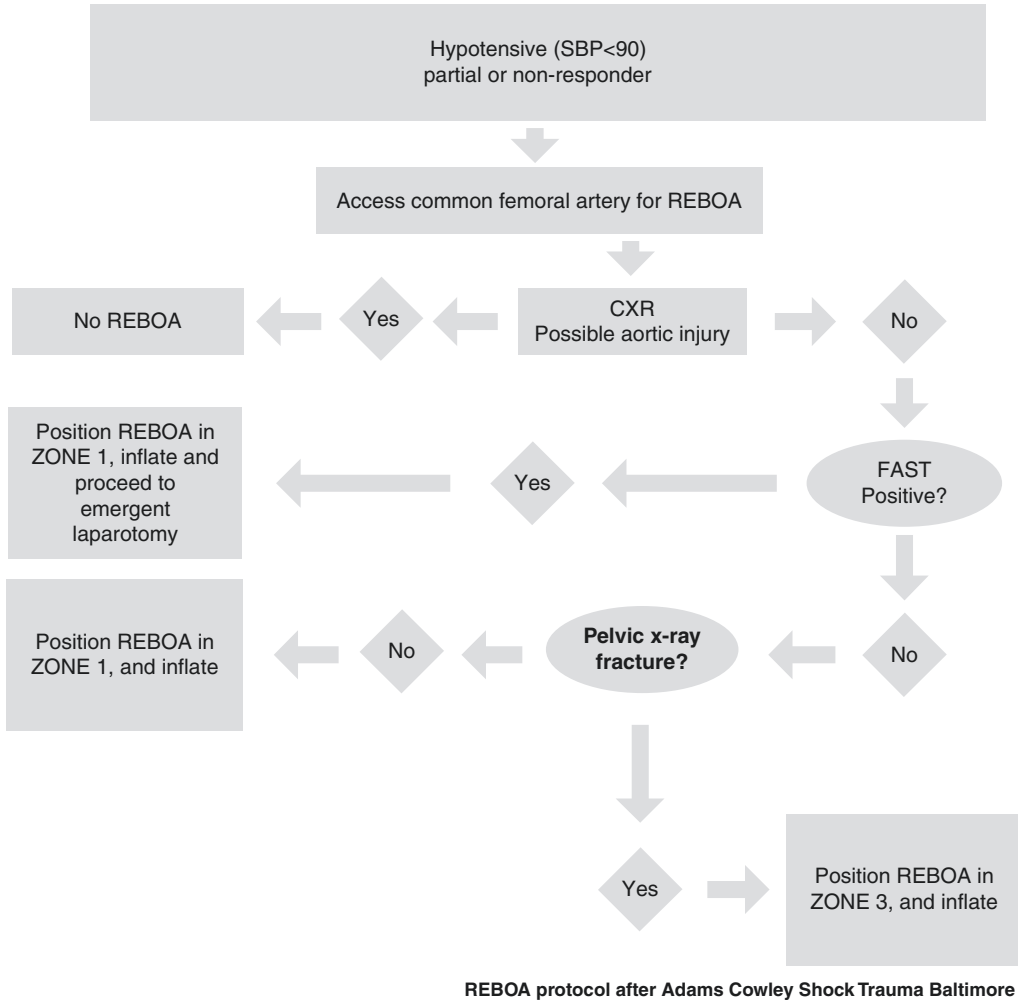


Fig. 24.1 REBOA protocol. Modified after Shock Trauma Baltimore

24.1.2 Technique

The technique can be performed safely in the resuscitation suite using X-ray or in the operating room using fluoroscopy. Recently a non-fluoroscopic technique was introduced [9].

The technique is among others nicely outlined in the article of Stannard et al. [10]. It consists of five steps: arterial access, balloon selection and positioning, balloon inflation, balloon deflation, and sheet removal.

The procedure usually is performed as a Seldinger technique. The femoral artery is approached percutaneously however preferably by cut down, and a 12 French catheter is advanced over the wire through the artery into the aorta. Some authors prefer to use ultrasound to localize the artery in the severely hypotensive patients, while others rely on the rather invariable anatomic landmarks.

Typically there are two zones of interest (Fig. 24.1), zone 1 where the balloon is placed just above the diaphragm, to control both the abdominal viscera and the pelvis, or in zone 3 just above the bifurcation to control the pelvis and the lower extremities.

Consequently injuries can be addressed, whereafter the balloon can be withdrawn, to avoid further metabolic problems and/or severe repercussion injury and a severe systemic inflammatory response.

24.1.3 Complications

As with any invasive procedure, complications can occur. In the first place, the catheter is a fairly large-bore catheter, with the usual problems of discrepancy between vessel diameter and the catheter. Advancement in older people with tortuous iliac vessels can be a problem and in the hectic circumstances in the ER could cause perforation of the vessel. However many of these problems have been noted in the endovascular procedures currently in vogue in vascular surgery.

As noted in the paragraph above, if the balloon remains too long in place, severe repercussion problems can occur, leading to severe inflammatory sequelae.

24.1.4 Courses

Currently REBOA is coming to adulthood. There are currently several published training courses like the Basic Endovascular Skills for Trauma (BEST™) and Endovascular Skills for Trauma and Resuscitative Surgery (ESTARS™) designed to familiarize physicians with the basic endovascular techniques required to perform the REBOA.

24.2 Hybrid Rooms

The advent of the hybrid operating room coupled with the benefits of endovascular techniques in the setting of trauma will likely result in an increasing number of patients being diagnosed and treated with catheter-based interventions.

The combination of both catheter-guided and damage control operative care makes it possible to use both techniques in the same patient without the need for dangerous transport of a hemodynamically jeopardized patient. In the case of a damage control procedure, catheters can be used intraoperatively for additional diagnostics, e.g., in a patient after packing of the liver and ongoing intraparenchymal arterial bleeding, followed by catheter-guided therapy with balloon occlusion or embolization.

Over the years an increasing indication was noted for intra-arterial interventions already for over a decade as documented, e.g., by Pryer and co-workers [11]. A combination of open operative intervention and intra-arterial adjuncts like stenting and embolization is more and more found to be indicated trying to combine less invasive and more targeted treatment of visceral bleeding [12] as well as pelvic bleeding.

Different authors have presented combined solutions where optimized combined care can be delivered [13].

The ultimate trauma diagnostics, resuscitation, and treatment room have been designed and realized in Switzerland [14], where CT, angio, and operating facilities have been realized (Fig. 24.3).

24.2.1 General Operative Techniques

In general in damage control, simple techniques have to be employed in order to gain control as

quick as possible. Time-consuming complex repairs are most times not indicated and result in the loss of the patient and most times on the operating table.

Draping in case of suspicion of major vessel injury should be from sternal notch to the knees, as it might be possible to regain control of in- and outflow in adjacent compartments.

In damage control situations, only a limited number of preoperative measures and diagnostic procedures are possible. In modern trauma care, airway, breathing, and circulatory management should be simultaneous.

24.2.2 Temporary Occlusion

In the prehospital or preoperative situation with extensive external blood loss, temporary occlusion with simple manual or digital pressure provides a simple effective measure for reduce further blood loss. Alternatively a tourniquet in extremity injury can be applied. After being banned from clinical practice, because of the danger of venous congestion and the imminent danger of further damage by injudicious application, it is back as a result of the Iraqi conflict where it found renewed interest [15–17].

Another adjunct to temporary tamponade is the extraluminal balloon tamponade, which can be utilized in a wide variety of situations and anatomic localizations. A Foley catheter is placed through the trajectory of the injury and the balloon is inflated. Slight traction may bolster the effect. If the opening in the skin is too wide, it can be sewn together to minimize the opening.

Intraoperatively bleeding can be stopped or diminished by manual compression or swabs on the in- and outflow trajectory. In low-pressure systems, e.g., veins, which are easily damaged by clamping or attempts to dissect this provides a quick and effective approach to bleeding control (Fig. 24.2). For immediate control of abdominal aorta, an aortic occluder (Fig. 24.3) can be used (see section on abdominal vessel bleeding), which is placed in the diaphragmatic aperture.

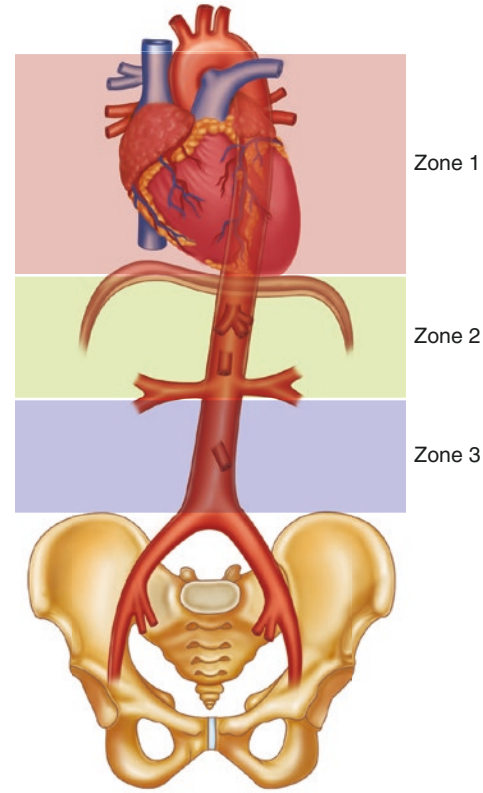


Fig. 24.2 Aortic zones: *Zone 1* from subclavian arteries to the diaphragm, *Zone 2* visceral arteries, and *Zone 3* (from renal arteries to iliac bifurcation)

Intraluminal balloon occlusion can be used intraoperatively if the vascular structure can be readily identified. Also in- and outflow control can be obtained with rubber tourniquets, without further damaging vulnerable vascular structures (Fig. 24.4).

24.3 Flow Restoration

24.3.1 Shunts

In recent years intraluminal shunts have been used more and more as a temporary vascular conduit for almost any anatomic location. Basic research shows that (Fig. 24.5) even under low-pressure circumstances, the shunt remains patent for a considerable amount of time [18]. Recent experiences in Iraq showed a

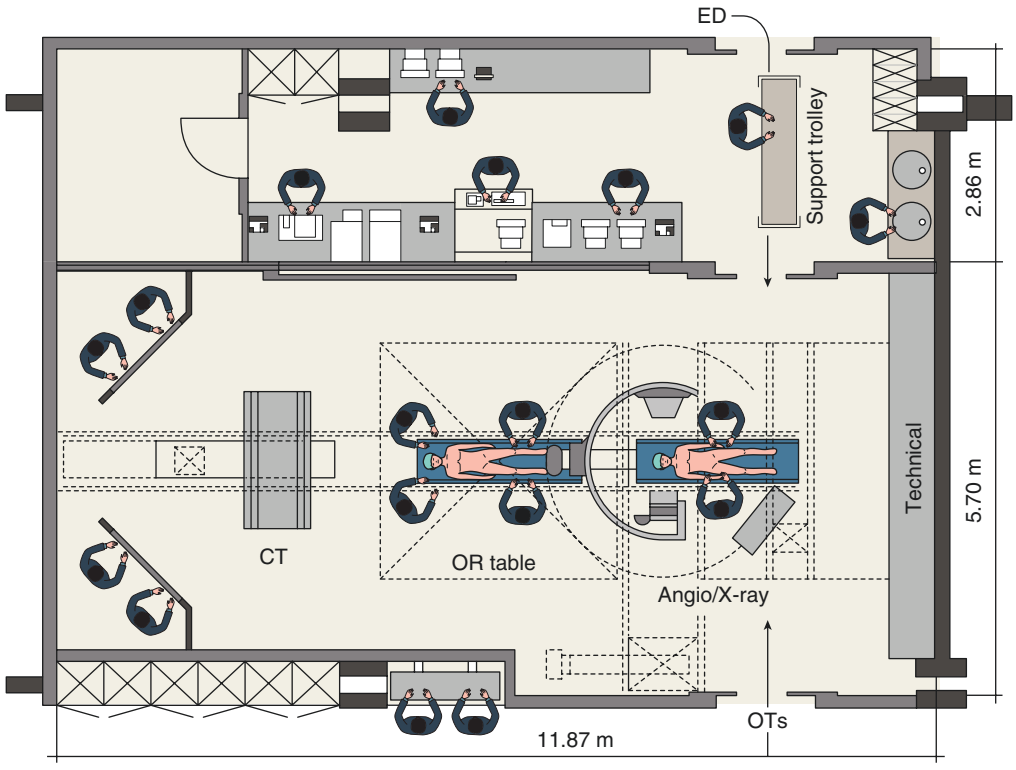


Fig. 24.3 Combined CT, angio, and OR facilities in one room (From Gross et al., Br J Surg 2010)

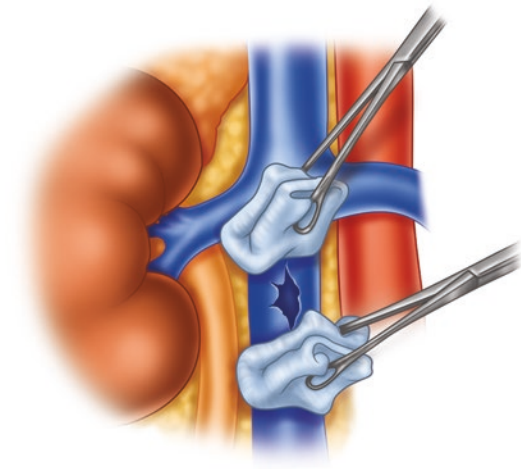


Fig. 24.4 Swab sticks (Carrico, Thal, Weigelt)

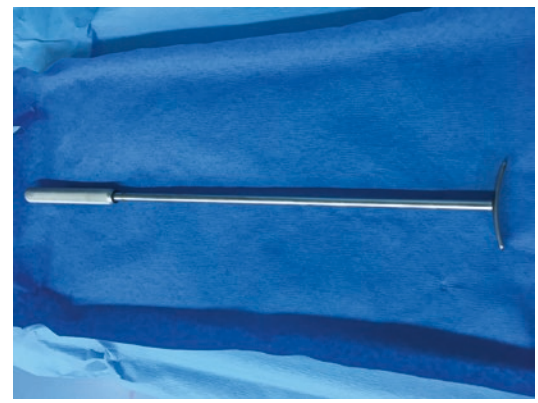
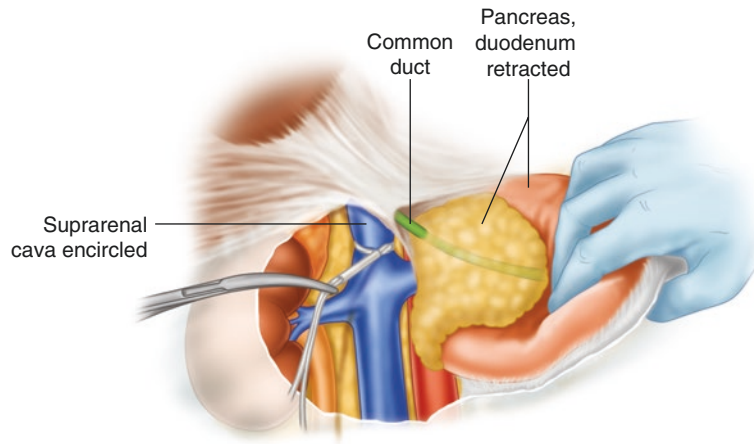


Fig. 24.5 Aortic occluder (Stamper)

huge success for introduction of shunts even in the field. In a series of 54 shunts placed in the field and 43 in the proximal limb, 37 (85%) remained patent until arrival in the definitive care area [19]. Even shunts placed in the venous system remained patent [20]. Commercially available shunts, used for, e.g., carotid surgery (Fig. 24.6), can be utilized; however they also can be constructed from simple iv line or endotracheal suctioning tubing. The shunt is secured from dislodging with a simple tie of any kind, but also sophisticated clamps can be used when available.

Shunts can be left in place for a considerable amount of time without major drawbacks. The use of anticoagulants is not advised as most patients are coagulopatic anyhow and have other, potentially bleeding, injuries. The patency of the shunts depends on the physiological situation, local circumstances, and time of the distal ischemia. Shunts give the opportunity of quick revascularization of the organ or limb, minimizing the acidotic load to the patient and minimizing the reperfusion reaction. The skin can be closed over the shunt (Fig. 24.7) temporarily, whereafter other injuries can be addressed and/or the patient can be further resuscitated in the ICU. Repair of the vessel can be attempted when the patient is in a more favorable condition and an optimal plan for repair with the optimal operation team can be worked out. In case of a concomitant orthopedic injury, vascular repair can ensue the repair of the fracture [21] (Fig. 24.7).

Fig. 24.6 Tourniquets, vessel loop with tubing



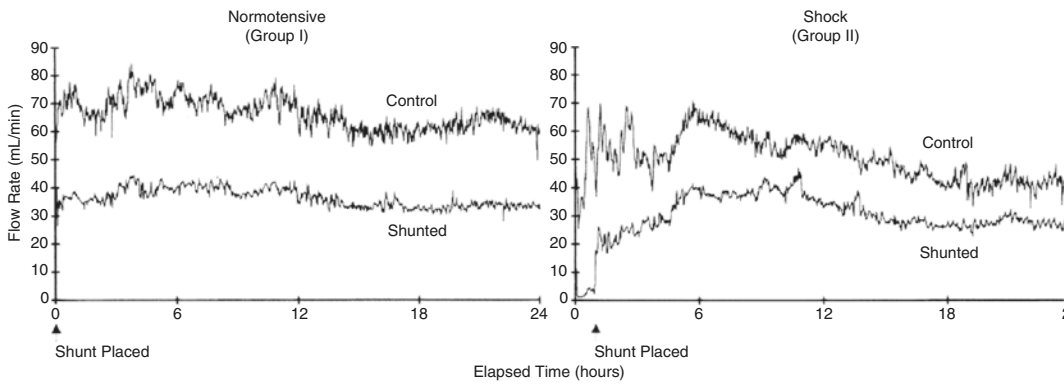


Fig. 24.7 Animal lab shunts illustration article

24.3.2 Lateral Repair

Simple lateral repair, in suitable cases, is preferable as a quick and effective measure. Lesions of larger truncal or extremity vessels can benefit from this technique. The lesion has to be clean and no devitalization of the vessel wall should be present. Also frayed ends or complete transaction are a contraindication for lateral repair. Major disadvantage is the high chance to create a stenosis, even with larger vessels like the aorta or vena cava. If possible the repair should be transverse, even in the case of a length tear. Revision of the repair at a second instance is advised and where needed revision before thrombosis is apparent.

24.3.3 Stents

With the increased availability of intraluminal stents, the use in selected cases has picked up. For numerous indications stents can be used, with the major advantage of reducing the operative trauma. Of course in the case of bleeding, covered stents should be used. Main indication for stenting is the thoracic aorta, but also in other regions, it has become the method of choice for vascular repair. Upper thoracic aperture vessels and axillary but also iliac vessel injuries can be treated in this way in a damage control fashion. There is considerable debate whether these stent procedures should be regarded as bridge procedures, with removal of the stent and direct repair in a later phase. As most patients are young and the natural course of these stents is still unknown, there is a tendency to the latter.

24.3.4 Complex Repairs and Grafts

In the context of damage control, surgery complex repairs and the use of extended repair and grafts are a bad choice. The lengthy operations needed are ill advised in a cold coagulopathic patient, whereas the quality of the repair in many cases is not optimal because of the time pressure the surgeon is confronted with.

24.3.5 Definitive Occlusion

24.3.5.1 Ligation

The most simple method for regaining bleeding control is clamping and ligation of the bleeder. In a large range of bleeding problems, this remains a very attractive measure; however every named artery has its own rules, whether a simple tie will be tolerated. Care has to be taken in the procedure of clamping. Wild undirected clamping in a pool of blood results in more damage and vulnerable structures like veins are the first to be severed. Moreover the venous structures are most difficult to repair. Controlling inflow and outflow at some distance in an untouched area can be of great help to gain overview. For this a vessel loop passed twice around a vessel and held in place with a clip, clamp, or tubing (Fig. 24.4) can be used.

24.3.5.2 Coiling

A modern way of occlusion of the bleeding vessel is coiling through the intravascular route. Although a hemodynamic unstable patient in the angio suite is a bad combination, the disadvantages of additional operative trauma may lead to this approach. Moreover in a modern combined operation-angio suite, which should be state of the art in a level 1 trauma center, the best of these two worlds can be combined and a versatile approach to vascular trauma can be utilized. Of course the nature of the bleeding must be arterial.

24.3.5.3 Hemostatic Agents and Glues

In the case of severe bleeding and devastating wounds, sorting out the exact bleeding focus can be very demanding. As these patients are mostly already coagulopathic, everything bleeds and discriminating between the structures is mostly not possible. Mainly for the use outside the hospital, hemostatic agents have been developed, which stops the bleeding immediately. These mineral hemostatic agents mainly draw water from the surroundings in an exothermic process developing temperatures up to 55 °C. In a comparative analysis in an animal model of lethal groin injury, the efficacy of zeolite was compared to classic dressings and other commercially available

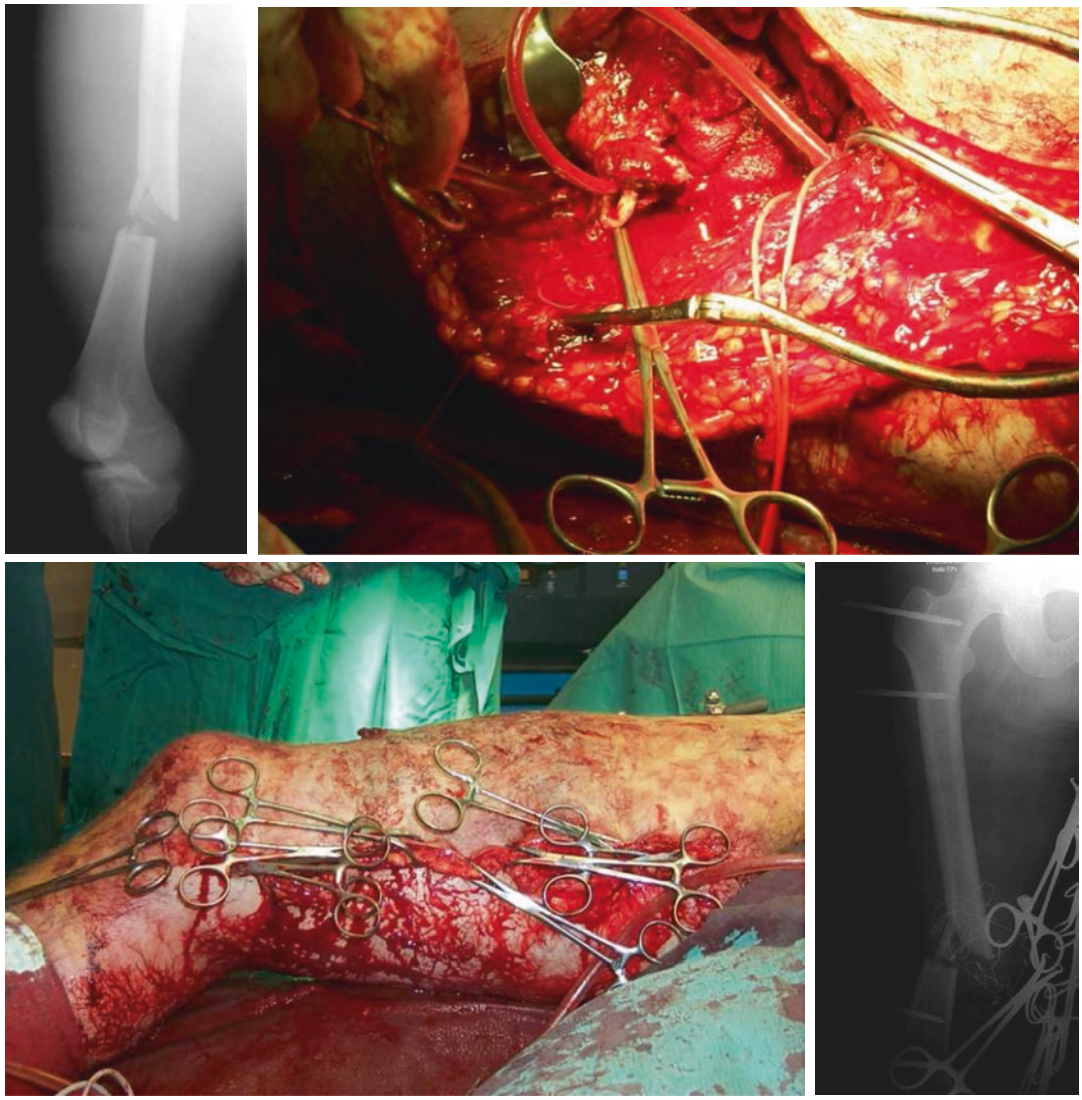


Fig. 24.8 Shunt

hemostatic agents. The results were astonishing where zeolite reduced blood loss 4–180 min after application to 10 ml/kg body weight and no deaths however at the cost of high exothermic reaction with temperatures up to 55 °C [22] (Fig. 24.8). This could be attenuated by modification of the zeolite hemostatic dressing [23].

Another adjunct to damage control in vascular lesions is the use of fibrin sealant. Kheirabadi et al. [24] evaluated the use of fibrin sealant dressing in a high-pressure vascular lesion animal model and concluded that fibrin sealant can seal an arterial bleeding and prevent rebleeding for at least 7 days. It therefore can be used as a bridging procedure for subsequent stenting or open repair procedures.

24.3.5.4 Amputation

A very definitive way of dealing with a major bleeding problem can be amputation. In the case of a mangled extremity, with multiple injuries and severe hemorrhage, it is wise to go for an amputation, in order to save the patient. This team decision should be made early and expeditiously to gain time and prevent needless blood loss and additional shedding of waste products into the circulation.

The preferred technique is a guillotine amputation, with compressive dressing afterward, to prevent a lengthy procedure of modeling and flap creation. The guillotine amputation offers also the opportunity to have a second look and to judge whether the remaining tissues are viable and suitable for the creation of an adequate amputation stump.

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Principles for Damage Control in Military Casualties

25

John B. Holcomb and Thomas A. Mitchell

25.1 Introduction

Conceptually, damage control principles are rooted in military origins; the surgical application is analogous to US Navy terminology for "the capacity of a ship to absorb damage and maintain mission integrity" [1]. The interval termination of an operation has been described with liver packing as early as 1908; however, the practice was ended soon after high infectious rates were identified [2]. The introduction of a formal laparotomy by World War I improved mortality; however, the essence of damage control surgery was further captured in World War II, as exploratory laparotomy became the standard of care for penetrating abdominal trauma, and surgeons gained vast experience in comprehending the necessity for expedited abdominal exploration hemodynamically unstable patients [2]. Specifically, in the Western desert in 1942, Watts highlighted that a war surgeon "must evacuate the wounded with all possible speed, both to clear the unit and to restore its mobility…that he must wherever possible, avoid the procedures that will prevent the early evacuation of the patient" [2]. This resonated on the battlefield; however, it wasn't until 1983 that Dr. Harlan Stone, a civilian, reinstituted rapid packing and termination of the laparotomy in civilian trauma patients when intraoperative coagulopathy became excessive [2]. Ten years later, Rotondo specifically termed the phrase "damage control surgery" where patients that underwent damage control surgery with two or more visceral injuries and/or had a major vascular injury had a markedly higher survival in a small nonrandomized cohort [77% (10 of 13) vs. 11% (1 of 9), $p < 0.02$]. Currently, more recent publications have demonstrated an improved 30-day survival [73.6% vs. 54.8%; $p = 0.009$] and a decreased mean trauma intensive care unit [11 v 20 days; $p = 0.01$] stay in patients receiving a massive transfusion that underwent damage control laparotomy and damage control resuscitation [3]. Although this methodology to appropriately care for severely injured civilian patients would appear to extrapolate well to an austere environment, the transition to military austere environments was initially questioned by many.

Damage control surgery was first utilized in Somalia in 1993 during the battle of the Black Sea fought by the US Army Rangers in the streets of Mogadishu, Somalia; two patients underwent damage control procedures and subsequently died [4]. Retrospectively, the utilization of damage control methodologies prior to the conflicts of

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Operation Enduring Freedom and Operation Iraqi Freedom was questioned "because the manpower and material resources are often limited during large military mass casualty events, it is questionable whether traditional damage control procedures should be performed," as the author notes that the two patients who underwent damage control laparotomies would likely have been categorized as "expectant" if they would have presented to the combat support hospital later in the fight on October 3, 1993 [4]. This logistical concern was propagated in November 2000 by Eiseman et al. who postulated that the "enormous logistic requirements for such strategies are contrary to the demands of the usual wartime scenario. On the basis of experience in civilian trauma centers and combat casualty management, we question the suggested extensive role of damage control surgery during wartime" [5].

Despite such criticisms, the US military taught and adopted damage control surgery operative and resuscitative principles at their trauma training centers during the conflicts of Operation Iraqi Freedom, Operation New Dawn, and Operation Enduring Freedom in Iraq and Afghanistan. The damage control techniques and logistics have been termed "global combat damage-control surgery" and encompass "multiple separate surgical facilities, multiple surgeons, multiple resuscitation and stabilization episodes, helicopter evacuation, and fixed-wing evacuations by the Critical Care Air Transport Team during a multi-stage global transit" [6]. Specifically, the operative techniques remain similar to the civilian setting to include the surgeon implementing damage control techniques must be cognizant of their surgical resources, personnel, blood supplies, throughput of current conflict, geographical location, and external weather that plays a direct role in patient transport in comparison to most Level I hospitals in the United States that have ample personnel, surgical resources, and blood supplies and do not have to be concerned about transcontinental transfer of their patient. Secondly, the US military adopted a policy of damage control resuscitation recommending a 1:1:1 balanced resuscitation of packed red blood cells/fresh frozen plasma/platelets codified on December 18, 2004 [JTTS CPG: damage control resuscitation at Level IIb/III treatment facilities].

Furthermore, in order to ensure appropriate implementation, evaluation, and scientific inquiry into the evolution of these damage control techniques during these conflicts, the "Joint Trauma System" was born in May 2004 in order to fulfill a mission to ensure "that every soldier, marine, sailor, or airman injured in the battlefield or in the theater of operations has the optimal chance for survival and maximal potential for functional recovery" [7, 8]. The collaboration of Surgeons General of the US Military, the US Central Command, the US Army Institute of Surgical Research, and the American College of Surgeons Committee on Trauma formally implemented the system in November 2004 [8]. Most importantly, this effective utilization and organization of resources has led to a decreased number of soldiers killed from combat wounds, 8.8% compared to 16.5% during Vietnam [8]. Additionally, a large focus of the Joint Theater Trauma System has been to decrease killed-in-action and died-of-wound rates in combat wounded. The Joint Theater Trauma Registry has allowed elucidation of the specific mechanisms/etiologies of these two aforementioned categories such that they may catalyze future research endeavors to improve overall combat casualty prevention and medical intervention.

Specifically, the dominant injury mechanisms during the current conflicts of Operation Enduring Freedom and Operation Iraqi Freedom between October 2001 and June 2011 to include 4,596 battlefield fatalities demonstrated that 73.7% were explosive and 22.1% were gunshot wounds [7]. Notably, 87.3% of all injury mortality occurred prior to arriving at a medical treatment facility with three distinct peaks in mortality [35.2% instantaneous, 52.1% were acute (minutes to hours: pre-MTF), and 12.7% dying of wounds after reaching an MTF) [7]. Most importantly, of the pre-MTF deaths, 24.3% (n = 976) were deemed potentially survivable with hemorrhage (90.9%) and airway compromise (8.0%) accounting for the vast majority of potentially survivable mortality. Specifically, truncal (67.3%), junctional (19.2%), and peripheral

(13.5%) extremity characterized anatomical distribution of potentially survivable mortality [7].

In succession to this study analyzing death on the battlefield, Eastridge et al. also evaluated military patients who "died of wounds" after reaching a military treatment facility. From October 2001 to June 2009, 558 soldiers died of wounds [9]. Notably, 51.4% (n = 287) were potentially survivable to include 80% which were from acute hemorrhage (48% truncal, 31% peripheral extremity, and 21% junctional) [9].

The development of the Joint Theater Trauma System and Joint Theater Trauma Registry and the deployed research teams has enabled the evolution and scientific inquiry regarding each specific aspect of military damage control principles. These concepts will be further elucidated in the upcoming sections for which acute hemorrhagic shock remains at the forefront of future research to mitigate potentially survivable death at each facet of military healthcare which comprises damage control principles.

25.2 Prehospital

The most impactful focus of damage control surgery/resuscitation remains the prehospital phase of care, as previously mentioned, 87.3% of fatalities occurred prior to reach a medical treatment facility of which 24.3% were potentially survivable [7]. The combination of "temperature and weather extremes, severe visual limitations imposed by night operations, logistical and combat related delays in treatment and evacuation, lack of specialized medical care providers and equipment near the scene, and lethal implications of opposing forces" renders prehospital pickup and transportation while simultaneously delivering optimal healthcare challenging [10]. The implementation of civilian Advanced Trauma Life Support into the austere setting revealed certain deficiencies during Iraq and Somalia in the early 1990s [10]. After extensive congressional inquiries into these disparities in care, an article entitled "Combat Casualty in Special Operations" was borne emphasizing three main objectives: (1) treat the patient, (2) prevent additional casualties, and (3) complete the mission [10]. Furthermore, it defined the three modalities of prehospital care – (1) care under fire, (2) tactical field care, and (3) casualty evacuation care – with a major emphasis on preventing death from the following potentially survivable mechanisms: (1) extremity hemorrhage exsanguination, (2) tension pneumothorax, and (3) airway obstruction [10]. These principles were initially captured by the US Special Operations Command to include Army Rangers and Navy SEALS (Sea, Air, and Land Teams) [10]. A study of the 75th Ranger Regiment from October 1, 2001, to March 31, 2010, revealed a 3% potentially survivable death (1 in 32) compared to previous estimations of 24% (232 in 982). Furthermore, no casualties died of airway obstruction or a tension pneumothorax. The illustration of these important data points accelerated TCCC guideline recommendation by the Defense Health Board for implementation by combatant units throughout the DOD [10]. In addition to fundamentally changing the training of military medics/soldiers using TCCC, the prehospital environment provides ample opportunities to implement patient care: (1) tourniquets, (2) hemostatic dressings, (3) hypothermia prevention, (4) airway protection, (5) tension pneumothorax, and (6) fluid resuscitation.

First, tourniquets were sporadically utilized on the battlefield during the initial conflicts in Afghanistan with the exception of the Special Operation Forces in 2001. However, the preliminary evaluation of preventable death on the battlefield from extremity hemorrhage revisited the necessity of tourniquet implementation. Holcomb et al. demonstrated from 2001 to 2004 in the US Special Operations Forces that 15% (n = 12) died from potentially survivable etiologies to include noncompressible hemorrhage (n = 8) and hemorrhage amenable to a tourniquet (n = 3) [11]. USSOCOM was the first to mandate tourniquets for its deploying combatants in March 2005 [12]. By 2006, acceptance of tourniquet utilization had become widespread. Specifically, Eastridge et al. estimated that the universal implementation of tourniquets decreased the death rate from peripheral-extremity hemorrhage from 23.3 deaths per year (pre-2006) to 17.5 deaths per year

(2006–2007). Furthermore, Eastridge et al. demonstrated that 19.2% of potentially survivable death resulted from junctional hemorrhage. Thus, current research through the Medical Research and Materiel Command initiated pursuit of a junctional tourniquet for the inguinal region. There are now multiple junctional tourniquets approved for use on the battlefield and in civilian EMS.

Secondly, hemostatic dressings have undergone an evolution in care throughout Operation Iraqi Freedom and Enduring Freedom. First, dry fibrin sealant dressings were utilized, but were withdrawn secondary to regulatory challenges and expenses [12]. Secondly, the US Army moved to the HemCon bandage which utilizes freeze-dried chitosan working through tissue adhesion of the positively charged chitosan with the negatively charged red blood cells [12]. Wedmore et al. demonstrated a 97% (62/64) success rate in the cessation or improvement in hemostasis after the application of a HemCon bandage from direct verbal reports from combat medics [13]. Concurrently, the US Navy utilized QuikClot, which was shown efficacious against venous and mixed venous-arterial hemorrhage via a mineral-based (zeolite) agent that absorbs water and concentrates blood clotting proteins with cells rendering hemostasis [12]. Unfortunately, the resulting exothermic reaction caused several severe burns. Further research has demonstrated that Combat Gauze is superior and has been adopted in the current 2008 TCCC guidelines [12, 14]. Combat Gauze was identified to be the most efficacious in a femoral arterial injury porcine model resulting in an 80% survival (8/10) compared to other bandage models; Combat gauze, a product of Z-Medica Corporation (Wallingford, CT), is impregnated with contact (intrinsic) pathway activated clotting agent known as kaolin [14]. Based on recommendations from TCCC, HemCon and QuikClot have largely been abandoned by the DoD.

Hypothermia (<35 °C) prevention techniques include wrapping casualties in both a wool blanket and a "space blanket"; however, more recent evidence and innovation demonstrated that Hypothermia Prevention and Management Kit (HPMK) can provide external warmth without a power source for several hours [14]. In 2006, the JTTS published a CPG for hypothermia prevention and was associated with a decrease in the incidence of hypothermia a year after implementation ([15]/include JTTS hypothermia prevention CPG).

Airway protection including tension pneumothorax decompression with a 5 in. needle and/or proficiency with cricothyroidotomy if endotracheal intubation cannot be safely performed are further instructive pieces in the TCCC training to thwart potentially survivable deaths [11, 16].

Prehospital fluid resuscitations per TCCC guidelines specify for the medic to evaluate the radial pulse character and/or mental status (in the absence of traumatic brain injury) to administer 500 ml of Hextend (Hospira, Inc., Lake Forest, Illinois), intravenously if the casualty is in shock [17]. A repeat bolus is to be given if there is a persistence of shock after 30 min of duration. Crystalloid and colloid resuscitations are to be minimized guided by the principles of a "hypotensive" resuscitation (90 mm Hg) to minimize the sequelae of popping the clot, dilutional coagulopathy, and perpetuating further bleeding, especially in noncompressible torso hemorrhage [17, 18].

Finally, the implementation of rapid evacuation teams within the military prior to arrival at a medical treatment facility led to the creation of a physician-led evacuation team entitled Medical Emergency Response Team (MERT) [12]. The team consists of a critical care nurse with emergency medicine background, physician, and a paramedic on a CH-47 Chinook in Afghanistan that is capable of carrying eight stretcher cases or 20 ambulatory casualties [12]. The guiding principle of moving advanced healthcare team members farther forward is to implement further damage control resuscitative and intervention principles earlier in the care of the wounded soldiers. Furthermore, many of the MERT-E (enhanced) have four units of O-positive packed red blood cells and four units of thawed plasma on board to give balanced blood product transfusion instead of colloid/crystalloid earlier in the resuscitations [12]. Finally, the MERT physician has the option for patients with noncompressible

torso hemorrhage to administer the antifibrinolytic TXA (tranexamic acid) [12].

25.3 Hospital

Upon arriving at the medical treatment facility, damage control principles are continued. First, the resuscitation of patients arriving parallels the damage control resuscitation CPG which emphasized two major components: permissive hypotension and 1:1:1 of plasma/platelet/RBC resuscitation [19]. The evaluation of a combat support hospital between November 2003 and September 2005 of 246 patients who received a massive transfusion, defined by a low ratio (1:8), medium ratio (1:2.5), and high ratio (1:1.4) of pRBCs to FFP demonstrated an overall survival of 19%, 34%, and 65%, respectively [19]. Furthermore, mortality rates from hemorrhage were 92.5% (low ratio), 78% (medium ratio), and 37% (high ratio), respectively [19]. In massive transfusion resuscitations in 466 civilian trauma patients between July 2005 and June 2006, the combination of high plasma/pRBC (59.6% vs. 40.4%, $p < 0.01$) and high platelet/pRBC (59.9% vs. 40.1%, $p < 0.01$) ratios improved 30-day survival compared to low plasma/pRBC and platelet/pRBC ratios [20]. Furthermore, the implementation of DCR principles (2006–2011) versus pre-DCR (2002–2006) demonstrated a decrease in the plasma/RBC ratio from 2.6:1 pre-DCR to 1.4:1, while the concomitant mean ISS in the cohort increased from 23 to 27 ($p < 0.05$), TRISS decreased to 0.64 (DCR) from 0.77 (pre-DCR), and an average AIS head (4.3 (DCR) vs. 3.9 (pre-DCR), $p < 0.01$) indicates that patients post-DCR implementation were more severely injured including a greater predominance of traumatic brain injury while minimizing the potentially preventable deaths [21]. Ultimately, the implementation of DCR principles appeared to have improved survival in less injured potentially survivable population that may have succumbed during the pre-DCR era. Additionally, from March 2003 to February 2012, 3,632 soldiers received at least one unit of transfusion, the lowest mortality was identified in the high-FFP to high-PLT group (12%) compared to low FFP/low PLT (16%), high FFP/low PLT(17%), and low FFP/high PLT (16%). Notably, the high FFP/high PLT had the highest ISS of 26. The summation of this presented data gives credence to the military's 1:1:1 resuscitation strategy. Most importantly, in massively transfused patients prior to DCR CPG compared to post-DCR implementation, the mortality rate decreased from 32% to 21% [22].

Additional adjuncts to DCR include TXA (tranexamic acid). In 896 casualties at a NATO Role III in Bastion, Afghanistan, 896 casualties received TXA, and those receiving TXA had a higher survival rate (82.6 vs. 76.1%; $p = 0.028$). Additionally, in the massive transfusion subgroup, the survival rate was 28.1% vs. 14.4%, $p = 0.004$. Finally, TXA was demonstrated to be independently associated with survival odds ratio of 7.28 [23].

Finally, in addition to traditional coagulation studies such as Fibrinogen, pT, apTT, and INR, newer modalities such as thromboelastography and rotational thromboelastometry have been utilized to further guide resuscitative efforts in theater as early as 2004 [24]. Specifically, Doran et al. demonstrated that rotational thromboelastometry compared to traditional coagulation tests (pT/apTT) increased the identification of coagulopathic patients (64% vs. 10%) in 31 patients in a deployed setting [25].

As a direct adjunct to the DCR means of resuscitation, the operative techniques largely are similar to civilian counterparts. However, the one main divergence is the large preponderance of explosion injuries that render multiple components of injury. Explosion injuries induce four different means to cause injury (primary, secondary, tertiary, quaternary). Primary injury is caused by the contact of the blast shockwave on the body such as occasional tympanic membrane rupture, lung injury, and concussion [26]. Secondary injuries are caused by ballistic wounds such as fragments that induce penetrating injuries, traumatic amputations, and laceration [26]. Tertiary injuries are induced from the blast wave propelling soldiers onto surfaces and/or objects causing a blunt injury, crush syndrome, and/or compartment

syndrome [26]. Finally, quaternary injury results from other explosion-related injuries/illnesses such as burns or toxic gases [26]. Therefore, in comparison to standard civilian injuries that can be divergently categorized as blunt or penetrating and treated accordingly, the explosion injury is a complex hybrid that often has components of both penetrating and blunt injurious mechanisms. This mechanism has been dominant on most battlegrounds since World War I and the physics well described.

Interestingly, a primary modality to evaluate penetrating abdominal trauma from World War I to 2004 was to explore all patients; however, the implementation of ultrasound and computed tomography has enabled visualization and/or estimation of fragment/bullet trajectories to aid the surgeon in their decision to explore or to monitor [6]. Beekley et al. demonstrated that 60% of all stable patients with penetrating fragments could undergo selective nonoperative management in the absence of frank peritoneal signs on physical examination and based on CT findings had no intraperitoneal nor retroperitoneal penetration of the fragments [6]. Therefore, the deployed military surgeon may utilize focused abdominal sonography for trauma, physical examination, hemodynamic stability, and computed tomography which are screening tests in an austere environment to assist the decision-making of the surgeon whether or not to explore an abdomen. If exploratory laparotomy is performed, damage control principles of controlling contamination and hemorrhage should be considered especially for those who are acidotic, hypothermic, coagulopathic or undergoing a massive resuscitation. A temporary abdominal closure should be utilized for these patients to allow for further resuscitation in conjunction with a second-look operation to perform definitive management of all identified injuries either at a higher echelon of care (NATO Role III from initial NATO Role II) or a second-look operation at a NATO Role III MTF, or transferring to a higher level of care (NATO Role IV or NATO Role V) with a temporary abdominal closure. Furthermore, damage control modalities can also be extrapolated to the thoracic cavity if major vascular injury at the thoracic outlet, loss of chest wall requiring subsequent debridement and closure, massive air leak, tracheobronchial injury, esophageal injury, and mediastinal are identified [2]. However, the unnecessary overutilization of DCS should be avoided, as inappropriately leaving the abdomen open predisposes patients to subsequent complications (Hatch, Holcomb et al.).

Major vascular injuries can be diagnosed 90% of the time based on history and physical examination evaluating for hard signs of vascular injury to include pulsatile bleeding, expanding hematoma, palpable thrill, audible bruit, and evidence of ischemia [27]. An ankle-brachial index and/or injured extremity index should be performed in conjunction with a physical examination. Arteriography if available may also be useful for diagnostic purposes. Finally, the Mangled Extremity Score may be used as a guide for the surgeon in a deployed setting to guide decision-making whether to amputate or not which includes criteria such as (1) skeletal/soft tissue injury, (2) limb ischemia, (3) shock, and (4) patient age with a combined score of 7 or more that predicts a 100% amputation rate [27]. The utilization of these tools while in theater allows the surgeon to utilize three different generalized modalities: (1) attempted limb salvage with or with vascular repair, (2) damage control with the use of shunts or placement of pneumatic tourniquets, and (3) debridement alone [27]. Furthermore, important principles while deployed include adequate debridement of wounds and/or placing bypasses through non-contaminated fields [27]. Additionally, the utilization of systemic heparinization (50–75 units/kg IV) must be juxtaposed against the totality of injuries the patient has suffered, as this is rarely required in theater (28: Starnes). Saphenous vein as opposed to vascular synthetic grafts should be considered in likely contaminated fields. Finally, shunts such as the Sundt shunt (Integra Lifesciences, Plainsboro, NJ) are recommended if damage control vascular techniques are to be employed in conjunction with the liberal utilization of fasciotomy incisions [27]. Importantly, the implementation of damage control resuscitative strategies has enabled increased

efforts toward limb salvage, as opposed to prior doctrine in Vietnam/beginning of conflicts (2001–2003) which stated that amputation was often necessary in severely injured casualties [28]. Dua et al. document a graft patency of 84.9% in 96 grafts with a follow-up between 29 and 1,079 days with an amputation-free survival of 84% [28]. Overall, 1,221 soldiers underwent amputations sustaining a total of 1,631 amputations from January 2001 through July 30, 2011, with transtibial (683, 41.8%) and transfemoral (564, 34.5%) being the two most common [25]. The overall mean amputation rate was 5.29 per 100,000 deployed troops [25]. Furthermore, this study identified that 30% of all amputees had multiple amputations of the lower extremities, and this was higher than the multiple-amputee rate from World War I, World War II, the Korean War, and Vietnam War [25]. Furthermore, Standsbury et al. identified from October 1, 2001, to June 1, 2006, a major amputation rate of 5.2% for all serious injuries and 7.4% for all major limb injuries compared to an 8.3% amputation rate in Vietnam [29].

Orthopedic damage control principles entail early, rapid, temporary stabilization of a fracture to minimize blood loss subsequently followed by physiological resuscitation and then definitive management [6]. External fixation is the most common means of rapidly stabilizing a pelvis and/or long bone fracture. In conjunction with extremity fractures, soft tissue injuries have prevailed within the current conflicts of Operation Iraqi Freedom and Operation Enduring Freedom secondary to the high-velocity gunshot wounds and explosive blasts that mandate early aggressive operative debridement [30]. A newer modality to dress these wounds compared to conventional wet-to-dry gauze dressings includes the utilization of negative pressure wound therapy (V.A.C. Therapy, KCI Licensing, Inc., San Antonio, TX). For instance, one retrospective study evaluated 218 patients in combat who had 298 separate sites and 1.37 wound sites per patient; Pollak et al. demonstrated that negative pressure wound therapy could be utilized effectively and safely in an austere environment that involves a transcontinental flight with an average transit time of 53 h from the initial time of application in a deployed environment to an operating room at a NATO Level IV (Outside the Continental United States) well within the standard of 72 h device recommendation [30]. Fang et al. demonstrated a similar effectiveness and safety with the negative pressure wound therapy device in 30 patients who underwent successful transcontinental aeromedical evacuation with a negative pressure wound therapy. Finally, the aggressive debridement of these austere wounds emanates from the desire to diminish the superimposition of bacterial and/or fungal superinfections to these wounds which prompted the "Treatment of Suspected Invasive Fungal Infection in War Wounds" Clinical Practice Guideline which was first published on November 1, 2012, which advocates three main principles with war wounds: debridement of infected tissue, minimization of immunosuppression, and utilization of systemic anti-mold medications [JTTS CPG]. Furthermore, this CPG advocates early topical antifungal therapy with 0.0025% Dakin's solution if a patient has three of the following risk factors: dismounted blast injury; above-the-knee amputation; extensive perineal, genitourinary, or rectal injury; and/or super massive transfusion of greater than 25 units packed red blood cells including whole blood. Finally, a histopathological specimen will be taken at NATO Role IV medical treatment facilities of the wounds, and if two operative trips have not improved the wound, the patient will be empirically started on voriconazole and/or liposomal amphotericin B [JTTS CPG].

The major advances in burn care reflect implementation of a Burn Resuscitation Flow Sheet and use of a simplified formulation called the Rule of 10 [6]. The Burn Resuscitation Flow Sheet enables continuity of care throughout the geographical movement, such that every provider will know specifically how much fluid the patient has received in transit to avoid complications such as abdominal compartment syndrome from over-resuscitations. From January 2003 to June 2007, 598 military casualties of which 118 had greater than 30% total body surface area burns [31]. The implementation of a

burn resuscitation guideline in January 2006 resulted in a non-statistically significant decrease in overall abdominal compartment syndrome (5% vs. 16%; $p = 0.06$) and mortality (18% vs. 31%; $p = 0.11$); however, the composite end point of abdominal compartment syndrome and mortality was statistically significant (18% vs. 36%; $p = 0.03$) [31]. Additionally, the Rule of 10 enables for calculation of the initial fluid rate in the resuscitation which is 10 cc/h * % t to avoid arbitrary starting points [6]. Furthermore, the most common topical antimicrobial is silver sulfadiazine and Sulfamylon cream in addition to negative pressure wound therapies that are being utilized more frequently after early excision of burn wounds [6].

25.4 Aeromedical Evacuation to OCONUS/CONUS

The transport of critically ill patients from austere environment (NATO Level II and III) medical treatment facilities to definitive care at NATO Level IV (OCONUS) and V (CONUS) medical treatment facilities is predominantly controlled by the US Air Force [32]. The creation of Critical Care Air Transport Teams (CCATTs) by the US Air Force met the demands of current Operation Enduring Freedom and Operation Iraqi Freedom. The CCATT is comprised of an intensive care physician, intensive care unit nurse, and a respiratory therapist which may take care of up to six critically ill patients (three mechanically ventilated) for evacuation trips that may last from 30 min to 16 h or longer [32]. Ingalls et al. evaluated 2,899 CCATT transport records between September 11, 2001, and December 31, 2010, and evaluated 975 injured soldiers with an overall mortality en route of less than 0.02% and an overall 30-day mortality of 2.1% [32]. More impressively, 93% of all CCATT patients arrived to NATO Level IV (OCONUS) within 72 h of injury and 98.5% arrived to NATO Level IV by the 96 h mark [32]. The rapidity of safe and effective evacuation of military soldiers remains an upmost importance for the implementation of global combat damage control surgery.

25.5 Summary

The preceding sections outline the changes that have occurred in battlefield care from 2001 to 2014. Largely, these changes have been centered on the damage control concept of early aggressive intervention, pushing interventions into the prehospital area, a focus on restoration of normal physiology, multiple operations, and rapid evacuation to a higher level of care. Under the leadership of the Joint Trauma System, these integrated changes have resulted in a low case fatality rate. However, it is incumbent on us to recognize that there are still far too many potentially preventable deaths and avoidable morbidity, and we must work together to improve outcomes on the next battleground.

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26 Penetrating Injuries and Damage Control Surgery: Considerations and Treatment Options

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26.1 Introduction

For most trauma care providers, patients in need of DCS due to penetrating injury are still a rarity. This certainly accounts for the bigger part of Western Europe [1–4]. There are, however, some indications that the number of patients suffering from PI is slightly rising [5–7], if not for the least under victims of terrorist attacks [8, 9]. Another argument to elaborate on this specific trauma mechanism is that these patients are known self-referrals who can present themselves at any emergency department of even the smaller peripheral hospitals with a seemingly minor injury rapidly deteriorating into a resuscitative setting. Hence the authors will present in this chapter general considerations and body region specific treatment options for patients suffering from penetrating injury (PI) and who are in need of DCS.

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26.2 General Considerations with Regard to Penetrating Injuries and Damage Control Management

Trauma mechanisms for penetrating injury (PI) are classically described as being either high- or low-velocity injuries. Stab injuries with knives or sharpened object were recognized as low-velocity PI. As for gunshot wounds a differentiation was proposed based on the muzzle velocity of the projectile. A more sensible discrimination can however be made by the amount of energy the projectile transfers to the body [10]. So it is possible that, for example, an AK-47 bullet, with a muzzle velocity of 1,100 m/s, will hit a victim placed several hundred meters away from the assailant only with enough energy to penetrate the skin and subcutaneous tissue. This gunshot wound (GSW) can then be considered a low-energy transfer (LET) injury, similar to a small caliber pistol injury. However, a close range pistol GSW can reveal a high-energy transfer (HET) concomitant injury such as devitalization seen by the temporary cavitation caused by the shockwave of the passing projectile (Fig. 26.1). Though notably inconclusive ("minding his own business when suddenly attacked by strangers"), a history of a patient suffering from PI might render information whether he might suffer from HET or LET. This information could, for example,

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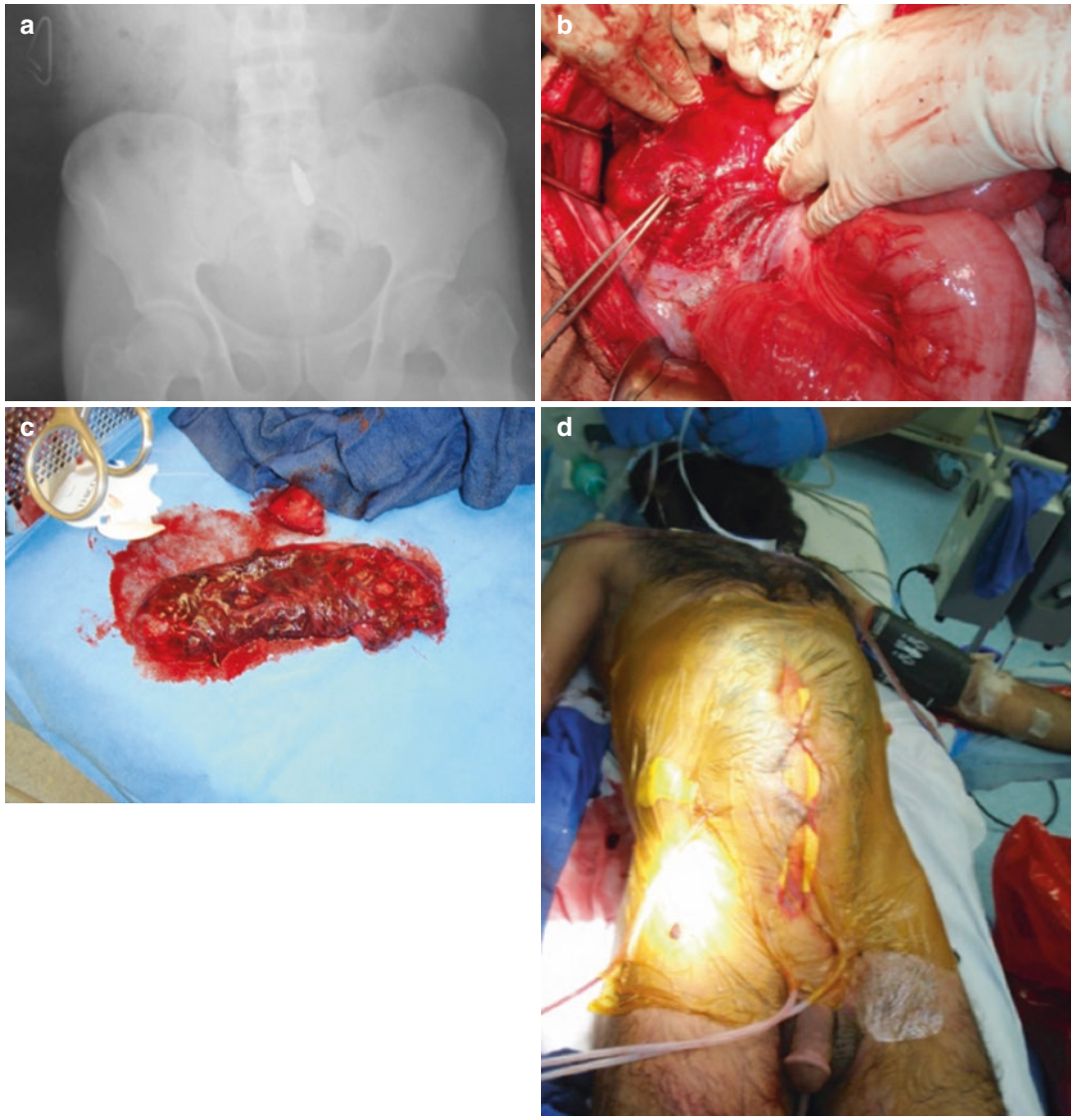

Fig. 26.1 (**a**–**c**) A HET projectile (**a**) causing not only penetrating injury to the cecum (**b**) but also ischemia of the whole cecum and ascending colon (**c**); hence the patient was treated with staged surgery (**d**). After 12 h definitive surgery with ileocolic anastomosis was performed

support the surgeon's decision to perform staged surgery for injuries to the gastrointestinal tract to assess the vitality before performing a definitive anastomosis in (suspected) HET PI. On physical examination all clothing should be removed as soon as possible to exclude additional injuries of which the patient or prehospital emergency services personnel was not aware. Special care should be given to junctional areas (axillae, groin, neck) and skin folds, since these areas are prone to miss PI. It is advisory to mark all wounds. Paperclips can be bend a P or A shape, as to indicate the posterior or anterior side of the patient (Fig. 26.2). This is helpful to assess which body cavities or organs might be injured. It should be mentioned though that the projectiles might not have traveled through the tissue in a straight line. In case of GSW the number of paperclips should be an even number (entry wound corresponding with exit wound) or corresponding with a bullet.

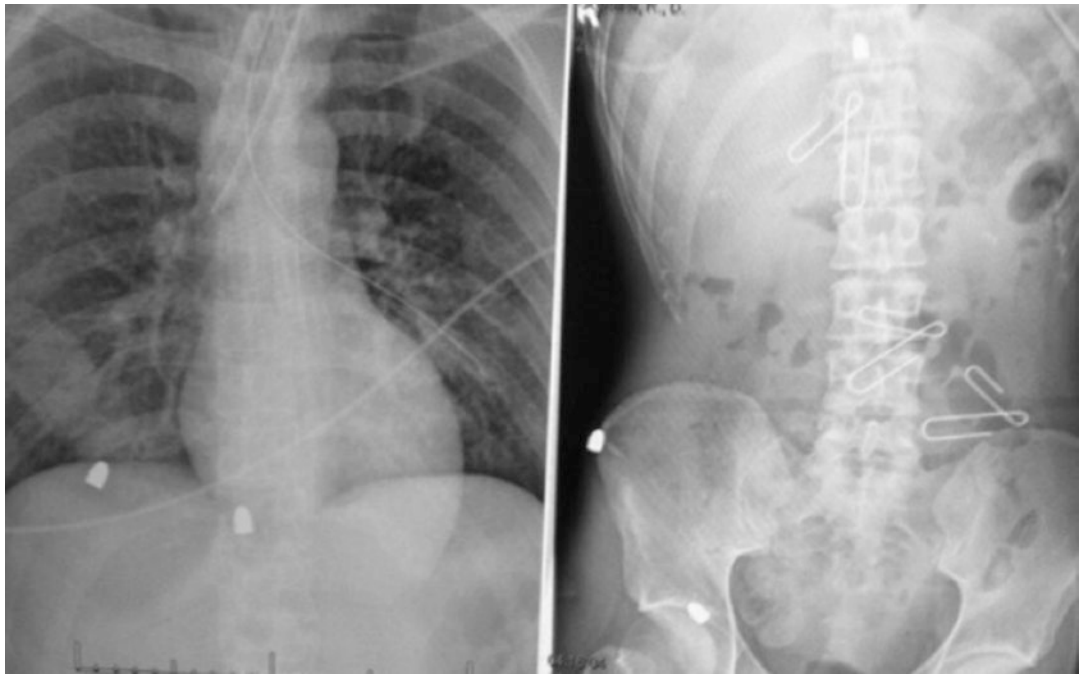

Fig. 26.2 X-ray without (**a**) and with paperclip markers (**b**). The A-shape bend paperclips indicate three anterior gunshot wounds corresponding with three bullets

When an uneven number is counted, not matched with a projectile, the examiners should depict the adjacent body regions with X-rays until the bullet is found, if the hemodynamic status of the patient allows [11, 12].

26.3 Damage Control Options for Penetrating Injuries to the Head and Neck

As mentioned prior the amount of energy, which is transferred to patient's tissue, indicates the outcome. This is certainly true for gunshot wounds to the brain. Though it should be mentioned that not all gunshot wounds to the brain are lethal. The recent military conflicts in Iraq and Afghanistan produced data in which rapid damage control craniectomies for penetrating brain injuries produced an increase in survival [13–15]. In civilian practice these aggressive damage control resuscitation and neurosurgical treatment strategies have also been successfully implemented, under the adagium "time is brain" [16, 17]. Patients with brainstem reflexes and a Glasgow coma score of 3 and over, after successful resuscitation with CT scan proven mass lesion effect, should undergo decompression via craniectomy (Fig. 26.3) as soon as possible to improve survival and outcome. Patients who display a "tram-track sign" caused by cavitation of a passing projectile, or those with transventricular injury approximately 4 cm above the dorsum sellae, the so-called zona fatalis, will not benefit from decompression and should be treated expectantly [18–20]. If the patient's hemodynamic status does not permit a CT scan of the brain, synchronous damage control surgery by both trauma surgeon and neurosurgeon can be performed if a mass effect due to the penetrating brain injury is suspected. If the measured intracranial opening pressure does not warrant diagnostic burr holes and damage control surgery has been successful, a CT scan of the brain should be acquired as soon as possible to assess brain injury and treatment options [16]. For stab wounds to brain with sharpened objects, apply the same indications for neurosurgical treatment as for gunshot wounds with exteriorized objects in situ as additional indications (Fig. 26.4)

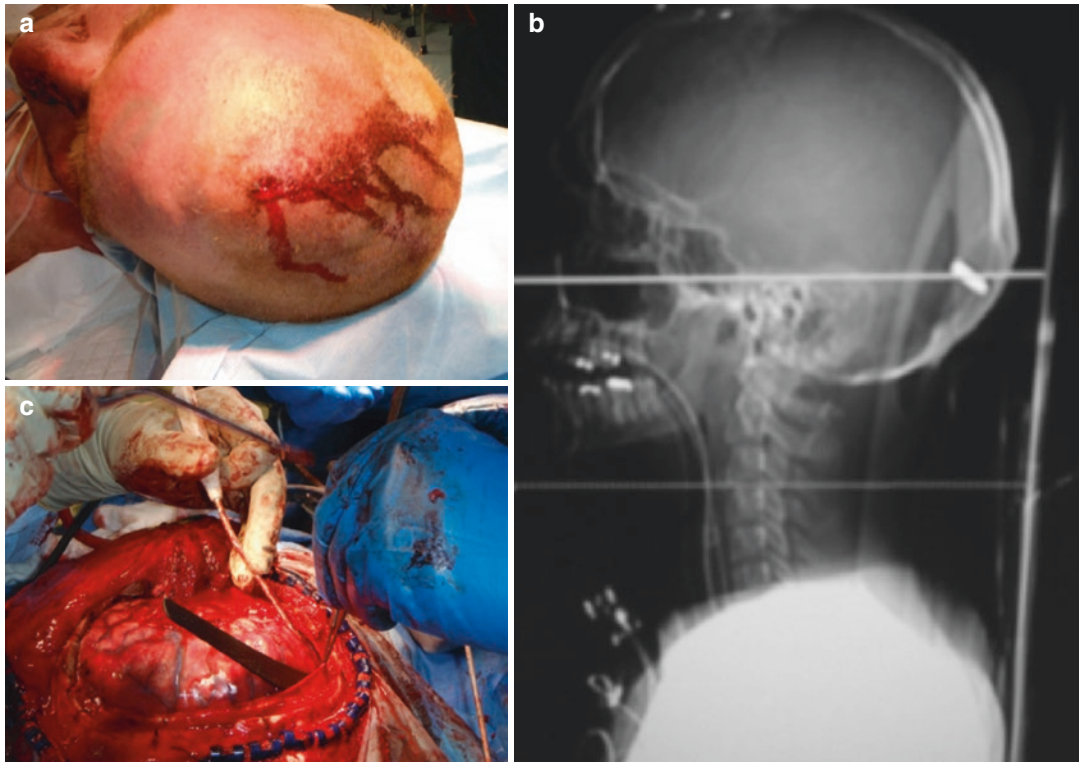

Fig. 26.3 Clinical presentation (**a**) and CT planogram (**b**) of HET gunshot wound limited to one hemisphere as indication for decompression via craniectomy (**c**)

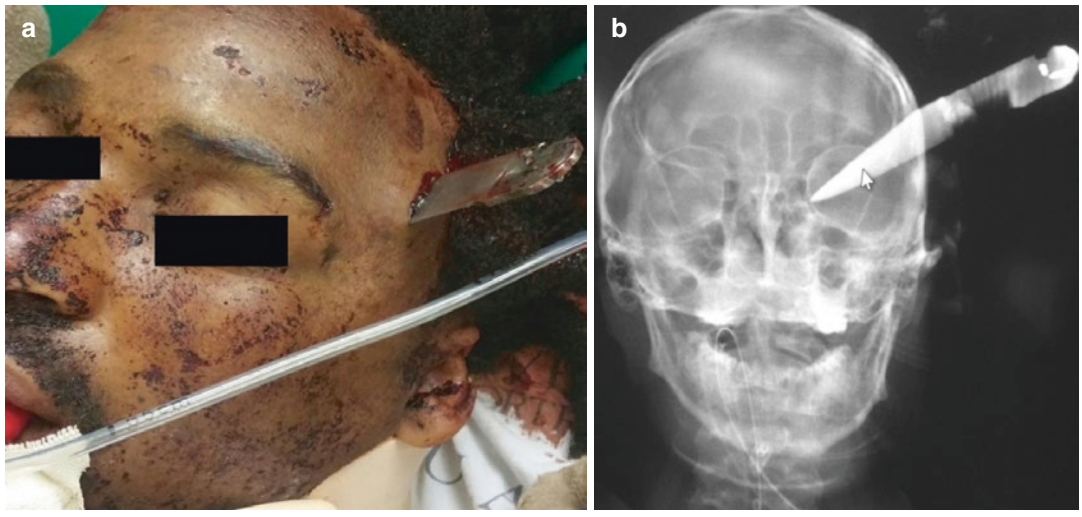

Fig. 26.4 Clinical presentation (**a**) and X-ray (**b**) of an exteriorized knife still in situ

[21–23]. Penetrating injury to the neck can generate a predicament, especially when gross bleeding is noted [24, 25]. In low-volume centers for penetrating injury, there might be the tendency to rush to theater for surgical exploration, with an increased risk on iatrogenic injury [26, 27]. Foley catheter balloon tamponade can be used as a damage control resuscitative tool to regain

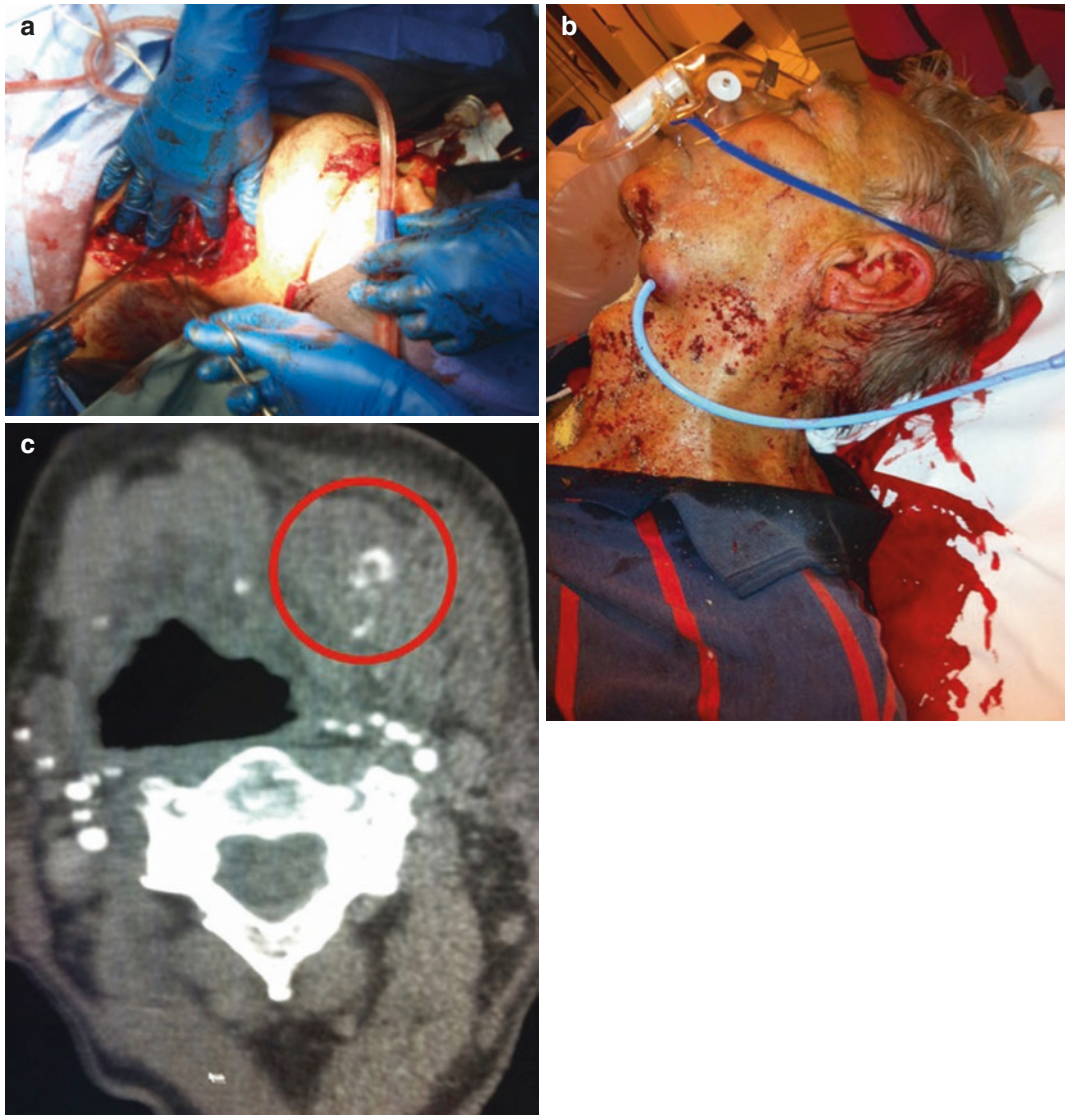

Fig. 26.5 Rushed into exploration of a penetrating neck injury with considerable change of iatrogenic injury (**a**). Foley catheter balloon tamponade achieving hemostasis (**b**), thus creating a controlled situation for the patient to be assessed for vascular or hollow organ injury via computed tomography angiogram of the neck (**c**)

hemodynamic stability and temporary hemostasis to bridge to endovascular or surgical treatment after CTA assessment of the neck (Figs. 26.5 and 26.6). When no arterial bleeding needs to be addressed, the catheter is deflated and removed in controlled surroundings. Successful conservative treatment for penetrating neck injury, when using this strategy, can be 87% [28]. When surgical exploration is indicated, the surgeon should be familiar with shunts suitable for the carotid vessels if the patient's condition does not allow definitive primary repair or an interposition graft. For bailout options in case of persistent bleeding in which suture techniques might not be successful, such as injury to vertebral artery, again Foley catheter balloon tamponade can be used or hemostatic granules or bone wax. Esophageal injury repairs, especially in combination with tracheal injury, should be protected with mobilized strap muscle and a drain.

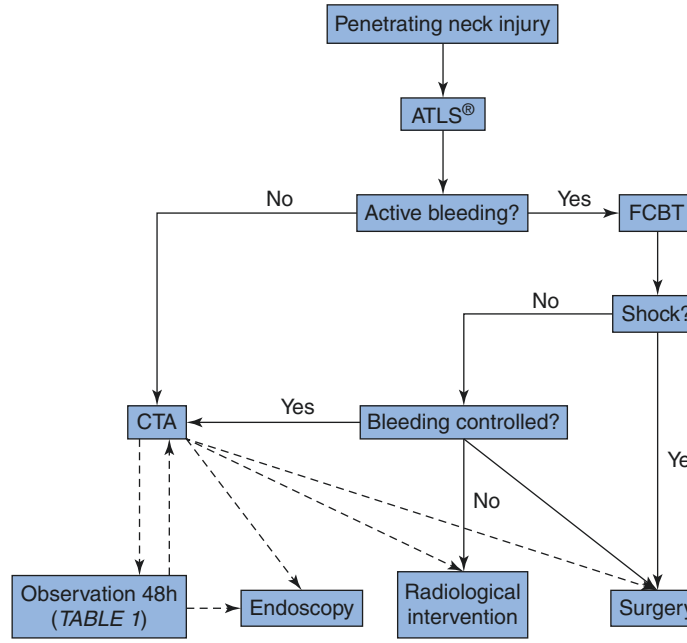

Fig. 26.6 Flowchart for management of penetrating neck injury with optional Foley catheter balloon tamponade

26.4 Damage Control Options for Penetrating Injuries to the Chest

For penetrating injuries of the chest, it should be mentioned that gunshot wounds, especially with an oblique trajectory, are likely to perforate the diaphragm and thus might inflict intra-abdominal injury. This indicates that patients should have sterile exposure and surgical draping that, if needed, allows an additional laparotomy when the primary surgical exploration is a sternotomy or thoracotomy (Fig. 26.7). In case of a transient or nonresponder to resuscitation with a systolic blood pressure that cannot be raised over 60 mmHg in the shock room, a resuscitative thoracotomy is indicated [29, 30]. This also applies for patients suffering from penetrating chest injury with witnessed cardiac arrest. Though there is no global consensus with regard to the "downtime" after which an emergency thoracotomy is still justified, it is generally accepted that survival for patients with ongoing cardiopulmonary resuscitation longer than 15 min is nil [31]. Unless the injury is clinically limited to the right side of the chest, a left-sided anterolateral approach is standard for an emergency department thoracotomy. The pericardium should always be opened as soon as possible, since from the outside it cannot be assessed for the presence of a hemopericardium. Opening of the pericardium, ventral to the phrenic nerve, has the additional benefit of being able to perform more efficient cardiac compressions. For massive pulmonary bleeding, clamping of the lung parenchyma or hilium is an option. Another alternative is the so-called pulmonary hilar twist, in which the apex of the lung is twisted downward to the diaphragm and the lower lobe upward after release of the inferior pulmonary ligament [32]. Simple suture closure of perforating brisk bleeding lung injuries is not an option, and the development of intrapulmonary hematoma or a possible air embolism can only be prevented by performing a pulmonary tractotomy. This can be achieved by "connecting" the entrance and exit (or creating an exit) wound of the lung parenchyma using a GIA stapler. The injured vessels and bronchi which have not been sealed by staples can then be selectively ligated (Fig. 26.8) [33]. In order to limit the circulation to the chest and head while other resuscitation techniques are

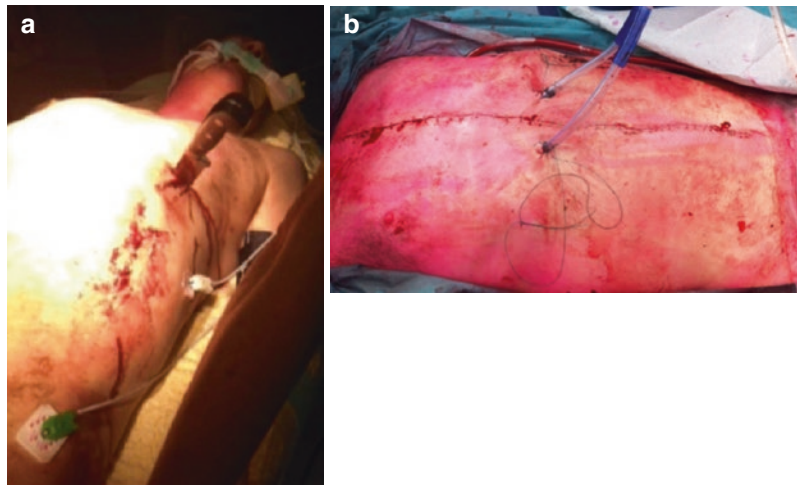

Fig. 26.7 Penetrating injury to the chest with both pericardial and subdiaphragmatic injury (**a**), indicating a sternolaparotomy (**b**). Full sterile exposure facilitated a swift extension from sternotomy to laparotomy

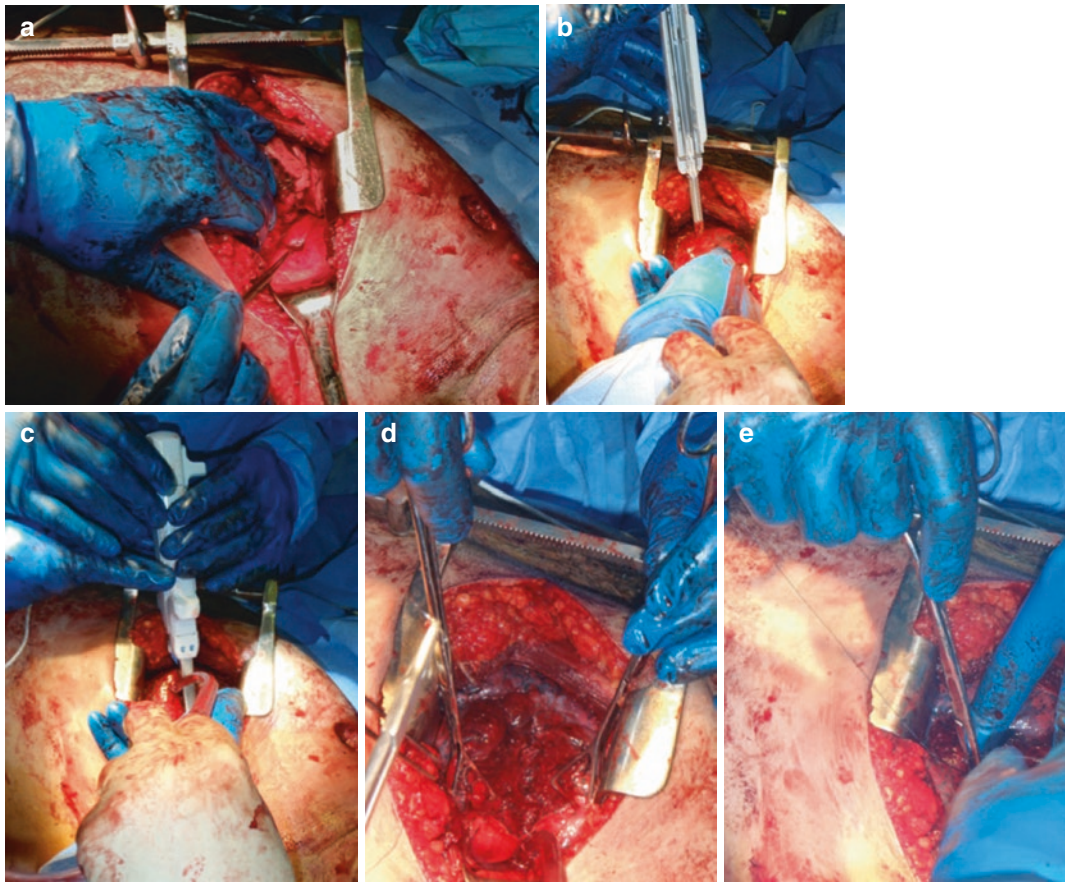

Fig. 26.8 A gunshot wound to the left lung (**a**). A tractotomy using a GIA stapler (**b**, **c**) and selective ligation of bronchi and vessels with persistent leakage (**d**, **e**)

applied such as placement of central lines, the aorta can be clamped. Positioning of the clamp can be cumbersome in a flaccid aorta. It is more practical to compress the aorta to the vertebral column with fingertips. After successful administration of circulating volume, the aorta will be pulsatile again with a normal caliber and easier to clamp. If the injury is trans-mediastinal or injury to other side of the chest is suspected, the thoracotomy can be extended by cutting the sternum and the intercostal musculature of the right side of the chest into a so-called clamshell thoracotomy with an excellent exposure of both left and right side of the thorax and mediastinum [34]. In patients in extremis with a penetrating injury trajectory that is suspected for pericardial injury and in whom the clinical status did not allow assessment of the pericardium via CTA or ultrasonography, a "subxiphoid window" procedure should be performed prior to laparotomy. The pericardial sac is approached preperitoneal, aided by elevating the xyphoid process with a clamp. If the evacuated fluid from the pericardium is clear or serosanguinolent and remains clear after rinsing with saline, the drain production can be monitored. When blood is encountered, the procedure should be converted to sternotomy for most likely myocardial repair [35–37] (Fig. 26.9). Small injury of the right side of the diaphragm can be repaired if easily reached by limited mobilization of the liver to prevent the possibility of bile leakage in the pleural cavity. Left-sided diaphragmatic injuries always need closure with nonabsorbable sutures to prevent future complication (e.g., intrathoracic herniation of peritoneal content). In case of gross intra-abdominal fecal spillage, the laceration can be enlarged, parallel to the phrenic innervation, or using an additional incision posterolaterally in a curvilinear orientation to facilitate washout of the pleural cavity

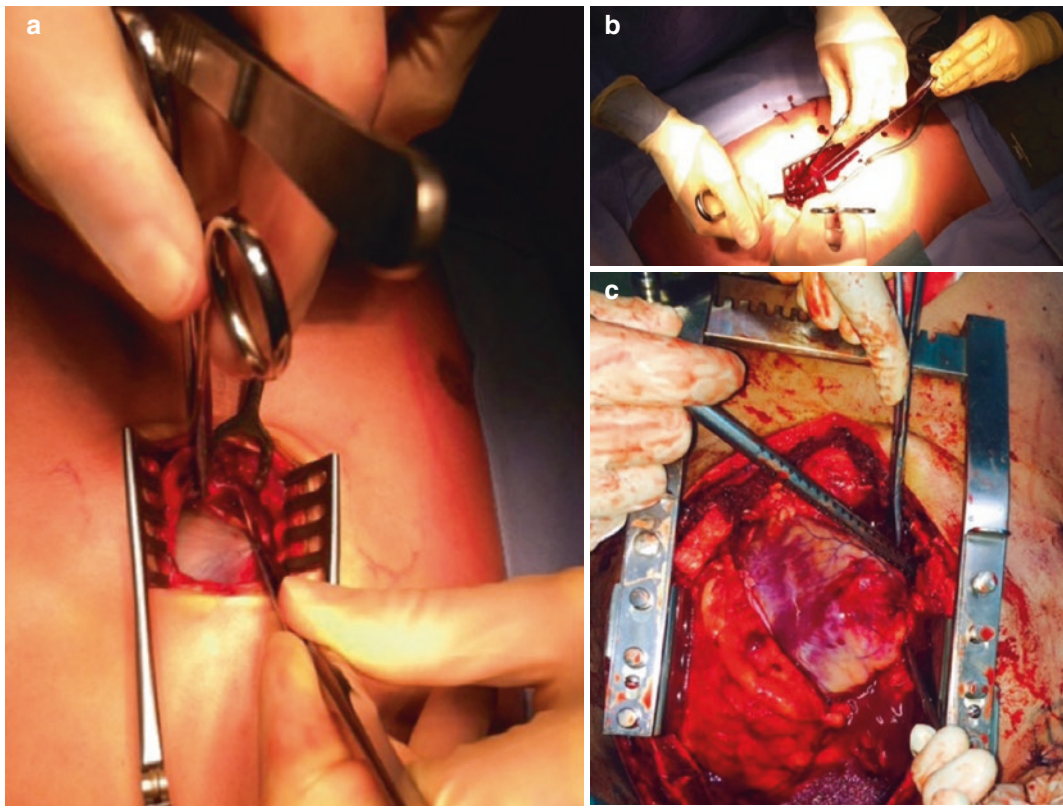

Fig. 26.9 A subxiphoid window procedure for a suspected pericardial injury (**a**). Clear fluid can be monitored using a drain for 12 h. Bloody effusion (**b**) indicates conversion to a sternotomy (**c**)

with several liters of saline to diminish the bacterial load and change of formation of pleural empyema [38].

26.5 Damage Control Options for Penetrating Injuries to the Abdomen

A fair number of tangential abdominal gunshot wounds and the majority of abdominal stab wounds can be treated via selected nonoperative management principles of careful examination and repetitive clinical reassessment. Exploratory laparotomies are indicated for patients with peritoneal signs with or without shock [39]. Venous (liver) bleeding, encountered during clock- or counterclockwise inspection of the four quadrants of the peritoneal cavity, is amenable for packing. Arterial bleeding, such as mesenterial vascular injuries, cannot be packed but should be temporary clamped and repaired or suture ligated as soon as possible. Since penetrating injuries to the gastrointestinal tract are easily missed, especially at the border of the mesenterium with the bowel wall, both the lead surgeon and his assistant should "flip flop" the entire bowel with mesenterium from side to side to inspect the whole circumference. If the patient's condition dictates staged surgery, injured bowel segments can be stapled or resected and temporary tied for later definitive anastomosis during relook laparotomy. Kocher's maneuver is mandatory if a duodenal injury is suspected. Primary tensionless repair of duodenal lacerations should be attempted and concomitant pancreatic injuries are to be drained. The suture line can be protected with decompressive jejunal-cutaneous fistula using (Foley) catheters and optional a more distal one for enteral feeding. Another possibility to spare the duodenal repair is by pyloric exclusion [40]. However, these procedures are time demanding and not recommended in a DCS modus. The lesser sac is always to be explored to exclude injury to the posterior gastric wall and pancreas. Indications for an emergency Whipple's procedure are scarce and not recommended since most patients suffering from pancreatic head injuries, in combination with duodenal injury, are usually in need of a DCS approach due to additional vascular injuries. A prompt but sound assessment of the extent of gland and duct injury dictates further future surgical management. Minor injuries without visible duct involvement are drained. Injury to body and tail of the pancreas with duct laceration are treated with a distal pancreatectomy, en bloc with the spleen using a linear stapler, gaining rapid control of bleeding and leakage (Fig. 26.10) [41]. Liver lacerations treated with "packing," in which six abdominal sponges should suffice, dictate a relook laparotomy for pack removal preferably after 48 h post placement to prevent rebleeding [42]. Another option for liver lacerations in a non-shocked patient, to prevent open

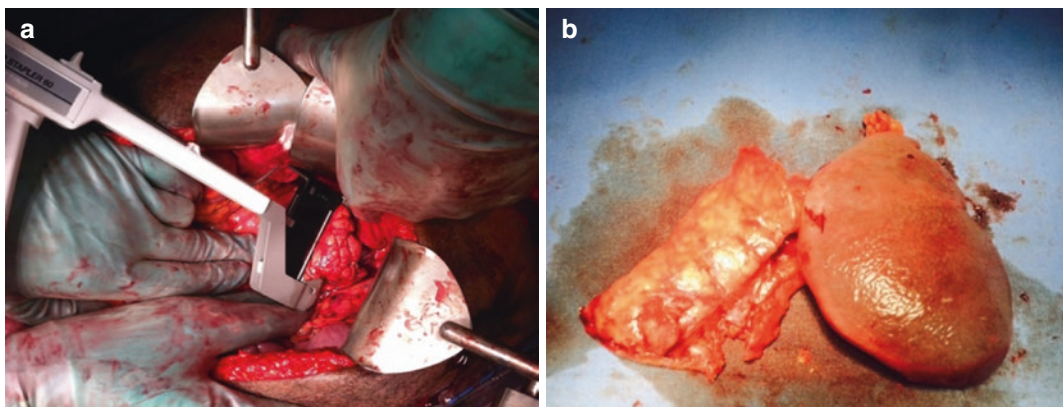

Fig. 26.10 A penetrating injury to the tail of the pancreas swiftly controlled using a linear stapler (**a**) for en bloc resection of pancreas tail and spleen (**b**)

abdominal treatment, is careful placement of several large diameter monofilament sutures using the figure of eight configuration to gain hemostasis and diminish bile leakage (Fig. 26.11). Central retroperitoneal hematomas are in need of exploration to exclude injury to the duodenum, pancreas, aorta, and inferior vena cava [43]. Injury to the latter can be masked by a low flow state in combination with containment by the peritoneum, especially in through and through perivertebral gunshot injuries. Cava injuries are notorious for rapid exsanguination once the tamponading effect of the peritoneum is released. Hence it is advocated, if inferior vena cava injury is suspected, to compress proximal and distal of the injury onto the vertebral column by an extra assistant before opening the peritoneum. If the injury is not amenable for primary repair, ligation is an option for hemorrhage control, which will be tolerated by the patient [44]. A non-expanding lateral retroperitoneal hematoma does not need surgical exploration, unless colonic injury is suspected. Large expanding lateral hematomas are most likely to be caused by kidney injury beyond repair (AAST injury scoring scale grade 4 and 5). Nephrectomy is best performed via a lateral approach using the dissection established by the hematoma, after which the hilum and ureter can be ligated [45]. Repair of ureter injury in a DCS setting is not advisable. Instead the injury can be drained and tacked for repair in relook surgery. Percutaneous nephrostomy can be used as a bridge to definitive ureter repair. Simple intraperitoneal bladder injuries should be repaired with transurethral and/or suprapubic drainage. More complex or extraperitoneal injuries receive a para-cystic drain after provisional hemostatic suturing [46]. In contrast to pelvic retroperitoneal hematoma caused by blunt force, hematoma by gunshot or stabbing will need exploration. These haematoma are usually caused by injury to iliac artery, vein, or predicamentally a combination of both. Shunting, with optional fasciotomy of the lower leg, can be a limb-saving damage control strategy in these injuries. When the trajectory is suspected for injury to the rectum, a negative digital rectal examination should always be followed by rigid rectosigmoidoscopy prior to laparotomy (Fig. 26.12). When blood or injury to the rectum is confirmed, pelvic sepsis should be prevented by a diverting colostomy. Injury to the rectosigmoid should be assessed, during laparotomy, for primary repair or "bailout" diverting colostomy and drainage [47]. Damage control laparotomies should always finish with a (provisional) closure of the abdominal wall wounds caused by the firearm or blade, to prevent future herniation of abdominal viscera, and a thorough washout with several liters of warmed saline. In order to protect the viscera till the subsequent

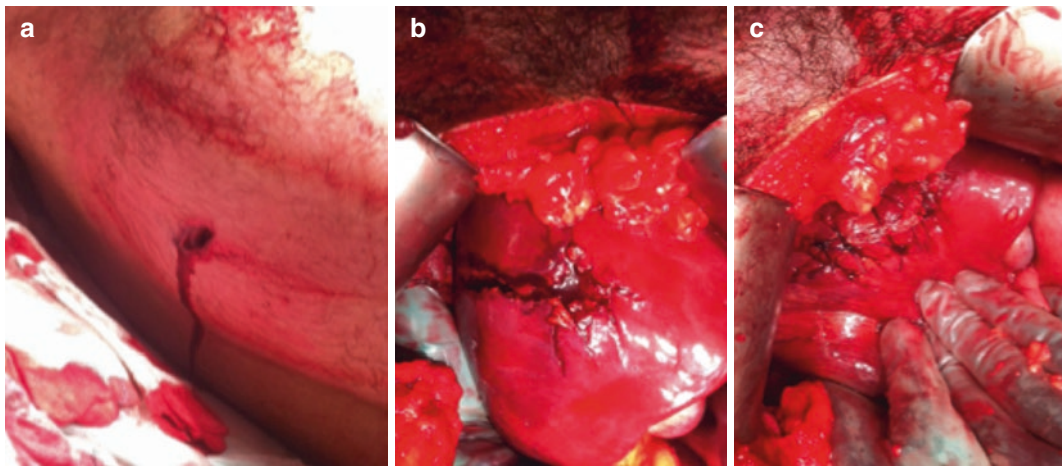

Fig. 26.11 A gunshot injury (**a**) to the liver. Instead of packing, large monofilament sutures in a figure of eight configuration were used to treat the laceration (**b**, **c**). Thus avoiding the need for pack removal and relook surgery

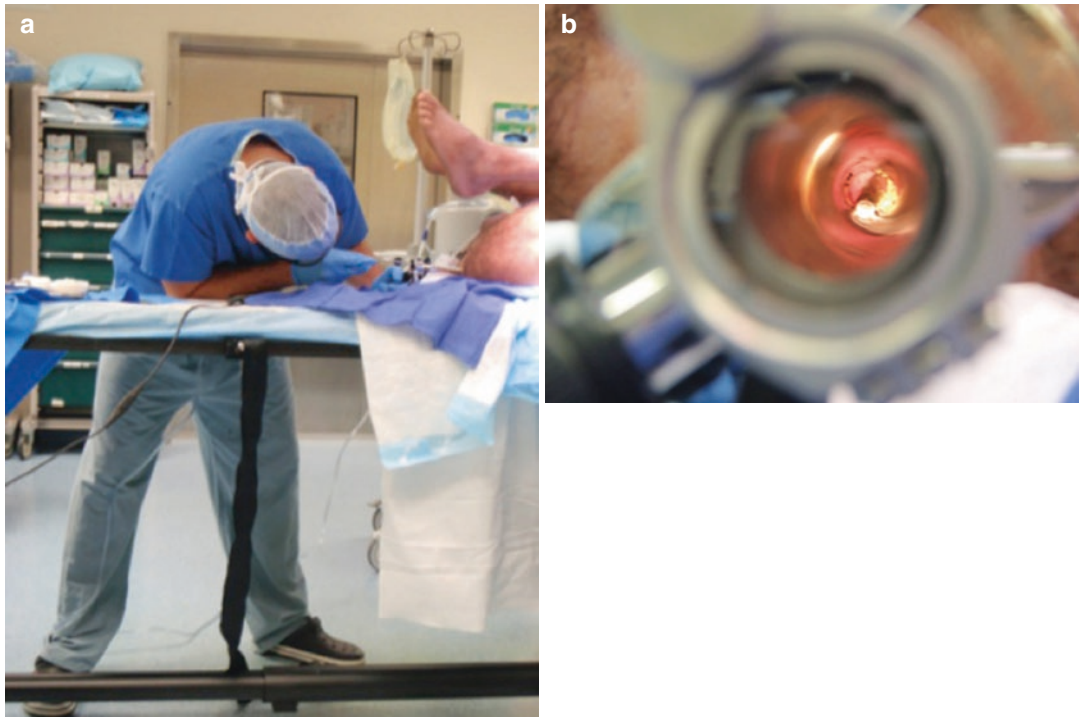

Fig. 26.12 Rigid rectosigmoidoscopy prior to laparotomy (**a**), revealing a bullet in the rectum (**b**)

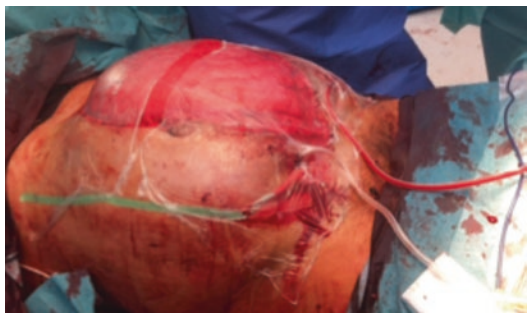

Fig. 26.13 A temporary abdominal closure device (TAC) fashioned from two percutaneous drains connected to wall suction and abdominal packs covered by adhesive plastic sheets. A low-cost solution in patients prone for an abdominal compartment syndrome or in need of relook abdominal surgery as part of DCS

surgery and prevent an abdominal compartment syndrome, a temporary abdominal closure device can be fashioned from a combination of (adhesive) plastic sheets, gauze, and percutaneous drains connected to a suction device (Fig. 26.13) as a (cheaper) alternative to commercial negative pressure wound therapy [48].

26.6 Damage Control Options for Penetrating Injuries to the Extremities

Though the doctrine of damage control surgery dictates "the life over limb" principle, it should be stressed that seemingly insignificant injuries to the extremities can be life threatening. Junctional penetrating injuries (e.g., groin, axillae) can be difficult to control since these injuries, in contrast to the more distal injuries, are not suitable for temporary hemorrhage control using a standard tourniquet. Not only in a prehospital or emergency department setting but also in the operation theater can hemostatic bandages or granules and catheter balloon tamponade render provisional hemorrhage control for these injuries in which rapid access for vascular control is difficult [49, 50]. As mentioned prior, the surgeon should be familiarized with shunt options to bridge vascular injuries. Most vascular injuries due to stabbing or projectiles are not fit for primary repair and thus will need an interposition

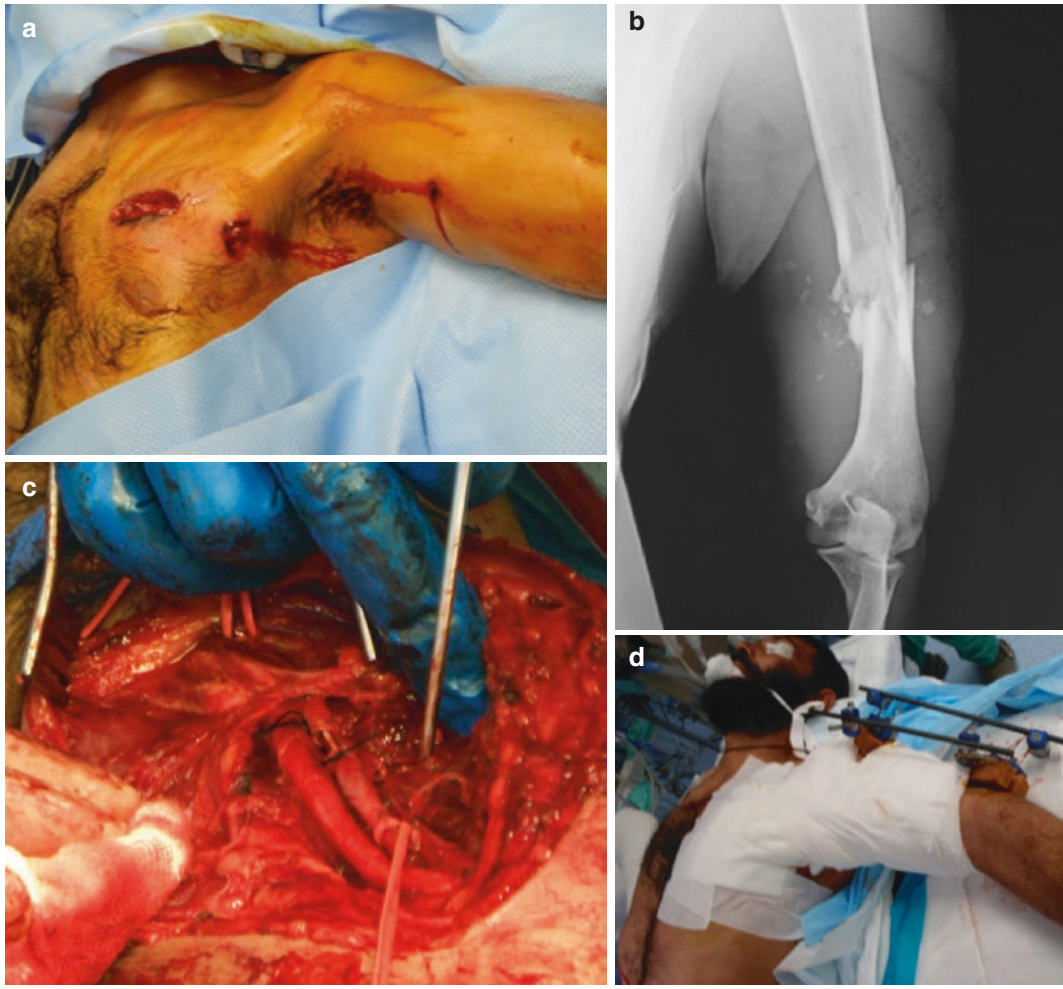

Fig. 26.14 A gunshot injury to the left upper extremity (**a**) with a concomitant fracture of the humerus (**b**). In DCS treated with a shunt for the brachial artery injury (**c**) and external fixator (**d**)

graft. Patients in need of DCS are not in the condition to undergo the lengthy procedure of gaining proximal and distal vascular control followed by harvesting and preparing a vein graft, which can then be sutured into the defect. A more realistic scenario is a patient with multiple penetrating injuries in need of DCS. When confronted with a penetrating injury to the extremities with a vascular deficit, it is more than likely to be accompanied with a fracture and nervous injury. The latter can be tagged with 5:0 monofilament suture for later definitive repair. The most practical approach to these combined injuries is to gain vascular control and shunt the defect to preserve distal flow. This is followed by placement of an external fixator, in case of a fracture (Fig. 26.14). Prophylactic fasciotomy of the affected limb is highly advocated, prior to definitive repair or when using a shunt in a DCS case [51, 52].

Conclusion

Patients with penetrating injuries can present themselves to any emergency department. Hence all trauma care providers should be familiar with the injury patterns (HET versus LET) and treatment options. Hemorrhage control techniques used in prehospital or emergency department settings (e.g., hemostatic

agents and catheter balloon tamponade) can be used as "bailout" options in theater as well. When DCS is needed, it is advised to return the patient to theater for definitive repair as soon as the preset resuscitation values are established to avoid the detrimental effects of missed injury for which this patient group is prone.

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27 Complications After Damage Control Surgery: Pin-Tract Infection

Peter V. Giannoudis and Paul Harwood

27.1 Introduction

Damage control is an approach to care for patients in extremis [1]. The primary aim is to avoid worsening the patient's situation by only undertaking interventions in the acute phase that are life or limb saving. Complex, prolonged or specifically risky or immune stimulatory procedures are avoided. The patient then undergoes staged reconstruction once their physiology has sufficiently recovered for them to withstand this. As regards to orthopaedic surgery, this usually involves the initial application of spanning external fixation to rapidly stabilise skeletal injuries [2–4]. Intramedullary nailing has particularly been associated with immune stimulation and systemic complications [5]. The patient then undergoes further definitive procedures at an appropriate time point once initial concerns regarding life-threatening physiologic disturbances have passed. This can be termed systemic damage control. Moreover, it has been recognised lately that immediate definitive care of complex isolated extremity injury may also be inappropriate (Fig. 27.1) [6–8]. Large incisions and internal fixation in swollen, compromised or violated soft tissues have been associated with an excess of wound and infective complications [9, 10]. Examples include open and complex periarticular injuries, particularly in the lower limb [11]. It is often sensible to temporarily impart secure skeletal stabilisation using an external fixator in such situations in order to allow the soft tissue swelling to subside and any open injuries to be treated prior to definitive fixation. Furthermore, such injuries are increasingly being treated in expert centres by specialists. This means that temporary stabilisation may be relevant in certain situations where it would not otherwise be required, allowing safe transfer to facilities where appropriately skilled personnel and equipment are available. This is termed local damage control.

A major consideration when utilising temporary external fixation is pin-site care and infection. Particularly where patients are to undergo definitive internal fixation, violation and potential bacterial contamination of the soft tissue envelope raise concerns regarding subsequent deep infective complications which are a major

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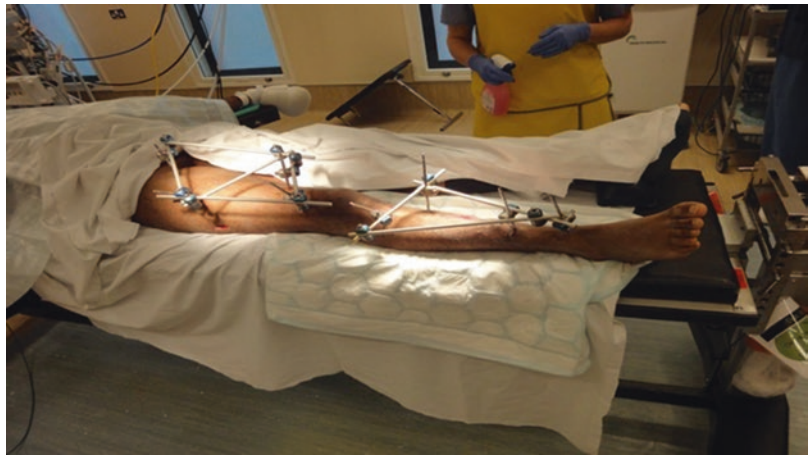

Fig. 27.1 Male patient 26 years of age that sustained a right floating knee injury which was initially managed with external fixators applied to the ipsilateral femoral and tibial fracture

source of morbidity and extremely expensive to treat. Here we review current evidence regarding pin-site care in damage control orthopaedics, infection rates following such procedures and practices which may help reduce such problems. Indications and effectiveness are explored elsewhere.

Whilst specific literature on damage control applications of external fixation exist, it is important to note that much of the evidence and knowledge on pin-site care and infection are based on experience from definitive external fixation including fine wires. In the absence of other sources of information, it is reasonable to apply such findings to this practice, but it is appropriate to consider that important differences apply, not least the nature of the fine wire/tissue interface in comparison with more rigid half pins and the length of time that the fixators are applied.

27.2 Indications for Damage Control

In attempting to avoid infective complications when using external fixation for damage control, certain precautions should be taken from the outset. A fundamental aspect of this is applying the indications for damage control correctly [12]. Whilst various studies have identified little or no excess of deep infective complications following such an approach, the presence of external fixation pin sites logically must introduce the potential for this to occur. It is therefore important that these decisions are not taken lightly, and external fixators are only applied where they provide true benefit. Counterargument to this is that when applied carefully, this approach is safe and reduces the risk of more serious, potentially life-threatening complications. It is important that the balance of risk versus benefit is carefully and individually assessed. The level of concern is related to the planned definitive fixation method. If the injury is to be managed by internal fixation, particularly intramedullary nailing, more careful consideration is required. There has perhaps been a tendency to adopt a systemic damage control approach too readily at times, and this has been a source of criticism of the technique [12, 13]. Clearly these decisions must be taken individually and the indications for systemic damage control applied with caution. Regarding local damage control, the literature would tend to support that early definitive internal fixation in a limb where the soft tissues are suboptimal leads to an increase in wound and infective complications compared with a period of immobilisation to allow soft tissue care [6, 10, 11]. Whether this can be achieved by plaster immobilisation should be carefully considered but, particularly in the lower limb and

absolutely where the soft tissue envelope is breached or severely compromised, it is the author's experience that thoughtfully applied external fixation is almost always preferable in unstable injury patterns. This provides much more stable immobilisation, is usually more comfortable for the patient, allows easy access to soft tissues for care and inspection and usually helps to provisionally reduce fractures, making definitive fixation more straightforward. Similarly, it has been demonstrated that management of complex injury by non-specialists without adequate imaging, planning or equipment will lead to an increase in complications and poor outcome, including infection. It is therefore reasonable to adopt such an approach to allow treatment to be planned and patients transferred where necessary to appropriate facilities [14].

27.3 Considerations in Application of Spanning External Fixation for Damage Control

Steps can be taken when applying the external fixator to reduce the risk of serious infective complication. Careful technique is critical, and potentially minor problems can quickly escalate into serious complications.

27.3.1 Priorities When Placing Pins

The process of applying stable external fixation is well described elsewhere [15]. There are specific considerations when considering the risk of infection following subsequent conversion to definitive fixation. It is useful to think of the competing priorities in pin placement when applying such fixators in these terms. One should remember at all times that these factors are relative; the need to comply with anatomic constraints and avoid injuring critical neurovascular structures during pin insertion remains paramount. Imparting stability to the construct is usually the next priority; without this, the act of applying the fixator becomes futile, and indeed, a poorly applied unstable fixator may represent more of a risk than no fixator at all. Even this is relative however, no temporary fixator will afford absolute stability, and at times the user may elect to accept a degree of instability in order to fulfil one of the following priorities. This is however a difficult judgement call, and the balance of these competing interests must be carefully considered in each case. It is generally recommended to avoid inserting pins through traumatised tissue – *the zone of injury*. Certainly placing pins in open fracture sites is not advised unless absolutely no other option exists. To avoid violating closed soft tissue injuries is a more relative recommendation, though such pins likely are at increased risk of infection. If the pin is inserted in areas of soft tissue stripping and closed (or open) degloving, these pins will effectively be in communication with the fracture site and associated haematoma. If such pins become infected, this can result in serious complication and would likely be a very significant risk if conversion to internal fixation were planned. Less severely injured tissues present a more relative problem and, whilst it is always preferable to place pins through normal tissues, if this results in a situation where a stable construct cannot be constructed then ongoing injury due to fracture site motion may well increase the risk of infection on conversion to internal fixation more than placing the pins through damaged tissue. Pin placement through areas of compromised soft tissue is at times inevitable to impart stability in severe injury. Placing pins outside the zone of proposed fixation is a more debatable aspiration. In some situations, for example where intramedullary fixation is planned, this is not possible as the nail effectively has a zone of fixation that traverses the entire bone. In fractures to be treated by plating the area where the plate will lie, particularly in the line of planned incision, should logically be avoided where possible. The evidence supporting this is however contradictory, with some studies finding an increased rate of deep infection in such situations whilst others do not [16, 17]. These are however relatively small retrospective studies, and negative results may be subject to type II sampling error and selection bias. It makes

logical sense that placing pins where internal fixation will lie should increase risk of infection. The question is to what degree this is a problem. Compromising pin placement to avoid the zone of fixation may render the construct so unstable that the effect of ongoing motion on soft tissue swelling and recovery might well outweigh any perceived advantage and actually make the risk of infection higher. Construct design in such cases needs careful consideration; a conscious effort should be made to place pins outside the zone of fixation where possible.

Where definitive external fixation is planned, final construct design still needs to be considered during pin placement though this is less of a concern. Deep infection in pin sites can become a problem even in the absence of internal fixation, and therefore sensible steps should always be taken to minimise this risk. It is also sensible to avoid pin insertion in areas where definitive external fixation will ultimately be placed, as infection with resultant soft tissue and bone damage here may compromise this. This is even more important in complex cases where corticotomy for bone transport might be required and careful consideration to the placement of external pin must be given in these situations. Furthermore, it is sometimes helpful to leave the temporary fixator in place whilst applying definitive fixation, particularly with complex injury patterns and articular injuries. Thoughtful fixator construction is required to facilitate this.

27.3.2 Pin Insertion Technique

Meticulous surgical technique can help minimise soft tissue damage and ultimately reduce the risk of infection. This is described elsewhere [15]. In terms of reducing the risk of pin-site infection, the main emphasis is protection of the soft tissues by careful placement of incisions, blunt dissection and appropriate skin release to reduce pressure effects. Care should be taken during drilling and pin insertion to protect soft tissues to prevent mechanical and thermal damage. Unstable pins lead to a great increase in infection rates and add nothing to construct mechanics. It is therefore imperative that pins are inserted carefully to create a stable pin-bone interface. Pre-drilling to create a pilot hole followed by manual pin insertion is recommended. Secure bi-cortical purchase should be achieved and checked radiographically and the stability of the pin checked manually directly after insertion. If the pin is not completely stable, it should be resited. Haematoma formation at pin sites results in a very effective potential culture medium with a portal for inoculation. It is advisable to carefully clean the pin immediately following insertion and apply a dressing with a clip or bung to hold this in place to minimise this. If these become soiled, they should be changed at the end of the procedure. During fracture manipulation to achieve reduction, tissue tensions can change. It is therefore important to carefully check each pin again at the end of the procedure and further release any soft tissues as appropriate.

27.3.3 Stability

Ongoing motion of injured tissues, particularly at the fracture site, causes pain and leads to ongoing tissue trauma. This can result in persistent or increasing swelling and blistering and in patients with external fixation pins in place will potentially increase the risk of pin-site infection due to motion of the tissues around the pin. As highlighted above, it is therefore pertinent to afford as much stability as possible to the injured limb segment when applying external fixation in such circumstances. Placing pins appropriately and prioritising stability are discussed above and summarised elsewhere. It is important to critically assess construct stability following fixator application. This can be improved by adding fixation, improving working length (termed near-far pin arrangement), spanning joints with small periarticular fragments and altering arrangements of rods and bars. It is therefore critical that surgeons applying damage control external fixators are well versed in the principles of building stable constructs and understand the basic mechanics which contribute to this [15].

27.4 Pin-Site Care

Limited objective evidence regarding different pin-site care regimens is available. The majority relates to the care of pin sites for definitive rather than temporary fixators. The objective, to minimise the risk of infection, remains the same in either scenario. The principles are identical and to use the same approach is logical. A Cochrane review in 2013 concluded that there was insufficient evidence to recommend pin-site care strategy to minimise infection rates and what did exist was of generally poor quality [18]. The authors recommended that further clinical studies were undertaken to help answer this question. A more recent literature review reached the same conclusion [19].

In the absence of robust evidence on pin-site management, an expert group in the United Kingdom met in 2010 to form a consensus statement to help guide care [20]. They recommend the following:

- Sterile non-shedding dressings should be used to keep pin sites covered at all times (Fig. 27.2). These should be of a type that draws exudate away from the wound.
- Gentle compression should be applied to the dressing using a bung or clip.
- First dressing change should be carried out between 1 and 3 days post-operatively.
- Pin sites should then be cleaned and dressings changed every 7 days unless there is excessive discharge or infection is suspected. Under these circumstances, dressing should be changed when there is strikethrough.
- A non-shedding material should be used to clean the pin sites with chlorhexidine in alcohol. If this is contraindicated or the patient develops sensitivity, then saline should be used.
- Patients should keep pin sites dry and never immerse them in order to bathe but may shower immediately before dressings are changed (once a week).

The publication relates specifically to the care of patients with definitive care fixators. Important differences exist between these and the type of temporary devices under discussion here. Probably most important is overall stability – definitive construct will usually be much more rigid as most are designed to allow motion and weight-bearing – and for this reason will have many more pin-tissue interfaces. They will be in place for much longer periods than damage control fixators, and there is usually no intention to replace the fixator with internal implants, and therefore the potential consequences of minor pin-site infections are less. Advice regarding showering is also probably not appropriate for those with damage control fixators.

27.5 Diagnosis and Treatment of Pin-Site Infection

Definitive diagnosis of pin-site infection can be troublesome and is on the whole reliant on clinical signs and symptoms. In general, culture swabs

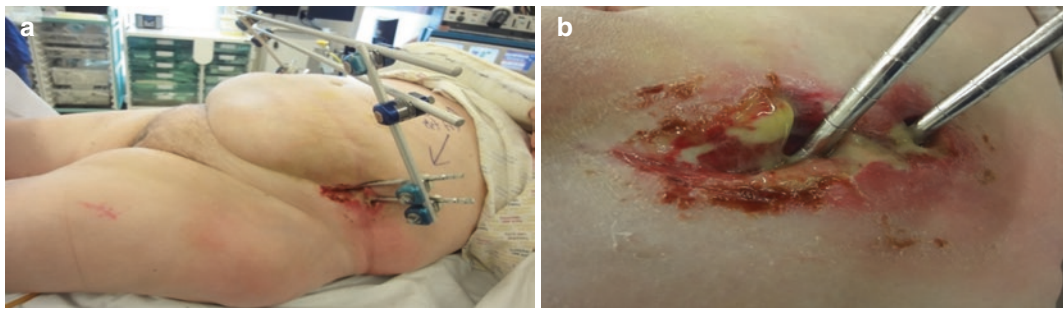

Fig. 27.2 Female patient sustained an open book pelvic ring injury which was initially stabilised with a Hoffman II external fixator and was transferred to our institution for definitive care. (**a**)The skin around the iliac crest pin sites looks inflamed. (**b**) There is also discharge of pus

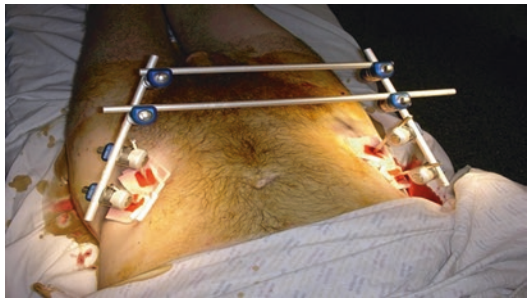

Fig. 27.3 A Hoffman II external fixator was applied in a male patient with an open book pelvic ring injury for temporarily stabilisation. The pin sites are covered with Allevyn dressing as per our unit's protocol

are not helpful as any external fixator pin will rapidly become contaminated and not only will a positive result not be diagnostic of infection but the cultured bacteria from the pin may well not be representative of any organism causing an infection. Previous studies in damage control patients found that rates of positive culture from fixator pins increased rapidly the longer a fixator was in place but that there was no correlation between positive results and subsequent infective complications [21]. Infection should be diagnosed based on the presence of increasing pain at a pin site, decreased tolerance of limb segment motion, spreading redness, increased swelling and discharge (Fig. 27.3) [20]. It is important to note that not all infected pin sites will discharge and not all discharging pin sites are infected. Serous discharge in the absence of other signs of infection is usually inflammatory in nature; purulent discharge is more indicative of infection. Various systems have been proposed in an attempt to standardise this clinical approach to diagnosis, and these may have some utility in clinical practice but on the whole are used for research purposes [22, 23]. Diagnosis is therefore based on classical signs of infection as outlined above.

In general, pin-site infections have been found to be effectively treated by increased frequency of pin-site care, relief of any predisposing factors and oral antibiotics. Spreading infection or signs of systemic infection should prompt investigation for other sources or local collections. The pins and overall construct should be critically assessed. Any causes of soft tissue pressure should be relieved; this may require skin release at the pin sites once the infection has settled which can usually be achieved using local anaesthetic. Soft tissue motion around pin sites is a frequent cause of infection; if the construct is unstable or pins are close to joints, it may be that revision is required to improve this, possibly spanning adjacent joints. If this does not address the issue, then pins should be removed and resited. If infection is serious or recurrent, then serious consideration should be given to the safety of proceeding with exchange to definitive fixation, and this should certainly be delayed until the infection has been treated. It may be that the injury is suitable for treatment with definitive external fixation, which would likely be safer under such circumstances. Some studies have failed to find correlation between previously clinically infected pin sites and late deep infection [24, 25]. Others have found a strong link between cases where pin sites became infected prior to exchange and subsequent deep infective complications [26–28]. This body of literature is difficult to interpret and the numbers are relatively small, and therefore subgroup analysis examining only those in the external fixation groups who suffered pin-site infections is even smaller and prone to error. The studies are heterogeneous and on the main retrospective with divergent inclusion criteria, making grouped interpretation very troublesome. Authors in some studies did not advocate exchange in patients with previously infected pin sites at all or removed the fixator for a period prior to the secondary procedure [2, 29, 30]. Another excluded patients with infected pin sites from analysis [31]. It is clear that each case must be taken on its merits and great care taken in decision-making with patients who do suffer pin-site infection.

27.6 Exchange from Temporary to Definitive Fixation and Results

Decision-making around exchange to definitive fixation is critical if the advantages of adopting this approach are to be realised. The definitive

fixation method should be chosen as appropriate, not just for the bony injury in question, but taking into account the patients systemic status, soft tissue envelope and the presence and state of the spanning external fixator. It may be, for example, that an injury which in other circumstances might be best treated by intramedullary nailing would be better treated using a circular frame in a patient who has been critically ill in intensive care following multiple injuries with an external fixator in place for 6 weeks. A patient with a distal tibial fracture in whom definitive intramedullary nailing was planned might be better served by plate or Ilizarov fixation in order to move the implants out of the zone of fixation if the proximal pin sites have been recurrently infected and the plate would involve these.

Table 27.1 summarises infective outcomes and timing of exchange from external to internal fixation in major publications on systemic and local damage control. These studies are extremely diverse, and this is reflected in the variability of

Table 27.1 Summary of results from published studies reporting results of local and systemic damage control

Study	Date	Type of damage control	Patients/fractures	*N*	Infection rate (%)	Exchange time (days)
Van den Bossche [7]	1995	Systemic and local	Open femoral fracture	16	0.0	Mean 21
Yokoyama et al. [27]	2008	Systemic and local	Open tibial fractures	21	13	18.3 (planned), 52.1 (non-union in ex-fix)
Mody et al. [32]	2009	Systemic and local	Polytrauma, tibia/femur	58	40.0	Median 9 (4–414)
			All battlefield injuries			
Suzuki et al. [33]	2010	Systemic and local	Polytrauma, humerus	17	11.8	Mean 6.2 (2–14)
Nowotarski et al. [2]	2000	Systemic	Femur, polytrauma	59	1.8	7–49
Scalea et al. [3]	2000	Systemic	Femur, polytrauma	43	2.9	Mean 4, IQR 2–6
Taeger et al. [34]	2005	Systemic	Polytrauma	101	7.0	Mean 13.7, 3–46
			49 femur, 39 tibia, 25 pelvis, 22 upper limb			
Harwood et al. [21]	2006	Systemic	Polytrauma, femur	111	7.2	Mean 14, 1–61
Lavini et al. [30]	2007	Systemic	Polytrauma, femur	39	5.1	Group A – 5.6/7 (4–7)
						Group B – 4-6/12 – ex-fix removed – MRI/WC scan
						Group 3 – treated with EF
Stojiljkovic et al. [35]	2008	Systemic	Polytrauma, femur	24	5.5	Only 6 converted – 16 % of these infected
Mathieu et al. [29]	2011	Systemic	Polytrauma, long bones, pelvis	16	6.3	Mean 84 (60–90)
Metsemakers et al. [36]	2015	Systemic	Polytrauma, femur	87	0.0	5–15
Maurer et al. [26]	1989	Local	Open tibial fracture	24	29.2	Mean 52 (3–360)
Sirkin et al. [6]	2004	Local	Tibial pilon	56	5.4	Mean 12.7
Barei et al. [10]	2004	Local	Tibial plateau	83	8.4	Mean 9
Egol et al. [11]	2005	Local	Tibial plateau	49	6.1	
Yokoyama et al. [25]	2006	Local	Open tibial fracture	42	16.7	Mean 52.4 (2–135) and 114 (60–240)
Parekh et al. [37]	2008	Local	Tibia (36)/femur (16)	47	16.0	Mean 5 (1–23)
Oh et al. [38]	2011	Local	Periarticular lower limb	59	3.4	Mean 15.3 (4–81)
Laible et al. [16]	2012	Local	Tibial plateau	79	7.6	
Japjec et al. [39]	2013	Local	Tibial pilon	15	0.0	Mean 7
Roussignol et al. [31]	2014	Local	Tibial fracture	55	7.3	Mean 64 (14–365)
Shah et al. [17]	2014	Local	Tibial pilon (97)/ plateau (85)	182	13.7	Mean 20 (2–156)

their reported outcomes . Some include only or a very high proportion of open fractures. In others, the external fixators were left in place for very long periods in some patients. There are high proportions of multiply injured patients. Despite this, the majority report results with infection rates of less than 10% which is very reasonable in that context. Those with high infection rates usually have a peculiarity in design which explains this. For example, the study by Mody et al. contains exclusively battlefield casualties with a very high proportion of open blast injuries [32]. This factor was highly associated with deep infection, and despite this, overall results were good. Studies by Maurer and Yokoyama contain only high-grade open fractures and include patients in whom the external fixators were left in place for extremely long periods; in some of these the intention was to treat by external fixation definitively, the exchange only occurring when the fracture failed to unite [25, 26].

Determining the optimum time for exchange requires balance of the competing advantages of early exchange to reduce the risk of pin sites becoming contaminated against delaying exchange whilst the patient's systemic physiology and local soft tissues recover. Clearly patients should never be placed at undue risk of life-threatening complication by undertaking complex secondary surgery too soon. Similarly, performing definitive internal fixation in a local damage control situation before the soft tissues have adequately recovered will likely increase the risk of infection more than waiting in the presence of external fixation pins. It is therefore generally recommended to perform definitive surgery as early as possible provided that the patient's systemic state and soft tissues have adequately recovered. It is important to be critical in this decision-making and avoid performing secondary surgery too early for convenience's sake.

Evidence regarding the timing of exchange and risk of infection is difficult to interpret. Whilst a number of studies examining such patient groups have been published, these are very diverse and retrospective in nature. Some series have patients in whom the external fixators were in place for many months. In some, internal fixation was only applied if the fracture failed to unite in the external fixator. Few studies have specifically addressed the timing of exchange specifically, and no randomised studies exist. Given the individualised decision-making that is required in these patients, a study randomising the timing of exchange would be very difficult to design ethically. Harwood et al. examined a group of polytrauma patients with femoral shaft fracture treated either by primary intramedullary nailing (81 fractures) or initial damage control (111 fractures) followed by staged internal fixation [21]. The mean time of exchange procedure was 14 days (range of 1–61). Despite the fact that the damage control group had more severe injuries, overall there were no significant differences in deep infection rates, with 7.2% in the damage control group and 6.2% in the primary nailing group. Further examination of the data found that whilst more patients had positive microbiology swabs for contaminated pin sites if external fixators were in place for more than 14 days, this did not result in higher rates of clinically important infection in these patients. It would seem likely that this simply represented the fact that there was more time to send swabs in patients with external fixators in place for longer, given that no specific protocol for timing of microbiological testing was employed. A multivariate analysis controlling for other factors did however reveal a weak association between external fixation for more than 14 days and infective complications. Other studies have examined infection rates based upon the time between external fixator application and exchange to internal fixation. The majority found no relationship between these variables [6, 16, 24–26, 31, 37]. Only a few studies have found a positive relationship between duration of fixation and subsequent deep infection [27]. In interpreting these results, it is important to consider the limitations of the studies in this outcome. The decision on when to exchange in these studies was almost certainly taken individually in each case, based on previous experience. Furthermore, various protocols were employed, including opting for definitive external fixation in high-risk patients and excluding patients from the studies with infected pin sites.

Some authors have advocated use of specific criteria to determine whether exchange is appropriate including the absence of clinical infection, the absence of previously infected pin sites, normal inflammatory markers and no evidence of infection on MRI or radio-labelled nuclear medicine examinations [26, 30, 31]. Our group have not found these investigations to be routinely necessary, preferring instead to exchange in a short time frame, as soon as the patient has physiologically recovered, where possible within 2 weeks. In selected cases, where the patient's local or systemic physiology is felt insufficiently improved within this time frame, such an approach might be helpful. It may be that in these situations an alternate definitive fixation method should be employed as detailed above.

Overall, it would therefore seem logical to undertake exchange from temporary to definitive fixation as soon as is possible within constraints detailed elsewhere. When this is delayed, very careful consideration should be given to the chosen mode of fixation.

27.7 Technique for Exchange

Various methods of exchange have been described. Whilst it may occasionally seem advisable to remove the fixator for a period prior to secondary procedures and give the external fixation pin sites time to heal, on the whole this is not advocated. In most cases, this would remove many of the benefits of adopting a damage control approach approach in the first place. The soft tissues are usually better served by the stability of a correctly applied external fixator rather than removing this and applying a splint. This also facilitates ongoing inspection and treatment of the soft tissues. As detailed above, it is usually better to revise the fixator to render it more stable and move offending pins rather than remove the fixator entirely. Clinical results suggest that very acceptable rates of infective complication can be achieved using such an approach.

Most authors advocate removal of the fixator in the operating theatre following anaesthesia, immediately before internal fixation with curettage and/or excision of the pin sites. Protocols have been described though these are not based on specific evidence; an example is detailed as follows [21]:

1. The external fixator is removed in the anaesthetic room:
   (a) External fixator rods and clamps are removed leaving only the pins in place.
   (b) These are prepped and removed under aseptic conditions.
   (c) Pin tracks are excised to subcutaneous fat and superficially irrigated with sterile saline; avoid introducing this under pressure driving potential contaminants deeper.
   (d) Some surgeons choose to leave pin sites open, covering these with waterproof sterile dressings at this stage and leaving these in place during the definitive surgery, prepping over them.
   (e) Some surgeons choose to close pin sites following excision, potentially at this stage or following the definitive procedure.
2. The patient is taken into theatre and then completely re-draped; all previously used surgical equipment is discarded:
   (a) Surgical team re-scrub and re-gown.
   (b) Some surgeons choose to over-drill pin sites, either at first or second prep, particularly when the fixator has been in place for more than 14 days.
   (c) Surgery is undertaken in a standard manner; incisions should be placed away from previous pin sites.

In a study of patients treated for tibial fracture with local damage control, Rossignol et al. reported results after sending reaming debris for culture in every case. Twenty-two percent of these samples were positive on microbiological culture; none of these patients, who received targeted antibiotics for 6–8 weeks, went on to develop deep infection following intramedullary nailing. This would seem a reasonable approach, though it is not possible to comment upon whether it prevented clinical infection in this study.

Conclusions

Damage control external fixation requires careful decision-making and meticulous planning from the outset. This may be challenging in the context of major trauma. Consideration of the likely definitive treatment method is important in deciding on pin placement. Surgeons involved in such cases should have a good understanding and experience of monolateral external fixation techniques in order to allow versatility in frame application and appropriate surgical technique. Pin-site care should begin during pin insertion and be carefully maintained until exchange to definitive fixation. Pin-site infections should be aggressively treated and the fixator examined for problems, particularly instability, and revised as necessary. Exchange procedures should be undertaken as soon as the patient is ready, within 2 weeks if possible. Evidence on timing of exchange does not however appear to demonstrate a large increase in the risk of deep infection if exchange is undertaken after this period. Careful individual decision-making is critical in these patients if infective complications are to be minimised. If doubts remain about the suitability of a patient's soft tissues for internal fixation, alternative methods including definitive external fixation should be considered.

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Complications Status Post Damage Control for the General Surgeon

28

Anastasia Kunac and David H. Livingston

28.1 Damage Control for the General Surgeon

Damage control laparotomy is widely practiced as a temporizing measure to salvage surgical patients whose physiologic derangements do not permit the completion of an intended operation. The more complete description of the development and evaluation of damage control laparotomy following severe trauma is outlined in Chaps. 1 and 2. However, it is worth briefly reviewing the genesis of the procedure in light of developing of early and late complications. The technique was popularized in the early 1980s by Stone and associates for surgical treatment of coagulopathic trauma patients. Stone described the technique of packing, control of hemorrhage, bowel resection without anastomosis or stoma formation to control contamination, and biliary or pancreatic drainage if necessary. Patients returned to the operating room for definitive operative repair of injuries following correction of coagulopathy. As the widespread use of non-warfarin anticoagulants, many with no or complicated reversal agents, is increasingly encountered in patients with emergent surgical conditions, the need for damage control for uncontrolled coagulopathy will not be an uncommon event.

The term "damage control laparotomy" was coined by Rotondo et al. in 1993 who reported a salvage rate of greater than 70% in patients with major abdominal vascular injuries and concomitant shock, acidosis, and hypothermia. This lethal triad of core temperature <35 °C, disruption of hemostatic mechanisms, and metabolic acidosis as a marker of impaired oxygen delivery may occur not only from hemorrhagic shock in the setting of acute traumatic injury but most certainly can apply to general surgery patients suffering from profound shock whether the source is hemorrhage or sepsis. The concept of abbreviated laparotomy or "damage control" in critically ill general surgery was first supported in a study by Finlay et al. in 2004. Finlay demonstrates a lower than expected mortality rate for surgical conditions such as peritonitis, infarcted bowel, severe pancreatitis, ruptured abdominal aortic aneurysm, and postoperative/post-procedural hemorrhage. The observed mortality rate was 7% utilizing damage control techniques, as compared to predicted 50–65% mortality utilizing the Physiological and Operative Severity Score for enumeration of Mortality and morbidity (POSSUM) scoring system.

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As more general surgeons are employing use of the open abdomen, additional literature is available to describe when these techniques should be applied and what the anticipated complications may be. A recent prospective observational study by Bruns et al. describes indications for nontrauma open abdomens in 338 patients with abdominal pathology managed by general surgeons with laparotomies performed over 1 year (June 2013–June 2014). Bruns describes demographics, surgical site complications, and mortality in these patients. The patient population included primary laparotomies performed by the acute care emergency surgery team, and in this patient population, 28% were left with open abdomens. Indications included "damage control" (acidosis, coagulopathy, or hypothermia), planned second look, excessive contamination, decompression, and combinations of the aforementioned. Common complications include surgical site infection (14%), fascial dehiscence (10%), and fistula formation (10%). The in-hospital mortality of patients with an open abdomen was 30% indicating the severity of illness and physiologic derangements and organ failure present for emergency surgical patients managed in this manner. These data suggest that management of general surgical conditions with an open abdomen following index operation is becoming more common, and familiarity with associated techniques and anticipated complications is prudent.

Over the last 10 years, a phased approach to damage control surgery in both trauma and nontraumatic abdominal emergencies has been advocated. As outlined in earlier chapters, the initial phase focuses on goal-directed resuscitation, hemostasis, and source control in the setting of sepsis. Phase II requires correction of hypothermia, coagulopathy, and acidosis in the ICU. This is followed by definitive repair of all injuries/pathology and ultimately closure of the abdominal wall. While numerous authors have demonstrated that utilization of these techniques results in successful patient salvage, few studies have looked at the pitfalls of damage control techniques. This chapter will focus on complications post damage control as they pertain to the general surgeon.

28.2 Intra-abdominal Hypertension and Abdominal Compartment Syndrome

Perhaps the most common reason for which a general surgeon might utilize damage control laparotomy is profound intra-abdominal sepsis with or without septic shock. Under such circumstances, the focus would be source control followed by restoration of normal physiologic and homeostatic parameters with consideration given to blood and fluid resuscitation. Particularly when fluid resuscitation is substantial, development of primary or secondary abdominal compartment syndrome is possible. Primary abdominal compartment syndrome (ACS) can occur in the preoperative or pre-intervention time period where the patient is undergoing aggressive fluid resuscitation. Hypotension, worsening pulmonary mechanics, and decreased urine output can all be mistakenly attributed to septic shock. Serial physical examination is the key to ensure that ACS is not developing. Appropriate blood and fluid resuscitation is discussed in detail by *Dr. Cotton in* Chap. 6, and *Dr. Ivatury* has outlined abdominal compartment syndrome in Chap. 9.

Secondary intra-abdominal hypertension and abdominal compartment syndrome may develop even with an open abdomen following initial damage control surgery. In the initial abbreviated surgery, common abdominal dressings utilized include sterile plastic sheets (such as sterile x-ray cassette covers) or more recently sophisticated vacuum-assisted closure (VAC) dressings. Intra-abdominal hypertension may develop irrespective of the dressing utilized on an open abdomen. If the abdomen is packed too tightly, towel packs are too stiff or a VAC dressing too

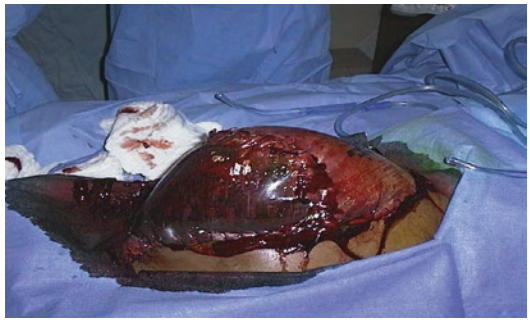

Fig. 28.1 "Silo"-type dressing sewn to the skin of the abdominal wall in setting of significant bowel edema. This was the second closure in this patient as he developed secondary ACS following ongoing resuscitation. As can be observed, it required two 3 L IV bags to achieve closure. Lap pads are placed in the "lumen" of the IV bags to prevent sutures from cutting through the plastic

tight, the patient may not tolerate even a temporary closure. A number of clinical parameters may be followed intraoperatively and postoperatively to monitor for signs of intra-abdominal hypertension.

Intraoperatively, during the initial surgery for control of hemorrhage or source control for sepsis, peak pulmonary pressures can be monitored. The patient is optimally sedated and paralyzed at this time. If upon application of the temporary abdominal closure dressing the peak pulmonary pressures rise to greater than 30 mmHg, particularly if pressures were substantially lower prior to application of the dressing, one must consider the possibility that the dressing is too tight. Some patients, particularly those in septic shock, have such profound bowel edema as to require a "silo"-type dressing (see Fig. 28.1). It is our practice to avoid VAC-type dressings in these patients if the bowel protrudes above the anterior abdomen at initial exploration. These patients are much better treated with the application of a loose silo-type dressing to avoid unplanned and often emergent bedside re-exploration as resuscitation with fluid and blood products is likely to continue in the intensive care unit postoperatively.

Ongoing monitoring of pulmonary pressures as well as bladder pressure is appropriate in the immediate postoperative period. The procedure we employ for measurement of bladder pressure is outlined in detail in Fig. 28.2. Utilizing aseptic technique, a pressure transducer is set up utilizing the specimen port from a Foley catheter collection system. The urine collection tubing is clamped distal to the specimen port, and via the system depicted in the figure, 25 mL of sterile saline is instilled into the patient's bladder. An intra-abdominal pressure measurement is then transduced. After the measurement is taken, one must ensure that the clamp is removed from the urinary drainage tubing. Consideration should be given to removal of the dressing and replacement with a looser dressing for abdominal pressure measurements >20 mmHg. While we recognize that this value is below the classic threshold for abdominal compartment syndrome, we strongly believe that ongoing intra-abdominal hypertension in this setting results in prolonged visceral ischemia which perpetuates the SIRS response.

Alternately, a "quick and dirty" bladder pressure measurement can be executed by laying the patient supine and raising the Foley catheter drainage system tubing vertically over the patient's pelvis. The height of the fluid column from the pubic symphysis can then be measured in centimeters (1.00 cm H_2O=0.74 mmHg). A measurement of >27 cm H_2O=>20 mmHg and warrants concern for intra-abdominal hypertension.

28.3 Rebleeding

Another common complication is bleeding following initial operative intervention. As mentioned earlier, surgical procedures such as emergent repair of a ruptured abdominal aortic aneurysm exploration for postoperative or postprocedural bleeding or procedures in the already coagulopathic patient may lend themselves to

Equipment Needed
Indwelling urinary catheter with drainage bag and needleless specimen port
Disinfectant (povidone-iodine solution, chlorhexidine, or alcohol)
Standard intravenous setup with 500 mL of normal saline
20 mL syringe
Transducer and pressure tubing
Clamp for urinary drainage tubing
Setup
Using aseptic technique, set up the manifold system, intravenous container of normal saline, the 20 mL syringe, and the pressure tubing and transducer as shown in the figure below.
Open the intravenous fluid and flush the system.
Connect the pressure system setup to the needleless specimen port.
To obtain a measurement, clamp the urinary drainage tubing distal to the specimen port.
Aspirate 25 mL of the normal saline.
Instill 25 mL of normal saline into the urinary bladder and obtain a pressure reading.
AFTER THE MEASUREMENT, ENSURE THAT THE CLAMP IS REMOVED FROM THE TUBING.

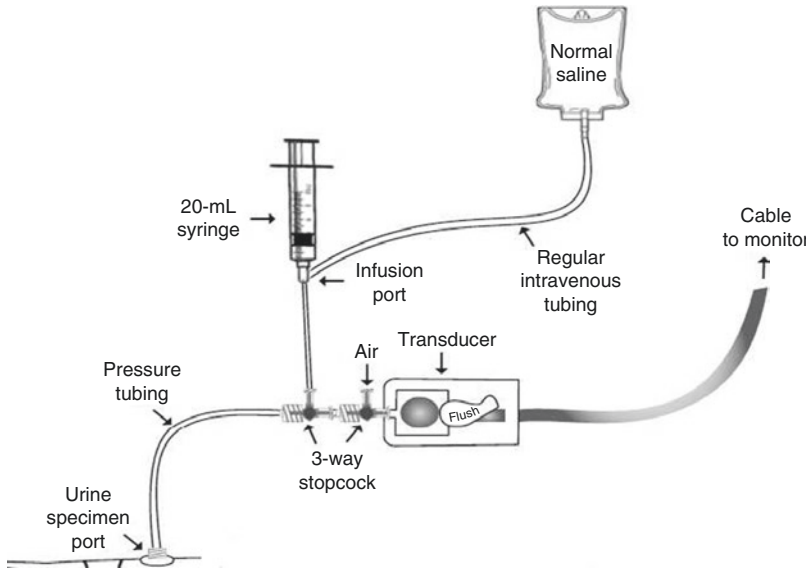

Fig. 28.2 How to measure bladder pressure (Adapted from *Critical Care Nurse* 2012; 32:19–31)

damage control laparotomy. Rebleeding following initial damage control is either from coagulopathy associated with acidosis and hypothermia and/or circulating undetected anticoagulants or "surgical" bleeding. In the former instance, correction of temperature, acidosis, and coagulation factors are what is required, while the latter mandates early reoperation. In the early postoperative period, this can be challenging to determine as ongoing surgical bleeding will further compound coagulopathy and can make correction of hypothermia and acidosis impossible. Advanced coagulation measurements (TEG, ROTEM) may be useful in helping identify coagulation defects but should not be used to determine the need for re-exploration. Evidence of early blood loss from negative pressure canisters and a need for continuous blood and factor replacement to maintain blood pressure are definite indications for emergent reoperation. Worsening acidosis as measured by base deficit or lactate, the need for the institution of vasopressor therapy to maintain blood pressure, and dropping hemoglobin levels despite blood administration are also signs that

warrant urgent re-exploration. Often, even without identifying a surgical source of bleeding, removal of clotted and non-clotted blood and repacking with fresh lap pads can result in marked improvements in hemostasis.

28.4 Ischemia

Acute mesenteric ischemia is a condition for which initial damage control and planned second-look operation is appropriate. Ischemia of the bowel secondary to a low flow state (nonocclusive mesenteric ischemia) is also a potential complication in patients who are so profoundly ill as to require damage control laparotomy. Each of these warrants careful consideration.

First, patients who undergo abdominal exploration for acute infarction of the small bowel may benefit from a planned open abdomen and early second look. A planned open abdomen decreases the risk of developing intra-abdominal hypertension/abdominal compartment syndrome as the patient is resuscitated. Necrotic bowel can be resected without performing anastomoses using bowel of questionable integrity at initial operation. Progressive bowel ischemia is certainly a potential problem with this particular disease process; a clear plastic dressing may be utilized as a temporary abdominal closure allowing a "window" into the peritoneal cavity should the patient suffer progression of their bowel ischemia. Finally, the planned second look allows early reevaluation of segments of the bowel with questionable viability and potential preservation of intestinal segments that may have otherwise been resected and avoidance of possible short gut.

In performing damage control for any indication, one must also consider that intestinal ischemia may also occur secondary to the physiologic state of the patient. Diminished splanchnic circulation secondary to profound shock is common and may be exacerbated by vasopressor use. The only certain treatment is correction of the underlying arterial hypoperfusion—this may be by virtue of fluid and blood product infusion in cases of hypovolemic/hemorrhagic shock or source control and antibiotics in the septic patient. Unexplained and worsening metabolic acidosis may lead to suspicion of nonocclusive mesenteric ischemia. Alternately, the intestine may be visible via a temporary abdominal dressing and could appear to be poorly perfused. If bowel ischemia is suspected for any reason, the underlying cause must be reversed.

28.5 Anastomotic Leaks and Fistulae

Perhaps the most feared complications following DCL are anastomotic leaks and fistulae. Whether or not to perform anastomoses and how to construct them certainly plays a role in development of anastomotic leaks. When small bowel is resected, with the exception of the distal ileum, creation of anastomosis to restore intestinal continuity is almost always necessary. In accordance with usual surgical tenets, anastomoses are constructed such that they should be tension-free with good blood supply. While there remains no definitive answer as to whether hand sewn or stapled anastomosis is superior in this setting, in our experience the marked bowel edema that is almost uniformly present makes hand-sewn anastomosis our construction of choice. More importantly and not often taken into account, following elective general surgery is where the anastomosis lies within the peritoneal cavity as it relates to the abdominal wound. It is imperative to "bury" the anastomosis behind the abdominal wall or other viscera. An anastomosis that ends up in the center of the wound, particularly when the abdomen cannot be closed, is a "fistula in waiting." For this reason, we strongly avoid any midline anastomosis such as gastrojejunostomies or those in the transverse colon in this patient population.

Whether or not to perform a colonic anastomosis or create an ileostomy or colostomy should be given careful thought. Anjaria et al. studied trauma patients with colonic injuries who underwent a single laparotomy versus DCL and subsequent re-exploration. Anjaria found

that repair or delayed anastomosis following DCL is feasible, with complication rates similar to single laparotomy when successful fascial closure is completed on the first post-DCL reoperation. However, if fascial closure is not possible on the second operation, patients should be treated with a stoma because there is an eightfold increase in the incidence of anastomotic leak. This suggests that there is a single opportunity to establish colonic continuity after DCL. For those patients who require placement of a colostomy, we strongly advocate placement lateral to the rectus sheath. Retaining the fascial and muscular integrity of the rectus abdominis is important as these patients have significant wound complications as well as the potential need for later abdominal wall reconstruction with component separation.

Fistulae formation following damage control laparotomy has also been studied. The American Association for the Surgery of Trauma established an open abdomen registry and found that large bowel resection, large-volume fluid resuscitation, and an increasing number of abdominal re-explorations were statistically significant predictors of enterocutaneous fistula, entero-atmospheric fistula, or intra-abdominal sepsis in patients with an open abdomen after damage control laparotomy. For those who require multiple re-explorations, fistulae are fairly common complications with fistula rates ranging from 5% to 25%.

Management of the entero-atmospheric fistula is one of the most challenging complications following damage control surgery as these often occur within an open granulating wound. It is also the reason that there is a significant emphasis on techniques or approaches to prevent or minimize this complication. However, once a patient has developed a fistula, both the patient and the surgeon must realize that they are in for a long and complicated treatment course as there are no quick fixes to this problem. Attempts to suture, glue, or otherwise close the fistula are destined to fail and are likely to make the opening larger. Even the use of tubes and catheters to attempt to capture the effluent is problematic and prone to almost certain leakage as the enteric contents are often too thick to be captured. At this juncture, it is also almost impossible to close the abdominal wall any further, and surgeons should realize that a late abdominal wall reconstruction will be needed. Fortunately, given patience on the part of the surgeon, time, nutrition, and a bit of ingenuity, almost all fistulae can be treated successfully.

As stated above, the goal of entero-atmospheric fistula management is control of effluent and good nutrition. As the opening in the bowel is to the outside world, the fistula opening will always have a lower pressure than the distal intestine and thus will most commonly remain open. One exception are those fistulae at the edge of the open abdomen-native abdominal wall where wound contraction may increase the pressure gradient in favor of fistula closure. In the early stage of management, we believe and advocate achieving as much coverage over the remaining viscera as possible. The easiest and most reliable way to achieve that goal is through the use of thin split-thickness skin grafts while making allowance to capture as best as possible the effluent from the fistula. We eschew the traditional 3–5-day period before uncovering the graft as this is associated with a 100% graft failure. Rather we nurse these grafted wounds open which prevents effluent from building up under the new grafts. While some graft loss is inevitable, we often achieve $\geq$80% coverage. As the skin graft heals, the wound contracts and allows for eventual management of fistula effluent with a traditional colostomy appliance. It is during this time period that a fistula at the edge of the wound may actually close.

Fistulae that are away from the edge of the wound or those that have become “stomas” always require eventual surgical intervention. These secondary operative closures are always challenging but can be made more so by an overly aggressive timeline. The longer one can wait and temporize, the less hostile the abdomen will be and the less likely for creation of inadvertent enterotomies in the dissection and closure of the fistula.

In our experience it is 3 months at the earliest following closure or healing of all other

wounds before reoperation is even considered. While not necessarily completely nutritionally replete, patients must be demonstrating markedly improved nutritional parameters (prealbumin, transferrin, albumin) prior to embarking on closure. We advocate nutritional optimization via enteral feeds whenever possible. Patients with distal fistulae that have a significant length of functional small bowel, especially in cases where eating does not significantly increase fistula output, should be allowed to eat (Fig. 28.3). In patients with fistulae are tolerating regular diet and can manage any fluid losses, one can wait even longer for repair until the patient is nutritionally optimized. Some patients with proximal fistula, complete distal obstruction, or other issues that require significant supplemental fluid and/or TPN require intervention as soon as it is feasible. Quite often these patients do not have the resources and ability to be treated at home or at a skilled nursing facility and therefore remain in the acute care hospital. For these patients in particular, surgeons have to resist the pressure and temptation to operate prematurely.

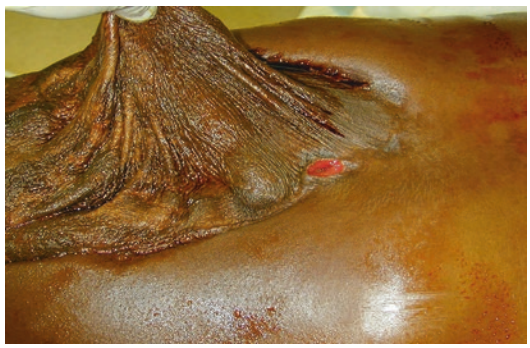

Fig. 28.3 A "simple" mid-small bowel fistula at the edge of an open abdomen treated with a skin graft. The patient has been home eating a regular diet with a stoma bag placed over the fistula. He is now about 9 months and the graft can be easily lifted away from underlying viscera. In this stage, the fistula was easily dissected free and closed. The skin graft was sewn to the edge of the wound, and the patient underwent formal abdominal wall reconstruction in a clean field, 6 months following fistula closure

28.6 Inability to Close the Abdomen

Following DCL, there is a small group of patients where achieving abdominal fascial closure seems impossible. This may be due to loss of domain in cases where the patient had a destructive lesion of their abdominal wall or an infection requiring significant debridement or may be due to retraction of the fascia. Others have retroperitoneal processes (hemorrhage or pancreatitis) that decrease the intra-abdominal domain.

A number of techniques have been discussed for the early closure and late reconstruction of the abdominal wall—many of these are discussed in detail elsewhere (Chaps. 17 and 19). The most common approach is delayed primary closure. If delayed closure is possible on the first reoperation, this is preferred. Even sequential closure of the fascia over two or three operations is acceptable. Despite success of closing many damage control trauma laparotomies, the same success has not been necessarily achieved in the setting of sepsis or other acute care surgical disasters. Given the risk of fistula formation if the abdomen remains open, we must accept that other techniques may be employed to achieve closure over the abdominal contents. In patients who have the omentum covering the abdominal contents, the concern for the development of a fistula is much less, and thus more options for closure are available. These alternate techniques come with an accepted need to return at a later date to offer definitive repair of the intentional hernia.

In patients who have a wide defect that has not been amenable to sequential secondary closure and insufficient omentum that leads to exposed bowel, a split-thickness skin graft may be applied directly over the bowel. There is no need for granulation tissue to be present, and in fact the longer one allows for granulation tissue to occur, the more likely that a fistula will occur. In patients whose viscera are not "stuck," we routinely sew a Vicryl mesh to the fascial edge so that the mesh overlies the intestine. The mesh remains in place for about 3 days allowing the bowel to become fixed within the peritoneal

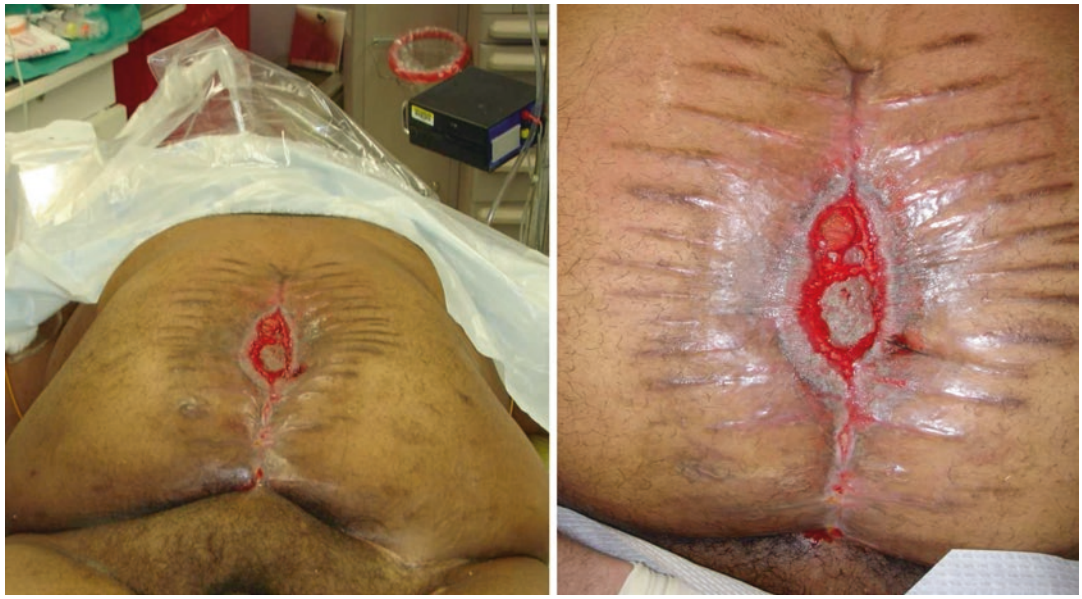

Fig. 28.4 Patient who developed complex entero-atmospheric fistulae during ongoing care and secondary closure of an open abdomen. Fistula (top of granulating wound; right photograph, incision) has proximal high output and the distal limb could not be intubated to deliver enteral feeds. Patient managed with TPN until fistula closure when nutritional replete. Wounds (except for fistula) healed at 3 months following last operation

cavity as many of the modern plastic dressings prevent adherence of the bowel to the abdominal wall. Remember that at the time of skin grafting, minimal movement is ideal, and the bowel that is adherent is unlikely to eviscerate from beneath a graft. The Vicryl mesh is easily removed, and thin skin grafts are harvested from available donor sites, most commonly the anterior thigh, and applied directly over the bowel. Commonly the proteins on the bowel will allow the skin graft to "stick" without any other adhesive necessary. In other cases we have employed other tissue adhesives in small areas to prevent skin graft movement. We routinely use nonadherent gauze followed by the hydrophilic sponge and then a commercial negative pressure dressing to minimize shear forces. The graft is routinely unveiled on post-op day 3 or 4. Skin grafting over bowel results in a giant ventral hernia but usually abrogates fistula formation. Frankly we believe that fixing a hernia without a fistula is far preferred over dealing with both a late hernia and an entero-atmospheric fistula! Six to 12 months following skin grafting, the graft is no longer adhered to the bowel and can be easily lifted away from the underlying viscera (see Fig. 28.3). At this point, the patient is a candidate for abdominal wall reconstruction.

Ideally, abdominal wall reconstruction should be performed independent of fistula repair or stoma reversal. Fistulae should be repaired prior to planned hernia repairs. Figure 28.4 depicts a patient with a complicated midline abdominal wound, multiple fistulae, and an underlying hernia. This example reflects a patient in whom nutritional status has been optimized, and timing is appropriate for definitive repair of his abdominal wall. Given the fistulae, this is accomplished in two stages. The first operation involves tedious adhesiolysis along with isolation and resection of the fistulae (Fig. 28.5). The final operation will be repair of the hernia—thus, if mesh is required, this can be accomplished as a "clean" case. Following similar logic, intentional stomas should also be reversed prior to abdominal wall reconstruction.

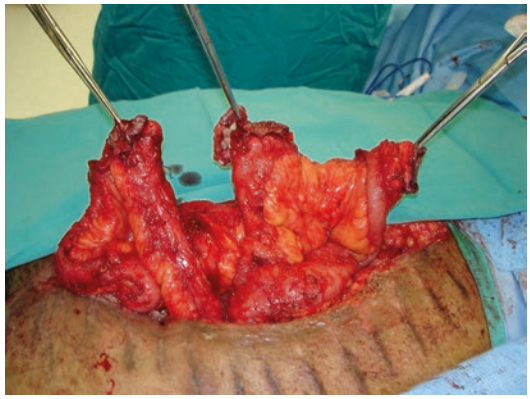

Fig. 28.5 Intraoperative appearance of following isolation of two distinct entero-atmospheric fistulae from patient in Fig. 28.4. The reason for the inability to intubate the distal limb is now obvious as it was an obstructed blind limb between the two fistulae. To achieve successful outcomes in closing these fistulae, careful and complete dissection of the viscera is required to ensure all components are identified

28.7 Setting the Stage for a Good Outcome

Damage control laparotomy has applications for the general surgeon and specifically has been shown to be efficacious for laparotomies performed for conditions such as peritonitis, infarcted bowel, severe pancreatitis, ruptured abdominal aortic aneurysm, and postoperative/post-procedural hemorrhage. In fact, as the use of massive transfusion protocols emphasizing early blood loss and factors with minimal crystalloid resuscitation has decreased the use and need of damage control/open abdomens following trauma, it is the general surgeon treating intra-abdominal sepsis and other acute surgical catastrophes that are performing this technique most commonly.

Successful interventions rely on correcting derangements in coagulation, acid, base, and body temperature following DCL and before reoperation. When employing DCL, one must be aware that there are a number of possible complications. While DCL decreases the likelihood of abdominal compartment syndrome, intra-abdominal hypertension may develop even with a temporary abdominal closure from a dressing applied too tightly. One must also consider that post-op patients may suffer ongoing hemorrhage and/or bowel ischemia from poor gut perfusion.

Enterocutaneous fistulae, entero-atmospheric fistulae, and intra-abdominal sepsis from anastomotic leaks are fairly common complications of the open abdomen. Colonic diversion should be considered if fascial closure cannot be achieved at the first re-exploration following DCL. Further, skin coverage over the abdomen should be sought as soon as possible—the surgeon must be willing to admit when timely primary fascial closure is not possible and should move to an alternate plan such as skin grafting under such circumstances. There is no doubt that this group of patients will require more thought and long-term planning and will tax a surgeon's ability and ingenuity. However, adherence to the principles and concepts outlined in this chapter and elsewhere will result in excellent outcomes in this critically ill and injured population.

Suggested Reading

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DC2 Outcomes of Damage Control Surgery: General

29

Ben Kautza and Jason Sperry

29.1 Background

Trauma is one of the leading causes of death in individuals 1–44 years of age. Significant abdominal trauma, whether penetrating or blunt, is a major factor in determining outcomes. During initial resuscitative and operative procedures, the presence of acidosis, hypothermia, and coagulopathy has been shown to be associated with higher mortality (Mikhail 1999; Moore 1996). In individuals with major abdominal trauma development of coagulopathy, metabolic acidosis, hemodynamic instability, infections, and pulmonary complications have been shown to significantly increase morbidity and mortality (Moore 1998). Since the early 1980s operative techniques such as damage control laparotomy (DCL) or staged laparotomies have evolved to help reduce the morbidity and mortality associated with severe abdominal trauma. DCL has shown benefit in case series and smaller studies; no definitive randomized control trial exists comparing DCL to immediate and definitive repair in the treatment of severe abdominal trauma. Outcomes of DCL discussed below will help delineate the data known to date and highlight the future direction of therapy and research needed.

29.2 Damage Control Laparotomy

Damage control is characterized by initial resuscitation, brief laparotomy when necessary, continued intensive care unit resuscitation followed by planned reoperation. The operative approach focuses on arresting hemorrhage (use of vascular clamps, packing, etc.) for vascular or parenchymal injuries, the avoidance of further peritoneal contamination with stapling of hollow viscus injuries (often left in discontinuity), and in the final stages leaving the abdomen open to avoid abdominal compartment syndrome (ACS). ACS refers to impaired organ function attributed to increased intra-abdominal pressure (Richardson 1976). The main aim of damage control laparotomy is to reestablish physiologic stability and avoid the development or worsening of acidosis, coagulopathy, and hypothermia (Cirrochi 2013). This technique aims to avoid prolonged surgical intervention and potentially complex definitive repairs in an unstable patient. A potential disadvantage is the need for further operative intervention the potentially increase in morbidity associated with open abdomens (OAs).

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29.3 Early Experience and Outcomes in Damage Control Laparotomies

The contemporary use of DCL began in the early 1980s. A study from Emory University in 1983 first described the use of aborted laparotomy with intra-abdominal pack tamponade in the treatment of 17 patients who required laparotomy for abdominal trauma (Stone 1983). The technique described involved controlling major hemorrhage with vessel ligation or repair and purse-string closure of hollow viscus injury and packing of the abdomen with abdominal closure under tension. These patients were then reexplored at 15–69 h later with 11 patients deemed to have a "lethal coagulopathy" surviving (Stone 1983). Compared with matched controls undergoing the then contemporary standard of care, those undergoing aborted laparotomies with packing had a mortality rate of 35% vs 93%, respectively. Interestingly, considering todays knowledge of massive transfusion protocols, there were no significant differences in the volume of blood used between the groups. However significantly greater volumes of plasma, red cell mass, and platelets were required in the group who underwent initial definitive repair (Stone 1983).

A study out of the University of Pennsylvania in the early 1990s first defined "damage control" as the initial control of hemorrhage and contamination followed by packing and rapid closure (Rontondo 1993). This series investigated 46 patients who required laparotomy for penetrating injuries who required greater than ten units of red blood cells for exsanguination. Overall survival between those undergoing DCL and standard of care was not significantly different. However in a subgroup of patients who had major vascular injury as well as two or more visceral injuries, DCL showed significantly increased survival (77% vs 11%) when compared to initial definitive operation (Rotondo 1993). This study was also able to validate that postoperative resuscitation after DCL was able to achieve correction in coagulopathy and acid-base balance as well as restore normothermia prior to planned reoperation. Results showing in penetrating abdominal trauma with hemorrhage with vascular and hollow viscus injuries, DCL contributes to increased survival.

The use of DCL showed increase survival and offered a viable operative strategy for dealing with severely injured and exsanguinating trauma patient. Questions on optimal timing of definitive operative intervention as well as the recognizing morbidity associated with the use of DCL were investigated. The development of ACS in these patients was identified, and strategies to recognize and predict those at risk were questioned.

A series out of Vanderbilt University consisting of 107 patients undergoing DCL investigated timing of definitive operation as well as development of ACS (Morris 1993). In this series survival to discharge was 74.1% with development of ACS in 15%. Development of ACS increased mortality significantly (62.5%). Echoing Rotondo et al. (1993), reversal of hypothermia, coagulopathy, and acidosis was shown to be correctable early after initial laparotomy and usually within 36 h. The majority of patients were able to have definitive surgery and packing removal within 24–36 h. Interestingly abdominal fascial closure was achieved in 90% of cases (Morris 1993). This series showed the decision to proceed with a DCL should be made early and the presence of hypothermia (<35 °C), acidosis (base deficit <−14 mmol/l), and observation of medical bleeding were indications to do such (Morris 1993).

The potential morbidity of DCL and complications such as ACS led to novel strategies to recognize these early as well as novel treatment strategies. Initial aggressive resuscitation of trauma patients in extremis undergoing DCL can lead to substantial bowel edema making, at times, even skin closure challenging. Temporary abdominal closure after DCL early on was typically described and achieved with either suture to achieve tension (Stone 1983) or towel clip skin closure (Feliciano 1988; Morris 1993; Burch 1992). Burch et al. (1992) were one of the first to describe the use of a synthetic material to achieve coverage of abdominal contents after DCL. Synthetic material, typically in the form of modified intravenous bags, was sewn to the skin

to achieve coverage and temporary abdominal closure in these cases (Burch 1992; Rotondo 1993; Hirschberg 1994; Raeburn 2001). The use of synthetic material to achieve coverage does not reduce the risk of ACS development (Raeburn 2001). The use of these strategies led to patients with open abdomens whose abdominal fascia could not be closed. The use of absorbable mesh sewn to fascia to achieve abdominal closure after definitive operative intervention was described (Morris 1993). Not until later use and development of negative-pressure wound therapy (NPWT) systems was ACS risk reduced and treatment of open abdomens facilitated (Barker 2000; Subramonia 2009; Godat 2013; Chiara 2016).

29.4 Contemporary Use of Damage Control Laparotomies, the Open Abdomen and Outcomes

Damage control operative strategies occur in 10–15% of current trauma laparotomies (Demetriades 2014). As mentioned previously the use of DCL involves truncated initial operation with a temporary abdominal closure strategy; correction of physiologic derangements followed by definitive operative intervention and abdominal closure. When DCL strategies are undertaken, typically the abdomen is left open with some type of temporary abdominal closure technique in the effort to avoid intra-abdominal hypertension and the development of ACS. This strategy leads to the relatively common condition of the open abdomen (OA) in the care of the severely injured trauma patient. As noted earlier, the decision to perform a DCL and the subsequent advancements in trauma resuscitation have greatly increased overall survival in this cohort. However, the care and management of OAs has needed to focus on strategies to allow for primary fascial closure, decrease enterocutaneous fistula (ECF) formation, and decrease overall morbidity.

Temporary abdominal closure strategies have become instrumental in the treatment of severely truncal injured trauma patient in conjunction with DCL. Early experience with the use of intravenous bag closures (e.g., Bogota bags) did not decrease the development of ACS or increase PFC rates (Raeburn 2001). Barker et al. (2000) describe the ideal temporary abdominal wound closure as one that provides containment of abdominal contents, protects abdominal contents from mechanical injury, prevents contamination of the peritoneal cavity, controls peritoneal fluid, and can be rapidly applied and relatively inexpensive. The development of commercially available negative-pressure wound therapy (NPWT) systems have become widely developed and meet majority of ideals described by Barker et al. (2000).

The recognized success of DCL use has led to the increase in OAs, not only in trauma patients, but other surgical specialties as well. Care of an OA patient still remains a great challenge and sparks debate, despite improved surgical interventions and advancements in ICU care and nutritional support (Al-Khoury 2008; Becker 2007). Challenges in the OA patients' care have spurred the development of national and international committees and organizations dedicated to addressing these issues. Techniques for temporary abdominal closure, management of OA complications, and challenges as well as achieving definitive abdominal closure will be reviewed.

29.4.1 Temporary Abdominal Closure in DCL

The use of DCL, as described earlier, allows for rapid termination of the initial operation for correction in physiologic derangements as well as eases planned reoperations in cases of hemorrhage, abdominal contamination, ischemia, or other indications. Abdominal fascial closure is ideal at the conclusion of planned reoperations after OA and can be achieved in the majority of cases (Barker 2000; Navsaria 2013). Rates of OA complications increase as the period between initial operative intervention and definitive closure increases (Redden 2013). Abdominal

fascial closure is best achieved after bowel edema subsides and prior to the formation of dense adhesions and granulation tissue between viscera and the abdominal wall (Chiara 2015). However; lateral retraction of the fascial edges in prolonged OA scenarios becomes problematic and at times fascial closure unachievable. Current temporary abdominal closure strategies aim to provide visceral protection, reduce or delay development of significant intra-abdominal adhesions, and prevent fascial retraction and thus abdominal domain loss.

Since the implementation of DCL, various strategies for temporary abdominal closure have been used including skin approximation, interposition material sutured to fascial edges, or more recently NPWT systems. The use of skin approximation techniques (i.e., towel clip closure, Bogota bags, silo techniques) (Feliciano 1988; Morris 1993; Burch 1992; Cipolla 2005; Tremblay 2001) has largely fallen out of favor. Skin approximation techniques are prone to significant fluid leakage and evisceration and do not allow for optimal measurement or egress of peritoneal fluid and do not prevent fascial retraction (Chiara 2015) and can be challenging from a nursing care standpoint. These techniques still place OA patients at risk of ACS development and have been shown to be a risk for fistula formation (Regner 2012).

Interposition patches sewn to fascial edges (e.g., Whitman patch; Keramati 2008) do provide a mechanical advantage to reduce fascial retraction. However, they also do not allow for egress of peritoneal fluid, place viscera at increased risk of mechanical injury, and do not prevent the formation of intra-abdominal adhesions. Interposition patch use is known to promote high rate of fistula formation as well as low rates of primary fascial closure (Campbell 2010; Open 2009). These patches are best used when fascial closure is known to be unachievable and granulation tissue is needed for eventual skin grafting (Chiara 2015).

NPWT systems and their iterations typically involve a porous plastic material that covers the bowel to prevent adhesions and allow egress of peritoneal fluid. The following layer is typically followed by a macroporous material or sponge followed by an occlusive dressing and negative pressure suction. NPWT have become the primary choice for the management of OAs (MacLean 2008) and are associated with the highest rates of fascial closure and lowest mortality (Quyn 2012; Voele van Hensbroek 2009). Known benefits of NPWT systems include prevention fascial retraction, decreased adhesion and fistula formation, titratable suction, peritoneal fluid egress, as well as overall ease of use. In trauma patients undergoing DCL, peritoneal fluid leak has been shown to contain inflammatory cytokines with NPWT use allowing a decrease in cytokine levels (Roberts 2013). Standard suction settings of negative 125 mmHg allow for adequate peritoneal fluid measurement and collection; in OA patients with coagulopathy, negative 75 mmHg pressure aids in coagulopathy correction (Open 2009). Potential drawbacks of NPWT are their availability in certain areas, technical issues with suction maintenance, and the need for frequent dressing changes (typically every 48 h) and cost. The increased experience and use of NPWT systems has led a recent international consensus conference to state that NPWT is the "best option" in OA management (Chiara 2015).

No investigations to date have rigorously evaluated these methods of temporary abdominal closure in a comparative manner. One study out of Italy compared the use of a skin approximation technique (Bogota bag) to NPWT system in the treatment of patients with ACS (Batacchi 2009). This study looked at prospective use of NPWT compared to historical controls (Bogota bag) and found that NPWT was significantly more effective in managing intra-abdominal hypertension as well as normalizing serum lactate in the first 24 h after laparotomy. In addition, NPWT system showed faster abdominal closure rate, decreased duration of mechanical ventilation, as well as ICU and hospital length of stay; however no significant differences in mortality were shown (Batacchi 2009). The applicability to the DCL trauma patient is unknown and highlights the need for further investigation.

29.4.2 OA Management and Complications

Outcomes in DCL use and subsequent OAs are defined to an extent by the development of complications and associated morbidity. Individuals undergoing DCL are, by definition, critically ill; improvement in outcomes has been largely secondary to advances in trauma resuscitation, ICU care, nutrition, and improved surgical techniques. Steps to prevent complications are needed; their early recognition is imperative to improve outcomes in patients undergoing DCL.

Individuals post DCL and with OA are in an extreme catabolic state making nutritional support paramount. Initiation of nutritional support, particularly enteral nutrition, has been shown to greatly improve outcomes in DCL and OAs. Development of malnutrition in this cohort has been shown to significantly increase morbidity and mortality (Open 2009; Bartlett 1982; Collier 2007; Kudsk 1992). It has long been shown that early enteral nutrition in trauma patients without OAs is associated with improved outcomes (Moore 1986, 1992). Similarly in DCL patients with OA, early enteral nutrition improves outcomes. A retrospective review from the University of Washington found that early enteral feeding (within 4 days of initial laparotomy) slightly improved rates of early abdominal closure, lower fistula development rates, and hospital costs, as well as being independently associated with reduction in pneumonia development (Dissanaike 2008). The use of enteral feeding formulas containing glutamine and 3-fatty acids may be of benefit through anti-inflammatory effects (Polk 2012). The use of total parenteral nutrition is generally reserved for only the extremely injured (McKibbin 2003) and those with less than 75 cm of usable bowel (Majorcik 2012).

Achieving early abdominal fascial closure has been shown to improve outcomes and avoid the dreaded complication of fistula formation. Fistula formation can occur in 15% of OA patients when early fascial closure is unable to be done within 8 days of initial laparotomy (Dubose 2010). Independent risk factors for fistula formation have been shown to be need for large bowel resection, need for large-volume resuscitation, and need for repeated manipulation of friable and edematous bowel (Bradley 2013). As mentioned earlier, techniques for temporary abdominal closure can also impact the rate of fistula formation.

Fascial closure is preferred and the most desirable option in DCL and OAs. Ideally fascia is closed primarily within the first week after initial laparotomy. The use of NPWT systems has been shown to allow for fascial closure even 3 weeks after initial laparotomy (Miller 2002). Fascial closure at times becomes unattainable and in certain patients can be inappropriate to perform. Patients with prolonged OA who develop fixed bowel edema, significant loss of domain, respiratory distress, or tertiary peritonitis should not have their fascia attempted to close (Open 2009). In instances of prolonged OA, a planned ventral hernia is a viable and safe option. The use of a synthetic absorbable mesh with subsequent granulation tissue formation and skin grafting or early skin-only closures allows for patient recovery and a planned definitive fascial closure in the future.

Conclusion

Recognition of severely injured trauma patients and development of coagulopathy, hypothermia, and acidosis should lead the trauma surgeon to employ damage control strategies in the treatment and resuscitation of these patients. Damage control laparotomies allow for correction of the lethal triad and definitive operative interventions under more control and most ideal circumstances. The increased use of these strategies has led to the complex and challenging scenario of the open abdomen in trauma patients. Developments in temporary abdominal wound closure with negative pressure systems has allowed for improved care and outcomes early on in damage control scenarios. These improvements have led to decreased rates of abdominal compartment syndrome and dreaded fistula formation. Advances in ICU care and nutritional support have played a large impact as well. Further investigations into damage control resuscitation and operative strategies are needed and ongoing and will lead to even better care and outcomes in the future.

Late Outcome After Severe Fractures

30

Roman Pfeifer and Christian Fang

Over the past decades, numerous improvements have been made in the delivery of trauma care and rehabilitation, such as injury prevention, rescue systems, in-hospital diagnostics, and various surgical techniques. The mortality rate of multiple trauma patients decreases from 37% to 18% during the past two decades [1–6]. Polytraumatized patients are likely to have persisting functional handicaps, long-term work disability [7–9], and psychosocial impairment. The socioeconomic burden on the affected individuals, their families, and society are often severe and long lasting [7–12].

30.1 General Long-Term Outcomes

It is important to have tools that longitudinally look beyond mortality and predict the patients' long-term function. Identifying the early factors that influence long-term outcome following major injuries can help physicians identify those that may require special interventions. Several projects [7–9, 13–16] evidence that individual, socioeconomic factors and health habits have a strong impact on the outcome on top of injury-related factors, such as injury severity, injury location, and treatment methods [8, 13, 17]. The most important poor prognostic factors identified are as follows [18–20]:

- *Patient related*
  - Female patients
  - Old age
  - High BMI (body mass index)
- *Injury related*
  - Brain injury
  - Lower extremity injuries
- *Socioeconomic-related factors*
  - Lower socioeconomic environment
  - Lower level of education

Functional deficits and chronic pain represent the main factors jeopardizing the quality of life after polytrauma [21]. Up to 60% of severely injured patients report chronic pain, often caused by injuries

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to the lower extremity. Thirty-five percent of patients require long-term analgesic treatment [19, 21].

Twenty-four to 39% of patients after having multiple injuries were diagnosed with posttraumatic distress syndrome, 32–70% with anxiety disorder, and 35–68% with depression. Additionally, cognitive defects, such as memory impairment, difficulty with concentration, and emotional problems have been reported [7, 13, 22–24]. These studies stress the strong need for routine early posttraumatic psychological support.

Self-efficacy was shown to be one of the strongest predictors of the Sickness Impact Profile and return to work [7–9, 13]. It is hypothesized that patients with low self-efficacy are more likely to be disengaged from the physical rehabilitation and recovery process. To address this issue, self-efficacy and self-management training should be prescribed to polytrauma patients, especially as benefits have already been demonstrated in the treatment of patients with other chronic diseases such arthritis, diabetes, and chronic pain syndromes [25, 26].

Several groups have shown gender-related differences in the well-being after severe injuries [16, 27]. Premenopausal women seem to have better short-term outcome than men in the acute phase [16, 27]. However, long-term results demonstrate the opposite, as females are more likely to have a higher incidence of psychological disorders and poorer long-term results [28–31].

A lower socioeconomic status has been reported to be associated with negative long-term outcomes [32]. In patients that are injured during their working age (20–50 years), financial impact and unemployment rate were frequently substantial [32]. In contrast, younger patients (≤18 years) appear to be more capable of adapting to disabilities and sustained life changes and are more likely to return to work. Despite this fact, social deprivation (loss of friends or social environment) was more often reported by younger patients. This includes failure of exams in students and the requirement to switch school [32].

Many of the findings reported below were generated in a study with a minimum follow-up of 10 years (mean 17.5 years) [14–16]: It summarizes data from 637 patients that were reexamined at least 10 years after their initial injuries. One of its main findings was that head and extremity injuries accounted for the most frequent causes of long-term disability [16]. At follow-up, 33% of patients required a medical aid for their disability, and 20.1% reported disability due to their injury. The results from this study are summarized in the following sections.

30.2 Upper Extremity Injuries

A limited number of information is available regarding the outcome of upper extremity injuries. Some isolated fractures [33, 34] are associated with low-energy mechanisms [33] and are thereby different from those of severely injured patients who have sustained high-energy trauma. At long term, patients with injuries to the upper limb generally have better outcomes than patients with lower extremity injuries [35, 36]. Nevertheless, concomitant vascular and neurological injuries (involvement of brachial plexus and peripheral nerves) were found to be a major determinant for poor recovery [37]. Additional adverse sequelae following upper extremity trauma include nonunion, heterotopic ossification, and impaired range of motion [34, 38, 39].

Mkandawire and coauthors analyzed the 5-year musculoskeletal recovery in survivors of severe injuries (ISS >15) [11]. They performed a reexamination of 158 severely injured patients (>15 years old) treated between 1989 and 1990. According to this multicenter investigation (16 hospitals), 48% of patients with shoulder girdle injury and 66% of those with arm and forearm fractures had persistent disabilities. Displaced fractures and articular fractures were identified as poor prognostic factors. Remarkably, at 5 years following trauma, 45% of patients with shoulder girdle and 62% of those with upper extremity fractures still complained of chronic pain. In particular, 75% of those with combined ipsilateral fractures of the shoulder girdle and arm had long-term disabilities. Moreover, authors have hypothesized that associated head, facial, and thoracic injuries can potentially interfere with rehabilitation, resulting in continuing disability and pain.

The 10-year follow-up study addressed above also looked at long-term outcomes following

Table 30.1 Functional status of the upper extremities following polytrauma with fractures at different localizations

	Articular fractures $N = 60$	Shaft fractures $N = 37$	Combined fractures $N = 52$
ROM >50%	88.3%	94.6%[a]	73.1%[b]
Contractures	8.3%[a]	10.8%	25%[c]
Stiffness	1.6%	2.7%	5.8%
Neurological impairment	11.7%	10.8%	13.5%
Full muscle force shoulder	90%	97.3%	88.5%
Full muscle force elbow	86.7%	100%[a]	80.8%[b]
HASPOC—total	70.2 ± 48.9[b]	47 ± 34.3[a, c]	69.4 ± 44.4[b]
SF-12 Phy	43 ± 11.9[b]	47.9 ± 9.8[a, c]	43.6 ± 9.9[b]

HASPOC Hannover Score for Polytrauma Outcome, *SF-12 Phy* Short-Form 12 items health survey, Physical Component Summary

[a]Significantly worse outcome vs. combined fractures ($p < 0.05$)

[b]Significantly worse outcome vs. shaft fractures ($p < 0.05$)

[c]Significantly worse outcome vs. articular fractures ($p < 0.05$)

upper extremity fractures and separated three groups: (1) isolated upper extremity articular fractures, (2) isolated shaft fractures, and (3) combined shaft and articular fractures (Table 30.1). Out of the three groups, those with combined shaft and articular fractures had significantly worse outcomes in terms of limitations in range of motion (ROM), presence of contractures (25%), and muscle weakness (10–20%). Patients with isolated shaft fractures have the best prognosis in a general rehab score and in SF-12 scores when compared with those that had articular and combined injuries. This may be a result of a high-energy injury background, complicated rehabilitation, and degenerative joint diseases that commonly follow articular fractures [40–44].

30.3 Pelvic Fractures

Pelvic fractures are often caused by high-energy trauma and are associated with multiple concomitant injuries of the lower limb, spine, abdomen, thorax, and head [45, 46]. Because of these accompanying injuries, it is harder to analyze the long-term outcomes with regard to the pelvis alone [47–49]. It has been shown that both the severity of fracture (stable vs. unstable) and the presence of associated injuries contribute to poor long-term outcome [50]. Patients with unstable pelvic ring injuries and additional associated injuries typically do poorly [49, 51]. Incomplete recovery and functional impairments are commonly observed following unstable pelvic ring fractures, while stable pelvic injuries rarely lead to major long-term problems [45, 51, 52]. Open pelvic fractures, in particular, carry poor prognosis [53]. Patients with combined anterior and posterior pelvic ring injuries typically have inferior outcomes (SF 12) versus those with isolated anterior or posterior ring injuries. In the long term, 39% of patients who suffered pelvic ring injuries had a limp and 11% required crutches [54].

Chronic pain syndrome, neurological impairment, and nonunion are negative factors that influence the long-term outcomes after pelvic fractures (Table 30.2) [52]. Pohlemann and coworkers reviewed the incidence of pelvic fracture patients with chronic pain [50, 52, 55]. At 2 years, 45% of patients with type A-Type fractures, 59% with B-Type, and 73% with C-Type fractures fracture had pain [50, 52, 55]. Nonanatomic reduction or insufficient fixation leads to chronic back pain, instability, malunion, and nonunion [46, 56, 57].

There is a close correlation between neurological injury and long-term functional outcome [48]. At 2 years, 21% of patients with B-Type and 60% with C-Type fractures continued to have at least some degree of neurological impairment [58]. In particular, vertically unstable injuries and transforaminal sacral fractures were shown to be associated with more severe neurological deficits [59, 60]. The sequelae of neurological compromise include peripheral motor and sensory disturbances, incontinence, and sexual dysfunction [47, 50–53, 55, 57, 58], and these sequelae result in work disability [46]. Approximately 50–75% of pelvic fracture patients are expected to be able to return to their previous occupation [47, 51, 53].

Table 30.2 Clinical examination of pelvic ring fractures following polytrauma

Study	Fracture type	Follow-up	Patient[a]	Pain	Functional disability	RTW	Neurologic impairments
Pohlemann 1994	Unstable fractures	2.2 years	58	11–66%	No data	No data	21–60%
Miranda 1996	Pelvic ring fracture	5 years	80	16–35%	8–21%	75–81%	No data
Tornetta 1996	Unstable fracture	3.7 years	48	37%	37%	67%	35%
Brenneman 1997	Open fracture	4 years	27	No data	No data	64%	18%
Kabak 2003	Unstable fracture	3.8 years	36	31%	No data	72%	16–31%[b]
Suzuki 2007	Unstable fracture	3.9 years	57	No data	No data	84%	28%

RTW Return to work
[a]Skeletally immature patients
[b]Sexual and urinary dysfunction

30.4 Lower Extremity Fractures

Lower extremity injuries are especially important in causing long-term loss of function [35, 61–63]. Polytrauma patients with lower limb involvement have a poorer prognosis than those without [61, 62]. Even after 2–5 years, patients rarely have full recovery and overall satisfaction [11, 12]. Especially those with complex fractures, dysvascular limbs, major soft tissue injuries, and severe foot and ankle injuries often experience prolonged recovery with a high incidence of complications and frequently require reoperations including amputations [64]. In addition, those with concomitant acetabular and lower extremity fractures seem to also do particularly worse. As with upper limb injuries, patients with articular involvement have significantly worse outcomes than those with isolated shaft fractures [65].

The Lower Extremity Assessment Project (LEAP) [7–9, 13] is a prospective study that included eight level I trauma centers. Long-term functional outcomes of 601 patients were assessed; patients with major leg-threatening injuries were grouped under two main treatment categories: (1) limb salvage or (2) amputation. The study included patients with open fractures, dysvascular limbs, major soft tissue injury, and severe foot and ankle injuries. Outcomes were graded using the Sickness Impact Profile, a multidimensional self-reported measurement tool [7–9, 13]. The results demonstrated comparable functional outcomes in both groups. However, regardless of the treatment option, both the limb salvage and amputation groups had severe long-term disabilities. One-half of all patients had physical subscores on the Sickness Impact Profile $\geq$10, indicative of significant disability; only 34% of patients achieved scores comparable to the uninjured. Only 58% of those working before the injury remained employed after 7 years. Of those who returned to work, 20–25% were unable to perform tasks at their pre-injury level. Moreover, no significant improvements were observed from the second-year to the seventh-year follow-up.

The 10-year study addressed before [14–16] demonstrated the following long-term results (Table 30.3): 30–45% of patients experienced posttraumatic pain, and approximately 10–30% reported limited range of motion. High rates of gait abnormality were observed in patients who sustained acetabular fractures, while lower rates of gait abnormalities were observed following femur shaft fractures. Outcome scores were significantly better following an isolated femur shaft fracture as compared to those with acetabulum and knee joint involvement. The observed rates of arthroplasty were 7.5% for the hip joint and 15.1% for the knee, and 12.3% had ankle

Table 30.3 Functional status of the lower extremities following polytrauma with fractures at different localizations

	Acetabulum $N = 20$	Prox. femur $N = 20$	Femoral shaft $N = 107$	Knee[e] $N = 48$	Tibial shaft $N = 34$
Persistent pain	50.0%	45.0%	32.7%	43.8%	26.5%
Abnormal gait	35.0%[a, b]	20.0%[a]	3.7%	8.3%	14.7%[a]
Work disability	27.8%[a, c, d]	10.0%	7.6%	19.8%	8.8%
Successful rehabilitation	70.0%	60.0%[a]	80.4%	56.3%[a]	67.7%
HASPOC—total	78.78[a]	70.07[a]	49.71	79.28[a]	65.73
SF-12 PCU	40.91[a]	40.95	46.05	39.81[a]	44.06

HASPOC Hannover Score for Polytrauma Outcome, *SF- 12 PCU* Short-Form 12 items health survey, Physical Component Summary

[a]Significantly worse outcome vs. femoral shaft fractures ($p < 0.05$)

[b]Significantly worse outcome vs. injuries at the knee joint ($p < 0.05$)

[c]Significantly worse outcome vs. fractures of the proximal femur ($p < 0.05$)

[d]Significantly worse outcome vs. tibial shaft fractures ($p < 0.05$)

[e]Knee: including fractures of the distal femur and proximal tibia

fusion. In all, patients with lower extremity injuries below the knee demonstrated significantly lower outcome scores than patients with lower extremity fractures above the knee joint, as measured by general rehab scoring, SF-12, the Tegner Activity Score, and the ability to return to work. The authors assumed that factors such as the thin soft tissue envelope, unfavorable blood supply, and complex fracture patterns contributed to inferior outcomes below the knee [15].

Conclusion

Along with the improvements in medical care, the long-term functional outcome, quality of life, and satisfaction of severely injured patients are increasingly studied. Social reintegration and return to work are the major long-term goals. Those with articular fractures, concomitant injuries, neurovascular compromise, and lower extremity injuries have poorer long-term results. The importance of psychosocial variables is emphasized, and early psychological intervention has been suggested to address the issue. As prognostic factors are now better defined, patients with severe injuries who are at risk should be identified with intensive interventions initiated early.

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