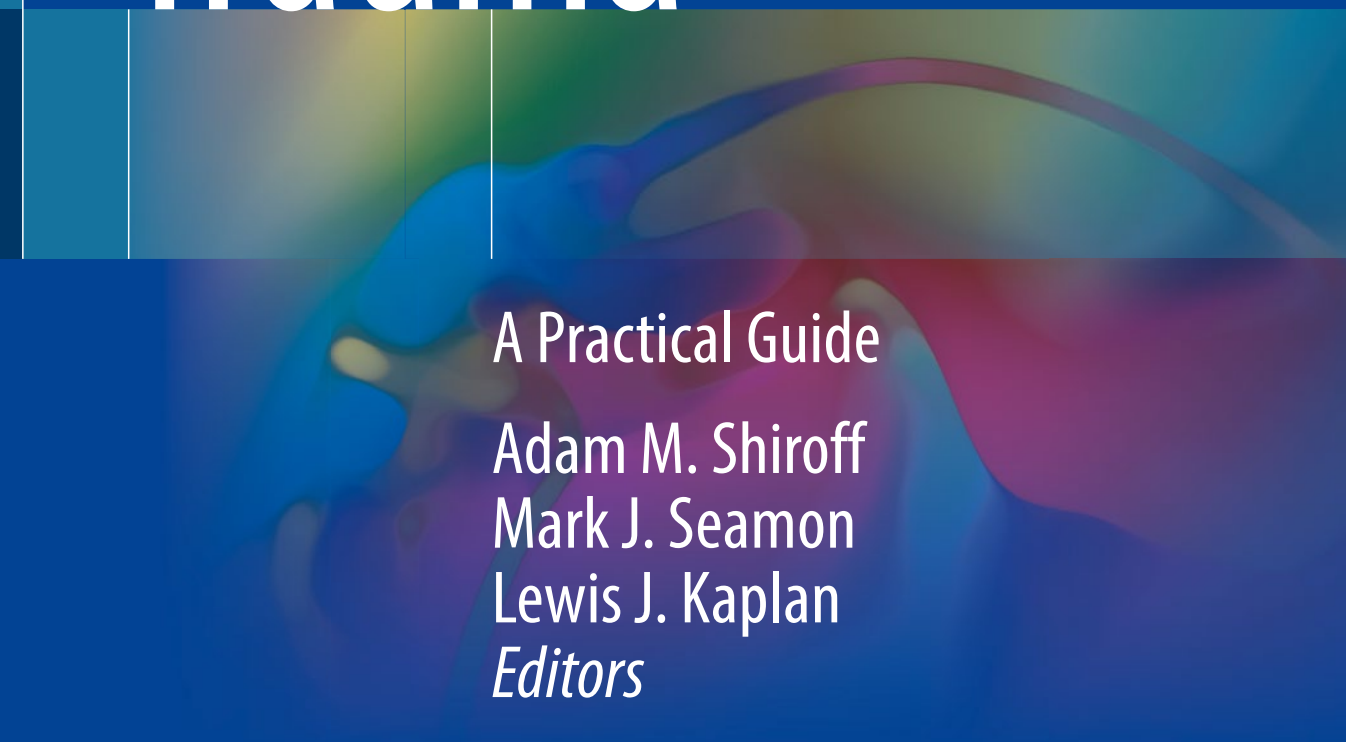
Management of Chest Trauma



A Practical Guide

Adam M. Shiroff
Mark J. Seamon
Lewis J. Kaplan
Editors

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This book is dedicated to our colleagues—in and out of the hospital—who help us care for patients with chest trauma that occurs in isolation, or within the setting of multicavity complex injury. Injury care leverages a team to rescue patients including those in law enforcement, Emergency Medical Services, Fire Rescue, Nursing, Advanced Practice, Pharmacy, Pastoral Care, Case Management, Social Work, and every medical and surgical discipline with the trauma center—we are indebted to you. On behalf of the authors, we dedicate this work to our author's families whose support in developing the skills used every day to rescue patients has required time to be spent apart. We most importantly dedicate the wisdom within this text to those who taught us how to save lives—our patients and their families. The pandemic has reinforced that injury is not bound by time, social circumstance, politics, nor public health approaches to viral pathogen containment. Patients and their family members are our partners in survival and recovery whether local to our trauma center or transported to us for specialty care after injury. This textbook is indeed driven by our dedication to you.

Foreword

CHEST Trauma is a novel approach to educating clinicians, especially trauma surgeons and those caring for patients with thoracic injury. The work delivers an inspiring argument that there is specialty knowledge needed to best manage this particular spectrum of injuries. Indeed *CHEST Trauma* is a first of its kind injury management textbook that addresses a single, but complex anatomic compartment. It focuses management using evidence-based care and state-of-the-art approaches that aid even the most seasoned traumatologist during complex decision-making and management prioritization. Contained within its pages is a robust amount of data and experience that is organized using an embraceable but partitioned approach to evaluating and managing the panoply of thoracic injuries we might all encounter. Clinical guidance flows from experiential leaders at major Level 1 regional resource trauma centers in the USA as well as similar centers abroad. Its well-researched topics deliver what clinicians challenged with chest trauma patients would expect from an in-depth examination of relevant topics that drive patient rescue. To that end, the authors deliver dense information in a readily digestible format. Best practices are presented in a way that supports rapid incorporation into your center whether urban, suburban, rural, or military in nature. The SARS-CoV-2 pandemic has underscored the need to think, plan, and deliver integrated care in a team-based fashion; *CHEST Trauma* hews to that approach with unprecedented deliverables. Importantly, the role of the trauma center in disaster planning and preparation, as well as resource allocation, serves as a capstone cementing the trauma center as a foundation for the communities and the region it serves. The trauma center as nexus to coordinate public health emergency care is cemented within the disaster management of the future. Similarly, I believe that *CHEST Trauma* will serve as a foundation to improve practice and spur new knowledge discovery. I have no doubt it will catalyze innovation and drive debates regarding how we implement those discoveries as we continue to improve how we as a trauma community deliver and improve the care of

victims of thoracic injury. Cheers to its authors for delivering a needed treatise for these challenging times.

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Preface

Advances in prehospital care, technology, inpatient management, and operative care benefit patients with thoracic injury regardless of age. This textbook explores those underpinning advances in three domains: chest wall injury, thoracic content injury, and critical care. In order to improve the care of those with thoracic injuries associated with high risk of mortality or morbidity—including natural and man-made disasters—we present an entirely new textbook exclusively focused on managing patients with chest trauma. Authors are acclaimed experts within the segments that they present, and offer in-depth analyses and best practices to influence care. Woven throughout this book is the theme of team-based care linking partners in the prehospital and inhospital spaces across the spectrum of post-injury patient rescue and care. We trust that this textbook serves as a guide for those embarking upon a career in trauma, but also as a touchstone for those with established careers to refine existing practice.

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1 Centers of Excellence, Trauma and Health-Care Systems, and Regionalization

Kristen M. Chreiman, Madhu Subramanian, and Patrick M. Reilly

Introduction

More than five million people, or 9% of the global population, die of traumatic related injuries annually causing this to be the third largest source of disease burden worldwide [4]. In the USA alone, trauma is the third leading cause of death and continues to be the leading cause of death for those under the age of 44; accounting for 25% of all life years lost [5]. In fact, in 2013 health-care costs and lost productivity from traumatic injuries were estimated at $675 Billion annually. Thoracic trauma alone makes up 25% of all trauma related deaths with rib fractures being the most common (20%) injury within this subset [6–10]. Trauma mortality can be divided into three timeframes: 50% of deaths occur at the scene, whereas 30% die within hours of presentation to trauma centers due to neurologic injury or hemorrhage, and 20% die of infection or multiorgan failure within days to weeks of injury [11, 12].The former scenario can be addressed through injury prevention mechanisms but the latter two scenarios are dependent on rapid and quality trauma care and are the focus of in-hospital efforts. These staggering statistics have fostered the drive for the development of trauma systems, regionalized trauma care and the need for establishing centers of excellence in injury care [6, 13, 14].

Application of Combat Casualty Care to the Civilian Sector

Much of our current knowledge regarding triage and injury care stems from early experiences on the battlefield. Such encounters have led to developments in care delivery and process improvements like accurate and timely triage and appropriate resource allocation [15]. Evolving practices during the two world wars introduced new technology such as motorized transport vehicles, early delivery of blood and antibiotics, as well as the application of hemostatic dressings. The Korean and Vietnam wars continued to advance trauma care by moving stabilization efforts closer to the injured patient and utilizing aeromedical transport in difficult to reach areas. Iraq and Afghan conflicts improved field trauma

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care further adding new levels of expeditious scene care, improved triage and regionalization of trauma care resulting in a reduction in mortality to 1.7% [16]. Lessons learned during combat casualty care highlighted the need to optimize care delivery and develop trauma systems in the civilian setting [14, 16, 17].

In the years following the wars, a series of pivotal, parallel events ensued. Originally treatment of injured patients was largely performed at large municipal hospitals associated with university medical schools and staffed by house officers and faculty. Quality care was provided at these hospitals through lessons learned in conflict and reinforced by civilian volume and research. Unfortunately, this same level of care could not be found once outside of urban settings [18]. In 1966, the National Academy of Sciences published "Accidental Death and Disability: The Neglected Disease of Modern Society," which advocated for the organization of trauma care and the development of a care delivery model with systematic resource allocation [19]. This article was pivotal, as it described the foundational underpinnings of the structure we know today. Further, this publication suggested the need for centralized data collection, quality metric capture, and formalized education in trauma care. Shortly thereafter, the National Highway Safety Act (1966) appropriated funding for the creation of the National Highway Traffic Safety Administration and mandated state-level safety mechanisms such as licensing and vehicle inspections as well as programs for improved prehospital communication and scene transport. The Emergency Medical Service Systems Act of 1973 was created to help develop comprehensive EMS systems throughout the country guided by the Department of Health [20]. During the 1970s several US states implemented these strategies and demonstrated a reduction in mortality with organized trauma care [15]. However, there was still a pressing need for identification of hospitals with specialized resources designed for trauma care distribution and effective bidirectional communication between those hospitals and prehospital providers. In 1976 the American College of Surgeons Committee on Trauma (ACS-COT) published an unprecedented historical document, "Optimal Hospital Resources for the Care of the Injured Patient" later named the "Resources for Optimal Care of the Injured Patient." This guided pursuing centers on what key elements would be expected from within a trauma center as well as how that trauma center would function within the larger network. Pillars included commitment, readiness, access, triage, care delivery and coordination, quality improvement, rehabilitation injury prevention and outreach, and research [15]. "Resources for Optimal Care of the Injured Patients" remains the gold standard guiding trauma center functions, resource allocation and benchmarks covering public access to services through 911; prehospital care, triage and transport via emergency medical services; emergency and inpatient care; rehabilitation, as well as public health measures including injury prevention, community reintegration and social services, and methods for performance improvement [16, 21, 22].

Regionalization

Although the ACS-COT developed criteria for trauma center designation, few states had a body to designate trauma centers, largely due to funding issues that were the result of sharp federal funding cuts as a result of the Omnibus Budget Reconciliation Act of 1981 [23]. In 1990, the Trauma Care Systems and Development Act laid out a Model Trauma Systems plan and provided a source of funding for trauma systems development, although the act proved to be unsuccessful due to stringent rules and limited funding. As a result, the American trauma system is comprised of several independent regional trauma systems run by state and local governments and with varying standards.

Inclusive and Exclusive Trauma Systems

The core of a trauma system is the network of hospitals that provide trauma care. Trauma systems fall into two categories: inclusive and exclusive [24]. Originally, trauma systems were framed

around an exclusive model, where a region designates one hospital (or a few) as the primary center within a set geography for the diagnosis and treatment of injury care. While this model facilitates and promotes volume requirements to maintain proficiency in surgical skill, it is not always the most cost-effective. This model has made way for more mature, "inclusive" systems where all around-the-clock emergency rooms in a region are able to participate in trauma care. Inclusive systems envelope a collection of hospitals into a more sophisticated arrangement that enables a leveling classification; Level I and II serve as regional resource tertiary care facilities and provide care for the most severely injured, whereas Levels III and V provide initial treatment/stabilization and transfer [14]. Higher leveled centers (Level 1 and 2) accept the liability of the outcomes of those transferred for resource intensive complex injuries. These centers commit to continual readiness with an investment in a multidisciplinary care delivery model. This network of hospitals function together to embody appropriate triage, facilitate timely definitive care to improve morbidities and mortalities, reduce risk with transfers, and reduce cost to those within the system [25].

Outcomes for patients in state-mandated trauma systems outperform states without regionalized care [26]. Further, nationwide data demonstrates mortality reductions of about 25% when treated at trauma centers [27]. Although volume of patients (>650 annual admissions) was directly related to outcomes [15, 16, 28–30]; outcomes only appear to be improved for complex injury care in systems defined as being established for over 8 years [29]. Moore et al. (2017) performed a systematic review of the literature to examine which key components of a trauma system positively impacted outcomes. During their study they reported that an inclusive, mature trauma system with continuous coverage of subspecialists were shown to lower mortalities from motor vehicle collisions. Other key components included decision-making skills which directed the right patients to the right place using rapid transport. These aspects should not be overlooked when examining factors reducing mortality [9, 31, 32].

Economic Factors

Trauma Systems in the USA are not without their shortcomings. Although regionalization is one way to create cost-effective care delivery models, it also can come with a cost not only to the patient in travel and expense, but to the higher level hospitals as patients tend to be sicker and underfunded [16, 21]. Due to a lack of a federally mandated governing body that regulates trauma care or allocation, disparities in access exist nationally and within states. For example, one study demonstrated about 40 million Americans live in proximity to approximately 20 trauma centers in the northeast, compared to over 50 million Americans that remain outside a 60-min window and without timely access to a Level 1 or 2 trauma center [14].

In addition, trauma care can be expensive, and reimbursement may be underwhelming in many urban environments. In states where designation and/or participation in national benchmarking databases is mandatory, annual fees may be a large deterrent or not sustainable. Excessive unmonitored over-triage rates within an organization can also stress the system with unwarranted costs. However, more recently certain areas in the USA located in more favorable payor mix arenas and coupled with the ability to charge for an uncapitated trauma activation fee, tend to have an overabundance of trauma centers. In fact, ACS-COT warned that uncontrolled growth of trauma centers had the potential to worsen regional trauma care by spreading resources thin, increasing personnel costs without improvements in outcome as well as ignoring large swathes of the country [33–36]. This together with the rising cost of health-care readiness and falling reimbursement rates leaves centers at the mercy of national professional medical societies advocating for change [14].

Regionalization Based on Needs Assessment

In the USA, access to trauma care is variable. There are sections of the country whose access to injury care remains outside of the "golden hour" rule. Accrediting bodies are tasked with ensuring institutional commitment to support comprehen-

sive infrastructures which includes robust performance improvement processes. These national agencies attempt to reduce variability of care delivery and equitable access to high quality trauma care.

Modern research describes a more tactical approach to the deployment of systems within geographical regions to reduce oversaturation and more evenly distribute the burden of care. Geospatial mapping could be a valuable tool to inform decisions for the placement of trauma centers in areas without established trauma systems in place. Taking a blank slate, Horst et al. combined geospatial technology with experience and bed capacity to determine the best placement for Level 1 and 2 adult trauma centers. They were able to divert focus away from individualized hospital interest and instead focus on largest population served [37]. In 2015 the Needs Based Trauma Center Designation Consensus Conference, convened by the ACS-COT, developed a needs-based assessment of trauma systems tool (ACS NBATS). By examining population concentration, transport times, injury volumes and hospital resources; this team's mission was to develop a measurable way to determine the number of trauma centers and their strategic distribution individualized to a specific region. This needs-based guide could establish a nonbiased direction for trauma system development inclusive of urban- and rural-level injury care while mitigating the effects of the double transfer [16, 38, 39].

Evolution in Modern Health-Care Landscapes

Over the last decade, the USA experienced fundamental changes to its health-care landscape. In 2007, Schwab described the increasing dilemma of the current environment of trauma and emergency care by which there were not enough surgical specialists to meet the demands for around-the-clock specialty care in two thirds to three-quarters of the country [30]. This followed at a time where acute care facilities were already experiencing record high visits leading to overcrowding, increased diversion hours, and uncompensated care [40, 41]. In response, consolidation of health-care systems has accelerated in the last decade. Surgical subspecialty and trauma care has centralized the most complex and resource intensive care to only a few medical centers [1, 2]. Standalone hospitals are finding it challenging, if not impossible, to remain independent with only 13% of independent hospitals left in 2012 [3].

Coordinated care delivery with a focus on system integration has the potential to foster better developed relationships between community-based hospital and academic tertiary care centers. Hospital mergers have the potential to reduce cost through efficiencies—alleviating financial burdens from individual centers and reducing duplication through a shared system of services. These savings though are not always immediate and may require several years for recovery in both patient outcomes and financial gains [42–44]. Health-care systems are charged with constant monitoring of their existing market share and forecasting future growth opportunities to ensure economic viability. This approach involves integrating culture, strategic planning and thoughtful execution; failures of mergers and acquisitions most frequently occur when the cultures of the involved organizations are incompatible [3, 10, 42]. In such scenarios, systems yield increased pricing for commercially insured patients and declines in patient satisfaction [45]. Further, lack of competition within a geographic area can lead to complacency in performance and quality [45–48]. A successful merger requires adapting inherent practices, providing early adoption of innovative technologies that expand reach into greater communities and enhancing patient experience via nontraditional routes like telemedicine; all without adding additional costs to patients [1, 3, 49, 50].

Centers of Excellence in Health Care

In modern times with more informed patients, limited compensation, and competitive markets, health systems are examining ways to differenti-

ate themselves and increase market share. Instituting a center of excellence (CoE) is one such strategy. Organizational culture is the engine for the success or failure of a CoE. Key stakeholders who are considered experts in their field must embody a shared vision and align their mission for quality excellence in care delivery and outcomes. Cancer centers are an example of existing CoEs in the USA. They house physician experts from a variety of disciplines all related to cancer care. Not only do these centers include disease site experts in medical oncology, hematology, radiation oncology, and ancillary services (social work, nutrition), but they also provide multiple treatment modalities and additional resources for labs, pharmacy, patient education, and wellness (pet therapy, yoga, virtual reality). Some favorable outcomes have been described by more coordinated care from shared electronic medical records, decreased variations in practice patterns, and prevention of morbidity and mortality by cohorting surgical procedures to one facility, thereby increasing volume and provider experience and eliminating the risks often caused by inexperience from low volume procedures [51, 52].

Benchmarking outcomes against national standards is one way to validate effective care delivery practices, prove exceptional outcomes, and identify industry leaders for a specific disease. An article by Halm et al. in 2002 examined relationships between hospital volumes and surgical outcomes for cardiovascular, thoracic, and abdominal surgical procedures [53].The study determined that mortality rates improved in high volume centers which demonstrated the benefits of regionalized care delivery models [16]. This same concept rings true for trauma centers. Chest injury could also benefit from replicating these synergies within a regional resource trauma center and should be considered as a CoE of the future. The development of a CoE for chest trauma would operate in parallel coordinating acute care surgery (emergency general surgery and trauma surgery), cardiothoracic surgery, vascular surgery, spine/orthopedic surgery, anesthesia/pain management, social work, plastic surgery and a designated call center (Fig. 1.1).

Designation of CoE are currently unrestricted and unmonitored with no overarching governing body mandating standards of care. This means that health systems have the ability to self-assign as a CoE. There are two different methods seen that health-care organizations use to market this venture to the public. One is to include the CoE in the formal brand of the organization and the

Fig. 1.1 Chest injury center of excellence

other can serve as a reference within a title for a division or department within the organization [51]. Marketing aside, the organization needs to be able to deliver the quality of care that will eventually draw and maintain a referral base [51]. Centers of excellence are differentiators in that they improve efficiencies by centralizing operations and cohorting resources; bundle payments; improve quality and reduce cost; and ultimately improve quality and satisfaction of the patients they serve.

Innovating Market Strategies Build and Sustain

When considering new technology, staying ahead of the curve is critical to cornering the market share. Partnering with industry can be that effective strategy. For example, when deployed in the appropriate patient cohort, stabilization of rib fractures can reduce or eliminate pain, minimize prescription drug use, and expedite reentry into the workforce. Zhou et al. (2019), described their experiences with integrating modern technology into surgical practice. Three-dimensional modeling/printing, historically used in orthopedic procedures, such as long-bone fractures or clavicle repairs, was found to be useful to help surgeons visually understand fracture patterns and patient anatomy prior to the operation [54, 55]. The creation of a rib model using 3D printing facilitated minimally invasive techniques and reduced operative time and pulmonary complications [54, 55].

In the spring of 2020, as the COVID-19 pandemic hit the USA, the value of telemedicine and connected health strategies became apparent. Adaptability is one of the inherent strengths of those involved in the delivery of trauma care. During this time, telehealth strategies rapidly evolved and relaxed HIPAA regulations allowed for expanded use. Previous to 2020, telemedicine in trauma was limited to specific use cases such as consultation between burn centers and acute care facilities for the discussion of treatment and transfer needs. In one east coast urban regional trauma center, telemedicine services were used to connect remote OB-GYN care teams and trauma teams as an interim method of direct care until response teams arrived to assist in the resuscitation of the pregnant trauma patient. Other examples include telemedicine remote monitoring in intensive care units, virtual communication for morning report, and teleconferencing for education. After COVID-19, telemedicine and mobile health strategies will be reimagined facilitating sustainment of some of these initiatives and engage in efforts to improve reimbursement models. As access to subspecialty care continues to evolve, connectivity through telemedicine is a viable option and is projected to be more widely adopted.

Summary

National efforts to reduce disparities to access for trauma care as well as monitoring the need for new centers should continue. In an environment where the general public are more engaged in their health care, the adaptability of surgeons and their continued evolution of skills is essential. Personalized medicine with integrated technology and excellent outcomes will continue to be an adopted strategy of health-care systems. By centralizing services into centers of excellence (CoE), health systems can optimize efficiencies and enhance patient satisfaction and outcomes. Chest injury could benefit from replicating these synergies within a regional resource trauma center to enhance efficiencies and patient satisfaction and outcomes, and should be considered as a CoE of the future.

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2 Introduction, Definitions, and Significance of Chest Trauma

Michael A. Vella, Yanjie Qi, and Adam M. Shiroff

A textbook dedicated entirely to chest trauma highlights the importance of these injuries in the overall care and outcomes of the trauma patient. Injuries ranging from single rib fractures to those of the heart and great vessels can be associated with significant short- and long-term morbidity and mortality. While the introduction of novel and less invasive techniques has expanded the options available to those with chest injuries, these advancements have increased the complexity of decision-making, and, in some instances, introduced ongoing controversy. Treatment strategies previously considered dogma have been scrutinized with both retrospective and prospective studies. Something as "simple" as chest tube size illustrates this well. A 2012 study comparing small (29–32 French)- to large (36–40 French)-bore chest tubes found no significant differences in complications or need for additional procedures [1]. Several years later, Bauman et al. demonstrated in a prospective study that percutaneously placed, smaller diameter tubes were as efficacious as traditional chest tubes with respect to drainage, insertion-related complications, and failure rates [2]. While these studies have certainly contributed to the literature in an important way, the debate over the best method of chest drainage is far from settled.

The goal of this book is to provide trauma practitioners with up-to-date, evidence-based strategies for the care of injured patients with chest trauma. The following are brief descriptions of the history of chest trauma management, the anatomy of the chest, the epidemiology of chest trauma, and the short- and long-term sequelae of these injuries.

History of Chest Trauma Management

Descriptions of chest injury resulting from war, construction activities, and farming date back to ancient Egypt (4000–3500 BCE) [3]. Cuneiform etchings circa 3000 BCE suggest that the *Asu*, or Sumerian general practitioner, may have been performing thoracotomy for pleural drainage [1]. Writings from 1600 BCE Egypt first describe rib injuries and open chest wounds, which were treated with binding devices [3, 4].

Thoracostomy for chest trauma was described by fourteenth-century surgeons, although the procedure was rarely performed at that time. Drainage

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of traumatic hemothoraxes using "flexible tubes" was described by Boerhaave in the early 1700s [5]. Modern chest tube drainage via a continuous water-seal device for empyema was first described by Playfair in 1875, and active suction was employed in 1910 [6]. Despite these relatively early descriptions, continuous thoracostomy tube drainage was not routinely used until after the world wars and evolved in both the civilian and military arenas. The mortality rate of chest injuries during World War I was at least 25%, most commonly from bleeding and empyema [7]. Treatment at that time typically consisted of repeated thoracentesis for removal of blood, closure of open pneumothoraxes, and occasional thoracotomy (typically for foreign body removal or late compilations of chest injury). While advancements in airway management, general anesthesia, antibiotics, and blood banking improved the overall mortality of chest wounds, routine chest tube drainage was not commonplace due to actual or perceived procedural-related complications. By World War II, the morality of chest injuries sustained by American soldiers decreased to 5–8%. The use of thoracostomy tubes was typically reserved for air leaks that persisted after needle aspiration as it was usually of short duration. It was not until the 1960s that routine use of thoracostomy tubes was the norm, with more rapid adaptation in the civilian population and during the Vietnam War. At that time, mortality from chest injuries in combat was reported to be 5.6% and related to the relatively high mortality of tracheal and bronchial injuries [5, 7].

Continuing advancements in diagnostics, mechanical ventilation, treatment of infection, operative techniques (especially related to aortic and great vessel injury) and a better understanding of acute respiratory failure have greatly improved the care of the injured thoracic trauma patient [4]. Mortality from chest trauma in modern warfare has been less than 2%, and majority of injuries in both the military and civilian populations can be managed with supportive care and chest tube drainage alone [7].

Thoracic Anatomy and Patterns of Chest Injury

The musculoskeletal cage of the chest includes the clavicles, manubrium, and sternum anteriorly as well as ribs that wrap around to the spinal elements posteriorly, all supported by muscles used for respiration and movement. Contained within this protective cage are the mediastinum (pericardium, heart, esophagus, trachea, great vessels, thoracic duct, and thymus) as well as the bilateral pleural cavities containing the lungs and their associated vasculature [8, 9].

Thoracic injuries occur from both blunt and penetrating mechanisms, with blunt trauma often resulting from rapid deceleration forces (causing organ shearing/tearing), crush, and intrusion. Severity of injury is likely related to a combination of these factors as well as the velocity of tissue deformation and amount of tissue compression [4, 10]. An early study of thoracic injury patterns in fatal motor vehicles crashes (MVC) illustrates the broad range of injuries the can occur from significant blunt chest trauma [11]. From 1954–1959, 294 of 585 (54%) MVC casualties in the New Orleans area sustained significant thoracic trauma, and 133 (45%) were thought to have died as a direct result of those injuries. Common injuries included rib fractures (39%), hemothorax (28%), lung laceration (10%), ruptured great vessel (10%, most commonly the thoracic aorta), lung contusion (6%), diaphragm laceration (5%), sternal fracture (5%), myocardial laceration (4%, most commonly the right ventricle), cardiac contusion (2%), tracheal laceration (1%), and esophageal laceration (0.2%). Only 2 of 22 (9%) patients with cardiac laceration and 11 of 58 (19%) patients with thoracic great vessel injury reached the hospital alive. There have been marked improvements in preventative efforts, motor vehicle safety, and the care of injured patients since that time, although morbidity and mortality remain considerable.

Epidemiology of Chest Trauma

Unintentional injury is the fifth leading cause of death in the USA, and the third leading cause when suicide and homicide are included [12]. Trauma is the leading cause of death in Americans ages 1 to 44, with injuries killing more people between the ages of 1 to 34 than all other major causes combined [12]. In survivors, injury also results in significant lost wages and productivity.

The thorax makes up a fourth of total human body mass and is frequently injured by both blunt and penetrating mechanisms, which account for 7% and 8% of all US trauma admissions, respectively [8, 13]. Seventy to 80% of all thoracic trauma results from MVC, with significant injuries associated with high vehicular speed, lack of seatbelt use, and extensive damage to both the vehicle and steering wheel [8, 14–20]. Additional common mechanisms include motorcycle crashes, pedestrians struck by vehicles, and falls [4].

According to the 2016 National Trauma Data Bank report, 194,622 patients sustained thoracic injuries in 2015 (23% of patients) [21]. The case fatality rate was 9.5% for those with chest Abbreviated Injury Severity (AIS) scores ≥3. Overall, thoracic trauma results in about 16,000 annual deaths in the USA [8]. Blunt thoracic aortic injury is the most common lethal injury of the chest (especially if left untreated), with majority of deaths occurring immediately [22, 23]. In a 2004 study of 1359 consecutive chest trauma patients (90% blunt) at a level I center, the overall mortality rate was 9.4%; 56% of deaths occurred immediately upon arrival or within 24 h of admission [18]. Low GCS and advanced age were the most significant predictors of mortality. It is important to note that isolated chest injury was uncommon (as it is in many series), with about 20% of patients sustaining significant abdominal, long bone, and spine injuries.

Mortality from penetrating chest trauma can approach 30% and has been associated with degree of pulmonary resection and need for damage control operations [24]. Interestingly, Horst et al. found that, while the morbidity of chest injuries in their population has decreased, overall mortality has not [25].

Less than 10% of patients with chest trauma require thoracotomy, most commonly for ongoing hemorrhage or hemodynamic instability. Penetrating injuries of the chest are associated with higher need for operative intervention, in the range of 15–20% [4, 13, 26]. In a large study of primarily blunt chest trauma patients, 18% of patients required immediate chest tube placement, and only 2.6% required thoracotomy [18]. In a recent report on some 3000 penetrating chest trauma patients from a single urban level I trauma center, 23.4% of patients required thoracotomy or median sternotomy (the authors excluded emergency department thoracotomy) [24]. Fortunately, majority of blunt and penetrating injuries can be managed with supportive care and chest tube placement when indicated [8, 13, 26–28].

Brief Introduction to the Sequela of Chest Trauma

Chest injuries often result in both short- and long-term complications that will be discussed in relation to specific injuries elsewhere. In brief, significant chest injury is a predictor of other injuries and is associated with pneumonia, sepsis, venous thromboembolism, and respiratory decompensation often requiring ICU admission, prolonged mechanical ventilation, and tracheostomy [29–33]. These complications are more pronounced in older individuals. In a study of 1621 patients older than 50 years (mean 70 years) with at least 1 rib fracture (mean 3.7 fractures), overall mortality was 4.6% [32]. Mortality was associated with admission to a high volume trauma center, need for intubation, increased injury severity score (ISS), development of pneumonia, number of rib fractures, and a history of coronary artery disease and congestive heart failure. The use of patient controlled analgesia (PCA) and tracheostomy were associated with

improved outcomes. Bulger et al. found that each additional rib fracture in patients ≥65 years of age increases the odds of pneumonia and mortality by 27% and 19%, respectively [31].

Chest injuries are also associated with adverse long-term outcomes. In a study of 105 French ICU patients with no prior pulmonary history sustaining blunt trauma (thoracic AIS ≥ 2), 39 of 55 (71%) subjects evaluated at 6 months post-discharge had at least one abnormal pulmonary function test (PFT) [34]. These individuals were also found to have decreased 6 min walk distances as well as impairments in pulmonary related quality of life factors when compared to normal controls. In another study of 203 patients with rib fractures (mean 5.4 fractures), 59% of the 187 subjects who were followed for 2 months postinjury had prolonged chest wall pain; 76% had prolonged disability [35]. Of the 111 patients with isolated rib fractures, 64% had prolonged chest wall pain, and 66% had prolonged disability. At 60 days, 66% of all patients were still using opioids, including 31% of those with isolated rib fractures. Blunt trauma to the bony thorax has also been associated with chronic chest wall deformities, decreased lung volumes, exertional dyspnea, and decreased ability to return to work [36]. Less is known about long term functional outcomes in penetrating trauma patients, although a study by Keller et al. of 16 survivors of emergency department thoracotomy found that, while unemployment and daily alcohol/drug use were common, few had long term evidence of long term functional and neurologic impairment [37]. More work is needed to better define the long term outcomes in those with penetrating injuries.

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The Initial Resuscitation of the Chest Trauma Patient

3

Catherine E. Sharoky and Mark J. Seamon

Abbreviations

ATLS	The advanced trauma life support
CPR	Cardiopulmonary resuscitation
CT	Computed tomography
CTA	Computed tomography angiography
CXR	Chest radiograph
EAST	The Eastern Association for the Surgery of Trauma
ECG	Electrocardiogram
EDT	Emergency department thoracotomy
FAST	Focused assessment with sonography in trauma
TEVAR	Thoracic endovascular aortic repair

Introduction

Trauma is the leading cause of death for people 46 years and younger and has increased for all-comers over the last decade [1]. Thoracic injuries account for 25% of trauma deaths. Penetrating trauma most commonly occurs secondary to gunshot wounds and stabbings, with gunshot wounds accounting for more death than stab wounds [2]. Industrial accidents, blast injuries, and impalements make up the majority of remaining penetrating chest trauma. Falls and motorbike and motor vehicle collisions are the most common cause of blunt thoracic trauma, accounting for >75% of cases [3].

Thoracic structures at risk of injury in chest trauma include the chest wall (ribs, intercostal vessels), lungs, tracheobronchial tree, heart, great vessels (superior and inferior vena cava, pulmonary arteries and veins, aorta), brachiocephalic trunk, left subclavian artery, left common carotid artery) esophagus, spinal cord, thoracic vertebrae, and the thoracic duct. Injuries including disruption of the great vessels, heart, and tracheobronchial tree often result in death on the scene.

The majority of chest trauma that reaches the hospital is ultimately managed nonoperatively. Only 10–15% of blunt thoracic trauma, and 15–30% of penetrating thoracic trauma require surgery [4].

Prehospital care of the chest trauma patient should focus on stabilization and transport to the closest trauma care facility with capacity to care for the injured patient. Patients with thoracic injuries and hemodynamic instability should be transferred to a Level I trauma center. Rib fractures occur in over 1/2 of blunt thoracic injuries [3], and are often associated with pneumothorax. Patients with penetrating or blunt thoracic injury

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and clear evidence of tension pneumothorax physiology (hemodynamic instability with respiratory distress, absence of breath sounds) may benefit from needle or finger thoracostomy in the prehospital setting. Though concerns about the inability to correctly identify tension pneumothorax in the prehospital environment exist, some evidence suggests that prehospital needle thoracostomy improves physiology and dyspnea with few iatrogenic complications [5]. The technique for needle thoracostomy is described later in the chapter. Patients with thoracic injury resulting in an open pneumothorax or "sucking" chest benefit from an occlusive dressing secured on three sides applied in the prehospital setting. Spine immobilization in penetrating trauma has not been shown to mitigate neurologic deficits and is not recommended for prehospital transport [6]. Blunt trauma is frequently associated with polytrauma including head and abdominal trauma, and as such spine immobilization is essential for blunt trauma patients.

While there are some principles common to all trauma resuscitations that guide the initial resuscitation of chest trauma patients, resuscitation varies widely depending on the mechanism of injury and clinical presentation.

Resuscitative Emergency Department Thoracotomy

Patients who present pulseless and unconscious after penetrating or blunt trauma are in traumatic circulatory arrest. The arrest may be secondary to severe hypoxia, tension pneumothorax, exsanguinating hemorrhage, cardiac herniation, or severe myocardial infarction [7]. The cardiac rhythm can be pulseless electrical activity, ventricular fibrillation, or asystole. Closed cardiopulmonary resuscitation (CPR) may be ongoing when the patient arrives if they are transported by emergency personnel. Immediate resuscitative emergency department thoracotomy (EDT) may be indicated depending on specific factors including elapsed time since arrest, mechanism of injury, and signs of life [8]. Signs of life include palpable pulse, measurable blood pressure, pupillary response, spontaneous movement, spontaneous respirations, and cardiac electric activity.

EDT allows for immediate access to the thoracic cavity with the goal of relieving cardiac tamponade, controlling cardiac and vascular injuries, and temporizing exsanguinating abdominal hemorrhage. Access to the chest is obtained through a left anterolateral thoracotomy in the fourth or fifth intercostal space. A vertical pericardiotomy (to avoid injury to the phrenic nerve) is made to release any tamponade, followed by aortic cross-clamping to temporarily control hemorrhage and to allow filling of the heart. Attempt is made to directly repair or temporize any cardiac injuries identified. Open cardiac massage and defibrillation is performed as indicated. Simultaneously, a right-sided tube thoracostomy is placed to evaluate for massive hemothorax. The thoracotomy incision should be extended across the sternum in a clamshell configuration to further evaluate for intrathoracic injuries if massive right-sided hemothorax is evident. Immediate transfer to the operating room for definitive control and repair should be pursued if the patient regains signs of life after EDT.

EDT is a controversial procedure with a survival rate of approximately 7% in all-comers [9, 10]. Highest survival rates (>20%) are seen with patients who undergo EDT after thoracic stab wounds with signs of life documented on arrival [8, 10]. EDT can expose healthcare providers to blood-borne pathogens. A recent prospective study of more than 300 EDTs found a 7.2% exposure rate per procedure, with trainees most often exposed [11]. The Eastern Association for the Surgery of Trauma (EAST) 2015 practice management guidelines provide recommendations for EDT in various clinical situations (Table 3.1).

Primary Survey

The principles of The Advanced Trauma Life Support (ATLS) resuscitation course of the American College of Surgeons are utilized in trauma centers around the world to guide the initial evaluation and management of all life-threatening injuries [7]. This protocol begins with

Table 3.1 The Eastern Association for the Surgery of Trauma (EAST) practice management guidelines on resuscitative emergency department thoracotomy (EDT). *Table extrapolated from:* Seamon MJ, Haut ER, Van Arendonk K, Barbosa RR, Chiu WC, Dente CJ, et al. An evidence-based approach to patient selection for emergency department thoracotomy: A practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2015;79(1):159–173

Guidelines on resuscitative Emergency Department Thoracotomy (EDT)			
Injury mechanism	Signs of life* on arrival	EDT	Level of recommendation
Penetrating—thoracic	Present	Yes	Strong
	Absent	Yes	Conditional
Penetrating—extra-thoracic	Present	Yes	Conditional
	Absent	Yes	Conditional
Blunt	Present	Yes	Conditional
	Absent	No	Conditional

*Organized cardiac motion or pericardial effusion on cardiac ultrasound, cardiac electrical activity, spontaneous respirations or movement, measurable blood preasure, pupillary reactivity

the primary survey, which functions to immediately identify and correct abnormal physiology.

1. Primary survey.

   Airway: Air exchange, voice, patency, cervical immobilization.

   Breathing: breath sounds present, chest wall motion, neck veins.

   Circulation: mentation, blood pressure, hemorrhage control, pulses.

   Disability: Glascow Coma Score (GCS) calculation, extremity gross motor function.

   Exposure: removal of clothing, log-rolling patient to identify location of all injuries and determine trajectory of penetrating injuries.

Immediate Life-Threatening Thoracic Injuries

Specific life-threatening injuries will be identified during the primary survey of a chest trauma patient. These immediate threats to life must be recognized and addressed. These threats include problems with the airway (obstruction, tracheobronchial tree injury), breathing (tension and open pneumothorax), and circulation (massive hemothorax, cardiac tamponade) [7].

1. Airway obstruction: Inability to exchange gas due to vomitus/blood or foreign body in the pharynx or larynx.
   (a) Causes: direct laryngeal injury, blood or emesis in airway, airway swelling, posterior clavicular head dislocation, penetrating neck trauma.
   (b) Diagnosis.
   - Clinical: air hunger, use of accessory muscles, oropharynx inspection revealing foreign body or vomitus/blood, lack of air movement at mouth, stridor, change in voice quality, crepitus, hemoptysis.
   - Imaging: none required.

   (c) Treatment: clearance of airway (suction, finger sweep), intubation, clavicular head reduction.
2. Tracheobronchial tree injury: Most commonly occurs 1 in. distal to carina, often results in death at the scene due to asphyxiation.
   (a) Causes: rapid deceleration, direct laceration.
   (b) Diagnosis.
   - Clinical: hemoptysis, cervical subcutaneous emphysema, associated tension pneumothorax, pneumopericardium, cyanosis.
   - Imaging: tension pneumothorax on CXR or thoracic ultrasound, visualization of injury with bronchoscopy.

   (c) Treatment: intubation of unaffected side (often challenging due to anatomic distortions), immediate operative repair. A double lumen endotracheal tube or bronchial blocker may be helpful.
3. Tension pneumothorax: Parenchymal lung injury that creates a "one-way" valve of air escaping into the pleural space, which compresses lung parenchyma and shifts mediastinal structures, impairing central venous return.
   (a) Causes: penetrating or blunt chest trauma, positive pressure ventilation with simple pneumothorax, iatrogenic injury, tracheobronchial injury.

(b) Diagnosis.
- Clinical: air hunger, absent lung sounds, tracheal deviation, lack of chest rise, hemodynamic collapse, hyperresonance to percussion over lung fields, hypoxia.
- Imaging: large pneumothorax with mediastinal shift and tracheal deviation on CXR (Fig. 3.1), absence of lung sliding with thoracic ultrasound [12].

(c) Treatment: needle decompression (14-gauge angiocatheter in fourth to fifth intercostal space, anterior to mid-axillary line) [13] or finger thoracostomy, followed by tube thoracostomy (described later in chapter).

4. Open pneumothorax: Large chest wall injury (>3 cm) that creates a "sucking" chest wound that pulls air into the thoracic cavity and collapses the lung.
   (a) Causes: blunt or penetrating chest trauma, often impalement injury.
   (b) Diagnosis.
   - Clinical: tachypnea, hypoxia, large wound on visual inspection of chest, distended neck veins, decreased breath sounds.
   - Imaging: pneumothorax on CXR, thoracic ultrasound.
   (c) Treatment: occlusive dressing secured on three sides to create a flutter valve (Fig. 3.2), tube thoracostomy on affected side away from wound site.
5. Massive hemothorax: Rapid accumulation of >1500 cc of blood into the thoracic cavity causing compression of lung parenchyma and shifting of mediastinal structures.
   (a) Causes: injury (often penetrating) to hilar structures or great vessels, intercostals.
   (b) Diagnosis.
   - Clinical: dullness to percussion over lung field, absence of breath sounds, hemorrhagic shock.
   - Imaging: white out on CXR, homogeneous echogenic effusion or anechoic area in the pleural space on thoracic ultrasound [12].
   (c) Treatment: tube thoracostomy, operative intervention (>1500 cc blood or >200 cc/h for ≥4 h or hemodynamic collapse after thoracostomy or failure to adequately drain hemothorax with placement of two well-positioned chest tubes).
6. Cardiac tamponade: Heart compression due to fluid in pericardial sac that impedes venous return and cardiac output.
   (a) Causes: penetrating wound medial to nipples (anterior) or scapulae (posterior), cardiac chamber rupture (blunt).

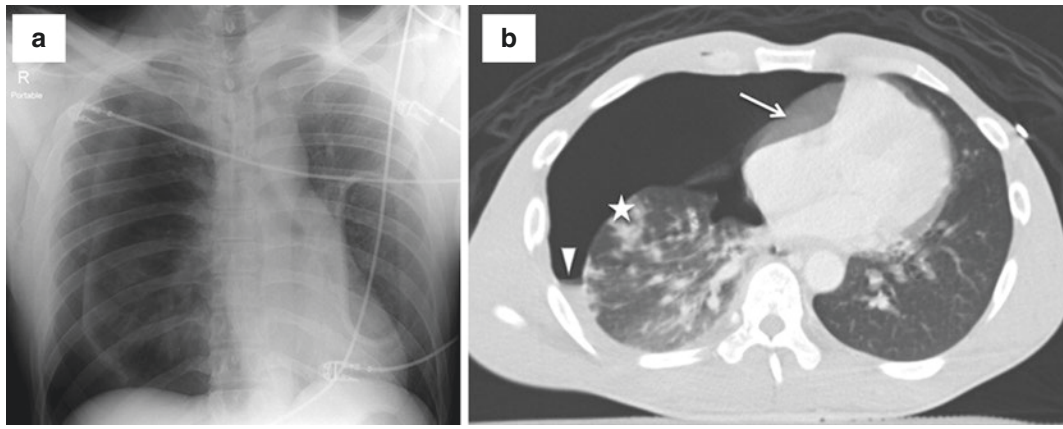

Fig. 3.1 Anteroposterior CXR (**a**) and axial computed tomography angiography (CTA) image (**b**) of large right-sided tension pneumothorax with mediastinal shift after stab wound. Arrow denotes mediastinal shift, arrowhead denotes hemothorax, white star denotes lung contusion. *From*: Nicolaou S. Emergency and Trauma Radiology, An Issue of Radiologic Clinics of North America, E-Book. Elsevier Health Sciences; 2015. 305 p.; with permission

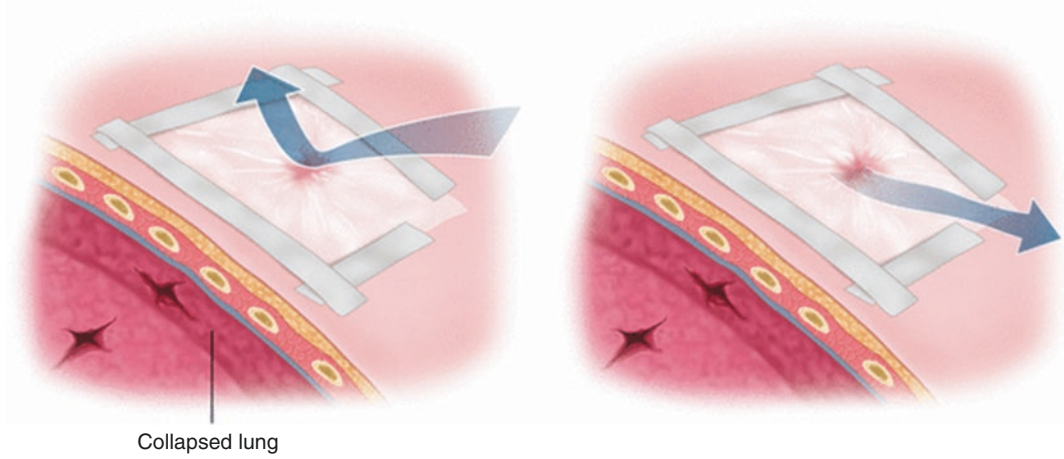

Fig. 3.2 Three-sided dressing for open pneumothorax wound. Dressing prevents continued air sucking into the chest cavity on inspiration, allows air to escape on expiration to prevent development of tension pneumothorax. *From:* Glaser JJ, Rodriguez CJ. Open Chest Wounds and Flail Chest. In: Ganti L, editor. Atlas of Emergency Medicine Procedures. New York, NY: Springer New York; 2016. p. 159–162. Available from: https://doi.org/10.1007/978-1-4939-2507-0_25; with permission

(b) Diagnosis.
- Clinical: distended neck veins, muffled heart sounds, hypotension, paradoxical rise in venous pressure with inspiration (Kussmaul sign).
- Imaging: cardiomegaly on chest X-ray, pericardial fluid visualized on focused assessment with sonography for trauma (FAST) exam.

(c) Treatment: resuscitative thoracotomy/sternotomy, pericardiocentesis in centers without surgical capability and prolonged transport to centers with surgical capability, pericardial window.

Primary Survey Adjuncts

In tandem with the primary survey, vascular access should be obtained. Preferentially two large-bore peripheral venous catheters should be placed, however interosseous infusion and/or central venous access may be necessary depending on the clinical situation. Additional adjuncts, including continuous electrocardiogram (ECG) monitoring and pulse oximetry should be utilized. Baseline laboratory panels should be drawn including a pregnancy test for all females of childbearing age and a type and screen for cross-matching of blood products. Urinary and gastric drainage catheters are also often utilized as well [7].

A supine anteroposterior CXR should be obtained as an adjunct to the primary survey in all chest trauma patients, as it can help diagnose or exclude life-threatening thoracic injuries. CXR should be assessed for lung expansion, pulmonary infiltrates or contusions, hemothorax or pneumothorax, mediastinal widening, apical capping, blunting of the aortic knob, diaphragm elevation, aberrant course of a nasogastric tube if present, and bony fractures. Any of these findings should raise suspicion for thoracic injury and prompt additional workup or intervention. Additional radiographs (head, neck, abdomen, pelvis, extremities) should be obtain depending on the constellation of injuries.

The FAST exam is an additional primary survey adjunct that greatly aids in identifying life-threatening injuries. It consists of four views: right upper quadrant (perihepatic), left upper quadrant (perisplenic), pelvic (retrovesical/retrouterine), and pericardial (subcostal/subxiphoid) (Fig. 3.3). The pericardial view is >90% accurate

in identifying pericardial fluid [7]. The pericardial FAST can be falsely negative when associated a left hemothorax, as lacerations of the pericardial sac with an associated cardiac injury can decompress into the thoracic cavity [14].

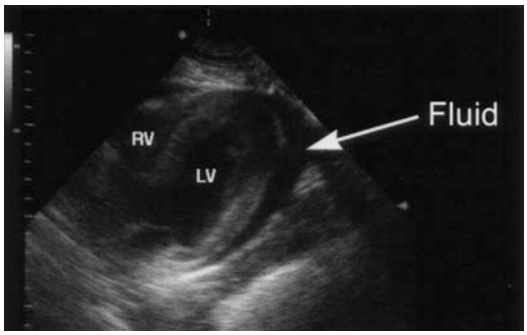

Fig. 3.3 FAST exam positive for pericardial fluid in the subcostal view. LV signifies left ventricle, RV signifies right ventricle, fluid with white arrow represents blood in pericardial sac. *From:* Chelly MR, Margulies DR, Mandavia D, Torbati SS, Mandavia S, Wilson MT. The Evolving Role of FAST Scan for the Diagnosis of Pericardial Fluid: J Trauma Inj Infect Crit Care. 2004;56(4):915–7; with permission

Thoracic ultrasound (i.e., extended FAST) has been more consistently incorporated in the initial evaluation of the trauma patient to identify pneumothorax and hemothorax [15], and may be more sensitive than CXR at identifying occult pneumothoraces [15]. Extended FAST (eFAST) can be performed using the same abdominal probe (2 MHz to 5 Mz curvilinear) used for the other FAST exam components to maximize efficiency. The anterior thoracic view is obtained by placing the probe at the midclavicular line in the third or fourth intercostal space with the patient in the supine position. The probe should be oriented longitudinally. The visceral and parietal pleural should be visualized sliding as they move against each other in a normal chest (Fig. 3.4). The sliding artifact is also called the "ants marching" sign as it looks like an army of ants marching along the white pleural line. Lack of sliding indicates air in the space, suggesting pneumothorax [16]. A lung point (the site where the lung adheres to the parietal pleura adjacent to the pneumothorax) is another highly specific ultrasound finding that indicates pneumothorax [17].

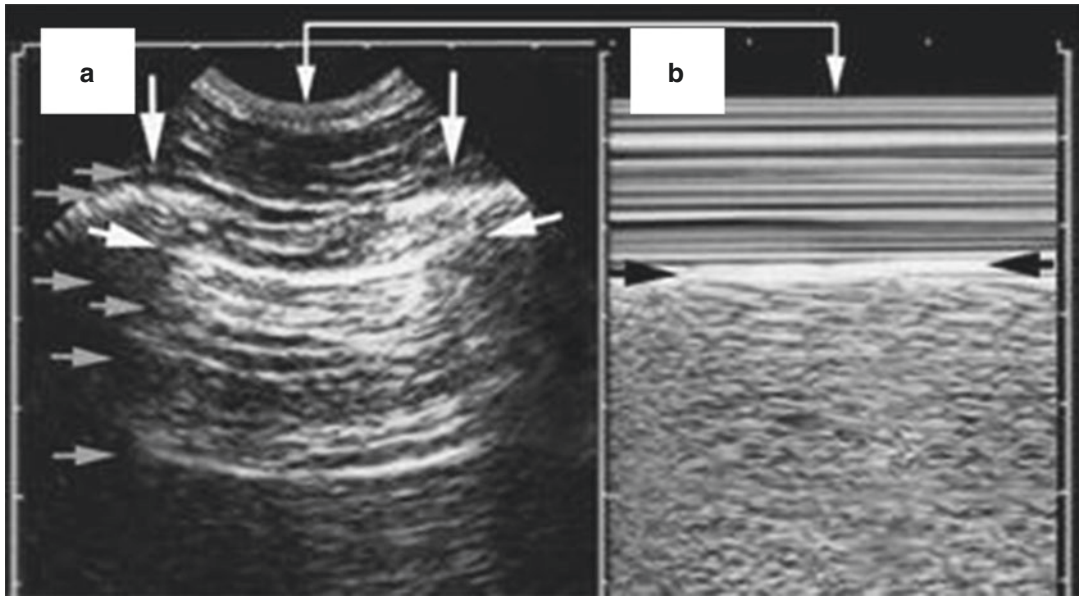

Fig. 3.4 Panel **a** shows the pleural line (horizontal arrows) with overlying ribs (large vertical arrows) and skin surface (small vertical arrow). Panel **b** shows the pleural line (black horizontal arrows) and illustrates a "seashore sign," a homogeneous sandy pattern below the pleural line generated by lung sliding. *From:* Lichtenstein DA. The A-profile (normal lung surface): 2. Lung sliding. In: Lichtenstein DA, editor. Lung Ultrasound in the Critically Ill: The BLUE Protocol. Cham: Springer International Publishing; 2016, p. 67–78. Available from: https://doi.org/10.1007/978-3-319-15371-1_10; with permission

Secondary Survey

The secondary survey includes a head-to-toe physical examination. Additional goals of the secondary survey include a thorough review of existing medical conditions, medications and other pertinent information that is necessary in determining treatment. AMPLE (allergies, medications, past illness, last meal, events related to the injury) is a helpful pneumonic to quickly and efficiently obtain this critical information [7].

1. Physical exam.

   For patients with chest trauma, examination of the torso and neck are of particular importance. Examination of the torso includes visual inspection, palpation, auscultation and percussion. Auscultation should be performed high on the anterior chest wall to detect pneumothorax, and at the lung bases to detect hemothorax. Auscultation of the heart can identify distant heart sounds that could be indicative of cardiac tamponade. For patients with blunt chest injury, particular attention is paid to palpation of the entire chest cage including the clavicles, ribs, and sternum for step-offs, bony prominences, or pain that may indicate fractures. Visual inspection of chest rise can help identify flail chest or pneumothorax. Contusions and hematomas can indicate underlying pulmonary or cardiac injuries. For penetrating trauma, inspection of the chest wall should include specification of the number and location of penetrating wounds. Some institutions apply paper clips or other markers to the site of penetrating injuries to best identify them on imaging studies and to help establish trajectory. This is often done during the primary survey in patient with penetrating chest trauma. Examination of the neck should assess for subcutaneous emphysema or tracheal deviation that could indicate tension pneumothorax. Distended neck veins can help identify cardiac tamponade.
2. Additional imaging.

   For those patients hemodynamically stable enough for additional imaging considerations, contrast-enhanced helical computed tomography (CT) has emerged as a cost-effective modality in comparison to historical diagnostic modalities (conventional angiography, endoscopy, pericardial window or pericardiocentesis) [18]. Penetrating thoracic injuries should be evaluated with a CT angiogram (CTA) of the chest and abdomen to assess transdiaphragmatic injuries [19]. CT can detect pneumothorax, hemothorax, pneumopericardium, pericardial effusion, pericardial and myocardial injury. It can also be used to determine trajectory and identify foreign bodies (Fig. 3.5) [19]. CTA has replaced conventional angiography for diagnosis of vascular injuries, and the use of three-dimensional and multiplaner reformatted images have increased accuracy [19]. CT is more sensitive than CXR at identifying pneumomediastinum from aerodigestive tract injuries as well as and tracheal ring or wall fractures.

Other Thoracic Injuries

Many thoracic injuries that are not immediately life-threatening but require intervention are identified during the primary and secondary survey. Some examples include flail chest, pulmonary contusion, blunt cardiac injury, traumatic aortic or diaphragmatic disruption, and esophageal rupture [7]. A high index of suspicion and appropriate imaging adjuncts help to quickly identify these injuries.

1. Flail chest: Three or more consecutive rib fractures in ≥2 places, which can alter chest wall mechanics (Fig. 3.6) by causing paradoxical chest wall motion [20] or costochondral separation of one rib from the thorax [7].
   (a) Causes: Blunt trauma.
   (b) Diagnosis:
   - Clinical: hypoxia, pain, paradoxical chest wall motion, overlying chest wall injury, bony step-offs, splinting.
   - Imaging: Three consecutive rib fractures in ≥2 places on CXR or CT, underlying pulmonary contusion.

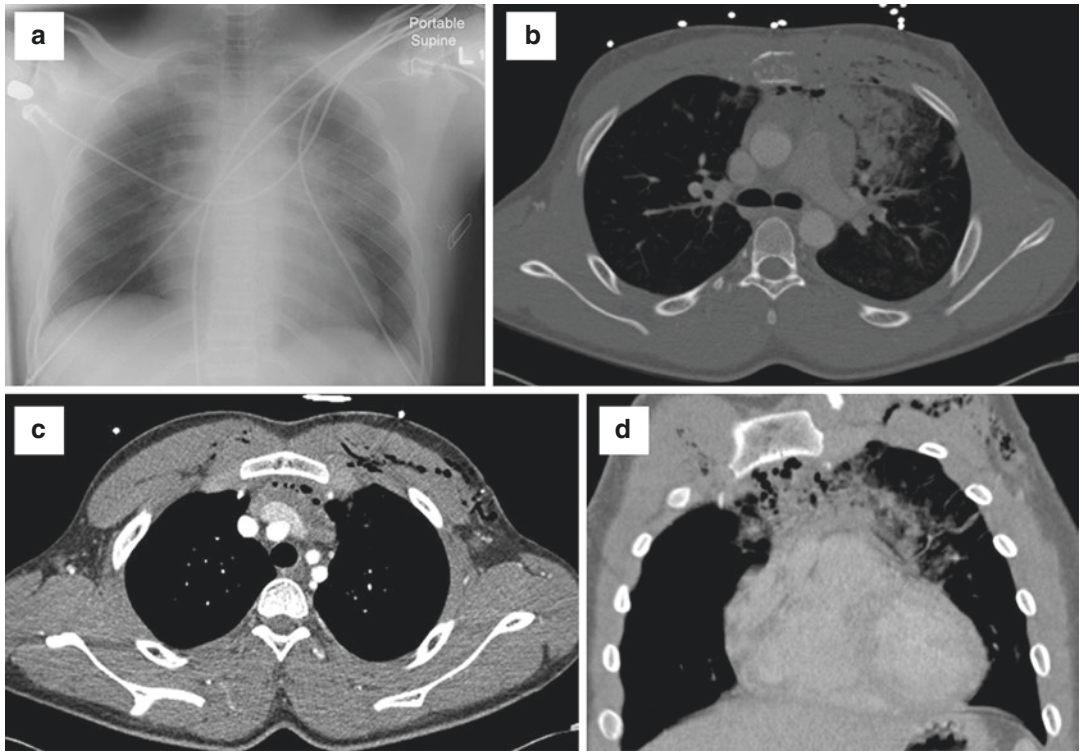

Fig. 3.5 Left chest wall gunshot wound. CXR (**a**) with paperclip left torso demarking wound. CTA images (**b**–**d**) show transmediastinal trajectory with lung contusion, pneumomediastinum, anterior mediastinal hematoma. *From:* Nicolaou S. Emergency and Trauma Radiology, An Issue of Radiologic Clinics of North America, E-Book. Elsevier Health Sciences; 2015. 305 p.; with permission

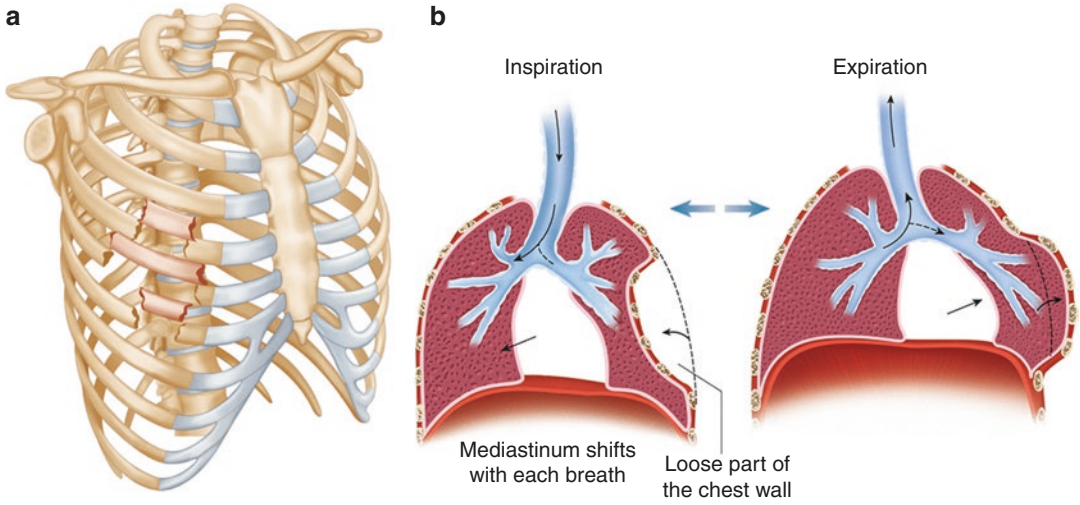

Fig. 3.6 Illustration of flail chest injury (**a**) with associated chest wall physiology (**b**). *From*: Tiwari A, Nair S, Baker A. The Pathophysiology of Flail Chest Injury. In: McKee MD, Schemitsch EH, editors. Injuries to the Chest Wall: Diagnosis and Management. Cham: Springer International Publishing; 2015; p. 19–32. Available from: https://doi.org/10.1007/978-3-319-18624-5_3; with permission

(c) Treatment: humidified oxygen and pulse oximetry monitoring, limited fluid resuscitation, intubation for respiratory failure (SaO_2 < 90%), multimodal pain control, aggressive chest physiotherapy, surgical fixation for severe cases with inability to wean from mechanical ventilation [21] (see Chap. 5).

2. Pulmonary contusion: Lung parenchymal disruption of alveolar-capillary membrane with accumulation of blood and fluid in the lung tissue, often associated with overlying rib fractures.
   (a) Causes: blunt trauma.
   (b) Diagnosis:
      • Clinical: hypoxia, respiratory failure, pain, splinting.
      • Imaging: overlying rib fractures, often associated with flail segments.
   (c) Treatment: humidified oxygen and pulse oximetry monitoring, limited fluid resuscitation, intubation for respiratory failure (SaO_2 < 90%), multimodal pain control, aggressive chest physiotherapy [21].
3. Blunt cardiac injury: Myocardial muscle contusion which can result in coronary artery dissection or rupture, valve or cardiac chamber rupture.
   (a) Causes: motor vehicle collision, falls from high heights, pedestrians struck by vehicles.
   (b) Diagnosis:
      • Clinical: dysrhythmia (sinus tachycardia most common), ST changes, ischemia, heart block, overlying sternal fracture.
      • Imaging: sternal fracture on CXR or CT, wall motion or valvular abnormalities on echocardiogram.
      • Laboratory: elevation of troponin I.
   (c) Treatment: ECG in all patients with suspected blunt cardiac injury, troponin I measurement, continuous ECG monitory and admission if abnormal, echocardiogram with hemodynamic instability (Fig. 3.7), management of dysrhythmia per Advanced Cardiac Life Support (ACLS) protocols [21].
4. Traumatic aortic disruption: Rupture of thoracic aorta near the ligamentum arteriosum due to shearing forces resulting in hematoma contained with the adventitial wall of the aorta or the within the mediastinum. Adventitial rupture occurs at an unpredictable rate (within seconds to years later). Approximately 80% of aortic transections die before reaching the hospital [22].
   (a) Causes: high-impact deceleration blunt force (motor vehicle crash, fall from great height).
   (b) Diagnosis.
      • Clinical: chest pain, intrascapular pain, seat belt or steering wheel lacerations, new-onset murmur, upper extremity hypertension with lower extremity hypotension, tracheal deviation, first, second rib or scapular fracture.
      • Imaging: left hemothorax, widened mediastinum, left mainstem bronchus depression, apical cap, esophageal deviation to the right, left hemothorax, left subclavian hematoma, aortopulmonary window obliteration on CXR, active aortic contrast extravasation, pseudoaneurysm, luminal filling defects, periaortic hematoma on CTA [22].
      • Aortic injury grade (based on imaging findings).
         – Grade 1: intimal tear.
         – Grade 2: intramural hematoma within vessel wall.
         – Grade 3: pseudoaneurysm formation.
         – Grade 4: complete wall rupture.
   (c) Treatment: blood pressure and heart rate control with beta-blockade, surgical repair (thoracic endovascular aortic repair (TEVAR) as standard of care vs. open repair when anatomy for TEVAR not feasible).
5. Diaphragm injury: Rupture of the diaphragm, which most commonly occurs on the left pos-

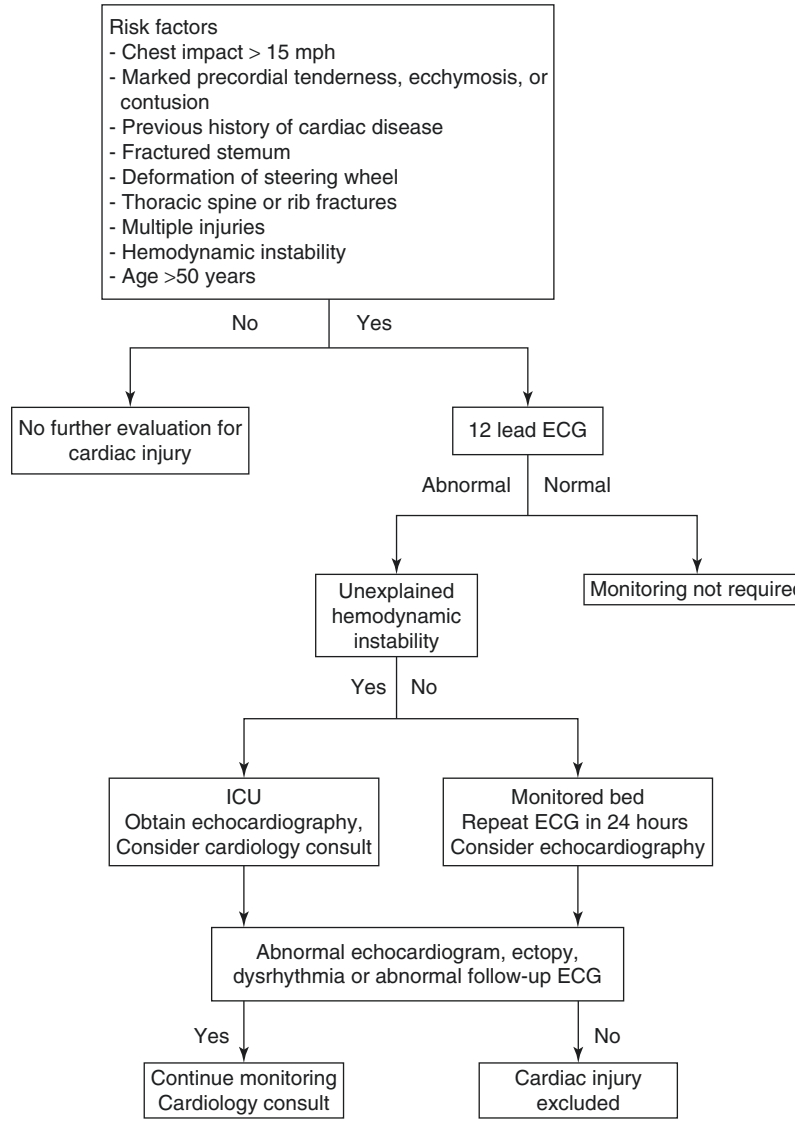

Fig. 3.7 Blunt cardiac injury risk factors and diagnostic algorithm. *From:* Peitzman AB, Yealy DM, Fabian TC, Schwab CW. The Trauma Manual: Trauma and Acute Care Surgery. 5th Edition. Philadelphia: Wolters Kluwer; 2020. 35: Thoracic Injury; p. 407; with permission

terolateral surface and is often associated with intrabdominal injury and herniation of intrabdominal organs into the thoracic cavity (Fig. 3.8). Often a delay in diagnosis as injury may enlarge over time and result in herniation.

(a) Causes: blunt injury causes larger radial force tears, penetrating injury causes direct lacerations which are often smaller, easy to miss and enlarge over time.

(b) Diagnosis.
- Clinical: herniation of intrabdominal contents, hemothorax, thoracic location of nasogastric tube, dyspnea, chest pain.
- Imaging: thoracic location of nasogastric tube, intrathoracic bowel gas, diaphragmatic elevation with lower lobe atelectasis on CXR, herniation of abdominal organs, "hourglass" sign (waist-like stricture of a partially herniated organ including stomach or bowel loop [23] on CT scan.

(c) Treatment: operative visualization via laparoscopy, thoracoscopy or open exploration remains the mainstay of diagnosis

and treatment as this injury can be easily missed and requires a high index of suspicion. Diaphragm repair is accomplished using nonabsorbable interrupted horizontal mattress sutures. Reduction of hernia contents may require thoracotomy.

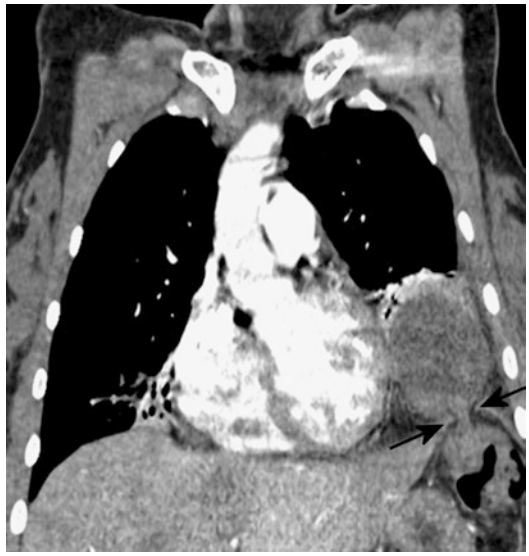

Fig. 3.8 Contrast-enhanced CT image of "hourglass" sign of herniated left colon through diaphragmatic injury (black arrows). *From:* Oikonomou A, Prassopoulos P. CT imaging of blunt chest trauma. Insights Imaging. 2011;2(3):281–95; with permission

6. Esophageal rupture.
   (a) Causes: direct laceration, forceful vomiting, blunt injury to lower sternum/epigastrum, hyperextension, vertebral body fracture.
   (b) Diagnosis.
      - Clinical: subcutaneous emphysema, hemoptysis, odynophagia, dysphagia, hoarseness, hematemesis, peritonitis (distal esophageal injury), mediastinal crunch on auscultation (Hamman's sign).
      - Imaging: subcutaneous air in the neck, left hemothorax, pneumomediastinum on CXR or CT, extravasation of oral contrast into the pleural spaces on CT.
   (c) Treatment: esophagoscopy required for visualization and anatomic location of injury, broad spectrum antibiotics, surgical repair (extend myotomy to expose full extent of injury, two-layer repair with inner layer absorbable suture, outer layer nonabsorbable suture) (Fig. 3.9).
      - Cervical esophagus: neck exploration with incision along anterior border of sternocleidomastoid muscle.
      - Thoracic esophagus: right posterolateral thoracotomy at fourth intercostal

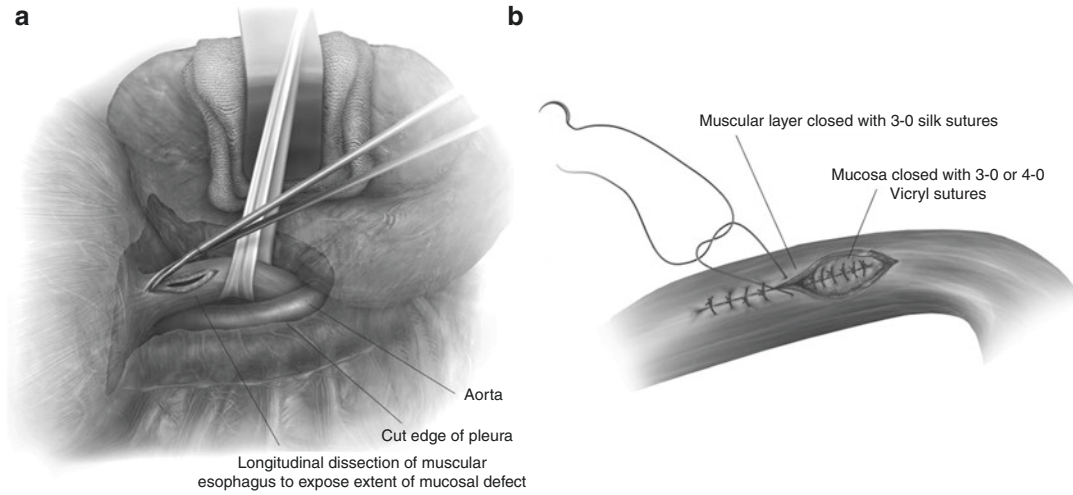

Fig. 3.9 Exposure of the thoracic esophagus via left posterolateral thoracotomy (**a**) with extension of myotomy using electrocautery to visualize full extent of mucosal injury. Two-layer repair (**b**) of thoracic esophageal injury with inner absorbable and outer nonabsorbable suture. *From:* Sancheti MS, Fernandez FG. Surgical Management of Esopahgeal Perforation. Oper Tech Thorac Cardiovasc Surg. 2015;20(3):234-50; with permission

space (proximal), left posterolateral thoracotomy at sixth intercostal space (distal).

- Emergent esophagectomy with diverting cervical esophagostomy and wide drainage if mediastinitis present may be required if repair not feasible, but is rarely necessary.
- Endoluminal esophageal stent placement for intrathoracic perforations <6 cm has become increasingly utilized for perforations secondary to Boerhaave's syndrome. Use of endoluminal stenting in trauma remains limited as there are often associated injuries that mandate exploration [24].

Trauma Bay Procedures

1. Needle thoracostomy.

   Needle thoracostomy (needle decompression) is performed for immediate decompression of life-threatening tension pneumothorax. It is often performed in prehospital setting or in the trauma bay on initial recognition of tension physiology. It involves insertion of a 14-gauge angiocatheter (5–8 cm in length) in the mid-axillary line at the fourth or fifth intercostal space (Fig. 3.10). It should be inserted over the top of the rib to avoid injury to the neurovascular bundle that runs under the ribs. This is the same site that is recommended for tube ad finger thoracostomy (described below). Recent studies have shown higher rates of successful entry into the pleural cavity in this position as compared to the midclavicular line [13, 25]. The skin at the insertion area should be cleaned with an alcohol or iodine-based solution prior to insertion. In its newest edition (2018), the ATLS Student Course Manual has formally adopted the recommendation to perform needle thoracostomy in the mid-axillary line. Alternatively, the needle can be inserted in the second intercostal space in the midclavicular line. An 8 cm angiocatheter is recommended for decompression at the second or third intercostal space as it has been shown to more effectively penetrate the full thickness of the chest wall than the 5 cm catheter in this position [26]. Tube thoracostomy must follow needle thoracostomy.
2. Finger thoracostomy.

   Needle thoracostomy may fail to improve the clinical condition of patients with massive hemothorax. Needle thoracostomy may also fail if the catheter becomes kinked in the chest wall, preventing air from venting through the catheter lumen. Some indications for finger thoracostomy in the prehospital setting include pneumothorax in a patient undergoing

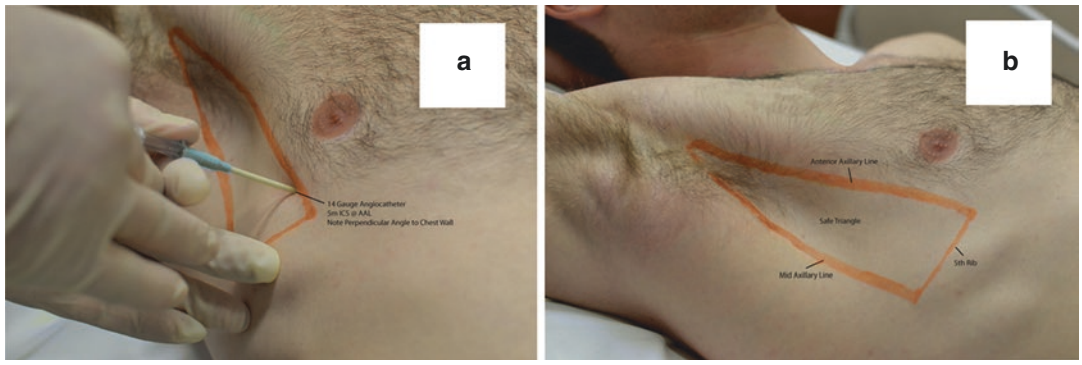

Fig. 3.10 Safe landmarks for needle, finger and chest thoracostomy (**a**) with insertion of angiocatheter (**b**) in fourth intercostal space anterior to mid-axillary line for needle thoracostomy. *From:* Greene C, Callaway DW. Needle Thoracostomy for decompression of Tension Pneumothorax. In: Taylor DA, Sherry SP, Sing RF, editors. Interventional Critical Care: A Manual for Advanced Care Practitioners. Cham: Springer International Publishing; 2016. p. 171–8. Available from: https://doi.org/10.1007/978-3-319-25286-5_19; with permission

positive pressure ventilation, traumatic cardiac arrest or periarrest, and unexplained hypoxia or hypotension in a ventilated patient [27]. Finger thoracostomy, using the same landmark of the fourth or fifth intercostal space in the mid-axillary line, can increase the effective evacuation of the chest and improve physiology quickly.

After the chest is prepped and draped in a sterile fashion, a 3–5 cm incision is made at the fourth or fifth intercostal space anterior to the mid-axillary line with the arm abducted. In patients where the ribs are not easily palpable, the incision site should be in a horizontal line with the level of the nipple. Local anesthetic should be infiltrated at the site prior to incision if the clinical scenario allows. Blunt dissection is then used to dissect down to the pleural space over the top of the rib. The pleural cavity is then entered bluntly, and a finger is inserted to sweep the chest cavity to aid in fluid evacuation and to feel for adhesions to the chest wall.

3. Chest thoracostomy.

Tube thoracostomy refers to the placement of a tube into the pleural cavity to drain air, blood, or infected fluid. Tube placement should be at the fifth intercostal space just anterior to the midaxillary line. Position the patient so their arm is extended over the head if possible or abducted from their side. Prep and drape the chest in the usual sterile fashion when possible, ensuring landmarks including the inferior aspect of the axilla, the inframammary crease, and nipple are visible. When time permits, use local anesthetic at the site (10–20 mL of 1% lidocaine) beginning at the skin surface and advancing down through the subcutaneous tissue to the pleural space and periosteum. Begin by making a small (3–5 cm) incision at the fifth intercostal space just anterior to the mid-axillary line and palpating the top of the rib (Fig. 3.11). In patients where the ribs are not easily palpable, the incision site should be in a horizontal line with the level of the nipple. Bluntly dissect with a Kelly clamp or finger down to the parietal pleural. In a controlled fashion, puncture the parietal pleura over the top of the rib space to avoid the neurovascular bundle that runs on the underside of the rib. Insert a finger into the space and perform a sweeping motion to ensure no lung parenchyma is adhesed to the chest wall. Insert the chest tube into the pleural cavity, aiming for the posterioapical aspect of the thoracic cavity. Use of a Kelly clamp on the end of the tube may help guidance into the proper position. Rotating the tube during advancement may also help guide it into preferred position. Advance the chest tube until all side holes are within the pleural cavity. Attach the tube to a Pleurovac container set to −20 mmHg suction and assess the container for evacuation of air and fluid and proper tidaling. Secure the chest tube in place using nonabsorbable suture. Obtain a chest X-ray to confirm proper placement.

The optimal size chest tube for emergent thoracostomy is a moving target. Traditionally smaller-bore (28–32Fr) tubes have been recommended for pneumothorax evacuation, while larger-bore tubes (36–40Fr) were recommended for hemothorax evacuation. Recent evidence has shown equivalent rates of hemothorax evacuation with the smaller (28–32Fr) tubes [28]. The newest ATLS edition now recommends 28–32Fr tube size for all tube thoracostomy procedures [7]. Some studies have even shown successful pneumothorax evacuation with smaller-bore tubes (14–22Fr) in the emergent setting [29, 30], however this has not become standard practice at most trauma centers.

There are currently no clear recommendations for timing and duration of antibiotics in the setting of chest thoracostomy. The EAST 1998 practice management guidelines supported use of a first-generation cephalosporin at the time of tube insertion, not to extend beyond 24 h from time of thoracostomy as there was some evidence to support a decrease in pneumonia with periprocedural antibiotics [31]. A 2012 EAST practice management update, however,

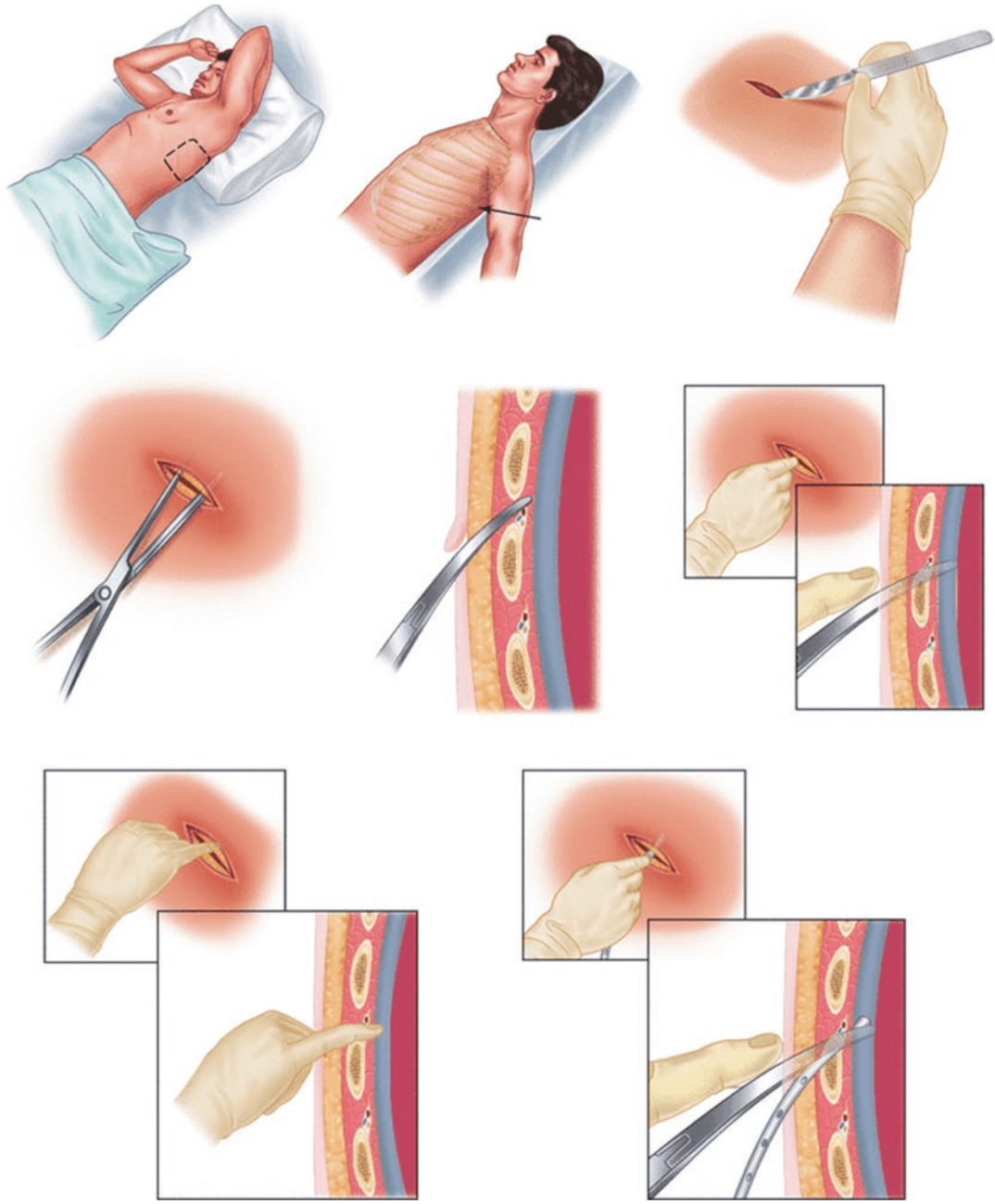

Fig. 3.11 Tube thoracostomy procedure. *From:* Allen BR, Ganti L. Chest Tube Thoracostomy. In: Ganti L, editor. Atlas of Emergency Medicine Procedures [Internet]. New York, NY: Springer; 2016 [cited 2020 Apr 26]. p. 149–53. Available from: https://doi.org/10.1007/978-1-4939-2507-0_23; with permission

concluded that there is insufficient evidence to recommend for or against periprocedural antibiotics in association with reduced incidence of pneumonia or empyema [32]. The practice at our trauma center is to administer one dose of antibiotics (cefazolin as first-line, clindamycin if known allergy) at the time of thoracostomy [33].

Resuscitation Algorithms

1. Penetrating thoracic injury.
   (a) Fully expose patient.
   (b) Role early (before traditional ABCD of ATLS protocol) to identify all penetrating injuries and begin to determine trajectory and anatomic injuries.
   (c) Address life-threatening injuries as listed above.
      • Consider empiric tube thoracostomy if imaging not rapidly available.
   (d) Continue with primary survey and adjuncts including CXR and FAST.
      • Neck, abdominal and pelvic X-rays as indicated.
   (e) Classify patient as hemodynamically stable or unstable.
      • Unstable: proceed directly to operating room (Fig. 3.12).
         – Transmediastinal injury with hemodynamic instability.
         – >1500 cc blood evacuated after tube thoracostomy.
         – Cardiac tamponade present.
         – Large undrained hemothorax after two well-placed chest tubes.
      • Stable: secondary survey, CTA chest and abdomen.
   (f) Monitor for delayed decompensation requiring intervention.
      • >200 cc/h blood for 2–4 h or retained hemothorax with well-positioned chest tubes.
      • Repeat CXR to identify occult pneumothorax.
      • Consider diagnostic laparoscopy/thoracoscopy for identification of diaphragm injury.

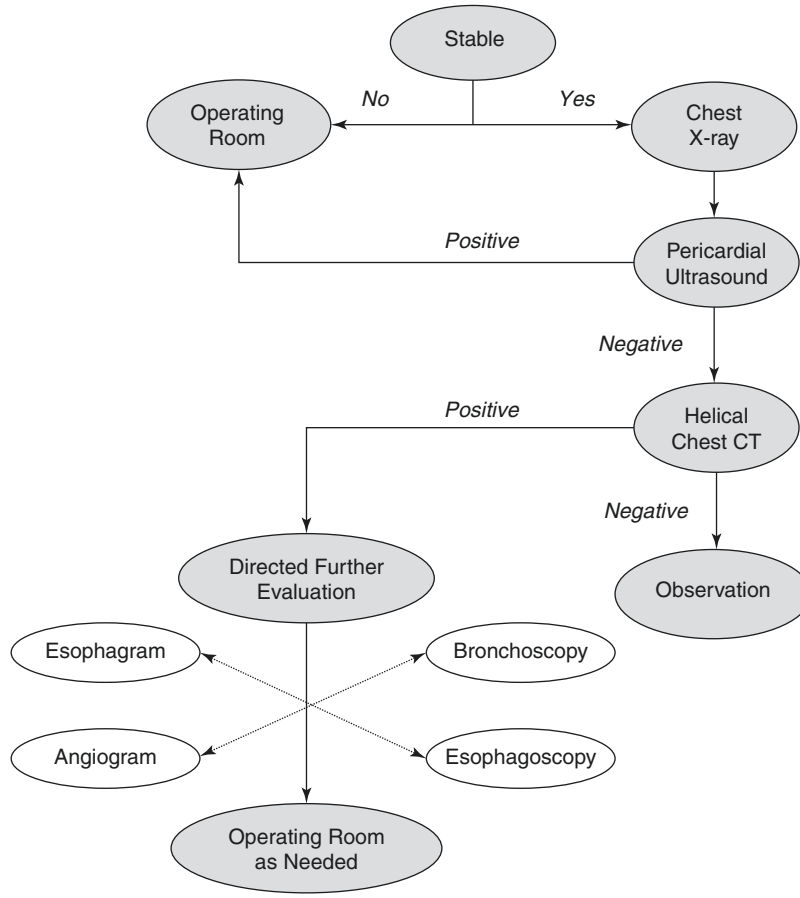

Fig. 3.12 Transmediastinal gunshot wound evaluation algorithm. *From:* Stassen NA, Lukan JK, Spain DA, Miller FB, Carrillo EH, Richardson JD, et al. Reevaluation of diagnostic procedures for transmediastinal gunshot wounds. J Trauma. 2002;53(4):635–8; with permission

2. Blunt thoracic injury.
   (a) Primary survey.
   (b) Adjuncts (CXR, FAST) with ECG and troponin lab work for blunt cardiac injury.
   (c) Secondary survey.
   (d) Consider additional imaging (CTA chest/abdomen, echocardiogram) based on mechanism and injury pattern.
   (e) Operative intervention for specific injuries.
      - Esophageal rupture.
      - Diaphragm injury.
      - Traumatic aortic disruption.
      - Large air leak/tracheobronchial tree injury identified.
   (f) Nonoperative management care bundle: respiratory support, analgesia, chest physiotherapy, appropriate monitoring.
      - Surgical fib fixation as potential adjunct.

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Nonoperative Rib Fracture Management

4

Vincent Butano, Adam Greenwood, Babak Sarani, and Paul Dangerfield

Introduction

Rib fractures are the most common form of chest injury. It is estimated that they are present in roughly 10% of trauma admissions [1]. These patients, if not adequately treated, can develop progressive respiratory failure and pneumonia, and can die. The likelihood of these adverse events increases as the severity of the chest wall injury and the patient's age increase. In Bulger et al.'s seminal paper regarding the mortality and morbidity of rib fractures in trauma patients, they describe a twofold increase in morbidity in patients over 65 years of age and a 20% increase in mortality per rib fracture [2]. Holcomb et al. went on to describe that patients as young as 45 years of age with four or more rib fractures have an increase in morbidity [3]. Thus, it is imperative that these patients are identified and treated expeditiously.

Inadequate pain control resulting in poor pulmonary hygiene is the most common reason that patients with rib fractures develop the severe complications noted above. Rib fractures are uniquely challenging because the rib cage, as a whole, cannot be mechanically stabilized as may be done in extremity fractures (e.g., casting or splinting). Since the pain recurs with each breath, the rib fracture patient instinctively seeks to minimize the depth and force of breathing to minimize pain. Thus, in order to mitigate the risk of respiratory failure, pain control serves as the cornerstone of nonoperative management of rib fractures. As discussed below, a multimodal approach to pain control has a much higher efficacy and lower side effect profile than a regimen centered around narcotics. In this chapter, we will discuss current data regarding evaluation of the chest wall injury patient and nonoperative management.

Initial Evaluation

When carrying out the initial assessment of a patient with chest wall injury, the clinician should pay particular attention to the respiratory rate and depth of inhalation (tidal volume). Qualitative factors, such as whether or not the patient is able to speak in full sentences and whether or not the patient is able to move his or her body without splinting/stopping respiratory effort, can provide significant insight into the degree of pain present and probability of respiratory embarrassment.

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Use of accessory muscle or paradoxical movement of the chest (suggesting flail chest injury) increases the likelihood of respiratory failure. The past medical history, with particular attention paid to chronic heart or lung disease, can further augment the clinician's risk assessment for respiratory failure.

Imaging is paramount in the management of rib fractures. Although a chest X-ray is the most frequently ordered initial imaging test, it lacks sufficient sensitivity or resolution to allow for insightful assessment of the severity of chest wall injury present. Livingston et al. studied a cohort of 388 patients with rib fractures and found that 208 of these patients had an initial chest X-ray read of no fracture [4]. As such, patients who are deemed to be at risk for having severe chest wall injury require CT scan imaging of the chest.

Because the chest CT scan allows for excellent resolution of the nature of the rib fractures present, it is the test upon which various scoring systems for quantification of the probability of respiratory failure following rib fractures are based. As an example, one such scoring system, the RibScore, was developed by Chapman et al. to assess the probability for need for mechanical ventilation using these radiographic variables: six or more ribs fractured, bilateral fractures, flail chest, three or more bicortical displaced fractures, first rib fracture, and at least one fracture in each of the anatomic areas (anterior, lateral, and posterior) [5]. The composite RibScore was significantly and linearly associated with pneumonia, respiratory failure, and tracheostomy. This scoring system gives trauma providers objective data on the severity of the rib fractures and can assist with the decision to initiate interventions at an earlier point in their care. As such, scoring systems, such as the RibScore, may be a valuable tool in triaging patients and determining who may benefit from SSRF. Of note, currently no scoring systems has been validated prospectively. As well, although these scoring systems predict respiratory failure due to rib fractures, they have not been used to as a predictive tool to identify patients who may benefit from SSRF.

Care of the Admitted Patient with Rib Fractures

After the initial evaluation and resuscitation, the mainstay of nonoperative treatment is pain control. Early and aggressive pain control combined with respiratory therapy and nursing interventions are crucial to the recovery of rib fracture patients. Nursing and respiratory therapy provide education on pain control, incentive spirometry/deep breathing, and the importance of adequate coughing. Physical therapists assist the patient in maneuvers to increase mobility while reducing pain. Multiple studies have showed that rib fracture clinical pathways incorporating these interventions improve outcomes [6–9]. Todd et al. found that after implementing a clinical pathway, the study group had significantly lower ICU length of stay, hospital length of stay, pneumonia, and mortality [9].

Patients with pulmonary contusions or respiratory illnesses, such as COPD, are at increased risk for decompensation. Pulmonary contusions often worsen over the course of 72 h. It is our practice to keep these patients on continuous pulse oximetry and supplemental oxygen as necessary. Patients developing tachypnea, shortness of breath, or other signs of acute respiratory failure may require a higher level of care, noninvasive ventilation, or possibly mechanical ventilation.

Pain Control: Systemic

In the age of the opioid epidemic, pain control in the trauma patient becomes a fertile ground for utilizing multimodal analgesia and regional anesthesia options. Many commonly used analgesic medications have some opioid sparing effect. Although patients with severe chest wall injury will invariably require opioids, they should not be considered first-line agents. The section below is written using an escalating strategy of a multimodal pain regimen that intentionally puts opioid therapy last.

Polytrauma patients require special considerations. For these patients, it may be difficult to find the one or two most significant sources of pain that regional anesthesia could appropriately address. For example, placing a fascia iliaca nerve block for a hip fracture may reveal a second source of pain that precludes mobility. In these cases, a multimodal systemic approach as described below may be most efficacious.

Acetaminophen

Outside of advanced liver failure, acetaminophen is an analgesic medication with little risk for adverse events. It is a centrally acting cyclogoxygenase-3 (COX 3) inhibitor, which decreases opioid requirements [10]. Intravenous (IV) and rectal variations are available for patients deemed NPO. However, rectal suppositories should be avoided because of their unreliable absorption. Despite its label as a nonsteroidal anti-inflammatory drug (NSAID), it is safe to coadminister with COX1 and COX2 inhibitors. Its use is limited by the Food and Drug Administration (FDA) maximum dose of 3 g/day. The maximum daily dose is lower for elderly or cirrhotic patients. Acetaminophen should be dosed around-the-clock to minimize the need for opioid and breakthrough pain.

Nonsteroidal Anti-inflammatory Drugs

The mechanism of nonsteroidal anti-inflammatory drugs is inhibition of the COX 1 and 2 enzymes. Specific NSAIDs may be selective for a specific COX enzyme or nonselective for either. Celecoxib shows greater affinity for the COX 2 enzyme. As with acetaminophen, NSAIDs show an opioid sparing effect and can reduce the incidence of nausea, vomiting, and sedation [10].

Despite their value, NSAIDs have a variety of side effects and risks that should be considered. Among the most oft-cited are increased risk of bleeding and kidney failure. NSAIDs should be avoided in patients with cardiac disease, renal failure, history of gastric bypass surgery, and history of or current gastrointestinal bleeding. Because celecoxib has a sulfa moiety, it is contraindicated in patients with a sulfa allergy. Along with acetaminophen, NSAIDs should be dosed around-the-clock to minimize need for opioids and the risk of breakthrough pain. As noted above, NSAIDs can be coprescribed with acetaminophen.

Ketamine

Ketamine is an N-methyl-D-Aspartate receptor (NMDA) antagonist used with increasing frequency for both acute and chronic pain [11]. Due to its low cost and relatively safe side-effect profile, ketamine infusions are commonly used by the military in the prehospital setting. Ketamine is increasingly being used in the civilian emergency medical system (EMS) setting, and should be used in the emergency department, operating room, ICU, and general ward. Infusions can be run at a rate of 0.1–0.2 mg/kg of ideal body weight/hour. The dose can be titrated to effective pain control.

While most of the studies in patients with trauma and rib fractures are positive, some studies have suggested mixed results requiring further discussion. A small retrospective study of 30 ICU patients showed lower average and more severe Numeric Pain Scale/Behavioral Pain Scale (NPS/BPS), as well as lower opioid use for patients receiving ketamine [11]. The infusion rate was 0.1 mg/kg/h. The study did not find lower mortality or length of stay. A randomized controlled trial of 91 patients with three or more rib fractures showed that ketamine at an infusion rate of 2.5 mcg/kg/min (0.15 mg/kg/h) did not decrease pain score [12]. A subgroup analysis demonstrated significantly decreased opioid use at 24 h and 48 h in patients with an Injury Severity Score > 15, suggesting its efficacy in more severely injured patients. Secondary outcomes of length of stay, overall oral morphine equivalents, pulmonary complications, and adverse outcomes were not statistically different. The studies vary considerably in design and more research is required to show which patients will benefit from ketamine infusions.

An obvious consideration is patient tolerance of side effects, which become more apparent at higher doses. Infusions can be started

without bolus doses to mitigate the possibility of adverse effects and titrated to desired effect. Ketamine has cardiac depressant effects that should be considered in patients with preexisting cardiomyopathy. It can cause dysphoric reactions that should be considered in patients with preexisting psychiatric disorders. The dysphoric reaction can be mitigated with low dose benzodiazepine. Ketamine has been shown to decrease the incidence of post-traumatic stress disorder in severe burn and trauma patients, though the mechanism of action for this endpoint is not known [13]. At the authors' institution, ketamine infusion is a commonly used adjunct for pain control on the medical/surgical ward and its use does not necessitate continuous monitoring; however, patients receiving ketamine are seen on a daily basis by the Acute Pain Service.

Lidocaine

Lidocaine's mechanism of analgesia as an IV infusion is not fully described; however, current research suggests it plays a role in limiting priming of the pro-inflammatory polymorphonuclear granulocyte (PMN). Priming a PMN increases the response to inflammatory mediators and therefore increases the overall inflammatory response to pain [14]. Increased inflammation leads to an increase in sensitivity to and perception of pain. Possible additional benefits are due to lidocaine's ability to prevent pain beyond the initial insult. In a rat model, lidocaine blocks the excitatory response in wide dynamic range (WDR) neurons, which are involved in the transition from acute to chronic pain in the spinal cord [15]. Further studies are needed to elucidate whether this is an actual or theoretical benefit to patients.

Lidocaine infusions can be run at a rate of 0.5–1.5 mg/kg of ideal body weight/hour, and the drug can be coprescribed with any of the aforementioned adjuncts. Contraindications to IV lidocaine infusions include cardiac conduction abnormalities, concurrent regional anesthetic administration, as well as recent administration of liposomal bupivacaine (Exparel™). Exparel package information states that IV lidocaine should not be given for at least 96 h from time of Exparel dosing. Care should be used in elderly patients and those with impaired hepatic function. Aside from its analgesic properties, studies suggest lidocaine may have pro-motility effects, especially following bowel surgery [14]. If not available as an IV infusion, topical lidocaine patches may be administered, although efficacy is not well established in rib fractures.

Overall, lidocaine has proven effectiveness in acute pain management and can decrease opioid requirements. Combined ketamine and lidocaine infusions, with or without concomitant use of acetaminophen and/or NSAIDs, may be especially beneficial in patients who have contraindications to regional anesthesia.

Gabapentinoids

Gabapentinoids, such as Lyrica and Gabapentin, work by binding to the Alpha-2-Delta subunit of the voltage sensitive presynaptic calcium channels to reduce the release of excitatory neurotransmitters. Gabapentinoids have been shown to significantly reduce neuropathic pain. Gabapentin has been shown to be beneficial in thoracic and abdominal surgery and is a part of pain control protocols at many institutions [16, 17]. A meta-analysis of studies examining Gabapentin found a 35% reduction in total opioid consumption in the first 24 h after surgery [18]. These studies have made these compounds increasingly popular as an adjunct for pain control.

The use of gabapentinoids in thoracic surgery has led to interest in utilization for pain control in rib fractures as they have similar pain pathways. Rib fractures can be associated with a mixed pain picture. The fracture and associated muscle and soft tissue disruption causes somatic pain. Intercostal nerve damage or disruption can cause neuropathic pain.

There has been controversy regarding gabapentinoids efficacy in rib fractures. A recent double-blind, randomized study compared gabapentin to placebo in patients with rib fractures [19]. The study found no difference in numeric pain scores, opioid consumption, respiratory rate, oxygen requirements, or incentive spirometry

between the groups. Unfortunately, gabapentinoids are probably not effective in the management of patients with rib fractures unless there is a clear neuropathic component to the pain and should be used on a case by case basis.

The most common side effect of gabapentinoids is sedation. Other common adverse effects include dizziness, fatigue, abnormal gait, rash, blurry vision, and cough. There is an associated increased risk of respiratory depression and death in patients who are taking gabapentinoids and opioids. This is more likely to be seen at higher doses and in elderly patients with underlying obstructive sleep apnea or pulmonary dysfunction. Slow uptitration is required to avoid this complication. At our institution, we begin at 100 mg TID and increase as needed.

Alpha-2 Agonists

Alpha-2 agonists, including clonidine and dexmedetomidine, have analgesic properties. Clonidine, which is more commonly used as an antihypertensive agent, has the potential for rebound hypertension when discontinued abruptly. Dexmedetomidine is a commonly used sedative in the ICU setting, but its cost and risk of bradycardia limits its use as an analgesic agent.

Opioids

Opioids exert their effects through the mu-opioid receptor and are very effective in the acute setting for pain control. However, this class of medications has a significant side-effect profile including dose-dependent respiratory depression, constipation, nausea, and pruritus. The risk of respiratory depression is particularly concerning in patients who are at risk of respiratory failure due to rib fractures. Equally well-established is the relatively quick onset of tolerance, in which patients require an increasing dose to achieve the same analgesic effect. Physical dependence and the possibility of addiction may come later, but these concepts should always remain firmly in the thought process of all opioid prescribers. The Center for Disease Control (CDC) currently recommends that for acute pain, immediate release opioids should be started at the lowest effective dose and utilized for the shortest amount of time necessary. The CDC goes on to say that 3 days is often sufficient and more than 7 days is rarely needed [20].

Opioids can be prescribed in either oral and IV regimens, with the latter also offering the option of continuous or intermittent dosing. Patient-controlled analgesia (PCA) devices have been shown to improve patient satisfaction but patients should be transitioned to oral regimens as soon as possible.

At the authors' institution, continuous opioids are never prescribed (including intubated patients) and opioids are never used as a solitary regimen for pain control. Patients are transitioned as soon as possible from intravenous dosing to oral, and the opioid is the first drug to be curtailed as the overall multimodal pain control regimen is advanced.

Pain Control: Regional

Regional anesthesia is an increasingly attractive option for analgesia in trauma patients as it decreases the requirements for opioids and the associated systemic effects. Considerations for all of the following regional techniques include location of injury (e.g., anterior or posterior), number of rib fractures, therapeutic or prophylactic anticoagulation and their timing, uni- or bilateral fractures, NPO status, and the feasibility of leaving nerve block catheters. Regional techniques should always be cross-referenced with the most current American Society of Regional Anesthesia and Pain Medicine (ASRA) guidelines regarding appropriate timing of block around the time of anticoagulation. The guidelines must be verified both prior to the block placement and prior to removal of catheter if catheter is used.

Patient Selection for Regional Anesthesia

Not all patients with rib fractures will require regional intervention. According to one review, younger patients and patients with three or less rib fractures and nonsevere internal injuries may be managed with multimodal regimens outlined

above, but this conservative treatment plan should be reconsidered if the patient's condition deteriorates. Regional anesthesia should be used for patients aged 65 or older, those with 6/10 or higher pain at rest, four or more rib fractures, weak cough, or FVC of 15 ml/kg or less [21].

Epidural

Thoracic epidural analgesia (TEA) involves depositing local anesthetic into the epidural space. The technique is used frequently in thoracic and upper abdominal surgery and provides excellent pain control to bilateral chest walls. TEA can be associated with sympathetic blockade, hypotension, urinary retention, and potentially decreased patient mobility [22]. Absolute contraindications include infection at the intended site, severe coagulopathy, uncorrected hypovolemia, and patient refusal. Relative contraindications include less severe coagulopathic conditions, anticoagulation, sepsis, elevated intracranial pressure, and aortic stenosis. These should be weighed against benefits.

The Eastern Association for the Surgery of Trauma (EAST) 2016 guidelines for Pain Management of Blunt Thoracic Trauma recommend the use of an epidural for analgesia over nonregional modalities [23]. According to the guidelines, this recommendation is made despite low quality evidence citing the priority for patient preference of analgesia over weak and mixed results over length of stay, ventilator days, and mortality. Although TEA offers excellent pain relief, it also requires ongoing hospitalization and can impede mobility. Both factors contribute to prolonged hospital length of stay and can be associated with morbidity. As well, placement of a TEA can be technically challenging resulting in suboptimal pain relief.

Paravertebral

The paravertebral nerve block (PVB) involves injection of local anesthetic in the paravertebral space, which communicates with the intercostal and epidural space. A single catheter may provide analgesia for five to six dermatomal layers. Some authors recommend a second catheter for bilateral rib fractures or for unilateral fractures involving six or more ribs [22].

PVB have some advantages over TEA. PVB placement is technically easier to perform. Unlike with thoracic epidurals, patients with PVB catheters can be discharged home. These patients also have decreased risk of hypotension, urinary retention, and lower extremity weakness when compared to thoracic epidurals. As such, the authors' institution strongly prefers PVB placement to TEA for management of pain related to rib fractures.

Myofascial Plane

The following techniques have been described as potential alternatives to TEA and PVB. Their use is not widespread and there is ongoing debate regarding their efficacy [24]. Both are considered myofascial blocks, similar to the transversus abdominis plane (TAP) block.

Serratus Anterior

The serratus anterior plane (SAP) block is an option only for rib fractures in the anterior two-thirds of the chest wall. Potential benefits include ease of positioning (allowing for both supine or sitting), post-procedure patient mobility, and the possibility of performing the block in patients on anticoagulation [22].

Current reviews of the technique suggest a possibility of pneumothorax as well as a higher absorption of local anesthetic comparable that of an intercostal block.

Erector Spinae

The erector spinae plane (ESP) block can potentially cover the anterior, lateral, and posterior chest wall. It also may be used in anticoagulated patients. Ability to position the patient appropriately may present a challenge as the technique requires the patient sitting or lateral; however, the risk of pneumothorax is lessened with the technique [22].

Intercostal

The intercostal nerve block is performed between the ribs at the level of the rib fracture. The tech-

nique sometimes requires injections above or below the fracture and may require multiple injections. It is a simple block and may be performed by landmark or with ultrasound. The block is associated with a relatively high level of local anesthetic systemic absorption and, if single shots, may require multiple injections that may be uncomfortable to patients [22].

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5 Operative Rib Fracture Management

Alexander C. Schwed and Fredric M. Pieracci

Introduction

Rib fractures are common following blunt thoracic trauma. They are estimated to be present in 10% of admissions to trauma centers following blunt trauma [1], and are the most common injury following blunt chest trauma [2]. These injuries pose a legitimate morbidity and mortality risk, with some estimates of mortality that range from 12 to 20% [3, 4] and an overall complication rate of 33% or greater [5]. More recent data has further illustrated the significant mortality and morbidity that these injuries pose, especially in older adults, who are more likely to develop respiratory failure, pneumonia, and death as compared with younger adults [1]. The attendant risks of these injuries to younger patients should not be discounted, however; both the short-term [6] and long-term [7] outcomes of these injuries in all patients can be burdensome, and the successful recovery from these injuries can heavily depend on treatment rendered during the acute hospitalization [8–11].

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The spectrum of injury of rib fractures varies considerably, and the definitions of chest wall injury have undergone revision over the past few decades. Historically, flail chest was defined as three or more ribs fractured in two distinct places representing perhaps the most serious type of injury. This definition has undergone critical revision, and now is better defined as either "flail segment" which represents two or more consecutive ribs fractured in two or more places and "flail chest" which is a flail segment that causes paradoxical motion on inspiration [12]. Many prior studies have considered flail chest to be an indication for operative intervention, and the early randomized control trial data to support surgical stabilization of rib fractures (SSRF) included only patients with flail chest [13–15]. Flail chest is estimated to be present in as many as 7% of patients with rib fractures [8], and carries a mortality risk as high as 33% [16]. Concomitant underlying pulmonary contusion can further exacerbate these injuries; contemporary series have identified very high rates of mechanical ventilation (61%), ICU admission (84%), and pneumonia (22%), as well as longer time in the ICU and the hospital as compared to patients without underlying pulmonary contusions [17]. It should be noted, however, that even nondisplaced rib fractures contribute to poor outcomes in patients following blunt chest trauma. The pain from rib fractures can impede clearance of pulmonary secretions and can increase the likelihood of pneumonia and respiratory failure in the

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short term and can contribute to long-term chronic pain and opioid dependence in the long term [18].

Surgical stabilization of rib fractures (SSRF) has historically been underutilized in the treatment of severe rib fractures, which is likely polyfactorial. Early interest in the operative repair of flail chest and significant chest wall deformities following trauma is first described in the 1950s [19], and thereafter there was a decade or two of interest in external traction devices and percutaneous approaches [19]. Prior to this, the general treatment of rib fractures was nonoperative and sometimes involved splinting or casting as means of rib immobilization; as would be expected, these efforts were generally unsuccessful and often lead to pulmonary complications [19]. Despite advances in the medical technology, there has been a general under-utilization of SSRF even in severe flail chest, with a 2014 evaluation of the National Trauma Databank indicating that only 0.7% of all patients with flail chest had undergone SSRF between 2007 and 2009 [17]; a more recent analysis of similar data indicates that 5.8% of patients with flail chest have undergone SSRF between 2008 and 2014 [20].

The aims of this chapter are to review the operative indications, operative technique, and operative complications of SSRF. Our goal is to describe in detail how SSRF is performed at our institution with recommendations about patient selection, preoperative planning, intraoperative strategy, and postoperative management. We will also describe what is presently known as to the longer-term outcomes following SSRF in patients with blunt chest trauma and attempt to summarize the best-available evidence in support of SSRF for patients with complex rib fractures, flail chest, or both.

Operative Indications

The bulk of early evidence and experience with SSRF was in patients with flail chest. Indeed, the first three randomized trials of operative versus nonoperative management of rib fractures were performed exclusively in patients with flail chest [13–15]. More recently, another prospective trial undertaken by Liu and colleagues examined SSRF versus nonoperative management in 50 patients admitted to their hospital between 2015 and 2017 and, echoing prior authors' work, found that patients undergoing SSRF had a shorter duration of mechanical ventilation, shorter ICU stay, and a lower risk of ARDS or pneumonia [21]. Only recently have nonflail chest rib fracture patterns in patients who undergo SSRF been studied; the NONFLAIL trial undertaken by Pieracci and colleagues evaluated SSRF in patients with three or more ipsilateral, displaced rib fractures, in addition to pulmonary physiologic derangement following optimal locoregional analgesia, and compared operative versus nonoperative management in these patients. These authors demonstrated significantly lower pleural-space complications in the surgically managed group as well as better self-reported pain scores 2 weeks following hospitalization [22]. This work, as well as increasing experience in SSRF in general, and SSRF in expanded patient populations, have contributed to a greater understanding of patient selection and operative indications in patients who will benefit most from SSRF.

As with any surgical undertaking, patient selection for SSRF is paramount. Indeed, as has been demonstrated in a few smaller studies, SSRF may not be able to overcome certain magnitudes of underlying pulmonary contusion or concomitant injuries. Farquhar et al. retrospectively compared 19 patients undergoing SSRF with 36 case-matched control and were not able to demonstrate an improvement in hospital length of stay, ICU length of stay nor duration of mechanical ventilation [23]. It should be noted that 100% of patients undergoing SSRF had underlying pulmonary contusion while only 58% of the nonoperatively managed control patients had contusions. In addition, it is not clear from these authors' descriptions of their technique if these patients undergo routine bronchoscopy and video assisted thoracoscopic surgery (VATS) washout of the chest as has become routine at our institution. Similar retrospective reviews have also demonstrated no significant difference between operatively and nonoperatively man-

aged patients [24]. These smaller works stand in distinction to an ever-growing body of literature that supports SSRF in patients with flail chest [4, 8–10, 25, 26]; in addition, the Eastern Association for the Surgery of Trauma (EAST) has recently published a practice management guideline that conditionally recommends operative rib fracture fixation over nonoperative management to decrease mortality, shorten duration of mechanical ventilation, and decrease ICU length of stay and hospital length of stay, rate of pneumonia, and need for tracheostomy [27]. In combination, this recent change from EAST as well as mounting evidence and experience have helped popularize SSRF as a meaningful way of helping patients overcome blunt chest trauma and rib fractures.

Following the three early randomized controlled trials that focused solely on flail chest patients [13–15], our own institution and others have worked to expand the role for SSRF in patients with rib fractures without a flail pattern. We first described this expanded paradigm in 2015 and included patients with either: acute respiratory insufficiency despite optimal medical therapy; uncontrolled pain despite optimal medical therapy; or anticipated chronic pain/impaired pulmonary mechanics [28]. These indications have been simplified and refined, and currently are best described as follows: flail chest; three or more severely displaced ribs (bicortical displacement); 30% or greater hemithorax volume loss as measured on CT scan; or any displaced fracture pattern with failure of nonoperative management [29]. Following this, a multicenter, prospective, controlled trial of operative fixation in patients with rib fractures but without flail chest (the NONFLAIL trial) was undertaken and has helped reinforce and disseminate these expanded criteria [22]; it is our hope that additional centers will continue to use SSRF not only in flail chest patients but in this expanded cohort who, we believe, will benefit from operative intervention. In addition, it should be noted that the decision to perform SSRF relies heavily on surgical judgement and critical evaluation of the patient's physiology and how the patient is coping with their injuries. Though there are numerous clinical scoring systems that attempt to quantify response to locoregional analgesia and pulmonary toilet, our own institution relies on the sequential clinical assessment of respiratory function (SCARF) score to help guide clinicians in the evaluation of patients following blunt thoracic trauma [30]. Regardless of the method used, it is paramount that the surgeon undertake a thorough investigation of the patient's present physiologic status and determine the role that SSRF can play in helping that patient recover from their injuries.

Just as operative indications have expanded over the past decade as more work has gone into investigating the exact role that SSRF can serve in the management of blunt thoracic trauma, it is important to delineate operative contraindications to SSRF. Patients who are hemodynamically unstable, are unable to lie flat or be positioned prone or in lateral decubitus (including patients with intracranial hypertension, or with unstable cervical, thoracic, or lumbar spine injuries, or with external fixation hardware in place that would preclude appropriate positioning), patients with higher priority injuries (descending aortic injury, for example), prolonged ventilator-dependent respiratory failure unrelated to rib fractures, and patients under 18 years of age are generally excluded from consideration for SSRF at our institution. It is important to consider that patients with a present contraindication may become candidates for operation after a day or 2 in the hospital. The timing of SSRF has been controversial, though the most recent evidence supports attempting SSRF as early as possible in patients with blunt thoracic trauma, and ideally within 48 h [31]. In addition, patients may be able to tolerate multiple operations within a single anesthetic and could, in theory, undergo fixation of an unstable spinal fracture followed by SSRF assuming the patient's physiology would be tolerant of such an undertaking.

Age in and of itself is not an operative contraindication to SSRF. At our institution, we do not offer SSRF to patients under 18 years of age given concerns about physical development of the rib cage and the need for permanently implanted hardware. Elderly patients, on the

other hand, have at times been excluded from consideration for SSRF given concerns about their tolerance for thoracic surgery, despite well-described increased risks of mortality among this cohort [1]. Recent work by Zhu et al. helps dispel this misperception. These authors analyzed the National Trauma Data Bank for all geriatric patients undergoing SSRF and with a propensity-matched analysis demonstrated decreased mortality among patients undergoing SSRF [32]. In addition, though these authors did find an increased hospital length of stay and increased rate of tracheostomy in all patients undergoing SSRF, those who underwent fixation earlier had decreased rates of pneumonia, tracheostomy, and overall length of stay [32], findings which are congruent with others authors' experiences [31].

Operative Technique

The rationale for SSRF applies traditional orthopedic surgical principles of open reduction and internal fixation to the ribs. Because ribs are unique bony structures in the body in that they are flexible, twisted and conical, and because they are designed to move with respiration, operative fixation presents several challenges [33]. Traditional fixation devices such as mandibular or long bone plates are too rigid and prevent the natural excursion of the chest wall during respiration. Nonoperative immobilization of the chest is not feasible, and the pain of rib fractures directly impedes full chest excursion, thereby increasing the risk of poor clearance of secretions, impaired pulmonary toilet, atelectasis, and eventual pneumonia and possible respiratory failure. SSRF affords not only the realignment of displaced rib fractures but also the opportunity for the operating surgeon to provide a "thoracic tune-up" by combining multiple procedures in the operating room under one general anesthetic. That is to say, SSRF allows surgeons to intervene in multiple ways for patients with severe rib fractures, including pulmonary toilet via bronchoscopy, use of some form of locoregional analgesia (e.g., directed bupivacaine injection, pain catheter, or erector spinae block), and thoracoscopic interrogation and washout of the chest. The improved outcomes of this surgical intervention have been described in some series, including the reduced likelihood of empyema and hemothorax [34], and improved self-reported pain scores [22]. These described advantages, as well as additional opportunities for "tuning up" patients following blunt chest trauma, may, in part, explain the recent increase in interest and use of this surgical technology [35].

As described above, patient selection is an essential component of the successful use of SSRF technology in the management of patients with rib fractures. Patients without obvious contraindications to surgery should be thoughtfully evaluated by the operating surgeon with careful review of CT imaging to help with operative planning. Recent work by the Chest Wall Injury Society to help codify the language and taxonomy of rib fractures [12], with subsequent validation of this work [36] in its application to clinical use helps provide a common language of describing rib fracture location and degree of displacement. All rib fractures between the third and tenth ribs should be evaluated for intervention. The first and second ribs are technically quite challenging to access and, if fixed, are unlikely to provide significant benefit to a patient given the attendant morbidity of accessing the chest wall in that location. Ribs 11 and 12 do not generally participate in the chest wall mechanics of respiration and therefore are also not usually considered for operation. Anterior, lateral, and posterior ribs fractures of ribs three to ten should be considered for operative intervention, keeping in mind that posterior fractures within 3 cm of the transverse process should not be approached given proximity to the spine and likelihood of iatrogenic injury to the ligaments or muscles in this area. Anterior fractures, however, may be approached even if they fall within 3 cm of the costal cartilage or the sternum, as the current iteration of rib fixation plates allow for anchoring of the plates to these locations.

Preoperative planning for patients being considered for SSRF should involve critical assessment of rib fracture locations and operative plan for which ribs will be fixed. We have found that a

preoperative planning checklist used in the operating room can help assure that all rib fractures are assessed and fixed as planned (Figs. 5.1 and 5.2). It can be worthwhile to coordinate with other surgical teams involved in a given patient's care, as SSRF can be combined with other operative procedures, assuming said patient can tolerate multiple interventions at the same time. This can reduce the number of trips to the operating room and can potentially help speed a patient's recovery from complex polytrauma. It is also valuable to coordinate preoperatively with the anesthesia team, not only for questions about airway management and tube size selection but also with regard to intraoperative or postoperative pain management strategies. Ideally, surgical and anesthesia teams can strategize a way of helping provide multimodal locoregional analgesia either pre- or intraoperatively for patients undergoing SSRF.

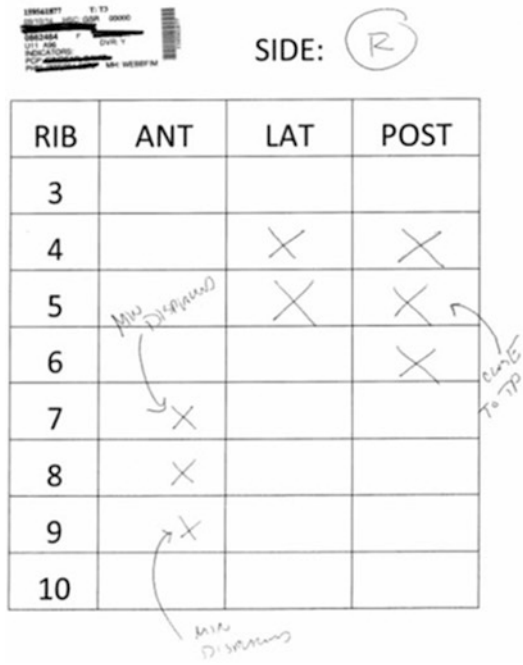

Fig. 5.1 Intraoperative checklist

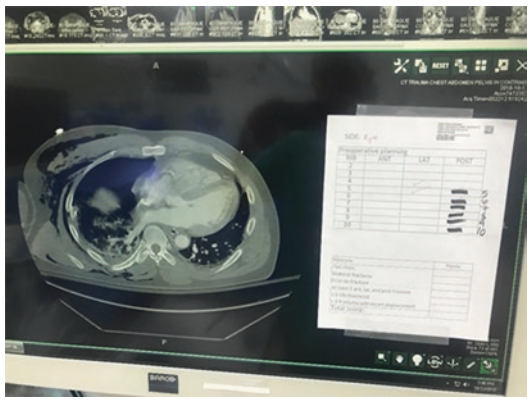

Fig. 5.2 Side-by-side use of intraoperative checklist and CT imaging

Patients selected for SSRF are positioned either prone, in lateral decubitus, or in supine position to access the anatomic location of the rib fractures (Figs. 5.3, 5.4, and 5.5). Intubation with the largest endotracheal tube tolerated by the patient will aid in bronchoscopy. A dual-lumen tube or a bronchial blocker system to allow lung isolation is helpful for the thoracoscopic portion of the procedure and can help protect the underlying lung while hardware is being applied. We and others have described a muscle-sparing technique that minimizes trauma to the soft tissues

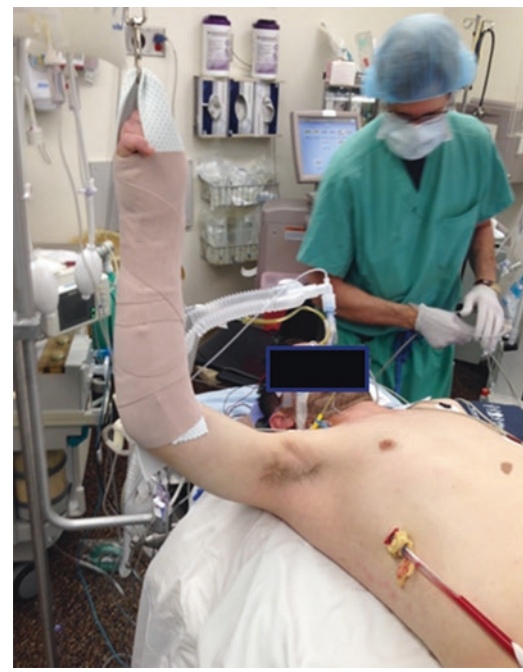

Fig. 5.3 Supine positioning for anterior rib fractures. Arm suspension with candy cane rods can assist in accessing these fractures

Fig. 5.4 Supine positioning for bilateral anterior rib fractures

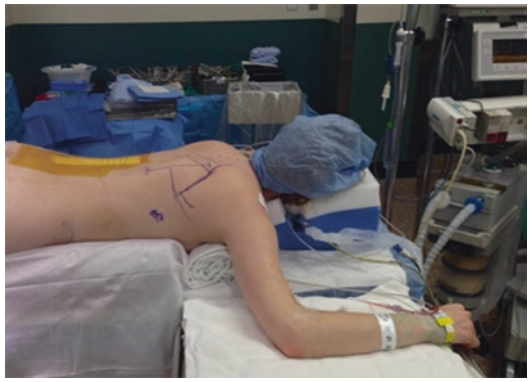

Fig. 5.5 Prone positioning for posterior rib fractures. Note that the arms are suspended below the level of the operating room table to assist in accessing these fractures

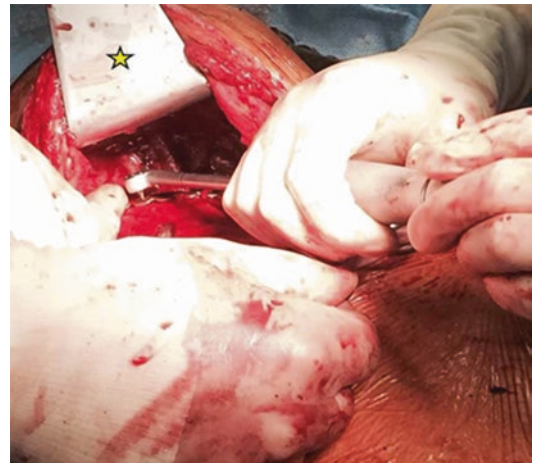

Fig. 5.6 Subpectoral approach for anterior rib fractures. Note the use of the right-angle screwdriver which can assist in accessing anterior fractures of the more superior ribs

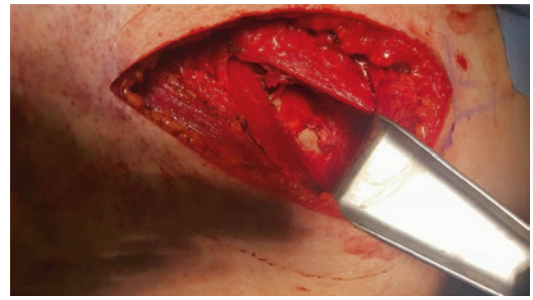

Fig. 5.7 Muscle-sparing approach for lateral rib fractures

and muscles of the chest while still allowing access to the ribs to apply the plates and screws [28, 37]. Anterior rib fractures are generally approached with an inframammary incision that allows the surgeon to reflect the pectoralis major muscle superiorly and to divide or split the fibers of the pectoralis minor (Fig. 5.6). Lateral rib fractures can be accessed via an oblique incision along the anterior border of the latissimus dorsi muscle, which allows the surgeon to raise a muscle flap and thereby preserve the latissimus while separating it from the serratus anterior muscle posteriorly and the subcutaneous fat anteriorly (Fig. 5.7). From there, the serratus muscle can be split while protecting the neurovascular bundle and the lateral rib fractures accessed. Finally, posterior rib fractures are approached with the patient placed in prone position with the arm positioned below the level of the OR table to allow the scapula to move laterally (Fig. 5.5). A longitudinal incision between the tip of the scapula and the spine will allow surgical access to the triangle of auscultation; access to this space will allow muscle-sparing access to the posterior ribs by having the surgeon raise flaps beneath the trapezius and latissimus muscles. We have found the use of a pediatric Bookwalter or an Alexis wound protector can be invaluable aids in retracting the soft tissues and muscles to expose the ribs and can free the surgeon or assistants from manually retracting the muscle flaps (Fig. 5.8).

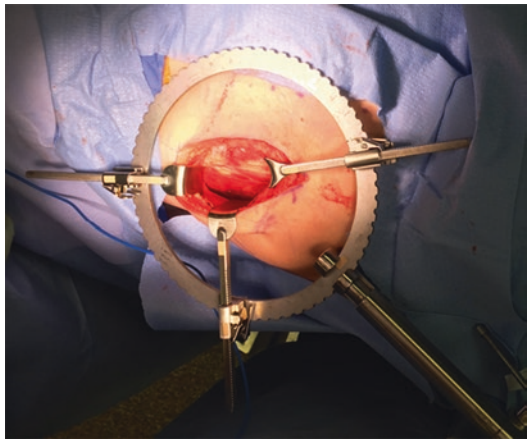

Fig. 5.8 Pediatric Bookwalter retractor to assist in exposure of the ribs for fixation

Once access has been gained to the rib fractures in a muscle-sparing manner, reduction of the fractured segments of rib can be accomplished with the aid of a right angle clamp or other instrument to grasp the ribs. Application of a commercially available plating system is then undertaken, generally with locking screws. There are several commercially available rib fixation systems, none of which has been shown to be superior to another. We generally approach fractures by first securing the furthest screw, which can generally aid in applying the plates flush with the ribs. The available systems differ with respect to recommended number of screws placed on either side of the fracture line and in uni- versus bicortical fixation of the ribs. Regardless of which system is used, we have found the intraoperative use of the preoperative planning tool invaluable in assuring that all fractures identified preoperatively are repaired.

While in the operating room, we have generally undertaken both bronchoscopic evaluation of the airways as well as thoracoscopic evaluation of the pleural space. These two interventions afford the operating surgeon numerous advantages. First, bronchoscopy will allow for aggressive pulmonary toilet and sampling of secretions for microbiologic analysis (if indicated). Thoracoscopy will allow for irrigation and drainage of the pleural space, as well as thoracoscopically guided tube thoracostomy placement and the use of locoregional analgesia. Our own practice has been to thoracoscopically infiltrate the rib fractures with liposomal bupivacaine which we have found provides multiple days of analgesia and can help patients with postoperative pulmonary toilet and mobility. The advantages of thoracoscopic irrigation of the pleural space has been described by other authors [34], and our own experience supports these conclusions.

Postoperatively, patients are managed similarly to other patients undergoing thoracic surgeries. We attempt to aid patients in mobility and pulmonary toilet, and generally try to remove thoracostomy tubes within 48 h of the operating room. A postoperative chest X-ray is routinely obtained in the recovery area to assure that tubes have not dislodged with transport and to obtain a baseline postoperative film to compare should the patient's pulmonary condition change. Preoperative antibiotics are discontinued and chemical VTE prophylaxis is maintained in the postoperative period.

Operative Complications

The overall reported rate of SSRF complications is quite low, and this fact may account for some of the recent increase in use and interest in this technology. Given that SSRF involves the use of implantable hardware, theoretical concerns about hardware infection and removing said hardware have been raised, especially in the setting of infected pleural fluid or open fractures [38]. Despite these concerns, there is relatively robust experience that indicates an overall low rate of hardware infection in patients treated with SSRF. In a prospective controlled study published by Pieracci et al., there was noted to be one patient with a hardware infection managed operatively at postoperative day #24 (1/35 patients = 2.9%) [29]. Similarly, Thiels and colleagues retrospectively reviewed their institutional experience with SSRF over a 5-year period and, in a cohort of 122 patients, observed a 4.1% hardware infection rate, all of whom underwent operative management of their infections [39]. In one of the largest published experiences with

hardware infection, Junker and colleagues identified an overall infection rate of 3.5% among a cohort of 285 patients who underwent SSRF over a 9-year period [38]. These authors also describe successful salvage of infected hardware through the use of antibiotic beads, an orthopedic surgical technique that has successfully been used in other areas of the body but, to our knowledge, has only been described in SSRF by these authors. These findings from single-center experiences are corroborated by a recent meta-analysis of SSRF outcomes undertaken by Peek et al. who report an overall surgical and implant-related complication rate of 10.3% (173/1690), with an overall wound infection rate of 2.2% and a revision surgery rate of 2.9% [40]. In addition to these larger data sets, there are also several case reports of successful use of SSRF in infected fields or with pleural infections without hardware infections that help support the use of this type of hardware for these patients [41, 42].

Beyond concerns about hardware infection, chronic irritation or discomfort from implanted hardware is another concern about implanted devices that are meant to work with a bony structure that is constantly in motion as the ribs participate in chest wall movement and respiration. Overall long-term data is not robust, though there have been reports of need for hardware revision or removal (see section "Long-Term Outcomes") [43]. Furthermore, rib nonunion or hardware failure is another concern about implanting static devices into a bony system that is designed to move with respiration. Sarani and coauthors undertook a retrospective review of more than 1200 patients who had undergone SSRF to identify risk factors for hardware failure. In this cohort, 38 patients were noted to have hardware failure (which these authors defined as migration or fracture of implanted devices) and found an overall 3% failure rate in this cohort. These patients were noted to be asymptomatic 40% of the time, though 55% of these patients underwent operative removal of hardware [44]. This review helps add to the body of literature that demonstrates an overall low complication rate in patients undergoing SSRF for acute rib fractures.

Finally, there are natural concerns about bleeding, postoperative empyema, or postoperative hemothorax when operating in the chest. Despite the fact that SSRF focuses primarily on the extrapleural space, this operation can potential cause bleeding or pleural space infection. Several authors have addressed these concerns. Majercik and colleagues examined their center's 4-year experience with SSRF and measured the rates of retained hemothorax and empyema following SSRF as compared with patients managed medically. These authors describe a retained hemothorax rate of 2.2% in patients undergoing SSRF as compared with 10.2% among patients managed medically, and no empyema in the SSRF group as compared with a 14.3% rate of empyema among medically managed patients [34]. These authors did not note a statistically significant difference in hospital length of stay or ICU length of stay between patients managed surgically or medically, although interestingly patients managed with SSRF were noted to have a greater number of rib fractures [34]. Supporting the conclusions of these authors, Peek et al. in their meta-analysis describe an overall rate of 1.4% risk of intra- or postoperative bleeding [40].

Long-Term Outcomes

As with many newer surgical techniques or technologies, the long-term outcomes of SSRF have not been extensively described. Prior work in many prospective studies [13–15, 29] has, by necessity, focused on short-term outcomes of patients undergoing SSRF as compared with patients managed nonoperatively. These outcomes, such as duration of mechanical ventilation, and ICU or hospital length of stay are important markers of the successful correction of underlying physical and physiologic derangements of patients with rib fractures, but do not necessarily inform discussions about long-term outcomes following surgery. Due to the focus on short-term outcomes among many of the larger series of descriptions of SSRF, the same improved

short-term outcomes are what is demonstrated in many of the systematic reviews that have been conducted to date examining the role of SSRF in the management of rib fractures following blunt trauma [4, 8–10, 25, 26].

There have been a few analyses of longer-term outcomes of patients managed with SSRF, though the overall results are somewhat mixed. Majercik and colleagues reviewed 50 patients managed with SSRF between April, 2010 and August, 2012 and contacted these patients with a follow-up telephone survey at least 6 months following surgery. This survey focused on patient satisfaction, presence of chronic pain, and willingness to recommend SSRF to friends or family [7]. Among patients surveyed, 70% underwent SSRF for flail chest, 20% for fracture displacement and 40% for intractable pain. Among this cohort, SSRF was performed on average 3.4 ± 0.5 days following admission. These authors describe an overall significant rate of satisfaction and resolution of acute pain in the vast majority of their patients, with an average reported abatement of acute pain at 5.4 ± 1 weeks. They do report 2 of the 50 patients surveyed who reported chronic pain and, as could be expected, these patients were also the only ones in the cohort who would not recommend SSRF to friends of family [7]. These results are promising results from a cohort of patients managed relatively early in the recent era of SSRF. That the vast majority of patients reported abatement of pain and overall high levels of satisfaction helps provide evidence to clinicians undertaking preoperative counseling of patients who are considering undergoing SSRF.

Likewise, Caragounis et al. report on a cohort of 56 patients who underwent SSRF for multiple rib fractures or flail chest and contacted these patients serially at 6 weeks, 3 months, 6 months, and 1 year following surgery. In addition to surveying patients as to their pain at rest or during activity and their use of oral pain medicines, these authors also objectively studied patient lung function with pulmonary function testing. These authors describe a significant decrease in the need for pain medicine from 6 weeks to 1 year following surgery, as well as a significant increase in forced vital capacity and peak expiratory flow following surgery [45]. These results help support the belief held among many surgeons who undertake SSRF that the correction of the physical misalignment of the rib cage can help ameliorate immediate and chronic pain as well as improve respiratory mechanics and prevent future complications following blunt thoracic trauma.

These positive results should be tempered with additional reports that indicate a less favorable long-term outcome for SSRF. Marasco and associates report a retrospective analysis of all patients admitted to their hospital with rib fractures between January, 2012 and April, 2015. Of these 1482 patients, 67 (4.5%) underwent SSRF. As compared with patients managed nonoperatively, these authors found that patients undergoing SSRF were older, and had higher abbreviated injury scores (AIS) of the chest and abdomen [46]. Patients were contacted following hospitalization at 6, 12, and 24 months following discharge and, compared with patients managed nonoperatively, these authors report no improvement in quality of life assessments of patients managed with SSRF. No differences were seen between the two groups with respect to return to work or gainful employment following hospitalization [46]. These results should be interpreted with caution, however, as the overall ability of the authors to contact patients was reduced to only 63% at 24 months. In addition, the SSRF cohort was older, sicker, and more injured. They were also much more likely to require mechanical ventilation, ICU admission, or both, and had a longer ICU length of stay compared with patients managed nonoperatively [46]. It would appear that these conclusions are likely influenced by significant selection bias as to who undergoes SSRF at these authors' institution.

Finally, Beks et al. report on the experience of SSRF at a Level One trauma center in the Netherlands. These authors report on the short- and long-term outcomes of 166 patients treated with SSRF over a 6 year period (2010–2016) [43]. Patients were considered for SSRF if they either presented with flail chest or had deranged

physiology (tachypnea or intractable pain) with multiple rib fractures; analysis was conducted on both the entire cohort as well as individual indications (flail chest or multiple rib fractures). Both the short-term and long-term outcomes among these reported cohorts are dispiriting. These authors report a 35% pneumonia rate among the entire cohort, and a 9% mortality among patients with flail chest. Both the multiple rib fracture and the flail chest cohort were noted to have a 3% hardware infection rate in the short-term. Long-term outcomes were reported as measured by incidence of chronic pain or prosthetic irritation, need for hardware removal, and presence of dyspnea. Among patients with flail chest, 21 (53%) reported chronic irritation at the hardware site, with 5 (13%) undergoing hardware removal at an average of 1 year postoperatively [43]. It should be noted that despite these results, the authors conclude that SSRF is well-tolerated, safe, and provides patient satisfaction in the majority of cases. It remains to be seen whether, with increased familiarity with the preoperative selection, intraoperative management, and postoperative care, patients undergoing SSRF presently will have improved long-term outcomes as compared with patients now 5–10 years out from their initial surgery. Future work by those currently undertaking SSRF will hopefully continue to address questions of long-term outcomes.

Conclusions

Operative intervention for rib fractures, though described nearly a century ago, has only recently become more widely practiced and understood. Recent work that has helped clarify and expand the operative indications for this procedure, as well as robust and encouraging evidence about the significant short-term and long-term gains that patients may experience by undergoing SSRF will hopefully help convince other trauma, orthopedic, and thoracic surgeons that SSRF is an important component of the multidisciplinary treatment of patients with rib fractures following thoracic trauma. It is our hope that this operative technique will continue to grow in popularity as we strongly believe that, in the correctly selected patients, SSRF offers significant advantages and helps speed recovery following trauma.

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Flail Chest

6

Silvana F. Marasco and Viju Abraham

Introduction

Traumatic thoracic injuries account for 10% of all hospital trauma admissions and are a frequent cause of trauma related deaths, second only to head injury [1]. Fractured ribs are present in over 20% of patients admitted to trauma centers with blunt chest trauma [2]. Flail chest injury represents the more severe end of the spectrum of rib fracture injury.

Definition

Flail chest injury is defined as the fracture of at least three consecutive ribs in more than one place, leading to a floating segment of chest wall. Flail chest injury can also include the sternum, whereby bilateral rib fractures, on either side of the sternum lead to a floating anterior chest wall [3]. A flail segment is a radiological definition of a floating segment of rib or chest wall and may not necessarily imply physiological changes of a flail chest injury.

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Anatomy

The rib cage consists of two sets of 12 ribs on each side. The first 10 ribs articulate with the vertebrae posteriorly and the sternum anteriorly either directly or via costal cartilages (Fig. 6.1a). The 11th and 12th rib are typically described as "floating" without an attachment to the sternum. Neurovascular bundles lie along the inferior edge of the ribs within a recess in the rib contour.

A typical rib (ribs 3–10) consists of a head which articulates posteriorly with the thoracic spine and a neck which connects to the main shaft or body of the rib. This curves laterally to the anterior end of the rib which articulates with the sternum (in the case of the first seven ribs). Like all bones, the ribs are made up of an outer cortex and an inner medulla which is a sponge like bone with little strength.

The rib head contains two joint facets. The lower facet is vertical and articulates with the upper border of its own vertebra whereas the upper facet faces up to the lower border of the vertebra above. Both facets form a synovial joint with the vertebral bodies (costovertebral joints). The anterior surface of these joints are reinforced by the three bands of the radiate ligament and an

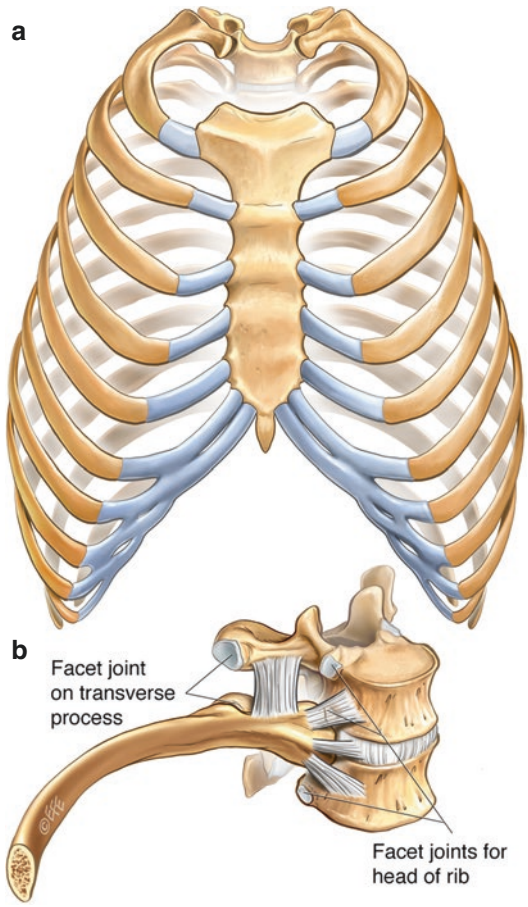

Fig. 6.1 (**a**) Thoracic cage, (**b**) Attachment of rib to vertebra

intra-articular ligament which inserts onto the intervertebral disc (Fig. 6.1b).

The neck of the rib extends from the head to the tubercle which forms a joint with the costal facet of the transverse process. The tubercle consists of two hyaline cartilage-covered facets; the medial facet forms a costotransverse joint with its own vertebra and the lateral facet receives the lateral costotransverse ligament from the tip of its own transverse process.

The most anterior part of each rib consists of costal cartilage which joins the rib proper by way of a primary cartilaginous joint at which no movement occurs. The anterior ends of the second to seventh ribs articulate with the sternum by means of synovial joints. The first rib joins the manubrium by a primary cartilaginous joint and the eighth to tenth ribs articulate with each other anteriorly via synovial interchondral joints.

The movements of the ribs are described by two main movements. A pump-handle movement elevates and depresses the ribs and has an axis of rotation through the neck of the rib. This movement, which is most prominent in the upper ribs, elevates and moves the sternum out thus increasing the cross-sectional area of the thorax at a given spinal level. The bucket-handle movement of the ribs is more prominent in the lower ribs. This movement occurs about an axis which runs from the costochondral joints anteriorly to the head of the ribs posteriorly. Thus, the bucket-handle action rotates the ribs out laterally increasing the transverse diameter of the thorax (Fig. 6.2).

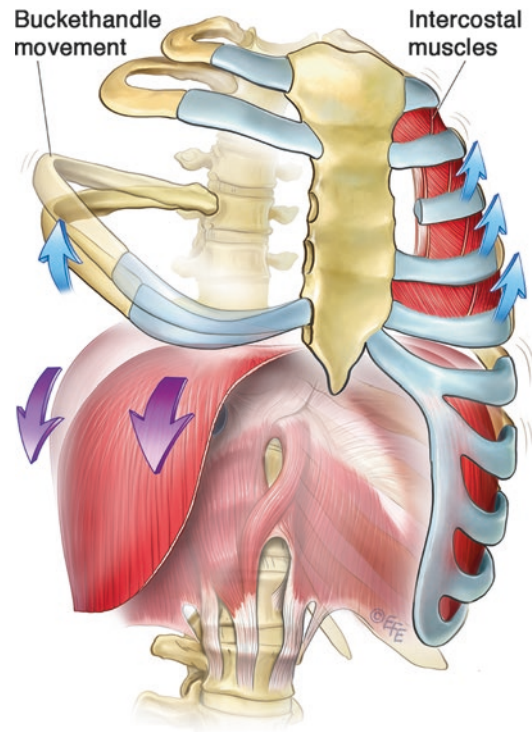

Fig. 6.2 Mechanics of breathing

Physiology

The respiratory cycle, comprising of a sequence of inspiration and expiration is dependent on the interplay of the atmospheric pressure and intra-alveolar and intra-pleural pressures. The primary muscle groups which are involved in breathing are the intercostal muscles and the diaphragm. The lungs themselves are passive, in that they have no contribution to the movement that aids inspiration and expiration. The pleural fluid has an adhesive effect which allows the lungs to be pulled out when the thoracic wall expands outward during inspiration. Contraction of the diaphragm causes it to move inferiorly creating a larger thoracic cavity, in turn contraction of the external intercostals cause the ribs to move upward and outward (Fig. 6.2).

The increase in volume decreases the intra-alveolar pressure, lower than the atmospheric pressure and causes air to move in passively via the bronchial tree through the open glottis. Expiration occurs by the elastic recoil of the lungs and relaxation of the diaphragm and external intercostal muscles. The intrapulmonary pressure rises above the atmospheric pressure and causes air to leave the lungs. Forced breathing in contrast, requires activation of other accessory muscle groups such as the scalenes in the neck, the intercostals of the thorax, and the obliques of the abdomen.

Other large muscles such as serratus anterior, latissimus dorsi, trapezius, and pectoralis which attach to the ribs do not contribute to breathing, but rather use the rib cage as stabilization to move other bones, specifically the upper limbs.

Pathophysiology

The discontinuity of the chest wall at the flail segment causes it to move inward during inspiration as a result of the generated negative intrathoracic pressure during breathing (Fig. 6.3). This occurs independent of the remainder of the chest wall which is moving out during inspiration. This is the classically described paradoxical movement of the flail chest, in response to the negative intrapleural pressure during inspiration, and can result in decreased tidal volume. In most cases, despite the inefficient respiration, the alveolar minute ventilation and the PCO2 are maintained. Changes in the tidal volume are compensated by an increase in the respiratory rate. In a canine model of a flail segment, it has been observed that in the absence of significant pulmonary parenchymal injury, effects on gas exchange and ventilation are minimal [4]. However, as loss of efficiency of the breathing effort progresses, (and pain contributes), there is atelectasis and compression of underlying lung leading to increased lung resistance and decreased compliance, further increasing the work of breathing.

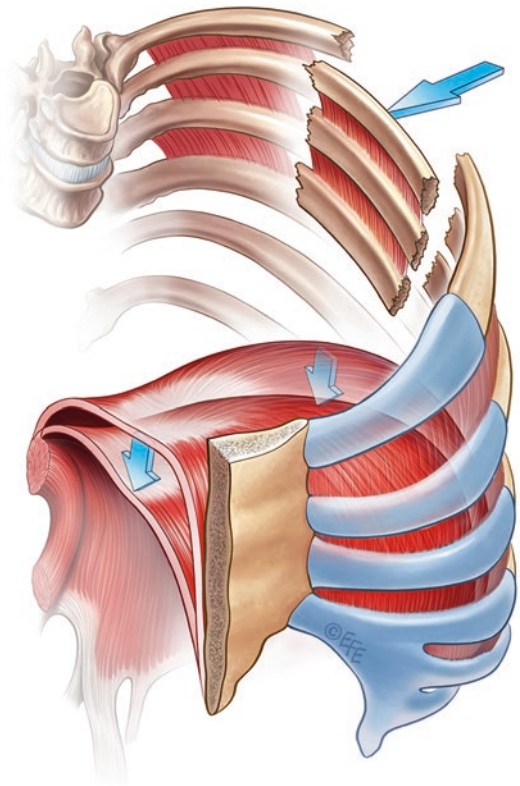

Fig. 6.3 Paradoxical movement in flail chest injury

Despite these disturbances in mechanics, it is clear that the injured lung plays a pivotal role in the pathophysiology of flail chest and this is not a condition affecting the fractured ribs alone [5, 6]. Pulmonary contusion causes leakage of plasma into the alveoli resulting in reduced lung compliance which in turn leads to increased shunt fraction and decreased ventilation per unit volume. First described in a dog model, it has been noted that parenchymal injury, particularly contusion is a progressive condition which worsens over 24–48 h and can be exacerbated by fluid resuscitation [7, 8]. An increase in pulmonary vascular resistance and subsequent decrease in blood flow has also been noted in the contused lung.

Other effects including edema, increased vacuolation, and thickened septae have also been demonstrated in the uninjured portion of the lung [9]. This has been said to occur as a result of release of systemic inflammatory cytokines and impairment of cellular immunity [10].

The negative intrathoracic pressure developed in the chest with normal breathing is also essential for venous return. Another passive process, movement of venous blood from the periphery into the chest relies heavily on the negative intrathoracic pressure developed in the chest with each breath. The high-capacitance, low-pressure chambers of the heart then fill (particularly the right atrium) and blood is then actively transported to the ventricle. Loss of venous return leads to hypotension and also contributes to mortality in the acute setting in these patients.

Blunt trauma to the chest is often associated with other injuries. Complications such as tension pneumothorax, cardiac tamponade, airway disruption, hemothorax, major vascular injury as well as liver and splenic injuries all impact on presentation and immediate management in these patients.

Mechanism

All rib fractures occur either due to a direct force (usually blunt) which causes rib fracture at the site of injury, or a force to the chest which causes fracture of ribs remote to the force impact site. Blunt force leads to the propagation of stresses and strains through the body, and will lead to injury depending on the force applied, the geometry, and material properties of the tissue to which the load is applied. The most common cause of flail chest injury is motor vehicle accidents. As a result, ribs fracture either at the site of impact (e.g., laterally in side car intrusion), or remotely due to compressive forces to the thorax as happens in a front collision in occupants wearing seat belts [11, 12] (Fig. 6.4b). In such an accident, the ribs bend and deform and this occurs maximally in the lateral aspect of the thorax. When the ability of the rib to deform is exceeded, fractures occur in the ribs, away from the actual site of the impact location [13] (Fig. 6.4).

Lower-velocity injuries such as chest compression during cardiopulmonary resuscitation can also cause flail chest injury. In the case of CPR, concentrated loads on the sternum lead to high stress peaks in the anterior parts of the ribs due to the strong deformation of the costal cartilage and subsequent bilateral anterolateral rib fractures [13] (Fig. 6.4a). The propensity for rib fractures in this group is also contributed to by the greater brittleness of ribs in older patients leading to rib fractures with less force. In contrast, the pediatric population with greater elasticity of their ribs, are less likely to develop rib fractures. However, this will often correspond to greater transmission of force to the underlying tissues in younger patients leading to higher incidences of lung contusion and other solid organ damage [14]. In elderly patients, in contrast, even a low velocity accident such as a fall can lead to rib fractures and even a flail chest injury without necessarily developing significant lung contusion (Fig. 6.4c).

Outcomes

Traumatic flail chest injury has been associated with a mortality rate of 33–80% quoted in early literature [15–17]; however, mortality rates have reduced substantially in more contemporary cohorts of national registry data with 20.6% quoted in a study of 262 flail chest patients in Israel [18] and 16% quoted in a Canadian study of 3467 patients [19].

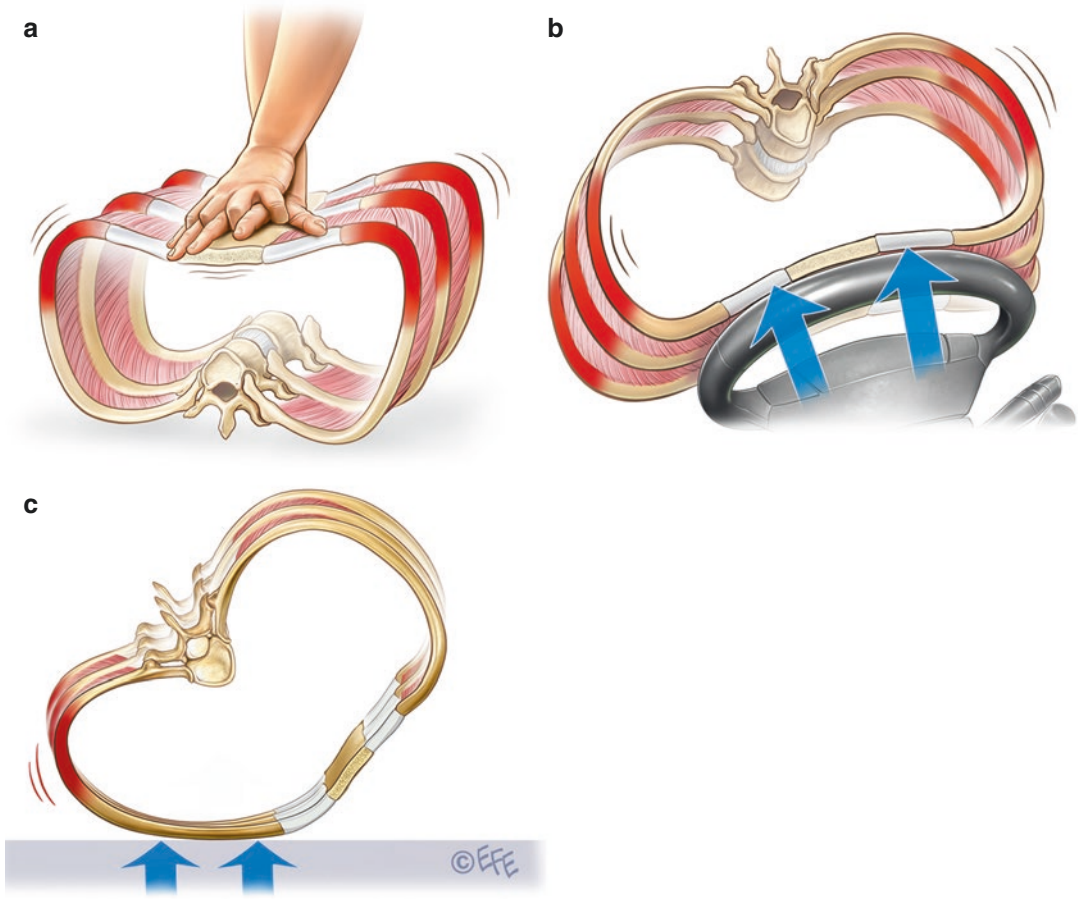

Fig. 6.4 The schematic of a flail chest fracture. (**a**, **b**) Demonstrate typical anterior compression injuries which lead to bilateral rib fractures and a central flail. (**c**) Demonstrates a more lateral injury which can lead to posterolateral rib fractures and costochondral disruptions, or a variety of other patterns of flail chest

The most common cause of flail chest injury is motor vehicle accident, accounting for 79% [19] and 76% [18] in registry studies. The high mortality rates of flail chest injury reflect the high impact nature of the injury as well as associated life-threatening injuries. Severe head injury is seen in 15–27%, pulmonary contusion in 54%, and hemothorax, liver lacerations and splenic lacerations are also commonly seen [18, 19]. Mortality risk has been shown in several studies to be increased with age ≥ 65 years, associated brain injury, and bilateral flail chest injury [20]. Presence of pulmonary contusion has not been shown to be associated with increased mortality in contemporary series [19].

Patients with flail chest often require invasive mechanical ventilation and prolonged intensive care unit stay. As a result there is an increased risk of complications such as tracheostomy, pneumonia, sepsis, and adult respiratory distress syndrome [19]. Long-term morbidity including chronic pain, deformity, disability, respiratory compromise, and failure to return to work have been well documented [21–25]. One of the mechanisms for ongoing deformity has been identified as the result of the action of serratus anterior which pulls any mobile flail segment posteriorly toward the scapula resulting in distraction at the anterior fracture line and comminution at the posterior fracture line [26, 27] (Fig. 6.5).

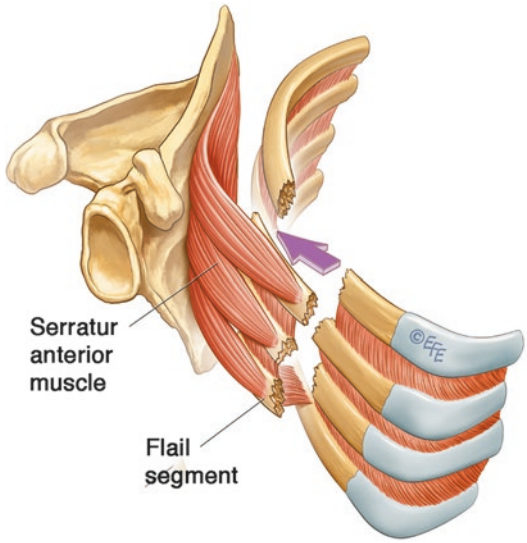

Fig. 6.5 Action of serratus anterior in flail chest injury

Treatment

Identification of the high mortality in this condition, and of the physiological derangement, led to early reports of external support by means of traction, years before mechanical ventilation was widely available [28, 29]. Strapping, sandbagging, and physical traction by attached weights via pulleys to the patient's skin with forceps were described in those early reports and have been reviewed in detail more recently [30].

Development of the mechanical ventilator and its increasing clinical use in the 1950s led to the concept of internal pneumatic splinting for flail chest injury [31, 32]. Surprisingly this treatment modality remained the standard management of flail chest injury for the next 50 years.

Optimal management of flail chest injury requires a multimodality approach aimed at relieving pain, supporting respiration and preventing complications. In the immediate resuscitative period of management, judicial use of parenteral fluids is required to support the circulation by limiting pulmonary oedema in contused tissue [33]. Intercostal drainage is a frequently used adjunct in treating hemothorax and pneumothorax and may be used preemptively in any patient who will undergo positive pressure ventilation.

Respiratory supportive measures are aimed at avoiding the need for intubation. Aggressive physiotherapy focusing on deep breathing and coughing, and incentive spirometry are essential. Positive pressure in the form of noninvasive ventilation can assist if it is tolerated. These patients need to be regularly monitored for signs of respiratory fatigue. Invasive mechanical ventilation is required in up to 59% of patients [19] and remains a mainstay of management in these severely injured patients.

Analgesia is a critical component of the management of these patients. Without adequate pain relief, it is unlikely that physiotherapy and noninvasive ventilatory support will be successful. Analgesic protocols include an escalating regime of oral or parenteral analgesia depending on the condition of the patient and the extent of injury. Typically these include regular acetaminophen, with a COX-2 inhibitor and a demand-only opioid such as fentanyl or morphine [34]. Regional analgesia is being used increasingly in those patients in whom oral and parenteral therapy do not provide sufficient analgesia. These blocks include intercostal nerve blocks, paravertebral blocks, or epidural infusions, with increasing focus on local muscle blocks such as serratus anterior blocks and erector spinae blocks [35, 36]. Routine use of epidural analgesia in blunt trauma patients has been shown to improve lung volumes and ventilatory function [37, 38]. Guidelines has suggested epidural analgesia for patients with four or more rib fractures and suggest its use in those with fewer fractures who are older than 65 years or who have significant cardiopulmonary disease or diabetes mellitus [39, 40].

Despite scattered reports of surgical open reduction and internal fixation of fractured ribs over the 1990s, lack of dedicated rib prostheses, hardware failures, and variable results have meant there was a general lack of take up of this operation in the surgical community [41]. However, in 2002, a small randomized controlled trial was published which showed significant

benefits to operative fixation of flail chest injury in terms of ventilator time, respiratory function, pain, return to work rates and costs of treatment [42]. Increasing interest in operative fixation of fractured ribs was developing around that time, although it was far from standard management. Over the next few years, there were multiple publications of successful operative fixation of flail chest injury, but only two more randomized controlled trials have been performed to this day [43, 44]. Since then a number of meta-analyses have combined the published outcomes of almost 6000 patients (although less than 1300 patients received operative fixation). These analyses are consistent in their findings that in flail chest patients, operative fixation offers benefits to mortality, hospital length of stay, duration of mechanical ventilation, incidence of pneumonia, and requirement for tracheostomy [45–52]. It is disappointing that there are not more RCTs from which to draw data and conclusions. Published meta-analyses outnumber RCTs by quite some way and there has even been a systemic review of meta-analyses on rib fixation in flail chest injury [53]. Perhaps this underscores the difficulty of performing surgical RCTs [54].

It is important to note that despite all this evidence for rib fixation in flail chest injury, there is a paucity of evidence for rib fixation in nonflail rib fractures [55]. This is an area that needs further study as enthusiasm for rib fixation is already expanding beyond the current evidence [56].

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Costal Cartilage Injury

7

John G. Edwards

Anatomy and Nomenclature

Knowledge of the anatomy of the costal cartilages (CC) is important in the assessment and management of injury. On each side, there are usually seven true ribs, which articulate posteriorly with the vertebrae and anteriorly with the sternum via individual CCs. The eighth to tenth ribs are false ribs, as the CCs do not articulate with the sternum directly, but the CC lying immediately above, forming the costal margin (CM). The 11th and 12th ribs are both false and floating, in that they have no CC connecting them to the sternum. There are three sets of joints relevant to CC injury. The *costochondral joints* are primary cartilaginous joints between the ribs and the CCs. Whereas the first *sternocostal joint* is a primary cartilaginous joint, the second to seventh are synovial joints. The lower sternocostal joints are supported by radiate sternocostal ligaments. The *interchondral joints*, which are also synovial, are those which allow for articulation between the CCs of the eighth, ninth and tenth ribs. There is variability in the configuration of the lower CCs, with "side to side" as well as "end to side" interchondral joints being present. The costal cartilages are related to several muscles, which can provide challenges to the surgeon. The upper six costal cartilages are overlaid by, but also form the sternocostal origin of, pectoralis major, with, more laterally, the third to fifth CCs and ribs giving the origin of pectoralis minor. The costal margin, formed by the lower border of the CCs of the false ribs, provides the costal origin of the diaphragm and the insertion for the internal oblique muscle fibres. Transverse abdominis, the innermost abdominal muscle, is attached to the inner surface of the lower six ribs and their CCs. On the outer surfaces of the CCs, there is the origin of rectus abdominis medially and, more laterally, the origin of external oblique. Thus the costal margin cartilage can be considered to be intersection of three musculoskeletal planes: the muscles of the diaphragm and abdominal wall, and the chest wall. Hence, injuries to the CCs can affect not only the individual cartilages themselves, but be associated with traumatic disruption of the sternocostal, costochondral and interchondral joints, as well as the integrity of the diaphragm, chest wall and abdominal wall muscles. Without consideration to all these anatomical features, the nature and associations of CC injuries can easily be overlooked.

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Mechanism of Injury and Presentation

While a history of (usually blunt) trauma is often present, this is not always the case. Injury to CCs can also occur in relation to coughing, sneezing or retching. Costal cartilage fractures may also occur as a result of cardiopulmonary resuscitation. Patients will often complain of a lump, which may be reducible, occurring after trauma. Hence, there should be a high suspicion of CC injury in patients who present with tenderness and swelling lateral to the sternum and rectus abdominis muscle, particularly if it is possible to induce clicking on examination. Patients with a costal margin rupture (CMR), in addition, may have signs of a cough-induced hernia or a deep palpable defect in the costal margin (Fig. 7.1).

Investigation and Classification

Plain chest radiography is typically unhelpful in the diagnosis of CC injury, although associated injuries (such as rib fractures, intercostal hernia, diaphragm rupture, sternal fracture) might be seen. A computed tomography (CT) scan is useful in the diagnosis of CC fractures [1], although there is a tendency for the injuries to be overlooked. Three-dimensional reconstructions rendered for costal cartilage may aid in the identification of anatomy of the injury and in planning for surgery (Fig. 7.2). Dynamic ultrasound may reveal more CC fractures than radiography and, indeed, may demonstrate CC injury in most patients presenting with rib fractures [2]. Magnetic resonance imaging has also been proposed as a sensitive investigation [3].

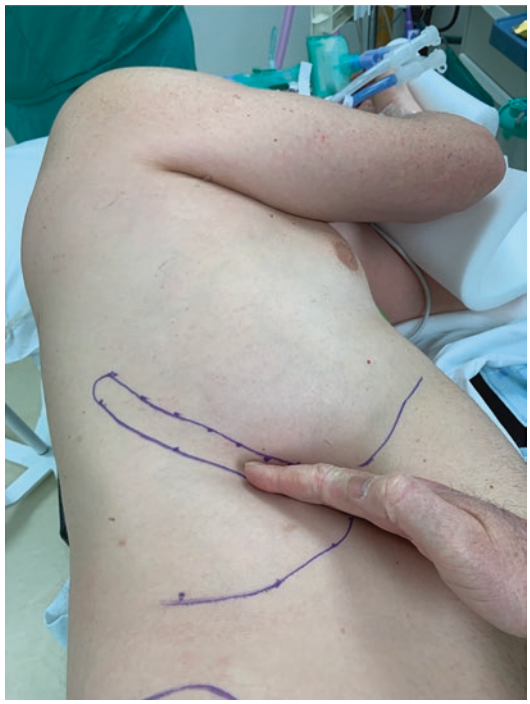

Fig. 7.1 Costal margin rupture and intercostal hernia (CMR + IH) in a 58-year-old male as a result of a coughing attack. The defect in the costal margin is clearly visible once the patient is positioned for surgery

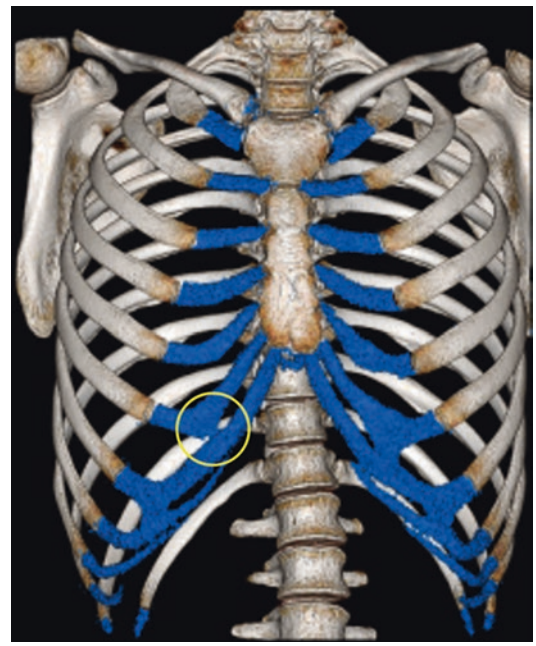

Fig. 7.2 An unenhanced computed tomography (CT) scan, visualised using composite volume rendering of the bone and costal cartilage in a 21-year-old female elite gymnast. The circle indicates irregularity of the interchondral joint between the right sixth and seventh costal cartilages. Dynamic ultrasound confirmed the presence of an unstable interchondral joint

Taxonomy of rib fracture patterns has been addressed by the Chest Wall Injury Society (CWIS) in a Delphi-based consensus exercise [4]. The inclusion of a costal cartilage sector was felt to be important in the description of blunt chest trauma. In an institutional analysis of the CWIS proposed definitions in 539 fully characterised patients with multiple rib fractures, there were, however, too few cases with CC injuries to determine the clinical significance of CC fractures [5]. CC fractures may contribute to an anterior flail segment, where three or more ribs are fractured on both sides of the sternum. This pattern of injury can be seen as a result of severe blunt trauma to the anterior chest or following cardiopulmonary resuscitation [6].

With respect to injuries affecting the costal margin, injuries are poorly and inconsistently described in the case reports or small case series which make up the literature. The Sheffield Classification has been proposed, describing the contribution of the different musculoskeletal planes associated with the cartilage: the diaphragm, chest wall and abdominal wall (Fig. 7.3) [7]. Sequential analysis of the anatomic structures enables accurate categorisation of the injury, allowing for appropriate determinisation of clinical management, as discussed in detail below.

Management

Whereas the mechanism of healing of bone fractures is well known, there are differences with cartilage injuries which affect the healing process. The rib periosteum and underlying bone tend to be very vascular, the costal cartilage perichondrium and particularly the cartilage within are less so. Hence, there is less hematoma formation, less acute inflammation and a slower generation of cartilaginous union. However, the goal of reducing the strain across the fracture to allow for healing to occur is the same. Elliott and colleagues have described this process eloquently [8]. It is important to take into account the amount of strain across the costal cartilage injury when determining the appropriate management. For example, in a young patient with compliant costal cartilage, there may be more movement, and

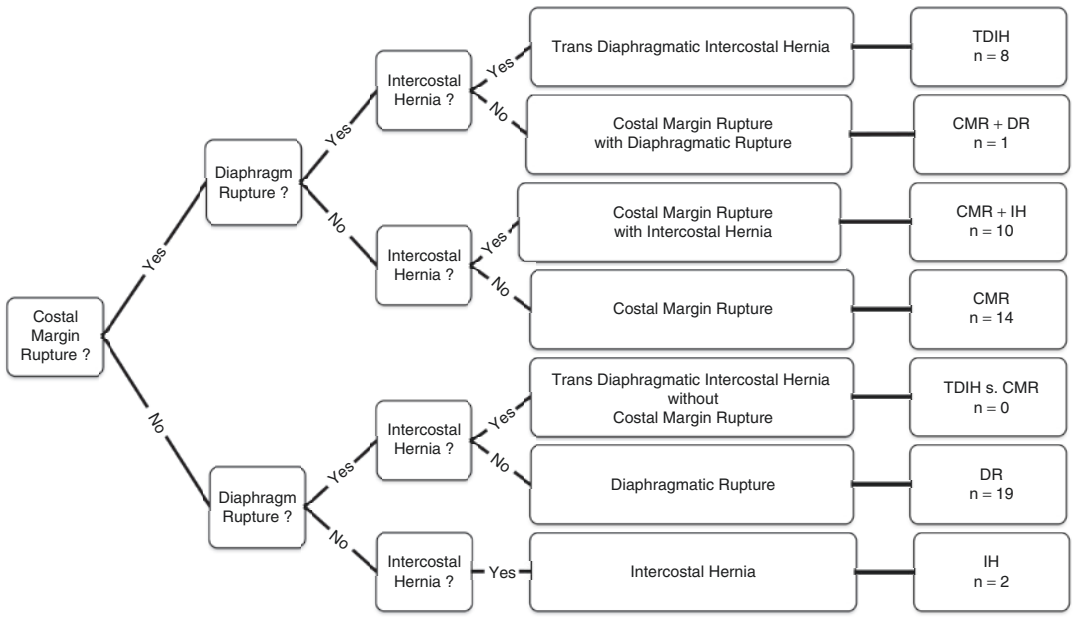

Fig. 7.3 Sheffield Classification of Injuries Involving the Costal Margin. Sequential segmental analysis of CT scans, directed at the costal margin, diaphragm and intercostal muscles leads to categories with discrete clinical characteristics and management options

hence more strain, present than in an older patient with heavily calcified costal cartilages [9]. Furthermore, if external cortical plate and screw fixation is planned, then the construct might be stronger and more resistant to "back out" in the latter patient. The forces across a costal margin rupture, particularly in the obese patient and in conjunction with an intercostal hernia, might be significantly greater than forces across an isolated costal margin rupture in a slim patient.

Non-operative management is likely to be successful in the vast majority of patients with costal cartilage injuries, partly because of the challenges in identifying the injuries and also because the majority of symptoms will settle with conservative management. Indications for surgical management will be similar to those for rib fractures, namely, respiratory compromise, instability, paradoxical motion, pain and deformity. Surgical management, typically the placement of extrathoracic cortical plates and screws, will depend on the injury categorisation. For the purposes of simplicity, isolated injuries to the costal cartilages of the "true ribs" will be considered separately from those affecting the costal margin (the costal cartilages of the "false ribs"). Of course, the situation is rarely simple, as there may well be combinations of different individual injuries and associated rib fractures to take into account.

Injuries of the True Rib Costal Cartilages, Costosternal Joints and Sternochondral Joints

These injuries may be considered as a group, as the surgical principles and techniques are the same. There are no published guidelines specifically directed at costal cartilage injuries of the true ribs. While surgeons may have concerns about fixing external cortical plates and screws to costal cartilage, there is growing experience of this, as evidenced by several case reports [10–12]. A large series of patients relevant to this application was published by Schulz-Drost and colleagues, in the setting of surgery for pectus deformity surgery [13]. They described the placement, after multiple angled osteotomies and mobilisation of the sternum, of transverse plates fashioned to follow the chest wall from the rib on one side of the chest, across costal cartilage and the sternum to the same structures on the other side of the chest. An "iatrogenic anterior flail chest" is stabilised effectively by this "elastic stable chest repair", which acknowledges the mobility and the strain characteristics of the chest wall in the name of procedure. Such surgery can be performed through several small incisions along the path of the plate, rather than with a single large incision. Costal cartilage (and rib) fractures occurring following cardiopulmonary resuscitation have the added complexity in decision making of surgical intervention in a patient who may have suffered a recent myocardial infraction, and who might typically be requiring dual anti-platelet therapy. These patients may have bilateral costal cartilage injuries, manifest by anterior chest wall paradoxical motion and respiratory compromise, requiring bilateral incisions (Fig. 7.4) [14, 15].

Interchondral Joint Injuries

Patients may present with anterior chest wall pain where the costal cartilages appear intact, without fracture. Figure 7.2 shows an unenhanced computed tomography (CT) scan, visualised using composite volume rendering of the bone and costal cartilage (TeraRecon iNtuition v4.4.8, TeraRecon [Foster, CA, USA]), in a 21-year-old female elite gymnast, who had been crushed at the base of an acrobatic stack 4 years previously. She complained of significant right lower anterior chest wall pain and clicking, which had halted her career. The scan detected irregularity of the cartilaginous joint between the right sixth and seventh costal cartilages, not visible on bone windows alone, with no fracture of the costal cartilages themselves. At surgery (Fig. 7.5), the clicking was located to instability of the interchondral joint visualised on the CT scan. The articulation was excised to prevent contact

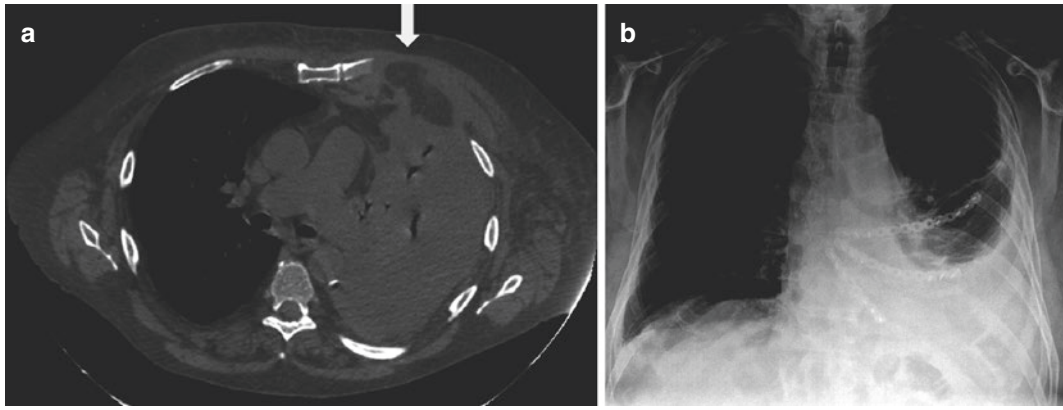

Fig. 7.4 (**a**) Axial CT scan of a patient following cardiopulmonary resuscitation after an out of hospital cardiac arrest, who was failing to wean from the ventilator due to gross paradoxical motion of the chest wall. There were displaced fractures of the left third to sixth costal cartilages, accompanied by lung herniation (arrow) and a large hemothorax. (**b**) Chest radiograph after surgical stabilisation of the fourth, fifth and sixth costal cartilage fractures, with titanium external cortical plates placed from the rib, across the cartilage to the body of the sternum, secured with variable angle locking screws

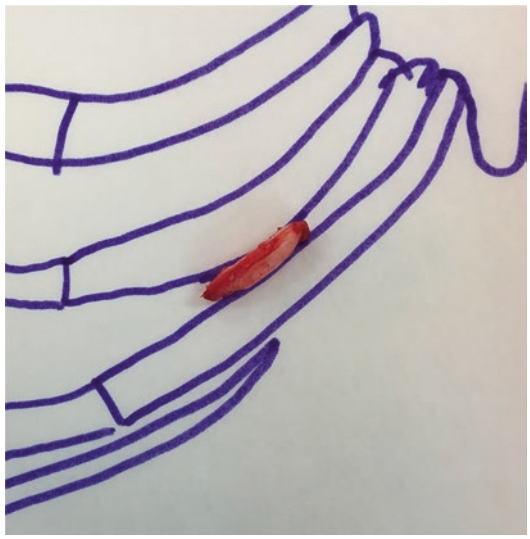

Fig. 7.5 Photograph of the excised interchondral joint of the patient described in Fig. 7.2. At surgery, this synovial joint was found to be unstable and responsible for the complaint of painful clicking. Excision of the joint resolved the symptoms

between the costal cartilages, which gave complete symptomatic resolution and allowed for resumption of her competitive career.

Figure 7.6 shows an axial CT scan of a 35-year-old female who presented with persistent pain and clicking after childbirth 3 years previously. She had a non-united fracture close to the left sixth costochondral joint, and subluxation of the sixth to seventh interchondral joint. At surgery, there was concern that separating the sixth and seventh interchondral joint by trimming the cartilage would compromise the strength of the costal margin and also the healing of the sixth rib. An external cortical plate was placed across both: over the sixth rib, the sixth rib fracture and the interchondral joint to the seventh costal cartilage anteriorly, held by variable angle locking screws (Fig. 7.6b). Her symptoms resolved.

Injuries Involving the Costal Margin

Injuries involving the costal margin are particularly challenging, in terms of diagnosis, assessment and management. These may present acutely, most often in the setting of blunt chest trauma, or long after the injury, typically in those occurring in relation to coughing, retching or vomiting. Taking note of the correct classification of the pattern of injury is critical in determining the appropriate management. Since we derived the Sheffield Classification and started recording cases prospectively in our Major Trauma Unit in the UK (serving a population of 1.8 million), we

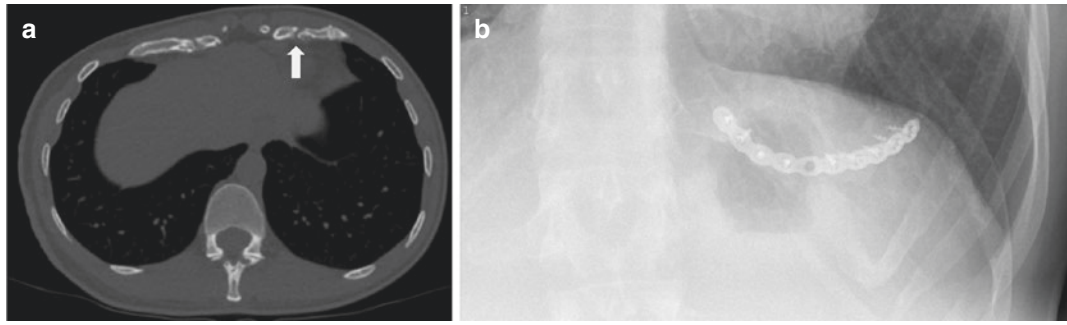

Fig. 7.6 (**a**) Axial CT scan of a patient with persistent pain and clicking following childbirth. The scan showed increased calcification around a sixth to seventh interchondral joint (arrow), close to a non-united fracture of the anterior sector of the sixth rib, which was unstable and subluxing at surgical exploration. (**b**) Postoperative chest radiograph, showing the external cortical plate passing from the sixth rib to the seventh costal cartilage, stabilising the non-united sixth rib fracture neighbouring the costochondral joint, and the subluxing sixth to seventh interchondral joint

Table 7.1 Categorisation, presentation and aetiology of injuries associated with the costal margin, according to the Sheffield Classification

	Number	Presentation		Cause				
Category name (abbreviation)		Acute	Chronic	High velocity trauma	Penetrating trauma	Fall	Cough, sneeze or retch	Crush/ twist
Traumatic diaphragmatic and intercostal hernia (TDIH)	8	2	6	2	–	–	6	–
Costal margin rupture with diaphragmatic rupture (CMR + DR)	1	1	–	1	–	–	–	–
Costal margin rupture with intercostal hernia (CMR + IH)	10	1	9	–	–	3	7	–
Costal margin rupture (CMR)	14	11	3	4	1	3	2	4
Traumatic diaphragmatic and intercostal hernia without costal margin rupture (TDIH s.CMR)	0	–	–	–	–	–	–	–
Intercostal hernia (IH)	2	1	1	–	1	–	–	1

have classified 35 accordingly patients and recorded the clinical outcomes, building on our published experience [7] (Fig. 7.3, Table 7.1). The proposed management options for each injury type are indicated in Table 7.3. The surgical principles to note are as follows.

1. Whenever there is splaying of the intercostal spaces on the chest radiograph or CT scan, the patient should be assumed to have a costal margin rupture (CMR) until proven otherwise. Sequential segmental analysis of the costal margin, diaphragm, intercostal muscles and ribs, and abdominal wall muscles allows for the correct diagnosis and management plan to be made.
2. The acute inflammation in the setting of an acute presentation promotes an environment of healing and tissue remodelling, which allows for the following.
   (a) A trial of conservative management for an isolated costal margin rupture (CMR),
   (b) The possibility of successful repair using sutures only for CMR with Intercostal Hernia (CMR + IH) or isolated intercostal hernia (IH).
3. In a non-acute presentation, which occurs typically in patients suffering the injury as a

result of a cough, sneeze or retch, a suture repair alone may not provide a strong enough repair, reducing strain to the degree required to allow healing of the costal margin and chest wall. Hence, failures of the surgical repair can occur. These cases can be considered to have an "atrophic" non-union of the CMR.

4. Fixation of acute or non-united fractures alone is insufficient to treat a patient with CMR + IH or trans-diaphragmatic intercostal hernia (TDIH).
5. Surgical stabilisation of the costal margin, such as with the titanium external cortical plates and screws used for surgical stabilisation of rib fractures (SSRF) provides symptomatic control for cases of isolated CMR who fail initial conservative management.
6. SSRF-style plating may not alone provide a sufficiently strong costal margin repair, particularly in cases of chronic presentation after cough, sneeze or retch where there is usually an IH component (either CMR + IH or TDIH). In these cases, we have found it difficult to gain close cartilage apposition with the expectation of cartilaginous union under the plate.
7. The double-layer mesh repair does provide a solid repair of the IH component of CMR + IH or TDIH, without relying on cartilaginous union of the costal margin.

Surgical Techniques

The vast majority of surgery for costal cartilage injuries relates to open surgery. Minimal access techniques have been described, although reports are few and follow-up limited [16]. The repair of the sequential components of injuries around the costal margin is as follows:

1. External mesh repair: either a biologic or a polypropylene mesh is placed using non-absorbable sutures.
2. Diaphragm rupture repair: a primary suture repair of the diaphragm is usually possible using continuous or interrupted Ethibond (Ethicon, Somerville, NJ, USA) sutures, rarely supported with interrupted Teflon pledgets (Ethicon, Somerville, NJ, USA).
3. Costal margin rupture: where surgery is part of a more complex repair in the acute setting (e.g. CMR + IH or TDIH), No. 5 Ethibond sutures are used to re-approximate the CMR. For surgical fixation of a symptomatic hypertrophic non-union of the CMR, external cortical plates with locking titanium screws are placed after contouring of the non-union. Our preference is to use a system with variable angle locking screws, as we believe that this provides a stronger plate–cartilage construct which is more resistant to "backing off" from the repaired costal margin.
4. Intercostal hernia: an isolated intercostal hernia can be repaired easily in the acute setting by the placement of pericostal non-absorbable sutures (e.g. Ethibond), or an extrathoracic polypropylene, absorbable or biologic mesh. Where "pericostal" sutures are placed, the preference is for sutures to be placed in holes punched or drilled into the rib caudal to the defect (intracostal sutures) in order to prevent entrapment of the intercostal nerve and risk chronic pain [17]. However, while an external mesh repair may be successful in the acute setting of CMR + IH and in cases of isolated IH, in those cases where there is chronic CMR + IH or acute/chronic TDIH, our preference is for a Double-layer Mesh Repair.
5. Double-layer mesh repair: We experienced failures of both IH suture only and also external thoracic mesh repairs in TDIH cases. The patients presenting following a cough, sneeze or retch are often obese, diabetic or taking inhaled corticosteroids for chronic obstructive pulmonary disease [7, 18]: all risk factors for impaired wounds healing. Hence, we created a double-layer mesh repair, which has developed further since the original description, the revisions being the preference for use of a biologic mesh and intracostal sutures drilled through holes in the ribs caudal to the IH defect (see above). These two developments obviate the need for extra layers of pledget material to reinforce the mattress sutures. It is

a challenging repair to undertake. The sequence of steps is as follows.

(a) Any associated rib fractures requiring surgical stabilisation are addressed first.
(b) The origins of the serratus anterior and external oblique muscles are noted on from the chest wall, mobilising the non-muscular tissues deep to these muscles to expose the three ribs caudal and two cephalad from the intercostal defect.
(c) Two layers of mesh are used. The extrathoracic mesh is typically 15 × 20 cm and cut into an H shape to account for the preservation of the serratus anterior and external oblique muscle origins, above and below the IH defect. The intrathoracic mesh is rectangular.
(d) Pairs of holes 1 cm apart are drilled with a 2.8 mm drill, as used typically for SSRF, into the two ribs caudal to the IH defect. A Sweet's sternal punch (GU Medical Ltd., Reading, UK) can be used for the caudal rib adjacent to the IH, but it will not reach the lower rib, hence the need to drill the holes.
(e) No. 1 Prolene sutures (Ethicon), are placed from the outside, passing through the outer mesh, the intercostal space and the inner mesh, returning back through those layers in reverse. The sutures are placed in the lowermost rib first and then secured.
(f) Cephalad to the IH, it is important to ensure that the meshes are placed smoothly and the load borne by the sutures shared equally. This is achieved with the temporary aid of a Tudor-Edwards rib approximator (GU Medical Ltd., Reading, UK), and the careful placement of the sutures through the layers. Drilling of the cephalad ribs is not necessary, as the sutures will be pulled down by the tension in the mesh onto the top of the cephalad ribs and intercostal nerve entrapment will not occur. We have found that this is easiest if the intrathoracic mesh is placed first, the needles cut off and each suture passed through a polyvinyl chloride (PVC) "snugger tube", as used for cardiopulmonary bypass cannulation, in order to confirm accurate placement of the sutures under tension (Fig. 7.7a). The extrathoracic patch is then overlaid and a needle passer used to thread the sutures through the patch.
(g) Once all sutures have been placed, the diaphragm rupture is repaired (if present) at this juncture, as approximation of the costal margin may be required to suture the diaphragm. Additional "intracostal" pericostal sutures are placed, each with two passages of the chest wall caudal and cephalad to the IH defect in equilateral triangular fashion, in order to prevent loosening.
(h) If there is a component of the injury extending to the abdominal wall, transverse abdominis and external oblique muscles are repaired with continuous No. 2 Ethibond sutures. In cases of chronic presentation and muscle loss, a mesh repair may be required in order to prevent residual upper quadrant abdominal herniation.
(i) The CMR may also itself be repaired with sutures, as described previously. Given the solid nature of the Double-layer Mesh Repair, and considering the frequent finding of atrophy or hypoplasia of the costal margin cartilages, we have not found it necessary to perform the additional step of plating the costal margin. Indeed, in one case re-explored a year after double-layer mesh repair due to chronic pain, with the intention of plating the non-united costal margin, there was such atrophy of the costal margin that plating was not possible.
(j) After tying the diaphragmatic, abdominal wall and intracostal sutures, the cephalad mesh retaining sutures are tied last (Figs. 7.7b and 7.8).

6. In all cases where the pleural cavity is open, the wound is washed out with a dilute iodine/povidone before a 28 Fr intercostal tube is placed. Muscle and skin layers are closed in the standard fashion. Small-bore suction drains may be placed deep to the muscle layers. The chest drain is connected typically to an underwater seal drain, to which is applied

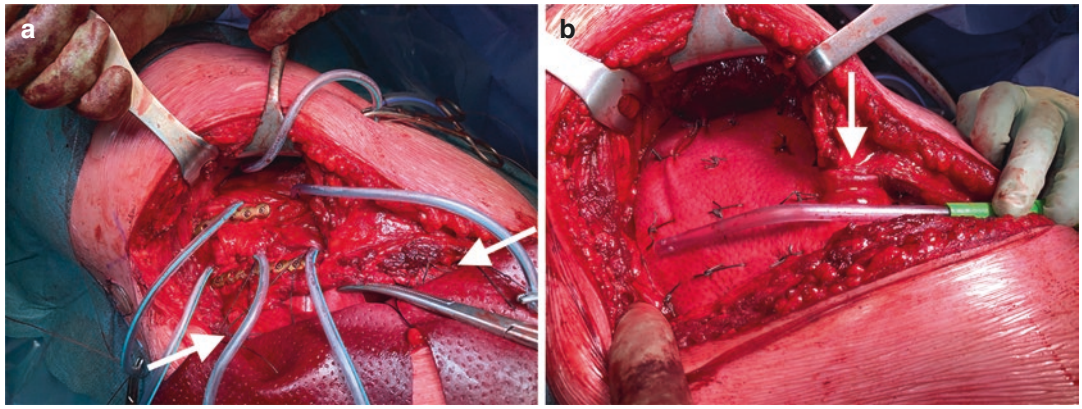

Fig. 7.7 (**a**) Intraoperative photographs of the repair of a case of Costal Margin Rupture with Intercostal Hernia (CMR + IH), as seen preoperatively in Fig. 7.1. There has been surgical stabilisation of two non-united rib fractures cephalad to the intercostal hernia defect, indicated by the arrows. The intrathoracic biologic patch (indicated by the artery clamp) has been sutured in place, with the sutures cephalad to the intercostal hernia held in tension by passing them through the PVC tube "snuggers". The wound retractors are deep to the serratus anterior muscle. The extrathoracic patch, already secured caudally, is reflected caudally, with the "H" shape visible. The tip of the arrow to left side overlies the intracostal/pericostal sutures, which have been placed. These will be tied when all the intrathoracic patch sutures have been placed. (**b**) The completed CMR + IH repair. The sucker marks the level of the intercostal hernia (IH). There are three rows of sutures placed in the three intercostal spaces cephalad to the IH. The arrow indicates the interdigitating origins of the serratus anterior and external oblique muscles, which have been preserved. The external patch cephalad to the IH is not seen, hidden where the sucker is held

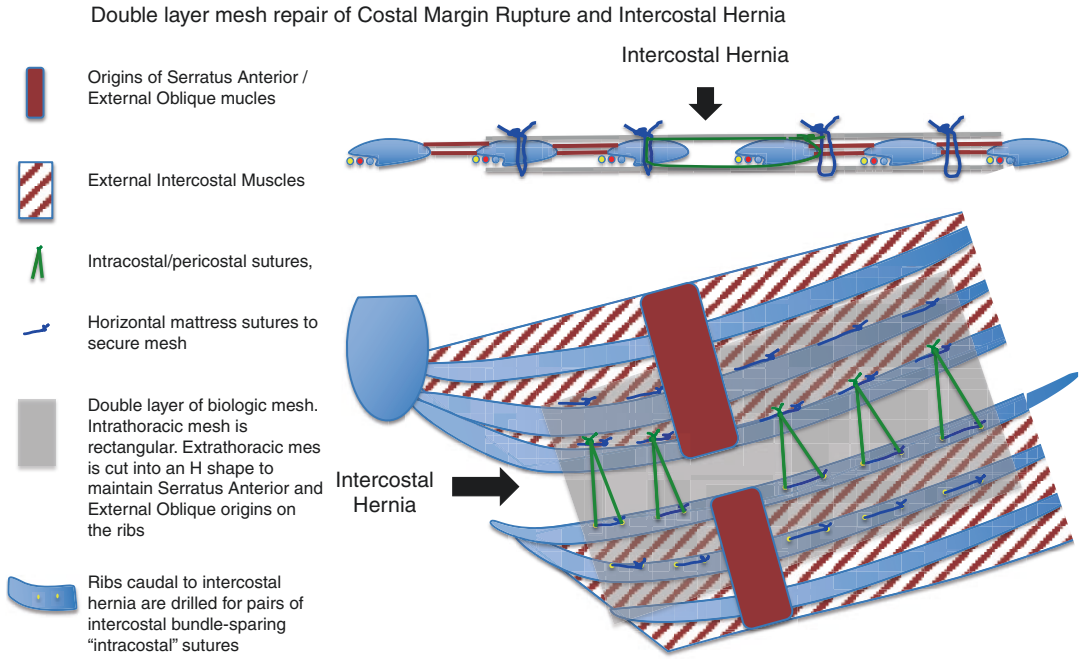

Fig. 7.8 Schematic diagram of the Double-layer Mesh Repair, used in cases of Trans Diaphragmatic Intercostal Hernia (TDIH) and Costal Margin Rupture with Intercostal Hernia (CMR + IH)

20 cmH_2O (2 kPa) of negative suction. Wherever possible, patients are extubated on the operating table and the routine "enhanced recovery after surgery programme" initiated, which involves early nutrition, physical therapy, drain removal and mobilisation.

Results of Surgery

There appears to be a positive publication bias for successful surgical results following surgery, particularly in the numerous case reports indicating good results with limited follow-up. Complications and the burden of further surgery have been noted in another small case series [19], with two thirds of the 12 patients described experiencing failure of the operative intervention. Our experience to date is updated here. The distribution and causation of the 35 cases we have recorded is given in Table 7.1. Of note is that the majority of cases of TDIH and CMR + IH occurred with a chronic presentation following a cough, sneeze or retch. Conversely, patients with CMR tended to present acutely with a more even distribution of causation: the trial of conservative management in patients was successful in 79% of cases (Table 7.2). All patients with TDIH and 60% of those with CMR + IH underwent surgical repair. 19

Table 7.2 Operative management of injuries associated with the costal margin

Category name (abbreviation)	Number of patients	Number managed conservatively	Number requiring surgery	Number of repeat surgical procedures	Extra-thoracic mesh repair	Double-layer mesh repair	Suture repair	Plate fixation
Traumatic diaphragmatic and intercostal hernia (TDIH)	8	1[a]	7	4	2	3	3	–
Costal margin rupture with diaphragmatic rupture (CMR + DR)	1	0	1[b]	–	–	–	1	1
Costal margin rupture with intercostal hernia (CMR + IH)	10	4[c]	6	2	1	3	4	1
Costal margin rupture (CMR)	14	11[c]	3	0	0	0	–	3
Traumatic diaphragmatic and intercostal hernia without costal margin rupture (TDIH s.CMR)	0	–	–	–	–	–	–	–
Intercostal hernia (IH)	2	–	2	–	2	–	–	–

Repeat operations

1. CMR + IH: originally SSRF + suture repair of IH: recurred and further suture repair
2. CMR + IH: originally SSNURF + suture repair of IH: recurred and extrathoracic Prolene mesh repair performed
3. TDIH: underwent suture repair of the DR at presentation but required repeat surgery with double-layer mesh repair for recurrence
4. TDIH: original suture repair but recurred, underwent double-layer mesh repair

5a. TDIH: original double-layer mesh repair: trimming of costal cartilage, left upper quadrant hernia

5b. TDIH: original double-layer mesh repair: removal of extra-thoracic polypropylene mesh

[a]One patient with TDIH underwent suture repair of the DR at presentation and the residual CMR + IH was managed conservatively

[b]One patient died from blunt cardiac injury on day 2

[c]One CMR and one CMR + IH patient are awaiting surgical repair

patients underwent surgery, and, at a median 38 (range 9–169) months of follow-up, there have been 6 repeat procedures in 5 patients, as detailed in Table 7.2. One patient underwent two repeat procedures, due to chronic pain, after a double-layer mesh repair with polypropylene mesh supported by intercostal Teflon pledgetted sutures caudal to the defect. Firstly, there was concern that the pain was due to impingement between the free ends of the costal margin rupture (which were not amenable to plate and screw fixation), in conjunction with an upper quadrant abdominal hernia. The cartilage was trimmed and the hernia repaired, without resolution of pain. At further surgery, the intercostal sutures retaining the patches, and the extrathoracic mesh were removed, but the pain persists. This case prompted the switch to biologic mesh and intracostal sutures: no further case has experienced chronic pain. Four patients had primary surgery without correct recognition of the Sheffield Classification of injury: three of these required further surgery, one did not.

Plate fixation of the CMR component of the injury was possible in five patients, without complication.

Of the 24 surgical procedures performed, after 21 the patients were extubated immediately in the operating room and transferred to the recovery area. Two patients were transferred to the intensive care unit (ICU) following a high oxygen concentration requirement intraoperatively and these were extubated the next day: one of these stayed there 6 further days without reintubation, the other remained there for 15 days recovering from orthopaedic injuries. One further patient with CMR + DR underwent repair of the DR only during a damage control laparotomy and remained intubated postoperatively until death: this case was the only mortality up to 1 year of follow-up.

Health-related quality of life was not recorded objectively and prospectively, although narrative outcomes were assessed during post-discharge hospital visits. As described above, five patients required a total of six repeat procedures: the other patients reported being symptom-free at the most recent follow-up and had returned to preinjury activities of daily living, including employment.

Conclusions

Comprehension of the anatomy and consistent taxonomy is crucial in the identification, diagnosis and treatment of patients with costal cartilage injuries. The Orthopaedic Trauma Association/AO Foundation Fracture and Dislocation Classification Compendium do allow for the coding of a costochondral fracture, but the associated soft tissue injuries are not included [20]. Furthermore, it is not possible to code accurately for the Sheffield Classification of costal margin injuries using with The American Association for the Surgery of Trauma (AAST) Organ Injury Scales (OIS) for diaphragmatic injuries and chest wall injuries. The AAST OIS for the diaphragm characterises the presence of contusion, presence and length of laceration and area of tissues loss, whereas that for the chest wall categorises the presence of contusions, lacerations and fractures [21, 22]. Therefore, while it is possible to account for the presence of adjacent rib fractures and flail segments, there is no OIS suitable category suitable for costal margin rupture which would allow data to be retrieved from national datasets for outcome analysis. This shortfall is therefore a barrier for further research, which must currently be carried out at a hospital level.

Further challenges exist in the identification of cases at the clinical and radiologic levels. Costal cartilage injuries can be difficult to characterise, compounded by the ease of missing subtle findings on the ubiquitous CT scan. Once appropriately classified and surgical management considered, there are few large studies with sufficient outcomes to guide surgical strategy. Currently, literature review for surgical management [7] leads to the recommendations made in Table 7.3. Only with carefully designed studies of sufficient size will it be possible to strengthen the evidence for the best strategy in each injury category. Given the apparent rarity of these injuries and the absence of specific diagnostic coding data, it is most likely that specific multi-institutional

Table 7.3 Recommended management of injuries associated with the costal margin, according to the Sheffield Classification and the type of presentation. We have not encountered any cases of TDIH where CMR is not present: we do not believe that this entity exists

Category name (abbreviation)	Acute presentation	Chronic presentation
Traumatic diaphragmatic and intercostal hernia (TDIH)	1. Five-layer double mesh technique for IH *and* 2. Suture repair of DR Optional: 3. Plate screw fixation of CMR	1. Five-layer double mesh technique for IH *and* 2. Suture repair of DR
Costal margin rupture with diaphragmatic rupture (CMR + DR)	1. Plate and screw fixation of CMR *and* 2. Suture repair of DR	1. Plate and screw fixation of CMR *and* 2. Suture repair of DR
Costal margin rupture with intercostal hernia (CMR + IH)	1. Suture repair of IH Optional: 2. Plate and screw fixation of CMR 3. External mesh repair of IH	1. Five-layer double mesh technique for IH Optional: 2. External mesh repair
Costal margin rupture (CMR)	1. Conservative management	1. Plate and screw fixation of CMR (also indicated if the patient fails conservative management)
Traumatic diaphragmatic and intercostal hernia without costal margin rupture (TDIH s. CMR)		
Intercostal hernia (IH)	1. Suture repair of IH	1. External mesh repair
DR	1. Suture repair	1. Suture repair, with or without mesh reinforcement

studies will need to be carried out for progress in the management of costal cartilage, and particularly costal margin, injuries to be achieved.

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8 Management of Rib and Sternal Fracture Nonunions

Katherine Kent and John Mayberry

Historical Perspective

The paleopathologic record suggests that fracture nonunions, including rib, were known to ancient communities [1–4]. The first written record of a possible chest wall fracture nonunion or at least a malunion is Case 43 of the Edwin Smith Surgical Papyrus (circa 1500 BCE) which refers to "dislocated" sternal costal articulations and "projecting" rib fractures (in contrast to merely "sprained" or "displaced") [5]. Hippocrates (circa 400 BCE) made clear the possibility of rib fracture nonunion especially in cases of more severe chest wall injury where "the flesh no longer adheres to the bone as formerly" [6]. His recommendations for binding, if followed, he stated, will prevent this possibility if "the extravasated blood forming in the bruise be dried up and absorbed, and the part be made up with sound flesh, and the flesh adhere to the bone" [6].

Galen (first century CE), who described callus formation from fracture hematoma [7], was familiar with the possibility of sternal fracture nonunion associated with infection. He described a gymnast who developed an abscess at a probable sternal fracture site 4 months post-injury. Galen drained the abscess and, recognizing that the sternum was necrotic, resected the sternum, exposing the heart [8].

Soranus (second century CE) and Albucasis (tenth century CE) may have seen patients with rib fracture nonunion [9, 10]. Their recommendations to resect comminuted and/or depressed rib fracture fragments at the time of injury (Soranus & Albucasis) or during subsequent suppuration (Albucasis) would certainly have led to nonunions in many cases. Albucasis recommended manipulation and binding of closed fractures of the sternum and ribs, but his only comment regarding the ultimate outcome was for the practitioner to continue the treatment until the fractures heal or abscess [11].

Ambrois Pare' (sixteenth century) recognized the possibility of fracture nonunion when the fracture was not allowed to "rest" during healing or when the injury was more severe.

> For the Callus is easily dissolved, if they be moved before their perfect and solid agglutination. … Otherwise there will be no Callus, or certainly it will grow more slowly. … Fractures are thought dangerous, whose fragments are great, and fly out, especially in these bones which are filled with marrow on the inside [12].

Osteoarcheologic examination of eighteenth to early nineteenth century exhumed graves in England revealed several rib fracture nonunions (Fig. 8.1) [13, 14]. In 1859, Malgaigne reported an instance of rib fracture nonunion with a classic pseudoarthrosis discovered at autopsy [15].

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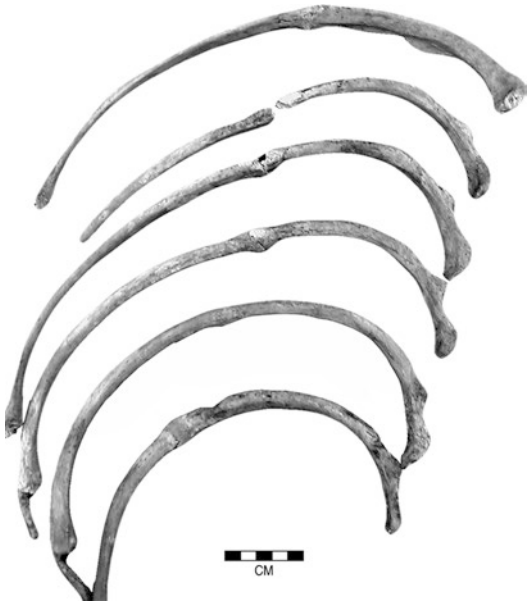

Fig. 8.1 Multiple right posterolateral rib fractures in a male aged over 50 years at death, buried in an earth-cut grave in early nineteenthcentury Birmingham, England. At least two and perhaps three of the fractures appear to be nonunions. Courtesy of Dr. Megan Brickley, with permission John Wiley and Sons

In the early twentieth century the likelihood and potential disability of chest wall fracture nonunion was frequently dismissed or minimized [16–19]. In 1928, Holderman was perhaps the first in modern times to mention sternal fracture nonunion stating, "Occasionally bony union fails to occur … This does not cause any serious disability other than temporary inability to abduct and adduct the arms" [20].

In 1941 Knoepp presented what may be the first record of surgical intervention for symptomatic rib fracture nonunion [21]. He reported the outcomes of 386 inpatients with rib fractures at University Hospital, Shreveport, USA, half of whom presented from 1 month to more than 1 year post injury, stating, "there were delayed union in one, nonunion in four, osteomalacia in one, osteomyelitis in two, excessive callus in two and bridging of the ribs in one" [21]. Four patients had "late excision of the fracture site and intercostal neurectomy for painful rib" [21]. His report was immediately followed by Leavitt's intriguing description in 1942 of a patient with multiple rib fracture nonunions persisting 1 year after a fall [22]. Leavitt harvested cortical tibial grafts and fashioned them into "shuttles" that he inserted into the "freshened" rib medullary canals. Subsequent reports of surgical intervention for rib fracture nonunion did not appear until 40 years later, and traumatic sternal fracture nonunion fixation (autograft/wiring) was not reported until 1975 [23–26].

Epidemiology

Recent healthcare administrative databases in the UK and the USA indicate that the risk of reported fracture nonunion for all fractures is 1.9–2.6%, ranging as high as 8% for clavicle/scapula fractures in the 35- to 44-year age group [27–29]. The risk of symptomatic or detected rib fracture nonunion in the US Medicare population is estimated to be 0.6% per fracture [28].

Diagnosis

The criteria for the diagnosis of fracture nonunion lack consensus among orthopaedic clinicians and is not uniform throughout the skeletal system [30, 31]. Clinical prudence and experience indicate that it can be apparent within 4–6 months that a chest wall fracture patient is not likely to progress to bony union, especially in cases of severe fracture comminution or displacement/distraction. The formation of a synovial pseudoarthrosis is a strong indicator that further union is unlikely [30].

Patients with symptomatic rib or sternal fracture nonunion complain of chronic pain focal to the site of the nonunion(s) exacerbated by physical exertion that requires inactivity and/or higher doses of analgesics or opioids to relieve [32–39]. Many but not all patients perceive fracture motion with breathing or physical activity. Symptoms of intercostal nerve entrapment with pain radiating by dermatome has been reported [39]. Imaging by CT scan is the norm, but MRI and bone scanning may also be obtained [40].

There are two main types of nonunion: hypotrophic and hypertrophic [31, 41]. Hypotrophic

nonunions show no evidence of healing because the fracture site, in particular the periosteum, has been devascularized (from irreversible soft tissue damage) and/or the fracture ends are so displaced/distracted that a bridging callus cannot form (Fig. 8.2). Hypertrophic nonunions have an adequate blood supply but the healing process is impeded by excessive motion (Fig. 8.3). Both of these presentations are observed in rib and sternal fracture nonunions and in both types a pseudoarthrosis may or may not be present [39].

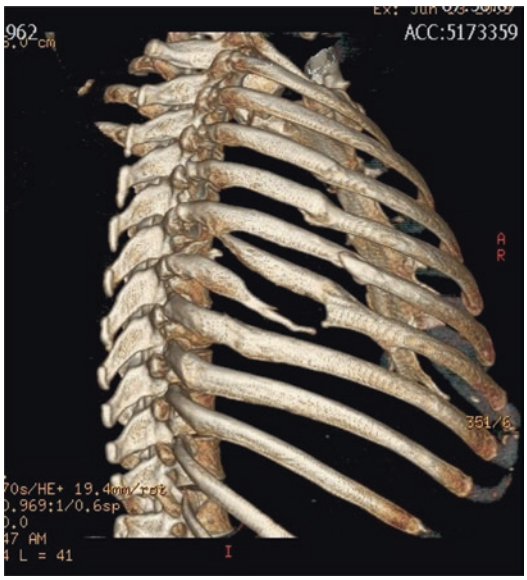

Fig. 8.2 3D CT image of hypotrophic and severely misshapen right seventh rib fracture nonunion in 30 year old female 18 months following motor vehicle crash where severe soft tissue and bony damage was incurred

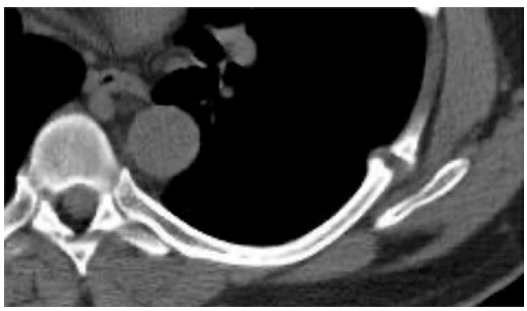

Fig. 8.3 CT image of hypertrophic rib fracture nonunion in a middle-aged male 10 months following cough fracture

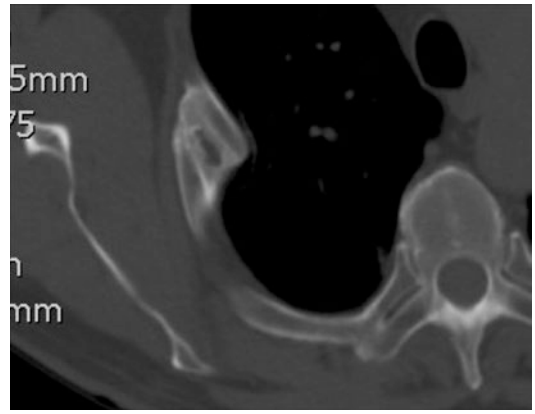

Fig. 8.4 Subscapular rib fracture nonunion/malunion deformity several years following kayaking incident where patient struck his right chest wall on rocks within river rapids

Malunions and fusion/bridging of adjacent ribs also occur (Fig. 8.4) and the errant fusion of a fractured rib end to the incorrect adjacent rib end is possible. In severe cases with multiple malunions and nonunions, the damaged chest wall can barely rise and expand.

Cartilaginous fracture nonunions also occur and may be exceptionally symptomatic. Since CT scan cannot reliably detect cartilage injuries, an MRI with or without a bone scan may assist with diagnosis. Caution is advised, however, against overaggressive operative intervention for cartilaginous injuries since cartilage is also prone to a variety of nonoperative inflammatory pathologies [42].

Risk Factors for Fracture Nonunions

Five sequential steps occur in normal fracture healing: hematoma formation, local inflammatory response, soft callous generation, hard callous formation, and remodeling [39, 43]. Hematoma formation and direct tissue injury initiate local tissue hypoxia, resulting in a rapid release of inflammatory cytokines and chemokines, and attracting neutrophils and eventually macrophages to the fracture site to phagocytize cellular debris and defend against pathogens [43–45]. Recruitment of mesenchymal precursors and

endothelial cells into the fracture site leads to formation of soft callous with eventual hard callous formation and remodeling [39, 43, 44, 46]. Disruption of these steps due to either injury-related or patient-related factors can lead to delayed healing, nonunion, or malunion [39].

Injury-Related Risk Factors

The likelihood of nonunion increases with the severity of the injury. High-energy trauma causing extensive soft-tissue injury, fracture displacement, distraction, comminution, or impingement of muscle or nerve between fractured ends will delay or prevent union [39, 47, 48]. In general, defects greater than 2 cm or loss of 50% of the bone circumference are unlikely to heal without intervention [49]. Open fractures and bacterial contamination also substantially increase the risk of delayed union [27, 43, 47, 50]. Excessive motion at the fracture site can lead to nonunion by an overabundance of callus formation since callus formation is dependent on a minimal degree of movement [46]. Too rigid of fixation leads to little or no callus formation [39, 46].

Polytrauma is also an important injury-related risk factor for nonunion [45, 51]. Polytrauma generally consists of multiple injuries to areas of the body with high injury severity scores (>15) through a combination of blunt force, fracture, burns, hemorrhage, ischemia/reperfusion, surgery, or infection [45, 52]. In polytrauma patients, the highly regulated process of inflammation and immune response goes awry due to aberrant inflammatory signaling leading to prolonged inflammation and impaired fracture healing [45, 53]. Mangum et al. and Recknagel et al. have found in murine models with blunt chest trauma and polytrauma that the number of neutrophils, macrophages and IL-6 levels at the fracture site are substantially altered in polytraumatic injuries [45, 53]. In particular, blunt chest trauma leads to early healing disruption at fracture sites leading to impaired bone regeneration [45, 53]. Histologically, Recknagel et al. found reduced bone formation and inferior callus quality at 5 weeks after the initial trauma compared to rats with fracture and without blunt chest trauma [53]. Bone failure and hyperresorption have also been found to be highly prevalent among critically ill patients in the ICU, resulting in greater fragility and reduction in bone mass [45, 54].

Patient-Related Risk Factors

There are a number of patient-related risk factors, separated into environmental-related factors and host-related factors, that predispose a fracture to nonunion. Among the most important environmental-related risk factors for impaired fracture healing and nonunion are smoking, alcohol intake, and medications [28, 50, 55]. Smoking impairs bone healing through multiple mechanisms including the presence of reactive oxygen species, low antioxidant levels, nicotine-induced vasoconstriction and hypoxia, and by the associated burden of atherosclerosis [50, 55]. In addition, high-dose nicotine inhibits proliferating osteoblasts and has been associated in rabbit models with diminished strength of repair [55]. These results have been echoed in human clinical studies, where smokers with compound tibial fractures had higher rates of delayed union/nonunion and were twice as likely to develop infection [55]. This effect on nonunion and infection was also observed in ex-smokers [55]. Among the 56,492 fractures reported in Medicare patients analyzed by Zura et al. where the overall incidence of nonunion was 2.5% of fractures, 13.1% of patients with nonunion were also past or current smokers ($p < 0.0001$) [28]. Alcohol use, especially chronic or excessive, has also been implicated in poor fracture healing [28, 50, 55]. Chronic alcohol consumption has been linked with osteopenia and decreased bone formation [55]. Human and animal studies suggest that alcohol works through a dose-dependent relationship to suppress osseous matrix perhaps through cell proliferation inhibition and decreased differentiation of mesenchymal precursors resulting in less strength, lower stiffness, and decreased mineral content [55]. Zura et al. also found an increased prevalence of alcoholism in patients with nonunion ($p < 0.01$) [28].

Table 8.1 Medications associated with impaired fracture healing

Medication	Mechanism
Corticosteroids[a]	Osteocyte and osteoblast apoptosis and inhibition of osteoblast formation [50, 56]
Chemotherapeutics	Numerous: impaired vasculogenesis, impaired DNA synthesis, impaired callus formation [50, 56]
Antibiotics (quinolones[a], gentamicin, tetracycline[a], rifampicin)	Multiple mechanisms: chondrocyte death, decreased osteoblast progenitors, impaired bone growth in early stages, inhibition of osteoblast proliferation [50, 55, 56]
NSAIDs	Via COX-2 inhibition, inhibited endochondral ossification, impaired angiogenesis [55, 56]
Anticoagulants[a]	Decreased trabecular volume, increased resorption, decreased osseous formation [56]
Aspirin[a]	Irreversible inhibition of COX-2, inhibited endochondral ossification, impaired angiogenesis [57]

[a]Observed only in animal models

Numerous medications are also implicated in impaired fracture healing and are listed in Table 8.1 along with their proposed mechanism, although some have only been proven in animal models [50, 55–57].

Host-related factors for nonunion include comorbidities such as osteoarthritis with rheumatoid arthritis, diabetes mellitus, obesity, malnutrition, renal insufficiency, anemia, and hypothyroidism [27, 39, 50, 55]. Of these, rheumatoid arthritis and diabetes are considered major risk factors for development for nonunion with odds ratios of 1.58 (95% CI 1.38–1.82) and 1.40 (95% CI 1.21–1.61) respectively [27]. In clinical studies, patients with diabetes have also showed higher nonunion and a doubling of healing time of diabetic compared to nondiabetic patients [55]. However, tight glycemic control has been shown to mitigate these effects [55]. The role of osteoporosis in the development of nonunion is currently under debate, as bone mineral density has not been shown to influence nonunion risk [58, 59].

Increased age has traditionally been associated with an increased risk of nonunion; however, this association has come into question by Zura et al. who looked at age as predictor of nonunion while controlling for other comorbidities [27, 28]. Patients >85 years of age were less likely to have nonunion (1.3%) than those who are younger (55–59 years of age, 5.5%), a finding confirmed by additional prospective studies [27–29]. Zura et al. postulated that patients who survive to advanced age may have less risk factors for nonunion, many of which are risk factors for premature death [28]. Gender does not appear to be an independent risk factor for nonunion, although more nonunions are found in males due to higher incidence of high-energy trauma [55]. Additional host-related risk factors specific to sternum and rib nonunion also include chronic obstructive pulmonary disease (COPD), persistent coughing, and prior radiotherapy to the chest wall [35, 60, 61].

Nonoperative Management

The nonoperative outcome of symptomatic rib and sternal nonunions has not been specifically reported. The assumption of the authors is that when over several months or years the chronic pain and disability stabilizes and no further healing is demonstrated on imaging, the patient is not likely to improve further.

Electromagnetic stimulation (ESTIM), low-level laser therapy (LLLT), and low-intensity pulse ultrasound (LIPUS) have been advocated for acute fractures and nonunions although their efficacy has been contested [62, 63]. A recent meta-analysis and systematic review of 15 trials by Ebrahim et al. suggested benefit to ESTIM over standard care at 3 months; however, this was not statistically significant [62]. Randomized trials have shown mixed results and a Cochrane systematic review in 2011 conducted on ESTIM for nonunion of long bone fractures in adults concluded that its use is inconclusive and insufficient to guide current clinical practice [62, 63]. Gauger et al. included adjunctive stimulation before operative rib fracture nonunion repair [37]. Nine

patients underwent LIPUS for 3 months before surgery [37]. In one patient, one of three of their nonunions healed [37]. A case report by Severson et al. mentioned the use of ESTIM for 8 weeks before operative intervention of a sternal nonunion in a 50-year-old female that ultimately failed [64]. Interestingly, a case report by Chintamaneni et al. reported successful treatment of sternal atrophic nonunion of a 67-year-old male with teriparatide, a synthetic parathyroid hormone, with radiographic union at 9 months [65]. Teriparatide has been reported to be successful in the treatment of nonunion long bone fractures, all though more studies are needed to justify its use and demonstrate its efficacy in the routine treatment of nonunions [66].

Surgical Indications

The ideal rib fracture nonunion patient to offer surgical intervention has a nonunion(s) confirmed by CT scan with discrete, "jabbing" pain at the nonunion site(s), pain on palpation at the site(s), perceived motion at the site(s) with deep breathing that exacerbates the pain, has had the pain for a year or less, and is a nonsmoker [32–39]. Of these, the most indicative patient characteristics predicting a positive response to surgery are focal pain at the nonunion site and nonsmoking [32, 39]. In most situations, prior to consideration of surgery, the patient should be evaluated by a pain management physician, have attempted a work-hardening program mentored by a physical therapist, and be committed to weaning opioids and returning to work. The patient is counseled that surgical intervention requires a several month commitment to progressive recovery that cannot be hurried. The surgeon does not guarantee a positive outcome given that chronic pain due to fracture nonunion can be well-set and may not respond to removal or fixation. In addition, the surgeon should be experienced in acute rib or sternal fracture repair since nonunion surgical intervention is of a magnitude of order more challenging than acute repair.

Sternal nonunion intervention is considered no earlier than 6 weeks after initial injury if instability (clicking, popping, grinding sensations) and debilitating focal pain and tenderness persist [35, 36]. Limitations of activities of daily living and altered pulmonary function are also considerations [35, 67].

Surgical Techniques

Surgical approaches to rib or sternal fracture nonunion mirror orthopedic approaches to fracture nonunions of both long and short bones with the caveat that ribs and sternum are flat and membranous and respond to fixation similarly to the bones of the oral/maxillofacial region, foot, ankle, hand, or wrist. It is not necessary to resect en bloc the entire nonunion. Vascularized bone, even if pocketed with whirls of fibrous tissue, can be left in place and the pockets of fibrous tissue, which may impede healing progression, can be discretely removed with a small nosed rongeur [68]. The edges of bone are "freshened" to stimulate the return of osteogenesis. Orthobiologics with tiny bits of bone autograft harvested from the site are inserted into the spaces at the discretion of the surgeon (Figs. 8.5 and 8.6) and a reliable locking plate expected to withstand the rigors of the fixation for several weeks is placed [37, 39, 69].

During exposure of rib nonunion/malunions, care is taken to avoid further injury to the intercostal nerve; if resected the proximal end should be carefully tied with nonabsorbable suture. Bony connections of adjacent ribs, as seen in the

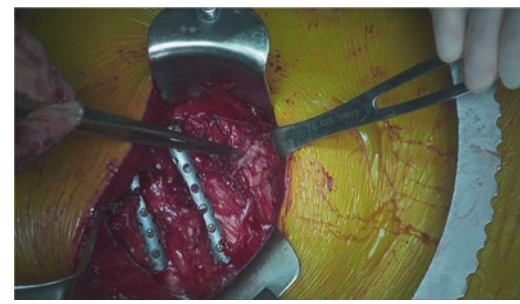

Fig. 8.5 A putty consisting of demineralized bone matrix mixed with bits of bone harvested from the site is placed within the small gaps of the nonunion either prior to or following plate fixation

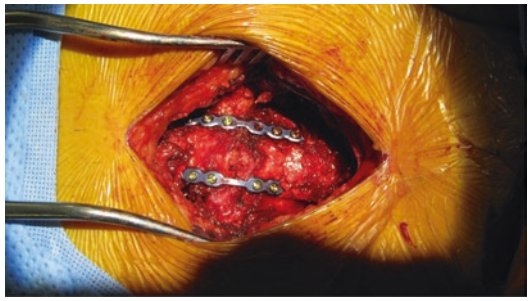

Fig. 8.6 Following plate fixation, demineralized bone matrix mixed with bits of bone harvested from site is placed within the small nonunion gap

more severe cases of malunited flail injuries, are resected to allow for the possibility of improved chest wall expansion.

Absorbable plates have been used for temporary stabilization or as scaffolding for grafting [39, 70]. There is suggestive evidence that polylactide absorbable plates may aid fracture healing [71]. Composite polylactide and hydroxyapatite sheeting that may be osteoconductive is also available [72].

Nonunions with an exuberant pseudoarthrosis or malunions in unusual locations such as the first or second rib with impingement on the brachial plexus or with associated pain that prevent an athlete from continuing in their chosen sport can be considered for complete resection since they are not essential to thoracic physiologic function (Fig. 8.7) [73]. Malunions that impinge on adjacent structures such as the scapula or adjacent ribs or are perceived by the patient, for example, when sitting in a chair or lying in bed, can be burred flat (Fig. 8.8).

Symptomatic cartilaginous nonunions require special attention since they are not likely ever to heal. One approach is resect any exuberant surrounding fibrous tissues and construct a "sandwich" repair with 1 layer of polypropylene mesh and the injured area "sandwiched" by an exterior and interior absorbable plates (Fig. 8.9). This repair provides flexibility and durability thus mimicking expected chest wall cartilaginous function.

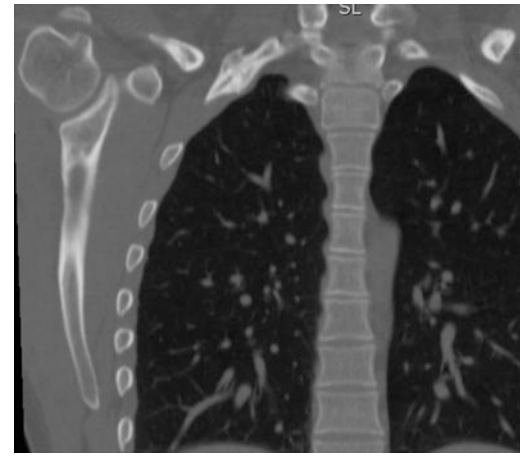

Fig. 8.7 First rib fracture nonunion in a 20 year old female collegiate tennis player 2 years post injury. Symptomatic improvement followed debulking of hypertrophic bone, resection of fibrous nonunion, and compression plating through a posterior lateral cervical approach

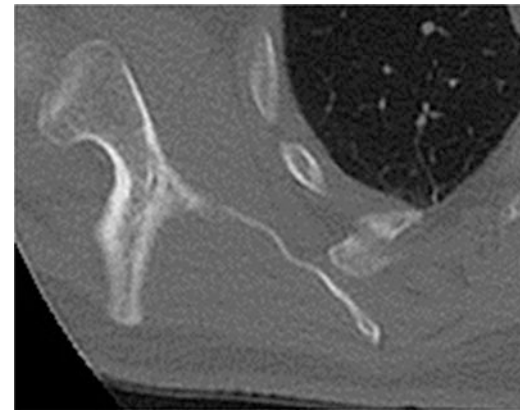

Fig. 8.8 Symptomatic impingement of hypertrophic rib fracture malunion beneath the scapula. Leveling of the malunion with a bur was curative

Adjunctive Orthobiologics

Orthobiologic substances such as packaged allograft, demineralized bone matrix (DBM), calcium ceramics, bone morphogenetic protein (BMP), bone marrow aspirate, and platelet-rich plasma with or without bone allograft are frequently used adjunctively in the operative man-

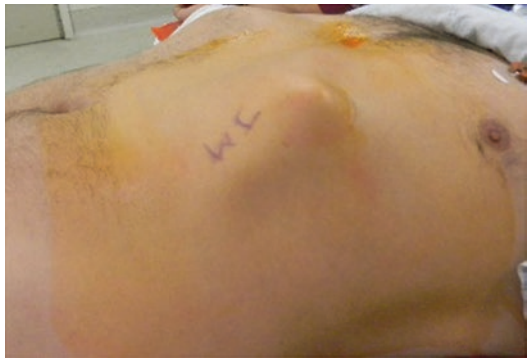

Fig. 8.9 Left costal margin cartilaginous fracture nonunion/malunion 1 year following a wrestling injury. Minimal resection, anatomic reduction, and "sandwich" plating with polylactide plates and polypropylene mesh provided symptomatic relief and durability

agement of challenging fractures, delayed unions, nonunions, and in bone defects [74]. These substances are expected to stimulate or augment bone formation by one or more of three properties: osteoconduction, osteoinduction, or osteogenesis. Bone autograft promotes all three properties and is the gold standard. DBM and calcium ceramics, in contrast, provide passive osteoconductive scaffolding and BMP is purely osteoinductive. Packaged allograft bone is osteoconductive and osteoinductive whereas bone marrow aspirate and platelet-rich plasma are osteoinductive and osteogenic.

Operative Outcomes

Rib Fracture Nonunion

Table 8.2 outlines the results of the several prospective and retrospective case series of surgical intervention for symptomatic rib fracture nonunions [32, 33, 37–39]. All studies listed reported improvements in pain, function, or activity in the majority of patients. The aggregate mean time between injury and intervention reported was approximately 15 months (range 4–197 months). Fabricant et al. is the only prospective study that reported validated quality of life metrics (RAND36 and MPQ) and pain medication usage before and after intervention [39]. The majority of patients increased their daily activity and morphine equivalent dosing (MEDs) decreased from an average of 20.3 MEDs to 9.4 MEDs ($p = 0.054$) [39]. These results were echoed by Hernandez et al., where the majority (five of six patients) experienced reduction in pain scores 6–12 months postoperative [33]. De Jong et al. reported 5 out of 19 patients with persistent neuropathic pain that was difficult to treat that was thought to be secondary to intercostal nerve entrapment, highlighting the need to consider intercostal nerve damage as a source of pain for nonunion patients [32].

Two of six studies reported 100% union after intervention, although radiographic evidence of union was not uniformly confirmed [32, 33, 37–39]. Complications rates varied by study and ranged from infection to implant failure and request for removal due to irritation (Table 8.2) [32, 33, 37–39]. Fabricant et al. reported a single case of pulmonary hernia after adjacent rib resections and de Jong et al. reported two cases of new fractures ventral to the plate repair that required reoperation [32, 39].

Sternal Fracture Nonunion

Overall, the operative outcomes of sternal nonunion repair are favorable with the vast majority of patients receiving symptomatic relief, reduced pain, improvement in activity and functional levels, and reduced analgesic requirements after fixation [75–84]. Table 8.3 provides an outcomes summary of several case series, case reports, and one prospective cohort study of traumatic sternal fracture nonunion operative and nonoperative intervention [64, 65, 75–84]. Queitsch et al. prospectively studied 12 patients who underwent operative repair of traumatic sternal fracture acutely (two patients) and those with nonunion (ten patients) [75]. The majority of patients (10 of 12) were able to return to work at their previous occupational level, were pain

Table 8.2 Literature review of rib fracture nonunion operative intervention

Author/year	Study type	No.	Intervention (No.)	Follow-up	Outcomes (No.)	Complications (No.)
Fabricant 2014 [39]	PC	24	Resection Locking plates Absorbable plate with cerclage Intercostal nerve release (9)	60, 120, 180 days	Lower MEDs Reduced pain Improved activity levels No change in functional or work status	Wound infection (1) Screw dislodged (2) Pulmonary hernia (1)
Gauger 2015 [37]	RC	10	Locking plates(9) Rib resection Mesh repair (2) Bone graft (10)	3–46 months	80% of patients returned to work[a]	Wound infection (1) Implant removal (1)
de Jong 2018 [32]	RC	19	Locking plates Bone graft (2)	38 months	Reduced pain specialist use Persistent neuropathic pain (5)	Implant removal (2) Nonunion (1) Implant failure and new fracture (2) Neuroma (1)
Hernandez 2018 [33]	RC	6	Locking plates Bone graft (6)	2–24 months	Reduced pain (5)[a]	Hematoma (2) Seroma (1) Implant failure (3)
Edwards 2018 [38]	CS	24	Locking plates (6) Bone graft	Unknown	Modest pain reduction	Implant failure (1) Screw dislodged (1)

Abbreviations: *PC* prospective cohort, *RC* retrospective cohort, *CS* case series, *No.* number of patients, *MEDS* morphine equivalent dose
[a]Documented 100% union rate

free (10 of 12), and demonstrated union at follow up (12 of 12) [75]. A reduction in pain was further supported by a study by Zhao et al. who looked at 64 patients who had traumatic fracture of sternum and 13 resultant nonunions [77]. After operative repair with plating and allogenic bone graft, there was pain reduction from an average of 7.74 to 3.8 on the NRS and a statistically significant improvement in pulmonary function as measured by FEV1 and FVC [77]. Molina et al. reported a series of ten patients who underwent sternal wiring and pinning for sternal fracture nonunion [79]. All patients were able to return to their previous level of activity and function [79]. Richardson et al. in a case series of 16 patients with delayed sternal union found that operative fixation with locking plates and bone grafting resulted in significantly reduced pain and no narcotic requirements in patients after fixation [80]. Morgan et al. reported an interesting case of a 26-year-old male professional rugby player who experienced a nonunion after an injury resulting in significant pain [81]. He underwent locking plate fixation with continued symptoms of nonunion 10 weeks after the initial operation [81]. A second operation was performed where BMP was added to a collagen sponge to stimulate bone growth [81]. Successful union was reported 4 months later, and he was able to return to his career as a professional rugby player [81].

In most studies, operative fixation of the nonunion was completed via locking plates with or without bone grafting [64, 75–84]. Molina et al. used sternal wires and pinning to achieve nonunion in 10 patients with good result [79]. Complications were relatively infrequent and of low severity and listed in Table 8.3. Of note, as with rib nonunion hardware, sternal nonunion hardware occasionally required removal due to irritation/discomfort and at the patient's request [75–86].

Table 8.3 Literature review of traumatic sternal fracture nonunion intervention

Author/year	Study type	No.	Intervention (No.)	Follow-up	Outcomes (No.)	Complications (No.)
Mayba 1985 [84]	CR	2	Locking plates Bone graft	3–4 years	Pain free 100% Union	None
Coons 2002 [83]	CR	2	Sternal wires Bone graft	3 years	Pain free 100% Union	None
Molina 2005 [79]	CS	10[a]	Sternal wires and pinning	0.5–17 years	Return to activity No restrictions	Pin migration (1) Wire discomfort (1)
Wu 2005 [76]	CS	2[a]	Locking plates	6–18 months	Pain free and improved stability 100% Union	None
Gallo 2006 [78]	CR	1	Locking plates Bone graft	9 months	Pain free Return to activity	None
Lundy 2007 [82]	CS	1[a]	Locking plates	1 year	Stability Pain free	None
Richardson 2007 [80]	CS	16[a]	Locking plates Bone graft	NR	Pain reduction No narcotic requirements 100% Union	Hardware migration (1)
Morgan 2008 [81]	CR	1	Locking plates BMP	4 months	Return to activity and work	Persistence of nonunion after operation requiring BMP
Chintamaneni 2009 [65]	CR	1	Teriperatide	9 months	Union achieved Resumed activity	None
Severson 2009 [64]	CR	1	ESTIM Locking plates	1 year	100% Union Reduced pain	Hardware migration
Queitsch 2011 [75]	PC	10[a]	Locking plates Bone graft (7)	NR	Return to work (10) Reduced pain Persistent pain in two patients 100% Union	Wound infection (1) Hypertrophic scar (1)
Zhao 2017 [77]	CS	13[a]	Locking plates Allogenic bone graft	6 months	Reduced pain (avg 7.74 to 3.80 on 10 pt. scale) Increase in lung function 100% union	None

Abbreviations: *PC* prospective cohort, *CR* case report, *CS* case series, *No.* number of patients, *NR* not reported, *BMP* bone morphogenetic protein, *ESTIM* electromagnetic stimulation, *avg* average, *pt* point
[a]Reflects only traumatic nonunion patients

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Sternal Fracture Repair

9

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Introduction and Review of Literature

Traumatic sternal fractures are an uncommon but potentially debilitating injury. The most common mechanism is direct blunt trauma to the anterior chest wall caused by motor vehicle crashes (MVCs) [1, 2]. The overall incidence of sternal fractures amongst all broken bones is less than 0.5%, and the incidence of sternal fractures directly related to blunt trauma is estimated at 3–8% [1–4]. Traumatic sternal dislocations have an even lower incidence, one that is hard to ascertain from current literature [1]. There has been a rise in sternal injuries attributable to universal seatbelt legislation. Sternal fractures are also frequently seen with falls from height or indirect trauma due to spinal flexion-compression injuries [1–4]. CPR survivorship with unstable sternal fracture is another category of indication with growing interest and experience.

Morbidity and mortality from sternal fractures is determined primarily by associated intrathoracic injuries. The mortality from sternal fractures ranges from 4% to 45% [1, 3]. Most commonly associated injures include vertebral fractures (specifically cervical and thoracic spine fractures), rib fractures, clavicular fractures, scapular fractures, pulmonary contusion, hemopneumothorax, cardiac and mediastinal injury, and aortic dissection [1]. These concomitant injuries, coupled with severe chest pain, can lead to respiratory insufficiency/failure, organ failure, and ultimately death [1, 3, 4].

The treatment of traumatic sternal fractures has remained relatively consistent over the years. Although there is growing interest in and demand for a general treatment algorithm of sternal fractures that include operative and nonoperative management, there is a paucity of literature or general agreement among experts on the ideal management strategy [4]. The majority of sternal fractures (>95%) are treated nonoperatively with pain control, corset fixation, rest, and passive reduction of displacement if severe [1, 2, 4]. Adequate pain control is the mainstay of nonoperative management as uncontrolled pain can directly lead to respiratory compromise and/or failure and its attendant complications [1, 4]. Recently, operative sternal fixation has emerged as a viable management option for sternal frac-

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tures. It should be considered for patients with sternal or chest wall instability, fracture displacement, sternal deformity, respiratory insufficiency, uncontrolled severe pain, or fracture nonunion [1].

Several fixation techniques have been described in the literature, of which wiring and plating are most common [1]. A recent literature review demonstrated that of the 191 patients managed operatively over a 10 year period, 83% of patients underwent surgical sternal fixation using only plates and the remainder (16%) undergoing surgical fixation with a combination of plates and bone graft [1]. Simple wire fixation, (still the most frequently utilized technique for midline sternotomy fixation) has essentially been supplanted by plates and screws. Surgeons have developed an appreciation of the difference between transverse and vertical sternal defects and the biomechanical advantages of vertical plate constructs for the former [1]. These advantages include superior stability, better restoration of chest wall mechanics, improved pain control, and improved bone healing, resulting in less risk of fracture nonunion [1–4]. Furthermore, sternal fixation is accompanied with a low complication rate of only 3% in current literature, whereas only 1% of patients required reoperation [1]. Complications include hardware failure or dislodgement, infection, wound dehiscence, or fracture nonunion. Most patients requiring reoperation had hardware failure or dislodgement [1]. While sternal fracture nonunion is rare (incidence of <1%), bone grafting, plus surgical fixation with plates, is often reserved for these cases due do its osteoinductive properties [1, 4].

Pain relief and hospital length of stay after sternal fixation remain difficult to quantify. This is largely due to the high frequency of concomitant injures. A recent study by Zhao et al. examined 64 patients, all undergoing sternal fixation with a plate and locking screw system. They found a statistically significant reduction in pain scores after surgery by almost 4, however, these same results have not been reported in other studies [2]. Furthermore, a recent systematic literature review study quoted an average hospital length of stay 15 days with a range of 3–59 days [1]. These data underscore the difficulty in teasing out the benefits of sternal fixation from retrospective analyses. The authors of this chapter have anecdotally witnessed, on multiple occasions, immediate improvements in pain scores, enhanced mobility, and decreased hospital length of stay after sternal fracture stabilization surgery. These outcomes coupled with improvements in upper extremity range of motion and the potential for earlier return to activities of daily living, make surgical stabilization of traumatic sternal fractures an attractive option for these patients.

The chest wall surgeon needs a strong working knowledge of sternal anatomy and sternal fracture patterns to maximize success and minimize complications. As previously discussed, semirigid plates and locking screws currently afford the best results and will be described here in further detail [1].

Techniques of Repair

The surgical repair of a sternal fracture is a relatively straight forward and quick procedure. A recent study of 64 patients undergoing sternal fracture fixation demonstrated a mean operative time of 42.62 ± 10.23 min [2]. To help simplify the procedure itself, it can be broken down into five main steps: (1) preoperative planning, (2) exposure, (3) fracture reduction, (4) fracture stabilization, and (5) closure.

Preoperative Planning

If it has not already been obtained, a helical computed tomography (CT) is highly recommended prior to surgical intervention. Although a standard chest X-ray can provide some information about chest wall injury, a CT scan provides significantly more information which will optimize planning. In addition to identifying associated injuries such as rib fractures, pulmonary contusions, retrosternal hematoma, etc., the sagittal views will quantify any anterior–posterior disassociation of the fracture segments. The coronal views will help characterize fractures with

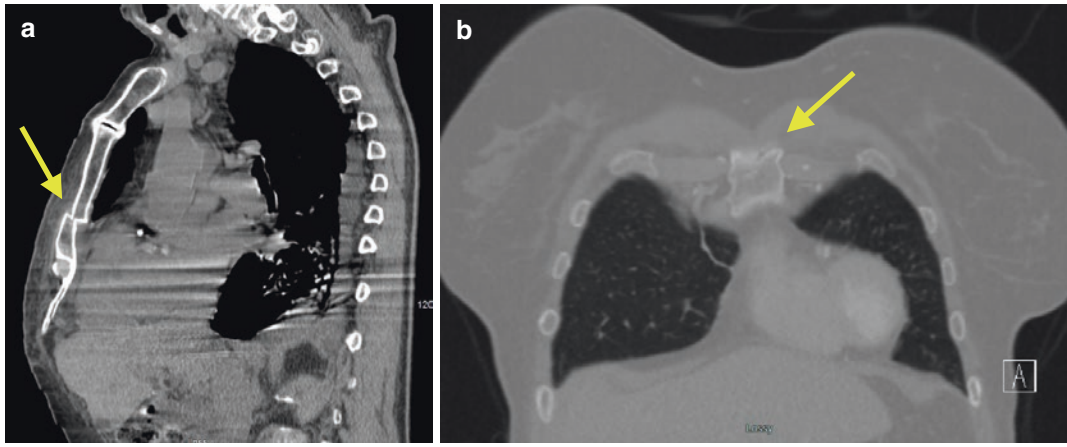

Fig. 9.1 CT scan of the chest for preoperative planning. (**a**) Sagittal view showing displacement of fracture (arrow) and the relationship to the xiphoid and sternal angle. This view can also be used to measure sternal bone thickness for bicortical screws if needed. (**b**) Coronal view showing oblique sternal fracture (arrow)

oblique or vertical elements (Fig. 9.1) [5]. The CT scan allows the surgeon to measure the distance from the sternal angle, jugular notch, or xiphoid process directly to the fracture itself, allowing for more accurate placement of the incision [5]. If the sternal fracture is more oblique, this should be considered in the preoperative planning as it may influence the length of your incision in order to provide adequate exposure to secure plates to the sternum.

Exposure

The patient should be under general anesthetic and in the supine position on the operating room table. Tucked arms provide the most room for the surgeon to maneuver around the patient's torso. Although tucking the arms can make placement of a chest tube difficult, the authors find it rare for a patient undergoing isolated sternal fixation to require a chest tube. It is important to identify and mark anatomical landmarks such as the jugular notch, sternal angle, and xiphoid process. It is also important to stay directly over the middle of the sternum and to minimize incision length for optimal cosmesis. Although the fracture is often palpable once the patient is asleep (it is very difficult to palpate the fracture when the patient is awake given the amount of pain associated with these fractures), using the patient's anatomical landmarks and the measurements obtained from the CT scan, the operating surgeon can be very accurate in locating the site of the fracture. For those individuals proficient at ultrasonography, utilization of this technology can also be very helpful when trying to identify the fracture site.

Once the fracture site is identified, the surgical field should be prepped and draped in standard sterile fashion for your institution. The incision should be made directly over top of the fracture site utilizing the fracture site as the midpoint of the incision. The incision does not need to be the entire length of the sternum. Typically, a 6–8 cm incision is adequate (Fig. 9.2). It is important to remember that you can always make the incision bigger if better exposure is required; however, you cannot make the incision smaller once it is created.

The pectoral muscles insert onto the anterior sternum and these should be cautiously lifted off the sternum toward the lateral sternal borders. It is important to respect the anatomical soft tissue planes as this will help in the eventual closure of the incision. One should minimize damage to the periosteum. Expose the fracture and remove any intervening tissue or clot (Fig. 9.2). Expose the anterior sternal surface of both segments to allow

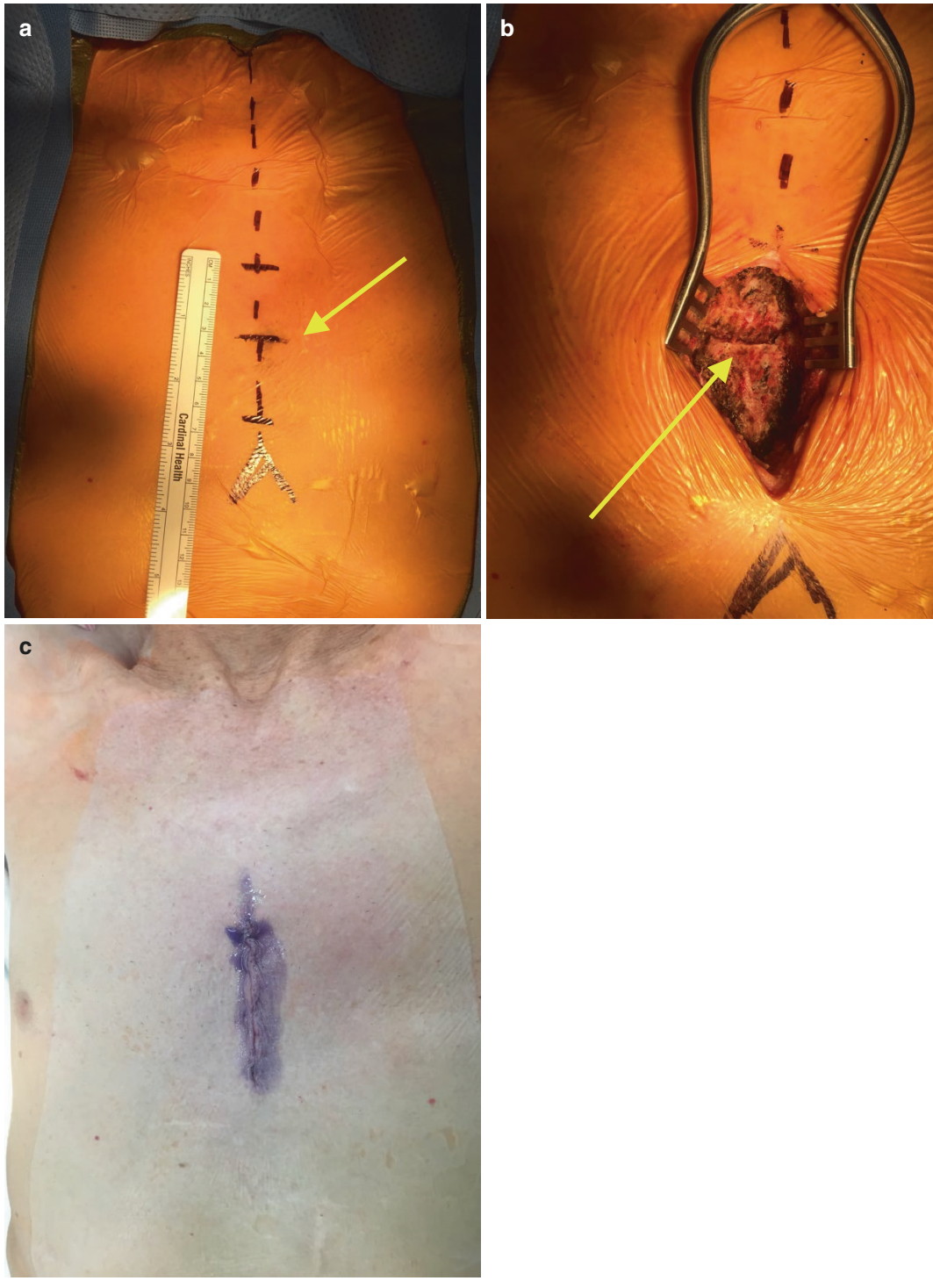

Fig. 9.2 Incision and exposure. (**a**) Incisional planning (6 cm) with the underlying fracture palpable in the middle at the arrow. (**b**) Exposure of sternal fracture (arrow) after the pectoral muscles have been dissected laterally. (**c**) Closed incision with skin glue. (Top of all pictures is jugular notch and the bottom is the xiphoid process)

for precise apposition of plates to bone. This generally requires a minimum of 2–4 cm on either side of the fracture line.

Fracture Reduction

This is often the most challenging aspect of this operation, especially if there is significant displacement with overlap of fracture segments. Prior to incision, the surgeon may place a roll behind the patient's back at the level of the scapula. This provides some arch and stretch to the sternum and can aid with sternal fracture reduction.

Once exposure is obtained, several methods can be utilized to help with reduction. A single screw can be placed in the middle of the posteriorly depressed portion of sternum. This screw should be much longer than intended for securing the plate and left protruding out of the bone approximately 0.5–1 cm. The surgeon can then grasp this protruding screw with a hemostat and apply light traction helping to bring the depressed bone back into a reduced position (Fig. 9.3). Care should be taken during this process as pulling to hard can dislodge the screw altogether resulting in bleeding from the bone marrow of the sternum.

Additional reduction methods include the use of bone reducing forceps to grasp the lateral sides of the posteriorly depressed portion of the bone and applying gentle anterior traction until the fracture is aligned properly. This may require simultaneous manipulation of the anteriorly raised portion of the fractured bone in order to properly realign the fracture. Lastly, a plane can be created posterior to the depressed portion of the sternal fracture, making sure to stay as close to the posterior periosteum as possible. Then using an elevating device (long right angle, compression wires, pointed ball forceps, etc.), gentle anterior pressure is applied to the posterior aspect of the posteriorly depressed bone, raising it back into a reduced position [5]. Care should be taken utilizing this last method given that the pericardium and heart lie directly posterior to the sternum. Furthermore, it is important to remember the internal mammary arteries run near the lateral border of the sternum and can easily be injured with aggressive dissection or manipulation. Of note, many patients with sternal fracture will have a retrosternal hematoma which may be encountered during this step and should be irrigated out thoroughly.

Another technique for reducing these fractures is to use an orthopedic foot distractor. This reduction technique involves placing two

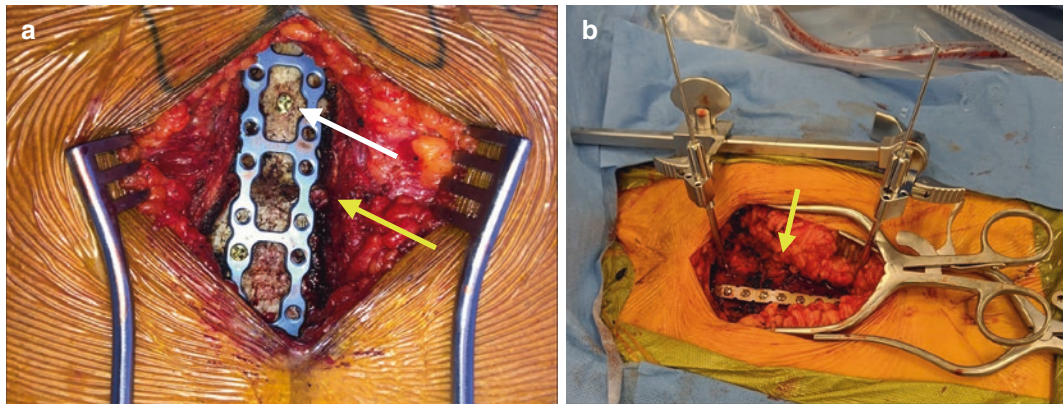

Fig. 9.3 Reduction. (**a**) Single screw (white arrow) is placed in the middle of the depressed portion of the sternal bone and gentle traction applied anteriorly to reduce the fracture while the plate is secured in place. (Yellow arrow is the sternal fracture). (**b**) Use of the orthopedic foot distractor for reduction of the sternum. (Yellow arrow is at the level of the fracture)

2.5 mm threaded k-wires into the bony plate of the sternum or manubrium superior and inferior to the fracture line. Care must be used to ensure that the k-wires do not penetrate any mediastinal structures. It is extremely important to ensure that measurements of the thickness of the sternum are exact and the wires are not inserted deep to the posterior wall of the sternum. These are then attached to the distractor. The distraction knob is turned to pull the superior and inferior sternal bones apart and get them to length. When reduced to full length, often the fracture will auto reduce into its normal anatomic position. Occasionally additional elevation using a freer elevator is needed to make the anterior cortex of the sternum align. After the distractor has been extended to length, it remains in place while a plate is attached to the two sternal fracture pieces holding the reduced fracture line in place (Fig. 9.3). The distractor is then released, and the k-wires removed from the sternal body.

Fracture Stabilization

As stated previously in this chapter, the authors recommend a plate and locking screw system for this portion of the procedure given the proven superior biomechanical advantages [1]. There are several systems currently available for sternal fixation and the choice of plate and locking screw should be based on surgeon comfort and experience. Most of the plates and screws are made of commercially pure titanium or titanium alloy, which affords acceptable strength with some flexibility. Allergy to metal is rare but can result in intolerance and lead to plate removal. There are no published head to head comparison studies to inform the use of one system over another. A common technique is the use of two parallel straight plates, like "railroad tracks," secured to the lateral aspects of the sternum. Single long ladder-shaped plates are also commonly used (Fig. 9.4). One key is to contour the plate(s) to the patient's sternum without "over bending" which

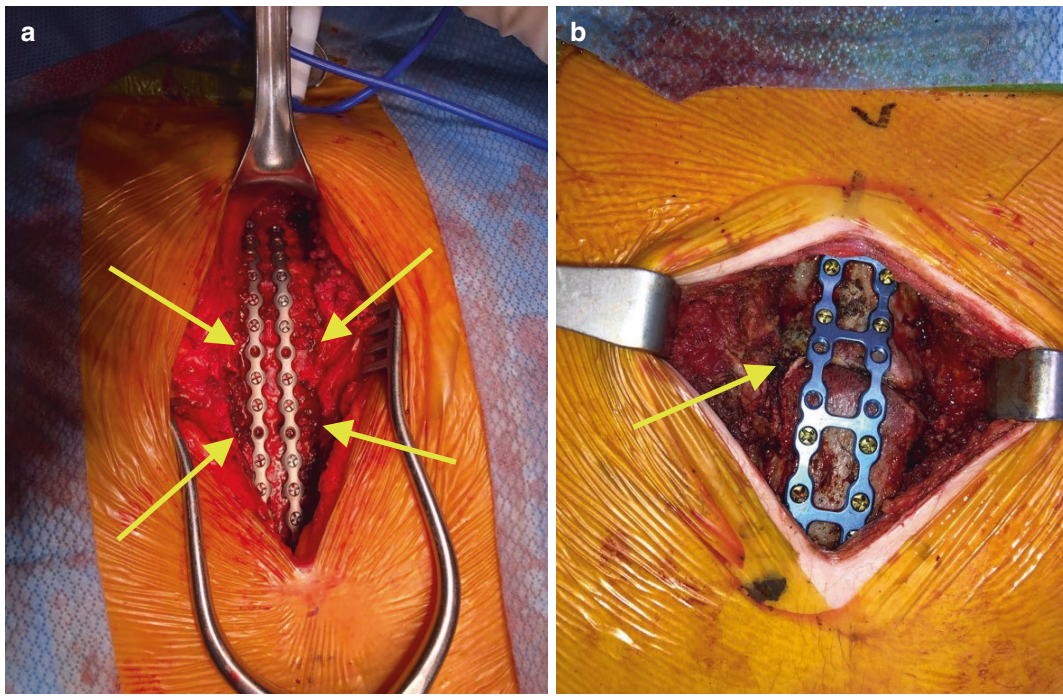

Fig. 9.4 Secured plates. (**a**) Two straight plates running parallel to each other like "railroad tracks." There are two fractures present so four screws were placed in the middle segment of sternal bone. (**b**) Single ladder plate spanning the fracture site. (Fractures at arrows)

may induce plate stress and the potential for plate failure. This shaping process can be time consuming but is critical to prevent gaps between plate and bone or undo stress on the construct with clamping to "force" a good plate to bone match. Furthermore, it is important to keep the sternal fracture centered within the span of the plate(s) to allow for adequate coverage of the fracture and solid fixation. One advantage of using two separate straight plates is the ability to independently shift the plates superiorly or inferiorly on the sternum to maintain adequate coverage of obliquely oriented fractures. Another theoretical advantage of parallel plates is the opportunity for future median sternotomy without plate removal.

Locking screws are used with all current systems. Locking screws are designed to "weld" to the plate, producing a situation that is prone to failure only if the entire construct fails. This design is the standard for modern rib fracture plating systems. There are systems that utilize monocortical or bicortical screws. If bicortical screws are utilized, the bone thickness of the sternum must be determined. A ready and reliable way to do this is by obtaining measurements of the sternum thickness from the CT images. Most plating systems have a bone measuring caliper or other blunt device for measuring sternal thickness but access to the posterior sternum must be obtained to accurately obtain this measurement.

The plate(s) must be held secure to the sternum while pacing screws as locking screws do not "lag" the bone and plate together. This can be done using a small ball pointed forceps or sometimes just gentle posterior pressure of the plate onto the sternum. Most plating systems also have temporary screws which can be used to hold the plate in place but also allow for manipulation of the fracture site, allowing for other permanent screws to be placed. Once the plate is secure enough, the temporary screws are removed and replaced with permanent screws. With recent advances in technology, most plating systems utilize a self-drilling screw, however not all. Three to four screws are recommended on either side of the fracture line to adequately secure the plate in place. Longer lengths of fixation provide for more "load sharing" and likely lower failure rates.

More complex fracture patterns may require transsternal fixation to adjacent ribs. Sternal repair may be one element, along with surgical stabilization of rib fractures, to anterior flail chest stabilization. This may also require a larger "anchor-type" incision to provide adequate exposure of both the sternal fracture(s) and bilateral rib fractures (Fig. 9.5). Utilization of this type of incision allows for creation of pectoral flaps in a medial to lateral direction while preserving normal pectoral function. It is usually recommended to utilize a bone graft if there is a 0.5 cm gap or larger at the fracture reduction site. An autograft can be harvested from various locations in the body (most common being the iliac crest) or there are several manufactured bone matrices that can be utilized. Which product to use for the graft is based on surgeon familiarity and experience.

Closure

Once you have adequately reduced, stabilized and plated the sternal fracture, it is now time to close the incision. Although very rare, the surgeon should assess for a pleural lesion bilaterally. If hemothorax is identified or highly suspected, a chest tube should be inserted to maintain lung expansion. The chest tube can likely be removed within the next 24–48 h. We recommend closing the incision in multiple layers to provide adequate coverage. A chest X-ray should be obtained in the recovery room to assess proper placement of the sternal plate(s) and that no hemopneumothorax is present. Continuation of a pain regimen is usually required but should be tailored as these patients often will have significant pain reduction postoperatively. Furthermore, these patients do not usually require significant activity restriction postoperatively.

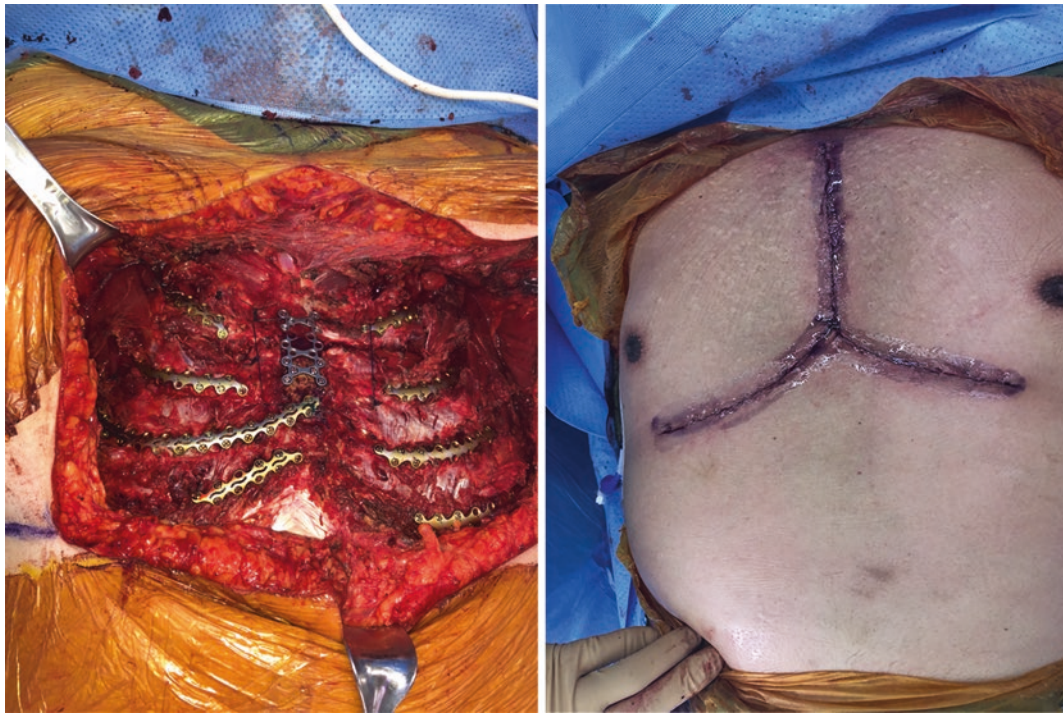

Fig. 9.5 Combined sternal and anterior rib exposure. Anchor-shaped incision with medial to lateral creation of pectoral flaps allows for great exposure to surgical stabilize the sternal fracture(s) (midline) and anterior bilateral rib fractures simultaneously, relieving the patient's anterior flail chest physiology. Some rib fracture require stabilization to the sternum itself. (Sternal notch at top of picture and xiphoid process at bottom)

Conclusion

The surgical management of traumatic sternal fractures is maturing rapidly. Current literature supports the utilization of a plate and locking screw systems which are demonstrably safe and effective in restoring normal chest wall mechanics, reducing pain, and promoting bone healing. As the management of sternal fracture patients continues to evolve, so too must the guidelines and indications for operative intervention. Sternal fracture repair is generally a straightforward, safe, and gratifying procedure. Surgical fixation should strongly be considered for patients with traumatic sternal fracture in the settings of chest wall instability, respiratory insufficiency, severe pain, fracture displacement, or fracture nonunion.

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Clavicle and Scapula Fractures

10

D. A. Carlson, L. K. Schroder, and P. A. Cole

Introduction

In the acute phase of severe chest wall trauma, hemostasis, ventilation, and survival are the primary concern. At times, chest wall stabilization may play a key role in this acute phase treatment. Once the patient is physiologically stable, an interdisciplinary discussion is helpful to time subsequent surgeries in the polytrauma patient. There is a titration of treatment that should occur based on prioritization of injuries and the tolerance of the patient to withstand the surgical challenge. Whether or not open reduction and internal fixation of flail segments have already been addressed, the diagnostic workup of concomitant orthopedic injuries to the shoulder girdle should be completed as a diagnostic sequence.

There are times when concomitant operative treatment of ribs and the fractured scapula or clavicle would be most appropriate. Certainly, if the patient can tolerate the extended operative session and is under physiologic control, it makes sense to time surgeries together, especially when in the same patient position and operative field. The combined procedures may take coordination between services if rib fixation is not a part of the orthopedic surgeon's domain. Our goal is certainly to progress the patient to as fast a recovery as possible, beginning with optimum ventilatory mechanics, to full range of motion of the upper extremities and ambulation as possible given lower extremity injuries, "Get 'em extubated and get 'em going!"

The importance of the shoulder for positioning the hand in space where it can perform both high-dexterity tasks, as well as lifting and resistance, cannot be overstated for patient independence. The scapula, clavicle, and complex of associated joints provide an intricate linkage between the axial and appendicular skeleton. The sternoclavicular joint for example allows for coordinated movement of up to 55° and in multiple planes, whereas the more restricted acromioclavicular joint allows for approximately 30° of elevation, along with translation and axis for coronal and sagittal plane movement. Combined injuries of any or possibly all

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of these structures should be ruled out particularly in the "forequarter lateral implosion" injuries to the chest wall in which segmental rib fractures often occur.

At the worst end of the spectrum is scapulothoracic dissociation with brachial plexus palsies and vascular injury. In such patients, above-elbow amputation may be in the patient's best interest to allow for restoration of maximum independence through the use of an above elbow prosthesis. While this is the most extreme example of loss of function, it does underscore the importance of the shoulder girdle attachments to the thorax. As discussed below, there are many more common scenarios in which injury to the shoulder girdle and thoracic attachments can lead to chronic pain, weakness, dysfunction, and overall decreased quality of life. For this reason, there must be a high index of suspicion and a thoughtful treatment plan for shoulder girdle injury in association with chest wall trauma. Below, we divide these injuries into the broad categories of clavicle fractures and scapula fractures; however, it should be noted that the surrounding ligaments, muscles, tendons, and cartilage are also paramount and deserve careful consideration. Injuries to the sternoclavicular and acromioclavicular joints represent common breaches in the shoulder girdle during this injury mechanism.

Even if not providing the definitive treatment for these shoulder girdle injuries all treating physicians should be aware of them for a number of reasons: (1) shoulder girdle injuries often are first identified on initial trauma imaging including chest radiographs and CT scans; (2) while many clavicle and scapula injuries can be treated nonoperatively it is clear that some injury patterns meet surgical indications; (3) there is often the opportunity for concurrent surgical treatment of clavicle and scapula fractures with rib fracture fixation or other procedures in the same surgical setting and possibly with a multidisciplinary approach.

The superior shoulder suspensory complex (SSSC), as described by Goss [1], refers to the bony, muscular, and ligamentous structures that attach the upper extremity to the thorax (Fig. 10.1). This includes the clavicle and scapula, which serve as bony struts, and the surrounding soft tissue ring including the acromioclavicular and coracoclavicular ligaments. The medial clavicle anchors the SSSC to the sternum at the sternoclavicular joint, while the scapula interfaces with the thorax through a large bursa that facilitates gliding as the surrounding muscular envelope stabilizes and moves it. In this way the SSSC serves as a dynamic foundation to position the upper extremity in space. The SSSC can have a singular injury, as with a clavicle fracture, or in multiple places at once. If there is injury to both the clavicle and scapular body or neck, then the shoulder girdle becomes significantly unstable and the injury has been named a double disruption of the SSSC [1] or a "floating shoulder" [2–5].

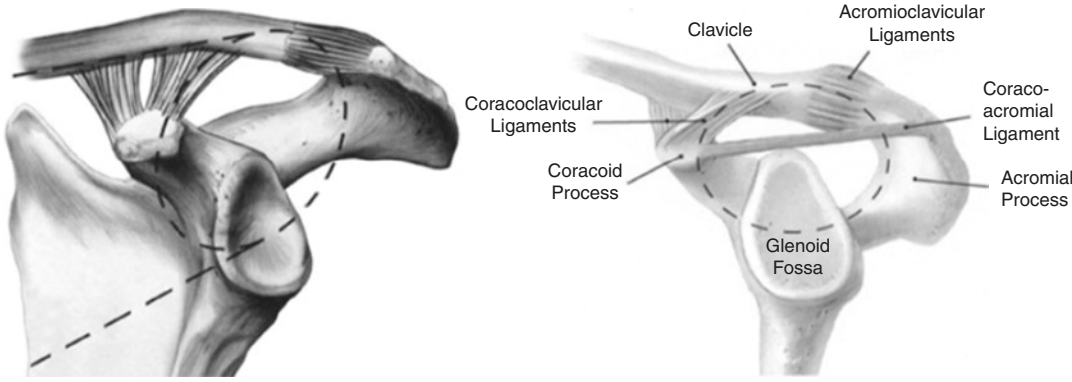

Fig. 10.1 Superior shoulder suspensory complex illustration from Goss et al. [1] (*with permission*)

Clavicle Fractures

Background

Epidemiology

The clavicle is the most commonly fractured bone in adults with an incidence of 2–10% [6–9]. Clavicle fractures have a bimodal incidence distribution with more high energy traumatic injuries occurring in young adults and low energy mechanisms in the older geriatric population [6]. The most common mechanism of injury is a fall onto an outstretched arm which leads to axial loading of the bony strut. However, when associated with chest wall trauma the clavicle often fractures as the result of a direct blow to the diaphysis or a lateral compression impact to the shoulder and thorax.

Development

The clavicle is the first bone in the human embryo to begin to ossify, at 5–6 weeks of gestation, and the last to fully ossify, which can occur as late as age 25 at the medial physis and approximately age 18 at the lateral physis [10, 11].

Osteology

When viewed from superior, the clavicle has a distinct "S" shape. It is significantly wider at the medial and lateral metaphases and thinner in the diaphysis. When viewed from directly anterior, on the other hand, it has a straighter appearance with a thicker medial end becoming thinner toward the lateral side. The detailed three-dimensional bony morphology is better appreciated in Fig. 10.2. The clavicle is often divided into the medial, middle, and lateral thirds when describing the location of a fracture.

Deforming Forces

The sternocleidomastoid muscle inserts onto the superomedial clavicle, which often pulls the medial fragment of a fracture upward [13, 14]. Conversely, the lateral clavicle provides the bony origin for the clavicular head of the pectoralis major, which acts as an inferior deforming force. The coracoclavicular ligaments, including the conoid and trapezoid, are attached to the inferolateral clavicular border and provide a tether which impacts the deformity by virtue of the relative location of the fracture [15, 16]. The trapezius and deltoid muscles also have attachments to the far lateral clavicle.

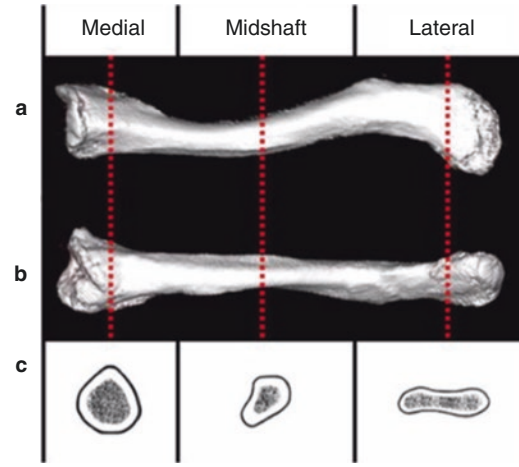

Fig. 10.2 Clavicle bony morphology. (**a**) superior surface view, (**b**) anterior surface view and (**c**) cross-sectional view. (Graves et al. [12]. *With permissions*)

Surrounding Structures

Directly anterior to the clavicle in the subcutaneous layer lie usually three branches of supraclavicular nerves [17]. These nerves run from superior to inferior and provide sensation to the skin of the surrounding anterior chest. These are almost always encountered during the surgical approach to the clavicle. With careful dissection it is possible to isolate and preserve them; however, they are often sacrificed producing a small patch of postoperative numbness. Just posterior to the medial clavicle lies the carotid artery, vagus nerve, and jugular vein. These structures can be at risk with medial clavicular injuries, or with a posteriorly displaced sternoclavicular dislocation. At the middle third of the clavicle, the brachial plexus, subclavian artery, and subclavian vein course posteriorly in close proximity to the bony cortex. These can be injured by a displaced fracture fragment and can be at risk during surgical exposure and fixation of fractures [18, 19]. The lateral third of the clavicle generally does not

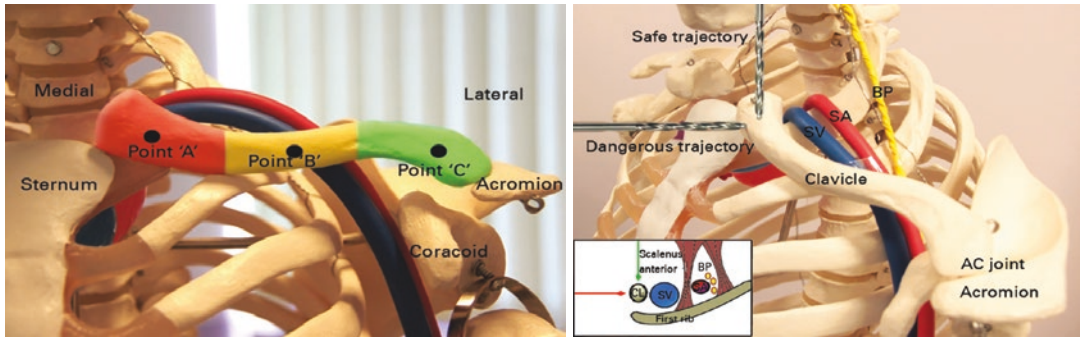

Fig. 10.3 Illustration of anatomical relationships of the clavicle. (Sinha et al. [19]. *With permissions*)

have any large neurovascular structures in close proximity; however, the smaller subacromial artery is occasionally encountered during inferior dissection of the lateral clavicle (Fig. 10.3).

Diagnosis

Clinical Evaluation

Clinical evaluation should always begin with a detailed history and physical exam. The patient's overall health and comorbidities should be assessed to help determine surgical candidacy. Additionally, it is useful to know if the patient has high demand function, as with an athlete, or if they are low demand, as with a homebound geriatric patient to state the extremes of profile.

Particular attention should be given to the neurovascular status of the extremity to asses for possible brachial plexus or vascular injury. An audible bruit at the fracture site indicates turbulent arterial flow and arterial injury [20]. In such cases a CT angiogram would be warranted. Jugulovenous distension may indicate venous obstruction at the fracture. The skin surrounding the entire shoulder should be fully exposed and examined to assess for open fractures or tenting skin that is threatened of possible necrosis, both of which would be indications for urgent surgical intervention. The skin, however, is very mobile in this region and despite tenting of skin it is extremely rare for skin to be actually threatened. If open fractures are identified prompt administration of IV antibiotics is warranted. The location and severity of skin abrasions should be noted as this may affect surgical incision planning. For temporary stabilization a sling may be given to increase comfort; however, this may be omitted in the acute phase if the patient is obtunded or the extremity is needed for access during initial stabilization of the patient.

Radiographic Evaluation

Clavicular fractures are often first discovered on chest radiograph or CT scan of the chest. Once discovered, however, there should be consideration for obtaining more specific imaging. Two views centered on the clavicle (AP and Zanca) should be obtained, which places the X-ray beam at a superior and then inferior angle to obtain different view profiles. When possible, AP clavicle radiographs should be obtained in both the supine and then upright positions. Direct comparison of the fracture characteristics in these two positions may reveal significantly more displacement when gravity exerts an effect on the thorax and upper extremity and may alter the decision of operative or nonoperative treatment [21]. Our protocol includes an AP view of bilateral clavicles so that clavicular distance from AC joint to midline can be compared (or at least estimated) as well as for symmetry and coracoclavicular distances (Fig. 10.4). AP, axillary, and scapular Y shoulder radiographs should also be obtained for complete trauma imaging of the shoulder. In general, dedicated CT imaging of clavicle fractures is not use-

ful unless there is concern for vascular injury, in which case CT-angiogram may be warranted (Fig. 10.5).

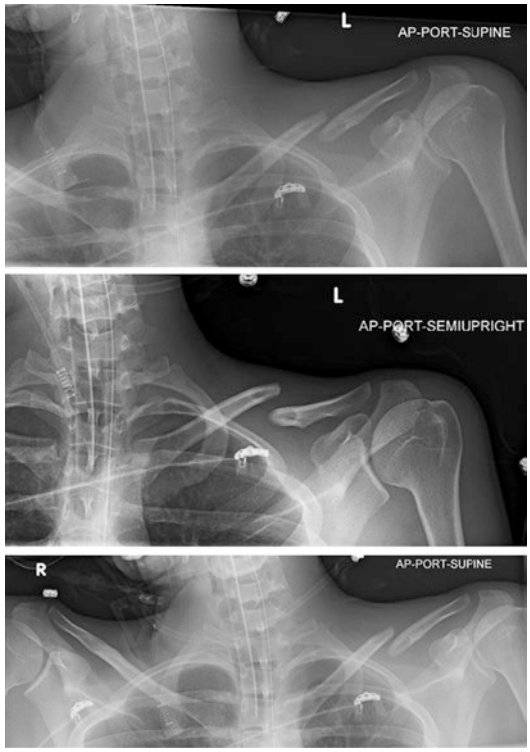

Fig. 10.4 Supine and upright radiographs demonstrating displacement

Fracture Classification

While several different clavicle fracture classifications have been proposed, the Allman classification is the most frequently used. This divides the fractures into thirds. Group 1 corresponds to injuries in the middle third and is the most common injury pattern, encompassing 81% of injuries. Group 2 represents injuries in the lateral third and makes up 17% of injuries. Group 3 injuries occur in the medial third of the clavicle and are the rarest, making up only 2% of all fractures [7]. Recent evidence, however, challenges this and argues that medial clavicle fractures may have been previously underestimated. In the middle age population (peak at 50 years old) medial clavicle fractures may comprise as much as 11.6% of all clavicle fractures [22] (Fig. 10.6).

Clavicle fractures in the lateral third (Group 2) are often subclassified based on their proximity to the coracoclavicular (CC) ligaments as this has a significant effect on fracture stability [24] (Fig. 10.6b). If these ligaments are intact and acting as stabilizing forces, then fracture displacement and subsequent nonunion rates are significantly lower, allowing for increased success with nonoperative treatment. If on the other hand the ligaments are not acting to stabilize the fracture, then fixation is often considered to prevent future malunion or nonunion.

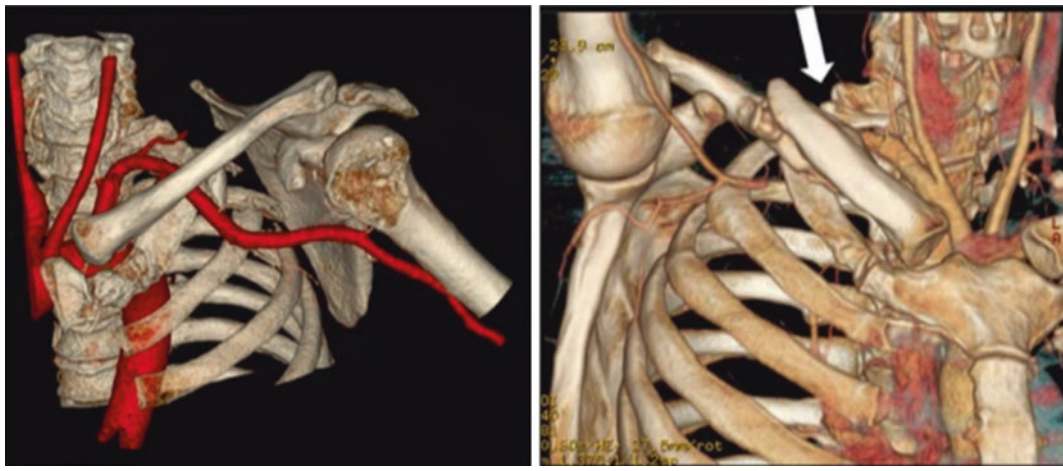

Fig. 10.5 CTA 3D reconstruction showing location of great vessels. (Graves et al. [12]. *With permissions*)

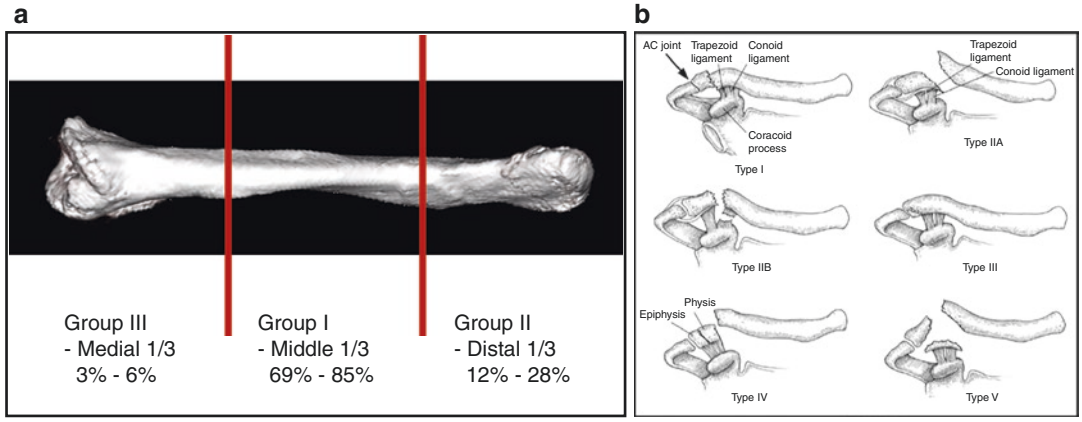

Fig. 10.6 (**a**) Illustration of Allman classification groups [23], (**b**) Neer lateral third classification [24, 25] (*with permissions*)

Treatment

Nonoperative Treatment

For fractures that are minimally displaced and do not meet indications for surgical fixation (discussed below), nonoperative treatment is appropriate. The patient should be kept non–weight bearing for 4–6 weeks, followed by a gradual return to full activities. A sling is given for patient comfort and they are encouraged to come out of it multiple times per day to do wrist, elbow, and gentle shoulder range of motion exercises. The patient should be followed weekly with repeat radiographs until fracture consolidation as 32% of patients will develop progressive displacement in the weeks after their injury [21, 26, 27]. Physical therapy can be prescribed in the event of weakness or stiffness. Figure of eight braces have been used in the past but have failed to show any clinical benefit studies [28].

Operative Indications

The classic absolute indications for surgical fixation of clavicle fractures includes (1) open fractures, (2) displacement with threatened skin (3) associated neurovascular injury, or (4) associated scapular neck fracture or floating shoulders [3, 29]. Some recent studies have shown that chest wall injuries with associated clavicle fractures portend worse respiratory outcomes, and propose that restoring the bony strut of the clavicle may help to restore respiratory function in patients with chest wall trauma [30, 31]. Conversely, there is also evidence that the presence of rib fractures indicates that midshaft clavicle fractures are more likely to be significantly displaced and comminuted, thereby requiring fixation [32]. More studies are needed to determine the merit of clavicle fixation with regards to flail chest injuries and respiratory compromise.

For midshaft clavicle fractures (Group 1) demonstrating 100% displacement and/or significant shortening there is prospective randomized evidence that surgical fixation results in a lower nonunion rate and higher functional outcome of the shoulder with regard to strength and endurance [33, 34]. In these midshaft displaced fracture types, the nonunion rate without fixation is reported in retrospective studies as 5–25% [35]. Specific risk factors for nonunion include female gender, age, fracture displacement, fracture stability, and comminution [36, 37]. Surgical fixation can also lower the rate of both nonunion and symptomatic malunion [38]. Late reconstruction of resultant clavicular malunions or nonunions is possible but is more costly and technically demanding [39, 40]. A recent large metanalysis reports that 17% of patient treated surgically for midshaft clavicle fractures require a second surgery, most often for removal of symptomatic

hardware, while 13% of patients initially treated nonoperatively require subsequent surgery for nonunion or malunion [41].

Lateral third fractures (group 2) that are widely displaced due to concomitant rupture of the CC ligaments are at increased risk for nonunion and should be considered for fixation [38]. Medial third fractures (group 3) are rare injuries, and as such there are no large studies guiding treatment. Surgical fixation should be considered for fractures with greater than 50% translatory displacement or if there is concern for posterior encroachment of the clavicle on mediastinal structures.

Surgical Technique

There is a wide variety of fixation tools and techniques which will be only briefly described here. The most common mode of fixation for clavicle fractures is with a plate and screw construct utilizing standard fracture fixation principals. The incision is placed directly over the anterosuperior fracture. The supraclavicular nerves can be identified and spared or sacrificed. The platysma is then divided until the clavicle is identified. The fracture ends are then further exposed and defined carefully. In simple patterns a bone clamp can be employed to achieve reduction and compression at the fracture site while the plate and screws are deployed. Alternatively, if there is significant comminution the approximate length, rotation, and alignment of the proximal and distal fragments can be held manually, and a bridging construct used to avoid the need for fixation within the comminution. The plate can be applied to either the superior or anterior surface of the clavicle [42]. Care is taken to avoid the surrounding neurovascular structures when dissecting or drilling. After satisfactory fixation is achieved the platysma layer is tightly closed with resorbable suture, and skin closed with a subcuticular suture for cosmesis.

Intramedullary fixation devices (screws, pins, or titanium nails) are also used on occasion. These intramedullary devices allow for smaller incisions, but are biomechanically inferior, can migrate postoperatively, and can be technically challenging with higher complication rates reported (32% vs 10% for plating) [43, 44]. If there are associated unstable rib or scapula fractures, then all of these can be fixed concurrently while in the lateral decubitus position.

Scapula Fractures

Background

Epidemiology

Scapula fractures are less common then clavicle fractures. Their incidence is reported in the literature as 0.01% of the general population, making up roughly 1% of all fractures [45]. Because the body of the scapula is highly mobile and protected by the surrounding musculature, it commonly takes a high energy direct blow to cause fractures, which is why injuries to this region are associated with other chest wall trauma in 85–95% of cases [46–49]. A review of 9453 scapula fractures in the National Trauma Database found that patients presenting with these injuries have associated rib fractures in 52.9% of cases, lung injury in 47.1%, and head injury in 39.1% [48, 50].

Development and Osteology

The thin scapular body initially forms through intramembranous ossification in at 6–8 weeks of gestation. After birth the cartilaginous perimeter and processes of the scapula continue outward growth through endochondral ossification until final ossification at ages 14–20 [51]. When the ossification center of the lateral acromion fails to fully ossify, a persistent fibrous gap can develop and is termed an os acromiale. Os acromiale is present in 8% of the population and can be unilateral or bilateral. It is usually asymptomatic, but can become mobile and painful after direct trauma. On imaging, it can appear to be acute fractures, but characteristically has rounded or sclerotic margins.

The three-dimensional bony morphology of the scapula is complex. It is therefore useful to divide the scapula into distinct regions including the body, the glenoid, the acromion, and the coracoid process (Fig. 10.7).

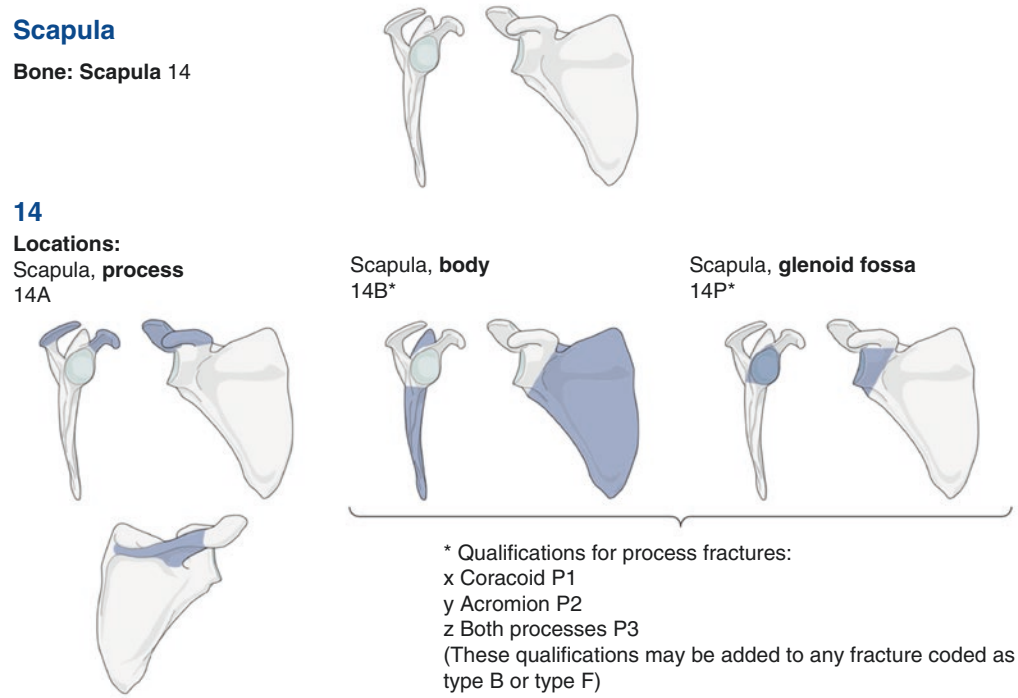

Fig. 10.7 Illustration of scapula bony morphology from the AO/OTA Classification Compendium [52], *with permissions*

Body

The scapular body is a large flat surface providing for numerous muscular attachments. The anterior and posterior surfaces are largely covered by the rotator cuff musculature (supraspinatus, infraspinatus, subscapularis). The perimeter vertebral border of the scapular body is thicker, measuring roughly 6–10 mm, while the inner surface of the scapular body is often as thin as 1–2 mm. The perimeter serves as the attachment site for the rhomboids, serratus anterior, trapezius, and other muscles that aid in protraction, retraction, translation, and other gliding movements.

Glenoid

The glenoid contains articular (hyaline) cartilage for articulation with the humeral head. The glenohumeral joint has very little inherent bony constraint and has been likened to a golf ball on a tee. Instead, it relies on the surrounding ligaments and labrum, as well as the muscles to create dynamic stabilizing forces throughout a wide range of movements and positions. Potential long-term sequalae of glenoid fractures include shoulder instability/recurrent dislocations or glenohumeral arthritis.

Acromion

The acromion process articulates with the lateral end of the clavicle. It is the primary origin of the large deltoid muscle and therefore is subject to large bending forces during active abduction of the arm. The acromion can be likened to a diving board as it withstands significant moment arm related forces to its base. Fractures of this bony process are therefore difficult to heal and can lead to significant shoulder weakness and dysfunction, particularly with overhead activities. It can be fractured by a direct blow to the superior shoulder, severe traction from the deltoid origin, or from extension from a related scapular body fracture line.

Coracoid

The coracoid process protrudes anteriorly. It is the site of origin of the short head of the biceps as well as the coracobrachialis and pectoralis minor muscles, which exert a downward force. At the superior base of the coracoid lie the attachments of the coracoclavicular and coracoacromial ligaments which help to anchor the shoulder girdle from superior displacement. The coracoid can be fractured by traction from the originating muscles or by extension of scapular body fractures to the base of the coracoid.

Diagnosis

Clinical Evaluation

Similarly, to clavicle fractures, the clinical evaluation of scapula fractures begins with the ATLS protocol, followed by a detailed history and physical exam. Scapula fractures are often overlooked on chest radiograph and the diagnosis may be missed entirely if a detailed second physical exam is not completed [53].

Radiographic Evaluation

AP, scapular Y, and axillary radiographs are the standard initial radiographs for scapula fractures. Unlike imaging for the clavicle, however, a dedicated CT scan of the shoulder can be immensely helpful for assessment of the scapula, especially in conjunction with a three-dimensional reconstruction of the scapula with humeral head subtraction. The inter and intra observer reliability of plain radiographs is markedly lower than with 3D reconstructions of a CT scan [54]. Additionally, CT scans allow for accurate measurement of any articular step-off in the glenoid, give a more detailed assessment of the degree of comminution present, and assist with planning a three-dimensional surgical fixation strategy. It is recommended to obtain this advanced imaging in any scapula fracture that shows signs of displacement for the measurement to determine surgical indications.

Fracture Assessment and Classification

Body

Prognosis and treatment are guided by certain validated radiographic measurements of the injury on X-ray and/or CT scan. These measurements include the glenopolar angle, lateral border offset sometimes called medialization, and sagittal plane angulation [54] (Fig. 10.8). The glenopolar angle is the angle created by a line drawn from the inferior pole of the scapular body, to the superior edge of the glenoid, and then along the face of the glenoid. Lateral border offset is a measurement of displacement between fracture fragments in the medial-lateral direction at the lateral border of the scapular body. It essentially measures the amount of medial displacement of the glenoid and glenohumeral articulation, which in turn affects the excursion and pull of the rotator cuff musculature. Sagittal plane angulation is assessed on the scapular Y radiograph. It is measured by drawing the angle between the superior and inferior scapular body fragments.

Glenoid

The most commonly used classification system for fractures of the glenoid fossa is the modified Ideberg classification [55] (Fig. 10.9). This system groups injury patterns primarily based on the direction that fracture lines extend away from the glenoid.

Acromion

The Ogawa acromion fracture classification (not to be confused with the Ogawa coracoid classification) as well as the Kuhn classification system have been described to classify acromial injuries based on their fracture location and degree of displacement [56, 57]. These are not commonly used in day-to-day clinical practice.

Coracoid

The Ogawa coracoid classification of coracoid fractures distinguishes between two types [58]. Type I fractures occur close to the base of the

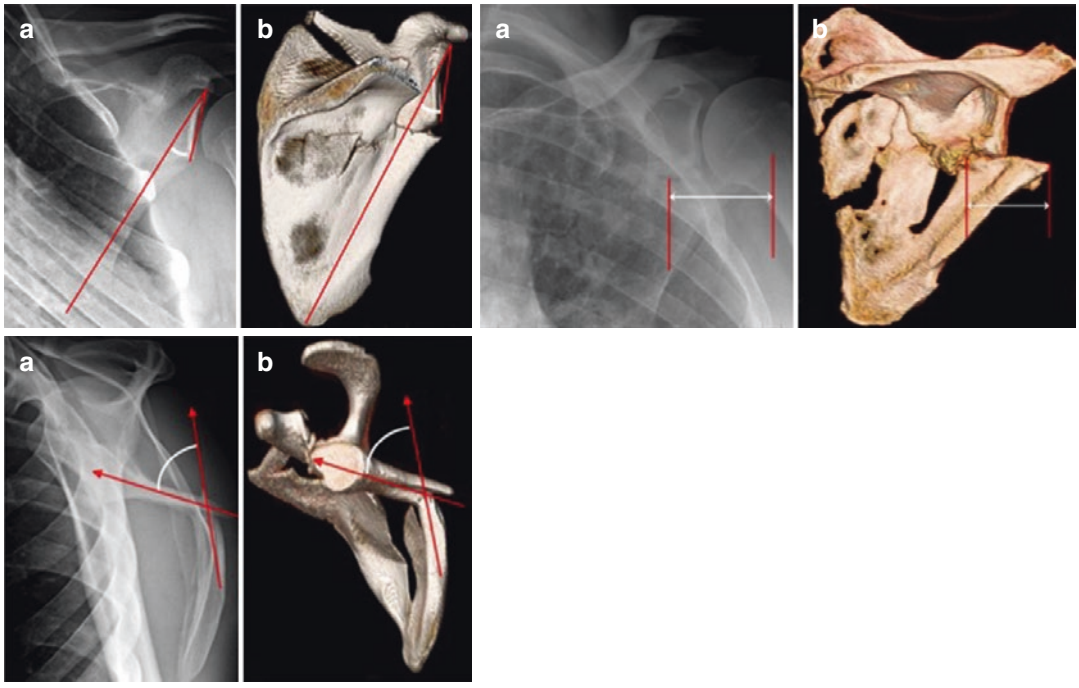

Fig. 10.8 (**a**) Radiographs and (**b**) 3D CT demonstrating glenopolar angle, lateral border offset, and sagittal plane angulation

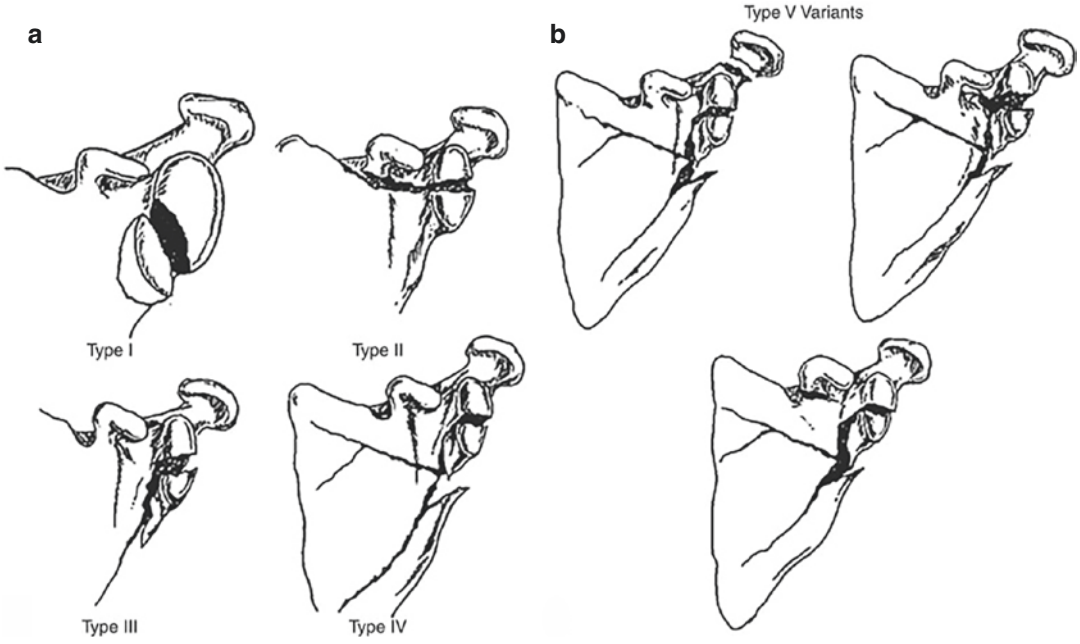

Fig. 10.9 Illustration of modified Ideberg classification (**a**) simple patterns and (**b**) associated patterns from Mayo et al. [55]. (*With permissions*)

coracoid and posterior to the CC ligaments, while type II fractures are anterior to the CC ligaments. The Eyres classification can also be used; it identifies five different injury patterns for a more detailed description and categorization of injuries [59].

Treatment

Nonoperative Treatment

Scapula fractures with minimal displacement can be treated nonoperatively [27, 60, 61]. The richly vascularized surrounding muscle bed usually leads to rapid fracture consolidation and subsequent osseous healing in 6–8 weeks. There is a rich anastomosis between circumflex scapular, suprascapular, and dorsal scapular arteries. A sling should be given for comfort and early elbow and shoulder range of motion encouraged. Weekly radiographs until fracture consolidation are advised to monitor for potential ongoing displacement.

Surgical Indications and Technique

Body

Untreated displaced scapular body fractures can lead to a number of sequelae including decreased rotator cuff strength from relative glenoid medialization, bony impingement of the glenohumeral joint space, scapulothoracic impingement, chronic pain, and other shoulder dysfunction [62–68]. While malunion of untreated scapular body fractures is common, nonunions are extremely rare due to the rich surrounding blood supply. Indications for surgical fixation include (1) angular deformity greater than 45° on scapular Y view or 3D reconstruction, (2) lateral border offset greater than 2 cm, (3) glenopolar angle less than 22° as measured on AP Grashey radiograph or 3D reconstruction, (4) displaced double disruptions of the SSSC [69]. Use of these surgical indications have demonstrated good functional outcomes with low complication rates [70, 71]. If indicated, surgical fixation should be completed within 2 weeks, before appreciable fracture callus is formed, though late surgery is well documented. Ideally, it may be opportune for the thorax and scapula to be operated in the same setting.

The preferred fixation technique for scapular body fractures is as follows. The patient is positioned in the lateral decubitus position with the arm over a bolster. This allows for adequate posterior exposure of the scapula and also affords the opportunity for concurrent ipsilateral rib or clavicle fracture fixation. A large curved skin and subcutaneous tissue flap is raised from medial to lateral and secured with a temporary suture for retraction throughout the case. When maximal visualization is needed a full Judet exposure is completed by taking the deltoid muscle off of its posterior scapula origin and then elevating the entire infraspinatus muscle off of the scapular body. Alternatively, if less visualization is needed a modified Judet approach can be used, in which the deltoid origin is spared and fixation completed through the interval between the infraspinatus and teres minor interval [72, 73]. Of note, the rhomboid insertion at the medial scapula border can be split or taken down to enable fixation of rib fractures deep to the scapula.

Fracture mapping studies reveal a common "T" pattern of injury as demonstrated in Fig. 10.10 [74]. As such, it is most common to utilize two 2.7 mm mini fragment locking plates for adequate stabilization. One plate is placed along the lateral border of the scapular body and extends up to the posterior glenoid. Another 2.7 reconstruction plate is contoured to fit superior and medial at the base of the spine of the scapula (Figs. 10.11 and 10.12). This configuration takes advantage of the thicker bone at the perimeter of the scapula and provides adequate strength for fracture stabilization and healing. A Bankardt shoulder awl, a large Lamina spreader, Schantz pins, and bone clamps are useful reduction aids.

Glenoid

Displaced intraarticular glenoid fractures warrant fixation to prevent the development of glenohu-

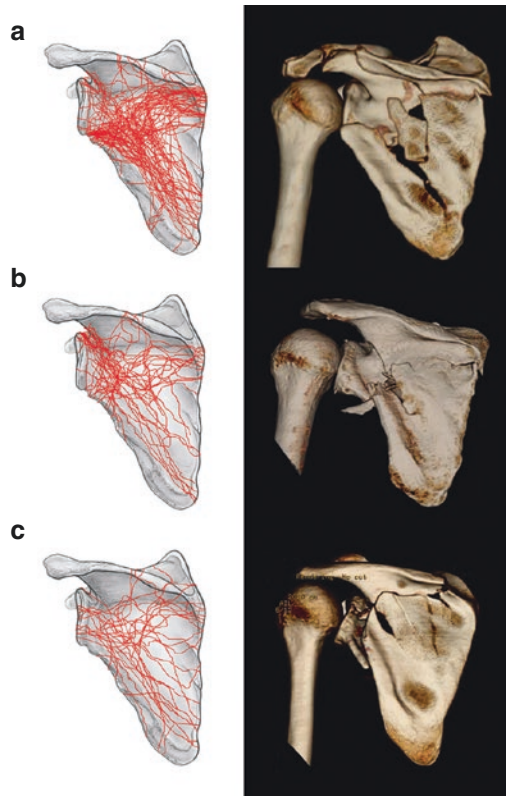

Fig. 10.10 Fracture mapping illustration demonstrating common patterns (**a**) Inferior Glenoid Neck, (**b**) Spinoglenoid Notch, and (**c**) Glenoid Articular Surface. (Armitage et al. 2009 JBJS Am. [74]. *With permissions*)

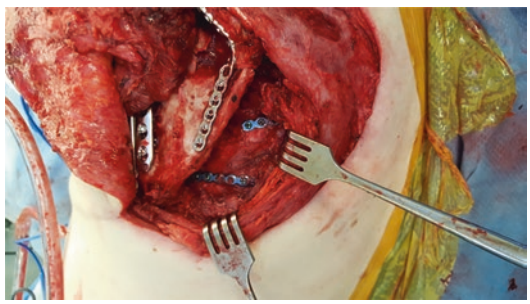

Fig. 10.11 Operative photo showing fixation of scapula and ribs in lateral position

meral arthritis as well as to prevent instability with recurrent humeral subluxation or dislocation. Generally accepted surgical criteria include 2–4 mm of articular step off or fragments greater than 25% of the articular surface. Additionally, any apparent glenohumeral subluxation on radiograph would also be an indication for fracture fixation. Smaller glenoid rim avulsion fractures, often referred to as "bony Bankart" lesions may be amenable to arthroscopic stabilization techniques. Larger fractures, however, will require an open approach, usually through an anterior deltopectoral approach utilizing either a split in the subscapularis tendon, or a complete tenotomy to visualize the base of the glenoid. Alternatively, if the majority of the fracture fragment lies posteroinferior then a posterior modified Judet approach or a straight posterior approach will afford better access. Specific fixation usually requires a combination of small or mini fragment lag screws and buttress plates [75].

Acromion

Acromion fractures are subject to the large deforming forces of the deltoid muscle and gravity of the arm, and therefore often require surgical fixation to prevent progressive displacement, narrowing of the subacromial space, and persistent nonunion. The proposed specific indications for surgical treatment include 5 mm of fracture displacement, narrowing of the subacromial space, open fractures, multiple disruptions of the SSSC, or symptomatic nonunions as diagnosed by CT scan and clinical exam [4, 76]. These injuries are often best stabilized with a sequence of direct anatomic reduction and compression using bone clamps, lag screw preliminary fixation, superior tension band plating, and adjunctive perpendicular plates as need to obtain multiple points of fixation [76]. Locking plates should be utilized when possible to help increase the pullout strength of the construct.

Coracoid

Due to the various ligamentous and muscular attachments onto the coracoid process, displaced fractures can be a source of chronic pain and discomfort. Recommended surgical indications include more than 1 cm of displacement, multiple disruptions of the SSSC, and symptomatic nonunion [4, 77]. These fractures are usually best approached anteriorly through a deltopectoral approach. A 4 mm Schantz pin and shoulder hook can be useful to help gain control and reduce the fracture. Fixation can be in the form of 3.5 mm

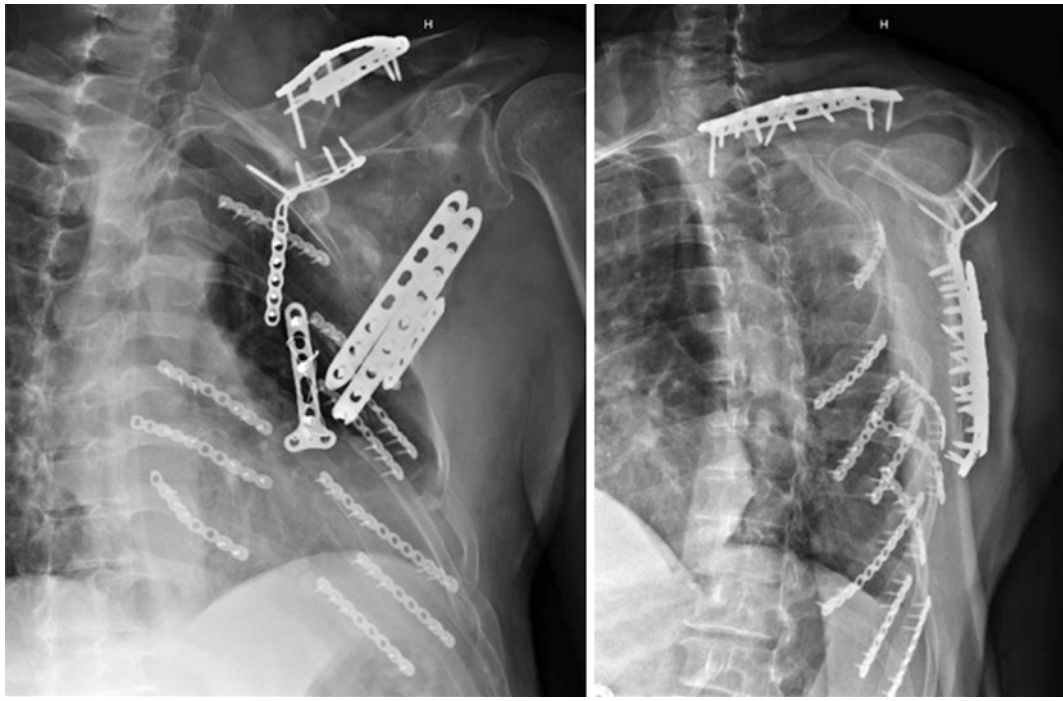

Fig. 10.12 Postoperative radiographs in a patient whose ribs were fixed 3 months earlier at another institution, but presented with a scapula malunion and severe shoulder pain and dysfunction. Scapular osteotomy and correction requires more robust fixation, in this case with dual 3.5 mm plates along the lateral column

lag screws and/or 1/3 tubular plates; however, specific fixation will vary based on fracture morphology. On occasion, the coracoid process will need to be reduced concurrently with associated scapular body or glenoid fractures.

Conclusions

- Clavicle and scapula fractures occur commonly with chest wall trauma.
- Effective diagnosis and treatment require proper imaging and careful consideration of surgical indications.
- Surgical intervention, when indicated, has a low complication rate and results in superior functional outcomes.
- Concurrent treatment of rib, clavicle, and scapula fractures in the same surgical setting is feasible and should be considered whenever possible.
- Interdisciplinary communication is necessary to ensure optimal treatment of complex chest wall trauma with multiple injuries.

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11 Nonsurgical Aids in Fracture Healing

Adam J. Kaye

Abbreviations

CW	Continuous wave
LIPUS	Low-intensity pulsed ultrasound
LLLT	Low-level laser therapy
PW	Pulsed wave
SSRF	Surgical stabilization of rib fractures

Introduction

In the previous chapters the authors have discussed the operative and nonoperative management of rib fractures. The indications for operative management are still fluid as more indications are becoming apparent over time. Once the decision to operate or not has been made, there are still therapeutic options that the physician can use to help with fracture healing. The majority of the therapies discussed have been shown to help with bony fracture healing, but they have not been studied on ribs. The research necessary to determine efficacy of these therapies for rib fractures has not been done or is in progress, and the number of participants necessary for some studies may not be attainable. Furthermore, it is unclear if rib fractures heal like other weight bearing bones and so it may not be prudent to equate the two. However, this is the only data we have. The following therapies have been used with anecdotal successes and no adverse events. But again, no randomized controlled trials have been done to prove their effectiveness. In this chapter, we will discuss the therapies in order of their scientific evidence, from the strongest to the weakest. We will also discuss options to better study these therapies as they pertain to rib fractures.

Vitamin D

Although named vitamin D, the chemical cholecalciferol is actually a hormone and not a vitamin [1]. The skin when exposed to sunlight uses the ultraviolet radiation to photolyze provitamin D3 to previtamin D3. Previtamin D3 is then converted to vitamin D3 or a number of other isomers or its photolyzed chemicals not involved in the vitamin D pathway. It is this vitamin D3 that enters the circulation and is then metabolized to 25-hydroxyvitamin D3 in the liver. It is further hydroxylated to 1,25-dihydroxyvitamin D3 (1,25-[OH]2-D3), its biologically active form, in the kidney [2]. This new compound exhibits its effects on multiple tissues. For this discussion, we will discuss bone and more specifically bone fracture repair.

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Within the general population there is a high prevalence of vitamin D deficiency. It is estimated that one billion people have vitamin D deficiency or insufficiency worldwide [3]. Clinically there is an association of delayed union with vitamin D deficiency; however, the incidence of radiographically evident delayed union is not vitamin D dependent [4].

Two case-controlled studies showed a predominance of vitamin D deficiency in patients with delayed healing or nonunion (60%) compared to those with normal fracture healing [5, 6]. These two studies evaluated the vitamin D level after the diagnosis of the fracture pathology was already known. A third study assessed the vitamin D status prior to the injury as well as after supplementation. If the patient still remained vitamin D deficient, despite additional vitamin D administration, the incidence of delayed union was 9.7% [4]. These studies all show the need for normal vitamin D levels to help ensure proper fracture consolidation.

Three further studies show a positive influence of vitamin D supplementation on fracture healing [7–9]. These studies were in the elderly postmenopausal female population with accompanied calcium supplementation as well. In the general population, vitamin D supplementation did not show a significant difference in the rate of nonunion or delayed union and normal fracture repair [4].

In our practices, the patient population is diverse and there will be subsets of patients for whom vitamin D supplementation will benefit their rib fracture healing. For this reason, it seems prudent to assess all patients' vitamin D status as soon as a diagnosis of rib fracture has been made and supplement them with vitamin D if they are found to be deficient.

The goal for therapy is a vitamin D concentration of >75 nmol/l as the optimal concentration for fracture repair [1, 4]. The usual dosing depends on BMI and initial levels. A good simple supplementation strategy is 50,000 IU vitamin D2 weekly for 8 weeks, with reassessment of vitamin D levels to determine need for further supplementation [1].

A multi-institutional randomized controlled trial of vitamin D supplementation in patients with vitamin D deficiency and concomitant rib fractures will need to be performed to better understand this simple and hopefully effective therapy for our patients.

Low-Intensity Pulsed Ultrasound

Low-intensity pulsed ultrasound (LIPUS) has been developed as an alternative and adjunct to common methods of fracture fixation and repair. LIPUS uses a probe with a frequency of 1.5 MHz, a signal burst width of 200 ms, a signal repetition frequency of 1 kHz, and an intensity of 30 mW/cm^2 for 20 min a day [10, 11]. The mechanism of action of this therapy is postulated to be related to cox2 upregulation from mechanoreceptors at the fracture site stimulated by ultrasound waves [12].

There have been multiple randomized clinical trials evaluating LIPUS's use on primary fractures of the tibia and distal radius as an adjunct to closed reduction and cast immobilization. These studies showed that LIPUS can accelerate the fracture healing rates from 24 to 42% for fresh fractures [13, 14]. For delayed unions and nonunions LIPUS's benefits have been shown to benefit certain fracture subsets [11, 15, 16]. Specifically, one study of long bone nonunions showed instability, fracture gap size of >8 mm, and atrophic or oligotrophic nonunion as predictors of failure of LIPUS alone [11]. The authors recommended LIPUS as an adjunct to surgical interventions (bone graft, chipping, and decortication with secure fixation) in these cases.

All of these studies, like most of the data we have for adjuncts to fracture repair, have been done in long bone models. There is one case report of LIPUS for rib fracture management [17]. Two patients were presented, one with LIPUS as an adjunct to surgical stabilization of rib fractures (SSRF), and the other with LIPUS as the sole treatment for the fractures. The use of LIPUS as an adjunct was used in a fracture that was addressed 8 weeks after the injury. The LIPUS was started 2 weeks post-SSRF; the

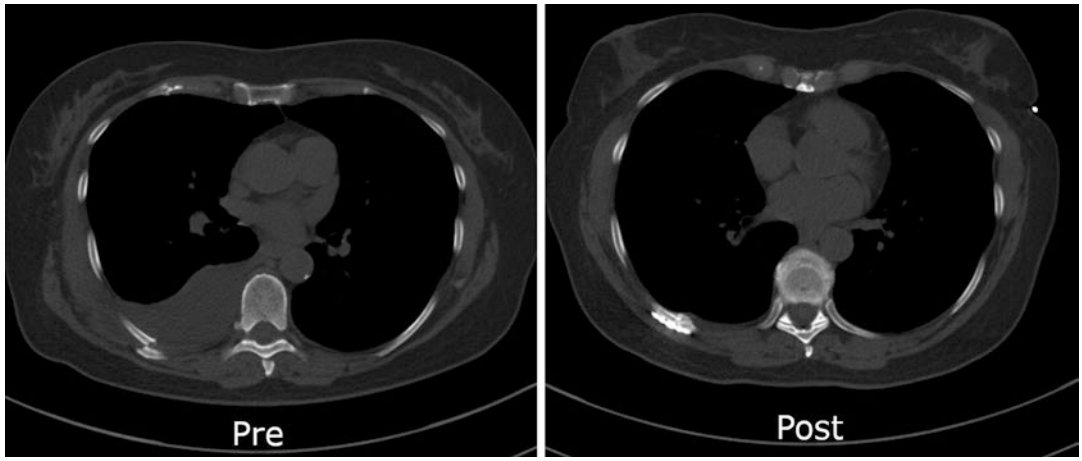

Fig. 11.1 CT scan of patient prior to surgery and then 4 months post-op after undergoing SSRF and LIPUS therapy

repeat CT scan to document the fractures was performed after 4 months of stimulation (Fig. 11.1). The other patient had multiple rib fracture nonunions 5 months after her injury still causing considerable pain. SSRF was offered but was declined and LIPUS was offered as an alternative option. A repeat CT was again performed after 4 months of LIPUS therapy (Fig. 11.2). Both patients reported resolution of previous rib pain. The CT scans showed in both cases good bone deposition at the fracture sites and complete cortical formation. Both patients used a combination of bone stimulation, oral narcotic pain medication, and physical therapy during their healing.

In clinical practice, currently, LIPUS has been used in a limited fashion. Patients who are deemed too high a risk for operative management or refuse operative management of their acute fractures are offered LIPUS. The therapy has also become an adjunct therapy in the care of nonunions and malunions, and is started 2 weeks after SSRF and chest wall reconstruction is performed.

This case report, although only two cases, is intriguing and deserves more study. Unfortunately, evaluation of rib fractures over time and their degree of healing are not as simple as a two-view X-ray for long bones. Therefore, a longitudinal study in the clinical environment is not feasible. Future large animal studies may be able to elucidate whether LIPUS is beneficial in the healing of rib fractures, both acute and chronic.

Low-Level Laser Therapy

Low-level laser therapy (LLLT) was initiated in the late 1960s when increased collagen synthesis was found in skin wounds treated with laser therapy [18]. The biostimulatory effect and the mechanism of action of LLLT are still under study, but some believe the LLLT forms free radicals of oxygen that influence the formation of adenosine triphosphate [19]. Cell functions and homeostasis reportedly were promoted by the LLLT in the healing of nonhealed wounds. Numerous studies of the effects of LLLT on tissue healing have shown good results, but those on fracture repair have resulted in a mixed message as to its effectiveness clinically [18–20]. Most studies on LLLT use a number of different laser systems, with different chemical reactants, different mechanisms of laser production, and therefore different wavelengths and different energy levels [18–20]. LIPUS, on the other hand, has a specific ultrasound setting used for all studies and therefore a more uniform consensus has been able to be reached.

To make this therapeutic option more confusing, LLLT can be administered as a continuous wave (CW) as well as a pulsed wave (PW)

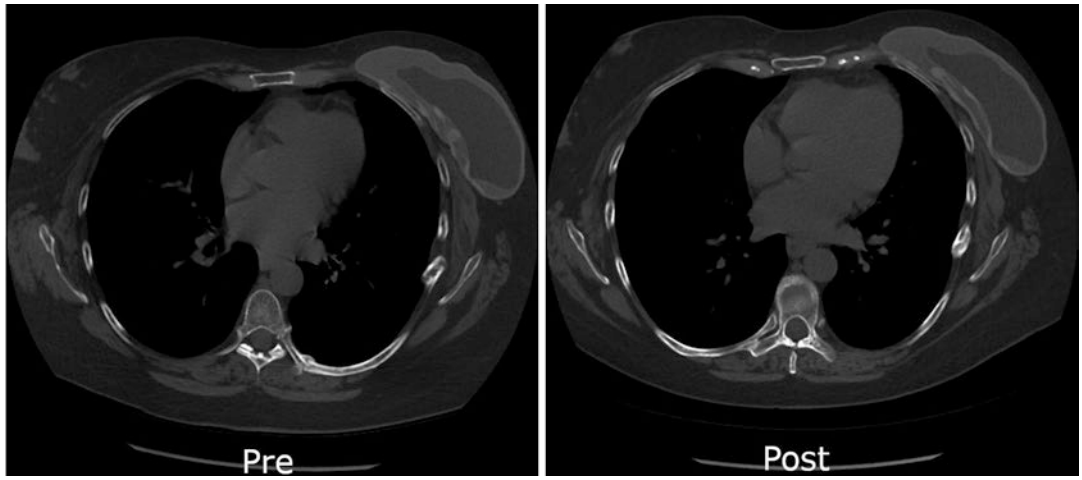

Fig. 11.2 CT scan before and 4 months after undergoing LIPUS therapy without any surgical intervention

[21]. A study comparing CW LLLT and PW LLLT has shown some convincing evidence that PW LLLT with its varied parameters can be more effective in obtaining the proper therapeutic settings [22]. In a study in rabbits, CW had no biomechanical effect on osteochondral defect healing, while PW significantly increased the stiffness of repaired osteochondral tissue. A literature search for LLLT (CW or PW) as a treatment option of bone fractures has shown no reported studies of human or even large animal models at this time. Similar to LIPUS, a large animal study of the therapeutic options of LLLT on rib fracture healing is currently being considered to elucidate if this too may be another noninvasive therapeutic option for our patients.

Kinesiotape

Kinesiology tape was advanced in Japan by Dr. Kenso Kase, a chiropractor. It was developed to help heal traumatized tissue and muscles. Although the kinesiotape application appears similar to other sports taping methods it has the added benefit of not restricting range of motion as well as supporting the overlying facia [23]. Kinesio taping saw worldwide application during the 1988 Seoul Olympics, and since then it has become a popular form of muscle taping used by health professionals the world over.

There are multiple theories about the benefits of kinesiology tape. Kinesiology tape can be used during any phase of an injury (acute/chronic) to help initially reduce swelling and inflammation by improving circulation. It reportedly changes the proprioception input of the sensory nervous system in the muscles, joints, and skin. It improves the interaction between the skin and the underlying structures improving muscular activation and performance. The taping method inhibits nociceptors in muscles, skin, and joint structures, which decreases painful input and is thought to normalize muscle tone. This results in decreased pain and muscular spasm and overcontraction. Finally, kinesiology tape is also thought to realign joint positions, and it may be useful in remodeling collagen tissues as well [24].

Sareen et al. in 2015 used kinesiotape on patients with nondisplaced rib fractures [25]. He showed a decrease in pain scores while deep breathing and coughing that was statistically significant. Another paper from Cyczewski et al. also showed a significant decrease in pain score, after kinesiotape application, with transitions from sitting to standing, deep breathing, and coughing [26]. This paper also showed a trend to significance in improvement of respiratory parameters (FVC, FEV1, and PEF).

Although no studies have shown the benefits of kinesiology tape specifically in the context of rib fracture healing, use of kinesiology tape with and without SSRF may be beneficial. The taping should help with pain and stabilize the chest. The added benefits of fascial support, lymphatic drainage, and collagen remodeling may assist in fracture repair.

Kinesiology tape is applied to the affected side. There are multiple different approaches to taping. One approach frames the area of pain with the tapes. After determining the location of maximal pain, the patient is asked to abduct his/her arm as much as possible to reveal the chest wall. The tape is then placed at a 50% stretch along the length of the rib from posterior to anterior above the area of pain. A second strip is placed along the ribs below the area of pain again with a 50% stretch from posterior to anterior as well. Finally, a third piece of tape is placed with a 50% stretch vertically along the length of the chest wall over the area of pain (Fig. 11.3).

Fig. 11.3 One approach of kinesiology tape application for rib fractures

Conclusion

Although none of these therapies have been extensively studied in the rib fractures, their possible benefits definitely outweigh their risks. Vitamin D deficiency affects almost 1/8 of the world's population and is more prevalent in the elderly [27]. Assessing vitamin D levels or empirically treating patients with rib fractures seems a simple therapy with no major downside. LIPUS and LLLT also appear to be benign therapies; however, there is a larger cost to society for the equipment and time needed to provide these therapies. These two modalities need to be further studied to assess their efficacy. Use of these therapies should be done either in a select patient population with goals of studying their therapeutic value. Finally, kinesiology tape is being employed mainly for assisting in postoperative pain management, but it may be beneficial in those not operated on as well; more research is needed to support this.

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12 Anesthesia and Analgesia Options in Chest Wall Injury

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Introduction

Chest wall injury is common and responsible for 10–15% of all trauma admissions [1]. Chest wall injuries put patients at higher risk for morbidity and mortality due to the primary injury or development of secondary complications due to the injury. The most common presentation of blunt chest trauma is rib fractures, which may be associated with complications including hemothorax, pneumothorax, and lung contusions. Pain from rib fractures may burden respiratory effort in patients and thus may lead to respiratory complications including hypoventilation, atelectasis, pneumonia, aspiration, and acute respiratory distress syndrome. Avoiding intubation and mechanical ventilation in these patients is prudent especially if patients are not surgical candidates for fracture repair. Therefore, optimal analgesia for chest wall injuries, especially rib fractures, is essential in decreasing morbidity and mortality.

Systemic analgesia entails a multimodal approach with a combination of opioids, acetaminophen, nonsteroidal anti-inflammatory drugs, and other opioid-sparing analgesics including ketamine and lidocaine infusions. Along with systemic analgesia, many regional anesthesia techniques have the ability to provide adequate analgesia and aids in the decrease of systemic opioid use. This is essential as there are various unwanted side effects associated with systemic analgesics, especially opioids, including sedation, respiratory depression, nausea/vomiting, and ileus, to name a few. Using regional anesthesia techniques may provide dense localized analgesia while minimizing systemic effects; however, they are not without their side effects. Regional techniques employed include thoracic epidural, paravertebral block, intercostal block, pectoralis nerve block, serratus anterior plane block, and erector spinae plane block. Depending on the type of block will determine what other side effects patients are at risk for, and thus, it is essential to weigh the risks and benefits of the various analgesic approaches. In this chapter, we review the various common analgesic techniques for trauma patients presenting with chest wall injury.

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Neuraxial Analgesia

Thoracic epidural analgesia (TEA) utilizing local anesthetic with or without opioid can confer great analgesic benefit to those with chest wall trauma. TEA involves placement of a catheter within the epidural space often times placed via landmark or ultrasound-guided techniques. Placement may be difficult depending on provider experience and patient anatomy. TEA anesthetizes the spinal nerves in the epidural space which decreases nociceptive pain signals from intercostal nerves. When compared to parenteral opioids for pain management, TEA reduced mortality in patients sustaining rib fractures [1–3], decreased incidence of pulmonary complications [4, 5], decreased length of ICU stay [6], and decreased incidence of somnolence and respiratory depression [7]. Other advantages of TEA include improvement in respiratory function [7], decreased subjective pain scores [8], reduced cost of inpatient care [9], and ability for bilateral and multilevel analgesia. Because of this, TEA has traditionally been considered the gold standard for bilateral multilevel rib fractures and remains the conditionally recommended method of analgesia over nonregional modalities of pain control according to the management guidelines of the Eastern Association for the Surgery of Trauma (EAST) and the Trauma Anesthesiology Society [10].

However, more recent data suggests that the advantages of TEA over other analgesic modalities may not be as pronounced as previously believed. Several meta-analyses and systematic reviews of RCTs have concluded that epidural placement was not associated with improvement in mortality or ICU and hospital length of stay when compared to other analgesic interventions [11–14]. Limitations of TEA include many contraindications to placement, including coagulopathy, significant spinal or traumatic brain injuries, systemic infection, significant hypotension or hypovolemia, as well as high rates of failure reportedly ranging from 13% to 47% [15] and catheter-related problems including early catheter dislodgment, leakage, or occlusion. Epidurals should not be placed in patients with increased risk of coagulopathy to reduce the risk of an epidural hematoma, as such complication may lead to somatosensory deficits and consequently permanent lower extremity paralysis. Furthermore, placement of TEA can be complicated by hypotension due to local anesthetic-induced sympathectomy which can prevent sufficient analgesia, risk of dural puncture, motor blockade, urinary retention, and epidural hematoma. Additionally, TEA placement is considered one of the more difficult regional anesthetic procedures which can be potentiated by challenges in positioning a patient with multiple painful injuries.

PECS Block

The PECS blocks are aimed to anesthetize the pectoral nerves, intercostal nerves, intercostobrachial nerves, and the long thoracic nerve. The PECS block was initially described for analgesia for breast surgery and its use extrapolated to anesthetize the chest wall for chest wall trauma. Such block is performed via ultrasound guidance. In the PECS 1 block, local anesthetic is placed between the pectoralis major and pectoralis minor muscles at the third rib to block the lateral and medial pectoral nerves. In the PECS 2 block, PECS 1 block is performed along with depositing local anesthetic between the pectoralis minor and serratus anterior to block the anterior cutaneous branches of intercostal nerves 3 to 6, the intercostobrachial nerves and the long thoracic nerve. The PECS blocks are technically more simple than thoracic epidural, paravertebral and intercostal nerve blocks as well as the advantage of easier patient positioning in the supine position, and less serious risk of coagulopathy issues. Furthermore, with PECS blocks, various complications are minimized compared to neuraxial analgesia including pneumothorax, hemodynamic instability, and epidural hematoma. Providers may be less conservative in regards to coagulopathy when placing PECS blocks versus neuraxial blocks. Disadvantages of PECS block includes being a newer block and few random-

ized controlled trials to demonstrate noninferior or superior analgesia compared to other established blocks for rib fractures and no studies of continuous infusion of PECS block. Furthermore, the use of catheters for continuous infusion of local anesthetic has not been as well established as that with epidural analgesia.

Paravertebral Block

Thoracic paravertebral blocks (TPVB) provide ipsilateral, segmental, somatic and sympathetic nerve blockade in contiguous thoracic dermatomes by preventing nociception from ventral and dorsal rami of spinal nerve roots in the paravertebral space. The paravertebral space communicates with the intercostal and epidural spaces and is amenable to catheter placement for continuous infusion of local anesthetics. The use of TPVB vs. TEA for rib fractures have been studied extensively. A meta-analysis comparing TPVB and TEA in patients with rib fractures showed similar pain scores and length of ICU or hospital stay [13]. When compared to patient-controlled intravenous analgesia, patients with TPVB reported lower pain scores and incidence of nausea and vomiting and superior respiratory parameters [16]. Risks during TPVB placement include pneumothorax, pleural puncture, and inadvertent bilateral blockade.

Intercostal Nerve Block

Intercostal nerve blocks (ICB) may be used for localized and temporary pain relief of chest wall injuries. ICB provide reliable unilateral dermatomal analgesia to the area of localization and have been shown to improve respiratory mechanics [17, 18]. They are technically simple to perform compared to TEA and TPVB placement and do not carry the risk of neurologic injury or sympathectomy. However, because of the segmental overlap of intercostal nerves, multiple injections are required above and below the affected rib for adequate analgesia. Unlike TPBV, these blocks tend not to be facilitate contiguous spread across dermatomes, and therefore, multiple injections may be required. This can increase patient discomfort due to multiple injections and increase risk of vascular uptake and systemic toxicity of local anesthetic and pneumothorax and hemothorax [19]. Additionally, catheter placement for ICB have not been well studied [20] and repeated injections every 6-8 hours may be needed for continued adequate pain control. The risks of complications between TPVB, neuraxial, and ICB need to be considered when choosing which nerve block to perform. With ICB, the pleura is more superficial and therefore, the risk of pneumothorax may theoretically be increased. However, compared to TPVB and neuraxial approaches, ICB may be associated with less sympathetic blockade, and thus less post-block hemodynamic instability.

Serratus Anterior Plane Block

Serratus anterior plane block (SAPB) is performed by depositing local anesthetic either superficial to the serratus anterior muscle (between the latissimus dorsi muscles and serratus anterior) or deep to the serratus anterior muscle (between the serratus anterior muscle and the external intercostal muscles and ribs). Local anesthetic deposition to either plane will achieve analgesia to the anterolateral chest with similar efficacy [21–23]. The SAPB blocks nociception from the lateral cutaneous branches of the thoracic intercostal nerves as they travel through the serratus anterior muscle providing analgesia to the anterior two-thirds of the thorax. Local anesthetic deposition above or below the serratus anterior muscle results in spread throughout the lateral chest wall with resulting analgesia of multiple dermatomal levels depending on the injectate volume. The dorsal ramus providing sensation to the posterior thorax and the anterior cutaneous nerve providing sensation to the anterior thorax are not covered by SAPB. SAPB offers technically simplicity with a favorable side effect profile with little to no risk for hemody-

namic changes, motor blockade, vascular injury or inadvertent intrathecal injection as well as ability to perform the block in coagulopathic patients. Several case reports have shown that SAPB decreased pain scores and opioid use in patients with multiple rib fractures [24, 25]. When deciding between SAPB versus TPBV or neuraxial approaches, a few considerations should be acknowledged. SAPB is potentially technically less challenging and because of that, the risk of complications such as pneumothorax should be decreased. Furthermore, there is less to no hemodynamic instability associated with this nerve block. Considerations for anticoagulation may be less conservative for this block as there is essentially no risk for neuraxial hematoma in these cases. However, because the use of continuous nerve blocks in the SAPB are less studied, this may not be a viable option.

Erector Spinae Plane Block

Erector spinae plane block (ESPB) provides analgesia to the anterior, lateral and posterior ipsilateral thorax by blocking the dorsal and ventral rami at their thoracic spinal nerve origins in the paravertebral space. The erector spinae muscle is formed by the spinalis, longissimus thoracis and iliocostalis muscles that run vertically along the spine. The ESPB is performed by advancing the needle through the trapezius, rhomboid major and erector spinae muscles to contact the transverse process where the local anesthetic is deposited and leads to cephalocaudad spread [26]. This potential space is amenable to catheter placement and can provide prolonged analgesia for chest wall injuries. In patients with rib fractures, ESPB was shown to decrease pain scores and improve spirometry in the first 24 h [27]. This approach is technically easier than TPVB and thus, the risk of pneumothorax and hemodynamic instability is lessened. This is what makes this block more attractive in the less experienced hands. More studies are needed to test the efficacy of ESPB when compared to either TPVB or neuraxial approaches in trauma patients.

Chest Wall Blocks Vs. Thoracic Epidural Analgesia

With adequate analgesia for chest wall injuries, pulmonary hygiene can be maintained leading to a mortality benefit. Current research in modalities to improve patient outcomes after chest wall injuries has explored the efficacy of various regional anesthetics for potential superiority. In a systematic review and meta-analysis of both observational studies and randomized controlled trials, epidural analgesia and chest wall blocks inclusive of ICB and TPVB were compared to intravenous analgesia and found better pain relief with regional anesthesia [13]. In studies comparing TPVB and TEA, no significant differences between the two modalities were found as both interventions provided good pain relief and improved respiratory function and similar length of ICU and hospital stay [13, 28, 29].

In a retrospective chart review study, ICB with liposomal bupivacaine was compared to TEA for treatment of traumatic rib fractures and found that patients receiving intercostal nerve blocks with liposomal bupivacaine were less likely to require intubation, had shorter hospital and ICU stays. Patients who received epidurals had more minor complications but there was no difference in mortality between the two groups [30].

There is a paucity of studies comparing the newer myofascial plane blocks such as PECS block, SAPB and ESPB between each other and with the established current modalities including TEA and TPVB. While the myofascial plane blocks would theoretically be inferior to TEA and TPVB, these plane blocks offer technical simplicity with a favorable side effect profile.

Intravenous Infusions

Ketamine is an intravenous anesthetic that provides analgesic, sedative and dissociative properties that works via antagonism of the N-methyl-D-aspartate receptor. In contrast to opioid analgesics ketamine does not depress the respiratory drive and stimulate the cardiovascular

system. The use of ketamine infusion as an adjunct to patient with rib fractures has become a topic of increasing interest due to its noninvasive method of analgesia, efficacy against other pain etiologies, relatively low financial burden, and favorable side effect profile. While studies have been contradictory, in multiple studies, ketamine infusions have been associated with reduced pain scores and opioid use [31–33].

Intravenous lidocaine has been well-studied in other analgesic situations and is a potent anti-inflammatory, antihyperalgesic, and gastrointestinal pro-peristaltic drug. Intravenous lidocaine in the perioperative period has been shown to be a useful adjunct with reduced pain scores, opioid consumption and side effects. While the use of lidocaine infusions for chest wall injuries is not well studied [34], there are ongoing randomized controlled trials aimed to quantify the analgesic efficacy of intravenous lidocaine infusions for patients with traumatic rib fractures [35].

Conclusions

Optimal analgesia remains an essential component for treatment of chest wall injuries including traumatic rib fractures as it maintains pulmonary hygiene and early mobility. Guidelines from the Eastern Association for the Surgery of Trauma and the Trauma Anesthesiology Society concerning the treatment of blunt thoracic trauma place an emphasis on patient preference and conditionally recommend epidural analgesia and a multimodal approach. As epidurals have more stringent contraindications, side effects and failure rates compared to other regional anesthetic procedures, the use of epidurals may not be feasible in some patient populations. In these instances, TPVB, ICB, or myofascial plane blocks including PECS, SAPB, and ESPB can be considered to optimize analgesia. Another area of interest and current research include intravenous infusions of ketamine and lidocaine to augment analgesia in populations where regional anesthetics may not feasible or to further decrease pain scores and opioid use. The choice of analgesia should be in conjunction with patient and family preferences, physician expertise, and consideration of side effect profile and risk of complications with the ultimate goal of minimizing pain to improve outcomes.

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Blunt Cardiac and Aortic Injuries

13

Amy V. Gore and Jamie J. Coleman

Introduction

Blunt chest trauma (BCT) is estimated to be present in 15% of trauma admissions and is the second leading cause of death following motor vehicle crash (MVC). It is associated with a broad spectrum of injury to the heart and aorta, with marked variation in character and severity. While some individuals will have no or mild symptoms at most, others have severe injuries resulting in immediate death or presentation to the emergency department in extremis. Despite this wide range of severity, the high mortality rate associated with these injuries warrants rapid evaluation and expeditious expert consultation [1].

BCT most commonly occurs as a direct impact to the anterior chest wall during a MVC with a sudden deceleration and compression to the thorax, although it is seen with a myriad of mechanisms, including pedestrians struck by motor vehicles, fall from height, crush injury, and sports injuries. In some cases, the mobile intrathoracic viscera are injured by the immobile bony structures composing the thoracic cage. Given their close anatomic relationship, sharp sternal fracture fragments can injure the right ventricle or ascending aorta [2, 3]. The least mobile part of the descending aorta is at the level of the ligamentum arteriosum, where it is fixed to the pulmonary artery. Torsion or traction forces against this site of fixation can result in aortic injury [2]. Blunt forces that occur at the time of end diastole can abruptly increase intraventricular pressures, injuring cardiac valves or the myocardium itself [4]. Electrical or structural injury to the heart can result in conduction delay or complete heart block [5].

While patients sustaining severe blunt injury to the heart and aorta rarely survive to hospital transport, it must always be a consideration in the multiply injured patient allowing for rapid recognition and treatment. This chapter describes common injury patterns, appropriate diagnostic algorithms, and optimal treatment strategies for these relatively uncommon, but highly mortal injuries.

Blunt Cardiac Injury

Blunt cardiac injury (BCI) is an elusive entity as it lacks an accepted gold standard test for diagnosis, thereby making this injury difficult to quantify and diagnose. BCI is actually a heterogeneous

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group of cardiac conditions resulting from blunt trauma, ranging from minor ECG/cardiac enzyme abnormalities, to complex arrhythmias, cardiac failure, septal or free wall rupture, coronary artery thrombosis and/or disssection [6, 7].

The most common mechanism of BCI is MVC, where it is implicated in up to 20% of deaths [6, 8]. While MVC is the most common mechanism, there are multiple other sources of blunt force which can result in BCI, including: direct, indirect, crush injury, bidirectional/compressive, decelerative, blast, and concussive mechanisms [6, 8]. BCI is often associated with other thoracic injuries, including rib fracture (57%), pulmonary contusion (41%), sternal fracture (22%), and pneumothorax (21%), as well as extrathoracic injuries including traumatic brain injury (41%) [9].

Diagnosis

Given this heterogeneity of injury patterns, BCI can be difficult to diagnose. History and physical exam findings are generally nonspecific or masked by the presence of other injuries. Complaints of chest pain or shortness of breath and findings of ecchymosis, chest wall tenderness or crepitus, or flail chest are markers for a high-risk mechanism, although are poorly specific [10]. More subtle findings, including jugular venous distension, distant heart sounds, friction rubs, and new murmurs may not be appreciated in the acute trauma setting. It is important to maintain a high index of suspicion for BCI in injured patients in whom the level of hemodynamic instability does not correlate with suspected blood loss. Cardiac risk factors, including prior myocardial infarction (MI), recent angioplasty or stress test, angina, hypertension, hyperlipidemia, tobacco or other drug use, current medications, and family history of MI should be elicited [11].

Immediate bedside ultrasonography should be performed in all hypotensive patients sustaining chest trauma in order to identify hemopericardium [1]. The bedside Focused Assessment with Sonography for Trauma (FAST) exam has been shown to evaluate the pericardium with 97–100% sensitivity [12]. Once immediately life-threatening injuries have been ruled out, all patients sustaining blunt chest injuries should undergo screening ECG (Fig. 13.1) [13]. The most common ECG abnormalities include sinus

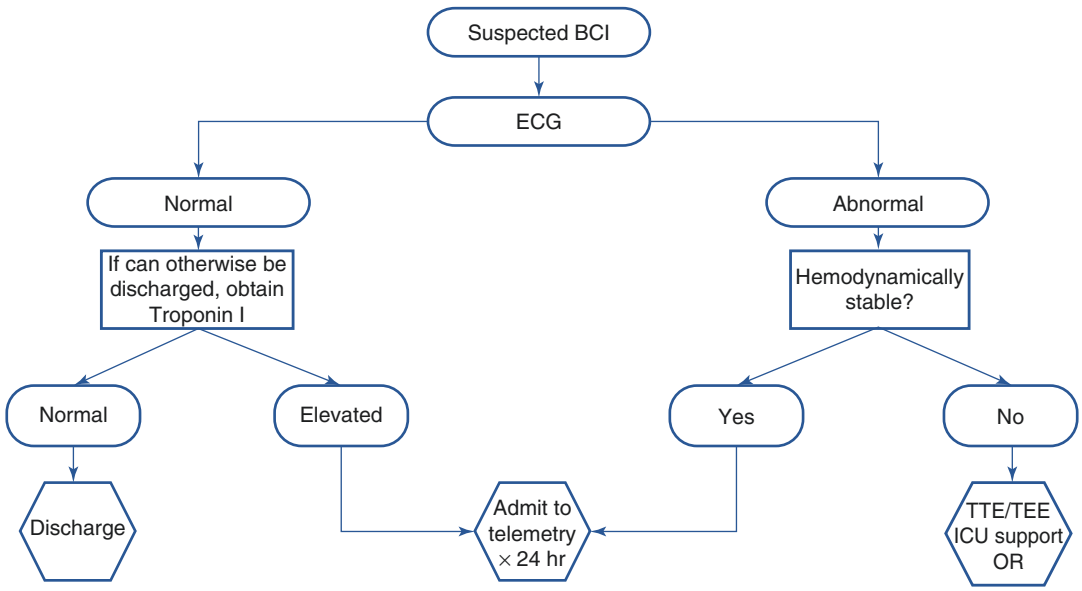

Fig. 13.1 Proposed diagnostic and management algorithm for blunt cardiac injury. *BCI* blunt cardiac injury, *ECG* electrocardiogram, *TTE* transthoracic echocardiogram, *TEE* transesophageal echocardiogram, *ICU* intensive care unit, *OR* operating room

tachycardia, atrial fibrillation, and ST segment changes [14, 15]. These typically manifest within 48 h of injury. Of all arrhythmias requiring intervention, up to 80% are detected on admission ECG in the emergency department [11]. While guidelines historically concluded a normal ECG was sufficient to exclude BCI, clinically significant BCI has been described despite normal ECG findings [14–18]. To this end, the addition of a Troponin I level has been found to aid in the exclusion of BCI. In combination, a normal ECG and normal Troponin I exclude BCI with 100% sensitivity and 100% negative predictive value [16, 19].

Chest radiography (CXR) should be performed as an adjunct to the primary survey. While lacking in sensitivity and specificity for cardiac injury, it may identify other injuries, such as pulmonary contusion, rib and sternal fractures, a widened mediastinum, hemothorax and/or pneumothorax, all of which are associated with BCI [9, 11].

Cardiac imaging, in the form of transthoracic or transesophageal echocardiography, may be a helpful adjunct in the appropriate patient. Echocardiography characterizes the degree of left ventricular dysfunction and identifies associated valvular disorders or pericardial fluid, if present [18, 20]. It is important to note that echocardiography may appear normal despite depressed cardiac activity [21]. Other imaging modalities, including CT, MRI, magnetic resonance coronary angiography, and nuclear medicine studies should only be used on a case-by-case basis after multidisciplinary consultation [22].

Specific Injury Patterns

Cardiac Concussion/Contusion

Cardiac contusion loosely describes a spectrum of injury with ranging from a slight elevation in Troponin I to cardiogenic shock. Given the inconsistency in the definition of BCI, incidence in the literature ranges from 0 to 76%. Histologically, cardiac contusion is characterized by localized edema, myocardial hemorrhage, and necrosis, although it is diagnosed by either symptoms of chest pain or elevation in biomarkers following chest trauma [22]. While largely mild or asymptomatic and self-limited with resolution in 24–48 h, symptomatic contusions resulting in hypotension, pulmonary edema, arrhythmias, or conduction delays should prompt immediate further investigation and admission to ICU level of care [23].

Dysrhythmia

Dysrhythmia is the most common manifestation of BCI, typically presenting as sinus tachycardia, premature atrial or ventricular contractions, and atrial fibrillation. Excluding sinus tachycardia, dysrhythmia on ECG is present in 1–6% of patients with chest trauma [23]. Severe injury can be associated with high-degree heart block, and ventricular tachycardia or fibrillation [24].

Commotio Cordis

Commotio cordis is a form of sudden cardiac death resulting from BCI. Pathophysiologically, this is thought to result from a high-velocity chest impact that occurs during ventricular repolarization, resulting in ventricular fibrillation secondary to an R-on-T phenomenon [25]. On autopsy there is no evidence of morphologic injury or preexisting heart disease. Commotio cordis is most prevalent in boys less than 18 years old, especially in young athletes where it is the second most common cause of sudden cardiac death [26].

Myocardial Rupture

Blunt cardiac rupture is rapidly fatal, as the pericardium rapidly fills with blood, resulting in tamponade and cardiac arrest. Mortality approaches 80% even among those surviving transport. In those with contained rupture, hypotension may be protective, with worsening of tamponade upon fluid resuscitation [1]. A pseudoaneurysm may form, sealing off the myocardium, however, this defect is prone to spontaneous rupture without prompt surgical repair. Ventricular rupture is more common than atrial rupture, although atrial rupture is more survivable. Right sided injuries are more common in either chamber type [24, 27].

Septal injuries may be identified by the presence of a murmur, thrill, arrhythmia, or conduction disturbance and are confirmed by echocardiography. Severe injury results in overt cardiogenic shock, whereas milder injury has a more insidious course—contusion leading to necrosis and delayed rupture.

Valvular Injuries

Valvular injuries can similarly present in the acute setting or with a more indolent course, with degree of hemodynamic derangement correlating to the severity of injury. In all cases injury results in valvular insufficiency. Injury to the aortic valve is most common, resulting from a sudden increase in intrathoracic pressure against a closed valve. The resultant acute valvular insufficiency results in cardiogenic shock. Mitral valve injury typically presents with pulmonary edema and hypotension [1]. Delayed disruption of the mitral valve may occur as papillary muscle contusion leads to necrosis and rupture, resulting in a more indolent course. Tricuspid injury manifests as right ventricular dysfunction progressing over months to years. Pulmonic valve injury is rarely described and seemingly well tolerated [28].

While frank pericardial rupture is unlikely in BCI, laceration can result in pericarditis with delayed tamponade from an accumulated effusion.

Coronary Artery Injury

Most coronary artery injuries are comprised of lacerations, dissection, and aneurysm formation with or without rupture [1]. Given its anterior position, the left anterior descending (LAD) coronary artery is the most commonly injured of these vessels, comprising 76% of coronary injuries [29].

Treatment

Treatment of BCI varies widely according to the specific injury subtype and severity. All patients with abnormal ECG or elevated troponin I levels should be admitted with telemetry monitoring for at least 24 h [24]. Severe myocardial contusions may result in significant arrhythmia, heart failure, or cardiogenic shock, and as such, patients with severe BCI should be admitted to an intensive care unit (ICU) and treated with dysrhythmics, inotropes, or mechanical support as indicated [30]. A standard approach to dysrhythmia management should be employed. Those with rapid atrial fibrillation and hemodynamic compromise should undergo cardioversion. Hemodynamically stable patients should undergo electrolyte repletion, acidosis correction, and avoidance of hypoxia. Additionally, antidysrhythmic medications should be used as appropriate with consideration of anticoagulation and cardioversion if atrial fibrillation persists. Right bundle branch block (RBBB) is the most common conduction disturbance following trauma and usually is not associated with long-term sequelae [1]. While uncommon, complete heart block necessitating temporary or permanent pacemaker placement has been described [31, 32]. ST segment elevations provide a management quandary in that they can be a result of myocardial contusion, or a more dire traumatic myocardial infarction (MI). Although no standardized recommendations exist, many experts would recommend coronary angiography in patients with ST elevation and concern for MI [33].

Structural injuries to the myocardium, pericardium, valves, or coronary arteries require surgical intervention with timing dependent on the degree of hemodynamic instability and the acuity of decompensation. Mobilization of a perfusion team for cardiopulmonary bypass may be necessary. Emergent thoracotomy may be indicated for blunt cardiac rupture resulting in hemopericardium and cardiac arrest secondary to tamponade. It is important to remember pericardiotomy should be performed parallel to the phrenic nerve to prevent injury. Once the blood has been evacuated, the heart is assessed for injury. Temporizing measures to attain hemostasis include use of digital pressure, a surgical clamp, suture, Foley catheter balloon, or staples according to location, size, and operator preference. Bimanual cardiac compressions can be started as soon as hemorrhage is controlled with transfer to the OR and

definitive repair undertaken [11]. In the hemodynamically stable patient with tamponade, some authors suggest pericardiocentesis with or without drain placement as a temporizing measure, however, this should never delay definitive operative intervention. Aortic valve injuries typically require urgent operative repair, whereas mitral valve injuries can often be temporized with afterload reduction and mechanical support with an intra-aortic balloon pump. Percutaneous coronary intervention with stenting is optimal therapy for uncomplicated coronary lesions, however, surgical revascularization may be indicated.

Blunt Aortic Injury

Blunt traumatic aortic injury (BTAI) occurs when shearing forces generated by abrupt deceleration result in a tear in the descending thoracic aorta at the level of the ligamentum arteriosum [2]. It is frequently seen in MVCs, pedestrians struck by motor vehicles, and falls from height >3 m [1]. Uncontained BTAI is rapidly fatal, and even in the setting of rapid definitive treatment, 30% of patients that survive to the hospital die within 24 h and more than 50% die within the first week after injury [34]. Some of this significant mortality rate can be attributed to the high incidence (40%) of severe concomitant injuries [35]. With contained aortic rupture, hypotension is likely a result of associated injuries that should be addressed prior to consideration of aortic repair.

Diagnosis

Patients with suspected BTAI should undergo workup according to the advanced trauma life support (ATLS) protocol. As an adjunct to the primary and secondary survey, a single-view CXR is often the first diagnostic test obtained. CXR may reveal a widened or abnormal mediastinum, blurred aortopulmonary window, loss of aortic knob, apical capping, a trachea shifted to the right, or depressed left main stem bronchus, all signs of aortic injury. Reports of the prevalence of abnormal CXR findings are varied, with some groups reporting false-negative radiographs in 44% and others reporting abnormal mediastinum in 93% of patients with BTAI surviving to hospitalization [1, 36, 37]. As such, any patient with clinical suspicion of BTAI should undergo further imaging regardless of CXR findings. While aortography was the gold standard for diagnosis of BTAI for 40 years following Parmley's landmark study, modern helical CT has an estimated sensitivity of 100% and specificity of 99.7% for detecting even minor aortic injuries, and has the additional benefits of being rapidly available, able to identify other injuries, and less invasive [34–36, 38, 39].

Specific Injury Patterns

Aortic Rupture and Contained Rupture (Pseudoaneurysm)

Aortic transection and free rupture carries a greater than 80% mortality rate within the first 30 min of injury [34, 40]. Survivors typically present with a full thickness tear contained by surrounding adventitia or perivascular hematoma and connective tissue or an aortic pseudoaneurysm and remain at risk for free rupture [34].

Aortic Intimal Tear and Dissection

Classic dissection following blunt trauma is rare; however, similar to rupture, it frequently begins at the aortic isthmus at the level of the ligamentum arteriosum with an intimal tear [34]. This can be associated with a hematoma within the vessel wall. Low grade intimal injuries often resolve, however, some do progress to dissection. As with classic dissection, systemic hypertension is the most common risk factor [1]. Complications of dissection include acute and severe aortic regurgitation, coronary ischemia, free rupture, and cerebral, extremity, and visceral ischemia.

Treatment

Emergent repair is required for cases of frank or impending rupture. Impending rupture may be heralded by pseudoaneurysms occupying >50%

of aortic circumference with significant mediastinal hematoma, large left hemothorax, or significant lactic acidosis (>4 mmol/L). Endovascular repair is recommended in most patients, although open repair, ideally via a left posterolateral thoracotomy in the fourth intercostal space, may be required [35, 41, 42]. The Society for Vascular Surgery issued clinical practice guidelines for endovascular repair of traumatic thoracic aortic injury in 2011, citing lower mortality (9% vs 19%) as well as lower risk of paraplegia with endovascular repair vs. open repair with comparable stroke rates [43]. Technical contraindications to endovascular repair include small aortic diameter (<15 m), involvement of the arch proximal to the left common carotid, and anomalous left vertebral artery origin on the aortic arch without an intact circle of Willis [35]. Left upper extremity ischemia is uncommon in the trauma setting, however, can be treated with carotid–subclavian bypass. Carotid–subclavian bypass may also be required after endovascular repair if the left subclavian artery is covered in patients with history of CABG and graft off the left internal mammary artery, as abrupt occlusion puts them at risk for catastrophic MI. Device-related complications are estimated to occur in 2.4% of patients and long-term graft follow-up to assess for migration and endoleak is required [44]. Open repair consists of reconstruction of the injured aorta with an interposition graft. This can be performed using a "clamp and sew" technique, left heart bypass, or full cardiopulmonary bypass, with decreasing rates of paraplegia, but increasing need for heparinization respectively. Postoperatively, patients are placed on aspirin and maintained with a goal of normotension.

When other injuries or severe comorbidities preclude immediate aortic repair, initial nonoperative management of aortic injury is an acceptable treatment strategy. This consists of antihypertensive therapy with a targeted systolic blood pressure goal of less than 120 mmHg, MAP goal of less than 80 mmHg, and target heart rate of 60–80. This treatment strategy has been shown to significantly improve outcomes in those undergoing delayed repair [45]. The goal of therapy is to reduce shear stress on the aortic wall, in a principle similar to the management of acute aortic dissection. A titratable beta-blocker, such as esmolol, is preferred given the high risk of hemodynamic instability [1]. There have been no case reports of aortic rupture in a patient receiving appropriate beta-blocker therapy, and it is well tolerated in most patients. Those who undergo definitive repair >24 h after presentation may have lower mortality than those undergoing immediate repair, especially in patients with TBI [46].

While clinical guidelines from vascular and trauma societies have recommended early definitive repair of aortic pseudoaneurysms, select small lesions, particularly those <50% of the aortic circumference, can likely be managed medically or with interval follow-up and elective repair [47]. While the data suggest infrequent failure of nonoperative management on the short term, long-term data is lacking, and in the majority of cases definitive repair is recommended.

As with classic dissection, surgical management is the mainstay for proximal aortic dissection due to trauma, whereas dissection distal to the left subclavian artery can be managed medically. Endovascular repair may be warranted if distal dissection is complicated by threatened rupture, progressive dissection despite optimal medical therapy, or malperfusion [1].

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Penetrating Cardiac Injury

14

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Epidemiology

Penetrating cardiac injury remains a highly lethal condition that is challenging to study. Historically, less than 10% of patients with penetrating cardiac injuries reached the hospital alive, though this is likely improving with rapid prehospital transport [1]. For patients with penetrating cardiac injury who are treated at a hospital, overall mortality ranges from 22 to 67%, depending on the context [2–6]. Factors associated with increased risk of mortality following penetrating cardiac injury include prehospital cardiopulmonary resuscitation, prolonged prehospital time, resuscitative thoracotomy, multichamber cardiac wounds, and multiple body cavities affected [5–9]. In some studies, cardiac stab wounds and tamponade appear protective against mortality when compared to gunshot wounds and massive hemothorax respectively [5–9]. Timely transport, diagnosis, and definitive surgical management of penetrating cardiac injuries are required to optimize patient survival [10].

Mechanism and Anatomy

Most penetrating cardiac injuries result from wounds caused by firearms or knives; however, multiple other implements have been described to cause cardiac stab wounds. Cardiac gunshot wounds range from through-and-through injuries to blast injuries. Bullets, shrapnel, or shotgun pellets can result in retained fragments and subsequent embolization to the pulmonary artery (right-sided fragments) or systemically (left-sided fragments) [11]. Thus, a high clinical suspicion of penetrating cardiac injury must be maintained in any situation with an unclear trajectory.

The most commonly injured chambers of the heart are the right ventricle (40%), the left ventricle (40%), and the right atrium (24%) [12]. Less commonly, penetrating cardiac injuries can involve the coronary arteries, valves, or multiple chambers, and such complex injuries are more likely following gunshot wounds compared to stab wounds. Penetrating cardiac wounds are graded on a scale of severity from I to V, with grade I injuries involving the pericardium only and grade V injuries defined as wounds that result in >50% tissue loss of a cardiac chamber [13].

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Prehospital Intervention

Minimizing prehospital time and interventions is critical to the survivability of penetrating cardiac injuries. Hemodynamic decompensation due to tamponade and/or exsanguination occurs rapidly and requires prompt operative intervention for definitive management. In one study, 14% of patients with penetrating cardiac injury survived following out of hospital cardiac arrest, compared with a 76% survival rate for those who did not arrest [5]. Surgical intervention within 30 min has been shown to be associated with improved survival while scene and transit times of greater than 10 min have been shown to be associated with increased mortality for penetrating cardiac injury [1, 5].

Prehospital intubation should be avoided when possible. Intubation leads to increased intrathoracic pressure and a resultant drop in preload caused by relative compression of the inferior and superior vena cava. In patients with penetrating cardiac injury, any degree of hemodynamic instability caused by cardiac tamponade or exsanguination will be exacerbated by intubation and may result in cardiac arrest [14].

External chest compressions have minimal benefit in the case of cardiac arrest caused by pericardial tamponade or exsanguination. In pericardial tamponade, the increase in intrapericardial pressure leaves little space for additional filling of the heart [15]. In exsanguination, the chambers of the heart are empty; therefore, external compression will not lead to demonstrable cardiac output.

For patients with penetrating trauma, we advocate for minimizing prehospital and emergency department crystalloid resuscitation in favor of relative hypotension (target systolic blood pressure $\leq$90 mmHg) until definitive surgical hemorrhage control can be obtained [16, 17].

Presentation

Classically, the "cardiac box" on the anterior chest wall was used to determine anatomic risk of penetrating cardiac injury. The box is defined superiorly by the clavicles, laterally by the midclavicular lines and inferiorly by the costal margins. It is important to note, however, that penetrating cardiac injury cannot be excluded when wounds lie outside the cardiac box [18]. If fact, penetrating cardiac injuries from wounds outside the box are associated with higher mortality than wounds inside the box [19]. A recent analysis of 263 patient autopsies found that the cardiac box is inadequate to predict anatomic risk for penetrating cardiac injury caused by gunshot wounds [20]. Authors of this study contend that cardiac injury should be considered in any gunshot wound to the thorax [20].

Patients with penetrating cardiac injuries can present on a clinical spectrum from asymptomatic with normal vital signs to cardiac arrest. Physiologically, bleeding into the pericardial sac causes compression of all cardiac chambers, resulting in decreased ventricular filling, decreased stroke volume, decreased venous return, septal shift to the left, and ultimately cardiovascular collapse. A relatively small amount of blood within the pericardial sac (100–150 mL) can result in cardiac tamponade in the acute setting [21]. Beck's triad of muffled heart sounds, hypotension and distended neck veins is a classic finding in tamponade but may be difficult to examine for in the trauma resuscitation area. Pulsus paradoxus, which occurs when the systolic blood pressure falls during inspiration, may also be present in tamponade, and is most readily apparent in intubated patients in our experience. Tachycardia and a narrowed pulse pressure, which may be easier to assess in the trauma patients, should raise suspicion for tamponade with cardiac injury in a patient with penetrating wounds to the thorax. Importantly, cardiac gunshot wounds may not result in tamponade but instead present with massive hemothorax caused by decompression of the bleeding injury into the chest. Thus, a penetrating cardiac injury cannot be excluded, even with negative ultrasonographic imaging of the pericardium, in a patient with a hemothorax and a wound with trajectory suspicious for cardiac injury [22].

Evaluation

When a patient with suspected penetrating cardiac injury arrives to the hospital, a rapid evaluation should commence. We advocate for early and complete exposure of penetrating trauma patients to facilitate the rapid identification of wounds and immediate initiation of cavitary triage. Thoracic gunshot trajectories and stab wounds within the cardiac "box" (and beyond) should prompt evaluation for penetrating cardiac injury [20, 23].

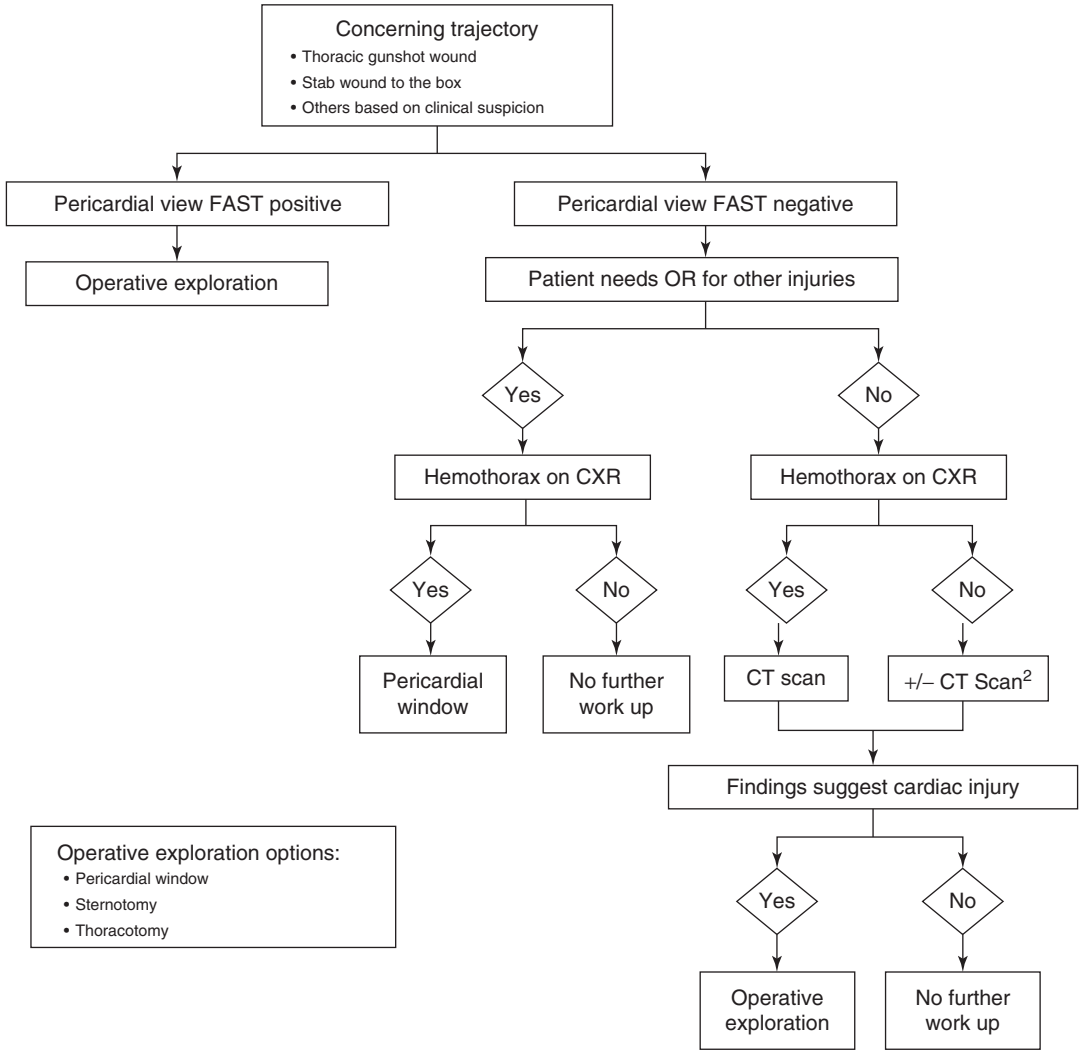
As with all trauma patients, the principles of Advanced Trauma Life Support (ATLS) should guide evaluation and resuscitation. Physical examination should include assessment for the classic physical exam findings of pericardial tamponade. The presence of hemorrhagic shock in a patient with a suspicious trajectory should heighten clinical suspicion for penetrating cardiac injury. Chest X-ray and the Focused Assessment with Sonography for Trauma (FAST) exam should be completed as adjuncts to the primary survey. Surgeon performed ultrasound has been shown to be a rapid and accurate tool to diagnose hemopericardium and should be the part of the work up of all patients with suspected penetrating cardiac injury [24, 25]. A FAST examination that demonstrates pericardial fluid is highly suspicious for cardiac injury. Hemothorax on chest X-ray or ultrasound may represent decompression of a cardiac injury into the chest, even in the setting of a negative pericardial FAST [22].

An unstable patient with suspected penetrating cardiac injury based on any combination of physical examination, chest X-ray (hemothorax), or FAST exam should be managed in the operating room. Our approach to the evaluation of the stable patient with suspected penetrating cardiac injury is summarized in Fig. 14.1. Stable patients with a concerning trajectory and positive pericardial FAST exam should receive operative intervention. In stable patients whose examination and radiography data are inconclusive, a chest computed tomography (CT) scan is a useful adjunct [26]. In these patients, the chest CT Scan can help to clarify trajectory and assist in evaluation for secondary findings concerning for penetrating cardiac injury, including occult hemopericardium, pneumopericardium, or retained hemothorax [27].

Emergency Department Management

Patients with penetrating cardiac injury who are pulseless or in imminent cardiac arrest should receive a resuscitative thoracotomy in the emergency department. This procedure should be performed in conjunction with endotracheal intubation, large bore intravenous and/or central line placement, a right chest tube, and resuscitation with blood products when appropriate. Advanced cardiac life support (ACLS) should be considered, along with administration of bicarbonate, calcium, and intracardiac epinephrine.

The goals of the resuscitative thoracotomy for penetrating cardiac injury in order of importance are: to relieve tamponade, control cardiac hemorrhage, and restore perfusion with aortic cross clamp, cardiac massage, and resuscitation. To access the heart, a left anterolateral thoracotomy through the fifth intercostal space is performed. The pericardium is opened anterior and parallel to the phrenic nerve to relieve pericardial tamponade. In the case of tense tamponade, a scalpel may be preferred over scissors to enter the pericardium. Once inside the pericardium, the cardiac injury is identified, and hemorrhage controlled. Although foley balloon placement, suturing, and stapling have been described in the management of penetrating cardiac wounds in the ED, we believe "less is more" in this setting and prefer either judicious clamping for atrial injuries (see Fig. 14.2) and/or finger occlusion for ventricular injuries. Once tamponade is relieved and hemorrhage controlled, an aortic cross clamp may be applied; however, this maneuver may not be necessary in the case of isolated tamponade and restoration of a perfusing rhythm. If there is blood in the right chest or an atrial injury, increased exposure may be achieved by extending the incision across the sternum either



[1]Hemodynamically unstable patients should be managed in the operating room
[2]CT scan may be performed based on clinical suspicion of cardiac injury

Fig. 14.1 Evaluation of suspected cardiac injury in the stable patient

partially or completely with a bilateral anterior "clamshell" thoracotomy [28]. In the case of massive hemothorax and resulting exsanguination causing cardiac arrest, massive transfusion should be initiated. Other intrathoracic injuries causing bleeding should also be managed at this time.

For patients with penetrating cardiac injury who are not in imminent cardiac arrest, large bore intravenous and/or central line access should be obtained. In tamponade, optimizing preload is critical to maintain cardiac output. Temporary volume loading to overcome external compression of the right ventricle should be considered in patients with clinical features of cardiac tamponade. To prevent hemodynamic compromise associated with endotracheal intubation, we recommend avoiding ED intubation in favor of intubation in the operating room when possible [29]. Tube thoracostomy should be placed for a hemothorax and/or pneumothorax identified on physical exam, Chest X-ray, or

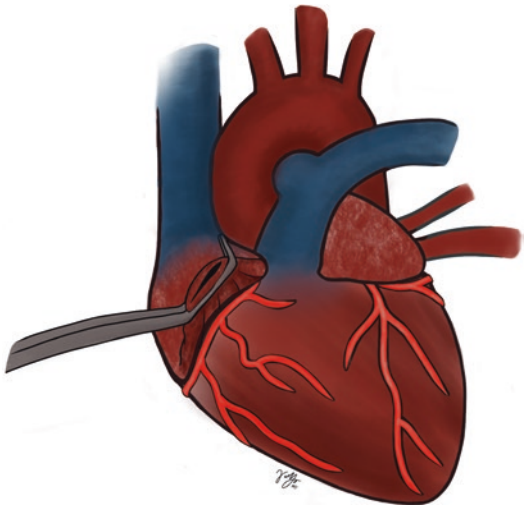


Fig. 14.2 Judicious clamping of a penetrating injury to the right atrium provides temporary control of bleeding

FAST exam. Today, subxiphoid pericardial window and pericardiocentesis are rarely if ever indicated in the emergency department. For patients in hemorrhagic shock, administration of IV vasopressin should be considered and activation of the hospital massive transfusion protocol should be carried out [30]. Interventions in the ED should be as expeditious as possible to allow rapid transport to the operating room for definitive surgical management.

Pericardial Window

In patients with suspected penetrating cardiac injury and equivocal FAST or in cases with suspicious trajectory, hemothorax, and negative FAST, a pericardial window should be performed to evaluate for cardiac injury (Fig. 14.1). There are several techniques described for the pericardial window procedure, including subxiphoid, parasternal, and transdiaphragmatic (laparoscopic and open) [31]. In general, we prefer subxiphoid or parasternal approaches for isolated thoracic trauma, while the transdiaphragmatic approach may be used to evaluate the pericardium during laparotomy. The sensitivity and specificity of subxiphoid pericardial window to detect a cardiac injury are 100% and 92%, respectively [32]. To prevent a false positive window, care must be taken to ensure a bloodless field prior to incision of the pericardium. If there is blood within the pericardium, this suggests a cardiac injury. We avoid closure of the pericardium following pericardial window to reduce risk of tamponade and pericarditis. Following a transdiaphragmatic pericardial window, we generally close the diaphragm with a figure-of-eight 0 polypropylene suture. This is especially important in cases with concomitant intrabdominal injury and contamination.

Although pericardial window and washout with and without drain placement has been described as definitive treatment for a positive pericardial window in hemodynamically stable patients, studies of this procedure are limited by design, size, long term follow-up and in some cases, external validity given prolonged prehospital and observation times which are not present in or suitable for all contexts [33–35]. With the available evidence, we recommend exploration for all patients with positive pericardial window via median sternotomy or left anterolateral thoracotomy.

Operative Management

Once in the operating room with a patient suspected to have a cardiac injury, we routinely prep and drape prior to intubation. In the case of an isolated stab wound to the precordium, a median sternotomy may be performed. This incision is carried from the suprasternal notch to the xiphoid, using a sternal saw to divide the sternum down the middle. This incision allows access to anterior cardiac structures but may limit exposure of posterior mediastinal structures and the aorta. In the case of gunshot wounds or posterior cardiac injuries, a left anterolateral thoracotomy with the patient in the supine position is preferred. This is performed with similar technique to the resuscitative thoracotomy described above and may be extended across the sternum to perform a bilateral anterior "clamshell" thoracotomy if needed. As previously described, tamponade is relieved by entering the pericardium.

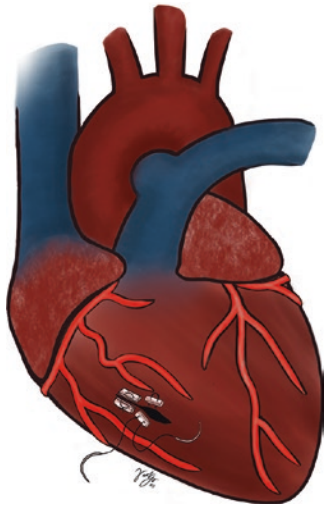


Fig. 14.3 Definitive repair of penetrating cardiac injury is best performed with pledgetted horizontal mattress sutures

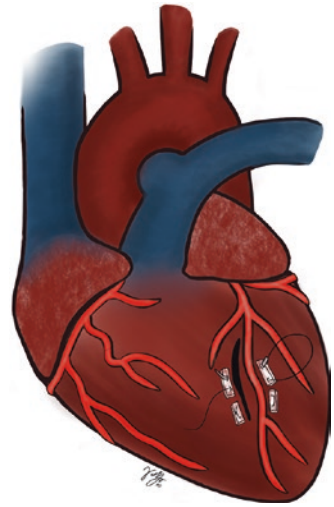


Fig. 14.4 Injuries adjacent to coronary vessels may be safely repaired with horizontal mattress sutures placed under the vessel

Digital pressure for ventricular injuries and vascular clamp application (e.g., Satinsky clamp) for atrial injuries should be used to allow time for resuscitation prior to definitive repair (Fig. 14.2). As illustrated in Fig. 14.3, definitive cardiorrhaphy for ventricular injuries should be performed using horizontal mattress sutures buttressed with polytetrafluoroethylene pledgets to prevent tissue tearing. Injuries adjacent to coronary vessels should be repaired with a horizontal mattress suture placed under the vessel (see Fig. 14.4). Pledgets are most useful for repairing injuries to the thicker-walled ventricles. Lower pressure venous and atrial lacerations may be repaired with simple running sutures without pledgets. If a clamp has been utilized to temporarily control these injuries, running suture under the clamp is very effective. For the cardiac repair, we use a 3-0 polypropylene suture on a large tapered needle to permit full thickness bites with minimal tissue damage. A double-armed suture is preferred when using pledgets. As with the pericardial window, we generally do not close the pericardium following open pericardial exploration in order to reduce risk of tamponade and pericarditis.

Complex cardiac injuries, including those to the coronary vessels, valves, or multiple chambers require special consideration. In these cases, it can be helpful to involve a cardiac surgery specialist. Distal coronary artery injuries may be ligated and monitored for arrhythmia and/or significant hypokinesis, while proximal injuries require repair with or without cardiopulmonary bypass. Ideally, transesophageal echocardiography (TEE) should be performed in every case of penetrating cardiac injury; however, it is most important in cases of suspected valvular or transchamber injuries. When identified either intraoperatively or postoperatively, valvular injuries and intracardiac fistulas should be repaired based on the clinical situation with assistance from cardiac surgery. Posterior cardiac wounds can be challenging to repair as elevation of the heart can cause cardiac arrest or arrhythmia. Methods for cardiac retraction include apical suturing, clamping, and sequential elevation with laparotomy pads. We have had some recent success in repairing posterior injuries with the aid of a cardiac positioning device and suggest consideration of their use if available.

Postoperative Management and Complications

Systemic inflammation following penetrating cardiac injury is common, particularly in the patient who presents in extremis. Postoperative management should minimize oxidative stress, coagulopathy and end organ dysfunction [36]. Ventilator settings should be customized to the patient, with preference for lung protective, low stretch settings and adequate ventilation for maintenance of normal acid–base status. Avoidance of acute hypercapnia is critical, particularly in patients with right ventricular dysfunction, since elevations in carbon dioxide can increase pulmonary arterial vasoconstriction and right ventricular afterload. Conversely, patients with right ventricular dysfunction related to cardiac injury may benefit from the use of inhaled nitrous oxide or epoprostenol to reduce pulmonary vasoconstriction and right ventricular afterload. These agents should be administered as soon as right ventricular dysfunction is recognized in the operating room. When possible, postoperative fluid status should be managed judiciously in favor of a restrictive resuscitation strategy.

Postoperative transthoracic echocardiography (TTE) is recommended for all patients with penetrating cardiac injury and is usually an acceptable alternative to intraoperative TEE. An abnormal postoperative echocardiogram is common and can demonstrate pericardial effusion, wall motion abnormality, reduced ejection fraction, intramural thrombus, valve injury, conduction abnormality, pseudoaneurysm, or septal defects [37]. A transiently depressed ejection fraction is a common finding on postoperative TTE. Supportive measures should be employed as the early phase of postoperative systemic inflammation resolves.

Atrial or ventricular dysrhythmias after repair of penetrating cardiac injury are uncommon, but can represent life-threatening postoperative complications [38]. Cardiac electrical pathways can be disrupted primarily related to the injury, secondarily by the repair, or in a delayed fashion due to scar tissue formation. Treatment for postoperative dysrhythmias should be tailored to the patient and can include medical management or invasive therapies. Beta blockade can be useful to mitigate postoperative catecholamine surge, reducing the risk of dysrhythmias and preventing development of tachycardia-induced cardiomyopathy.

Pericarditis after operative management of cardiac injury is common, presenting in up to 30% of patients [39]. Management should include consideration for NSAIDs and colchicine, as well as interval repeat echocardiography due to the increased risk of delayed pericardial effusion [40]. Development of pericardial effusion after repair of penetrating cardiac injury can occur as early as one day and as late as several months after surgery. Patients with pericarditis are at increased risk. Use of colchicine reduces the risk of developing postpericardiotomy pericardial effusion [41]. Echocardiography prior to discharge and in a delayed fashion for symptomatic patients should be employed [37].

Follow-Up

The literature on long term outcomes after penetrating cardiac injuries is limited to small descriptive studies. Secondary sequelae following penetrating cardiac injury are relatively common, affecting 23–52% of patients [37, 42–44]. Complications reported in the literature include coronary damage, conduction system injury, intracardiac fistula, septal defects, valvular injury, pseudoaneurysm, and pericardial effusion/pericarditis [37, 42–44]. Although some patients with secondary sequelae will present with clinical symptoms or an abnormal cardiac examination, many will be asymptomatic. Echocardiography is the cornerstone for diagnosis of secondary complications following penetrating cardiac injury. Therefore, we recommend obtaining an echocardiogram prior to discharge on all patients with penetrating cardiac injury. Consideration for follow-up echocardiogram may be made on a case-by-case basis. Once diagnosed, most secondary sequelae may be safely managed expectantly [37]. When complications require surgical repair, outcomes are generally good [44]. For

patients who survive penetrating cardiac injury without coronary or valvular injury, long-term outcomes are expected to be excellent [45]. Of note, long-term outcomes following cardiac injuries managed by pericardial window and washout with or without drain placement are unknown, and no current recommendations exist for follow-up of these patients [33–35].

Conclusions and Future Directions

Despite advances in prehospital and hospital care, penetrating cardiac injury remains a highly lethal condition. Rapid transport, diagnosis, and management are required for survival. In this chapter, we reviewed the epidemiology, mechanism of injury, anatomy, presentation, evaluation, and management of penetrating cardiac injury using past and present literature and our own clinical experience. Future research should examine optimal modes of prehospital resuscitation and transport and develop and test novel methods of cardiac stabilization during repair and the postoperative period. Unfortunately, some penetrating cardiac injuries will continue to be nonsurvivable due to the immediate tissue destruction caused by the trauma. For these injuries, the solution is prevention. A public health approach to violence prevention should be at the center of our trauma system in the USA with programs present at trauma centers and in communities nationwide.

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15 Penetrating Vascular Injuries of the Thorax

Matt Strickland and Kenji Inaba

Introduction

In the care of injured patients, there are few insults as immediately life-threatening and technically challenging as penetrating vascular injuries to the thorax. It is an area of the body distinctive for large, high-flow vessels, vast potential spaces, and narrow margins of error. While most abdominal injuries can be easily accessed through a midline laparotomy, the bony thorax and fixed mediastinal structures require the surgeon to consider and master multiple incisions and approaches. Finally, most clinicians caring for trauma patients are, by virtue of their training and elective surgical practice, more accustomed to operating in the abdomen. They are thus doubly tested by having to operate on highly lethal problems in a body cavity they are less accustomed to.

This chapter offers an organized approach to the most important vascular injuries in the chest, including the subclavian vessels, the aorta and its major branches, and the pulmonary vessels. Specific considerations are offered for the assessment and resuscitation of these patients. The chapter focuses on the core knowledge and techniques to manage the vast majority of such injuries but several important, related topics such as cardiopulmonary bypass, systemic hypothermia, and complex shunting procedures are outside the scope of detail presented here.

General Principles and Initial Management

The approach to patients with suspected thoracic vascular injuries begins with a rapid primary survey as organized and advocated by the Advanced Trauma Life Support curriculum. Many of these patients will already be in advanced stages of hemorrhagic shock and it is imperative to identify this early. With penetrating injuries, clinicians will generally have a high suspicion of thoracic trauma based on external wounds. Chest X-ray should be performed early in the assessment as it may reveal hemothoraxes, widened mediastinum, and information about the trajectory of any ballistics. An e-FAST is another useful adjunct that, in trained hands, can diagnose hemothoraxes and pericardial blood. If trauma bay imaging reveals evidence of bleeding in the pleural space, chest tube thoracostomy should be performed and the amount of bleeding carefully observed. High volume bloody output from the chest tubes usually necessitates operative intervention.

The thoracic inlet and extremities should be examined carefully for the vascular hard and soft

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signs, summarized in Table 15.1. Injuries to the mediastinal and subclavian arteries can commonly result in expanding hematomas at the thoracic inlet or clavicular area or pulselessness in an upper extremity. A hard sign has traditionally been considered an indication for immediate operative management [1]. However, any patient who can safely undergo CT angiography should have this extra imaging performed as it may guide the surgical approach and possibility of endovascular interventions. Patients with soft signs who are hemodynamically normal should undergo CT angiography since many of them will not require further intervention.

In the unstable patient, priority should be placed on initiating hemostatic resuscitation protocols and obtaining operative control of the bleeding [2, 3]. Where possible, upper extremity intravenous lines should be inserted in the contralateral side from the injury. This helps avoid ineffective transfusions either because of a poorly perfused arm in the case of an arterial injury or extravasation of the infused fluids through a proximal injury in the case of a venous injury. For central venous catheterization it also avoids the potential for a concomitant in-line iatrogenic injury [4].

In a landmark paper of hypotensive patients with penetrating injuries to the torso, a third of which were to the thoracic area, mortality was improved by delaying resuscitation until bleeding was controlled [5]. In this permissive hypotension strategy, crystalloid or blood for resuscitation is restricted and blood pressures below normal are accepted until definitive hemostasis is achieved. For massively exsanguinating patients, the administration of blood products in a 1:1:1 ratio of packed red blood cells, fresh-frozen plasma, and platelets is associated with improved mortality [6].

Although most thoracic vascular trauma is noncompressible, direct pressure may be effective in more superficial injuries and for those to the thoracic inlet and clavicular areas. If pressure is unsuccessful, balloon tamponade is a good option. To perform this maneuver, a large Foley catheter is inserted as far into the tract as possible and the balloon is then inflated with water (Fig. 15.1). The catheter is clamped at the level of

Table 15.1 Hard and soft signs of vascular injury [1]

Hard signs	Soft signs
1. Brisk external bleeding 2. Pulsatile or rapidly expanding hematoma 3. Loss of pulse 4. A palpable thrill or audible bruit 5. Shock attributable to the injury	1. A history of arterial bleeding at the scene or en route 2. A small nonpulsatile, nonexpanding hematoma 3. Minor bleeding 4. A pulse discrepancy as measured by ABI or BBI

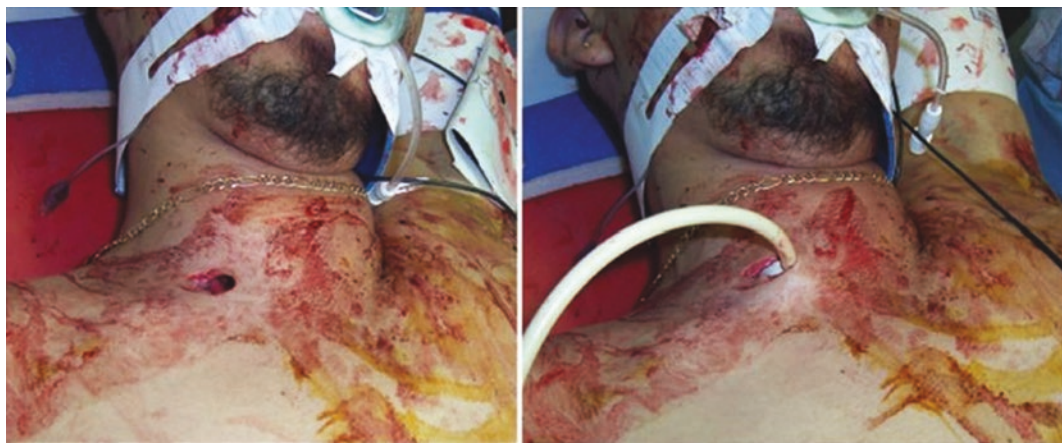

Fig. 15.1 Balloon tamponade in a patient with penetrating injury near the thoracic outlet. With permission [7]

the skin to produce tension and keep the device in place. For large cavities, additional catheters may be required, and the skin opening partially closed to prevent balloon extrusion. For wounds with communication into the pleural cavity, two catheters may be used with the first passing beyond the parietal pleura, inflated and pulled back until seated against the inner chest wall sealing the internal opening, with a second used to occlude the bleeding source [4]. These are measures to slow the bleeding while the patient is prepared for further imaging or intervention.

For large volume blood loss, autotransfusion has been shown to be safe while decreasing the need for allogeneic transfusions and reducing hospital costs. This can be achieved in the resuscitation area by using chest tube collection chambers that are designed for autotransfusion and, once in the operating room, using intraoperative cell salvage devices. The need for these systems should be anticipated so that the equipment can be obtained early in the course of care [8, 9].

If the patient needs operative intervention, some thought must be put into which surgical approach will yield the best exposure of the injured structures. Although this relies on a number of factors, as discussed throughout this chapter, Table 15.2 summarizes the major types of vascular injuries and the optimal incision to control them.

Table 15.2 Preferred incision for visualization of thoracic vascular structures

Structure	Optimal approach
Ascending aorta and aortic arch	Median sternotomy
Brachiocephalic artery and vein	Median sternotomy
Right subclavian artery or vein	Clavicular incision +/– median sternotomy
Left subclavian artery or vein	Clavicular incision +/– median sternotomy
Descending thoracic aorta	Left posterolateral thoracotomy
Pulmonary vessels	Posterolateral thoracotomy
Superior vena cava	Median sternotomy

Subclavian Vessel Injury

Epidemiology

Penetrating injury to the subclavian vessels remains an uncommon injury in both military and civilian settings. In a large analysis of 2471 arterial injuries from American troops during World War II, only 21 (0.85% of the total) of these injuries were reported, with six leading to loss of limb [10]. In large American civilian series, penetrating subclavian artery injuries are equally rare. The Elvis Presley Regional Trauma Center in Memphis, Tennessee, reported a mean of one case per year between 2000 and 2013 [11]. Similarly, when the Jefferson Davis/Ben Taub General Hospitals reported their 30 years experience, they noted only about five subclavian injuries annually, or 2.9% of their penetrating cardiovascular trauma [12]. The majority of subclavian vessel injuries in the USA are from gunshot wounds (53–80%), but stab wounds (25%), iatrogenic injuries, and shotgun wounds also contribute [11–13]. In countries with less access to firearms, stab wounds predominate [14]. Approximately 25% of injuries will involve both the artery and the vein [4].

Anatomy

The right subclavian artery originates from the brachiocephalic (innominate) artery where it divides into the right common carotid and subclavian. On the left, it usually originates directly from the aortic arch as the third and final major branch. Bilaterally, the artery can be divided into three segments, relative to the anterior scalene muscle. The first portion is from the artery's origin to the medial edge of the anterior scalene, the second portion lies deep to the scalene, and the third portion lies lateral to the scalene. This relationship is illustrated in Fig. 15.2. The first portion gives rise to several important branches including the vertebral, internal thoracic, and

thyrocervical arteries as shown in Fig. 15.3. After it passes over the first rib, the subclavian artery becomes the axillary artery.

The subclavian vein is the medial continuation of the axillary vein as it passes over the first rib. Its course is anterior to the anterior scalene. Near the medial border of this muscle, it joins with the internal jugular vein and becomes the brachiocephalic (innominate) vein. On the posterior aspect of this junction, the left and right thoracic ducts, on their respective side, drain into the venous system, as illustrated in Fig. 15.4.

Very closely related to the subclavian arteries are the branches of the brachial plexus. Other relevant nerves include the phrenic, which lies on the anterior scalene muscle, and the vagus nerve that runs anteriorly in close proximity to the first part of the subclavian. On the right, the vagus gives off the recurrent laryngeal nerve which loops posteriorly under the subclavian artery before continuing back into the neck with the carotid artery.

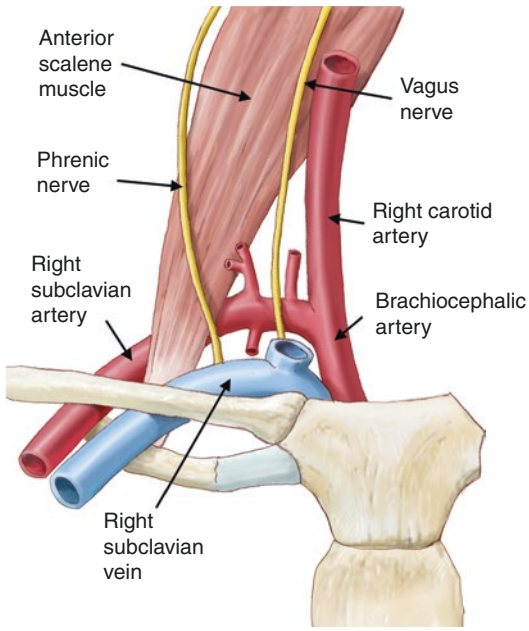

Fig. 15.2 Relationship of the subclavian artery and vein to the anterior scalene muscle. With permission [7]

Presentation and Diagnosis

Patient presentation is variable and depends on how well the bleeding is contained, transport time, and whether the injury is in direct communication with the pleural cavity. In hemodynamically normal patients, physical exam findings may include a diminished or absent ipsilateral pulse, a large hematoma, findings consistent with a brachial plexus injury, and thrill or bruit. In patients who have only soft signs of vascular injury and are sufficiently resuscitated, a CT angiography of the chest is the most valuable screening investigation [4, 14]. Figure 15.5 shows a subclavian artery pseudoaneurysm in a patient

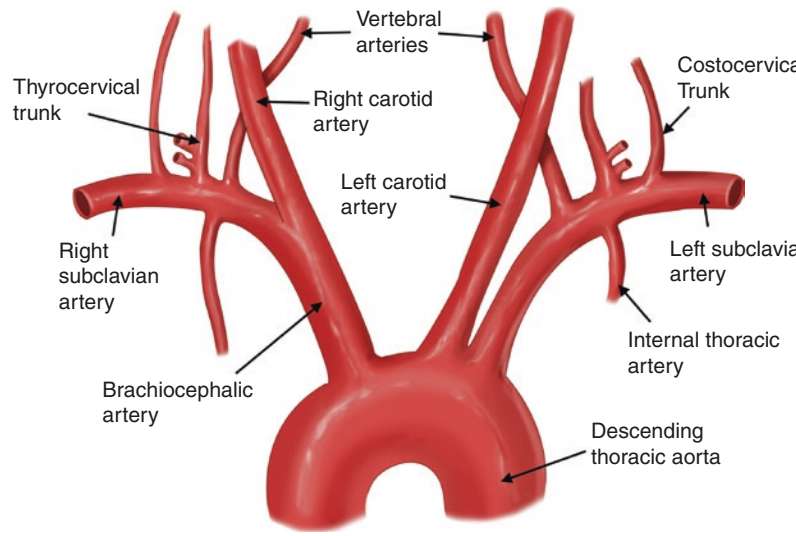

Fig. 15.3 Anatomy of the aortic arch and subclavian artery branches. With permission [7]

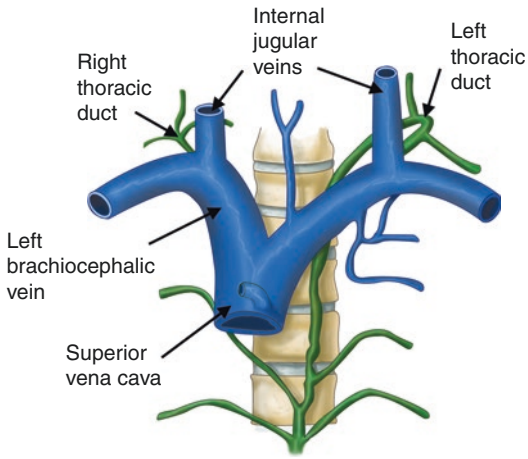

Fig. 15.4 Subclavian venous anatomy and relationship to thoracic ducts. With permission [7]

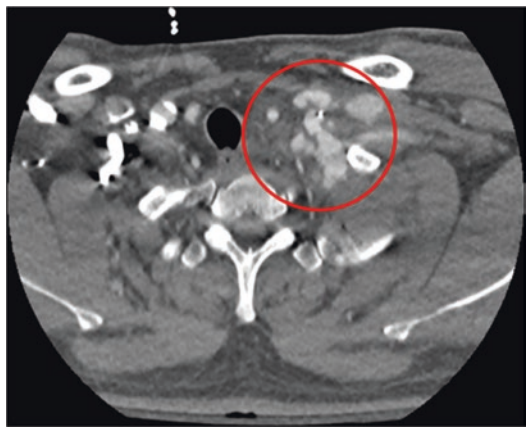

Fig. 15.5 Pseudoaneurysm in a patient with subclavian artery and vein injury from a gunshot wound

with a GSW and diminished pulses in his left arm. For injuries where there are retained metallic fragments creating artifacts, common after shotgun injuries, catheter-based angiography can be helpful.

Operative Management

While the majority of contained, minor venous injuries may be managed nonoperatively, any subclavian artery or major venous injury will require operative intervention. Exposure is critically important for addressing subclavian vascular injuries, and while several different approaches are often discussed, for both right and left sided injuries, the clavicular incision (Fig. 15.6) is highly effective, extensible, and provides excellent access to the second and third part of the subclavian. For injuries that require more proximal control, combining the clavicular incision with a sternotomy gives excellent exposure to the full length of the vessels. One alternative, a trap door incision, is associated with greater morbidity secondary to rib fractures, pain, respiratory complications, and bleeding, and is therefore of limited utility. Finally, a supraclavicular incision is rarely suitable for trauma because of its more limited exposure.

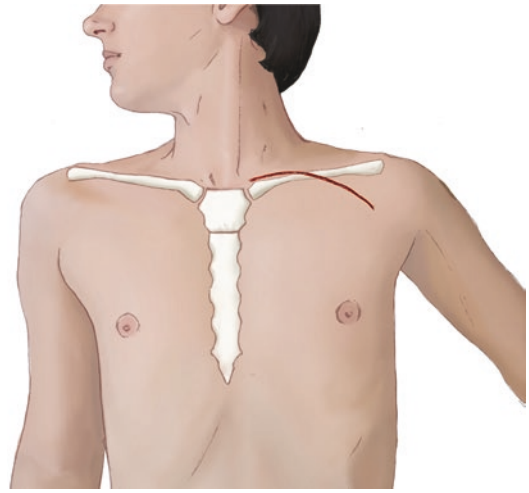

Fig. 15.6 The clavicular incision. With permission [7]

A clavicular incision begins at the sternoclavicular junction and extends along the superior aspect of the clavicle until the midclavicular line when it courses gently inferior and onto the deltopectoral groove. The clavicular head of the sternocleidomastoid, subclavius, and medial portion of the pectoralis major are detached from the medial half of the clavicle. Any remaining soft tissue on the clavicle is then cleared with a periosteal elevator and Doyen rasp (Fig. 15.7). The clavicle can be disarticulated at the sternoclavicular joint or, preferably, divided using a Gigli saw. Once this has been accomplished, the bone can be rotated externally around its lateral attachments to expose the vascular structures posteri-

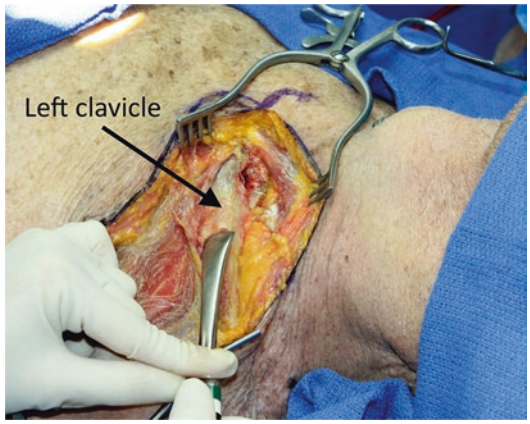

Fig. 15.7 A periosteal elevator is used to clear soft tissue from the clavicle. With permission [7]

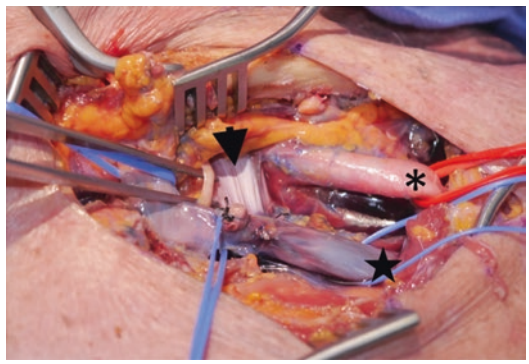

Fig. 15.8 Exposure of the subclavian vein and artery after the clavicle has been divided and retracted superiorly. Subclavian artery marked with asterisk, subclavian vein with star, and anterior scalene with arrowhead. With permission [7]

orly. The vein will be the anterior-most vascular structure and is usually located inferiorly relative to the artery. To expose the artery, the anterior scalene muscle must be transected, making sure to spare the phrenic nerve that runs along its anterior aspect. Figure 15.8 illustrates the anatomy once the clavicle has been divided and retracted from the field.

Once the vascular injury has been identified and controlled, a decision must be made between damage control procedures and definitive reconstruction. As a general principle, ligation of the subclavian artery should be avoided because of high rates of limb loss [15, 16]. A better damage control option is to perform temporary shunting and delayed reconstruction [17]. Primary repair is seldomly possible so vascular reconstruction with a synthetic or autologous graft is the procedure of choice. While both graft materials are acceptable, PTFE is often used because saphenous veins are often too small and lead to graft–artery size mismatch.

Injuries to the subclavian vein are of less consequence and the vein can be ligated without significant morbidity. If it is a simple injury, the vein can be repaired primarily as long as the repair does not cause greater than 50% stenosis as this would increase the chance of thrombosis and pulmonary embolism. For any patient that would require a complex venous reconstruction, especially in a damage control setting, ligation should be performed.

For cases that have combined arterial and venous injury, prolonged ischemic time, or associated injuries to the extremity, a prophylactic fasciotomy should be considered. If the clavicle was transected or disarticulated, it can be reapproximated with steel wire or an orthopedic plate. If the patient's physiologic condition will not support this, replacement of the clavicle is not mandatory [18].

Endovascular Management

Endovascular treatment for penetrating subclavian artery trauma is described but remains uncommonly used [19]. In trauma, the experience on this approach comes mainly from case reports and small case series. While short term outcomes appear comparable to open repair for subclavian injuries, data about long-term results, including stent patency, remains sparse [20–22]. In a large review of the contemporary management of subclavian artery injuries reported between 2004 and 2014, there was no increase in the rate of endovascular management [23]. In centers with appropriate expertise, endovascular repair can be considered in stable patients with contained injuries or factors that complicate open repair.

Prognosis

Outcomes for subclavian injuries are generally good. Modern mortality rates are approximately 10%, and long-term limb salvage is achieved in 97% of survivors. Late complications related to stent or graft placement are uncommon [23]. Those patients who do go on to have limb loss after subclavian vessel injury are more likely to have combined injuries to the artery and vein, severe tissue loss, and early graft thrombosis [23]. Specific anatomic complications associated with subclavian injuries that warrant special attention include damage to the brachial plexus, phrenic nerve, recurrent laryngeal nerve, or thoracic duct. Ligation of the vertebral artery can cause embolization and posterior strokes [24].

Aorta and Brachiocephalic Injuries

Epidemiology

The majority of patients with penetrating thoracic aortic injury die before they can be brought to hospital.

In Debakey's series of 2471 vascular injuries from WWII, there were only three aortic injuries recorded [10]. Reported in-hospital mortality rates, then, are largely dependent on the patient population and injury pattern arriving to care. For example, the first major series of penetrating aortic injuries reported by a large American trauma center with rapid transport time described a 38.9% mortality [13]. In a large South African series, published in the same era, the mortality rate was only 5%, likely on account of a lower rate of gunshot wounds and poor prehospital triage and transport [25]. Only a small proportion of penetrating thoracic trauma that reaches hospital will have injury to the aorta or brachiocephalic [26]. In a series from Los Angeles, 2% of all thoracic GSWs and 1% of all thoracic SWs had injury to the aorta. The same study demonstrated that, like the penetrating trauma population at large, the majority of victims are young men (95%) with a mean age of 27.4 years [27].

Anatomy

The aorta has three major divisions in the chest: the ascending aorta, the aortic arch, and the descending aorta. The ascending aorta is approximately 5 cm in length and is partially covered in pericardium. The right and left coronary arteries are its only branches, originating just distal to the aortic valve. The aortic arch curves leftward and posteriorly in the superior mediastinum. Its three major branches, illustrated in Fig. 15.3, are the brachiocephalic, the left carotid, and the left subclavian. The brachiocephalic, in turn, branches into the right subclavian and right carotid arteries. The descending thoracic aorta is the longest segment and extends from just distal to the left subclavian artery to the aortic hiatus in the diaphragm, located adjacent to the T12 vertebra. There are numerous small branches from the descending aorta, including nine paired intercostal arteries, the esophageal arteries, and the bronchial arteries.

Presentation and Diagnosis

Patient presentation will depend largely on prehospital time and whether the injury is contained. In urban environments, patients will usually arrive in severe hemorrhagic shock or having already lost vital signs [27]. Where there is a longer transport time, some patients may be hemodynamically stable [25]. Thus, in the majority of patients with significant injuries, there will not be time for advanced medical imaging and they should proceed directly to the operating room. If the patient is normotensive and appears stable enough to undergo investigations, a CTA of the chest is the best choice and has been shown to be accurate when compared to angiography [28].

Operative Management

The optimal surgical approach depends on the location of the injury and the stability of the patient. As most patients are hemodynamically

unstable at presentation, information about the vascular injury must be inferred from the hypothesized trajectory, the chest tube output, and whether the patient needs resuscitative maneuvers such as aortic cross-clamping or open cardiac massage. Usually, for versatility, the patient will be placed supine on the operating table with arms abducted to 90°. For injuries to the ascending aorta and aortic arch, a sternotomy offers the best access to the mediastinum. The optimal incision for exposure of a descending thoracic aortic injury is a posterolateral thoracotomy in the 4th intercostal space [29], however, this is generally inadvisable in unstable patients so an extended anterolateral thoracotomy is usually performed.

Because the ascending aorta lies partly in the pericardium, some patients with a contained injury will have an associated cardiac tamponade [30]. If this is the case, the pericardium should be opened in the midline to avoid injury to the phrenic nerves and the hemopericardium evacuated while controlling the injury with digital pressure. To better expose the arch, the thymus may need to be dissected and then bluntly retracted out of the way. The left brachiocephalic vein may also impede the surgeon's access to the arch. This can be ligated and divided with no major repercussions but this is usually unnecessary [31]. Repair of penetrating wounds to the ascending aorta and arch is usually accomplished by lateral aortorrhaphy. If necessary, partially occluding clamps can be used to control the damaged vessel and a 4-0 nonabsorbable monofilament can be used for simple repair. For larger injuries, Teflon or pericardial pledgets may help reapproximate the vessel edges. In cases requiring more complex repair or cross-clamping, the patient will need to be placed on cardiopulmonary bypass (CPB) with femoral cannulation before a patch angioplasty or interposition graft can be performed [32].

Injuries of the brachiocephalic artery and the other major branches from the aortic arch all follow the same basic principles. First, exposure may require extension of the median sternotomy toward the neck (Fig. 15.9). Once proximal and distal control have been obtained, the injury can be fully assessed. Injuries less than 30% of the circumference can generally be repaired primarily, those between 30 and 50% can undergo patch repair, and those greater than 50% should be repaired with end-to-end anastomosis or interposition graft. Figure 15.10 illustrates a brachiocephalic injury being repaired with a synthetic interposition graft. Before reperfusing either a brachiocephalic or proximal left carotid injury, it is important to clear any air or debris so that these do not embolize and cause strokes.

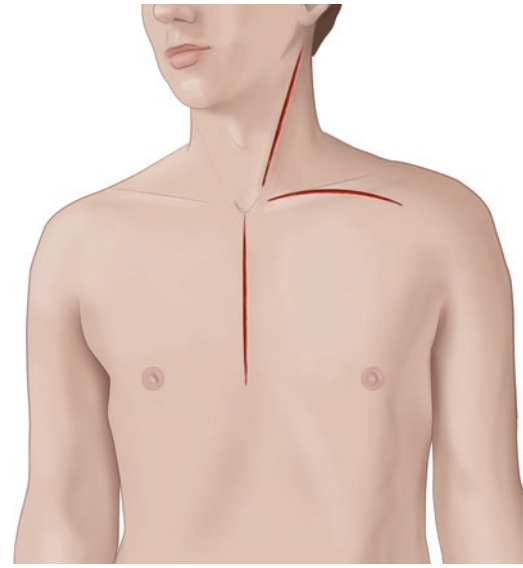

Fig. 15.9 The median sternotomy can be extended toward the neck, if necessary. With permission [7]

The descending aorta can be repaired using the same techniques as the ascending aorta and arch, but with some special considerations. Injuries to the descending aorta are usually surrounded by large mediastinal hematomas, complicating localization of the injury [31]. Because the injury is more distal along the arterial system, CPB is rarely required. More injuries, however, will require a Dacron or PTFE interposition graft (Fig. 15.11). Proximal control can be achieved by opening the pleura overlying the left subclavian and then mobilizing the nearby aorta with blunt dissection. Once sufficiently mobilized, a clamp can be placed just distal to the subclavian for proximal control. If cross-clamping is required, acute heart failure, paralysis, and acute kidney injury are the major complications. Risk of para-

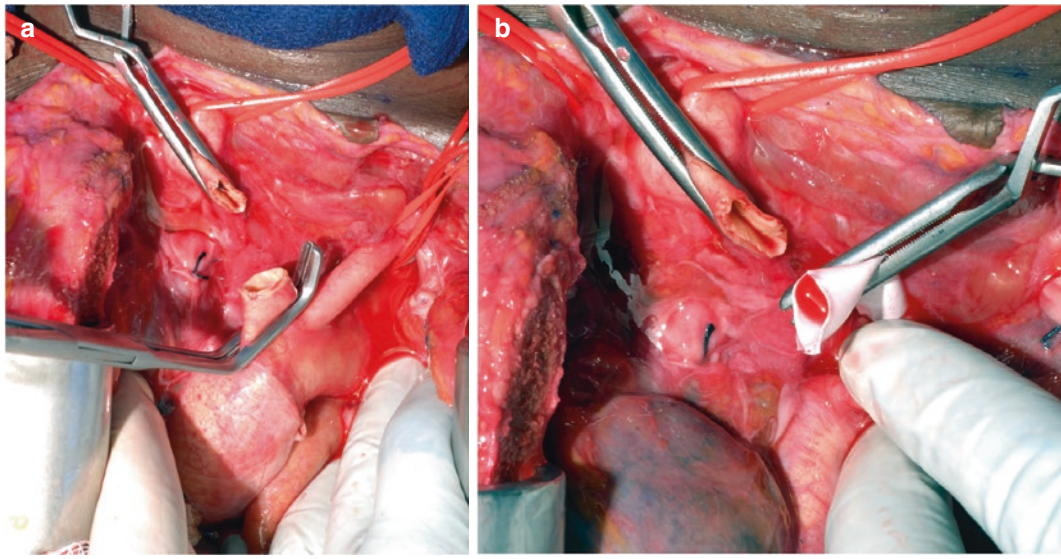

Fig. 15.10 Repair of a complex brachiocephalic injury. After resection of injured segment (**a**) and with synthetic interposition graft (**b**). With permission [7]

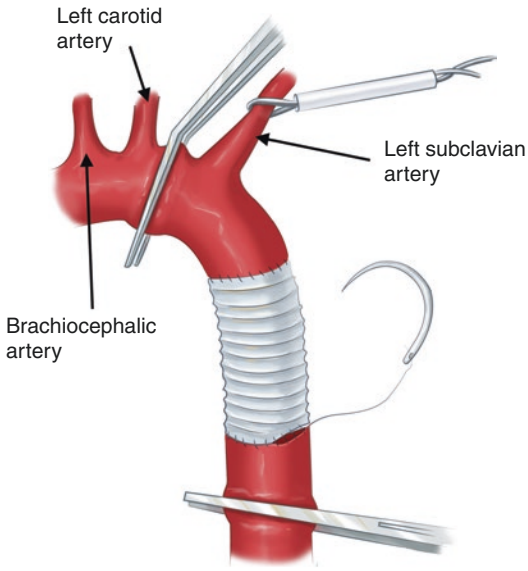

Fig. 15.11 Figure of proximal control and interposition graft repair of descending aorta. With permission [7]

plegia is approximately 8% and is related to the length and depth of hypotension, the number of intercostal arteries injured or ligated, and the duration of cross-clamping [33]. Thus, the surgeon should move efficiently to repair the aorta rapidly, ligate only those intercostals necessary for exposure, and aim for a cross-clamp time of less than 30 min [30, 33].

Regardless of location, if a single penetrating injury to the aorta is seen, it is important to consider a second injury on the opposite wall. For patients with gunshot wounds, the possibility of bullet embolism should also be considered especially if there are distant signs or symptoms, unaccounted for by the known trajectory [31].

Endovascular Management

Although the endovascular management of blunt traumatic thoracic aortic injuries is now standard of care and associated with improved outcomes [34], its role in penetrating trauma is reserved for a small subset of stable patients. Several case reports have reported successful short term outcomes [35, 36] with stents, however longer term outcomes remain unclear. Endovascular approaches should be considered only in hemodynamically stable patients with tomographic imaging and with available expertise in the necessary techniques.

Pulmonary Vascular Injuries

Epidemiology

Patients who undergo injuries to the pulmonary hilum and survive to hospital presentation are rare. In a large series of 4459 cardiovascular injuries from a major trauma center in Houston, there were 40 pulmonary vein injuries and 79 pulmonary artery injuries over a 30 years period [12]. In a review of cases reported since 1990, 62% were from penetrating trauma and 80% of patients were male. Survival in patients making it to hospital was 92% [37], but this likely reflects a publication and survivorship bias as autopsy studies of patients who die in the community have suggested a much higher mortality rate [38].

Anatomy

The pulmonary hilum normally consists of a bronchus, a pulmonary artery, and two pulmonary veins. These arborize into lobar and then segmental divisions as they extend into the lung parenchyma. The pulmonary artery (PA) trunk originates at the pulmonary valve at the outflow of the right ventricle. The PA carries deoxygenated blood at low pressures, usually with a peak systolic pressure less than 25 mmHg, but with high flows [39]. It divides into a right and left branch within the pericardium. The left PA enters the hilum immediately, lying anterosuperior to the left bronchus. The right PA passes posterior to the ascending aorta and superior vena cava before entering the right hilum (Fig. 15.12). Venous drainage of the lungs occurs through the pulmonary veins (PV) which carry oxygenated blood from the alveoli back to the left atrium. There are usually two PVs per side. The arrangement of these structures varies slightly between the left and right sides, but the bronchi are usually the most posterior structure, the veins lie anteroinferiorly, and the arteries lie anterosuperiorly (Fig. 15.13). The lung's other blood supply, the bronchial arteries, are substantially smaller and are located posterior to the bronchi. Although they carry less blood than the

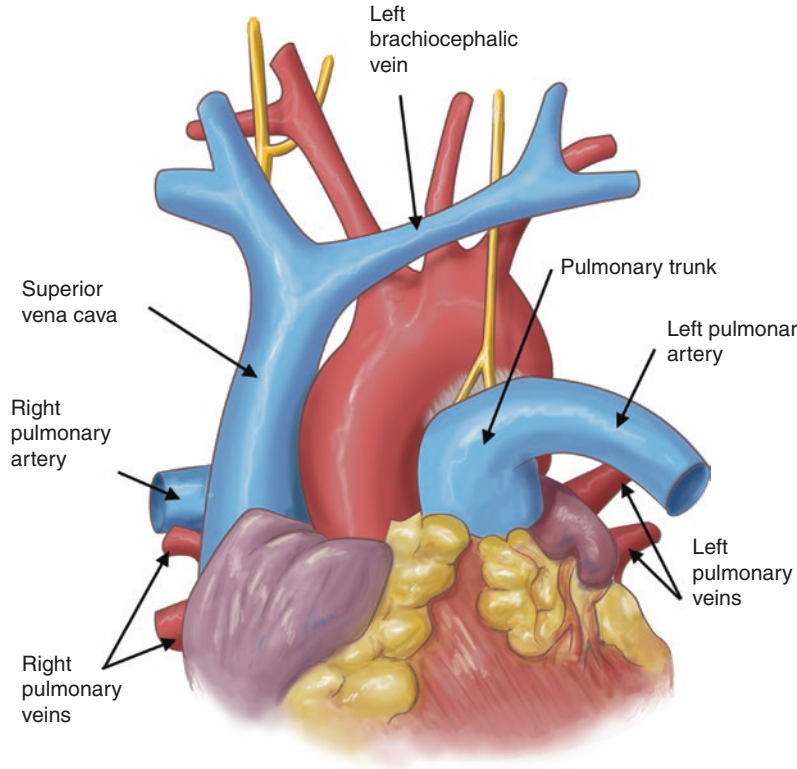

Fig. 15.12 Anatomy of the pulmonary vessels in relation to the heart and other great vessels. With permission [7]

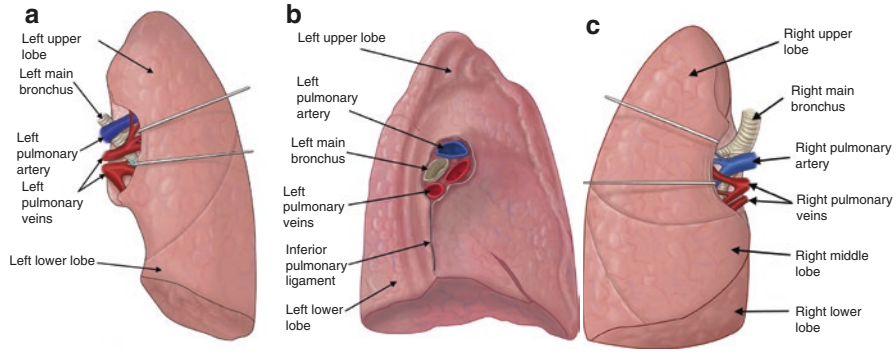

Fig. 15.13 (**a**) Relationship of pulmonary hilar structures—Left lung. (**b**) Relationship of pulmonary hilar structures—Left lung. (**c**) Relationship of pulmonary hilar structures—Right lung. With permission [7]

pulmonary vessels, they are part of the body's systemic circulatory system and are thus higher pressure.

Presentation and Diagnosis

The pulmonary vessels can cause bleeding in both the pericardial and pleural spaces, depending on the specific site of the injury. Thus, patients may present with signs and symptoms consistent with cardiac tamponade, massive hemothorax, or both. A small subset will present with normal hemodynamics because of a contained injury or embedded foreign body [37, 40]. In patients who are stable enough to undergo medical imaging, CT pulmonary angiography is the modality of choice.

Operative Management

The surgical approach will depend on the patient presentation and whether any imaging has been done to guide management. For patients with cardiac tamponade where the injury is either cardiac or to the intrapericardial pulmonary vessels, a median sternotomy offers the best exposure. For patients with massive hemothorax, an anterolateral thoracotomy offers good compromise between versatility and exposure. Injuries to the intrapericardial pulmonary vessels can be repaired with simple techniques including digital compression and lateral arteriorrhaphy with 4-0 or 5-0 monofilament, nonabsorbable sutures [41].

Control of injuries to the intrapleural pulmonary vessels first requires dissection of the inferior pulmonary ligament. This ligament extends inferiorly from the inferior pulmonary vein, attaching the lower lobe of the lung to the mediastinum medially and the diaphragm inferiorly (Fig. 15.13a). Care must be taken to not injure the vein as the dissection approaches the hilum. Control of vascular injuries is best achieved with digital compression or clamping. Although the "hilar twist," where the lung is grasped and rotated 180° around the hilum, is described for temporary control of bleeding, practically, this is less effective than the other methods and risks injury to hilar structures.

The two major options for definitive management of the bleeding vessel are simple repair or pneumonectomy. Simple repair is again reserved for smaller injuries, usually from stab wounds. If the injury is destructive or cannot be controlled, a pneumonectomy can be performed. Although pneumonectomy usually involves careful dissection of the individual structures, this may be prohibitively difficult and time-consuming in complex injuries. Patients that present alive with this injury are often critically ill and require rapid intervention. A transverse surgical stapler can be used to quickly encircle the entire hilum (Fig. 15.14) and perform an en masse pneumonectomy. The main bronchus should be taken as close as possible to the carina to avoid a long

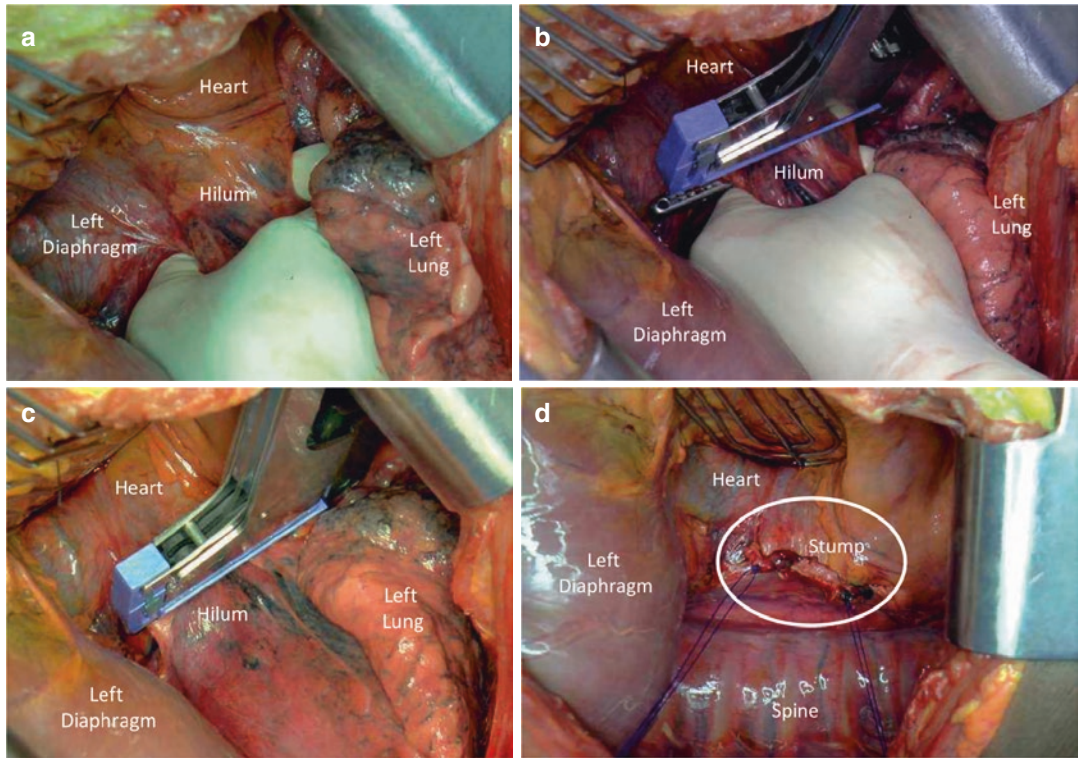

Fig. 15.14 The steps of an en masse pneumonectomy. (**a**) The inferior pulmonary ligament is taken down and structures are encircled. (**b**) A TA stapler is positioned around the entire hilum. (**c**) The staple is fired. (**d**) Stay sutures are placed on the corners of the stump before releasing. With permission [7]

blind end where secretions will accumulate and contribute to stump breakdown. Before releasing the stapler, stay sutures are placed at each corner, distal to the stapler, to prevent retraction of the pedicle. The transection is then performed, leaving a 5–10 mm cuff distal to the staple line so that it can be oversewn with absorbable suture if necessary [42].

Trauma pneumonectomy is a highly morbid procedure. In a national registry-based study of 261 patients undergoing pneumonectomy, 62.5% sustained penetrating trauma and their in-hospital mortality was 49.1% [43]. Acute right heart failure is a common complication and should be anticipated if pneumonectomy is performed. Clear communication with anesthesia, judicious use of fluids, and cardiac support with inotropes may help mitigate this.

Injury to Other Vessels

Superior Vena Cava and Inferior Vena Cava

Disruptions of the SVC or thoracic IVC usually lead to death before hospital arrival. In a 5-year review of vascular injuries in the National Trauma Databank, there were 166 penetrating and 17 blunt injuries to the SVC out of 10,677 reported adult injuries [44]. In-hospital mortality has been reported as high as 60% [2].

Repair of these injuries is extremely challenging. High volume venous bleeding is difficult to control and application of vascular clamps may aggravate the injury. Repair is further hampered by cardiovascular collapse as any clamping or attempts to visualize large injuries will cause

venous return, and thus preload to the right ventricle, to drop precipitously [45]. Since proximal and distal control are so hard to secure, where possible, a partially occluding clamp is favored. If the injury is small, a simple lateral venorrhaphy can be performed with a 5-0 polypropylene suture. For more complex injuries, a patch with autologous pericardium or an interposition graft with Dacron or PTFE prosthesis may be used to bridge the defect. Still, these techniques may require CPB to be successful [2, 46]. Using an autotransfusion device may help outcomes.

The risk of air embolism is especially notable with large venous injuries since the low intraluminal pressure may rapidly entrain large amounts of air.

Internal Thoracic Artery

Injury to the internal thoracic artery (ITA) can lead to clinically significant hemothorax, hemomediastinum, and extra-cardiac tamponade. While relatively small and protected, the ITAs have mean flow rates of about 110 ml/min [47] and thus can rapidly lead to hemorrhagic shock [48]. The injury is usually found on thoracotomy and can be ligated without major consequence. If extravasation or pseudoaneurysm is revealed on CT scan, embolization is a well-described option [49].

Intercostal Artery

Intercostal artery bleeding is usually associated with blunt injury and rib fractures, but can be the product of penetrating injury. Blood loss is often self-limited, but in those patients who continue to exsanguinate, thoracotomy or angiography and embolization should be performed. In the operating room, intercostals can usually be ligated without difficulty. Posterior injuries can be especially challenging because the ribs are closest together, the artery is largest in diameter, and access may be limited with an anterolateral thoracotomy [50]. In these cases, the injury can be temporized with pressure and ongoing resuscitation while endovascular expertise and equipment are mobilized [51].

Azygos and Hemiazygos Vein

The azygos and hemiazygos veins are relatively large diameter vessels that, when injured, can cause significant bleeding from the posterior chest. Patients generally present with hemothorax, hypotension, and associated injuries to other thoracic structures [52, 53]. Injury to these veins should be suspected intraoperatively when faced with significant bleeding coming from a region posterior to the pulmonary hilum. Ligation or oversewing are well tolerated and their safety is supported by a large experience of elective esophagectomies where taking the vein is performed routinely.

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Esophageal Injuries

16

Kelsey A. Musgrove and Daniel J. Grabo

Background

Traumatic injuries of the esophagus are relatively rare with many large trauma centers treating an average of 1–5 cases annually [1, 2]. Trauma comprises approximately 9% of all esophageal perforations with the remainder largely iatrogenic secondary to endoscopy (more than 50%), 12% foreign body ingestion, and 1% malignancy [3].

Penetrating injuries represent the majority of traumatic esophageal injuries in the USA with the majority secondary to gunshot wounds (70–80%) followed by stab injuries (15–20%) [1, 4, 5]. Esophageal injury after blunt mechanism is exceedingly rare with less than 0.1% incidence [6, 7]. Penetrating esophageal injury accounts for only 0.14% of all penetrating trauma with an increased risk in males (OR 2.62) and African Americans (OR 4.61) [8, 9]. Mortality was higher in patients with esophageal injury (20.5% vs 1.4%) and morbidity was reported to be as high as 46% [9]. A review of the National Trauma Data Bank reported incidence for esophageal injury of 37 per 100,000 trauma patients and found esophageal injury to be more common in younger (38 vs 52 year old), male patients (81% vs 62%) and more severely injured (Injury Severity Score [ISS] >25 in 45% vs 7%) [10]. Penetrating injuries were 16 times more frequent than blunt injuries with in-hospital mortality 19% [10]. Esophageal injury was again independently associated with increase in mortality after adjusting for age, gender, ISS with no difference between blunt and penetrating mechanisms [10].

The esophagus has three anatomical points of narrowing that are more commonly ruptured: the cricopharyngeus muscle of the cervical esophagus, the broncho-aortic constriction at the proximal one-third of the esophagus, and the distal esophagogastric junction (Fig. 16.1) [11]. The esophagus is located in the posterior mediastinum and is divided into the cervical, thoracic, and intra-abdominal esophagus and location of injury alters diagnosis and treatment. The cervical esophagus is the most commonly injured followed by thoracic and less frequently intra-abdominal. In cases of blunt injury, motor vehicle accidents are the most common etiology with cervical and upper thoracic injured in 82% in one study, likely due to esophageal fixation at these locations [7, 12]. Biffl et al. noted 43% of traumatic esophageal injuries involved the cervical esophagus and 57% involved the thoracic esophagus [8]. Asensio et al. analyzed 34 institutions and 405 patients with traumatic esophageal injuries between 1988 and 1998. They noted 56.6% involved the cervical esophagus, 30% thoracic, 17% abdominal, 2% combined thoracic

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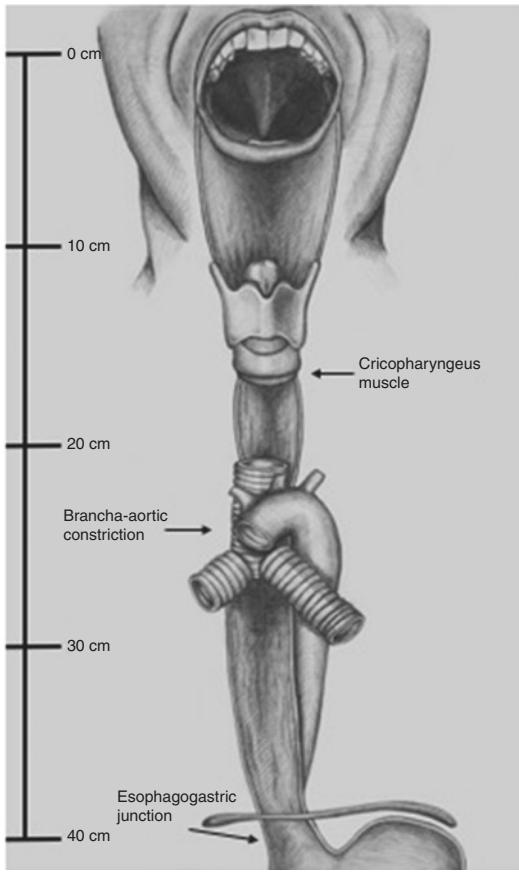

Fig. 16.1 Demonstration locations of esophageal anatomic narrowing include the cricopharyngeus muscle, broncho-aortic constriction, and esophagogastric junction. Original sketch by Mr. Michael Cameron, BFA

and abdominal, 1.2% cervicothoracic, and 0.3% cervical and abdominal [5]. Associated injuries were found in 88%, with intra-abdominal injuries including liver and spleen most common in patients who went directly to the operating room from the trauma bay [4]. Weiman et al. additionally found the majority of injuries were in the cervical esophagus (63%) with 94.7% penetrating. In cases of cervical esophageal injury, the most common associated injury was the trachea in 47% [13]. The high mortality rate of 19-30% noted with esophageal injuries is in part secondary to high grade associated injuries [1, 5, 8]. Mortality also varied with site of the perforation with one study showing lowest mortality with cervical perforation (6%), 34% with thoracic, and 29% with intra-abdominal [14].

Diagnosis

Clinical diagnosis and treatment of esophageal injury is time sensitive, with delay significantly increasing the rate of esophageal-related complications and increasing mortality after a delay of 12 h [3]. Delay in management over 24 h can result in mortality increase from 14% to 27% [3]. Other studies have shown similar trends with reported mortality of 10–25% if therapy is initiated within 24 h compared to 40–60% after 24 h [12].

Signs and symptoms of esophageal injury are not reliably present and include dysphagia, odynophagia, neck pain or swelling, hypersalivation, retrosternal fullness, hematemesis, and subcutaneous emphysema. Chest pain was noted in up to 70% of intrathoracic perforations, Hamman sign indicative of subcutaneous emphysema was noted in up to half of cases, and vomiting or dyspnea was seen in about 25% [15]. Mackler's Triad of subcutaneous emphysema, chest pain, and vomiting was only seen in 1 of 7 cases [15]. Common signs of subcutaneous emphysema and dysphagia were only identified in 19% and 7%, respectively, mandating a high level of clinical suspicion in the absence of these findings [4]. Chest pain, fever, and dyspnea were common presenting signs of acute perforation, whereas dysphagia and supraventricular arrhythmia were more common in chronic perforations (>48 h) [16].

The most common initial study that may note esophageal injury is the chest or abdominal X-ray performed in the trauma bay. Findings include air within the soft tissues of the neck, pneumomediastinum, or pneumoperitoneum. A CXR may detect up to 90% of cases, but the development of significant subcutaneous emphysema to be identified may take up to 1 h to develop and an effusion may take several hours [15]. Advanced imaging includes CT which may show paraesophageal fluid or air suggesting injury [7]. All are non-specific findings and the overall sensitivity of CT is low with one retrospective review of zone 2 penetrating injuries detected only two of the four perforations [7, 17].

Two options for definitive diagnosis are flexible esophagoscopy and esophagography. Flexible esophagoscopy is used to directly visualize the

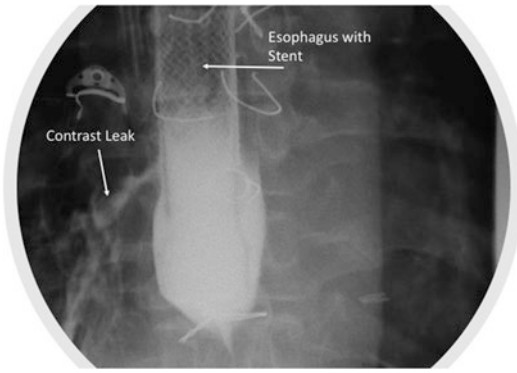

Fig. 16.2 Esophagram demonstrating leak from thoracic esophagus in a patient with iatrogenic esophageal injury previously managed with stent

esophagus and has sensitivity ranging from 96 to 100% and specificity 92–100% [8, 18–20]. If endoscopy cannot be performed, esophagography can be performed first with water soluble contrast followed by thinned barium contrast if no leak is identified. Figure 16.2 demonstrates esophogram with contrast leak. In one study, 22% perforation was noted on follow-up thinned barium swallow [21]. However this method has higher rates of false negatives between 10 and 43% [8, 18, 22, 23]. Combination of esophagogram with esophagoscopy provides nearly 100% specificity [5]. In evaluation of 231,694 patients between 2004 and 2010 only 10.4% with esophageal injury received an upper endoscopy as most injuries were discovered on CT, surgery, or autopsy [9].

Esophageal injury is graded according to the American Association for the Surgery of Trauma (AAST). Grade I consists of contusion/hematoma, partial thickness tear, grade II is a laceration less than 50%, grade III is laceration greater than 50%, grade IV is less than 2 cm disruption of tissue or vasculature, and grade V is greater than 2 cm disruption [15].

Management

Initial treatment involves management of the overall trauma patient including assessment and securing of the airway and resuscitation. If the patient is stable and esophageal injury is suspected, an endoscopic assessment of the esophagus is necessary with consideration of evaluation of the airway due to frequent concomitant injury [5]. The patient should remain nil per os (NPO) with placement of nasogastric tube for decompression under direct visualization at the time of endoscopy. Blind insertion should not be performed as this could exacerbate injury. Broad-spectrum antibiotics including antifungal coverage should be administered [5, 16].

Operative approach depends on location of the injury (cervical, thoracic, or abdominal), severity of damage, hemodynamic status, and associated injuries. Primary repair with or without buttress reinforcement is preferred if feasible and can reduce mortality by 50–70% compared to other interventions [3, 16, 24, 25]. The repair is also time sensitive and although several studies show that repair can be done safely without increased mortality after a delayed diagnosis, there is a higher overall morbidity and leak rate (0% leak if repaired in <6 h, 67% 6–24 h, and 83% if >24 h) [26].

General operative principles include exposure, debridement of devitalized tissue, closure of the defect, use of buttress with muscle preferred, adequate drainage around the repair, decompression of the esophagus and stomach, and consideration of distal enteral nutrition access [7].

After debridement of devitalized tissue, the muscular layer must be incised longitudinally to reveal the extent of the mucosal injury [11, 15]. Failure to perform this step adequately is the most common cause of post-operative leak [11]. Next, the mucosa is closed with interrupted absorbable suture and the muscularis layer closed with interrupted non-absorbable suture, see Fig. 16.3 [11]. The repair must be tension free but mobilization should be minimized due to its segmental blood supply and high risk for ischemia of the anastomosis with over-mobilization [15]. The repair can be reinforced with a buttress flap, most commonly an intercostal muscle flap in the chest and cervical strap muscle or SCM in the neck [11, 15]. Pericardium is a less ideal choice due to its fragility and relative decreased blood supply

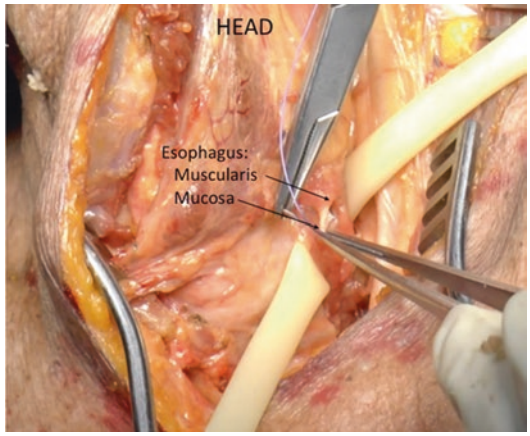

Fig. 16.3 Cadaveric specimen demonstrating repair of cervical esophageal injury. These injuries are repaired in two layers (mucosa followed by muscularis) using 3-0 vicryl sutures. Placement of buttress of vascularized muscle over the repair can be helpful

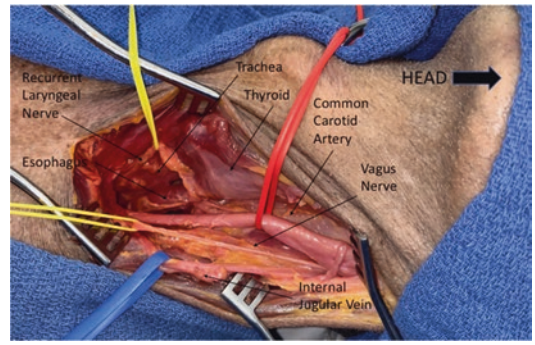

Fig. 16.4 Representative anatomic dissection of left anterior sternocleidomastoid (SMC) incision performed on a fresh, perfused cadaver specimen. This is the preferred approach for a known or suspected cervical esophageal injury. The carotid sheath has been opened to demonstrate the relationship of the common carotid artery (red vessel loop), vagus nerve (yellow vessel loop), and internal jugular vein (blue vessel loop). Anteriorly, the relationship between the thyroid (superiorly), trachea (anteriorly), and esophagus (posteriorly) is demonstrated as is the recurrent laryngeal nerve (yellow vessel loop) retracted anteriorly and out of the trachea-esophageal groove

[15]. Muscle flaps are especially important if the repair is delayed greater than 8–12 h from injury in order to improve vascular supply to a highly inflamed and contaminated field or when there is adjacent injury or suture line to avoid fistula formation [15]. Wright et al. noted that patients presenting with mediastinal sepsis should always have the repair buttressed due to increased risk of post-operative leak developing in the setting of preoperative sepsis compared to primary closure alone (70 vs 11%) [25]. One study reported successful reinforcement using collagen patches coated with human fibrinogen and thrombin with no leaks in two patients; however, there is no statistically significant data to support this [27]. After repair, distal feeding access via jejunostomy tube should be considered, especially in patients with malnutrition [11].

Technique for repair also can vary slightly depending on location, with cervical perforations typically easier to treat [11]. Primary repair is performed if the injury is easily seen; however, wide drainage in the neck is typically adequate if the injury cannot be located [11]. The esophagus is approached via the left neck where the recurrent laryngeal nerve can more easily be preserved and the incision placed at the anterior border of the sternocleidomastoid muscle [15]. Cooke et al. describe exposure of the cervical esophagus (Fig. 16.4) including retracting the sternocleidomastoid muscle and carotid sheath laterally, division of the middle thyroid vein and omohyoid muscle, medial retraction of the trachea and esophagus, dissection of the esophagus in the retropharyngeal plane, and debridement of devitalized tissue [11].

An upper or mid-thoracic perforation is approached via a right thoracotomy at the fourth or fifth intercostal space (Fig. 16.5), whereas a distal thoracic perforation is approached through a left thoracotomy at the fifth through seventh intercostal space [12, 28]. Upon entry, an intercostal muscle flap is salvaged as potential buttress, the inferior pulmonary ligament is divided and pleural reflection mobilized allowing anterior retraction of the lung. The pleural space is drained and any mediastinal devitalized tissue is debrided. The esophagus can be encircled by a penrose drain to facilitate dissection, then repair is again performed as detailed above with placement of chest tubes for wide drainage. A nasogastric tube is typically placed distal to the site of repair under direct visualization [28].

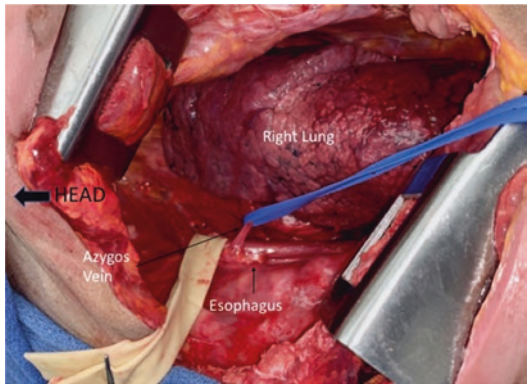

Fig. 16.5 Representative anatomic dissection of a right postero-lateral thoracotomy performed on a fresh, perfused cadaver specimen. This is the preferred approach for known or suspected upper or mid-thoracic esophageal injury. The right lung is gently retracted anteriorly. The azygos vein is demonstrated by the blue vessel loop and thoracic esophagus is encircled in and retracted posteriorly with a penrose drain

Abdominal esophageal perforation is accessed via a laparotomy. The left triangular ligament of the liver is divided and the liver retracted laterally, thus exposing the hiatus. Division of the short gastric vessels allows mobilization of the gastroesophageal junction. After primary repair, the hiatus is closed posteriorly and a fundoplication is performed to reinforce the site of repair. Wide drainage is again performed [28].

Alternatives to Primary Repair

Primary repair is avoided only if the patient is clinically unstable, the perforation is too large, there is diffuse mediastinal necrosis, there is a cervical perforation that cannot be accessed but can be adequately drained, or the patient has known or documented pre-existing esophageal disease including achalasia or malignancy [16, 24, 25]. Alternatives to primary surgical repair include drainage, diversion and exclusion procedures, esophagectomy if the patient has concomitant achalasia or malignancy, esophageal endoscopic stent placement, or non-operative management if the perforation is contained or there is limited contamination in a stable patient [3, 15].

Drainage alone is usually reserved for cervical perforations due to inability to control contamination in this fashion within the pleura and peritoneum [29]. One study noted 43% mortality for drainage of intrathoracic perforation alone versus 11% with primary repair and buttress [30]. Diversion and exclusion with drainage include: proximal diversion with cervical esophagostomy and distal diversion with a gastrostomy tube for drainage in addition to resection of the remaining esophagus [31]. Exclusion with closure of the GE junction can be performed if there is significant contamination. This approach is typically required for injury encountered at the mid to distal esophagus with or without an end cervical esophagostomy [15]. The excluded esophagus however can be a source of sepsis and the patient must be monitored for "blind loop syndrome" usually treated by drainage [15]. T-Tube diversion can also be considered and is most useful for smaller injuries near the GE junction [15]. The T-Tube is placed directly into the defect, secured and placed to suction over several weeks to ensure tract formation with simultaneous gastrostomy tube for decompression and jejunostomy tube for feeding.

Esophagectomy with immediate reconstruction can be considered with the assistance of expert consultation and in stable patients with achalasia, caustic ingestion, chronic or severe stricture that cannot be dilated, or a malignancy that would otherwise be resectable [12, 32, 33]. If the patient is unstable, delayed reconstruction can be performed, again with expert consultation [12]. If the patient has achalasia that is not end-stage or causing considerable symptoms, a primary repair can be considered with contralateral myotomy and partial fundoplication to prevent reflux [11, 12]. Esophagectomy was also noted to have improved outcomes in the setting of severe mediastinal sepsis. Salo et al. analyzed 90 patients with esophageal perforation and noted overall 44% mortality; in patients with delayed esophageal perforation with mediastinal sepsis, esophagectomy was superior to primary repair alone due to higher rates of mediastinal leakage, sepsis, and death after primary repair in these cases [24].

Endoscopic stent placement by endoscopic specialists can be considered in patients with multiple comorbidities who are unable to tolerate surgery in combination with debridement, muscle flap coverage, and wide drainage. Freeman et al. analyzed 187 patients undergoing esophageal stent placements and 8% required repair after the stent failed to resolve the leak after an average of 3 days [34]. Factors that predicted stent failure included injury longer than 6 cm, injury to the proximal cervical esophagus, injury traversing the gastroesophageal junction, and anastomotic leak associated with a more distal conduit leak [34]. Malignancy or radiation therapy did not increase likelihood of stent failure [34]. Diagnostic endoscopy is used to localize the perforation and a covered stent 4 cm longer than the injury is used to ensure 2cm proximal and distal overlap of the defect [34].

Non-operative management can also be considered in patients with contained mediastinal disruption and minimal clinical symptoms without signs of sepsis. Altorjay et al. showed the rate of complications to be lower in the non-operative treatment group compared to operative (20 vs 50%) and suggested non-operative management for intramural perforations or contained transmural perforations with no signs of sepsis or underlying esophageal disease or malignancy [29, 32]. From 1992 to 2004, Vogel et al. analyzed 47 patients with esophageal perforation. 72% (4 cervical, 28 thoracic) with no signs of sepsis and contained leaks were treated non-operatively with no mortality and decreased morbidity overall [35].

Post-operative Management and Complications

Post-operative management involves intensive care unit monitoring with close attention to cardiovascular compromise and management of infectious complications, with sepsis having the highest rate of associated mortality [15]. Contrast swallow evaluations typically take place 5–7 days post-operatively with early initiation of distal enteral (jejunostomy tube) feeds or total parenteral nutrition and maintenance of the operative drain until the repair is confirmed to be patent without leak [15]. However, esophageal anastomotic leak is relatively common, with rates up to 83% if there is a delay in repair [15, 26]. Other esophageal-related complications include wound infection, abscess, mediastinitis, empyema, pneumonia, and tracheoesophageal fistula [6]. The most common of these in one study was wound infection in 8%, empyema in 7%, and fistula in 5%. [6]. Longer term complications include diverticulum around the repair and anastomotic stricture which can be managed with endoscopic dilations or esophagectomy if persistent [15].

Summary

In summary, traumatic esophageal injury is rare with associated high morbidity and mortality. Timely diagnosis and treatment are essential to mitigate the risks of complications. Primary repair is the mainstay of treatment in the trauma patient with consideration of alternatives in an unstable patient with multiple comorbidities. The main operative principles include exposure, debridement of devitalized tissue, closure of the defect, use of buttress with muscle preferred, adequate drainage around the repair, decompression of the esophagus and stomach, and consideration of distal enteral nutrition access. Finally, non-operative management is increasingly considered a viable alternative with decreased complications compared to operative intervention. This approach should be considered more frequently in stable patients with minimal symptoms and no signs of clinical sepsis.

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Tracheal and Pulmonary Injury

17

Collin Stewart, Mohamad Chehab, and Bellal Joseph

Tracheal Injury

Tracheobronchial injuries are rare, occurring in less than 1% of all trauma patients, but carry a disproportionally high risk of morbidity and mortality [1]. They encompass a heterogenous group of injuries that are often concomitant with other injuries, making their diagnosis and management challenging. Therefore, a high index of suspicion and the ability to recognize the clinical signs and symptoms of such injuries early on are warranted. Although definitive indications for surgical intervention remain, minimally invasive techniques for a select group of patients or for patients who are poor surgical candidates have emerged.

Presentation

Tracheobronchial injuries can occur in the neck or in the chest. The cervical trachea is more likely to be injured in penetrating trauma, while the intrathoracic trachea is more often injured in blunt trauma, usually within 2.5 cm of the carina. In penetrating trauma, injuries are most likely due to missile or knife wounds, but can also be due to bony fragments secondary to blunt trauma. In blunt trauma, injury may occur by one of the three mechanisms: laceration from shear forces, rupture from compressive forces against the rigid vertebral column, or rupture from increased airway pressures against a closed glottis [2]. Associated injuries of the esophagus, great vessels, nerves, and thoracic duct must be ruled out [3].

Patients with tracheobronchial injuries typically present with dyspnea, tachypnea, hoarseness, stridor, or hemoptysis. Large amounts of subcutaneous emphysema may be present, with air bubbles seen in cases of open wounds. A distal tracheobronchial injury must be suspected in patients with a large pneumothorax and a massive continuous air leak that persists after tube thoracostomy placement. In severe cases, patients may present with respiratory distress requiring emergent definitive airway management.

Diagnostic Workup

Radiographically, the fallen lung sign on chest X-ray imaging may be present with mainstem bronchus transection. This sign describes an absent hilum, ipsilateral atelectasis, and pneumothorax with collapse of the lung toward the diaphragm (peripheral displacement). This is in contrast to the collapse of the lung toward the

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hilum seen in cases of pneumothorax without a tracheobronchial injury (central displacement) [4]. The fallen lung sign is pathognomonic for tracheobronchial injuries, but is evident only in cases of severe injury. Other more sensitive signs include abnormal mediastinal shadow, pneumomediastinum, subcutaneous emphysema, pneumothorax, or pleural effusion. Computerized tomography (CT) scan imaging may also demonstrate tracheobronchial wall discontinuity or mediastinal emphysema. This is of particular importance in penetrating trauma where the trajectory of the wound can give insight into possible tracheal injury. In physiologically stable patients, a 3-dimensional CT scan reconstruction of the tracheal surface and lumen can further delineate such injury.

The gold standard diagnostic modality for tracheobronchial injuries is bronchoscopy. Careful inspection of the entirety of the trachea is important for a thorough diagnosis. Flexible fiber-optic bronchoscopy can serve to determine the extent of the injury and aid in the intubation of critically ill patients in respiratory distress. However, a flexible bronchoscope can miss the injury if passed through an endotracheal tube that itself traverses the injury. Therefore, evaluation of the cervical trachea may necessitate withdrawal of the endotracheal tube to allow further visualization. The bronchoscope can then be used as a bougie to readvance the endotracheal tube. Rigid bronchoscopy can overcome that by allowing ventilation through the bronchoscope itself and also has the advantage of evacuating blood and debris.

Management

The first step in management of tracheobronchial injuries is securing the airway. Depending on the location of the injury, this may involve direct laryngoscopy, fiber-optic intubation, or a surgical airway. In severe cases, placing the endotracheal tube or tracheostomy directly through the injury may be necessary. Once the airway is secured, the full extent of the injury must be ascertained. This requires careful coordination with anesthesia. Cervical injuries can be managed with a single lumen tube, but more distal injuries require a double lumen endotracheal tube to facilitate repair. Due to the size and rigidity of double lumen tubes, care must be taken when intubating a patient to not worsen the situation by converting a partial injury into a circumferential one.

Tracheobronchial injuries that are large (usually greater than 2 cm) or full thickness are repaired primarily with the aim to restore airway continuity. Operative exposure of the trachea is determined by the location of the injury. A collar incision can be used to approach the proximal half of the trachea. The distal trachea as well as the right mainstem and proximal left mainstem can be approached using a right posterolateral thoracotomy. A distal left mainstem injury should be accessed with a left posterolateral thoracotomy. If severe mediastinal, parenchymal, or pleural injury requiring surgical intervention are also present, median sternotomy or clamshell incision can be considered. In cases of mainstem bronchus injury, lung-sparing repair procedures should be attempted before opting to proceed with the highly morbid pneumonectomy procedure [5].

Intubation of the contralateral bronchus during repair may prove helpful, with intermittent apnea and decreased tidal volumes also providing a more friendly operative field. Simple lacerations to the trachea are repaired with interrupted absorbable suture. More extensive injuries require debridement to healthy tissue before repair. If additional length is needed to facilitate repair, the trachea may be mobilized on the pretracheal plane. Tracheal hooks may be used to grasp a transected, retracted trachea, thereby allowing reapproximation and repair. If tracheal injury is not amenable of simple repair, tracheal resection may be necessary. In all repairs, care must be taken to avoid involving the endotracheal tube or the cuff. If separate repairs of the surrounding vascular structures or esophagus are done, an intercostal, sternocleidomastoid, or strap muscle may be placed between the suture lines to help prevent fistula formation.

Small tracheobronchial injuries (usually less than 2 cm) that are only partial thickness can be

managed nonoperatively. Conservative management may involve tube thoracostomy, covered stent placement, selective intubation beyond the injury, or even observation alone. However, close monitoring for any signs and symptoms of clinical deterioration is mandatory to allow for prompt surgical intervention. Red flags may include respiratory compromise even when on mechanical ventilation, signs of mediastinitis, or rapid progression of subcutaneous or mediastinal emphysema.

Complications

Aside from the overall complications of tracheobronchial-associated injuries of polytrauma patients, anastomosis-related complications are the most common. The rates of anastomotic dehiscence and stricture formation following repair have been reported to exceed 5% [6]. Initial management is recurring the airway with delayed reconstruction or multiple dilations. Fistula formation is another potential complication. Tracheoesophageal fistula is managed by gastric decompression, supplemental nutrition, and treatment of pneumonia. Once the patient has stabilized, then operative repair or resection can be pursued. A trachea-innominate fistula is a surgical emergency that is often fatal [7]. Other delayed complications may include bronchiectasis, post-obstructive pneumonia, mediastinitis, and cervical abscess.

Pulmonary Injury

Because of the anatomic design of the lungs, pulmonary injuries can be classified as either pneumatic or hemorrhagic. Pneumatic injuries are injuries to the lung parenchyma with rupture of alveoli that lead to a pneumothorax. These may range from the sometimes occult simple pneumothorax to the life-threatening tension pneumothorax. Conversely, hemorrhagic injuries are injuries to the bronchial, pulmonary, or intercostal vessels that lead to a hemothorax. Because of limited tissue to provide tamponade, these vessels can bleed extensively when injured. Central injuries to the lung hilum and associated major pulmonary vessels are technically challenging and often fatal if not addressed promptly.

Presentation

Lung injury should be suspected in any patient with blunt or penetrating chest trauma. History from patients must be sought whenever possible; otherwise, field history must be obtained from prehospital providers. This includes details of mechanism of injury, type of impact, type of weapon, vital signs, transport times, neurologic status, and fluid resuscitation, if any.

In patients with a pneumothorax, initial presentation can vary depending on the size and type of the pneumothorax. Patients with a simple pneumothorax may present with minimal complaints, if any. However, care must be taken not to miss such cases because a simple pneumothorax can evolve into a tension pneumothorax. Classical findings of patients with tension pneumothorax are respiratory distress, decreased or absent breath sounds on the affected side with hyperresonance on percussion, distended neck veins, tracheal deviation away from the affected side, chest wall crepitus, and paradoxical chest wall motion. Elevated intrathoracic pressure causes decreased venous return, decreased cardiac output, and ultimately cardiac arrest. Patients may also develop hypoxia, which manifests as dyspnea, confusion, anxiety, and use of accessory muscles. In patients with vital sign instability or profound hypoxia, the physical exam should suffice, and further investigation with imaging should not delay intervention with a tube thoracostomy.

The combined pathology of hemopneumothorax is not uncommon, given the shared traumatic etiology. Therefore, in patients with a suspected pneumothorax, evaluation for a possible concomitant hemothorax is warranted. Clinical presentation is usually similar, but with dullness on percussion. Hemodynamic instability is an additional factor that is contingent upon the size of the hemothorax and the rate of bleeding into the

thoracic cavity. In general, hemodynamically unstable patients require immediate intervention, but stable patients may benefit from further diagnostic imaging.

Pulmonary contusions are common following blunt trauma and are highly associated with rib fractures, especially flail chest. Such injuries can be clinically silent and not apparent on initial chest X-ray. In more severe forms, patients may present in respiratory distress and increased work of breathing. Pulmonary contusions are unique in that clinical symptoms and radiographic findings worsen over the first few days, before resolving in a 1-week period.

Diagnostic Workup

Arterial blood gas analysis must be sent for initial laboratory studies, as it yields critical information on oxygenation, ventilation, and degree of shock. Portable chest X-ray may reveal a pneumothorax or hemothorax, although controversy exists about the utility of this exam in the stable patient. In supine patients, liquid blood will spread along the posterior border of the thorax appearing as a subtle haziness that can be missed if the blood volume is small. In upright chest X-ray, liquid blood will manifest as fluid at the costovertebral angle and, depending on the volume, may displace lung tissue. Extended Focused Assessment with Sonography for Trauma (EFAST) exam with ultrasound (US) is a rapid, radiation-free alternative that is highly sensitive and specific in detecting a pneumothorax with the absence of lung sliding [8]. EFAST also offers the advantage of concomitantly evaluating the heart. In the hemodynamically stable patient, CT scan will allow greater inspection of the thoracic cavity. Hemopneumothoraces not seen on chest X-ray may be revealed, whether because of their small size or anterior nature. Further evaluation of the lung parenchyma is also achieved, revealing pulmonary contusions, lacerations, and pneumatoceles. CT scan also allows further evaluation of the great vessels and aorta [9].

Management

Initial management of traumatic pulmonary injury is guided by the principles of advanced trauma life support (ATLS). Injuries to the chest are prioritized and are addressed early in the evaluation of the trauma patient. Many thoracic injuries, such as pneumothorax, hemothorax, or pulmonary contusion can often be managed nonoperatively with a tube thoracostomy which allows re-expansion of the lung and drainage of the surrounding air and blood. Thoracic trauma patients with decreased breath sounds, hypoxia, and hypotension are presumed to have a tension pneumothorax until proven otherwise and require immediate decompression. In the field, needle decompression through the fourth or fifth intercostal space at the anterior axillary line or through the second intercostal space at the midaxillary line was found to have the lowest predicted failure rates [10]. In the hospital, most patients with a pneumothorax can be definitively treated with a tube thoracostomy, but some will have a persistent air leak requiring surgical intervention through either a thoracotomy or the less invasive video-assisted thoracoscopic surgery (VATS).

In patients with a hemothorax or a hemopneumothorax, 14 French pigtail catheters have been shown to be as effective as large bore chest tubes [11]. If the retained hemothorax is large, a second chest tube may prove helpful. However, a massive hemothorax, defined as more than 1500 mL of initial chest tube output or 200 mL/h for the first 3 h, should prompt evaluation for operative intervention. Thoracic exploration for hemorrhage control should not be delayed, as early intervention is associated with improved outcomes [12]. Emergent exploration should be done via a sternotomy or a thoracotomy. In a select group of stable patients with pulmonary vascular tree injury, or patients who are poor surgical candidates, transcatheter embolization is a feasible alternative to thoracotomy. When evaluating chest tube output, care must be taken not to confuse resolved intrathoracic bleeding with organized blood clots or tube mispositioning causing drainage cessation.

For stable patients with pulmonary contusions, treatment usually consists of adequate analgesia, ventilatory support in the form of supplemental oxygen, frequent pulmonary toilet, and judicious volume administration. In cases of respiratory failure refractory to less invasive therapies, mechanical ventilation is indicated.

In unstable patient, operative management must not be delayed. Choice of operative approach is dictated by the patient's overall clinical condition and hemodynamics, presence of concomitant injuries that will also necessitate repair, and findings of imaging studies. In the hemodynamically unstable patient, a median sternotomy, and an anterolateral thoracotomy, which can be extended as bilateral anterior thoracotomies (clamshell), provide adequate exposure to the pleural space and anterior mediastinum. Additionally, such approaches can be continued as a laparotomy for abdominal exploration, but offer little exposure of the posterior compartment. This is in contrast to the posterolateral thoracotomy most often used in elective thoracic surgery, which provides adequate exposure of the posterior compartment and is therefore the preferred approach for intrathoracic tracheal and esophageal injury repairs.

If tolerated by the patient, a double lumen instead of a single lumen endotracheal tube is preferred to improve the exposure and the intended repair or resection of the injured lung. However, in emergent settings, single lung ventilation may not be tolerated by the hemodynamically unstable patient, and lung isolation should therefore be avoided. In such cases where a single lumen tube is used, holding ventilation intermittently or manual compression of the adjacent lung can aid in optimizing exposure.

Once in the chest, blood and clots must be evacuated, and the lung is mobilized by incising the inferior pulmonary ligament and lysing any adhesions. Bleeding from the hilum can be controlled with digital pressure, a vascular clamp, or placing a Penrose drain around the hilum as a tourniquet. In cases of massive hemothorax, sources of bleeding may include a large parenchymal laceration, pulmonary vessel injury, or great vessel injury, especially in cases of penetrating trauma. If bleeding persists, a hilar twist may be performed. This should be attempted as a last resort as it can lead to further decompensation and precipitate rapid heart failure secondary to the rapid increase in pulmonary arterial pressure.

After exsanguinating hemorrhage is controlled and the patient's hemodynamics are improved, lung injuries can be attended to. Similar to the decision for operative approach, the choice of repair technique is dictated by the type and severity of the parenchymal injury, chest wall, cardiac and vascular injuries, and the patient's overall status. Simple lung injuries can be primarily repaired via pneumonorrhaphy. In more complex injuries, pulmonary tractotomy must be attempted, as tissue sparing techniques have consistently shown to improve outcomes and thus remain the mainstay for management [13]. The stapler is placed through the tract of the injury and fired, thereby exposing any injured blood vessels and airways that can be oversewn. Extensive injuries that involve unsalvageable lung tissue should be treated with a wedge resection or lobectomy. When attempting a lobectomy, the arterial and venous lobar branches should be dissected and either stapled or ligated. The lobar bronchus must also be identified and stapled prior to resection. To ensure the correct bronchus is to be transected, the lung is inflated while the bronchus is occluded by the stapling device. The lobe to be resected must not inflate. When proceeding with the resection, care must be taken not to fire the stapler through major pulmonary artery branches, especially in cases of more central lesions. Proximal hilar injuries are extremely difficult to manage and may necessitate the often fatal pneumonectomy. To further strengthen the bronchial stump following lobectomy, and especially pneumonectomy, a viable tissue flap can be used for reinforcement. This may include an intercostal muscle flap, diaphragmatic flap, or pericardial fat pad.

Growing interest and experience with minimally invasive techniques have made VATS an important part of the trauma surgeon's armamentarium in managing patients with chest injuries. VATS can be useful both as a diagnostic tool in

the assessment of continued hemorrhage and air leaks and as a therapeutic tool in the repair of small diaphragmatic injuries, control of bleeding from intercostal vessels, and empyema decortication. However, the greatest utility of VATS has been in the evacuation of retained blood clots. Ideally, patients need to be placed in the lateral decubitus position with the injured side facing up. A double lumen endotracheal tube is almost always required to allow deflation of the affected lung. The operative field must be widely prepped and draped in case a conversion to thoracotomy is warranted. The thoracoscope can be inserted through the existing chest tube site after tube removal. Otherwise, it can be inserted in the fifth intercostal space with the tip of the scapula serving as a convenient landmark. This would permit excellent visualization of the inferior and posterior portions of the thoracic cavity where blood clots are most likely to accumulate. Additional ports can then be added as needed under direct visualization. By means of high-pressure pulse irrigation and suctioning, the blood clot can be broken up and evacuated. Therefore, the success of VATS in evacuating blood clots is contingent upon the earlier timing of the procedure, i.e. before the clot becomes organized with adhesions and loculations.

Complications

The most common complication following pulmonary injury is pneumonia. Patients requiring intubation and mechanical ventilation in the field were 12 times more likely to develop pneumonia compared to those who did not. Patients with pulmonary contusion, aspiration, or hemothorax were also at an increased risk for pneumonia and acute respiratory distress syndrome [14]. There are also the complications of the chest tube insertion itself, which not uncommonly requires readjustment or reinsertion. In addition, patients with an inadequately drained hemothorax have been shown to develop an empyema in 26.8% of the cases [15]. A retained hemothorax may also lead to a fibrothorax and an entrapped lung. In general, hemothorax volumes of 300 mL and less estimated on CT scan can be safely observed if not complicated by infection. Volumes greater than 300 mL will likely require evacuation. If a lobectomy was performed, a bronchial stump dehiscence may also occur. The risk of this may be lessened by using a flap, such as an intercostal flap, to buttress the site of resection. Other uncommon complications in patients with pulmonary injury include persistent air leak, bronchopleural fistulae, and chylothorax.

Conclusion

Learning the principles in diagnosis and management of tracheal and pulmonary injuries is incumbent on all trauma surgeons and surgical residents, as such injuries can be challenging and often fatal. Tracheobronchial injuries are infrequent, but remain a serious event with significant associated morbidity and mortality. Pneumatic and hemorrhagic pulmonary injuries are commonly encountered injuries in the trauma patient and must be addressed early on to restore adequate ventilation and circulation.

1. Bronchoscopy remains the gold standard diagnostic modality for tracheobronchial injuries.
2. Small (<2 cm) partial thickness tracheobronchial injuries can be managed nonoperatively.
3. Extensive tracheal injuries require debridement to healthy tissue before repair.
4. In cases of hemothorax or hemopneumothorax, 14 French pigtail catheters are as effective as large bore chest tubes.
5. Massive hemothorax (>1500 mL of initial chest tube output or >200 mL/h for the first 3 h) warrants operative intervention.
6. Uncomplicated small volume (<300 mL) hemothorax can be observed.
7. If lung resection is necessary, preference remains to the tissue sparing techniques.
8. Choices of operative approach and repair technique are to be dictated by the injury type, injury location, and overall patient status.

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18 Transmediastinal and Thoracoabdominal Injuries: Damage Control and Surgical Techniques for Their Management

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Transmediastinal Injuries for Transmediastinal Injuries

It is difficult to envision how a missile can cross the midline of the chest without causing injuries to the vital structures of the cardiovascular, pulmonary, and gastrointestinal systems that reside within the tight confines of the mediastinum. In fact, the majority of missile wounds to the mediastinum are rapidly lethal because of exsanguination or cardiac tamponade [1]. Patients that do survive to reach definitive medical care often present with tachycardia, hypotension, large-volume tube thoracostomy output, or other dramatic signs of life-threatening mediastinal injury that mandate immediate exploration [2].

However, a significant percentage of patients present with occult injuries to mediastinal structures [3–6]. Delayed recognition and treatment of these injuries results in significantly increased morbidity and mortality [3, 7]. Thus, any patient in whom the missile trajectory crosses the midline with potential mediastinal violation requires rapid but deliberate evaluation.

Traditionally, this evaluation has consisted of chest radiography (CXR) in the emergency department (ED) followed byE-FAST and/or echocardiography or, in the past, subxiphoid pericardial window, aortography, esophagoscopy, or esophagography, and bronchoscopy [3, 7]. Some have even suggested mandatory exploration in this clinical scenario [2]. As has been pointed out, evaluation of the mediastinum is an invasive, labor-intensive, expensive, and time-consuming endeavor [8], with the consequent potential for clinical deterioration while the multistage evaluation proceeds.

Computed tomographic (CT) scanning is an increasingly useful tool, as speed and resolution improve with each subsequent generation of scanners and as trauma surgeons have gained greater experience with this modality. Recently, there have been several reports assessing the utility of CT scanning in the evaluation of gunshot wound trajectories [9, 10]. Although in the past trauma surgeons have previously relied on traditional, invasive evaluation of transmediastinal injuries [8], as time has evolved, a rapid evolution in our reliance on CT scanning for the evaluation of both blunt and penetrating injuries in general and thoracic injuries in particular.

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Over the last 30 years, there has been a remarkable change in the approach to penetrating trauma, from that of aggressive operative intervention to selective exploration of penetrating injuries [11]. This evolution has been driven by the significant morbidity associated with nontherapeutic interventions [12], concerns raised by overextended and at times untrained personnel, and mounting economic pressure to deliver cost-effective care [13]. This evolution in philosophy addresses both stab wounds and gunshot wounds [14] and includes all regions of the body [15]. As with therapeutic interventions, there has been a movement toward less-invasive diagnostic modalities. This also is driven by concerns for lowered patient morbidity and increased cost-effectiveness as well as by the improvements in noninvasive technologies and the accumulation of evidence confirming the efficacy of these technologies.

This evolution in diagnostics is perhaps most clearly exemplified by the approach to potential vascular injuries [16, 17].The use of CT scans for the evaluation of penetrating injuries is not new. It has been used for the assessment of both gunshot wounds and stab wounds of the back and flank for some time [18]. More recent reports also address the evaluation of anterior abdominal stab wounds as well [19, 20]. However, most of the reports regarding CT scans have focused on the diagnosis of specific organ injury. Only recently have studies appeared that specifically address the definition of missile trajectory and determination of body cavity penetration, yielding a secondary assessment of risk to organs along the missile tract [9, 10]. As early as 1981, Bryant [3] queried the potential application of CT scanning to mediastinal injuries.

In 1996, Fleming [8] anecdotally reported the use of CT scans for the assessment of missile trajectories in the chest. In 1998, Grossman [9] retrospectively reviewed the University of Pennsylvania experience with CT scan evaluation of truncal injuries, including 15 patients with gunshot wounds to the chest. In these 15 patients, CT scans provided definitive information regarding potential mediastinal injury for nine patients who required no additional evaluation. The remaining six patients underwent a limited evaluation of the mediastinum. In this study two thoracic injuries were detected: a subclavian artery injury and a pericardial vein laceration. Grossman [9] concluded that CT scan evaluation of thoracic gunshot wounds was a safe and effective screening tool to limit the use of invasive investigations.

Previous publications have questioned the traditional priorities of mediastinal injury investigation [8]. Nonetheless, we have evolved to rely on a variation of traditional, multimodality investigations of the mediastinal structures [21–23]. As recently as 1996, CT scan of the torso was little used for the evaluation of penetrating trauma in our practice. However, it was noticed shortly thereafter that there was a remarkably rapid increase in our reliance on CT scan to evaluate the hemodynamically stable patient with gunshot wound to the torso [22, 24, 25].

Hanpeter and Asensio prospectively analyzed the use of CT scans in this setting to verify the safety and efficacy of this change of practice [25]. The goal of the study was not to alter the therapeutic approach to mediastinal organ injuries but rather to streamline the diagnosis of these injuries in a select group of hemodynamically stable patients felt to be at risk of mediastinal injury by virtue of missile trajectories crossing the midline of the chest. Specifically, this study sought to define the findings of missile trajectory visible on CT scans, evaluate how easily and reliably CT scans can elucidate missile trajectory, assess the clinical utility of CT scans in decision-making regarding additional diagnostic evaluation and treatment, and determine whether the use of CT scans could decrease the reliance on more expensive, invasive, and time-consuming investigative modalities without an adverse effect on patient morbidity and mortality [25].

Although the literature is replete with studies describing injuries to individual organ systems, there are few studies that address the regional assessment of mediastinal injury. Without question, injuries to the heart or great vessels are highly lethal [1, 26]; and patients who survive to reach definitive medical care usually present with tachycardia, hypotension, or large volume hemothoraces [2, 27].

Few cardiac and major vascular injuries present in a hemodynamically stable condition. Aerodigestive tract injuries occur in association with cardiovascular injuries, with the presenting symptoms dominated by the cardiovascular injury. Isolated aerodigestive injury may well present in an occult fashion, but such isolated injuries are infrequent; and published reports in general describe rather small series of patients. Because of the paucity of publications addressing mediastinal injuries, it is entirely unclear what percentage of patients succumb in the field, how many arrive in unstable or in stable condition with occult injuries, and how many escape any significant injury. It seems certain that the patients with a true mediastinal traverse but without clinically significant organ injury constitute a minority of mediastinal injuries.

Although it would be interesting to answer these questions regarding outcomes with true mediastinal missile trajectories, the issue of real clinical relevance is the evaluation of these patients who would on bedside evaluation appear to be at significant risk of having a missile tract that traverses the mediastinum. This population, as defined by Richardson [3], is comprised of patients with missile entry and exit wounds or missile entry wounds and retained missiles localized on radiography in opposite hemithoraces. We have expanded Richardson's definition slightly by including patients in whom the missile overlies the mediastinal silhouette on chest radiographs.

The significant morbidity and mortality and the delayed recognition of mediastinal injuries mandate that all patients at risk of a mediastinal injury undergo prompt evaluation of all mediastinal structures.

In Hanpeter and Asensio's study [25], patients sustaining gunshot wounds to the chest crossing the midline who did not have clinical indications for immediate operative intervention, such as hypotension, signs of pericardial tamponade, significant ongoing blood loss from chest tubes, or need for immediate cervical or abdominal exploration underwent thoracic evaluation with helical CT scans of the chest. CT scans were evaluated by the in-house surgical staff, examining the films for the findings of missile tracts (Table 18.1). Of 24 patients who met entry criteria, nine required operative intervention (Table 18.2) [25].

One patient underwent sternotomy and cardiorrhaphy to remove a missile embedded in the

Table 18.1 CT scan findings to delineate missile trajectory and organ injury

Chest wall
Skin violation
Subcutaneous fat stranding
Soft tissue air or hematoma
Scapular, sternal, rib, or vertebral fracture
Soft tissue missile or bone fragments
Pulmonary parenchymal contusion
Mediastinal organ injury
Mediastinal air
Contrast extravasation
Missile location

Table 18.2 Studies performed in addition to CT scan and operative interventions required within each group

Additional evaluation	Patients (no.)	Interventions
No additional studies	12	Sternotomy for myocardial missile
		Thoracotomy for persistent pulmonary parenchymal bleeding
		Laparoscopic evaluation of left hemidiaphragm
		Exploratory laparotomy for diaphragm, stomach, liver, spleen
		Exploratory laparotomy for diaphragm, small bowel
Angiography, esophageal evaluation, bronchoscopy	1	Thoracotomy for persistent intercostal bleeding
Angiography, esophageal evaluation	5	Exploratory laparotomy for small bowel
Angiography alone	2	Sternotomy for para-aortic missile
		Thoracoscopy for retained hemothorax
Esophageal evaluation alone	3	None
Bronchoscopy alone	1	None

myocardium diagnosed by CT scan, and a second patient underwent sternotomy and removal of a missile abutting the pericardium overlying the aortic root confirmed by angiography after CT scan. Two patients underwent thoracotomy for persistent intrathoracic bleeding, and one patient had thoracoscopic evacuation of a retained hemothorax on the basis of the CT scan. Three patients required laparotomy for intraabdominal injury, and one patient had negative laparoscopic examination of the left hemidiaphragm. In summary, two of 24 patients (8.3%) sustained mediastinal injury requiring operative intervention.

In contrast, Richardson [3] reported a 63% incidence of significant injury in hemodynamically stable patients. Of 43 patients that were initially stable, three became unstable during evaluation and 24 had significant findings on evaluation requiring surgical intervention. Injuries included: 26 pulmonary, 11 great vessel, nine esophageal, six cardiac, and six tracheobronchial injuries. The reason for the marked difference in frequency of significant injuries is unclear, because the definitions of potential transmediastinal gunshot wounds would seem to be similar in Hanperter and Asensio's study compared to Richardson's.

In Hanpeter and Asensio's study of 24 patients with suspicious injuries, 12 patients avoided any additional diagnostic evaluation [25]. One patient required complete diagnostic evaluation with angiography, bronchoscopy, and esophagography. The remaining 11 patients underwent a limited evaluation of the mediastinum, as directed by CT scan results. Overall, 16 patients (67%) were spared aortography and 15 patients (63%) were able to avoid esophageal evaluation. In this study of 24 patients, no injuries were missed. During the course of this study, several observations were made. Although the missile entry and exit sites are usually identifiable on CT scan by the means outlined above, they are occasionally indiscernible. This is most often attributable to subcutaneous dissection of air from pneumothoraces or tissue plane disruption caused by chest tube insertion. In addition, some missile tracts leave few telltale signs, or the site may lie between cuts of the CT scan.

Radiographic identification of chest wall violation is most easily facilitated with the marking of these sites with a small metal object taped to the site, such as a or paper clip. Similarly, trajectory in proximity to the esophagus on occasion can be difficult to assess because of the small profile of the collapsed esophagus. This is made easier by routine placement of a nasogastric tube to unambiguously identify the esophagus on CT scan. Intravenous contrast is, of course, mandatory. Because missile trajectory is more clearly identified within the pulmonary parenchyma than in chest wall soft tissues and because the pulmonary parenchyma is immediately adjacent to the mediastinum, this aspect of the CT scan missile tract is the most important evaluation [9, 24].

However, in Hanpeter and Asensio's study the authors found that in several patients, the pulmonary parenchymal tract may be completely obscured by large pulmonary contusions. Large contusions may be attributable to a large amount of damage from the primary injury, injury from multiple missiles, or evolution of the contusion because of significant time delay from time of injury to time of CT scan. Therefore, a large pulmonary contusion on chest radiograph may predict failure of the CT scan to accurately identify missile trajectory [25].

Thus, Hanpeter and Asensio concluded helical CT scan in the evaluation of patients with potential transmediastinal gunshot wounds can frequently elucidate missile trajectory. In one-half of these patients, CT scans eliminated the need for additional evaluations. In the other half of these patient populations, CT scans facilitated a limited, organ-specific evaluation of the mediastinum. Overall, the use of CT scan permitted a two-thirds reduction in labor-intensive, expensive, time-consuming, invasive radiographic evaluation of the mediastinal structures. The authors further concluded that CT scans have proven to be a safe, effective, and readily available tool to facilitate rapid evaluation of potential transmediastinal gunshot wounds. This seminal study set the tone for the future evaluation of stable trasmediastinal injuries [25].

Damage Control

Damage control as a surgical concept and/or technique has evolved over the past 25 years to become a mainstay of the trauma surgical armamentarium. Given its importance, it is meritorious to review its origins and indications. While the physiologic entity of the lethal triad (acidosis, hypothermia, and coagulopathy) has always existed, the surgical management of the bleeding diathesis in trauma did not change until Stone's hallmark work describing the "Bailout" approach [28–30]. His 1986 seminal paper recognized a physiological "cluster" of intraoperative signs, i.e., coagulopathy, prompting interruption of trauma surgical procedures after institution of hemorrhage containing measures and packing of the abdominal cavity [28]. Following the interrupted laparotomy, patients were transported to the intensive care unit setting where resuscitative measures, correction of coagulopathy, hypothermia, and acidosis took place prior to return to the operating room for definitive surgery.

This "Bailout" approached ushered the area of staged surgical procedures for trauma. With this approach, Stone [28] reported a 65% versus 7% survival rate in favor of patients with abdominal packing versus those undergoing definitive surgical management. In 1992, Burch described the abbreviated laparotomy with planned reoperation for critically ill patients recognized to have the lethal triad showing a similar 67% survival rate [31].

Later described in 1993 as "Damage Control" by Rotondo [32], this manuscript outlined a methodology for the management of critically injured trauma patients. In this study consisting of 46 patients, the authors identified a maximum injury subset of 22 patients, of which nine underwent definitive laparotomy (DL) and 13 damage controlled laparotomy (DCL). In this group of patients, survival rate for the damage control group was 77% versus an 11% survival rate for the definitive laparotomy (DL) group. It should be noted that in Rotondo's study recommendations were made on the basis of 22 patients described as the maximum injury subset. Furthermore, the study did not have any statistical analysis.

In reality, damage control as a methodology emerged to deal with exsanguination, an ill-defined, easily recognized, feared entity; but not foreign to trauma surgeons. Initial attempts by Anderson to define this syndrome: " Patients losing their entire blood volume" and Trunkey who described it within the context of flow, defined outcomes for patients with severe hemorrhage and rates of blood loss exceeding 250 mL/min [33, 34]. Asensio and Ierardi later rethought and redefined this syndrome and described it as: "Exsanguination is the most extreme form of hemorrhage. It is usually caused by injuries to major components of the cardiovascular system, injuries to parenchymatous organs or both. It is a hemorrhage in which there is an initial loss of 40% of the patient's blood volume with an ongoing rate of blood loss….. Exceeding 150 mls per minute. If this hemorrhage is not controlled, the patient may lose over half of his or her entire blood volume within 10 minutes." [35–37]

While many had previously described the lethal triad, Moore [38] subsequently described "The Bloody Vicious" cycle of acidosis, hypothermia, and coagulopathy, Cosgriff postulated that prediction and correction of the coagulopathy was perhaps the most critical decision-making component of the "Bloody Vicious Cycle." Asensio and colleagues later went onto describe statistically validated indications for the institution damage control and described predictors of outcome based on a study of 548 patients presenting with exsanguination. All of their patients were admitted with very low revised trauma scores (RTS) and very high injury severity scores (ISS) consisting of thoracic, abdominal, and multiple injuries in critically injured patients with profound shock with a mean admission pH of 7.15 and a mean estimated blood loss of 7.3 L [39–41].

The "Lethal Triad" was later expanded by Asensio [39] to include cardiac dysrhythmias and described as the "Lethal Tetrad." While the indications for DCL described by Asensio have been universally adopted, validated, and instituted for abdominal injuries. However, no study has sought to apply them solely for the management of cardiothoracic injuries.

Damage control is now considered standard in the management of exsanguinating abdominal trauma. However, its uses have been recently scrutinized and its frequency has decreased especially with the advent of hemostatic resuscitation. Its use has found a much smaller place in the management of exsanguinating cardiothoracic trauma with abbreviation of surgery in order to restore the patient's deranged coagulopathic and biochemical abnormalities. Thoracic trauma, though, is frequently and rapidly lethal with definitive surgical repair required if the patient is to survive. When controlled, implementation of damage control techniques for cardiothoracic injuries in order to restore patient physiology can have benefits outcome wise [42–45].

Damage control in cardiothoracic surgery (DCS) has been limited by several factors. First, cavitary packing, a hallmark of DCL, when applied in the thorax may impair cardiac filling and impede pulmonary expansion. Second, while abbreviated thoracotomy as a damage control technique consists of rapid hemorrhage control requiring a return to the OR, there is no clear definition of damage control in the thorax. Lastly, while the literature describes the operative management of specific thoracic injuries there is a paucity of physiologic data or outcomes [8, 44–48].

Patients most likely to require damage control thoracotomy (DCT) are unstable and have sustained penetrating thoracic injuries from gunshots (GSWs), stab wounds (SWs), or rarely shotgun wounds (STWs). While there are obvious differences between DCL and DCT, the decision to perform either must be based on the patients altered physiology. In both cases, acidosis, hypothermia, and coagulopathy are individual predictors of mortality [39, 49]. These physiologic derangements as well as control of the exsanguinating hemorrhage must be considered important selection criteria [39]. The divergence between cardiothoracic and abdominal surgery lies, from the standpoint of damage control, in the inherent anatomic differences between these two cavities [50].

One of the most important principles of DCL involves not only the control of hemorrhage but control of contamination from gastrointestinal track injuries which may affect the vascular repairs. This complication is less of a concern in the thorax as esophageal injuries are rare and thus decreases the risk of contamination and complications there in. Another important difference is that almost all abdominal and retroperitoneal injuries can be accessed from a single midline laparotomy incision, whereas the thorax is compartmentalized and thus necessitates different incisions to afford optimal access, exposure, and visualization of specific injuries.

Therefore, DCT depends on the anatomic location of the injury. Left anterolateral thoracotomy allows rapid access to the left hemithoracic, pericardium, heart, and the thoracic aorta, whereas median sternotomy provides optimal exposure to the heart, mediastinum, and injured vessels. Extension of this incision as, bilateral anterolateral thoracotomies or "clamshell thoracotomy," has also been used. Regardless of the approach, it cannot be overemphasized that the incision must provide adequate exposure to all injuries [51, 52].

Damage Control in the Trauma Center for Transmediastinal Injuries

The trauma center and specifically the trauma bay is the first area to assess and manage exsanguinating trauma patients. It is here where injuries are first identified and triaged. Addressing the ABCs of penetrating thoracic trauma are unique in that they are both therapeutic and diagnostic within this setting. Survival from this point mandates an expeditious approach to injury, trajectory identification, management of exsanguination, and surgical decision-making. Inherent to this process in the trauma bay is strict adherence to the principles of advanced trauma life support (ATLS) where definitive airway control and placement of large bore intravenous access for rapid blood product are paramount. Indication for activation of massive transfusion protocols (MRP) is based on the patients admitting vital signs which include tachycardia, hypotension, penetrating injuries, and positive focused abdom-

inal sonography in trauma (FAST) or E-Fast exam findings. These techniques aid decision-making and early and rapid release of blood products. Placement of thoracostomy tubes is both therapeutic and diagnostic. Arterial blood gases (ABGs) and lactic acid levels are also important.

Radiography can aid in determining trajectory and therefore identify potential injuries. A chest X-ray at a minimum can be obtained in many patients prior to OR unless more aggressive maneuvers such as an emergency department thoracotomy (EDT) is undertaken (Fig. 18.1). Thoracostomy tube output, E-FAST in both pericardial and pleural views, as well as chest radiographs (CXR) should be sufficient to establish the diagnosis of injuries requiring immediate surgical intervention.

EDT involves rapid access to the left hemithorax through an anterolateral thoracotomy in order to control hemorrhage, relieve cardiac tamponade, institute resuscitation, or repair cardiac injuries until these patients can be rapidly transported to the operating room (OR). EDT also serves as a triage maneuver and can identify non-survivable injuries prior to transport to OR, thus ensuring that patients with non-survivable injuries are not unnecessarily transported to the OR. Performance of EDT must be based on strict criteria as it is associated with a significant exposure risk [53]. The indications for EDT have been well described, thus the decision to risk exposure and perform this maximally invasive procedure should be driven by the potential for survival [54, 55].

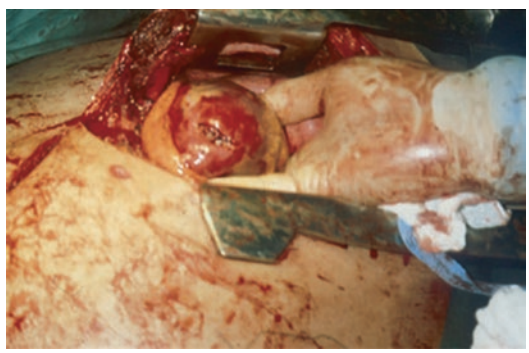

Fig. 18.1 Left anterolateral thoracotomy for gunshot wound in the left ventricle

Decision-making for EDT is based on the presence or absence of signs of life on the scene and on arrival to the trauma center as well as presence or absence of a carotid pulse. Those with signs of life in the field and on arrival and lose their pulses in the trauma bay have the greatest chance of survival and extreme efforts such as EDT should be made to save these patients. Following this scenario, those with signs of life in the field but no signs of life or pulse on arrival constitute the most difficult patients in the process of decision-making. Chances of survival in this group are approximately 8% after pooled analysis [56]. Patients presenting without a pulse after sustaining blunt trauma have extremely low survival rates and thus EDT is not recommended [56, 57].

The primary goals of EDT are the same in damage control: rapid control of hemorrhage, control of potential sources of air embolism, release of pericardial tamponade, and cross-clamping of the aorta to redistribute blood flow to the coronary and carotid arteries in order to maintain cardiac and cerebral perfusion [52, 58, 59]. The procedure begins with an anterolateral thoracotomy incision which starts at the left sternocostal junction in the fifth intercostal space and extending to the anterior border of the latissimus dorsi (Fig. 18.1). In females, the left breast is displaced cehpalad to prevent injury to the breast.

The skin, subcutaneous tissues, and chest wall musculature are rapidly transected with a #10 or 21 scalpel. A small incision is made through the intercostal muscles to enter the thoracic cavity, followed by complete transection of the three layers of the intercostal musculature with Metzenbaum thoracic scissors. Simultaneously, a right side thoracostomy tube must be inserted for identification of right sided hemorrhage and should prompt exposure of the right hemithorax through a bilateral anterolateral thoracotomy incision if significant bleeding is identified. This is known as a "clamshell thoracotomy." A Lebsche knife or Bethune shears can be used to divide the sternum if extension is required for better visualization of thoracic structures. A Finochietto Rib Retractor is then placed and positioned with the handle toward the table to facilitate sternal division if indicated.

Upon entrance into the thoracic cavity, the trauma surgeon should note the presence of arterial or venous blood as well as the presence of a bulging pericardium indicating the presence of tamponade. This signifies a potential cardiac injury and release of the tamponade is often lifesaving. The lung is then retracted anteriorly to allow for better exposure of the aorta. This is done by placing the left hand posterior and lateral to the lung with the palm against the parenchyma. Using Metzenbaum scissors, the mediastinal pleura is then divided immediately anterior to the aorta, avoiding injury to the esophagus. Placement of a nasogastric tube following intubation can aid in identification of the esophagus (Fig. 18.2). Division of the mediastinal pleura enveloping the aorta is particularly important; if not done properly, cross-clamping of the aorta may be ineffective. The space between the esophagus and the aorta is developed digitally and a Crafoord-DeBakey Aortic Clamp is placed (Fig. 18.3).

If present, pericardial tamponade is released by incising the pericardium with a scalpel anterior and parallel to the phrenic nerve which must be preserved. The incision is then extended with Metzenbaum scissors to the root of the Ascending Aorta. Extension from the root of the aorta to the apex of the heart allows for complete delivery of the heart into the field for repair and open cardiac massage [60, 61]. The presence of an air embolus in the coronary vasculature is an ominous finding and a negative predictor of outcome (Fig. 18.4). This finding is very rare and can only be observed in the coronary vein [62].

Once these techniques have been implemented and open cardiopulmonary massage has commenced, attention should be turned to ensuring adequate resuscitation and correction of acidosis and hypocalcemia. Additional procedures may be indicated based on injuries and the physiologic status of the patient. Hemorrhage from cardiac injuries is controlled with digital occlusion prior to performing either atrial or ventricular cardiorraphy with 2–0 polypropylene sutures on an MH needle [51, 52, 63–65]. Initial management of pulmonary injuries begins with mobilization of the lung by sharply dividing the inferior pulmonary ligament meticulously so as to not injure the inferior pulmonary vein.

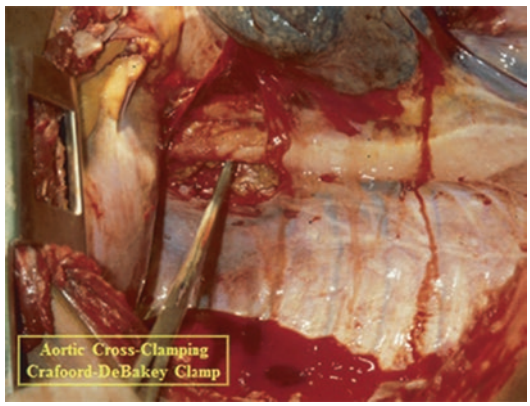

Fig. 18.2 Resuscitative thoracotomy on a patient that succumbed. Notice the left hemithoracic cavity which can harbor the entire blood volume. Thoracic aorta is dissected. Esophagus is above

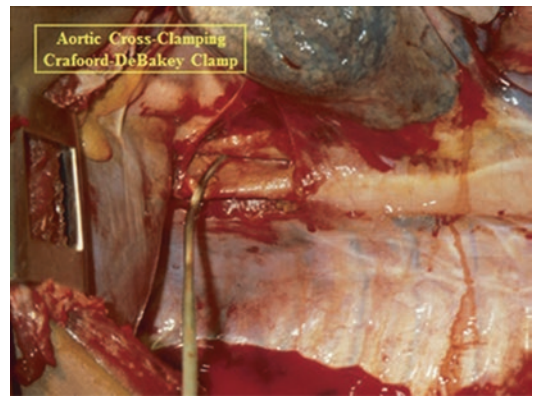

Fig. 18.3 Resuscitative thoracotomy on a patient that succumbed. Descending thoracic aorta has been clamped

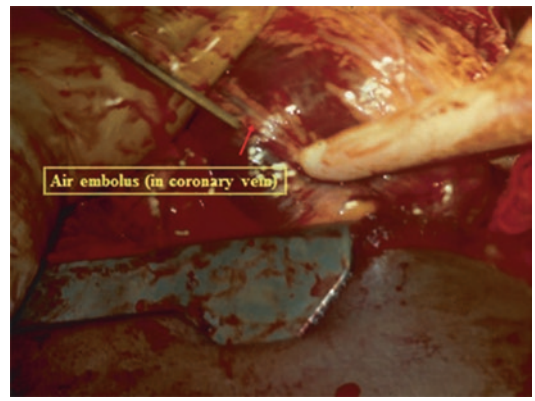

Fig. 18.4 This is an ominous finding

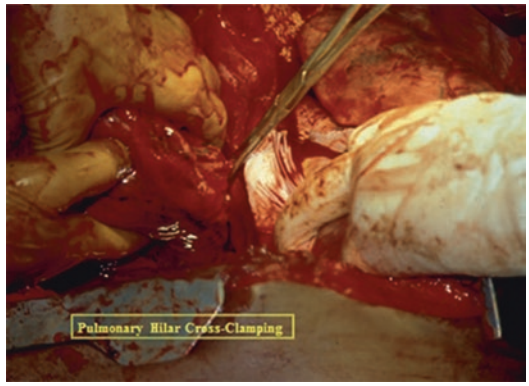

Fig. 18.5 Cross-clamping pulmonary hilum

Hemorrhage control and prevention of air embolism can be achieved by using Duval clamps to compress the injured pulmonary parenchyma. Injuries to major venous structures can be controlled with Satinsky partial occlusion vascular clamps. If a central hilar hematoma is encountered, rapid dissection is instituted and a Crafoord-DeBakey Aortic cross clamp is placed across the pulmonary hilum (Fig. 18.5). Arterial injuries can be controlled with both Satinsky or DeBakey Vascular clamps. The goal is to control rather than institute definitive repair. When controlled, expedient transport to the OR is based on the patient's ability to regain and maintain an organized and perfusing cardiac rhythm and pulse.

As mentioned above, this life-saving technique is not without risk and pitfalls. Overall, survival remains low and patient selection is critical. Asensio, Wall, and others produced the Practice Management Guidelines of the American College of Surgeons (ACS) Committee on Trauma (COT), described indications and outcomes for EDT [50, 66–68]. An overall survival rate from EDT for survivors was 7.83%. When stratified based on mechanism of injuries, 11.6% and 1.6% survival rates were noted for penetrating and blunt trauma mechanisms, respectively. More recently, Seamon reviewed and provided Practice Managements Guidelines for the Eastern Association for the Surgery of Trauma (EAST), similarly noting an overall survival of 8.5%, with 10.6% and 2.3 survival rates for both penetrating and blunt injuries, respectively [56, 58, 68–71]. These must be taken into consideration of exposure risks, which are noted to be greater than 7% for those involved in EDT and by the reported HIV seropositivity rates as high as 4% in some urban trauma centers.

Cardiothoracic Damage Control in the Operating Room for Transmediastinal Injuries

Overall Considerations

If the patient is able to regain and maintain an organized and perfusing cardiac rhythm, they are transported immediately to the OR where hemorrhage control and damage control techniques are implemented. Speed is paramount. Patient positioning and prepping should follow standard protocols with prepping from the chin to knees in order to access both the thoracic and abdominal cavities. Appropriate thoracic instruments should be available (Fig. 18.6). In the case of EDT, MTP should have been initiated in the Trauma Bay and extended to the OR where a balanced hemostatic resuscitation with 1:1:1 (packed red blood cell:fresh frozen plasma: platelet) ratio of blood products should be carried out with guidance from intraoperative viscoelastic hematologic studies such as thromboelastography (TEG) or rotational thromboelastography (ROTEM) [72–76].

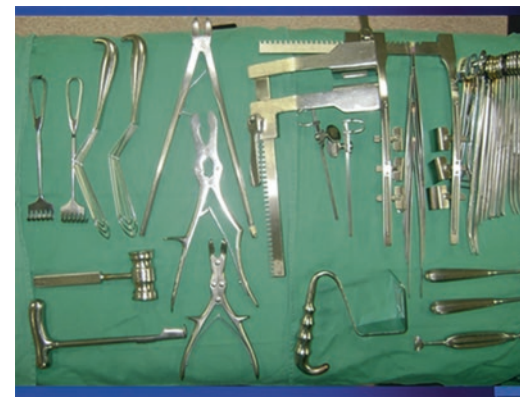

Fig. 18.6 Thoracic surgical instruments

Crystalloids must be minimized to avoid accelerating coagulopathy [75, 77]. Additional warming devices are used to minimize heat loss and facilitate rewarming in the hypothermic patient. Use of autotranfusion should be utilized given its low risk of contamination, however, consideration should be given to the fact that the autotransfused blood from hemothoraces contains less coagulation factors specifically fibrinogen (f II) [78–80]. Placement of invasive hemodynamic monitoring devices such as arterial and large bore central venous access lines is of the utmost importance. Exchanging to a dual lumen endotracheal tube (ETT) is usually not imperative, feasible, or recommended. If improved visualization is required, a bronchial blocker may be placed into the mainstem bronchi of the desired lung to facilitate unilateral deflation.

Cardiac Injuries: Technical Aspects for Repairs and Outcomes

Cardiac injuries are particularly lethal and mandate immediate hemorrhage control. Following EDT in the trauma bay, initial digital control is required to prevent further exsanguination once the injury is recognized. This is followed by primary repair in the majority of cases. Atrial injuries can be controlled by a Satinsky partial occlusion Clamp and primarily repaired with 2–0 or 3–0 polypropylene monofilament sutures on an MH needle with horizontal mattress sutures of Halsted. Ventricular injuries can also be dealt with in a similar fashion. In the case of destructive gunshot wounds, the use of Teflon plegets may be warranted (Fig. 18.7). Control in the Trauma Bay setting does not have to be definitive and a pledgeted repair if deemed necessary can be performed in the more controlled setting of the OR.

The technical demands of suturing a beating heart are obvious. Entry of the needle perpendicular to the tissue with wrist supination following the curve of the MH needle so that exit of the tissue occur exactly opposite entry is necessary. Several techniques have been described

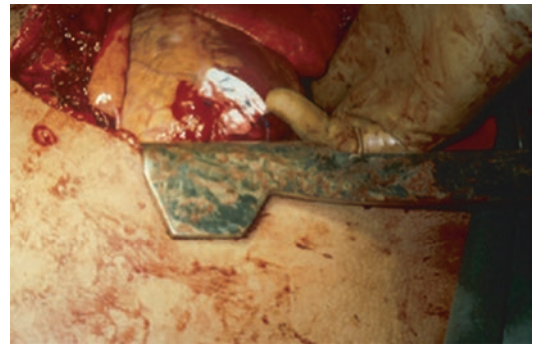

Fig. 18.7 Lefat anterolateral thoracotomy revealing a AAST-OIS Grade V Cardiac Injury secondary to gunshot wound (GSW) successfully repaired. Left Ventricular Cardiorraphy with Teflon Strips

that reduce the risk of lacerating the myocardium or aggravating a concurrent injury [51, 64, 65, 81].

Regarding coronary vessel injuries, distal injuries compose the majority of survival injuries that survive to reach the OR. Distal coronary vessel injuries are amenable to ligation. The trauma surgeon must be aware of the potential for the development of intraoperative infarction and likewise must be aware of the potential development of ischemia in the post-operative setting. Proximal coronary injuries are rare, mostly lethal, and those very rare to survive to the OR will require aorto-coronary artery bypass with a reverse autogenous saphenous vein graft (RSVG). In this setting, cardiopulmonary bypass is required [51, 64, 65, 82, 83]. These injuries are also extremely rare and rarely reported in the literature.

For injuries of the atrio-caval junction and the lateral most portion of the right atrium, total inflow occlusion is indicated to allow for proper repair. Total inflow occlusion of the artio-caval junction is achieved by cross-clamping both the superior and the inferior vena cavae at the space of Gibbon's (insertion of the IVC in the right heart) (Shumacker's maneuver) (Fig. 18.8). Cardiac arrest will ensue allowing a brief window of time to perform repairs. However, this window of time is only 1–3 min, if exceeded, as Asensio has previously warned; re-establishment of a sinus rhythm is improbable.

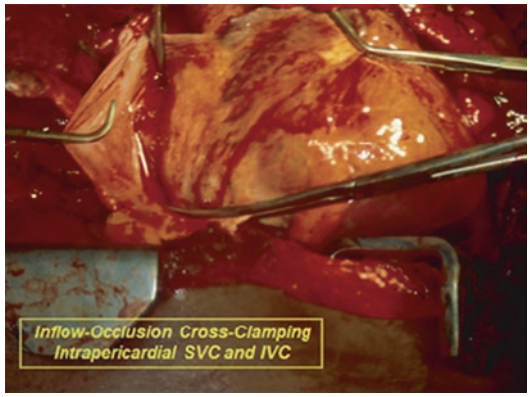

Fig. 18.8 Shumacker's maneuver

When repair is complete, closure of the pericardium is not recommended to avoid the re-creation of tamponade physiology. Swelling of the heart following "the stunned myocardium syndrome" and re-perfusion injury occurs following this injury and pericardial closure can restrict cardiac filling in this setting. Following repair, a transesophageal echocardiogram is recommended to rule out any septal injury from the penetrating missile.

Penetrating cardiac injuries are uncommon and lethal. The majority of these patients succumb at the scene of the traumatic incident. Those that survive to arrive at a trauma center present with impending or cardiopulmonary arrest and incur high mortality. Evaluation of data in the literature reveals favorable outcomes in selected studies, for these injuries. Close scrutiny of these series reveals many to be retrospective and overlapping. Frequently, there is a lack of reported data describing the initial physiologic status upon arrival of these patients. No series has graded cardiac injuries utilizing the American Association for the Surgery of Trauma Organ Injury Scale (AAST-OIS). Similarly, there is a lack in reporting independent predictors of outcomes. To the best of our knowledge, there are only three prospective cardiac injury series in the literature providing statistical analysis of predictors of outcomes and only one validating the AAST-OIS cardiac injury scale [52, 64, 65].

Cardiac injuries remain uncommon, and thus few trauma centers and trauma surgeons have developed significant experience with these injuries. This is validated by the paucity of series reported in the recent literature. A search of studies on penetrating cardiac injuries for the past 20 years yielded 323 publications. This search revealed that the last large series were published between 1998 and 2000, while the remaining series consisted of small studies and single case reports describing unusual wounding agents. Since 2004 there have been at least three series, reporting between 64 and 117 patients whose injury mechanism was mostly stab wounds [46, 83–86].

There are currently three prospective cardiac injuries series in the literature. The first reported by Buckman and Asensio analyzed factors influencing their initial resuscitation. This 27 month study included 66 patients evaluated with the cardiovascular respiratory score (CVRS) component of the trauma score (TS), 70% sustained GSWs, and 71% of the patients required an emergency department thoracotomy (EDT) and for the first time validated the CVRS as a physiologic predictor of outcome with reported survival rates of 20% for GSWs and 80% for SWs, respectively [87].

Subsequently, Asensio reported the second prospective study in the literature consisting of 60 patients admitted in a 1-year period, validating the CVRS score, and statistically validating the physiologic conditions of these patients in the field, during transport and upon arrival; reported a 68% incidence of GSWs and an overall survival rate of 36.6%. Stratified to mechanism of injury the survival rate for GSWs was 14% and SWs was 68%, respectively; with an EDT survival rate of 16%. This study did not accumulate sufficient number of patients to perform stepwise logistic regression to generate a predictive model [65].

In the third prospective series in the literature, Asensio reported a 2-year study consisting of 105 patients with an incidence of 65% GSWs and a survival rate of 33% [52]. In this series 71 (68%) of the patients required EDT with a survival rate of 14%. Survival stratified to mechanism of

injury was 16% for GSWs and 65% for SWs. This study also prospectively validated the CVRS score and the physiologic condition of patients in the field, during transport and upon arrival as predictors of outcome. It graded cardiac injuries utilizing the AAST-OIS for cardiac injuries, stratifying mortality rates for each injury grade and per cardiac chamber injured. Of the 105 patients 99 (94%) of the patients sustained grade IV–VI injuries. Stepwise logistic regression identified GSWs, exsanguination, and restoration of blood pressure to be the most independent predictive variables for mortality. This model had an overall predictive power of 95% [52].

The most recent retrospective study was published by Soto in 2015, reporting 135 patients from a Level II Trauma Center over the span of 15 years, from an institution receiving approximately nine cardiac injuries per year [88]. Of these 135 patients, 96 (71%) did not survive past the ED; and 89 of these 96 (93%) patients were considered DOA (dead on arrival), thus leaving 39 patients to undergo thoracotomy. This small series reports similar survival data reported in the only three prospective cardiac injury series in the literature.

Difficulties exist in evaluating the results of series reported over the past three decades. Over 30 series have been reported in the English language literature [51, 52, 64, 65, 82, 86, 89–128]. Close scrutiny reveals that most series are retrospective, reporting small volumes with many serial and overlapping studies from selected institutions. Although some of these selected studies have reported favorable outcomes, many provide no data on the physiologic conditions of these patients upon presentation, nor do they report statistically validated predictors of outcome. Unfortunately, none of these series have graded these injuries utilizing the AAST-OIS cardiac injury scale.

Asensio in 2018 examined the national profile of cardiac injuries to identify and validate independent predictors of outcome, create a more robust predictive model, and compare and validate previous predictive models determining outcomes [84]. Asensio hypothesized that the NTDB, with its large number of patients would allow the creation of such predictive model and validate current models. The other objectives were to report the incidence of these injuries and overall survival and mortality rates.

Upon review of the NTDB, there were 2016 patients identified from a total of 1,310,720 patients. The national incidence of penetrating cardiac injuries is estimated as 0.16%. This does not take into account that many patients sustaining cardiac injuries succumb at the scene of the traumatic incident prior to transport. This data does not reflect penetrating cardiac injury deaths in non-trauma centers that do not submit data to the NTDB. Breakdown according to gender includes 1203 males (59.4%) and 813 females (40.8%). Mean age for this patient population is 38 ± 23. Prehospital procedures performed include administration of intravenous fluids in 1689 (83.8%) and thoracic needle decompression in 267 (13.3%) [84].

There were a total of 1264 (63%) gunshot wounds (GSWs), 717 (36%) stab wounds (SWs), and 19 (0.9%) patients with impalement injuries, while 16 (0.8%) sustained shotgun wounds (SGWs). The mean RTS was 1.75 ± 3.22 (range 0–7.84), mean ISS 27.19 ± 23 (range 1–75), mean admission systolic blood pressure (SBP) (mmHg) 42.72 ± 5.03 (range 0–150), mean admission diastolic blood pressure (DBP) (mmHg) 29.26 ± 34.29 (range 0–88), ED heart rate (HR) 58.71 ± 71.82 (range 0–238) beats/min, and mean respiratory rate (RR) was 8.53 ± 10.69 (range 0–68), mean temperature was 35.4 ± 0.09 °C (range 33.1–36.4), while the mean GCS was 5.7 ± 4.89 (range 3–15) [84].

Of the 2016 patients, 212 (10.5%) were pronounced dead upon arrival, 1804 (89.5%) survived to receive further management. Of these, 830 patients underwent ED thoracotomy with 47 (5.7%) survivors. The remaining patients succumbed in the ED immediately post resuscitative thoracotomy; while the remaining 974 (54%) survived to reach an operating room (OR) to undergo OR thoracotomy (ORT) 628 (64.5%) survived (see Fig. I). There were 675 (33%) survivors. Survival stratified by mechanism of injury: GSWs 114/1264 (10%), SWs 564/717 (76%).

The mortality rate of patients with RTS = 0 was 94% versus those with RTS ≥ 1–38.6%. The mortality for GSWs was 90.6% versus SWs—23.8%. Those requiring EDT had a mortality rate of 94.3% versus reaching the OR to undergo ORT—35.6%. For patients requiring aortic cross-clamping, the mortality rate was 94.3%.

There were statistically significant differences between survivors and non-survivors including initial vital signs, GCS, RTS, and ISS ($p < 0.0001$). Other statistically significant predictors of outcome for mortality included need for prehospital cardiopulmonary resuscitation (CPR), need for emergency department intubation, ED thoracotomy (EDT), and aortic cross-clamping ($p < 0.001$).

Stepwise logistic regression identified GSW ($p < 0.001$; AOR 26.85; 95% CI 17.21–41.89), field CPR ($p = 0.003$; AOR 3.65; 95% CI 1.53–8.69), absent spontaneous ventilation ($p = 0.008$; AOR 1.0, 95% CI 1.02–1.14), need for an ED airway ($p = 0.0003$ AOR 1386.30; 95% CI 126.0–15,251.71), and need for aortic cross-clamping ($p = 0.0003$ AOR 0.18; 95% CI 0.11–0.28) as independent predictors of outcome for mortality. Overall predictive power of model: 93%. Complications among the survivors ranged from 1.4 to 6.5%.

Penetrating cardiac injuries are thus uncommon and highly lethal. The majority of patients succumb at the scene of the traumatic incident. Because of rapid transport, many generally arrive at urban trauma centers either in impending or cardiopulmonary arrest.

In Asensio's analysis of 2018 patients from the NTDB, the incidence of penetrating cardiac injuries has estimated at 0.16%. Stratified to mechanism of injury, GSWs account for 63%. This is consistent with series which range from 65 to 70% and also with the study reported by Soto of 72%, whereas the incidence of SWs is 36%, again consistent with data reported by Buckman, Asensio, and Soto, ranging from 28 to 35%. Series by Gao, Rodrigues, and Topal, however, report the majority of their patients sustained stab wounds ranging from 61 to 87%.

The mean RTS of 1.75 ± 3.22, ISS 27 ± 23, and GCS of 5.70 ± 4.89 is consistent with data reported by the three prospective series in the literature. The series by Soto reports a mean ISS of 53; however, 96 (93%) of their 135 patients were DOA [88, 129]. Series by Gao, Rodrigues, and Topal do not report these data [128, 130, 131]. The low RTS, ISS, and GCS, which is an indirect indicator of perfusion, reveal a severely physiologically compromised population with a high degree of anatomic injury.

Physiologic condition of patients upon arrival proved to be statistically significant between survivors versus non-survivors; some of these parameters have been confirmed in previous series [52, 65, 87, 128, 130–135]. The CVRS score could not be calculated for the 2016 patients in Asensio's latest series. Similarly, it is known that the need for field CPR and EDT are also strong predictors of outcome. Aortic cross-clamping, however, has only been statistically proven as a predictor of outcome in two series. In this study the need for EDT and aortic cross-clamping were strong predictors of outcome [84].

Stepwise logistic regression confirmed a larger number of strong independent predictors of outcome, which include presence of an associated abdominal GSW ($n = 6$), cardiac gunshot wounds, need for field CPR, absence of spontaneous ventilation, need for ED airway, and need for aortic cross-clamping as independent predictors of outcome with a predictive power for this model of 93% [65]. These data are consistent with another series whose logistic regression model identified a triad ($n = 3$) consisting of gunshot wounds exsanguination and restoration of blood pressure as the strongest independent predictors for mortality in a model with a max-rescaled R^2 of 0.81 and a predictive power of 95%. No doubt, the larger number of patients in the NTDB allowed other independent predictors of outcome to emerge and be validated.

The overall survival rate of 33% is comparable to the largest prospective series in the literature [65]. Survival rate stratified to mechanism of injury GSWs 10% is slightly lower than reported by Asensio [52, 65] 14–16% but within statistical range. Whereas survival for SWs 76% in this study is slightly higher than previously reported 65–68%, again, within statistical range, and is

similar to that reported in Buckman's prospective series 80% [87]. ED thoracotomy survival rate for this series is 5.7% versus Asensio survival rate of 14–16%; this is lower, but may be accounted by the fact that in these two series there was one group of trauma surgeons performing these procedures under strict protocols [51, 52, 65].

From this study, predictors of outcomes were identified. Overall survival rates are lower than most retrospective studies reported. Data suggests that patients can be selected for salvage and outcomes predicted. Decisions must be undertaken to direct salvage efforts including ED thoracotomy to patients presenting with signs of life in the field and short transport times to 10 min versus 12 min prior to arrival at the trauma center. Consideration should be given to patients that have been successfully intubated in the field and have a secure airway. For these patients resuscitative thoracotomy may be undertaken based on institutional experience upon their arrival at trauma centers, to improve outcomes and decrease health care costs. The predictive model generated from the NTDB, with all of its previously cited limitations, generated a larger number of strong independent predictors as outcome (n = 6) validating previous predictive models reported prospectively in the literature.

Pulmonary Injuries: Technical Aspects for Repairs

The management of pulmonary injuries can be approached based on location of injuries moving from a peripheral injury to more central or hilar injuries. Techniques utilized in these constellations of injuries include pneumorraphy, non-anatomic resections, stapled pulmonary tractotomy, lobectomy, and pneumonectomy. Small peripheral injuries can be managed with non-anatomic resection using a GIA or TA stapler.

Intraparenchymal through and through injuries without involvement of the hilum are amenable to stapled pulmonary tractotomy (Fig. 18.9). Clamp tractotomy described by Wall utilized aortic clamps placed through the injury tract and the tissue between the clamps divided [136–138]. This approach resulted in crushed pulmonary parenchyma and was rapidly abandoned. Asensio first described stapled tractotomy utilizing a GIA stapler as a tissue sparing technique to identify and selectively ligate bleeding sources for control of hemorrhage [138]. Once the injury track is identified, the stapler is placed within the track and the stapler is fired to open the pulmonary parenchyma and allow for identification of bleeding vessels and transected bronchi (see Figs. 18.9 and 18.10). More contemporary staplers with staple line reinforcement may aid in minimizing air-leaks from the staple line. Multiple studies have both validated and demonstrated this technique

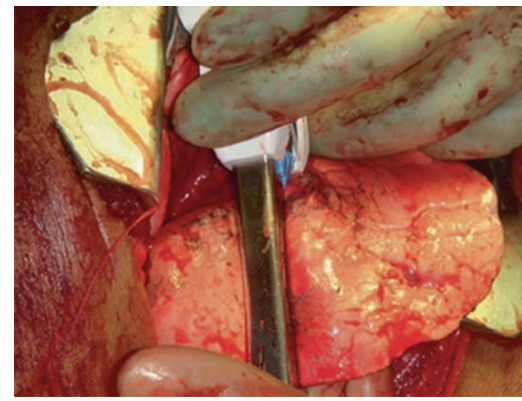

Fig. 18.9 Central Stapled Pulmonary Tractotomy, as described by Asensio for a AAST-OIS Grade V Pulmonary injury

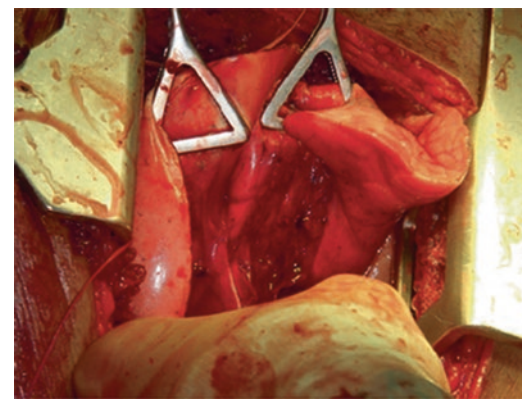

Fig. 18.10 Duval clamps separating the pulmonary parenchyma after stapled pulmonary tractotomy as described by Asensio

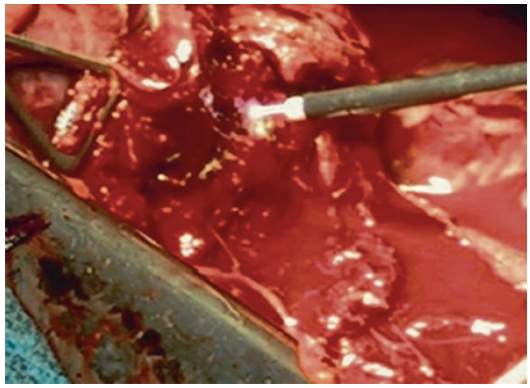

Fig. 18.11 Use of the Argon beam coagulator as an adjunct to stapled pulmonary tractotomy both procedures described by Asensio to control diffuse intraparenchymal Bleeding

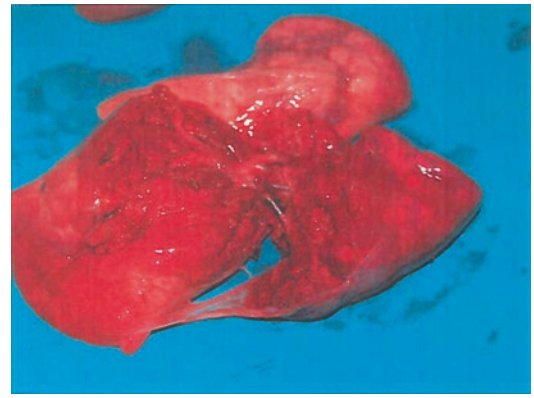

Fig. 18.12 Left pneumonectomy for the control of massive pulmonary hemorrhage. Patient survived. Pneumonectomy carries a 72–75% mortality

to not only be safe but effective. Furthermore, stapled pulmonary tractotomy can be safely extended as a central hilar tractotomy.

Given the dense microvascularity of the lung parenchyma and the finability of electrocautery regular to control diffuse hemorrhage, Asensio described the argon beam coagulator to be effective in controlling diffuse hemorrhage of the opened tract once major vessels have been ligated [48, 136]. This has been accepted as a useful adjunct to the stapled pulmonary tractotomy (see Fig. 18.11).

Central injuries of the hilum or hilar structures require hilar control. This can be achieved with cross-clamping with meticulous attention to prevent crushing the main bronchus. Hilar twist has been described but similarly, can result in injuries to the hilar structures and is definitely not recommended [43]. Lobectomy or pneumonectomy may be required in order to save the patient (see Fig. 18.12). If patients physiologic status and time allows, pulmonary vessels and bronchial isolation should be attempted to avoid these extremely morbid procedures in the trauma setting (see Figs. 18.13 and 18.14).

Thoracic damage control due to patient physiology may not allow for these technically difficult dissections, in which case lobectomy or pneumonectomy using a large green load TA stapler may be required [138]. Care should be taken to avoid these procedures as they are associated with poorer outcomes when compared to lung-sparing techniques such as stapled pulmonary tractotomy [139].

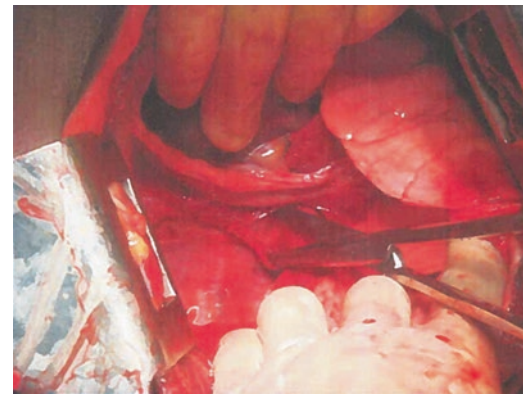

Fig. 18.13 Extra pericardial dissection to selectively isolate and control left pulmonary artery

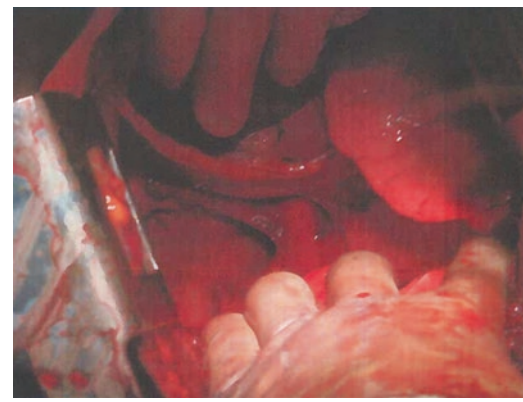

Fig. 18.14 Left main pulmonary artery

In 2018, Asensio and colleagues reported a large study consisting of 101 patients with penetrating pulmonary injuries, all of which required emergent thoracotomy. There were 96 males (95%) and five females (5%), with a mean age of 30 ± 10.29. The mean RTS was 6.25 ± 2.77, mean ISS was 36 ± 22, and the mean AIS for chest was 3.97 ± 0.78. A total of 73 (72%) sustained gunshot wounds (GSWs) and 28 (28%) sustained stab wounds (SW) [140].

Mean admission systolic blood pressure was 97 ± 47 mmHg, mean admission diastolic blood pressure was 53 ± 34, mean admission heart rate (HR) was 98 ± 47 beats/min, mean admission respiratory rate (RR) was 22 ± 11 breaths/min, and mean admission temperature 35.8 ± 1.07 °C. Predictors of outcome were identified for initial conditions on arrival, including systolic blood pressure ($p = 0.0019$), respiratory rate ($p = 0.0043$), RTS ($p < 0.0001$), and admission pH ($p = 0.0014$). Arterial blood gases (ABGs) were obtained in 88 of the 101 patients (87%) revealing a mean pH of 7.22 ± 0.17 and a mean base deficit of −6.8 ± 5.8. The mean total volume of fluids administered in the ED was 2716 mL, including 2076 ± 604 mL of crystalloids and 540 ± 181 mL packed red blood cells (PRBCs). There were nine (9%) patients that arrived in cardiopulmonary arrest, all required EDT, aortic cross-clamping, and open cardiopulmonary resuscitation; two patients (22%) ultimately survived [140].

All patients were subsequently transported to the operating room (OR) for definitive surgical intervention. In the OR, 83 (84%) were intubated with a single lumen tube, 11 (11%) had double lumen tubes and in six, (6%) the type of endotracheal tube was not recorded. Most patients underwent anterolateral thoracotomy 83 (82%), 18 (18%) required median sternotomy, four (4%) underwent posterolateral thoracotomy, while another four patients (4%) underwent a combination of incisions other than median sternotomy [140].

Operating room (OR) findings confirmed the presence of pulmonary injuries in all patients. Anatomic distribution of injuries revealed 66 (65.3%) patients that sustained left lung injuries, while 35 (34.6%) sustained right lung injuries. There were 143 operative procedures required in 101 patients. Many required more than one technique for definitive repair and control of bleeding, for a total of 1.4 procedures per patient. There were a total of 32 (31%) pneumonorrhaphies, 41 (41%) stapled pulmonary tractotomies and/or wedge resections, 23 (23%) lobectomies, and 6 (5%) pneumonectomies. There were a total of 114 (80%) tissue sparing versus 29 (20%) resective procedures. Pneumonectomy predicted mortality ($p = 0.024$).

All injuries were graded utilizing the American Association for the Surgery of Trauma Organ Injury Scale (AAST-OIS) 16 lung injury scale. There were 11 (10.9%) grade II; 51 (50.5%) grade III; 30 (29.7%) grade IV; six (5.9%) grade V; and three (3.0%) grade VI injuries. When comparing survival rates AAST-OIS injury grades I–III versus IV–V predicted survival ($p < 0.001$). The more complex surgical procedures including resective procedures were required for the definitive management of higher injury grades.

The mean estimated blood loss (EBL) was 5277 + 4455 mL. The total mean volume of intraoperative fluid replaced included 17,794 mL. This consisted of a mean total crystalloid volume of 6895 + 4372 mL and 961 + 634 mL of colloids. The mean total volume of blood and blood products administered in the OR included: packed red blood cells 3463 + 2700 mL; whole blood 3300 + 2693 mL; fresh frozen plasma 1724 + 1413 mL; cryoprecipitate 220 + 96 mL; and platelets 1541 + 1868 mL. Intraoperative complications included acidosis in 49 (49%) patients, hypothermia in 40 (40%), disrythmias in 18 (18%), and coagulopathy in 12 (12%). Multiple intraoperative factors such as EBL were predictive of outcome ($p = 0.02$) as was the presence of intraoperative disrhythmias ($p = 0.0001$).

There were a total of 179 associated injuries for an average of 1.77 associated injuries per patient of which there were 39 (22%) thoracic and 140 (78%) of the intrathoracic injuries, there were 24 (24%) cardiac and 15 (15%) large vessel injuries. Associated cardiac injuries were a strong single independent predictor of outcome for mor-

tality in stepwise logistic regression analysis (p = 0.02, OR 8.74, 95% CI 1.37–55.79). Associated intraabdominal injuries included diaphragmatic injuries 43 (42.5%), hepatic 26 (25.7%), gastric 19 (18.8%), splenic and small bowel 15 (14.8%), large bowel 9 (8.9%), and major abdominal blood vessels 7 (6.9%) as well as renal, duodenal, pancreas, gallbladder, and ureter all ranging from 1.9 to 5.9%.

A total of 64 (64%) patients survived for an overall survival rate of 64%. Adjusted survival rate excluding patients requiring emergency thoracotomy was 68%. Survival stratified to AAST-OIS injury grade revealed a higher survival rate for grades II–III versus IV–VI ($p < 0.001$). Survival was also stratified to surgical procedures; with pneumonectomy incurring a very high mortality (83%). One or more post-operative complications occurred in 22 (34%) patients, including infections/sepsis in nine (14%), pneumonia in seven (11%), post-operative hemorrhage in five (8%), bronchopleural fistulas in four (6.25%), and empyema in three (4.7%). Seven (11%) required tracheostomy with a mean number of 24 + 14 tracheostomy days. The mean total SICU-length of stay was 5.54 + 9.05, and the mean hospital length of stay was 11.7 + 14 days.

Stepwise logistic regression analysis identified AAST-OIS injury grades IV–VI (p = 0.007; OR 6.38, 95% CI 1.64–24.78), presence of intraoperative disrhythmias (p = 0.003; OR 17.38, 95% CI 2.59–116.94), and associated cardiac injuries (p = 0.02; OR 8.74, 95% CI 1.37–55.79) as the most important independent predictors of outcome for penetrating pulmonary injuries.

Asensio's study describes one of the largest series, consisting of 101 patients with penetrating pulmonary injuries, all requiring emergent thoracotomy secondary to their clinical presentation with a low mean blood pressure 97, RTS 6.25, mean pH of 7.22, significant base deficit 6.8, and a high ISS 36, denoting a physiologically compromised and anatomically severely injured patient population [140].

This study described and validated predictors of outcome for patients sustaining penetrating pulmonary injuries requiring surgical intervention. These predictors of outcome include physiologic condition upon arrival, such as vital signs, pH, and base deficit. It is also worthwhile to note, that the initial PaO_2 level after intubation was a strong predictor of outcome. Anatomically, AIS was also noted to be a strong predictor of outcome. To the best of our knowledge, this has not been previously reported. Also, to the best of our knowledge no other series has validated or graded these injuries utilizing the AAST-OIS lung injury scale. Patients with AAST-OIS IV–VI had statistically significant higher mortality rates.

Asensio's study also describes intraoperative predictors of outcome including estimated blood loss as well as the need for blood and blood products. Similarly, the presence of any intraoperative complications such as acidosis, hypothermia, coagulopathy, and the presence of dysrhythmias also predicted outcome.

Historically, penetrating pulmonary injuries were managed by resective procedures including both lobectomy and/or pneumonectomy. These procedures still carry significant mortality. Since the description of lung-sparing procedures by Asensio—Stapled pulmonary tractotomy and the advent of the argon beam coagulator as an adjunct to tractotomy—also described by Asensio—significant decreases in both morbidity and mortality have been reported [48, 138, 141].

Both Velmahos and Cothren reported that up to 85% of their penetrating pulmonary injuries could be managed with tissue sparring techniques [139, 142]. In this series, 80% of patients were managed with lung sparring techniques; however, higher injury grades required resective techniques. Karmy-Jones' study reports a higher mortality as the extent of resection increases [143]. This correlation was validated in our study and was a strong independent predictor of outcome [140]. Similarly, the need for pneumonectomy was highly predictive of mortality.

Martin reviewed the National Trauma Data Bank (NTDB) of the American College of Surgeons (ACS) data of 669 patients and reported lower mortality rates for patients undergoing non-resective versus resective procedures, with mortality rates for lobectomy 27% and pneumonectomy 53% [144]. In our series, lobectomy and pneumonectomy had 48% and 83% mortality,

respectively. These differences may be accounted for by the much higher ISS of the patients in Asensio's series [36] versus in Martin's series (24) [145], as well as by the higher number of thoracic and extra-thoracic associated injuries.

Unfortunately, mortality rates for patients requiring pneumonectomy are very high, ranging from 50% to 100%. The vast majority of patients requiring pneumonectomy usually present in profound shock, or already experiencing the exsanguination syndrome. In order to determine the true mortality of patients requiring pneumonectomy for penetrating injuries we reviewed 20 series from the literature and included our own experience [139, 142, 145–155]. There were a total of 117 patients, 82 succumbed for a mortality rate of 70%. The mechanism responsible for this high mortality was proposed by Bowling who postulated that these patients succumb to acute right ventricular failure. This was confirmed in a porcine model by Cryer and has also been described clinically by Asensio.

Asensio's overall survival rate excluding patients arriving in cardiopulmonary arrest requiring EDT was 68% and compares favorably with rates reported in the literature, given the significant numbers of both thoracic and extra-thoracic injuries especially a 24% and 15% incidence of associated cardiac and large thoracic blood vessel injuries, respectively. The presence of an associated cardiac injury was a strong predictor of outcome in our series as well as in Karmy-Jones' series.

In Asensio's study, the authors reported predictors of outcome for penetrating pulmonary injuries that must be taken into account during their operative management while validating the AAST-OIS lung injury scale. Tissue sparring techniques may be utilized to manage between 80 and 85% of these patients as previously reported in the literature. According to our data, every effort should be made to utilize lung-sparing techniques. This study once again validates stapled pulmonary tractotomy as a valuable technique to manage these injuries. This procedure is now uniformly used worldwide and is estimated that approximately 85% of patients sustaining penetrating pulmonary injuries can be managed this way.

Unfortunately, lobectomy and pneumonectomy still carry significant morbidity and mortality as evidenced by our review of the literature. Because only a small percentage of penetrating pulmonary injuries require thoracotomy, for definitive management, the challenges of decreasing their mortality await a concentrated effort to develop animal models to define newer strategies within a translational model approach.

Intrathoracic Vascular Injuries: Technical Aspects

In the damage control setting the majority of patients presenting with these exsanguinating injuries may have already undergone EDT (left anterolateral thoracotomy) or clamshell thoracotomy (bilateral anterolateral thoracotomies) in the trauma bay as a rescue maneuver. Ultimately, the diagnosis of the injured vessel dictates the incision and subsequent maneuvers to expose proximal and distal control.

In the trauma bay, control is key and the main objective. On the left, the proximal subclavian can be controlled intrathoracically as it originates lateral and posterior on the aorta, although this is very difficult. On the right side control of the right subclavian and innominate artery is also difficult and often only digital control is obtained. If unable to obtain control digitally, descending thoracic aortic and proximal subclavian control can be achieved through the standard left anterolateral thoracotomy incision in damage control to allow for at least proximal control. While this incision provides adequate exposure for proximal control of other left sided structures it lacks exposure for right sided injuries [51, 156]. Injuries of the aortic arch and the proximal great vessels require median sternotomy for proximal control which can be extended to a standard cervical incision anterior to the sternocleidomastoid or as a subclavicular incisions (Fig. 18.15).

Subclavian injuries are most easily accessed via a supraclavicular or subclavicular incision. If a more proximal injury is noted, clavicular head removal is indicated with or without replacement of the clavicle post-repair (see Figs. 18.16 and 18.17). If digital compression of the subclavian artery against the clavicle was required to maintain control, then the person maintaining digital

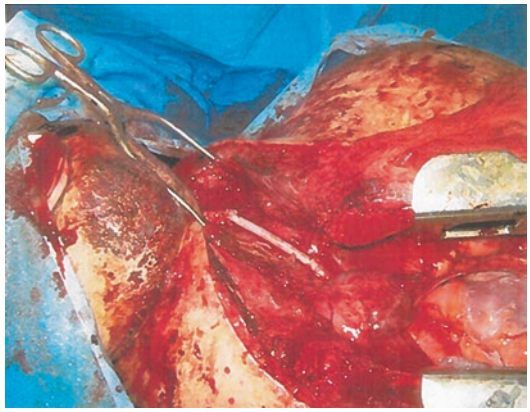

Fig. 18.15 Median sternotomy and left neck exploration depicting a non-reversed autogenous left saphenous vein interposition graft originating 2 cm from the origin of the left common carotid artery off the arch of the aorta to the distal left common carotid artery. Patient survived

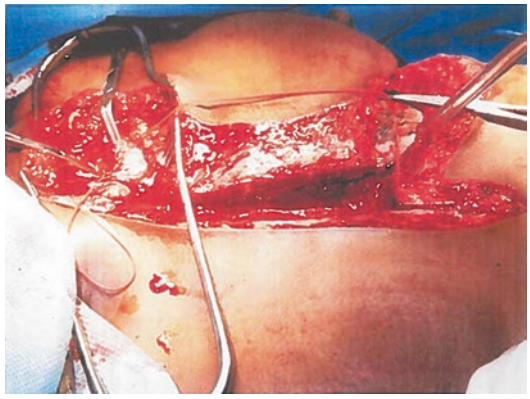

Fig. 18.16 Left anterolateral thoracotomy initially performed for a patient that sustained a left proximal subclavian artery injury secondary to a tangential gunshot wound (GSW). The patient arrived in cardiopulmonary arrest. Digital pressure was applied at the origin of the left subclavian artery and the patient transported to the Operating Room requiring a Median sternotomy and left supraclavicular approach with left clavicular removal for control of the left subclavian artery injury. Left subclavian artery controlled. Patient survived

control should be prepped into the field, while more proximal and distal control is achieved [51, 156, 157]. Ligation is not an option for the proximal subclavian artery. Injuries to the subclavian, innominate, and jugular veins can be safely ligated [64, 67, 157].

When exposure is obtained, primary repair is the preferred method of reconstruction. This

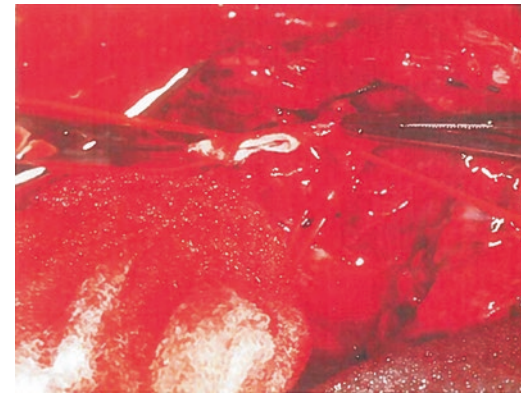

Fig. 18.17 The same patient as in Figure 18.17 reveling a close-up picture of the left subclavian artery clamped in anticipation of a successful placement of a 6 mm PTFE graft. Patient survived

choice is often not possible given the tissue damage caused from the missile. In this case reconstruction with a synthetic graft is preferred. Polytetrafluoroethylene (PTFE) or knitted Dacron grafts are the conduits of choice for vessels larger than 5 mm in diameter. Aortic injuries secondary to penetrating injuries can be repaired primarily or with synthetic grafts.

For subclavian injuries, the placement of shunt has been described to be a useful adjunct in damage control. Although described, there is a very limited number of reports in the literature, placement of an intraluminal shunt has been associated with limb salvage. We have described the technique for shunt placement for the very rare occasion in which they may be needed.

Choice of shunt size will depend on the vessel diameter and availability at your institution but Argyle shunt has shown success and is preferred [158, 159]. When placing Argyle shunts first do not trim the damaged arteries to be shunted (Fig. 18.18). This will allow maximal length to be utilized for reconstruction. Passage of a Fogarty catheter to remove any distal clot and ensure unobstructed flow. A 0 silk should be placed in the middle of the shunt to allow for maneuverability into the proximal and distal ends. Trim the shunt to allow for easier placement into the vessel so that it can be secured to the ends of the damaged ends of the vessel. Shunt dwell time should be limited to less than 6 h but can be left in longer with little injury [158–160].

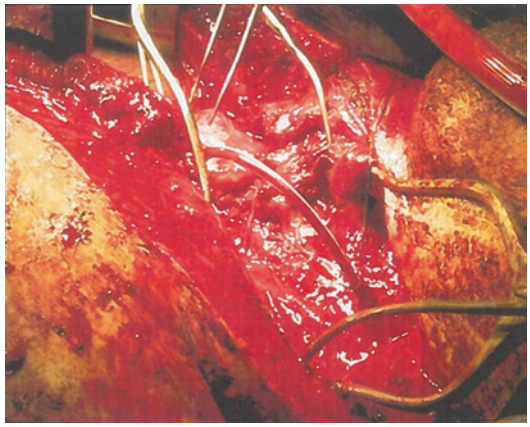

Fig. 18.18 Temporary Argyle shunt placed in left common carotid artery to restore ipsilateral cerebral blood flow prior to placement of an autogenous non-reverse saphenous vein graft. Patient survived with intact neurological function

The shunt must be secured in place with 0 silk and check flow with doppler ultrasound.

Tracheobronchial Injuries: Technical Aspects for Repair

Tracheobronchial injuries can be categorized based on location: as distal or proximal. This may dictate the treatment. Distal tracheobronchial tree injuries are rare and may be repaired primarily in the setting of penetrating trauma. These are often encountered during stapled pulmonary tractotomy. More proximal tractotomy injuries must be repaired; otherwise, bronchopleural fistulas may develop. In all cases of tracheobronchial injuries an airway must be secured prior to intervention. This must be obtained in the trauma bay.

Tracheal injuries can be devastating and ensuring adequate oxygenation and ventilation is key. Advancement of the endotracheal tube past the level of the injury will allow this and provide the surgeon time for repair. Penetrating tracheal wounds should be primarily repaired. Permanent sutures can be placed through or around the tracheal rings with the knots tied externally to prevent to formation of granulomas or strictures.

Bronchial injuries likewise are rare and should be repaired primarily using interrupted sutures with absorbable sutures. If proximal bronchial injuries are unable to be repaired, in a damage control setting pneumonectomy may be indicated. Post-operative suture or staple line dehiscence and airleaks are potential complications. Particularly in proximal bronchial and tracheal repairs a muscle or Brewer's fat flap can be used to buttress the repair and minimize fistulation [139, 142].

Esophageal Injuries: Technical Aspects for Repair

Penetrating esophageal injuries are rare and carry a high risk for morbidity and mortality [161]. The primary goal in the management of these disastrous injuries is primary repair without stenosis. Because of the high risk for leak and subsequent mediastinitis, wide drainage with chest tubes is necessary. Meticulous surgical technique will prevent suture line, dehisance, or anastomotic failures, thus avoiding risks the mediastinitis, mediastinal abscess, or empyema.

Time to diagnosis and operative management must be kept at a minimum as most patient with esophageal injuries who dies, die secondary to other causes. This injury is rare and in centers practicing selective management of penetrating neck or transmediastinal injuries rapid diagnosis and repair should be made a high priority. In his series, Asensio reviewed injuries over a 72-month period where 43 patients were identified representing 0.11% of all trauma admissions [7]. This rare injury, though, carries high morbidity and mortality when repair is delayed. The most common mechanism was penetrating injury due to gunshot wound with the most common location being cervical. Of those who survived the initial resuscitation in the emergency department, 35 were able to reach the OR for definitive surgical repair. Within this cohort, the group was divided into those who received pre-operative evaluation (17 patients) and those who went directly to the OR (18 patients). Greater than 94% of the patients in this cohort had a systolic blood pressure greater than 90 mmHg [89]. There were no differences in complication or ICU length of stay. The only difference was time to the OR which averaged 16.7 h in the pre-operative evaluation group and

1.42 h in the OR direct group. While not significant, there were more complications in those who delayed in going to the OR. These findings suggested that, while the groups were small, delay in operative intervention for esophageal injury results in significant morbidity. Diagnostic maneuvers are prudent and must be done expeditiously with the intent to go directly to OR for repair once diagnosed [7].

In a subsequent multicenter study, Asensio and the American Association for the Surgery of Trauma attempted to define the time after which delays in operative management of esopheal injurycauses increased morbidity and mortality [47]. In this study, patients were collected over a 10.5-year period and 405 patients from 34 institutions were enrolled. Again, cervical esophageal injuries were the most common and the most common mechanism was gunshot wounds. The overall mortality rate was 19% or 78 patients. Half of those who died, died in the emergency department following resuscitative thoracotomy. 381 patients made it to the operating table and 35 of those patient died in the OR. The group of survivors totaling 346 patients were then divided into those who received pre-operative evaluation and those who went directly to OR. Of those who underwent pre-operative evulation, each patient averged 1.7 procedures, the most common of which was esophagoscopy (73%). There were little differences between the groups. Blood loss though was found to be significantly greater in the no pre-operative evaluation group 1696 mL vs 505 mL in the pre-operative evaluation group. In patients with complications related to the esophageal injury, there were marked differences between the groups. There were 74 (41%) patients that experienced esophageal related complications in the pre-operative operation group versus 32 (19%) patients in the direct OR group. The majority of the complications were infectious in nature and included abscess, mediastinitis, and empyema. When evaluating the length of stay in the ICU the pre-operative evaluation group had longer length of stay 11 days vs 7 days. Similarly, the hospital length of stay was longer in the pre-operative evaluation group compared to the no pre-operative evaluation group 22 versus 11 days, respectively. These findings suggest that immediate operative intervention is favored [47].

Primary double layer repair should be attempted at the initial operation followed by definitive Grillo pleural or intercostal muscle flaps [7, 47]. Those injuries that cannot achieve repair or are deemed to be unreconstructable should be ligated and a nasogastric tube is placed immediately proximal to the level of the injury with wide drainage and bilateral thoracostomy tubes. Reconstruction over a T-tube (Kehr tube) has been reported for complex injuries [7, 47]. Diversion with cervical esophagostomy is another option but is time-consuming and requires additional proximal dissection. This should not be considered during damage control and can be an option at the second look operations. To facilitate enteral feeding in all cases of esophageal injury gastrostomy tube placement should be strongly considered [7, 47].

Thoracic Packing

Packing is a long held and accepted practice in the damage control setting. This is true for abdominal damage control where concerns for venous return of pulmonary expansion are of no concern. In the thoracic cavity attention should be directed to these circumstances where packing may impede blood and air flow in the critically ill patient. Thoracic packing has been described as a means to control bleeding after cardiac procedures and some pulmonary resections but has been utilized less in the trauma setting given concerns over intrathoracic pressure impeding venous return, oxygenation, and ventilation [63, 156]. Reports by both Caceres and Lang described the application of this technique in thoracic damage control with some success [162, 163].

These reports, though, are limited to case reports and larger studies are needed. Packing of the thorax should be reserved for the most extreme situations where bleeding secondary to coagulopathy and where not surgical corrected causes is encountered. In this case damage con-

trol and bailout are necessary for survival with correction of coagulopathy, restoration of normothermia and correction of acidosis is achieved in the ICU. In the setting of stapled pulmonary, a tractotomy topical hemostatic agents are useful adjuncts as is the use of the Argon beam coagulator.

Temporary Chest Wall Closure

Proper closure of the chest wall involves a multilayer closure with anatomic re-approximation. This is not feasible in the damage control setting, thus more expeditious approaches to chest wall closure are recommended. In this setting temporary chest wall closure can be achieved with closure of the skin alone. This can be done with suture in a running fashion. In those whom skin closure is not possible, an adhesive type bandage such as Steri-Drape™ or Ioban™ can be placed over the incision after placement of moist laparotomy pads to provide closure. Ensure drainage of the thorax with thoracostomy tubes. Other methods that can be utilized are towel clips a running en-masse suture, a Bogota bag or a negative atmospheric pressure device (Wound Vac™) [142, 164], may also be used.

Cardiothoracic Damage Control in the Intensive Care Unit for Transmediastinal Injuries: Post-Operative Management

The next steps in damage control may take place in the trauma surgical intensive care unit (TICU) setting. Management in this setting is as critical as in the trauma bay or operating room. Additionally, diagnostic modalities such as computed tomographic scan and angiography should be performed prior to arrival in the ICU to minimize transport. On arrival to the TICU, diagnostic and/or therapeutic bronchoscopy should also be used in patients with tracheobronchial and bronchial injuries.

Rapid correction of acidosis, hypothermia, and coagulopathy is vital to success. Oxygen perfusion mismatching is the cause of acidosis and should be corrected with balanced resuscitation and optimization of oxygen delivery. Because of the pulmonary compromise associated with pulmonary injuries minimization and judicious use of crystalloids along with hemostatic resuscitation will decrease pulmonary edema and improve oxygenation. Many of these patients have decreased pulmonary reserve due to intraparenchymal hemorrhage, pulmonary contusions, and/or airleaks resulting from their initial injury. Monitoring is also key in this setting. A low threshold for return to the operating room must be strictly adhered to. Inertia to re-operate may be costly and lead to downstream unwanted complications.

Thoracostomy tube output should be monitored closely; bleeding from the chest in the thoracostomy tubes should decrease with time and goal directed correction of the coagulopathy using the results of TEG. Abrupt decreases and/or no output from the drainage tubes should prompt investigation as blood clots may have occluded the system. If thoracostomy tube output remains high despite correction of hematologic abnormalities, inadequate hemostasis should be suspected and a return to the OR or reoperation at the bedside should be strongly considered. A threshold of four to six units of blood transfused in the setting of ongoing bleeding should prompt consideration of a return to the OR as advocated by Martin [60]. This number though should be used as a guide and clinical judgment at the bedside taken together with the patient's physiologic parameters should take precedent to hard-and-fast rules.

Return to the Operating Room

Patients who have required temporizing procedures should be returned to the operating room once normal physiology and endpoints of resuscitation have been restored and met. The goal of the second look operation is complete closure of the chest wall. The patient's physiology will, again, dictate this and thus close monitoring of intrathoracic pressures can guide this.

As lung compliance falls and cardiac swelling ensues from a stunned myocardium following EDT and other associated thoracic injuries, elevated intrathoracic pressures may develop following attempts at closure. Thus, a staged approach can be employed. If closure is possible, two thoracostomy tubes should be placed and directed posteriorly one toward the costophrenic sulcus and the second apically to allow for maximal drainage. Additional tubes and drains are dictated by the constellation of injuries incurred by the patient. Esophageal injuries in particular may require additional mediastinal drainage to prevent mediastinitis. The thoracic cavity and incision are vigorously irrigated and hemostasis is obtained prior to closing the chest wall in layers.

Complications

Complications following thoracic damage control are common, severe, and often multiple. Cardiac tamponade and airleaks are particularly unique to this population for reasons stated above. Cardiac tamponade, described as Beck's triad of distended neck veins, muffled heart sounds, and hypotension is rare, as it pertains to cardiac injuries, does not classically appear in this patient population. Presentation is subtle manifesting initially with decreased cardiac output and cessation of mediastinal drainage output. Therefore the use of noninvasive hemodynamic monitoring such as pulse wave analyzer, assessment of cardiac function with transthoracic, and/or transesophageal echocardiography is strongly recommended. Definitive management of pericardial tamponade requires re-opening of the thorax, and evacuation of hematomas and hemostasis if ongoing surgical bleeding sources are encountered.

Airleaks are common following pulmonary procedures and often are self-limiting. Conservative management of thoracostomy tube output should be initially attempted in conjunction with lung-sparing ventilation and low positive end expiratory pressures. High frequency percussive ventilation has been employed by the senior author in some patients successfully. Persistent leaks may require reoperation to repair or resect the portion of lung parenchyma involved.

Mortality in this population is quite high and is reported to range from 23 to 69%. Attention to detail is of great importance in this population and variation has been attributed to differences in patient age, damage control techniques employed, and severity and mechanism of injury.

Conclusions

Patients with severe thoracic injuries and unstable physiology may benefit for cardiothoracic damage control. Initial management occurs in the trauma bay with establishment of an airway and control of hemorrhage. EDT is useful and may save lives in the trauma bay as well. In the operating room, control of hemorrhage and contamination are the first priorities. This is followed by goal directed critical care including appropriate intravascular volume replacement, normalization of end points of resuscitation, and a planned second look via reexploration once the physiological derangements have been corrected. Management of the lethal tetrad of acidosis, hypothermia, coagulopathy, and cardiac dysrhythmias in the OR and in the TICU will minimize deaths and complications. Cardiothoracic damage control has placed emphasis on simple and rapid techniques to control hemorrhage and correct patient physiology to allow for subsequent stages of definitive surgical repair and reconstruction.

Thoracoabdominal Injuries

Thoracoabdominal injuries represent some of the most challenging injuries facing trauma surgeons. American wartime experience has shown them to be among the most critical injuries incurred by battlefield casualties [2, 3, 5]. The diagnostic challenge of multiple body cavity injuries, the notorious difficulty of establishing the proper sequence for intervention, their high injury severity, frequent hemodynamic instability, and the inherent dangers of cross-cavity con-

tamination conspire to increase morbidity and mortality for these injuries. The diagnosis of penetrating thoracoabdominal injuries is often predicated on the presence or absence of diaphragmatic penetration, which at times can be difficult to establish pre-operatively [50].

Errors in diagnosis often occur, as these injuries vex even the most experienced trauma surgeons [165]. A particularly difficult scenario presents with the unstable patient whose operative findings on one side of the diaphragm cannot account for the patient's hemodynamic instability or blood losses.

Given the clinical challenge and large volume of these injuries Asensio examined his institutional experience with their management. In this study, he sought to define this patient population clinically, describe the sequence of surgical interventions using combined procedures (i.e., thoracotomy and laparotomy), as well as describe the difficulties and pitfalls leading to inappropriate choices of surgical interventions for thoracoabdominal injuries [50].

In this 4 year study of 254 patients sustaining penetrating thoracoabdominal injuries all requiring surgical intervention were identified and studied. In patients sustaining torso injuries, violation of both the thoracic and abdominal cavities was established on the basis of physical examination, location of injuries, investigative studies, chest tube output, and operative findings.

In this study, data collected included demographics, Revised Trauma Score (RTS), Glasgow Coma Scale (GCS), and Injury Severity Score (ISS). Field data such as the need for intubation and cardiopulmonary resuscitation (CPR) were also recorded. Other data included pre-operative investigations such as radiography (chest, abdomen, and pelvic films), echocardiography (ECHO), ultrasonography (USN), and diagnostic peritoneal lavage (DPL). In addition, the number and types of emergency department (ED) thoracotomies (left or right, anterolateral or bilateral) were recorded. Indications for the performance of thoracotomy or laparotomy were recorded. The types and combinations of surgical procedures were also tracked [61].

Patients were grouped according to the sequence of procedures: laparotomy followed by thoracotomy (Lap + Thor) or thoracotomy followed by laparotomy (Thor + Lap). Patients subjected to laparotomy plus chest tube thoracostomy were also identified. Determination of the direction and trajectory of the injury and whether the injury crossed the thoracic or abdominal midline were recorded. Estimated blood loss (EBL), number of reoperations, and associated injuries were also examined [50].

Further analysis of patients undergoing combined procedures was undertaken. Two patient groups were compared. Group I consisted of patients undergoing laparotomy first then thoracotomy (Lap + Thor) versus group II patients undergoing thoracotomy then laparotomy (Thor + Lap). The number of times that either of these procedures had to be interrupted to convert to the other procedure was analyzed. The reasons for these interruptions were also recorded and classified as errors or pitfalls. Outcome was measured by calculating the overall mortality with particular emphasis on the patient population who underwent combined thoracotomy and laparotomy.

Over the span of this 4-year study there were 254 patients that sustained penetrating thoracoabdominal injuries meeting the inclusion criteria. In all patients there was confirmation of both chest and abdominal cavity involvement by investigative studies, operative interventions in one body cavity, or both. There were 233 males (92%) and 21 females (8%). The mean age was 27 years (range 7–69 years). The mechanism of injury was gunshot wounds (GSWs) in 187 patients (73%), shotgun wounds (STWs) in 3 (2%), and stab wounds (SWs) in 64 (25%). The mean RTS was 6.04 (range 0–7.84); the mean GCS was 12; and the mean ISS was 27 (range 4–75), indicating a severely injured patient population. Field data revealed the use of CPR in 33 patients (14%), and 26 patients (10%) required field intubation.

Investigative studies included radiography (chest, abdomen, and pelvic films), FAST, and DPL when needed. Radiography was the most

commonly used investigational procedure and DPL the least used. ED thoracotomy was performed in 51 patients (20%): 34 (67%) left anterolateral, 1 (2%) right anterolateral, and 16 (32%) bilateral anterolateral thoracotomy. Only three patients (6%) survived. All patients underwent immediate surgical intervention: 103 (41%) thoracotomy, 224 (88%) laparotomy, and 73 (29%) both. The most common indications for thoracotomy and laparotomy were resuscitation (56%) and the presence of peritoneal signs (46%).

Information was available to establish the direction and trajectory of the injury for 156 (62%) of the patients. Mortality rates were consistently higher for these patients. There were multiple combinations of surgical procedures and interventions in this severely injured patient population. A total of 327 major surgical interventions (thoracotomy, laparotomy, thoracotomy + laparotomy) were performed, representing a mean of 1.3 surgical interventions per patient. Of the 103 thoracotomies, 23 (22%) revealed no thoracic pathology, including 9 of the 51 ED thoracotomies, which were considered resuscitative. Thus, resuscitation was the most frequent reason for a negative thoracotomy. If both ED and operating room (OR) resuscitative thoracotomies are excluded, negative thoracotomies decrease to 13% [50].

Other important reasons were a misleading chest tube output and suspected cardiac tamponade. Altogether, 26 (11%) of the 224 laparotomies were also negative. The mechanism of injury (i.e., missiles or stab wounds suspected of, but not causing, intraabdominal injury) was the most important reason for performing a negative laparotomy. The mean estimated blood loss was 3004 mL (range 100–30,000 mL). Altogether, 38 patients (15%) required reoperation: 22 (58%) laparotomy, 10 (26%) other surgical interventions; 5 (13%) laparotomy and thoracotomy combined; 1 (3%) thoracotomy. Six of the reoperated patients died. There were seven missed injuries (3%), including one diaphragmatic and splenic injury each and five mesenteric arterial injuries.

The mortality rate among those with missed injuries was 57%. There were 462 injuries, including 179 (39%) solid organs, 116 (25%) hollow viscera, 61 (13%) pulmonary, 34 (7%) abdominal vascular, 32 (7%) cardiac, and 22 (4.8%) thoracic vascular, representing a mean of 1.8 associated injuries per patient. Altogether, 175 of 254 patients survived, yielding a 69% survival rate. Analysis of those undergoing combined procedures (laparotomy + thoracotomy) revealed a total of 73 (29%) patients, which included 70 males (96%) and 3 females (4%). The mean age for this patient population was 27 years (range 14–50 years). There were 59 patients (81%) who sustained GSWs, 1 (1%) sustained an STW, and 13 (18%) sustained SWs. The mean RTS was 5.02 (range 0–7.84), the mean GCS was 10, and the mean ISS was 34 (range 9–75), revealing greater physiologic compromise and degree of anatomic injury for this subset of patients.

In this group of patients, ED thoracotomy as a resuscitative procedure was performed in 21 (29%); 3 (14%) survived. All 73 patients underwent immediate surgical intervention. Altogether, 53 (73%) underwent laparotomy and thoracotomy, 17 (23%) laparotomy and median sternotomy, and 3 (4%) laparotomy, thoracotomy, and median sternotomy. The mean estimated blood loss was 6827 mL (range 500–30,000 mL).

Twelve patients (16%) underwent reoperation: seven (58%) had a laparotomy, three (25%) had laparotomy and thoracotomy combined, and two (17%) had other surgical interventions. Two of the reoperated patients died. There was a total of three missed injuries (4%). All of those missed were mesenteric arterial injuries. The mortality rate among those with missed injuries was 67%. There were 196 associated injuries, representing a mean of 2.7 injuries per patient. A total of 30 of 73 patients survived, for a 41% survival rate.

Among the patients who underwent combined procedures, 32 of 73 (44%) had inappropriate surgical sequencing defined by the number of times either of these procedures had to be interrupted to convert to another procedure as the patients deteriorated. In group I (Lap + Thor) the initial procedure (i.e., laparotomy) was

interrupted in 18 of 34 patients (53%). In group II (Thor + Lap) the initial procedure (i.e., thoracotomy) was interrupted in 14 of 39 patients (36%). The most frequent pitfalls leading to inappropriate surgical sequencing were persistent, unexplained hypotension in 13 patients (18%) unaccounted for by surgical findings in the initial cavity accessed and misleading chest tube output (i.e., high output from abdominal injuries through diaphragmatic lacerations) in eight patients (10%).

Penetrating thoracoabdominal injuries pose a significant challenge to trauma surgeons. Involvement of the two largest cavities of the body confronts the trauma surgeon with a critical dilemma: Which body cavity should be accessed first, and when? This dilemma is compounded by the critical nature of these patients and the hemodynamic instability that often accompanies these injuries. Establishing whether an injury trajectory has crossed the diaphragm to involve an adjacent cavity is often confusing and imposes a risk to the patient if the wrong body cavity is accessed first.

The high mortality incurred by these injuries is corroborated by both military and civilian reports, although there is a paucity of data in the literature dealing with these injuries, Brewer [2] reported World War II data on 983 thoracoabdominal injuries with a 27% mortality rate. Artz [3] reported a Korean conflict experience of 129 patients with a 13% mortality rate.

In the civilian arena, both Borja and Ransdell reported 20% mortality among 44 patients who sustained thoracoabdominal injuries [166]. Similarly, Hirshberg [167] reported a 41% mortality rate for 82 patients who required combined laparotomy and thoracotomy. The incidence of thoracoabdominal injury varies depending on the patient population and the mechanism of the injury [168]. It has been stated that gunshot wounds to the thorax involve the abdominal cavity approximately 30–40% of the time [165]. Despite the critical nature of these injuries, few data have been reported in the literature describing their management. In series reported by Borja and Ransdell [166] Moore [169], Oparah and Mandal [170], and Hirshberg [171] there were 20 patients who underwent combined thoracotomy and laparotomy.

Asensio's study report represents a large experience with these injuries managed in a busy urban Trauma Center. This group represents a critically injured patient population who required immediate surgical intervention. The critical nature of these patients is evident by the frequent use of EDT as a resuscitative measure. Their low RTS and high ISS, the large number of surgical procedures and combinations necessary to care for these patients, elevated blood loss, and mortality rate certainly attest to the need for life-saving surgical interventions.

Most of the initial diagnostic procedures include plain radiography, which is often time-consuming and yields suboptimal results. Although these films may be helpful, they generally do not alter indications for surgical intervention.

Given the advances in the field of ultrasonography we strongly recommend the use of E-FAST as an initial evaluation tool whose use may be the first step in decreasing the number of negative explorations. The pattern of injury most frequently observed in this group of patients is in a downward direction, from thorax to abdomen. Regardless of the direction of the missile, transdiaphragmatic injury increases the chances for cross-cavity contamination, as it permits passage of gastrointestinal contents into the affected hemithoracic cavity, increasing the risk for empyema. The crossing of midline structures by injury patterns occurs frequently, increasing the scope and duration of surgical exploration, which also has significant implications.

As exploration is broadened, i.e., opening another body cavity the chances for the development of hypothermia, acidosis, and coagulopathy increase; similarly, so do the chances for missed and iatrogenic injuries. The two most critical decisions that must be made during the management of these injuries are which body cavity must be accessed first and the timing. These decisions are difficult and often wrong.

In Asensio's series, the most frequent indications for thoracotomy were resuscitation and elevated chest tube outputs, producing an over-

all rate of 22% negative thoracotomies. If resuscitative thoracotomies are excluded, the negative thoracotomy rate is still significant: 13%. An 11% negative laparotomy rate is also significant. This relatively high frequency of negative explorations is similar to those reported by Hirshberg [167]. Whom reported 11% and 22% rates for negative thoracotomy and laparotomy, respectively. These figures point to the difficulties during surgical decision-making. A 15% reoperation rate for unpacking/repacking and for completion of abbreviated surgical procedures (i.e., damage control) is similar to that in Hirshberg's series [167].

In Asensio's study the rate of missed injuries was lower (3% vs. 9%); nevertheless, the mortality rate for missed injuries was 57%. Missed injuries are generally the result of failure to explore the correct body cavity initially because of unclear indications or less than thorough explorations due to the critical nature of these patients and the need for damage control. In this series, 73 patients underwent combined surgical procedures. This group of patients incurred more physiologic compromise and injury severity than the main group, as evidenced by their RTS, ISS, and greater mean EBL (6827 vs 3004 mL), although they experienced similar rates for reexploration, missed injuries, and mortality from missed injuries as the main group. It is in this group of patients, however, where the potential for surgical error and pitfalls is highest.

Given the high potential for surgical errors in diagnosis; Asensio examined this issue by analyzing the number of times the primary surgical procedure had to be interrupted to convert to another as demanded by the patient's acuity. This situation was considered inappropriate surgical sequencing. In this group, 44% of the patients had inappropriate surgical sequencing. In the two subgroups of patients analyzed, group I (Lap + Thor) had a 53% rate of interruption of the primary procedure, and group II (Thor + Lap) had a 36% rate of interruption. The most frequent causes of inappropriate sequencing were persistent unexplained hypotension unaccounted for by the surgical findings in the initial cavity accessed and misleading chest tube output, both of which were considered indications that the wrong body cavity had been initially accessed.

Other important pitfalls included the need to access another body cavity for exposure or mobilization of the liver, as well as injuries missed during the early evaluation that later manifested during the intraoperative course. The pattern leading to inappropriate surgical sequencing usually begins during the initial assessment and resuscitation of these patients. Trauma surgeons must be aware that abdominal examination can be unreliable in the presence of thoracic injury.

Likewise, chest tube output can be highly unreliable for reasons such as incomplete evacuation of the thoracic cavity, clotted hemothorax, and a high output that may originate from abdominal bleeding in the presence of an associated diaphragmatic laceration that may initially go undetected. Similarly, caution must be taken when interpreting the initial set of films obtained during the resuscitation period. Often they are unreliable and misleading. Intraoperatively, the surgeon must be prepared for all contingencies. The patient must be prepared from neck to midthigh in the event that another body cavity must be accessed. Chest tube output must be tracked intraoperatively as well as peak airway pressures. This demands close communication with both anesthesia and nursing personnel.

Intraoperatively, the trauma surgeon must attempt to follow injury trajectories and examine the diaphragm and pericardium for bulging or penetration. Transabdominal pericardial window, intraoperative diagnostic peritoneal lavage, reinsertion of a new chest tube, and even intraoperative chest or abdominal films are valuable tools for diagnosing an injury in an adjacent body cavity. They should be employed selectively. The wide use of these procedures or combinations of these procedures may avoid opening another body cavity.

Opening another body cavity to exclude injury (i.e., "quick" thoracotomy or laparotomy) should not be practiced indiscriminately nor routinely. The physiologic implications can be devastating and may promote hypothermia and its sequelae, increase the operating time in severely compromised patients, and place the patient at risk for

iatrogenic injuries. When a thoracic cavity injury cannot be definitively excluded employing the previously outlined procedures and strategies, thoracotomy should be undertaken. We advocate preservation of the thoracoabdominal barrier to prevent thoracic contamination and preserve diaphragmatic function [166]. We prefer an anterolateral thoracotomy rather than a diaphragmatic incision, which is often inadequate for full exploration. In cases where damage control has been necessary, the trauma surgeon must entertain the possibility of missed injuries and be ready to intervene rapidly as indications develop.

Return trips to the operating room impose further risk for the patient. Asensio's study points to the difficulties dealing with penetrating thoracoabdominal injuries. It defines this severely injured patient population along with the pitfalls and potential areas of inappropriate management. Clearly a 31% mortality that increases to 59% in patients undergoing combined procedures should alert trauma surgeons to the complexity and the need for flexibility and surgical creativity demanded for the management of these injuries [50].

Conclusions

Thoracoabdominal injuries are synonymous with high injury severity. They pose great challenges as accessing the wrong body cavity for hemorrhage control may lead to further physiologic compromise of these severely injured patients which often present with acidosis, hypothermia, and coagulopathy. These injuries incur both high morbidity and mortality rates. Similarly, not many trauma surgeons or trauma centers have developed significant experiences with their management.

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19 Principles of ICU Resuscitation and Team-Based Care

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Introduction

Thoracic injury is a frequent occurrence following both blunt and penetrating trauma. Any thoracic organ system can be injured, including the aerodigestive tract, great vessels, and mediastinum, in addition to the lungs and chest wall. Patients can present anywhere along the clinical spectrum from asymptomatic to severely symptomatic and hemodynamically unstable. In critically injured patients, management and ongoing evaluation should be conducted simultaneously.

Injured patients with significant degrees of shock, pulmonary dysfunction, alterations in organ function, or who are at high risk of progression of organ dysfunction benefit from resuscitation and management within an ICU. Obtaining optimal outcomes in these critically injured patients requires a high degree of coordinated, anticipatory, and precisely delivered care in multiple areas of patient management. An overview of these important management areas will be discussed, including:

- Initial evaluation and management.
- Assessment and management of shock and hypoperfusion.
- Pulmonary support and avoidance of lung injury.
- Approach to pain control.
- Surgical stabilization of rib fractures.
- Team-based ICU care.
- Ancillary aspects of ICU management.
  - Tertiary survey.
  - ICU liberation bundle.
  - Adequate pain control.
  - Sedation and delirium management.
  - Daily spontaneous awakening and breathing trials.
  - Early mobility.
  - Nutritional support and glycemic control.
  - Deep vein thrombosis prophylaxis.

Initial Evaluation and Management

ICU care frequently begins in the trauma bay. Following arrival in the trauma bay, all patients should be assessed rapidly according to ATLS protocol, with evaluation of airway, breathing, and circulation. Patients with chest trauma can present with acute life-threatening injuries that require immediate intervention, including tension pneumothorax, massive hemothorax, tracheobronchial injury, or cardiac injuries.

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After ruling out these acutely life-threatening injuries, other thoracic injuries including simple pneumothorax, hemothorax, and pulmonary contusion not associated with acute respiratory compromise can be addressed and managed. Even in the absence of these specific injuries, rib fractures themselves are associated with increased morbidity and mortality, with more fractures being predictive of worse outcomes [1].

Following initial evaluation and treatment, disposition from the emergency department depends on patient physiology and suspected or established diagnoses. Patients with chest trauma are frequently critically ill from multi-system injuries, and these patients require careful evaluation with ongoing resuscitation and treatment. Patients with hemodynamic instability, a severe injury burden, or geriatric patients with significant co-morbidities often require admission to a surgical trauma ICU. Specific thoracic trauma criteria that suggest consideration for ICU admission include age $\geq$ 65 years old, severe injury pattern with complex rib fractures and/or pulmonary contusion, and evidence of poor pulmonary status (pulse oximetry <92% on room air and incentive spirometry $\leq$1 L) [2]. Patients with flail chest are also at high risk for requiring ICU management, with nearly 60% of patients requiring mechanical ventilation and over 20% requiring tracheostomy [3].

Assessment and Management of Shock and Hypoperfusion

Ongoing cellular hypoperfusion can contribute to organ system dysfunction and mortality following severe trauma. Shock, which is defined by inadequate global perfusion, may be overt or occult depending on the presence or absence of hypotension. While overt shock is readily recognizable by assessment of hemodynamic status, occult (compensated) shock is assessed by measures of inadequate cellular delivery such as elevated lactate, more negative base deficit, or low $ScVO_2$. Persistent shock contributes to oxidative stress and potentiates activation of the innate immune system and up-regulation of proinflammatory cytokines, exacerbating subsequent organ system injury and dysfunction. While hemorrhage is the most common etiology of shock following thoracic trauma, other etiologies such as cardiogenic, obstructive, and/or distributive shock can occur either in isolation or concurrent with hemorrhagic shock. It is important to recognize that severe hemorrhagic shock may directly contribute to cardiogenic dysfunction and a reactive distributive state due to the physiologic changes induced by hypoperfusion.

Ensuring adequate resuscitation in the ICU entails monitoring various surrogates of global oxygen delivery, including vital signs, laboratory values, and assessment of cardiac function and volume responsiveness. Close monitoring of trends in hemodynamic parameters can determine the patient's response to interventions and guide further interventions. There is a wide spectrum of monitoring options, ranging from non-invasive to minimally invasive and invasive techniques. However, there is no one perfect test or measurement for determining successful resuscitation. The critical care team must understand the strengths and limitations of the available tests and integrate the different parameters into a comprehensive management plan tailored to each patient.

Non-invasive Monitoring: Vital Signs and Ultrasound

Heart rate, blood pressure, and urine output are readily available non-invasive measurements that can be easily monitored for each patient. Normalization of blood pressure and heart rate, as well as adequate urine output, are simple markers of adequate resuscitation. However, there are significant limitations to relying only on vital signs, and therefore vital signs should not be used in isolation.

Bedside cardiac ultrasound can be used as a dynamic study to assess fluid responsiveness (volume status) and basic cardiac function, because response to interventions can be observed in real time [4]. The Society of Critical Care

Medicine recommends using bedside cardiac ultrasound for gross assessment of ventricle function, in addition guiding fluid resuscitation by measuring relative change in IVC diameter with respiration for ventilated patients [5]. Ultrasound is portable, repeatable, and non-invasive, and it is useful for determining intra-vascular volume status and contractility. However, it is limited because it is not able to determine adequacy of perfusion.

Minimally Invasive Monitoring

Minimally invasive monitoring includes parameters measured from an arterial line, such as continuous arterial blood pressure and stroke volume variation, as well as central venous pressure, which is measured from a central venous catheter. Arterial lines allow continuous monitoring of blood pressure, in comparison to intermittent measurements with a blood pressure cuff. Arterial waveform parameters specifically stroke volume variation (SVV), pulse pressure variation (PPV), and systolic pressure variation (SPV) can more reliably predict volume responsiveness compared to static parameters such as central venous pressure or pulmonary artery occlusion pressure.

If there is evidence of organ system dysfunction, such as decreased urine output, central venous pressure (CVP) can help determine the next steps in resuscitation. CVP approximates right ventricular end diastolic volume, and it is a helpful supplement in conjunction with other markers, particularly when trended. However, many factors can affect the accuracy of CVP. If CVP is used to monitor resuscitation, it should be trended and should be considered in the context of other measurements.

Invasive Monitoring

Invasive monitoring, now used infrequently, can be obtained from pulmonary artery catheters that measure venous oxygen saturation (SvO_2) continuously. Venous oxygenation saturation is a marker of oxygen utilization and is also helpful when used in conjunction with lactate. SvO_2 is a dynamic parameter, and once catheter access is established, it can be followed real time at the bedside to evaluate response to clinical interventions. It can serve as an early marker for hypoperfusion, as low SvO_2 can be detected even before derangements in vital signs [6]. In addition, normalized SvO_2 is a better marker for survival than driving oxygen delivery to supranormal values [7].

Serial Laboratory Assessment

Trending laboratory values, including base deficit and lactate, is another component of resuscitation assessment. Higher lactate and more negative base deficit on admission are both associated with increased mortality [8]. Lactate, a marker of global perfusion, is a useful guide when poor oxygen delivery is secondary to hypovolemia or low cardiac output [4]. It should be followed at least every 6 h, and potentially more frequently based on the clinical scenario. In addition, resolution of elevated lactate within 6–24 h is associated with decreased mortality [9, 10]. Negative base deficit on admission is a sensitive marker for hypoperfusion on admission and is also a marker for ongoing hemorrhage and increased need for volume resuscitation [11].

Pulmonary Support and Avoidance of Lung Injury

All patients with chest trauma requiring admission must be assessed for pulmonary functional status and need for supplemental oxygenation and/or ventilatory support. Different patterns of thoracic trauma have different likelihoods for requiring intubation. Patients with impaired oxygenation and ventilation secondary to an open pneumothorax, massive hemothorax, or flail chest may require urgent or emergent intubation. The combination of multiple rib fractures and ventilation perfusion mismatch secondary to pulmonary contusions may also lead to acute respiratory failure and need for intubation [12, 13].

Common indications for intubation include major chest trauma (abbreviated injury score 3 or greater) and evidence of respiratory failure, including respiratory rate < 9 or >25–30, PaO_2/FiO_2 ratio < 280, pulse oximetry <90%, or abnormal arterial blood gas values (PaO_2 < 60 mmHg, $PaCO_2$ > 45, pH < 7.2) [14, 15]. However, each patient may respond differently to the same injury, and pre-existing conditions must be considered. A patient with poor underlying physiology (pre-existing pulmonary disease, geriatric patient with poor reserve) may require intubation for an injury pattern that might be more easily tolerated by a young healthy patient. Guidelines recommend early tracheostomy for patients who are predicted to require >7 days of mechanical ventilation [16].

Acute respiratory distress syndrome (ARDS) is a common sequelae of trauma, in particular chest trauma. Trauma patients are also at risk for severe hypoxemia due to pulmonary contusions. Careful ventilator management, minimizing barotrauma, volutrauma, and atelectrauma, is key to preventing further pulmonary insult [12]. This involves minimizing tidal volume (target of 6–8 mL/kg), decreased plateau pressure (<30 cm H_2O), and utilizing recruitment maneuvers and positive end expiratory pressure (PEEP) to promote open lung ventilation. For patients who develop ARDS, using ventilatory protocols, such as the ARDSNet protocol, is important to optimal patient care. Airway pressure release ventilation (maximizing mean airway pressure to improve oxygenation), prone positioning, pulmonary vasodilators, and extracorporeal membrane oxygenation (ECMO) are options for patients unable to be managed by conventional ventilation.

For patients who do not require intubation, aggressive non-invasive pulmonary interventions may be necessary to prevent physiologic collapse leading to the need for intubation. Volume expansion therapies (e.g. incentive spirometry and intermittent positive pressure breathing [IPPB]) and secretion clearance therapies (e.g. chest physical therapy, positive expiratory pressure [PEP], and oscillating PEP [acapella]).

Approach to Pain Control

Pain control is fundamental in the care of blunt thoracic trauma. Chest wall pain leads to inadequate inspiratory function (splinting). Inadequate pain control limits patient participation in respiratory therapies required for pulmonary hygiene, such as coughing, use of volume expansion devices, and ambulation. Patients with poor pain control are at a higher risk of post-traumatic pneumonia and atelectasis.

Pain management has evolved from monotherapy with opiates to a true multi-modal approach. The 2016 EAST guidelines recommend that epidural analgesia and multi-modal analgesia are likely better than opioids alone for pain control [17]. There are various drug classes that can be incorporated into a multi-modal regimen [18], including non-steroidal anti-inflammatory drugs (NSAIDs), acetaminophen, neuropathic agents (gabapentin, pregabalin, tricyclic anti-depressants, anti-convulsants), alpha 2 agonists (clonidine and dexmedetomidine), muscle relaxants (cyclobenzaprine, methocarbamol), and anesthetics (topical lidocaine) [19]. In addition, methocarbamol has been shown to decrease hospital length of stay for patients with rib fractures [20]. Another alternative is intravenous ketamine, which has been demonstrated to decrease opioid requirements for patients with injury severity score > 15 [21]. Non-pharmacologic therapies such as cryotherapy, hypnosis, massage, and music can also be useful adjuncts [19].

If pain remains poorly controlled despite multi-modal systemic therapy, locoregional pain management strategies should be considered. Partnership and consultation with an anesthesia/pain management service can offer access to advanced techniques for improving pain control, with subsequent improvement in pulmonary function. Catheter-based analgesia (epidural or paravertebral) and other regional anesthesia techniques, such as erector spinae plane block (ESP), offer additional opiate sparing therapeutics options and have been shown to reduce delirium

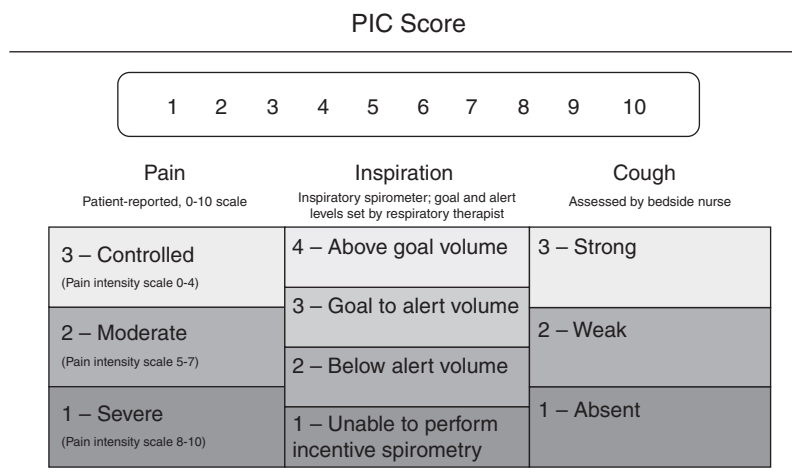

Fig. 19.1 PIC score

in geriatric patients with rib fractures. Epidural anesthesia has been shown to be associated with decreased mortality for patient with three or more rib fractures [22]. ESP is an inter-fascial paraspinal approach for regional anesthesia [23]. Under ultrasound guidance, local anesthesia is infiltrated below the erector spinae muscles, with the goal of blocking the ventral and dorsal rami of the spinal nerves. At our facility, we previously used epidurals, but we now have multiple providers, including emergency department physicians and anesthesiologists, who are performing erector spinae plane block for our patients following blunt chest trauma. We are currently awaiting approval of a study protocol designed to evaluate the impact of ESP on incentive spirometry, pain score, and opioid requirements.

Monitoring response to adjustments in analgesic regimen is necessary to assess effectiveness of pain control. The numeric pain scale is probably the most widely utilized method to assess pain. For patients who are unable to verbalize their pain, which is a common occurrence in the ICU, the behavioral pain scale and the critical care pain observation tool are reliable alternatives [19]. Improvement in pulmonary function can serve as a surrogate for adequacy of pain control. The PIC score, developed in 2015, incorporates pain, incentive spirometer volumes, and cough strength as an indication of pulmonary function [24]. A higher PIC score indicates less pain, better incentive spirometry, and stronger cough (Fig. 19.1).

Surgical Stabilization of Rib Fractures

Patients with rib fractures may benefit from operative fixation. There are various characteristics that may suggest a potential for benefit from surgery. Indications for operative fixation of rib fractures remain controversial. Proposed indications include flail chest, failed ventilator weaning, paradoxical chest movement, poorly controlled pain, significant chest wall deformity with collapsed chest wall or severely displaced ribs, symptomatic non-union of rib fractures, or patient undergoing thoracotomy for another indication [25–30]. The 2016 EAST Guidelines, based on a review of 22 publications, recommend consideration of surgical fixation to decrease morbidity, but simultaneously report that the ideal patient subset that would benefit from operative management is currently undefined. The Western Trauma Algorithm recommends considering early operative fixation for patients with >2 acute rib fractures and >65 years of age without other injury that would require prolonged intubation or immobility [2].

Team-Based ICU Care

Critical care management of severely injured patients is optimized through a team-based approach. Bundled care is not a new concept—injury specific clinical pathways have been implemented as early as 1998, and several studies have documented improved outcomes for patients with thoracic trauma managed in a clinical pathway [31, 32]. The ICU liberation bundle, frequently known as the A-F bundle, incorporates early mobilization, sedation holidays with spontaneous awakening and breathing trials, and delirium prevention and treatment. The bundle encourages multi-disciplinary care for patients, and adoption of the bundle has been shown to improve clinical outcomes [33]. Following implementation of a multi-professional acute trauma health care (mPATH) team at our facility, we were able to demonstrate decreased length of stay for neurologically injured patients requiring tracheostomy [34]. This approach clearly has benefits that extend beyond just the neurologic trauma population and should be utilized in the care of chest trauma patients as well.

The key members of the team include the provider team (intensivist, residents, advanced practice providers), the bedside nurse, respiratory therapist, and physical therapist. This team can be augmented by anesthesia, clinical nutritionists, clinical pharmacy specialists, and other ancillary teams, depending on resource availability.

The critical care nurse plays a key role in the overall care of the patient. Nursing interventions for patients with thoracic trauma include assessment of parameters of respiratory function (pulse oximetry, respiratory rate, strength of cough). Identification of improvement or decline in ability to cough or use incentive spirometer provides an objective measurement of pulmonary function. Nurses are also key to the process of assessing pain and relative effectiveness of analgesic regimen [13]. They often have more frequent interaction with the patient at the bedside, and they can provide a more descriptive assessment of the patients comfort over several hours, which can be more informative than the numeric pain score in isolation. Additionally, nurses encourage participation in pulmonary therapy and physical therapy, in close coordination with the respiratory therapist and physical therapist, respectively. Early mobilization improves pulmonary function, and it is a key component of the ICU bundle.

Respiratory therapists play an integral role in the evaluation and management of patients with thoracic trauma, beginning in the trauma bay and extending to the ICU. For non-intubated patients, they can provide individualized therapy plans to optimize pulmonary physiology. For intubated patients, coordination between the intensivist and the respiratory therapist is vital for targeted approach to ventilator mechanics. Respiratory therapists offer vital insights into pulmonary compliance, effectiveness of adjustments in ventilator settings, and suggestions for additional therapeutic interventions.

Ancillary Aspects of ICU Management

In addition to the specific management strategies directed at the sequelae of chest trauma, there are several additional elements of ICU care that need to be addressed in these patients, including ongoing assessment for injuries, with the tertiary survey, incorporating the components of the A-F bundle, providing appropriate nutrition as well as implementing DVT prophylaxis.

The tertiary survey is a key component of the ongoing assessment of patients requiring ICU admission. Severely injured patients frequently have additional injuries that can be overlooked initially, especially when life-threatening injuries are present. It is important to complete a thorough exam to identify other injuries. Diagnosing injuries in the tertiary survey reflects appropriate triage, with initial assessment and intervention directed most life-threatening injuries.

The ICU liberation bundle is a key component of ICU care [35]. As we have increased our awareness of the detrimental effects of ICU delirium, prolonged mechanical ventilation and excess sedation, the ICU bundle is a helpful tool

to incorporate protocols to improve patient outcomes. The bundle includes daily spontaneous awakening/ breathing trials, which have been shown to decrease duration of mechanical ventilation. In addition, the bundle emphasizes adequate pain control, minimizing sedation, avoiding delirium and early mobility. Benzodiazepines were previously a common choice for sedation in the ICU, but they have been shown to be associated with increased incidence of delirium [36]. Other sedative options include anti-psychotics, propofol, and dexmedetomidine. Mobility is important for several reasons. Not only does it combat the muscle wasting and weakness that frequently accompany ICU stays, but it is also associated with a decreased duration of delirium.

Ensuring adequate early enteral nutrition is crucial [37]. The preferred route of administration is oral, then enteral followed by parenteral if the enteral route is inadequate by 7 days. Gastric feeding is the standard approach for initiating enteral support, but post-pyloric is preferred for patients who are intolerant to gastric feeds or have a high risk for aspiration. Optimizing glycemic control is another component of optimal patient management. Stress induced insulin resistance is a common sequelae following trauma, and outcomes are improved with glycemic control. Current recommendation targets a glucose range of 140–180, with a goal of avoiding hypoglycemia and severe hyperglycemia.

Appropriate venous thromboprophylaxis is vital to appropriate care. Low molecular weight heparin is the recommended therapy [38]. Clinical nutritionists are key members of the ICU team, as they advocate for appropriate and adequate nutritional support. Clinical pharmacy specialists lend their expertise in appropriate dosing of medications for glycemic control as well as venous thromboprophylaxis.

Trauma Practice Guidelines

At our institution, we have developed an algorithmic approach to blunt chest wall trauma, and it is similar to other institutional protocols, such as the Rib Fracture Management Protocol at Harborview Medical Center [1]. Our algorithm (Fig. 19.2) begins during initial assessment in the emergency department, with evaluation for admission to the regular ward or the ICU. Frail or geriatric patients, patients with underlying pulmonary dysfunction, or patients with pulse oximetry <92% on room air and/or incentive spirometry <1 L are considered for ICU admission.

We have an aggressive pain management protocol, based on a three-tier system of increasing multi-modal analgesic regimens for more severe pain, with dosing adjustment for the geriatric population to minimize risk of adverse medication effects (Table 19.1). If patients are on the highest tier or have weak cough and/or poor incentive spirometry, the pain management service is consulted to evaluate the patient for regional anesthesia (intercostal blocks, epidural, ESB, etc.). Patients with complex fracture patterns (3 or more fractures, 2 or more displaced fractures, flail segment, or chest wall deformity) are referred for consideration for operative rib fixation. We monitor patients with our modified version of the PIC score, which is an inversion of the original PIC score, with the hope of paralleling the universally understood numeric pain score (i.e., higher modified PIC score indicates poorer pulmonary physiology, including more severe pain, less incentive spirometry volume, and weaker cough) (Table 19.2). Patients with persistently elevated modified PIC score also receive consultation by our rib plating service.

Conclusion

Thoracic injury following blunt or penetrating trauma is common. Depending on injury severity, patient stability, and presence of multi-system trauma, patients may require ICU admission for optimal ongoing resuscitation, evaluation, and treatment. Patient care can be optimized with adherence to simple protocols, which can be facilitated by a multi-disciplinary team approach to ensure comprehensive management.

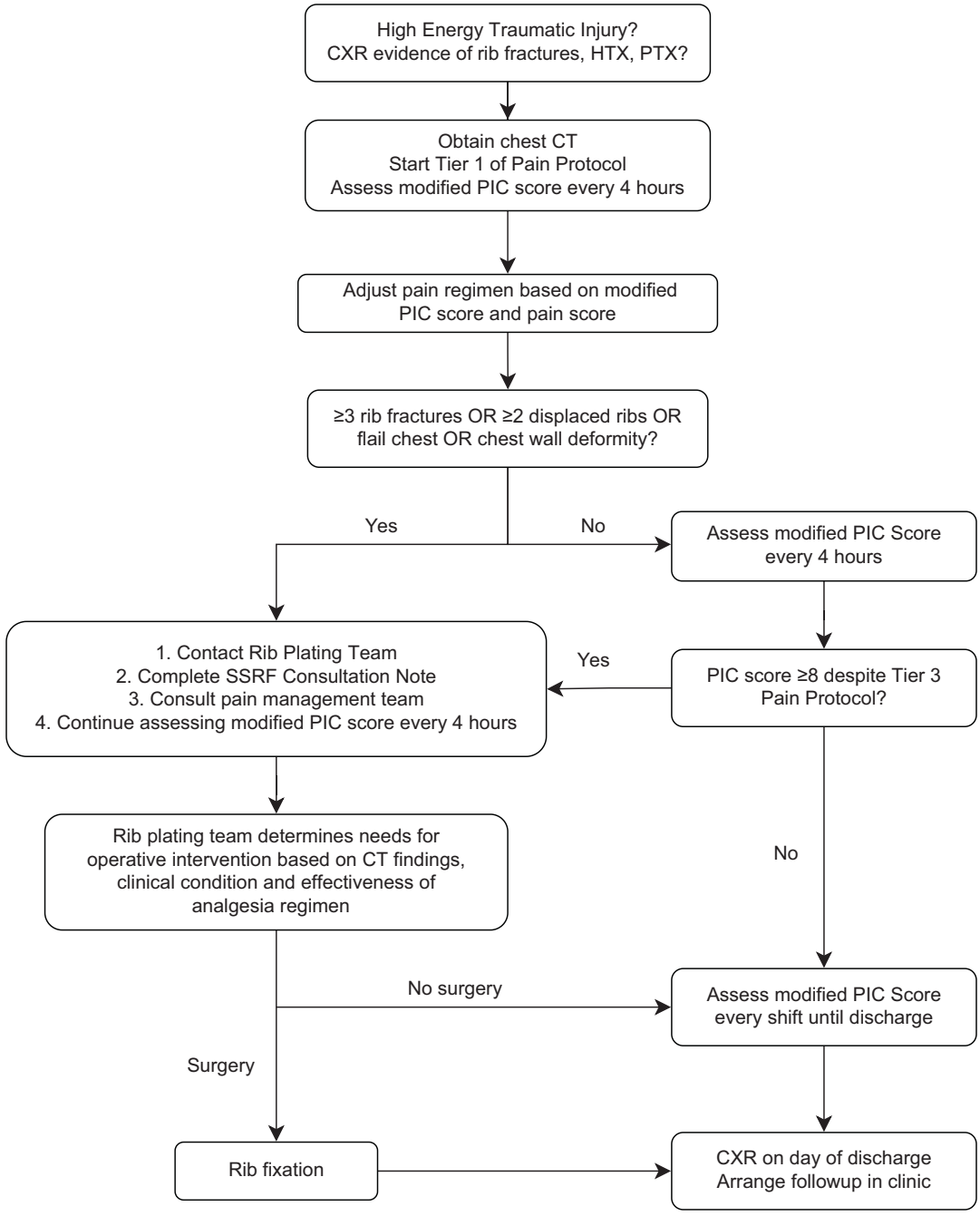

Fig. 19.2 Carolinas medical center blunt chest trauma clinical practice guidelines

Table 19.1 Blunt chest trauma pain protocol. Patients with blunt chest trauma are started on the therapies included in Tier 1, at a minimum. If their modified PIC score remains elevated, their regimen is increased through Tier 2 and Tier 3 as needed

Tier 1
Ibuprofen 600–800 mg PO q6h
Acetaminophen 650 mg PO q6h
Oxycodone 2.5 mg PO q4h PRN
Topical lidocaine patches
Gabapentin 300 mg PO q8h
Cryotherapy (ice packs)
Breathing coach
Tier 2
Tier 1 agents PLUS
Methocarbamol 500 mg PO q8h
Ketorolac 15 mg IV q6h for 5 days (stop ibuprofen)
Increase to oxycodone 5 mg PO q4h PRN
Hydromorphone 0.25 mg IV q2h prn
Tier 3
Tier 2 agents PLUS
Consult pain service and rib plating team
Consider opiate PCA
Consider long acting opiate

Table 19.2 Modified PIC score. Scale is inverted relative to the original PIC Score, but the scores are calculated similarly. Lower pain scores, stronger cough and higher incentive spirometer (IS) volumes achieved are associated with better pulmonary physiology

Pain	IS	Cough
Pain 0–3 (1 pt) Pain 4–7 (2 pts) Pain 8–10 (3 pts)	>1.5 L (1 pt) >1–1.5 L (2 pts) 500 mL to 1 L (3 pts) <500 mL (4 pts)	Strong (1 pt) Weak (2 pts) Absent (3 pts)

3–4 points = ideal pulmonary physiology
5–7 points = moderate pulmonary physiology
8–10 points = poor pulmonary physiology

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Coagulopathy Management and VTE Prophylaxis

20

Sean T. Dieffenbaugher, Bruce A. Crookes, and Heather L. Evans

Trauma Induced Coagulopathy

Injury represents a major cause of death and disability in adults despite continued advances in trauma systems, injury care, and critical care. Besides the direct consequences of blunt or penetrating injury, other clinically relevant abnormalities are engendered that impact injury care. Clinically significant coagulation abnormalities are evident in up to one-quarter of all injured patients upon acute care facility arrival. Coagulation abnormalities have been extensively studied and collectively described in the post-injury period as Trauma Induced Coagulopathy (TIC), Acute Traumatic Coagulopathy (ATC), and the Acute Coagulopathy of Traumatic Shock (AcoTS). The relevance of identifying post-injury coagulopathy directly relates to both resource utilization and outcome. This novel coagulopathy engenders accelerated component transfusion volume, increased hospital, ICU, and mechanical ventilation lengths of stay, increased incidence of multiple-organ dysfunction syndrome, and four-fold greater index admission mortality [1, 2]. Understanding the genesis of TIC, current methods of management, and their impact on patient and trauma center relevant outcomes is essential while pursuing optimal outcomes.

Etiology and Pathophysiology

Normal coagulation processes involve complex, coordinated, and balanced hemostatic and antithrombotic mechanisms. Injury causes direct—and induces indirect—endothelial injury. Both of those processes influence the formation of a platelet plug and clot propagation as well as antithrombotic controls that terminate clotting and subsequently initiate fibrinolysis and clot dissolution. These adaptive processes are impacted by the self-propagating classic "lethal triad" of acidosis, hypothermia, and coagulopathy. Acidosis has a variety of etiologies including most notably hemorrhagic or hypovolemic shock accompanied by hypoperfusion and anaerobic metabolism. Intravenous fluids with supra-physiologic chloride concentration as well as direct tissue injury are additional important sources. Acidosis decreases the reaction rate of plasma serine proteases and may maladaptively influence membrane fluidity, compromising the integrity of the coagulation cascade and platelet degranulation.

Hypothermia similarly impairs coagulation. Hypothermia may reflect environmental expo-

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sure, prolonged transport, and the infusion of resuscitation fluids—including unwarmed or inadequately warmed component blood transfusion products. Coagulopathy additionally stems from external factors including large plasma volume expansion related hemodilution as well as active consumption or maladaptive fibrinolysis. Key goals for resuscitation are to prevent the triad from becoming established or mitigate it if present. Such goals have helped establish the conceptual underpinning of "damage control" or "hemostatic" resuscitation. Reductions in crystalloid volume, as well as 1:1:1 component transfusion goals for those requiring massive transfusion are direct consequences of such an approach to coagulopathy management. While those measures support clotting, they do not reverse undesirable fibrinolysis.

Trauma induced coagulopathy stands as a distinct phenotype characterized by hyperfibrinolysis, hypocoagulability, and impaired hemostasis. The mechanism underlying TIC has been extensively studied and demonstrates a complex, interwoven relationship between biochemical factors and endothelial characteristics without a clear causative relationship. Activated protein C is reported as a significant mediator of TIC. Circulating protein C is activated when binding endothelial protein C receptors in the presence of thrombin–thrombomodulin complex and protein S. The resultant complex initiates anticoagulant, profibrinolytic, and cytoprotective responses. Quantitative platelet defects and qualitative platelet dysfunction after injury also comprise additional features of TIC [3].

Diagnosis and Laboratory Quantification

Standard laboratory profiling after injury includes complete blood count, complete metabolic panel, arterial blood gas, and coagulation assays including prothrombin time, international normalized ratio, activated partial thromboplastin time, fibrinogen concentration, and D-dimer concentration. While the rest of the profile is broadly applicable to patients requiring acute hospitalization, coagulation competency assessment is particularly important after injury. Nonetheless, the aforementioned assays provide less information than desired and engender an obligate interval between acquisition, lab transport, and assay completion. This interval can be up to an hour after the samples have been obtained. Moreover, the specific assays are principally targeted to monitoring therapeutic agent activity or complementing the evaluation of potentially hereditable coagulopathies. Furthermore, standard coagulation profiling is unable to establish or exclude the presence of TIC. Thus, the limitations of standard coagulation laboratory assessments impede clinical decision-making in acutely bleeding patient. Instead, timely assays that provide real-time data regarding how the elements of the coagulation system function are required to guide acute therapy in the setting of injury and associated hemorrhage. Such assays are known as viscoelastic hemostatic assays (VHAs).

VHAs provide rapid functional assessments of clot initiation, clot formation, clot strength, clot stability, and ultimately, the degree of breakdown or lysis. These point of care tests are available in two dominant forms: thromboelastography (TEG) and rotational thromboelastometry (ROTEM). As a functional test of clot properties, viscoelastic testing can be utilized for the initial recognition and guided therapeutic intervention for coagulopathy correction. Repeated assay—performed in the trauma bay, OR, or ICU, allows for the rapid recognition of coagulopathy resolution and the termination of component therapy transfusion [4]. The clinician can assess coagulopathy related to factor deficiency, hemodilution, hypofibrinogenemia, platelet dysfunction, and fibrinolytic activity, including hyperfibrinolysis and fibrinolytic shutdown. Moreover, instead of only receiving numeric values, TEG and ROTEM both provide a graphic analysis of the dynamics of clotting supporting pattern recognition as the graph evolves (Fig. 20.1). While TEG and ROTEM provide similar kinds of information, the traces and nomenclature have relevant differences (Fig. 20.2) [5].

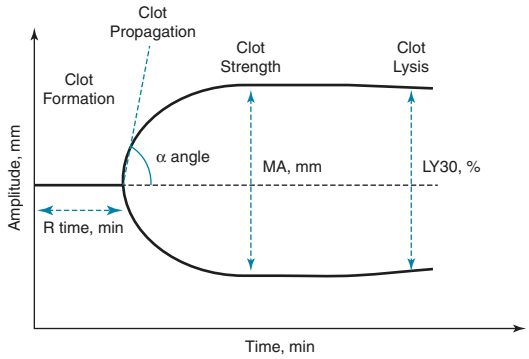

Fig. 20.1 This graphic depicts the features of a normal thromboelastogram tracing. Reproduced with permission from: Subramanian M, Kaplan LJ, Cannon JW. Thromboelastography-guided resuscitation of the trauma patient. JAMA Surgery 2019 Dec 1;154(12):1152–3

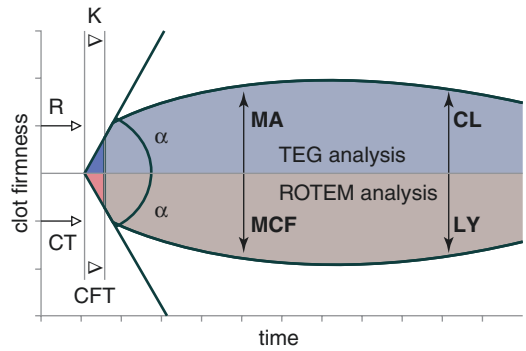

Fig. 20.2 This graphic depicts values and parameters obtained using TEG and ROTEM to facilitate comparison between the two techniques. TEG® and ROTEM® tracing TEG® parameters: *R* reaction time, *k* kinetics, ∝ alpha angle, *MA* maximum amplitude, *CL* clot lysis. ROTEM® parameters: *CT* clotting time, *CFT* clot formation time, ∝ alpha angle, *MCF* maximum clot firmness, *LY* clot lysis. Reproduced with permission using the Creative Commons license from: Sankarankutty, A., Nascimento, B., Teodoro da Luz, L. et al. TEG® and ROTEM® in trauma: similar test but different results?. *World J Emerg Surg* 7, S3 (2012). https://doi.org/10.1186/1749-7922-7-S1-S3

Treatment of Trauma Induced Coagulopathy

Early recognition of hemorrhagic shock coupled with prompt resuscitation and rapid hemorrhage control are essential components of managing the injured patient with hemorrhagic shock. Resuscitation ideally incorporates damage control resuscitation (DCR) techniques to address trauma induced coagulopathy as well. DCR limits crystalloid fluid administration, preserves permissive hypotension until hemorrhage control is achieved, and engages early balanced component therapy transfusion and avoids hypothermia [6]. In this way, DCR is reasonably known as "hemostatic" resuscitation.

Liberal plasma volume expansion using crystalloid-based solutions to restore deranged hemodynamics was previously commonplace. With careful study, unrestricted crystalloid-based resuscitation incites a number of untoward effects whose effects may be magnified in the setting of hemorrhagic shock. Besides exacerbating acidosis and coagulopathy, crystalloid-driven large volume plasma expansion impacts multiple-organ systems—most notably cardiac, pulmonary, gastrointestinal, and renal—but also induces salt and water excess complications related to capillary leak. Indeed, secondary abdominal compartment syndrome and acute respiratory distress syndrome (ARDS) are linked to fluid overload—as is mortality [7, 8]. As a corollary, deresuscitation appears to improve outcomes in those who have acquired fluid overload in the setting of septic shock and the perioperative period that is relevant to trauma [9, 10]. Importantly, as we learn more about genotypes and phenotypes, immune dysfunction may be driven, or at least deranged, by excessive crystalloid-based resuscitation and may be positively impacted by evolving therapeutics such as peritoneal resuscitation [11]. Thus, replacing liberal crystalloid resuscitation strategies with balanced biologically active colloid resuscitation may help mitigate against potentially avoidable secondary effects [12].

Permissive hypotension confers a protective effect by directing resuscitation efforts to maintain a systolic blood pressure of approximately 90 mmHg. This relative hypotension has been demonstrated to confer a protective effect and limit hemorrhage by decreasing new hemorrhage from recently clotted vessels. This principle hinges on decreasing intravascular hydrostatic pressure and preventing dislodgment of hemostatic clot in addition to avoiding dilutional coagulopathy and hypothermia associated with

crystalloid resuscitation. This strategy balances organ perfusion with hemostasis in the acute resuscitation period until definitive hemorrhage control is obtained [13, 14]. Hemorrhage control relies on having a functional clotting system to maintain control from non-surgical hemorrhage sites and to support tissues controlled using electrocautery or argon-beam therapy as well.

Strategies for the transfusion of blood components in the setting of post-injury hemorrhage have been extensively studied in military and civilian populations. Damage control resuscitation hinges on the balanced infusion of PRBC, plasma, and platelets in a 1:1:1 ratio. Early administration of this hemostatic, balanced resuscitation strategy decreases mortality without downstream mortality from component therapy transfusion [15, 16]. Subsequently, the PROPPR randomized trial compared ratios of 1:1:1 to 1:1:2 and demonstrated no improvement in 24 h survival but did demonstrate improved hemostasis and fewer early deaths by exsanguination without transfusion associated safety concerns in the 1:1:1 patient cohort [17]. This empiric resuscitation strategy improves platelet function and clot formation while decreasing inflammation, edema, and vascular endothelial dysfunction [18]. Institutional massive transfusion protocols adopting this balanced ratio of blood products should be employed for the most severely injured trauma patients [19]. Additionally, VHAs should be employed within massive transfusion protocols to facilitate goal directed resuscitation and to curtail component transfusion when hemostasis is achieved [20].

Massive transfusion most commonly relies on the rapid delivery and infusion of components from the blood bank. Such products are stored cooled and may induce iatrogenic hypothermia or exacerbate existing hypothermia if they are infused without using a blood product warmer. A notable exception is the military where "buddy" transfusion is well documented—a process that maintains clotting component activity while delivering a body temperature resuscitative fluid [21]. The success of whole blood transfusion at the point of injury has also supported civilian trauma centers to use fresh whole blood for the most critically injured patients who are believed to have hope of salvage [22]. Civilian fresh whole blood is stored cool and benefits from rewarming as well. Complementary efforts to avoid hypothermia, or help regain normothermia, include but are not limited to increased ambient temperature, warmed endotracheal gas, warm lavage solutions, as well as active and passive external warming systems [6]. For those patients who require thoracotomy for emergency department resuscitation, the period of transport to the OR represents a period of thermal vulnerability. The exposed heart is especially sensitive to hypothermia-induced dysrhythmia and should be bathed in warm lavage solution en route. The component of hepatic function that is related to cardiac performance to support the elaboration of new clotting factors may be underappreciated.

Hemostatic Adjuncts

While packing of laparotomy pads served as a standard means of helping create tamponade during operative intervention, such pads did not support coagulation. Adjuncts such as thrombin, gelfoam, and oxidized cellulose were also employed in smaller areas of non-surgical bleeding such as around vascular anastomoses. However, none of those was suitable for large areas, and all exert only local effects. The notion of being able to locally support clotting led to the development of agents that could be directly applied to wounds or embedded in wound packing or wound dressings. Initially pioneered in the military domain, procoagulant dressings are now widely available for acute care facility and layperson use [23]. Crafted in a variety of fashions and using an array of materials, the specifics are beyond the scope of this chapter and several reviews are available [24]. None of those adjuncts requires knowledge of a specific patient's clotting competency, while pharmacologic adjuncts benefit from obtaining that precise information for targeted administration and cessation.

The increasing availability of viscoelastic assays enables expeditiously assessing a given patient's precise state of fibrinolysis. VHAs can

signal hyperfibrinolysis characterized by excessive clot degradation with hemorrhagic consequences, physiologic (normal) fibrinolysis, as well as fibrinolytic shutdown characterized by localized thrombotic consequences. The distribution of fibrinolytic profiles described by the Denver group includes 64% shutdown, 18% physiologic, and 18% hyperfibrinolysis [25]. Importantly, increased mortality was noted for those with fibrinolysis shutdown (17%) as well as much accelerated mortality with hyperfibrinolysis (44%) establishing the need for acute intervention [25]. Patients noted to demonstrate hyperfibrinolysis should be treated with antifibrinolytic therapy.

Tranexamic acid (TXA) is the most extensively studied antifibrinolytic agent after injury. TXA serves to inhibit fibrinolysis by blocking the interaction of plasma plasminogen to fibrin, thus inhibiting clot dissolution. The CRASH-2 trial demonstrated a decrease in both all-cause and hemorrhage related mortality without increased thrombotic complications in a large civilian population [26]. Notable limitations included patient selection with large number needed to treat, inciting much debate about appropriate TXA utilization. Upon confirmation of a hyperfibrinolytic phenotype with LY-30 > 3%, and a post-injury time frame of less than 3 h, TXA administration demonstrates improved outcomes [27, 28]. Accordingly, the Eastern Association for the Surgery of Trauma practice management guidelines conditionally recommend early TXA administration as an adjunct for hemorrhage control in severely injured patient with hemorrhagic shock [19]. Relatedly, the CRASH-3 trial demonstrated improved survival for those with hyperfibrinolysis after mild-to-moderate traumatic brain injury who received treatment with TXA within 3 h of injury compared to placebo [29]. Thus, VHA driven assessment coupled with pharmacologic intervention support survival in the setting of hyperfibrinolysis.

Fibrinolysis shutdown and untoward clotting is not a new concept or finding. Recently, these findings have been widely explored during COVID-19 further expanding our understanding of its underpinning molecular mechanisms [30]. Therapeutic agents directed at management span streptokinase, urokinase, stanozolol, sequential compression devices with and without unfractionated IV heparin but none has demonstrated efficacy, and many—especially plasminogen activators—are contraindicated in those with TBI. Disparities in assessment tools, platelet activity, as well as controversy whether systemic fibrinolysis shutdown reflects local dynamics instead spur further inquiry and overlap with studies of post-injury endotheliopathy [31]. The recognition of fibrinolysis resistance also impedes discovering an appropriate therapeutic and may reflect increases in platelet activator inhibitor-1 (PAI-1) activity. All of this is impacted by widespread use of aspirin and other platelet inhibitors, especially in the elderly. While the impact of antiplatelet therapy is best studied in those with TBI, the implications for therapy are broadly applicable to those with chest injury as platelet transfusion has been associated with reduced progression of intracranial hemorrhage but also with increased mortality [32]. It is unclear if those who are on antiplatelet therapy have a decreased or increased risk of fibrinolysis shutdown. Regardless, there is no commonly accepted therapeutic intervention or consensus to guide clincial care.

Special Considerations

Addressing the reversal or management of outpatient anticoagulation, including therapeutic anticoagulation with vitamin K antagonists and with direct oral anticoagulants (DOACs) has become increasingly common in injured patient care. As direct thrombin (dabigatran) or factor Xa inhibitors (all others as of this chapter's writing), DOACs present an added level of complexity when assessing for coagulopathy. Therapeutic decision-making will reflect the presence of hemorrhage as well as the time since the last dose was ingested. Since the latter piece of information is often unavailable to the bedside clinician, and therapeutic reversal agents bear some risk of untoward clotting, decision-making often relates to hemorrhage location, rate, and the presence of

concomitant injuries that require therapy. Additionally, as technology evolves, VHAs or plasma concentration assays may soon be able to identify the presence of a DOAC to direct the need for therapeutic rescue.

Rescue strategies for those receiving warfarin are well described. While vitamin K is a slow reversal agent, fresh frozen plasma (FFP) is more rapid but engenders plasma volume expansion—an issue that does not usually impact the injured patient. On occasion, reversal is indicated due to over-anticoagulation (INR greater than the therapeutic window) in the absence of hemorrhage, but the patient is volume replete or has hypervolemia due to concomitant heart failure and cannot tolerate the required volume of FFP. Instead, prothrombin complex concentrate (both 3 and 4-factor formulations) can achieve identical results but at a much reduced plasma volume expansion cost [33]. Some DOACs may be reversed by pharmacologic antagonists. FDA approved reversal agents exist for dabigatran (idarucizumab) as well as apixaban and rivaroxaban (andexanet-alpha) [34]. Activated PCC may also be used to reverse Xa inhibitor DOACs [35]. There are reports of using factor 8 bypassing agent (FEIBA; approved to treat hemophilia A) to reverse DOAC-based anticoagulation as well [36]. Algorithms and management approaches have been articulated for the interested reader; note is made of the important influence of technologic advances in detecting coagulation abnormalities and their precise etiology [37, 38]. Finally, the clinician must carefully weigh the timely resumption of venous thromboembolism prophylaxis and subsequent therapeutic anticoagulation to avoid thrombotic complications after achieving hemorrhage control.

Venous Thromboembolism

Venous thromboembolism (VTE) remains a common complication for injured patients [39]. The clinical spectrum of disease encompasses silent thrombosis, incidental imaging findings, symptomatic venous thrombosis, and clinically significant embolic phenomena including life threatening pulmonary embolism. The clinical manifestations of VTE may be sufficiently vague that they overlap with symptoms of other commonly encountered diagnoses in the injured patient supporting the use of a clinical guideline to inform and reduce variation in clinician practice [40]. Indeed, robust prophylaxis regimens appear warranted since diagnosis may be challenging especially in a patient with injury, surgical intervention, or therapeutic agent effect that renders symptom reporting problematic or without fidelity [41]. Since thoracic injury may be accompanied by spinal cord injury, such patients may be incapable of reporting symptoms of venous thrombosis at all.

Incidence and Significance

The incidence of VTE, including both DVT and PE, is widely variable, ranging from 1.8% to 58% [39]. The timeline of diagnosis of pulmonary embolism is variable, with early diagnosis comprising a significant portion of trauma associated VTE. Up to 37% are diagnosed within the early period of hospitalization (1–4 days) while approximately 18% are diagnosed between hospital days 5–7. Pulmonary embolism occurring during the second week (days 8–14) and later both comprise an additional 21%. A further confounder includes the difficulty in discerning whether the pulmonary embolism flows from a remote clot locus or is instead a reflection of local injury. In the aggregate, VTE engenders a significant degree of morbidity and mortality including but not limited to prolonged length of stay, increased hospital costs, and the potential need for additional imaging or therapeutic intervention. These costs are augmented by repeated laboratory monitoring, especially for those managed using vitamin K antagonists. Additionally, short- and long-term management of VTE further complicates patient management, both in the perioperative inpatient period and the outpatient setting as it influences lab profiling, care frequency, and the timing of subsequent reconstructive or restorative procedures [42].

Etiology and Risk Factors

Virchow's classic triad (stasis, endothelial injury, hypercoagulable state) explains the predisposition for VTE after injury. Prolonged immobility regardless of cause contributes to venous stasis. Endothelial and tissue injury coupled with hypercoagulability—especially in those with fibrinolysis shutdown, as well as reduced protein C and antithrombin concentrations—all increase the risk of venous thrombosis [43]. While the endotheliopathy of trauma seems more related to hyperfibrinolysis—and may respond to TXA administration—the role of procoagulant therapy in subsequent VTE remains unclear [44]. In patients without hemorrhage—or after definitive hemorrhage control—early mechanical and chemical thromboprophylaxis remain the gold standard to reduce the incidence and consequences of VTE after injury.

Adherence to an institutional protocol to reduce VTE prophylaxis is informed by identifying high-risk features for VTE as well as those who may be harmed by early VTE prophylaxis. It is the latter aspect that drives the well documented reduced adherence to established VTE protocols. It is principally the chemoprophylaxis that is omitted in those who are assessed as high-risk for recurrent bleeding (i.e. packing around a solid organ) or bleeding within a space not amenable to external control or with unacceptable consequences (i.e. brain or spinal cord) [45]. Recent Trauma Quality Improvement Program analysis of just over 20,000 patients revealed that even with severe blunt TBI, chemoprophylaxis was safely administered within 48 h of admission in nearly half, and within 72 h in an additional 19%; slightly more than one-third received chemoprophylaxis later than 72 h [46]. It is important to note that the data was derived from those with TBI—a presumed higher risk injury than most thoracic injuries—and it is therefore applicable to those with thoracic injury who are at risk of venous thrombosis.

Conditions that are associated with venous clotting are well described and include but are not limited to admission >24 h, accelerated age, major TBI, spinal cord injury, pelvic fracture, major vascular injury, venous instrumentation, hypotension, and shock. Simplified risk assessment models can be used by the clinician to predict VTE risk, but some will advocate for a universal approach to VTE prophylaxis regardless of risk profile assessment [47]. Additionally, pre-existing conditions including prior history of VTE, hypercoagulable disease, malignancy, and obesity confer increased risk for in-hospital thromboembolic complications [48]. Thus, adherence to an institutional protocol to assure VTE prophylaxis is essential to help reduce post-injury clotting. For those seeking a precision approach to chemoprophylaxis, admission VHA assessment can be utilized to stratify VTE risk and help guide decision-making [49].

Little concern arises regarding the mechano-prophylaxis aspect of VTE prophylaxis. Historically, mechano-prophylaxis utilized both thrombo-embolic deterrent (TED) compression stockings and sequential compression devices (SCDs). Ideally, compression stockings should conform to a graduated compression construction rather than a single pressure gradient throughout. Acute inpatient care stockings may have less maximal compression and less of a compression gradient than what is available for outpatient continuing care. Note is made of recommendations that advocate for not using graduated compression stockings alone, but support their use with chemoprophylaxis [50]. Moreover, graduated compression stockings are optimally coupled with intermittent, sequential pneumatic compression devices. The major contraindication to not placing an SCD on any limb is the lack of a limb, large open extremity wounds, non-fixated extremity fractures, inaccessibility due to essential devices such as an external fixator, with a relative contraindication—for some—with a biologic vascular bypass conduit in the subcutaneous space. If the lower extremities are not available, upper extremity SCD use is supported. VTE prophylaxis ideally temporally couples both chemo- and mechano-prophylaxis. Recent data suggests improved outcome with VTE prophylaxis that is initiated within 24 h of admission in stable patients compared to later initiation intervals [51].

Therapeutic Agent Selection and Dose Considerations

The optimal agent, dose, and timing for initiation of chemoprophylaxis have been long and hotly debated. Current ACCP guidelines detail low-dose unfractionated heparin (LDUH) as therapeutically equivalent to low molecular weight heparin (LMWH). However, there is data supporting LMWH as a superior agent in the post-injury setting, especially for those with lesser degrees of injury [52]. Similar findings have been noted for those with TBI as well [46]. Regimens of enoxaparin 30 or 40 mg bid, as well as 40 mg daily appear both safe and efficacious but are ideally guided once they are initiated using anti-Xa levels with peak target concentrations of 0.2–0.4 IU/ml for prophylaxis. Regimens may be selected as a fixed dose, but the rising incidence of obesity has articulated weight-based dosing approaches for therapy initiation as well [53]. In patients with chronic kidney disease, or those with acute kidney injury, LDUH should be the preferred agent. Given the untoward impact of hypoperfusion on renal function, abnormalities of renal function should acutely prioritize LDUH as opposed to LMWH for safety.

While standard dosing initiation regimens exist, a higher starting dose of enoxaparin with monitoring of anti-Xa levels has been reported to provide improved protection in patients with features that increase venous thrombosis risk [54–57]. Clinical features that suggest the need for increased dose levels include male sex, younger age, increased creatinine clearance, obesity, and increased injury severity [58]. Relatedly, the Western Association for The Surgery of Trauma has evolved a clinical decision guide to aid in dose and agent selection but also includes consideration for adding antiplatelet therapy (aspirin 81 mg) in those with VHA identified hypercoagulability related to platelet function [59]. Decision-making and guidance may also benefit from input from a PharmD embedded in the critical care team, especially with regard to agent selection, dosing, and monitoring for interactions with other agents or organ systems.

Conclusions

Severely injured patients often manifest coagulopathy in conjunction with hemorrhagic shock. A variety of mechanisms underpin coagulopathy spanning consumption, dilution, acidosis, hypothermia, and hyperfibrinolysis. Increasingly, therapeutic anticoagulant agents and antiplatelet agents complicate hemorrhage and coagulopathy management in the acute setting. Rapid assessment supports evidence-based therapy for patients demonstrating abnormal clotting. Viscoelastic hemostatic assays are at the forefront of the currently available assessment tools and deliver rapid, readily interpretable visual data that informs clinical practice. These assays can also identify patients at high risk of untoward clotting and as such, supplement the clinician's assessment regarding initiation of venous thromboembolism prophylaxis after hemorrhage control. Chemoprophylaxis regimens continue to evolve and currently focus on low molecular weight heparin therapy guided by anti-Xa levels in injured patients. An institutional approach to identifying and managing coagulopathy to support hemostatic resuscitation should be coupled with a protocolized approach to preventing subsequent undesirable clotting in this unique patient population.

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21

Monitoring Strategy for the Operating Room and Intensive Care Unit After Thoracic Injury

Caoimhe C. Duffy, Gary A. Bass, and Meghan Lane-Fall

Introduction

Damage to the thoracic cage and its contents is common in the injured patient. This can occur when the injuries are confined to this single cavity or as part of the overall injury burden of the polytraumatized patient [1, 2]. The recent TRIPP Study (Trauma ICU Prevalence Project) reported that rib fractures were the leading indication for intensive care unit (ICU) admission in these patients, accounting for 41.65% of post-thoracic injury patients requiring critical care [3]. In a subset of these patients, the severity of injury to the intrathoracic viscera is either underappreciated at admission or blossom over the following 48–72 h [4–8]. The incidence of complications such as pneumonia, pulmonary contusion, delayed hemorrhage, or blunt cardiac injury appear to increase with the number of ribs fractured, the location of fractures, the degree of impairment of chest wall mechanics, as well as patient factors such as medical co-morbidities, cognitive impairment, and advancing age [1, 6, 9–12]. While the pattern of evolution of tissue damage and loss of function in patients may be predictable, identifying which patient will follow that pathway is less clear [1, 13]. The ability to promptly identify and abrogate complications arising from thoracic injuries can be reflected in the inter- and intra-hospital quality metric "failure to rescue" [13, 14]. At present, there remains a need to over-triage at-risk patients for enhanced monitoring in a critical care environment to mitigate the devastating consequences of unmonitored decompensation.

Several triage tools have been proposed as guides to clinical prognostication, such as the RibScore, SCARF, PIC Score (Fig. 21.1), and a vast array of elements including the number of rib fractures and metrics of pulmonary mechanics (such as Forced Vital Capacity (FVC) or Forced Expiratory Volume in 1 s (FEV_1)). Despite their intuitive attractiveness as prognostication tools, these have rarely been validated outside of their home institutions [1, 15–18]. Furthermore, many of the tools, such as the PIC Score, generate composite unitless scores from nomograms which aggregate several subjective variables, such as the pain visual analog scale (VAS), incentive spirometry effort, and the adequacy of cough [12]. In the absence of high-fidelity data-driven predictors of outcome, the best-available surrogates are active monitoring in a critical care setting and an abundance of caution [1, 4, 19]. To

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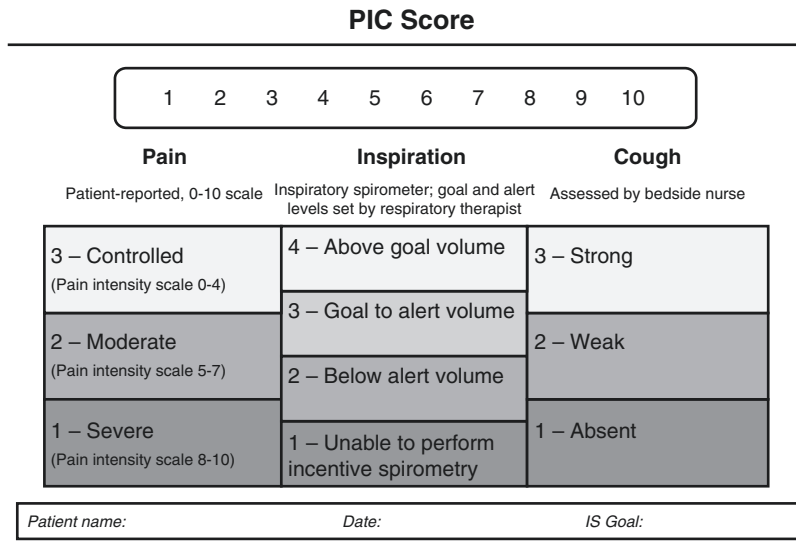

Fig. 21.1 Standard PIC score elements. The standard pain, incentive spirometry, cough (PIC) score for triage of patients with rib fractures that is currently in use in the authors' institution. Witt CE, Bulger EM. Comprehensive approach to the management of the patient with multiple rib fractures: a review and introduction of a bundled rib fracture management protocol. Trauma surgery & acute care open. 2017 Jan 1;2(1):e000064. Open access image use

that end, this chapter will review common monitoring techniques that may inform care in the ICU, the operating room (OR), and during intra- and inter-facility transport for patients with thoracic injuries.

Monitoring in the OR and ICU

Some monitoring modalities are standard across OR and ICU domains, while others are unique to the OR environment. Comprehensive post-thoracic injury care must accommodate or incorporate both, particularly if there is a need for high-flow nasal oxygen supplementation ($HFNO_2$), non-invasive positive pressure ventilation (NIV), or invasive mechanical ventilation, as well as during transitions of care. It is vital to understand the mechanism and physiology surrounding traumatic thoracic injuries, as the application of many monitors is routine and comprises a generic "safety net" approach, while others are driven by unique injury patterns or complexes. Careful monitoring within the ICU may prevent or reduce the impact of associated complications of thoracic injuries, such as progression towards the acute respiratory distress syndrome (ARDS) or the development of intracranial hypertension driven by hypercarbia [20, 21]. Monitoring the adequacy of hemodynamics as well as gas exchange in thoracic injury patients, regardless of intubation status, is therefore a cornerstone of care.

Routine Monitors in the OR

The American Society of Anesthesiologists (ASA) and the Association of Anesthetists in Great Britain and Ireland (AAGBI) provide guidance on best practice standard monitors for all patients undergoing anesthesia, including injured patients [22, 23]. Continuous assessment of the physiological state and depth of anesthesia of the patient is routinely necessary. This monitoring is twofold, involving direct clinical assessment of the patient that is supported by electronic monitoring devices (standard monitoring required for all cases, and "as needed" specialized monitoring; Fig. 21.2). Appropriate clinical observations include cardiovascular, respiratory, and neurologic assessments of mucosal color, urinary

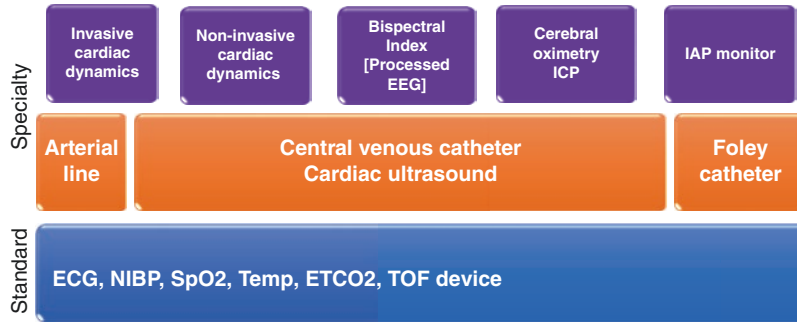

Fig. 21.2 OR standard and specialty monitors. This graphic depicts standard monitors on the bottom (blue) appropriate for all OR cases with specialty monitors used for select cases in orange and purple as tiers above. *OR* operating room, *ECG* electrocardiogram, continuous, *NIBP* non-invasive blood pressure, SpO_2 pulse oximeter, *Temp* temperature, $ETCO_2$ end-tidal carbon dioxide, *TOF* train-of-four, *ICP* intracranial pressure, *EEG* electroencephalogram, *IAP* intra-abdominal pressure

output, estimated blood loss, chest wall movement, pupil size, and response to painful stimuli. It is a core essential safety minimum standard to ensure that all electronic monitor alarms are set to appropriate values and that both visual and auditory alarms are enabled. Perioperative and critical care spaces should establish a single common consensus-based default set of alarms with guidance for adjusting alarm limits for specific patient physiologic states. "Smart alarms" use pre-defined algorithms to assess alarm priority and communicate clinical importance more efficiently. This approach serves as one means of enhancing information transfer while simultaneously decreasing alarm fatigue.

Operative management may be required to control hemorrhage, pursue source control, repair tracheobronchial injury, or mechanically stabilize the thoracic cage. Vigilant monitoring in these patients must account for the cardiopulmonary effects of general anesthesia, hemorrhage, bacteremia, prone or lateral positioning, and intermittent single lung ventilation to facilitate surgical exposure. In the OR setting, standard monitors include the continual observation of oxygenation, ventilation, circulation, and temperature of the patient. In all cases, basic monitoring should include pulse oximetry with plethysmography, non-invasive intermittent blood pressure measurement, as well as ECG and temperature, and then every 30 min until completion of surgery. During general anesthesia, inspired and expired oxygen concentration should be monitored along with adequacy of ventilation (airway pressure, tidal volume, respiratory rate of mechanical ventilator) and end-tidal carbon dioxide ($ETCO_2$) waveform capnography and capnometry [24]. Alarm monitoring should be activated to alert the provider to potential disconnections or leaks within the ventilatory circuit.

Quantitative neuromuscular monitoring is ideally present throughout all phases of anesthesia whenever neuromuscular blockade (NMB) drugs are used. Depending on the anesthesia technique, the depth of anesthesia should be monitored by assessment of inspired and end-tidal inhalational anesthetic drug concentration or by processed electroencephalography (EEG) when total intravenous anesthesia (TIVA) is administered. Thoracic surgery patients are at higher risk of awareness in the OR compared to the general surgical population as thoracic spine procedures are typically performed using TIVA, which removes the inhalational component of anesthesia routinely utilized in other types of cases [25]. Thoracic patients are also likely to require pharmacologic neuromuscular blockade to facilitate one-lung ventilation. Patients with thoracic injury who do not require spine procedures also may undergo bronchoscopy prior to surgery via a single lumen tube and then require re-intubation

with a double lumen tube. The tube exchange necessitates brief periods of discontinuing single lung ventilation and volatile anesthetic agent. These interventions also increase the likelihood of awareness. It is important to note, however, that the interpretation of processed EEG (a surrogate for depth of anesthesia, the reciprocal of likelihood of awareness) may be hindered in the trauma setting by use of induction agents (e.g., ketamine) or by concomitant intracranial injury.

Routine Monitors in the ICU

Many invasive procedures are performed in the ICU setting, under general anesthesia or monitored deep sedation. Undesirable high levels of awareness have been identified during these time frames. Monitoring of the adequacy of sedation or anesthesia should be implemented during these time frames and may be facilitated using a processed EEG approach [26]. In general, intensivists who are not also anesthesiologists may pursue deep sedation coupled with analgesia, but may require an anesthesiologist to administer general anesthesia depending on hospital credentialing and state licensure requirements. The cooperative nature of many ICU services facilitates leveraging a suitably trained individual who is already on service in the ICU instead of requiring relocation of an anesthesiologist from the OR.

Clinicians must be cognizant that both noninvasive blood pressure and pulse oximetric fidelity may be compromised in patients whose physiology is altered by hypothermia, as well as malperfusion related to hemorrhagic or septic shock. These perturbations occur most commonly during the initial resuscitation or during the subsequent management of complications. Furthermore, skin pigmentation may also influence the fidelity of pulse oximeters—a known phenomenon recently recognized to be relevant at SpO_2 levels near 90% [27]. Therefore, invasive monitoring in the OR and ICU often includes intra-arterial radial or brachial catheterization for real-time blood pressure monitoring, frequent sampling—including arterial blood gasses, and the assessment of intravascular volume status through systolic and pulse pressure variability analysis. Central venous pressure monitoring may help to trend intravascular volume, diagnose pericardial tamponade, or aid in placing temporary transvenous pacing wires. Some catheters also provide continuous subclavian venous O_2 saturation ($ScvO_2$) monitoring obviating repeated individual samples to assess this surrogate for mixed venous oxygen saturation (SvO_2). Indeed, such monitoring may provide clues regarding the need for component transfusion therapy to augment arteriolar O_2 carrying capacity during active resuscitation when hemoglobin concentration may be rapidly in flux [28].

Transthoracic (TTE) or transesophageal echocardiography (TEE) may also be beneficial for monitoring cardiac function and intravascular volume status as part of a precision-based resuscitation strategy or in response to unexpected hemodynamics. A key difference between TEE and invasive pressure monitoring is that echocardiography allows direct assessment of volume, systolic function, and filling pressure rather than using secondary measures or proprietary algorithmic assessments. The TEE acquired direct measurements support the evaluation of preload adequacy, the likelihood of volume recruitable cardiac performance, and global ventricular function including ventricular compliance. These assessments are critical in adjusting plasma volume expansion, the initiation or titration as well as the cessation of vasoactive infusions. In the critical care domain, a new certification has arisen—critical care echocardiography—to support acquiring and refining this essential skill [29]. Bedside and repeated ultrasound assessments may be particularly critical in evaluating those with pulmonary hypertension, right heart failure, or blunt myocardial injury—all of which are conditions commonly encountered in those with thoracic injury [30].

Contraindications to TEE for hemodynamic assessment in the OR or the ICU for the anesthesiologist or intensivist include oral, pharyngeal, or esophageal injury, active upper gastrointestinal hemorrhage, history of esophageal stricture, and recent history of resectional esophageal or

gastric surgery. There are a plethora of less, or non-invasive devices that also evaluate cardiac performance and they are detailed in Chap. 19. Note is made that some conditions, including, but not limited to pulmonary resection or the open thorax, may impair the fidelity of specific devices. The bedside clinician must be cognizant of available device limitations and have an alternative approach enabled when such circumstances are encountered. When data fidelity is unclear, insertion of a pulmonary artery catheter with assessment of cardiac output using thermodilution (intermittent bolus or "continuous" approaches) coupled with SvO_2 determination may afford clarity.

Transitions of Care

Care transitions represent a period of vulnerability for the critically ill and injured patient. Three specific circumstances are encountered including: (1) movement to the OR or procedure suite for ongoing care, (2) movement to a higher level or care within the same facility, or (3) movement out of the ICU to a less intensive monitored and resourced setting. In each of these circumstances, communication is essential between team members to facilitate safe hand-off. Standardized approaches such as HATRICC (Handoffs And TRansitions In Critical Care) help ensure that there is inter-team based communication at a single place and a single point in time to support information transfer as well as fidelity [31, 32]. Formalized hand-offs—as well as informal ones—are supported by objective data that are displayed on "safety net" kinds of monitoring devices. In this way, all team members may identify and evaluate the data on which the hand-off is being based. This approach is important in transferring patients between settings even when the patient is being moved to a less monitored setting. Anecdotes abound regarding changes in vital signs as patients move between care areas, all of which are noted only after the transferring team has departed. Rescue via a Rapid Response Team (RRT) or Medical Emergency Response Team (MERT) activation is not uncommon as a consequence [33]. Those who require rescue often need a change in medication therapy but also often require interventions ranging from supplemental oxygen to diuretic therapy to invasive mechanical ventilation but may include operation for source control regardless of admitting service [34]. Those with thoracic injury are especially vulnerable to processes such as atelectasis, pleural space occupying lesions, hypoxic pulmonary vasoconstriction, and hospital acquired pneumonia that drive acute respiratory distress or failure.

Electronic Health Record (EHR) Embedded Detection Systems

While most RRT or MERT activations require recognition of a potentially dangerous event by a clinician or family member, a multiplicity of efforts have worked to create data-driven detection systems that can anticipate an event and prompt intervention before rescue is required. A number of such systems are known as the Early Warning System, the National Early Warning Score, and the Medical Early Warning System [35, 36]. While some were initially focused on detecting cardiac arrest harbingers, or sepsis-driven abnormalities, a more broad approach now predominates and strives to recognize patterns that may impact morbidity, ICU admission, or mortality and embrace other indices such as the Rothman Index [37]. Data indicate that such systems may be better at detecting cardiac arrest and death within 48 h compared to other relevant events such as requiring NIV or invasive mechanical ventilation [38]. The Rothman Index, in particular, seems to perform well in predicting unplanned ICU admissions [39]. Based on the lack of ideal performance related to data acquirable and analyzable in terms of trends in the EHR, future systems are anticipated to leverage machine learning and artificial intelligence (ML/AI) approaches. Model development, validation, and training may await large volume of data sharing—as has already been accomplished from one ICU in a way that respects patient data, privacy, and international law—to inform well perform-

ing models in multiple locations and patient populations [40]. For now, the delivery and deployment of an actionable ML/AI detection system remain on the horizon as a highly desirable monitor for non-ICU care requiring patients at risk of acute decompensation.

Monitoring During Non-invasive and Invasive Mechanical Ventilation

A subset of thoracic trauma patients, such as those with isolated or few rib fractures, may be treated with pulmonary hygiene measures as well as supplemental O_2 including High-Flow Nasal Cannula O_2 ($HFNCO_2$) [41]. Close monitoring of respiratory physiology is paramount to rapidly identify the presence of acute respiratory distress and initiate interventions to prevent the progression to acute respiratory failure. Clinical observation of respiratory rate, synchrony between thoracic cage and abdominal wall movement offers a gross evaluation of tidal volume, minute ventilation, and most importantly, work of breathing (WOB). $HFNCO_2$ support may impact WOB but not to the same extent as will an NIV technique such as BiPAP [42]. Importantly, BiPAP will also garner estimates of tidal volume and minute ventilation, providing greater precision in the monitoring of patients who require pulmonary support (Fig. 21.3). Non-invasive positive pressure ventilation (NIPPV) techniques, such as high-flow nasal cannula O_2, CPAP by face mask or helmet, as well as BiPAP all reduce the need for intubation and invasive mechanical ventilation rescue following thoracic injury [43, 44].

Patient management using invasive mechanical ventilation benefits from a variety of specific monitors, many of which are intrinsic to the ventilator as opposed to externally applied devices. The reader is referred to the chapter on Mechanical Ventilation for a detailed exploration of monitoring as well as liberation from mechanical ventilation approaches. Specific note, however, is made of the subpopulation with a known or presumed difficult airway. When acute invasive support needs have resolved, liberation from

Fig 21.3 Non-invasive ventilation data. This image depicts the kinds of data that one may obtain from a BiPAP device. Open source

endotracheal intubation should be performed in a controlled manner, following an "all hazards" risk assessment, as detailed within joint as well as individual society guidelines [45, 46]. Specific vigilance and pre-planning are required in the extubation of a patient with thoracic injury who has concomitant SARS-CoV-2 (COVID-19) infection; adaptations of existing difficult airway guidelines were required to account for aerosol generation [47, 48].

Monitoring of Neuraxial Integrity

Epidural analgesia is the most commonly used regional technique in the ICU, particularly among elective thoracic surgery patients. A variety of options besides epidural analgesia are reviewed in the Approach to Analgesia chapter. Nonetheless, a major underpinning assumption is that the patient can participate in the examination and assessment of analgesia adequacy. At times, deeper sedation is required for mechanical ventilation and may impair the neurologic examination outside of sedation holidays. Patients with thoracic trauma may present with potential or evolving spinal cord injury who are unable to

participate in a neurologic examination due to such sedation, monitoring of motor-evoked potentials (MEPs) of lower extremities and/or somatosensory-evoked potentials (SSEPS) of the tibial nerve may serve as indicators of distal neuraxial integrity. Advanced monitoring that includes MEP or SSEP as part of a blended approach to care benefits from consultation with a neurointensivist; it is anticipated that a spine surgeon (Neurosurgery or Orthopedic Surgery) has occurred as part of a team-based approach to injury management [49]. Since specialty neurocritical care and neurosurgical or spine orthopedic surgery services are not uniformly available in every acute care facility, inter-facility transfer is often required. Safety and quality during inter-hospital transport builds on similar practices utilized for transport within a healthcare facility.

Monitoring During Intra-facility and Inter-facility Transport

Transferring a patient to the ICU, OR, or other procedural areas such as Interventional Radiology requires uninterrupted monitoring as such transport is well-chronicled as a period of great risk to safety, principally through human factors errors [50]. The recent COVID-19 pandemic has driven the evolution of transport practices to also help ensure safety when there is a bioemergency such as the recent COVID-19 pandemic [51]. In all circumstances, a standard monitoring approach helps identify deterioration, track the results on newly applied or existing therapy, and provides a common basis for team interactions to address abnormal physiology. During a bioemergency, well-identified and unique pathways for patient ingress and egress should be developed ahead of time to facilitate safe transport and avoid cross-contamination. Those pathways should be evaluated in terms of proximity to rescue equipment as device failure and patient disease evolution are both noteworthy events that occur with a non-zero frequency.

The standard of care and monitoring during the transfer of a patient from the ICU to the OR and back again should be identical to what was in place prior to transfer. The minimum monitoring that should be implemented includes continuous ECG, continuous SpO_2, and intermittent NIBP. This safe monitoring approach extends to the mechanical ventilator for those who require invasive mechanical ventilation. If a transport ventilator can replicate the ICU settings, or the newly revised ones from the OR, then such a device is ideal as it generally occupies a smaller footprint. If the capabilities of an ICU ventilator are required to support advanced ventilation, then the patient's transport must include the ventilator, O_2 tank as needed, and a respiratory care practitioner as well. The inability to provide such transport, or the inability to deliver the advanced mode in the OR may drive care—even abdominal or thoracic re-exploration—to occur in the ICU [52]. The risk of awareness appears to be higher during intra-facility transport, requiring the clinician to ensure that adequate sedation is provided, particularly if the patient has received a recent dose of a neuromuscular blocking agent shortly prior to transfer [26].

Inter-facility transport adds a layer of complexity to transport concerns based on the need for hand-off to an unfamiliar team, space constraints on both ground and air transport, and the obligate time for transit between resource replete locations in the event of acute deterioration [53, 54]. Vasoactive infusions, mechanical ventilation, component transfusion therapy, packed and open body cavity, as well as hemodynamic lability are but a few of the common indicators for advanced life support capable transport [55]. Total transport time may influence the mode of transportation selected and may be related to distance, road traffic, road quality, and ongoing care including the deployment of a time sensitive device such as a resuscitative endovascular balloon occlusion of the aorta (REBOA) catheter, or an evolving aortic or neurologic injury [56]. Transport for specialized care no longer uniformly awaits arrival at a destination facility based on the increasing frequency of field deployment of extracorporeal membrane oxygenation (ECMO) rescue teams. Such teams travel to the

patient's originating facility, cannulate the patient on-site for veno-venous ECMO, and then return to the destination facility providing ECMO en route. Complex acute rescue care such as that described above benefits from team members who have developed transactive memory and who are facile with advanced monitoring devices suitable for both ground and air transport [57, 58]. ECMO rescue is well described in both civilian and military domains and has been deployed for eCPR rescue—a technique that may be appropriate for select patients with arrest after blunt trauma arrest [59–61].

Conclusions

Basic monitoring provides a "safety net" across all locations in the acute care facility for patients who demonstrate altered physiology. A standardized approach to routine monitoring offers a foundation upon which specialty and unique monitors may be added in specific patient populations, often in a tiered-fashion. Care devices such as NIV machines and invasive ventilators provide both therapy and add data into the monitoring mélange to assess patients' responses and provide a signaling system that alerts clinicians to deterioration. Early warning systems continue to expand the data about consequences they strive to detect, but likely await developing ML/AI approaches to be ideally effective. Intra- and inter-facility transport present unique challenges to patient safety that are mitigated against by monitoring but also by structured communication between engaged teams. Adaptations of existing approaches to monitoring should be anticipated during bioemergencies such as the COVID-19 pandemic and serve as examples of successful modifications that may be utilized in future high-consequence infectious disease outbreaks. Clinicians should be familiar with the specific capabilities and limitations of their monitoring devices and complement to understand when additional data is required and how to best secure that data to guide high quality care.

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22 Mechanical Ventilation After Thoracic Injury

Lewis J. Kaplan, Elena Costantini, and Maurizio Cecconi

Introduction

Chest injury may compromise a patient's ability to oxygenate, clear CO_2, or meet their work of breathing in isolation or combination with one another. These derangements arise from damage to the thoracic cage, direct or indirect lung injury, airway disruption, or injury to the diaphragm or innervation that supports generating a transpulmonary pressure gradient [1]. While some patients may present with acute respiratory failure, others present with acute respiratory distress. In general, the latter may be acutely managed using non-invasive ventilatory support, while the former requires invasive mechanical ventilation [2, 3]. Patients with serious thoracic injury are less well managed without invasive ventilation due to a constellation of features that span pain, ventilation impairment, hemodynamic imbalance, and the need for operative intervention [1]. This chapter will solely address invasive ventilation in the context of chest trauma. The management of specific injuries such as major bronchial disruption is addressed elsewhere in this text.

Approach

One major decision to be made on Emergency Department admission is whether airway control and invasive mechanical ventilation are required concomitant with initial resuscitation. Certain conditions make rapid airway control appropriate and are presented in Fig. 22.1. In general, such conditions are related to shock, hemorrhage, and the need for acute intervention. One notable exception is the patient with penetrating thoracic injury who has a supportable mean arterial pressure but who needs operative hemorrhage control. This patient should be carefully evaluated to determine if airway control—and its known decrease in venous return—may be delayed until the patient has arrived in the OR and the chest is prepped and draped—so that any precipitous MAP decrease may be met with expeditious operative control.

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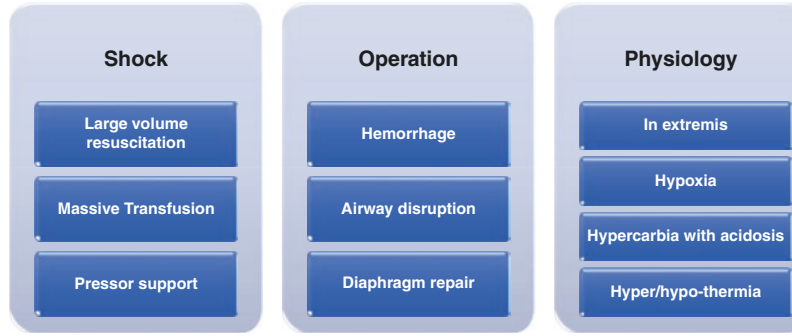

Fig. 22.1 Acute airway control and mechanical ventilation indications

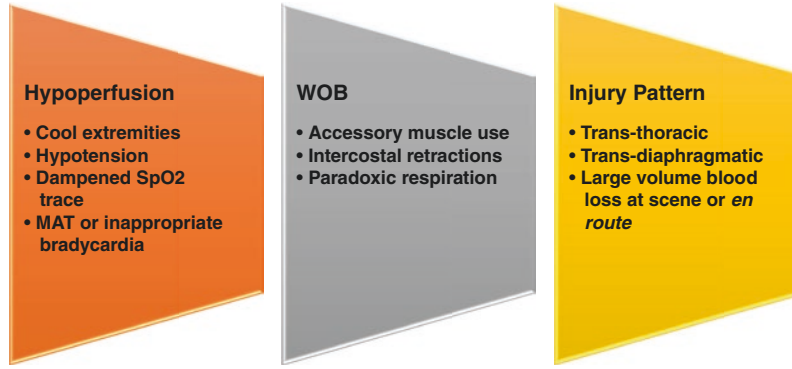

Fig. 22.2 Clinical cues to airway control

The decision to initially secure the airway is ideally enacted using clinical cues rather than data from an arterial or venous blood gas. Clinical cues take many forms but a list of common ones that direct rapid airway control and the initiation of invasive mechanical ventilation are presented in Fig. 22.2. Oral endotracheal intubation is preferred over the nasal route, but the team should always be prepared for the potential need for a surgical airway control; internally ringed difficult to kink tubes are particularly useful for airway management via cricothyroidotomy.

Respiratory Physiology During Spontaneous Breathing

Respiratory physiology normally places emphasis on the respiratory pump muscles such as the diaphragm and the intercostal muscles in generating effective breathing [4]. The contraction of these muscles during inspiration expands the thoracic cavity and generates negative pressure in the pleural space and across the alveoli. The difference between atmosphere pressure and alveolar pressure generates airflow that is responsible for lung ventilation (i.e. tidal volume). Because of the decrease in the intrathoracic pressure (ITP) there is an increase in the venous return (VR) from all extra-thoracic sites. This flow supports the stroke volume of the right ventricle. With minimal to no atelectasis, hypoxic pulmonary vasoconstriction is not active and the right ventricle (RV) faces a minimum of resistance for the ejection of blood into the pulmonary vascular bed. Conversely, during expiration relaxation of the inspiratory muscles results in a decrease in the volume of the thoracic cavity. The elastic recoil of the previously expanded lung tissue allows them to return to their original size. A decrease in lung volume due to the elastic recoil results in an increase in the pressure greater than the external environment pressure so that gas moves out of the lungs according to the pressure gradient. During expiration, because of the increased intrathoracic pressure there is a decrease in the VR and a corresponding decrease in right ventricle performance. Positive pressure ventilation, such as occurs using a bag-

valve approach, or invasive mechanical ventilation changes some of these relationships and their dynamics.

Respiratory Physiology During Positive Pressure Ventilation

Positive pressure ventilation (PPV) reverses the spontaneous ventilation dynamic. In ventilated patients inspiration occurs during an increase in endoalveolar and intrathoracic pressure and therefore creates a decrease in venous return. It is this relationship that drives the need to avoid a PEEP valve during the initial period of hand-ventilation when there is no hemorrhage control. Increased ITP will decrease venous return and when that pressure is sustained—such as with the use of PEEP, or exacerbated by rapid bagging with little time for exhalation—it may lead to cardiovascular collapse [5]. Exhalation results in a decrease in intrathoracic pressure that enables venous return. Understanding these relationships also informs the initial ventilator prescription.

Understanding the Ventilator Prescription

The ventilator prescription is readily understood in terms of its component parts, each of which is presented in Fig. 22.3. These elements are mode, rate, target, FIO_2, PEEP, waveform, flow rate, and inspiratory and expiratory time; these are specific to the two most common approaches to mechanical ventilation—volume and pressure cycled ventilation. Starting from these two key modalities, other variations exist as well (Fig. 22.4) and will be discussed later in the chapter. The aforementioned prescriptive elements interface with the patient's lung and chest wall to generate resultant pressures as well as graphics that help the clinician understand how the ventilator and the respiratory system interact. Below we discuss the features that one may manually set within the prescription, as opposed to those that are reflective of the prescription.

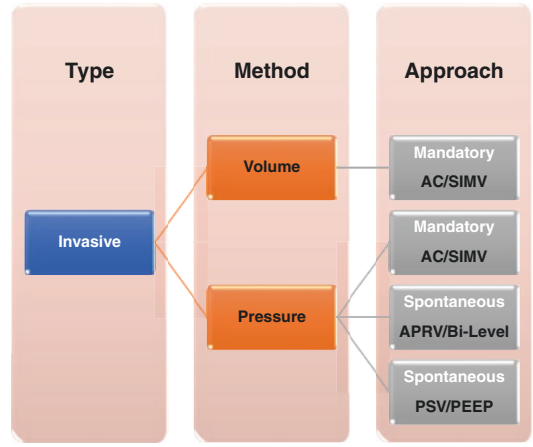

Fig. 22.4 Major methods of mechanical ventilation

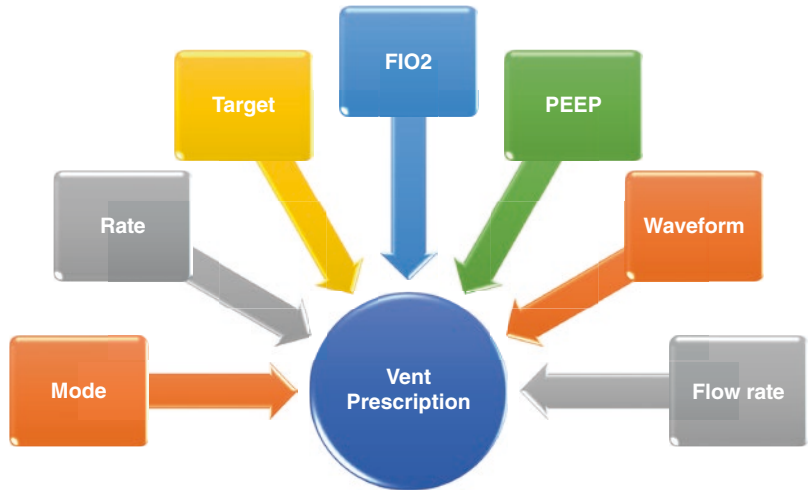

Fig. 22.3 Key elements for VCV and PCV ventilator prescriptions

Directly Set Elements

Mode: The most common mode is assist control (AC) which means that the ventilator will reach its target every time it is triggered to perform an action. The most common trigger is time and is established by the set rate; spontaneous breaths will also serve as a trigger in this mode. In the synchronized intermittent mandatory ventilation (SIMV) mode, the sole trigger is time as set by the rate; spontaneous breaths will not receive support unless coupled with an additional setting such as pressure support ventilation (see below).

Rate: The number of times each minute that the ventilator reaches its target.

Target: In general, there are two targets: volume or pressure. In volume cycled ventilation (VCV) the ventilator delivers a set volume of gas. In pressure cycled—or pressure controlled—ventilation, the ventilator delivers gas until a set pressure is achieved. It is clear that the time spent in inspiration (Ti) and therefore, the time available for exhalation (Te) will be determined by how gas is delivered in VCV, or by a time-based metric for PCV. The set VT, gas delivery waveform, and peak gas inspiratory flow impact Ti in VCV, while either a fixed Inspiratory:Expiratory (I:E) ratio or a set Ti is used during PCV.

Waveform: Gas delivery for adults occurs using either a square or a decelerating waveform. There are important differences in how each presents gas to the lung. While both waveforms start by having gas flow rise to its maximum or peak flow rate, the square waveform maintains the gas flow at that same rate until the set VT is delivered. The decelerating waveform begins immediately to decrease the flow rate in a preprogrammed fashion until the set VT is also delivered. Based on the planned decrease in flow rate, for a given VT, the Ti will be longer when using a decelerating gas delivery plan.

Peak inspiratory flow: This is measured in LPM and is the maximum rate at which gas may flow out from the ventilator. The faster the flow rate, regardless of waveform, the shorter the Ti and vice versa.

I:E ratio: This ratio specifies the proportion of time spent in inspiration compared to expiration. A normal I:E ratio for a spontaneously breathing, healthy individual at rest is 1:4–1:5. This means that in a 60 s cycle, with a 12 breath/min respiratory rate, and therefore 5 s/breath cycle, about 1 s would be spent in inspiration and 4 s would be spent in exhalation. In fact, in health, there are periods of no flow before the next breath. With critical illness, the I:E ratio closes and is often in the range of 1:2 reflecting either the need for a longer Ti or more breaths per minute, or both. When using I:E ratio ventilation one must be cautious to avoid simply increasing the RR. When so doing, the Ti and Te will each shorten and may lead to increased alveolar collapse as well as CO_2 retention despite the desire to increase CO_2 clearance. Unlike in VCV where the waveform and the gas flow rate are manually set, in PCV using I:E ratio ventilation, the device adjusts these features automatically.

Fixed Ti: This method manually sets the Ti, and therefore for a given RR, the Te as well. The waveform and flow rate are also adjusted by the ventilator. In this mode, increases in RR will only shorter the Te as the Ti remains invariate. Care should be exercised to avoid compromising the ability to clear CO_2 when the RR is increased.

FIO_2: Oxygen is delivered based on concentration from room air at 21% (0.21) to 100% (1.0) to achieve a goal pO_2 or target SpO_2. While previous SpO_2 targets have focused on maintaining $SpO_2 > 92\%$, conservative SpO_2 targets have recently emerged using 88% as a lower limit for those without baseline COPD [6].

PEEP: Positive end-expiratory pressure is generated by maintaining a volume of gas that creates a desired pressure in the airway at the end of expiration. PEEP is established in cm H_2O pressure and ranges from 0 on up, but commonly begins at 5 cm H_2O pressure denoted as (+5). While there is not an upper limit for

PEEP, recent data suggests that the previous era of super-PEEP was unnecessary and potentially harmful [7].

Each of these manually determined settings leads to specific pressures and graphics that should be specifically evaluated to determine whether the prescription is appropriate for a given patient's pulmonary system. These are explored below.

Derived Pressures and Indices

Minute Ventilation (*VE*): defined as tidal volume times respiratory rate. A normal VE is 6–8 LPM.This is the total gas that is moved across the patient's pulmonary system reflecting the mandatory component, as well as the spontaneous component of the total minute ventilation. A normal VE is 6–8 LPM.

Peak Airway Pressure/Peak Inspiratory Pressure (*Pawpeak or PIP*): This is the highest pressure reached in the respiratory cycle generally correlating to the maximal gas flow.

Mean Airway Pressure (*Pawmean*): This is the mean pressure experienced by the patient's pulmonary circuit. It reflects the area under the gas delivery waveform curve and is the strongest measure that correlates with pO_2. Ventilator prescription changes, other than FIO_2, designed to increase pO_2 and correct hypoxemia should also result in an increase in Pawmean.

Plateau Pressure (*Pplat*): This is the pressure in the patient's pulmonary system when there is zero flow and at the end of inspiration so that the pulmonary tree is full of gas. Measurement generally requires an inspiratory pause to be created at the end of inspiration in order for flow to cease and the pressure to be measured. It is believed to be consistent with lung protective ventilation when <30 cm H_2O pressure.

Peak to Plateau Gradient (*Pawpeak-plat*): This derived index provides insight into conditions such as bronchospasm, a large secretion burden, and tube dysfunction. A normal value is 4 cm H_2O pressure. Higher values should prompt tube interrogation, an evaluation of volume excess and of the I:E, but it is quite commonly associated with bronchospasm that responds to beta-agonists or supplemental magnesium as a smooth muscle relaxant, especially in those with pre-existing reactive airway disease.

Driving Pressure (*DP*): DP represents the difference between the plateau pressure and the total PEEP, i.e. Pplat – PEEP [8]. It is related to the patient's pulmonary compliance described by the following equation: Crs = VT/DP. Therefore, DP may be expressed in the following way: DP = VT/Crs. In this way, DP takes into account both dynamic and static stress on the pulmonary system. A related measure, transpulmonary DP requires measurement of esophageal pressure to calculate and only assess pulmonary stress. Since esophageal pressure is less commonly measured, DP is much more frequently used as a guide to management. DP is believed to be consistent with lung protective ventilation when it is <15 cm H_2O [9].

Key Graphics

There are three essential waveforms to routinely assess when evaluating the adequacy and appropriateness of the ventilator prescription. These are the pressure-time trace, flow-time trace, and the pressure-volume loop. Only assessing numeric data provides a partial view of how the patient's pulmonary system interfaces with how the ventilator is delivering gas.

Pressure-Time trace: This graphic (Figs. 22.5 and 22.6) in VCV allows the clinician to determine how pressure changes in the system over time. Bronchospasm, tube obstruction, and tube malposition are all evident on this profile.

Flow-Time trace: This graphic (Figs. 22.5 and 22.6) follows a pattern reflective of the gas delivery profile. It is an important clue to the presence of undesirable auto-PEEP in that auto-PEEP is indicated by failure of the trace to return to baseline prior to the next breath.

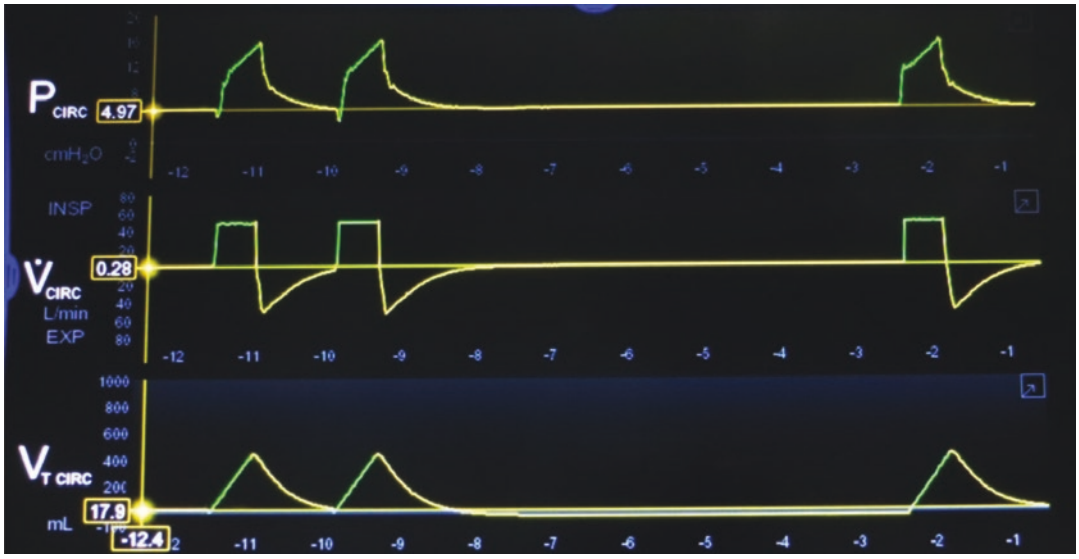

Fig. 22.5 Pressure, flow, and volume with square waveform gas delivery

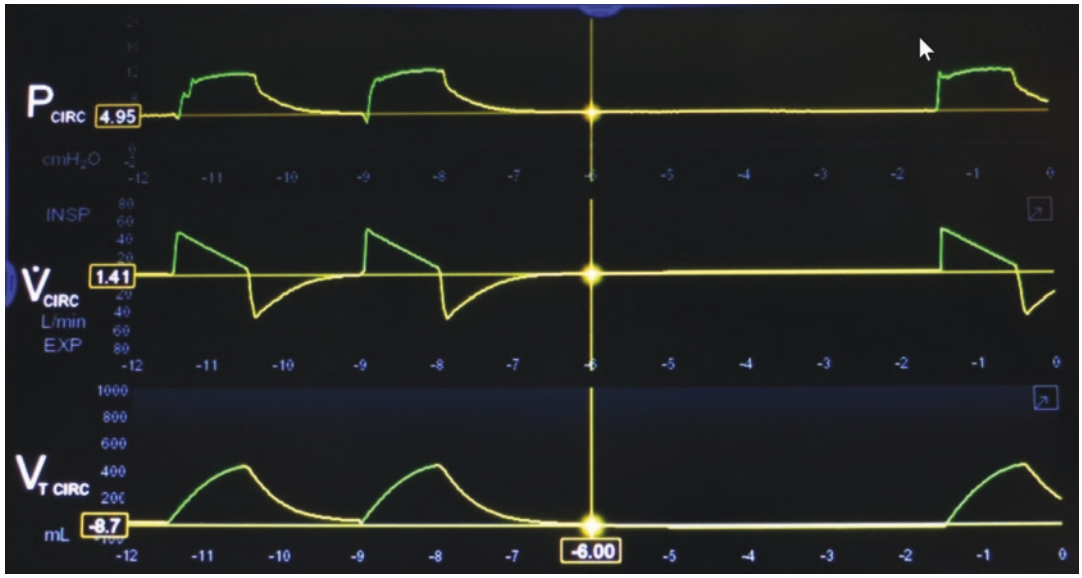

Fig. 22.6 Pressure, flow, and volume with decelerating waveform gas delivery

Pressure-Volume loop: This is an integrative trace (Fig. 22.7) that is the dynamic expression of the static pressure-volume curve. It is important to recognize the conformation of a normal trace as well as key abnormalities including inadequate PEEP, alveolar overdistension (Fig. 22.8), and under-recruitment.

Each of the ventilator prescription elements, including pressures and graphics forms the basis of establishing an appropriate prescription. The adequacy of that prescription must be interrogated using supplemental tools including laboratory analysis, portable radiography and, increasingly, bedside ultrasound.

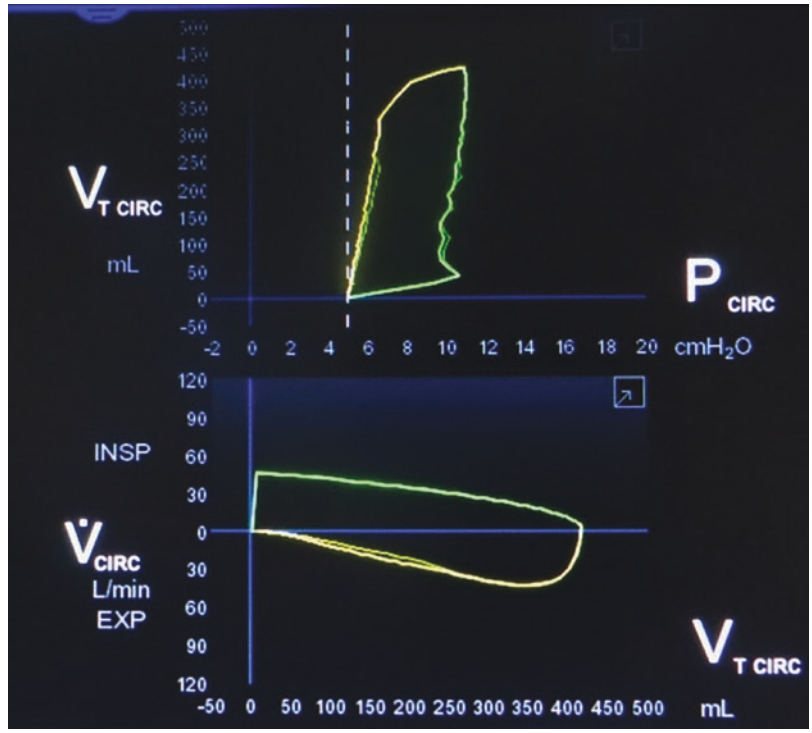

Fig. 22.7 Pressure volume and flow volume loops (normal)

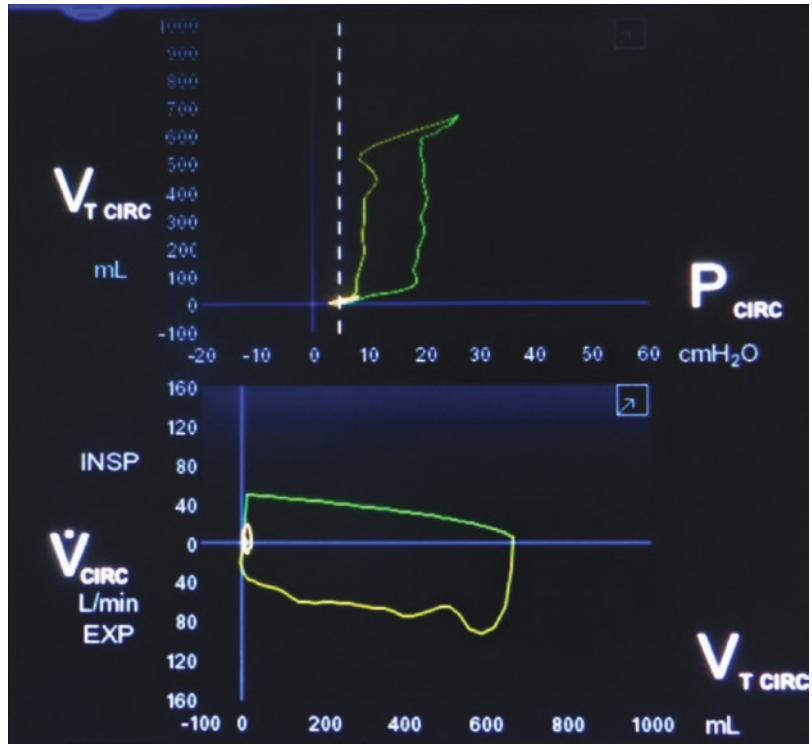

Fig. 22.8 PV curve demonstrating overdistension

Analysis Tools

Arterial Blood Gas (ABG): paralleling the ventilator prescription to ensure an adequate blood gas exchange it is fundamental to perform an arterial blood gas analysis. The ABG is reported in a specific format: $pH/pCO_2/pO_2/HCO_3$. Only an ABG will provide a pO_2 which may be required to determine the Alveolar-arterial gradient (A-a gradient) as well as the PaO_2/FIO_2 ratio. Baseline values may be key in assessing the progression of direct or indirect lung injury after thoracic trauma.

Venous Blood Gas (VBG): A VBG may be used instead of an ABG for most patients but will not allow one to interpret the pO_2 as it is venous and often initially obtained from a peripheral vein. There is an extensive body of literature addressing the utility of a superior vena cava O_2 saturation. Interconversion from a VBG to an ABG (i.e. arterialization) for most patients may occur recognizing that in general, the venous pCO_2 is 7 Torr higher than on the arterial side, and therefore the pH is 0.05 pH units lower (10 Torr change in pCO_2 = inverse 0.08 unit pH change). Hence the arterialized VBG is obtained by the following addition or subtraction from the venous values:

$$pH + 0.05 / pCO_2 - 7 / pO_2 \left(\text{uninterpretable}\right) = \text{arterialized venous ABG}$$

Portable Chest Radiography (PCXR): Besides systematic auscultation for breath sound equivalency, as well as confirmation of sustained end-tidal CO_2 concentration and waveform, one should always confirm ETT placement using PCXR. PCXR also provides key information regarding the state of the pulmonary parenchyma as well as the presence of space occupying lesions such as a pneumothorax or hemothorax. Mediastinal interrogation necessity may be highlighted by CXR findings, but CT scan evaluation of thoracic injury is common and provides precise detail and will not be reviewed within this chapter.

Bedside Ultrasound (U/S): Repeated assessment has value after injury. U/S brings that ability to interrogate the pleural space as well as the pulmonary parenchyma to the bedside in an on-demand fashion. Its value in readily identifying pneumothorax, hemothorax, and pericardial blood has been well established [10]. The recent SARS-CoV-2 pandemic has underscored its utility in defining parenchymal abnormalities well beyond the traditional boundaries of the Focused Assessment by Sonography in Trauma (FAST) examination [11, 12]. As part of the evaluation of the pulmonary system, the bedside ultrasonographer should also assess cardiac filling and function consistent with the recent expansion in critical care ultrasonography. Recall that a key element of O_2 onloading and CO_2 offloading is pulmonary flow, and that cardiovascular performance will be an integral driver of how the ventilator prescription interacts with the patient's cardio-pulmonary system.

Now that the basics of establishing and interrogating a ventilator prescription are understood, it is appropriate to establish the goals for mechanical ventilation after chest injury.

Mechanical Ventilation Goals and Approaches

The goals for mechanical ventilation after thoracic injury are readily agreed upon—adequate oxygenation to support cellular respiration and sufficient CO_2 clearance to manage pH. These goals should be obtained avoiding ventilator induced lung injury (VILI) [13]. The damage caused by the ventilation depends on two components: first how much the lung is injured and second how the ventilator is set. Our knowledge of the molecular underpinnings of the pulmonary parenchymal and vascular response to the ventilator prescription continues to evolve, but does not yet offer clear therapeutic targets by which one may influence outcome [14].

In order to address those sweeping goals, there are two broad approaches: recruitment of available pulmonary parenchyma versus ventilate using only the available lung units. It is clear that the latter is directly related to lessons learned from ARDS management and reflects the tenets of an ARDSNet approach [15]. It is not certain that otherwise normal lung initially requires a low tidal volume approach. To date, low tidal volume ventilation used as a prophylactic approach for those at-risk for ARDS has not improved outcomes [16]. Therefore, in the injured patient population, one must determine whether there is—or is not—recruitable lung.

When recruiting lung, there remains a standard to which one must hew—the provision of lung protective ventilation—a concept that has been extended to include diaphragm protection as well [17]. It is important to differentiate the standard of care (provide lung protective ventilation) from the commonly utilized approach to that standard (low tidal volume ventilation). Other approaches to lung protective ventilation such as airway pressure release ventilation, for example, have been demonstrated to be non-inferior to low tidal volume approaches in those with ARDS [18, 19]. These data support avoiding lung injury regardless of the approach, and method, of ventilation utilized. Given that those with thoracic injury who require mechanical ventilation generally also require resuscitation, alveolar recruitment seems a reasonable strategy to support venous return, unload the right side of the heart, and enhance cardiac performance all while perhaps offsetting the need for pressor support. Let us explore what is currently known about mechanical ventilation after thoracic injury.

Pitfalls in the Thoracic Trauma Patient

The interplay of several factors relevant for mechanical ventilation after thoracic injury influences outcome. Age, thoracic injury severity, extra-thoracic injury, massive transfusion, acidosis, body temperature, fluid excess, analgesia adequacy, pulmonary contusion volume, diaphragm function, cardiac or valve injury, new organ failure, infection with sepsis or septic shock, reduced lung volume from pulmonary resection (pre-existing or as a result of injury), pre-existing pulmonary disease, pulmonary hypertension, interstitial lung disease as well as pre-injury chronic organ dysfunction or failure all impact outcome [20]. Conditions that may impact outcome interface with how mechanical ventilation is utilized after chest trauma, each of which describes a different but related approach.

Recruit Available Lung

There are two related major approaches to the recruitment of available lung including longer Ti and higher VT, increased PEEP, and constant pressure but variable flow. This approach is one that may be undertaken to embrace what is termed the open lung model [21]. In this model, the maximal number of alveolar units that can participate in gas exchange are recruited to do so and maintained open and ready to exchange gas. Those units, therefore, are also devoid of hypoxic vasoconstriction and maintain alveolar capillary blood flow in support of oxygenation and CO_2 clearance. Such an approach may also leverage using a recruitment maneuver [22].

The aim of the recruitment maneuver is to recruit all the healthy alveolar units that can participate to gas exchange. Moreover, recruitment increases ventilated lung volume and will decrease airway pressure while improving the compliance of the respiratory system. There are several approaches to perform a recruitment maneuver both with volume and pressure controlled ventilation. One approach is not more effective than the other and will instead reflect institutional preference.

When using VCV, a higher VT than would be prescribed using low tidal volume ventilation is prescribed in conjunction with a decelerating waveform and a slower flow rate. These three elements increase the Pawmean and should both increase oxygenation as well as better match the physical characteristics of more alveolar units than using a lower VT and a higher flow rate—even when using a decelerating waveform. PCV

prescriptions would also embrace a longer Ti to improve alveolar recruitment rendering using fixed Ti ventilation as a preferred approach. The characteristic of the alveolar unit is termed the regional time constant [23]. This constant varies across units and is influenced by a host of factors including the alveolar unit critical closing volume, critical closing pressure, and the status of the alveoli around the unit in question.

The latter is key as alveolar interdependency that is mediated across the pores of Kohn described as the process whereby a relatively closed alveolus is able to be opened by other units with which it shares a common wall if those other units are more open [24]. Recall that the pores of Kohn transmit fluid, gas, and alveolar macrophages. The ability of the alveolus to open is also impacted by the health of the Type II pneumocytes (Clara cells) as they are the source of pulmonary surfactant. Since alveoli are polyhedrons, they have angles, corners, and can have walls that coapt. Surfactant, effectively soap, decreases surface tension and allows walls to separate more easily under the influence of gas influx as well as alveolar interdependency.

PEEP may be readily used to augment end alveolar pressure and rectuitment by moving the zero-pressure point more proximally to a cartilaginously supported portion of the airway. This helps prevent alveolar collapse. The pressure-volume loop is instrumental in evaluating the adequacy of PEEP in support of recruitment. Recall that the ventilator prescription may result in an appropriate ABG as well as appropriate pressures, but that the gas delivery and PEEP may not be ideal and will benefit from waveform analysis to titrate appropriately.

Constant pressure with variable flow is familiar to virtually all as continuous positive airway pressure or CPAP. However, with chest injury that requires invasive ventilation, a different configuration is necessary to meet oxygenation and CO_2 clearance goals. If instead of providing only a single low level of pressure (i.e. 5 or 10 cm H_2O pressure), one provided a single high level of pressure (i.e. 25 or 30 cm H_2O pressure), alveolar recruitment to total lung capacity could be achieved, but would occur at the expense of CO_2 clearance; it is quite difficult to exhale against a high pressure. This failure is repaired by simply turning off the pressure for a brief period of time. That time may be quite short (even less than 0.8 s) as those recruited to a great degree benefit from lung and chest wall elastic recoil and can generate high gas flow rates.

This prescription is embraced as airway pressure release ventilation (APRV) as ventilation, CO_2 clearance, occurs when the gas flow is stopped and the airway pressure is released (Fig. 22.9) [25]. The prescription elements are fewer than with VCV or PCV as there is the high pressure, the time the patient is maintained at the high pressure, the time allowed for airway pressure release, and if desired, a low pressure is to be maintained. Many will titrate the time for pressure release to flow dynamics and not set a low pressure at all. Of course, FIO_2 must be set as well. The time for release is sufficiently short that patients cannot breathe during the period of pressure release. A related mode called BiLevel Ventilation uses two separate pressure levels for breathing and provides sufficient time to breathe at the lower level (Fig. 22.10). In that way if is more akin to BiPAP than APRV with regard to

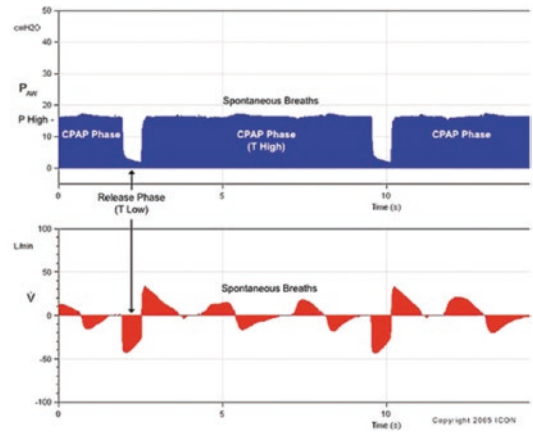

Fig. 22.9 Airway pressure release ventilation

breathing dynamics. Both modes may be used with success after thoracic injury.

Constant pressure and variable flow modes recruit pulmonary parenchyma by increasing Pawmean and keeping it there for a relatively long time (>4.5 s by convention, but is commonly 6 s or longer when addressing hypoxemia). This combination of pressure and time supports excellent matching of regional time constants. Indeed, data supports such matching and recruitment of posterior and basal segments as well as relocation of blood flow to those domains as well [26]. Unsurprisingly, but as anticipated based on physiology, improved venous return and cardiac performance have been documented, as has a decreased need for pressor support in studied patients [27].

Potential Risks with Using a Deliberate Recruitment Approach

Regardless of the ventilation method used to recruit available lung, we would like to point out two key concepts:

1. The pressure in the respiratory system should not exceed 35–40 cm H_2O especially in thoracic traumatic patients—the risk of barotrauma and pneumothorax is not to be underestimated.
2. The increasing pressure in the lungs reduces the VR and increases pulmonary vascular resistance leading to reduced cardiovascular performance. A major consequence of increased intrathoracic pressure may be right ventricle failure and systemic hypotension.

It is equally important to recognize that during the process of recruitment there will be some stress across alveolar walls that are shared by more and less open alveoli. This stress may be identified as intra-tidal shear and may be magnified by rapid changes from a high pressure to a much lower pressure that may injure structural microelements that support alveolar structure and function [28]. While these concerns are theoretic and intuitively attractive there is contradictory data, especially in robustly evaluated animal models. Regardless of mode, recruitment must adhere to lung protective ventilation guidelines as noted earlier [22]. An infrequently discussed risk is that with near total lung capacity recruitment, the chest radiograph may underrepresent

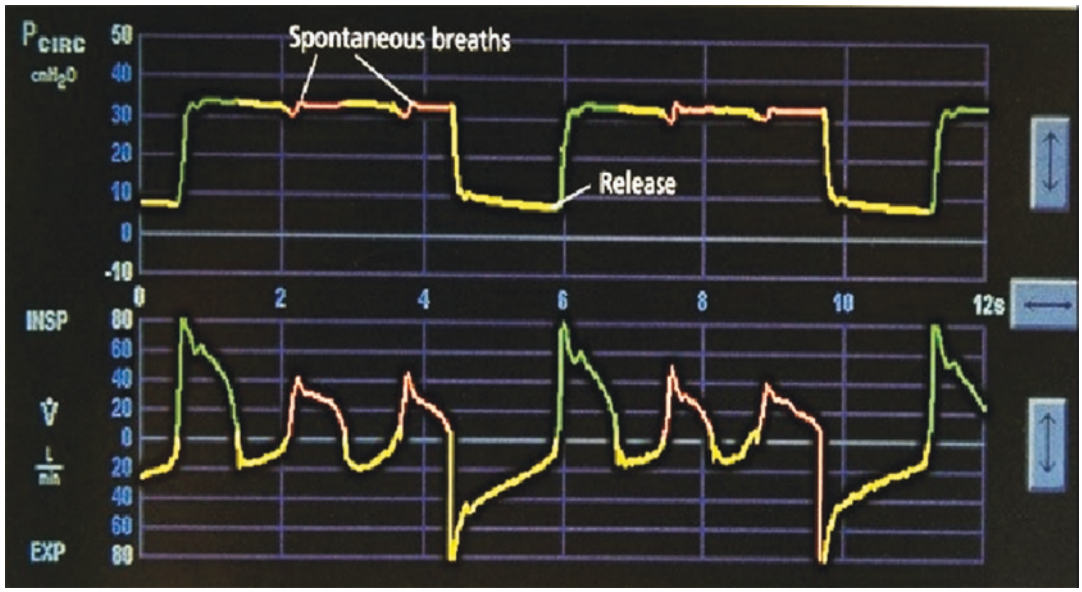

Fig. 22.10 Bilevel ventilation

the degree of pulmonary injury or infection and misinform team decisions regarding therapy. This may be especially true when using APRV or BiLevel Ventilation. The converse concern—potential overestimation of pulmonary injury or infection—may occur when the other approach is embraced where only the available lung is ventilated and no efforts are made to recruit the rest of the pulmonary parenchyma.

Ventilate only the Available Lung

This approach reflects lessons learned from the ARDSNet trial where a reduced VT demonstrated improved survival compared to a much higher VT in a principally medical patient population with a significant proportion of patients with pneumonia. Recognizing that there is a smaller than usual volume of pulmonary parenchyma and airspace that is available to participate in oxygenation and CO_2 clearance compared to the pre-illness time frame, the ventilator prescription is adjusted to deliver smaller volumes of gas. Conceptually, the reduced lung volume is termed a "baby lung." As the abnormal lung recovers from infection or injury, volumes may be increased, but do not necessarily need to be augmented to support liberation from mechanical ventilation. Since the reduced delivered gas volume is designed to also reduce the wall stress on participating alveoli, the approach is also known as low tidal ventilation or low stretch ventilation. The prescription using this approach keys from predicted body weight (PBW) and delivers 6 ml/kg PBW as the planned tidal volume, but may be reduced to 4 ml/kg PBW based upon pressure-volume dynamics. Easy to follow tables support routine use and specify step-wise adjustments.

A consequence of this approach is that in order to reach a desired VE to support CO_2 clearance, the RR must be higher than usual as VE = RR × VT. At high RR and small VT, air hunger is not infrequent and may require the use of increased sedation [29]. This is especially common with the high-flow rates that are required to deliver the specified VT so as to avoid compromising Te; flow rates of 80–100 LPM are not uncommon in conjunction with RR of 30. While the majority of the early data supporting this approach was generated in medical patients, it has been successfully utilized across the spectrum of surgical patients including those with injury. Indeed, in many locations, this approach has been embraced as the ideal fashion in which to engage in lung protective ventilation and avoid ventilator induced lung injury (VILI).

The benefit derived from using low (compared to higher) tidal volume ventilation vastly exceeds benefits in trials evaluating a specific CO_2 range, pre-planned PEEP levels. Some additional benefit appears to accrue for those with severe ARDS when a low stretch approach is combined with short-term neuromuscular blockade (and sedation), especially when a very high RR and low VT are prescribed [30].

Potential Risks with Using a Low Tidal Volume/Low Stretch Approach

Despite using this approach to support lung protective ventilation, with progressive lung injury, the volume of the baby lung can continue to decrease. If this occurs and is unrecognized, providing the same VE and VT to an even smaller volume of available lung can overdistend and injure the vulnerable alveoli and inadvertently promote VILI. Since cycling frequency can worsen intra-tidal shear and create inflammation, high RR can also worsen injury. As with the approach outlined above, the use of a low VT prescription can overestimate pulmonary injury or infection when evaluated using only chest radiography. Vascular crowding and hilar fullness may be difficult to evaluate as well. To improve the advantages of mechanical ventilation, adequate sedation and neuromuscular blockade may be very helpful. Of course both the depth of sedation and the degree of neuromuscular blockade should be monitored. To assess the sedative prescription we suggest using the BiSpectral (BIS™) Index while the extent of neuromuscular blockade is best tracked and titrated using train-of-four (TOF) stimulation [31].

Non-ventilator Prescription Specific Impactors of Mechanical Ventilation

Space Occupying Lesions

Pleural space occupying lesions adversely impact the ability to recruit available pulmonary parenchyma, may impede liberation from mechanical ventilation, and serve as potential sources of infection when they have been accessed and incompletely evacuated, especially with an indwelling drainage catheter. Not all pneumothoraces require drainage, especially when asymptomatic and identified in a spontaneously breathing, negative pressure ventilating patient. However, when the decision is made to drain pleural space lesions, they should be completely evacuated as they may compress parenchyma, augment ventilation/perfusion mismatching, and increase pulmonary shunt. Failure to resolve space occupying lesions should prompt an investigation into why evacuation has failed to resolve the lesion. Such inquiry may disclose bronchial injury, diaphragmatic herniation, or leaking pseudoaneurysm from an intercostal artery or other channel.

Cardiac Performance, Acid-Base Balance, Sedation, and Fluid Balance

A host of elements influence mechanical ventilation efficacy including cardiac performance, sedation, and fluid balance. Cardiac output, which may be supported by pressor agents or inotropic agents, influences the efficacy of mechanical ventilation through its impact on pulmonary flow. Increased flow improves O_2 onloading and CO_2 offloading, provided that the pressure gradient is not so high as to promote fluid movement into the alveolar space. Recall that pH also changes pulmonary artery tone. Tone is increased with acidosis and decreased with alkalosis. In this way, deliberate alkalosis (pH ~ 7.45) may be helpful to support RV ejection fraction and improve overall pulmonary flow and cardiac performance. While not necessary as a routine, patients with pulmonary hypertension, or those with elevated CO_2 despite a bioappropriate VE, may derive specific benefit.

Sedation also interacts with the movement of fluid into alveoli. Inadequate sedation leads to increased negative pressure at the start of a breath and may promote alveolar flooding. More commonly, inadequate sedation leads to either increased chest wall resistance or asynchrony with delivered breaths. Anxiety can also lead to an inappropriately high cycling frequency and therefore increase intra-tidal, overall shear, as well as worsen asymmetric distribution of gas to more compliant alveoli. Alveolar fracture, increased inflammation, and inadvertent lung injury may result.

Perhaps no other aspect of usual care has enjoyed as much scrutiny as fluid resuscitation. The last two decades have witnessed a major shift in management from large volume crystalloid-based resuscitation to damage control or hemostatic resuscitation using either specific ratios of component transfusion products (i.e. 1:1:1::PRBC:FFP:PLT) or, increasingly, whole blood [32, 33]. At the same time, substantial attention has been paid to decreasing overall salt and water loading even for conditions that are unassociated with hemorrhage. The recognition of untoward consequences such as secondary abdominal compartment syndrome, anasarca, anastomotic dehiscence, pleural effusion, and prolonged mechanical ventilation as a result of pulmonary and interstitial edema has revised acute and critical care management [34]. Ultrasound guided assessment, passive leg raise assessment, and a host of more invasive tools help clinicians to identify those who are likely to benefit from resuscitation and demonstrate volume recruitable cardiac performance.

This pivot towards less salt and less water is singularly helpful in supporting pulmonary compliance and elastance. As such, when the pulmonary parenchyma is already directly injured from penetrating or blunt chest trauma, the added deleterious impact of extravascular lung water, as well as alveolar edema is anticipated to prolong both ventilator and ICU length of stay. With judicious fluid management, and perhaps the adjunctive use of vasopressor agents with euvolemia, excess pulmonary morbidity is avoidable.

Prone Positioning

In the midst of the SARS-CoV-2 pandemic, prone positioning has gained more prominence for both intubated and non-intubated patients with hypoxemic acute respiratory failure or distress [35]. As for the ARDS patients, evidence suggests that prone position therapy for up to 16 h/day, for 3 or 4 days, improves alveolar recruitment and therefore helps resolve hypoxemia and hypercapnia. Similar findings are also noted for those who do not require intubation [36, 37]. Prone positioning may be combined with a variety of forms on non-invasive ventilation including CPAP, helmet CPAP, or high-flow nasal cannula therapy [38].

While specialty beds are available, they are not necessary to engage in safe prone position therapy. Careful attention to endotracheal tube, vascular catheters, chest drainage tubes, and bony prominences is essential especially in traumatic patients. In order to keep devices and tubes and lines aligned, it is a best practice to place arterial access and central venous access on the same side of the body. Since prone position therapy may be less common in many ICUs, many facilities have assembled prone position therapy teams leveraging the expertise of teams that routinely prone patients in the OR [39]. Such teams include, but are not limited to, those of OR nursing, anesthesia, orthopedics, neurosurgery, and general surgery.

Liberation from Mechanical Ventilation

Once a patient is placed on mechanical ventilation, the critical care team has several sequential tasks. First, ventilation should match the patient's needs in terms of WOB support, oxygenation, and CO_2 clearance. Second, with every interaction, the team should assess whether the current level of support is still required. Third, the team should assess whether the patient is ready to pursue liberation from mechanical ventilation. Many approaches to these elements have been advanced. One integrated approach is that of the ICU Liberation group that has crafted, deployed, and assessed a bundle to help guide teams and patients towards liberation from mechanical ventilation as a means of decreasing the risk for the post-intensive care syndrome [40]. The bundle is alphabet based and appears to be highly effective. Other approaches exist as well. Regardless of approach, the method of supporting liberation from mechanical ventilation with a given ICU, and often within a facility, should be internally consistent so that every team member understands the approach and their role within it.

Weaning

For injured patients needing only short-term mechanical ventilation, weaning may be as straightforward as ensuring that sedation is minimized (or stopped), analgesia is minimized, and a spontaneous breathing trial (SBT) is undertaken. A SBT is designed to ask and answer whether the patient is able to support their own work of breathing if the resistance imposed by the decreased diameter of the endotracheal tube (compared to their native airway) as well as that of the ventilator tubing is abrogated [41]. The SBT is typically provided for 30 min and is accompanied by an overall assessment of secretion management, WOB, oxygenation, hemodynamics, and the rapid-shallow-breathing-index (RSBI). The RSBI is a numeric score derived by calculating the RR/VT (L). For instance, a RR of 20 and a VT of 600 yield a RSBI of 20/0.6 = 33.3. RSBI <108 is associated with liberation success; higher scores are more strongly associated with the need for reintubation. Other metrics have been previously utilized but are strongly influenced by patient effort and especially neurologic compromise.

Pressure support and PEEP are the most commonly used methods of engaging in a SBT. The specific PSV used is generally adjusted based upon the diameter of the indwelling endotracheal tube. The smaller the tube, the greater the work of breathing. The following ETT sizes and PSV combinations are commonly—but not exclusively—employed: ETT 8, PSV 5; ETT 7.5, PSV 8; ETT 7, PSV 10; ETT 6.5, PSV 12; ETT 6, PSV 15. PEEP is often weaned down to a "minimum" level of 5 cm H_2O pressure prior to engaging in a SBT. Patients with rib fractures may have weaning enabled by rib fracture fixation [42].

Patients with longer periods of mechanical ventilation may be concomitantly deconditioned and have sustained lean body mass reduction as a result of hypercatabolism after injury or infection. Such patients may benefit from a more prolonged approach to weaning. Several approaches are appropriate including, but not limited to: several 30 min SBTs per day, progressive SBT lengthening, transition to only PSV/PEEP ventilation support with gradual reduction in PS each day. Other approaches will depend on the specific approach to ventilation that is being utilized. For example, patients managed using ARPV do not need to change to PSV/PEEP for weaning. Instead, progressive decreases in the high pressure combined with progressive increases in the time at the high pressure decrease the amount of ventilator support and lead to settings that appear quite similar to CPAP. When the high pressure reaches 10 cm H_2O pressure, liberation may be entertained (total pressure is similar to PSV 5/+5 PEEP).

Patients who require prolonged ventilation (> 2 weeks) often benefit from tracheostomy placement.

Tracheostomy

The specific indications for tracheostomy placement after chest injury are beyond the scope of this chapter [43]. Instead, we will explore weaning patients who have undergone tracheostomy placement. While a SBT and a RSBI may remain useful in those with a tracheostomy, tracheostomy collar (TC) trials are more commonly utilized, especially for those who are deconditioned [44]. A TC trial consists of a special collar attached to a humidified air/oxygen blended source that blows the humidified gas mixture past the tracheostomy orifice. Three general approaches are used to liberate tracheostomy patients from mechanical ventilation: (1) short, frequent periods that are repeated several times per day, (2) one or two longer TC trials per day (but not to the point of fatigue), and (3) scheduled short to progressively longer periods of TC trial interspersed with a fixed but short period of rest. One of the authors (L.J.K.) has used the latter to great effect. An example is provided in Table 22.1.

Table 22.1 A schedule for tracheostomy trial weaning for deconditioned patients

Day	TC time (hours)	Rest time (hours)	Cycles/ day	Recovery time on support
1	1	3	3	12
2	2	2	3	12
3	3	1	3	12
4	12	0	1	12
5	16	0	1	8
6	20	0	1	4
7	24	0	1	0

Recall that while on TC, the patient's PEEP is atmospheric pressure which is generally 0 cm H_2O pressure. The lack of PEEP worsens derecruitment by increasing atelectasis and initiating all of the linked untoward effects on hypoxic pulmonary vasoconstriction. The undesirable effect of extravascular lung water on pulmonary compliance will be readily identified in patients undergoing TC trials as an increased WOB, fatigue, and a lack of progress.

Regardless of which method is selected to wean a patient, care must be ensured to avoid patient fatigue. Should this occur, weaning will likely be derailed and require a period of recovery that may exceed 24 h. Moreover, weaning could be impeded by inadequate nutritional support that should be tailored to the patient's specific needs.

Conclusions

Thoracic injury that requires invasive mechanical ventilation to help manage may be straightforward or quite complex. The critical care team must be conversant with multiple modes and methods of invasive ventilation support to best match the type of support the patient requires. The plethora of critical care support techniques that help manage the rest of the patient including analgesia, sedation, and fluid resuscitation also directly influence the efficacy of mechanical ventilation and should be integrated into a single internally consistent approach to care. These kinds of management elements may be embraced, leveraged, and applied by specifically trained intensivists of all types regardless

of parent specialty training to achieve the best outcomes for the most critically ill and injured patients.

Disclosures M.C. is the President of the European Society of Critical Care Medicine; L.J.K. is a Past-President of the Society of Critical Care Medicine.

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Respiratory Adjuncts

23

Maurizio Cereda, Andrew Gold, and Brian Luu

Introduction

Trauma patients are at risk of pulmonary complications including pneumonia, acute lung injury, ARDS, and unplanned intubation after injury. These risks are magnified in the setting of chest injury whether penetrating or blunt [1]. Blunt chest injury resulting in rib fractures and/or pulmonary contusion impedes respiratory function by several mechanisms including but not limited to mechanical disadvantage, increased inspiratory work, and decreased maximal cough strength [1]. Decreased respiratory competency coupled with injury-associated pain impedes a patient's willingness and/or ability to support deep inspiration leading to low atelectasis and reduced available and perfused lung volume. Trauma patients often spend an extended period of time immobilized during and after complex operative management and concomitantly receive medications that impede respiratory drive [1]. Trauma patients—especially the elderly—demonstrate comorbid pre-injury conditions such as COPD or asthma that may be exacerbated after injury. In our aging population, patients with pre-existing lung disease coupled with age-related pulmonary changes comprise an increasingly larger proportion of the trauma population [1]. In addition, tobacco use is a major trigger of chronic mucus hypersecretion [2]. In this population, respiratory therapy that combines lung expansion strategies, secretion clearance, and leverages the utility of inhaled medications underpins improved outcomes.

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Lung Expansion

Incentive Spirometry

Incentive spirometry (IS) devices encourage patients to take deep breaths using a visual target on the device in order to improve lung volumes, mucus clearance, and prevent respiratory complications [3]. Instructions for proper use are relatively simple and allow patients to use the device without direct, active therapy provided by a respiratory therapist or bedside nurse. Instead, the bedside nurse is often critical in encouraging, rather than guiding use. Disadvantages of this technique include dependence on patient effort, cooperation, and technique. Ideal use involves slow inspiration to a target volume, holding the breath for 2–3 s, followed by slow, controlled expiration [3]. A number of systematic reviews have evaluated the efficacy of IS in preventing postoperative pulmonary complications. Several

fail to demonstrate sufficient evidence to support the routine use of IS [4, 5], while one older [6] and one recent review [3] of patients following thoracic surgery found much weak and strong evidence supporting its use, respectively. While IS use makes intuitive sense and is embedded in clinical practice, the data quality is intertwined with study heterogeneity and methodologic failures. Moreover, since IS is often combined with other pulmonary support techniques, data that can be solely attributed to the impact of IS on pulmonary outcomes is difficult to parse.

Chest Physiotherapy

Chest physiotherapy (chest PT) is a broad term describing interventions aimed at promoting mucolysis and clearance via expectoration, improving lung volumes, and reducing or preventing atelectasis and subsequent pneumonia. Chest PT does not refer to a specific regimen and thus associated interventions vary widely between institutions as well as clinicians. Given the imprecise nature of the term, heterogeneity of studies assessing chest PT limits evaluation of available evidence. Nevertheless, the use of focused chest PT to prevent respiratory complications is widely used in clinical practice and is a level 2 recommendation from the 2012 EAST practice management guidelines to address the sequelae of pulmonary contusion and flail chest [7].

Traditionally, chest PT consists of manual chest percussion, postural drainage, and teaching cough and breathing techniques by respiratory therapists. This approach has been shown to be effective in promoting mucus clearance and supports reduction in bacterial bioburden as a primary therapy for pulmonary infection [8]. However, this particular approach can be time-consuming for the respiratory therapists or bedside nurses (if appropriately trained) and uncomfortable for the patient. Moreover, to effectively use gravity as an aid in drainage from dorsal domains, patients need to be positioned prone to do so. While this has become common during COVID-19 care, the percussion that is required to support mucous liberation is often at odds with rib or spine fracture associated pain. High frequency chest wall oscillatory devices, most commonly worn as a vest, provide automated, mechanical chest physiotherapy and have been shown to be more effective in mucus clearance than manual percussion and more comfortable for patients, albeit in those without thoracic cage fracture [9, 10]. These vests use high frequency pulses of positive pressure to cause flow through the airways in order to generate shear forces to break up mucus [3].

Some adverse events have been associated with chest PT. These include an increase in oxygen consumption, bronchospasm, and even rib fractures [11]. A Cochrane review assessing chest PT for adults with pneumonia did not find evidence of reduced mortality, cure rate, or improvement of chest X-ray with any interventions, but did show decreased hospital length of stay with exogenously applied positive expiratory pressure [11]. Relatedly, while IS focuses on supporting and sustaining negative inspiratory forces, other devices focus on exhalation against a fixed or variable resistance valve. Exhalation may be accompanied by vibration to mimic the effects of oscillation to support pulmonary toilet.

Handheld Oscillatory Devices

A number of handheld devices have been developed combining vibratory principles with positive expiratory pressure including the Flutter device, Acapella (Smiths Medical Inc., Carlsbad, California, USA), and Cornet (R. Cegla, Montabaur, Germany) [3]. Devices in this category use an expiratory valve that opens and closes at a high frequency which simultaneously creates expiratory positive pressure akin to PEEP as well as oscillatory flow to aid with mucolysis and clearance similar to oscillatory vests. These handheld devices are an attractive alternative/adjunct to conventional chest PT because they avoid direct percussion of the injured chest wall and can be performed by the patient independent of a respiratory therapist or bedside nurse. Devices such as the MetaNeb System (Hill-Room)

have a pneumatic compressor designed to deliver oscillation and lung expansion (OLE) using a combination of high frequency oscillation and PEEP [12]. This combination of OLE and PEEP has been used to aid in secretion mobilization, lung expansion, and treatment and prevention of atelectasis [12]. A recent study utilizing the MetaNeb system demonstrated reduced postoperative pulmonary complications in patients undergoing high risk procedures [12].

Mucus Clearance

Mucus is a mixture of glycoproteins (mucins), lipids, and water that form a viscoelastic gel-like substance. Mucus provides airway protection as it forms a barrier that promotes epithelial hydration and gas humidification [13]. It also separates airway surfaces from inhaled irritants including those found in tobacco smoke [13]. The surface liquid that covers the epithelial lining of the airways is composed of two layers: the mucus gel layer and the periciliary layer consisting of mucins and polysaccharides [2]. The role of mucus is to trap inhaled particles and pathogens [14] and inhibit bacterial growth including biofilm formation [13]. One natural defense mechanism is pulmonary mucus clearance that expels inhaled pathogens and particles by means of airflow and ciliary hair activity [14]. Defective or impaired mucus clearance can cause lung dysfunction as in patients with chronic obstructive pulmonary disease (COPD), chronic bronchitis, and intubated or postoperative patients. In addition, pathologic mucus often demonstrates increased viscosity and elasticity causing it to be more difficult to clear and more likely to become inspissated [2]. There are several mucoactive pharmacological medications that serve to aid mucus clearance. These fall into the categories of expectorants, mucolytics, and mucokinetic drugs [13]. Expectorants are designed to increase the efficacy of cough by increasing the volume of airway water/secretions [13]. The most common expectorants are guaifenesin and aerosolized saline—and in particular, hypertonic saline. Mucolytics work to depolymerize mucin [13]; these include acetylcysteine and dornase-alfa (a cystic fibrosis specific drug) [14]. Mucokinetic drugs serve to increase the effectiveness of cough via increased expiratory airway flow and by reducing the adhesive properties of mucus; aerosolized surfactant is a promising medication from the mucokinetic class that is not yet in clinical practice [13].

Expectorants

Expectorant agents were initially defined by their ability to aid in purulent secretion expulsion [13]. In practice, this has come to include medications that increase airway water/secretions (i.e. saline nebulizers) in order to improve luminal hydration and mucus viscoelasticity reduction. Expectorants do not alter mucociliary clearance by the beat frequency of airway cilia [13]. Hypertonic saline actually serves to increase the volume of both mucus and water in the airways, but has been proven to increase pulmonary function in patients with bronchiectasis by inducing cough as well [13]. However, the unpleasant taste of hypertonic saline nebulizers may limit its acceptance by patients. Guaifenesin is sold over the counter (i.e. Robitussin or Mucinex) and is designed to stimulate the secretion of airway fluid by cholinergic stimulation to increase mucus secretion from submucosal glands [13]. This serves to increase hydration and decrease mucus viscosity leading to improved secretion clearance from both upper and lower airways [2]. Guaifenesin is thought to exert its primary pharmacologic effect by the gastro-pulmonary reflex (aka. The neurogenic theory) by stimulating receptors in the gastric mucosa [2]. Guaifenesin may also directly stimulate vagal centers attached to bronchial secretory glands which enhance respiratory secretion [2]. Together, these actions of guaifenesin act to reduce mucus surface tension and viscosity, increase mucociliary clearance, inhibit cough reflex sensitivity, decrease mucin production, and increase mucociliary transport [2]. However, guaifenesin has not been proved to be clinically beneficial in any randomized controlled trial. Nonetheless, it serves as a reasonable adjunct to

support secretion clearance as it has a quite favorable side-effect profile and is easily administered by mouth or enteral access catheter.

Acetylcysteine

Mucolytics work by altering mucus biophysical properties. The most widely recognized mucolytic agent is acetylcysteine (also known as N-acetylcysteine) which facilitates mucus clearance by decreasing viscosity. It works by hydrolyzing mucin disulfide bonds that link mucin monomers via a free thiol group [13, 14]. A report compiled by the Canadian Agency for Drugs and Technologies in Health (CADTH) [14] examined evidence-based guidelines addressing the use of acetylcysteine and noted that recommendations vary widely between medical professional organizations. This report included recommendations from the American Association of Respiratory Care (AARC), American Thoracic Society (ATS), American College of Chest Physicians (CHEST), Canadian Thoracic Society (CTS), European Thoracic Society (ERS), National Institute of Health Care and Excellence (NICE), the Department of Veterans Affairs and Department of Defense (VA/DoD). While there is a lack of guideline uniformity with regard to inhaled therapy for mucolysis, there are conditional recommendations (based on moderate evidence) to use oral acetylcysteine for patients with COPD [14]. However, other guidelines are equivocal due to reported insufficient evidence, and still other guidelines recommend against the use of acetylcysteine for acute cough and the use of aerosolized acetylcysteine for hospitalized patients [14]. Nonetheless, N-acetylcysteine use seems entrenched in practices around the globe. Relatedly, the CADTH report does mention guidelines from countries including England, Wales, Poland, Russia, Germany, and Spain where acetylcysteine use is recommended for patients with viscous secretions or those with prior COPD [14]. There is a theoretic but unsubstantiated risk that inhaled acetylcysteine can increase airway inflammation and infection by disrupting the natural protective mucin layer [13]. The vast heterogeneity of these guidelines is an indication that strong evidence does not exist either supporting or rejecting the regular use of acetylcysteine as a mucolytic respiratory adjunct. It is commonly available and readily deliverable by nebulizer.

Bronchodilators

Beta2-Agonists

Bronchodilators work via beta-2 agonism to relax airway smooth muscle. Through bronchodilation, beta-2 agonist relieves bronchospasm and reduces airway resistance [15]. Most beta-2 agonists are delivered by inhalation where there is minimal systemic absorption. Inhaled bronchodilators are useful in invasively ventilated patients with reversible airway obstruction (i.e. COPD and/or asthma) [16] and can enhance the effectiveness of cough in patients who have experienced augmented expiratory airflow after administration [13]. While beta-2 agonist does increase ciliary beat frequency, they only have minimal effect on mucociliary clearance and may theoretically induce airway collapse in patients with bronchomalacia [13]. Common beta-2 agonists include albuterol, salmeterol, and levalbuterol; other formulations exist without superiority demonstrated for a particular agent. There is quite little evidence to support bronchodilators use in patients with acute respiratory failure without airway obstruction.

In addition to bronchodilation, beta-2 agonists have been shown in vivo to exert some anti-inflammatory effects. The articulated mechanism includes inhibiting plasma exudation in the airways, inhibiting mast cells from secreting bronchoconstrictor mediators such as histamine and leukotrienes, and retarding the release of mediators from eosinophils, macrophages, T-lymphocytes, and neutrophils [17]. It should be noted that while salbutamol use in ARDS was associated with reduced extravascular lung water and reduced plateau pressures, it is also associated with increased 28-day mortality and thus is not recommended [18].

Muscarinic Antagonist

Acetylcholine, the main neurotransmitter of the parasympathetic nervous system, plays a critical role in the regulation of airway tone, smooth muscle contraction, and mucus secretion [19]. Acetylcholine primarily exerts its effects on airway smooth muscle via interaction with muscarinic receptors. Inhaled muscarinic antagonist includes medications such as ipratropium (short acting) and tiotropium (long acting). These inhaled anticholinergic medications work via blockade of the muscarinic receptors resulting in decreased cyclic guanosine monophosphate (cGMP) production [20]. This results in decreased airway secretions and bronchial smooth muscle dilation. Inhaled anticholinergics have been FDA approved for the treatment of bronchospasm associated with COPD, but are also indicated for the use of asthma exacerbations and as an aid in the clearance of secretions in intubated ICU patients [20]. Inhaled muscarinic antagonists may also modulate airway inflammatory responses suppressing acetylcholine mediated release of chemotactic substances [21].

Rescue Treatment

Heliox

Heliox is a mixture of the noble gas helium and oxygen usually delivered in ratios of helium:oxygen of 80:20 or 70:30 [16]. Helium has a much lower density than oxygen or nitrogen; thus, using helium as a carrier gas with oxygen increases laminar flow which may help to reduce the work of breathing and prove useful in the setting of partial upper airway obstruction or obstructive lung diseases such as asthma or COPD [16]. There is some evidence to suggest that heliox may be a superior driving gas for delivering nebulized medications to distal airways by facilitating the movement of aerosolized drug through partially obstructed regions of the bronchial tree [16].

Inhaled Pulmonary Vasodilators

The two most commonly used inhaled pulmonary vasodilators include inhaled nitric oxide (iNO) and inhaled prostacyclin (epoprostenol). iNO is a colorless and odorless gas that works by activating cyclic GMP to cause pulmonary vasodilation and decrease pulmonary vascular resistance [16]. When inhaled, nitric oxide diffuses across alveolar cells to its site of action in pulmonary arteriole smooth muscle. Side effects of iNO include platelet dysfunction, methemoglobinemia, pulmonary edema, and rebound pulmonary hypertension when it is not appropriately tapered [16]. Its use is safe at concentrations less than 80 ppm [16]. Inhaled prostacyclins work via upregulation of cyclic AMP resulting in pulmonary vasculature smooth muscle relaxation. Common side effects include headache, jaw pain, nausea, vomiting, flushing, and platelet dysfunction [22]. Both inhaled prostacyclin and iNO can help unload the right ventricle by reducing right ventricular afterload via pulmonary vasodilation. This is a key action in those who demonstrate hypoxic pulmonary vasoconstriction with secondary pulmonary hypertension, those with preexisting pulmonary hypertension, and if those with acidosis induced pulmonary artery vasoconstriction. These vasodilators also can improve ventilation-perfusion matching by preferentially increasing blood flow to well ventilated areas of the lung and can improve oxygenation in cases of severe hypoxemia [16]. Both iNO and epoprostenol have been shown to improve oxygenation in patients with ARDS, but this has not correlated to improved mortality or ventilator free days. Thus, not all centers use these agents, and in centers that have them available, use is often focused on specific patient populations as noted above.

Magnesium

Magnesium, a ubiquitous intracellular cation plays a key role in muscle tone [23], especially that of smooth muscle. Long used as a tocolytic to arrest pre-term labor [24], and as an

antihypertensive agent to manage pre-eclampsia or eclampsia [25], magnesium also has an adjunctive role in managing bronchoconstriction. Accordingly, a variety of studies have assessed the ability of intravenous magnesium to suppress cough during anesthesia, as well as rescue pediatric and adult patients with severe asthma that is refractory to standard therapy [26]. Reductions in fentanyl-induced cough during general anesthesia [27], as well as hospital admission for asthma exacerbation have been noted [28]. The limited data available regarding magnesium therapy for bronchoconstriction in COPD patients supports magnesium as an enabler of other brondilators rather than an efficacious solo therapeutic [29]. While there is reasonable data on IV magnesium supplementation, there is little supportive data on nebulized magnesium as a viable therapeutic [30].

Noninvasive Respiratory Support

High Flow Nasal Cannula (HFNC)

HFNC systems are designed to administer heated humidified high airflow through nasal prongs. The high gas flow—up to 60 L/min—ensures minimal entrainment of room air, thus allowing more precise FiO_2 delivery compared to standard nasal cannula therapy [16]. HFNC also serves to decrease dead space ventilation by washing out expired gas from the upper airways, thus increasing the inspired oxygen concentration during subsequent breaths. This results in a reduced minute ventilation requirement and is often manifested by a lower respiratory rate in patients being treated with HFNC [16, 23, 31]. The high flow may relieve dyspnea by reducing airway inspiratory resistance while impeding expiratory flow resulting in continuous positive airway pressure analogous to low levels of positive end expiratory pressure (PEEP) [16]. According to Prake and colleagues [32], hypopharyngeal pressure increases by about 1 cm H_2O per 10 L/min of HFNC. However, these low levels of PEEP may only be relevant with mouth closed breathing as Chanques and colleagues [33] demonstrated this effect of HFNC to be lost with an open mouth [16]. In patients with hypoxic respiratory failure, the use of HFNC has been shown to decrease the need for invasive mechanical ventilation compared to standard oxygen therapy; improved tolerance is also noted compared to mask-based forms of NIV [16]. HFNC is also beneficial in the post-liberation setting resulting in fewer re-intubations at 72 h compared to conventional oxygen therapy [34]. However, in a recent large multicenter study comparing patients at high risk for re-intubation, a combined approach with immediate NIV followed by HFNC therapy significantly reduced the re-intubation rate within the first 7 days compared to HFNC alone post-extubation [26]. This was in contrast to a previous study that reported that HFNC was non-inferior to noninvasive ventilation in preventing extubation failure in high risk patients [34, 35]. While controversy exists regarding the optimal use of HFNC, it is readily deployable, well tolerated, and appears efficacious in addressing hypoxic acute respiratory distress. When CO_2 clearance is the dominant aspect of acute respiratory distress, NIV modalities appear more appropriate.

Continuous Positive Airway Pressure (CPAP) and Bi-level Positive Airway Pressure (BiPAP)

CPAP and BiPAP are both methods of noninvasive mechanical ventilation (NIV) that are useful in patients with hypoxemic or hypercapnic respiratory failure [36]. CPAP, like HFNC, reduces work of breathing by providing higher oxygen concentrations, reducing airway resistance, and preventing atelectasis [37]. CPAP is also used for obstructive sleep apnea by maintaining patency of the upper airway [38]. Unlike HFNC, CPAP for acute respiratory distress management uses a facemask that covers both oral and nasal cavities in order to create a seal over the respiratory passages. Therefore, CPAP provides positive airway pressure regardless of open-mouth breathing. Alternatively, CPAP can be provided using a helmet device that encircles the head and seals

around the neck. CPAP delivered by helmet has been shown to be as effective as CPAP via facemask, though these devices are not approved by the FDA for use in the USA [39]. Emergency Use Authorization during the early phase of COVID-19 witnessed widespread use of helmet-based CPAP for those requiring oxygenation support with, or without prone position therapy [40]. BiPAP differs from CPAP in that a higher level of positive airway pressure is used during inspiration (IPAP) and a lower pressure (or no positive pressure) is used during expiration (EPAP) in order to further reduce the work of breathing and support pulmonary recruitment. Inspiratory pressure support with BiPAP is associated with decreased work of breathing with an additional benefit when expiratory positive pressure is applied [41]. In patients with acute pulmonary edema, BiPAP compared to CPAP has been shown to improve pH, $PaCO_2$, respiratory rate, and heart rate [36]. Both CPAP and BiPAP are ideally targeted to support while there is an underlying problem that may be concomitantly addressed—pulmonary edema is a typical condition that is well managed using NIV in combination with adjunctive therapy.

A systematic review assessing NIV (either CPAP or BiPAP) in patients with chest trauma by Chiumello and colleagues, compared to invasive mechanical ventilation or unassisted oxygen therapy noted that NIV resulted in reduced mortality, intubation rate, ICU length of stay, and improved oxygenation [42]. A Cochrane review evaluating NIV as a weaning strategy from invasive ventilation found overwhelmingly positive results including significantly improved mortality, reduced weaning failure, reduced ICU and hospital length of stay, reduced duration of intubation, rates of re-intubation, ventilator-associated pneumonia, and tracheostomy [43].

The limitations of NIV should be considered when deciding between noninvasive mechanical ventilation or invasive mechanical ventilation. Leaks around the mask and upper airway resistance can decrease both the delivered gas volume and pressure as well as the efficacy of CPAP and BiPAP in reducing airway resistance and alveolar recruitment [37]. The risk of aspiration should also be considered in patients without airway protective reflexes. NIV is also associated with skin breakdown due to the mask pressure required to maintain an adequate seal which may be impossible in patients with certain anatomical features [41]. CPAP via helmet circumvents some of those disadvantages by providing a seal around the neck rather than the face [39]. There is little evidence supporting BiPAP for patients with pneumonia after chest injury as expectoration of the bacterial bioburden is impeded by EPAP and the facemask seal. Thus, for those with acute respiratory failure related to pneumonia coupled with a large secretion burden, invasive mechanical ventilation appears more appropriate and readily facilitates secretion clearance using standard in-line suction devices or flexible bronchoscopy.

Conclusions

Patients with chest wall injury are at particularly high risk for pulmonary complications. Fortunately, there are a host of respiratory adjuncts that help to facilitate gas exchange, work of breathing, and prevent and treat complications. Incentive spirometry and handheld oscillatory devices help re-expand atelectatic lung. Expectorants and mucolytics such as aerosolized saline and inhaled acetylcysteine aid with clearance of respiratory secretions. Bronchodilators are widely used, although their utility is limited in patients without airway obstruction. HFNC is an attractive tool to reduce work of breathing and is excellent at oxygenation support, but only provides limited CO_2 clearance. CPAP and BiPAP are both useful NIV modalities that decrease work of breathing, improve respiratory mechanics, and support both oxygenation and CO_2 clearance. Both CPAP and BiPAP may be limited by aspiration risk in those with disordered consciousness, poor mask seal, or a large secretion burden. Overall, a multimodal approach that combines several interventions optimally targets improved patient outcomes.

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Lung Rescue and ECMO

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Abbreviations

ACT	Activated clotting time
AMICAR	Aminocaproic acid
aPTT	activated partial thromboplastin time
ARDS	Acute respiratory distress syndrome
CESAR	Conventional ventilation or ECMO for severe adult respiratory failure
CO_2	Carbon dioxide
DOD	Department of Defense
ECMO	Extracorporeal membrane oxygenation
ELSO	Extracorporeal Life Support Organization
EOLIA	EcmO to rescue Lung Injury in severe ARDS
IBW	Ideal body weight
ICD-9	International Classification of Diseases-9
IJ	Internal jugular
INR	International normalized ratio
IVC	Inferior vena cava
LMWH	Low molecular weight heparin
LVAD	Left ventricular assist device
P_aCO_2	Partial pressure of arterial carbon dioxide
P_aO_2	Partial pressure of arterial oxygen
PEEP	Positive end expiratory pressure
SVC	Superior vena cava
TBI	Traumatic brain injury
TBSA	Total body surface area
TXA	Tranexamic acid
UNFH	Unfractionated heparin
VA	Venoarterial
VA-V	Venoarterial-venous
VILI	Ventilator induced lung injury
VV	Venovenous
VV-A	Venoveno-arterial

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Introduction

Extracorporeal membrane oxygenation (ECMO) evolved following the development of the first cardiopulmonary bypass machine. Kolff and Kolobow demonstrated that a cellophane or silicone membrane oxygenator could be used for gas exchange while limiting hemolysis, platelet destruction, and systematic inflammation [1, 2]. The first documented case of extracorporeal support after injury occurred in 1972. The case involved a 24-year-old patient who developed trauma-induced acute respiratory distress syndrome (ARDS) refractory to conventional ventilator support (tidal volume of 1 L, PEEP of 8 cmH_2O, and fraction of inspired oxygen 100%). Venoarterial ECMO (VA-ECMO) was initiated, and the patient was successfully weaned from the circuit after 75 h, making a full recovery [3]. Then in 1979, Luciano Gattinoni developed the concept of venovenous extracorporeal pulmonary support (VV-ECMO) for hypercarbic and/or hypoxemic respiratory failure [4].

Several thorough reviews address the current management of severe acute respiratory failure, including the optimal application of ECMO [5, 6]. In addition, the Extracorporeal Life Support Organization (ELSO) offers online resources on ECMO management [7] as well as a comprehensive text [8]. The following chapter briefly summarizes the principles of ECMO, the different ECMO modalities, and the application of this support or rescue therapy to critically injured patients with particular focus on those with thoracic injuries.

ECMO Configuration and Modes

ECMO can provide cardiac support, respiratory support, or both. Typical configurations are: (1) VV which provides respiratory support, (2) VA which provides cardiac and some respiratory support, and (3) venoveno-arterial (VV-A) which provides both cardiac and respiratory support [9]. Mode selection flows from patient needs as the approach must be individualized for optimal outcome.

Venovenous ECMO

VV-ECMO is accomplished by draining blood via a large central vein, passing it through a pump and oxygenator, and returning it to a large central vein. In doing so, the work of the lungs is augmented as blood rich in oxygen and devoid of carbon dioxide (CO_2) is returned to the right heart. There is no "bypassing" of the cardiopulmonary system in VV-ECMO. Rather there is a net zero flux in blood flow, with oxygenated blood returned directly to either the vena cavae or right atrium for continued flow through the pulmonary vascular bed.

VV-ECMO can be accomplished via one of the several cannulation strategies [10] (Fig. 24.1). Single site VV-ECMO can be accomplished with a single dual lumen cannula which drains blood from both the inferior vena cava (IVC) and superior vena cava (SVC) and returns it directly to the right atrium [11, 12]. This cannula is placed into either the right or left internal jugular (IJ) vein and obviates the need for groin access [13]. There is a theoretical risk of less recirculation with this cannulation technique because the reinfused blood is returned directly across the tricuspid valve, thus mitigating the risk of well oxygenated blood being recirculated through one of the IVC or SVC drainage ports.

Additionally, VV-ECMO can be performed via a two site technique with drainage from the IVC, requiring access via the femoral vein. When drainage is accomplished via the femoral vein, the reinfusion cannula can be placed in the right or left IJ, depending on patient factors. Those factors include central line placement location, venous thrombosis, or other flow limiting or anatomically limiting conditions. The reinfusion cannula may also be placed in the contralateral femoral vein, but must be positioned so that the reinfusion cannula tip is more proximal to the heart than the drainage cannula. Recirculation is a common problem in dual site techniques, but can be mitigated with optimal positioning, with catheter placement often benefitting from image guidance [10].

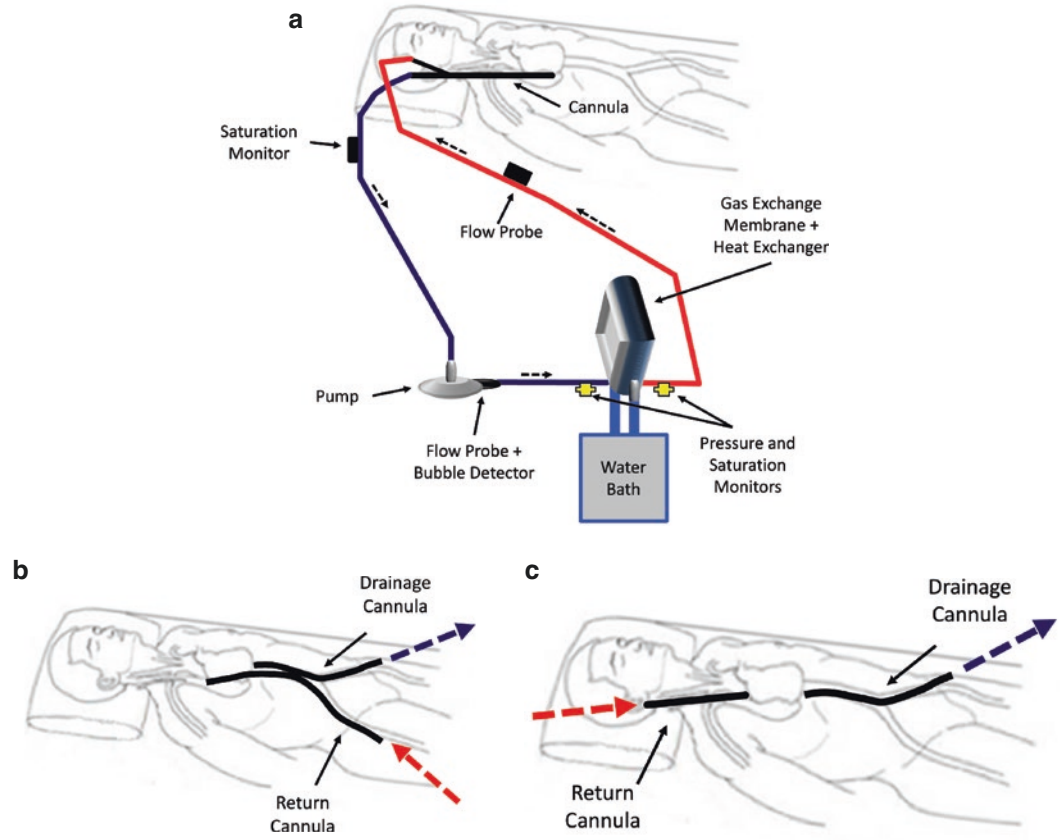

Fig. 24.1 Overview of venovenous extracorporeal membrane oxygenation (VV-ECMO). Typical components in a VV-ECMO system (**a**). Cannulation strategies include single site cannulation (**a**), dual femoral venous cannulation (**b**), or femoral-jugular cannulation (**c**). (Reprinted from MacKay EM, Cannon JW. Extracorporeal Membrane Oxygenation in the Unstable Trauma Patient. In: Hörer T, DuBose JJ, Rasmussen TE, White JM, editors. Endovascular Resuscitation and Trauma Management. Berlin: Springer International Publishing; 2020. p. 216, with permission)

Venoarterial ECMO

VA-ECMO is also accomplished by draining blood from a large central vein, or the right atrium, but the oxygenated blood is returned from the device via a peripheral or central artery. In doing so, the drained blood will bypass the cardiopulmonary system, thus offloading the work of the heart and lungs, and providing hemodynamic, and some respiratory, support to the rest of the body. Two configurations are currently employed: peripheral and central.

In peripheral VA-ECMO, venous drainage is often accomplished via the femoral veins, with drainage from the Right atrium/IVC. Additionally, it is possible to drain from an IJ vein approach with drainage from the right atrium/SVC. Reinfusion is most commonly performed via femoral arterial access, as this is the most easily accessible artery for rapid and efficient cannulation. This method of peripheral VA-ECMO provides excellent hemodynamic support, but does not provide much respiratory support to the upper half of the body. There are some centers that prefer to drain from the IJ vein when cannulating peripherally for VA-ECMO because the upper half of the body, which drains into the SVC, will have more poorly oxygenated blood, thus mitigating the chance of having a "dual circulation" system [10].

VA-ECMO can also be performed centrally, with access via the right atrium for drainage and aorta for reinfusion. This method is most commonly employed either post-cardiotomy, or if the chest is already opened for another reason such as for thoracic injury management. This provides excellent hemodynamic and respiratory support to the entire body, but is much more invasive and time consuming and is largely unsuitable for out of OR application in most sites.

Hybrid ECMO

At times, a secondary insult may occur in critically ill patients on ECMO support. In these cases, additional measures can be embraced beyond conventional ECMO. In patients on VV-ECMO who suffer from new onset cardiogenic shock it is possible to convert them to VV-A ECMO, which would then provide both respiratory and cardiac support. This is accomplished by having two reinfusion cannulas, split with a "Y-connector." In this configuration blood is directed to a central vein for respiratory support, and to a central or peripheral artery, to provide cardiac support.

Additionally, patients on peripheral VA-ECMO may suffer evolving acute lung injury, where their oxygenation and ventilation needs are not met by VA support. Such patients benefit from the placement of a second reinfusion cannula into a central vein. Again, in this VA-V ECMO mode, the reinfused blood is split with a "Y-connector" with blood being directed to a central vein for respiratory support and a peripheral artery for oxygenation support. All of the above techniques require safe and efficient cannula placement—a process that may even occur outside of an acute care facility as has been demonstrated by the French SAMU organization.

Cannula Insertion Technique

ECMO cannulation can be accomplished by one of the several approaches—percutaneous, open, or hybrid. Percutaneous ECMO cannulation is the most common technique and is often employed by surgeons and non-surgeons alike. This technique is similar to central line placement, with vascular access being achieved with a needle and wire using a Seldinger technique. Several commercially available kits match vessel dilator size with cannula size and support safe insertion.

The open approach to cannulation can be helpful in certain subsets of patients, including the morbidly obese, those without palpable pulses, or cases in which there is no ultrasound equipment available. This approach is achieved with a surgical cutdown over the desired vessel with the target vessel being cannulated under direct visualization.

The hybrid cannulation technique requires a small skin incision directly over the target vessel. The Seldinger technique is then used, as in the percutaneous approach, via the skin at a site adjacent to, but outside of, the cutdown field. This approach allows for direct visualization of percutaneous cannulation and then allows for closure of the skin incision. This approach is much less commonly used than the two aforementioned techniques.

ECMO for Pulmonary Failure

All-Cause ARDS.

ARDS is a heterogenous inflammatory pulmonary insult first described in 1967 that is often triggered by infection or injury [14]. The syndrome is defined by arterial hypoxemia, hypercapnia, and loss of pulmonary compliance, without a predominant cardiac etiology, but is instead accompanied by increased permeability of the alveoli-capillary membrane. The diagnostic criteria for ARDS have undergone multiple revisions and are currently articulated as the "Berlin Definition" [15] (Table 24.1). ARDS is defined as the sequela of a pulmonary insult either direct or indirect. Direct insults to the alveolar epithelium include bacterial, fungal, or viral pneumonia (such as SARS-Co-V-2), direct pulmonary injury (i.e. thoracic crush or blast injury),

Table 24.1 The Berlin Criteria for acute respiratory distress syndrome

Timing	Within 1 week of a known clinical insult of worening respiratory symptoms
Chest imaging (CT or CXR)	Bilateral opacities—Not completely explained by effusions, lobar/lung collapse, or nodules
Origin of opacities	Respiratory failure is not fully explained by cardiac failure or fluid overload
Oxygenation	
– Mild	$PaO_2/FiO_2 \leq 300$ mm Hg with PEEP or CPAP ≥ 5 cm H_2O
– Moderate	$PaO_2/FiO_2 \leq 200$ mm Hg with PEEP or CPAP ≥ 5 cm H_2O
– Severe	$PaO_2/FiO_2 \leq 100$ mm Hg with PEEP or CPAP ≥ 5 cm H_2O

CPAP, continuous positive airway pressure; FiO_2, fraction on inspired oxygen; PaO_2, partial pressure of oxygen; PEEP, positive end expiratory pressure

or aspiration of gastric acid. Indirect or extra-pulmonary insults may result from non-pulmonary sepsis, pancreatitis, or extra-thoracic injury.

Optimal conventional ARDS management employs a variety of approaches that deliver lung-protective ventilation (Fig. 24.2). Despite optimal ventilator management and supportive strategies, ARDS-related mortality ranges from 30 to 46% [16]. If conventional therapy proves to be inadequate to meet oxygenation and carbon dioxide (CO_2) clearance needs VV-ECMO is a recognized salvage therapy. VV-ECMO allows for sufficient total blood oxygenation and CO_2clearance while allowing for lung-protective or "ultra-protective" ventilation strategies. Although the initial trials investing ECMO in ARDS did not identify a clear benefit but noted a

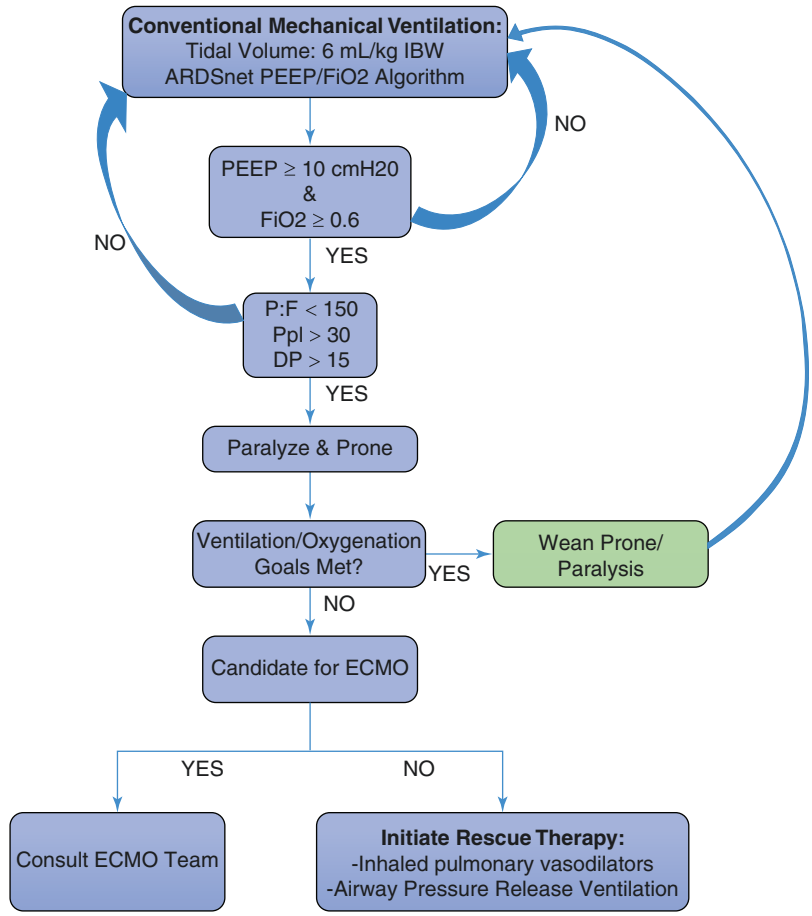

Fig. 24.2 One approach to ARDS management. IBW, ideal body weight; ARDSnet, acute respiratory distress syndrome network; PEEP, positive end expiratory pressure; FiO_2, fraction of inspired oxygen; Ppl, plateau pressure; DP, driving pressure; ECMO, extracorporeal membrane oxygenation

high risk of severe hemorrhagic complications, advancements in ECMO circuit technology renewed the interest in VV-ECMO [17]. The 2009 H1N1 influenza epidemic demonstrated benefits in initiating ECMO early in the course of ARDS refractory to conventional invasive mechanical ventilation management. That experience led to ECMO sites, teams, and competency blossoming around the world. Increased familiarity spurred additional research to better define optimal patient selection, indications, and timing.

The CESAR trial in the UK randomized 180 patients with severe ARDS to a strategy of transfer to a single ECMO referral center versus conventional therapy at designated medical centers [18]. Although the primary endpoint of death and severe disability at six months was significantly lower in the ECMO referral center group vs. conventional group (37% vs. 53%, $P = 0.03$), the notable caveat was that only two-thirds of the patients referred to the ECMO center received extracorporeal support. Additionally, the lack of a lung-protective ventilation protocol and low rates of prone position therapy established methodologic limitations and inhibited interpretation of the results. The study suggested that the mortality benefit may have been incurred as a result of the transfer to a center that utilizes guideline-based conventional strategies for ARDS compared to the use of ECMO as a rescue modality.

Since the CESAR trial, most studies on ECMO have been single center and retrospective. However, in 2018 the ECMO to Rescue Lung Injury in Severe ARDS (EOLIA) study was completed [19]. The study compared early ECMO to a conventional mechanical ventilation strategy in a cohort of 249 patients with severe ARDS on invasive mechanical ventilation for less than seven days. The inclusion criteria were severe hypoxemia ($P_aO_2 < 60$ or 80 mmHg for 3 to 6 h, respectively), severe hypercapnia ($P_aCO_2 > 60$ mmHg or pH <7.25 for >6 h). The study allowed for cross-over of patients in the control group with severe enough hypoxia to require ECMO support ($PaO_2 < 80\%$ for >6 h). The study was terminated for failure to demonstrate a 20% mortality difference at 60 days. There was a non-significant but perhaps clinically relevant reduction in mortality in the ECMO group versus the conventional strategy group (35% vs. 43%, $P = 0.09$).

Although the EOLIA trial failed to demonstrate the intended 20% mortality benefit, the 11% mortality difference in a study with high rates of patient cross-over has merit and warrants further consideration. We believe this trial suggests some clinical benefit from ECMO over standard care for patients with refractory hypoxia. It is important to note that adverse events in the ECMO group were rare, and there was no difference in the incidence of intracranial hemorrhage. In further interpreting the EOLIA study results, Goligher et al. performed a Bayesian probability analysis [20]. The analysis demonstrated a high posterior probability of mortality benefit with VV-ECMO. Given the following caveats and our own institutional experience, we feel that ECMO is an appropriate rescue therapy to offer select patients with refractory ARDS.

ECMO After Injury

The use of ECMO in injured patients has been fraught with controversy since ECMO's inception. Despite the survival of the initial ECMO patient—after injury—concerns regarding anticoagulation and subsequent hemorrhage had impeded the adoption of ECMO into the armamentarium of rescue techniques. Early use of ECMO in trauma patients was described by Michaels et al. in 1999 from a single level 1 trauma center. In that series, survival to discharge was 50%, but 58.6% of patients had bleeding complications. The modest survival and high risk of bleeding complications gave many providers pause when it came to using ECMO in this subset of critically injured patients [21]. However, in a retrospective review of the Extracorporeal Life Support Organization (ELSO) registry from 1989 to 2016, a total of 279 trauma patients were placed on ECMO, with 62% of those patients being in the last 5 years of the study. In this series survival to decannulation was noted in 70% and bleeding complications occurred in only 29%—

definite improvements from earlier experiences [22]. As ECMO familiarity has increased, and technology has flourished, ECMO use for post-injury rescue is increasingly common.

While the ELSO registry is a good starting point for studying global ECMO use, cases from non-ELSO centers are not represented. An additional study in 2019 aimed to describe ECMO use in trauma patients using the National Inpatient Sample from 2002 to 2012. In this study ICD-9 codes for trauma and ECMO were queried and 1347 patients were included for review. During the study period there was a 66-fold increase in ECMO use in injured patients with a total in-hospital mortality of 48%, though there was a decreasing trend during the study period [23]. Again, these findings substantiate more widespread use of ECMO as management strategies and circuit biocompatibility improved.

An additional retrospective review from 2001 to 2009 evaluated outcomes for trauma patients with acute hypoxemic respiratory failure treated with ECMO compared to those managed using conventional invasive ventilatory strategies [24]. Among the 102 patient cohort (26 received ECMO), the adjusted survival was greater in the ECMO group. Furthermore, when a cohort of 17 ECMO and 17 conventional patients (matched for age and lung injury severity) were compared, there was significantly greater survival in the ECMO group [24]. While these studies demonstrate reasonably favorable outcomes in the injured patients over the past decade, they do not specifically delineate indications for ECMO after injury. Trauma patients considered for ECMO often fall into two broad categories: cardiac or respiratory failure occurring as a direct result of injury or secondary sequelae of injury that leads ARDS or hemodynamic failure.

Patients who sustain chest trauma can present with severe, acute cardiopulmonary failure as a direct consequence of injury or can develop significant acute respiratory failure from a central airway injury presenting as a major air leak without or without tension physiology. These patients do not fit well into the inclusion criteria defined in the seminal trials such as CESAR and EOLIA. The injury pattern is hyperacute and the decision to institute ECMO must be made quickly. There is often not ample time to leverage adjunctive measures such as prone positioning, neuromuscular blockade, inhaled nitric oxide, or even standard lung-protective ventilator strategies. In a review out of Regensburg in 2013, they studied 52 patients who were placed on ECMO for severe thoracic injury. The Injury Severity Score-predicted mortality was 59% in these patients, while the overall survival in the study group was an impressive 79% [25]. This again demonstrates reasonable outcomes in the traumatically injured patient with chest wall trauma. While the average time from initial trauma to ECMO initiation was 5.2 days in this study, many patients with severe chest trauma present much more acutely. Several case reports and series have demonstrated successful ECMO rescue in the immediate post-injury setting with good success. Bronchial disruptions, massive hemoptysis, and uncontrollable bronchopleural fistulae all leading to an immediate inability to oxygenate or ventilate have been successfully treated with ECMO support. Additionally, this therapeutic strategy has been deployed with little to no anticoagulation in the adult and pediatric trauma populations [26–30].

A separate subset of injured patients will develop acute respiratory distress syndrome (ARDS) secondary to trauma, but not as a direct result of acute thoracic injury. These patients fit more appropriately into the well-defined inclusion and exclusion criteria of the CESAR and EOLIA trials, although they are not as well studied as other populations such as those with bacterial or viral pneumonia. In 2017 a group from Korea assessed the outcomes of trauma and non-trauma ARDS patients. In their review they describe a mortality of 21.7% and 13%, respectively, further informing the discussion about the feasibility of ECMO use in the injured patient [31].

While patients with chest wall injury and ARDS secondary to injury have enjoyed modest success with ECMO rescue, the use of ECMO for those with concomitant traumatic brain injury (TBI) remains questionable. TBI and intracranial hemorrhage in particular have long been considered contraindications to ECMO due to therapeu-

tic anticoagulation required to maintain circuit patency [18]. Technological advancements have reduced the need for anticoagulation and therefore raised the potential for ECMO rescue in this unique patient population. A 2017 systematic review examined survival, causes of mortality, and hemorrhage-related mortality in injured patients treated with ECMO. In this study, patients with TBI and intracranial hemorrhage demonstrated survival ranging from 60% to 93% with no mortality attributed to anticoagulation exacerbated intracranial hemorrhage. It was also notable that there was a trend towards less heparin use and lower than standard anticoagulation goals in this group [32]. While the data regarding ECMO and anticoagulation in patients with TBI remains sparse, and it is difficult to draw robust conclusions, current data suggests that the benefit of ECMO correction of correct acidosis, hypoxia, and hypercapnia may outweigh the potential for intracranial hemorrhage expansion or progression [33].

ECMO rescue after thermal injury (such as burns) is similar to ECMO use after TBI in that the data is more limited than desired and principally stems from single center retrospective reviews. It is worthwhile to note that active rewarming for survivors of acute hypothermia on the other hand is well established. In a 2016 French, 11 patients with a mean total body surface area (TBSA) burn of 31% were placed on ECMO for refractory ARDS. Of this group, only 28% achieved 90-day survival [34]. Relatedly, in a separate review of 8 patients at a single burn center with a median TBSA burn of 17%, there was only 1 in-hospital mortality [35]. Additionally, in 2018, a study at the US Army burn center at San Antonio Military Medical Center reviewed 14 patients with a mean TBSA burn of 27% and demonstrated a 57% survival to hospital discharge [36]. These data demonstrate that there may be efficacy in treating burn patients suffering from ARDS with ECMO support but also suggest that the underlying degree of burn injury—as well as burn injury related care—may principally drive outcome.

During post-burn recovery, an interesting ECMO application has been described. For patients who have severe contractures of the oral stoma or the head and neck region precluding intubation, per-procedural ECMO can support gas exchange during surgical exposure of the trachea for definitive airway insertion, as well as for completion of related reconstructive procedures [37]. Such creative approaches to non-ventilatory gas exchange will likely expand as extracorporeal technology improves and the risk profile of such interventions decreases.

ECMO Transport

As discussed in previous chapters of this text, the health systems, centers of excellence, and regionalization of trauma systems lead to improved care for the injured patient. Using a hub and spoke model to cohort the most critically injured patients at Level I trauma centers supports establishing appropriately focused multi-disciplinary teams in the ED, OR, ICU, med-surg units, and clinics. Broadly embracing the impact of critical illness and injury is important to improve functional recovery, evaluate for sequelae of critical illness and injury—including chronic critical illness or the post-intensive care syndrome—and reducing care recidivism.

The same practice technique—multiprofessional care teams organized around a specific service line—has been proven effective for those patients with severe acute lung injury in need of pulmonary rescue. In one of the most seminal trials in the management of ARDS, the CESAR trial demonstrated that the implantation of a hub and spoke model in managing severe acute respiratory failure improved survival as well as cost-effectiveness [18]. With the CESAR trial as validation, Bryner et al. described more than two decades of experience in transporting ECMO patients from remote locations to an ECMO center of excellence. Patients were cannulated remotely and transported via ground, rotary wing, or fixed wing aircraft to a single institution for continued management. In comparing the survival of transported patients to that of all ECMO patients in the ELSO registry, the transported patients demonstrated enhanced sur-

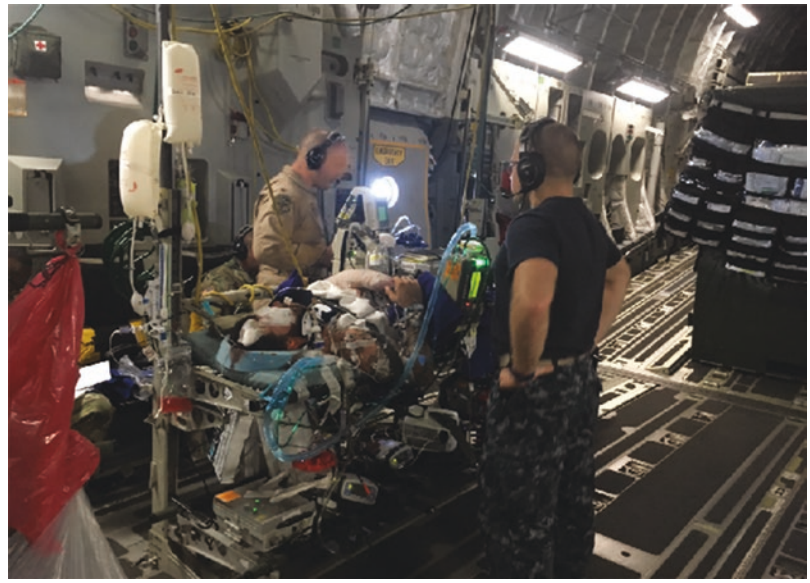

Fig. 24.3 ECMO transport from theater in the Middle East to San Antonio, TX aboard U.S. Air Force C-17 Aircraft with DOD ECMO and Critical Care Air Transport Team in coordination

vival (62%) at hospital discharge, at 62% [38]. A similar review in 2019 by Tipograf et al. described the transport of 265 patients over a ten-year period and demonstrated a 64% survival rate at hospital discharge [39].

These data collectively document that the management of patients with severe acute lung injury using ECMO is both safe and effective during transport especially when definitive care is rendered at a regional center of excellence. This closely parallels the US trauma experience with regionalization of care and designation of level I trauma centers for the management of the most severely injured patients. Accordingly, some have recommended regionalizing ECMO capability to ensure adequate case volume and collocated multi-professional expertise to support patient-relevant outcomes for this resource-intensive rescue modality [40]. Therefore, locating a level 1 trauma center and a regional ECMO center of excellence within the same facility is both intuitively attractive and allows high intensity resources and skilled personnel to interdigitate across service lines.

The Department of Defense (DOD) has embraced regionalization to direct the transport of critically injured combat casualties who require advanced lung rescue intervention, including ECMO support to specific centers for high intensity care. Built upon a historic long-range transport capability for neonatal and pediatric patients [41], the DOD has now successfully transported adults—including both beneficiaries and active duty personnel—around the globe using personnel and resources housed in the San Antonio, TX operations hub (Fig. 24.3) [42]. Outcomes for patients cannulated remotely and transported on ECMO were excellent, with 76% of patients surviving to decannulation and 73.3% surviving to acute care facility discharge [43].

ECMO Management

Anticoagulation

The management of patients on extracorporeal support is a constant balance between hemostasis and thrombosis. Blood interfaces with the ECMO circuit non-biologic surfaces and triggers an inflammatory cascade that activates elements of the intrinsic and extrinsic coagulation pathways, endothelial cells, platelet surfaces, and complement that is pro-thrombogenic. These influences are opposed by therapeutic anticoagulation whose management strives to avoid both thromboembolism and hemorrhage. The optimal anticoagulant approach, therapeutic goal, as well as

titration and monitoring strategy is unknown, but several approaches demonstrate success. The ELSO anticoagulation guideline recommends systemic anticoagulation; however, it leaves it up to individual centers to determine agent selection, titration intensity, titration frequency, and monitoring strategies [44].

The most common anticoagulant used in extracorporeal circuits is unfractionated heparin (UNFH). UNFH binds to antithrombin and increases the enzyme's inhibition of Factor Xa and thrombin by 1000-fold, preventing the conversion of fibrinogen to fibrin and the formation of a cross-linked fibrin clot. The typical bolus dose upon cannulation is 50 to 100 units/kg body weight. A bolus administered during vessel access and before the cannula insertion is given to the patient but does not circulate within the circuit. Before starting anticoagulation, coagulopathy should be corrected to the extent that it is clinically possible so that there is a common point of embarkation upon anticoagulation—and to ensure that injury site-related bleeding is arrestable without additional intervention. The author's practice guidelines recommend a goal of INR $\leq$1.5, platelet count $\geq$50 K, and fibrinogen $\geq$150 mg/dL as sufficient correction prior to initiating therapeutic anticoagulation for ECMO. Bolus dose and infusion rate may be decreased if there is a high risk of bleeding at the team's discretion. Eliminating the bolus dose, however, significantly increases the risk of circuit or cannula thrombosis. UFNH maintenance infusion should start within 2–4 h at a dose of 6–20 units/kg bw/h and is administered to the circuit in a pre-membrane fashion.

Multiple assays may be used to monitor the therapeutic effect of UFNH. Practices vary widely between centers and include assays such as activated clotting time (ACT), activated partial thromboplastin time (aPTT), anti-Xa levels, and viscoelastic testing as single monitoring assays or in combination. A review of eighteen studies by Sklar et al. was not able to determine an ideal anticoagulation monitoring strategy. The authors cited the vast heterogeneity of practices and small sample sizes among the reviewed studies and could not make recommendations [45]. Therefore, there are no uniformly embraced and standardized protocols that guide optimal anticoagulation monitoring. The authors practice combines anti-Xa activity levels and aPTT as complementary guides to inform UNFH titration. Anti-factor Xa activity level (anti-Xa) is a measure of UNFH effect and not its concentration as it solely measures UNFH's ability to catalyze antithrombin inhibition of factor Xa. In many institutions, anti-Xa serves as the gold standard for therapeutic low molecular weight heparin (LMWH) and UNHF management. The anti-Xa assay is not affected by coagulopathy, dilution, or thrombocytopenia. It is important to note, however, that some assays can result in a falsely low level due to hyperbilirubinemia, hyperlipidemia, as well as hemolysis.

Additionally, since other factors besides fibrin formation contribute to hemostasis, anti-Xa levels may be misleading. For this reason, we monitor aPTT as a complementary assay. Anti-Xa and aPTT levels are checked every 6 h and infusion is titrated to achieve a standard anti-Xa range of 0.2–0.4 for VV-ECMO thrombosis mitigation. Patients with bleeding or high risk of bleeding benefit from a lower range of 0.1–0.3. Those with known thromboembolism, flow limiting atherosclerotic disease, prior circuit thrombosis, or requiring VA-ECMO appear to have reduced clotting when titrated to a higher therapeutic range (0.4–0.7). The upper limit of aPTT at the author's institution is 85 s, regardless of anti-Xa levels; if aPTT level exceeds this range, the dose is reduced or held. The reader should note that the upper limit of the aPTT therapeutic range of UNFH infusion will depend on the specific aPTT assay employed in the clinical laboratory. Ranges may therefore vary between institutions within a hospital system if the clinical laboratory assays are not harmonized.

Patients with evidence of bleeding while systemically anticoagulated on any ECMO modality require urgent evaluation for surgical or procedural intervention. When significant bleeding is noted and a surgical intervention is not required, the author's practice is to stop systemic anticoagulation and start aminocaproic acid (AMICAR)

infusion (10 g bolus followed by 1 g per hour infusion). The infusion is continued for at least 12–24 h after the bleeding has resolved, and continued until anticoagulation has been restarted and reached desired therapeutic levels. An acceptable alternative is tranexamic acid (TXA), given as a loading dose followed by continuous infusion. No trials compare the effectiveness of antifibrinolytic therapies in a direct fashion. Both institutional experience and retrospective studies demonstrate that AMICAR and TXA may successfully reduce the incidence of surgical bleeding in ECMO patients [46, 47]. Holding UNFH infusion while antifibrinolytic therapy is provided for more than 24 h is discouraged but preferable to continuous component transfusion therapy. The state of circuit clotting and the patient's level of dependency on ECMO need to play a factor in the decision to stop anticoagulation. Holding anticoagulation may not be possible in patients that already have evidence of clotting or hemolysis in the circuit and are entirely dependent on ECMO for oxygenation as circuit failure would be catastrophic. Some such patients may benefit from revisiting goals of care with the family or surrogate. Discussions of this nature are complex and may benefit from Palliative Care Medicine consultation as well.

Invasive surgical procedures can be performed while the patient remains on anticoagulation with the addition of antifibrinolytic therapy. Typically, AMICAR is started 6 h prior to any surgical procedure on ECMO and continued for 12–24 h postoperatively as above. Alternatively, holding anticoagulation prior to a procedure decreases the risk of bleeding and may be appropriate depending on the nature of the required intervention. The decision to hold anticoagulation should be made through a discussion between the ECMO physician, the intensivist (if there is a separate intensivist who is not managing the ECMO circuit), and the operating surgeon. Factors such as the nature of the surgical procedure, issues with circuit clotting, and degree of dependence on ECMO should be factored into case-by-case decisions. Leveraging ECMO rescue as team-based care is an essential factor in optimizing outcome.

Ventilator Management

There is no single best strategy by which to pursue mechanical ventilation while patients receive ECMO support. Regardless of approach, all patients should be ventilated using lung-protective ventilation (see Mechanical Ventilation chapter). A common approach is to use low tidal volume ventilation as was undertaken in the ARDSNet study. That approach demonstrated a clear mortality benefit in ARDS compared to high tidal volume ventilation [48, 49]. ECMO replaces native pulmonary function, allows for lung recovery, and may reduce the incidence of VILI, in particular by reducing intra-tidal shear and alveolar inflammation. Relatedly, a systematic review by Marghon et al. demonstrated a lower mortality in ECMO patients treated with lower intensity mechanical ventilator strategies [50].

The LIFEGARDS study is an international multi-center prospective cohort of 350 patients on ECMO in 23 high volume ECMO ICUs [51]. Most centers utilized a pressure control mode (82% of patients by ECMO day 14), a driving pressure <15 cm H_2O, and an average resultant tidal volume of 3.7 ± 2 mL/kg of IBW. The study did not show any association between specific ventilator settings and outcomes; however, almost universally, the centers in the study used "ultra-protective" ventilator settings that targeted avoiding alveolar collapse. The benefit of an "ultra-protective" strategy is echoed in the EOLIA study in which a sub-group analysis showed an incurred mortality benefit in ECMO (24%) vs non-ECMO (55%) patients in that the ECMO patients were able to achieve reduced driving pressures and plateau pressures. Therefore ECMO appears to facilitate a lung-protective as well as an "ultra" lung-protective ventilation strategy, while pulmonary function is supported on the extracorporeal circuit.

The authors practice "ultra-lung protective" ventilation for ECMO patients. Pressure control ventilation is the approach of choice with settings that target a driving pressure < 14 cm H_2O, using a PEEP of 10 cm H_2O, and a respiratory rate between 10 and 14 breaths per minute. The tidal

volume goal is <4 mL/kg of ideal body weight. Ultimately, the ventilation strategy should be individualized and reflect the underlining insult causing ARDS and resulting in ECMO support. Approaches may differ in patients with bronchospasm, clinically severe obesity, or a structural injury that establishes a bronchopleural fistula.

Given that some patients require prolonged ECMO support, the authors consider bedside tracheostomy if ECMO support is anticipated to be longer than 5 days. Early tracheostomy supports reductions in sedation, enhanced mobilization, and patient participation in physical rehabilitation. This approach is supported by the author's empirical experience coupled with a single center retrospective review of 50 patients that demonstrated similar benefits from early tracheostomy for ECMO patients [52]. The study compared 21 patients who underwent early tracheostomy (median time of 4 days of ECMO) and 29 patients who underwent "late" tracheostomy (median time 21 days of ECMO). Duration of ECMO in the early tracheostomy group was significantly shorter (12 vs. 21 days, $p = 0.005$). Time to discharge and days to liberation from mechanical ventilation trended towards significance in the early tracheostomy group.

Operative Intervention

Since repeated operative intervention is common after severe injury—especially for patients with multicavity injury as well as those with bony injuries—avoiding thrombosis while on ECMO and supporting hemostasis around operation are competing interests. There have been several reports of operations being performed on ECMO patients before, during, or after ECMO initiation. In the review published by Ried et al. they describe 45 of 52 patients requiring some form of surgical intervention. Sixteen of these interventions were performed during ECMO. The procedures performed covered the spectrum of those typical for injured patients and included orthopedic procedures, spine surgery, abdominal surgery (packing, splenectomy), and neurosurgical procedures. Additionally, 8 patients underwent non-cardiac thoracic surgical procedures. In all patients who underwent an operation, there were no life-threatening bleeding complications noted and the median amount of transfused packed red blood cells was three. While there were 12 patients with abdominal bleeding after surgery, all were successfully managed [25]. Some centers advocate for leaving body cavities (i.e. the chest and/or abdomen) open in a damage control fashion if surgery is required on ECMO due to the risk of a post-closure major bleeding complication. This decision should be made on a case-by-case basis and in a team-based fashion.

As discussed above, another common surgical procedure for ECMO patients is tracheostomy. One of the largest published series to date examined the results of 127 patients undergoing tracheostomy while on ECMO at a single institution. They noted the median packed red blood cell transfusion was 2 within the first 48 h after surgery, and no peri-procedural mortalities [52]. Indeed, the risk of tracheotomy with loss of PEEP that is common with the typical patient undergoing tracheostomy is irrelevant for those on ECMO as oxygenation and CO_2 clearance is managed by the circuit.

Regardless of the reported successes noted above, the benefits of acute operative intervention while anticoagulated on ECMO must be weighted against the risk of operative site bleeding. As there are no evidence-based guides to rendering such decisions, team-based assessment helps to examine the decision-making around operative therapy from multiple approaches. Undertaking essential procedures with careful planning and delaying non-essential procedures seems most prudent.

Weaning Strategies

Once a patient is placed on ECMO support—regardless of specific modality—the next step is deciding on an exit strategy which may also include a time frame. ECMO provides time for the patient's pulmonary and/or cardiac system to recover from the index insult, but at present, is not destination therapy. The decision to wean a

patient from ECMO should ideally occur when the patient's native cardiopulmonary circuit can provide physiologically adequate support for the required hemodynamics for their expectedly hypermetabolic state. It is important to recall that hypercatabolism will augment CO_2 production and that during weaning, the adequacy of ventilation may be more problematic than the adequacy of oxygenation.

Patients placed on VA-ECMO are typically weaned to one of the three destinations: (1) no support, (2) durable device therapy such as an LVAD, or (3) cardiac transplantation. However, the injured population is often different than the characteristic VA-ECMO patient. Most patients placed on VA-ECMO after injury suffer from a presumedly recoverable cardiac and/or pulmonary insult and may be weaned from ECMO to either a low dose inotropic infusion or a less invasive mechanical support device such as an intra-aortic balloon pump. The timing of weaning from VA-ECMO often occurs after 3–8 days of therapy. Common weaning triggers include the presence of a pulsatile arterial waveform, the ability to tolerate ECMO flow that is reduced to 1–1.5 L/min, and when the echocardiography assessed left ventricular ejection fraction exceeds 25% [53].

Injured patients placed on VV-ECMO often fare similarly to those placed on VA-ECMO. Most patients are unencumbered by pre-injury intrinsic lung disease that would drive bridging them to lung transplantation using VV-ECMO. Instead, most injured patients who need VV-ECMO rescue—upon pulmonary recovery—may be transitioned to lung-protective invasive mechanical ventilation or liberation from invasive device support entirely. Weaning strategies for VV-ECMO differ between centers, with no consensus among experts with regard to a single ideal strategy [54]. However, nearly all strategies involve some combination of weaning ECMO blood flow as well as ECMO sweep flow. A reasonable strategy, and one that the authors prefer, is to reduce the ECMO blood flow to 2.5–3 L/min of flow. At this level there is a low risk for circuit hemolysis that is balanced with low risk of circuit thrombosis. As the patient's intrinsic lung function improves they will begin to clear more CO_2 and the ECMO sweep flow can be reduced accordingly. Once the VV-ECMO sweep flow is off, the patient is receiving no ECMO support and a "trial off" ECMO is undertaken. The "trial off" ECMO can last as little as a half-hour or as long as a day depending on the degree of injury and the extent of recovery. If the patient can maintain physiologically acceptable CO_2 clearance and oxygenation in lung-protective ventilator settings, ECMO support can be removed [54].

Summary

Acute respiratory failure in injured patients can be multifaceted resulting from direct or indirect etiologies, both of which are characterized by inflammation. Despite hewing to lung-protective ventilation strategies and optimal injury management, acute respiratory failure may be inadequately manageable using invasive ventilation strategies. Some injured patients suffer concomitant cardiac failure as well. ECMO provides an increasingly deployable rescue therapy for patients in refractory severe acute respiratory failure. Balancing ECMO management and coexisting injury management appears to have optimal outcomes when such high intensity care is rendered at centers of excellence using a team-based approach.

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25 Approaches to Analgesia After Chest Injury

Bryce R. H. Robinson

Introduction

Despite the wide variety of injury patterns and patient characteristics, common to all patients is the emergent need for early and effective pain control following chest wall injury [1]. Pain control begins immediately post-injury and continues through the inpatient stay with anticipated needs that follow hospital discharge. An ideal patient regimen should deliver a measurable benefit to the patient without driving to oversedation or inadequate pulmonary hygiene.

The negative consequences of poor pain control are well established. Inadequate pain management can increase the metabolic needs while concomitantly reduce effective pulmonary hygiene leading to a patient with tachypnea, atelectasis, and resultant hypoxemia that necessitates non-invasive or invasive mechanical ventilation rescue. Poorly managed pain reduces the patient's quality of life and can exacerbate delays in returning to work, impede engaging in activities of daily living, and increase the risks of both depression and post-traumatic stress disorders [2].

The landscape of acute and chronic pain control is dramatically changing due to the evolving opiate crisis that is well-chronicled in the USA. There is a clear link to the use prescription opiates and the evolution to abuse of illicit opiates. As such, modern analgesia techniques focus on multimodal analgesia (MMA) with an emphasis on the de-escalation of opiate exposures and the addition of non-opiate alternatives across the spectrum of pain care. Furthermore, the early articulation of pain control expectations is a foundation of current management. Clinicians need to explicitly re-enforce to their patients that they will likely not eliminate all pain, but rather work with the patient to mitigate it and achieve a pain level that is acceptable to the patient and that embraces a safely deliverable MMA prescription.

Pain Physiology

It is important to understand basic pain physiology as many interventions aim to modulate the perception of pain from the local activation of nociceptors through pathway endpoints in the cerebral cortex. Despite identifying a well-preserved and common physiologic neuronal pathway, the individual patient's response to pain can vary greatly and is modulated by the context of pain, prior pain experiences, and the patient's unique psychology and emotional state.

The perception of thoracic pain begins with the activation of nociceptors in the skin, soft tissue, and muscle of the chest wall. These special-

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ized receptors are activated by pressure, heat, or chemical stimulation at the site of tissue injury. Afferent nerve fibers carry the signal from the nociceptors to the spinal cord and synapse on second-order pain neurons which ascend into the thalamus. Within the thalamus, third order neurons are activated and terminate in the primary somatosensory cortex of the brain providing the sensation of pain (Fig. 25.1). It is the pathway from the afferent nerve fibers to the spinal cord that is responsible for our reflexive nature to withdraw from pain by the activation of discrete muscle bodies in the affected area causing movement away from the appropriately localized and painful stimulus.

Pain signal activation and pain perception have a complicated interplay that differ from patient to patient. Pain can be exaggerated or diminished given the context of the signal. The circumstances of the pain event can modulate how pain is perceived due to the overlap of brain regions responsible for pain perception with those involved in emotion and attention. Pain can be exaggerated in patients with chronic pain issues, depression, or anxiety while it can be diminished in those with extreme adrenergic responses during the injury. In others with chronic pain, what might be assumed to result in significant acute pain may be masked by existing pain. Therefore, pain management benefits from an individualized approach despite having common pathways and cellular responses.

At the cellular level, continued pain can upregulate spinal cord receptors and alter brain chemistry resulting in the amplification of pain signaling leading to chronic pain. Chronic pain, commonly defined as pain that persists for greater than three months is a debilitating condition and is a leading cause of disability and disease burden worldwide [3]. The greater the pain event and number of pain locations, the more likely chronic pain is to occur. Unfortunately, the transition from acute to chronic pain is not an uncommon event after injury. Nonetheless, effective, acute treatment of pain has been demonstrated to be protective against the development of chronic pain rather than predisposing patients to long-term substance use disorders [4].

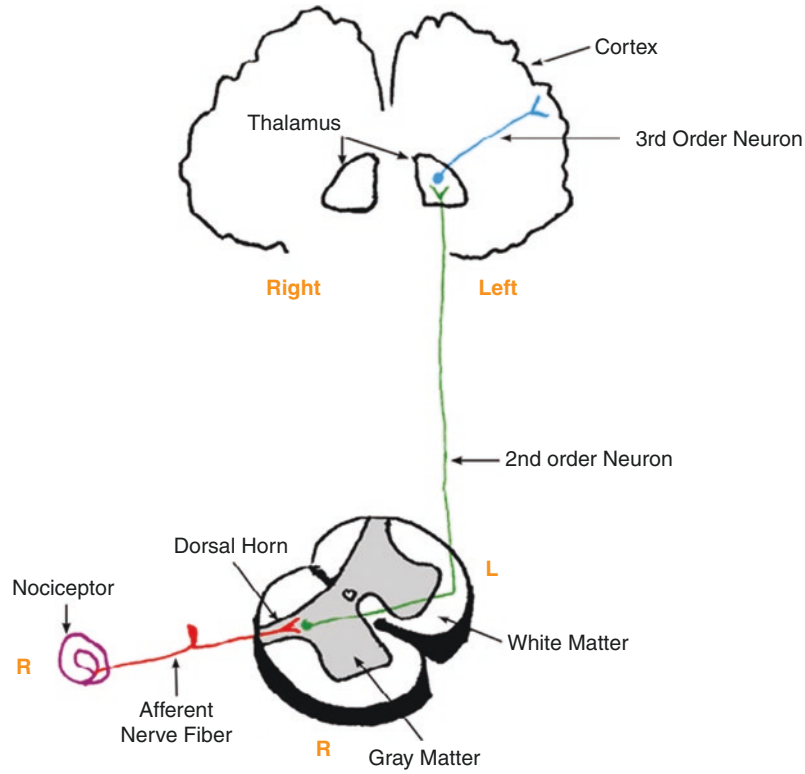

Fig. 25.1 Typical nociception pathway. This diagram depicts the typical neural connections that enable nocioception. (By Bettina Guebeli [CC BY-SA 4.0 (https://creativecommons.org/licenses/by-sa/4.0)], from Wikimedia Common)

Pain Assessment

Effective pain control begins with the ability of the treating clinician to perform a validated, reproducible pain assessment. Assessments need to be repeatedly performed to determine the effects of interventions and to understand the temporal nature of a patient's typically multifactorial discomfort. For patients that are able to be assessed in different settings, assessments should occur during rest and during physical effort including pulmonary hygiene (e.g. incentive spirometry, respiratory physiotherapy, deep coughing). The "gold standard" of assessment is the patient's self-report of pain. Though subjective in nature, validated unidimensional assessment tools are easily deployed and demonstrate high intra- and inter-rater reliability. Patients with the cognitive ability to participate should be serially and systematically assessed using the same tool over their acute care course. Common assessment tools for cognitively intact adults include the Numeric Rating Scale (NRS), the Visual Analogue Scale (VAS), and the Defense and Veterans Pain Rating Scale (DVPRS). While similar to one another, they are not precisely identical.

The NRS is an 11-point scale rating pain from 0 to 10 by the patient. Zero represents no pain, while 10 is the worst imaginable pain. Because of its ease and familiarity, the NRS is widely applied in medical and non-medical environments. The VAS parallels the 0–10 process of the NRS but instead uses a 10 cm line on a piece of paper. The patient marks his or her pain on a line where 0 as a representation of no pain is positioned on the left, while 10, representing the worst pain possible, is positioned on the right. Some VAS tools have the numeric markers at the bottom of the line, while others require physical measurement of the patients mark on the line. In the context of acute and critical illness and injury, the VAS presented in a horizontal fashion was found to be the most valid and feasible instrument in over 100 patients [5]. The DVPRS is also a 0–10 scale (ranging from no pain to worst pain) and requires patient engagement for completion. This assessment tool builds on the VAS in that it includes language describing the pain level under the number, color codes the various levels, and utilizes a changing face graphic to further help define each pain level [6].

Besides assessing their current pain score, timing of onset, prior experience of similar pain, and exacerbating or mitigation factors, it is also useful to understand what condition represents the patients score of "10." Eliciting such information is quite relevant as the patients "10" is being used as the benchmark against which current pain is being compared. This understanding may provide some insight into how pain is perceived and spur a conversation about prior experiences and expectations. Furthermore, in patients with chronic pain, it is ideal to understand how their baseline pain is rated. This knowledge helps inform what pain score represents an acceptable and manageable level of pain for that specific patient. Pain management is enabled by a patient who can participate in their own care—an interaction that is often derailed after acute injury.

Patients who cannot self-report pain are commonly encountered by trauma team members. This may be due to the nature of their injury, the need for critical care management (e.g. invasive mechanical ventilation with sedation and analgesia), or patient comorbidities such as prior stroke. Intoxicants, psychiatric diagnoses, as well as cognitive compromise—including the autism spectrum of disorders—may impede self-reporting fidelity or self-reporting in its entirety. Unfortunately, no validated objective pain monitors or devices exist for patients that cannot participate in their pain assessment. Validated, behavioral pain assessment tools exist and serve as alternatives for patients that cannot self-report [7, 8].

Like patients that can self-report, routine, serial monitoring with a single pain assessment tool should occur. Vital signs should not be used as the sole metric to assess for pain but should instead trigger the application of a validated pain assessment [9]. The most valid and reliable tools in critical care environments for patients that cannot self-report are the Behavioral Pain Scale (BPS) and the Critical-Care Pain Observation Tool (CPOT). The BPS score is the sum of three

items, ranging from 1 to 4 (3–12 total score), to include facial expressions, upper limb movements, and compliance with mechanical ventilation [10]. A BPS score of >5 indicates the presence of pain. The CPOT has four indicators to include facial expressions, body movements, muscle tension, and compliance with the ventilator or vocalization, if extubated. Each indicator is scored 0–2 (0–8 total score) [11]. A CPOT score > 2 represents a patient with pain. Both the BPS and the CPOT should then link with pain focused interventions, many of which start in a tiered fashion with non-pharmacologic therapies.

Non-Pharmacologic Therapies

The penetrance of non-pharmacologic therapies in the ICU is increasing as multimodal analgesia (MMA) therapies are recommended by a variety of medical professional organizations [12]. MMA refers to the simultaneous use of both non-pharmacologic and pharmacologic interventions to synergistically optimize pain control with an emphasis on decreasing opiate exposures. By reducing single class medication pain management, clinicians may reduce the dependence on that class, mitigate dosing escalation, and untoward side effects or medication interactions that are dose-dependent. Common MMA components include acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), opiates, gabapentinoids, regional anesthetics, and non-pharmacologic therapies. When multiple agents and interventions are administered, it is important that the patient remains engaged in their analgesia management and that goals of such interventions are understood by all members of the care team [13]. While the goals for medications are readily embraced, those for non-pharmacologic therapies may be more nebulous.

Non-pharmacologic therapies are commonly divided into cognitive and physical strategies. Although the evidence for non-pharmacologic therapies is less robust than for medication-based approaches, risks from their application are quite low. Common therapies include cognitive behavioral therapy, animal-assisted intervention, and music therapy. Cognitive behavioral therapy includes techniques that allow the patient to control their perception of a painful stimulus. This includes setting expectations, as well as mental imagery or conversation to distract attention from pain. Cognitive behavioral therapies require instruction and guidance at their outset and may benefit from device-based biofeedback to help sustain intervention efficacy [14]. To wit, cognitive therapy for patients following injury may reduce the long-term risk of post-traumatic stress disorder and support optimal recovery [15, 16].

Animal-assisted interventions are increasingly popular, especially in institutions with large pediatric or geriatric populations. Interactions with appropriately trained animals handled by qualified pet therapists reduce anxiety and pain through distraction as well as tactile—and with appropriate therapy animals—auditory stimulation. Dogs, cats, mink, hedgehogs, birds, miniature horses, and others have served in this role [17]. While the benefits of animal-assisted interventions have been reported, the robustness of the data is impacted by small numbers, single center observations as well as methodological challenges [18]. Nonetheless, therapy animals are generally well received. The use of these interventions is highly dependent on patient participation, the capacity of the institution to facilitate these interventions, and local as well as federal restrictions for animals in clinical environments. It is essential to distinguish therapy animals from emotional support animals. Therapy animals are typically bonded with and live with their handlers as part of their home. Note is made of therapy animals who are sourced from a local zoo and work with their handler as well. Emotional support animals are the patient's pet and need not be admitted to the hospital at all as they are not covered under the Americans with Disabilities Act as are service animals (only dogs and miniature horses may serve as service animals at present) [19].

Music therapies are also increasingly common in trauma care environments. Previously limited to hospital audio channel selected music, modern technology has supported CD and now digital

music selections delivered on the patient's smartphone, tablet, laptop, or dedicated media player. Given the success of music as a distraction, live "medical musicians" have intervened in settings from the OR to the ICU [20]. Specific programs have arisen to meet this need and include workshops that foster Music, Empathy, Teamwork, and Assessment skill training [21]. Medical musicians are unlike curated music in that there is also a human interaction that is required to help guide the kind of music—and its adaptation—that may be helpful to the patient even in the emergency department [22]. Reduced anxiety as well as pain is regularly reported—and may deliver benefits beyond the patient [23]. Staff also report reduced fatigue, and parents of children also report reduced anxiety around or following interventions.

Common physical, non-pharmacologic strategies include acupuncture and temperature modulation therapy. Though widely applied to chronic and neuropathic pain syndromes, acupuncture is being considered for acute pain relief after injury. This therapy requires specifically licensed and credentialled therapists whose training is complemented by blood-borne pathogen education. Furthermore, reports of pneumothorax after chest wall acupuncture have been reported. A non-traditional application of acupuncture has arisen and is known as "Battlefield Acupuncture" or BFA. Pioneered within the military domain, it has made its way to related sites such as the Veteran's Health Administration group of facilities. Unlike traditional acupuncture that emplaces needles along body or limb meridians, BFA is limited to the earlobe [24]. BFA is readily taught, easily deployed, is time limited, and appears effective in reducing pain as well as opioid exposure in a variety of settings.

Topical temperature modulation therapy is often employed for patients with chest wall injuries. Cold therapy—also termed cryotherapy—at the site of injury aims to reduce vascular permeability, edema, and inflammation to reduce pain promotion and perception. Ice bags, ice packs, or chemical cold packs all appear to demonstrate equal efficacy. Reduced pain scores after applying cryotherapy to the chest wall even during the removal of chest tubes have been reported [25, 26]. At the other end of the thermal spectrum, topical heat therapy seeks to increase blood flow to the affected area and reduce muscle spasm. Often, cryotherapy and heat therapy are applied in sequence after acute injury, with cryotherapy dominating during the first 48 h to reduce inflammation. In comparison to cryotherapy, heat-wrap therapy may be more effective in providing temporary pain relief for patients with lower back pain (especially in combination with a NSAID) and those with delayed-onset myalgias [27, 28]. Clinicians prescribing cyro- or heat therapy must be cognizant of the duration of application, especially in patients with altered mental status or impaired sensation in order to avoid local thermal injury.

Internal or tissue-directed temperature modulation therapy provides a more durable solution to analgesia. Best known as cryoablation or cryonerve block, the deliberate but temporary rapid freezing of nerve bundles to establish a neurapraxia can provide prolonged pain relief without other concomitant therapy. This approach has been used to address sternotomy pain as well as rib fracture pain in adults and children [29, 30]. For the latter, cryotherapy may be utilized intraoperatively or in a percutaneous fashion to supplant using oral, IV, local, or regional techniques [31]. A probe and delivery system is commercially available to support this opioid sparing analgesic technique [32].

Pharmacologic Therapies

A tiered approach to pain control interventions is recommended for all with chest injury. Even with concomitant non-pharmacologic therapies, most patients will require pharmacologically supported pain control. Common pharmacologic agents include acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), gabapentinoids, ketamine, and opiates (both short and long-acting). Adjunctive agents such as transdermal lidocaine delivery patches may be helpful, especially bracketing linear incisions. Due to the escalating opioid use crisis in the USA, a tiered

approach to analgesia that promotes non-opioid agents—usually in combination with one another—targets reducing opioid exposure. An intuitively attractive approach to the use of non-opioid agents is to initiate therapy with agents with the best safety profile. Using that approach, many tiers will rest on non-pharmacologic therapies to which acetaminophen and NSAIDs are initially added.

Acetaminophen and NSAIDs

Acetaminophen and NSAIDs are common first-line medications and may serve as the sole agents needed for those with low level pain intensity. Acetaminophen is low cost, widely available, and offers multiple routes of administration. Intravenous acetaminophen appears equi-efficacious to oral formulations, but supports therapeutic administration when patients are unable to ingest or tolerate oral agents [33]. Those with acute liver injury or chronic failure need to be carefully monitored for toxicity and warrant dose adjustment. Acetaminophen should be avoided in those with acute hepatic failure. Of note, many over-the-counter medications contain acetaminophen. Thus, total dosing of all ingested formulations will need to be calculated to avoid exceeding the recommended maximum daily dose during outpatient care.

Nonsteroidal anti-inflammatory drugs can be used alone or in combination with other MMA interventions and are also deliverable via multiple routes of administration. Common risks of NSAID use include gastrointestinal irritation and hemorrhage, as well as decreased platelet aggregation and post-procedure or post-injury hemorrhage. Newer formulations such as meloxicam demonstrate no significant impact on coagulation competence and are supported during epidural catheter use and removal in the American Society for Regional Anesthesia guideline [34]. Historically, great concern has been raised about increased rates of fracture non-union with the use of NSAIDs. However, recent studies contradict these claims enabling modern, orthopedic trauma guidelines to strongly recommend their routine use [13]. The benefits of acetaminophen and NSAIDs commonly outweigh the negative side effects, which are most likely to occur with chronic rather than acute temporary use. Because of low cost, ubiquity, and safety, acetaminophen and NSAIDs are commonly included in MMA therapies in an effort to decrease the need for opioid agents for post-injury analgesia.

Gabapentinoids

Gabapentinoids (e.g. gabapentin and pregabalin) have been associated with improved pain control in those with neuropathic etiologies. Although gabapentinoids have been shown to reduce pain following elective thoracic procedures, outcomes in critically ill patients with rib fractures are less robust [35, 36]. Nonetheless, due to an excellent safety profile and wide dosing range, gabapentinoids appear to best serve in an off-label adjunct role rather than a single agent approach to pain management during acute injury analgesia and chronic pain management. Care should be taken in providing gabapentinoids to those with acute kidney injury, chronic kidney disease, and in the elderly (likely related to decreasing renal clearance) as drowsiness and balance instability are common side effects with dose escalation. Atelectasis, secretion clearance failure, and fall-risk all increase with untoward sedation.

Ketamine

The use of ketamine as an analgesic adjunct is re-emerging both by bolus administration and continuous infusion for acute management—especially for those with concomitant chronic pain [37]. Ketamine's ability to control pain coupled with reduced risks of apnea and hypotension makes it an ideal option to control pain and support patient mobilization in patients without airway control if other therapies are inadequate, and opioids are either to be avoided or have demonstrated untoward side effects (see below) [1]. Ketamine decreases oral morphine use in those with severe chest injury with rib fractures and

leads to lower post-thoracotomy pain scores. Importantly, higher doses may induce hallucination and dissociation [37, 38]. Caution should be employed in those with hypertension as it may transiently raise blood pressure due to sympathetic excitation. In that context, historic concerns have been raised about the potential to increase intracranial pressure (ICP) in those with traumatic brain injury. Recent analyses suggest that the risk is quite minimal, and other data indicates that ketamine decreases ICP [39, 40].

Opioids

Opioids—both oral and IV formulations—continue to be the widely prescribed to manage acute, intense pain after chest injury [1]. Increasingly, opioids occupy the third tier in pain management after resuscitation, but IVP opioids remain standard practice during the initial evaluation for acute pain control (Fig. 25.2). Common short-acting opioids used in the acute, inpatient setting include morphine, fentanyl, hydromorphone, and oxycodone. Intra-operative methadone seems to have some benefits in reducing post-operative opioid needs and perhaps underpins its use outside of the OR [41]. Specific note is made of increasingly common use of methadone for acute pain management in the ICU for those with opioid use disorder, intractable acute pain, or who are already on methadone maintenance [42]. Specific recognition is merited with regard to the near total abrogation of prescription of long-acting opioids such as oxycontin in light of the opioid epidemic. Transdermal preparations are generally of limited value in the acute setting with regard to pain control, but instead offer the potential for medication error and patient harm, leading to their general lack of use during acute hospitalization. With the panoply of opioids available for prescription, titration to garner benefit must be balanced with avoidance of undesirable side effects.

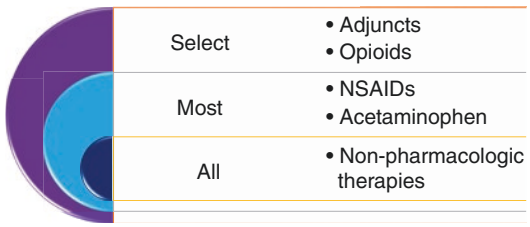

Fig. 25.2 Tiered approach to analgesia. This figure depicts a tiered-approach to analgesia that is appropriate for all patients, most patients, or only select patients. Original figure by LJ Kaplan. *NSAIDs* non-steroidal anti-inflammatory drugs. Adjuncts include lidocaine patches, ketamine by bolus or continuous infusion

Indeed, pain control efficacy varies from patient to patient. Accordingly, establishing order sets that tie escalating opioid doses to a specific range of pain scores is one useful approach. Relatedly, escalating doses of oral agents may be coupled with a rescue agent of an IV agent. Care must be taken to ensure that when prescribing an IV rescue dose that one prescribes an increase in opioid biopotency. This is a common error if one does not assess the pharmacologic equivalency between agents using readily available print or digital resources including tables, charts, or APPs. Of course, one must be vigilant for untoward consequences of rescue doses, especially in the clinically severely obese who may saturate their adipose stores and demonstrate opioid release.

The undesirable effects of opiates are well described and include respiratory and central nervous system depression as well as delayed gastrointestinal function. The sedative effects on the central nervous system can be compounded by concurrent administration of benzodiazepines, gabapentinoids, as well as intra-operative epidurally or intrathecally delivered long-acting opioid such as DuraMorph®. Comorbid conditions, including, but not limited to, clinically severe obesity, obstructive sleep apnea, chronic obstructive pulmonary disease appear to increase the risk of excess sedation and subsequent hypercarbic acute respiratory distress and failure. Titration of agents to achieve the patient's pain goal may ideally occur in a monitored setting such as the Emergency Department, Step-Down Unit, or ICU to facilitate careful dosing and enhance surveillance frequency and efficacy.

An opioid tapering plan should be formulated with the first dose of medication. In opioid naïve

patients the discharge target is pain control without ongoing opioid prescription. This goal is supported using a MMA to analgesia. Careful monitoring of dosing is key, especially in the patient with polytrauma as their hospital course is generally long, and fraught with multiple therapeutic interventions—all of which may drive additional pain. Dose escalation to achieve the same pain score goal is an important indicator of opioid analgesic agent tolerance. Rather than continuing to increase dosing, leveraging adjunctive agents if they have not already been used may be helpful. For some, increasing opioid need may indicate developing depression, PTSD, or other psychiatric concerns and specific inquiry is warranted. Opioid tolerance is a challenging problem to address and consultation with an acute pain service (if available) may be invaluable.

One circumstance deserves specific mention—the increased pain that comes with acute physical therapy. It is important to not confuse exercise induced pain with tolerance and to assess for focal muscle spasm that may be addressed using agents such as methocarbamol or nighttime diazepam. Therefore, context is important in evaluating opioid dose requirements in order to achieve the patient's desired pain goal in a safe and effective fashion that also supports opioid tapering and cessation. For patients who are maintained on a chronic pain regimen that includes opioids, discharge without opioid therapy is generally not achievable, but dose decreases may be feasible. This unique patient population is aided by direct discourse with their chronic pain clinician prior to discharge to ensure care coordination.

Transdermal and Intra-Operative Intravenous Lidocaine

Lidocaine patches are commonly placed on patients with chest wall injury, specifically those with rib fractures. These patches, impregnated with 5% lidocaine, allow for a prolonged topical anesthetic effect in the affected thoracic wall and demonstrate a remarkably safe risk profile. Despite their common application in the acute, critical care, and outpatient settings lidocaine patches have questionable efficacy. In a prospective, randomized trial of trauma patients with rib fractures, lidocaine patches failed to decrease the use of both enteral and parental opioids, nor did it alter pain assessment scores, length of stay, or the rate of pulmonary complications [43]. Accordingly, the clinical experience is that it is unpredictable which patient will derive benefit, leading to the liberal application of lidocaine patches. Alternatively, IV lidocaine infusion in the OR appears to have a beneficial impact on post-operative opioid use, especially in those with opioid use disorder [44]. More broad use outside of the OR, including the PACU, to reduce post-operative pain has been documented [45, 46]. Careful monitoring for lidocaine toxicity is required and is generally readily managed with dose reduction or infusion cessation.

Regional Pain Control Therapies

Because of the energy transference underpinning of chest injuries—and specifically rib fractures—regional techniques present unique opportunities for pain control. Regional, neuraxial therapies are considered a significant component of MMA therapies in those with compromised pulmonary dynamics after chest injury. Systemic, intravenous analgesic agents evidence risk profiles unfavorable to a patient's need for pulmonary hygiene. Specific regional techniques include the placement of epidural or paravertebral catheters with continuous or patient-controlled administration options. Infusate or bolus dosing routinely delivers a local anesthetic, but may be also accompanied by an opioid.

Ultrasound guided fascial plane blocks of the serratus anterior and erector spinae are gaining in popularity. Nonetheless, the use of regional techniques remains controversial. The 2016 Eastern Association for the Surgery of Trauma (EAST) pain management for blunt thoracic trauma guidelines conditionally recommended epidural analgesia and multimodal techniques due to the low quality of evidence available at that time

[12]. However, a more recent systematic review and meta-analysis of 19 studies of analgesia therapy in those with traumatic rib fractures demonstrated that epidural analgesia provided improved pain control compared to intravenous or paravertebral techniques [47]. Epidural placement was not associated with changes in the length of intensive care unit stay, mechanical ventilation use, or the occurrence of pulmonary complications compared to the other techniques. When epidural analgesia was compared directly to paravertebral techniques, no differences in the length of hospital or intensive care unit stays were noted. Like the EAST guideline, the quality of the literature reviewed remained low. Regardless, each of these techniques is a part of the clinicians armamentarium to address acute pain after thoracic injury.

Epidural Anesthesia

The placement of an epidural catheter with a continuous infusion or on-demand patient-controlled analgesia is common in facilities caring for trauma patients and the gold standard for pain control after elective thoracotomy. The catheter is aseptically inserted into the epidural space of the spinal canal and provides a safe approach to pain control over a large body domain including the thorax and abdomen (Fig. 25.3). Catheter insertion is common but also bears insertion, removal, and use risks. Use risks are the most evident in the ICU and include vasodilatation hypotension as well as nerve blockade driven urinary retention. Patchy or single side thoracic pain control can occur if the catheter becomes malpositioned—a common event as the catheter is secured using an adhesive dressing but not suture. Connection issues with tubing are not common but do occur including connection fracture, leakage, and disconnection.

Insertion is more common in the OR but does occur in the ICU for analgesia rescue. Peri-spinal, paradural, as well as intra-spinal hematomas have all been reported as epidural catheter insertion complications. Additionally, prophylactic anticoagulation appears to increase bleeding risks during insertion as well as during removal [48]. The ASRA guidelines detail when such catheters may be placed or removed relative to administration of a dose of a prophylactic anticoagulant be it unfractionated or fractionated heparin [49]. Epidural catheter insertion is contraindicated in those with therapeutic anticoagulation and relatively contraindicated in those

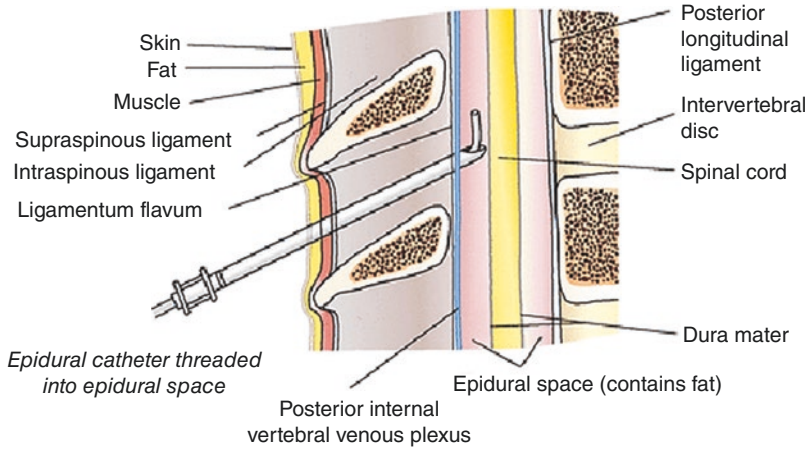

Fig. 25.3 Diagram of epidural catheter placement. This diagram demonstrates the proper placement of an epidural catheter for epidural anesthesia. Note the use of a Tuohy needle for access and catheter direction. (Open access publication download: Duc H. Do. Journal of the American Heart Association. Thoracic Epidural Anesthesia Can Be Effective for the Short-Term Management of Ventricular Tachycardia Storm, Volume: 6, Issue: 11, https://doi.org/10.1161/JAHA.117.007080)

with spinal cord injury, vertebral fracture, or a mental status that precludes participation in the procedure with the care team.

Paravertebral Anesthesia

Single dose injection or catheter placement can achieve ipsilateral pain control if the paravertebral space, a potential space, is utilized for therapeutic agent or continuous infusion delivery. The paravertebral space is lateral to the vertebral column and posterior to the parietal pleural but anterior to the costotransverse ligament (Fig. 25.4). Ultrasound guidance is often used for percutaneous placement, while open access is afforded during thoracotomy. Paravertebral analgesia efficacy is strongly influenced by operator skill as ideal placement appears more challenging than epidural catheter insertion. Depending on the dermatome levels required for pain control, multiple levels may need to be accessed. In a 2016 Cochrane review of 14 studies with a total of 698 participants undergoing thoracotomy, paravertebral blocks appear to have comparable analgesic efficacy compared to thoracic epidural analgesia [50]. Importantly, paravertebral analgesia carries a reduced risk of hypotension, nausea, vomiting, and pruritis compared to the epidural approach [50]. Paravertebral catheter placement is subject to the same guidance with regard to anticoagulant medication administration as with epidural catheter insertion.

Fascial Plane Anesthesia

With the advent of more sophisticated yet economical bedside ultrasound devices, nerve planes are becoming more accessible for those practicing regional anesthesia techniques. Fascial plane blocks are less technically challenging than epidural or paravertebral techniques but require appreciation of operator acquired ultrasound findings to guide safe and efficacious local anesthetic injection. Because injections of these planes occur in compressible spaces, fascial plane blocks can be considered for those receiving prophylactic anticoagulant therapy. A serratus anterior block utilizes the injection of

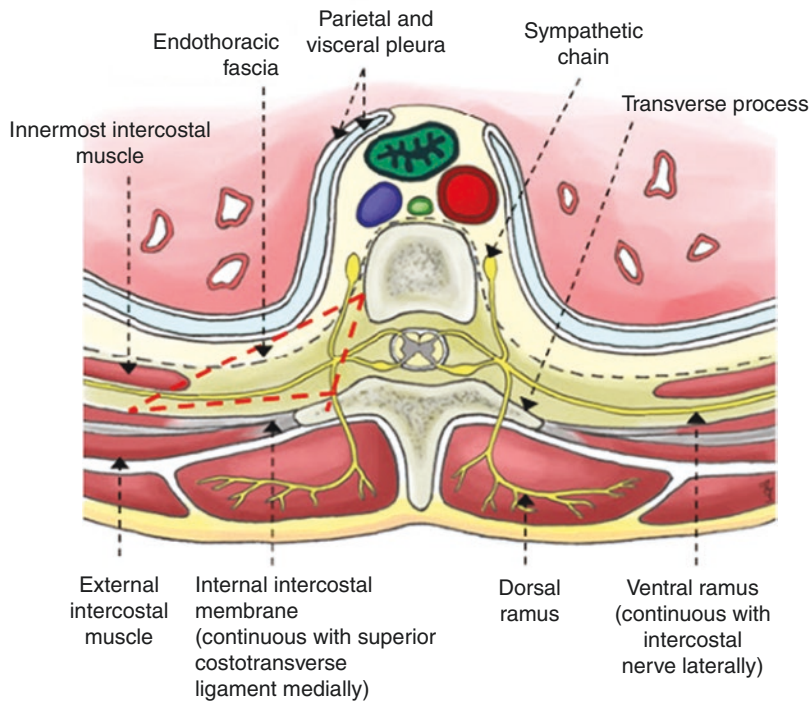

Fig. 25.4 Paravertebral block placement. This cross-sectional diagram indicates the space into which paravertebral local anesthetic is delivered via single access bolus or indwelling catheter. From: Thoracic Paravertebral Block. Thoracic Paravertebral Block (us http://www.usra.ca/regional-anesthesia/specific-blocks/trunk/thoracicparavertebral.phpra.ca). Open access

long-acting local anesthetic—or indwelling catheter placement—either deep or superficial to the serratus anterior muscle at the midaxillary line along ribs 2 to 7. Serratus anterior blocks reduce intravenous opioid dosing needs as well as inadvertent hypotension compared to epidural or paravertebral techniques. Serratus anterior blocks used for post-thoracotomy rescue therapy can be complex due to the need for injection near the operative site, and the unpredictable nature of anesthetic agent spread due to the operative disruption of thoracic wall tissue planes.

The erector spinae block is a recently described technique that utilizes the injection of long-acting local anesthetic—or indwelling catheter placement—deep to the erector spinae muscles but superficial to the transverse processes. Ultrasound guidance is similarly critical to ensure proper placement. Injections in this area spread to the paravertebral and epidural spaces but also involve the posterior and lateral cutaneous intercostal nerves of the chest establishing the potential to cover dermatomes T_2 to T_{10} [51]. The risk profile of this block is advantageous in that its placement is not near major anatomic structures such as pleura, major vasculature, or the spinal cord. Moreover, the erector spinae block is also appropriate for those receiving prophylactic anticoagulant therapy. In a small retrospective study of patients with multiple rib fractures, erector spinae blocks were associated with improvements in incentive spirometry volumes and pain scores without inducing hemodynamic lability [52]. Despite the safety profile and apparent efficacy of fascial blocks, evidence promoting their use over more traditional regional techniques remains to be obtained. The decision regarding the specific approach—or approaches—is often informed by consultation with a multi-professional pain care team.

Multi-Professional Pain Care

Effective pain control following chest injuries remains a complex aspect of trauma care. The intersection of boney and soft tissue injuries with pulmonary function creates a delicate balance between pain control and undesirable side effects of commonly prescribed analgesics that may exacerbate, rather than improve, pulmonary dysfunction. This balance is even more precarious in the growing elderly population who present after injury with multiple comorbidities—including frailty—that influence therapeutic agent selection, dosing, medication interactions, and outcome [53]. The opioid crisis that has plagued the USA over the last 15 years is magnified at trauma centers and underscores the complexity of modern pain care. Approximately 15% of injured patients presenting to a verified trauma center have a history of opioid use prior to injury. Furthermore, 1 in 16 patients becomes a long-term opioid user during the peri-operative period—an event linked to opioid exposure during their acute care episode. Injured patients with an opioid use disorder (OUD) typically require more hospital resources—specifically trackable as ICU and hospital lengths of stay—than the opioid naïve. Because of these complexities, a multi-professional approach to care of those with a chest injury is ideal, supports decision-making for inpatient care, and may extend into the outpatient domain for those with ongoing needs.

Acute pain management services (APMS) are increasingly represented within tertiary and quaternary centers and may be considered to be necessary at high-volume trauma centers. Often lead by anesthesiologists with expertise in pain management, APMS provide consultative aid to address the acute pain needs of unique hospital-based populations. In those with OUD, APMS offer more than therapeutic agent selection of adjustment recommendations. APMS members commonly engage in screening brief interventions referral to treatment (SBIRT) assessment, or referral for psychiatric services with the common goal of understanding the patient's pre-injury needs and behaviors. For complex analgesia need patients at the end of life, APMS often coordinate with palliative care medicine services to help prioritize comfort during the process of dying. Members of APMS are often facile with the regional techniques described above and

practice MMA as a standard approach to care. Formal pain services like APMS may also design and implement educational programs enhancing clinician knowledge and skill sets regarding pain control. They also inform institutional protocols for both inpatient and outpatient prescribing to implement MMA approaches, improve opioid use safety, create triggers for pain management consultation, and chart de-escalation pathways to facilitate opioid cessation.

Patients who are on long-term maintenance therapy such as methadone or buprenorphine present a challenge in avoiding withdrawal symptoms while adequately treating acute pain. APMS are invaluable in helping guide care, but there are some generalities that are useful to recognize. Patients maintained on a daily methadone dose prior to injury need to have their therapy continued, but the dose should be confirmed with the outpatient prescriber. Continuing methadone will prevent acute withdrawal symptoms and avoid destabilizing the psychiatric component of their OUD that is managed by maintenance therapy [54]. Clinicians must ensure that despite providing methadone maintenance therapy, additional agents are also provided to address acute pain but should hew to a MMA approach.

Those using buprenorphine as medication-assisted treatment (MAT) demonstrate more complexity with their post-chest injury pain needs than those on methadone maintenance regimens. Buprenorphine will limit the effect of other opioids provided for acute pain control. Like methadone, standard care at many centers is to continue buprenorphine in light of challenges encountered in restarting medication-assisted treatment if it is temporarily stopped [55]. Note is made of previous recommendations that guided buprenorphine weaning and then cessation prior to elective admissions for surgical care. Similar issues with managing pain and resuming MAT were noted and led to the current recommendation to continue existing MAT. Anticipatedly, these patients will require higher doses of opioids for their acute needs and will therefore need to be closely monitored for undesirable side effects such as respiratory depression.

Atypical analgesia adjuncts may also benefit from consultation with and guidance by a specialty pain management service and team. These agents include antidepressants as well as alpha-2 agonists such as clonidine and dexmedetomidine. The latter two are merit specific comment within the acute injury period [56]. Clonidine and dexmedetomidine act both peripherally and centrally to reduce pain, mitigate anxiety, and induce variable amounts of sedation. Clonidine is routinely suitable for oral use (epidural and topical formulations also exist), while dexmedetomidine is only available for continuous infusion. Clonidine is perhaps best positioned as continuation of preexisting therapy (withdrawal may occur with abrupt cessation), while dexmedetomidine is often used to facilitate safe liberation from invasive mechanical ventilation. Neither are suitable for single agent therapy for pain control and as such are part of a tiered approach to pain management.

Conclusions

Regardless of the presence of an acute pain management service, effective communication between all members of the multi-professional care team as well as the patient is essential for optimal pain control after thoracic injury. This enables a detailed pain history to be obtained, serial and focused pain assessments to be performed, and therapeutic goal expectations to be set. Clinical pathways and guidelines help leverage evidence-based approaches—including multimodal analgesia—to inform order sets and enhance patient care and outcomes. Importantly, within a given center or hospital system, pain management that unifies inpatient and outpatient practice may help reduce overall opioid exposure and embrace non-opioid approaches as well as non-pharmacologic therapies to help modulate pain, its perception, and its impact during acute care through recovery.

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26

The Role of Advanced Practice Providers (APPs) in the Intensive Care Unit (ICU)

Karah Sickler, Marina Trevisani, Philip Efron, and Reka Somodi

Introduction

Over the last two decades, the number of patients requiring medical care far outpaced the number of physicians available to provide that increasingly complex care. This is true in both the outpatient and inpatient settings, including patients requiring critical care medicine services. Each year, approximately six million patients are admitted to North American ICUs, and this number continues to grow [1]. However, the Association of American Medical Colleges (2019) predicts a shortage of 24,800–65,800 physicians in nonprimary care specialties by the year 2032 [2]. Due to the rising acuity of patients' needs, ICU specialization within centralized hospitals, and residency work-hour restrictions, it is estimated that staffing shortages in the acute care setting will be of particular concern [3].

In response to this issue and with greater availability of APPs [4], institutions have increasingly utilized an APP complement to augment care in nonprimary care specialties, including trauma, surgery, and critical care. Studies evaluating the efficacy of utilizing APPs in various care settings document excellent patient outcomes, specifically while assessing care quality, mortality, financial reimbursement, and assistance with trainee education. The use of APPs in intensive care and trauma is comparatively recent, and therefore less intensively delineated. This chapter will focus on defining APPs, exploring APP training requirements, evaluating the utility of APPs in the ICU, and imperatives in supporting ongoing education for critical care APPs.

What Is an APP?

The term advanced practice provider, or APP, is used to describe individuals, typically physician assistants (PAs) and nurse practitioners (NPs), who are collegiately trained to perform tasks—both cognitive and procedural—that were traditionally or historically performed by physicians. The development of the accepted nomenclature for APP has evolved multiple times and continues to vary state by state. The terms physician

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assistant, nurse practitioner, physician extender, mid-level provider, non-physician provider, acute care provider, and most recently advanced practice provider have been utilized to describe these clinicians. The term advanced practice provider is the most appropriately descriptive of the care provided by these individuals. While some domains support independent practice (commonly primary care), others require physician collaboration (commonly subspecialty care such as critical care); requirements vary by state. Each nursing-based APP specialty has its own unique educational requirements, national boarding requirements, and licensure requirements. PAs, on the other hand, currently hew to a single profile. It is appropriate to delineate differences in training approaches for APPs as they collectively function within the ICU in a similar fashion.

Nurse Practitioner Training and Qualifications

Acute care nurse practitioners comprise the majority of NPs that are employed within acute care facilities. Acute care nurse practitioners (ACNPs) are registered nurses who have completed a graduate-level nursing education program, with didactic and practical requirements specifically related to acute care including critical care. Many ACNPs worked in the ICU as bedside registered nurses prior to and during their education program, although such experience is not a consistent requirement for all ACNP educational programs. Their graduate education can be either master's level (MSN) or doctoral level (DNP) education, and all programs must fulfill state requirements to qualify as a higher education institutions. The spectrum of NP training includes the following roles: certified registered nurse anesthetist, certified nurse midwife, clinical nurse specialist, and certified nurse practitioner. NP training also established the following foci: family, adult-gerontology, neonatal, pediatric, women's health/gender related, psychiatric-mental health. NPs who practice in adult ICUs are therefore certified nurse practitioners with focused training in the adult-gerontology population in the acute care setting.

Once the graduate training program is completed, successful completion and maintenance of national board certification is required prior to state licensure. There are three main certifying organizations that allow nurse practitioners to complete national board certification. These three organizations are the American Academy of Nurse Practitioners Certification Program, the American Association of Critical Care Nurses, and the American Nurses Credentialing Center. After passing one of the accredited certification exams, the ACNP can apply for state licensure in the state that they wish to practice. Unfortunately, there is no accepted standard for licensure that is common to all states and the roles of the ACNP vary dependent on practice location and state laws. Increasingly, the need to match training with licensure, accreditation, certification, and education (LACE) as advanced through the ARPN consensus model drives the alignment of skill sets with care settings for advanced practice nurses [5].

Physician Assistant Training and Qualifications

Similar to NPs, physician assistants (PAs) are utilized in outpatient and inpatient settings. PAs complete a Master's Degree Program in Physician Assistant Studies (MSPA). Admission requirements for MSPA vary by institution, with most programs requiring a science or health science baccalaureate degree prior to admission. MSPA curricula are not specific to intensive care or surgical specialties; however, they encompass patients in multiple settings and across the spectrum of age. PA programs often provide 1 year of didactic education and a subsequent year of clinical rotations lasting 4–6 weeks in each location, depending on the school attended. The availability of intensive care rotations during PA school is often included as an elective for master's degree candidates, and most programs are focused on other specialties. One advantage of PA programs is the technical surgical training provided. Once a candidate has graduated from an accredited

degree program, they qualify to take the Physician Assistant National Certifying Examination (PANCE) that is offered through the National Commission on Certification of Physician Assistants. Once the PANCE is complete, the PA is then nationally certified and may apply for state licensure. Unfortunately, there is no reciprocity for licensure in each state, and the roles of the PA may vary dependent on practice location and state law. Unlike NP training and LACE requirements, PA licensure is not driven by specialty or focus and requires a physician collaborator. Independent PA practice is not currently supported in any state.

APP Fellowship Programs

Over the last several years, APP fellowship programs have blossomed across the country [6]. These programs provide fellowship-style education to APPs and often consist of 1 year of intense training in a specific specialty. Specialty programs that exist include both critical care fellowships and surgical fellowships, where APPs can learn the skills necessary to provide care in environments that have traditionally been provided by physicians. The fellowship programs are of particular value for APPs who did not have substantial prior in-hospital experience. The fellowships are structured like a residency program and include clinical rotations through multiple areas, mandatory education, and many hours of bedside care time, while developing a practice profile in a supervised setting. Fellowship programs can be invaluable to those who are looking for jobs with complex patient populations where pre-employment-specific cognitive and procedural skill domains are essential.

Prescribing Laws

There are no national standards that govern therapeutic agent prescription practices for APPs. Some states require specific licensure to be obtained in order to prescribe, and some states include prescribing authority as part of the general licensure. Also, each state has its own laws regarding opioid agent prescribing, which may also differ between nurse practitioner laws and physician assistant laws. NP prescribing laws can be obtained from the respective state's Board of Nursing. PA prescribing laws can be obtained by the state licensure board or the Department of Health. Articulating a standardized approach to prescriptive authority would be of benefit to clinicians, healthcare facilities, as well as patients. Recently there has been national attention focused on APP opioid prescriptive privileges. The number of states that allow APPs to prescribe controlled substances independent of physicians has expanded. In states where APPs are granted privileges to prescribe controlled substances, Drug Enforcement Administration licenses must be obtained. Both nurse practitioners and physician assistant graduate programs encompass multiple hours of training in pharmacology. According to the American Association of Physician Assistants (2019), some states have allowed PAs to prescribe for over 30 years, and there has been no record of increase in malpractice claims or liability in the states where this capability has been added [7].

Quality Improvement

Several studies evaluate the role of APPs in quality improvement initiatives. APPs can and have been utilized to successfully lead quality initiatives in both medical and nursing domains [8, 9]. Expanding APP staffing is tightly associated with decreased rates of urinary tract infections, increased rates of deep vein thrombosis (DVT) prophylaxis utilization, and earlier identification of sepsis [4, 11]. As they serve on hospital or specific nursing quality improvement committees or teams, APPs link nursing and physician efforts in pursuit of high-quality care. As APPs typically do not rotate to other care environments, they are able to efficiently become experts in a given specialty. That expertise renders them ideal clinicians to identify specific areas or practices that would benefit from quality improvement activities. This allows the APP to become expert cham-

pions on unit policies and protocols, develop and deploy standardized order sets, and provide guidance and education for rotating trainees about quality metrics relevant to their patient(s). This is particularly vital when rotating house staff may be unfamiliar with unit processes (activating a stroke alert) or specific points of contacts, which may therefore delay appropriate patient care. Time-sensitive environments benefit from a stable APP presence that improves efficiency. Such environments include, but are not limited to, injury care (golden hour), stroke (time to CT scan and tissue plasminogen activator (TPA)), and acute coronary syndrome (door to balloon time) [10]. High reliability in specific service line performance also supports institutional certification and center of excellence designation.

Procedural Skills

APPs are trained to perform a wide array of invasive procedures, and do so in a safe manner [4]. As each graduate institution often maintains an independent institutional credentialing system, procedures taught in APP programs include, but are not limited to, arterial line insertion, chest tube insertion and removal, bronchoscopy, wound repair and suturing, central line insertion, oral endotracheal intubation, paracentesis, thoracentesis, bone marrow biopsy, lumbar puncture, and point of care ultrasound. One study evaluated the quality and safety of tube thoracostomies performed by APPs versus attending trauma surgeons. The study found that tube thoracostomies performed by APPs were comparable to those performed by trauma surgeons [11]. Similar findings have been noted in comparison to medical residents [12]. Since many APPs regularly perform invasive procedures, they must be held to the same credentialing standards for procedural competence as physicians within their institution. APPs may also be included as part of an institutional procedure team, as APPs have proven to be skilled in performing procedures as well as teaching those procedures to new staff or trainees.

Cost-Effectiveness and Billing

APPs are able to bill for reimbursement as part of the critical care or surgical team, but not all institutions opt to do so [13, 14]. Billable services provided by APPs include bedside patient care, monitoring patients of varying acuity with physician supervision, providing consultations with or without the physician, and conducting advanced planning conversations with patients and families. APPs can also bill for procedural interventions. Each institution has different bylaws as to how APPs are utilized within the institution based on state practice acts. When an APP provides service, the APP can document appropriately and bill for those services, thus adding otherwise uncaptured revenue to the institution.

It is not possible for both the APP and the physician from the same service to bill on the same patient in the same day. APPs are reimbursed at 85% of the Medicare allowable physician rate. Although this is less reimbursement than that of a physician, the utility of APPs to independently bill can add additional revenue to the institution by allowing more patients to be seen in a shorter span of time, which generates higher revenues and may also improve patient flow. In addition, APPs can be utilized to care for patients when physicians are not immediately available [15]. One study evaluated the utility of adding an APP to a rapid response team to allow for care of patients when a physician was not immediately available. The study found that there was a 30% increase in billing, solely because the APP documented and billed for time that would not have been otherwise documented or billed [4]. Education regarding billing and coding should be offered to new APPs—and updated on a yearly basis—as it is not a focus of APP educational programs beyond a basic introduction.

Patient Satisfaction

Patient satisfaction has become a key metric of patient care and permeates efforts to augment the patient and family experience in both inpatient and outpatient sectors. Since ICU physicians are

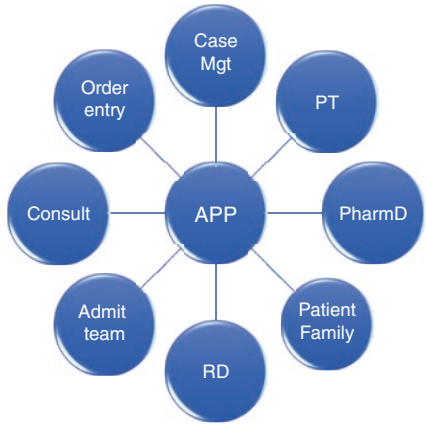

Fig. 26.1 APP communication support roles. *Case Mgt* case management, *PT* physical therapy, *PharmD* doctor of pharmacy, *RD* registered dietitian, *Consult* consultant teams, *APP* advanced practice provider

often responsible for more than one area at a time, and APPs are most commonly geographically fixed ICU assets, APPs can serve as excellent ambassadors for building relationships with patients and families especially during episodes of critical illness [9, 16]. Nonetheless, the value of APPs as relationship builders is not limited to the ICU setting. One study of postoperative cardiac patients was performed that evaluated the difference between NP care and hospitalist care. It was found that patients felt that NPs were superior at patient education, answering questions, addressing pain management, and listening to individual concerns [17]. Accordingly, the APP can also serve as the conduit to share ongoing or difficult to address concerns with the admitting or operating physician as well as consultants. This approach leads to increased patient satisfaction scores and improved team communication [18] (Fig. 26.1).

Impact on Trainee Education

The increasing shortage of physicians may adversely impact trainee education. APPs can enhance resident and fellow learning in teaching institutions and augment attending physician-based education as one mechanism to address physician shortages. Furthermore, the Accreditation Council for Graduate Medical Education (ACGME) places restrictions on the number of hours that resident physicians are allowed to work, which has led to the increased utilization of APPs in teaching institutions in a wide variety of roles. A 2015 study evaluated trainee opinions regarding the outcomes of APPs in providing trainee education [19]. The study found that APPs reduced resident workload, taught unit or service-line-specific guidelines and protocols, enhanced global patient care, and improved communication within care units. As noted above, APPs are able to teach procedures as well as cognitive elements to both trainees and new hires. Nonetheless, there remains some controversy regarding the specific value of APP support of trainee education that may reflect local practice rather than APP capability [20] as different sites identify different value [21].

ICU APP-Focused Education

Creating ongoing educational opportunities for APPs is a necessity to support ongoing professional development as well as satisfaction. Not all APPs have similar educational experiences prior to graduation, and clinical experiences also differ between programs. For example, acute care nurse practitioners often have years of ICU experience before and during their graduate degree programs, while PAs may have only limited ICU experience since ICU rotations are generally classified as electives in many PA programs. Therefore, institution-specific on-boarding programs are one mechanism to establish a point of embarkation for new hires. On-boarding addresses APP competency to provide care, use the electronic health record, and become familiar with unit-specific protocols, guidelines, and pathways including billing as appropriate [22]. Such an approach does not support APP professional development.

Instead, some institutions have established monthly didactic or practical sessions that cover a year-long curriculum. There are two broad-based approaches to ongoing APP education: APP focused education versus education that is for APPs and trainees alike. While there are propo-

nents of each approach, neither has demonstrated superiority. Similarly, didactics may be provided by an attending, APP or a trainee. Teaching institutions may also enlist fellow expertise as well. A key element is that at least one individual who has topic-based expertise is engaged in the session to provide perspective and answer questions, especially if the lecturer is not expert in that topic. Practical sessions ideally support small group didactics and often focus on devices and procedures including ventilator changes, point-of-care-ultrasound (POCUS), and airway management. Given the ubiquitous nature of digital learning, it is of value to record sessions so that learners who cannot attend may review key information as well as the post-didactic discussion.

APP Professional Leadership

ICUs benefit from leadership that includes a medical director, medical co-directors, as well as a nurse manager. In ICUs underpinned by APPs, there is benefit in identifying a Lead APP who becomes part of the leadership team. This person becomes the point of contact for the APP team and ensures that unique elements that influence APP practice are incorporated into new initiatives. This position can be appointed or elected and may be time limited; rotation strategies have been also described. Articulating a Lead APP position requires ICU leadership support as well as institutional support to help provide both time and financial support for nonclinical administrative activities [23].

Conclusion

In conclusion, APPs are clinicians who can help provide high-quality care to a host of patient populations including the most critically ill or injured. Augmented by the increasing physician shortage, and the increased need for critical care clinicians, APPs can capably aid multiprofessional ICU teams to meet patient care needs, ICU workflow efficiency, as well as rotating trainee education needs. Furthermore, APPs may also serve as a bridge between clinical care teams and patients and their family members. There is state-specific governance that impacts APP practice and provides an opportunity for a standardized approach to licensure and credentialing. ICU directors should be engaged in APP education, professional development, and professional advancement. APPs are integral partners in clinical care, compliance, quality improvement, relational dynamics, and leadership in acute inpatient care.

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Managing Conflict in the Surgical Intensive Care Unit

27

Joshua B. Kayser

Introduction

Over the last two decades, patient-centered care has been accepted as a core tenet of intensive care unit (ICU) practice. Central to this practice is the provision of high-quality communication and engagement in shared decision-making (SDM) to align participants in a therapeutic relationship [1]. Major professional critical care societies now strongly recommend incorporation of SDM into patient care [2–4]. As such, it should be considered a core competency for ICU clinicians. Despite efforts at communication, it is unsurprising that conflict will emerge in the ICU setting [5–7]. The surgical intensive care unit (SICU) is a uniquely intense environment. Patients have severe, potentially life-threatening injuries, require complex surgical procedures, and may have uncertain outcomes. Further, patient conditions can abruptly diverge from that which is expected and may come as a surprise to both families and healthcare team members, especially after injury. Since many thoracic injuries result in acute respiratory distress or failure, patient rescue often renders communication difficult in terms of consent, understanding, and goals of care. Of necessity, family members, and often surrogates, become interwoven with the ICU team, the admitting team, and a variety of consultants as care and the patient's clinical condition evolve.

Given the panoply of stakeholders in a given patient's care, aligning all of the participants to ensure effective communication and a therapeutic relationship should be viewed as an integral component of ICU care. However, conflict may emerge between—or among—any of these stakeholders. Communication may be additionally challenged by a host of factors including prognostic uncertainty, severity of injury, surgical complexity, need for surgical buy-in, prior patient and family healthcare experiences, family or healthcare team expectations for recovery, and inconsistency in needs among healthcare team members or between families and healthcare teams [8–13]. Structural elements such as the type of SICU care model (closed vs. open vs. collaborative) may also play a particular role in the prevalence of conflict between healthcare teams [9]. Numerous avenues exist for resolving conflict, each with their own advantages, but all applicable to resolving conflict between health-

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care teams and patients/families as well as among healthcare team members. This chapter reviews the origins and drivers of conflict, and outlines the various avenues for conflict resolution, highlighting conflict management techniques as a teachable skill that can be employed across a variety of ICU scenarios, from acute trauma to end-of-life care.

Conflict Genesis

While conflict can emerge across the spectrum of health care, it is predictably prevalent in the ICU setting owing to a mixture of intersecting factors [6]. The ICU is a stressful environment requiring complex, dynamic, and rapid decision-making to rescue patients from impending death. The complex nature of ICU care necessitates the involvement of numerous stakeholders including the healthcare team, consultants, patient, and family. Inevitably, variability in values, preferences, and expectations will emerge. Moreover, variability exists among patients/families and healthcare teams in terms of preference for communication style and decision-making authority [14]. Many patients may not be able to participate in medical decision-making [15, 16], requiring family members to act as surrogates for care decisions including goals of care. Surrogates may struggle in this role for a number of reasons including lack of knowledge of the patient's wishes as well as cognitive biases [17–21]. Although approximately 20% of patients will die in the ICU [15, 22], fewer than one-in-three patients will have previously discussed broad goals of care or preferences for life-sustaining interventions with their families prior to their ICU admission [23, 24]. Surrogates are subject to cognitive biases including optimism bias around likelihood of survival [17] and may lack strongly held preferences that are expressed as both inconsistency and uncertainty [25, 26]. They may also struggle with affective reasoning or ambivalent preferences for particular outcomes due to emotional strain [27, 28]. The resulting decision-making paralysis may generate conflict between surrogates and healthcare teams.

Challenges related to patient and surrogate decision-making are exacerbated by the unpredictable nature of critical care. Patients are often unstable, and conditions can change rapidly, necessitating dynamic decision-making with little margin for error. Clinical equipoise is not always attainable, and prognostic uncertainty may result in inaccurate predictions for patient recovery [29], undermining trust in the healthcare team. Further, the multiprofessional collaborative nature of critical care results in numerous points of contact for the patient and family. Various healthcare team members and consultants regularly enter rooms and numerous stakeholders may speak with the patient or family, creating opportunities for inconsistency. Such variability produces an appearance of siloed or fragmented care and further undermines trust. Additionally, team members have variable communication capabilities and may disagree about the "best" option for patient care. When interprofessional disagreements emerge, it can further exacerbate conflict. Studies demonstrate variability in physician approaches to end-of-life decision-making is common and suggest that conflict among healthcare teams frequently arise as a consequence [30–32]. The overarching result is communication failure and trust erosion.

Understanding Conflict

Conflict can be defined as a competitive or opposing action of incompatibles [33, 34]. Conflict generally arises when disputants become entrenched in opposing or at least noncongruent positions for a particular outcome. Positions can be described as what an individual or group of individuals want and are typically binary with a presumed "right" and "wrong" option perceived by each disputant. Recognizing the uncertainty of outcomes existing around ICU care and the plurality of values and preferences around care in critical illness, it is reasonable to posit that the concept of right vs. wrong is inherently flawed and should not be used as a standard for medical decision-making. In fact, bioethicists have described the concept of moral aporia, a state of

ethical ambiguity where equally justifiable outcomes may be ethically permissible, even if one or more disputants have a strong moral opposition to that outcome [35, 36]. At its root, therefore, conflict resolution necessitates identifying mechanisms for disputants to abandon their positions in favor of focusing on the underlying interests driving those positions, i.e., why the disputant is anchoring on a particular stated outcome [37, 38]. Focusing on interests allows for consideration of the "why" underpinning the stated position and may provide disputants with an opportunity to consider a variety of outcomes that may achieve the stated interests, rather than addressing a single position.

Conflict may be practically divided into three broad domains—substantive, process, and relational [39]. *Substantive conflict* results when real or perceived questions of competency or motivation emerge. Examples include unethical behavior of healthcare team members, medical misadventures such as errors or adverse events, or questionable motivations of family members (e.g., secondary gain) impacting judgment or decision-making. *Process conflict* results from unclear or inconsistently stated patient preferences, as may occur when a patient requests care that is misaligned with prior advance directives or stated wishes, when conflicting patient and family preferences exist, or when there is decisional disagreement among family members. It may also surface due to local hospital policy or prior legal precedent that informs options for care but may significantly diverge from patient or family wishes. Perhaps the most frequently encountered domain is *relational conflict* resulting from communication failures [39, 40]. While it is the most common among the conflict domains, relational conflict is anticipatedly the most correctable through dedicated communication training and skill-building.

Numerous triggers of relational conflict exist and can be further subdivided into three categories—environmental, systemic, and interpersonal. Of note, the ICU environment is challenging for anyone lacking familiarity with complex care spaces. In addition, the ICU can overwhelm the visual and auditory senses of patients and family as well as their emotional balance. It is highly stimulating with loud and frequent alarms, complex machinery, and limited privacy. The environment and nature of critical illness may impair reasoning and impede surrogate decision-making [28]. Systemic triggers include prior patient and family experiences in healthcare including traumatic experiences and systemic disenfranchisement based on personal identifiers such as race, gender identity, and religion.

In recent years, researchers have begun to explore and codify the concept of trauma-informed care where prior healthcare interactions, layered on top of systemic racism and other micro- and macro-aggressions, can profoundly impact perceptions of care resulting in lack of trust, suspicion, and perceived moral harms manifesting as frustration, resentment, or anger [10]. Prior traumatic experiences may be further exacerbated by existing power imbalances and hierarchy between the healthcare team and patient/family. Interpersonal factors including discordant views of prognosis or outcome, variability in values and preferences, and language or cultural barriers may further derail high quality communication [40]. Clinicians are also subject to bias (both implicit and explicit) which can also undermine relationships [41]. When considering the layers of complexity, it is no surprise that effective communication around critical illness and injury is challenging for even the most skilled communicators [42].

Ultimately, if not addressed early, a cycle of conflict emerges, perpetuating discord [43]. As communication breaks down, disputants become further entrenched in their positions engendering moral distress, disengagement, and intractable conflict. The impact on both patient and family is characterized by complicated grief, anxiety, depression, PTSD, and general dissatisfaction with care [6, 44–46]. For healthcare team members, feelings of powerlessness, cynicism, and detachment arise that may render them vulnerable to burnout syndrome (BOS) and negatively influence future patient care [47–49]. For the profession of medicine, it could result in loss of societal trust, magnifying traumatic experiences for

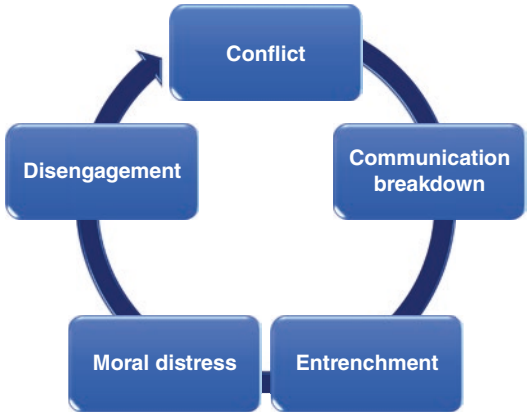

Fig. 27.1 Conflict cycle resulting in intractable conflict. Failure to identify and address disagreement and ensuing conflict results in a cycle perpetuating conflict and further disrupting communication and eroding trust

patients and families, and potentially increase the frequency of pursuing legal recourse. Fig. 27.1 outlines the conflict cycle and ensuing intractable conflict resulting from failure to identify and remediate significant disagreements.

Managing Conflict

Mechanisms to address conflict first emerged in business and law through mediation and arbitration, whereby a neutral third party facilitates a negotiated resolution between disputants [37]. Bioethics mediation in the healthcare setting was first described in the 1990s and has been identified as a core competency by the American Society of Bioethics and Humanities (ASBH) since 1998 [50–53]. The mediator's role is to bring disputants together to reduce emotion, facilitate dialogue, and aid in shared problem solving to ameliorate conflict. In doing so, mediators help disputants compromise and achieve a "win-win" rather than "win-lose" outcome [37]. Consequently, mediators allow disputants to resolve their own conflict and achieve what has been described as a "catharsis," leaving the dispute resolution process empowered and satisfied with the outcome [54–59].

While the availability of bioethics mediation and appropriately trained mediators may be limited in most healthcare settings, mediation techniques can be readily deployed by ICU teams [57, 60–63]. Prevention of conflict begins by having an awareness of the previously described triggers and a recognition of perceived harms that may result in moral emotions (e.g., anger, frustration, indignation, resentment) [38, 57]. De-escalation of conflict requires the clinician to dedicate face-to-face time with the patient or family to address the conflict as it emerges, and before it escalates. Doing so requires time and patience to move past the stated positions (e.g., "what I want") and discover the underlying interests (e.g., "why I want it") [37, 38]. Although this may seem an arduous and time-consuming task, addressing conflict early and transparently is likely to both reduce total time expenditure and achieve a mutually agreeable resolution.

Conflict management—as opposed to conflict mediation—skills are readily teachable and rooted in relational communication techniques that have been well articulated and codified [63–68]. Relational communication necessitates empathy, respect, and a desire to build relationships and trust between the healthcare team and patient/family. It requires clinicians to be inquisitive and curious and to move away from goal- or task-oriented conversations around medical decision-making and instead focus on understanding the individual's values, preferences, and opinions. This includes validating emotions, acknowledging errors or perceptions of harm, and abandoning the moral high ground in favor of seeking to understand the patient or family's reality [69, 70]. Table 27.1 lists communication skills with example language that may be used to facilitate dialogue [63].

Communication tools hold promise in standardizing communication, improving family engagement, aligning goals, and reducing family bereavement [71]. Two have been widely adopted across ICU settings to aid clinicians in effectively communicating with patients and families and are complimentary to the conflict management

Table 27.1 Relational communication skills and example language to facilitate dialogue and de-escalate conflict [63]

Skill	Description/sample language
Active listening	Reframing or restatements to demonstrate you have heard what they are saying and are engaged "It sounds like…" "From what I understand…"
Demonstrating inquisitiveness	Asking questions to probe the individual's concerns and better delineate positions and interests "Tell me more about…" "What are you hopeful for?"
Empathizing	Acknowledging, validating, and respecting the individual's struggle "I can imagine this has been difficult" "I'm sorry things have been so challenging"
Acknowledging different perspectives	Messaging the plurality of moral positions around decision-making "As you see it…" "I imagine from your point of view…" "It's ok for us to disagree about the best option for [you/your loved one]"
Naming emotions	Identifying and respecting the primary emotion driving the conflict "I sense you are [emotion]. Is that correct?" "Many people in your situation would feel [emotion]. Is that how you feel?" "I imagine if I were in your situation, I would feel [emotion]. Are you feeling that way?"
Abandoning the moral high ground	Recognizing that regardless of whether you are right, telling them you are right will further inflame and alienate the disputant (e.g., check your ego at the door)
Righting wrongs (perceived or real) and expressing regret	Further validates the individual's experience "I'm sorry that…" "I wish things were different"
Compromising	Finding common ground based on interests rather than positions in order to achieve a "win-win" outcome that achieves catharsis and healing. This includes demonstrating respect for the outcome at the end "If this outcome is meaningful to you, it is meaningful to me as well"

skills described above. Originally described in the Oncology literature [72], NURSE (Naming, Understanding, Respecting, Supporting, Exploring) is a communication technique for empathetically responding to emotions and is an effective means of mitigating conflict by acknowledging and validating existing emotions to enhance patient-centered care (Fig. 27.2). Similarly, VALUE (Value, Acknowledge, Listen, Understand, Elicit) [71] was developed for the ICU as a communication tool to improve clinician communication and provide a framework for organizing family meetings to elicit and validate emotions and perceptions (Table 27.2). Utilizing both techniques enables clinicians to cultivate their communication skills and more effectively engage patients and families to build trusting relationships and avoid conflict around decision-making—particularly when meaningful recovery is believed unlikely. Additional techniques focused on improving communication and reducing conflict include incorporating family on rounds [73, 74] and enlisting consultative expertise.

Consultative Assistance

When communication and conflict management strategies fail despite the best efforts of the ICU team, multiple avenues provide consultative assistance. Healthcare ethics consultation (HEC, aka Clinical Ethics Consultation) has existed for many years [75, 76] and is ideal for addressing ethical dilemmas and clarifying legal precedent [77, 78]. Some ethics consultants may have prior training in mediation and conflict resolution, but this training is not uniform. The ASBH now

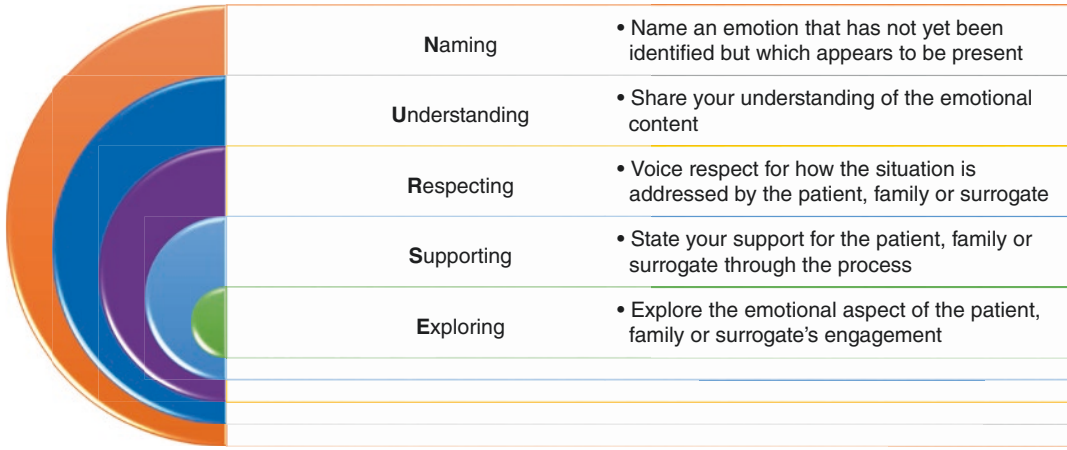

Fig. 27.2 NURSE Mnemonic for Emotions and Empathy. This figure depicts the elements of the NURSE mnemonic that informs clinicians of an integrated approach to participating in emotionally charged conversations while expressing empathy. (Adapted from Ref. 72. Note: this is an original figure and did not appear in Ref. 72. It is an adaptation of the content published in Ref. 72 and thus does not need publisher reproduction permission)

Table 27.2 VALUE mnemonic for organizing relational objectives in a family meeting [71]

	Objective
VALUE	To demonstrate appreciation for the information the patient or family member is sharing
ACKNOWLEDGE	To respect the patient or family member's emotions
LISTEN	To demonstrate active listening as a sign of engagement with the patient or family member
UNDERSTAND	To ask reflective questions so the clinician gains awareness of the patient as a person
ELICIT	To give patients and family members the opportunity to ask questions

identifies basic bioethics training and certification of ethics consultants as a strategic goal to ensure adequate consultative skill [79]. Although effective at clarifying ethical ambiguity, the majority of conflicts in the ICU are not purely ethical and therefore may limit the effectiveness of HEC [80, 81].

Another valuable resource is hospice and palliative medicine (HPM) consultation [82]. HPM specialists are skilled at relational communication and are highly effective in eliciting values and preferences for care [83–85]. Utilizing collaborative techniques, HPM consultants can enhance trust and improve patient/family buy-in. Secondary HPM consultation has thus been endorsed as an essential resource by critical care societies [2, 86–88]. Though lateral to the primary team, HPM consultation can be viewed as additive to patient- and family-centered care and the process of building and sustaining relationships. This approach may be especially valuable when there is a change of life circumstance and an ongoing therapeutic dynamic is anticipated to be of benefit after acute care facility discharge.

Ultimately, when efforts to improve communication and ameliorate conflict fail, patients and families or healthcare teams can request that the patient's care be transferred to another clinician, service, or hospital. While feasible in application, this approach fails to resolve the underlying conflict, re-establish trust, or improve patient care. Instead, service or facility transfer only serves to permanently divide disputants. This kind of avoidant approach is likely to perpetuate traumatic experiences for patients and family as well as healthcare team members and should be reserved for only the most extreme cases. Unfortunately, when transfer occurs, the patient

and family may be labeled as a "difficult family," when in reality, they represent a vulnerable group in need of rescue, understanding, and conflict resolution.

Conclusions

Conflict is inevitable in the ICU. The critical care environment is intense, requires dynamic decision-making, and outcomes may be uncertain. It can be overwhelming for the patient and family as well as healthcare team members. Conflict should be appreciated as a common occurrence that requires prompt attention and dedicated efforts at effective communication and collaboration to mitigate it. When conflict remains unaddressed, it may result in breakdowns in trust and psychological harm for patients/families and healthcare team members. There are numerous factors that may increase or exacerbate conflict including implicit and explicit bias, prior traumatic experiences, power inequity, disparate values and preferences, and prognostic ambiguity. The environment of care and the size of ICU teams can also perpetuate conflict, as well as failures in communication style and substance by clinicians. When conflict results, deploying relational communication and conflict management techniques may help repair trust and reveal opportunities for compromise that achieves the patient or family goals. HEC and HPM consultation are invaluable resources whose focused application supports the ICU team's efforts at patient- and family-centered care when it is threatened by conflict. Ultimately, skill development in communication and conflict management techniques should be viewed as an essential component of critical care training and practice to ensure high-quality care.

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Pulmonary Infections

28

Mayur Narayan, Nicole Meredyth, Victoria Aveson, and Philip S. Barie

Trauma remains the leading cause of death and disability in Americans aged 1–44 years. Chest trauma, as a result of either blunt or penetrating injury, is a major source of morbidity and mortality, accounting for 20–25% of all deaths due to trauma, or approximately 16,000 deaths/year in the United States [1]. Thoracic trauma predisposes patients to infectious complications through a number of mechanisms: disruption of respiratory mechanics, contamination of sterile tissues, and iatrogenesis from life-saving interventions. Understanding the anatomy and physiology of the chest, including the bony skeleton, the lungs and pleurae, the tracheobronchial tree, the esophagus, and the cardiovascular system is crucial. Furthermore, as the lung is a large target organ for secondary damage by post-traumatic inflammation, lung injury can contribute substantially to the development of multiple organ dysfunction syndrome (MODS).

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Respiration Physiology

Normal Control Mechanisms

Respiratory complications are common after multiple trauma—especially when it involves chest injury—and often manifest as sepsis or other attributable complications. Prevention and management of infection following chest trauma require a sound knowledge of normal pulmonary function and its mechanisms of control, as well as disease pathophysiology. At-risk patients for postinjury pulmonary infections should be expeditiously identified and closely monitored.

Infection may develop after chest trauma due to the injury itself or from therapeutic modalities necessary for support of the patient, including operative intervention. Factors that contribute to postoperative acute respiratory distress or failure include inhalational anesthetic agents, which depress mucociliary clearance mechanisms and decrease functional residual capacity (FRC). Postoperative pain and opioid analgesics compound these effects by depressing respiratory drive, predisposing to atelectasis and airway obstruction by retained secretions. Chest and upper abdominal incisions, in particular, predispose to pulmonary complications via this mechanism.

Respiratory responses play a major role in maintaining acid–base homeostasis. Short-term regulation of acid–base balance is largely a

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function of feedback mechanisms that govern the regulation of ventilation. The principal regulators are aortic arch and carotid body chemoreceptors, afferent and efferent impulses from medullary respiratory centers, and mechanoreceptors in the chest wall, diaphragm, and lung parenchyma. Excretion of CO_2 is the principal mechanism by which the lung maintains normal pH, which in turn is affected by red blood cell (RBC) transcapillary transit time, alveolar-capillary transmembrane diffusion of O_2 and CO_2, and matching of the distributions of alveolar ventilation ($\dot{V}A$) and perfusion (Q). Abnormalities of any of these domains may impair CO_2 excretion in those with preexisting pulmonary disease, following injury, or in the increasingly elderly patient population, a combination of both.

Central excitatory and inhibitory neurons receive afferent impulses from chest wall and lung mechanoreceptors [2]. Responding to mechanoreceptors, neural mechanisms regulate cyclic inhalation and exhalation. Neurons of the dorsal and ventral medulla integrate neural and chemical stimulation. Dorsal respiratory neurons regulate neural and chemical signals, and principally affect inspiration, while ventral respiratory neurons regulate both inspiratory and expiratory activities. Many of the ventral neurons project to the thoracic spinal cord to innervate the intercostal and abdominal musculature, whereas the dorsal motor neurons coalesce in the phrenic nerves. Other neurons located in the rostral pons inhibit the firing of the medullary neurons, acting as a physiologic filter to produce smooth, regular ventilatory activity [3].

Tendon mechanoreceptors in the diaphragm and intercostal muscles are principally inhibitory, but their effect is modest compared with pulmonary mechanoreceptors, of which at least three types modify ventilation via vagal afferents [4]. Airway epithelial mechanoreceptors respond to local irritants or mechanical deformation of the tracheobronchial tree, producing bronchoconstriction and hyperventilation. Airway smooth muscle stretch receptors enhance bronchodilation during lung expansion and act with irritant receptors to regulate airway diameter during breathing. Juxtacapillary ("J") receptors in the alveolar interstitial space are stimulated by interstitial edema or fibrosis [2, 5]. Pulmonary and chest wall mechanoreceptors increase expiratory muscle activity during lung inflation to reduce end-expiratory lung volume to normal. Afferent signals from these receptors are processed in the brainstem to adjust both respiratory frequency and tidal volume (V_t) to minimize energy expenditure. Stimulation of almost any afferent somatic nerve, especially pain fibers, results in a reflex increase in minute ventilation ($\dot{V}E$). Increased body temperature is also a potent stimulus of hypothalamic efferents that, when transmitted directly to excitatory neurons, produce an increase in $\dot{V}E$ independent of the increase in CO_2 production caused by fever.

Adjustments of ventilation and perfusion in response to alterations in P_{O_2}, P_{CO_2}, and pH are mediated by two distinct chemoreceptor systems [6]. Central chemoreceptors respond to changes in cerebrospinal fluid proton concentration and are primarily responsible for determining resting $\dot{V}E$, as well as ventilatory and circulatory adjustments during hypercapnia and chronic disturbances of acid–base balance [7]. However, owing to the blood–brain barrier, the brain is affected slowly by changes in plasma $[H^+]$ or $[HCO_3^-]$. Thus, central chemoreceptors respond differently to acidosis resulting from hypercapnia or from organic acids (e.g., lactic acidosis) [8]. Increases in arterial P_{CO_2} ($PaCO_2$) produce linear increases in $\dot{V}E$, which are predominantly driven by central chemoreceptor response to medullary proton concentration. Central chemoreceptors can suppress peripheral chemoreceptor responses by suppressing autonomic efferents. Therefore, there may be no response in mild hypoxia while hypercarbia remains a powerful catecholamine triggering event.

Peripheral chemoreceptors respond to changes in PaO_2 rather than O_2 content (CaO_2). Stimulation of peripheral chemoreceptors is responsible for increased $\dot{V}E$ produced by hypoxia and for some of the increase caused by hypercapnia [8]. Carotid body chemoreceptors are probably more important than those in the aortic body [6]. The

carotid body receives sympathetic and parasympathetic efferent stimulation. Afferent impulses to the brainstem as well as inhibitory efferents are transmitted via the carotid sinus nerve. The effects of increased Pa_{CO_2} and, to a lesser extent, low pH on carotid body activity are greater during hypoxia than hyperoxia, which accounts for the synergistic effects of hypoxia and hypercapnia on ventilatory drive.

Gas Exchange

After alveolar ventilation with fresh gas, O_2 and CO_2 are exchanged by diffusion, which is inversely proportional to membrane thickness and also depends on gas solubility and molecular weight. Higher solubility allows CO_2 to diffuse 20 times faster than O_2. Normal values for gas concentrations are indexed at sea-level with adjustments required based on elevation above or depression below sea level. The alveolar P_{O_2} (P_AO_2) is approximately 105 mm Hg (14.0 kPa), whereas the venous O_2 tension ($P_{\bar{v}O_2}$) is 40 mm Hg (5.3 kPa), creating a diffusion gradient for O_2 across the alveolar-capillary membrane. Pulmonary arteriolar P_{CO_2} is 46 mm Hg (6.1 kPa), equilibrating to an end-capillary P_{CO_2} of approximately 40 mm Hg (5.3 kPa).

The lung has a large reserve before diffusion becomes impaired. Mean pulmonary capillary transit time is approximately 0.5 s. The diffusion equilibrium for O_2 occurs within 0.25 s and CO_2 reaches equilibrium even faster. Diffusion limitation can be demonstrated if transit time is decreased (e.g., exercise, increased perfusion), the diffusion gradient is narrowed (e.g., high altitude); or functional membrane area is lost (e.g., after resection, in fibroproliferative disease, or pneumonia). However, abnormalities of gas exchange caused purely by diffusion abnormalities are rare. Rather, V_A/Q mismatching is the major culprit.

The distribution of V is not uniform, even in normal lung. Pleural pressure (P_{pl}) in the upright position is further below atmospheric pressure at the apex than at the base of the lung. Consequently, transpulmonary pressure (P_t) [alveolar pressure (P_{alv}) − P_{pl}] is greater at the base of the lung and alveoli at the apex are generally more expanded. However, the distribution of inspired gas is not passively determined by anatomy. Regional variations in lung compliance caused by normal airway elasticity or inflammation, bronchial hyperreactivity, mechanical airway obstruction, or pulmonary edema can influence the distribution of V. The particular lung volume from which an inspiration is begun is also important. Within the range of normal V_T, alveoli are ventilated better at the bottom than at the top of the lung. At very low lung volumes (near the residual volume [RV]), P_{pl} at the dependent portion of the lung exceeds airway pressure (P_{aw}), which results in atelectasis. During a breath taken at or near RV (e.g., during acute lung injury (ALI)/acute respiratory distress syndrome (ARDS), which decreases FRC), gas entering the lungs is preferentially distributed to nondependent areas.

Distribution of pulmonary Q is complex by comparison [9]. Blood flow is influenced by gravity, which provides a gradient of increasing blood pressure and perfusion toward the lung bases (or dorsal lung in supine patients) that is greater than the distribution gradient of V. The distribution of Q is also influenced by pulmonary blood volume, right ventricular output, mean pulmonary artery pressure (P_{pa}), pulmonary vascular resistance, and lung volume (physiologic dilation of extra-alveolar capillaries keeps vascular resistance low at higher lung volumes). The application of positive-pressure ventilation, particularly positive end-expiratory pressure (PEEP), accentuates redistribution of Q toward the most gravity-dependent portion of the lung because Q ceases when alveolar pressure (P_A) exceeds perfusion pressure in nondependent lung [10].

Other than gravity, vascular responses mediated by both local and extrapulmonary mechanisms are the most important determinants of pulmonary perfusion distribution. Both alveolar hypoxia and arteriolar hypoxemia cause pulmonary vasoconstriction [11]. Hypoxemia results in

chemoreceptor-mediated vasoconstriction via adrenergic efferent pathways [11, 12]. Acidosis also causes vasoconstriction and enhances the pressor effect of hypoxia (effects that are attenuated by alkalosis). Indeed, metabolic acidosis drives pulmonary hypertension and decreases right ventricle ejection fraction by increasing the afterload against which the ventricle must pump. Atelectasis with hypoxic pulmonary vasoconstriction demonstrates similar untoward effects on right ventricle function.

Efficient gas exchange depends upon V_A/Q matching. Abnormalities may develop of V, Q, or both. Inequality of V_A/Q is the most common cause of hypoxemia in people with lung disease or injury. The major factor in determining SaO_2 is the $V_A{:}Q$ ratio, not the absolute quantity of each. Even if V_A/Q distribution is abnormal, arterial blood gases are little affected if they remain matched. The normal $V_A{:}Q$ in the whole lung is about 0.85, with the low V_A/Q areas in the lung base compensating for the higher ratios in the relatively nonperfused apex. In disease, dead space (V_D) as well as shunt (Q_s/Q_t) may develop. Matching of V_A/Q is maintained better in the supine position because gravity-induced differences of perfusion are attenuated, but ARDS can cause V_A/Q mismatching even in supine lung, which may be ameliorated transiently by placing the patient prone. Also, hypoxic vasoconstriction is the most potent mechanism for normalizing an abnormal $V_A{:}Q$ [11]. Drugs that abolish hypoxic vasoconstriction (e.g., epoprostenol, nitroglycerin, nitroprusside) may worsen P_aO_2 when given to a patient with ARDS. Thus, understanding physiology in health, disease, and injury allows the bedside clinician to appropriately select adjuncts when complications arise.

Lung Mechanics and Pulmonary Dysfunction After Injury

Anesthesia, the anatomic location of an operative procedure, and preexisting conditions impact postoperative ventilatory function by altering respiratory muscle function and disrupting lung mechanics. Understanding these processes requires a knowledge of lung mechanics and how surgery and anesthesia alter these mechanics, often resulting in pulmonary complications. Thoracic and abdominal procedures cause restrictive lung function abnormalities that reduce all lung volumes. Vital capacity (VC) and FRC can be reduced as much as 50–70% during the first 24 hours after operation. These changes occur shortly after anesthesia induction and can last as long as 14 days postoperatively. General anesthesia itself is not causative, as similar abnormalities may be seen in patients receiving epidural anesthesia. Pain and muscle splinting contribute to the reduced lung volumes, but effective pain control does not immediately restore lung volumes.

General anesthesia predisposes to upper airway obstruction, impairs protective airway reflexes, and depresses mucociliary clearance mechanisms. Virtually all anesthetic agents share this property, which is caused by changes in both chest wall conformation and motion regardless of whether there is neuromuscular blockade. The FRC decreases after induction [13] but remains stable for the duration of the anesthetic. Anesthetics also decrease diaphragmatic tone at end-exhalation, further decreasing thoracic volume [14]. Atelectasis begins to develop in dependent portions of lung within 10 min of induction [15]. Breathing at lower lung volumes increases the elastic recoil of the lung and decreases lung compliance [15], but healthy, non-neuromuscularly blocked patients can compensate for this and the partial airway obstruction caused by the endotracheal tube (smaller lumen than the native trachea) and maintain V_E by increasing the work of breathing.

The alveolar-arterial O_2 gradient ($D[A\text{-}a]O_2$) decreases in direct proportion to decreased FRC in anesthetized subjects because of intrapulmonary shunting and perfusion of low V_A/Q areas [16]. The phenomenon is accentuated in elderly patients and by preexisting lung disease. Inhibition of hypoxic vasoconstriction by inhalational agents is the primary mechanism of V_A/Q mismatching under anesthesia. All halogenated anesthetic agents impair normal responses to

hypoxemia and hypercapnia by depressing carotid body chemoreceptors and impair peripheral chemoreceptor-mediated ventilatory response to acidemia [17, 18]. Atelectasis, decreased FRC, and increased $D[A\text{-}a]O_2$ persist in the postoperative period only in patients who have undergone general anesthesia for a thoracic or abdominal procedure [19]. Diaphragm dysfunction, rather than the persistent effects of anesthesia or the restrictive effects of incisional pain, is most important in the pathogenesis of prolonged abnormalities. Diaphragm dysfunction is not abolished by epidural fentanyl, and the response to phrenic nerve stimulation is normal [20, 21].

Postoperative Lung, Chest Wall, and Diaphragm Dysfunction

Cough and mucociliary transport protect lungs and airway from environmental and infectious agents, but endotracheal intubation suppresses airway reflexes, which increases the risk of respiratory tract infection. During the induction of general anesthesia in supine patients, cephalad diaphragm displacement accounts for a loss of 350–750 mL of thoracic volume. Intravenous anesthetics (except ketamine) depress diaphragmatic tonic activity and volatile anesthetics depress synaptic transmission, affecting the intercostal muscles preferentially. The loss of respiratory muscle tone, mediated by inhibitory phrenic nerve efferents, rapidly results in atelectasis. At 1 and 24 h postoperatively, 90% and 50% of the anesthetic-induced atelectasis is still present, respectively. Coughing is inhibited because of pain and opioid analgesic use. Respiratory muscle dysfunction reduces the expulsive force and effectiveness of coughing. Mucociliary clearance is reduced for 2–6 days following general anesthesia because of ciliary damage from dry anesthetic gases, increased mucus viscosity, and reduced clearance from atelectatic areas. Epidural local anesthetics ablate the inhibitory signals from visceral sympathetic receptors, but prolonged recumbency and sedation mean that epidural analgesia does not prevent abrogate atelectasis. By contrast, epidural opioids ameliorate pain and may centrally depress ventilatory drive, phrenic nerve function remains intact.

In thoracic operations, reduced surfactant activity can result from mechanical compression of the lung and accumulation of extravascular lung water. Changes in pulmonary function after a thoracotomy may last as long as 3 weeks [22]. In addition to loss of functional lung tissue, chest wall compliance decreases as much as 75%. These changes are exacerbated by opioid analgesics. After abdominal operations, the patient adapts to a pattern of breathing with little abdominal volume change but increased rib cage excursion. The accessory muscles of ventilation assume increased importance during this phase of recovery. Contraction of abdominal muscles is prominent in exhalation, but its importance in generating V_T is not well characterized. The shift from using the diaphragm to intercostal muscles is accompanied by a re-distribution of V and less inspiratory gas delivery to the lower lobes. Abdominal muscle activity during exhalation decreases the FRC. Accordingly, alveolar closing capacity (CC) may be reached during a tidal breath and result in atelectasis.

The Immune Response and Pathophysiology

The respiratory tract is a portal for invasion by potential pathogens and thus must play a major role in protecting the body against infectious diseases. The vibrissae in the nose, along with the ciliated epithelia of airway mucosa (including the nares and pharynx), trap particles and microorganisms, moving them cephalad to the pharynx to be swallowed, expectorated, or eliminated by cough. This local host defense response is orchestrated primarily by four cell types (i.e., ciliated epithelium, mucus-secreting goblet cells, club cells, and basal cells) to produce a physicochemical barrier to respiratory infection. Club

cells produce antimicrobial compounds and, in conjunction with basal cells, serve as regional progenitor cells to replenish other cell types [23, 24] (Fig. 28.1).

Following respiratory invasion by viral, bacterial, or fungal pathogens, type 1 immune responses are engaged [23] (Fig. 28.2), consisting of T-bet+ interferon (IFN)-g-producing group 1 innate lymphoid cells (ILCs) (ILC1 and natural killer [NK] cells), cluster of differentiation (CD) 8+ cytotoxic T cells (TC1), and CD4+ T helper (TH)1 cells, which protect against intracellular microbes through activation of mononuclear phagocytes. These infectious agents are recognized through pattern recognition receptors in sensor cells including airway epithelium, macrophages, dendritic cells, and plasmacytoid dendritic cells. Respiratory parenchymal macrophages phagocytize particulates and microorganisms and, if overwhelmed, recruit neutrophils from alveolar septal capillaries. Immunoglobulin (Ig) A in mucosal secretions supports humoral immunity. Inhalation of allergens can trigger a type 2 immune response where epithelial cells and mast cells secrete cytokines (Interleukin (IL)-4, -5, -9, and -13) that perpetuate the immune response [23] (Fig. 28.3).

In general, suboptimal (ineffective) humoral immunity or nonimmune defense systems increase the risk of bacterial infection, whereas suboptimal innate immunity increases the risk of infection by intracellular or low-virulence organisms principally represents by viruses and much less robustly by bacteria. Other risk factors for

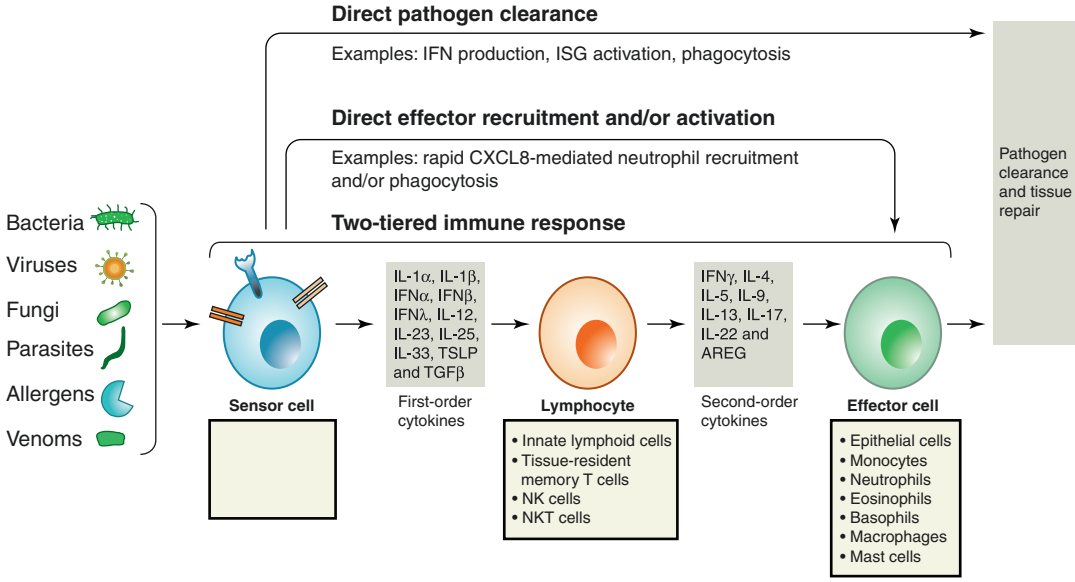

Fig. 28.1 Stepwise engagement of tiered responses to respiratory infection. Pathogens and certain noxious compounds are detected by sensor cells located within the respiratory tract. Sensor cells immediately initiate innate immune responses that may be sufficient to clear localized infections. For example, sensor cells may secrete factors such as interferons (IFNs) that lead to pathogen clearance (direct pathogen clearance, top arrow). In some cases, first-order cytokines directly recruit effector cells that clear pathogens; for example, CXCL8 mediates recruitment of neutrophils to clear bacteria (direct effector recruitment and/or activation, second arrow from the top). In addition, a two-tiered response can be engaged, in which sensor cells secrete first-order cytokines that act on tissue-resident lymphoid cell populations, which integrate these signals and release appropriate second-order cytokines. These cytokines in turn recruit and activate effector cells and effector functions specific to the pathogen type, which serve to promote pathogen clearance and tissue repair. (Reproduced with permission from Ref. 23)

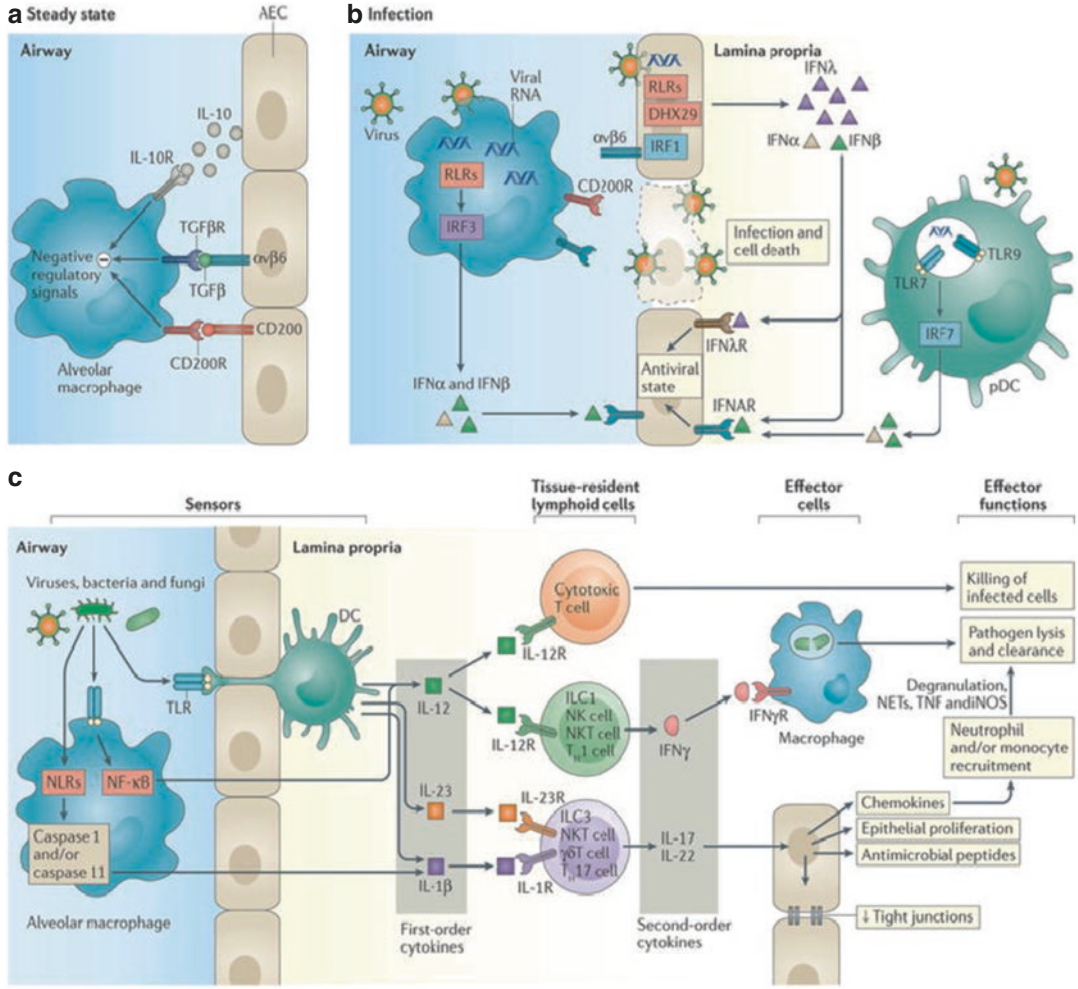

Fig. 28.2 Single- and two-tiered responses in type 1 immunity. (**a**) At steady state in the airways, alveolar macrophage activation is suppressed by negative regulatory signals in part delivered by CD200–CD200R and recognition of TGFβ presented by αvβ6 on airway epithelial cells (AECs). (**b**) During infection, disruption of these interactions due to death of AECs enables activation of macrophages. Recognition of viruses by pattern recognition receptors expressed by airway epithelial cells (AECs) leads to secretion of interferon-λ (IFNλ), whereas recognition by endosomal Toll-like receptors (TLRs) in plasmacytoid DCs (pDCs) and cytosolic RIG-I-like receptors (RLRs) and DNA sensors in alveolar macrophages leads to IFNα/β production. These IFNs induce an antiviral state in proximal AECs, inducing IFN-stimulated genes that help constrain viral spread. (**c**) TLRs expressed by alveolar macrophages and DCs that extend trans-epithelial processes enable the recognition of viral, fungal, and bacterial molecules, and bacterial pathogens in the airway leading to the production of first-order cytokines including interleukin-12 (IL-12) and IL-23. Additional pathogen recognition via inflammasome activation leads to caspase-1-mediated activation and release of the first order cytokine IL-1β. These first-order cytokines act on tissue-resident lymphoid populations of cytotoxic T lymphocytes to enhance direct killing of infected cells, and on innate lymphoid cells (ILCs), natural killer (NK) cells, NK T cells, and T cells to induce the production of appropriate second order cytokines including IFNγ, IL-17, and IL-22. These second-order cytokines in turn act on AECs to induce chemokine production, antimicrobial peptide release, and increased proliferation and/or tight junction formation to enhance airway integrity and constrain pathogen spread. Local and chemokine-recruited phagocytes including neutrophils and monocytes are additionally activated by IFNγ, enhancing their phagocytic capabilities and leading to enhanced pathogen lysis and clearance. (Reproduced with permission from Ref. 23)

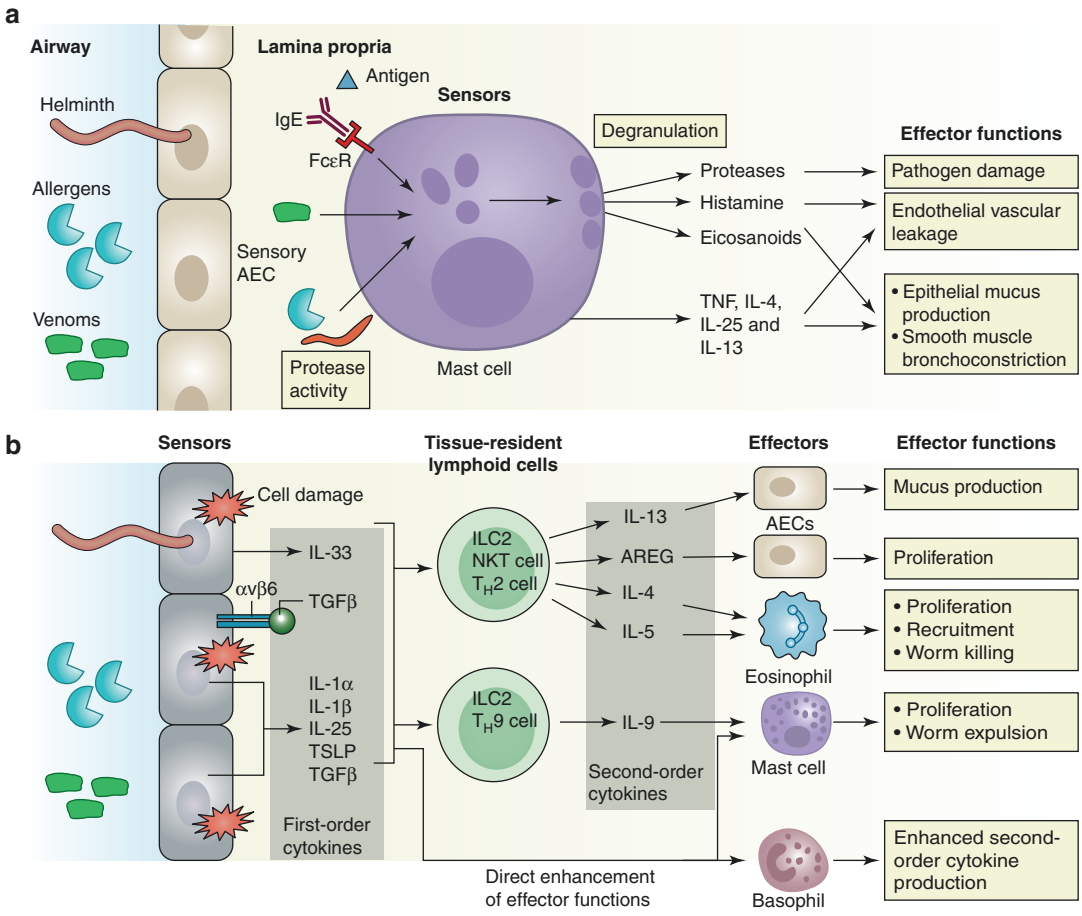

Fig. 28.3 Single- and two-tiered responses in type 2 immunity. Mast cells can be activated directly in response to certain protease activities, venom proteins homologous to mammalian mast cell-activating proteins, or through antigen-specific IgE-mediated signaling through FcεR, whereupon they can form a single-tiered immune response to helminths and allergens. (**a**) Activation leads to degranulation and release of proteases, histamine, and eicosanoids (including prostaglandins), as well as the production of certain effector cytokines. These compounds can directly initiate effector mechanisms that can promote worm expulsion but which are also associated with anaphylaxis in severe instances of allergy. (**b**) Cell damage and protease activity from helminth infection or exposure to allergens and venoms leads to the secretion and release of the first-order cytokines interleukin-25 (IL-25), TSLP, IL-33, IL-1β, and TGFβ (presented by αvβ6) from sensory airway epithelial cells (AECs). These cytokines in turn act on tissue-resident lymphoid cells including innate lymphoid cells (ILC2s), natural killer T cells, T helper 2 (Th2) cells, and Th9 cells to drive secretion of appropriate second-order cytokines, which act on mast cells, AECs, basophils, and eosinophils to initiate effector mechanisms aimed at worm expulsion and tissue repair. First-order cytokines can also enhance basophil and mast cell recruitment and activation in order to appropriately calibrate the immune response. (Reproduced with permission from Ref. 23)

the development of respiratory tract infection include impaired cough reflex, reduced phagocytic/bactericidal activity of alveolar macrophages (e.g., impairment by alcohol, smoking, anoxia, or oxygen toxicity), pulmonary edema, and mucociliary dysfunction leading to accumulation of airway secretions and airway obstruction [23] (Fig. 28.4).

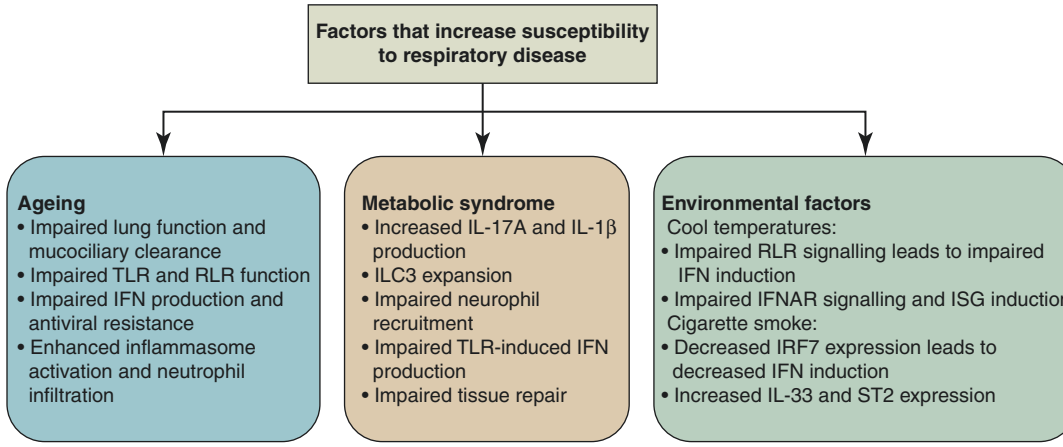

Fig. 28.4 Internal and external factors increase respiratory disease susceptibility. Both internal and external influences can alter early respiratory immune responses. External factors like cold temperatures and cigarette smoke can both impair sensor cell functionality and antiviral responses. Chronic conditions such as aging and metabolic syndrome can also have profound effects, altering the functions of sensors, lymphoid cells, and effector responses, thus disrupting various stages of the tiered respiratory immune response. Together these factors contribute to the increases in respiratory morbidity and mortality observed in populations affected by these factors. (Reproduced with permission from Ref. 23)

Injury Patterns in Chest Trauma that Predispose to Infection

Pulmonary Contusion

Pulmonary contusion, the most common type of lung injury in blunt trauma, is a term credited to the French military surgeon and anatomist Baron Guillaume Dupuytren in the nineteenth century, although reports acknowledge its description in the late 1700s by the Italian anatomist Giovanni Morgagni. Pulmonary contusion results by rapid deceleration when the moving chest strikes a fixed object leading to disruption of capillaries of the alveolar walls and septae, with extravasation of blood and fluid into alveolar spaces and the lung interstitium [25]. The contused lung is commonly surrounded by an area of edema. Fluid accumulation in alveoli interferes with normal gas exchange as a result of alveolar flooding, accumulation of proteinaceous debris, and collapse from disruption of surfactant production by type II pneumocytes.

Pulmonary contusion is often the initial insult ("first hit") in chest trauma patients, as it creates an environment conducive for pathogens to become established and proliferate. The acute inflammatory response to contusion involves recruitment of leukocytes, tissue macrophage activation, and the production of a series of mediators including cytokines, chemokines, oxygen radicals, eicosanoids, and components of the complement and coagulation cascades. Neutrophil-induced lung injury predominates in the first 24 h after lung contusion, but transitions quickly to a monocytic response by 48 h postcontusion that is associated with increased type II pneumocyte apoptosis. This response is stereotypical of injured lung, similar to the insults that lead to acute lung injury/acute respiratory distress syndrome (ALI/ARDS) [26]. A contusion volume of 20% of the pulmonary parenchyma is a robust predictor for ARDS (positive predictive value, 80%) (Fig. 28.5). Additionally, freely accessible iron supports lipid peroxidation and reestablishes nutrient sources for pathogens that

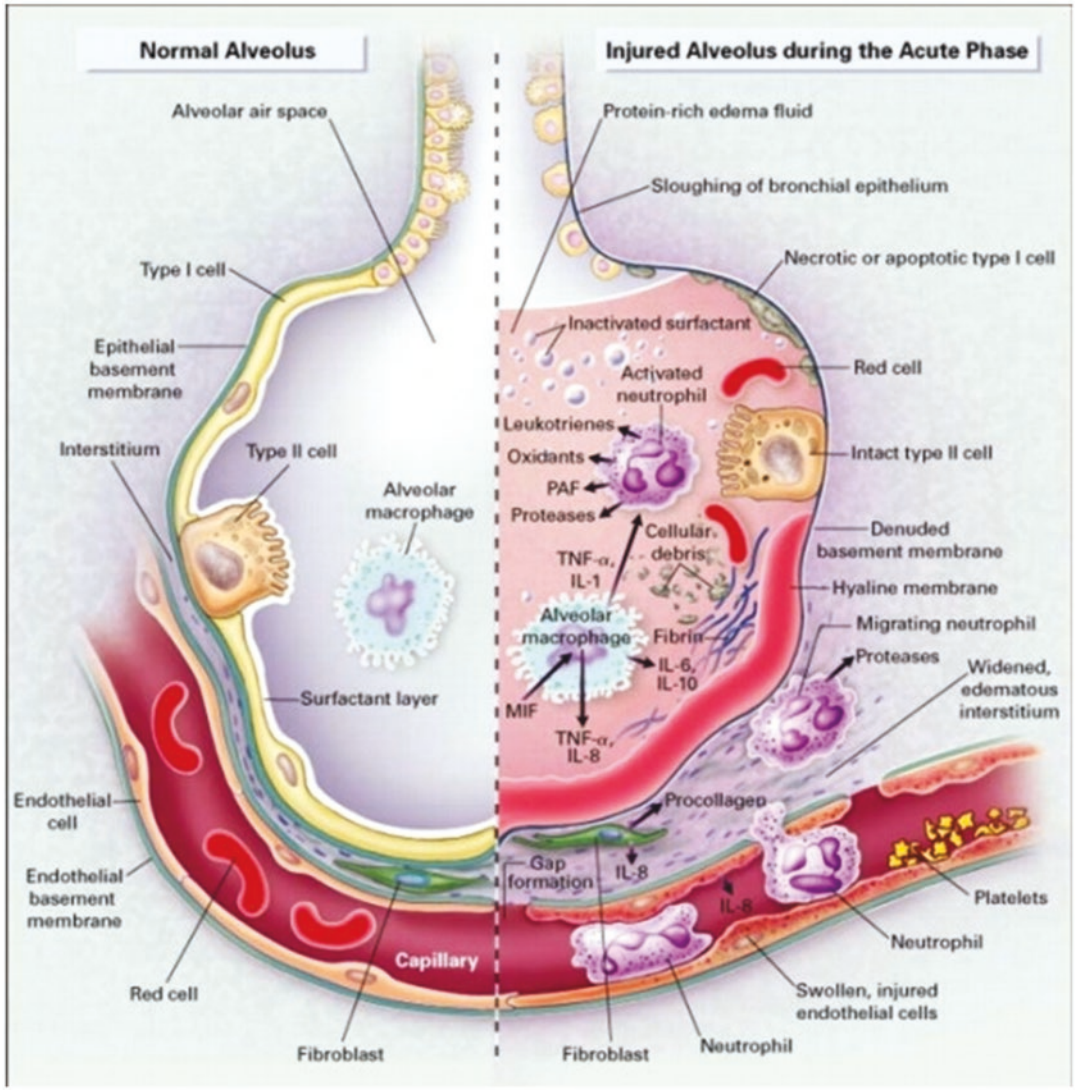

Fig. 28.5 The normal alveolus (left panel) and the injured alveolus in the acute phase of acute lung injury and the acute respiratory distress syndrome (right panel). This graphic depicts important differences between the alveolus in health and during acute inflammation

reach the lung by a variety of mechanisms, perhaps most notably, by aspiration.

Aspiration

Aspiration is defined as the inhalation of foreign material into the airways distal to the vocal cords. The aspirate may contain various substances such as food particles, oral medication, or blood or may be principally composed of gastric secretions [27]. The lower the pH of the aspirate, the higher the likelihood of injury; a pH > 4.0 is unlikely to cause lung injury. Injury, if it occurs, is instantaneous, but the local and systemic inflammatory responses can be protracted and may mimic findings consistent with sepsis or ARDS, even though, in its early stages, the injury from gastric acid aspiration is a sterile chemical pneumonitis (Mendelson syndrome). About 30%

of patients who aspirate will progress to a severe pulmonary inflammatory response from gastric aspiration or develop aspiration pneumonia if the aspirate also contained pathogens. Gastric acid suppression or preexisting gastric achlorhydria impede normal gastric acid elaboration that suppresses swallowed pathogen survival. Accordingly, both the events may enable aspiration pneumonia after injury.

Aspiration events are common after injury, including in patients sustaining chest trauma. Major risk factors associated with aspiration include increased age, nursing home care (due to increased prevalence of gastro-esophageal reflux disease and dysphagia), neurologic disorders (prior stroke, seizure or head/spinal cord injury), and disordered consciousness (brain injury, hypothermia, alcohol or illicit substance intoxication) [28]. Endotracheal intubation, even electively, is also associated with aspiration, and many patients presenting with chest trauma with multiple rib fractures or pulmonary contusions ultimately require airway control and invasive mechanical ventilation. Patients with facial fractures or trauma that results in increased intra-abdominal pressure are also at high risk for aspiration [29].

Aspiration Pneumonitis

Aspiration (chemical) pneumonitis is the ALI that occurs after the inhalation of regurgitated gastric contents. Within minutes after acidic fluid enters the lower airway, bronchoconstriction or spasm, atelectasis, peribronchial hemorrhage, pulmonary edema, and the degeneration of bronchial epithelium are consequential. In hours-to-days thereafter, alveoli fill with leukocytes, fibrin, and hyaline membranes in a chemokine-mediated phenomenon secondary to tumor necrosis factor (TNF)-alpha and IL-8 that also mediates the systemic inflammatory response [30, 31]. Associated symptoms and signs include fever, acute-onset dyspnea, and tachycardia. Hypoxemia is variable but may be profound. On auscultation, there may be diffuse crackles or wheezing. Imaging may demonstrate consolidation of dependent pulmonary segments, depending on the position the patient was in at the time of the aspiration event and the time elapsed after the event.

Treatment is mainly supportive, including head-of-bed elevation to at least 30°, judicious fluid resuscitation to support perfusion but not accelerate extravascular lung water, supplemental O_2, nebulized bronchodilators, and pulmonary hygiene [32]. Gastric decompression by nasogastric tube should be considered but is not mandatory [33]. Bronchoscopy is indicated initially only for airway obstruction by particulate debris, but may be subsequently useful (with quantitative microbiology) to help differentiate ongoing inflammation from new onset infection. Neither antibiotic prophylaxis nor glucocorticoids are recommended for pneumonitis as neither demonstrates benefit [33–35]. Noninvasive ventilation may support those with acute respiratory distress, and few require invasive mechanical ventilation. Invasive support is much more commonly required in those who develop pneumonia.

Aspiration

Pneumonia

While aspiration pneumonia may follow aspiration pneumonitis after trauma, those who do develop pneumonia may demonstrate additional risk factors including large-volume aspiration, poor dental hygiene, and proton-pump inhibitor or histamine receptor-2 antagonist therapy derailed gastric aid inhibition with subsequent gastric colonization [35, 36]. Local host defense mechanisms that normally protect against aspiration, including cough and mucociliary clearance, are impaired by pain, endotracheal intubation, analgesia/sedation/emergence from anesthesia, and a host of other factors that can depress the sensorium or disrupt local host defenses [28]. The bacterial burden in oropharyngeal secretions is typically small, but colonization of pharyngeal mucosa occurs rapidly after hospitalization, is often dominated by gram-negative bacilli, and creates another reservoir of potential pathogens [37, 38]. When host defenses are disrupted, especially if oral care is suboptimal, the risk of devel-

oping aspiration pneumonia increases [33, 39]. Pneumonia pathogens detected commonly after chest trauma include Enterobacteriaceae, non-fermenting Gram-negative bacilli (e.g., *Pseudomonas aeruginosa*, *Stenotrophomonas maltophilia*, *Acinetobacter baumannii* complex), and *Staphylococcus aureus*. [40–42]. The prevalence of Gram-negative organism pneumonia is related to the increased incidence of aspiration events in patients sustaining thoracic trauma [33, 43, 44].

Pneumonia

Pneumonia is the most common infection-related complication after chest injury and is closely related to both invasive mechanical ventilation and large-volume component transfusion therapy [45]. Those requiring ventilatory support are seven times more likely to develop pneumonia. The estimated risk of developing pneumonia increases by 1.2%–3.5% per day of mechanical ventilation [46]. Hospital-acquired (HABP) and ventilator-associated bacterial pneumonia (VABP) are often difficult to diagnose in patients who have sustained chest trauma. Plain chest radiography may be confounded by numerous causes of lung infiltrates (e.g., pulmonary contusion or laceration, atelectasis, hemothorax or other pleural effusions, ALI/ARDS). Chest computed tomography (CT) can help to distinguish among those confounders, but ultimately the diagnosis of HABP/VAPB requires identification of a pathogen.

A diagnosis of nosocomial pneumonia (pneumonia that develops more than 72 h after hospital admission) poses clinical and microbiologic challenges, especially patients with acute respiratory failure or preexisting ventilator-dependence [47–49]. Neither routine sputum aspirates nor portable chest X-ray demonstrates diagnostic specificity [50, 51]. The classic findings of fever, purulent sputum, leukocytosis, and a new pulmonary infiltrate on chest X-ray are caused by pneumonia in fewer than 50% of cases [52]. Sputum collected via endotracheal suction catheter is unreliable because such specimens can be contaminated by upper airway flora or by pathogens sequestered in the biofilm that accumulates in the lumen of an artificial airway. It is especially difficult to diagnose pneumonia in the presence of acute respiratory failure [49]; estimates from bronchoscopic surveillance data suggest that bacterial pneumonia complicates 20%–65% of cases of ARDS [53]. Overdiagnosis results in over-prescription of empiric antibiotic therapy, which is expensive, potentially morbid (there are a myriad antibiotic-related complications), and powerfully promotes the emergence of resistant pathogens [54, 55].

Sputum collection using a technique that avoids contamination is controversial. Options to avoid contamination include bronchoalveolar lavage (BAL) with or without fiberoptic bronchoscopy, or the protected-specimen brush technique, which has a sensitivity of 70–90%. Quantitative bacteriology showing 10^4 colony-forming units (CFU)/mL of BAL fluid collected is a consensus indicator of invasive infection [56]. BAL increases the overall accuracy of bronchoscopic diagnosis by increasing the specificity [57–59]. Unfortunately, validation of the diagnosis does not appear to improve outcome perhaps related to delays before initiating appropriate therapy [60, 61].

New Definitions

Frustration over the diagnosis of nosocomial pneumonia has prompted the US Centers for Disease Control and Prevention (CDC) to proffer new definitions. The frustrations include the confusion caused by a lack of consensus definitions for HABP and VABP and the lack of sensitivity and specificity of existing definitions. Moreover, extant definitions include a chest x-ray component, despite the fact that chest x-rays are unreliable for the diagnosis of VABP and the recognition that pneumonia is not the only pulmonary complication that may afflict the invasively mechanically ventilated patient.

New definitions include *ventilator-associated conditions* (VAC) and *ventilator-associated events* (VAE) (Tables 28.1, 28.2, 28.3, 28.4, 28.5).

Table 28.1 Ventilator-associated events (VAE) surveillance algorithm

Ventilator-associated condition (VAC)
After a period of stability or improvement on the ventilator, the patient has at least one of the following indicators of worsening oxygenation:
(1) Increase in daily minimum* FiO_2 of ≥0.20 (20 percentage points) over the daily minimum FiO_2 in the baseline period, sustained for ≥2 calendar days
(2) Increase in daily minimum* PEEP values of ≥3 cm H_2O over the daily minimum PEEP in the baseline period†, sustained for ≥2 calendar days
* Daily minimum defined by lowest value of FiO_2 or PEEP during a calendar day that is maintained for at least 1 h†. Daily minimum PEEP values of 0–5 cm H_2O are considered equivalent for the purposes of VAE surveillance
On or after calendar day 3 of mechanical ventilation and within 2 calendar days before or after the onset of worsening oxygenation, the patient meets both of the following criteria:
(1) Temperature > 38 °C or <36 °C, OR white blood cell count ≥12,000 cells/mm^3 or ≤4000 cells/mm^3 AND
(2) A new antimicrobial agent(s) is started and is continued for ≥4 calendar days
Infection-related ventilator-associated complication (IVAC)
On or after calendar day 3 of mechanical ventilation and within 2 calendar days before or after the onset of worsening oxygenation, ONE of the following criteria is met:
(1) Purulent respiratory secretions (from one or more specimen collections and defined as for possible VAP) AND one of the following:
• Positive culture of endotracheal aspirate*, ≥10^5 CFU/mL or equivalent semi-quantitative result
• Positive culture of bronchoalveolar lavage*, ≥10^4 CFU/mL or equivalent semi-quantitative result
• Positive culture of lung tissue, ≥10^4 CFU/g or equivalent semi-quantitative result
• Positive culture of protected specimen brush*, ≥10^3 CFU/mL or equivalent semi-quantitative result*
(2) One of the following (without requirement for purulent respiratory secretions):
• Positive pleural fluid culture (where specimen was obtained during thoracentesis or initial placement of chest tube and NOT from an indwelling chest tube)
• Positive lung histopathology
• Positive diagnostic test for *Legionella* spp.
• Positive diagnostic test on respiratory secretions for influenza virus, respiratory syncytial virus, adenovirus, parainfluenza virus, rhinovirus, human metapneumovirus, coronavirus
On or after calendar day 3 of mechanical ventilation and within 2 calendar days before or after the onset of worsening oxygenation, ONE of the following criteria is met:
(1) Purulent respiratory secretions (from one or more specimen collections)
• Defined as secretions from the lungs, bronchi, or trachea that contain >25 neutrophils and <10 squamous epithelial cells per low power field (lpf, ×100)
• If the laboratory reports semi-quantitative results, those results must correspond to the above quantitative thresholds
(2) Positive culture (qualitative, semi-quantitative, or quantitative) of sputum*, endotracheal aspirate*, bronchoalveolar lavage*, lung tissue, or protected specimen brushing*
*Excludes the following:
• Normal respiratory/oral flora, mixed respiratory/oral flora, or equivalent
• *Candida* species or yeast not otherwise specified
• Coagulase-negative *Staphylococcus* spp.
•*Enterococcus* spp.
Possible ventilator-associated pneumonia (see Table 28.4**)**
Probable ventilator-associated pneumonia (see Table 28.5**)**
Patient has a baseline period of stability or improvement on the ventilator, defined by ≥2 calendar days of stable or decreasing daily minimum* FiO_2 or PEEP values. The baseline period is defined as the 2 calendar days immediately preceding the first day of increased daily minimum PEEP or FiO_2. Information is in the public domain
*Daily minimum defined by lowest value of FiO_2 or PEEP during a calendar day that is maintained for at least 1 h

PEEP, positive end-expiratory pressure. www.cdc.gov. Information is in the public domain

Table 28.2 Ventilator-associated condition (VAC)

After a period of stability or improvement on the ventilator, the patient has at least one of the following indicators of worsening oxygenation:
(1) Increase in daily minimum* FiO_2 of ≥0.20 (20 points) over the daily minimum FiO_2 in the baseline period, sustained for ≥2 calendar days
(2) Increase in daily minimum* PEEP values of ≥3 cm H_2O over the daily minimum PEEP in the baseline period†, sustained for ≥2 calendar days
*Daily minimum defined by lowest value of FiO_2 or PEEP during a calendar day that is maintained for at least 1 h
Daily minimum PEEP values of 0–5 cm H_2O are considered equivalent for the purposes of VAE surveillance
Patient has a baseline period of stability or improvement on the ventilator, defined by ≥2 calendar days of stable or decreasing daily minimum* FiO_2 or PEEP values. The baseline period is defined as the 2 calendar days immediately preceding the first day of increased daily minimum PEEP or FiO_2
*Daily minimum defined by lowest value of FiO2 or PEEP during a calendar day that is maintained for at least 1 h

PEEP, positive end-expiratory pressure. www.cdc.gov. Information is in the public domain

Table 28.3 Infection-related ventilator-associated complication (IVAC)

Patient meets criteria for VAC
On or after calendar day 3 of mechanical ventilation and within 2 calendar days before or after the onset of worsening oxygenation, the patient meets both of the following criteria:
(1) Temperature > 38 °C or <36 °C, OR white blood cell count ≥12,000 cells/mm^3 or ≤4000 cells/mm^3 AND
(2) A new antimicrobial agent(s) is started and is continued for ≥4 calendar days

www.cdc.gov. Information is in the public domain

Table 28.4 Possible ventilator-associated pneumonia (VAP)

Patient meets criteria for VAC and IVAC
On or after calendar day 3 of mechanical ventilation and within 2 calendar days before or after the onset of worsening oxygenation, ONE of the following criteria is met:
(1) Purulent respiratory secretions (from one or more specimen collections)
• Defined as secretions from the lungs, bronchi, or trachea that contain >25 neutrophils and <10 squamous epithelial cells per low power field (lpf, ×100)
• If the laboratory reports semi-quantitative results, those results must be equivalent to the above quantitative thresholds OR
(2) Positive culture (qualitative, semi-quantitative, or quantitative) of sputum, endotracheal aspirate, bronchoalveolar lavage, lung tissue, or protected specimen brushing
Excludes the following:
• Normal respiratory/oral flora, mixed respiratory/oral flora, or equivalent
• *Candida* spp. or yeast not otherwise specified
• Coagulase-negative *Staphylococcus* spp
• *Enterococcus* spp

www.cdc.gov. Information is in the public domain

VAP is classified as possible or probable under the new definitions. The new definitions are detailed and cumbersome; the reader is directed elsewhere for in-depth information. It is likely that this terminology will remain a construct for epidemiologic reporting to the National Healthcare Safety Network (NHSN) of the CDC, since it seems too unwieldy to guide clinical care. An initial analysis of the utility of this approach for classifying critically ill surgical patients suggests that the system is both inaccurate and only minimally useful [62].

Microbiology

The pathogens of nosocomial pneumonia can be unit-specific and change over time. Therefore,

Table 28.5 Probable ventilator-associated pneumonia (VAP)

Patient meets criteria for VAC and IVAC
On or after calendar day 3 of mechanical ventilation and within 2 calendar days before or after the onset of worsening oxygenation, ONE of the following criteria is met:
(1) Purulent respiratory secretions (from one or more specimen collections and defined as for possible VAP) AND one of the following:
• Positive culture of endotracheal aspirate, $\geq 10^5$ CFU/mL or equivalent semi-quantitative result
• Positive culture of bronchoalveolar lavage, $\geq 10^4$ CFU/mL or equivalent semi-quantitative result
• Positive culture of lung tissue, $\geq 10^4$ CFU/g or equivalent semi-quantitative result
• Positive culture of protected specimen brush, $\geq 10^3$ CFU/mL or equivalent semi-quantitative result
*Same organism exclusions as noted for possible VAP, OR
(2) One of the following (without requirement for purulent respiratory secretions):
• Positive pleural fluid culture (where specimen was obtained during thoracentesis or initial placement of chest tube and NOT from an indwelling chest tube)
• Positive lung histopathology
• Positive diagnostic test for *Legionella* spp.
• Positive diagnostic test on respiratory secretions for influenza virus, respiratory syncytial virus, adenovirus, parainfluenza virus, rhinovirus, human metapneumovirus, coronavirus

CFU, colony-forming units. www.cdc.gov. Information is in the public domain

hospital-wide antibiograms may be less accurate than desired when selecting empiric therapy for those suspected of manifesting pulmonary infection. Common pneumonia etiologic agents include Gram-positive cocci (GPCs) that are more often methicillin-resistant *Staphylococcus aureus* (MRSA), followed by methicillin-sensitive *S. aureus* (MSSA) [63]. The most common causative Gram-negative bacilli (GNBs) are Enterobacteriaceae and *P. aeruginosa* [63]. Other non-fermenting GNBs such as *A. baumannii* complex and *S. maltophilia* can also cause pneumonia. The microbiology can differ depending on when pneumonia develops following injury. Within the first 5 days post-injury (early VAP), staphylococci (usually MSSA) and *Haemophilus influenzae* are commonplace. Patients with late VAP are more likely to have multi-drug-resistant (MDR) bacteria, including *P. aeruginosa*, A. *baumannii* complex, or extended-spectrum β-lactamase-producing organisms such as *Klebsiella* spp. or *Escherichia coli* [64]. The combination of bacterial virulence and resistance and impaired host defenses can make pneumonia a highly lethal infection following chest injury. For example, *Acinetobacter* can have an associated mortality that exceeds 50% in patients in the trauma intensive care unit [65, 66].

Viral and fungal causes of pneumonia are rare (0–7% of HAP and VAP). Immunocompromised patients (i.e., patients with HIV/AIDS or receiving immunosuppressants posttransplant), are at an increased risk for viral and fungal pneumonia. The most common fungal pathogens for pneumonia are *Pneumocystis jirovecii*, *Aspergillus* spp., and *Cryptococcus neoformans* [67, 68]. Viral pneumonia is caused most commonly by influenza viruses, rhinoviruses, and coronaviruses, is more common in pediatric patients, and typically requires only supportive treatment [69]. Note is made of specific therapeutic regimens that continue to evolve to treat SARS-CoV-2 infection.

Current recommendations for antimicrobial therapy of pneumonia consist of a broad-spectrum antibiotic regimen, with de-escalation of therapy once culture and susceptibility testing have been finalized [70]. Those patients at risk for infections caused by MDR bacteria should receive coverage with an anti-MRSA (e.g., vancomycin, linezolid) and anti-pseudomonal agent in combination, such as piperacillin–tazobactam, cefepime, or a carbapenem such as imipenem or meropenem. Therapy for confirmed VAP should continue for a total of 7 days [71]. Empiric antifungal therapy is not indicated.

Empyema

Pleural Space Infections and Empyema

Pleural space infection after injury can arise from parapneumonic effusion associated with pneumonia, pulmonary contusion, lung abscess, secondary infection of hemothorax, violation of the pleural cavity by penetrating injury, esophageal or bronchial rupture, or thoracic surgical interventions. The incidence of purulent fluid in the pleural cavity or *empyema* after thoracic injury ranges from 3% to 4% with an associated mortality rate as high at 23% [72]. Early recognition that drives rapid intervention that achieves source control is key in reducing mortality.

Pleural space infections presenting as parapneumonic effusions progress through three stages of development. Early effusions are driven by local inflammation, which increases the permeability of pleural vasculature and leads to the accumulation of free-flowing, transudative pleural fluid that is initially sterile. When bacteria invade the pleural fluid, the host response drives further immune cell infiltration and a pro-coagulant/anti-fibrinolytic response that marks a transition to the fibrino-purulent stage (exudative). Over time, fibrin deposition and fibroblast proliferation can produce a thick "rind" on the visceral and parietal pleura, restricting the expansion of the lung and chest wall [73]. The latter two stages are considered "complicated" effusions, and empyema may accompany either.

Pleural fluid analysis is the definitive method of establishing the diagnosis of pleural space infection. Normal pleural fluid has a pH ~7.6, protein concentration similar to interstitial fluid, a low cell count, and glucose and bicarbonate concentrations similar to serum [74]. Infection alters these characteristics, lowers pH and glucose, and can increase markers of inflammation such as lactate dehydrogenase. The diagnosis of empyema can be made with an abnormal Gram stain or culture, as well as grossly based on aspiration of purulent fluid during thoracentesis.

Bacterial isolates from pleural infections vary based on setting. In community-acquired empyema, aerobic Gram-positive cocci represent the majority of pathogens isolated by pleural fluid culture, followed by anaerobes and Gram-negative bacilli. In healthcare-associated pleural space infections, the incidence of Gram-negative pathogens in fluid culture more than doubles, and anaerobes are less frequently identified [75]. In traumatic empyema, the organism most commonly identified is *S. aureus*, and discordance between bronchial aspirate culture and pleural fluid is common perhaps reflecting differences between the microbiomes of the chest wall compared to the aerodigestive tracks [76].

Management of pleural space infections depends on the stage of infection [77]. Uncomplicated effusions are often successfully managed with antibiotics alone, while complicated effusions require drainage. Drainage typically progresses in a stepwise manner, starting with tube thoracostomy placement; more recently, percutaneous smaller bore catheters have been deployed with great success for more thin fluid management. However, about one-half of patients so treated will require additional intervention, drain manipulation (e.g., repositioning, upsizing), placement of a second drain, or surgical intervention [78]. Complicated effusions are often characterized by loculations. Several studies have shown a benefit from intrapleural fibrinolytic therapy to degrade loculations due to fibrin (e.g., instillation of recombinant tissue plasminogen activator [rtPA] via the tube to promote drainage), data are mixed, and routine use is not recommended [79].

In patients who fail to improve with antibiotics and percutaneous drainage, formal surgical drainage is recommended. Surgical management of pleural infection is directed at the complete evacuation of infected material, with the goals of obtaining source control and decortication of the pleural rind to allow lung re-expansion and unrestricted movement relative to the chest wall (at least initially). Decortication also allows the pleural space to be "open" supporting dependent suction-assisted drainage of fluid and apical

suction-assisted drainage of gas since multiple small visceral pleural breaches are common. Video-assisted thoracoscopic surgery (VATS) has been increasingly used for the surgical management of empyema and is associated with improved postoperative pain control, reduced respiratory compromise, and reduced morbidity when compared to open surgery (thoracotomy) for empyema. Accordingly, a VATS approach is recommended for the initial surgical management of empyema [79].

In patients for whom thoracostomy tube management of empyema fails and who are too ill to tolerate decortication, or those with persistent infection after decortication, a thoracic drainage window can provide long-term drainage of the pleural space. Thoracic wall windows are surgically created openings into the pleural space that act as one-way valves allowing for the drainage of pus without influx of air. The Eloesser flap, which was originally described in 1953 for the management of tubercular empyema, is the most well-known. It involves resection of portions of one to three of the posterolateral ribs of the affected side and marsupialization of the skin flap to the parietal pleura to establish an epithelialized drainage track. Subsequent modifications have reduced the complexity and morbidity of the procedure [80].

Empyema due to hemothorax appears unique to trauma patients. Numerous studies have identified hemothorax as an independent risk factor for the development of empyema in patients with thoracic injury [81, 82]. This contributes to the recommendation that all hemothoraces be evacuated to prevent the development of empyema. If initial hemothorax drainage is inadequate, retained blood presents a risk, with some reports of the incidence of empyema developing in as many as one-third of patients with retained hemothorax after tube thoracostomy [83]. Several randomized studies demonstrate the superiority of surgical intervention over protracted catheter drainage or fibrinolytic therapy in preventing the development of empyema in trauma patients. Therefore, early VATS is recommended for retained hemothorax management [84].

Lung Abscess

It is important to distinguish empyema from lung abscess, which is typically described as a circumscribed area of purulence or necrosis bounded by lung parenchyma. Many are caused by polymicrobial infection, often including anaerobes. Primary lung abscesses result from direct infection of the pulmonary parenchyma such as from aspiration, whereas secondary lung abscesses are typically due to an underlying condition such as presence of a foreign body or neoplasm leading to bronchial obstruction. Other causes of secondary lung abscesses include complicated bacterial pneumonia, bacteremia, or endocarditis (particularly right-sided) leading to septic emboli, or as a result of direct inoculation after penetrating thoracic injury [85].

Most patients who develop a lung abscess do not do so within an index hospitalization, but instead present later with nonspecific symptoms of cough (often with foul-smelling sputum), chest pain, or dyspnea. Patients may also present with systemic symptoms such as fever, chills, or night sweats. The evaluation of lung abscesses typically starts with a chest X-ray or thoracic CT scan. Findings concerning for abscess include a thick-walled cavity with an air-fluid level (Fig. 28.6). Sputum Gram stain and culture and blood cultures should be obtained to help direct

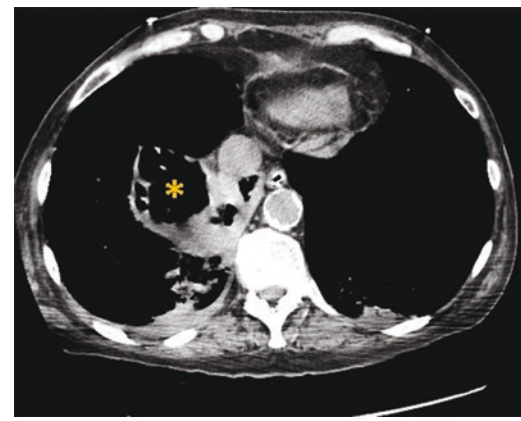

Fig. 28.6 Thoracic CT scan of a pulmonary abscess. Chest CT scan demonstrating pulmonary abscess (*). Original patient image for LJK (Editor)

therapy. The use of bronchoscopy to obtain sputum samples is recommended [86].

The microbiology of lung abscesses varies based on the route of infection that led to abscess development. For example, if resulting from aspiration, most abscesses will be polymicrobial due to the presence of oral and gingival flora. The most common pathogens encountered will typically be streptococci and anaerobes (*Bacteroides* spp., *Prevotella* spp., or *Peptostreptococcus* spp.) [87]. In immunocompromised patients, Gram-negative species can also be identified. Aerobic bacteria can lead to monomicrobial lung abscesses with typical agents including *S. aureus*, *K pneumoniae*, *P. aeruginosa*, *Streptococcus pyogenes*, or *H. influenzae* although this is less common. Select nonbacterial pathogens can also result in cavitary lesions that resemble lung abscess. These include *Aspergillus* spp., *Cryptococcus* spp., *Coccidioides* spp., and also mycobacteria, fungi, or parasites [87, 88]. Therefore, bronchoscopic sampling is key in establishing the diagnosis and guiding therapeutic agent tailoring.

Initial antimicrobial treatment of a lung abscess begins with an empiric broad-spectrum regimen, with de-escalation based on culture and susceptibility test results. Chosen agents should have adequate lung parenchymal penetration (acceptable partition coefficient) and target both anaerobes and streptococci. Examples could include a beta-lactam-beta-lactamase inhibitor agent with anaerobic coverage (e.g., piperacillin–tazobactam) or an anti-pseudomonal carbapenem. In patients with penicillin allergy, consider alternative regimens that incorporate moxifloxacin or a combination of levofloxacin with metronidazole. The optimal duration of antibiotics for the treatment of lung abscesses is unknown and must be determined on a case-by-case basis driven most importantly by patient condition and response to therapy [85, 88].

Antibiotics alone will suffice for most cases. Failure of improvement benefits from re-imaging of the chest to look for progression or the development of complications. Approximately 10% of patients will require a drainage procedure or surgical resection. Options for drainage include either catheter drainage or needle aspiration, the latter either through a percutaneous or transbronchial approach. For lung abscesses resulting from bronchial obstruction, abscess resolution will not occur unless the obstruction is relieved. As a last resort, patients who fail to improve with antibiotic therapy and drainage approaches, or who develop further complications such as bronchopleural fistula or pulmonary hemorrhage, may be candidates for resection (lobectomy or, in rare cases, pneumonectomy) [89]. Such management is rarely encountered and is associated with accelerated morbidity and mortality.

Mediastinitis/Ludwig's Angina

Ludwig's angina, also known as *morbus strangularis* and *angina maligna*, is a rapidly progressive bilateral cellulitis of the submandibular space associated with elevation and posterior displacement of the tongue that usually occurs in adults with concomitant dental infections. Cases have been also reported following facial trauma, specifically mandibular fracture. It is named after the Stuttgart physician Karl Friedrich Wilhelm von Ludwig, who first described the condition in 1836 [90]. His description was based on the observation of five patients with "*gangrenous induration of the connective tissues of the neck that advanced to involve the tissues that cover the small muscles between the larynx and the floor of the mouth*." Untreated, an abscess may form that can invade along tissue planes and track into the mediastinum.

After chest injury, the most common cause of mediastinitis is esophageal perforation. The most lethal form of mediastinitis is descending necrotizing mediastinitis (DNM) [91]. DNM, which occurs as a complication of oropharyngeal abscesses, or as a complication of cervical/thoracic inlet trauma, occurs when infection spreads along fascial planes into the mediastinum (Figs. 28.7 and 28.8) [91]. Endo et al. [92, 93] proposed a classification scheme to facilitate DNM management based on CT assessment of the extent of infection. Type I DNM—infection localized to the upper mediastinum above the tra-

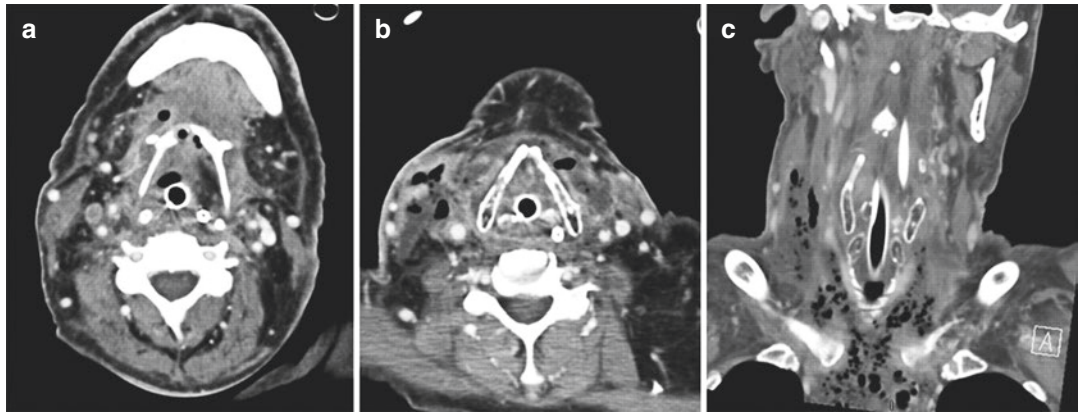

Fig. 28.7 Contrast CT of Ludwig's angina and descending necrotizing mediastinitis. Contrast-enhanced computed tomography of the neck of a case of Ludwig angina with progression to descending necrotizing mediastinitis. Note the substantial retromandibular edema (panel **a**), extensive infrahyoid phlegmon and gas, especially on the right (panel **b**), and extension of gas below the thoracic inlet and into the mediastinum (panel **c**). (Reproduced from Ref. 91, with permission)

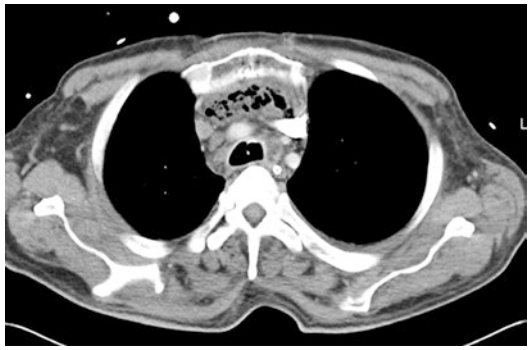

Fig. 28.8 Thoracic CT scan of descending necrotizing mediastinitis. Contrast-enhanced chest computed tomography of the case depicted in Fig. 28.7, demonstrating a complex gas and fluid collection in the anterior mediastinum, consistent with mediastinitis. (Reproduced from Ref. 91, with permission)

cheal bifurcation—may be managed with mediastinal drainage via a transcervical approach without opening the chest. Diffuse DNM is subclassified as type IIA when infection involves the lower anterior mediastinum or as type IIB if both the anterior and posterior lower mediastinum are involved. In type IIA infection, cervicotomy and a subxiphoid anterior mediastinotomy may provide adequate exposure for debridement and drainage. For type IIB infection, combined cervicotomy and thoracotomy with wide debridement is recommended to obtain source control.

Acute mediastinitis is a serious infection involving the areolar tissue that fills the interpleural spaces and surrounds the midline thoracic organs. As infection spreads along deep cervical fascial planes into the mediastinum or access that same space by direct extension after esophageal perforation, widespread cellulitis, tissue necrosis, and abscess formation are a continuum that benefits from early diagnosis and rapid rescue. Delayed diagnosis and delayed or incomplete mediastinal drainage are the main reasons for the high mortality rate of this life-threatening condition.

Microbiology and Antibiotic Therapy

Mixed aerobic and anaerobic bacteria found in the oral flora are the predominant microorganisms isolated from DNM patients. The most common aerobic bacteria include alpha hemolytic *Streptococcus*, *S. aureus*, and K. *pneumoniae*. The most common anaerobic bacteria include *Peptostreptococcus* spp., *Bacteroides fragilis*, *Prevotella* spp., and *Porphyromonas* spp. Other organisms reported include viridans group *Streptococcus*, *Serratia marcescens*, *Enterobacter* spp., *Neisseria* spp., *Fusobacterium* spp., *P. aeruginosa*, *E. coli*, *S. maltophilia*, and *Veilonella*

spp. Antibiotic therapy should be started immediately and be sufficiently broad to cover Gram-positive cocci, Gram-negative bacilli, and anaerobic bacteria. Empiric treatment regimens include piperacillin–tazobactam and vancomycin, a third- or fourth-generation cephalosporin plus an antianaerobic agent, or for penicillin-allergic patients, a fluoroquinolone plus an antianaerobic agent. Once susceptibilities are known, therapy can be tailored accordingly. Based on hypothesized effects of increased O_2 tension on anaerobic microbial growth, hyperbaric O_2 (HBO) therapy has been suggested as a potential treatment for DNM. Whereas HBO may ultimately prove useful, more study is required to establish therapeutic benefit.

Infectious Complications After Trauma Pneumonectomy

Trauma pneumonectomy engenders substantial morbidity and mortality. Immediately following pneumonectomy, air fills the space previously occupied by the lung (i.e., the postpneumonectomy space [PPS]). Unlike most other thoracic procedures, a chest tube is *not* inserted following pneumonectomy. The physiologic rationale for this is to allow the PPS to slowly fill with serosanguinous fluid so as to balance pressures in the two hemithoraces, eventually leading to complete opacification. A chest tube, in this situation, would therefore be counterproductive, in that negative pressure in the evacuated hemithorax could over-shift the mediastinum ipsilaterally, compromising venous return. Over time, a number of additional important changes occur that lead to a slow decrease in the size of the PPS. These including elevation of the hemidiaphragm, hyperinflation of the remaining lung, and some shifting of the mediastinum toward the PPS [94]. As a general rule, fluid accumulates at a rate of one to two intercostal spaces/day in the immediate postoperative period. Unexpected rapid accumulation of fluid into the PPS in the immediate postoperative period should raise the possibility of postoperative hemorrhage or the development of chylothorax [94]. By chest X-ray, complete opacification of the hemithorax after pneumonectomy takes approximately 4 months (range, 3 weeks to 7 months) [95].

There are three main complications following pneumonectomy. First is postpneumonectomy pulmonary edema of the contralateral lung that may require diuresis or high-flow nasal cannula oxygen therapy to manage; reintubation is uncommon but should be done under bronchoscopic control to avoid bronchial cuff disruption. Second is postpneumonectomy syndrome which includes excessive mediastinal shift resulting in compression and stretching of the tracheo-bronchial tree and the esophagus, and displacement of the trachea that leads to exertional dyspnea and inspiratory stridor on rapid inspiration [96]; rescue often requires airway control as well as PPS decompression (albeit without suction). When due to hemothorax, repeat operation is indicated. Third is intraoperative contamination of the contralateral lung which is in part mitigated against by double lumen tube placement, or deliberate mainstem intubation of the uninjured lung.

The main categories of pleural space complications of either the PPS or the contralateral pleural space include infection, fistula formation, bleeding, chylothorax, and contralateral pneumothorax. Postpneumonectomy empyema (PPE) occurs after pneumonectomy in approximately 5% of patients [97]. Early empyema, occurring within 10–14 days postprocedure, is commonly associated with a bronchopleural fistula. Risk factors for developing a bronchopleural fistula include older age (>60 years), right-sided procedures, immunocompromise (receiving either radiation or chemotherapy or with poor wound healing), bronchial stump diameter >25 mm, or the presence of residual tumor (generally not applicable to postinjury patients) [95].

Late empyema typically occurs more than 3 months after pneumonectomy and has been reported up to decades following surgery. The etiology is most often acquired via a hematogenous route. The most common organisms causing postpneumonectomy empyema are *S. aureus* and *P. aeruginosa*. Almost 50% of cases of both early and late empyema are polymicrobial. The presen-

tation of patients with PPE is similar to patients who develop empyema for other causes as described above in the empyema section of this chapter.

Chest X-ray or CT scan aids in establishing the diagnosis of PPE. Specific radiographic findings suggestive of the diagnosis include mediastinal shift away from the PPS, failure of the mediastinum to shift normally in the immediate postoperative period, development of a new air-liquid level, or a sudden change in a preexisting air-liquid level. Regardless of imaging presentation, the definitive diagnosis of PPE is confirmed by PPS fluid sampling. Treatment of PPE includes systemic antibiotics, drainage of the PPS, and repair of any coexisting bronchopleural/esophagopleural fistula once the PPS is sterilized [98]. Patients with large (>3 mm) bronchopleural fistulas will require surgical debridement, irrigation of the pleural cavity, and closure of the pneumonectomy stump with an omental patch (Clagett procedure) or a musculofascial pedicled flap (i.e., intercostal flap). Smaller fistulas (<3 mm) may be approached using VATS.

Chylothorax occurs in fewer than 1% of cases and is usually observed in patients undergoing pneumonectomy with lymphadenectomy—generally not a procedure undertaken after injury. The diagnosis should be considered when there is rapid filling of the PPS in the immediate postoperative period. Asymptomatic patients with slow accumulation of chyle can be treated with bowel rest for anticipated resolution. In patients with signs and symptoms of elevated central venous pressure, tachycardia, dyspnea, or hypotension as well as radiographic evidence of rapid filling of the PPS, drainage and surgical repair may be required. Chyle is usually considered to be bacteriostatic, and chylothorax rarely becomes infected. However, case reports of infected chylothorax have been described, and the clinician must be wary of clinical deterioration in this setting. Fluid sampling for laboratory profiling and culture can aid in the diagnosis [97, 98]. Other modalities to manage chylothorax include surgical control as well as duct embolization, although the latter may not be universally available.

Summary

Pulmonary infections in patients sustaining chest trauma require increased vigilance for proper diagnosis and management. Understanding the anatomy and physiology of the chest including the bony skeleton, the lungs and pleurae, the tracheobronchial tree, the esophagus, and the cardiovascular system is crucial. This review of the physiology and pathophysiology of chest injury creates a construct for diagnosing, managing, and ideally preventing pulmonary infection following chest injury.

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29

Blast Injury, Blast Lung, and Pulmonary Aspiration

John Hunninghake, Michael Gonzalez, and Jeremy C. Pamplin

Introduction

Although explosion-related injuries are historically less commonly encountered in civilian practice, the use of explosive weaponry in both military operations and civilian-relevant violent extremism appears to be increasing in frequency and scope [1–4]. Unlike other mechanisms of injury, explosions often inflict both blunt and penetrating injury. Importantly, the combined patterns of injury are often unfamiliar for clinicians without military-relevant experience. Moreover, besides the readily identifiable penetrating and related blunt injury, explosions are accompanied by blast pressure waves which present a different injury pattern than what is commonly encountered in civilian practice. Therefore, examining the care of the thoracic trauma patient who has sustained penetrating, blunt, and blast pressure injury is essential in order to ensure thorough evaluation, but to also allow the treating intensive care team to anticipate potential complications, prevent them when possible, and expeditiously treat them when they arise.

Blast pressure wave effects may cause extensive soft tissue damage through deformation or over-pressure, which may seem innocuous at first but evolve over time. The lungs and their gas and tissue/fluid interfaces are particularly vulnerable to blast injury. For example, a retrospective review of the United Kingdom's joint theater trauma registry from 2003 to 2009 revealed 1678 injuries secondary to explosions and 113 cases of blast lung. Notably, only 50 of those 113 patients survived to arrival at a care facility, and 80% of initial survivors required invasive mechanical ventilation [5]. This chapter explores explosion physics and the mechanisms of blast injury, followed by pathophysiology and management approaches specific to blast lung injury.

Explosion Physics

Explosions rapidly convert explosive material into a large volume of gas causing a rapid increase in local pressure that generates a blast wave. This wave propagates in all directions away from the source; such waves may have their effects magni-

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fied when they arise within confined spaces. When the traveling wave interacts with the human body, the results vary depending on the characteristics of the tissue the wave encounters. As the wave impacts gas-filled organs (such as lungs, bowel, ears) energy transmission results in damaging stress and shear forces as well as gas expansion [6, 7]. Damage may be augmented by gas expansion within a closed space such as the airway when the glottis is closed. Tympanic membrane rupture, pneumothorax, hollow viscus rupture are all well-described sequelae. Less at risk than gas-filled spaces are liquid-filled spaces (such as blood vessels and solid organs) at their junction with solid organs due to wall shear stress. While blast wave injury seems like a discrete event, blast injury encompasses multiple dimensions and phases.

Injuries inflicted by explosive devices may be conveniently divided into at least five separate mechanisms that relate to the kinds of injuries that may be evident: primary, secondary, tertiary, quaternary, and quinary (Fig. 29.1). The primary mechanism of injury is blast wave and mainly results in injuries to the ears, lungs, and abdominal hollow viscus. After the shock front of the blast wave quickly dissipates, the subsequent blast wind causes secondary injuries by propelling surrounding debris and fragments that strike victims and cause principally penetrating injuries; blunt injury may occur in those wearing body armor that retains integrity. Tertiary injuries occur when the blast wind propels large objects into victims or fling victims against fixed structures mainly establishing blunt impact injury. Quaternary injuries are caused by the heat, flames, gas, and smoke generated during explosions, while quinary injuries are attributable to bacteria or explosion-related radiation damage. The prevalence and magnitude of these injuries are affected directly by the size of the explosion and inversely by the distance from the

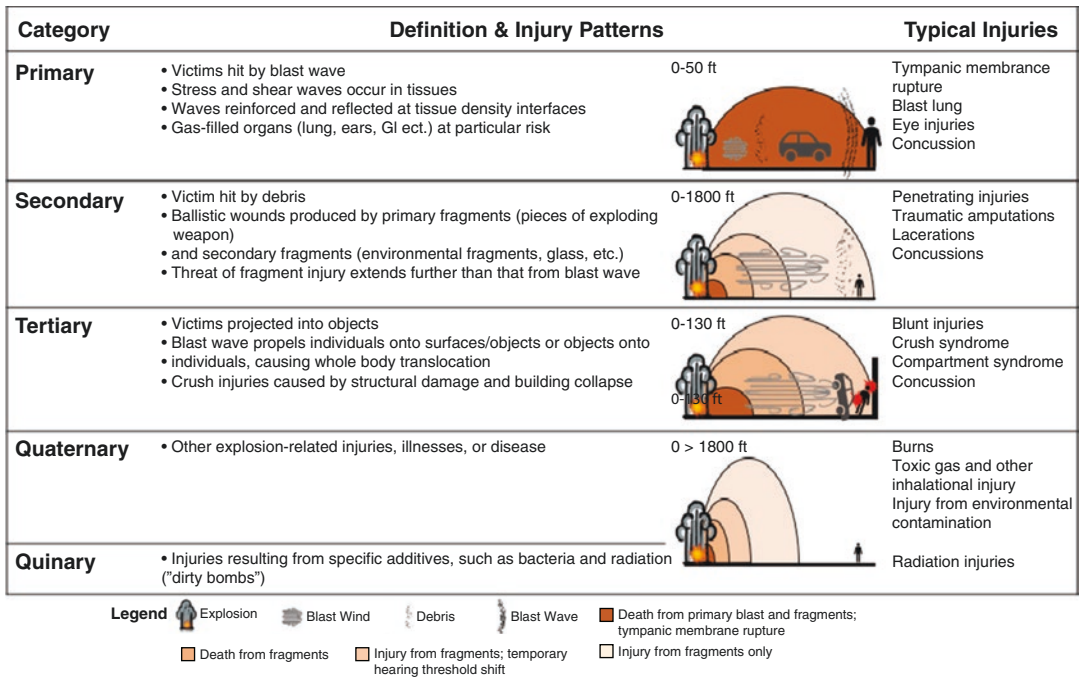

Fig. 29.1 Blast explosion injuries. The morbidity and mortality related to blast injuries are directly affected by the size of the explosion and inversely by the distance from the explosion. Distance from an explosion determines the amount of energy adsorbed from penetrating missiles and is the single most important influence on survival. (*Adapted from Champion HR et al, "Injuries from Explosions: Physics, Biophysics, Pathology, and Required Research Focus" J Trauma 2009 and Geiling J, Burns SM, eds. Fundamental Disaster Management, 3rd ed. Mount Prospect, IL: Society of Critical Care Medicine; 2009*)

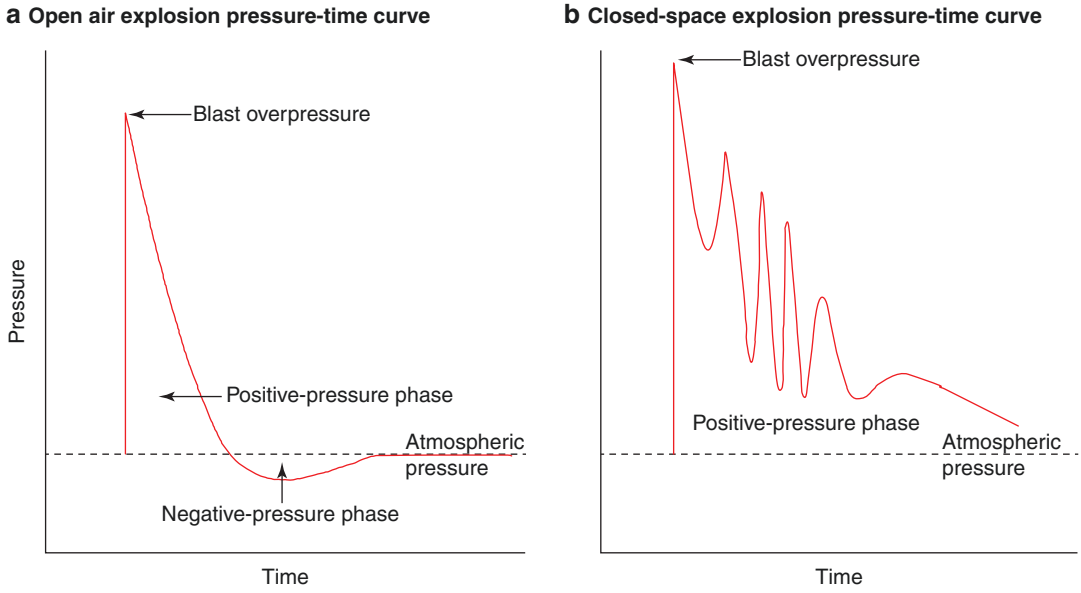

Fig. 29.2 Explosion pressure-time curves in open and closed spaces. Pressure changes differ in an open-air explosion (**a**) compared to a closed-space explosion (**b**). The blast overpressure and the positive-pressure phase are responsible for causing the range of blast injuries. (Used with permission from Wolf SJ, Bebarta VS, Bonnett CJ, Pons PT, Cantrill SV. Blast injuries. Lancet. 2009 Aug 1. 374(9687):405–15)

explosion [8]. The distance from an explosion locus determines the amount of energy adsorbed from penetrating missiles and is the single most important influence on survival [9].

The type of space in which injury occurs also influences outcome with clear differences between open air and enclosed space explosive injuries. Fig. 29.2 graphically plots the pressure changes over time and demonstrates the various pressures for simple open-space and closed-space explosions. Notably, the blast overpressure and positivepressure phase are key elements in establishing blast injuries [1]. Pressure waves in a closed-space explosion do not dissipate as do those in open air, but instead reflect off surfaces causing increased pressure over time and enhance the destructive power of the wave [10]. Accordingly, closed space pressure–time curve is denoted as complex compared to those which occur in open spaces. Increased blast pressure over time in enclosed spaces and variable blast wave directions make injury patterns more unpredictable and more severe. Unsurprisingly, mortality from closed space explosions seemly uniquely related to blast lung injury compared to mortality after open-air explosions that is more related to secondary, tertiary, and quaternary mechanisms [11–13].

Blast Lung Injury

The lungs are particularly vulnerable to primary blast effects, which have been reported in up to 38% of victims in conventional explosions [9]. Primary blast lung injury (PBLI) is characterized by a rapid onset after explosion exposure and requires that features that would suggest secondary or tertiary injury are absent [14]. PBLI occurs as a consequence of the blast pressure causing rapid over-distension of pulmonary parenchyma with disruption of the inter-alveolar septae. The intense barotrauma leads to alveolar damage through enlargement, over-distention, and rupture, which clinically manifests as pulmonary contusion, pneumothorax, pneumatocele, subcutaneous emphysema, tracheal injury, as well as pneumomediastinum [15]. For survivors of the initial blast, the extent of pulmonary injury is a significant determinant of later mortality [9].

This injury rarely occurs in isolation and should therefore prompt a thorough examination for related injuries as would be appropriate for victims of multiple trauma. However, given its clinical significance as a key driver of outcome, it is appropriate to further explore PBLI pathophysiology and clinical manifestations to help guide optimal treatment.

Pathophysiology

PBLI is the result of rapid and excessive pressures changes at tissue–density interfaces, occurring most notably with air and fluid-containing organs. Explosion-driven pressure changes lead to significant damage through both stress and shear forces that act upon tissue interfaces. Organs with substantial air content coupled with large air–tissue surfaces are particularly vulnerable to the acceleration/deceleration forces produced by blast injury. PBLI has further implications given the airway is contiguous with the atmosphere. The blast wave travels via the tracheobronchial tree and simultaneously applies compressive forces on the thorax. These pressure changes travel at different speeds and produce multiple pressure differentials across the pulmonary parenchyma and airways. With unequal transmission of pressure, tissue deforms and leads to the spectrum of PBLI that spans lung contusion, pneumothorax, parenchymal laceration, and alveolo-venous fistulas which promote air emboli [16]. Other organs with similar gas–tissue interfaces should also be interrogated for blast induced injury, but those evaluations exceed the scope of this chapter.

Clinical Presentation and Diagnosis

PBLI is a clinical diagnosis and can be difficult to establish in the presence of secondary and tertiary blast injuries. However, diagnosis is imperative given the accelerated mortality that can reach 60% in initial PBLI survivors [9]. Much like the acute respiratory distress syndrome (ARDS), blast lung injury may be categorized as mild, moderate, and severe based on clinical signs and symptoms (Table 29.1). Notable differences exist in the PaO_2/FIO_2 ratios that demarcate each grouping for those with PBLI compared to the Berlin modification of the ARDS criteria. Nonetheless, patients typically present with hypoxic respiratory distress within 12 h of initial injury, and once established, progression is common.

PBLI signs and symptoms are unfortunately nonspecific and include tachypnea, tachycardia, dyspnea, cyanosis, and hemoptysis. Physical examination findings also suggest a number of related diagnosis based on diminished breath sounds, crackles, and crepitus from subcutaneous gas [14, 16]. Radiologic findings are generally more helpful in establishing a diagnosis as plain chest radiography demonstrates bilateral perihilar infiltrates ("batwing" pattern) (Fig. 29.3), while CT imaging consistently presents diffuse

Table 29.1 Primary blast lung injury severity classification

Classification	Mild	Moderate	Severe
Radiographic appearance	Unilateral or limited opacities	Asymmetric bilateral opacities	Diffuse bilateral infiltrates, "batwing appearance"
PaO_2/FiO_2 ratio	>200	60–200	<60
Bronchopleural fistula (BPF)	Not present	Moderate BPF (no major BPF present)	Major BPF
Mechanical ventilation	None	Yes, routine settings	Yes, advanced settings with PEEP >10 cmH_2O Consider ECMO

PaO_2, partial pressure of oxygen in arterial blood; FiO_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure; ECMO, extracorporeal membrane oxygenation

Adapted from: Geiling J, Burns SM, eds. *Fundamental Disaster Management*, third ed. Mount Prospect, IL: Society of Critical Care Medicine; 2009

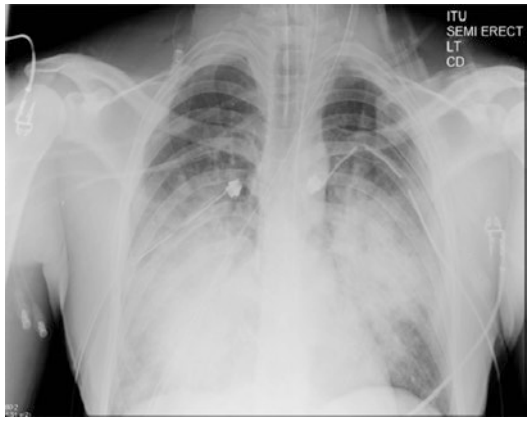

Fig. 29.3 Primary blast lung injury radiograph. Characteristic batwing appearance of PBLI on frontal plain chest radiograph. (Used with permission from Johnston, A., Alderman, J. Thoracic Injury in Patients Injured by Explosions on the Battlefield and in Terrorist Incidents Chest 2019)

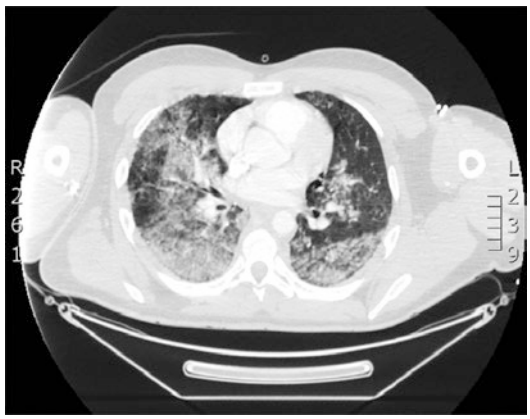

Fig. 29.4 Primary blast lung injury CT scan. Contrast-enhanced chest computed tomography (CT) scan demonstrating bilateral diffuse alveolar infiltrates consistent with PBLI. (Used with permission from Johnston, A., Alderman, J. Thoracic Injury in Patients Injured by Explosions on the Battlefield and in Terrorist Incidents Chest 2019

alveolar infiltrates consistent with alveolar hemorrhage (Fig. 29.4) [17]. Variably present CT findings span the gamut of lung injury diagnoses including pulmonary laceration, pneumatocele formation, interstitial emphysema, and pneumo- or hemothorax, some of which may be subtle and not detectable on plain radiography [16]. Therefore, all patients who are injured in proximity to an explosive detonation should undergo thoracic CT scanning for injury assessment. Air embolism is also a known complication of blast injury that is frequently seen within 30 min following the blast, and may be one of the main causes of cardiac dysfunction, stroke, intestinal ischemia, blindness, spinal cord injury, and immediate death [9].

Management

PBLI patients may demonstrate acute respiratory distress or acute respiratory failure, but may also progress from the former to the latter as injuries evolve. Nearly one-quarter of such patients are successfully managed using supplemental oxygen—including high-flow nasal cannula oxygen—or other forms of noninvasive ventilation (NIV). It is important to recognize that if a PBLI patient is able to be managed using solely NIV, their improved prognosis is related to reduced injury severity compared to those who require invasive mechanical ventilation (IMV). Up to 76% of patients with PBLI will require IMV to address acute respiratory failure [12, 18]. The need for IMV is juxtaposed with the injury that drives the need for IMV, making avoidance of ventilator-induced lung injury both an imperative and a substantial challenge [12, 18]. There is limited data to guide VILI avoidance after PBLI in humans, and therefore, extrapolation from other well-studied conditions such as ARDS is intuitively attractive [19]. Regardless, PBLI patients who require IMV should receive lung protective ventilation with careful attention paid to complication surveillance as well as the adequacy of oxygenation and CO_2 clearance (see Mechanical Ventilation Chap. 22). When conventional ventilation and oxygenation approaches fail to meet patient needs, rescue approaches are required.

Rescue approaches include changing the mode of ventilation, adding a short course of neuromuscular blockade, evaluating for unanticipated intra-abdominal hypertension, as well as extracorporeal membrane oxygenation. With regard to mode changes, both pressure-controlled ventilation (PCV) with prolonged inspiratory

time and inverse ratio (IRV) ventilation have been well utilized to pursue salvage. More recently, airway pressure release ventilation (APRV) has been increasingly leveraged to address refractory hypoxemia more than refractory hypercarbia [20]. Both PCV and APRV hew to lung protective ventilation tenets. Recent APRV meta-analysis data coupled with a recent randomized controlled trial in ARDS patients suggests improved outcome for both mortality and secondary outcome endpoints [21, 22]. APRV has not been specifically evaluated in the context of PBLI but offers a putative superiority compared to PCV or PCV-IRV based upon its efficacy in achieving pulmonary recruitment. Critics offer three detractors to APRV. First, there is a theoretical but unproven risk of increased air embolism during the high-pressure period. Second, there is the potential for increased intratidal shear at recruitment close to total lung capacity as gas release occurs at high flow rate and over a short, often sub-second time frame; note is made of substantial controversy around this potential event based on animal data [23, 24]. Third, APRV settings may be less familiar to practitioners rendering prescription manipulation potentially more difficult. Since ARPV is simply modified CPAP, a small study on a blast lung injury simulator suggests acute effective management with ambient air from continuous positive airway pressure (CPAP) [25].

High-frequency oscillatory ventilation (HFOV) is another mode of IMV that may mitigate over distension and reduce alveolar damage and has been explored in the context of ARDS. HFOV's current role in managing patients with ARDS is quite uncommon due to a lack of efficacy compared to other modes and lack of mortality benefit and is therefore not recommended outside of clinical trials [26, 27]. However, HFOV is difficult to manage, is often limited by reduced CO_2 clearance compared to oxygenation, and is problematic for patient transport.

When a patient's oxygenation and/or ventilation needs are unable to be met with IMV, extracorporeal membrane oxygenation (ECMO) offers a treatment alternative in critically injured patients [28]. Multiple case reports and case series document successful ECMO rescue for combat casualties and provides an alternative strategy for managing patients with severe PBLI not responding to more conventional therapies [29, 30]. Previously tied to systemic therapeutic anticoagulation, ECMO may proceed without systemic anticoagulation for limited time periods that would be relevant for improving the risk profile for patients with PBLI [29–34].

Pulmonary Aspiration

Pulmonary aspiration is a well-described process that is associated with underlying clinical conditions that vary with age as well as acute conditions that impact mental status and airway protective reflex integrity. Intense research surrounds efforts to reduce aspiration risk in the perioperative setting in particular [35]. Particularly relevant to blast injury is that injuries caused by the variety of explosive injury mechanisms also often result in altered mental status making such patients at high risk for aspiration and subsequent aspiration pneumonia. While the typical aspiration encountered in acute care facilities drives therapy for gastric acid (a.k.a. Mendelson's syndrome), oral flora, and GI track contents (with macroaspiration), victims of blast injury must be evaluated for an additional component [36]. The blast wind may force dirt, debris, and quinary products (radiation, biologics) into the airway—or may suspend them in the air so that they are inhaled—and may contribute to complications from aspiration in the setting of blast injuries [16, 37].

Therefore, blast injury victims who have evidence of macroaspiration including airway debris on inspection during airway control or volume loss on plain chest radiography should undergo bronchoscopic evaluation to accomplish two goals: (1) debris clearance and (2) directed specimen acquisition for therapeutic agent guidance [38]. Empiric therapy may hew to existing guidelines as an initial treatment approach and should start with coverage for community-acquired pathogens (Table 29.2) [39]. For those who

Table 29.2 Empiric antibiotic therapy for aspiration pneumonia [36, 39]

Community acquired without risk for multidrug-resistant pathogens	Hospital acquired or risk for multidrug-resistant pathogens
Ampicillin–subactam 1.5–3 g every 6 h or Amoxicillin–clavulanate 875 mg twice daily or Levofloxacin 750 mg daily or Moxifloxacin 400 mg daily	Piperacillin–tazobactam 3.376 g every 6 h or Cefepime 2 g every 8–12 h or Imipenem 1 g every 8 h or Meropenem 1 g every 8 h or Plus Vancomycin 15 mg/kg every 12 h or Linezolid 600 mg every 12 h

develop pneumonia later in their management course, nosocomial pathogens—especially those with a multidrug resistance (MDR) profile–are the dominant concern and are outside of the scope of this review [40]. In both the circumstances, every effort should be made to obtain cultures prior to initiating antibiotic therapy. Furthermore, while a broad spectrum approach is appropriate, culture data should define a tailored approach that deescalates the spectrum of pathogens covered to address only those identified leveraging an antimicrobial stewardship approach [41]. De-escalation helps decrease the selection pressure for MDR pathogens, preserves the utility of high-risk antimicrobial classes (i.e., those with high utilization rate or high potential to induce resistance such as extended spectrum beta-lactamase production), and helps reduce the need for intensive infection control practices.

Military members who sustain blast injury are at specific risk of aspirating bacteria or fungi that are not commonly encountered in US civilian practice. Therefore, empiric therapy should also take into account the location in which injury occurred. For instance, during certain seasons, the Mali seasonal dust storms bear a well-known fungus (*Aspergillus* spp.) but also two unique fungi, *Cladosporium* spp. (generally non-toxic) and *Alternaria* spp. (responsible for invasive infection) [42, 43]. Additionally, in closed space explosions, building materials including, but not limited to, insulation, plastics, and latex debris may become airborne leading to mucosal inflammation or toxicity. It is essential to recall that those with latex allergy may suffer anaphylaxis or what appears to be severe asthma upon aerosolized exposure, especially in a vulnerable airway that manifests blast lung injury [44].

Survival Concerns

Many with isolated PBLI completely recover principally related to absent comorbidities and absent concomitant injury [45]. However, the influence of preexisting pulmonary disease or other comorbidities will predictably negatively influence outcome in the civilian setting and presents a sharp contrast with the otherwise young and healthy military combatant. Comorbid conditions—especially preexisting pulmonary disease—as well as immune competency altering therapies, and frailty will all enhance mortality as well as morbidity rates after blast injury. Due to complex injury care, nonresolving organ dysfunction, complication management, or chronic critical illness multiply injured patients often require prolonged ICU care. Survivors of lengthy ICU care are at high risk of the post-intensive care syndrome (PICS) [46, 47]. After injury, there is substantial overlap with features of the post-traumatic stress disorder as well [46]. Survivors, family members, and primary care clinicians who provide after-care should be specifically counseled regarding PICS to facilitate early identification and remediation (Fig. 29.5).

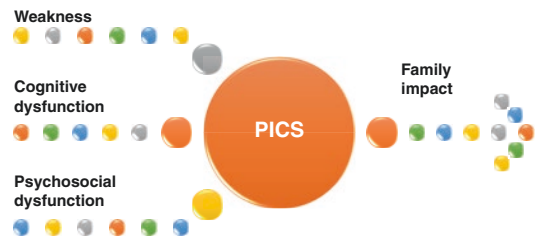

Fig. 29.5 Key features of PICS. This figures depicts the key features of the Post-Intensive Care Syndrome (PICS) that may affect ICU survivors, and of which patients, families, and primary care clinicians should be aware. (Original figure by LJ Kaplan)

The transition out of the ICU appears to be a particularly useful period to identify those at-risk or to begin interventions aimed at mitigating PICS in ICU survivors [48]. While there are specific pulmonary rehabilitation centers that are well established, post-ICU clinics are in their relative infancy. Therefore, it is essential that ICU clinicians communicate concerns regarding PICS to caregivers who will support those at-risk for PICS after discharge from the acute care, long-term acute care, or rehabilitation facility [49]. Of note, and particularly relevant for those who staff trauma clinics, PICS may impact family members as well, offering another opportunity for evaluation and potential intervention [50].

Conclusion

Blast injuries span a vast spectrum of injury types and mechanisms including most notably blast lung injury. The injury profiles may be readily understood in terms of explosion physics and its impact on organs, especially those that are gas filled, and at locations where there are gas and fluid interfaces. Relatedly, open air and closed space explosions bear different implications for human injury and may guide injury interrogation. Management depends on the specific injury, but the need for invasive mechanical ventilation portends a higher mortality related to injury severity. Given the dynamics of explosions, aerosolization or forcible airway entry by atypical agents should be considered and may influence the genesis or progression of aspiration pneumonitis or aspiration pneumonia. Since those who survive blast injury may require complex care within an ICU, they are at high-risk for the post-intensive care syndrome as well as post-traumatic stress disorder related to the index injury. After care is as important as acute care in supporting complete recovery and may leverage family members and primary care clinicians as key members of the care team. Trauma clinic, in particular, represents a unique opportunity to screen for PICS and to initiate focused rehabilitation.

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Thoracic Crush Injury After Natural or Man-Made Disasters

30

Alexandria Robbins, Matthew Robertson, and Gregory Beilman

Crush Injury and Syndrome

Crush Injury

Crush injury results from sustained or repeated forceful compression between two objects [1]. Crush injuries most frequently occur secondary to entrapment under rubble or debris after buildings collapse [2]. Yet, there are many mechanisms that can lead to crush injury including motor vehicle crash, natural disasters such as earthquakes, mass crowd stampedes, severe blunt assaults, as well as industrial, mining, construction, or agricultural injuries [3, 4]. Crush injury can also occur in patients with altered mental status with prolonged immobility after a fall where body weight can create sufficient pressure to establish injury [3].

Crush Syndrome

Crush syndrome describes the series of pathophysiologic events that result from widespread muscle injury which can occur during crush injury. Typically, crush syndrome requires 4–6 hours of direct compression to develop—but with severe pressure it can occur within an hour [5]. This phenomenon was initially characterized during the world wars of the first half of the twentieth century. More recently, post-earthquake care has led to the most comprehensive study of the clinical sequelae and treatment regimens for crush syndrome. The incidence is estimated to range from 2% to 40% in crush injuries but varies by type of disaster [6]. Regarding specific mechanisms of injury, estimates include 3–20% of earthquake victims, and 40% of multi-story building collapse survivors develop crush syndrome [5]. In mass disasters, crush syndrome is the second most common cause of death behind direct impact [1]. A summary of the timing and causes of mortality associated with crush syndrome are presented in Fig. 30.1.

Pathophysiology (Fig. 30.2)

During a crush event, muscle damage occurs through two primary mechanisms: direct muscle compression and ischemia/reperfusion injury. First, direct mechanical compression of the sarcolemma opens stretch-activated channels in the cell membrane causing an intracellular influx of sodium and calcium as they flow down their electrochemical gradients. This leads to cellular swelling as water is drawn in with solutes. The

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increased cytoplasmic calcium concentration activates a variety of cellular mechanisms that inhibit cellular respiration and degrade cellular proteins and membranes. This creates an ongoing cycle of muscle damage as decreased adenosine triphosphate (ATP) production depletes cellular energy stores, further decreasing the cell's ability to maintain proper electrochemical gradients through Na/K ATPase activity [7].

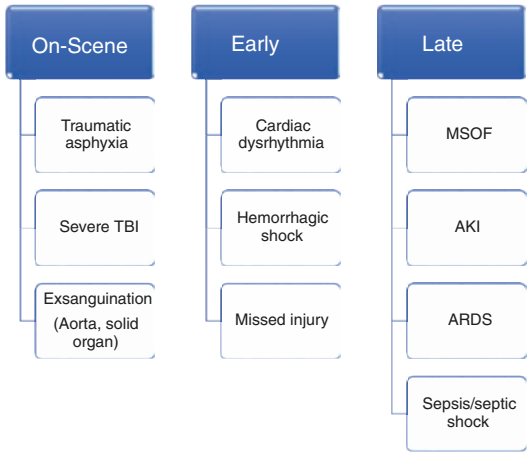

Fig. 30.1 Key on-scene, early and late causes of mortality after crush injury

In addition to the injury from direct impact, damage to soft tissues including muscles or nerves can occur from ischemia related to sustained pressure above diastolic pressure [4]. Crush victims may remain trapped for extended periods and suffer ischemic injury. Without prior mechanical injury, muscle may tolerate 2 h of ischemia without untoward sequelae. Irreversible damage begins after 4 h while tissue death is common by 6 h. Direct injury increases the susceptibility to ischemia. While ischemic, the myocyte switches to anaerobic respiration which decreases the availability of ATP, degrading the cell's ability to adequately sustain physiologic electrochemical gradients [7]. Arterial inflow inadequacy (crush, arterial disruption, intimal flap, thrombosis) is compounded by concomitant venous outflow obstruction which leads to extremity edema but also local increases in ischemic metabolites including acids such as lactate [4]. All of these events precede rescue and resuscitation. Special note is made of those who are

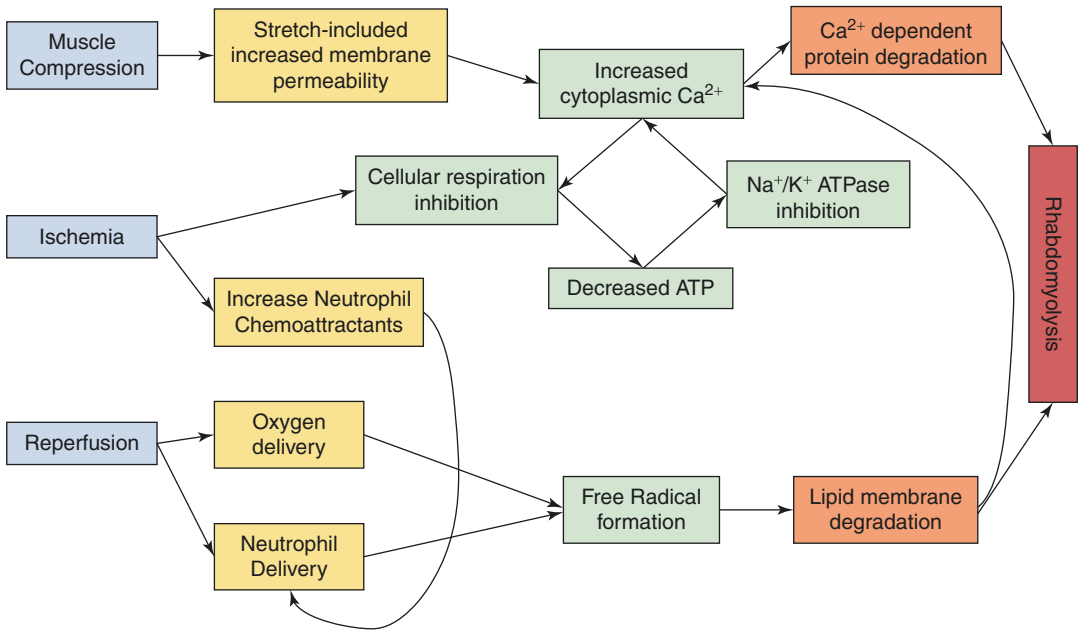

Fig. 30.2 Pathophysiology of rhabdomyolysis (Adapted from Malinsky 2004)

injured and entrapped with an extremity or body cavity that remains compressed until extrication.

Upon extrication and relief of compression—an event usually accompanied by fluid resuscitation—oxygenated reperfusion occurs and generates highly reactive oxygen-derived free radicals (aka toxic oxygen metabolites, TOMs). Such TOMs include but are not limited to singlet oxygen, hypochlorous acid, superoxide, and the final metabolite of degradation pathways, peroxide. TOMs attack membranes in a nonselective fashion, and reperfused tissue is at high risk due to injuries from ischemia and direct compression. Sarcolemmal disruption leads to maladaptive increases in cytoplasmic calcium concentration further. If sarcolemmal damage is sufficiently severe, myocyte lysis releases intracellular contents are released into both the local environment and the systemic circulation. Myocyte lysis also releases myoglobin, urate, phosphate, and potassium leading to well-chronicled sequelae of crush injury [3, 8]. Laboratory findings include hyperkalemia, hypocalcemia, hyperphosphatemia, and acidosis. Surviving myocytes support neutrophil trafficking to sites of injury increasing local damage from activated cell-mediated immune mechanisms [7]. Capillary leak is common and leads to extravascular salt, solute, and water excess that adversely influences lymphatic drainage.

The impact of crush injury may also impact other organ systems, most notably influencing renal function, and identified as acute kidney injury and defining the condition known as rhabdomyolysis. Figure 30.1 displays the overall pathophysiology of crush-induced rhabdomyolysis which leads to crush syndrome. When more than one organ system is adversely impacted, crush syndrome is present and bears some similarities to multi-system organ failure (MSOF) in this way. When persistent, MSOF can also be known as nonresolving organ dysfunction syndrome (nRODS). Regardless, those with crush syndrome commonly require high-intensity critical care including organ support techniques including but not limited to invasive mechanical ventilation, extracorporeal renal support, or extracorporeal membrane oxygenation.

On-Scene Management Issues

Trauma Asphyxiation

Direct thorax or upper abdominal crush injury can lead to traumatic asphyxia, also known as Perthes syndrome [2]. This can occur result from heavy object compression or during crowd crush/stampede events with repeated compression [4]. Chest excursion limitations prevent gas exchange and increased intrathoracic pressure compresses the superior vena cava and depresses cardiac performance [2, 4]. Patients with this syndrome typically demonstrate cervicofacial cyanosis, neurologic symptoms ranging from agitation to depressed level of consciousness, subconjunctival hemorrhage, and petechiae of the head, neck, and chest from increased thoracic pressure and forceful inspiration attempts against a closed glottis [4]. Other associated injuries include anoxic or hypoxic brain injury, thoracic cage fracture, vertebral fracture, and solid organ lacerations [4]. In some, death results within minutes of syndrome onset establishing a narrow window of opportunity for recognition and rescue.

Head and Traumatic Brain Injury

Cranial sheath, vault, and content injury is another cause of immediate mortality. In post-earthquake analyses, up to one third of immediate mortalities result from traumatic head and brain injury [9]. Not all crush-related cranial injuries are immediately life-threatening, but many have the potential to lead to major morbidity with a lesser frequency of mortality. Indeed, most earthquake-associated cranial injuries are mild-to-moderate (85%) and include concussions, superficial contusions, as well as scalp lacerations. It is estimated that up to 15% of earthquake-related cranial injuries trauma include an intracranial component with 34% of those requiring surgery management [9]. External hemorrhage control, resuscitation to euvolemia, prompt airway management, and avoidance of

secondary brain injuries are all appropriate to support optimal outcome.

Prolonged Extraction and Confined Space Medicine

Prolonged extraction times may trigger the need to utilize confined space medicine (CSM), as patients—or even a single patient—are treated while entrapped [4]. This is a particularly hazardous form of medical care that requires specialized training and benefits from coordination through an incident command center [4]. Airway management may require atypical positioning with the aid of oxygen and airway adjunctions from oral or nasal airways but may also include placing a surgical airway [4]. Inhalation injury from combustibles, hot gases, or dust may inform imperatives in airway management including the timing of artificial airway placement [4]. Finally, chest decompression may be required to manage space occupying lesions that exert intrathoracic compression such as pneumothorax or hemothorax related to bony, soft tissue or parenchymal injury, or hemorrhage [4].

Mortality and extrication time are linked, as is the likelihood of developing acute crush syndrome with a compression time greater than 1 h [2, 4]. The care in the immediate period after extrication is vital to avoid conditions colloquially referred to as "smiling death" or the "grateful dead syndrome." The exuberance patients experience once they are extricated—often having been buried under rubble—may not lead to survival. Instead, they may succumb to the ravages of reperfusion, including cardiovascular failure, despite being "rescued" [2].

The cardiovascular system is disadvantaged by volume shifts and cardiotoxicity from the significant metabolic acidosis and electrolyte burden that floods the systemic circulation during reperfusion [3, 5]. Potassium released from dead and injured muscles can cause significant hyperkalemia which can be exacerbated by acute kidney injury. Additionally, the myocardium may be more susceptible to arrythmias due to systemic hypocalcemia as well as an increase in endogenous catecholamine tone. To monitor these complications, continuous cardiac monitoring is recommended in CSM and should be emplaced early [5]. Evidence of hyperkalemia on electrocardiogram should prompt aggressive management including plasma dilution, intracellular displacement, conduction stability support, and definitive elimination [4, 5]. Hypocalcemia should prompt intravenous calcium supplementation and may be recognized by physical examination signs of hypocalcemia or via point-of-care assessment in the field [5].

Hypovolemic shock can occur secondary to both internal and external hemorrhage. Interstitial fluid loss across capillary beds exacerbates evolving or established hypovolemia and appears to be a process that is most prominent in the initial hours after injury [3]. Therefore, intravenous fluid resuscitation should begin on-scene and continue through extrication to definitive facility arrival [5]. Several different treatment algorithms have been proposed with variations in fluid composition, rates of administration, adjunctive treatments, and clinical endpoints [3, 7, 10, 11]; no single approach is clearly superior. General principles include obtaining large bore intravenous access and providing initial resuscitation with isotonic crystalloid solution. There is some suggestion that a balanced salt solution may exert a beneficial influence by reducing the likelihood of inducing hyperchloremic metabolic acidosis. Fluid titration to abrogated hypovolemia may be informed by urine flow, noninvasive assessments such as Point-Of-Care-Ultra-Sound (POCUS), or invasive modalities. Prior concerns regarding the use of lactated Ringer's solution have been debunked in part by noting that the infusion of a solution with a potassium concentration less than the fluid into which it is being infused predictably decreases—not increases—the resultant potassium concentration [12]. Avoiding precipitation of solute within glomeruli or tubules has been a long-standing concern. Previously routinely prescribed, alkalinization may be best reserved for those who produce urine but at a rate less than desired (<1.5 mL/kg/h in some studies and

300 mL/h in others). Patients who are anuric are generally not aided by alkalinization but instead benefit from intermittent or continuous dialysis to manage electrolyte abnormalities or life-threatening acidosis. Those with robust urine flow appear to not benefit from alkalinization.

Facility Care

Initial Stabilization

Advanced trauma life support provides a universal platform from which to pursue initial stabilization. When performing primary and secondary surveys in the population, one should consider the possibility of hemorrhage, solid organ damage, or spinal injury [3]. Some clinical findings that point to thoracic crush injury include acute respiratory distress, thoracic pain, paradoxical respiratory movements, cough, hemoptysis, dyspnea, hypoxia, or hypercarbia [13]. This is not an exhaustive list but instead lists common findings on presentation to the Emergency Department (ED).

If not obtained in the field, intravenous or intra-osseus access should be obtained to support plasma volume expansion [3, 14]. Routine monitors should be placed as for any trauma resuscitation including an indwelling urinary catheter. Besides the routine laboratory profile, serum CPK and creatinine, as well as urine myoglobin and creatine kinase should be obtained [3, 4]. Routine chest plain radiography is useful to screen for life-threatening injury and when abnormal may be further refined using computed tomography [13]. CT scans should be obtained with IV contrast when there is concern for arterial injury, and appear safe with regard to contrast-associated acute kidney in unselected injured and ED patients [15]. Some concerns regarding the safety of iodinated radiocontrast have been articulated particularly in those who have required prolonged extrication. No high-quality data in this unique patient population guides a uniform approach instead supporting individualized care.

Chest-Specific Crush Injury

Thoracic injury occurs in approximately 10–15% of all injuries, with an associated mortality rate ranging from 8% to 33% [13, 16]. The four most common causes of chest injury include motor vehicle crash, falls, blunt or penetrating assault, and occupational injury [16]. Mass casualty events such as earthquakes disclose that from 8% to 16% of hospitalized patients require treatment for lung or thoracic cage injury [13]. Indeed, thoracic crush injuries are well studied in survivors of earthquakes. When comparing earthquake-related thoracic trauma to other causes, earthquake-related thoracic trauma patients are more likely to be older and female [17]. Some of the unique attributes of crush chest trauma are highlighted in Table 30.1.

Rib Fractures

Rib fracture is present in 27–36% of earthquake-injured patients with thoracic trauma [13]. Dong et al. reviewed 215 patients who presented with crush thoracic trauma and severe enough symptoms to prompt multidetector chest CT evaluation after the 2008 earthquake in Sichuan, China. They reported that 70% of patients presented with a thoracic fracture, most commonly impacting the ribs (67% of all patients). Notably, the

Table 30.1 Features specific to thoracic crush injury

Features specific to crush type thoracic trauma
Rib fractures
More commonly bilateral
Involve first and second rib
Multiple
Flail chest
Pulmonary parenchyma
Contusion more common than laceration
Thoracic cage
More common T-spine fracture
Spinal cord injury with better neurologic outcomes
Sternum/clavicle fracture more common
Vascular/cardiothoracic
Better outcomes in acute aortic traumatic injury

average number of rib fractures per patient was 11 (range 1–21), and 46% were bilateral. This is significant because of the known correlation between the number of ribs fractured and the severity of injury. Indeed, three rib fractures drove the greatest prognostic difference compared to no fractures, with every subsequent fracture further increasing the mortality rate [13]. The incidence of greater than seven rib fractures was higher than in those reported other studies which prompted the authors to postulate that earthquake victims may fall and be trapped in a prone position with persistent bilateral compression resulting in different kinetic energy transfer compared to other causes of thoracic injury [13]. While putatively attractive, this notion has yet to be formally studied. Nonetheless, the most common locations for fracture were posterolateral in ribs 3–7, but 49% of patients also had first or second rib fractures. Similar to the number of fractures, first and second rib fractures imply severe injury and raise the potential for colocated vascular injuries [13]. Finally, 32% of patients with rib fractures were complicated by concomitant flail chest. Prompt recognition and rescue is essential as flail chest bears a 10–20% mortality rate [13].

Both unilateral and bilateral fractures occur with crush mechanisms, with bilaterality and flail chest seemingly prominent features of earthquake-related injury [17]. Another study compared all rib fractures from different causes of blunt chest trauma. They report that the "squashing/burying accident group" had more rib fractures per patient and more flail chest (23%) and required more thoracic drainage compared to other types of injury. This group also had the most heterogeneous pattern of rib fracture location which the authors attributed to complex loading mechanisms from more than one direction [18].

Nonrib Thoracic Cage Fractures

Other bony elements of the thoracic cage are also at risk of injury. Earthquake victims who required emergency care were noted to sustain fractures of the vertebral body (21%), sternum (6%), scapula (16%), and clavicle (10%) less frequently than they sustained rib fractures [13]. Vertebral body fractures spanned T3 to L2, and most (44/46) patients had concomitant rib fractures. Vertebral fractures most commonly occurred at T3 through T10 (47%), followed by T11–12 (27%) and L1–2 (26%). This pattern implies a high-energy injury profile as the thoracic spine is stiffer in sagittal and lateral flexion-extension relative to the lumbar spine [13]. Of the vertebral body fractures, one-third also had associated spinal stenosis. There were transverse processes or spinous processes fractures noted in 22% of patients as well (47/215) [13]. Nonetheless, in a 2011 follow-up study comparing earthquakes to other causes of thoracic injury, no statistically significant difference in the incidence of T1-T10 vertebral fractures was noted [17]. Another study of earthquake victims noted that multilevel thoracolumbar spinal injuries were common (22–30%). Burst fractures predominated (49–55%) followed by nearly equal prevalence of fracture dislocations, compression and nondisplaced fracture [9]. From a prognostication perspective, thoracic fractures had worse outcomes than lumbar fractures perhaps related to associated injuries and associated respiratory failure [9]. Relatedly, patients with spinal cord injury related to earthquake injury demonstrate better neurologic recovery (33%) relative to other causes (2–5%) which may be related to the kinetic energy transferred to earthquake survivors in comparison to MVCs or penetrating injury [9].

Sternal fractures were also noted in the Dong et al. study, with 58% discovered in the manubrium and half were also associated with anterior rib fractures [13]. All patients with scapula or clavicle fractures also had rib fractures and 48% of these patients had concurrent flail chest—a substantial injury complex in terms of acute injury but also rehabilitation. In both scapula and clavicle fractures, 9% of each were bilateral, less common than with rib fractures. In general, sternal, scapular, and clavicular fractures are uncommon in thoracic trauma associated with crush injuries—even while such fractures may be more commonly noted after falls from height and high-speed MVCs.

Pleural and Pulmonary Parenchymal Injury

Unsurprisingly, nonbony thorax injuries are also common with thoracic crush injury including pulmonary contusions (53% with 3% of such patients demonstrating pulmonary laceration), pneumothorax (4%), hemothorax (43%), and hemopneumothorax (21%) [13]. Most patients (86%) with parenchymal injury had associated rib fractures, but the presence of a parenchymal injury did not appear to influence the mortality related to rib fractures [13]. Pulmonary parenchymal injury occurs with blunt injury from contact with sharp elements of displaced fractured ribs, impact-related shearing forces around points of fixation, as well as direct and contra-coup compression—especially against a closed glottis. Data from earthquakes is subject to survivor bias in that those with severe injury are more likely to die on-scene rendering the impact of pulmonary laceration on survival or morbidity less clear than desired. Regardless, when compared with non-earthquake-related injuries, pulmonary parenchymal injury appears more common (54% vs. 37%) [19].

Most, but not all patients with pleural injury demonstrate concurrent rib fracture as well as pneumothorax, hemothorax, or combined hemopneumothorax [13, 16–18]. Slightly more than half (52%) of those with a hemopneumothorax demonstrated moderate or large amounts of pleural blood. Earthquake care reports document pleural drainage rates spanning 2–52% of patients will benefit from pleural drainage [9, 13]. In one study, tension pneumothorax was noted in 4%, another observation that may reflect survivor bias [13].

Other Thoracic Injuries

While all of the thoracic contents are at risk, aortic injury seems less common and tends to demonstrate better survival compared to noncrush aortic injury patients who presented after a deceleration event (fall from height or MVC) [19]. They highlight the differences in mechanism of injury with shearing stress to the aorta in deceleration and direct load with medial tears in crush injury. There is a large impact of survivor bias in the aortic data in that major injury leads to rapid death from exsanguination. Therefore, crush injury patients presented with low-severity injury type (vs. 44% in noncrush), required only medical management (vs. 57% in noncrush) and demonstrated no in-hospital mortality (vs. 26% in noncrush). The authors emphasized that there can be rapid progression of traumatic aortic injury later in recovery, thus serial follow-up imaging is required in these patients [19]. Concomitant esophageal injury was not described. Other anticipated injuries such as diaphragmatic hernia are rare, while soft tissue contusions and superficial lacerations are common.

Acute Respiratory Failure

A study of more than 1800 patients who were transferred to a university hospital after the Sichuan earthquake of 2008 found that 10% of patients had thoracic trauma [16]. Notably, 90% of these thoracic trauma patients had a crush injury by a heavy object and 85% had more than one organ system involved. Of the thoracic trauma patients, 21% went on to develop respiratory failure requiring invasive mechanical ventilation. The average duration of mechanical ventilation was 19 days, with 71% requiring tracheostomy for prolonged respiratory failure management. Flail chest, pulmonary contusion, and crush syndrome were all significantly more likely to be associated with respiratory failure. The role of noninvasive ventilation or high-flow nasal cannula oxygen therapy in managing thoracic crush injury remains underexplored while their efficacy in noncrush thoracic injury has been well investigated [20, 21].

Secondary Complications Related to Crush Syndrome

Acute Kidney Injury and Rhabdomyolysis

Acute kidney injury (AKI) precipitated by crush injury has long been recognized as one of the most serious complications that affects crush vic-

tims who survive their initial injuries [22]. Recent advances in our understanding of the pathophysiology of AKI underscores the diverse etiologies that can lead to AKI including most notably, sepsis. How those drivers impact cell cycle physiology, immune cell function, and interorgan cross-talk are increasingly clear [15, 23–26]. Postcrush injury AKI is multifactorial and includes but is not limited to decreased renal perfusion secondary to hypovolemia as well as renal toxicity from the products of muscle breakdown including myoglobin, urate, and phosphate. Acidosis also seems to drive renal arteriolar vasoconstriction and worsens low flow-induced AKI. Importantly, exertional rhabdomyolysis is different from postcrush AKI in that recovery to preexertion serum creatinine baseline is routine without intervention—an observation that merits specific investigation [27].

Myoglobin is a heme-containing protein and is thought to play a major role in renal injury secondary to crush syndrome. At low blood concentrations, myoglobin is primarily bound to haptoglobin and is cleared via the reticuloendothelial system [7]. As levels increase, unbound myoglobin is filtered in the kidney and is not reabsorbed. High levels of myoglobin in the urine lead to the characteristic "tea-colored" urine seen in patients with rhabdomyolysis. Myoglobin, urate, and phosphate may precipitate in the renal tubules forming casts, resulting in tubular obstruction, an event that is promoted by both aciduria and urinary concentration. Additionally, myoglobin can directly induce renal injury via lipid peroxidation through the action of unbound iron as it interfaces with oxygen and induces ferroptosis [7, 28, 29]. A summary of this pathophysiology is demonstrated in Fig. 30.3.

Elderly patients with crush injury demonstrate a higher rate of AKI after earthquake injury compared to younger patients [30]. This is unsurprising given the age-related changes in glomerular

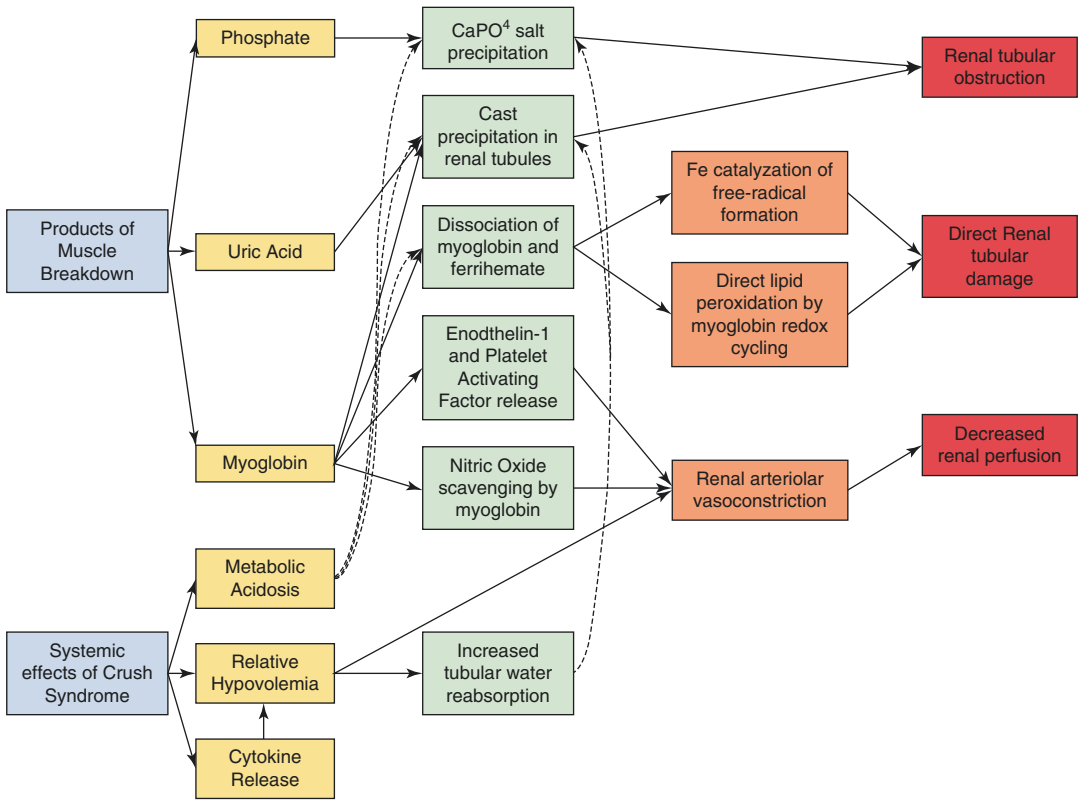

Fig. 30.3 Post-crush injury AKI drivers. (Adapted from Sever 2011 and Bartels 2011)

filtration rate, solute clearance, and comorbid conditions that impact overall renal function including atherosclerotic disease as well as diabetes mellitus [31]. Therefore, it is imperative to understand how to prevent AKI from occurring to the extent possible, recognizing that those who have sustained crush injury—especially those who have required prolonged extrication—may have already sustained AKI prior to rescue. Such patients may demonstrate evidence of subclinical AKI that will only be discernable by biomarker assessment [32, 33].

Management of Acute Kidney Injury

Abrogation of hypovolemia is essential and may be guided using noninvasive or invasive methods depending on local resources. Crystalloid resuscitation provides the mainstay of management to restore plasma circulating volume to correct hypoperfusion and help clear metabolites of muscle injury. Recognizing that crush injury patients are highly likely to have already been launched on the path to clinically evident AKI, linking management with the precise stage of AKI becomes therapeutically relevant. Such an approach—stage-based management—has been articulated by the Kidney Disease Improving Global Outcomes group [34]. Importantly, the approach is comprehensive guiding fluid management, level of care, pharmacologic dose adjustment, nephrotoxin avoidance, as well as the use of renal support techniques. Early nephrology consultation is important for this vulnerable patient population triggered by an increase in serum creatine as little as 0.3 mg/dL especially when accompanied by oliguria. Despite active therapy, severe oliguria, anuria, severe acidosis, hyperkalemia, and other electrolyte derangements including severe hyponatremia may drive the need for an extracorporeal support technique. The need for renal support may be informed by the results from a furosemide stress test with nonresponders appearing to demonstrate acute need for a renal support technique [35]. Recent data does not find a selective advantage with intermittent compared to continuous renal support, with modality selection driven by the need for concomitant vasopressor infusion to support mean arterial pressure goals [36]. Furthermore, emerging phenotypes may help guide-specific therapeutic agent selection as well [37].

Compartment Syndrome

The most common musculoskeletal injuries in crush injury include lacerations, fractures, and soft tissue damage [9]. Muscle damage from direct crush injury, vascular injury, and ischemia-reperfusion can impact compartment pressures, with pressures reaching up to 240 mmHg in crushed compartments [6]. Compartment syndrome may be defined as an intracompartment pressure >30 mmHg for 8 h or if the difference between the intracompartment pressure (delta pressure) and the diastolic pressure is <30 mmHg [38]. The skeletal muscle of the forearm and lower leg surrounded by inelastic fascial sheaths are especially prone [6]. On examination, patients may present with marked pain associated with passive flexion, flaccid paralysis, decreased sensation, and decreased or absent pulses if the compartment pressure equals the diastolic pressure [6]. Therefore, some advance that the diagnosis of compartment syndrome should be clinically driven and does not require invasive compartment pressure measurement. However, in the presence of traumatic brain injury, stroke, encephalopathy, spinal cord injury, or other conditions that derange the fidelity of the clinical examination, compartment pressure measurement is essential. Relevantly, noninvasive techniques (near infrared spectroscopy perfusion assessment, tissue mechanical property measurement devices) have arisen that can help establish or refute compartment hypertension and syndrome that may be particularly useful in those with coagulopathy or in setting where the skill to invasively measure compartment pressures is absent [39].

Early identification and treatment of compartment syndrome are essential to preserve tissue viability including nerves and leverages fasciotomy to relieve untoward pressure. On occasion,

hematoma evacuation or tissue debridement occurs at the same time. Delayed fasciotomy is associated with accelerated morbidity, including crush syndrome, neurapraxia, and limb loss [4, 40]. Prophylactic fasciotomy in patients with crush injury without intracompartment hypertension is not recommended given the risk for hemorrhage and surgical site infection which can lead to soft tissue infection and potentially amputation [4, 6]. Late fasciotomy where there is evidence of tissue necrosis does not enhance outcome and is not recommended [40]. Relatedly, in-field amputation is generally not recommended outside of the need to extricate an individual with an entrapped limb that cannot be freed [9, 41].

Other Complications

Crush injury survivors are at risk for a variety of complications during their ICU stay as well as during convalescence. Critical illness is frequently associated with sepsis, septic shock, acute respiratory distress syndrome, organ failure, and disorders of coagulation. Injury triggers an endotheliopathy that is related to the above untoward sequelae [18, 42]. Advances in resuscitation including hemostatic resuscitation may ameliorate some of the consequences of the endotheliopathy of trauma [43]. A number of guides to managing sepsis, septic shock, and ARDS in particular exist elsewhere, and the interested reader is referred to relevant sections of this book as well as other published works.

Crush injury survivors who depart an acute care facility to either a Long-Term Acute Care Hospital, a rehabilitation center, or home may experience intrusive memories of the index event, depression regarding changes in body habitus after reconstruction (or lack thereof), debridement or amputation, as well as the postintensive care syndrome. Chest wall deformity is common with multiple rib fractures and may be quite prominent in those without substantial body mass. Posttraumatic stress disorder may follow entrapment and has been well described after acute injury and entrapment [44]. Similar findings have been described in firefighters who have become entrapped in buildings, as well as emergency medicine services technicians who respond to disasters [45].

The Postintensive care syndrome (PICS) is increasingly well recognized in survivors of critical illness as well as in their family members (PICS-F). PICS is characterized by derangement in cognition, strength, and psychosocial domains. PICS-F reflects the dysfunction that occurs in family members of patients who have PICS while they provide uncompensated care [46]. Post-ICU clinics appear to be helpful in diagnosing and addressing PICS as do ICU survivor support groups [47, 48]. Survivors of crush injury may be ideal candidates for postacute care support using these approaches [49].

Conclusion

Earthquakes, structure collapse, and compression between immobile heavy objects can create crush injury. Thoracic crush injury is characterized by a variety of injuries to the thoracic cage as well as its contents. Muscle damage management and sequelae are informed by data derived from extremity crush injury care. Remote organ systems may be adversely impacted including, most notably, the renal system. Rhabdomyolysis-induced AKI may be managed using well-established approaches to muscle as well as acid, electrolyte, and muscle degradation product release into the local and systemic circulation. Entrapment and prolonged extrication engender risks to both the patient and the rescuer when confined space medicine is required. Reperfusion injury and restoration of flow after prolonged entrapment may lead to acute cardiovascular collapse. Definitive care typically requires ICU care but may also benefit from operative care. Critically injured patients should be anticipated to be at risk for common sequelae of acute injury and critical illness including sepsis, septic shock, and MSOF. Critical illness survivors should be surveilled for PICS and their family members should be assessed for PICS-F. When available, a post-ICU clinic or post-ICU support group should be engaged to help support recovery.

In summary, thoracic trauma secondary to crush injury is more likely to result in bilateral and multiple rib fractures, flail chest, and other thoracic cage fractures. There is also a higher rate of pulmonary parenchymal injury with prolonged respiratory failure. Fortunately, patients who develop acute traumatic aortic injury or spinal cord injury appear to have better outcomes than other causes of trauma. A unique feature of crush injuries is the potential for prolonged entrapment that requires confined space medicine and may result in the systemic sequelae of direct injury, ischemia, and reperfusion known as crush syndrome. Crush syndrome has both immediate (cardiac dysrhythmias) and delayed complications (acute renal failure) that trauma providers should be able to anticipate and initiate early treatment.

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Toxic Inhalations

31

T. Jacob Lee and Matthew A. Borgman

Introduction

While chest injury typically results from penetrating or blunt trauma, it may also arise from remote injuries or infections leading to the need for pulmonary rescue. Much less commonly, and perhaps principally relevant for industrial accidents, military operations, or urban violent extremism, toxic injury to the pulmonary parenchyma may be driven by toxic inhalation. Toxic smoke and burning sulfur were deployed in warfare more than 4000 years prior in a host of countries including but not limited to China, India, Greece, and Sparta. Incendiary deployment of toxins classically known as "Greek Fire" combined resin pitch sulfur, naphtha, quicklime, and saltpeter into a potent combination that ignites in an aqueous milieu and was used to great advantage in naval warfare. Due to the devastating effects of inhaled toxins, the earliest known accord to limit their use in military conflict was the 1675 Strasbourg agreement that launched the modern era of international toxic regulation. The era of modern chemical warfare began in the industrial revolution when chemical manufacturing facilities produced the variety of agents used during World War 1.

Currently, toxic exposures have evolved in terms of complexity, spectrum of injury, ability to detect potential exposure, and the setting in which one may encounter such exposures. Agents are sometimes classified as toxic industrial chemicals (TIC) or as agents of chemical warfare (CW). However, such a scheme does not include other elements that may lead to toxic exposure. The current umbrella approach to the larger array of substances that can induce injury is known as Chemical, Biologic, Radiologic, Nuclear, and Explosive (CBRN-E) exposures (Fig. 31.1). This chapter will address chemicals that trigger inflammation, chemical irritants elaborated from combustion or explosion, physical and chemical

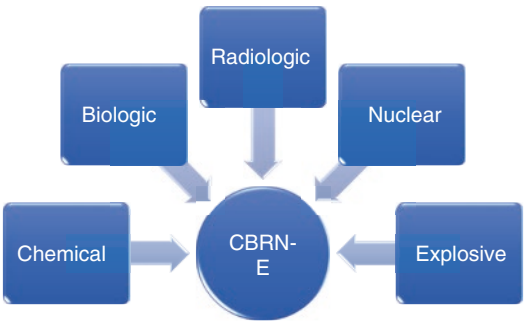

Fig. 31.1 CBRN-E components

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Table 31.1 Summary of toxic exposures and acute therapeutics

Toxic agent	Signs and symptoms	Treatment/antidote
Simple asphyxiants: e.g., carbon dioxide, methane, ethane	Hypoxia	Supportive care with 100% inspired oxygen (FiO_2)
Carbon monoxide	Headache, dizziness, weakness, cherry red lips or skin (severe), normal pulse oximetry	Supportive care with 100% FiO_2, hyperbaric therapy
Cyanide	Tachycardia, seizure, coma, death, lactic acidosis	Sodium/amyl nitrite & sodium thiosulfate OR hydroxocobalamin
Hydrogen sulfide	Rotten egg smell, headache, dizziness, nausea and vomiting, dysrhythmias	Supportive care, possibly sodium nitrite
Methemoglobin	Normal/elevated PaO_2, grayish skin discoloration, chocolate-colored blood	Methylene blue
Irritants: Ammonia, hydrogen fluoride, hydrogen chloride, riot control agents, chlorine, phosgene, nitrogen oxides	Copious secretions, dyspnea, upper and lower airway obstruction, ARDS	Supportive care, remove from offending agent
Sulfur mustard (vesicant)	Loss of smell, rhinorrhea, cough/dyspnea, burns/blisters followed by skin necrosis	Supportive care; burn wound care
Organophosphates/nerve agents	Classic toxidromes of salivation, lacrimation urination, diaphoresis, diarrhea, bronchoconstriction, bradycardia, muscle fasciculations, seizure, coma	Atropinization, pralidoxime, benzodiazepines (for seizures), supportive care
Radiologic dispersal devices	Acute radiation sickness based on exposure dose; fatigue, weakness, nausea, vomiting, death	Decorporation therapy, supportive care

asphyxiants, as well as radiological dispersion devices, resulting in inhalation of radioactive materials (Table 31.1). Understanding the signs and symptoms as well as rescue therapies for each of these domains is relevant for every member of the healthcare team spanning first responders, emergency department personnel, inpatient care clinicians, veterinarians, administrators, as well as family members of impacted patients or dyadic service animals or pets.

Initial Evaluation and Workup

Early identification of the agent of toxicity will enhance intervention specificity and yield the greatest chance of improved outcomes. The precise agent of toxicity may be suggested by analyzing readily collected data including age, exposure or event location, physical examination findings, and laboratory investigations. Unfortunately, such data provides little utility in precisely identifying the etiologic agent in most toxic inhalations. In the remainder of this section, we will review the key elements of the initial history and evaluation that may assist the team in determining the agent of toxicity and its most appropriate management.

Age

Age is perhaps the least reliable element when assessing toxic inhalation. Its primary utility is by defining likely locations in which exposure may have occurred. For example, a 5-year-old child is unlikely to be exposed to TIC outside of the context of an industrial catastrophe or military action. Thus, age is most helpful as an element in database evaluations of outcome rather than as an element within the evolution of acute care.

Location

With toxic inhalations or pulmonary pathology resulting from toxic exposure, the location may

be too broad to lead to suspicion of a specific agent of exposure outside of industrial and other facilities that utilize potentially hazardous chemicals. For individuals who work in such locations, the medical team should rapidly gather information regarding the chemicals utilized at the exposure location. Sites are required by law to have descriptors of such chemicals available as a Material Safety Data Sheet specific for each chemical.

Oxygenation, Partial Pressure of Arterial Oxygen (PaO_2)

Respiratory toxicants often cause hypoxemia due to direct airway or pulmonary injury, or relative displacement of inhaled oxygen. Therefore, hypoxemia, while strongly suggestive of a toxic inhalation, is not a finding specific to a particular agent or exposure. Conversely, normal arterial oxygen saturation in the setting of strong cues for toxic exposure (e.g., fire or explosion, shock, lactic acidosis) may, in fact, be strongly suggestive of several toxicants that should be immediately investigated. Toxic inhalants that do not result in hypoxemia notably include carbon monoxide, cyanide, hydrogen sulfide, as well as agents that result in methemoglobin [1, 2]. Methemoglobinemia is strongly suggested by an invariant oxygen saturation of 85% despite high PaO_2. Several newer noninvasive devices can detect carbon monoxide and methemoglobin via pulse co-oximetry [3]. Regardless, arterial blood gas analysis with co-oximetry should be rapidly obtained to help confirm the causative agent [1, 4, 5].

Respiratory

Tachypnea commonly accompanies all clinically significant toxic inhalations due to bronchospasm, airway inflammation, and alveolar and pulmonary parenchymal edema that may reduce the spontaneous tidal volume. Minute ventilation maintenance requires an increase in respiratory rate to maintain a normal carbon dioxide (CO_2) clearance. When there is shock and lactic acidosis, hyperpnea (increased minute ventilation) is required to clear additional CO_2 or to buffer a decreased pH. Hypoxemia is a powerful catecholamine trigger and will also drive hyperpnea. Any evidence of pulmonary dysfunction on presentation and exam should immediately raise suspicion of an inhalational toxicant. More specifically, bronchospasm and bronchorrhea in particular are associated with organophosphate poisoning, and bronchospasm in isolation is associated with toluene (commonly present in solvents) inhalation.

Cardiac

Hemodynamic lability may ensue after toxic inhalation, and most are accompanied by some degree of tachycardia resulting from increased endogenous catecholamine elaboration after injury. Bradycardia, on the other hand, is well known to result from organophosphate poisoning and is a key feature of the cholinergic toxidrome [6].

Agents of toxic inhalation or radiation exposure do not typically cause arrhythmias. Nonetheless, if EKG changes or arrhythmia is thought due to toxic inhalation, antiarrhythmic agents should be initially avoided due to the potential for unintended and maladaptive effects. Instead, first-line treatments include intravenous sodium bicarbonate to offset induced acidosis and magnesium sulfate (4 g $MgSO_4$) to support maintenance of myocardial conduction stability. These two agents should be accompanied by electrolyte imbalance correction with a focus on repairing concomitant hypokalemia with a goal of a potassium concentration greater than 4 mEq/L to support conduction stability [7].

Ocular Examination

Mydriasis and nystagmus are not associated with a specific inhalational toxidrome. Miosis (pupillary diameter less than 2 mm), however, can be associated with organophosphates [8].

Conjunctival injection may suggest an airborne toxicant as a reflection of aerosolized agent induced inflammation, but is equally nonspecific.

Mental Status and Neurological Examination

Mental status examination is typically of little help with inhalational toxicants, as nearly none primarily affects mental status or causes secondary mental status depression or other disordered consciousness. Organophosphate agents are a notable exception in that they cause mental status depression, seizures, and significant muscular fasciculation [2]. Toluene huffing also causes direct mental status depression, but is a nonspecific finding [9–11].

Temperature

Body temperature alterations are not typically associated with toxic inhalations or radiation exposure. It is worth nothing, though, that hyperthermia resulting from poisoning or overdose should not be treated with antipyretics due to lack of efficacy and must be instead treated with active cooling measures [7].

Skin

The skin examination, particularly coloration, warmth, moisture, and cyanosis, can be very helpful. The presence of cyanosis with a possible slate-gray discoloration is indicative of methemoglobinemia. Additionally, cyanosis and reduced pulse oximetry will be present despite supplemental oxygen therapy and normal arterial partial pressure of oxygen (P_aO_2). Cherry-red coloration of the skin is a well-known feature of severe carbon monoxide poisoning, as may occur with closed space fires. Profuse diaphoresis occurs with clinically significant organophosphate exposure.

Laboratory Analysis

Currently, no urinary or serum analyses exist to detect a specific inhalational toxicant and the clinician must rely on other laboratory data to help discover the specific toxicant, as well as identify sequelae that must be actively managed. Arterial blood gas analysis, with co-oximetry and lactate concentration, in addition to the basic metabolic panel are essential in not just evaluating for inhalational toxidromes, but nearly all toxidromes. While low arterial oxygen pressure may result from pulmonary failure due to inhaled toxicant effects, it is not specific to a particular toxin. Conversely, normal arterial oxygen saturation, in the setting of concern for inhaled toxicant, may suggest exposure to carbon monoxide, cyanide, sulfur containing chemicals (hydrogen sulfide gas), or methemoglobinemia [4, 12]. Blood gas co-oximetry measures levels of oxygenated hemoglobin, deoxygenated hemoglobin, carboxyhemoglobin, and methemoglobin, and is critical to confirm these states.

Of further use is calculation of the arterial oxygen saturation gap, which is the difference between the arterial blood gas calculated oxygen saturation and the arterial blood gas co-oximetry measured percentage of oxygenated hemoglobin. Significant levels of carboxyhemoglobin (carbon monoxide exposure), sulfhemoglobin (sulfur-containing compound exposure), and methemoglobinemia, collectively referred to as dyshemoglobinemias, will typically result in an arterial oxygen saturation gap of greater than 5%. In contrast, poisoning from cyanide exposure does not create such a gap because oxygen binding to hemoglobin remains unaffected [4, 12]. Normal arterial and venous bound hemoglobin values are presented in Fig. 31.2. Arterial blood gas pH may indicate respiratory acidosis with pulmonary failure (acute respiratory distress or acute respiratory failure), but the evaluating team should pay particular attention to concomitant metabolic acidosis as a manifestation of shock as well as toxicant consequence.

Electrolyte abnormalities will not routinely occur with toxic inhalations. However, positive

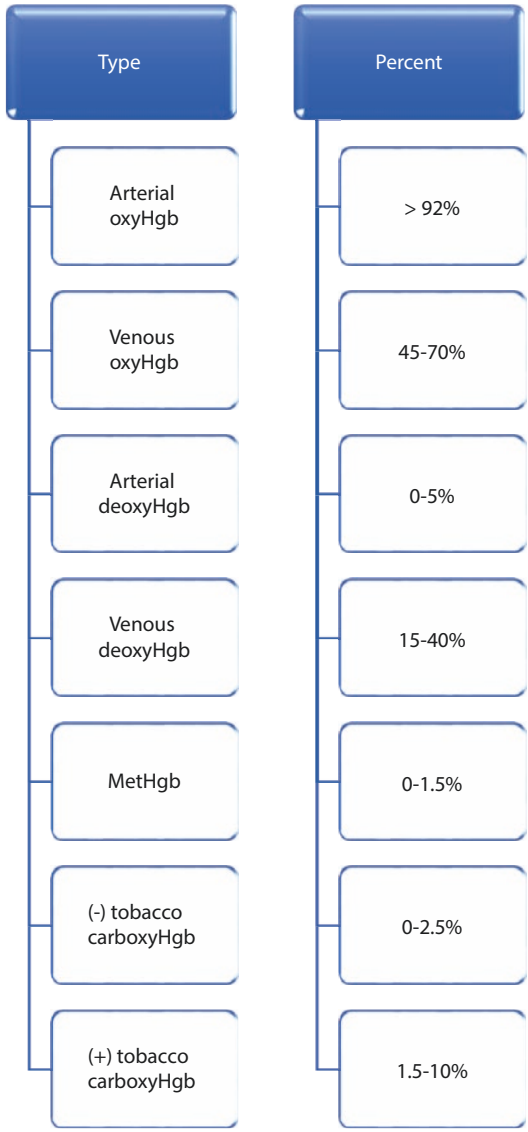

Fig. 31.2 Arterial and venous bound hemoglobin (Hgb)

anion gap acidosis may occur due to shock in the setting of lactic acidosis. Hypoxemic shock with decrease in arterial partial pressure of oxygen and lactic acidosis will be nonspecific and remains associated with a variety of agents causing pulmonary failure. Conversely, lactic acidosis with normal arterial partial pressure of oxygen is suggestive of poisoning from methemoglobin, cyanide, carbon monoxide, or sulfur-containing agents.

Toluene toxicity demonstrates several unique metabolic features. Hypokalemia frequently occurs [11]. Toluene may cause a positive anion gap acidosis or a nongap acidosis. The anion gap acidosis will not be due to lactate elevation, but rather hippuric acid, which is not routinely measured. The nongap acidosis stems from impaired renal elimination of ammonium ions, resulting in renal tubular acidosis [9, 10].

Specific Inhalation Agents of Toxicity

The Swedish physician and scientist, Paracelsus, is often quoted as having stated that "Sola dosis facit venenum"—the dose makes the poison. The complete concise and profound statement, however, reads: "All things are poison, and nothing is without poison, the dosage alone makes it so a thing is not a poison" [13].

Discussions of inhalational agents conveniently group agents based on the mechanism of harm. Prominent examples from each classification are discussed, and the reader can infer similar identification and treatment modalities for the unmentioned chemicals that exhibit similar effects. The relevant classes of agents to be discussed are:

Asphyxiants: simple and systemic (chemical).
Irritants.
Vesicants.
Cholinergic agents.
Radiological dispersal device.

Asphyxiants: Simple

Simple asphyxiants displace inspired oxygen, thereby lowering the inhaled partial pressure of oxygen. According to Dalton's Law, reducing the partial pressure of oxygen, assuming an unchanged total ambient gaseous pressure, will reduce the concentration of oxygen (i.e., the fraction of inspired oxygen (FiO_2)). Simple asphyxiants do not demonstrate further airway or

pulmonary parenchymal injury, an observation that simplifies treatment.

Common examples of simple asphyxiants include carbon dioxide, natural gas, nitrogen, acetylene, helium, hydrogen, and the petroleum-derived gases methane, ethane, propane, and butane. An important physical attribute to consider with asphyxiant gasses is their density compared to air. Gases less dense than air, which include methane, nitrogen, acetylene, helium, and hydrogen, will typically accumulate to dangerous levels only when dispersed within closed, confined spaces with poor ventilation—circumstances that increase the dose of the asphyxiant. Conversely, gases more dense than air include carbon dioxide (used in some fire suppressant systems), natural gas, ethane, propane, and butane. These dense gasses will potentially accumulate in any space, therefore representing an accelerated danger without proper precautions. While most medical care will likely occur away from the scene of injury, ensuring scene safety during rescue should be prioritized and merit-specific training and equipment. Treatment for simple asphyxiants includes removal from exposure, general Basic Life Support (BLS) care, and supportive care utilizing supplemental 100% oxygen (FiO_2 = 1.0) to displace the asphyxiant [1, 2, 14, 15].

Asphyxiants: Systemic (Chemical)

Chemical asphyxiants exert systemic toxic effects that fall into two categories: (1) agents that decrease oxygen carrying capacity and (2) agents that inhibit tissue oxygen utilization [1, 2, 15].

Carbon Monoxide

Carbon monoxide (CO) causes significant toxic effects by decreasing oxygen-carrying capacity and impairing tissue oxygen utilization. CO has increased binding affinity for all heme molecules compared to oxygen, including hemoglobin, myoglobin, fibrinogen, and cytochrome molecules—all with deleterious effects [16, 17]. The affinity of hemoglobin for CO is 200–270 times that of oxygen, thereby displacing oxygen from hemoglobin and forming carboxyhemoglobin. Fetal hemoglobin has even higher affinity for CO, subsequently increasing effects on the developing fetus [14]. Carboxyhemoglobin shifts the oxygen–hemoglobin dissociation curve leftward, which increases the binding of oxygen to hemoglobin, diminishing the release of oxygen to tissue. Myoglobin binding of CO causes further dysfunction of tissue oxygen transport, increasing effects on myoglobin-rich skeletal and cardiac muscle. Binding of CO to mitochondrial cytochrome C oxidase results in anaerobic metabolism increasing toxic effects on more metabolically active organs, such as the heart, liver, kidneys, and brain [16, 17]. Cardio and neurotoxicity are further increased by CO-induced apoptosis [16]. Increased inflammatory system activation due to increased oxidative stress, along with the formation of carboxyhemefibrinogen, results in hypercoagulability and an increased risk of intravascular thrombosis which further impairs end organ microvascular flow and O_2 delivery. United States National Poison Data System (NPDS) statistics describe the significant cumulative toxicity effects of CO. For the years 2016–2018, CO exposures totaled on average 13,272 events per year, just 1.2% of the reported nonpharmaceutical exposures. However, average per annum deaths from CO exposure were 45, representing 16% of total deaths reported for nonpharmaceutical exposures.

CO results from the incomplete combustion of carbonaceous organic material, making potential exposure quite common especially given our reliance on burning petroleum-derived fuels in vehicles and homes, as well as wood product combustion for home heating. Unfortunately, CO is colorless, odorless, and nonirritating, rendering it undetectable by human senses. CO is less dense than dry, pure air, which should improve dispersal in an arid environment. However, humidity lowers air density, causing CO to be relatively equivalent in density, and at times more dense than air, increasing the risk of exposure and toxicity from dose increases [16]. Exposure to combusted materials along with consistent symptoms should raise the clinical suspicion for CO exposure.

The most common symptoms of CO toxicity are headache, dizziness, and weakness. The classic finding of cherry-red lips occurs in only the most severe cases reflecting an accelerated dose [17]. Cyanosis and diminished pulse oximetry will usually not be present, regardless of PaO_2 and oxyhemoglobin levels, because CO and oxygen have similar light absorption spectra. This similarity causes cutaneous pulse oximetry, when utilizing a dual wavelength sensor (most common), to read normal or possibly elevated oxygen saturation levels, and arterial blood to visually appear normal and oxygenated. Carboxyhemoglobin levels can be directly determined by arterial or venous blood gas analysis using co-oximetry. Furthermore, the arterial blood saturation index gap will be elevated, as previously described. Traditionally, increasing carboxyhemoglobin levels correlate with progression and severity of symptoms as follows: 10–20% headache, fatigue, tinnitus; 20–30% headache, weakness, nausea and vomiting; 30–40% severe headache, dizziness, nausea and vomiting; 40–50% syncope, confusion, tachypnea, tachycardia; 50–60% coma, seizure, bradypnea; 60–70% coma, seizures, cardiopulmonary compromise; >70% respiratory failure, death [16, 17]. Preexisting cardiac comorbidities may lead to increased cardiac injury biomarkers such as troponin on the basis of cardiac-specific toxicity with CO more commonly than with other asphyxiants [17]. Pulmonary edema and cerebral edema are reported to occur with more significant exposures (carboxyhemoglobin >50%), but are nonspecific findings related to increased vascular permeability due to tissue hypoxia and shock rather than a direct effect of CO intoxication [16].

Patients should be treated based on symptoms, as opposed to carboxyhemoglobin levels, as serum levels do not necessarily reliably correlate with degree of toxicity. Indeed, patient susceptibility varies in relation to age, underlying physiology, and concomitant comorbidities [14, 16, 17]. In particular, victims with underlying vascular disease and those at the extremes of age may have increased toxic effects at lower CO levels; in those groups, mortality is reported at carboxyhemoglobin concentrations as low as 25%. Assuming the patient is being treated at a healthcare facility and basic BLS management has occurred, initial treatment involves administration of oxygen at an FiO_2 of 1.0. Hyperbaric oxygen therapy (HBO) will clinically significantly decrease carboxyhemoglobin half-life, but this therapy is not routinely available and uniformly definitive recommendations remain elusive [17]. High FiO_2 therapy should be continued until carboxyhemoglobin levels normalize (<2.5% for nonsmokers, <10% for smokers). Hemodynamic and other supportive measures should be applied based on patient needs. Other therapeutic measures, such as systemic or inhaled corticosteroids are not recommended.

Cyanide

While cyanide is an inhalational toxin common in household and industrial fires, it should be noted that it can also be ingested as well as transcutaneously or transocularly absorbed (skin and ocular exposure may lead to quite rapid toxicity due to rapid absorption). Cyanide solutions or cyanide salts are uncommonly ingested; vomiting should not be induced and the patient should be treated as if they had an inhalational or intravenous exposure. Rescuers should don chemical-protective clothing to prevent inadvertent exposure.

Cyanide impairs cellular oxygen utilization by inhibiting the electron transport chain through binding the ferric portion of mitochondrial cytochrome oxidase. This toxicity mechanism affects all tissues creating a profound lactic acidosis due to aerobic metabolism failure. Cyanide is metabolized by hepatic rhodanese to produce renally excreted thiocyanate; toxicity occurs when the delivered dose exceeds the metabolic clearance capability. The most metabolically active tissues, such as the brain and heart, will most be readily affected [2, 5, 14, 15, 18].

United States NPDS data from 2016 to 2018 shows a total of 765 cyanide exposures out of 3,225,142 nonpharmaceutical exposures, with only 11 deaths [19–21]. Therefore, cyanide exposure in the United States is uncommon, and applied therapies are effective. Even so, cyanide toxicity should be considered whenever a patient

has been exposed to combustion of materials or is employed in an industrial field with known cyanide exposure risk.

Cyanide vapor forms from combustion of synthetic materials that are commonly present in furnishings and other standard household products. Most US exposures, therefore, result from household fires but industrial accidents form a substantial portion of fatal exposures. Therefore, the medical professional should be aware of potential industrial exposures, which include electroplating, fumigation, jewelry crafting, laboratory workers, pesticide manufacture, some textile and plastics manufacturing, as well as metallurgy [15, 18]. Cyanide, as hydrogen cyanide or cyanogen chloride, can be used in small-scale terrorist attacks [22]. Secondary cyanide toxicity may flow from acute intravenous inpatient hypertensive emergency management when sodium nitroprusside is employed. Sodium nitroprusside reacts with oxygen releasing nitric oxide, methemoglobin, and cyanide in a dose-related fashion. Therefore, delivered doses should not crest more than 2 μg/kg/min to avoid iatrogenic cyanide toxicity [23, 24].

Because the heart and brain are most affected due to their high metabolic activity, rapid onset of coma, seizure, tachycardia, and hypotension characterize cyanide toxicity [14]. Symptoms of mild or early toxicity include dizziness, headache, anxiety, and flushing [5]. Significant lactic acidosis will be prominent, often >8–10 mmol/L, and is considered a diagnostic feature of cyanide toxicity [5, 15]. Arterial PaO_2 will be normal, and, unlike the dyshemoglobinemias, the arterial blood saturation index gap will not be elevated. A second unique feature of cyanide poisoning is a normal, or elevated, mixed-venous oxygen saturation (which must be measured by blood gas cooximetry for accuracy), due to the significant impairment of the electron transport chain and oxygen utilization [2, 5, 15]. Cyanide serum level measurement is possible, but is often not available at most installations and analysis results too slowly for proper initiation of therapy.

Cyanide toxicity will be unresponsive to supportive measures, including supplemental oxygen administration. Prompt recognition and administration of a specific antidote is essential. Classical treatment consisted of IV administration of sodium nitrite with or without inhaled amyl nitrite, followed by sodium thiosulfate. Nitrite administration induces methemoglobin concentration increase to approximately 20–30%. In turn, this induces cyanomethemoglobin formation and dissociates cyanide from cytochrome oxidase. Thiosulfate then reacts with cyanide to form thiocyanate, which is renally excreted. With concomitant acute kidney injury stage 3, or dialysis-dependent chronic kidney disease, thiocyanate could potentially accumulate, causing neurotoxicity [15]. Accordingly, hydroxocobalamin is now the preferred and the most common treatment for cyanide toxicity (after 2006) due to diminished undesirable side effects despite increased cost. The typical treatment dose is 70 mg/kg up to a maximum of 5 g IV. Cyanide combines with hydroxocobalamin to form cyanocobalamin, which is nontoxic and readily excreted. Treatment with hydroxycobalamin is superior to traditional treatment with amyl nitrite/sodium nitrite/thiosulfate treatment; however, the higher cost of hydroxocobalamin limits its use in low- and middle-income countries (LMIC) [5, 12, 14, 15, 18].

Hydrogen Sulfide (H_2S)

Hydrogen sulfide acts similarly to cyanide by inhibiting mitochondrial cytochrome oxidase, and therefore the electron transport chain and oxygen utilization [15, 18]. Similarly, highly metabolically active tissues are the most rapidly affected tissues, with coma swiftly followed by death. In addition to these shared effects, H_2S is an irritant of medium water solubility and has correspondingly immediate effects on skin, mucus membranes (especially the eyes), and the entirety of the respiratory tract [15]. Also unlike other asphyxiants, H_2S is highly flammable, but at concentrations far exceeding those attributed to death [25].

H_2S is a colorless gas recognized for the characteristic “rotten egg” smell. This aspect makes it less useful as part of violent extremism and serves as a readily identifiable clue to prompt

worker and rescuer safety in industrial accidents. H_2S density is greater than air, and can therefore travel at ground level, accumulate quickly even in open spaces, but concentrates rapidly in enclosed spaces [2, 25]. H_2S naturally occurs in sewers, manure pits, well water, and oil and gas wells and is used or produced industrially at petrochemical, mining, tanning, paper processing, and rayon manufacturing facilities. According to the US Occupational Safety and Health Administration and the Bureau of Labor Statistics, H_2S is one of the most common causes of inhalant-related deaths, with 46 worker deaths reported from 2011 to 2017 [25].

The characteristic odor threshold is approximately 0.01 ppm, with the earliest of toxicity symptoms occurring with ambient concentrations of 2–5 ppm. Early or mild symptoms, much like cyanide, reflect effects on the heart and brain and include headache, dizziness, nausea and vomiting, and cardiac dysrhythmias. Additionally, because of its irritant properties, cough, dyspnea, and skin and mucous membrane irritation are common, including a characteristic keratoconjunctivitis known as "gas eye." This unique finding occurs with environmental exposure concentrations of 50–100 ppm and is an excellent guide to exposure dose. At 100 ppm, loss of smell will occur after 5–10 min of exposure, drowsiness over 15–30 min, and pharyngitis after 60 min. At 150 ppm, loss of smell will occur nearly instantaneously. At 200–300 ppm, conjunctivitis becomes more marked, and lower respiratory tract irritation occurs after approximately 60 min. At 500 ppm, collapse occurs within minutes, significant eye damage within 30 min, and a high likelihood of death within 60 min. At 700 ppm, death occurs within minutes. At 1000–2000 ppm, death is nearly instantaneous [25].

Treatment regimens are not well understood or documented. Assuming treatment occurs at a medical facility, care is primarily supportive, and supplemental oxygen may have little effect given the mechanism of toxicity. No known antidote exists, but use of sodium nitrite administration (300 mg IV over 5–7 min) has shown benefit in anecdotal case reports.

Methemoglobin

Methemoglobinemia generated as a consequence of metabolism functions as a chemical asphyxiant by displacing O_2 from hemoglobin. Oxidative stressors constantly oxidize ferrous iron contained within hemoglobin to ferric iron and generate methemoglobin, an entity devoid of oxygen-carrying capacity. Normal enzymatic pathways in red blood cells reduce methemoglobin back to hemoglobin, with a physiologic concentration of 0% to 1.5–2% [4, 5, 14, 26]. Processes and exposures that drive increased methemoglobin exert toxicity on the basis of impaired O_2 delivery and subsequent tissue hypoxia. As with the other systemic asphyxiants, all organ systems are affected with impact paralleling organ metabolic activity.

Methemoglobinemia has been attributed to ingestion of well water containing nitrite due to fertilizer contamination, sepsis with increased endogenous nitric oxide release (converted to nitrate and methemoglobin), exogenous administration of nitric oxide (inhalation), gastroenteritis in infants, administration of nitrite or nitrate containing medications such as sodium nitrite and nitroprusside, and administration of local anesthetics, phenazopyridine, and dapsone. Multiple other medications or ingested agents have been documented to induce methemoglobinemia, as well as a variety of genetic disorders [4, 5, 14, 26, 27].

Common findings include cyanosis despite normal or elevated PaO_2, and cutaneous dual-waveform pulse oximetry measurements that trend toward 85% that remains unresponsive to supportive therapy (i.e., exogenous O_2) and is insensitive to increased methemoglobinemia as well. Unique to methemoglobinemia will be grayish skin discoloration and chocolate coloration of both arterial and venous blood due to the dark pigmentation of methemoglobin [5, 14, 26]. Arterial blood gas co-oximetry will determine methemoglobin concentrations and an elevated arterial oxygen saturation gap [4]. Methemoglobin symptoms increase in direct correlation with concentration: <10% relatively asymptomatic with cyanosis; >20% anxiety, lightheadedness, headache, tachycardia, nausea; 30–50% fatigue, diz-

ziness, confusion, tachypnea; >50% coma, seizures, dysrhythmias, acidosis; >70% death. However, severity of symptoms may increase based on physiologic conditions, age, and preexisting cardiovascular compromise [4, 5, 14, 26].

Treatment includes supportive measures, resolving the causative agent or condition, and administration of methylene blue. Intravenous methylene blue is indicated when methemoglobin concentrations are greater than 20%–30%, or if symptoms are prominent despite lower methemoglobin concentration [5, 14, 26]. Methylene blue dosing recommendations are 1–2 mg/kg (maximum 100 mg) IV over 5–30 min, with repeat administration 1 h later if methemoglobin levels remain >30% or if symptoms persist [27, 28]. Methylene blue is a vesicant and requires secure IV access to minimize extravasation risk.

Irritants

Inhalational irritants cause damage and injury at varying levels of the respiratory tract primarily driven by toxicant water solubility and secondarily by exposure duration. These agents are some of the most common involved in industrial accidents, but may be also deployed as riot control agents, and have been used in military conflicts or terrorist attacks [2, 22, 29]. Irritants cause morbidity and mortality due to acute respiratory failure, either due to upper airway obstruction or lower respiratory tract pulmonary edema and acute respiratory distress syndrome.

Highly Water-Soluble Irritants

Ammonia, as the third most produced chemical worldwide, is the most common irritant. This class—highly water-soluble irritants—also includes hydrogen chloride, hydrogen fluoride, riot control agents (tear gas) and chloramines, a household toxicant produced by the inappropriate mixing of bleach with ammonia, which parallels irritants generated by cat urine in a litter box [2, 15, 29].

Ammonia is used in industrial production of paper, plastics, dyes, petroleum, methamphetamine, fertilizers, explosives, as well as industrial refrigeration, and household cleaners [15, 18]. Ammonia has a density significantly lighter than air and should therefore disperse rapidly with enhanced ventilation. When stored in liquid form, it is characteristically maintained under pressure, rendering explosive dispersal an issue that encompasses an anticipatedly large volume of space.

Hydrogen fluoride gas is colorless, has a pungent odor detectable below the threshold for toxic effects, and is primarily utilized in industrial chemical processes. Hydrofluorocarbons, in industrial fire suppressants, may release hydrogen fluoride gas at high temperatures including those reached in close space fires [18, 30, 31]. In addition to respiratory tract injury, hydrogen fluoride inhalation may result in life-threatening hypocalcemia and hyperkalemia [15]. Liquid hydrogen fluoride is utilized in a variety of etching and cleaning processes but does not result in significant inhalation toxicity when used in noncommercial venues.

Hydrogen chloride gas is colorless to slightly yellow, has a pungent odor, is denser than air and readily concentrates in low-lying areas demonstrating slow dispersion without forced high flow ventilation. Of particular importance, only 50% of persons can detect the pungent odor at the lower toxic threshold. Hydrogen chloride is present in chemical laboratories, commercially available pool cleaning and disinfectant chemicals, industrial processes involving cleaning, electroplating metals, pickling, leather tanning, metal refining, petroleum well extraction, fat refining, soap manufacture, and edible oil generation, and plastics production. Hydrogen chloride gas may also be released by plastic combustion as may occur in industrial and house fires [32].

Riot-control agents are solids aerosolized by a small explosive, have a density heavier than air, and are characterized by collection in low-lying areas and slow dispersal. Traditionally riot control agents are referred to as "tear gas," consisting of chloroacetophenone (CN) and chlorobenzylidenemalononitrile (CS) [1].

Toxicants with high water solubility dissolve quickly into water of the mucus membranes of

the eyes, oropharynx, and upper respiratory tract producing immediate symptoms of burning, tearing, sneezing, rhinorrhea, and cough, with possible progression to upper airway obstruction. Because of the immediacy of symptoms, exposure is usually self-limiting so long as the exposed individual can flee, and the irritant is completely absorbed in the upper respiratory tract. With sudden, large exposures, or if the individual cannot escape the exposure location, effects may impact the lower respiratory tract and trigger dyspnea, bronchospasm, acute respiratory distress or failure, as well as the acute respiratory distress syndrome [2, 15, 29].

Initial treatment priorities, assuming the patient has been safely removed from the toxicant exposure, are exposed area decontamination, include copious ocular irrigation until a neutral corneal surface pH has been achieved [30]. Supplemental O_2 is indicated for dyspnea, tachypnea, or hypoxemia. Bronchospasm should be treated with nebulized beta-agonist therapy. Stridor should prompt expeditious airway control with a cuffed endotracheal tube. Glucocorticoid therapy may be considered for lower respiratory tract involvement and should be dosed as for acute asthma exacerbation. Current evidence does not establish a definitive benefit for inhaled or IV glucocorticoid therapy, but it should be considered for those with preexisting reactive airway disease or COPD. ARDS should be managed according to current guidelines (see Chap. 22). Ocular effects may be significant depending on the dose and duration of exposure, with ophthalmologic evaluation and follow-up to assess corneal healing recommended. Since hyperkalemia often accompanies hydrogen fluoride intoxication, this specific entity should be anticipated and managed based on concomitant evaluation of renal function. Like hyperkalemia, hydrogen fluoride exposure may induce hypocalcemia that benefits from intravenous calcium administration [1, 2, 15, 29].

Intermediate Water-Soluble Irritants

Chlorine is the most common medium water-soluble irritant. Chlorine gas has a greenish-yellow color, a pungent odor, and a density greater than air, causing collection in low-lying areas and slow dispersal. Chlorine is utilized directly in the production of a many products and is typically stored under pressure when in liquid form for industrial applications. Roughly, 85% of municipal water systems use chlorine for disinfection of public drinking water, and chlorine is used to prevent *Legionella species* transmission from water-cooling towers, which are utilized by large residential and commercial buildings, including hospitals, power generating facilities, and chemical, petrochemical, and petroleum production facilities [33, 34]. The Public Health Security and Bioterrorism Response Act of 2002 required that all municipal water systems assess security against terrorist attack for hazardous chemical release, directly illustrating the potential public health risk related to chlorine [35]. Railroad accidents involving chlorine transport tankers reported in the United States in 2004 and 2005 resulted in 11 deaths due to chlorine exposure [33]. Further, chlorine is a well-recognized chemical warfare agent having been employed in World War I as well as Syria in 2017 [2, 22, 29, 33].

Because of its intermediate water solubility, chlorine damages the entirety of the respiratory tract. The net effects will depend upon exposure concentration and duration. With exposure at a concentration of 1–3 ppm, only mild nasal irritation occurs. With an exposure concentration of 5 ppm, effects will usually be limited to the eyes and upper respiratory tract, with symptoms primarily consisting of ocular irritation, tearing, and rhinorrhea. With exposure concentrations of 15–30 ppm, headache and pharyngitis emerge. Exposure at 30 ppm produces immediate chest pain, nausea, vomiting, dyspnea, and cough. Exposure to concentrations of 40–60 ppm produces toxic pneumonitis and pulmonary edema, while death occurs within 30 min at 430 ppm and within a few minutes at 1000 ppm. The damaging effects of chlorine gas on the lower respiratory tract triggers a robust inflammatory response, commonly demonstrating a brisk leukocytosis and rapidly progressive ARDS [33].

Assuming patient treatment is occurring in a medical facility, treatment includes bronchodila-

tors for bronchospasm, airway management for acute respiratory failure due to lower respiratory tract involvement, and standard lung protective ARDS management. Some sources recommend corticosteroid administration, utilizing standard asthma exacerbation dosing, or inhaled corticosteroids, both of which are reasonable options, although definitive benefit has not been established. Inhaled sodium bicarbonate treatments, presumably to neutralize hydrochloric acid that may have formed with chlorine inhalation and subsequent water absorption are also a reasonable treatment option, although evidence of definitive benefit is lacking. Lastly, given that chlorine produces oxidative damage, inhaled *N*-acetylcysteine as an antioxidant therapeutic is a reasonable treatment option for the initial 24–48 h, although definitive benefit has not been established [1, 15, 33].

Low Water-Soluble Irritants

Phosgene and nitrogen oxides are the two most commonly encountered toxic inhalants with low water solubility. Phosgene is not natural occurring, is a gas at ambient temperature and pressure, is colorless, and emits the odor of freshly mowed hay. It has a density greater than air, causing collection in low-lying areas and slow dispersion. It is extensively used in industrial production of dyes, isocyanates for polyurethane production, polycarbonates, acid chlorides, insecticides, and pharmaceutical chemicals. It is stored as a liquid under pressure for these purposes. Phosgene has been deployed as a chemical warfare agent, most notably in World War I, and is known by the military designation "CG" [1, 36, 37]. Nitrogen oxides include, most notably, nitrogen dioxide, which forms from the decomposition of any plant material, especially those with high nitrate composition such as corn. Nitrogen dioxide is a gas at ambient temperature and pressure, has a yellowish-reddish color, a bleach-like odor, and a density greater than air [15, 38]. Therefore, nitrogen oxides have similar inhaled toxic effects to phosgene.

Because of low water solubility, phosgene does not react upon contacting mucosal surfaces. Toxic inhalations typically produce very few initial symptoms, unless the exposure concentration was extremely high resulting in ocular and upper respiratory symptoms similar to irritants discussed previously [15, 36, 39]. Phosgene (carbonyl chloride) reacts with mucosal surfaces in the lower respiratory tract to generate hydrochloric acid, which denatures lipids (lipid bilayer of mammalian cells) and proteins, disrupts cellular membranes, degrades enzyme function, depletes pulmonary glutathione, augments lipid peroxidation, and increases leukotriene synthesis [37]. The net effects, which take time to develop, are pulmonary edema, acute respiratory distress that leads to acute respiratory failure and ARDS [15].

The US Environmental Protection Agency (EPA) has developed very accurate data and guidance on phosgene exposure and toxicity, both of which are based on acute atmospheric concentration and cumulative exposure dose [37]. Odor can be perceived at 0.4 ppm, and recognized at concentrations >1.5 ppm, at which point mild ocular and nasal symptoms may occur. With acute exposures to concentrations >3 ppm, pharyngeal irritation develops, at >4 ppm ocular irritation occurs, at >5 ppm cough develops, and >10 ppm severe ocular and airway irritation predominate promoting cough, subjective chest tightness and dyspnea, headache, dizziness, as well as nausea and vomiting [1, 37]. At a cumulative exposure of >30 ppm-min (i.e., 3 ppm for 10 min), pulmonary mucosal injury is detectable, and at cumulative doses of >150 ppm-min, pulmonary edema will manifest. Pulmonary injury can progress to ARDS, but usually has a delayed onset termed a "latency period" that ranges from 2 to 24 h post-exposure. Pulmonary edema that develops within 2–6 h correlates with severe pulmonary injury [1, 37]. If respiratory symptoms have not developed and chest X-ray changes have not occurred within 8 h of estimated exposure, significant symptoms are unlikely to develop [15]. An incident occurring in 1928 in Germany illustrates the potential significant dangers of even accidental phosgene exposure, with victims developing symptoms up to 6 miles from the inadvertent release site [37].

Treatment for phosgene exposure primarily involves symptomatic care, similar to treatment

for the previously discussed irritants, with one notable exception: administration of supplemental oxygen should be avoided unless clinically necessary due to potential for increasing ongoing alveolar oxidative damage. When supplemental oxygen is utilized, goal oxygen saturation should be as low as feasibly tolerated and dovetails with the use of conservative oxygenation targets that has recently emerged (i.e., SpO_2 = 88–92%) [2]. Otherwise, management of ARDS should follow a standard approach. Traditional approaches to activity have typically enforced bed rest post-exposure to avoid driving inflammation on the basis of increased work of breathing (WOB), although the merits of this approach may be offset by noninvasive approaches to supporting WOB [2, 29]. Other adjuvant therapies to be considered include nebulized beta-agonist therapy, inhaled N-acetylcysteine, oral ibuprofen or intravenous ketorolac, as well as intravenous glucocorticoids, each of which is underpinned by only limited supporting evidence [1, 2, 15, 29].

Vesicants

Vesicants are incredibly toxic, commonly referred to as blistering agents because of the characteristic dermal effects, but also demonstrate broad-ranging respiratory and systemic effects. These agents will injure any contacted epithelial or mucosal surface. Vesicants are well-known chemical warfare agents and include nitrogen mustard, lewisite, and phosgene oxime. Sulfur mustard serves as the prototype for this group, and bears responsibility for the greatest number of casualties related to chemical warfare agent deployment [2, 29].

Sulfur Mustard

Sulfur mustard is not naturally occurring, is a liquid at ambient temperature and pressure, and has a high volatility with rapid vapor generation. It transmits the odor of garlic, onions, or mustard, has a gaseous density heavier than air causing collection in low-lying areas and slow dispersion. Furthermore, its oil base coupled with low water solubility establishes environmental persistence. Sulfur mustard is used exclusively as a chemical warfare agent and goes by the military designations HD or H [40, 41]. Sulfur mustard is an alkylating agent, reacting with DNA, RNA, and other macromolecules, as well as triggering cell membrane disruption. Because of the mechanism of action, sulfur mustard is particularly toxic to proliferating cells, especially endothelial cells. Due to its lipophilic properties, sulfur mustard readily absorbs transdermally, demonstrates broad systemic distribution, and partitions particularly well to the lungs, liver, bone marrow, and spleen [40]. Due to the absence of a barrier stratum corner layer, the eyes and respiratory tract (unlike the barrier rich skin) are especially susceptible to the damaging effects of sulfur mustard [41]. Absorption rates proportionally increase with temperature and humidity, with intertriginous areas showing greater local effect [2, 29, 40]. For example, an increase in ambient temperature from 72 F to 87 F results in absorption doubling [40].

The toxic effects of sulfur mustard are proportional to cumulative dose. Odor threshold has been determined to be 0.023–0.092 ppm, but blunting of olfactory sensation within minutes of exposure has been reported [40]. Additionally, all pathological effects have a latency period from the time of exposure, rendering victim recognition of severe exposure problematic until the latency period has passed. Ocular exposures have the shortest latency period with 1–2 h after severe exposure and 3–12 h following moderate exposure [42]. Respiratory tract injury has a latency period of 2–6 h with severe exposure and 12–24 h with mild exposure. Regardless, the maximal impact on the pulmonary parenchyma and airways may not be evident for up to 14 days. The latency period for dermal injury may be 6–24 h, with effects progressing for up to 48 h [40, 42].

Exposure level grading has been articulated by the EPA and encoded in the Acute Exposure Guideline Levels rubric for a host of different airborne chemicals ranging from low level (1) to high level (3) across the duration of exposure (10 min to 8 h) to establish the total exposure dose. Very low exposure (0.6 ppm/min) determined by the Acute Exposure Guideline Level-1

(AEGL-1) generally lead to nondisabling effects, while lethal effects occur with high exposure at AEGL-3 (6 ppm/min). It is important to note that exposure that may lead to severe injury may occur at concentrations below the olfactory threshold if exposure occurs for a sufficiently long period of time. Military studies identified widespread conjunctivitis accompanied by chemosis, photophobia, and irritation with cumulative doses of 9.2–11.6 ppm/min. More severe ocular effects, including corneal ulceration and temporary vision loss, occurred with cumulative doses of 11.6–13.9 ppm/min, while disabling ocular effects including permanent vision loss occurs with a cumulative dose exceeding 15 ppm/min [29, 40]. Mild respiratory tract effects, caused by both direct exposure and systemic absorption after dermal exposure include rhinorrhea, epistaxis, nasal and sinus pain, hoarseness, barking cough, wheezing, and dyspnea. Severe respiratory tract effects include severe ARDS, necrosis of the respiratory epithelium, and pseudomembranous obstruction of the trachea and distal airways, with most deaths occurring due to complications of respiratory failure [40, 42]. Dermal effects range from erythema, blistering, and pruritus, to skin necrosis. Lethal exposure correlates with an affected total body surface area exceeding 25% [40, 42]. Systemic effects include gastrointestinal symptoms such as nausea and vomiting, as well as bone marrow suppression that establishes leukopenia but may also include pancytopenia [29, 40, 42].

Sulfur mustard toxicity has no antidote. Therefore, early exposure recognition and immediate decontamination are paramount. Rescuers require chemical protective gear. All clothing should be removed, exposed skin should be washed with copious soap and water, and eyes should be irrigated with copious water. Patient care is entirely symptomatic, and no therapies have any convincing supportive evidence [42]. Skin lesions can be treated leveraging burn management protocols. ARDS should be treated using standard approaches. Adjuncts including n-acetylcysteine inhalational therapy as well as systemic glucocorticoids may be of potential benefit. Early treatments for ocular effects may include topical anticholinergic agents, topical antibiotics, and petrolatum. All ocular injuries should be evaluated and managed by an ophthalmologist, whenever possible. If absolute neutropenia occurs, the patient is at risk for systemic infection and granulocyte colony-stimulating factor and prophylactic broad-spectrum antibiotics may be considered drawing on data from bone marrow transplant patients as well as those with malignancy therapy-associated neutropenic fever [2, 29].

Cholinergic (Nerve) Agents

Organophosphates

Cholinergic-stimulating agents have broad use in medicine (e.g., neostigmine), agriculture, and military warfare, of which pesticides and nerve agents are most important for our consideration. Examples of commonly used home and agriculture pesticides are malathion, diazinon, parathion, chlorpyrifos, phosmet, and carbamates. Chemical warfare nerve agents include Tabun (GA), Sarin (GB), Soman (GD), VX, and the Novichok agents. The “G” agents were developed by Germany for use in World War I, VX in Britain, and Novichok agents by the former Soviet Union. The chemical properties of these agents vary widely, but pesticides are certainly far less toxic than nerve agents, possessing low volatility and requiring far higher doses to achieve toxicity. In contrast, nerve agents require extraordinarily low doses to achieve toxic effect and lethality [40]. Nonetheless, pesticides are more widely employed and therefore account for the majority of mortality and morbidity from this class of toxicants. Pesticides are responsible for approximately 200,000 deaths and one in six suicides globally per year; US data identifies 4300 exposures and four deaths on average per year [19, 20, 21, 43, 44].

The nerve agents are considered to be odorless, since, even if the emitted odor, the olfactory threshold exceeds the toxicity threshold. All are liquid at ambient temperature and pressure, but

all are quite volatile. Sarin demonstrates the highest volatility of the nerve agents. VX was specifically designed with a volatility 2000 times *less* than Sarin to enable prolonged environmental persistence to provide "terrain denial" in military warfare. All nerve agents are significantly more dense than air, causing collection in low-lying areas and slow dispersion [40].

Cholinergic agents uniformly exert toxic effects through acetylcholinesterase inhibition causing accumulation of acetylcholine, the primary neurotransmitter in cholinergic synapses of the central, peripheral, and autonomic nervous systems. This results in overstimulation of muscarinic and nicotinic receptors, as well as central nervous system effects [2, 14, 45]. Muscarinic effects with parasympathetic activation cause salivation, lacrimation, urination, diaphoresis, defecation or diarrhea, emesis, miosis, bronchorrhea, bronchoconstriction, and bradycardia. Nicotinic effects include muscle fasciculation's, weakness, diaphragm fatigue, hypertension, tachycardia, hyperglycemia, and hypokalemia. Central nervous system effects include confusion, ataxia, seizures, and coma [2, 14, 29, 45]. Acetylcholinesterase inhibition by all cholinergic agents—except for carbamates which produce a reversible inhibition—will become irreversible over varying time intervals, a process called "aging." Faster rates of aging equate to lower chance of survival. Rates of aging of pesticides range widely based on chemical structure and span 3.7–33 h [45]. Aging rates for nerve agents are 1.3 min for Soman, 5 h for Sarin, 46 h for Tabun, and 48 h for VX. Most deaths from cholinergic agents will be from cumulative effects causing acute respiratory failure.

Muscarinic effects will predominate with low-dose exposures and occur early with high-dose exposures. More severe symptoms progress to nicotinic receptor-driven and CNS effects. Acute Exposure Guideline Levels (AEGL) are the most comprehensive and delineated values available for understanding the severity of exposure and comparison of potency of the nerve agents. The AEGL-1 (mild, nondisabling effects) for an inhalational exposure time of 10 min for GA is 0.001 ppm, GB 0.0012 ppm, GD 0.00046 ppm, and VX 0.000052 ppm. The AEGL-3 (lethal) for an inhalational exposure time of 10 min for GA is 0.11 ppm, GB 0.064 ppm, GD 0.049 ppm, and VX 0.0027 ppm. Additionally, dermal exposure to 1–10 mL of GB and 1 drop of VX are reportedly fatal [40].

As with the other toxic exposures described in this chapter, assuming the patient is being treated safely away from the site of exposure, decontamination should occur first, noting that any liquid nerve agent absorbed into clothing may continue to cause exposure. Three therapeutic medication classes are well recognized (and United States Food and Drug Administration approved) to treat acute cholinergic agent toxicity: atropine, oximes (pralidoxime or obidoxime), and benzodiazepines. The first and paramount goal of medical therapy is achieving atropinization—clinically clear lungs, systolic blood pressure greater than 80 mm Hg, heart greater than 80 beats per minute, dry axillae, and pupils no longer pinpoint [45]. Standard adult atropine administration is 2 mg IV every 5–10 min. Relatively newer studies suggest incremental increases in atropine dose by doubling the dose every 5 min reduces the time to reach atropinization by 600%. Effective atropinization was then maintained by continuous atropine infusion at 20–30% of the total dose to determine the atropine infusion rate [45]. Once atropine has been effectively administered, an oxime should be administered (usually pralidoxime in the US). Intramuscular or subcutaneous administration is preferred, but intravenous regimens are available. Atropine will reverse muscarinic effects, while oximes will reverse nicotinic muscular as well as CNS effects. Benzodiazepines are recommended for acute seizure termination. Glycopyrrolate IV may be an alternative anticholinergic agent if atropine is unavailable, but a dosing regimen is not well established [14]. Other adjunctive therapies with potential benefit include intravenous magnesium sulfate, calcium channel blockers, as well as serum alkalization (target pH = 7.5) using sodium bicarbonate or sodium acetate during periods of bicarbonate shortage [45].

Radiation Inhalational Injury

Nuclear reactor meltdown or radiation release as well as thermonuclear warhead detonation demonstrate noninhalation mechanisms of acute radiation toxicity as well as blunt and potentially penetrating injury mechanisms. These aspects are outside of the scope of this chapter. Instead, this section will focus on radiological dispersal devices that may create airborne radioactive particulates.

Radiological dispersal devices (RDD) (aka. "dirty bomb") are improvised explosive devices combined with radioactive materials to further enhance physical, environmental, and psychological effects. Despite the added radioactive material creating a quinary category of blast injury effect, the most significant health effects from RDDs will be through quaternary effects (see Chap. 29) [46]. Numerous simulations and studies have been performed by emergency preparedness organizations and the military to understand the expected effects, and therefore required responses to RDDs. Effective response may require large-scale distribution and administration of chelating antidotes to prevent acute radiation sickness, besides environmental decontamination. The effects of RDDs depend on many variables, the most important of which seem to be the explosion design (plume height and radius), setting of the explosion (open air versus closed space), and choice of radioactive material (metallic versus nonmetallic) [47]. Simulations suggest that the vast majority of RDDs detonated in an open-air space will produce minimal acute radiation exposure effects, although smaller dispersion plumes will raise the risk of acute radiation sickness and inhalation of radioactive particles in those nearest the epicenter. Use of nonmetallic radioactive materials, such as radio-cesium, will disperse more readily with detonation and raise the risk of significant inhalation. Relatedly, the risk of significant inhalation of radioactive particles and subsequent acute radiation sickness is vastly increased with RDD detonation within an enclosed space [47].

Acute radiation sickness has a latency period with effects manifesting days to weeks after exposure. Therefore, reducing the absorption of radioactive material is key during initial management (see below). Hematopoietic syndrome occurs with bone marrow suppression at low doses resulting in fatigue, weakness, fever, and an increased risk of infection and spontaneous hemorrhage. Moderate exposures result in additional gastrointestinal symptoms including diarrhea and vomiting, as well as high fever, dizziness, disorientation, and hypotension. Higher level exposures drive the neuromuscular syndrome, with unconsciousness, high fever, diarrhea, and an accelerated likelihood of death [47].

For RDD attack victims, acute life-threatening injuries due to blast effects must be initially addressed [46]. Expected radiation exposure levels from RDDs do not cause acute life-threatening effects, but rescuers involved with RDD victims prior to decontamination merit radiation protection. Dermal decontamination should be completed prior to admission within a definitive care site. After rescue and surface decontamination, the next treatment priority is to minimize systemic distribution and deposition of inhaled radioactive particulates. If high-dose exposure is likely based on event details, the victim should undergo decorporation therapy, a chelating antidote therapy, with either Prussian Blue or Ca(DTPA), based on the suspected RDD radionuclide. Dosimetry can be performed to assess the radionuclide burden prior to decorporation therapy. However, testing is not widely available, and results may not return until after the therapeutic window has closed (typically 24–36 h post exposure). While acute radiation sickness effects can usually be prevented with appropriate and timely decorporation therapy, long-term stochastic effects such as malignancy require vigilant surveillance [47–49].

Conclusion

Inhaled toxicants span a wide range of origins from industrial chemicals to specifically designed nerve agents. The wide array of potentially inhaled toxicants for part of the CBRN-E approach to violent extremism (terrorism) but are

not exclusively associated with such activities. House fires, industrial catastrophes, and on rare occasion, medical therapy, may all lead to toxicant exposure. Therefore, every clinician (prehospital and in-hospital) who addresses patients with chest trauma should be conversant with the origins, signs, symptoms, and therapy for those who sustain toxic inhalational injury. The clinical team must identify those at risk for toxicant exposure and recognize the need for toxicant-specific antidote therapy as opposed to supportive care (Table 31.1). Both patient outcome and clinician safety benefit from understanding this broad grouping of inhaled toxicants, many of which also demonstrate noninhalational routes of ingress.

Resources

Key resources available to clinicians include the following:

Poison Control Center – This is the central communication agency of the American Association of Poison Control Centers. The national phone number **(800)222–1222** should be committed to memory and can be accessed by patient family and practitioner alike for real-time guidance with toxicological emergencies. A website is also available (www.poison.org) for general information and prevention strategies.

PubChem—This search engine within the National Library of Medicine contains free and accessible safety and toxicity information for patients and practitioners. Available at https://pubchem.ncbi.nlm.nih.gov

National Poison Data System—Maintained by the American Association of Poison Control Centers and utilized to publish annual reports on poisonings within the United States (36 to date), offering a trove of valuable information to providers (National Poison Data System (aapcc.org)).

International Toxicity Estimates and Risk (ITER)—Provides risk estimates compiled from a variety of sources in the form of comparison charts to help explain peer-reviewed risk values of different agents. Available at: http://www.iter.tera.org

Agency for Toxic Substances and Disease Registry (ATSDR)—Peer-reviewed toxicological profiles. Available at: https://www.atsdr.cdc.gov

Center for Disease Control and Prevention—Offers relatively little information, mainly relating to poisoning prevention. Available at: https://www.cdc.gov/homeandrecreationalsafety/poisoning/

Environmental Protection Agency (EPA) Acute Exposure Guideline Levels for Airborne Chemicals—Provides an extensive array of exposure data for individual airborne chemicals to determine exposure toxicity risk based on concentration and duration. Available at: https://www.epa.gov/aegl

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32 The Role of Local Facility in National Disaster Planning and the Ethics of Resource Allocation

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Introduction

Timely access to appropriate care is a vital aspect of modern healthcare systems and is particularly pertinant during critical illness when it may determine the likelihood of patient survival. Under normal circumstances, most countries uphold the principle of 'the right to health' (https://www.ohchr.org/Documents/Publications/Factsheet31.pdf) wherein every patient should have equal access to the resources required to meet their clinical needs within reason. However, should healthcare consumption increase exponentially—such as in a time of national disaster—a struggle for resources will likely develop. Unfortunately, healthcare resources are finite and during times of increased demand healthcare providers face challenging questions regarding resource allocation.

A gradual or sudden decrease in resource availability requires changes to be made to standard care algorithms. These challenges are particularly poignant if discrepancies arise between public's and physician's perceptions regarding the division of resources in a time of need. Such discrepancies originate from fundamental differences in the understanding of distributive justice [1]. In general, explicit rationing (i.e., decisions based on stated principles and rules) raises less concern regarding fairness than implicit rationing. With the latter, the basis of decision-making is undisclosed and may be influenced by either unconscious or even conscious bias [2]. Plans for national disasters should therefore consider resource allocation in a dynamic manner that is able to adapt to remaining system capacities. Strict adherence to allocation decisions is the only way to ensure a consistent level of care and prevent healthcare system collapse. How these resources are divided and to whom they are provided forms the basis of the discussion within this chapter.

Ethical Principles of Resource Allocation

Resource allocation refers to the assignment of various resources to specific patients for specific purposes. Resource rationing refers to the resource allocation strategies employed when supplies are inadequate to meet demand [3]. Providing the greatest amount of resources to those in greatest need is an approach likely to garner the support of many clinicians. In fact, this option constitutes the main ethical justification for the existence of intensive care units (ICU). However, in times of national disaster, this approach may result in dedication of a great

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amount of resources towards patients who ultimately have a poor chance of survival. Moreover, directing so many resources towards very unwell patients may negatively affect the chance of survival of other less-injured patients who require fewer resources to ensure survival. The ethical framework for decisions regarding resource allocation is therefore rooted in several treatment prioritisation options [3].

The option of 'first-come, first-served' is based on the principle that the first patients to attend hospital receive resources appropriate to their needs until such time that all resources are consumed. It is often the case that those patients who present earlier to hospital are those less severely ill. This has been shown to happen in terror-related events [4] and in earthquakes where severely injured patients requiring complex extraction may present to the acute care hospital relatively late [5]. Therefore, early arrivals may require less-intensive and time-sensitive care than the more severely injured who present later. As a result, a situation may arise that the early arrival of many lightly injured patients will result in resource consumption before the arrival of those most critically ill but for whom few resources remain.

The general perception is that such an approach could result in inappropriate early resource consumption and thus higher rates of mortality. When quantitative models simulating a surge in demand for ventilators during a time of pandemic compare various triage tools, a 'first-come, first-served' approach to ventilator allocation is associated with unacceptable mortality. Only by using high-quality predictors of survival with a sensitivity and specificity of 90%, could mortality be substantially reduced [6].

The approach of 'first-come, first-served' also fails to care for vulnerable patients for whom transport to hospital is more challenging especially during major disasters. In order to equally care for this group of patients, the option of 'lottery based' prioritisation of resource allocation has been proposed. If such an approach was adopted, resources would either be randomly allocated—or not-allocated—to patients irrespective of their time of presentation, urgency of care need or severity of injury or illness. However, this method of resource division is difficult to justify in an age of evidence-based medicine during which physicians strive to provide efficacious care, and there is little data demonstrating lottery superiority compared to other approaches. Moreover, a lottery is unlikely to garner much public support.

The option of providing the greatest amount of care to the greatest number of patients is a reasonable compromise between the two options presented above. This approach can meet the expectations of both physicians and the public whilst somewhat mitigating the challenge of early resource consumption. However, this approach requires some ability to predict the future case load as this is prerequisite for planning resource requirements. If the time frame of patient presentation to hospital varies, and if changes occur in patient needs over time, the required resources cannot be planned and allocation may become ineffective at the point of delivery. Furthermore, the performance of most triage systems during times of disaster remains unknown outside the world of theoretical modelling as they are rarely tested. Therefore, much remains to be learned regarding how the accuracy of these systems responds to disaster evolution. Whilst the responsibility for such research does not lie directly on the shoulders of individual facilities, their contribution to the learning process in such circumstances could be invaluable.

Often included in triage decisions are patient-specific factors which complicates matters further. For example, the 'fair innings' principle relates to the need to prioritise patients with the most life years left and takes into consideration the patients' stage in life [7]. Priority is given to younger patients and greater benefit is attributed to those who have lived through fewer life stages. Preference can also be given to those with a larger number of dependants compared to those who are dependent on others for their care. Whilst age should not factor directly into the decision-making process, Biddison et al. propose that priority should be given to those aged <49 years, followed by those aged 50–69 years and finally to

those aged >70 years because of the relation between age and the factors discussed above [7].

Strategies that leverage age as a determining factor are commonly labelled as 'ageist' and note that a healthy elderly individual may be less physiologically challenged than a younger patient with an inherited metabolic disorder such as insulin-dependent diabetes or a concomitant malignancy. Anti-ageism proponents contend that comorbid conditions are more relevant than age and that age should be expressly eliminated from decision support tools that guide resource access and allocation during disaster or crisis care. Clearly, age as an indicator of appropriateness of care incites substantial controversy as there is no uniformly accepted guidance on how age may or may not inform decision-making either in the field or on arrival at a definitive care facility.

Relatedly, priority may also be given to those patients without life-limiting pre-existing conditions. These patients have an improved prospect of long-term survival compared to patients with significant medical comorbidities. Injury-specific variables should also be considered in the decision regarding resource allocation. In the setting of blunt chest trauma, factors associated with prolonged ICU admission and days ventilated include male gender, admission systolic blood pressure of <90 mmHg, bilateral rib fractures and a concurrent requirement for axial skeletal surgery [8]. Accordingly, the presence of these injuries could be included when prioritising patients and resources. Injuries, and hence need for resources, may also vary depending on the nature of the disaster. Patients with chest trauma following earthquakes have been found to be older and have an increased incidence of pneumothorax requiring pleural space decompression [9]. Local facilities should be prepared to treat injuries most likely to be seen in the disaster scenarios they are more likely to encounter.

Decisions regarding resource allocation should be medical at their core. However, it is clear that when resources are limited, consideration of patient-specific factors alone does not suffice. Hence, including para-medical factors into the decision-making process may be required. The unifying theme of the approaches outlined above is the attempt to save as many lives as possible given the available resources. This myriad of ethical considerations highlights the difficulty underlying the decision-making processes of resource allocation. It is therefore unsurprising that community engagement is expressly desired to create disaster guidelines to ensure that diverse approaches, perspectives, values and considerations are captured and shared [10].

Triage

Manning the frontline of the ethical allocation of resources during a time of national disaster is the triage officer. Triage is comprised of two components—prioritising patient care based on the severity of illness and directing existing resources to the patients most likely to benefit from receiving them. The triage process takes place parallel to the provision of first aid to those patients who desperately require it.

In its most simple format, triage involves dividing patients into four categories that will identify what treatment each patient will receive. Red demarcates those requiring immediate life-saving care, yellow for urgent care, green for non-urgent care and black, the most troubling, for those patients with such devastating injuries they are not expected to survive with the resources available (i.e., expectant care) [5, 11]. Department of Emergency Medicine (ED) triage protocols that are based on the severity of patient injury, such as the Injury Severity Score developed by the Karolinska University Hospital, are well validated and easily implementable [12]. As opposed to patient severity scores commonly used to describe the severity of illness (e.g., the Acute Physiology and Chronic Health Evaluation II [APACHE II]), the scoring methods used in ED triage protocols need to be very simple and rely on data readily available in the early stages of patient care. Therefore, triage scores are mostly based on clinical rather than laboratory data which enables rapid patient classification whilst patient severity scores include a more complex mix of both. Given the increasing availability of

point-of-care testing, one may expect that lab values will be acquirable outside of the acute care facility and may impact triage approaches.

During a national disaster, decisions regarding resource allocation should be made as soon as possible. Ideally this should start in the pre-hospital setting prior to the triage process at the treating hospital. An effective, efficient and easily implementable triage system will streamline patient flow and ensure that the required resources are continuously being matched to those in greatest need of them whilst maintaining realistic expectations of survival. Both the triage process and the relevant decisions made by the triage officer should be clearly communicated with the medical staff designated to care for the patient and, if possible, with the individual patient and their family. It is important to note that the triage officer and the treating clinician(s) must be different individuals to ensure that there is no conflict or bias in their assessment.

The use of universal scores creates a common language between different hospitals and departments. This aids regional and national communication between healthcare centres and either local or central government, which is vital in formulating a coordinated response during a national disaster. As a result, these tools need to be selected beforehand, relevant staff should be trained in their use, and they should be speedily implemented in the event of a national disaster [13].

Triage is an evolving process as decisions may change during patient treatment. Such change may occur due to a change in a patient's condition (including clinical deterioration or improvement), developments in the information available regarding the patient or a change in resource availability. As noted above, triage should start in the pre-hospital environment, continue in the ED and subsequently be taken up by subspecialists such as surgeons or intensivists when deciding on definitive care [3]. Appeals to initial triage decisions should therefore be considered a part of the decision-making framework. An efficient and effective appeals process should be created as part of the triage plan and strictly implemented at a time of national disaster. Importantly, the appeals arbiters should not include any of the treating clinicians. Last minute introduction of stakeholders into triaging patients, the creation of an increasingly complex decision making process at any point during triage and decentralisation of patient-related decisions from the triage officer is the antithesis of efficient triage and should be discouraged [14]. As such, appeals to triage decisions must not come at the expense of the efficiency of the triage process.

Despite the use of triage protocols, over- and under-triage are common and should be avoided to prevent inappropriate resource allocation. Over-triaging occurs when a patient is presumed to be more unwell than they actually are [15]. This results in patients receiving higher levels of care than they require, potentially leading to an inappropriate allocation of resources, staff exhaustion and impaired patient flow [16]. This trend has even been found in specialist triage systems created for specific hospital networks [17]. Furthermore, over-triage has been associated with increased hospital cost when it results in unnecessary transfer to a tertiary or quaternary trauma centre [18] and may lead to overburdening of the limited resources available at specialist centres [19].

Conversely, under-triage occurs when patients are considered to be less unwell than they actually are. Evidence suggests that the association between this cohort of patients and their increased incidence of mortality starts in the pre-hospital setting [20]. Elderly patients seem to be at increased risk of under-triage, especially after suffering chest trauma and have higher rates of preventable morbidity and mortality [21]. Under-triage may reflect the influence of therapeutic agents that modify hemodynamics or may reflect individual phenotypes that manifest different responses to physiological challenges.

There is a historical preference for over-triage which may be appropriate when resource allocation is not being rationed [22]. However, at times of national disaster and increased resource demand, over-triage has been associated with increased morbidity and mortality for the reasons suggested above [23]. This emphasises the need for accurate triaging to maximise efficient

resource allocation based on the previously mentioned ethical foundations in an effort to avoid unnecessary deaths [24]. Furthermore, it also highlights that triage systems generally need to change their focus and approach between times of limited and times of plentiful resources.

Impact on Clinical Practice Outside of Injury Care

It is also important to consider the impact that national disaster may have on other non-related conditions. In times of resource shortage, clinical practice outside of direct injury care needs to be adjusted.

Allocation of ICU Beds

With increased resource demand, concurrent medical or surgical conditions that may not have previously precluded ICU admission, may now serve as contraindications to being accepted to high-intensity care spaces. Although not exhaustive, this may include out of hospital unwitnessed cardiac arrest, metastatic malignancy or severe polytrauma as all of these conditions substantially and deleteriously impact either survival or resource allocation or both [11]. The ability to change longstanding habits regarding allocation of ICU beds represents a significant challenge in resource allocation during a disaster. Additionally, regardless of the indication for ICU admission, patients who fail to progress despite maximal therapy may also need to be discharged from the unit in order to make space for a patient with a more favourable prognosis as part of an equitable resource triage and allocation process [25].

In addition to limitations on bed space in regional hospitals, patients who otherwise would have been treated locally may need to be transferred to more distant facilities during a time of national disaster. When hospitals reach maximum capacity in terms of bed occupancy, patient transfer to hospitals where there are empty beds may provide patient surge relief. Transfer may occur from both the ED and the inpatient space. Since trauma centre beds may be in higher demand than beds for uninjured patients, it may be necessary to transfer a non-trauma patient out of a trauma hospital. Transfer agreements that would address such patient flow and repatriation—when feasible—should be part of routine disaster planning. This will act to increase capacity at those centres that have the support services needed to treat major trauma patients.

Allocation of Mechanical Ventilation

With appropriate planning, the disparity between the availability of equipment and the ability to use spare equipment may be reduced. Although the absolute number of ventilators at a given hospital may be sufficient, the number of staff trained to use them may be limited. As noted during the early phase of COVID-19, both equipment and trained operators were in limited supply. It is therefore imperative, as part of disaster planning, to identify staff with training that would enable them—in conjunction with trained critical care staff—to help care for critically ill and injured patients. Several major medical professional organisations including the European Society of Intensive Care Medicine and the Society of Critical Care Medicine developed and deployed training regimens to enable non-ICU clinicians to participate in critical care alongside critical care professionals. This approach was key in staffing novel ICU spaces and serves as a model for the acute expansion of critical care services that may be applicable to natural or man-made disasters as well. Many clinicians do not receive sufficient education and training in disaster medicine and public health preparedness to be properly prepared to effectively participate in disaster care [26]. Therefore, establishing a capable and effective medical workforce for a disaster response, including the ability to provide mechanical ventilation, requires education and training to the relevant staff and is one of the responsibilities of the local facility and national specialist organisations in preparing for mass casualty scenarios.

The appropriate location for treating these patients is also important. Considerations should include the need for sufficient space to expand critical care environments in a safe and effective fashion. Appropriate space must be able to accommodate for supplies including but not limited to monitors, invasive and non-invasive ventilators, medical grade gas containers, suction, patient care supplies, computer access, communications, sterile equipment including procedure and difficult airway carts, medication lockers and nursing and physician workspaces. Reconfiguring existing space and creating entirely new spaces have been embraced during the COVID-19 pandemic [27]. Specifically, with regard to chest trauma, the ability to connect chest drains to a negative pressure vacuum is a prerequisite.

Finally, some patients with chest trauma and acute respiratory distress accompanied by hypoxemia may be managed conservatively. Although this approach has only been endorsed as a low-grade recommendation by the British Thoracic Society, the use of non-invasive ventilation in patients with blunt chest trauma may be beneficial for certain conditions and may be reasonable during disaster care when invasive ventilation is not available [28]. Conservative management may be a very effective tool for the preservation of resources, but the use of this tool should be tempered with caution as delays in care may be associated with worsening of patient outcomes [29]. In order to ensure that decisions regarding conservative treatment are made wisely, those those making these decisions must be both informed and trained in the use of such measures.

Command, Control, Communication and Business Continuity

The ideal management of disasters is centralised, both at a national (Fig. 32.1) and local level. Such centralisation requires preparation and commitment at multiple levels of healthcare and government. Nevertheless, the onus is on each individual facility to plan and prepare.

Across facilities, barriers to communication may include language and technological difficulties, differences in roles and responsibilities and failure to co-locate and coordinate activities [30]. Developing a common understanding of responsibilities and sharing of information will minimise deliberation and improve the relevance and

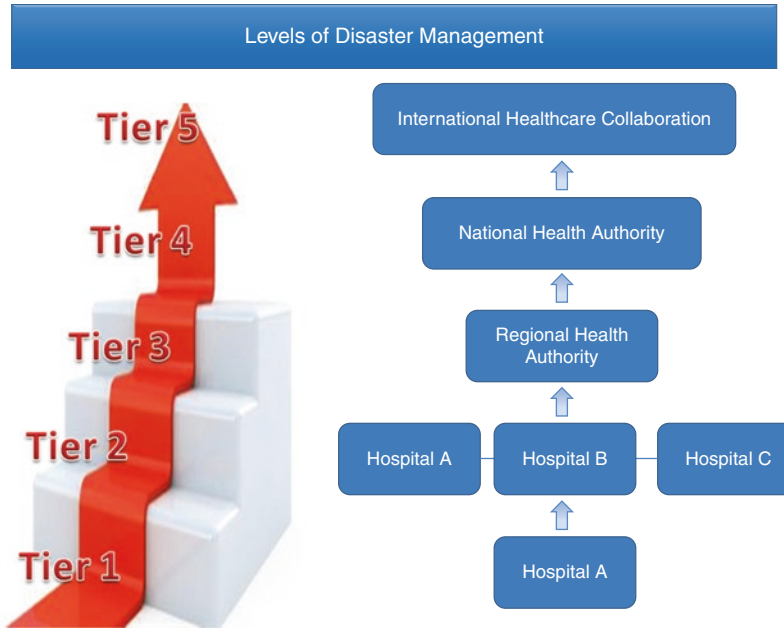

Fig. 32.1 Levels of disaster management. This figure demonstrates the tiers of disaster response starting at the local facility and moving through increasing levels of complexity and engagement with related organisations. The highest tier represents international collaboration. Not all disasters require all tiers to be engaged and tier step optimally reflects the scope and duration of the disaster

speed of the medical and surgical response [31]. Although specialist teams may be deployed to provide aid, this only reinforces the need for training and protocol development to allow such integration to occur at the onset of a disaster [32]. In both the resource replete and resource-limited spaces, these teams may incorporate governmental and non-governmental organisations into the local or regional facility response driving the need for a coordinated communications networks [33, 34].

Facilities must also be prepared to contribute to the overall effort of disaster response even at the cost of potential loss of income. For many trauma centres, such undertaking are part of their remit and are viewed as essential to their mission. Nonetheless, disaster medicine is notoriously financially costly [35]. Therefore, local commitment to help with disasters should receive support at a national level in terms of philosophy as well as resource and financial assistance. Durable resources that may be lifesaving during disaster may support non-disaster care and facility expenditure once the disaster has been successfully managed [36]. Indeed, both resources and finances underpin delivering high-quality care.

The Donabedian model proposes that three components be used for evaluating care quality: structure, process and outcome [37]. Without data on these three system characteristics, improvements in care quality are difficult to align with specific requests or processes. For this reason, it is also vital to fully divulge local data during a time of national disaster. Transparency regarding resource consumption enables future planning and informs patient movement and resource allocation. This can also have an impact on the focus and intensity by which aid may be optimally provided by either external or internal agencies [38]. Such data acquisition should be routine so that data tracking and analysis is not unique to crises or disasters, but is instead part and parcel of continuous quality improvement efforts.

Indeed, data collection and patient tracking constitute major challenges during disasters [39, 40]. Collecting patient data becomes difficult due to environmental hazards, communication disruption or incompatibility between treatment locations and compromised basic infrastructure (e.g. electricity, internet, computerised systems). Therefore, disaster planning must also encompass efficient data collection, data transmission, data sharing and data fidelity (i.e. cybersecurity and intrusion prevention). Backup approaches should also be available to ensure consistent operations should any of the above issues be encountered [41]. Some adaptations include shortened medical record entries, 'down-time' chart procedures if the electronic record is inaccessible as well as enabling a system to work with individuals devoid of identification at the time of facility entry akin to established trauma system approaches.

Finally, consideration should also be given to those patients who are not directly involved in the national disaster as their care may also be affected indirectly. Patients with acute medical and surgical conditions will continue to present to hospital. Patients with cancer diagnoses will need to continue both workup and treatment, and those with chronic illness will need ongoing care. The local facility must plan to accommodate the needs of such patients parallel to managing those directly involved in the disaster. Vulnerable populations typically include the elderly, children, those with cognitive dysfunction, those with chronic illness and the gravid [42–46]. Disasters exert powerful influences on routine care and divert attention from those whose needs are not directly impacted by the disaster. It takes little to disrupt the delicately balanced care of patients who rely on a consistently delivered service that may be crippled by the overwhelming needs of disaster care that captures national and, often, international attention.

Summary

The role of the local facility in a national disaster is comprised of its responsibility towards national level preparations and internal preparedness. At a national level, development of communication routes, shared understanding and the division of responsibilities and roles

alongside the establishment of modes of receiving and delivering data updates are crucial. At a local level, preparations should cover equipment, space and staff. Stockpiling of equipment has not been discussed in the current chapter as this is a topic unto itself. However, preparations within the hospital should include equipment allocation to pre-selected areas to be accessible for the anticipated surge. Finally, staff requirements should include disaster education and training.

Whilst trauma-focused clinicians strive to render high-quality care, triage officers need to help clinicians understand which patients' care imperatives can be matched with resources when space, supplies and clinicians are in short supply. Decisions regarding ethical resource allocation are complex not only because of the ethical foundation on which they are based but also because of the requirement to make medical rather than emotional decisions. Decisional frameworks benefit from being inclusive in their development and transparent in their distribution so that those who will deploy these guidelines—and individuals who will be subject to them—are well informed and in agreement.

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