Chest Trauma and Lung Contusions

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Introduction

Injury to the thorax contributes uniquely to outcome in the multitrauma patient with orthopedic injuries. It has long been recognized that chest injury is an independent predictor of morbidity and mortality in this setting [1]. That being said, the precise factors in thoracic trauma and extrathoracic bony injury that combine to put patients at risk remain an important area of investigation. Two themes emerge. First, primary pulmonary injury from an injured chest wall and lung makes operative management of bony injuries potentially unsafe. For example, a severe pulmonary contusion resulting in marked hypoxemia is a pivotal organ dysfunction that may increase the risk of any planned intervention. Second, the systemic response effected by blunt multitrauma, and exacerbated by bony injury, puts the lung at risk for a secondary inflammatory injury manifesting as the acute respiratory distress syndrome (ARDS). This second phenomenon, though incompletely understood, has substantial implications about the proper timing and nature of our interventions.

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Injury Severity Scoring

The most common descriptive tool in use today remains the *Injury Severity Score* (ISS). This provides a framework for description of the anatomic extent of injuries. For each of six defined body regions, individual injuries are assigned an *Abbreviated Injury Scale* (AIS) graded from zero (no injury) to six (unsurvivable). The highest AIS for each of three regions is squared and added to calculate the overall ISS. The AIS assigned to common chest injuries is shown in Table 14.1. While this system provides a common language to describe individual patients and allows for comparisons between groups, it does have some drawbacks for initial decision making. First, it largely ignores the underlying physiology of the patient. For example, while the number of rib fractures certainly has some correlation with the physiology of the patient, the ability of any individual patient to execute the work of breathing cannot reliably be predicted based on number of fractures alone. Second, not every injury is

Table 14.1 Abbreviated injury scale for some common chest injuries

Injury description	AIS
Pulmonary contusion	3
Fracture <3 ribs	1–2
Flail chest, unilateral	3
Blunt cardiac injury, minor ECG changes	3
Torn descending thoracic aorta	4–5

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immediately recognized and categorized; so many times, the "final" AIS/ISS is not appreciated until further imaging, assessment, or repair is done. Other scoring systems specific to chest injuries may have more utility for early decision making and are discussed later in this chapter.

Epidemiology

Thoracic injury is the primary cause of death in about 1 of 4 patients who succumb to trauma and contributes to the death of another 1 out of those 4 [2]. Most mortality directly attributable to chest trauma occurs very early (within minutes of injury) due to major cardiovascular disruption or major lacerations of the tracheobronchial tree. Excluding these early deaths, less than 5 % of patients with blunt thoracic injuries will require an operative intervention in the chest. For example, in a study of over 1,500 patients with combined blunt thoracoabdominal injuries, only 4.3 % of patients underwent thoracotomy (excluding resuscitative thoracotomies) [3]. For practical purposes, tube thoracostomy is the most invasive thoracic procedure needed in the vast majority of patients. Despite the rarity of operative intervention, patients with major chest injuries frequently have major cardiopulmonary dysfunction.

Blunt mechanisms of thoracic injury predominate in most centers, with motor vehicle accidents accounting for the vast majority. Penetrating injuries are less common and are likely to be of limited interest to those clinicians enjoying this book; they will not be discussed further. Blunt chest injuries usually occur in association with multiple injuries to other anatomic regions. Indeed—patients with major thoracic injuries typify the multiply injured patient. For example, in a 1987 study of over 500 patients admitted to the Maryland Institute of Emergency Medical Services with blunt chest injuries, only 16 % of patients had injuries limited to the chest [4]. In the Quebec trauma registry, approximately 25 % of patients with chest trauma had concomitant abdominal injuries [5]. The structures most commonly injured in the chest are the ribs, the pleura, and the lung. Major cardiac and vascular injuries, while certainly important, are in fact uncommon in patients who survive the initial insult. Blunt esophageal injuries are vanishingly rare. A summary of the distribution of injuries from three large studies is provided in Table 14.2.

Table 14.2 Relative frequency of injuries in the chest in three large published series

Injury	Cited frequency (%)
Rib fractures	35–64
Pulmonary contusion	16–30
Hemo-/pneumothorax	11–50
Flail chest	5–10
Heart/great vessels	2–6

Given that thoracic injuries are rarely an immediate threat to life in the patient who survives the initial insult, the primary challenge for the clinician is optimizing supportive care of the cardiopulmonary system and preventing pulmonary complications such as pneumonia, ARDS, fat emboli syndrome, and prolonged ventilator dependence. The sequelae of both direct and indirect cardiopulmonary injury can substantially complicate the care of multiply injured patients. In the Hannover experience, outcome was described in 278 multiply injured patients with chest trauma (ISS > 15 and Chest AIS >2, excluding severe brain injuries) [6]. They found that length of stay averaged 33 days and rates of pneumonia and ARDS and multiple organ failure (MOF) were 22 and 13 %, respectively. In general, then, given the likelihood of prolonged and complex hospital course, the optimal care of a patient with orthopedic injuries and concomitant thoracic injuries involves proper risk assessment (is it safe to take this patient to the operating room?) and planning interventions (is the secondary insult from an orthopedic procedure likely to worsen cardiopulmonary physiology?).

Pathophysiology of Pulmonary Dysfunction

Multiply injured patients are at risk for major pulmonary dysfunction because of disruption of three key elements. First, brain injury is common

in patients with chest injuries, resulting in inadequate respiratory drive, or inability to maintain patent proximal airways. Second, injury to the torso can produce changes in compliance, ineffective respiratory effort, and pain that impact the patient's ability to complete the work of breathing. Third, insults to the lungs themselves result in ineffective gas exchange and hypoxemia. In polytrauma patients, it is likely the clinician must consider simultaneous insults affecting all three elements. Impaired airway patency (e.g., diminished level of consciousness), increased work of breathing (e.g., multiple rib fractures), and impaired gas exchange (e.g., pulmonary contusion, fat emboli syndrome) often coexist.

At the same time that these patients experience impaired gas exchange, they actually have a marked increase in respiratory demand because the neurohormonal response to injury results in increased cellular metabolism. This creates a substantial increase in CO_2 production that must be matched by increased elimination from the lungs. While a resting adult eliminates 200 cc/kg/min of CO_2, postinjury hypermetabolism results in CO_2 production in the range of 425 cc/kg/min [7]. Thus, the minute ventilation required to maintain a normal pH may rise from a resting rate of approximately 5 L/min to more than 10 L/min. This represents a 100 % increase in ventilation simply to meet metabolic demands. To make matters worse, injured patients typically have an increase in physiologic and anatomic pulmonary dead space—ventilated regions of the lung that do not participate in gas exchange. In a normal adult, the proportion of each breath that is dead space (V_d/V_t) is approximately 0.35. For injured patients with pulmonary failure, the V_d/V_t often exceeds 0.6. Simply put, extra dead space means each breath is less effective at eliminating CO_2. Therefore minute ventilation requirements in the 12–20 L/min range are not uncommon in the postinjury setting.

In this light, secondary insults that further impair gas exchange or further increase metabolic rate may cause a stable patient to decompensate; as discussed below, orthopedic interventions are uniformly associated with worsening gas exchange. This makes timing of bony fixation a challenging puzzle. If we do not definitively repair fractures, we impair the respiratory system by immobilizing the patient (impaired work of breathing, increasing dead space, ineffective cough). Alternatively, If we opt for definitive fixation in a tenuous patient, we may impair the pulmonary system by worsening gas exchange and increasing metabolic demand.

The patient with chest injuries faces hurdles in meeting increased respiratory demand. Respiratory drive may be impaired by brain injury and by medications routinely used for sedation and analgesia. The energy required to complete a respiratory cycle is increased by chest wall edema and recumbent positioning, which is often prolonged in patients with major bony injuries. Muscular weakness from impaired energetics (acidosis, cardiovascular failure, mitochondrial dysfunction, oxidant stress) or fatigue may be an insurmountable challenge. Decreased pulmonary compliance from an increase in extravascular lung water and pleural collections (effusions/hemothorax) also contributes. Lastly, and perhaps most importantly, pain from torso injuries or operative interventions make the increased ventilatory demand a substantial burden to the patient.

Primary Injury Patterns

Rib Fractures and Flail Chest

Rib fractures are the most commonly identified chest injury in the multiply injured patient. Crude rates of morbidity and mortality are consistently associated with the number of broken ribs, particularly in elderly patients [8, 9]. Patients with multiple rib fractures are thought to be at high risk for pulmonary failure and pneumonia—likely from impaired cough, atelectasis from splinting, and inability to execute the work of breathing if pain control is poor. A recently published analysis of over 40,000 patients queries this association a bit more closely [10]. This work by Jones et al. highlights that in patients whose only injuries are rib fractures, mortality is less than 6 %. Further, when early (<24 h) deaths

are excluded, crude mortality, while still related to number of fractures, is less than 10 % across all groups. The most powerful predictor of mortality was the abbreviated injury score for the chest region, reflecting the potential importance of flail chest (below) and injuries involving the pleural space, lung, and mediastinal structures. The theme again here is that patients who surviving long enough to warrant orthopedic interventions are unlikely to die from their thoracic injury, and thus minimizing secondary insults becomes pivotal in achieving excellent outcomes.

Flail chest is a pattern of injury wherein a portion of the chest wall loses bony continuity with the rest of the respiratory pump. This most commonly occurs when multiple adjacent ribs are fractured in more than one location. It can also occur in association with sternal fractures or disruptions of costochondral junctions. When the patient expands their chest to take a breath, creating negative intrathoracic pressure, the disconnected area ("flail segment") moves inward in a paradoxical fashion. Particularly when this injury occurs in concert with a major loss of thoracic volume ("caved in chest"), the expansion of the underlying lung is attenuated, and there may be decreased effective tidal volume and therefore impaired ventilation.

Acutely, flail chest injuries per se are not frequently an early threat to life, with mortality reported as less than 10 % in modern series [11, 12]. In isolated flail chest, for example, most patients will not need mechanical ventilation. The major initial challenge is pain control and pulmonary hygiene as ineffective cough and ability to execute increased ventilatory demands are common. In long-term follow-up [13], this is a morbid injury pattern to be sure, as chronic pain, chronic dyspnea, and disability are a common outcome. The significance of a flail chest in the acute setting largely relates to the fact that it denotes major energy transfer to the thorax. This is particularly true in younger patients, where ribs are relatively elastic—more likely to transiently deform than to fracture. Major bony injuries to the chest wall in a young patient with a major mechanism of injury (e.g., high-velocity motor vehicle crash) signify a high likelihood of underlying pulmonary contusion (see below) and extrathoracic injuries [14].

Supportive care remains the mainstay of treatment in rib injuries, with or without flail chest, though there is renewed and justified interest in rib fixation. Acute mortality for chest wall injury is low, but long-term morbidity is substantial and is largely related to malunion—which would appear eminently preventable. Design of rib-specific hardware permits a more practical approach, and newer techniques involving plates with some elasticity as well as minimally invasive approaches may continue to fuel enthusiasm for operative treatment. A number of small published series suggest improved short-term outcomes [15, 16]. The current challenge is identifying patients who are likely to substantially benefit. For example, the patient with other major injuries that may result in prolonged ventilator dependence (brain injury, open abdomen, spinal cord injury) may not benefit acutely or long term from chest wall fixation.

Pulmonary Contusion

Pulmonary contusion, simply put, is a bruise of the lung. The most common presentation is a young passenger struck on the nearside compartment; rapid deceleration and frontal crashes into fixed objects are frequent contributors [17]. Three different types of forces combine to produce injury to the lung. First, direct transmission of energy through the chest wall can bruise the lung. Secondly, the lung can be bruised by shearing forces, for example, when a high-energy missile passes through the lung parenchyma, there is a zone of contusion around the tract of the missile. Thirdly, blast or concussive injury can produce significant lung contusion without obvious chest wall damage. An isolated pulmonary contusion is pathologically similar to bruises elsewhere. The initial response is edema and hemorrhage. This is followed by inflammation, recruitment of cellular elements to the zone of injury, and then by repair. The clinical course follows a similar pattern. As the swelling and inflammation evolves, there is worsening of

pulmonary compliance and gas exchange. This continues for 48–72 h after which improvement should be expected. Some mild hemoptysis can be expected as hemorrhagic secretions are cleared from the distal airways.

An initial chest x-ray is diagnostic in patients with large contusions. Smaller injuries may not become evident until later—when swelling and inflammation occur. Approximately one third of patients with blunt chest injuries will have evidence of pulmonary contusion on Computed Tomography (CT) that was not appreciated on initial plain radiographs [18]. CT has thus been promulgated as a more sensitive tool for diagnosis, and a number of scoring systems have been developed. Strumwasser et al. [19] analyzed 106 consecutive patients undergoing CT of the chest for blunt multitrauma. They observed that a computed tomography volume index (estimating the fraction of total lung involved by contusion) was an independent predictor of ICU length of stay. Additionally, pts with a CT volume index >0.2 had, on aggregate, a higher risk of pneumonia, ARDS, and death. In a larger retrospective series from Boston (almost 400 pts), a score of 1–6 was used, based on presence or absence of contusion in three zones of each lung [20]. They observed that mechanical ventilation was required more often in patients with a score >2, and this was an independent risk factor for the need for ventilation (odds ratio = 13); this can be thought of as 50 % or more of lung zones involved with contusion. That being said, only 35 % of patients with BPC6 >2 required mechanical ventilation. Additional factors also predictive of mechanical ventilation included diminished Glasgow Coma Scale (GCS) score and >4 rib fractures. Wang et al. observed that PC volume predicted chest trauma patients who would later meet criteria for adult respiratory distress syndrome [21].

It would seem logical, given these studies, that patients with large pulmonary contusions evident on radiographs should be recognized as at increased risk for secondary pulmonary insults. A different and as yet unanswered question is whether broad application of CT scanning for blunt chest injuries is cost effective for pulmonary contusion, as management is entirely expectant and treatment entirely supportive. Some studies strongly suggest that contusions identified only on CT scanning are of limited clinical significance [22]. Further, areas of dependent edema, consolidation, or aspiration pneumonitis may be mistaken for contusion. Certainly, if imaging is already done and available, it should be used to guide decision making, but in the tenuous patient, a trip to CT scan may represent an unnecessary risk.

Early evolution of the patient's gas exchange must be taken into account. Patients with early (<6 h) impairment in oxygenation should be approached with caution. The most common tool for describing impairment in oxygenation is the ratio of arterial partial pressure of oxygen (paO_2—the tension of oxygen in the blood) to the percentage (fraction) of oxygen the patient is inhaling (FiO_2—how much oxygen is the patient on). This is commonly referred to as the P/F ratio. At sea level, a normal P/F is about 400. Impairment in gas exchange results in progressively lower values, with mild impairment being <300, moderate <200, and severe <100.

Blunt Cardiovascular Injuries

Like the lung, the heart may be bruised by direct, shear, or blast forces. Since the true "gold standard" for myocardial contusion would be direct examination or biopsy, it is difficult to assess any particular diagnostic approach for sensitivity and specificity. Thus, for practical purposes, one should consider that there are only two common sequelae of blunt cardiac injury: arrhythmia and pump failure. Many of the arrhythmias associated with blunt trauma are relatively benign (sinus tachycardia, atrial fibrillation), and an initial EKG that is normal is associated with a very low chance of a malignant arrhythmia [23]. Thus, an early EKG can be advocated to identify patients at risk. With respect to pump failure, the most common cause is a major contusion of the right ventricle (which lies more anterior), and this typically presents early as hypotension refractory to volume replacement. In these rare cases, early echocardiography can be recommended to confirm the

diagnosis. In patients without clinical evidence of pump failure, the utility and clinical significance of cardiac enzyme measurement, while advocated by some, is a matter of some debate.

Other cardiovascular injuries such as great vessel injury, pericardial rupture, and cardiac rupture are remarkably rare and are largely beyond the scope of this chapter. One injury worth mentioning both in terms of incidence and significance is the torn descending thoracic aorta. While historically described as an immediate threat to life, many of these injuries can be safely observed in the stable patient, and intervention planned for a time when the patient is physiologically well enough to sustain an additional insult [24–26]. Minimally invasive approaches using stent grafts are rapidly replacing operative repair, though long-term follow-up is far from complete. There are some concerns about the ultimate fate of stent grafts placed in young patients, as graft collapse and migration have been described, and it is unclear what will evolve as the young aorta gradually dilates with age and a fixed stent graft remains in place. With respect to operative repair of the torn aorta, many of the risk factors that make any secondary procedure potentially unwise apply: major pulmonary contusion, poor gas exchange, and injuries that would make anticoagulation contraindicated.

Secondary Pulmonary Injury

Acute Respiratory Distress Syndrome (ARDS)

The syndrome of ARDS was outlined in a small series of patients by Ashbaugh and Petty in 1967 [27], and the essence of this description remains today. The main components in the clinical setting include (1) hypoxemia refractory to oxygen administration; (2) diffuse, bilateral infiltrates on imaging of the lungs; and (3) decreased lung compliance. A standard definition of ARDS has been in use for almost 20 years. The Consensus Conference of North American and European investigators (NAECC) agreed that ARDS should be viewed as the most severe end of a spectrum of an acute lung injury (ALI) [28]. The diagnostic criteria for ARDS include acute onset, the PaO_2/F_iO_2 200 mmHg or less (<300 for ALI), bilateral infiltrates on chest radiograph, and no evidence of left atrial hypertension (either clinical or with direct measurement). During the last two decades, some limitations of this definition have been apparent, including an unclear meaning of "acute," the unclear role of transient changes in the P/F ratio in establishing the diagnosis, and potential inclusion of a broad array of patients with hypoxemia. Additionally, recruitment of collapsed lung tissue (predominantly with positive end-expiratory pressure, PEEP) may result in a remarkable improvement in P/F ratio in a short period of time—does this patient no longer have the syndrome ARDS? Lastly, while the NAECC definition excludes patients with left atrial pressure (LAP) >18, Ferguson et al. [29] showed that patients with no risk factors for congestive heart failure but with a clinical syndrome of ARDS commonly had LAP >18.

Table 14.3 Summary of the proposed "Berlin" definition of ARDS

Factor	Description
Onset	Within 1 week of known risk factor
Imaging	Bilateral opacities not explained by effusion, collapse, or nodules
Type of pulmonary edema	Not explained by cardiac failure or fluid overload. If no clinical risk factor identified, objective assessment required
Severity (with PEEP≥5)	Mild: P/F ≤ 300 Moderate: P/F ≤ 200 Severe: P/F ≤ 100

Recently proposed changes to the ARDS definition have been developed using consensus methodology in a series of meetings in Germany. This new "Berlin definition" of ARDS is likely to be widely embraced and offers significant advantages over the 1994 definition [30]. In particular, it defines "acute" more precisely, drops the term ALI (which may be confused as a separate entity), and provides for a larger consideration of precipitating factors when CHF and ARDS may coexist. This modern definition of ARDS is shown in Table 14.3.

Clinical risk factors for ARDS can be broadly categorized into direct and indirect groups. Direct

Table 14.4 Commonly observed risk factors for the adult respiratory distress syndrome

Direct	Indirect
Pulmonary contusion	Severe sepsis
Aspiration	Severe trauma
Pneumonia	Pancreatitis
Pulmonary ischemia/reperfusion	Extrapulmonary (e.g., splanchnic) Ischemia/reperfusion
Fat emboli syndrome	Transfusion

factors are those primarily associated with local pulmonary parenchymal injury and include pulmonary contusion, aspiration, and pulmonary infection. Indirect factors are those thought to be associated with systemic inflammation and resultant lung injury. These include severe sepsis, transfusion of banked red cells, transfusion of FFP, and multiple long bone fractures [31]. Unless shock is associated with significant tissue injury or other known risk factors (e.g., transfusion), it is generally not known to precipitate ARDS. Orthopedic injuries are consistently found to be an independent risk factor for ARDS, particularly in the case of femur fractures [32–35]. Commonly observed risk factors for ARDS are shown in Table 14.4.

We currently understand ARDS as an immuno-inflammatory injury to lung tissue which produces markedly impaired gas exchange [36]. This paradigm posits that both infectious and noninfectious insults initiate a generalized inflammatory response that subsequently injures the lung in an autotoxic fashion. Mediators proposed to initiate this response include danger-associated molecular patterns (DAMPs, released from soft tissue injury), leukocytes/lipid/protein mediators from stored blood components and leukotrienes elaborated from gut lymph. Most relevant to the current discussion is the observation that several components of bone marrow and fracture serum can initiate or exacerbate this phenomenon, including particulate matter, arachidonic acid metabolites, and proinflammatory cytokines [34, 37, 38]. These mediators can initiate indiscriminate activation of effector cells (predominantly macrophages and neutrophils) that subsequently release oxidants, proteinases, and other factors that promote tissue injury.

If the initial insult is severe enough, early organ dysfunction results ("one-hit" or single insult model). More often, a less severe insult results in a systemic inflammatory response syndrome (SIRS) that may not be injurious. These patients appear, however, to be "primed" such that they have an exaggerated response to a second insult, which leads to an augmented/amplified systemic inflammatory response and multiple-organ dysfunction [39]. Fixation of fractures, which may represent additional soft tissue injury, blood loss, and release of mediators from bone marrow/fracture sites, is often thought to be a second insult. In that light, understanding which patients are at risk for ARDS from insults which can be planned may be pivotal in the care of the multiply injured patient.

The characteristic lesion of ARDS affects the interface between alveoli and pulmonary capillaries, with both epithelial and endothelial damage, resulting in a high permeability pulmonary edema. Changes in this lesion—and in the patient's physiology—follow a typical pattern, usually divided into three overlapping phases: (1) the exudative phase, with edema and hemorrhage; (2) the proliferative phase, with organization and repair; and (3) the fibrotic phase [40]. The exudative phase is apparent in the first 3–7 days. Histologic changes include proteinaceous alveolar edema, interstitial edema, and intra-alveolar hemorrhage. The exudative phase is characterized by the appearance of hyaline membranes, which are composed of cellular debris and plasma proteins. Loss of the alveolar epithelial barrier results in alveolar edema, as the remaining cells are unable to drive sodium from the alveolar into the interstitial compartment.

During the proliferative phase, type II cells divide and re-cover the lining of the alveolar wall, beginning about 3 days after the onset of clinical ARDS. Fibroblasts and myofibroblasts proliferate and migrate into the alveolar space in the third phase. Fibroblasts change the alveolar exudate into granulation tissue, which subsequently organizes and forms dense fibrous tissue. Eventually, epithelial cells cover the granulation

tissue. The fibrotic stage is characterized by thickened, collagenous connective tissue in the alveolar septa and walls. Pulmonary vascular changes occur as well, with intimal thickening and medial hypertrophy of the pulmonary arterioles. Complete obliteration of portions of the pulmonary vascular bed can result.

Clinically, ARDS is characterized by tachypnea, hypoxemia refractory to oxygen, and then the development of diffuse, patchy, panlobar pulmonary infiltrates on plain chest radiograph. Computed tomography of the chest will demonstrate that the parenchymal changes are inhomogeneous with the dependent lung regions most affected. Thus, in management of the patient with ARDS, it must be recognized that overall the lung should be considered small (many alveolar spaces are flooded); in fact, the aerated lung volume able to participate in gas exchange may be markedly reduced to one third of the original volume [41]. Further, though the overall lung compliance is diminished, there are actually a variety of airway units ranging from normally compliant to completely collapsed. The inhomogeneous distribution of parenchymal densities led to the concept of a four-compartment model of the lung in ARDS [42]. One compartment is substantially normal (healthy zone), one is fully diseased without any possibility of recruitment (diseased zone), a third compartment is composed of collapsed alveoli potentially recruitable with increasing pressure (recruitable zone), and, finally, a fourth compartment contains overdistended airway units.

Because there is no proven specific treatment for ARDS, therapy primarily involves supportive measures to maintain life while the lung injury resolves. Such measures include identifying and treating predisposing conditions, mechanical ventilatory support with oxygen, nutritional support, nonpulmonary organ support, and hemodynamic monitoring as necessary. Attention to detail is necessary to avoid nosocomial infection and iatrogenic complications. Increased airway pressure is necessary to recruit collapsed alveoli, and thus application of positive pressure ventilation is key in supporting patients with severe ARDS. As early as the 1990s, however, it was recognized that in the heterogeneously injured lung, airway pressure or stretch may be damaging to the healthy zone. This ventilator-induced lung injury is now thought to be responsible for severe protracted ARDS, as well as perpetuation of systemic inflammation and multiple organ failure. This is thought to be why ventilator strategies that minimize volume and pressure are associated with decreased mortality in ARDS.

Currently, ventilator-induced lung injury can be thought of in terms of stress-related injury and strain-related injury [43, 44]. Stress can be thought of as tension on the lung skeleton related to static distension (transpulmonary pressures); higher pressures produce injury by overdistending normally compliant units. Strain can be thought of deformation of lung units through the respiratory cycle, including the potential for repetitive "opening" and "closing" of alveoli. This is related to tidal volumes used and the end-expiratory volume. Of note, since lung units share walls, and one unit may not have the same compliance as its neighbor, strain can result from the interaction of two or more adjacent units. Both stress and strain are thought to potentiate ongoing lung inflammation.

ARDS network trials published in 2006 addressed appropriate fluid management in patients with ARDS. In this study, a total of 1,000 patients was randomized to either liberal or conservative fluid strategies over a period of 7 days [45]. The conservative group received approximately a net one liter less per day and spent 2.5 fewer days on the ventilator. There was no mortality difference and no increase in other organ failures in the conservative group. While this may not be considered a profound effect related to fluid, it is one of very few "positive" trials in the ICU setting; it can be concluded that a conservative approach to volume administration is safe and associated with some improvement in outcome. The modest effect observed may relate to the fact that pressure-limited ventilation trumps any major effect of fluid balance in this patient population. With respect to colloids, while conceptually attractive, there is little evidence that their routine use for acute resuscitation improves outcome.

Fat Emboli Syndrome

Extravasation of bone marrow into the venous system can result in a striking syndrome that includes severe pulmonary failure. Approximately 75 % with this clinical syndrome will have acute or subacute impairment of oxygenation, presumably due to microparticles of fat that obstruct pulmonary vessels or produce vasomotor dysfunction. Global CNS dysfunction mimicking encephalopathy is common; while it can be dramatic, it generally resolves without permanent sequelae. Petechial rash of the upper torso, axillae, oral mucosa, or other sites is present in a minority of described cases. Hematologic changes, including acute unexplained anemia and thrombocytopenia, are relatively common. Fever, tachycardia, and tachypnea are common but nonspecific findings. While no diagnostic test has adequate sensitivity, the finding of fat globules in urine (present in a minority of cases) is considered confirmatory. Like ARDS, this syndrome is a constellation of symptoms and signs without a "gold standard" test, and there may be considerable overlap between these two conditions in the multiply injured patient. While three different sets of diagnostic criteria have been proposed, the 1974 description by Gurd and Wilson is the most commonly cited. In this composite, one major and four minor findings can be used to make the diagnosis [46].

Definitive fixation of long bone fractures is the most common risk factor for fat emboli syndrome. Prospective evaluation of patients at risk identifies the syndrome in as many as 10 % of patients [47, 48]. Interestingly, observation by transesophageal echocardiography suggests that particulate matter in the right heart is quite common in this scenario, yet only a fraction of patients with this finding go on to have clinical manifestations. While the involvement of the pulmonary circulation is somewhat intuitive, the mechanism by which fat in the pulmonary circulation creates systemic manifestations attributable to the systemic circulation (brain, skin, kidneys) is unclear. Some patients have an identifiable anatomic right to left shunt (such as patent foramen ovale), yet in others, fat—or biologically active catabolites—presumably reaches the system circulation by moving through pulmonary capillary beds or around those beds via anatomic intrapulmonary shunts.

Implications on Timing of Fracture Fixation

Given that the patient with the combination of chest and nonthoracic bony injuries is at high risk for pulmonary failure, the matter of fracture fixation timing has been an area of intense scrutiny. Conceptually, fracture fixation may represent a "second hit" that could convert a patient's systemic inflammatory response into an autotoxic state of ARDS and multiple organ failure. On the other hand, fracture fixation may reduce bleeding, bone marrow release, pain-associated systemic manifestations, and further tissue injury. Concerns about the second insult phenomenon appear warranted based on both animal models and human observations of increased circulating proinflammatory cytokines, increased SIRS, and increased pulmonary dysfunction after fracture fixation. Furthermore, the inflammatory response to external fixators applied in a damage control fashion is markedly blunted compared to intramedullary fixation [38, 49, 50]. This would suggest that a damage control approach should be safer in patients at risk for ARDS and multiple organ failure.

Despite these findings, comparative studies of early versus late fracture fixation in multiply injured patients have produced divergent results, ranging from studies suggesting that early fixation is beneficial to studies suggesting it is harmful [51, 52]. In the case of femur fractures, a recent review of the literature captures the lack of clarity quite nicely. In the eight high-quality studies identified where incidence of periprocedural ARDS was reported, exactly half favored early definitive treatment, whereas half suggested no difference. In the studies reporting length of stay and mortality data, the majority favor early definitive treatment [53]. Thus, the debate continues over which patients should receive "early total care" for fractures and which should undergo a "damage control" procedure.

Given the lack of clarity regarding timing of fracture fixation in *populations* of multiply injured patients, a selective approach to the *individual* patient appears highly advisable. Understanding which patients with chest injury are most at risk is pivotal to this concept. In that light, a number of factors and scoring systems have been reported to predict outcome. Mommsen et al. [6] evaluated 278 patients with chest injuries and an ISS >15 admitted to a single regional trauma center. They studied whether abbreviated injury scores for the chest, scores related to pulmonary contusion, or the thoracic trauma score (TTS, which combines both anatomic and physiologic parameters) best predicted clinical outcomes in this set of patients. The anatomic score (AIS) was least predictive of clinical outcomes, whereas the TTS, using a cut-off of 9, was most predictive. For example, TTS >9 was associated with an almost fivefold increase in the result of ARDS, sixfold increase in the likelihood of MOF, and a fourfold increase in the likelihood of death. The components of the TTS are P/F ratio, severity of pulmonary contusion, injury involving the pleurae, number of rib fractures, and age of the patient.

Battle et al. [54] published a meta-analysis in 2012 attempting to describe which factors in chest wall injury were best predictive of mortality. These authors made a concerted effort to include both mild and severe injuries, with the goal of reliably predicting patients who might be considered on a safe clinical trajectory (and might be managed as outpatients) and those who might be considered at risk. While this is an imperfect parallel to the multiply injured patient, it may help inform our decisions. They observed that the following predicted mortality: patients older than 65 (odds ratio = 2), patients with three or more rib fractures (odds ratio = 2), and presence of preexisting conditions (odds ratio = 2.3). Pneumonia was the best predictor of mortality, though for practical purposes this is a late occurrence and thus can rarely inform our decision about fracture care.

Wutzler et al. [55], utilizing the German trauma registry, analyzed 5,892 patients with pulmonary contusions or lacerations and ISS >15 admitted to the ICU. Using the lung component of a standard organ failure score as an end point, they sought to identify patients most at risk for severe pulmonary failure. In multivariate analysis, age, ISS, male gender, and >1 surgical intervention independently predicted severe pulmonary failure. While this is a large retrospective study, it might be criticized for including patients with severe head injuries; in this population of patients, prolonged coma and high incidence of pneumonia may dominate many other considered variables.

The task of identifying patients at the highest risk for perioperative complications has been uniquely championed through a series of outstanding works by Pape and associated investigators over the last 20 years. These investigators have matured a set of criteria that define the "at risk" or "borderline patient" in whom the chance of exacerbating systemic inflammation appeared unacceptably high for definitive orthopedic care and wherein a damage control approach is preferred. Sometimes referred to as the "Hannover criteria," one iteration of the components are as follows:(1) polytrauma with thoracic trauma (ISS >20 and chest AIS >2), (2) polytrauma with abdominopelvic injury (AIS >3 for this region) and shock (SBP <90), (3) ISS >40, (4) bilateral lung contusion, (5) pulmonary hypertension (mean pulmonary artery pressure >24), or (6) increase in mean pulmonary artery pressure of >6 during intramedullary nailing. These have substantial face validity, yet suffer from some practical drawbacks, including the fact that ISS is often undetermined early in the patient's course and pulmonary artery catheters have all but disappeared from routine use in the trauma setting. Using a modification of these criteria, a multi-institutional randomized study of damage control versus early definitive care was published in 2007 [49]. Interpretation of this "EPOFF" study is hampered somewhat by disparities in injury severity and concomitant brain injury in the (randomized) groups, yet it was observed that for patients in "borderline" condition, there was an increase in morbidity in the group undergoing an early definitive care approach. Specifically, the risk of acute lung injury in the early total care group was sixfold higher.

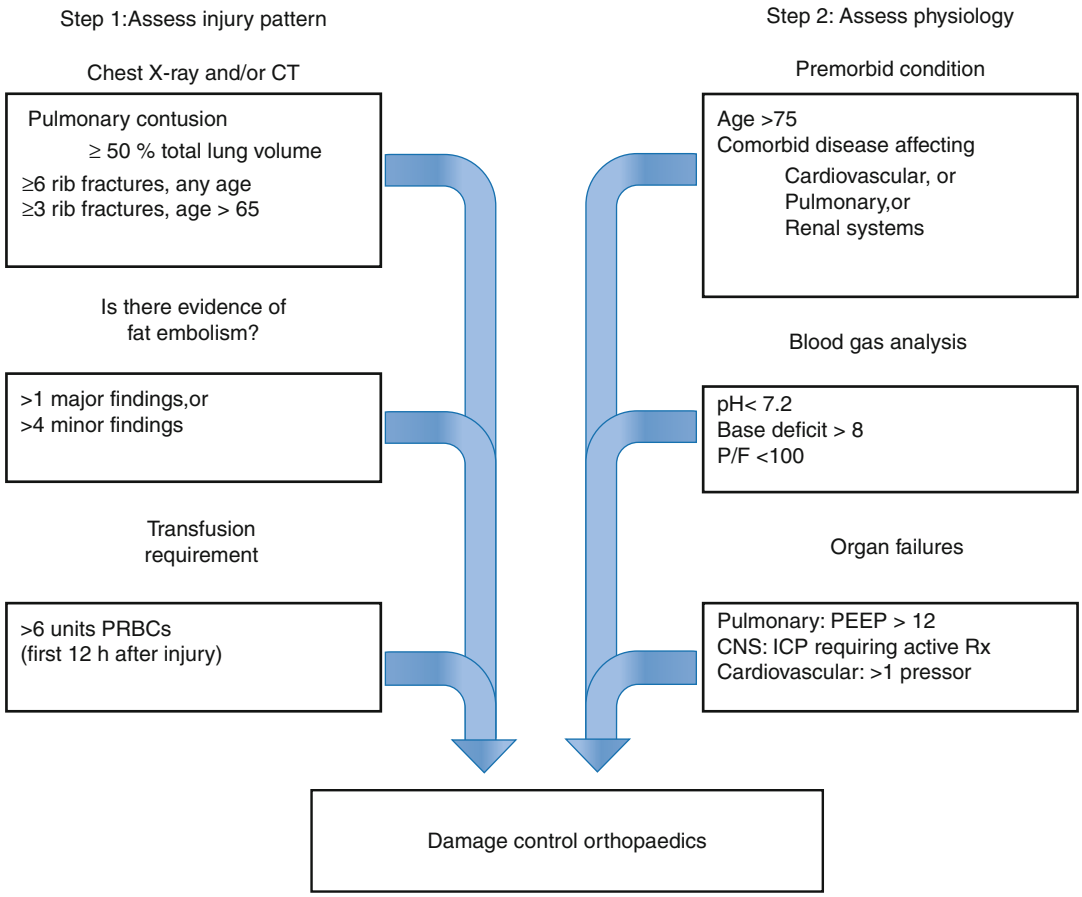

Fig. 14.1 Factors known to increase the risk of cardiopulmonary morbidity in the multiply injured patient with orthopedic injuries. It is proposed that the presence of any one of these factors should warrant consideration of a damage control approach to fracture care

Now that the reader understands the physiology of chest injury and the patients most at risk for secondary pulmonary injury, an algorithm-based approach can be proposed (Fig. 14.1). This will include our current understanding of the role of rib fractures, pulmonary contusion, ARDS risk factors, and physiologic parameters that might make major operative intervention unsuitable. Synthesizing the material up to this point, we can combine known risk factors to define the at risk patient. The following approach is proposed, concentrating on clinical variables that are in routine use or can be easily and rapidly calculated. The risk factors are subdivided into injury pattern and patient factors known in the early (first 6 h) phase of resuscitation. Based on current understanding of the literature, it can be stated that the presence of any one of these factors should prompt strong consideration of a damage control approach to fracture fixation in the patient with chest injuries. To be sure, this is not an exhaustive list of factors that should delay definitive fixation, yet it provides an approach that captures the majority of common events in the multiply injured patient.

The first consideration should be the pattern of injury, specifically evidence of pulmonary contusion, major chest wall injury, major hemorrhage, or fat emboli syndrome. With respect to contusion, those involving 50 % or more of the lung parenchyma suggest the patient will go on to have major gas exchange abnormalities in the ensuing

48 h and would therefore be unsuitable for early total fracture care. It is reasonable to place more weight on this finding when the contusion is evident on a plain radiograph as opposed to chest CT as the latter is clearly more sensitive. Pulmonary contusions evident on later (>24 h postinjury) radiographs may have less clinical importance as they may reflect the development of dependent atelectasis, aspiration pneumonitis, or the blossoming of an earlier smaller contusion.

Major chest wall injuries are a risk factor for early pulmonary dysfunction, in part because they are frequently associated with underlying pulmonary contusions and lacerations. Factors to take into consideration include age, number of rib fractures, and the likelihood of pulmonary failure. In aggregate, 3 or more rib fractures in a patient >65 or 6 or more fractures in any patient should warrant a damage control approach to orthopedic injuries.

In regard to hemorrhage, early (first 12 h) red cell transfusion is an important marker; furthermore, there is a linear relationship between number of units transfused in this time period and the risk for ARDS and multiple organ failure [56]. A precise threshold effect is not evident, yet for practical purposes a working "cutoff" for high risk of transfusion-associated ARDS would be useful; in this author's opinion, a 6-unit transfusion (first 12 h) is indicative of a substantial risk of subsequent organ failure; thus, delay of definitive orthopedic repair is the optimal approach. This is not to say that fracture fixation should not be pursued—some form of stabilization is likely to minimize ongoing blood loss. Finally, an injury pattern that produces the syndrome associated with fat embolization early in the patient's course should warrant fracture fixation with external devices as opposed to an intramedullary approach.

The second consideration in making a decision about early total care versus a damage control approach is patient factors. The emphasis should be on those that affect the cardiopulmonary reserve an individual patient. As previously described, the metabolic demand of postinjury physiology requires dramatic increases in carbon dioxide excretion. Furthermore, patients who fail to mount a hyperdynamic cardiovascular response appear to be at substantially increased risk of later organ failure [57]. This is likely a combination of diminished reserve and cardiovascular depressant factors present in the cytokine milieu in the critically injured patient. In any event, elderly patients and/or those with preinjury pulmonary, cardiac, or renal insufficiency are at higher risk and may tolerate major operative interventions poorly in the early postinjury period. To assess the patient at risk, pay particular attention to the early physiology—this is highly predictive of subsequent trajectory. This is both because a "second hit" may be injurious and because some patients are placed at risk simply from trying to transport them to an operating room environment. Five factors are proposed for consideration here: current pH, base deficit, P/F ratio, PEEP requirement, intracranial hypertension, and cardiovascular failure.

In the "at-risk" patient, arterial blood gas analysis is an invaluable insight into the current physiology of the patient. The particular elements of interest include the current pH, the base deficit, and the current pO_2 (and thus P/F ratio). With respect to pH, a patient who remains acidemic despite resuscitation is unlikely to tolerate further insults; $pH < 7.2$ suggests a damage control approach is warranted. All organ systems exhibit dysfunction at deranged pH and cardiopulmonary compromise; renal dysfunction and hepatic insufficiency can be expected in this scenario.

Base deficit can be thought of as the amount of base that would need to be given to regain a normal pH with normal pCO_2. It is a surrogate for the depth and duration of cellular shock. An elevated base deficit in the trauma setting implies impaired oxygen delivery/utilization in tissue beds, with resultant anaerobic production of lactate. Certainly other coexistent factors can produce a base deficit, and other measures such as lactate/pyruvate ratios or near-infrared spectroscopy can be used. Since blood gas analysis is so rapid and so widely available, in many ways it is the single best test to determine the patient's current state and likelihood of subsequent morbidity and mortality. An early (<6 h) base deficit of eight or more is independently associated with

organ failure and death and should prompt consideration of a damage control approach. Again, this is not to suggest that fracture fixation should not be pursued—merely that it should be done in a way that puts the patient at minimal risk.

The remaining patient factors that warrant a damage control approach include evidence of early organ failure. With respect to pulmonary failure, the arterial pO_2 gives us a measure of gas exchange and must be strongly considered as well. Patients' meeting criteria for severe ARDS (Berlin criteria, P/F < 100) are unlikely to tolerate additional insults and should be approached with caution. For practical purposes, a patient requiring an FiO_2 >0.6 to maintain oxygen saturations >90 % will fall in this category. Additionally, patients who require moderate or high levels of PEEP are likely to have very limited pulmonary reserve; a threshold of 12 is proposed. In most centers, it is not practicable to maintain such levels of PEEP during transport and anesthesia. While bag mask ventilators may have PEEP valves, and some transport and anesthesia ventilators can deliver advanced modes and pressures, attempts at transport to the operating room are likely to be met with de-recruitment of alveoli to a point where maintaining oxygenation is problematic. Creative strategies such as bedside external fixators in the ICU should be considered.

For CNS organ dysfunction, patients with intracranial hypertension requiring active treatment also fall into this category of transport risk as head elevation, maintenance of eucapnia, and judicious fluid administration are necessary. Lastly patients with severe cardiovascular instability (as measured by the requirement of more than one pressor) are poor candidates for definitive fixation.

Conclusion

In conclusion, patients with the combination of major chest injuries and fractures requiring orthopedic fixation represent some of the most challenging multitrauma patients. This overview may provide the reader with an improved understanding and approach. With our understanding of the interplay between the physiology of chest injury and the response of individual patients, the above guidelines can serve to guide clinicians on critical decision in individual patients. The author suggests a stepwise approach, considering injury pattern and early (patho) physiology.

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