

Anesthetic Considerations and Ventilation Strategies in Cardiothoracic Trauma

Craig S. Jabaley¹ · Roman Dudaryk² · Charles E. Smith³

Published online: 5 February 2016
© Springer Science + Business Media New York 2016

Abstract Cardiothoracic trauma represents a significant challenge for the anesthesiologist, and little robust investigational evidence exists to guide management decisions. Injury patterns are potentially diverse and often severe, and mortality rates are second only to those of traumatic brain injury. As such, anesthesiologists must be familiar both with the fundamentals of trauma patient care and considerations unique to cardiothoracic injury. Herein, we discuss anesthetic considerations related to traumatic injury of the heart, great vessels, conducting airways, and lungs. As the rate of respiratory failure and acute respiratory distress syndrome (ARDS) exceeds that of the general trauma population, we then review the

relevant fundamental aspects of mechanical ventilation and treatment of ARDS.

Keywords Thoracic trauma · Trauma anesthesia · Thoracic aortic injury · Blunt cardiac injury · Airway trauma · Pulmonary contusion

Introduction

The management of blunt thoracic trauma from both the anesthetic and surgical standpoints is complex owing to the unique challenges of certain injury patterns and the diverse nature of traumatic injury. Discussion regarding the perioperative management of these patients has been limited in the literature and is further constrained by a narrow foundation of investigational evidence. Thoracic trauma often involves inherent challenges that require high levels of expertise in the realms of airway management, interdisciplinary communication, perioperative mechanical ventilation, lung isolation, recognition and diagnosis of occult injuries, and treatment of obstructive shock. As such, the management of these patients involves concepts that extend beyond the conventional core trauma anesthesia topics of resuscitation, transfusion, and correction of coagulopathy. Mortality associated with thoracic trauma is second only to that caused by traumatic brain injury and is a leading preventable contributor to post-traumatic death [1]. To simplify the discussion, injuries to the heart and major vessels will be discussed separately from those to the lungs and conducting airways. We then conclude with a review of mechanical ventilation and acute respiratory distress syndrome (ARDS).

This article is part of the Topical Collection on *Anesthesia for Trauma*.

✉ Craig S. Jabaley
csjabaley@emory.edu
Roman Dudaryk
rdudaryk@med.miami.edu
Charles E. Smith
csmith@metrohealth.org

- ¹ Division of Critical Care Medicine, Department of Anesthesiology, Emory University Hospital, 1364 Clifton Road, NE, Atlanta, GA 30322, USA
- ² Department of Anesthesiology, Perioperative Medicine, and Pain Management, University of Miami/Jackson Memorial Hospital Ryder Trauma Center, 1611 NW 12th Avenue (C-301), Miami, FL 33136, USA
- ³ Department of Anesthesiology, MetroHealth Medical Center, Case Western Reserve University School of Medicine, 2500 MetroHealth Drive, Cleveland, OH 44144, USA

Vascular and Cardiac Trauma

Thoracic Aortic Injury

While penetrating injury may affect any segment of the thoracic aorta, blunt trauma most commonly occurs distal to the takeoff of the left subclavian artery where the proximal descending aorta is anchored by the ligamentum arteriosum at the aortic isthmus [2]. This relatively immobile segment is therefore susceptible to injury from abrupt deceleration. Blunt trauma can also manifest as injury throughout the entire continuum of the aorta. The incidence of blunt thoracic aortic injury is approximately 2 % of all patients who sustain thoracic trauma with the majority (80 %) being caused by motor vehicle collisions [3, 4]. Patients who sustain thoracic aortic injury after blunt trauma are typically severely injured owing to the high energies involved [5]. As demonstrated in one prospective series, operative repair has historically carried a high rate of mortality (31 %) and paraplegia (8.7 %); however, mortality in patients managed non-operatively was even greater at 55 % [6].

Initial excitement regarding the role of TEE in the diagnosis of acute aortic injury has subsided, and multi-detector computed tomography (CT) has emerged as the most common diagnostic modality. The sensitivity and specificity of CT with regard to the diagnosis of aortic injury approaches 100 % throughout the entire aorta. Further, CT is a critical tool to detect and catalog traumatic burden and reduces the likelihood of occult injury overlooked during clinical examination. The addition of intravenous contrast to facilitate CT angiography is easily accomplished and can be performed and interpreted rapidly at any time of the day. In contrast, TEE requires the presence of an experienced operator, and many institutions cannot support the continuous availability of a TEE service. Furthermore, it is often impractical to perform TEE in the resuscitation bay or emergency department, and TEE is ill-suited to rule out the gamut of potential non-vascular injuries. Compared to CT, TEE cannot visualize all portions of the ascending aorta due to the interposed air-filled distal trachea and left bronchi. Lastly, TEE is relatively contraindicated in the setting of unstable cervical spine injuries, severe craniofacial trauma, and suspected esophageal injury. Despite these shortcomings, TEE remains an invaluable intraoperative tool when cardiac or major vascular injury is suspected in patients brought emergently to the OR without previous CT imaging. Use of TEE in this scenario may quickly yield a potentially life-saving intraoperative diagnosis.

In addition to greater diagnostic accuracy, CT imaging also facilitates patient triage. This damage control

radiology approach uses CT to identify sites of bleeding and facilitate triage to either non-operative management, operative intervention, or hemorrhage control by interventional radiology [7, 8]. While current guidelines suggest that patients with intimal tears can be managed non-operatively, those with intramural hematomas, pseudoaneurysms, or contained rupture (periaortic hematoma) will likely require intervention [9]. Given the potential for high morbidity in the severely injured patient, surgical intervention can be delayed when the risk of rupture is low (e.g., no evidence of pseudoaneurysm) [10]. Numerous approaches to the open repair of thoracic aortic injuries have been described [11]. Over the last decade, the management of trauma to the great vessels has shifted from open to endovascular approaches such that open repair of aortic injury is becoming increasingly rare. Endovascular repair has been associated with lower mortality, decreased incidence of paraplegia, and reduced need for blood transfusion when compared with open approaches [12, 13].

The principles of anesthetic management for patients undergoing post-traumatic thoracic aortic repair are similar to those for associated elective procedures. Systemic blood pressure must be maintained within a narrow range to avoid worsened hemorrhage or extension of injury. In the absence of head injury, a systolic blood pressure of less than 100 mmHg is typically targeted. Although easily titratable agents (e.g., clevidipine, nicardipine, sevoflurane, and/or remifentanyl) can be employed for elective cases, the hemorrhaging trauma patient may require only close attention to hemodynamics during perioperative resuscitation. Likewise, beta blockade has long been a cornerstone of aortic injury management, as reduction in both the chronotropic and inotropic state of the heart is thought to reduce shear forces (i.e., the rate pressure product) and protect the site of injury. Esmolol has a favorable pharmacokinetic profile that affords a relative margin of safety in the compromised trauma patient. Periods of controlled hypotension, with mean arterial pressures of 50–55 mmHg, are sometimes required during endovascular stent deployment; however, the development of low-profile stents has largely obviated this need. The relative merits of controlled hypotension must be weighed against the risks when concomitant traumatic brain injury is known or suspected [14].

Open repairs of the aortic arch and ascending aorta typically require cardiopulmonary bypass and may require a period of circulatory arrest. On the other hand, open repair of the descending aorta requires one-lung ventilation (OLV) to facilitate exposure of the descending thoracic aorta. Placement of a single-lumen endotracheal tube (ETT) affords the greatest chance of timely first-pass success when a definitive airway is required emergently either in the resuscitation bay or operating room (Table 1). The

placement of a double-lumen tube may be challenging in the trauma patient due to cervical spine immobilization, airway contamination, and risk of aspiration. Moreover, tracheal anatomy may be distorted or externally compressed by a periaortic hematoma, which can complicate tube exchange and positioning. Furthermore, ETT exchange is typically ill-advised in the trauma patient as coagulopathy, airway edema, and limited pulmonary reserve are common. As such, a bronchial blocker is often the most expeditious and safest means by which to accomplish lung separation and OLV under emergent conditions after placement of a single-lumen ETT.

Blunt Cardiac Injury and Commotio Cordis

Blunt cardiac injury (BCI) should be suspected in patients with a decelerating mechanism of injury. Its overall incidence following thoracic trauma is difficult to discern

owing to inconsistent diagnostic criteria over the years; however, it is likely between 10 and 25 % with a proportional increase in likelihood that parallels injury severity [15, 16]. The diagnosis of BCI is challenging, and its sequelae can vary substantially. The spectrum of BCI can include cardiac contusion (with cardiac enzyme elevation and wall motion abnormalities), arrhythmia, acute coronary syndrome, valvular disruption, cardiogenic shock, and potentially catastrophic structural damage (Table 2). Comprehensive screening guidelines for BCI are available from the Eastern Association for the Surgery of Trauma (EAST) [17]. EAST guidelines recommend an admission EKG for all patients with suspicion of BCI. However, a normal EKG does not exclude BCI as was demonstrated in one study wherein 41 % of patients with abnormal echocardiographic findings had a normal EKG [18]. The addition of normal troponin I levels to an unremarkable EKG confers a negative predictive value of almost 100 %

Table 1 Advantages and disadvantages of selected lung isolation techniques

Options	Advantages	Disadvantages
Double-lumen tubes	<ul style="list-style-type: none"> • Quickest to place successfully under elective conditions • Repositioning rarely required • Facilitates bronchoscopy and suction of the isolated lung • CPAP easily added • Can alternate OLV to either lung easily • Placement still possible if bronchoscopy not available 	<ul style="list-style-type: none"> • Size selection more difficult • Challenging to place in the context of a difficult airway or abnormal trachea • Non-optimal postoperative two-lung ventilation • Risk of bronchial and/or laryngeal trauma
Bronchial blockers	<ul style="list-style-type: none"> • Size selection rarely an issue • Easily added to regular ETT • Allows ventilation during placement • Easier placement in patients with difficult airways and in children • Bilateral blocker balloons allow for sequential lung isolation without repositioning 	<ul style="list-style-type: none"> • More time needed for positioning • Repositioning needed more often • Bronchoscope essential for positioning • Non-optimal right lung isolation due to RUL anatomy • Limited availability and practical experience at some centers
1. Arndt		
2. Cohen		
3. Fuji		
4. EZ Blocker		
Univent	<ul style="list-style-type: none"> • Same as for bronchial blockers • Less repositioning than with bronchial blockers 	<ul style="list-style-type: none"> • Same as for bronchial blockers • Higher air flow resistance than regular ETT • Larger diameter than regular ETT
Endobronchial tubes	<ul style="list-style-type: none"> • Like regular ETTs, easier placement in patients with difficult airways • Longer than regular ETT • Short cuff designed for lung isolation • Tube is reinforced 	<ul style="list-style-type: none"> • Bronchoscopy necessary for placement • Does not allow for bronchoscopy, suctioning, or CPAP to isolated lung • Difficult right lung OLV
Endotracheal tube advanced into mainstem bronchus	<ul style="list-style-type: none"> • Easier placement in patients with difficult airways 	<ul style="list-style-type: none"> • Does not allow for bronchoscopy, suctioning, or CPAP to isolated lung • Cuff not designed for lung isolation • Difficult right lung OLV

Modified from [124] with permission from Cambridge University Press. ©2015 Cambridge University Press, NY
 CPAP continuous positive air pressure, ETT endotracheal tube, RUL right upper lobe, OLV one-lung ventilation

[19]. Contrary to previous beliefs, sternal fractures have not been reliably associated with BCI, and their presence or absence is not incorporated in current screening schema.

Management of BCI depends largely on the exact manifestations of injury. Patients with a concerning mechanism of injury, hemodynamic instability, and a positive FAST examination suggestive of tamponade will undergo immediate operative exploration. Formal echocardiography has been shown to have limited screening utility and is typically reserved for patients with hemodynamic compromise and arrhythmia [17]. Patients with BCI may come to the attention of an anesthesiologist during preoperative evaluation. Heightened perioperative mortality has been described for patients with BCI on an inconsistent basis in the literature; however, mortality was typically due to non-cardiac causes [20–22]. The ideal length of time, if any, to delay non-emergent surgery is unclear from the existing evidence and should rely on patient-specific factors and clinical judgement. In any case, the anesthesiologist must be vigilant as both arrhythmias and pump dysfunction with cardiogenic shock are known sequelae of BCI [21]. At a minimum, the anesthesiologist must be prepared to treat arrhythmias, such as ventricular tachycardia, ventricular fibrillation, atrial fibrillation with rapid ventricular response, conduction blocks, and heart failure. A defibrillator should be immediately available, and prophylactic placement of transcutaneous electrodes (i.e., pads) may be prudent. Non-emergent surgery should be delayed in the setting of clinically significant arrhythmias and/or heart failure.

Comotio cordis describes the development of malignant (e.g., ventricular) arrhythmia and cardiac arrest

following forceful impact to the precordium. The classic scenario involves young athletes injured while playing contact sports. Mechanical energy imparted to the myocardium during repolarization is thought to be the underlying mechanism; however, specific risk factors have not been fully elucidated [23]. The only intervention that has been associated with favorable outcomes is the immediate initiation of CPR and defibrillation [24]. Adverse legal consequences are not uncommon for involved parties and in instances of delayed diagnosis and treatment [25].

Considerations for Resuscitative Thoracotomy

Resuscitative thoracotomy (RT) in the ED and aortic cross-clamping are maneuvers that can be potentially life-saving for certain patients. However, efforts to establish evidence-based indications for this dramatic procedure have been frustrated by significant heterogeneity and limitations in the existing literature. The best available evidence supports the use of RT for patients who present to the ED with signs of life followed by rapid deterioration or cardiac arrest, especially for those with thoracic penetrating trauma or likely cardiac injury and tamponade [26, 27]. The use of RT following either out of hospital witnessed arrest and CPR, or CPR immediately on arrival by EMS, is more controversial [28]. However, Western Trauma Association guidelines argue that rapid transport may confer increased survivability in certain scenarios [29]. On this basis, the guidelines state that RT is potentially indicated following blunt trauma with less than 10 min of prehospital CPR, penetrating torso trauma with less than 15 min of prehospital CPR, and penetrating neck or extremity trauma with less than 5 min of prehospital CPR. In keeping with these findings, a recent review concluded that the RT is likely most beneficial for pulseless patients with signs of life after penetrating thoracic injury (e.g., likely tamponade) and least likely to be beneficial or pulseless patients without signs of life following blunt trauma [30].

RT is typically accomplished via a left anterolateral approach. For the anesthesiologist, endotracheal intubation is frequently performed concurrently. Visualization of lung expansion is a valuable means by which to confirm correct placement of the ETT as typical indicators (e.g., end-tidal carbon dioxide) will be unreliable. As patients are moribund, attention to analgesia and amnesia can be delayed initially. In the event that RT is successful in restoring signs of life, immediate preparations should be undertaken to facilitate ongoing resuscitation and prepare the operative suite. Temporary cannulation of the right atrial appendage and descending thoracic aorta have both been described as means by which to facilitate fluid resuscitation following RT when standard methods of vascular access prove to be impossible [31, 32].

Table 2 Clinical manifestations of blunt cardiac injury

Arrhythmias
Sinus tachycardia
Sinus bradycardia
Conduction blocks (e.g., right bundle branch block, third degree block)
Atrial fibrillation
Premature ventricular contractions
Ventricular tachycardia or fibrillation
Decreased cardiac function
Heart failure
Cardiogenic shock
Pericardial effusion and tamponade
Wall motion abnormalities
Valvular disruption
Cardiac chamber rupture
Elevated cardiac troponins

Modified from [125] with permission from Cambridge University Press. ©2008 Cambridge University Press, NY

Anesthetic Considerations for Damage Control Strategies

“Damage control” was originally used to describe the immediate but temporary correction of damage sustained by naval vessels to prevent loss of the ship through simple practical maneuvers. The term was first applied to trauma patients in the context of damage control laparotomy, whereby generous exposure was obtained, packs were placed to abate hemorrhage, and a temporary closure was effected [33]. Thereafter, the patient would be admitted to the ICU for resuscitation, correction of coagulopathy, and further optimization prior to definitive surgical intervention. A similar approach has been described for penetrating thoracic trauma, although mortality remains high (23–40 %) even at experienced centers [34–36]. A related concept is that of damage control resuscitation (DCR), which emphasizes means by which to facilitate early hemostasis. In addition to early surgical control of bleeding, the cornerstones of DCR are initial deliberate hypotension; resuscitation with transfusion of blood products rather than crystalloid fluids; avoidance and treatment of coagulopathy, acidosis, and hypothermia; and goal-directed delayed resuscitation [37–39].

One recently developed technique for hemorrhage control is resuscitative endovascular balloon occlusion of the aorta (REBOA), which acts as an endovascular aortic cross-clamp equivalent [40]. REBOA can be employed as a temporizing and life-saving measure in severely injured patients who continue to manifest profound hemodynamic instability and impending cardiac arrest despite aggressive resuscitation. Conceptually, deployment of a REBOA catheter will allow time to catch up with resuscitation while making arrangements for definitive hemorrhage control or further diagnostic studies. The success of this approach is likely patient and operator-dependent, and a recent prospective multicenter observational study demonstrated an increased risk of death in patients who underwent REBOA [41]. It has been argued that REBOA is likely contraindicated in the setting of thoracic hemorrhage as it could exacerbate bleeding from the great vessels [42]. Further experience and research is needed to better define the role of this technique for patients with thoracic injuries [43].

Pulmonary and Airway Trauma

Pneumothorax and Parenchymal Injury

The diagnosis of pneumothorax is typically made during the primary survey owing to obvious clinical signs or following radiographic studies as an element of the secondary survey. Occult pneumothorax that is not detected by plain

chest radiography, but is evident on CT scan, occurs in up to 16 % of all trauma patients and in 30 % of blunt chest trauma [44]. Point of care ultrasound is gaining increasing acceptance as the most expeditious means by which to diagnose pneumothorax in the prehospital and hospital settings with higher sensitivity and specificity than a plain chest radiograph [45, 46]. Similarly, ultrasound can facilitate intraoperative detection of pneumothorax by anesthesiologists [47]. The conventional approach of tube thoracostomy following the detection of even a small (e.g., less than 1.5 cm at the 3rd rib) or occult pneumothorax has been challenged as evidence mounts that patients with a small unilateral asymptomatic pneumothorax can be managed expectantly even in the setting of positive pressure ventilation [48–50]. However, this approach may prove to be inadvisable in the polytrauma patient slated to undergo emergent operative intervention. The frenetic intraoperative environment may lead to delayed appreciation of worsening pneumothorax on the basis of increased airway pressures (during volume-controlled ventilation [VCV]), reduced tidal volumes (during pressure-controlled ventilation [PCV]), impaired gas exchange, or hemodynamic compromise. As such, pre- or intraoperative tube thoracostomy is often the safest option in this circumstance.

Bleeding from the peripheral low-pressure pulmonary circulation resulting in hemothorax is typically self-limited. Tube thoracostomy in this setting is both therapeutic and diagnostic as the hemothorax can be evacuated and further bleeding monitored through suction of the pleural space. While bleeding from the lung parenchyma is often modest, intercostal, internal mammary, or hilar injury can result in brisk hemorrhage. The vast majority of pulmonary injury from blunt thoracic trauma is managed conservatively with tube thoracostomy, and less than 10 % of injuries require intervention [51]. Patients are typically taken to the operating room due to a massive air leak or uncontrolled bleeding, which is defined as a chest tube output exceeding 1500 ml/24 h or 3 consecutive hours of output >200 ml/h [52]. For the anesthesiologist, it is important to monitor chest tube output in the trauma patient who is undergoing repair of other coexisting injuries and alert the trauma team should the output exceed these thresholds.

Tracheobronchial Injury

Tracheal injury is rare but exceedingly challenging to manage both from a surgical and anesthetic perspective. As is frequently noted, the true incidence of such injuries is difficult to discern as many patients die promptly at the scene. Penetrating neck trauma typically entails injury to the cervical portion of the trachea as this segment is relatively poorly protected anatomically. Conversely, blunt injury typically entails either injury within 2 cm of the carina

(80 %) or at the laryngotracheal junction (20 %) as these two segments are vulnerable to crush injury by overlying osseous structures or barotrauma with a closed glottis, respectively [53]. In an otherwise stable patient, tracheal injury may present as unexplained dyspnea and subcutaneous emphysema (Table 3). Clinical suspicion should be further heightened when pneumothorax or pneumomediastinum is refractory to conventional management.

Patients with tracheobronchial injuries often require intubation due to hemodynamic instability and concomitant injuries before a diagnosis can be made. Care is warranted while inserting the ETT as intubation can either exacerbate the injury or create a false lumen. As such, when reasonable clinical suspicion for a tracheobronchial injury exists, fiberoptic bronchoscopy (FOB) should be performed at the time of intubation. Pre-loading an ETT onto the bronchoscope facilitates both an examination of the airway prior to intubation as well as insertion of an ETT into the trachea. This technique requires not only a skilled operator but also the aid of several trained assistants in a coordinated fashion. Simultaneous direct laryngoscopy (or videolaryngoscopy) by a separate provider allows for more complete visualization of the supraglottic airway and speeds insertion of the FOB into the trachea. Management of the traumatized airway has been recently reviewed in greater detail for interested readers [126••].

Significant injury to the post-carinal conducting airways will typically manifest as a pneumothorax. Continued air leak from a chest tube is highly suggestive of a bronchopleural fistula (BPF) related to either a bronchial tear or disruption. A BPF may lead to impaired gas exchange due to compromised lung expansion, loss of tidal volume, or the inability to provision positive end-expiratory pressure (PEEP) during mechanical ventilation. In such instances, it is preferable to preserve spontaneous ventilation unless respiratory failure develops. Otherwise, endotracheal intubation and emergent lung isolation can be used as a salvage maneuver until surgical intervention can be completed. If lung isolation cannot be accomplished, minimization of mean airway pressures during mechanical ventilation will reduce the pressure gradient across the fistula and minimize any associated air leak. Less obvious bronchial injuries may go unnoticed as radiographic and clinical findings can be subtle. Persistent atelectasis, otherwise unexplained consolidation, or segmental collapse should heighten clinical suspicion [54]. While discreet bronchial injuries can typically be repaired primarily, the operative approach to more complex injuries most often involves some degree of pulmonary resection.

Rib Fractures and Flail Chest

Rib fractures are the most common manifestation of blunt thoracic trauma. Rib fractures themselves are typically not

directly threatening and rarely require operative management aside from tube thoracostomy to manage associated pneumo- or hemothorax. However, they are painful and promote the development of splinting, ineffective airway clearance, progressive atelectasis, hypoxemia, and eventual respiratory failure. The incidence of respiratory complications has been estimated at up to one-third of patients with rib fractures [55]. As such, analgesia is an important element of management. Anesthesiologists are frequently asked to consider thoracic epidural (or paravertebral) analgesia as anecdotal evidence of its efficacy is prevalent. However, a clear role for epidural analgesia has not been borne out in the literature as evidenced by a recent systematic review and meta-analysis that did not demonstrate a benefit [56•]. As increasing age and number of fractures confer a higher risk of mortality, epidural placement is more likely to be beneficial among those who are over 55 years of age and sustain multiple fractures [57].

Flail chest describes the fracture of adjacent ribs at multiple points thus resulting in a freely mobile segment. Paradoxical expansion of the segment during exhalation and retraction during inspiration is commonly observed [58]. Patients with a flail segment are at an increased risk for respiratory failure compared to those with less threatening rib fracture patterns, and concomitant pulmonary contusion further increases the risk [59, 60]. Conservative management without positive pressure ventilation is possible in a subset of patients without polytrauma when coupled with judicious fluid administration, close attention to airway clearance, and multimodal analgesia [61••]. However, such an approach is likely impractical in the severely injured patient with a flail segment as evidenced in a recent retrospective series, which identified that mechanical ventilation (59 %) and ICU admission (82 %) were often required [62].

Pulmonary Contusion

The recognition of pulmonary contusion as a clinical entity began largely as a result of battlefield blast injuries after which pulmonary hemorrhage was evident on autopsy despite the absence of external trauma [63]. Following energy transmission to the thorax, three mechanisms of lung injury have been identified: shearing forces at gas–liquid interfaces, inertial injury between relatively fixed hilar structures and more mobile peripheral alveolar tissue, and expansion of gas in the wake of a pressure wave [64]. Disruption of the alveolocapillary membrane leads to loss of plasma and blood into the alveoli, which contributes to reduced compliance in conjunction with alveolar septal inflammation [65, 66]. These changes worsen the shunt fraction with resultant hypoxemia, and it has been postulated that increased pulmonary vascular resistance

Table 3 Selected findings in laryngeal and tracheobronchial trauma

Signs and symptoms:

Subcutaneous emphysema, crepitus, air escape, external bleeding and bruising, ecchymosis, hematoma, dyspnea, stridor, wheezing, cough, dysphonia, hoarseness, pain with phonation, hemoptysis, tracheal deviation

Bronchoscopic findings:

Tear, edema, hematoma, abnormality of vocal cords, compression or distortion of airway

Tracheal injury may be exterior to the visible mucosa, and evidence of injury may not be visible during fiberoptic bronchoscopy

Computed tomography findings:

Compression or distortion of airway and surrounding structures, fracture, tear, edema, hematoma, abnormal air pockets, pneumothorax, pneumomediastinum

Modified from [126••], with permission from Wolters Kluwer Health

develops as a compensatory mechanism [67]. Patients who suffer significant contusions may go on to develop fibrosis and sequelae of chronic lung disease [68].

Diagnosis relies initially on clinical suspicion in the setting of an appropriate mechanism of injury with impaired gas exchange. The development of infiltrates on a plain chest radiograph is often delayed, and radiographic findings typically worsen over the first 24 h [69]. As is the case with many injury patterns, CT offers heightened diagnostic sensitivity [70, 71]. Retrospective evidence suggest that patients with a GCS >13, ≤ 4 rib fractures, and pulmonary contusions totaling <1/3 of the total lung fields as assessed by CT scan are unlikely to deteriorate [72]. However, patients with polytrauma or extensive contusions evident on admission are a high risk for respiratory failure and progression to frank ARDS [73]. The evidence available to guide decision making for patients with pulmonary contusions is heterogeneous and of a limited quality; as such, the relevant EAST guidelines lack level 1 recommendations [61••]. Common interventions supported by EAST guidelines include limitation of fluid administration, administration of diuretics, and management of respiratory failure with positive pressure ventilation. The preferential use of colloids over crystalloids is not supported for this, or any other, subsegment of trauma patients [74]. Poor compliance of respiratory system often complicates mechanical ventilation as high inspiratory pressures may be needed. Inattention to ventilator parameters can lead to ventilator-induced lung injury, as is discussed subsequently.

Management of Mechanical Ventilation

Mechanisms of Lung Injury in Trauma

Traumatic injury initiates a complex neurohormonal inflammatory cascade that acts in concert with shock, surgical stress, sepsis, aspiration, and iatrogenic influences to produce pulmonary dysfunction [75]. The association between trauma, resuscitation, the development of

pulmonary infiltrates, and resultant poor gas exchange was first made during World War II [76]. Subsequent experience has shown this entity to be ARDS [77]. Current ARDS diagnostic criteria were established in 2012 and are outlined in Table 4 [78]. ARDS can develop after either direct (e.g., pulmonary contusion) or indirect (e.g., systemic inflammatory response to trauma) insults to the lung [79]. The common theme underlying both mechanisms is the development of a pro-inflammatory pulmonary milieu that either causes or worsens existing parenchymal injury [80].

Lung-Protective Mechanical Ventilation and Prevention of Lung Injury

ARDS frequently complicates the course of recovery after traumatic insult and represents a major source of morbidity and mortality in this patient population [81]. However, its incidence and associated mortality have significantly declined over the past decade in nosocomial settings, and a similar trend has been identified among trauma patients [82, 83]. These gains can be attributed largely to the widespread adoption of low tidal volume mechanical ventilation after publication of a landmark trial in 2000 [84]. Key aspects of this so-called “lung-protective” strategy are highlighted in Table 5. Critically, the application of these principles not only improves outcomes of patients with ARDS but also protects against the development of ARDS in at-risk patients [85–87]. As such, lung-protective strategies are now considered a standard of care in the ICU environment.

Unfortunately, these advances have not been universally adopted with regard to intraoperative mechanical ventilation, and the delivery of potentially injurious ventilation in the operating room remains common. In one recent retrospective study, 61 % of patients undergoing non-cardiac surgery received tidal volumes ≥ 8 ml/kg predicted body weight (PBW), and 39 % did not receive PEEP [88•]. (See Table 5 for calculation of PBW.) Multiple trials have assessed the relationship between intraoperative lung-

protective ventilation and postoperative pulmonary outcomes. When subjected to individual patient data meta-analysis, findings from relevant randomized controlled trials suggest that intraoperative delivery of tidal volumes in excess of 8–10 ml/kg PBW is associated with postoperative pulmonary complications [89••].

However, it is conceivable that patients undergoing short, elective procedures will be less vulnerable to ventilator-induced lung injury and can thus tolerate larger tidal volumes and greater degrees of atelectasis. As such, it has been argued that the most sensible approach to intraoperative ventilation is an individualized one in which lung-protective strategies are more stringently applied to patients with either a high likelihood of, or known, lung injury [90]. With that in mind, nearly all trauma patients are at a high risk for the development of ARDS, and the risk is further increased in patients with thoracic trauma. This point was well-elucidated during the development of a highly cited lung injury predictive score (LIPS) [91, 92]. Shock, aspiration, traumatic brain injury, smoke inhalation, pulmonary contusion, fractures, abdominal surgery, aortic surgery, and acidosis were among the identified risk factors (Table 6). Using a LIPS score cutoff of 4, the negative predictive value was 97 %; however, a positive predictive value of only 18 % likely limits its clinical utility [92]. Therefore, the authors feel it prudent to apply stringent perioperative lung-protective ventilation strategies to every trauma patient in an effort to reduce the risk of lung injury and ARDS.

Other ARDS Interventions

In an effort to standardize the care of patients enrolled in trials examining therapies for ARDS, a checklist for lung injury prevention has been developed as outlined in Table 7. Elements of the checklist are largely directed at avoidance of secondary lung injury through prevention of additional insults, such as unchecked septic shock or aspiration. Aside from appropriate mechanical ventilation, only two other interventions have been proven helpful in randomized trials to treat ARDS: prone positioning and neuromuscular blockade. Prone positioning allows for

dorsal lung recruitment and homogenization of ventilation, thus distributing the forces associated with positive pressure lung insufflation more uniformly [93]. The benefit to prone “sessions” has been demonstrated in patients with varying degrees of ARDS and is likely to be increasingly impactful as disease severity worsens [94–96]. However, placing critically ill patients prone is challenging under the best circumstances. The impact of fractures, injury burden, surgical wounds, body habitus, and hemodynamic instability must be weighed when considering the prone position for a trauma patient as various complications have been described [97]. The effects of neuromuscular blockade on ARDS outcomes were highlighted in a multicenter randomized control trial wherein patients who received a 48-h infusion of cisatracurium demonstrated reduced barotrauma and adjusted mortality rates [98]. Neuromuscular blocking drugs may serve to reduce ventilator asynchrony; however, their prolonged use contributes to the development of neuromuscular weakness and impaired airway clearance. These factors in addition to methodological criticisms have led some to argue that the benefit of neuromuscular blockade is not yet entirely convincing [99].

Practical Aspects of Intraoperative Ventilation for the Trauma Patient

VCV is the most common modality of intraoperative mechanical ventilation and thus is the most familiar to anesthesiologists [100]. Peak inspiratory pressure alarms are a common feature of anesthesia machine ventilators and are typically set to 40 cm H₂O. Driving pressures in excess of this value lead to cessation of inspiration on most modern ventilators and can result in hypoventilation. Peak airway pressures correspond to dynamic compliance and are affected by airway resistance. The ability to add an inspiratory pause during VCV is typically available but not always selected by default. An inspiratory pause allows determination of plateau pressures, which are unaffected by airway resistance and thus correspond to static respiratory system compliance (i.e., lung compliance plus chest wall compliance). A large difference between peak and plateau

Table 4 Acute respiratory distress syndrome diagnostic criteria [78]

Feature	
Onset within one week of a known insult or newly worsening respiratory symptoms	
Bilateral opacities on chest imaging in the absence of another etiology	
Pulmonary edema without heart failure or fluid overload	
Impaired oxygenation (with PEEP \geq 5 cm H ₂ O)	Mild 200 mmHg < PaO ₂ /FiO ₂ \leq 300 mmHg Moderate 100 mmHg < PaO ₂ /FiO ₂ \leq 200 mmHg Severe PaO ₂ /FiO ₂ \leq 100 mmHg

PaO₂ partial pressure of oxygen, FiO₂ fraction of inspired oxygen, PEEP positive end-expiratory pressure

Table 5 Principles of lung-protective ventilation for patients with acute respiratory distress syndrome

Parameter	Considerations
Calculation of predicted body weight	Males PBW = $50 + 0.91 \times (\text{height in CM} - 152.4)$ kg Females PBW = $45.5 + 0.91 \times (\text{height in CM} - 152.4)$ kg
Tidal volume	4–6 ml/kg PBW
Positive end-expiratory pressure	≥ 5 cm H ₂ O for PaO ₂ 55–80 mmHg, SpO ₂ 88–95 %
Peak airway pressures	Pplat <30 cm H ₂ O (via 0.5 s inspiratory pause where applicable)
Respiratory rate	Ideally ≤ 35 /min (for pH ≥ 7.30)

PBW predicted body weight, Pplat plateau pressure

Table 6 Lung injury prediction score calculation worksheet

Predisposing conditions		Risk modifiers	
Condition	Points	Modifier	Points
Shock	2	Alcohol abuse	1
Aspiration	2	Obesity (BMI >30)	1
Sepsis	1	Hypoalbuminemia	1
Pneumonia	1.5	Chemotherapy	1
High-risk surgery		FiO ₂ >0.35	2
Emergency surgery	1.5	Tachypnea (RR >30)	1.5
Orthopedic spine	1	SpO ₂ <95 %	1
Acute abdomen	2	Acidosis (pH <7.35)	1.5
Cardiac	2.5	Diabetes mellitus with sepsis	–1
Aortic vascular	3.5		
High-risk trauma			
Traumatic brain injury	2		
Smoke inhalation	2		
Near drowning	2		
Lung contusion	1.5		
Multiple fractures	1.5		

Reprinted with permission of the American Thoracic Society. Copyright © 2015 American Thoracic Society. From [92]

BMI body mass index, RR respiratory rate

pressures suggests increased airway resistance, whereas elevations in both values without a large delta suggest poor compliance of the respiratory system. The addition of an inspiratory pause is not only a valuable diagnostic maneuver but also allows for careful monitoring of plateau pressures in accordance with the principles of lung-protective ventilation. Elevated plateau pressures are indicative of impaired static respiratory system compliance, which can be due either to changes in lung or chest wall compliance.

Many anesthesiologists transition to PCV when peak airway pressures during VCV are encountered. Although this may ultimately be advisable, efforts should first be directed at ruling out an easily remedied cause. Elevation of peak pressure during VCV in excess of plateau pressure suggests increased airway resistance: secretions, bronchospasm, and circuit obstruction should be ruled out.

Elevation of both peak and plateau pressures with a narrow or absent delta suggests decreased compliance of the respiratory system, which could be due to endobronchial intubation, pneumothorax, ventilator asynchrony, or inadequate neuromuscular blockade. When these potentially reversible causes of elevated airway pressures have been excluded, common causes of respiratory system compliance impairment among trauma patients should then be considered: abdominal compartment syndrome, surgical retractors, pulmonary contusion or edema, and ARDS. In these instances, efforts should be made to limit plateau pressures to less than 30 cm H₂O. PCV offers more precise control over airway pressures; however, tidal volume becomes a dependent variable and must be monitored closely. The authors recommend tight expired tidal volume and minute ventilation alarm settings for this reason when using PCV to avoid hypo- or hyperventilation related to changes in respiratory

Table 7 Lung injury prevention checklist

Checklist elements	Definition
Lung-protective mechanical ventilation	Vt 4–6 ml/kg PBW, Pplat <30 cm H ₂ O, PEEP ≥5 mmHg
Aspiration precautions	RSI by experienced providers, elevate HOB, oral care with chlorhexidine, gastric acid neutralization in the absence of enteral feeding
Adequate empiric antimicrobial treatment and source control	Guided by suspected site, likelihood of nosocomial pathogens, and immune suppression status
Limiting fluid overload	Early fluid resuscitation in septic shock followed by goal-directed therapy
Restrictive transfusion	Hb target >7 g/dl in the absence of active bleeding and/or ischemia
Appropriate patient handoff	Structured ICU handoff that covers checklist elements for duration of stay

Adapted from [127] with permission from BMJ Publishing Group Ltd

Vt tidal volume, PBW predicted body weight, Pplat plateau pressure, PEEP positive end-expiratory pressure, RSI rapid sequence intubation, HOB head of bed, Hb hemoglobin, ICU intensive care unit

system compliance. Outcome differences between VCV and PCV have not been established; as such, provider familiarity and expertise should be considered when choosing intraoperative ventilation modes [101].

When elevated airway pressures limit ventilation, permissive hypercapnia should be considered. Bedside and investigational evidence both suggest that modest degrees of respiratory acidosis are relatively well-tolerated by most trauma patients [102, 103]. However, hypercapnia should be avoided in patients with traumatic brain injury as cerebral vasodilation may lead to elevated intracranial pressures. The adequacy of ventilation should be based on assessment of PaCO₂ by arterial blood gas (ABG) analysis instead of end-tidal carbon dioxide (EtCO₂). The absolute value of, and trends in, EtCO₂ often poorly correlate with PaCO₂ in trauma patients owing to increased alveolar dead space in the setting of hypovolemia, shock, and regional pulmonary ventilation/perfusion mismatch [104]. However, trends in EtCO₂ when minute ventilation is held constant can be a useful surrogate marker of cardiac output and adequacy of resuscitation [105, 106]. Efforts to increase minute ventilation when the respiratory system compliance is poor by manipulation of the respiratory rate can lead to incomplete exhalation, dynamic hyperinflation (i.e., auto-PEEP), increased intrathoracic pressures, decreased venous return, and cardiovascular collapse [107–109]. Similarly, vigorous ventilation via bag valve mask (BVM) devices must be avoided. Patients who prove difficult to ventilate intraoperatively or require high levels of PEEP should be transported with a mechanical ventilator (instead of BVM) and EtCO₂ monitor to reduce the risk of complications [110].

Considerations Related to One-Lung Ventilation

Management of OLV can be challenging even during elective operations, and these challenges are compounded following traumatic injury. Under normal circumstances, the shunt fraction induced by OLV is reduced from the expected

50 to 20–30 % by virtue of hypoxic pulmonary vasoconstriction, improved perfusion matching by lateral positioning, and manipulation of the operative lung [111]. Tolerance of OLV is generally much reduced in trauma patients owing to hypovolemia and either primary or secondary lung injury. Pulmonary contusions affecting the non-operative lung may make tolerance of OLV impossible owing to superimposed baseline shunt and poor compliance. As such, non-emergent procedures requiring OLV may require delay until gas exchange and pulmonary mechanics improve. Little definitive evidence exists to guide management of mechanical ventilation during OLV; however, lower tidal volumes (4–5 ml/kg PBW) have been associated with reduced inflammatory biomarkers and are likely most appropriate [112, 113]. When low tidal volumes are employed, higher levels of PEEP applied to the ventilated lung (averaging 10 cm H₂O) are needed to maximize recruitment and reduce lung strain [114]. Application of CPAP to the non-ventilated lung is often avoided during elective procedures such as video-assisted thoracoscopy (VATS) but may not necessarily interfere with emergent resection during open thoracotomy. CPAP not only minimizes shunt fraction but also prevents dense atelectasis that can be difficult to overcome postoperatively [115].

Advanced Ventilation Strategies

Airway pressure release ventilation (APRV [also known as bi-level ventilation]) provides high inspiratory pressures (P_{high}) for a set time period (T_{high}) with brief releases to a lower pressure (P_{low}) for a set time (T_{low}). APRV differs from PCV with an inverse I:E ratio in that it allows for spontaneous breathing throughout the respiratory cycle, and mean airway pressures are typically higher. A clear consensus has not been developed regarding the optimal approach to initial settings [116]. However, typical empiric settings entail a P_{high} of 20–30 cm H₂O, P_{low} 0–5 cm H₂O, T_{high} 5 s, and T_{low} 0.5 s. Hypoventilation requires a greater

pressure gradient or total proportion of the respiratory cycle spent at P_{low} . Hypoxemia requires either a greater inspired fraction of oxygen or higher mean airway pressure, which is dependent upon P_{high} and T_{high} . The hypothetical benefit of APRV is that it maximizes and maintains alveolar recruitment while avoiding collapse on exhalation. Although APRV has been studied in trauma patients, insufficient evidence exists to recommend its routine use, especially in centers without extensive experience [117, 118]. Similarly, excitement about the application of high-frequency oscillatory ventilation (HFOV) has been tempered by the publication of two large prospective trials in which mortality was either not improved or worsened in patients who received HFOV [119, 120].

Asymmetric or unilateral pulmonary contusions cause reduced compliance of the more injured lung, over-distention of the healthier lung during conventional mechanical ventilation, and ventilator-induced lung injury. Similarly, the applied PEEP may not be sufficient to recruit the injured lung. On this basis, independent lung ventilation (also known as differential ventilation) has been advocated as one means by which to manage patients with this challenging condition [121]. Lung isolation is typically best accomplished with a double-lumen ETT in this scenario. No difference in outcomes has been established regarding synchronous versus asynchronous ventilation, and an asynchronous strategy with two ventilators is typically less technically complex [122, 123].

Conclusion

The management of cardiothoracic trauma often requires a high degree of ingenuity and expertise owing to the diversity and severity of possible injuries. Anesthesiologists must understand the rationale underlying surgical management decisions and the operative approach to injuries in order to facilitate appropriate preparation. Unfortunately, there is a paucity of high-quality investigational evidence specifically related to thoracic trauma to help guide the anesthesiologist. As such, conclusions must be extrapolated from relevant literature concerning general trauma and critically ill adults. All trauma patients are at an increased risk of ARDS, and these risks are compounded in the setting of thoracic injury. Attention to appropriate mechanical ventilation not only helps limit the severity of ARDS, but also helps prevent its development in at-risk patients.

Compliance with Ethics Guidelines

Conflict of Interest Craig S. Jabaley, Roman Dudaryk, and Charles E. Smith declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Kleber C, Giesecke MT, Tsokos M, Haas NP, Buschmann CT. Trauma-related preventable deaths in Berlin 2010: need to change prehospital management strategies and trauma management education. *World J Surg.* 2013;37(5):1154–61.
2. Sevitt S. The mechanisms of traumatic rupture of the thoracic aorta. *Br J Surg.* 1977;64(3):166–73.
3. Dyer DS, Moore EE, Ilke DN, McIntyre RC, Bernstein SM, Durham JD, et al. Thoracic aortic injury: how predictive is mechanism and is chest computed tomography a reliable screening tool? A prospective study of 1,561 patients. *J Trauma Acute Care Surg.* 2000;48(4):673–83.
4. Ungar TC, Wolf SJ, Haukoos JS, Dyer DS, Moore EE. Derivation of a clinical decision rule to exclude thoracic aortic imaging in patients with blunt chest trauma after motor vehicle collisions. *J Trauma Acute Care Surg.* 2006;61(5):1150–5.
5. Azizzadeh A, Keyhani K, Miller CC, Coogan SM, Safi HJ, Estrera AL. Blunt traumatic aortic injury: initial experience with endovascular repair. *J Vasc Surg.* 2009;49(6):1403–8.
6. Fabian TC, Richardson JD, Croce MA, Smith JS, Rodman G, Kearney PA, et al. Prospective study of blunt aortic injury: multicenter trial of the American Association for the Surgery of Trauma. *J Trauma Acute Care Surg.* 1997;42(3):374–83.
7. Chakraverty S, Zealley I, Kessel D. Damage control radiology in the severely injured patient: what the anaesthetist needs to know. *Br J Anaesth.* 2014;113(2):250–7.
8. Gay D, Miles R. Use of imaging in trauma decision-making. *J R Army Med Corps.* 2011;157(Suppl 3):S289–92.
9. Lee WA, Matsumura JS, Mitchell RS, Farber MA, Greenberg RK, Azizzadeh A, et al. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. *J Vasc Surg.* 2011;53(1):187–92.
10. Fox N, Schwartz D, Salazar JH, Haut ER, Dahm P, Black JH, et al. Evaluation and management of blunt traumatic aortic injury: A practice management guideline from the Eastern Association for the Surgery of Trauma. *J Trauma Acute Care Surg.* 2015;78(1):136–46.
11. Neschis DG, Scalea TM, Flinn WR, Griffith BP. Blunt aortic injury. *NEJM.* 2008;359(16):1708–16.
12. Demetriades D, Velmahos GC, Scalea TM, Jurkovich GJ, Karmy-Jones R, Teixeira PG, et al. Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. *J Trauma.* 2008;64(3):561–70 **discussion 70-1**.
13. Neschis DG, Moaine S, Gutta R, Charles K, Scalea TM, Flinn WR, et al. Twenty consecutive cases of endograft repair of traumatic aortic disruption: lessons learned. *J Vasc Surg.* 2007;45(3):487–92.
14. Fabian TC, Davis KA, Gavant ML, Croce MA, Melton SM, Patton JH Jr, et al. Prospective study of blunt aortic injury:

- helical CT is diagnostic and antihypertensive therapy reduces rupture. *Ann Surg.* 1998;227(5):666–76.
15. Marcolini EG, Keegan J. Blunt cardiac injury. *Em Med Clin N Am.* 2015;33(3):519–27.
 16. Schultz JM, Trunkey DD. Blunt cardiac injury. *Crit Care Clin.* 2004;20(1):57–70.
 17. Clancy K, Velopulos C, Bilaniuk JW, Collier B, Crowley W, Kurek S, et al. Screening for blunt cardiac injury: an Eastern Association for the Surgery of Trauma practice management guideline. *J Trauma Acute Care Surg.* 2012;73(5 Suppl 4):S301–6.
 18. García-Fernández MA, López-Pérez JM, Pérez-Castellano N, Quero LF, Virgós-Lamela A, Otero-Ferreiro A, et al. Role of transesophageal echocardiography in the assessment of patients with blunt chest trauma: correlation of echocardiographic findings with the electrocardiogram and creatine kinase monoclonal antibody measurements. *Am Heart J.* 1998;135(3):476–81.
 19. Velmahos GC, Karaiskakis M, Salim A, Toutouzas KG, Murray J, Asensio J, et al. Normal electrocardiography and serum troponin I levels preclude the presence of clinically significant blunt cardiac injury. *J Trauma Acute Care Surg.* 2003;54(1):45–51.
 20. Devitt JH, McLean RF, McLellan BA. Perioperative cardiovascular complications associated with blunt thoracic trauma. *Can J Anaesth.* 1993;40(3):197–200.
 21. Healey MA, Brown R, Fleiszer D. Blunt cardiac injury: is this diagnosis necessary? *J Trauma Acute Care Surg.* 1990;30(2):137–46.
 22. Flancbaum L, Wright J, Siegel JH. Emergency surgery in patients with post-traumatic myocardial contusion. *J Trauma Acute Care Surg.* 1986;26(9):795–803.
 23. Maron BJ, Estes NAM. Commotio cordis. *NEJM.* 2010;362(10):917–27.
 24. Maron BJ, Gohman TE, Kyle SB, Estes NM III, Link MS. Clinical profile and spectrum of commotio cordis. *JAMA.* 2002;287(9):1142–6.
 25. Maron BJ, Mitten MJ, Greene Burnett C. Criminal consequences of commotio cordis. *Am J Card.* 2002;89(2):210–3.
 26. American College of Surgeons Committee on Trauma. Practice management guidelines for emergency department thoracotomy. *J Am Coll Surg.* 2001;193(3):303.
 27. Hall BL, Buchman TG. A visual, timeline-based display of evidence for emergency thoracotomy. *J Trauma Acute Care Surg.* 2005;59(3):773–7.
 28. Rabinovici R, Bugaev N. Resuscitative thoracotomy: an Update. *Scan J Surg.* 2014;1457496913514735.
 29. Burlew CC, Moore EE, Moore FA, Coimbra R, McIntyre RC Jr, Davis JW, et al. Western Trauma Association critical decisions in trauma: resuscitative thoracotomy. *J Trauma Acute Care Surg.* 2012;73(6):1359–63.
 30. 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):159–73.
 31. Renz BM, Stout MJ. Rapid right atrial cannulation for fluid infusion during resuscitative emergency department thoracotomy. *Am Surg.* 1994;60(12):946–9.
 32. Girardi LN, Magovern CJ, Fischer E, Barie PS. Descending aortic cannulation during emergent thoracotomy for blunt traumatic cardiac arrest. *J Trauma Acute Care Surg.* 1999;47(4):787.
 33. Rotondo MF, Schwab CW, McGonigal MD, Phillips GR, Fruchterman TM, Kauder DR, et al. ‘Damage control’: an approach for improved survival in exsanguinating penetrating abdominal injury. *J Trauma Acute Care Surg.* 1993;35(3):375–83.
 34. O’Connor JV, DuBose JJ, Scalea TM. Damage-control thoracic surgery: management and outcomes. *J Trauma Acute Care Surg.* 2014;77(5):660–5.
 35. Mackowski MJ, Barnett RE, Harbrecht BG, Miller KR, Franklin GA, Smith JW, et al. Damage control for thoracic trauma. *Am Surg.* 2014;80(9):910–3.
 36. Vargo DJ, Battistella FD. Abbreviated thoracotomy and temporary chest closure: an application of damage control after thoracic trauma. *Arch Surg.* 2001;136(1):21–4.
 37. Kaafarani H, Velmahos G. Damage control resuscitation in trauma. *Scan J Surg.* 2014;1457496914524388.
 38. Holcomb JB, Jenkins D, Rhee P, Johannigman J, Mahoney P, Mehta S, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma Acute Care Surg.* 2007;62(2):307–10.
 39. Jansen JO, Thomas R, Loudon MA, Brooks A. Damage control resuscitation for patients with major trauma. *BMJ.* 2009;338:b1778.
 40. Stannard A, Eliason JL, Rasmussen TE. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. *J Trauma Acute Care Surg.* 2011;71(6):1869–72.
 41. Norii T, Crandall C, Terasaka Y. Survival of severe blunt trauma patients treated with resuscitative endovascular balloon occlusion of the aorta compared with propensity score-adjusted untreated patients. *J Trauma Acute Care Surg.* 2015;78(4):721–8.
 42. Biffi WL, Fox CJ, Moore EE. The role of REBOA in the control of exsanguinating torso hemorrhage. *J Trauma Acute Care Surg.* 2015;78(5):1054–8. *The history, application, and role of REBOA is expertly summarized in this updated review.*
 43. DuBose JJ (Principle Investigator). Aortic occlusion for resuscitation in trauma and acute care surgery (AORTA): a prospective observational study of the Endovascular Skills in Trauma and Resuscitative Surgery. Study Protocol. 2013. Accessed 8 Nov 2015. <http://www.aast.org/Research/MultiInstitutionalStudies.aspx>.
 44. Guerrero-Lopez F, Vazquez-Mata G, Alcazar-Romero PP, Fernandez-Mondejar E, Aguayo-Hoyos E, Linde-Valverde CM. Evaluation of the utility of computed tomography in the initial assessment of the critical care patient with chest trauma. *Crit Care Med.* 2000;28(5):1370–5.
 45. Soult MC, Weireter LJ, Britt RC, Collins JN, Novosel TJ, Reed SF, et al. Can routine trauma bay chest X-ray be bypassed with an extended focused assessment with sonography for trauma examination? *Am Surg.* 2015;81(4):336–40.
 46. Ding W, Shen Y, Yang J, He X, Zhang M. Diagnosis of pneumothorax by radiography and ultrasonography: a meta-analysis. *Chest J.* 2011;140(4):859–66.
 47. Edrich T, Pojor C, Fritsch G, Hutter J, Hartigan PM, Stundner O, et al. Utility of intraoperative lung ultrasonography. *A A Case Rep.* 2015;4(6):71–4.
 48. Kirkpatrick AW, Rizoli S, Ouellet JF, Roberts DJ, Sirois M, Ball CG, et al. Occult pneumothoraces in critical care: a prospective multicenter randomized controlled trial of pleural drainage for mechanically ventilated trauma patients with occult pneumothoraces. *J Trauma Acute Care Surg.* 2013;74(3):747–54 **discussion 54-5**.
 49. Ouellet JF, Trottier V, Kmet L, Rizoli S, Laupland K, Ball CG, et al. The OPTICC trial: a multi-institutional study of occult pneumothoraces in critical care. *Am J Surg.* 2009;197(5):581–6.
 50. Yadav K, Jalili M, Zehtabchi S. Management of traumatic occult pneumothorax. *Resuscitation.* 2010;81(9):1063–8.
 51. Shorr RM, Crittenden M, Indeck M, Hartunian SL, Rodriguez A. Blunt thoracic trauma. Analysis of 515 patients. *Ann Surg.* 1987;206(2):200–5.

52. American College of Surgeons Committee on Trauma. Advanced trauma life support student course manual. Thoracic trauma. 9th ed. Chicago: American College of Surgeons; 2012.
53. de Lesquen H, Avaro JP, Gust L, Ford RM, Beranger F, Natale C, et al. Surgical management for the first 48 h following blunt chest trauma: state of the art (excluding vascular injuries). *Interact CardioVasc Thorac Surg*. 2015;20(3):399–408.
54. Glazer ES, Meyerson SL. Delayed presentation and treatment of tracheobronchial injuries due to blunt trauma. *J Surg Ed*. 2008;65(4):302–8.
55. Ziegler DW, Agarwal NN. The morbidity and mortality of rib fractures. *J Trauma Acute Care Surg*. 1994;37(6):975–9.
56. • Duch P, Møller M. Epidural analgesia in patients with traumatic rib fractures: a systematic review of randomised controlled trials. *Acta Anaesthesiol Scand*. 2015;59:698–709. *This systematic review examines the impact of epidural analgesia for patients with rib fractures.*
57. Fligel BT, Luchette FA, Reed RL, Esposito TJ, Davis KA, Santaniello JM, et al. Half-a-dozen ribs: the breakpoint for mortality. *Surgery*. 2005;138(4):717–25.
58. Mayberry JC, Trunkey DD. The fractured rib in chest wall trauma. *Chest Surg Clin N Am*. 1997;7(2):239–61.
59. Johnson JA, Cogbill TH, Winga ER. Determinants of outcome after pulmonary contusion. *J Trauma Acute Care Surg*. 1986;26(8):695–7.
60. Richardson JD, Adams L, Flint LM. Selective management of flail chest and pulmonary contusion. *Ann Surg*. 1982;196(4):481.
61. •• Simon B, Ebert J, Bokhari F, Capella J, Emhoff T, Hayward III T et al. Management of pulmonary contusion and flail chest: an Eastern Association for the Surgery of Trauma practice management guideline. *J Trauma Acute Care Surg*. 2012;73(5):S351–61. *The EAST guidelines for management of pulmonary contusions offers a comprehensive review of the relevant literature and important practice parameters.*
62. Dehghan N, de Mestral C, McKee MD, Schemitsch EH, Nathens A. Flail chest injuries: A review of outcomes and treatment practices from the National Trauma Data Bank. *J Trauma Acute Care Surg*. 2014;76(2):462–8.
63. Cohn SM, DuBose JJ. Pulmonary contusion: an update on recent advances in clinical management. *World J Surg*. 2010;34(8):1959–70.
64. Clemedson C-J. Blast injury. *Physiol Rev*. 1956;36(3):336–54.
65. Fulton RL, Peter E. The progressive nature of pulmonary contusion. *Surgery*. 1970;67(3):499–506.
66. Oppenheimer L, Craven K, Forkert L, Wood L. Pathophysiology of pulmonary contusion in dogs. *J App Phys*. 1979;47(4):718–28.
67. Wagner RB, Slivko B, Jamieson PM, Dills MS, Edwards FH. Effect of lung contusion on pulmonary hemodynamics. *Ann Thorac Surg*. 1991;52(1):51–8.
68. Bastos R, Calhoon JH, Baisden CE. Flail Chest and Pulmonary Contusion. *Sem Thorac Cardiovasc Surg*. 2008;20(1):39–45.
69. Tyburski JG, Collinge JD, Wilson RF, Eachempati SR. Pulmonary contusions: quantifying the lesions on chest X-ray films and the factors affecting prognosis. *J Trauma Acute Care Surg*. 1999;46(5):833–8.
70. Deunk J, Poels TC, Brink M, Dekker HM, Kool DR, Blickman JG, et al. The clinical outcome of occult pulmonary contusion on multidetector-row computed tomography in blunt trauma patients. *J Trauma Acute Care Surg*. 2010;68(2):387–94.
71. Poole GV, Morgan DB, Cranston PE, Muakkassa FF, Griswold JA. Computed tomography in the management of blunt thoracic trauma. *J Trauma Acute Care Surg*. 1993;35(2):296–302.
72. de Moya MA, Manolakaki D, Chang Y, Amygdalos I, Gao F, Alam HB, et al. Blunt pulmonary contusion: admission computed tomography scan predicts mechanical ventilation. *J Trauma Acute Care Surg*. 2011;71(6):1543–7.
73. Miller PR, Croce MA, Bee TK, Qaisi WG, Smith CP, Collins GL, et al. ARDS after pulmonary contusion: accurate measurement of contusion volume identifies high-risk patients. *J Trauma Acute Care Surg*. 2001;51(2):223–30.
74. Jabaley C, Dudaryk R. Fluid resuscitation for trauma patients: crystalloids versus colloids. *Curr Anesthesiol Rep*. 2014;4(3):216–24.
75. Bakowitz M, Bruns B, McCunn M. Acute lung injury and the acute respiratory distress syndrome in the injured patient. *Scand J Trauma Resusc Emerg Med*. 2012;20:54.
76. Burford TH, Burbank B. Traumatic wet lung; observations on certain physiologic fundamentals of thoracic trauma. *J Thorac Surg*. 1945;14:415.
77. Ashbaugh D, Bigelow DB, Petty T, Levine B. Acute respiratory distress in adults. *Lancet*. 1967;290(7511):319–23.
78. The ARDS Definition Task Force. Acute respiratory distress syndrome: the Berlin definition. *JAMA*. 2012;307(23):2526–33.
79. Ware LB, editor. Pathophysiology of acute lung injury and the acute respiratory distress syndrome. Seminars in respiratory and critical care medicine. New York: Thieme Medical Publishers, c1994, 2006.
80. Ware LB, Matthay MA. The acute respiratory distress syndrome. *NEJM*. 2000;342(18):1334–49.
81. Netzer G, Smith G, Murthi S, Afshar M. Trauma-associated acute respiratory distress syndrome case-fatality: 2004–2011. *Am J Respir Crit Care Med*. 2015;191:A1618.
82. Martin M, Salim A, Murray J, Demetriades D, Belzberg H, Rhee P. The decreasing incidence and mortality of acute respiratory distress syndrome after injury: a 5-year observational study. *J Trauma Acute Care Surg*. 2005;59(5):1107–13.
83. Li G, Malinchoc M, Cartin-Ceba R, Venkata CV, Kor DJ, Peters SG, et al. Eight-year trend of acute respiratory distress syndrome: a population-based study in Olmsted County, Minnesota. *Am J Respir Crit Care Med*. 2011;183(1):59–66.
84. Network The Acute Respiratory Distress Syndrome. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *NEJM*. 2000;342(18):1301–8.
85. Determann RM, Royakkers A, Wolthuis EK, Vlaar AP, Choi G, Paulus F, et al. Research Ventilation with lower tidal volumes as compared with conventional tidal volumes for patients without acute lung injury: a preventive randomized controlled trial. 2010;14:R1.
86. Futier E, Constantin J-M, Paugam-Burtz C, Pascal J, Eurin M, Neuschwander A, et al. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. *NEJM*. 2013;369(5):428–37.
87. Neto AS, Cardoso SO, Manetta JA, Pereira VGM, Espósito DC, Pasqualucci MdOP, et al. Association between use of lung-protective ventilation with lower tidal volumes and clinical outcomes among patients without acute respiratory distress syndrome: a meta-analysis. *JAMA*. 2012;308(16):1651–9.
88. • Ladha K, Vidal Melo MF, McLean DJ, Wanderer JP, Grabitz SD, Kurth T et al. Intraoperative protective mechanical ventilation and risk of postoperative respiratory complications: hospital based registry study. *BMJ*. 2015;351:h3646. *A robust retrospective analysis is offered regarding the impact of intraoperative ventilation on the development of postoperative pulmonary complications.*
89. •• Neto AS, Hemmes SNT, Barbas CSV, Beiderlinden M, Biehl M, Binnekade JM et al. Protective versus conventional ventilation for surgery: a systematic review and individual patient data meta-analysis. *Anesthesiology*. 2015;123(1):66–78. *This unique patient level meta-analysis examines the effect of*

- intraoperative ventilatory parameters on the development of postoperative pulmonary complications.*
90. Eikermann M, Kurth T. Apply protective mechanical ventilation in the operating room in an individualized approach to perioperative respiratory care. *Anesthesiology*. 2015;123(1):12–4.
 91. Trillo-Alvarez C, Cartin-Ceba R, Kor DJ, Kojacic M, Kashyap R, Thakur S, et al. Acute lung injury prediction score: derivation and validation in a population-based sample. *Eur Respir J*. 2011;37(3):604–9.
 92. Gajic O, Dabbagh O, Park PK, Adesanya A, Chang SY, Hou P, et al. Early identification of patients at risk of acute lung injury: evaluation of lung injury prediction score in a multicenter cohort study. *Am J Respir Crit Care Med*. 2011;183(4):462–70.
 93. Gattinoni L, Taccone P, Carlesso E, Marini JJ. Prone position in acute respiratory distress syndrome. Rationale, indications, and limits. *Am J Respir Crit Care Med*. 2013;188(11):1286–93.
 94. Sud S, Friedrich J, Taccone P, Polli F, Adhikari NJ, Latini R, et al. Prone ventilation reduces mortality in patients with acute respiratory failure and severe hypoxemia: systematic review and meta-analysis. *Intensive Care Med*. 2010;36(4):585–99.
 95. Guérin C, Reigner J, Richard J-C, Beuret P, Gacouin A, Bou-lain T, et al. Prone positioning in severe acute respiratory distress syndrome. *NEJM*. 2013;368(23):2159–68.
 96. Gattinoni L, Tognoni G, Pesenti A, Taccone P, Mascheroni D, Labarta V, et al. Effect of prone positioning on the survival of patients with acute respiratory failure. *NEJM*. 2001;345(8):568–73.
 97. Offner PJ, Haenel JB, Moore EE, Biff WL, Franciose RJ, Burch JM. Complications of prone ventilation in patients with multi-system trauma with fulminant acute respiratory distress syndrome. *J Trauma Acute Care Surg*. 2000;48(2):224–8.
 98. Papazian L, Forel J-M, Gacouin A, Penot-Ragon C, Perrin G, Loundou A, et al. Neuromuscular blockers in early acute respiratory distress syndrome. *NEJM*. 2010;363(12):1107–16.
 99. Gattinoni L, Marini JJ. Prone positioning and neuromuscular blocking agents are part of standard care in severe ARDS patients: we are not sure. *Intensive Care Med*. 2015;41:1–3.
 100. Jaber S, Coisel Y, Chanques G, Futier E, Constantin JM, Michelet P, et al. A multicentre observational study of intra-operative ventilatory management during general anaesthesia: tidal volumes and relation to body weight. *Anaesthesia*. 2012;67(9):999–1008.
 101. Rittayamai N, Katsios CM, Beloncle F, Friedrich JO, Mancebo J, Brochard L. Pressure-controlled vs volume-controlled ventilation in acute respiratory failure: A physiology-based narrative and systematic review. *Chest*. 2015;148(2):340–55.
 102. Sorkine P, Szold O, Kluger Y, Halpern P, Weinbroum AA, Fleishon R, et al. Permissive hypercapnia ventilation in patients with severe pulmonary blast injury. *J Trauma Acute Care Surg*. 1998;45(1):35–8.
 103. Gentilello LM, Anardi D, Mock C, Arreola-Risa C, Maier RV. Permissive hypercapnia in trauma patients. *J Trauma Acute Care Surg*. 1995;39(5):846–53.
 104. Russell GB, Graybeal JM. Reliability of the arterial to end-tidal carbon dioxide gradient in mechanically ventilated patients with multisystem trauma. *J Trauma Acute Care Surg*. 1994;36(3):317–22.
 105. • Belenkiy SM, Berry JS, Batchinsky AI, Kendrick C, Necsoiu C, Jordan BS et al. The noninvasive carbon dioxide gradient (NICO2G) during hemorrhagic shock. *Shock*. 2014;42(1): 38–43. *Belenkiy et al. offer an excellent description regarding the utility of end-tidal carbon dioxide monitoring as a surrogate marker for adequacy of resuscitation.*
 106. Tyburski JG, Collinge JD, Wilson RF, Carlin AM, Albaran RG, Steffes CP. End-tidal CO₂-derived values during emergency trauma surgery correlated with outcome: a prospective study. *J Trauma Acute Care Surg*. 2002;53(4):738–43.
 107. Berlin D. Hemodynamic consequences of auto-PEEP. *J Intensive Care Med*. 2014;29(2):81–6.
 108. Lapinsky SE, Leung RS. Auto-PEEP and electromechanical dissociation. *NEJM*. 1996;335(9):674–5.
 109. Myles P, Madder H, Morgan E. Intraoperative cardiac arrest after unrecognized dynamic hyperinflation. *Br J Anaesth*. 1995;74(3):340–2.
 110. Fanara B, Manzon C, Barbot O, Desmettre T, Capellier G. Research Recommendations for the intra-hospital transport of critically ill patients. *Crit Care*. 2010;14(3):R87.
 111. Karzai W, Schwarzkopf K. Hypoxemia during one-lung ventilation prediction, prevention, and treatment. *Anesthesiology*. 2009;110(6):1402–11.
 112. Lohser J, Slinger P. Lung injury after one-lung ventilation: a review of the pathophysiologic mechanisms affecting the ventilated and the collapsed lung. *Anesth Analg*. 2015;121(2):302–18.
 113. Yang M, Ahn HJ, Kim K, Kim JA, Chin AY, Kim MJ, et al. Does a protective ventilation strategy reduce the risk of pulmonary complications after lung cancer surgery?: a randomized controlled trial. *CHEST J*. 2011;139(3):530–7.
 114. Ferrando C, Mugarra A, Gutierrez A, Carbonell JA, García M, Soro M, et al. Setting individualized positive end-expiratory pressure level with a positive end-expiratory pressure decrement trial after a recruitment maneuver improves oxygenation and lung mechanics during one-lung ventilation. *Anesth Analg*. 2014;118(3):657–65.
 115. Verhage RJJ, Boone J, Rijkers GT, Cromheecke GJ, Kroese AC, Weijs TJ, et al. Reduced local immune response with continuous positive airway pressure during one-lung ventilation for oesophagectomy. *Br J Anaesth*. 2014;112(5):920–8.
 116. Daoud EG, Farag HL, Chatburn RL. Airway pressure release ventilation: what do we know? *Resp Care*. 2012;57(2):282–92.
 117. Rathor P, Kabak B, Shetty A, Hosur S, Sehgal V, Bajaj A. Efficacy of airway pressure release ventilation in critically ill patients: a meta-analysis of randomized controlled trials. *Am J Respir Crit Care Med*. 2015;191:A1195.
 118. Maung AA, Luckianow G, Kaplan LJ. Lessons learned from airway pressure release ventilation. *J Trauma Acute Care Surg*. 2012;72(3):624–8.
 119. Young D, Lamb SE, Shah S, MacKenzie I, Tunnicliffe W, Lall R, et al. High-frequency oscillation for acute respiratory distress syndrome. *NEJM*. 2013;368(9):806–13.
 120. Ferguson ND, Cook DJ, Guyatt GH, Mehta S, Hand L, Austin P, et al. High-frequency oscillation in early acute respiratory distress syndrome. *NEJM*. 2013;368(9):795–805.
 121. Cinnella G, Dambrosio M, Brienza N, Giuliani R, Bruno F, Fiore T, et al. Independent lung ventilation in patients with unilateral pulmonary contusion. Monitoring with compliance and EtCO₂. *Intensive Care Med*. 2001;27(12):1860–7.
 122. Ost D, Corbridge T. Independent lung ventilation. *Clin Chest Med*. 1996;17(3):591–601.
 123. Anantham D, Jagadesan R, Tiew PE. Clinical review: Independent lung ventilation in critical care. *Crit Care*. 2005;9(6): 594.
 124. Kanellakos GW, Slinger P. Intraoperative one-lung ventilation for trauma anesthesia. In: Smith CE, editor. *Trauma anesthesia*. 2nd ed. Cambridge: Cambridge University Press; 2015.
 125. Aydin NB, Moon MC, Gill I. Cardiac and great vessel trauma. In: Smith CE, editor. *Trauma anesthesia*. Cambridge: Cambridge University Press; 2008.
 126. •• Jain U, McCunn M, Smith CE, Pittet JF. Management of the traumatized airway. *Anesthesiology*. 2016;124(1):199–206. *The authors offer a comprehensive overview of airway management for patients with facial and neck trauma.*
 127. Kor DJ, Talmor DS, Banner-Goodspeed VM, et al. Lung Injury prevention with aspirin (LIPS-A): a protocol for a multicentre randomised clinical trial in medical patients at high risk of acute lung injury. *BMJ Open*. 2012;2(5):e001606.