






# Chest trauma

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<p><b>ATLS</b></p>	<p>The Advanced Trauma Life Support Course sequence allows rapid and systematic assessment and treatment of organ system injuries in the order of their potential threat to life: <b>airway</b> (with cervical spine protection), <b>breathing, circulation, disability</b> (neurologic status), full <b>exposure and environmental control</b> (avoidance of hypothermia). An initial primary survey of these systems is coupled with injury-specific resuscitation, and then followed by a more detailed history and physical examination (secondary survey). Many of the initial phases of this protocol focus on thoracic injuries.</p> <p>Injury or obstruction to the airway can rapidly be assessed by the “look, listen, feel” approach in which movement of air through the airway is assessed by inspection of the chest wall, auscultation for stridor, and direct palpation for air movement from the mouth and nose. All airway assessment and intervention is carried out with the head in a neutral position with in-line immobilization. If the airway is deemed to be at least temporarily patent and the patient’s neurologic status is sufficient to allow for airway protection (GCS <math>\geq</math> 8), the primary survey moves on to the assessment of breathing. This is accomplished by careful inspection of chest wall movement, auscultation for bilateral air entry in the axillae, and palpation of the chest wall for tenderness and deformity. Penetrating injuries to the chest and axillae must be comprehensively sought. Asymmetrical air entry in unstable patients must promptly be treated by pleural decompression, but in stable patients, equivocally reduced air entry may be further evaluated with chest x-ray.</p> <p>Adequacy of circulation may be estimated by palpation of all pulses, as well as blood pressure and urine output monitoring. Pulse oximetry and electrocardiographic monitoring may be used as adjuncts to vital sign data. Any evidence of hemodynamic instability is treated with crystalloid infusion: 1 – 2 litres of normal saline or lactated Ringer’s solution through wide bore intravenous access (14 gauge peripheral lines or 8 French central lines placed in the femoral, subclavian or internal jugular veins). If central venous access above the diaphragm is required, sites ipsilateral to existing chest tubes are preferable. It should be emphasized that assessment of stability must be placed in the context of ongoing fluid administration – high volume fluid infusion may mask ongoing blood loss by prolonging clinical euvolemia. Although most traumatic shock results from hypovolemia, inadequacy of venous return, which may be elusive, must be considered with thoracic injury. Classic presentations of obstructive shock, such as the triad of hypotension, muffled heart sounds and jugular venous distension in cardiac tamponade may be difficult to elicit in immobilized patients in noisy trauma departments, but if appreciated may prove to be lifesaving.</p>
<p><b>Diagnostic studies</b></p>	
<p><b>CXR</b></p>	<p>An AP supine chest radiograph, obtained as part of the primary survey provides a wealth of information about potential injuries. Almost all of the chest wall, pleural, diaphragmatic, lung parenchymal and mediastinal injuries can be diagnosed on chest x-ray. The sensitivity and specificity of this modality, are dependent on the nature of the injury and timing. For example, depending on the population, 15-70% of pneumothoraces may be missed by standard chest radiographs in supine trauma patients. Despite these limitations chest x-rays are widely employed: an early chest x-ray can often provide reassurance or find abnormalities that require prompt attention.</p>
<p><b>CT</b></p>	<p>Computed tomography (CT) is rapidly becoming the definitive study for the evaluation of blunt aortic injury (BAI). Currently, accuracy of CT for the diagnosis of BAI is in excess of 99%. At our institution, patients subjected to high risk mechanisms for BAI (rapid deceleration, side impact collision with intrusion) or those with suspicious findings on chest x-ray undergo chest CT scanning. CT of the chest is also gaining prominence in the evaluation of other intrathoracic injuries. CT has been shown to be highly sensitive in the detection of pneumothorax and early pulmonary contusion.</p>
<p><b>U/S</b></p>	<p>The utility of the focused abdominal sonography for trauma (FAST) examination for abdominal trauma has already been well established and is discussed elsewhere. Equally exciting are reports of successful applications of sonographic techniques quickly and portably in patients with chest injuries. Sensitivity and specificity of emergency ultrasound for the detection of cardiac injury are in excess of 96%. Surgeon performed ultrasound is comparable to conventional chest radiography in the detection of traumatic hemothoraces (sensitivity 97.5 %, specificity 99.7%). Extension of the FAST evaluation to the anterior chest at the fourth or fifth intercostals space was found to be useful in the detection of pneumothorax – when compared with chest x-ray, the sensitivity of this technique was 95% and the specificity was 100%.</p>
<p><b>Primary survey: Life threatening injuries</b></p>	
<p><b>Airway obstruction</b></p> 	<p>Patients found to have impending or active airway obstruction are often noted to have alteration of mental status, or to exhibit stridorous breathing, dysphonia, tachypnea, indrawing or accessory muscle use. Urgent placement of a definitive airway is indicated for those patients who are found to be apneic, hypoxemic, or obtunded (GCS <math>\leq</math> 8), or who are noted to have complex facial injuries, thermal inhalation injuries, penetrating injuries to the neck, or expanding or pulsatile cervical hematomas.</p> <p>A definitive airway is narrowly defined as a tube inserted in the trachea (with the cuff inflated in adults), attached to an oxygen source and a means of ventilatory assistance. All physicians involved in trauma care must become skilled in airway assessment and knowledgeable about the techniques and limitations of rapid sequence intubation (RSI). One member of the team is often committed to manual in-line cervical immobilization while efforts are made to open and inspect the airway using the chin lift or jaw thrust maneuvers. An oral airway may be placed in obtunded patients to maintain airway patency. Next, a bag-mask ventilation device with high flow oxygen is applied to the patient’s face and nose with care to create a seal between the face and mask. Firm cricoid pressure may reduce the severity of aspiration should this occur – this should not be withdrawn until tube placement has been confirmed. Ideally positive pressure ventilation should be avoided at this stage (in favor of spontaneous breathing) in order to prevent gastric distension and vomiting, except in hypoxic or apneic patients. During this phase of preoxygenation, the physician must confirm the adequacy of vital signs and saturation monitoring, and must prepare her intubation equipment. This includes a working laryngoscope, a variety of endotracheal tubes (sizes 6, 7 and 8) with intact cuffs, a stylet, suction, an end tidal carbon dioxide detector and/or a stethoscope to confirm placement, a 10 mL syringe for cuff inflation, and an oxygenation/ventilation source with appropriate connectors. Perhaps most importantly, an alternative means of airway access must be selected and prepared in advance if conventional endotracheal intubation proves to be problematic.</p> <p>Paralysis (and sedation) is a cornerstone of the RSI process. A quick “awake look” laryngoscopy prior to neuromuscular blockade may allow one to proceed more confidently with paralysis if it confirms that the airway is indeed amenable to orotracheal intubation. Short-acting paralytic agents (e.g. succinylcholine 1.5 mg/kg or rocuronium 1 mg/kg) take about 1 minute to achieve onset and last 3-60 minutes. Succinylcholine, which may result in potassium ion release, should not be used in potential hyperkalemic conditions (burns, rhabdomyolysis, renal failure). A variety of narcotics, benzodiazepines, and barbiturates have been used to achieve sedation in these patients, but all are associated with hypotension. However, etomidate (0.3 mg/kg) with an onset of action of 20 seconds, its short duration (7-14 minutes), its lack of hemodynamic effect, and its lack of interference with cerebral perfusion pressure, is gaining increasing popularity.</p>
<p><b>Tension pneumothorax</b></p> 	<p>Blunt or penetrating injuries to the thorax may result in airway, lung parenchymal or pleural disruption and subsequent escape of air to the pleural space. If the injury creates a “one way valve” with air entering the negative pressure environment of the pleural space, but prevented from returning to the airway, air trapping will occur. Positive pleural pressure creates an extremely dangerous situation of respiratory and hemodynamic compromise due to alterations in respiratory mechanics and venous return. Positive pressure ventilation can hasten this process, or convert apparently simple pneumothoraces to tension pneumothoraces (TPs).</p> <p>TP is a clinical diagnosis based on ipsilateral diminished air entry and hyperresonance, contralateral tracheal deviation, and jugular venous distension. In the presence of these findings, therapy must not be delayed by radiography. Patients with diminished air entry and evidence of hemodynamic instability should undergo immediate pleural decompression.</p> <p>TPs require immediate placement of chest tubes on the side of diminished air entry. These are usually placed in the mid axillary line at the fifth intercostal space (nipple or inframammary fold level) under sterile conditions. Since pneumothoraces are frequently associated with hemothoraces, 32 French or bigger tubes should be used to reliably evacuate of both air and blood. If the diagnosis of TP is suspected, and even brief delays in chest tube placement are anticipated, the pleural space can</p>

	<p>be temporarily decompressed by a 14G angiocatheter placed at the midclavicular line at the second intercostal space. Standard chest tubes are placed prior to removal of these decompression catheters, even if the catheters were placed empirically, as they can actually create iatrogenic pneumothoraces.</p> <p>Most pneumothoraces respond well to tube thoracostomy. But failure of the lung to re-expand with well-placed chest tubes on 20 cm H<sub>2</sub>O suction, persistent air leaks, or the observation of hemoptysis or blood in the endotracheal tube should raise the concern that a more proximal injury to the tracheobronchial tree has occurred. In such instances, a second chest tube may be required and evaluation for airway injuries by flexible bronchoscopy should be undertaken. Bronchoscopy-guided interventions in the operating room such as selective bronchial intubation, or placement of dual lumen tubes or bronchial blockers may allow protection and selective ventilation of the uninjured lung until definitive measures can be undertaken.</p> <p>Chest tubes may be safely removed once air leaks have stopped, and fluid drainage has diminished to less than 150 mL over a 24 hour period of observation. Follow-up radiographs may be obtained 6 hours after chest tube removal to exclude the presence of a residual pneumothorax.</p>
<p><b>Open pneumothorax</b></p> 	<p>High velocity weapons, or high speed blunt injury mechanisms can cause full thickness injury to the chest wall. Gaps in the chest wall may permit air entry into the pleural space, especially during spontaneous inspiration when intrapleural pressures are negative. When a chest wall defect reaches roughly 2/3 the diameter of the trachea, pleural ventilation may become the path of least resistance during inspiration and normal bronchial air flow may become extremely compromised. Patients with this injury present with respiratory distress and a “sucking chest wound”.</p> <p>Early therapeutic priorities for these patients include airway management, coverage of the wound and pleural drainage. Positive pressure ventilation may diminish airflow through the chest wall defect. More importantly, coverage of the defect with a square or rectangular plastic dressing taped on three sides prevents air flow through the wound during inspiration, but allows outflow during expiration. Once a dressing is applied, a chest tube placed via a separate incision allows more complete evacuation of intrapleural air. Wound contamination requires the short term administration of prophylactic antibiotics (cefazolin) and tetanus immunization. In the operating room, however, once a chest tube has been placed, wounds should be cleaned, debrided and closed if possible. Large areas of tissue loss may require more complicated measures for chest closure such as placement of prosthetic mesh or diaphragmatic transposition in lower, thoracoabdominal injuries.</p>
<p><b>Flail chest</b></p> 	<p>Multiple rib fractures may result in segmental instability of the chest wall. When blunt forces disrupt the continuity of ribs, costal cartilage, or sternum at more than one site, the intervening chest wall can remain stationary or even be drawn inward at inspiration. This phenomenon is known as flail chest (FC). The high energy transfers required to create a flail segment often result in significant associated injuries such as pulmonary contusion (50%) and hemopneumothorax (70%), as well as head, abdominal, pelvic and extremity injuries. Acute hypoxemia and respiratory failure is seen in those FC patients with associated pulmonary contusions, and flail segments may, in fact, become more evident as more respiratory effort is required with blossoming contusions. The acute and chronic severity of FC is primarily dependent on the magnitude of the underlying pulmonary contusion rather than on abnormalities of the chest wall.</p> <p>The diagnosis of FC is clinical – observation of segmental paradoxical chest wall movement with respiration requires adequate exposure and careful examination during all phases of spontaneous breathing. The observation of fractured ribs on plain x-rays or CT may, in some instances confirm the clinical impression, but should not be relied upon as fractures through the ribs and costal cartilages as well as the sternum often go unrecognized by these modalities.</p> <p>Once the diagnosis is confirmed, therapeutic efforts to treat and prevent worsening of respiratory compromise must commence and be carried on conscientiously until patients are pain free and are weaned from supplemental oxygen. The cornerstones of therapy for FC are close observation and reexamination for signs of deterioration of respiratory function, use of humidified supplemental oxygen and mechanical ventilation as necessary for support of gas exchange, optimization of analgesia, and prompt treatment of associated injuries.</p>
<p><b>Massive hemothorax</b></p> 	<p>Penetrating, and sometimes blunt injuries to the chest can cause the accumulation of greater than 1500 mL of blood in the pleural space. Blood loss of this magnitude in the chest can present with symptoms and signs of respiratory compromise and profound shock. When a chest tube, placed in the setting of hemodynamic instability, evacuates 1500 mL of blood immediately or continues to drain 200-300 mL/hour, ongoing bleeding is likely and preparations for exploratory thoracotomy should be made. Such preparations include reassessment of the patient’s airway, breathing and circulation, thorough evaluation for associated injuries, which may contribute to ongoing hemorrhage, and meticulous efforts to warm the patient and treat transfusion-associated coagulopathy.</p> <p>Note: Rapid exsanguinations can occur into the chest without significant chest tube output if the tube is clogged or kinked. Physicians attempting to rule out ongoing sources of hemorrhage should not be reassured by low volume chest tube output and patients must be frequently re-examined and re-x-rayed as necessary.</p>
<p><b>Cardiac tamponade</b></p> 	<p>Depending on the distensibility of the pericardium as little as 60-100 mL of blood in the pericardial sac can increase pressures around the heart sufficient to limit venous return to the right atrium. Obstructive shock from loss of cardiac preload can initially be compensated by sympathetic measures directed at maintaining cardiac output (tachycardia, vasoconstriction of the venous bed). However, unless mounting pressure is relieved, dramatic decreases in cardiac output and coronary perfusion can ensue. This condition is known as cardiac tamponade.</p> <p>Penetrating injuries to the chest, especially those at the precordium (the anterior chest bounded by the clavicles above, the midclavicular lines laterally and an imaginary line connecting the intersections of the midclavicular lines with the costal margin below) should raise the suspicion of cardiac injury. Many of these patients do not present with the classic features of Beck’s triad (jugular venous distension, muffled heart sounds, hypotension), but the presence of a high risk wound and hypotension confirms the diagnosis. Patients with high risk injuries who are normotensive should be further evaluated in order to exclude the presence of occult cardiac tamponade.</p> <p>Blood in the pericardial sac of hemodynamically stable patients is readily identified by the creation of a subxiphoid pericardial window. This is done in the operating room with preparation for immediate thoracotomy or median sternotomy should any blood be detected. More recently, transthoracic echocardiography and the incorporation of parasternal and subxiphoid views into the FAST ultrasound evaluation have been shown to be useful, non-invasive means to reliably exclude the presence of cardiac tamponade.</p> <p>Precordial penetrating trauma accompanied by severe hypotension refractory to efforts aimed at increasing preload, necessitates immediate (emergency department) left anterior thoracotomy for release of tamponade, along with cardiorrhaphy. After adequacy of the airway has been established, exposure of the heart can be obtained quickly with a left anterior thoracotomy using a generous incision at the fifth intercostal space. This should be carried medially across the sternum to the right chest to optimize exposure. Once a rib spreading retractor has been placed, the lung is reflected superiorly and the inferior pulmonary ligament is divided as necessary in order to improve exposure. The pericardium is divided longitudinally, anterior and parallel to the phrenic nerve. Cardiac lacerations can be occluded digitally or with the inflated balloon of a foley catheter while the patient is fluid resuscitated. It is often the right ventricle (RV) which is injured in penetrating trauma because of its anterior position – lacerations of the RV and other cardiac surfaces can be repaired using interrupted, pledgeted horizontal mattress sutures of 2-0 prolene, with care to avoid damage to the coronary arteries. If cardiac function does not return immediately after these measures, consideration should be given to clamping of the descending aorta to increase the impact of fluid resuscitation on preload. Hemodynamically compensated patients with tamponade can be emergently explored in the operating room. Once obvious injuries have been addressed, echocardiography to exclude intracardiac (valvular) injuries becomes a priority.</p> <p>Outcomes after emergency department thoracotomy are variable and are dependent on the mechanism of injury and the severity of hemodynamic compromise. Low velocity penetrating injuries causing cardiac tamponade rather than free bleeding in patients who are stable enough to be taken to the operating room for exploration are associated with the best outcomes (50% survival), while patients suffering high velocity penetrating or blunt injuries to the heart who lose their vital signs in the field have survival rates less than 1% in most series.</p>

**Secondary survey: Potentially life-threatening injuries**

**Simple pneumothorax**

Pneumothoraces undetectable by conventional supine, anteroposterior radiographs, but evident on abdominal computed tomography (occult pneumothorax OP) may account for as many as 55% of all pneumothoraces. With increasing use of CT scans more OP are being identified that in the past would have escaped detection and treatment with tube thoracostomy. The available literature on OP management is conflicting. Two randomized trials have reached opposite conclusions: that OP may progress to tension pneumothorax in patients receiving positive pressure ventilation, and that OP can be safely observed, even in mechanically ventilated patients. Until this issue is resolved, it would appear that an expectant approach can safely be taken for small OP in non-ventilated patients. Pneumothoraces usually resolve spontaneously (1.5%/day) over time, and these patients need not be subjected to the complications and costs associated with chest tube placement. For patients receiving positive pressure ventilation, the overall clinical picture and consequences of OP progression should be weighed before an expectant approach is taken.

Extra-anatomic air in the mediastinum, pericardium or subcutaneous tissue in the absence of pneumothorax must be carefully evaluated. Air in these positions is usually a benign finding, but may also signal the presence of occult esophageal, tracheobronchial or pulmonary injury. Our approach has been to place a chest tube on the same side as the subcutaneous emphysema. Patients with mediastinal air are investigated with bronchoscopy and esophagoscopy with or without esophagography.

**Hemothorax**

Hemothorax is usually caused by intercostals artery or pulmonary bleeding. Placement of a chest tube (32 or 36 F) evacuates blood, allows lung reexpansion, prevents the formation of a clotted hemothorax and allows assessment of ongoing bleeding. The decision to operate on a patient with a hemothorax is largely dependent on hemodynamic status and volume of blood loss (>1500mL).

**Blunt cardiac injury**

Typically, this injury is suspected in the presence of new post-traumatic arrhythmias and persistent hypotension in the absence of ongoing blood loss. Diagnosis requires a high degree of suspicion and is often confirmed by transthoracic or transesophageal echocardiography. Treatment in the absence of structural abnormality, is supportive and directed against specific abnormalities. Arrhythmias are controlled in using standard algorithms and shock is managed with pulmonary artery catheter monitoring and optimization of fluid resuscitation and inotropic support.

At most centers, ECG is used as the primary screening modality (NPV 98%) – patients with normal ECGs at admission are not evaluated further unless arrhythmias or hypotension develop. ECG abnormalities prompt a period of continuous electrocardiographic and blood pressure monitoring and echocardiography.

**Pulmonary contusion (PC)**

Pulmonary contusion (alveolar hemorrhage and parenchymal destruction) is common in patients sustaining multiple trauma (17%). Lung tissue with its many gas-fluid interfaces is thought to be uniquely at risk of disruptive phenomena such as shearing, inertial effects and implosion. Direct laceration from fractured ribs, and bleeding into normal lung areas with resultant bronchospasm may compound the above mechanisms. PC is diagnosed in dyspneic patients with hypoxemia who have chest infiltrates on x-ray. These infiltrates typically appear within 4-6 hours of injury, and may become more obvious over the next 48 hours. Computed tomography of the chest is more sensitive in the identification of PC.

With supportive care including supplemental oxygen and selective use of mechanical ventilation, chest physiotherapy, judicious use of intravenous fluids, and adequate analgesia, the effects of the contusion resolve in most cases. Severe contusions with dense pulmonary consolidation and those contusions evolving to ARDS may require prolonged support. Ventilator associated pneumonia must be suspected and treated promptly. There is no role for prophylactic antibiotic therapy and corticosteroids are currently not used in the treatment of PC.

**Blunt aortic injury**

Sudden deceleration, either from head on or side impact collisions can result in shear forces on the aorta at points of fixation such as the proximal descending aorta. Aortic transection is usually fatal at the scene, but if bleeding is contained in the overlying adventitial tissue, patients may survive to present at the trauma center. Because of the possibility of delayed rupture, aggressive measures to confirm the diagnosis and initiate treatment must be taken. The diagnosis is usually suspected based on mechanism. Chest x-ray may lend support to the diagnosis if the mediastinum is widened to 50% of the thoracic diameter, or if the aortic contour is obscured. Suspected cases of BAI should be further evaluated with CT scanning of the chest. Treatment of BAI depends in part on the impact of associated injuries. Severe compromise of airway and gas exchange, as always are addressed immediately. Active bleeding and contamination are controlled next, according to the principles of damage control. Care is taken to maintain normothermia, and correct coagulopathy and acidosis. Blood pressure and heart rate are aggressively controlled (mean arterial pressure less than 60, heart rate less than 60 if possible) using beta blockers (esmolol infusion) and sodium nitroprusside in an effort to reduce the likelihood of delayed aortic hemorrhage. If some degree of stability of associated injuries can be achieved, definitive aortic repair can be attempted (open interposition grafting with or without bypass or endovascular stenting).

**Injury to the diaphragm**

The movement, structure and span of the diaphragm make it uniquely susceptible to injury by both penetrating and blunt mechanisms. Thoracoabdominal penetrating injuries (i.e. injuries to the upper abdomen or lower chest) should raise the suspicion that the trajectory has transgressed the diaphragm. Sudden increases in abdominal pressures in blunt trauma can also cause blowouts of the diaphragm – usually on the weaker and less protected left side.

The diagnosis of diaphragmatic injuries is difficult as this structure is not easily imaged, and because the high frequency of associated injuries to the head, aorta, liver, spleen, stomach, pelvis and long bones may distract diagnostic efforts. If laparotomy is indicated for other reasons, both diaphragms should be thoroughly inspected. In patients initially managed non-operatively the diagnosis may be evident on conventional radiographs or CT scans demonstrating herniation of visceral structures to the hemithorax. However, small injuries are usually undetectable this way – these injuries may become more obvious over time as they grow in size under the influence of unremitting diaphragmatic contraction and negative intrathoracic pressure. Direct visualization using diagnostic laparoscopy is the best approach when diaphragmatic injury is suspected.

The treatment of small diaphragmatic injuries is straightforward. Careful placement of a chest tube as needed, reduction of abdominal viscera from the chest, direct suture of the diaphragmatic defect via a transabdominal approach using interrupted horizontal mattress sutures (0-prolene), and careful evaluation and treatment of associated injuries in the order of their severity



**Esophageal injuries**

Esophageal injuries should be suspected with any penetrating injuries of the chest. Clinically, these injuries may be characterized by respiratory distress, dysphagia, odynophagia, hemoptysis or hematemesis. The presence of any of these findings or mediastinal or subcutaneous air on chest x-ray should prompt a thorough evaluation for esophageal injury. Delays in diagnosis could allow progression of mediastinal contamination and severe sepsis. Thoracic injuries can be evaluated with rigid or flexible esophagoscopy, or contrast esophagography. The airway should be evaluated by endoscopy at the same time.

The main principles of therapy are early intervention (usually via a right thoracotomy for thoracic esophageal injuries), thorough debridement and drainage, and primary repair where possible with the liberal use of tissue flaps to buttress suture lines and prevent delayed formation of tracheoesophageal fistulas. Broad spectrum antibiotics and intensive care unit support will be required for variable periods depending on the severity of sepsis and underlying medical conditions.

