TRAUMA II: Diagnosis and Management

All trauma patients require a systematic approach to assessment and management using Advanced Trauma Life Support (ATLS) as a foundation to improve outcomes, regardless of the severity of their injury. ATLS requires that patients are triaged based on the mechanism of injury, severity of injury, hemodynamic stability and resources available. Hemodynamic stability is assessed by level of consciousness, skin color and pulse - and can be rapidly assessed during the primary survey (*see Trauma I*). It is important to understand that patients who are hemodynamically "stable" or normal does not always correlate with a normal physiologic state and, in fact, may represent a state of compensated shock. Prompt assessment and diagnosis of traumatic injuries combined with early intervention may reverse or even stop the traumatic cascade before it manifests or becomes irreversible.

Hemodynamic instability is broadly defined as global or regional perfusion that is not adequate to support normal organ function. Trauma patients who are hemodynamically unstable as a result of their injuries require early recognition of shock in order to prevent the most common causes of mortality from trauma: hemorrhage, multiorgan dysfunction, and cardiopulmonary arrest. Recognizing the physiologic state of shock and initiating treatment takes priority over the definitive determination of the extent of injury.

The most common cause of shock in trauma patients is hypovolemia from hemorrhage, but patients may also suffer from cardiogenic, obstructive, or neurogenic shock (*see Shock module*). Shock is a clinical diagnosis of inadequate organ perfusion and tissue oxygenation, and can be determined early in the primary survey through an expeditious and careful physical examination. The major physiologic determinants of tissue perfusion and systemic blood pressure are cardiac output and systemic vascular resistance. One cannot simply rely on systolic blood pressure to diagnose shock, as compensatory mechanisms can prevent hypotension until up to 30% of blood volume is lost.

The clinical signs of shock include tachycardia, peripheral vasoconstriction, dyspnea, restlessness, diaphoresis, cool and clammy skin, metabolic acidosis, hypotension, and oliguria. *Refer to the Shock module within this curriculum for a review of the different types and classes of shock.* Additional studies, such as chest or pelvis x-ray, or ultrasonography, may provide confirmatory evidence for the cause of shock, but should not delay appropriate resuscitation. The following content will focus on the systematic approach to the most common traumatic injuries.

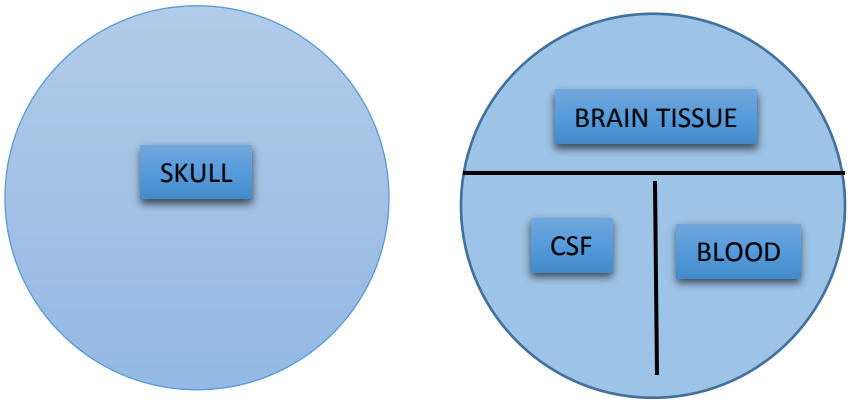
NEUROLOGIC INJURY

1. Traumatic Brain Injury

Epidemiology/Pathophysiology

The primary injury in traumatic brain injury is irreversible. It is induced by the mechanical force and occurs at the moment of injury. A contusion, hematoma, or laceration of the brain will

disrupt microtubules, neurofilaments, axons, and the blood brain barrier which lead to the immediate clinical effects. The brain tissue that surrounds the primary injury tissue is known as traumatic penumbra. The **penumbra** is the region at risk of secondary injury which is delayed from the time of impact and can extend the primary injury. Interventions and treatments attempt to mitigate secondary injury as the traumatic penumbra can be reversed and recovered. In order to understand the physiology behind traumatic brain injury, one must understand the concept of the **Monro-Kellie doctrine** which hypothesizes that the sum of volumes of brain, CSF, and intracranial blood must remain constant as the rigid vault of the cranial skull cannot change. An increase in one should cause a decrease in one or both of the remaining two. An imbalance or autonomic dysregulation can lead to increased intracranial pressure (ICP) which is the primary target of many therapies. Hypotension and hypoxia have been implicated as important extracranial insults that can aggravate this balance and cause further permanent injury. The goal of treatment is to maintain normal ranges of **cerebral perfusion pressure (CPP)** which is in the range of 60-70 by targeting ICP and mean arterial pressure (MAP) using the equation of **CPP=MAP-ICP**. Understanding how therapies, such as hypertonic saline, surgery, and hyperventilation work on a physiologic level to affect the volume of the brain, cerebrospinal fluid, and intracranial blood allows for targeted management.



Signs/Symptoms

The initial primary injury of traumatic brain injury leads to immediate clinical effects which can be assessed using the Glasgow Coma Scale (GCS). The GCS is a rapid and standardized tool for assessing focal and global brain dysfunction. It is reproducible and can be used to monitor a patient's neurologic status over time. In addition, papillary, corneal and gag responses are valuable in assessing in an unresponsive patient.

Glasgow Coma Scale

RESPONSE	SCALE	SCORE
EYE OPENING	SPONTANEOUS	4
	TO VOICE	3
	TO PAIN	2
	NONE	1

VERBAL	**ORIENTED** **CONFUSED** **INAPPROPORATE WORDS** **INCOMPREHENSIBLE WORDS** **NONE**	**5** **4** **3** **2** **1**
MOTOR	**OBEYS COMMANDS** **LOCALIZES PAIN** **WITHDRAWS** **ABNORMAL FLEXION** **ABNORMAL EXTENSION** **NONE**	**6** **5** **4** **3** **2** **1**

Traumatic Brain Injury signs and symptoms (increase with level of severity);

Mild TBI: GCS13-15

Nausea
Headache
Vertigo

Moderate TBI: GCS 9-12

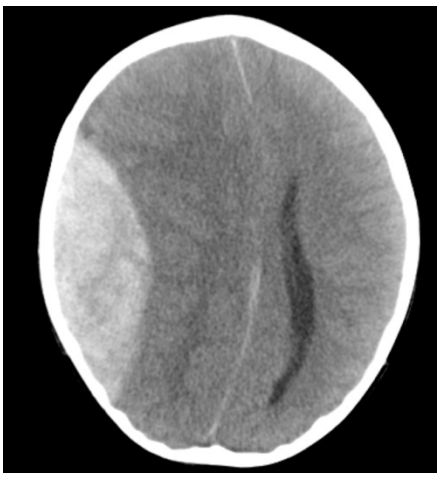
Vomiting
Memory loss
Unsteady gait

Severe TBI: GCS 3-8

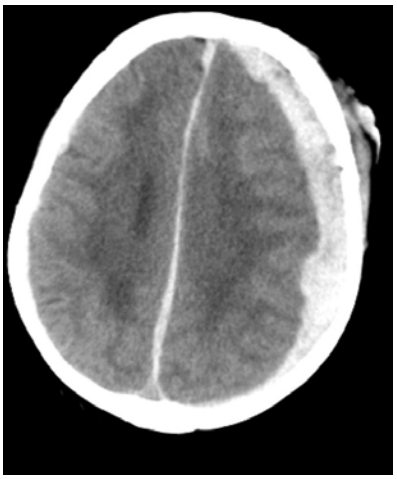
Obtunded (coma is defined as GCS $\leq$ 8)
Cushing reflex due to autonomic response to increased ICP ($\uparrow$BP, Irregular breathing, $\downarrow$Heart Rate)

Relevant Diagnostic Studies

The standard diagnostic imaging of traumatic brain injury is a non-contrast brain computed tomography (CT). Mass lesions have specific findings on CT and can be rapidly identified based on their location and shape. For example, an epidural hematoma has a biconvex shape, it usually located between the skull and dura mater in the temporal region from tearing of the meningeal arteries. In contrast, a subdural hematoma has a crescent shape, is usually located between the dura and arachnoid mater and results from tearing of bridging veins.



Epidural Hematoma



Subdural Hematoma

Other injuries detected on CT are intraparenchymal hemorrhage or contusions and subarachnoid hemorrhages. Diffuse axonal injury is best imaged using an MRI.

Non-operative Management

Not all patients with traumatic brain injury require operative management. However, patients that are postoperative and those for whom surgery was not indicated will require nonsurgical management. The Trauma Brain Foundation task force delineates the medical management of severe traumatic brain injury. Guidelines can be found on https://www.braintrauma.org. The mainstay of treatment depends on the severity of the injury and targets prevention of secondary traumatic brain injury. The primary target of treatment is control of the intracranial pressure in a normal range by maintaining adequate cerebral perfusion and oxygenation. Hypertonic saline, osmotic diuretics, elevation of the head of the bed, barbiturates, and sedation all can help to reduce ICP, each with their own advantages and disadvantages. Hypotension should be avoided and intravascular volume should be maintained, however, over-resuscitation should also be avoided. Over resuscitation with crystalloid solutions may worsen brain edema. In patients that have mild traumatic brain injuries, treatment might involve close observation and frequent neurologic checks to monitor for progression or deterioration.

Operative Management

Any patient with a severe TBI causing increased ICP's should be evaluated for possible operative management. Most of the management involves evacuation of the hematoma, via a craniotomy or craniectomy, which depends on the injury and determines if the skull flap can be left on or off. A decompressive craniectomy for the management of medically intractable intracranial hypertension remains controversial.

Basic Postoperative Care

Most common postoperative care involves medical management to control ICP's, and rare but severe complications involve brain edema and swelling. Much of the management focuses on early detection of postoperative complications, including those not always related to the brain but common to many postoperative patients such as pneumonia, deep vein thrombosis, urinary tract infections, and wound complications. Monitoring patients that are critically ill occurs in the intensive care unit and is multidisciplinary.

2. Blunt Cerebrovascular Injury

Epidemiology/Pathophysiology

Blunt injury to the carotid or vertebral arteries (BCVI) is relatively uncommon with an incidence of about 1% of patients sustaining direct trauma. Traumatic mechanisms of injury that cause rapid deceleration, hyperextension, rotation, or direct blows and manipulation to the head, face or neck may cause the arterial walls or intima to stretch and tear which produce intimal flap/dissection, occlusion/thrombosis, pseudoaneurysms or complete transection. Although the incidence is low, left untreated, these injuries can lead to significant and devastating neurologic morbidity and mortality. Maintaining a high index of suspicion based on patient risk factors and utilizing established screening criteria for patients suspected to have blunt cerebrovascular injuries can lead to improved outcomes. The goal is to diagnose and treat this injury prior to the patient becoming symptomatic.

Signs/Symptoms

Signs and symptoms of blunt cerebrovascular injury can be present on initial physical exam and late findings can lead to significant neurologic deficits. Emphasis is on early recognition of signs and symptoms and risk factors for BCVI.

Signs and symptoms of BCVI:

- Arterial hemorrhage from neck, nose or mouth
- Cervical bruit in a patient who is less than 50 years old
- Expanding cervical hematoma
- Focal neurologic deficit
  - Transient Ischemic Attack
  - Hemiparesis
  - Horner's Syndrome
- Any neurologic deficit not explained by a head CT
- Stroke on CT or MRI

Risk factors for BCVI:

- Facial fractures:
  - Displaced mid-face fracture such as LeFort II or III injuries
  - Mandible fracture
  - Complex skull fracture
  - Basilar skull fracture
  - Occipital condyle fracture
- Closed head injury:
  - Diffuse Axonal Injury
  - GCS < 6
- Cervical spine or spinal cord injury:
  - Subluxation
  - Ligamentous injury
  - Cervical spine fractures
- Neck injury:
  - Near hanging with anoxic brain injury
  - Clothesline injury
  - Seat belt abrasion
- Traumatic brain injury with thoracic injuries
- Scalp degloving
- Thoracic vascular injuries
- Blunt cardiac rupture

Relevant Diagnostic Studies

Once risk factors for BCVI have been identified, the standard diagnostic imaging is a CT angiogram (CTA) of the neck. Blunt cerebrovascular injuries are classified using a grading scale of I-V.

Denver Grading Scale for BCVI

Grade of Injury	Findings on CTA Imaging of the Neck
I	**Irregularity of the vessel wall or Dissection/Intramural hematoma with <25% luminal stenosis**
II	**Intramural thrombus or Raised intimal flap or Dissection/Intramural hematoma with >25% luminal stenosis**
III	**Pseudoaneurysm**
IV	**Vessel occlusion**
V	**Vessel transection**

Non-operative Management

Much of the cornerstone of therapy for BCVI is antithrombotic therapy, either with antiplatelet or anticoagulant therapy, as long as the patient does not have contraindications such as intracranial hemorrhage or active bleeding. Repeat imaging with a CTA of the neck evaluates whether the lesion has healed and if prolonged antithrombotic therapy is required.

Operative Management

Surgical or interventional therapy for patients with BCVI depends on the type and location of the vascular lesion, concomitant injuries precluding medical therapy, availability of interventional techniques, and anatomic feasibility for operative repair.

Basic Postoperative Care

Most patients will require immediate postoperative care in the intensive care unit with strict blood pressure parameters, ICP management, and frequent neurologic physical exams. The approach to patient care is multidisciplinary and involves care not just in the immediate postoperative period but also long term physical and mental rehabilitation.

3. Blunt Traumatic Cervical Spine Injuries

Epidemiology/Pathophysiology

Traumatic injuries to the spine can involve the bone, ligaments, discs, muscles, and spinal cord, and can affect any part of the spine. For the purposes of this chapter, we will focus on blunt traumatic cervical spine injuries. Spinal fractures can occur from a break or disruption of the spinal vertebrae with damage to the attached ligaments. Traumatic spinal cord injury is a major cause of morbidity and mortality in patients. Blunt traumatic injuries to the spine can lead to significant neurologic deficits, associated injuries, and shock. Patients with underlying spine disease are at higher risk for unstable injury. Blunt traumatic cervical spine injuries most commonly are due to flexion-extension type injuries in high energy motor collisions, although they can also occur from falls, diving, sports, and violence injuries.

Signs/Symptoms

As described in the *Trauma I module*, all patients suffering blunt trauma must be promptly assessed and spine immobilization must be maintained until the spine can be properly cleared of injury. Presence of midline cervical pain or tenderness, focal neurologic deficit, or altered mental status can raise suspicion for a cervical spine injury. Spinal cord injuries can vary in severity depending on the level of injury and must be carefully assessed. In addition, cervical spine injury has a high risk for concomitant injuries of the aerodigestive (vocal cord injury, hoarseness, esophageal injury) and neurovascular systems (BCVI), which must be assessed. **Complete spinal cord injury** is when all functions below the injured area are lost, whereas an

incomplete spinal cord injury involves preservation of motor or sensory function below the level of injury in the spinal cord. Quadriplegia (paralysis of all four limbs) or paraplegia (usually involving lower limbs) can be complete or incomplete. Complete quadriplegia due to a high spinal cord disruption can be life threatening as it can lead to respiratory compromise and neurogenic shock due to the autonomic disruption of the sympathetic fibers. Other signs and symptoms include loss of bladder or bowel function, inability to regulate body temperature, and sexual dysfunction.

Incomplete spinal cord injury syndromes are listed below:

Syndrome	Motor Function	Sensation
Central Cord	Preserved in lower extremities, diminished in upper extremities	Pain and temperature preserved in lower extremities, diminished in upper extremities
Anterior Cord	Diminished	Diminished pain and temperature Preserved position, vibration, crude touch
Brown-Sequard	Ipsilateral loss	Ipsilateral loss of proprioception and vibration Contralateral loss of pain and temperature

Relevant Diagnostic Studies

Patients who cannot be cleared clinically as having a cervical spine injury will require imaging. The diagnostic imaging of choice is a noncontrast CT scan of the neck. A ligamentous injury of the cervical spine is best imaged with an MRI.

Non-operative Management

Most patients with blunt traumatic cervical spine injuries that are considered neurologically stable or without neurologic deficits can be managed non-operatively with cervical spine precautions and a cervical spine collar. Patients who present with significant spinal cord injury usually require intensive care as they can have respiratory compromise which requires intubation and mechanical ventilator support. Likewise, patients in **neurogenic shock** (distributive shock resulting in low blood pressure and bradycardia), require close hemodynamic and neurologic monitoring and management with isotonic fluids and pressors to maintain intravascular volume and tone in the ICU.

Operative Management

Indications for operative intervention usually depend on the type of fracture and if it is considered unstable. Operative management for spinal cord injury depends on whether surgical intervention will provide improved neurologic outcomes.

Basic Postoperative Care

Postoperative care in the immediate setting involves cervical spine precautions, maintaining hemodynamics and frequent neurologic checks. Patients are also closely monitored for decubitus ulcers, urinary tract infections, and aspiration. Long term care involves physical rehabilitation once cleared by the spine surgeons.

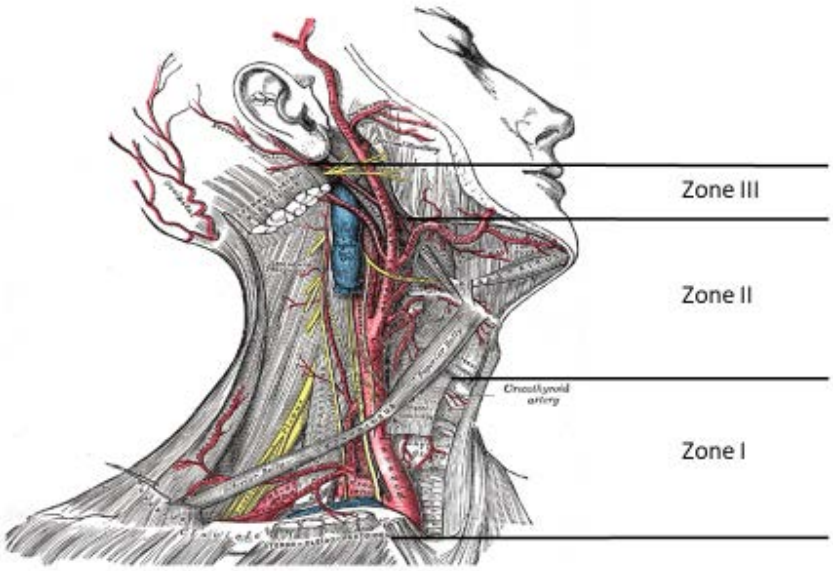
4. Penetrating Neck injury

Epidemiology/Pathophysiology

Penetrating neck injuries make up 5-10% of traumatic injuries in adults and are generally caused by bullets, knives, or other impaling objects, with high velocity guns causing more serious injuries. Blunt neck injuries are diagnosed in approximately 1% of blunt traumas, and typically include either cerebrovascular injury or airway trauma resulting from direct force to the neck, hyperextension with contralateral rotation of the head and neck, lacerations secondary to bony fractures, bleeding within or disruption of the soft tissue.

Signs/Symptoms

Hemodynamically unstable patients with blunt or penetrating neck injuries show obvious signs of aerodigestive or vascular injury. The signs and symptoms exhibited will vary depending on the zone of neck involved. Penetrating wounds to the neck have traditionally been grouped into three separate zones to assist in the evaluation of neck injuries.



Taken from: https://commons.wikimedia.org/wiki/File:External_carotid_a.gif
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Zone	Borders	Contents
1	Sternal notch and clavicles to the cricoid cartilage	• Subclavian arteries and veins • Internal jugular veins • Proximal carotid arteries • Vertebral artery • Apices of the lungs • Trachea • Esophagus • Spinal cord • Thoracic duct • Thyroid gland
2	Cricoid cartilage to the angle of the mandible	• Common carotid arteries • Internal and external branches of carotid arteries • Vertebral arteries • Jugular veins • Trachea • Esophagus • Larynx • Pharynx • Spinal cord • Vagus and recurrent laryngeal nerves
3	Above the angle of the mandible up to the base of the skull	• Distal portion of the internal carotid arteries • Vertebral arteries • Jugular veins • Pharynx • Spinal cord • Cranial nerves IX, X, XI, XII • Sympathetic chain

The presence of "hard signs" of injury signifies a serious injury to the neck that generally requires emergent intervention. "Hard signs" of vascular injury include:

- Active hemorrhage
- Expanding or pulsatile hematoma
- Thrills or bruits over wound
- Absent or diminished pulse (may manifest as stroke in the neck)
- Extremity ischemia (pain, pallor, paralysis, cool to touch)

"Hard signs" of aerodigestive injury include:

- Air bubbling from a wound
- Massive hemoptysis or hematemesis
- Respiratory distress

Every penetrating neck wound must be inspected (but not probed) to determine if the platysma muscle has been violated. The presence of violation of the platysma and the patient's signs/symptoms and stability will determine the next step in management. Hemodynamically unstable patients require rapid stabilization of the airway, breathing and circulation. In both penetrating and blunt neck injuries, if the airway is inadequate but anatomic structures are preserved, rapid sequence intubation is appropriate. If there is massive facial or neck trauma, hemoptysis, significant bleeding in the oropharynx or laryngeal obstruction, a surgical airway is recommended. With partial or complete transection and exposure of the trachea, the caudad portion of the exposed trachea should be stabilized (via a towel clip or clamp) and intubated directly.

Relevant Diagnostic Studies

Hemodynamically unstable patients with neck injury require operative intervention. Once the patient is stabilized, additional diagnostic studies may augment management. A rigid esophagoscopy or esophagogastroduodenoscopy may assist with localizing an esophageal injury. Alternatively, in the operating room, the esophagus can be filled with methylene blue dye in saline and the exposed esophagus inspected for full thickness dye staining. A bronchoscopy may assist with localizing a tracheal injury.

Operative Management

Hemodynamically unstable patients with "hard signs" of penetrating neck injury require immediate hemorrhage control and emergent neck exploration. For Zone 1 injuries, intrapleural hemorrhage is approached via a sternotomy with or without clavicular extension or high anterolateral thoracotomy (third or fourth intercostal space above the male nipple). For Zone II and III injuries, external hemorrhage can be initially controlled with finger compression or balloon inserted through injury. For Zone II injuries, standard anterior oblique neck incision

along the anterior border of the sternocleidomastoid muscle on the side of injury is performed, followed by a formal neck exploration with exposure and inspection of the carotid artery, internal jugular vein, esophagus and trachea. Carotid injuries are repaired with interrupted sutures, patch angioplasty, segmental resection with end-to-end anastomosis or segmental resection with interposition graft. Internal jugular vein injuries are repaired with a lateral venorrhaphy for wall defects or ligation for extensive injuries (should avoid bilateral ligation for fear of severe cerebral edema). Esophageal injuries are repaired with a 2 layer (mucosa and muscularis layer) closure with drainage. Tracheal injuries are repaired using absorbable interrupted sutures for an airtight seal. With extensive injury, a tracheostomy tube can be placed in the defect until formal reconstruction. For Zone III injuries, exposure of the distal internal carotid artery at the base of the skull is obtained by a "stepladder" mandibulotomy, subluxation of the temporomandibular joint, or a vertical ramus osteotomy. Vertebral artery injury can be temporarily managed with packing, with definitive proximal embolization in interventional radiology.

THORACIC INJURY

Blunt thoracic injuries account for approximately 8% of all trauma admissions in the United States, are often related to rapid-deceleration, from high-speed motor vehicle collision, falls from significant height, or direct force to the chest. The majority of patients with stable thoracic injuries can be managed non-operatively. This is likely due to the protection provided by the ribs and the limited parenchymal response following injury to the lungs. These injuries include 1) traumatic rib fractures, 2) traumatic pneumothorax, and 3) traumatic thoracic aortic injury. The presentation of penetrating thoracic trauma can vary widely, from stable patients with few complaints to hemodynamically unstable patients requiring immediate life-saving interventions. Even apparently stable patients with penetrating chest injuries can deteriorate precipitously and a focused evaluation must be rapidly performed to assess for life-threatening conditions.

1. Traumatic Rib Fractures

Epidemiology/Pathophysiology

Traumatic rib fractures generally occur when significant blunt forces impact the chest wall. Although most injuries occur after motor collisions, falls and assaults, traumatic rib fractures can also be sustained after a penetrating injury. Rib fractures are classified based on their location (right or left), number, and if they are displaced or nondisplaced. A **simple rib fracture** is one that occurs in a single rib and is nondisplaced. **Complex rib fractures** are usually misaligned or nondisplaced. When multiple adjacent ribs are broken in two or more places, this is termed **flail chest**, as it appears that the segment is free floating. This flail may be radiographic or mechanical (not all radiographic flails have a mechanical flail). A mechanical flail is when paradoxical movement of the chest wall is noted during the respiratory cycle. Given the proximity of the rib cage to many other vital structures, much care is given to early identification and treatment of associated injuries. For example, fractures of the first and second rib

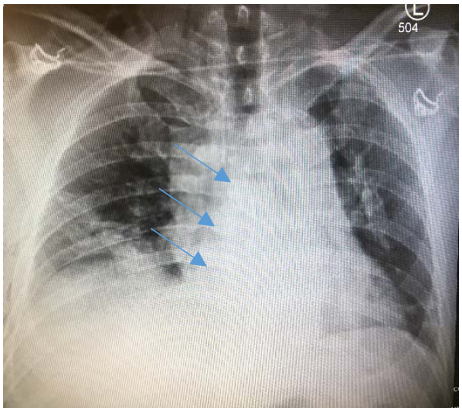
fractures, although rare, can be associated with injuries to the great vessels, brachial nerve plexus damage or maxillofacial trauma. Multiple traumatic rib fractures carry a high degree of morbidity and mortality, especially in the elderly population.

Signs/Symptoms

Many of the signs and symptoms associated with traumatic rib fractures depend on the location, number of ribs broken, and associated injuries. The most common symptom of rib fractures is pain that is usually elicited with breathing, coughing, or moving. Nondisplaced rib fractures can cause a pulmonary contusion, which can be asymptomatic or lead to shortness of breath and respiratory distress. Displaced rib fractures can cause a pulmonary laceration or tear an intercostal artery with subsequent pneumothorax or hemothorax, which can be life threatening. Fractured lower ribs can injure the liver, spleen, and diaphragm, and if significant enough, can lead to hemorrhagic shock. **Flail chest** due to the underlying contusion are associated with respiratory failure and subsequent Acute Respiratory Distress Syndrome (ARDS).

Relevant Diagnostic Studies

The sensitivity of the chest x-ray for rib fractures is 60%. A CT scan of the chest is more sensitive in identifying rib fractures and can help determine if there are associated injuries to the lung or other vital organs.



Example of right rib fractures

Non-operative Management

The mainstay of therapy for traumatic rib fractures is early and effective pain control. Pain control can be initiated with oral or intravenous narcotics, adjuvant muscle relaxants, nerve blocks, and epidural anesthesia. Geriatric trauma patients with multiple rib fractures are usually admitted to the intensive care unit for closer monitoring as they are at high risk for developing respiratory distress or pneumonia. Flail chest can sometimes alter the mechanics of breathing, called **paradoxical breathing**, and may require ventilator support if it is significantly affecting the patient's ventilation and oxygenation.

Operative Management

Operative fixation for traumatic rib fractures depends on the injury pattern and how the patient is doing clinically. Rib fixation may be warranted in extensive flail chest segments, open chest wounds or displaced rib fractures that are causing the patient significant morbidity, such as refractory pain despite maximum pain control, failure to wean from mechanical ventilation, chest wall instability due to nonunion of rib fractures.

Basic Postoperative Care

Postoperative treatment for traumatic rib fractures depends on whether the operative approach also involved repair of associated injuries. If a thoracotomy or Video Assisted Thoracoscopic Surgery (VATS) was performed to evacuate a hemothorax or repair a diaphragm injury, most patients will have a chest tube postoperatively and be closely monitored for rebleeding or respiratory distress. Not all rib fixations require violation of the pleural space or a thoracotomy, and patients can be treated without a tube thoracostomy. Regardless of the technique, pain control, is once again, the crux of early postoperative management, as patients are required to ambulate, clear secretions, and cough.

2. Traumatic Pneumothorax

Epidemiology/Pathophysiology

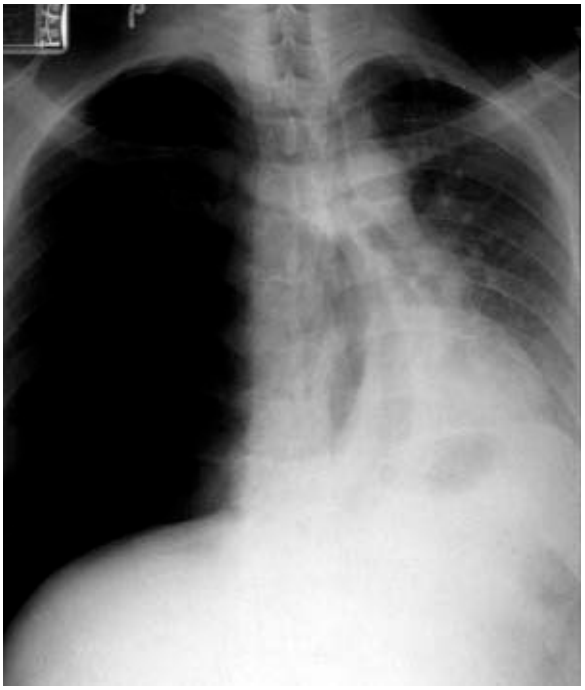
A pneumothorax is defined as air in the pleural space between the lung and chest wall, and it can result from blunt or penetrating trauma to the thoracic or thoracoabdominal cavity**.** Pneumothoraces can be clinically insignificant to life threatening. A **simple pneumothorax**, is usually due to traumatic rib fractures that have violated the pleural space or air from an injured lung that is escaping into the pleural cavity. An **open pneumothorax** is when air enters the thoracic cavity through an open chest wound and pressures between the thorax and atmosphere become equal. A **tension pneumothorax** is when air enters the thoracic cavity but cannot escape resulting in intrapleural pressure that is higher than atmospheric pressure, causing the heart and great vessels to shift from the side of injury, compressing return of blood to the heart, which can lead to complete circulatory arrest if left untreated. *See Trauma I Module.*

Signs/Symptoms

Patients with pneumothoraces can be asymptomatic or present in cardiac arrest if tension is present. Small simple pneumothoraces usually do not affect the patients clinically. Larger simple pneumothoraces can cause the patient to have chest pain, shortness of breath, dyspnea on exertion, tachypnea, and hypoxia. An open pneumothorax can cause the patient significant respiratory distress, hypoxia, hypercarbia, and pulmonary shunting. A tension pneumothorax causes hypotension, tachycardia, and hypoxia with imminent respiratory and cardiac collapse.

Relevant Diagnostic Studies

Most pneumothoraces can be diagnosed on a chest x-ray. A pneumothorax that is not seen on a chest x-ray but seen on a CT scan of the chest is termed **occult**. Open and tension pneumothoraces are usually diagnosed clinically and are apparent on chest x-rays.



Example of a Tension Pneumothorax

Non-operative Management

Most simple small pneumothoraces do not require any treatment other than management of the inciting cause such as rib fractures. Larger simple pneumothoraces that are symptomatic warrant a tube thoracostomy and management of the chest tube. An open pneumothorax requires temporary coverage of the wound with three sides occluded in order to only allow air to escape out of the thoracic cavity, as well as a tube thoracostomy. A tension pneumothorax is treated with needle decompression (in the field) followed by tube thoracostomy. If the patient is in respiratory or cardiac arrest, the patient will also require intubation for airway protection and intensive care.

Operative Management

Open pneumothoraces usually require definitive operative repair and closure of the chest wall defect.

Basic Postoperative Care

Following a tube thoracostomy procedure or thoracotomy, chest tubes remain in place until the pneumothorax has resolved, there is no air leak in the pleurevac and patient is asymptomatic. Patients are monitored closely for adequate pain control, development of an **empyema** or recurrence of symptoms.

3. Traumatic Thoracic Aortic Injury

Epidemiology/Pathophysiology

Traumatic aortic injury is rare, however, it is estimated that 80-85% of patients with this type of injury die before arrival to the hospital. Due to the anatomic attachments of the aorta at the aortic root, ligamentum arteriosum (just distal to the left subclavian artery) and diaphragmatic hiatus, high energy from a rapid deceleration, motor collisions with or without ejection, falls greater than 25 feet, or direct impact with blunt forces to the chest wall can cause the aorta to stretch and tear. Associated injuries are usually multiple rib fractures, sternal and first rib fractures. Although less common, cardiac, splenic, and liver injuries are also seen with aortic injuries. The most common site for aortic injury is in the proximal descending aorta, distal to the left subclavian artery, as the aorta is tethered by the ligamentum arteriosum. Aortic injury is described as intramural hematoma, intimal flap, or pseudoaneurysm.

Signs/Symptoms

Traumatic thoracic aortic injury is not easy to diagnose as many patients present with other signs and symptoms of associated injuries. Extreme diligence and high clinical suspicion must be exercised based on mechanism and associated injuries due to the very high mortality of a missed thoracic aortic injury. Patients may present with chest pain, hoarseness, and shortness of breath, especially when lying flat. Signs can include unequal blood pressures in the upper extremities, systolic murmur over the precordium and shock.

Relevant Diagnostic Studies

In blunt thoracic injury, the presence of other chest wall injuries on CXR increases one's risk for significant intrathoracic injury, including:

- Scapula fracture
- Flail chest (three or more adjacent ribs fractured in two places, creating a floating segment)
- Multiple rib fractures (≥3) and displaced rib fractures
- Sternal fracture
- Posterior sternoclavicular dislocation

Blunt aortic injury occurs most commonly at the aortic isthmus just distal to the left subclavian artery. Other locations include the transverse arch, proximal ascending aorta, and descending aorta just proximal to the diaphragm. Rupture of the intimal and medial layers with subsequent hemorrhage into the mediastinal tissues produces obvious radiographic signs. Signs consistent with thoracic aortic injury on CXR include:

- Wide mediastinum (supine CXR >8 cm; upright CXR >6 cm)
- Obscured, indistinct or enlarged aortic knob
- Left "apical cap" (pleural blood above apex of left lung)
- Large left hemothorax
- Displacement of the left main stem bronchus
- Deviation of the esophagus to the right
- Deviation of trachea rightward and/or right mainstem bronchus downward
- Wide left paravertebral stripe

The diagnosis of blunt aortic injury is confirmed by transesophageal echocardiogram or CTA Chest. Grading of aortic injury is based on CTA are intimal tear, intramural hematoma, pseudoaneurysm or rupture (free rupture or periaortic hematoma). For patients who are hemodynamically unstable, transesophageal echocardiography can be performed at the bedside, or in the operating room prior to or during operation.

Non-operative Management

The goal of management of traumatic aortic injuries depends on whether the patient is stable or unstable, and presence of associated life threatening injuries. Most patients that are stable are managed initially with aggressive blood pressure and heart rate control and serial imaging.

Operative Management

Patients who are deemed operative candidates may undergo open or endovascular aortic repair.

Basic Postoperative Care

Postoperative care involves strict blood pressure and heart rate control and monitoring, pain control, and serial imaging.

4. Penetrating thoracic injury

Epidemiology/Pathophysiology

Penetrating thoracic injury may be caused by missiles, fragments or knives, and account for 7% of all trauma admissions, and 16% of penetrating injuries. While penetrating thoracic trauma occurs less common, it is often more deadly than blunt chest trauma.

Signs/Symptoms

A detailed physical examination will provide clues of life-threatening thoracic injuries. Hypoxia and increased work of breathing may be manifested by anxiety, confusion, combative behavior, dyspnea, or the use of accessory muscles. Patients with significant hypoxia or dyspnea require intubation. The presence of specific physical findings in the hemodynamically unstable patient supports the diagnosis of intrathoracic injury:

Intrathoracic injury	Signs and symptoms
Tension pneumothorax or hemothorax	• Distended neck veins • Tracheal deviation • Subcutaneous emphysema • Chest wall instability • Absent breath sounds
Tracheobronchial injury	• Hemoptysis • Large air leak after tube thoracostomy
Blunt aortic injury	• Chest pain • Difficulty breathing or swallowing • Cardiac murmur • Left subclavicular hematoma • Upper extremity hypertension • Bilateral femoral pulse deficit
Cardiac tamponade	• Distended neck veins • Pulsus paradoxus • Muffled heart sounds

Relevant Diagnostic Studies

In a hemodynamically unstable patient, diagnostic studies should not preclude intervention. Rapid diagnostics may aid in decision making as to which body cavity to operate in, especially in cases with multi-organ injuries or presence of foreign bodies (bullets, fragments, knives, impaled objects). An extended Focused Assessment with Sonography in Trauma (eFAST) will identify pneumothorax and pericardial tamponade. Pneumothorax is identified on eFAST by the following findings:

- Absence of lung sliding (along the parietal and visceral pleuras)
- Absence of “comet tails” (reverberation artifact caused by areas of interstitial edema on the visceral pleura)
- Presence of a lung point sign (juxtaposition of lung sliding and the absence of lung sliding in the same space, representing the edge of a pneumothorax)

Pericardial tamponade is identified on eFAST by the following findings:

- Anechoic area between pericardium and epicardium, signifying presence of pericardial effusion
- Inverted right atrial free wall, indicating right atrial collapse
- Persistent inward motion of the right ventricle after mitral valve opens, indicating right ventricular collapse
- Septum movement towards left ventricle during diastole, signifying presence of elevated right heart pressures

A single view PA CXR will identify life threatening conditions such as tension pneumothorax, hemothorax, or widened mediastinum. In general, a tension pneumothorax or hemothorax is picked up on physical examination followed by rapid intervention, not requiring a CXR.

Non-operative Management

Asymptomatic and hemodynamically stable patients with penetrating thoracic injuries and a normal CXR on presentation are observed for development of a delayed pneumothorax or hemothorax. A repeat examination and CXR are performed six hours after arrival. If the reevaluation is negative, the patient can be discharged, with instructions to return immediately should any concerning symptoms (e.g., increasing shortness of breath, painful swallowing) develop. A simple pneumothorax or clinically significant hemothorax (volumes greater than 300 to 500 mL, where 300 mL is needed for hemothorax to be seen on an upright CXR) should be treated with tube thoracostomy.

Operative Management

With signs and symptoms consistent with a tension pneumothorax, a tube thoracostomy should be performed during *Breathing* of the primary survey. *Refer to Trauma 1 module within this Curriculum for a description of tube thoracostomy.* Simple tube thoracostomy evacuates accumulated air and blood, allowing complete lung re-expansion with apposition to the chest wall. Massive hemothorax, defined as 1500 cc or more of blood in the pleural cavity or persistent chest tube output of 200 to 250 cc per hour for 3 consecutive hours, is generally considered an indication for operative thoracotomy. In addition, large air leaks from tracheobronchial injuries resulting in respiratory compromise generally require thoracotomy. Thoracic trauma resulting in persistent hemodynamic instability, without another obvious source, should also prompt emergent resuscitative thoracotomy. *Refer to Trauma 1 module within this Curriculum for the indications for a resuscitative thoracotomy.*

As a general rule, a median sternotomy or anterolateral thoracotomy, which can be extended as a clamshell, are the preferred incisions for exploring the hemodynamically unstable patient. For an anterolateral thoracotomy, the skin incision begins just lateral to the sternal edge and follows the inframammary crease up to the anterior axillary line at the level of the 5th intercostal space. The dissection is carried down to the chest wall dividing the pectoralis major and minor, and the medial edge of the serratus anterior muscles. The external and internal intercostal muscles are

divided, staying superior on the rib to avoid the neurovascular bundle. Use of a rib spreader aids in exposure. Upon entering the chest, blood and clot should be evacuated allowing a thorough examination of the hemithorax. Pneumonorrhaphy via a running simple or mattress suture is used to repair superficial pulmonary lacerations. More extensive lung injuries require resection including simple wedge resection, tractotomy, nonanatomic and formal anatomic resections. With hemorrhage from penetrating thoracic injuries, a tractotomy (stapling through the tract of the laceration to expose the bleeding at the base of the wound) can be used to assist with hemorrhage control.

Hemodynamically unstable patients with blunt aortic injury should be taken to the operating room for emergent exploration to determine the source of hemorrhage, which could be in the chest and/or abdomen. If the source of hemodynamic instability is blunt aortic injury, it should be immediately repaired. If aortic injury is identified, but life-threatening bleeding from another source is controlled, aortic repair can be delayed to allow resuscitation of the patient. Repair of blunt thoracic aortic injury can be performed using open or endovascular techniques. Open thoracic repair involves primary repair of the aorta or replacement of the aortic segment with a tube prosthetic graft through a thoracotomy incision. Endovascular thoracic aortic repair involves the placement of modular graft components that are delivered via the iliac or femoral arteries to line the thoracic aorta and exclude the injury from the circulation.

Basic Postoperative Care

Retained hemothorax - Blood left within the pleural cavity will clot and will not be evacuated with a chest tube. A retained hemothorax may progress to fibrothorax with lung entrapment or become infected resulting in an empyema. Once diagnosed, a video assisted thorascopic surgery (VATS) can be done to evacuate the retained blood.

Empyema - Empyema is diagnosed by positive pleural cultures or frank purulence in the pleural space. The most common cause of post traumatic empyema is a retained hemothorax, which is why evacuating retained blood is so important. Empyemas are treated depending upon their severity. Mild cases can be treated with antibiotics and drainage alone, while drainage failures or more severe sepsis may require a VATS and decortication. Decortication is the removal of the inflammatory exudate on the lung via either a VATS or a thoracotomy

Persistent air leak from bronchopleural fistula - This communication from the lung parenchyma to the pleural space results in an air leak to the chest tube persistent 5-7 days after injury. Pleurodesis, sealants, continued chest tube with Heimlich valves, and operative therapy are among the management options to treat a persistent air leak. Bronchoscopy should be performed if there is concern for a major airway injury.

ABDOMINAL INJURY

Abdominal injury related to trauma affects the solid organs (liver, spleen, kidneys, pancreas) or hollow viscus (stomach, small bowel, colon, rectum, bladder) and can be due to blunt (motor

vehicle crash, pedestrian injury, falls) or penetrating trauma (knife, gunshot, shotgun). The risk of specific organ injury from penetrating abdominal trauma depends upon the projectile's mass, velocity, and trajectory. Lastly, one must also consider possible injuries to the bony structures in the torso, including the pelvic bones.

1. Abdominal viscera injuries

Epidemiology/Pathophysiology

Blunt abdominal trauma accounts for 80% of abdominal injuries seen in the emergency department. The mechanism of blunt injury is typically due to crushing of the organs between the solid structures of the body, or shearing forces from sudden deceleration. The spleen and liver are the most commonly injured solid organs. Solid organ injuries can occur with associated injuries to the thorax, such as rib fractures, hollow viscus injuries, such as the small bowel and stomach and the pelvis. The incidence of hollow viscus blunt injury with blunt trauma is approximately 3%.

Signs/Symptoms

In both penetrating and blunt abdominal trauma, the absence of abdominal pain or tenderness does not rule out the presence of significant intra-abdominal injury. Abdominal trauma can manifest a variety of presentations, and requires a high index of suspicion. The physical findings most strongly associated with intra-abdominal injury are:

- Seatbelt sign (ecchymosis over the abdominal wall in the distribution of the seat belt)
- Rebound tenderness
- Hypotension
- Abdominal distention
- Abdominal guarding

Solid organ injury can be a difficult clinical diagnosis as not all injuries are symptomatic. Major injuries of any solid organ can lead to significant hemoperitoneum, disruption to the pancreatic or biliary ducts, and avulsion to the renal calyx, leading to hemorrhagic shock, biliary sepsis and hematuria. Intra-abdominal injury may cause referred pain. For example, splenic injury with hemoperitoneum near the left hemidiaphragm may cause referred pain to the left shoulder (Kehr's sign), or a liver injury with hemoperitoneum near the right hemidiaphragm may cause referred pain to the right shoulder. The digital rectal examination has a poor sensitivity for bowel injuries and should only be performed to look for blood in the rectal vault related to rectal injury or to assess for sphincter tone in suspected spinal cord injury.

Relevant Diagnostic Studies

FAST serves as a screening tool for the detection of hemoperitoneum in blunt abdominal trauma. However, FAST is not sensitive for detecting abdominal injury unless there is significant fluid volume of spilled enteric contents or blood (generally >200 mL). FAST has little

role in the initial evaluation of penetrating abdominal trauma, but may be helpful for decision-making in multi-cavitary penetrating trauma. Hemodynamically unstable patients with a positive FAST exam should go directly to the operating room for emergent laparotomy. In hemodynamically unstable patients with a negative FAST exam, one must consider additional work up (i.e., falsely negative FAST) or search for alternative sites of hemorrhage or other non-hemorrhagic causes of shock.

When there is concern for pelvic injury and hemodynamic instability, a pelvic XR may assist the trauma surgeon with injury identification and early resuscitation.

Imaging, in particular, CT scanning, has changed the diagnosis and, in many ways, the management of solid abdominal organ injury. Injuries of specific solid organs are graded based on the CT findings using the American Association for the Surgery of Trauma (AAST) injury scale. The AAST Liver injury scale is provided below.

AAST Liver injury scale

GRADE	Type of Injury	Description of Injury
I	Hematoma Laceration	Subcapsular, <10% surface area Capsular tear, <1cm
II	Hematoma Laceration	Subcapsular, 10%-50% surface area Capsular tear, 1-3cm
III	Hematoma Laceration	Subcapsular, >50% surface area >3cm
IV	Laceration	Parenchymal disruption involving 25%-75% of the hepatic lobe
V	Laceration Vascular	Parenchymal disruption involving >75% of the hepatic lobe Injuries to the retrohepatic vena cava or hepatic veins
IV	Vascular	Hepatic avulsion

Non-operative Management

Stable patients and those with low grade injuries are usually managed nonoperatively. Patients with higher grade (>II) solid organ injuries are usually admitted to the intensive care unit for serial abdominal and laboratory exams. Injuries that used to mandate operative exploration historically are often now managed by employing multidisciplinary adjuncts such as angioembolization and endoscopy. Higher grade, liver, splenic and kidney injuries in a hemodynamically stable patient, can be managed exclusively with angioembolization of major vessels and pseudoaneurysms with good success rates. Injuries to the pancreatic duct can be managed endoscopically with an endoscopic retrograde cholangiopancreatogram (ERCP) and stents.

Operative Management

Hemodynamically unstable patients with suspected traumatic intra-abdominal injury should be explored via a trauma laparotomy. Absolute indications for immediate operative intervention

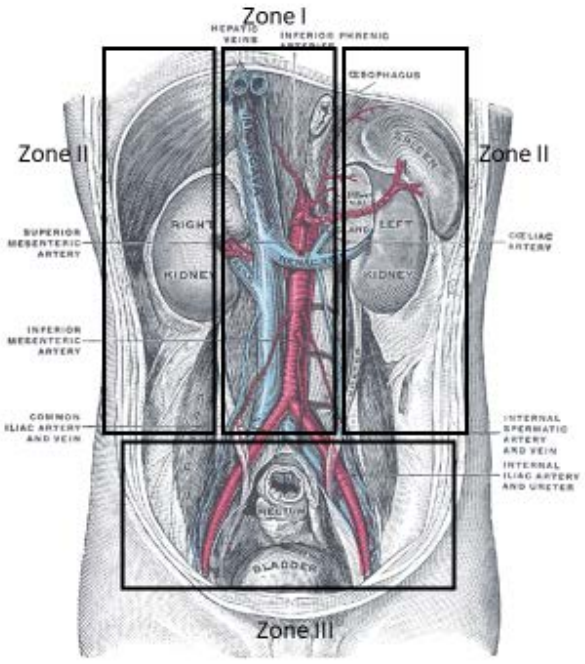
include hemodynamic instability, diffuse abdominal pain and/or peritonitis on clinical examination, findings on initial imaging studies consistent with gastrointestinal perforation (pneumoperitoneum, spilled intraluminal contrast, bowel infarction), or evisceration.

The goals of a trauma laparotomy are to control bleeding, control contamination, identify injuries and repair of injuries, if permissible. The patient is placed supine on the operating room table, and prepped from the chin to the knees, providing open access to the chest, abdomen and bilateral groins. A trauma laparotomy is started with a midline abdominal incision from the xiphoid to pubic symphysis, extending through the skin, subcutaneous tissue, fascia, and peritoneum. The transverse colon and omentum are retracted cephalad, and small bowel eviscerated. This gives the surgeon a view of Zone 1. The vasculature of the abdomen is divided into 3 zones:

Zone 1: Midline retroperitoneum from the aortic hiatus to the sacral promontory, containing aorta, inferior vena cava and all branches.

Zone 2: Left and right flanks, containing the kidneys, paracolic gutters and renal vessels

Zone 3: Pelvic retroperitoneum, containing the iliac vessels



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Methodical exploration of the peritoneal cavity allows the surgeon to identify injuries. With significant hemoperitoneum, packing of the four quadrants with lap pads will assist with temporary bleeding control. In penetrating trauma, following the presumed trajectory of the

object allows the surgeon to focus on a specific quadrant. In a rapidly bleeding patient, clamping the aorta above the celiac trunk may slow bleeding and provide more time to find the bleeding source. Zone 1 is examined first for hematoma. If hematoma is present, then the area should be formally dissected and explored. For stable hematomas in blunt injury in Zone 2 and 3, the injury can be observed, whereas expanding hematomas require exploration. For stable and expanding hematomas in penetrating injury in Zone 2 and 3, the area should be formally dissected and explored. Examination of the bowel from the ligament of Treitz to the rectum, including both sides of the bowel and mesentery, allows for identification of injuries. Spillage from perforations should be temporarily controlled using Babcock or Allis clamps. Definitive repair of the bowel should wait until the entire length of the bowel has been examined, as additional injuries may be discovered. Inspection of the stomach, 2nd and 3rd portions of the duodenum (after a Kocher maneuver, or dissection of the lateral peritoneal attachments of the duodenum), liver, spleen, pancreas (through the lesser sac), diaphragm and bladder can be done with direct visualization.

When gastrointestinal injury is identified, the injured bowel can be repaired primarily or resected with an immediate or delayed anastomosis. The choice depends upon the clinical status of the patient, the severity of the specific injury, site of injury, and number and relationship of hollow viscus injuries to each other. The kidneys are palpated to assess for hematoma or obvious injury. Right to left visceral medial rotation (also known as the Cattell-Braasch Maneuver) is used to expose injuries in the right retroperitoneum (right kidney and vessels, head of pancreas, duodenum, IVC, right iliac and pelvic vessels) and is completed by incising the parietal peritoneum at the white line of Toldt from the base of the cecum to the hepatic flexure. This commonly includes a Kocher maneuver to visualize the duodenum and head of the pancreas. Left to right visceral medial rotation (also known as a Mattox Maneuver) is indicated for left-sided Zone 1 and 2 injuries (aorta, left iliac and pelvic vessels) and is completed by incising the parietal peritoneum at the white line of Toldt from the sigmoid colon to the splenic flexure. The spleen, tail of the pancreas, left kidney, and stomach can also be reflected medially.

In a hemodynamically unstable patient where the definitive repair of injuries is not safe (i.e., hypotension at admission, vasopressor use during case, blood transfusions> 6 U, delay of operation >6 hours, severe peritoneal contamination), temporary closure of the abdomen at the end of a trauma laparotomy provides containment of the abdominal cavity, protection of bowel, and sparing the fascia for the definitive closure. This is commonly accomplished using a vacuum-assisted device including a sponge, polyethylene sheet, and vacuum machine. After temporary abdominal closure, the patient is further resuscitated to interrupt the lethal triad of trauma (hypothermia, coagulopathy, acidosis). Once stabilized, the patient returns to the OR for re-laparotomy, typically within 24-48 hours. Planned re-laparotomy begins with meticulous exploration of the abdominal cavity. Packing, if present, is removed and significant injuries that have been overlooked, or only partially defined in the first laparotomy, are now addressed with reconstruction. Major vascular reconstruction typically precedes GI tract repairs to avoid contamination. Formal abdominal closure, if possible, is then completed.

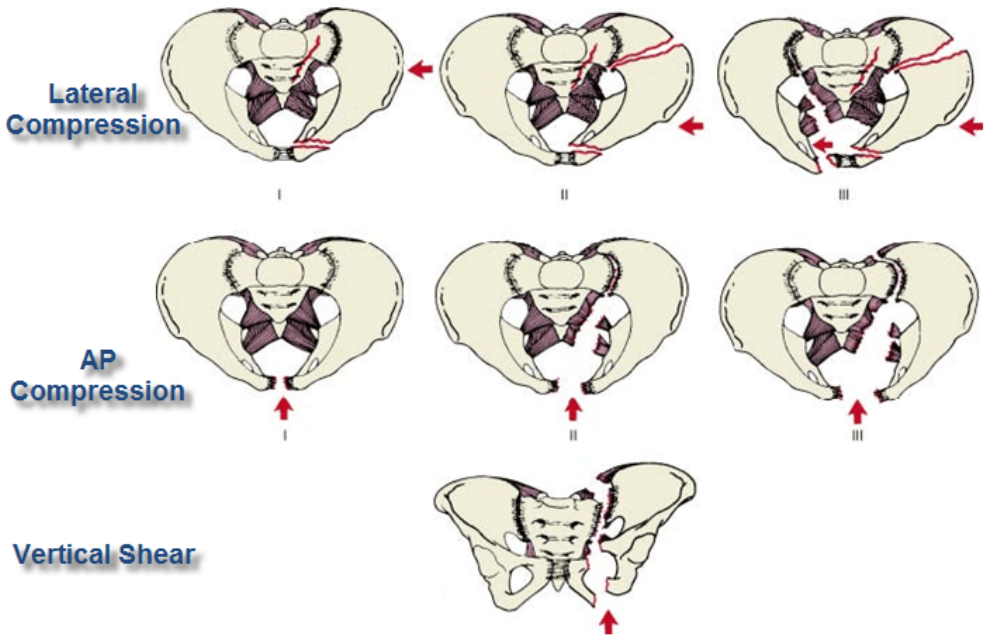
Basic Postoperative Care

Postoperative care involves intensive care monitoring, serial abdominal and laboratory exams for at least 24 hours until the patient is deemed to be hemodynamically stable.

2. Pelvic Fractures

Epidemiology/Pathophysiology

Pelvic fractures represent approximately 3% of skeletal injuries, with an overall mortality from 5-16%, with mortality related to unstable pelvic fractures at approximately 8%. Hemodynamic instability from pelvic fractures is typically due to life-threatening hemorrhage from the pre-sacral and lumbar venous plexus. Arterial bleeding can be related to injury to the iliac arteries, and are more common in penetrating trauma. The bladder is injured in approximately 3.4% and the urethra in 1% of pelvic trauma cases. Pelvic fractures are classified by the vector of force applied: anteroposterior compression/"open book", lateral compression, and vertical shear. The vector of force causes a predictable fracture pattern with known rotational and vertical stability. Fractures to the bony pelvis can be associated with vascular, colorectal, and urogenital injuries.



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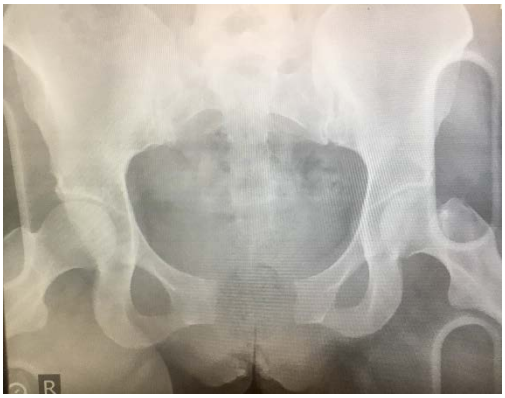
Signs/Symptoms

Most clinically significant pelvic fractures can be diagnosed on physical exam as patients will complain of pain. A good neurologic, urogenital, and anorectal exam is imperative in assessing for associated injuries that can be lethal, if missed. In pelvic injuries, the patient may present

with external bleeding, ecchymosis of the flank, perineal, and scrotal regions, blood at the uretheral meatus, vaginal bleeding, or altered positioning of the lower extremities and iliac crests. Compression of the pelvis to assess for stability should be done in a gentle manner, in order not to displace fracture fragments or exacerbate injuries. Once a pelvic injury is detected, repeated compression should be avoided. Signs and symptoms can range from inability to void, decreased sensation, tone, and reflexes, and blood in the rectal vault. Pelvic fractures can also be associated with vascular injuries which can lead to pelvic hematomas. Patient's hemodynamics are monitored as continuous or incontrollable bleeding can lead to hemorrhagic shock.

Relevant Diagnostic Studies

X-ray studies are still the mainstay of diagnosing significant pelvic fractures. CT scanning has improved the ability to diagnose occult pelvic and sacral injuries and provide 3D reconstruction which can aid in operative planning.



Example of an open book pelvic fracture with widened pubic symphysis

Non-operative Management

Pelvic fractures are managed in a multidisciplinary fashion, consisting of trauma and orthopedic surgeons, and interventional radiologists. Non-operative pelvic fractures with a pelvic hematoma and no hemodynamic compromise can be managed in the critical care setting with serial laboratory and physical exams. Down-trending hemoglobin values and ongoing transfusion requirements in the setting of continued hemodynamic stability warrant angiography with embolization of pelvic vessels.

Unstable pelvic fractures can cause ongoing hemorrhage because of: injury to small vessels (most commonly venous bleeding), movement of the fractured elements, and large pelvic volume. "Wrapping" the pelvis reduces pelvic volume (creating a tamponade effect), stabilizes fracture fragments (reducing hemorrhage from the fracture sites), and improves patient comfort. A pelvic binder should be placed early in the resuscitation of a hemodynamically unstable

patient with traumatic injury to the pelvis. When placing a pelvic binder:

- The patient is log rolled in order to place a sheet or commercial pelvic binder under the pelvis
- The legs should be together in internal rotation
- The sheet is wrapped circumferentially centered on the greater trochanters. Care should be taken NOT to center the wrap (sheet or commercial product) over the iliac crests.
- If possible, a pelvic XR should be obtained after splint application to assess the adequacy of reduction

Significant lateral compression fracture can actually be worsened by application of a pelvic binder, so an initial pelvic XR aids the surgeon with management decisions.

Operative Management

Pelvic fractures from blunt injury can cause hemodynamic instability in trauma patients. Two options exist for management of pelvic fracture related hemorrhage: angio-embolization or pre-peritoneal pelvic packing (PPP). Determining which management is appropriate is based on the patient's stability. If a patient's vital signs improve and stabilize with blood transfusion in the trauma bay, angio-embolization in Interventional Radiology provides a less invasive treatment. If the patient remains hemodynamically unstable, operative intervention is required. For PPP, the patient is prepped and draped similar to a trauma laparotomy. A vertical midline or low transverse incision is made from the pubic symphysis extending cephalad for 6-8 cm, and carried down thru the skin, subcutaneous tissue and fascia. Care is taken not to enter the peritoneum. Once in the pre-peritoneal space, hematoma is evacuated. The hematoma often has already dissected the pelvic space, facilitating packing. To place packs, the bladder is retracted to one side and laparotomy pads placed on either side. By not opening the peritoneum, the pelvis remains intact, providing tamponade to the forming pelvic hematomas. In patients who remain hemodynamically unstable following PPP, angio-embolization can be used to address arterial bleeding.

Basic Postoperative Care

Postoperative care of the patient with a pelvic fracture involves multiple specialties, including physical and rehabilitation medicine, to help the patient with mobility and adaptation of their lifestyle post injury.

3. Bladder injury

Epidemiology/Pathophysiology

Injuries to the bladder are extremely rare and most occur in the setting of blunt trauma and associated pelvic fractures. Bladder injuries are described as extraperitoneal and intraperitoneal. Extraperitoneal injuries are more common due to deceleration/shearing injury

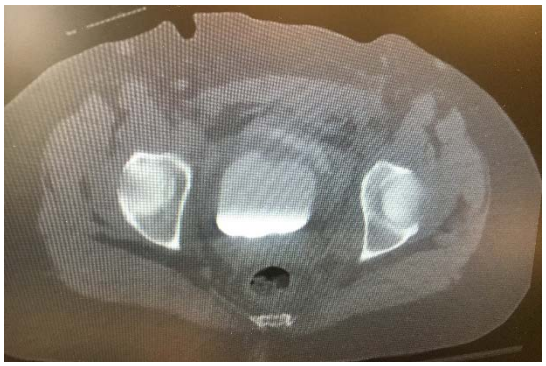
and direct bony injury from the pelvis. Intraperitoneal injuries occur in the setting of rapid deceleration with a full bladder causing a burst type of injury.

Signs/Symptoms

Most bladder injuries, if significant, can be diagnosed clinically. Gross hematuria is one of the hallmarks of bladder injury, but is not always present on physical exam. Patients may complain of the inability to void or suprapubic pain, but sometimes, the diagnosis can be missed due to associated and distracting injuries. Delayed presentations of bladder injuries involve abdominal distention and pain, and fever. Laboratory abnormalities, such as acidosis, hyperkalemia, hypernatremia, and hyperchloremia, can also occur.

Relevant Diagnostic Studies

Imaging, consists of cystography, which can be done with an x-ray or CT scan, utilizing instillation of 300-400ml of contrast into the bladder. Static cystography is usually employed when a retrograde urethrogram is performed when there is suspicion for a urethral or bladder neck injury. CT cystography is employed more frequently as it can be easily added on to a CT scan of the abdomen and pelvis and can distinguish between an extraperitoneal and intraperitoneal bladder injury.



Example of an extraperitoneal bladder injury on CT cystography

Non-operative Management

Most extraperitoneal bladder injuries without associated operative injuries are usually managed non-operatively with a Foley catheter for 7 days followed by a cystogram prior to removal of the catheter.

Operative Management

Intra-peritoneal bladder injuries require operative management. In addition, if bladder injuries are associated with another indication for an exploratory laparotomy (urogenital, anorectal, colon, vascular or pelvic injury), then they are repaired at the same time.

Basic Postoperative Care

In the postoperative period, most patients will have catheter drainage and monitoring for continued gross hematuria and lab abnormalities until normalization.

EXTREMITY INJURY

Epidemiology/Pathophysiology

Extremity injuries are common in patients sustaining blunt and penetrating trauma. Most common mechanisms involve high speed motor collisions and falls. Most simplified, fractures are described by location, type, displaced or nondisplaced, and open or closed. Comminuted fractures are where the bone is broken in multiple places, splintered, or shattered. Open fractures are when the bone breaks through the skin, usually causing soft tissue damage and contamination.

TYPE of FRACTURE	Examples
Complete	**Transverse** **Oblique** **Spiral** **Longitudinal** **Segmental** **Comminuted**
Incomplete	**Bowing** **Buckle** **Greenstick**
Compound	**Open**

Extremity dislocations of importance in trauma are the elbow, hip, and knee. Dislocations can be associated with fractures and neurovascular compromise.

Approximately 12% of extremity injuries occur as a result of a penetrating mechanism. When evaluating an extremity injury, one must assess the four functional components of the extremity - nerves, vessels, bones, and soft tissues. An open fracture (a fracture in which there is an open wound or break in the skin near the site of the broken bone) and vascular injury increases the risk for limb loss after injury. After assessing for Airway and Breathing, control of hemorrhage from an extremity injury takes priority.

Signs/Symptoms

Extremity fractures that are open and considerably displaced are usually found during the initial evaluation of the patient. Signs and symptoms depend on the location and distracting injuries. A neurologic and vascular exam are imperative in examining extremity injuries. Long bone fractures can cause significant bleeding leading to hemorrhagic shock, neurovascular damage, and compartment syndrome. Compartment syndrome is a clinical diagnosis and a surgical emergency as it can lead to ischemia and necrosis of the extremity. The signs/symptoms of compartment syndrome are similar to ischemic changes (see table below), however, pulselessness is a very late finding. In order to have a compartment syndrome, the compartment pressures must overcome the capillary perfusion pressure which is approximately 25mmHg. Above 25mmHg the microvasculature flow is affected and cellular ischemia develops. This process occurs with pressures well below the systolic pressures and, therefore, pulses are usually present.

Dislocations usually cause the patient significant pain and many are obvious on physical exam. A knee dislocation can cause damage to the popliteal artery. Unlike fractures, dislocations of the elbow, hip, and knee must be rapidly diagnosed and reduced to decrease further ischemia. Many times, extremity injuries, especially if minor, can be missed during the initial survey, which is why the tertiary exam is so vital in the complete assessment of the trauma patient. After the acute and life threatening injuries have been identified and managed, days later, patients might complain of pain in their hands or toes, or ecchymosis and swelling might appear that was not seen initially, prompting further investigation.

Limb Ischemic Signs/Symptoms

5 P's of Acute Ischemia
Pain
Paresthesia
Pallor
Pulselessness
Paralysis

Hemodynamically unstable patients who present with a penetrating extremity injury are most commonly in hypovolemic shock from blood loss. The patient may present with altered mental status, tachycardia, hypotension, tachypnea, diaphoresis and "hard signs" of arterial injury (see above). A detailed neurologic examination should be completed to look for signs and symptoms of nerve damage. In the lower extremity, function of the femoral, sciatic, tibial, and peroneal nerves should be assessed since these nerves are more likely to be injured:

- Injury to the femoral nerve results in decreased sensation on the anterior thigh and weakness of hip flexion and knee extension.
- Injury to the sciatic nerve causes decreased sensation in the lateral leg and the lateral, dorsal, and plantar aspects of the foot; weakness of knee flexion; and loss of motor

function of the leg and foot.

- Deep peroneal nerve injury causes decreased sensation in the first dorsal webspace and causes foot drop.
- Injury to the tibial nerve results in loss of sensation to the heel, inability to plantar flex the foot, and cavus deformity of the foot.

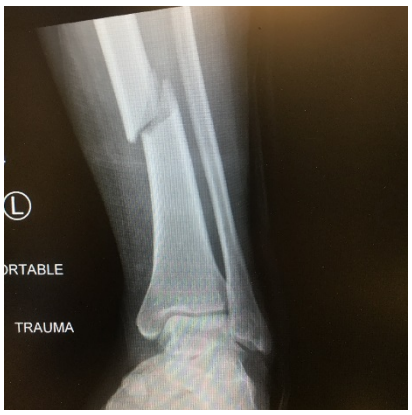
In the upper extremity, a game of "rock, paper, scissors" is a quick way to assess the motor function of the median, radial, and ulnar nerves, respectively.

- Injury to the axillary nerve (proximal humerus fractures) results in loss of arm abduction and an area of numbness or paresthesia along the lateral aspect of the upper arm.
- Radial nerve injury leads to loss of sensation on the dorsum of the hand and weakness of the wrist and finger extensors.
- Injury to the median nerve leads to decreased sensation on the palmar aspect of the first three digits and weakness of the thenar musculature.
- Injury to the ulnar nerve leads to decreased sensation on the palmar aspect of the fourth and fifth digits and weakness of the flexors of these digits.

The skin, subcutaneous fat, and muscle should be evaluated for signs of a potential underlying bony fracture, and to assess the extent of soft tissue damage. Assess the soft tissue to identify areas of missile entry and exit, soft tissue avulsion, skin or muscle flap formation, and evidence of contamination. In penetrating injuries, such as high-velocity gunshot wounds, the external wound may be relatively small; however, underlying soft tissue destruction can be significant. Signs of a potential bony fracture include: extremity deformity, point tenderness, ecchymosis, laceration deep to the muscle fascia or near a joint, and joint laxity.

Relevant Diagnostic Studies

As with most bony injuries, x-rays are by far the most utilized imaging technique. CT scans and MRIs are reserved for occult and ligamentous injury as well as operative planning for complex injuries.



Example of a comminuted tibia fracture

In a hemodynamically unstable patient with “hard signs” of vascular injury, diagnostic studies are not indicated as the presence of a hard sign of arterial injury is nearly 100% predictive of a vascular injury requiring surgical repair.

Non-operative Management

Many extremity fractures, if simple, nondisplaced, and closed can be treated with immobilization, using a splint or cast. If the fracture is displaced, it may first require closed reduction to realign the bone. Dislocations can usually be reduced with adequate sedation in the trauma or emergency room, but might require general anesthesia if unsuccessful or if there is vascular compromise. It is imperative to always check an x-ray to ensure proper alignment and to perform a neurovascular exam post reduction.

Control of hemorrhage is priority in extremity trauma associated with active bleeding. Direct pressure can be used. However, direct pressure does not always stop the bleeding, and is difficult when bleeding from junctional vessels (axillary artery, common femoral artery). Bleeding can also be controlled by direct clamping of visible vessels. Clamping vessels which cannot be clearly identified is not recommended, as it can lead to further injury. If the wound is large enough, one can pack the wound with gauze, then apply direct pressure to further tamponade the bleed. For life threatening bleeding from the upper or lower extremity, a tourniquet should be used to stop the bleeding. Ideally, a commercial grade tourniquet is used, as improvised tourniquets (made from a rope or piece of clothing) can be nonhemostatic or venous occlusion (tight enough to occlude the vein but not the artery - provides temporary hemorrhage control but are potentially deadly from paradoxical bleeding).

In order to properly apply the tourniquet:

1. Wrap the tourniquet around the bleeding arm or leg about 2 to 3 inches above the bleeding site. The tourniquet should not be placed over the joint, but rather go above the joint if necessary.
2. Pull the free end of the tourniquet to make it as tight as possible and secure the free end.
3. Twist or wind the windlass until bleeding stops.
4. Secure the windlass to keep the tourniquet tight. A tourniquet will likely cause pain but is necessary to stop life-threatening bleeding.
5. Note the time the tourniquet was applied (can write it on the tourniquet itself).
6. A second tourniquet can be placed if bleeding persists proximal to the tourniquet, or if bleeding control is inadequate distal to the first tourniquet.
7. With hemorrhage controlled, the patient should undergo operative repair emergently. A tourniquet can remain in place approximately 2 hours before neurovascular and tissue damage occur.

Operative Management

Extremity fractures which are complex and open usually require some sort of operative intervention. Operative interventions include internal or external fixation, the latter reserved for patients that require urgent stabilization but are either too injured to undergo surgery or the soft tissues are too swollen. Open fractures need irrigation, debridement, tetanus, and antibiotics to prevent infection and osteomyelitis. Dislocations that are not successfully reduced, or if associated with significant fractures or neurovascular compromise, usually require operative intervention.

Emergent operative intervention is required in hemodynamically unstable patients with extremity trauma. For known or suspected vascular injuries of the upper extremity, the injured arm should be extended on an arm board, and entire extremity prepped and draped. A new sterile tourniquet (typically a blood pressure cuff) can be used to control bleeding during dissection. During initial dissection, bleeding is usually controlled with direct pressure. The incision made is related to the suspected area of injury, and is usually along an imagined line from the sternal notch, running laterally on the clavicle (exposes the axillary artery), tracing the deltopectoral groove (exposes the brachial artery), crossing the elbow obliquely, and running on the radial side of the volar forearm (exposes the radial artery) to the wrist crease. In order to expose the ulnar artery, an incision is made along the ulnar side of the volar forearm.

For known or suspected vascular injuries of the lower extremity, the patient should be prepped and draped from the nipples to the knees to insure adequate exposure. Furthermore, a wide prep allows access to bilateral groins for saphenous vein graft harvesting and on-table angiograms post repair. For exposure of the iliac vessels, a “hockey stick” incision extending from the groin above the inguinal ligament and laterally above the iliac crest is used. For exposure of the common femoral vessels, profunda and proximal superficial femoral artery, a longitudinal incision along the medial border of the sartorius muscle above and below the inguinal ligament is used. Deep dissection of the artery should be lateral to the saphenous vein and inguinal lymph nodes. Opening the deep fascia and arterial sheath allows for proximal and distal control of the vessels. Hip abduction and slight lateral rotation with stacked towels placed under the knee can be used when attempting to expose the distal superficial femoral artery. The incision parallels the lateral border of the sartorius muscle. The sartorius muscle is retracted medially to expose the adductor canal which contains the superficial femoral vessels. The popliteal artery is located between the adductor hiatus (popliteal or Hunter’s canal) and the lower border of the popliteus muscle behind the knee joint.

A medial approach is often preferred for traumatic injury with an incision made between the vastus medialis and sartorius muscles. Division of the semimebranosis, semitendonosis, and adductor magnus can aid in further exposure of the popliteal artery without little postoperative disability, if the muscles are reapproximated. Retraction of the medial head of the gastrocnemius posteriorly allows exposure of the popliteal fossa. Once the vascular sheath is entered, the popliteal vein is first encountered, and can be retracted posteriorly to see the popliteal artery. The tibial nerve is located posteriomedial and should be protected.

A temporary vascular shunt (appropriately sized plastic tubing) can be used as the injury is

being assessed. This vascular damage control tactic provides oxygenated blood distal to a vascular injury, while other injuries are addressed or the patient is transferred to a higher level of care. Once the injured vessel is identified, proximal and distal control of blood flow is attained using vascular clamps or vessel loops. The vascular injury can be repaired primarily with interrupted sutures or reconstructed using an interposition graft.

Basic Postoperative Care

In the immediate postoperative period, patients are monitored for pain control and compartment syndrome. Long term management involves physical and occupational therapy as well as physical and medical rehabilitation.

Patients with severe lower extremity injuries have a high incidence of complication, including wound complications (infection, necrosis, nonunion, osteomyelitis), venous thromboembolism, rhabdomyolysis, and late complications including amputation and heterotopic ossification in residual limbs.

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