

Dr. Ali's Uworld Notes For Step 2 CK

Surgery

Trauma

Hemorrhagic Shock - Hemorrhagic shock may be divided into four classes based on the amount of blood loss.

Parameter	I	II	III	IV
Blood loss	<15% (750 ml)	15-30% (750-1500 ml)	30-40% (1500-2000ml)	> 40%
Heart rate	> 72	100-120	> 120	> 140
Blood pressure	Normal	Slightly decreased	Markedly decreased	Markedly decreased
Capillary refill	Normal	May be delayed	Delayed	Markedly delayed
Urine output (cc/hr)	> 30	20-30	5-15	< 5
CNS symptoms	Normal	Anxious	Confused	Lethargic

Patients with class I hemorrhage have lost less than 15% of their intravascular volume (or less than 750 cc) and are generally alert. The blood pressure is normal and the major organs are satisfactorily perfused as evidenced by a normal urine output. The patient compensates for blood loss through a sympathetic response that induces mild tachycardia and peripheral vascular constriction. Capillary refill is maintained (< 2 seconds).

Patients with a class II hemorrhage have lost between 15 and 30% of their blood volume (or 750-1500 cc) and are generally more anxious and agitated.

Pulse rate will be more than 1 DO/min. While the mean arterial blood pressure remains normal, the pulse pressure is narrowed and the blood pressure starts to trend downward. Urine output is slightly decreased and the skin is cool and moist. Capillary refill may be delayed. All of these manifestations can be attributed to further increases in sympathetic discharge and shunting of blood from less critical vascular beds such as the skin, leading to skin vasoconstriction.

Patients with class III hemorrhage have lost 30-40% of their blood volume (or 1500-2000 cc) and can no longer maintain their blood pressure at normal levels despite further increases in heart rate and peripheral vascular constriction. These patients will begin to have a decreased level of consciousness and a further decrease in urine output due to poor cerebral and renal perfusion, respectively.

Class IV hemorrhage is defined as a blood loss of more than 40% of the blood volume (or > 2000 cc). The patient appears lethargic, tachypneic and has markedly decreased urine output. At this point, circulatory failure and death are imminent without therapeutic intervention.

When hemorrhage occurs, **tachycardia** and peripheral vascular constriction are the first physiological changes. These responses act to maintain the blood pressure within normal limits until severe blood loss has occurred.

Tetanus Immunization -

Time of Latest Td Booster	Minor and Clean Wound	More Severe or Dirty Wound
Unimmunized	Td only	Td and TIG
≥10 years	Td only	Td and TIG
<10 years	None	Td if latest booster given ≥5 years ago

All patients with traumatic wounds should be assessed for the need of tetanus prophylaxis. Tetanus immune globulin (TIG) provides passive, temporary, and immediate immunity. Tetanus-diphtheria toxoid (Td) provides active, prolonged, and delayed immunity. Wounds at high risk for *Clostridium fefani* growth are those that provide an anaerobic environment for growth, such as puncture wounds, projectile wounds, wounds containing foreign bodies, sites of active infection by other organisms, and wounds containing necrotic tissue.

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A **Td booster** should be administered to individuals with more **severe or dirty wounds** (e.g .. puncture wounds and wounds contaminated with dirt, feces, or saliva) who received their latest dose **more than 5 years** ago and those with clean minor wounds who received their latest dose **more than 10 years** ago.

A **TIG** injection should be administered to individuals who have received **less than three doses** of tetanus vaccine and those with a more severe or dirty wounds who have an unknown immunization status.

Glasgow Coma Scale - All trauma patients should be first assessed using the GCS, which estimates the severity of the patient's neurologic injury for triage. The GCS can also give some prognostic information when used in conjunction with the patient's age and presence of concomitant adverse clinical findings, such as hypoxia, cardiovascular compromise, increased intracranial pressure, and radiographic evidence of a midline shift of the brain. Calculation of GCS score is shown below.

Eye Opening	
Spontaneous	4
To verbal command	3
To pain	2
None	1
Verbal Response	
Oriented	5
Disoriented / Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Motor Response	
Obeys	6
Localizes	5
Withdraws	4
Flexion posturing (Decorticate)	3
Extension posturing (Decerebrate)	2
None	1

The GCS is used to predict the prognosis of coma and other medical conditions, such as bacterial meningitis, traumatic brain injury, and subarachnoid hemorrhage. However, the GCS is not used to diagnose coma in a patient. Findings used to diagnose coma include impaired brainstem activity (e.g., disruption of the papillary light, extraocular, and corneal reflexes), motor dysfunction (e.g., decorticate or decerebrate posturing), and impaired level of consciousness,

Blunt Head Trauma -

These patients presents after blunt head trauma (look for scalp lacerations, skull depression). In such patients, look for signs/symptoms worrisome for transtentorial (uncal) herniation secondary to an epidural hematoma. Epidural hematomas result from rupture of the middle meningeal artery, and this higher arterial pressure can rapidly expand the hematoma and compress the temporal lobe. ***The fluid resuscitation in such patient likely increased the rate at which the epidural hematoma expanded.*** The presence of hypertension, bradycardia, and respiratory depression (Cushing's reflex) indicates elevated intracranial pressure.

The uncus is the innermost part of the temporal lobe and herniates through the tentorium to cause pressure on ***the ipsilateral oculomotor nerve,***

ipsilateral posterior cerebral artery, and contralateral cerebral peduncle against the edge of the tentorium. Patients typically present with the focal neurologic signs shown in the table above.

Transtentorial (Uncal) Herniation	
Lesion	Neurologic Signs
Compression of the contralateral crus cerebri against the tentorial edge	Ipsilateral hemiparesis
Compression of the ipsilateral oculomotor nerve (i.e., CN III) by the herniated uncus	Loss of parasympathetic innervation causes mydriasis (occurs early); loss of motor innervation causes ptosis and a down-and-out gaze of the ipsilateral pupil due to unopposed trochlear (i.e., CN IV) and abducent (i.e., CN VI) action (occurs late)
Compression of the ipsilateral posterior cerebral artery (i.e., ischemia of visual cortex)	Contralateral homonymous hemianopsia
Compression of the reticular formation	Altered level of consciousness; coma

Abducens nerve (i.e., CN VI) injury from uncal herniation usually occurs **later in the clinical presentation**, with a symptom of inability to abduct the eye.

Most head injuries are mild and will not progress to more serious clinical scenarios. Minor head trauma is defined as a head injury that is associated with a Glasgow coma scale (GCS) score of 15, normal mental status on examination, no abnormal neurologic or funduscopic examination findings and no physical evidence of skull fracture. These patients can be discharged with no further imaging or studies if a reliable individual can monitor them for 24 hours following the injury.

Mild traumatic brain injury (TBI) is defined as a head injury that is associated with a GCS score of 13-15 and brief loss of consciousness, vomiting, headache or disorientation. Patients with moderate TBI have a GCS score of 9-12, and those with severe TBI have a GCS score of less than 8. Patients with severe TBI, evidence of intracranial injury, focal neurologic signs, seizure, prolonged loss of consciousness, and evident skull fracture on examination should have a CT scan of the brain and be observed in the hospital with frequent neurologic examinations.

Patients with mild-to-moderate TBI who have vomiting, headache, or brief loss of consciousness should also have a CT scan of the head. If the CT scan is normal, these patients can be discharged with a reliable caretaker and printed instructions (with a list of symptoms) that describe when they should return to the hospital. Patients with mild-to-moderate TBI who do not have any of the above symptoms do not need neuroimaging and can be observed for 4-6 hours in the emergency department, with neuroimaging reserved for those who worsen.

Diffuse axonal injury is the most significant cause of morbidity in patients with traumatic brain injuries. It is frequently due to traumatic deceleration injury and results in vegetative state. Sudden acceleration-deceleration impact produces rotational forces that affect the brain areas where the density difference is the maximum, thus most of the diffuse axonal **injury occur at gray white matter junction.**

Clinical features of patients with diffuse axonal injury are out of proportion with the CT scan findings. Patient loses consciousness instantaneously and later develops **persistent vegetative state**. CT scan characteristically **shows numerous minute punctate hemorrhages with blurring of grey white interface**. However, MRI is more sensitive than CT scan for diagnosing diffuse axonal injury.

Intracranial pressure is a function of volume and compliance and determined by pressure in the brain parenchyma, CSF and blood. Brain parenchyma is fairly constant unless there is a mass lesion, and CSF is also fairly constant unless there is a ventricular obstruction. Cerebral blood flow (CBF) determines the volume of blood and increases with hypercapnia, increased metabolic demand and hypoxia through cerebral vasodilation. The brain autoregulates CBF and cerebral perfusion pressure to maintain a relatively constant flow, but this becomes dysfunctional in certain pathologic conditions (e.g .. trauma and stroke). Increased CBF from more cerebral vasodilation and elevated blood pressure raises the ICP. The interventions for lowering ICP are summarized below.

Intervention for Lowering ICP	Mechanism
Head elevation	Increased venous outflow from the head
Sedation	Decreased metabolic demand and control of HTN
Intravenous mannitol	Extraction of free water out of the brain tissue → osmotic diuresis
Hyperventilation	CO ₂ washout, leading to cerebral vasoconstriction

Short-term hyperventilation lowers the ICP by **lowering the vascular CO₂ concentration in the brain** (i.e .. CO₂ washout), leading to cerebral vasoconstriction. However, this should be closely monitored because extreme hyperventilation can decrease CBF too much and lead to marked iatrogenic brain ischemia.

Traumatic Amputation - All patients suffering traumatic amputations should be treated as candidates for reimplantation while in the field. As such, their amputated limb or digit should be **wrapped in sterile gauze, moistened with sterile saline** and placed in **a plastic bag**. The bag should be then **placed on ice and transported** with the patient to the nearest emergency department. The amputated part should not be allowed to freeze. Packaging of the amputated part in this manner prolongs the viability of the part for up to 24 hours. Younger patients suffering sharp amputations with no crush injury or avulsion are the best candidates for amputation reimplantation.

Cervical Spine Injury - The first step in evaluating this patient in the field is to **stabilize** the cervical spine and spinal column with a backboard, a rigid cervical collar, and lateral head supports until a spinal injury is excluded. The next step is to assess the airway because unstable lesions above the C3 level can cause immediate paralysis and lower lesions can damage the phrenic nerve. Cervical spine injuries can be associated with oral maxillofacial trauma, hemorrhage in the retropharyngeal space, and significant airway and neck edema.

Traumatic Lower Spinal Cord injury - It is most commonly caused by motor vehicle accidents. The trauma causes a primary injury to the spinal cord through mechanical compression, contusion, or shear injury. A secondary injury follows (within minutes to hours) and causes spinal cord

edema that eventually leads to central hemorrhagic necrosis. All trauma patients with suspected spinal injuries should first be hemodynamically stabilized and have a secure airway (possibly requiring intubation). The neck should be immobilized until spinal injury has been ruled out.

Orotracheal intubation with rapid sequence intubation (RSI) is the preferred route to manage unstable and apneic patients to protect the airway and provide oxygenation. Four people are involved in RSI; one manually stabilizes the patient, one induces the patient with anesthesia, one applies cricoid pressure to prevent passive regurgitation until endotracheal tube placement is confirmed and one places the endotracheal tube. Manual stabilization requires firmly holding either side of the patient's head with the neck midline and on a firm surface, without applying traction. This prevents any flexion or rotation of the neck during intubation.

Patients should then have a urinary catheter placed to assess for urinary retention and prevent possible bladder injury from acute distention. Imaging, such as CT scans and x-rays, might then be required to diagnose the injury and evaluate the extent of spinal cord damage. The use of high-dose intravenous steroids (e.g., methylprednisolone) is somewhat controversial, with conflicting evidence on benefit in spinal cord injury patients. Surgical decompression is indicated in patients who need acute spine stabilization.

Needle cricothyroidotomy is an excellent field procedure to establish an airway in **children**. It is not suitable in adults due to the risk of carbon dioxide retention, especially in patients with head injury in whom hyperventilation might be required to prevent or treat intracranial hypertension.

Tracheostomy is **no longer** a first option to establish an airway because of its complications. Surgical cricothyroidotomy is preferred over surgical tracheostomy but should be converted to formal tracheostomy in 5-7 days if prolonged airway control is needed. Prolonged use of cricothyroidotomy has a high incidence of tracheal stenosis.

CO Poisoning - CO (produced by incomplete combustion of carbon-containing compounds) is a tasteless, colorless and odorless gas whose poisoning should be considered in all patients who are **exposed to smoke** in a **closed space**. Carbon monoxide has over 200 times higher affinity for

hemoglobin than oxygen and impairs the delivery of oxygen to tissue by shifting the hemoglobin-oxygen dissociation curve to the **left**.

Symptoms and signs of mild to moderate CO toxicity include headache, nausea, dyspnea, malaise, altered mentation, and dizziness. Severe CO poisoning can present with **seizure, coma, syncope**, heart failure, or **arrhythmias**. Bright cherry lips can be a sign of CO poisoning on exam but is not specific. The diagnosis is confirmed clinically and by documenting an elevated carboxyhemoglobin level (> 3% in nonsmokers and > 15% in smokers). A pulse oximetry is unreliable and may appear normal since it cannot differentiate carboxyhemoglobin from oxyhemoglobin (as in this patient).

The treatment of carbon monoxide poisoning is administration of **100% oxygen** via nonrebreather facemask to competitively remove the binding of CO to hemoglobin and decrease the half life of CO from nearly 5 hours on room air to 1-2 hours. The patient should then be monitored for at least four hours and hospitalized if not improved. Hyperbaric oxygen can be used in severe cases not responsive to facemask-administered oxygen.

Sharp Abdominal Trauma - All hemodynamically **unstable** patients with sharp penetrating abdominal trauma and gunshot wounds that are believed to have entered the peritoneum must be treated with **emergent exploratory laparotomy** in order to prevent the development of sepsis resulting from hollow organ perforation and to prevent exsanguinating hemorrhage.

Blunt Abdominal Trauma - The most reliable symptoms of BAT in stable patients are abdominal pain, tenderness, and peritoneal signs. Intraabdominal injury should be suspected in patients with **abdominal wall ecchymosis, abdominal distention** and hypoactive bowel sounds.

The first step is fluid resuscitation. **If the patient is not responding to IV fluids (sustained low BP after IV fluids) continuous bleeding should be suspected**. The next step is to determine if a patient needs exploratory laparotomy. All patients with BAT should first be assessed for intraperitoneal free fluid or hemorrhage. A convenient and effective test is bedside ultrasonography to detect free intraperitoneal fluid in the hepatorenal space, splenorenal recess, and inferior portion of the intraperitoneal cavity. When combined with evaluation of the pericardium, this is known as the focused assessment with sonography for trauma (FAST) examination.

The FAST examination can be done in a few minutes and has a high sensitivity and specificity for detecting **hemoperitoneum, pericardial effusion, and intraperitoneal fluid**. If the FAST examination is limited or equivocal, a diagnostic peritoneal lavage (DPL) can be done to evaluate for hemoperitoneum. Patients with a positive finding on either DPL or FAST should undergo exploratory laparotomy. Hemodynamically stable patients with negative findings on FAST may undergo further testing with abdominal CT scan to determine need for laparotomy. This patient is hemodynamically unstable, should be quickly evaluated with FAST for intraperitoneal hemorrhage, and will likely need emergent exploratory laparotomy.

BAT - 1st give Fluid Resuscitation

See if the patient is hemodynamically stable or unstable & needs exploratory laparotomy.

UNSTABLE? - Do FAST. If FAST negative, do Diagnostic Peritoneal Lavage.

STABLE? - Do FAST. If Negative, Do CT.

In Penetrating Abdominal Trauma, If FAST is negative, directly go for Exploratory Laparotomy if the patient is unstable. Is stable, do DPL.

Hypotension not responsive to fluid administration following trauma is suggestive of ongoing occult blood loss. Patients in this scenario must be ***treated emergently with surgical intervention*** to stop further hemorrhage.

We don't do CT in unstable patients cuz it takes a lot of time and patient can die.

Spleen Injury - The spleen is the most commonly injured organ following blunt abdominal trauma. Initial CT can diagnose the injury. Delayed splenic rupture would cause ***acute left upper quadrant abdominal pain*** and ***possibly hypotension***.

Most common injuries are to the spleen and liver, and less common injuries are to the hollow viscus organs in the abdomen. Symptoms and signs suggesting splenic injury include ***left upper quadrant abdominal pain, abdominal wall contusion, left lower chest wall tenderness, hypotension, and left shoulder pain*** referred from splenic hemorrhage irritating the phrenic nerve and diaphragm (i.e .. Kehr sign). The initial examination after BAT can be ***unremarkable***, and the symptoms can occur ***hours later***, indicating ongoing splenic injury.

Hemodynamically stable patients should be suspected for intraabdominal injury if they develop hypotension, abdominal pain, concurrent femur or pelvic fracture, decreased hematocrit level, or hematuria. **If an initial ultrasound does not show free intraperitoneal fluid or pericardial fluid, the next step is an abdominal CT scan with intravenous contrast** (no oral contrast is needed because it adds little utility). This will define organ injury, assess for presence of bleeding in all abdominal compartments, and determine the need for surgery.

A gastric perforation would be more likely to occur in penetrating (rather than blunt) trauma. Furthermore, this condition would be evident during the initial presentation as an acute abdomen, with **free air under the diaphragm** on upright abdominal x-ray studies (or CT scan).

Duodenal hematomas most commonly occur following direct blunt abdominal trauma and are more commonly seen in children. Following trauma, blood collects between the submucosal and muscular layers of the duodenum causing obstruction. Patients classically present with epigastric pain and vomiting due to the failure to pass gastric secretions past the obstructing hematoma. Most hematomas will resolve spontaneously in 1-2 weeks, and the intervention of choice is **nasogastric suction and parenteral nutrition**. Surgery may be considered to evacuate the hematoma if this more conservative method fails.

Pancreatic Laceration - Any form of blunt abdominal trauma can compress the neck and/or body of the pancreas against the vertebral column over which it lies. A pancreatic **contusion, crush injury, laceration** or **transection** may result. Abdominal CT scans done early following the traumatic insult may **fail to detect** a pancreatic injury. Serial CT scans are often required to detect the evolution of the injury (i.e. enlargement of the gland, parenchymal disruption, areas of diminished contrast perfusion and peripancreatic fluid collections). Serum amylase values are nonspecific, and do not assist in the diagnosis of pancreatic trauma.

If blunt traumatic pancreatic injury is undetected initially, **devitalized tissue** or **a pseudocyst** resulting from such injury can become **secondarily infected**. A pancreatic abscess is a serious complication with a high mortality. Treatment is by immediate placement of a percutaneous drainage catheter, culture of the drained fluid, and ultimately surgical debridement.

Post Traumatic Pancreatic Abscess - **Hx of blunt abdominal trauma + Fever, chills and deep abdominal pain suggests a retroperitoneal pancreatic abscess.**

Post Traumatic Pancreatic Pseudocyst - Hx of blunt abdominal trauma + presents 4-6 weeks later with a palpable abdominal cyst like mass.

Hypovolemic shock & Positive Pressure Ventilation - Hypovolemia due to hemorrhage can occur in severe trauma cases. Severe hemorrhage causes **decreased venous return** and therefore decreased end diastolic volume and cardiac output. It also causes increased sympathetic activity to constrict the venous capacitance vessels, compensating for the hypovolemia by improving venous return.

Positive pressure mechanical ventilation acutely increases intrathoracic pressure, increasing right atrial pressure and **decreasing systemic venous return**. This sudden loss of venous return may cause acute circulatory failure and death. Additionally, the sedative medications used prior to intubation relax venous capacitance vessels and may themselves cause circulatory failure by acutely decreasing venous return in the hypovolemic patient.

BAT & Diaphragmatic Rupture - These patients presents after blunt abdominal trauma with mild respiratory distress and abnormal chest x-ray. Blunt abdominal trauma can cause a sudden increase in intraabdominal pressure that overcomes the muscular strength of the diaphragm and leads to large radial tears in the muscle. The resultant diaphragmatic rupture allows leakage of intraabdominal contents into the chest, causing compression of the lungs and mediastinal deviation. *It may present months after the trauma.*

Diaphragmatic rupture is more common on the **left side** because the right side tends to be protected by the liver. Patients can acutely present with respiratory distress, but some patients with smaller ruptures can have a delayed presentation with nausea and vomiting. Elevation of the hemidiaphragm on the chest x-ray might be the only abnormal finding, but ultrasonography or CT scan of the chest is sometimes required if the chest x-ray does not visualize the area well. The small bowel is sometimes present in the thoracic cavity.

Early recognition of diaphragmatic trauma is extremely important because the mortality rate of undiagnosed injury and subsequent strangulation of the bowel can be 30%-70% depending on the extent of the associated injuries. Most patients require surgical repair and exploration of the abdomen for other traumatic injuries. Best initial test is **a nasogastric tube in the pulmonary cavity, indicating a diaphragmatic hernia. A barium**

swallow or CT scan with oral contrast will be diagnostic. All diaphragmatic ruptures require operative repair.

BAT + Decreased Breath Sounds in Left Lower Lobe + Mediastinal Shift to opposite side = Diaphragmatic Rupture with herniation.

Chest Trauma -

Penetrating Thoracic Gun Shot Wound - The diaphragm can rise as high as the fourth thoracic dermatome on the right and fifth thoracic dermatome on the left (i.e., level of the nipples) on expiration and go down to the twelfth thoracic dermatome on both sides on inspiration. Any penetrating injury in the thorax below the level of the nipples has great potential to involve the abdomen through the diaphragm and is assumed to involve both compartments until proven otherwise.

A GSW involving the abdomen can cause significant injury, most commonly to the small bowel, colon, and liver. Patients who are hemodynamically unstable, have evidence of peritonitis, or have evisceration of any organ need **immediate exploratory laparotomy**. A focused assessment with sonography for trauma (FAST) can be done in a few minutes on all patients and has high sensitivity and specificity for detecting hemoperitoneum, pericardial effusion, and intraperitoneal fluid. If the FAST was equivocal in a hemodynamically unstable patient, he requires an immediate laparotomy.

1st - Fluids

Unstable - FAST. If Negative Immediate Laparotomy.

Stable - FAST. If Negative, DPL. If Negative, CT.

DO NOT MAKE ANOTHER HOLE BY DOING DPL WHEN A PATIENT ALREADY HAS A GSW.

Traumatic Fat Necrosis of the breast - This uncommon condition is associated with breast **trauma** or **surgery**, although frequently the patient **cannot recall** a history of trauma. Fat necrosis can mimic breast cancer in its clinical and radiographic presentation because it commonly presents as a fixed mass with skin or **nipple retraction**, has evidence of **calcification on mammography**, and appears solid on ultrasonography. The calcifications seen in breast malignancy, however, tend to be microcalcifications, while the calcifications seen in benign conditions such as fat necrosis tend to be **coarse**. Excisional, core, or fine-needle biopsy is diagnostic and typically shows **fat globules** and **foamy histiocytes**. Fat necrosis is not a

premalignant lesion and will resolve with time. **Standard follow-up** is sufficient because there is no increase in the risk of breast cancer. **No intervention is needed.**

Pulmonary contusion represents parenchymal bruising of the lung, which may or may not be associated with rib fractures. The clinical manifestations develop usually in the **first 24 hours** (often **within few minutes**); tachypnea, tachycardia, and hypoxia are characteristic. Physical examination typically reveals chest wall bruising and **decreased breath sounds** on the side of pulmonary contusion. **Chest x-ray reveals patchy irregular alveolar infiltrate** and a CT scan may be employed to make an early diagnosis. ABG typically shows hypoxemia and, itself, is an indication to suspect pulmonary contusion in trauma patients.

Pulmonary contusion is often not clinically evident immediately following an injury and initial radiographic studies may be **negative**. Patients typically develop hypoxia and respiratory distress hours later as pulmonary edema sets in. **Administration of large volumes of IV fluid may hasten this process.**

This is very important to differentiate pulmonary contusion from adult respiratory distress syndrome (**ARDS**). ARDS usually manifests **24-48 hours** from the trauma; besides that, **bilateral lung involvement** is present.

Cardiac Contusion - In the setting of trauma, hypovolemic shock from blood loss is most common. A low PCWP would be expected in hypovolemic shock. However, **elevated CVP/PCWP** or failure of hypotension to resolve after a bolus of intravenous fluids should suggest an alternative diagnosis. Myocardial contusion should be suspected in patients with evidence of injury to the anterior chest and can be confirmed with positive cardiac markers and EKG changes.

Rib fractures should be suspected in all patients with localized chest wall tenderness following trauma; up to half of rib fractures will not be evident on initial chest x-ray. Rib fracture is associated with significant pain, which causes hypoventilation that may ultimately result in atelectasis and pneumonia. Thus, **Pain management and respiratory support are the priorities** in the management of rib fractures. Oral agents, Such as opiates and/or NSAIDS are most commonly utilized, but an intercostal nerve block with a long-acting local anesthetic can be used if oral or systemic analgesics are not sufficiently effective. Intercostal nerve blocks provide pain relief

without affecting respiratory function, as opiate analgesics may, but it does carry some risk of pneumothorax.

Flail Chest - Flail chest is typically caused by **three or more adjacent rib fractures** that break in **two places** and create an unstable chest wall segment that moves in a paradoxical motion with respiration (Look for this on CXR!). The segment tends to retract during inspiration (instead of bulging out as normal) and bulge out during expiration (instead of retracting as normal).

Tachypnea and paradoxical thoracic wall movements that correct with positive pressure mechanical ventilation indicate a flail chest. Because of the associated pain, patients with flail chest take shallow breaths and compensate for the resulting hypoxemia with hyperventilation.

On examination, the isolated thoracic wall segment exhibits **paradoxical inward motion** on inspiration and outward movement on expiration. Pain control and supplemental oxygen are the most important early steps in managing this condition, but **intubation with mechanical positive pressure ventilation is required in many patients with this injury.**

Positive pressure mechanical ventilation replaces the normal negative intrapleural pressure during spontaneous ventilation with positive intrapleural pressure.

Because of the positive intrathoracic pressure induced by the ventilator, the previously paradoxically moving flail segment of the thoracic cage now moves out normally with the rest of the rib cage during inspiration.

Tension Pneumothorax - A clinical diagnosis of tension pneumothorax should be made in trauma patients who have **decreased breath sounds** and **tracheal shift** in the presence of **hypotension** and **jugular venous distention**. Needle **thoracostomy should be done emergently**; performing a chest radiograph for confirmation may lead to an unacceptable delay in treatment & should **NOT** be done!

Iatrogenic Pneumothorax - Placement of subclavian central venous catheters accounts for approximately one-fourth of iatrogenic tension pneumothorax. Tension pneumothorax is a life-threatening condition caused by air within the pleural space that displaces mediastinal structures and compromises cardiopulmonary function. This condition develops when injured tissue forms a one-way valve allowing air to enter the pleural space but preventing it from escaping naturally. It is characterized clinically by rapid onset severe shortness of breath, Tachycardia, tachypnea, hypotension and distention of the neck veins due to SVC compression. Tension

pneumothorax is a clinical diagnosis and treatment should be initiated immediately with needle thoracostomy to decompress the pleural cavity. This should be followed by an emergency tube thoracostomy with underwater seal.

Positive-pressure ventilation following intubation usually exacerbates an existing pneumothorax. Use of sedation for intubation can also worsen hypotension.

Chest tube placement in the fifth intercostal space in the left midaxillary line is usually performed after needle decompression to maintain lung expansion. Patients who continue to remain hemodynamically unstable after needle decompression should have a FAST (Focused Assessment with Sonography for Trauma) examination to look for pericardial tamponade. Hemothorax would also require chest tube placement. However, the **neck veins are collapsed**, not distended, in hemothorax.

Hemothorax - After blunt chest trauma, hemorrhagic shock associated with **decreased breath sounds** and **dullness to percussion** over one hemithorax and contralateral tracheal deviation is most likely due to a large ipsilateral hemothorax.

Pericardial Tamponade - these patients presents with hypotension (unresponsive to IV fluid bolus), tachycardia and elevated jugular venous pressure after blunt thoracic trauma & normal CXR. Cardiac tamponade occurs acutely in trauma because of bleeding into a stiff pericardium that has **no elasticity**. Only 100-200 ml of blood is needed to cause a sudden rise in intrapericardial pressure that compresses the cardiac chambers and compromises both venous return (causing elevated jugular venous pressure) and cardiac output (causing tachycardia and hypotension).

The chest x-ray in these patients can **appear normal** without a change in cardiac silhouette size due to the small amount of pericardial fluid. The resultant cardiogenic shock must be treated **immediately with decompression by pericardiocentesis** or **surgical pericardiotomy** to remove this small fluid and reduce the intrapericardial high pressure acutely.

In contrast, **chronic processes** (such as malignancy or renal failure) cause slow accumulation of pericardial fluid that gradually increases the intrapericardial pressure and allows the pericardial elasticity to adapt slowly. As a result, it may take **1-2 liters of fluid before** the intrapericardial pressure reaches a critical point that leads to the same physiologic changes described above in acute cardiac tamponade. The chest x-ray in these patients tends to show the classic findings of an enlarged cardiac silhouette in a globular shape.

Tracheobronchial Perforation - These patients suffer rapid deceleration chest trauma, likely in association with forceful impact with his vehicle's steering wheel. Chest radiography is the most important initial diagnostic study in all stabilized patients (airway, breathing and circulation secure) following blunt chest trauma. If the patient's chest x-ray shows a **persistent pneumothorax** despite chest tube placement and **pneumomediastinum**, and he has **subcutaneous emphysema** (palpable crepitus below the skin) on physical examination, then the diagnosis of Tracheobronchial Perforation is highly likely. The right main bronchus is most commonly injured in these cases. The diagnosis can be confirmed with **high-resolution CT scanning**, bronchoscopy or surgical exploration. Operative repair is indicated.

Blunt Aortic Injury - For patients involved in motor vehicle accidents or falls from > 10 feet, physicians must have a high suspicion for blunt aortic injury. Blunt aortic injury carries a high mortality rate, making expeditious detection and treatment critical. Though clinical signs and symptoms are highly variable, anxiety, tachycardia and hypertension are common. Therefore, radiographic imaging is critical to diagnosis and should be obtained whenever the mechanism of injury raises suspicion for blunt aortic injury. **Chest x-ray is an appropriate initial screening study.** **Medastinal widening** is the most sensitive finding for blunt aortic injury. Deviation of the trachea or nasogastric tube to the right or depression of the left mainstem bronchus may also be seen. Where the history and chest x-ray findings are equivocal, **chest CT and angiography are appropriate.**

Orthopedics

Meniscal injuries often result from **twisting injuries** with the foot fixed. The medial meniscus is more commonly injured than the lateral meniscus. Patients generally complain of a **popping** sound followed by severe pain at the time of injury. Because the meniscus is not directly perfused, effusion following injury typically is not clinically apparent for many hours following the injury. Examination reveals localized tenderness on the medial side of the knee.

Locking of the knee joint on extension is generally seen in "bucket handle" tears, while range of motion at the knee is limited by pain in all meniscal tears. McMurray's sign, which is indicative of a medial meniscus tear, refers to a palpable or audible snap occurring while slowly extending the leg at the knee from full flexion while simultaneously applying tibial torsion.

Medial Meniscal Tear - Injury to a number of different structures within the knee, including ligaments and cartilaginous menisci, can cause a **popping** or **snapping sensation**. Damage to the medial meniscus is a common knee injury. Meniscal injuries occur most commonly in patients in their thirties and forties while performing activities requiring axial loading and rotation. Meniscal injuries are most commonly caused by a twisting injury with the foot in a fixed position. Patients typically have swelling and pain (usually gradually occurs within 24 hours after the injury) in the knee made worse with twisting movements and occasionally causing a popping sensation. Untreated patients can develop symptoms weeks later consisting of **popping, catching, the knee giving out, and locking** (ie., inability to extend the knee). Physical examination findings that indicate possible meniscal injury are summarized below.

Knee function	<ul style="list-style-type: none"> Joint line tenderness Loss of smooth flexion or extension Inability to move forward and backward while squatting Effusion
Provocative tests	<ul style="list-style-type: none"> Thessaly test: Pain or locking with internal and external rotation of the knee while standing on one leg with the knee flexed to 20° McMurray test: Painful click with passive flexion and extension of the knee with the examiner's thumb and index finger placed on the medial and lateral joint lines Apley test: Pain with pressing the heel toward the floor while internally and externally rotating the foot with knee flexed to 90°

Ligamentous injuries cause rapid joint swelling due to hemarthrosis. (Ligaments have much greater vascular supply than menisci, which rely on diffusion for nourishment) Physical exam may show joint line tenderness in the **anterior & medial aspect of the knee**, decreased range of motion and a positive McMurray's test **MRI provides the definitive diagnosis.**

Vs

The anserine bursa underlies the conjoined tendons of the gracilis and semitendinosus muscles and separates them from the head of the tibia. Anserine bursitis causes tenderness over the **medial aspect** of the knee, and typically affects athletes and obese middle-aged to elderly women. A popping sensation is **not** typically reported.

In cases of **anterior cruciate ligament tear**, the patient gives a history of a **forceful hyperextension** injury to knee or a noncontact torsional injury of

the knee during deceleration. Effusion is seen rapidly following injury. Lachman's test, **anterior drawer test** and pivot shift test are used for clinical diagnosis.

Posterior cruciate ligament injury is classically seen in the "dashboard injury", which refers to forceful posterior-directed force on the tibia with the knee flexed at 90 degrees. The posterior drawer, reverse pivot shift and posterior sag tests will help in clinical diagnosis.

Medial collateral ligament injury is associated with **abduction injury** to the knee. **The valgus stress test** will help in clinical diagnosis of this condition.

Lateral collateral ligament injury is very rare and would be seen in **adduction injury** to the knee. The **varus stress** test will help in clinical diagnosis of these patients.

MRI is the investigation of choice for defining soft tissue injuries of the knee.

Hip Fractures - Falls are the most common cause of a hip fracture in the elderly. Hip fractures are classified by anatomic location and fracture type into either intracapsular (e.g .. femoral neck and head) or extracapsular (e.g .. intertrochanteric and subtrochanteric). Intracapsular fractures have a higher chance of avascular necrosis, while extracapsular fractures have a greater need for implant devices (e.g .. nails and rods).

Hip fractures are common in the elderly and **first require stabilization and treatment for pain control** and **deep vein thrombosis prophylaxis**.

The next step in management is to figure out the cause of the fall, particularly in elderly patients. Common causes include **syncope** (e.g .. **arrhythmias and valvular heart disease**), **other cardiac pathology** (e.g .. **acute coronary syndrome and heart failure**), **central nervous system pathology** (e.g .. **stroke and transient ischemic attack**), **infection** (e.g .. **pneumonia**), and **metabolic disorder** (e.g .. **hypoglycemia**).

Trochanteric bursitis is inflammation of the bursa surrounding the insertion of the gluteus medius onto the femur's greater trochanter. Excessive frictional forces secondary to overuse, trauma, joint crystals, or infection are responsible. Patients with this condition complain of hip pain when pressure is applied (as when sleeping on the effected side) and with external rotation or resisted abduction.

Vs

Hip osteoarthritis causes pain localized **deep within the joint** (in contrast to the superficial tenderness here) that may be referred to the inguinal area or rarely to the knee. Hip osteoarthritis is uncommon before the age of 50. Classically, internal rotation of the hip worsens this pain.

Slipped Capital Femoral Epiphysis (SCFE) - It is characterized by displacement of the femoral head on the femoral neck due to disruption of the proximal femoral growth plate. It is commonly seen in **obese adolescent boys**. The physis (i.e .. physical junction between the femoral head and neck) weakens during early adolescence because it is rapidly expanding and primarily composed of cartilage, which does not possess the strength of bone. When exposed to excessive shear stress, which is magnified by obesity. The physis fractures and the femoral head slips posteriorly and medially relative to the femoral neck.

Patients typically present with **hip** or **knee pain** of insidious onset that causes **limping**. Acute presentations can occur. Diagnosis requires a high degree of clinical suspicion because **knee pain (referred pain)**, not hip pain, is a common presenting complaint with this condition. Physical examination shows *loss of abduction and internal rotation of the hip as well as external rotation of the thigh* while the hip is being flexed. A frog-leg, lateral-view x-ray of the hip is the diagnostic imaging technique of choice.

Patients with SCFE should be promptly treated with **surgical pinning of the slipped epiphysis** where it lies (i.e .. in situ) in order to lessen the risks of avascular necrosis of the femoral head and chondrolysis.

Developmental Dysplasia of the Hip (DOH) - Risk factors are General ligamentous laxity, Family history, significantly more females, Firstborn, Breech, Oligohydramnios &Multiple gestation. Physical examination shows - **Barlow** is most important examination; will dislocate an unstable hip; is easily felt (clunk not a click) - **Ortolani**-reduces a recently dislocated hip (most at 1-2 months of age), but after 2 months, usually not possible because of soft-tissue contractions. Diagnosis-ALL (+) ~ refer immediately for orthopedic consult

If unsure, **Dynamic ultrasound of hips best test for age <4 months.** After 4 months, frog lateral x-ray. Treatment is **Pavlik harness for 1-2 months**, surgery & casting. Complications-acetabular dysplasia, leg length discrepancy.

Transient synovitis is the most common cause of **hip pain** in children, typically occurring in boys age 3-10 years. The cause is unknown but usually **follows a viral infection** or mild trauma. Synovial inflammation leads to pain, decreased range of motion, and limping. On examination, the affected hip is typically flexed, slightly abducted, and externally rotated. This position maximizes the joint space, thereby providing some pain relief.

Because characteristics of transient synovitis overlap with septic arthritis, laboratory studies should be sent to assess for severity of inflammation. In contrast to septic arthritis, children with transient synovitis **rarely** have fever or significant laboratory abnormalities.

Plain radiographs should be done to exclude bony lesions, fractures, and Legg-Calve-Perthes disease (LCP). Anteroposterior and frog-leg lateral views of both hips should be obtained to compare affected and normal sides for subtle changes. Additional workup is unnecessary unless symptoms persist or worsen. Treatment consists of **rest** and **nonsteroidal anti-inflammatory medications** (NSAIDs). NSAIDs (eg. ibuprofen) have both analgesic and anti-inflammatory properties and are recommended over other pain relievers (eg. acetaminophen, opioids). The exception is aspirin, which should be avoided due to the risk of Reye syndrome. Children usually recover within 1-4 weeks and have no complications.

If symptoms persist for more than 4 weeks, or worsen, a diagnosis of **LCP should be reconsidered** as initial radiographs may appear normal in early disease. Magnetic resonance imaging can detect early LCP as well as marrow changes suggestive of osteomyelitis.

Transient Sinovitis for more than 4 weeks = Consider Legg Calve Parthes Disease.

Legg-Calve-Perthes Disease - A 5-year-old boy has developed progressive limping. At first painless, it now hurts to run and walk. The pain is in the anterior thigh. The pain is relieved by rest. Parents recall no trauma.

Idiopathic avascular necrosis of the capital femoral epiphysis in immature, growing child, more in males; 20% bilateral, sometimes after trauma. Presentation is mild intermittent pain in anterior thigh with painless limp with restriction of motion.

Osgood-Schlatter Disease - *Traction apophysitis of tibial tubercle*

(overuse injury).

Look for active adolescent (running, jumping)

Swelling, tenderness, increased prominence of tubercle

Treatment-rest, restriction of activities, knee immobilization, isometric exercises

Complete resolution requires 12-24 months

Morton neuroma - It commonly occurs in runners and is not a true neuroma. The disorder is a mechanically induced neuropathic degeneration with symptoms including numbness and burning of the toes, aching and burning in the **distal forefoot** that radiates forward from the metatarsal heads to the third and fourth toes. The symptoms are worsened by walking on hard surfaces and wearing tight or high-heeled shoes.

Physical examination typically shows pain between the third and fourth toes on the plantar surface and a clicking sensation (Mulder sign) when simultaneously palpating this space and squeezing the metatarsal joints. The diagnosis is made clinically, and treatment involves using metatarsal support with a bar or padded shoe inserts to decrease pressure on the metatarsal head. The inserts should be placed in both shoes to ensure that the patient walks evenly. If conservative treatment fails, then the patient usually requires surgical treatment.

Tarsal tunnel syndrome involves compression of the **tibial nerve** as it passes through the ankle and is usually caused by a fracture of the bones around the ankle. Patients typically present with burning, numbness, and aching of the distal plantar surface of the foot or toes that sometimes radiate up to the calf.

Stress fractures - It most commonly occurs in athletes (up to 15% incidence in runners) or nonathletes who **suddenly increase their activity**. Stress fractures are also common in **ballet dancers, basketball and soccer players, and military recruits**. The causes of stress fractures are categorized as activity related (e.g., excessive training and improper footwear), biomechanical (e.g., weak calf muscles, high arched feet, etc.), or metabolic (e.g., demineralized bone from hormonal or nutritional diseases). The fractures occur due to a sudden increase in repeated tension or compression without adequate rest that eventually breaks the bone.

- **Metatarsal stress fractures** typically occur in athletes and military recruits, particularly due to the sudden and drastic increase in activity by the latter. The second metatarsal, which is subjected to significant extremes of loading during gait, is the most commonly involved

metatarsal. Patients present complaining of slow onset foot pain that initially only occurs with activity but later is present during rest as well. Point tenderness over the affected metatarsal is present on examination. **Fractures of the second, third and fourth metatarsals** are managed conservatively because the surrounding metatarsals act as splints and nonunion is uncommon. **Rest and pain control are the most appropriate treatment.** Patients may additionally be managed with a hard-sole shoe and light activity may be resumed immediately.

- The tibia is the major weight-bearing bone in the leg, and patients usually develop **medial tibial stress syndrome** (i.e., "shin splints" with no tibial tenderness on palpation). This can progress with further activity to a complete or incomplete fracture, resulting in pain to palpation of the tibia. The diagnosis of a stress fracture is clinically made on examination with **pain at a specific area that increases with jumping or running** and is associated with local swelling and point tenderness to palpation. X-rays are frequently **normal** but can reveal periosteal reaction in the site of the fracture. The injury is best defined radiographically using MRI or bone scan. Treatment involves **rest** and healing of the stress fracture.

Fat embolism is common in patients with polytrauma, especially with multiple fractures of long bones. It is characterized by **severe respiratory distress, petechial rash**, subconjunctival hemorrhage, tachycardia, tachypnea and fever. Diagnosis can be confirmed by presence of **fat droplets in urine** or presence of **intra-arterial fat globules on fundoscopy**. It may occur from 12 to 72 hours after the injury. Central nervous system dysfunction initially manifests as confusion and agitation but may progress to stupor, seizures, or coma and frequently is unresponsive to correction of hypoxia. Thrombocytopenia and hypofibrinogenemia are nonspecific findings. Serial x-rays shows increasing diffuse bilateral pulmonary infiltrates within 24-48 hours of onset of clinical findings.

Treatment should include prompt respiratory support. Use of heparin, steroids and low molecular weight dextran is controversial.

Scaphoid Fracture - This patient presents with acute injury of the right wrist concerning for a scaphoid fracture, which is the most common carpal bone fracture. The typical cause is a fall on an outstretched arm with a dorsiflexed wrist $>95^\circ$. The scaphoid is on the radial side of the wrist and can be forced against the dorsal part of the distal radius while falling, creating a

fulcrum for injury. Scaphoid fractures are particularly concerning because of the tenuous blood supply of the scaphoid, making the proximal fracture fragment vulnerable to avascular necrosis. Patients usually have pain on the radial aspect of the wrist in the anatomic snuffbox, minimally decreased range of motion (unless dislocated fracture is present), decreased grip strength, and possible swelling.

The next step in this patient is to confirm the diagnosis with **plain x-rays of the wrist** in full pronation and ulnar deviation to better expose the scaphoid. Initial x-rays of a patient with a suspected scaphoid fracture can show a fracture if it is displaced. **If the fracture is compressed or minimally displaced, it can take up to 10 days to show abnormalities on x-rays.** The next step in the management of a suspected scaphoid fracture includes **immobilizing the wrist with a thumb spica cast for 7-10 days followed by repeating the x-rays.** If the patient cannot tolerate immobilization or an immediate diagnosis is needed, either CT scan or MRI of the wrist can distinguish between fracture and ligament injuries.

Rotator Cuff Tear - The rotator cuff is formed by the tendons of the supraspinatus, infraspinatus, teres minor and subscapularis muscles. **The supraspinatus is most commonly injured**, due to repeated bouts of ischemia near its insertion on the humerus induced by compression between the humerus and the acromion.

A common cause of acute rotator cuff tears is a fall on outstretched hands. Patients usually have severe shoulder pain and edema following the traumatic event and are unable to abduct the arm past 90 degrees.

The drop arm test is a maneuver that can help to diagnose a rotator cuff tear. Here, the patient's arm is abducted passively to greater than 90 degrees, and the patient is then asked to lower the arm slowly. With a complete rotator cuff tear, the patient will be unable to lower the arm smoothly and it will appear to drop rapidly from near the 90 degree position.

Rupture of the tendon of the long head of the biceps produces a positive "**Popeye sign**" where the biceps muscle belly becomes prominent in the mid upper arm. Weakness with supination is prominent, and forearm flexion is typically preserved.

Injury to the long thoracic nerve causes a **winged scapula** due to paralysis of the serratus anterior muscle. The most common cause is iatrogenic injury during axillary lymphadenectomy.

Klumpke's Palsy - The lower (inferior) trunk of the brachial plexus originates from the C8 and T1 cervical roots. Injury to this trunk, which typically results from sudden upward pulling on the arm, produces Klumpke's palsy. This palsy primarily affects muscles innervated by the ulnar nerve, which supplies most of the intrinsic muscles of the hand. Weakness and atrophy of the **hypothenar** and **interosseous** muscles characterize this palsy and a "**claw hand**" deformity may also result.

Anterior Dislocation of Shoulder Joint - forceful abduction and external rotation of the arm results in anterior dislocation of the humeral head from the glenoid fossa. In anterior shoulder dislocations, the humeral head is displaced anteriorly relative to the glenoid fossa and the anterior capsule of the glenohumeral joint is often torn. Physical exam will reveal prominence of the acromion with an abnormal subacromial space where the humeral head normally resides.

In anterior dislocations, the patient holds the arm slightly **abducted** and **externally rotated**. Fullness of the anterior shoulder is noted on palpation. Anterior shoulder dislocations warrant neurologic examination of **axillary nerve function**, as the axillary nerve courses around the medial undersurface of the humeral head through the quadrangular space and can be injured by anteroinferior shoulder dislocations. Axillary nerve injury can cause paralysis of the deltoid and teres minor muscles as well as loss of sensation over the lateral upper arm.

Posterior Dislocation of Shoulder Joint - It is commonly caused by violent muscle contractions during a **tonic-clonic seizure** or **electric shock**. Examination will show flattening of the anterior shoulder, prominent coracoid process, and the patient holding the arm **adducted** and **internally rotated** with an inability of external rotation. Radiographs can show internal rotation of the humeral head with circular appearance (light bulb sign), widened joint space >6 mm (rim sign), or two parallel cortical bone lines on the medial aspect of the humeral head (trough line sign). Management involves closed reduction, usually in the operating room but sometimes in the emergency room.

Nursemaid elbow is a common injury among preschool children. It refers to subluxation of head of radius at elbow joint. The injury can occur innocently from swinging a young child by the arms or pulling a child's arm while in a hurry. The child is usually not in distress at presentation but would cry at any attempt to flex the elbow or supinate the forearm. Diagnosis is made clinically as radiographs are often normal.

Treatment of this condition is closed reduction in following steps:

- First extend and distract the elbow.
- Next supinate the forearm.
- Hyper flex the elbow with your thumb over the radial head in order to feel the reduction as it occurs.

No post reduction films are needed, since success is usually demonstrated by the infant who has resumed the use of previously unused extremity.

Important Nerves of Lower Extremity -

The femoral nerve innervates the muscles of the anterior compartment of the thigh, and is therefore responsible for ***knee extension*** and ***hip flexion***. The femoral nerve provides sensation to the ***anterior thigh*** and ***lower medial leg*** via the saphenous branch.

The tibial nerve supplies the muscles of the posterior compartment of the thigh, posterior compartment of the leg, and plantar muscles of the foot. These muscles control flexion of the knee and digits, and plantar flexion of the foot. The tibial nerve provides sensation to the leg (except medial side) and plantar foot.

The obturator nerve innervates the medial compartment of the thigh (i.e .. gracilis adductor Longus, adductor brevis, anterior portion of adductor magnus), and controls adduction of the thigh, It provides sensation over the ***middle medial thigh***.

The common peroneal nerve gives rise to the superficial and deep peroneal nerves. These two nerves supply the muscles of the anterior and lateral leg. These nerves provide sensation to the ***anterolateral leg*** and ***dorsum of the foot***.

Damage to Superior Gluteal Nerve - Trendelenburg sign, a drooping of the contralateral pelvis that occurs when the patient stands on one foot. The associated Trendelenburg gait is waddling in quality, caused by the trunk's rocking to compensate for this pelvic drooping during the stance phase of gait.

Normally, the gluteus medius and gluteus minimus muscles, which are both innervated by the superior gluteal nerve, function to abduct the thigh at the hip when standing on one foot or during normal ambulation when the body's weight rests on only one foot. Weakness of these muscles, as can occur in neuromuscular disease, impingement of or trauma to the superior gluteal

nerve, or inflammatory myopathies, results in a positive Trendelenburg sign and gait.

Paget's disease of bone, also known as osteitis deformans, is a disease of unknown etiology that is characterized by disordered bone remodeling. Specifically, osteoclast activity is increased leading to drastically enhanced bone resorption. This results in accelerated osteoblast activity in an effort to rebuild the degraded bone. The result is formation of structurally inferior woven bone at various sites throughout the body. The woven bone formed in Paget disease lesions is larger than normal bone and is prone to bowing and fracture.

The most common presenting symptom in patients with this condition is pain resulting from bowing or fracture of the long bones that may result in secondary arthritis of the hip or knee. Laboratory testing in patients with Paget disease of bone will typically show an **increase in alkaline phosphatase** and **normal serum calcium and phosphorus levels**. Lesions may be identified radiographically with x-rays or by bone scanning. Enlargement of the bones of the skull in this condition may cause **frontal bossing, increased head size** (old hats no longer fit the patient), **headaches** and **cranial nerve palsies**. Classically, **hearing loss** may occur due to damage to the cochlear nerve resulting from enlargement of the temporal bone and impingement in the internal auditory meatus. In one series, 37% of patients suffered from hearing loss.

Compartment syndrome may be caused by direct trauma (hemorrhage), **prolonged compression of an extremity** or **after revascularization of an acutely ischemic limb**. Muscles of the extremity are encased in fascial compartments that do not allow for expansion of tissue. Blood flow at the capillary level relies on the pressure difference between the arterial and venous systems. Hemorrhage or edema within muscle causes increased pressure within the non-distensible fascial compartment. This increased pressure interferes with perfusion by disallowing passage of blood from the arterial system into the capillary beds of the affected muscles. This eventually leads to muscle necrosis.

Patients with compartment syndrome classically complain of **severe pain that is worsened on passive range of motion**, paresthesias, pallor and paresis of the affected limb. Pressure in the compartments can be measured directly using a needle and pressure transducing catheter system. Pressures over 30 mmHg may result in cessation of blood flow through the capillaries and should be **treated emergently by fasciotomy**.

Injury to limb + Pain on Passive Motion = Compartment Syndrome.

If compartment syndrome has been ruled out and the leg swelling is due to edema, then elevation and ice may be a good choice. After compartment syndrome is relieved by fasciotomy, the leg is always elevated as well.

Supracondylar fracture of humerus is common in young children and adolescents secondary to fall on an outstretched hand. Compartment syndrome results from increased pressure within a limited anatomic space, acutely compromising the circulation and ultimately threatening the function of the tissue within that space. Diagnosis of compartment syndrome is made predominantly on clinical findings of pain, pallor, pulselessness, paralysis and paresthesia. Treatment consists of immediate fasciotomy. ***Volkmann's ischemic contracture*** is the final sequel of compartment syndrome in which the dead muscle has been replaced with fibrous tissue

Pre & Post Op Care

Pre Op INR Normalization - If a patient on Warfarin needs surgery, Then, reversal of anticoagulation must be rapidly achieved ***pre-operatively by infusion of fresh frozen plasma.***

Postop Atelectasis - Respiratory complications are a common cause of postoperative morbidity and mortality. After upper abdominal surgery, a combination of factors can cause the vital capacity (***VC***) to fall 50% and the functional residual capacity (***FRC***) to fall 30%. First, ***postoperative pain*** promotes shallow, rapid breathing. ***Narcotic analgesics*** further decrease respiratory drive, deep inspirations and coughing. Some anesthetic agents depress mucociliary clearance and may promote bronchiolar obstruction as well. Additionally, obese patients will experience a Pickwickian-like syndrome when kept chronically supine following surgery. These factors together promote alveolar atelectasis, a major cause of FRC reduction.

These patients have a drop in her oxygen saturation, and blood gas analysis reveals hypoxemia with a borderline low pCO₂ on the 1st post op day. Vital signs are otherwise normal. Thus, the most likely cause of her hypoxemia is post-operative atelectasis. Atelectasis is one of the most common postoperative pulmonary complications, and is particularly common after abdominal and thoracoabdominal surgery. Following such surgeries, pain and changes in lung compliance can cause ***impaired cough*** and ***shallow breathing.*** Shallow inhalations limit recruitment of alveoli at the lung bases and weak cough predisposes to small airway mucus plugging.

Large areas of atelectasis may cause significant **ventilation-perfusion mismatch**, leading to hypoxemia and increased work of breathing (eg, dyspnea, tachypnea). These manifestations typically start after the patient has left the post-anesthesia care unit, become most severe during the **second postoperative night**, and can last up to 5 days. As compensation for the hypoxemia, patients usually **hyperventilate** and develop **respiratory alkalosis** and **decreased arterial partial pressure of carbon dioxide (PaCO₂) & Hypoxia**. Acute pulmonary embolism can occur postoperatively and present with similar arterial blood gas (ABG) levels.

Chest physiotherapy, incentive spirometry, adequate pain control, coughing and frequent repositioning/early ambulation are all methods that can be used to increase the FRC and prevent atelectasis in the immediate postoperative period. **Simply moving the patient from supine to sitting** reduces the intraabdominal pressure acting on the undersurface of the diaphragm thereby permitting greater alveolar expansion at end expiration and increasing the FRC.

Postop Pneumonia Prevention – the risk factors for postop pneumonia include smoking, preexisting pulmonary disease, age >50, thoracic or abdominal surgery, Surgery lasting > 3 hours and poor general health. Postoperative measures used to decrease the risk of pneumonia are aimed at **encouraging lung expansion**. These include **incentive spirometry**, deep **breathing exercises**, **continuous positive airway pressure**, and **intermittent positive pressure breathing**. Of these, **incentive spirometry** has been shown to be the **most effective**, and is thus the first line preventive measure

Ventilator associated pneumonia (VAP) may occur in patients who are on assisted ventilation for **>48 hours**, and is a result of impairments in the lungs natural defenses. **Fever and hypoxia are common**, and *Pseudomonas* infection must be considered.

Mechanical Ventilation & RQ - The RQ is the steady-state ratio of carbon dioxide (CO₂) produced to oxygen (O₂) consumed per unit time and may be used to make assessments of the metabolism taking place in particular organs or in the body as a whole. In a steady resting state, this ratio depends mainly upon the major fuel being oxidized for ATP production. **A RQ close to 1.0 indicates that carbohydrate is the major nutrient being oxidized.**

Metabolism of proteins alone yields an RQ of approximately 0.8 and oxidation of fatty acids alone for ATP production yields an RQ of approximately 0.7. The RQ for a given fuel is calculated by dividing the number of moles of CO₂

produced by the number of moles of O₂ required to oxidize one mole of substrate. An example using glucose is shown below:



Thus, the RQ for glucose is $6\text{CO}_2/6\text{O}_2 = 1.0$. Because the body normally utilizes a combination of fuels, the normal full-body steady-state RQ is typically near 0.8. Assessment of the RQ is important when attempting to wean patients from mechanical ventilation, as overfeeding, especially with carbohydrates, can cause excessive CO₂ production and make weaning more challenging. This factor is especially important in patients with preexisting lung disease.

Post Op Ileus - An ileus is a functional defect in bowel motility without an associated physical obstruction. Abdominal surgery is the most common cause, with some degree of ileus occurring following most abdominal procedures. Signs and symptoms of postoperative ileus include nausea, vomiting, **abdominal distention, failure to pass flatus or stool**, and **hypoactive or absent bowel sounds** on physical examination. (In contrast, mechanical bowel obstruction causes hyperactive "tinkling" bowel sounds.) Contributors to defective bowel motility in the postoperative setting include: increased splanchnic nerve sympathetic tone following violation of the peritoneum, local release of inflammatory mediators, and **postoperative narcotic (opiate) analgesics**. Opiates contribute to poor bowel motility by causing disordered peristalsis.

Post Op Prerenal Failure - In patients without preexisting intrinsic kidney disease, oliguria is defined as less than 400cc. or less than 6cc/kg of urine output per day. Volume depletion secondary to intraoperative blood loss is the most likely diagnosis when there is **oliguria & anemia**. Volume depletion causes poor renal perfusion leading to prerenal azotemia and oliguria. The elevated BUN/Cr more than 20 supports this notion. Fractional excretion of sodium FENa will be < 1. **The first step** in the management of any patient with new-onset oliguria should be to **change the Foley catheter** to ensure that it is not clogged. **Next**, if prerenal azotemia is suspected, a **careful fluid challenge is indicated**.

Intravenous catheters are one of the most common causes of nosocomial infections. Femoral central venous catheters carry a higher risk of causing bacteremia than subclavian catheters. Intravenous catheter infections are most commonly caused by cutaneous organisms, such as Staphylococci, but femoral catheters may also cause gram-negative bacteremia.

Post Op Colonic ischemia is a known complication of abdominal aortic aneurysm repair. It is due to interference of blood flow to the **distal left colon** due to abdominal surgical procedures. Common causes include loss of collateral circulation, manipulation of vessels with surgical instruments, prolonged aortic clamping and impaired blood flow through the inferior mesenteric artery. Patients classically present acutely following the procedure with dull abdominal pain in the area overlying the ischemic bowel and bloody diarrhea. X-rays of colonic ischemia are usually nonspecific except in cases of advanced disease. **CT scan will show thickening of the bowel wall.** Colonoscopy characteristically shows cyanotic mucosa with hemorrhagic ulcerations. There is typically a sharp transition from affected to unaffected mucosa with only a segment of bowel affected by ischemia.

Postop Acute Bacterial Parotitis - These patients have fever, leukocytosis and parotid inflammation. **Dehydrated postoperative patients** and the elderly are most prone to develop this infection. Acute bacterial parotitis presents with painful swelling of the involved parotid gland that is aggravated by chewing. Prominent physical exam findings are a tender, swollen and erythematous gland; with purulent saliva expressed from the parotid duct. The most common infectious agent is ***Staphylococcus aureus***. **Adequate fluid hydration and oral hygiene, both pre- and post-operatively can prevent this complication.**

Perioperative use of beta-blockers in patients with coronary artery disease decreases the likelihood of myocardial ischemia.

Acute Adrenal Insufficiency - Acute onset of nausea, vomiting, abdominal pain, hypoglycemia and hypotension after a stressful event (e.g .. surgical procedure) in a patient who is steroid-dependent is typical. A very important clue to the correct diagnosis is the past medical history indicative of preoperative steroid use. Exogenous steroids depress pituitary-adrenal axis and a stressful situation can precipitate an acute adrenal insufficiency.

Recurrent parotid neoplasm - The two lobes of the parotid gland are separated by the facial nerve, which courses directly through the substance of the gland. If the facial nerve (CN VII) is involved by the tumor, then it may need to be sacrificed in order to achieve a cure. The extracranial facial nerve carries motor innervation to the muscles of facial expression. Its destruction will cause a **unilateral facial droop**.

Post Op Mediastenitis - Acute mediastinitis is a possible complication of cardiac surgery that is usually due to intraoperative wound contamination. Mediastinitis can complicate up to 5% of

sternotomies. Patients typically present post-operatively (usually within 14 days) with fever, tachycardia, chest pain, leukocytosis, and **sternal wound drainage or purulent discharge.**

Chest x-ray usually shows a widened mediastinum in non-postoperative mediastinitis, but this can also be seen in postoperative mediastinitis after cardiac surgery. The diagnosis is usually clinically made and confirmed during surgery when pus is noted in the mediastinum. Postoperative mediastinitis requires **drainage, surgical debridement with immediate closure, and prolonged antibiotic therapy.** Antibiotics alone do not appropriately treat mediastinitis. Acute mediastinitis has a mortality rate of 10%-50%, even with appropriate treatment.

Atrial fibrillation commonly (up to 15%-40%) occurs within a few days after CABG and is usually self-limited, with **resolution in <24 hours.** Rate control with beta-blockers or amiodarone is best. Anticoagulation and/or cardioversion is reserved for patients with atrial fibrillation lasting > 24 hours after CABG.

Post Op Necrotizing Surgical Infection - This patient presents with signs and symptoms suggestive of necrotizing surgical infection, as summarized below.

Signs/Symptoms
1. Intense pain in wound
2. Fever, hypotension, and tachycardia
3. Decreased sensitivity at edges of wound
4. Cloudy-gray discharge
5. Tense edema outside involved skin
6. Subcutaneous gas with crepitus

Necrotizing infections more commonly occur in diabetics and are usually caused by mixed gram-positive and gram-negative flora. These infections are considered emergencies if they involve the fascial plane and develop into fascitis. The most important step in the management of this condition is **early surgical exploration** to assess the extent of the process and **debride the necrotized tissues.** Adjunctive therapies include antibiotics, adequate hydration, and tight glycemic control.

Intravenous antibiotics are effective for wound infections with cellulitis.

General Surgery

Boerhaave Syndrome - Spontaneous rupture of the esophagus can occur during episodes of vomiting, particularly when the patient is resisting the vomiting reflex. This results because high intraabdominal pressures are transmitted into the mediastinal esophagus where the transmural difference in pressure is large due to negative intrathoracic pressure. Esophageal rupture in this setting typically occurs a few centimeters above the gastroesophageal junction. The retrosternal pain and **crepitus** in the suprasternal notch are the result of **pneumomediastinum**, which commonly occurs following rupture of the esophagus within the mediastinum.

Cases of esophageal perforation in the hospital are usually due to **medical instrumentation**, such as esophageal dilation procedures or surgery around the esophagus. Spontaneous rupture of the esophagus can be caused by a sudden increase in the intraesophageal pressure combined with negative intrathoracic pressure that can occur with **persistent vomiting** or excessive straining. Other causes include **pill esophagitis from potassium chloride**, Barrett esophagus leading to an esophageal ulcer, infectious esophageal ulcer (e.g *Candida* in HIV patients) and ingestion of a caustic substance.

Patients usually present with sudden-onset and severe pain that is located retrosternally or in the neck, back, and abdomen and made worse with swallowing. Physical examination sometimes shows **subcutaneous emphysema in the neck** or a characteristic **crunching sound** on auscultation of the heart due to mediastinal emphysema (i.e .. Hamman sign). Chest x-ray can show **air in the paraspinal muscles**, a widened mediastinum, pneumomediastinum, or pleural effusions with or without pneumothorax (usually hours to days later).

The diagnosis of esophageal perforation is confirmed with a contrast (usually **water-soluble**)

Gastrograffin esophagogram, which demonstrates contrast leakage at the site of the perforation. Patients with this condition typically require immediate broad-spectrum antibiotic therapy, parenteral nutrition, and surgical repair.

A Mallory-Weiss tear is an **incomplete** mucosal tear at the gastroesophageal junction usually resulting from protracted vomiting. The common presentation is self-limited hematemesis.

Pneumomediastinum **does not** occur in such tears because the rupture is incomplete.

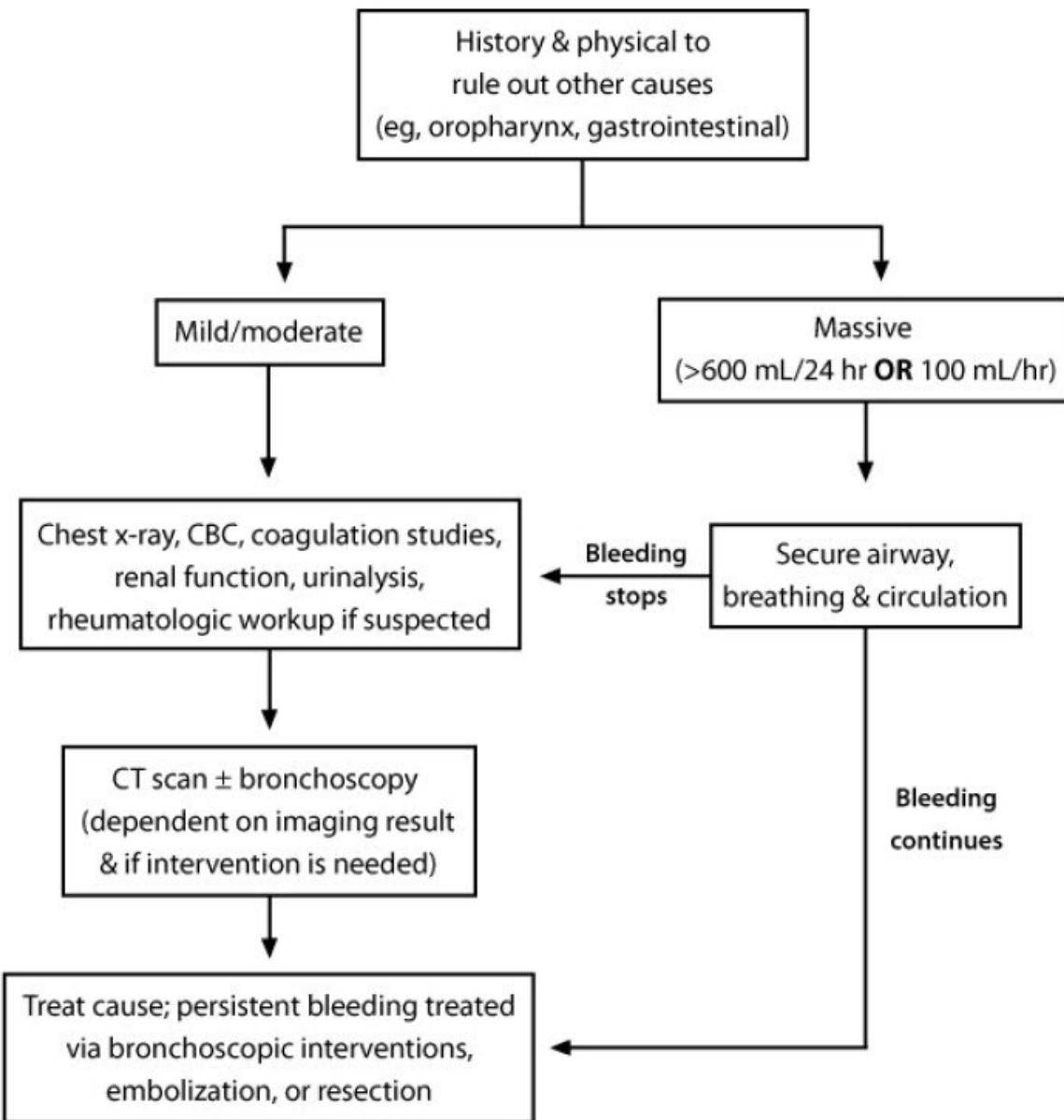
Esophageal perforation due to iatrogenic cause is not infrequent. Chest radiography frequently demonstrates a pleural effusion, pneumomediastinum, and/or pneumothorax. A **water-soluble contrast esophagogram** should be used to confirm the diagnosis.

Hemoptysis - Hemoptysis is defined as any expectoration of blood, with a wide spectrum from minimal blood streaking in sputum to the presence of frank blood and/or clots. Despite the many causes of hemoptysis, **pulmonary airway disease** (eg, chronic bronchitis, bronchogenic carcinoma, bronchiectasis) ranks as the **most common cause**.

Common causes of hemoptysis	
Pulmonary	<ul style="list-style-type: none">• Bronchitis• Pulmonary embolism• Bronchiectasis• Lung cancer
Cardiac	<ul style="list-style-type: none">• Mitral stenosis/acute pulmonary edema
Infectious	<ul style="list-style-type: none">• Tuberculosis• Lung abscess
Hematologic	<ul style="list-style-type: none">• Coagulopathy
Vascular	<ul style="list-style-type: none">• Arteriovenous malformations
Systemic diseases	<ul style="list-style-type: none">• Wegener's granulomatosis• Goodpasture's syndrome• Systemic lupus erythematosus, vasculitis

Massive Hemoptysis - It is defined as >600 ml of expectorated blood over a 24-hour period or a bleeding rate >100 ml/hour. The greatest danger in massive hemoptysis is not exsanguinations but asphyxiation due to the airway flooding with blood. Initial management involves establishing an **adequate patent airway**, maintaining adequate ventilation and gas exchange, and ensuring hemodynamic stability. The patient should be placed with the **bleeding lung in the dependent position** (lateral position) to avoid blood collection in the airways of the opposite lung.

Evaluation of hemoptysis



Woman of child bearing age presenting with Abdominal Pain - These patients presenting with acute onset of abdominal pain may have many etiologies. Including gastrointestinal (e.g .. peptic ulcer disease, cholecystitis, pancreatitis and bowel obstruction or perforation), genitourinary (e.g .. kidney stone and pelvic inflammatory disease) vascular (e.g .. bowel ischemia) or possibly musculoskeletal in origin.

A pregnancy test should be performed before exposing the patient to any form of radiation, such as x-ray or CT scan, used to evaluate her abdominal pain. A fetus exposed to ionizing radiation during the first 14 days after conception either survives undamaged or is resorbed. Exposure after the first 14 days can lead to developmental defects but usually not death. The serum pregnancy test can be positive within 4 days of implantation, while the urine pregnancy test can be negative at that time and take another week to be positive.

Paralytic Ileus is usually caused by an exaggerated intestinal reaction **after abdominal surgery** but can also be seen in other conditions, such as **retroperitoneal hemorrhage associated with vertebral fractures**. Ileus is thought to be due to a disruption of the normal neurologic and motor control of the gastrointestinal tract. Symptoms include **failure to pass stool or flatus, abdominal distention**, nausea and/or vomiting, distended abdomen with tympany, and **decreased or absent bowel sounds**. Abdominal x-rays in patients with paralytic ileus classically show **air-fluid levels** and **distended gas-filled loops** of both the small and large intestines. Management is conservative, with bowel rest, supportive care, and treatment of any secondary causes of the ileus.

Dumping syndrome –It is a common **postgastrectomy complication**. Up to 50% of patients with partial gastrectomy may experience this syndrome. The rate is lower for patients who underwent more conservative gastric surgery (e.g .. proximal vagotomy). The pathophysiology of this condition involves **rapid emptying of hypertonic gastric content** into the duodenum and small intestine. This process leads to the fluid shift from intravascular space to the small intestine, release of intestinal vasoactive polypeptides, and stimulation of autonomic reflexes. This leads to **postprandial abdominal cramps, weakness, light-headedness** and **diaphoresis**. The symptoms begin **25-30 minutes after eating**.

The diagnosis is made clinically, but provocative tests and contrast x-ray studies to demonstrate rapid gastric emptying are occasionally used.

The symptoms usually diminish over time and **dietary changes are helpful** to control the symptoms. In resistant cases, **octreotide** should be tried. Reconstructive surgery is reserved for intractable cases.

Gastric Outlet Obstruction is caused by mechanical obstruction, leading to **postprandial pain** and **vomiting** with **early satiety**. Common causes of gastric outlet obstruction include gastric malignancy, peptic ulcer disease, Crohn disease, **strictures (with pyloric stenosis) secondary to ingestion of caustic agents** and **gastric bezoars**.

Physical examination can show an abdominal succussion splash, which is elicited by placing the stethoscope over the upper abdomen and rocking the patient back and forth at the hips. Retained gastric material > 3 hours after a meal will generate a splash sound and indicates the presence of a hollow viscus filled with both fluid and gas. ***In a patient with a history of acid ingestion, pyloric stricture is the most likely cause.***

Diabetic Gastroparesis may present with similar symptoms but the disease is usually present for decades.

Mechanical Intestinal Obstruction - Colicky or paroxysmal abdominal pain with episodic hyperactive bowel sounds attributable to peristaltic rushes, abdominal distension, and diffuse abdominal tenderness are signs and symptoms of a mechanical intestinal obstruction. ***Nausea and vomiting may be more frequent with small bowel obstruction (SBO)*** as compared to large bowel obstruction (LBO) where the ileocecal valve is competent.

Adhesions are by far the most common cause of SBO. They may be congenital in children (e.g., Ladd's bands), but typically result from abdominal operations or inflammatory processes.

The contents of the vomitus are typically bilious in ***proximal*** SBO and feculent with more distal obstructions. Failure to pass stool or flatus (obstipation) indicates that a complete obstruction has occurred. ***A mild leukocytosis*** and modest increase in amylase are expected with any bowel obstruction.

Whereas a partial SBO may be managed conservatively with nasogastric tube decompression and observation, complete ***SBO requires surgical correction.*** If the patient has metabolic acidosis in this setting, it most likely representing ***lactic acidosis*** due to ischemia of a strangulated loop of small bowel. is a further indication for laparotomy. Strangulation leading to ischemic necrosis and perforation of a SB segment is the major life-threatening complication of SBO. The absence of peritoneal signs in this patient suggests that frank bowel necrosis has not yet occurred, but there are signs of ischemia that warrant ***emergent abdominal exploration*** to reverse the cause of his mechanical bowel obstruction.

MIO = Colicky Abdominal Pain + Nausea & Vomiting + Absent Flatus/Stool + Increase in bowel sounds during colicky pain + Fever & Leukocytosis + Metabolic (Lactate) Acidosis + Pain in all 4 quadrants.

Appendicitis - These patients presents with RLQ pain, nausea, vomiting, fever and leukocytosis. Appendicitis typically begins as a vague, dull, periumbilical pain caused by midgut visceral pain due to stretching of the appendiceal wall. As the peritoneum in the RLQ becomes inflamed, the pain becomes sharp and localizes to the RLQ. Patients can have subsequent examination findings of pain with palpation of McBurney point in the RLQ and Rovsing sign (RLQ pain with deep palpation of the LLQ). Laboratory studies can show leukocytosis or sometimes be nonspecific.

Appendicitis is a **clinical diagnosis** with many other etiologies in the differential diagnosis (e.g .. diverticulitis, ileitis, Crohn disease etc.). Patients who present with the classic signs, Symptoms, and laboratory data of appendicitis do not need further imaging to confirm the diagnosis. Studies have shown that **further imaging** with either CT scan or ultrasound **can delay treatment** in patients who have classic clinical presentation of appendicitis. Imaging with either CT scan or ultrasound of the abdomen is useful in patients who have a suspected diagnosis of appendicitis with atypical presentation. Women of childbearing age and older adults have a higher rate of negative exploratory laparotomy and would benefit more from initial imaging with ultrasound or CT scan. Pregnant women should be **initially evaluated with ultrasound of the abdomen to evaluate for pelvic pathology**.

Patients with a **classic presentation** of appendicitis should have **an immediate operation** to remove the inflamed appendix and prevent appendiceal rupture. Antibiotics should be given preoperatively and continued postoperatively if there is appendiceal rupture.

Patients who do not seek medical care until **>48 hours** after the onset of symptoms have a **high risk of perforation** into the peritoneal and retroperitoneal spaces, causing peritonitis or **an abscess**. Patients at this stage can have fever, leukocytosis (**leukocytes >15,000/mm³**), **reactive thrombocytosis**, right lower quadrant pain, nausea and abdominal signs on physical examination. A **positive psoas sign, suggestive** of a **psoas abscess**, is a known complication of perforated appendix.

Patients who are ill-appearing with suspected perforation typically present within 72 hours of symptom onset and should undergo an emergent laparotomy. **Patients who present with a longer duration of symptoms (>5 days) likely have a contained abscess** and appear more stable.

In males, appendicitis is a common cause of **pelvic abscess** while gynecologic issues more commonly cause pelvic abscess in females. The finding of a tender, fluctuant mass palpable only with the tip of the examining finger on rectal examination indicates an **abscess in the**

rectovesical pouch as the prostate is much more readily palpable and would cause different symptoms. Patients with pelvic abscesses typically present with fever, leukocytosis, painful defecation and diarrhea resulting from bowel irritation by the intraabdominal infection. Drainage of the abscess is the usual treatment in such cases.

They should be treated with antibiotics; intravenous fluids and bowel rest because surgery has been associated with a higher morbidity. A CT scan may reveal an abscess that can be percutaneously drained. Most patients will require an interval appendectomy 6-8 weeks later to prevent future appendicitis.

Acute Diverticulitis – It is an acute inflammation of the colonic wall that can be classified as **uncomplicated (75%)** or **complicated (25%)**.

Uncomplicated diverticulitis is characterized by colonic diverticular inflammation resulting in LLQ pain and tenderness, fever and leukocytosis. A CT scan can show associated inflammation as soft tissue stranding and colonic wall thickening. Uncomplicated diverticulitis in stable patients can be managed in the outpatient setting with **bowel rest, oral antibiotics, and observation**. Hospitalization, treatment with intravenous broad-spectrum antibiotics, and observation is recommended for patients who are elderly, are immunosuppressed, have high fever or significant leukocytosis, and with significant comorbidities.

Complicated diverticulitis refers to diverticulitis associated with an **abscess, perforation, obstruction or fistula formation**. Fluid collection **<3 cm** can be treated with intravenous antibiotics and observation, with surgery reserved for patients with worsening symptoms

Fluid collection **>3 cm** should initially be drained with **CT-guided drainage**. If the drain does not control symptoms by the fifth day, surgery for drainage and debridement is the next recommended step. **Surgery with sigmoid resection** is generally reserved for patients with **fistulas, perforation with peritonitis, obstruction or recurrent attacks of diverticulitis**.

Pancreatic cancer classically presents insidiously with a combination of **constant visceral epigastric pain** radiating to the back, **jaundice** due to extrahepatic biliary obstruction and **anorexia** with **weight loss**.

Upper GI Bleeding

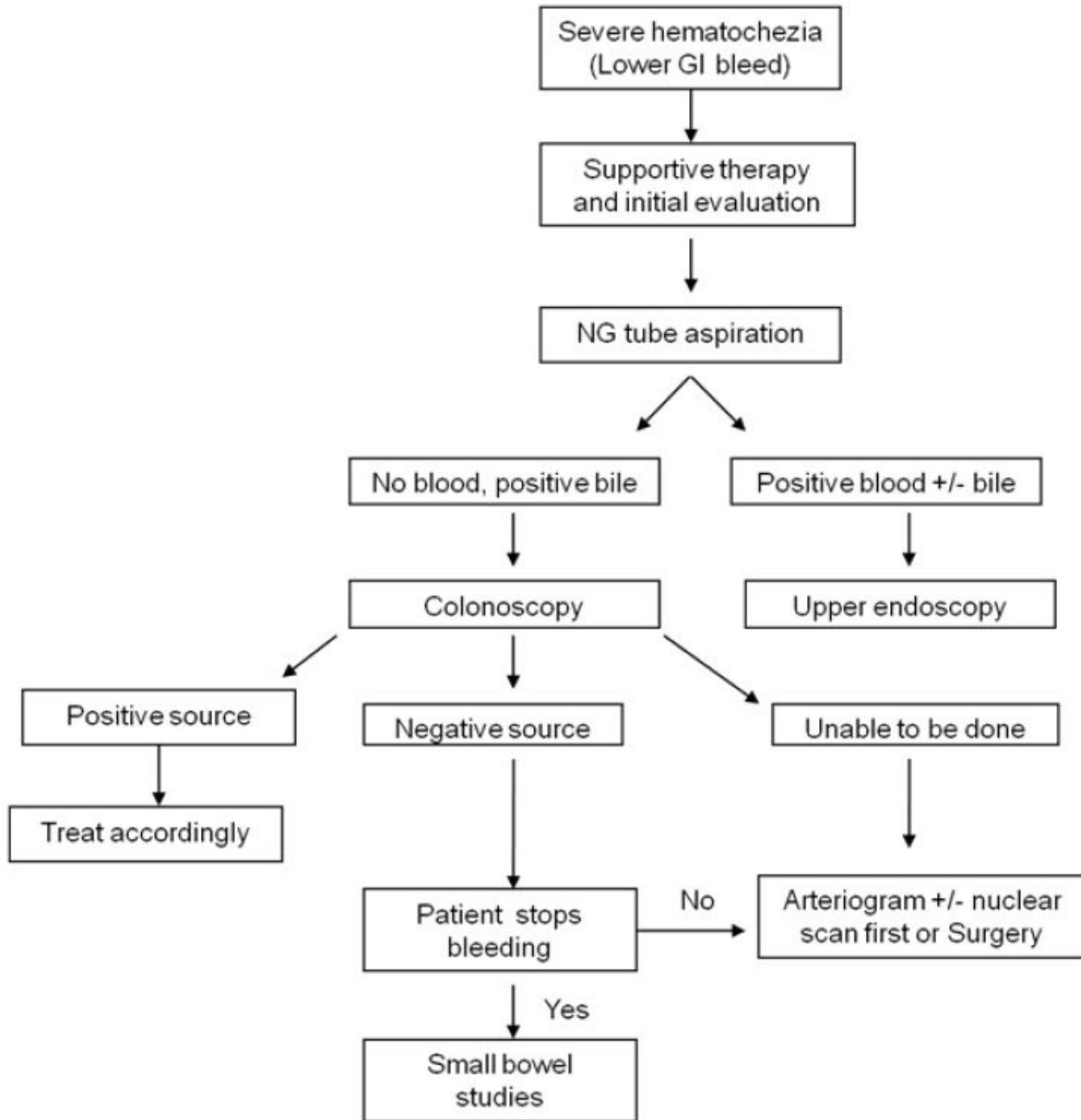
Non bleeding varices are managed with nonselective beta-adrenergic antagonists, such as propranolol. Such therapy can reduce the risk of bleeding by up to half.

Sclerotherapy, endoscopic band ligation and surgery are indicated after a patient has experienced a first episode of variceal bleeding.

A portosystemic shunt connects the portal venous system to the systemic venous system. This can be accomplished surgically or with a TIPS procedure. These procedures are considered a last resort in variceal bleeding unresponsive to medical and endoscopic interventions and may worsen the encephalopathy in patient.

Lower GI Bleeding -

Hematochezia is usually due to lower GI bleeding (distal to Ligament of Treitz), but it may occur in cases of very brisk upper GI bleeding. The most common causes of acute lower GI bleeding (LGIB) in patients over 50 years old are anatomic (diverticulosis), vascular (angiodysplasia, ischemia), infectious, and neoplasms.



The first step is to do a **NG suction** to rule out upper GI bleeding. If upper GI bleeding is ruled out, the next step is evaluation with a **colonoscopy**. If the colonoscopy is negative and the patient continues to bleed, the next step would be to consider **radionuclide imaging** to help identify the general site of bleeding. Although not a very specific study, labeled erythrocyte scintigraphy (tagged RBC scan) is less invasive and more sensitive than angiography. If the RBC scan identifies a bleed, it can be followed by angiography or repeat colonoscopy to identify and potentially treat the exact bleeding source. If all of the above tests are negative, an upper endoscopy with push enteroscopy to evaluate the small bowel or surgical therapy should be considered.

Diverticulosis is the most common cause of lower GI bleeding in an elderly patient. Colonic diverticula form due to high intraluminal pressure in the colon, which causes the mucosa and muscularis mucosa to herniate through the bowel wall at sites where it is penetrated by vasculature. Because they do not include all layers of the bowel wall, colonic diverticula are regarded as false diverticula. Chronic constipation resulting from a low-fiber diet is the most common predisposing factor for diverticulosis. Due to their proximity to the bowel vasculature, diverticula can erode a penetrating artery, resulting in profuse self-limited bleeding per rectum.

Colon cancer tends to present with **chronic occult blood loss**. Gross bleeding is less likely.

Gall Bladder -

Biliary colic secondary to gallstones - Ingestion of a fatty meal causes the gallbladder to contract, which can press gallstones against the cystic duct opening, increasing intragallbladder pressure and causing distension and colicky pain. Subsequent gallbladder relaxation allows the stone to fall back from the duct, causing the pain to resolve completely. Biliary colic is often accompanied by nausea, vomiting, and **right-sided shoulder or subscapular discomfort** (referred pain). The pain of biliary colic is distinguished from that of acute cholecystitis by its intermittent nature and relation to meals as well as the absence of fever.

Acute cholecystitis refers to inflammation and distention of the gallbladder, typically due to obstruction of the cystic duct by a gallstone. It presents with acute right upper quadrant pain and tenderness, plus fever and leukocytosis. Physical examination may reveal a positive Murphy's sign. The gallbladder is palpable in one-third of cases.

Ultrasonography is **more sensitive** than CT scan for making the diagnosis of acute cholecystitis.

A HIDA scan can be used in patients where **ultrasonogram cannot** make a clear diagnosis of acute cholecystitis.

It is appropriate to manage acute cholecystitis conservatively followed by **cholecystectomy within 72 hours**. Symptoms often subside within a few

days with volume resuscitation, Antibiotics, and pain medications. Early cholecystectomy (within 72 hours) reduces disease duration, duration of hospitalization, and cholecystitis-associated mortality when compared to delayed cholecystectomy. Laparoscopic cholecystectomy is the surgical procedure of choice in patients without contraindications.

Biliary Pancreatitis - The most common causes of acute pancreatitis are **gallstones** (biliary pancreatitis), alcohol consumption, hypertriglyceridemia, and recent endoscopic retrograde cholangiopancreatography (ERCP). Treatment of pancreatitis is primarily supportive, with intravenous fluids, nasogastric tube suction, restriction of nothing by mouth, and analgesia. In severe cases, antibiotics are indicated, and calcium and magnesium levels should be monitored and replaced as needed.

Pancreatitis + Elevated LFTs = Biliary Pancreatitis.

Ultrasound is the preferred test to detect gallstones, with a sensitivity of 72%-84% and specificity of 99%. Comparatively, CT scan has a sensitivity of 52% and specificity of almost 100% because most stones are isodense with bile and not easily visible on CT. As a result, CT scan can miss many gallstones and is inferior to ultrasound. Endoscopic ultrasound/ERCP is recommended for extraction of a visible common bile duct stone on ultrasound. Stable patients with biliary pancreatitis should also undergo a laparoscopic cholecystectomy prior to discharge to prevent recurrent pancreatitis, which can be as high as 25%-30% without cholecystectomy.

Acalculous cholecystitis - This condition is most often seen in patients chronically **hospitalized in the intensive care unit** with any of the following conditions: multiorgan failure, severe trauma, surgery, burns, sepsis or prolonged parenteral nutrition. The pathogenesis of this condition is unclear, but it is most likely due to cholestasis and gall bladder ischemia leading to secondary infection by enteric organisms, edema of the gall bladder serosa, and necrosis of the gall bladder. Most patients affected by this condition have no prior history of gall bladder disease.

Acalculous cholecystitis is a serious condition that can lead to sepsis and death if undetected. The clinical signs of disease (such as fever and leukocytosis) are vague, and patients most vulnerable to this condition are typically noncommunicative due to their general medical condition. The best way to make the diagnosis is to have a high degree of clinical suspicion and to confirm the diagnosis with imaging studies that demonstrate **gallbladder distention**, gall **bladder wall thickening**, and the **presence of pericholecystic fluid**. Since this patient is critically ill, the immediate

treatment is antibiotics followed by percutaneous cholecystostomy under radiologic guidance. Cholecystectomy with drainage of any associated abscesses is the definitive therapy once the patient's medical condition improves.

Psoas Abscess - Patients with a psoas abscess typically present with fever and lower abdominal or back pain. Deep abdominal palpation is required to elicit tenderness due to the deep location of the psoas on the posterior abdominal wall. As with any infectious process leukocytosis is evident on laboratory tests. **A CT scan is required to confirm the diagnosis.** A psoas abscess may also result from **contiguous spread from nearby bone or bowel.**

Patients with psoas abscess are treated with drainage and systemic antibiotics. Drainage may be accomplished without laparotomy or laparoscopy by placement of a percutaneous drainage tube.

Kidney -

Renal Colic - Patients typically present with severe flank or abdominal pain radiating to the groin, accompanied by nausea and vomiting. Unlike patients with an acute abdomen, patients with urinary stones are often writhing in pain and unable to sit still in the exam room (these patients do not have peritoneal irritation so movement does not worsen their pain).

The first-line modality for diagnosing a urinary stone is a non-contrast **spiral CT of the abdomen** and pelvis. This test can be obtained relatively quickly and will visualize calcium stones and the majority of non-calcium stones

Nephrolithiasis & Crohn's - It classically presents with flank pain and hematuria frequently accompanied by nausea and vomiting. Patients with Crohn disease, or any other small intestinal disorder resulting in fat malabsorption, are predisposed to hyperoxaluria. Oxalate is obtained from the diet and is a normal product of human metabolism. Symptomatic hyperoxaluria is classically the result of increased oxalate absorption in the gut. Under normal circumstances, calcium binds oxalate in the gut and prevents its absorption. In patients with fat malabsorption, **calcium is preferentially bound by fat leaving oxalate unbound** and free to be absorbed into the bloodstream. Failure to adequately absorb bile salts in states of fat malabsorption also cause decreased bile salt reabsorption in the small intestine. Excess bile salts may damage the colonic mucosa and contribute to increased oxalate absorption.

Pilonidal Cyst - Acute pain and swelling of the midline sacrococcygeal skin and subcutaneous tissues is most commonly due to infection of a pilonidal cyst. Pilonidal cysts are most prevalent in young males, particularly those with larger amounts of body hair. The precise etiology of pilonidal cysts and sinuses is not clearly described, but they are believed to develop following chronic activity involving sweating and friction of the skin overlying the coccyx within the superior gluteal cleft. Infection of hair follicles in this region may spread subcutaneously forming an abscess that then ruptures forming a pilonidal sinus tract. The chronic sinus tract may then collect hair and debris resulting in recurrent infections and foreign-body reactions. When the sinus becomes acutely infected, pain, swelling, and purulent discharge occur in the midline postsacral intergluteal region. Treatment is by drainage of abscesses and excision of sinus tracts.

Suppurative hidradenitis (hidradenitis suppurativa), pilonidal disease, dissecting folliculitis of the scalp and acne conglobata are members of the follicular occlusion tetrad. Affected patients present with **multiple painful nodules** and pustules of the axillae and groin. These lesions lead to sinus formation and fibrosis.

A perianal abscess presents with anal pain and a **tender, erythematous bulge** at the anal verge.

A perianal fistula due to chronic anal crypt infection or Crohn's disease would have an **external (cutaneous) opening draining purulent material**. Perianal fistulae are generally located within 3 cm of the anal margin.

Pediatric Surgery

Cardiothoracic Surgery

Cardiac catheterization is typically done by cannulating the femoral artery to access the cardiac vessels. A common complication is hematoma formation in the soft tissues of the upper thigh. If the initial arterial puncture was done above the inguinal ligament, this hematoma can extend directly into the retroperitoneal space and cause significant bleeding, with hypotension and tachycardia. Patients can also develop ipsilateral flank pain/back pain and neurologic deficits on the ipsilateral side.

The next step in management is to obtain a CT scan of the abdomen and pelvis without contrast to confirm the diagnosis. Treatment is mainly supportive (e.g., blood transfusion, intravenous fluids, and bed rest),

with intensive monitoring. If the bleeding continues or the patient is hemodynamically unstable, the patient might need systemic reversal of anticoagulation. Patients who develop neurologic deficits in the ipsilateral extremity require ***urgent decompression*** of the hematoma.

Cardiac Catheterization Followed by Tachycardia + Hypotension = Hematoma

Major Complications	<ul style="list-style-type: none">● Myocardial infarction (<0.1%)● Stroke/TIA (0.2%–0.4%)● Death (<0.1%)
Minor Complications	<ul style="list-style-type: none">● Hemostasis at access site● Hematoma formation● Arteriovenous fistula● Pseudoaneurysm● Arterial thrombosis● Perforation of heart or great vessels● Acute kidney injury● Contrast allergy

Vascular Surgery

PAD - Risk factors include increasing age (especially > 70 years), Diabetes mellitus, cigarette smoking, hypertension and hyperlipidemia. Patients usually present with either atypical leg pain or claudication, but up to 50% of the patients aged >50 years may be asymptomatic. Severe cases can result in limb ischemia and infarction.

Current guidelines recommend obtaining the ***ankle-brachial index (ABI) using Doppler as the first step*** to diagnose PAD in high-risk or symptomatic patients. The ABI is the ratio of the systolic blood pressure in the posterior tibial and dorsalis pedis arteries over the systolic blood pressure in the brachial artery. Ratios of 1- 1.3 are considered normal, while ***a ratio <0.9*** is highly sensitive and specific for ***>50% occlusion in a major vessel***. Ratios <0.4 are consistent with limb ischemia. After PAD is

diagnosed by ABI, a number of different imaging studies can be performed to more accurately identify the occluded vessel before pursuing treatment.

Patients who have **significant symptoms suggesting claudication** and a **normal ABI might** have mild disease at rest or arterial entrapment symptoms that **produce false-negative ABI results**.

They should undergo exercise testing with preexercise and postexercise ABI measurement. Exercise augments the flow across a moderately stenotic vessel and exposes a lower ABI compared to rest. If the exercise test ABI readings are normal, PAD is very unlikely to be the cause of the symptoms. If the postexercise ABI measurements are abnormal, further testing can be done to confirm the diagnosis of PAD.

Contrast arteriography is the gold standard for evaluating PAD because it is highly sensitive and specific for determining the specific vessels involved. However, it is an invasive procedure requiring arterial puncture and the use of contrast dye. Arteriography is best reserved for an **unclear diagnosis** or when **planning invasive interventions**.

Aspirin and **cilostazol** are antiplatelet agents that are effective together for the treatment of claudication once the diagnosis of PAD is established.

Leriche syndrome is characterized by the triad of **bilateral hip, thigh and buttock claudication, impotence** and **symmetric atrophy of the bilateral lower extremities** due to chronic ischemia. Impotence is almost always present in men with this condition; in the absence of impotence, an alternate diagnosis should be sought. The pulse is soft or absent bilaterally from the groin distally in this condition. Men with a predisposition for atherosclerosis, such as smokers, are at the greatest risk of this condition. Because impotence is not uncommon in this age group, and the complaints of hip and thigh pain with walking may also be attributed to osteoarthritis, there is a risk of missing this diagnosis if a thorough vascular examination is not performed.

Ruptured AAA – These patients present with hypotension, back/abdominal pain & syncope.

The abdominal aorta is 1-3 cm in diameter in most individuals, and a diameter > 3 cm at the level of the renal arteries is considered to be an aneurysm. Unlike thoracic aortic aneurysms, an AAA involves all aortal layers and does not create an intimal flap or false lumen. An AAA typically occurs in people aged >60 years and occurs at a higher rate in smokers, men and people with a history of coronary artery disease. Patients typically have few

symptoms with AAAs, which are usually incidentally found on screening ultrasound or CT scan of the abdomen.

Physical examination can reveal a pulsatile abdominal mass at or above the level of the umbilicus. Once the aneurysm ruptures, only about 50% of the patients survive to come to the hospital. They present with profound **hypotension, abdominal or back pain followed by syncope** and possible pulsatile mass on examination. **An AAA can rupture into the retroperitoneum and create an aortocaval fistula with the inferior vena cava, leading to venous congestion in retroperitoneal structures (e.g .. bladder). The fragile and distended veins in the bladder can rupture and cause gross hematuria.**

The symptoms and signs can also mimic other abdominal pathologies, such as renal colic, mesenteric ischemia, pancreatitis, diverticulitis and biliary disease. The patient should be immediately taken to the operating room for emergent surgical repair of the ruptured AAA. Mortality with this condition is approximately 50%. so early recognition and operative intervention is essential.

The CT scan shows a ruptured aorta with blood collection in the adventitial layer. This is a classic presentation of a ruptured abdominal aortic aneurysm. An abdominal aortic aneurysm can rupture freely into the peritoneal cavity or into the retroperitoneum. These ruptures are associated with significant abdominal pain and hypotension. Patients with a ruptured aortic aneurysm require **urgent surgery**. If the patient is unstable and rupture has been diagnosed, no further evaluation is required and the patient is transferred straight to the operating room. **CT scanning is only done in stable patients.** In patients **not stable** enough to undergo CT scan, the presence of an aneurysm can be confirmed at the bedside by **ultrasound**. Surgical repair of a ruptured aortic aneurysm carries a very high morbidity and mortality rate.

Hypotension, abdominal or back pain followed by syncope = Ruptured AAA.

Femoral Artery Aneurysm - A pulsatile groin mass below the inguinal ligament is characteristic; anterior thigh pain is due to the compression of the femoral nerve that runs lateral to the artery. Femoral artery aneurysm is the second most common peripheral artery aneurysm after popliteal aneurysm. It may be associated with an abdominal aortic aneurysm.

Ischemia-Reperfusion syndrome - It is a form of compartment syndrome that occurs following more than 4 to 6 hours of ischemia. Tissues can suffer

both intracellular and interstitial edema upon reperfusion. When the extremities are involved, this creates a risk for compartment syndrome. Compartment syndrome is defined as increased pressure within an enclosed fascial space causing ischemia of muscles and nerves. The increased pressure is the result of **acute edema within the muscular compartments of the leg**. Compartment pressures in excess of 30 mmHg indicate the presence of compartment syndrome and the need for emergent fasciotomy. Compartment syndrome most commonly affects the distal leg and forearm, and classically **follows fracture** or **crush injuries** or **after the removal of an embolus from a peripheral artery**. The most reliable indicators of early compartment syndrome include severe pain in the affected extremity that is out of proportion to the physical examination findings and pain that is exacerbated by passive stretch of the muscles in the affected compartment. The clinical findings in compartment syndrome can be remembered as the five

"P's":

- **Pain** is the earliest symptom. It is classically increased by passive stretch of the muscles in the affected compartment.
- **Paresthesias** (burning or tingling sensations) occur in the distribution of the affected peripheral nerve.
- **Pallor** of the overlying skin is the result of tense swelling and compromised perfusion.
- **Pulselessness** is a late finding. The presence of a pulse on exam does not rule out compartment syndrome.
- **Paresis/paralysis** is a late finding resulting from nerve and muscle ischemia and necrosis.

Arterial occlusion in the lower extremity may arise due to one of three major causes: embolus, thrombosis or trauma. All forms of arterial occlusion will cause pain, diminished pulses, pallor, coolness to touch, neurologic deficits and muscle dysfunction in the affected extremity. In embolic arterial occlusion, the pain classically occurs **suddenly** and is severe. The pulses tend to be diminished or absent in the affected limb and normal in the unaffected limb. The majority of emboli causing such occlusion originate from the heart either from the ventricles following a myocardial infarction or from the atria in the setting of atrial fibrillation.

Deep vein thromboses occur when the Virchow triad (i.e., stasis, endothelial injury, and hypercoagulability) is present. Major surgery is a significant risk factor. Patients should be treated with a **heparin product**

acutely and **warfarin for several months**. Stable patients can be treated with anticoagulation as early as 48-72 hours after surgery.

Lower extremity edema and Stasis Dermatitis - Both of these conditions are the result of lower extremity **venous valvular incompetence** resulting in pooling of venous blood and increased pressures in postcapillary venules. This increased pressure damages capillaries causing loss of fluid, plasma proteins and erythrocytes into the tissue. Erythrocyte extravasation causes hemosiderin deposition and the **classic coloration of stasis dermatitis**. Inflammation of venules and capillaries as well as fibrin deposition and platelet aggregation cause microvascular disease and ultimately ulcerations will occur. Stasis dermatitis most classically involves the medial leg below the knee and above the medial malleolus. **Xerosis** is the most common early finding; lipodermatosclerosis and ulcerations characterize late disease.

Patients present with unilateral lower extremity edema that worsens when the leg is dependent (i.e. while the patient is at work) and improves with leg elevation (i.e. when the patient is sleeping).

Lymphatic obstruction is an uncommon cause of edema. It may result from malignant obstruction of lymph nodes, lymph node resection, trauma and filariasis. It classically affects the dorsa of the feet and causes marked thickening and rigidity of the skin.

Impaired cardiac contraction and reduced diastolic filling of the heart may cause bilateral lower extremity edema due to pooling of blood in the venous circulation causing increased capillary hydrostatic pressure. However, respiratory symptoms and crackles on examination are common.

Increased urinary loss of protein and **decreased liver protein synthesis** causes decreased plasma oncotic pressure, which results in decreased reabsorption of interstitial fluid in distal capillaries and edema. Urinary protein loss occurs in nephrotic syndromes as well as in most cases of nephritis. Failure of liver protein synthesis typically occurs in the setting of cirrhosis or other forms of liver failure where the synthetic function of the liver is impaired. Other signs of liver failure and nephrotic syndrome are usually evident.

Arterial thrombosis classically presents with a patient complaining of severe pain in a single extremity. The onset of pain is less acute than that

seen in arterial occlusion due to embolus. The extremity typically exhibits coolness to touch, pallor, pulselessness and paralysis on examination.

Raynaud disease / phenomenon is characterized by arterial spasm in response to cold or emotional stress causing discoloration and discomfort of the distal digits. Raynaud phenomenon may ultimately result in **distal digital gangrene** if severe.

Diabetic foot ulcers occur due to three derangements present in all diabetics: neuropathy, Microvascular insufficiency and relative immunosuppression. Diabetic foot ulcers typically occur on the **sole of the foot** on **high-pressure weight bearing sites**, such as below the head of the first metatarsal. Peripheral neuropathy renders the foot and ankle relatively insensate. Due to this inability to sense minor foot trauma and foot pain due to high pressure on the skin, patients with diabetes are predisposed to ulcer formation at these sites. Poor perfusion due to microvascular insufficiency also promotes ulcer formation and predisposes to poor wound healing. Immunosuppression predisposes to infection of these wounds, which may not only slow healing but may also predispose to osteomyelitis.

Ulcers resulting from venous insufficiency (venous valvular incompetence) typically occur on the medial aspect of the leg above the medial malleolus. Such ulcers are often preceded by chronic lower extremity edema and stasis dermatitis.

Central venous catheters are commonly used for access in patients with difficult vascular access and for long term administration of medications. Complications from central venous catheter placement occur in 1 %-5% of the cases and include arterial puncture, pneumothorax, hemothorax, thrombosis air embolism, sepsis, vascular perforation and myocardial perforation leading to tamponade. **A chest x-ray should always be obtained to confirm proper placement of the catheter and absence of complications.**

To avoid myocardial perforation, the catheter tip should be located proximal to either the cardiac silhouette or the angle between the trachea and the right mainstem bronchus. Ideally, the catheter tip should lie in the superior vena cava. Tip placement in smaller veins (e.g .. subclavian, jugular, azygous and internal mammary) predisposes to vascular perforation. Proper placement is essential prior to administering medications and other agents due to the possibility of administration into the wrong anatomic space.

Clavicle Fracture - The clavicle is one of the most commonly injured bones in the body. The majority of clavicular fractures occur in the middle third of

the bone. Injury to this bone classically occurs during athletic events and follows a fall on an outstretched arm or a direct blow to the shoulder. Patients with clavicular fractures present with pain and immobility of the affected arm. The contralateral hand is classically used to support the weight of the affected arm. The shoulder on the affected side is displaced inferiorly and posteriorly. **A careful neurovascular exam** should accompany all fractures to the clavicle due to its proximity to the subclavian artery and brachial plexus. **If a bruit is heard, an angiogram is necessary to rule out injury to the underlying vessel.**

Fractures of the middle third of the clavicle, which account for most clavicular fractures, are treated nonoperatively with a brace, rest and ice. Fractures of the distal third of the clavicle may require open reduction and internal fixation to prevent nonunion. In cases managed nonoperatively, early range of motion and strengthening are recommended to prevent loss of motion at the shoulder.

Skin Surgery -

The initial management of burn injuries is identical to the management of all trauma patients- airway, breathing and circulation must always be secured first. Burn victims are at high risk for respiratory compromise because the **supraglottic airway**, which efficiently exchanges heat with inhaled air, is **very susceptible to direct thermal injury** and acute obstruction by edema and blistering. (In contrast, the subglottic airway is protected from injury by reflexive closure of the vocal cords upon exposure to extremely hot air.)

Clinical indicators of thermal and smoke inhalation injury include: **burns on the face**, singeing of the eyebrows, **oropharyngeal inflammation**, **blistering** or **carbon deposits**, **carbonaceous sputum**, **stridor**, **carboxyhemoglobin level > 10%**, and **a history of confinement in a burning building**. All burn victims should be treated initially with high-flow oxygen via a non-rebreather mask, though caregivers should maintain a low threshold for **intubation** in any patient with physical evidence of thermal damage to the upper airway. A key reason for early intubation is that progressive airway edema may preclude intubation later in the patient's clinical course, potentially necessitating an emergent surgical airway.

Eschar formation on a limb can sometimes compromise both blood and lymph circulation, resulting in a decrease in pulses and significant edema distal to the burn. Next, Doppler ultrasonography should be done to

document peripheral pulses and estimate the tissue compartment pressure. A pressure of 25-40 mm Hg is the threshold to perform escharotomy. Escharotomy relieves pressure on the vascular supply by making an incision (under sterile conditions) only through the eschar layer.

Escharotomy is indicated for ***circumferential full-thickness burns of an extremity*** with an eschar causing significant edema and constriction of the vascular supply and decreased peripheral pulses.

SCC - The diagnosis of malignancy should be suspected in all non-healing wounds. Squamous cell carcinoma (SCC) may arise within chronically wounded, scarred or inflamed skin. SCC arising within burn wounds is known as a ***Marjolin ulcer***. SCC has also been described arising in the skin overlying a focus of osteomyelitis, radiotherapy scars and venous ulcers. SCC arising within chronic wounds tend to exhibit more aggressive behavior, so early diagnosis is key to preventing metastatic disease in such patients. A biopsy should be obtained in all chronic wounds failing to heal in order to rule out malignancy.

Ophthalmology

ENT

Throat

Torus Palatinus - These patient presents with a chronic growth on his ***hard palate*** which is a benign bony growth (i.e .. exostosis) located on the midline suture of the hard palate. It is thought to be due to both ***genetic*** and ***environmental factors*** and is more common in younger patients, women, and Asians. Preceding trauma ***does not*** appear to be associated with TP. Although a TP is usually <2 cm in size, it can increase in size throughout a person's life.

Patients with TP usually say that the lesion has been present for some time and deny tenderness. The thin epithelium overlying the bony growth tends to ulcerate with normal trauma of the oral cavity and heal slowly due to a poor vascular supply. Surgery is indicated for patients in whom the mass becomes ***symptomatic, interferes with speech or eating*** or causes problems with fitting of dentures later in life.

Ludwig angina is a rapidly progressive bilateral cellulitis of the submandibular and sublingual spaces. The infection classically arises from an **infected second or third mandibular molar**; the organisms that typically cause this process are *Streptococcus* and anaerobes. Patients present with fever, dysphagia, odynophagia and drooling. These symptoms result from swelling of the submandibular space and **posterior displacement of the tongue**. Physical examination reveals firm induration of the submandibular space; the presence of anaerobes may cause crepitus due to gas formation. **The most common cause of death is asphyxiation**. Patients should be monitored for respiratory difficulty and **intubated if necessary**. Antibiotics and **removal of the infected tooth is the treatment of choice**.

Nose -

Rhinoplasty or, a **nose job**, is a plastic surgery procedure for correcting and reconstructing the form, restoring the functions, and aesthetically enhancing the nose, by resolving nasal trauma (blunt, penetrating, blast) congenital defect, respiratory impediment, or a failed primary rhinoplasty.

Complications are common following rhinoplasty, and up to one in four rhinoplasties may need revision. Common complications include **patient dissatisfaction, nasal obstruction** and **epistaxis**. Those that involve the nasal septum are less common but more serious. The septum is made up of cartilage and has **poor blood supply** contrasting sharply with the rich anastomosing blood supply of the nasal sidewall. The underlying cartilage relies completely on the overlying mucosa for nourishment by diffusion. Because of the poor regenerating capacity of the septal cartilage, trauma or surgery on the septum may result in **septal perforation**. The typical postoperative presentation is a **whistling noise** heard during respiration. Following nasal surgery, septal perforation is typically the result of a septal hematoma though a septal abscess may also be the cause. Additional conditions that can cause septal perforation are self-inflicted trauma (nose picking), syphilis, tuberculosis, intranasal cocaine use, sarcoidosis and granulomatosis with polyangiitis (Wegener's).

Nasal furunculosis results from staphylococcal folliculitis following nose picking or **nasal hair**

plucking. It is potentially life threatening as it can spread to the cavernous sinus. Patients complain of pain, tenderness and erythema in the nasal vestibule.

Nasal polyps are usually seen in patients with **asthma** and **allergic disorders** but may also occur in patients with other inflammatory conditions

of the nasal mucosa. They may cause chronic nasal obstruction and should be surgically removed in symptomatic patients.

Foreign bodies are common in children. On presentation, patients will have **nasal obstruction** and may have **a foul odor, halitosis** and **nasal bleeding**. Following surgery, a retained foreign body such as nasal packing most classically would cause toxic shock syndrome.

Nasopharyngeal carcinoma (NPC) - It is an undifferentiated carcinoma of squamous cell origin seen with higher frequency in people of Mediterranean or Far Eastern descent. These tumors are usually asymptomatic until disease is advanced; most NPC is metastatic at the time of diagnosis. Patients with NPC often present with recurrent otitis media (resulting from eustachian tube obstruction by tumor), Recurrent epistaxis, and/or nasal obstruction.

Undifferentiated NPC is strongly associated with positive serologies for **Epstein-Barr virus (EBV)**. The association is so strong that EBV titer levels may be used to track the progress of therapy for this malignancy. This cancer is also associated with smoking and with chronic nitrosamine consumption (as in diets rich in salted fish).

Neurosurgery

Anterior cord syndrome is commonly associated with **burst fracture of the vertebra** and is characterized by total loss of motor function below the level of lesion with loss of pain and temperature on both sides below the lesion. **MRI is the best investigation** to study the extent of neurological damage.

Central cord syndrome is characterized by burning pain and paralysis in upper extremities with relative **sparing of lower extremities**.

The central cord syndrome classically occurs with **hyperextension injuries in elderly patients** with **degenerative changes** in the cervical spine. Such a traumatic injury causes selective damage to the central portion of the anterior spinal cord, specifically the central portions of the corticospinal tracts and the decussating fibers of the lateral spinothalamic tract. Central cord syndrome is characterized by weakness that is more pronounced in the upper extremities than in the lower extremities. This unique motor deficit occurs because the motor fibers serving the arms are nearer to the central part of the corticospinal tract. Rarely, a patient may also have a selective loss of pain and temperature sensation in the arms due to damage to the spinothalamic tract.

Spinal Cord Ischemia with possible infarction is a rare complication of vascular surgery. The spinal cord derives its blood supply from the anterior spinal artery (ASA) and two posterior spinal arteries, which originate from the vertebral artery. The ASA has several small arteries feeding into it that originate from radicular arteries in the vertebral artery, intercostal arteries and sometimes the aorta. The thoracic spinal cord is particularly dependent on these radicular arteries and the most vulnerable to infarction. The artery of Adamkiewicz is the most prominent thoracic radicular artery and arises from the aorta to supply the ASA in the T9-T12 region.

Surgery to repair thoracic and thoracoabdominal aortic aneurysms is the most common cause of spinal cord ischemia and infarction.

Risk factors for infarction include systemic hypotension in the perioperative period, increased spinal canal pressure, and aortic cross clamping or occlusion that reduces flow through the radicular arteries and the artery of Adamkiewicz. These patients typically abruptly present with ASA spinal artery syndrome consisting of flaccid paralysis, ***bowel/bladder dysfunction***, sexual dysfunction, possible hypotension and loss of tendon reflexes.

Spasticity and hyperreflexia develop over the subsequent days to weeks. ***Vibratory and proprioceptive sensation is preserved*** because posterior circulation of the spinal cord is not affected. This patient needs emergent ***MRI for diagnosis*** and treatment with supportive care and lumbar drains to reduce spinal pressure.

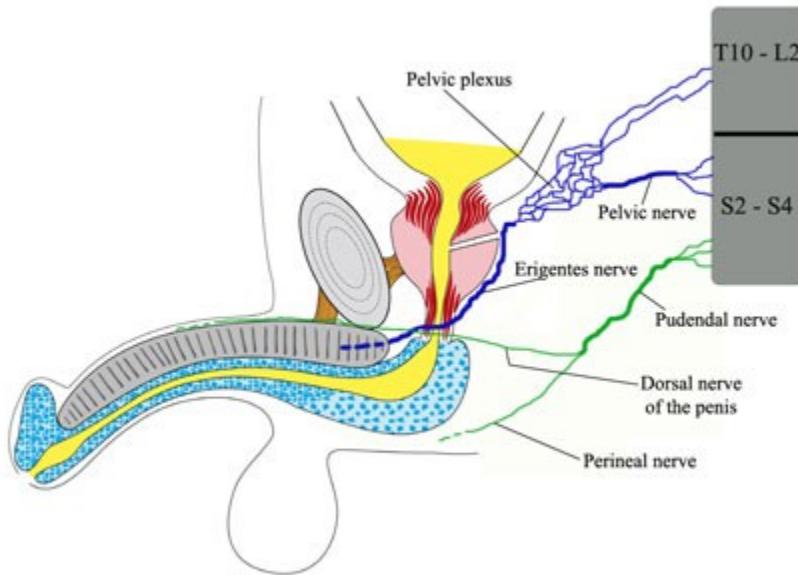
Urology

Urethral Injuries - Urethral injuries can be classified broadly based on anatomic location.

Injury to the anterior urethra, the portion of the urethra distal to the urogenital diaphragm, most commonly results from blunt trauma to the perineum (straddle injuries) or instrumentation of the urethra. Findings include perineal tenderness or hematoma, ***a normal prostate***, and bleeding from the urethra. Patients may not complain of inability to urinate, and delayed presentations may be complicated by sepsis secondary to extravasation of urine into the scrotum, perineum and/or abdominal wall.

The posterior urethra consists of the prostatic and membranous urethra. Posterior urethral injuries are commonly associated with fractures of the pelvis. Patients classically complain of suprapubic pain and an inability to

void following major trauma. Examination will show **blood at the urethral meatus, a high-riding prostate** due to displacement of the prostate by a pelvic hematoma, and scrotal hematoma, in addition to signs and symptoms of pelvic fracture.



When a urethral injury is suspected clinically, the urethra should be assessed with a **retrograde urethrogram** prior to insertion of a Foley catheter. The procedure can be used both to determine whether damage to the urethra has occurred and to determine the location of such damage within the urethra. Blind insertion of a Foley catheter is contraindicated in patients suspected of having a urethral injury because it can cause infection of a periurethral hematoma and can increase the severity of the urethral tear.

Patients with ureteral stones may suffer **paralytic ileus**. The ileus goes away once the stones pass out. This ileus is possibly due to a vagal reaction due to ureteral colic.

Bladder Dome Injury Abdominal pain that refers to the shoulder suggests an intraabdominal pathology that is causing peritonitis and irritation of the diaphragm (Kehr sign). Pain sensation resulting from irritation of the parietal peritoneum covering the undersurface of either hemidiaphragm can be referred to the ipsilateral shoulder because the phrenic nerve originates from the C3 through C5 spinal levels, which also mediate sensation for the shoulder region.

In the setting of blunt abdominal trauma, **hemoperitoneum** or **spillage of bowel contents, bile, pancreatic secretions** or **urine can cause an acute chemical peritonitis..**

Intraperitoneal rupture of the bladder occurs in blunt trauma to a **full, distended bladder**. The **dome of the bladder** is the only region covered by peritoneum; thus, it is the only injury that would permit leakage of urine into the peritoneum. Additionally, the dome of the bladder has a developmental hiatus where the urachus originates during embryonic life. Since the dome is attenuated in this region, it is also the segment of the bladder wall which is most susceptible to rupture caused by sudden increases in intravesical pressure.

Any possible blunt traumatic injury to bladder neck or anterior bladder wall or any part of the urethra **would not**, by itself, cause peritonitis. Injury to any of these structures is classically associated with severe trauma and pelvic fracture. The most common site of extraperitoneal bladder rupture is the bladder neck.

Penile Fracture - The penis, due to its mobility in the flaccid state, is typically not injured in cases of blunt trauma. It is far more vulnerable to trauma in its erect state, such as during sexual intercourse, and this is the setting where penile fracture is most commonly encountered. This injury most often occurs during intercourse where the woman is situated on top of the man because the penis may emerge from the vagina in this position and be subsequently injured in a bending fashion between its rigid fixation on the male and the downwardly moving female perineum.

Patients typically complain of a snapping sensation and / or sound when the injury occurs followed by severe pain. The injury and associated snapping sensation results from tearing of the tunica albuginea, which invests the corpus cavernosum. A hematoma rapidly forms at the site of injury causing bending of the shaft of the penis at the site of the fracture. Treatment is with an **emergent urethrogram to assess for urethral injury followed by emergent surgery** to evacuate the hematoma and mend the torn tunica albuginea.

A hydrocele is a fluid collection within the processus or tunica vaginalis -the peritoneal projection that accompanies the testis during its descent into the scrotum. When the processus vaginalis fails to obliterate, peritoneal fluid may accumulate within the processus vaginalis causing a **communicating hydrocele**. A collection of fluid within a tunica vaginalis that has properly obliterated its communication with the peritoneum is a noncommunicating hydrocele. Hydrocele can be differentiated from other testicular masses by

transillumination; a hydrocele will transilluminate while other masses will not.

Most hydroceles, both communicating and noncommunicating, will resolve spontaneously by the age of 12 months and can be safely observed during that period.

Communicating hydroceles **that persist beyond 12 months** of age are unlikely to resolve spontaneously and put the patient at increased risk of indirect inguinal hernia. Surgical intervention is indicated in such cases.

A Varicocele is a **tortuous dilation of the pampiniform plexus of veins** surrounding the spermatic cord and testis in the scrotum. A varicocele results from **incompetence of the valves of the testicular vein** and occurs most frequently on the left side, possibly because the left testicular vein enters the left renal vein inferiorly at a right angle thereby predisposing to impaired drainage. Patients are commonly asymptomatic. Those who do complain of symptoms may endorse a "**dull**" or "**dragging**" **discomfort** of the scrotum that is worse when standing. On examination, the affected side of the scrotum will feel similar to a "bag of worms" and the **Valsalva maneuver** **will typically cause the mass to enlarge**.

Breast -

Nursing Mastitis - Typically mastitis in this setting results from transmission of a bacterial organism from the infant's nasopharynx to a fissure on the mother's nipple or areola. Examination shows **a hard, red, tender and swollen area on her right breast**. There is **no** fluctuance noted. The most commonly isolated organism is *S. aureus*. **Treatment is with analgesics, antibiotics and continued nursing**. The most commonly used antibiotics are dicloxacillin or cephalosporins. Continued nursing from the affected breast has been shown to decrease the progression of mastitis to breast abscess and should be recommended in all cases.

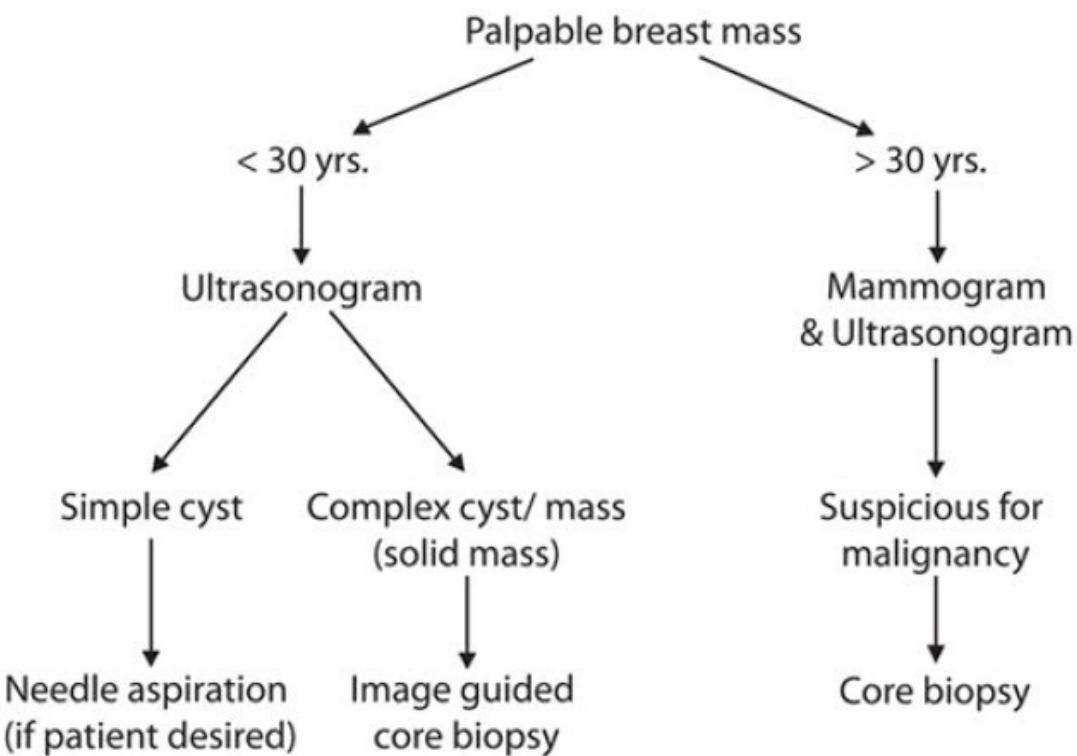
Incision and drainage would be indicated for an abscess.

Intraductal Papilloma, a form of benign breast disease that is most common in perimenopausal women. The classic presentation is **intermittent bloody discharge from one nipple**. Most intraductal papillomas are situated beneath the areola, and are difficult to palpate on physical exam due to their small size (no larger than 2 mm) and soft consistency.

Ultrasound is best at detecting masses greater than 1 cm in diameter; therefore, it is no surprise that the ultrasound finding might be normal if the size is less than 1 cm.

Palpable breast mass

In patients under the age of 30, ultrasound is typically the first imaging modality utilized for a palpable abnormality although mammography can still be used for further characterization if an abnormality is seen on ultrasound. Women over the age of 30 are usually evaluated with both mammography and ultrasound; typically the mammogram is performed first to help in targeting the abnormal location for the ultrasound.

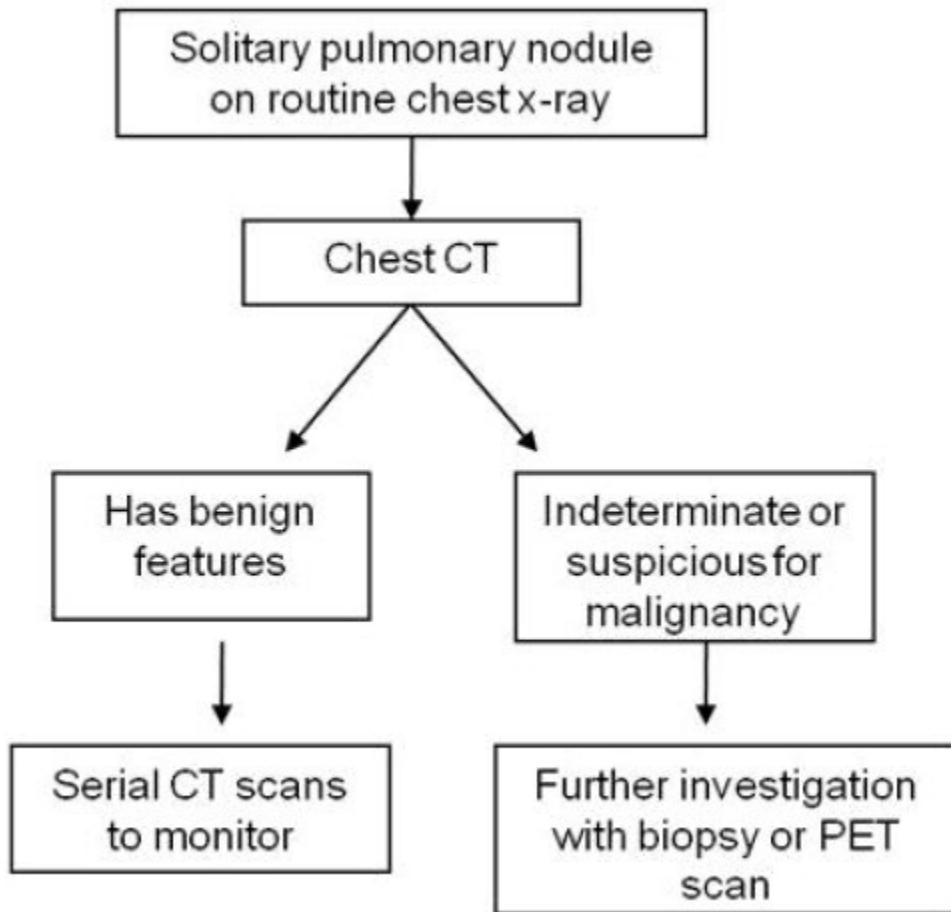


Inflammatory Breast Cancer - Classically, inflammatory breast carcinoma presents as a brawny edematous cutaneous plaque with a "**peau d'orange**" (orange peel) appearance **overlying a breast mass**. As this is an aggressive tumor, most patients also present with **axillary lymphadenopathy**, and one quarter of patients are subsequently found to have metastatic disease. Another sign that indicates the possibility of breast cancer in this patient is spontaneous nipple discharge. Nipple discharge in a non-lactating woman should always raise suspicion for breast cancer,

especially if spontaneous, unilateral, localized to a single duct, occurs in a patient over 40 years old, is bloody or is associated with a mass. Clinically, inflammatory breast cancer cannot be differentiated from an infectious process, such as a breast abscess, with 100% certainty. Therefore, a **biopsy for histology should be done first** to exclude or confirm that diagnosis.

Lungs

A solitary pulmonary nodule (SPN) is defined as a discrete lesion <3 cm in diameter incidentally discovered on chest x-ray that is completely surrounded by lung parenchyma and does not contact the pleura, hilum, or mediastinum. There must not be any associated pleural effusion, adenopathy, or atelectasis. The main causes of SPN are shown in a table. The main goals of following SPNs are to promptly detect and surgically resect malignancies, and to leave benign lesions intact. The initial history, physical examination, and chest x-ray should provide some clues regarding the etiology of the SPN. Malignancy is more likely in patients aged ~50 years, patients with a history of smoking, and lesions > 2 cm with **irregular and spiculated borders**.



CT scan is more sensitive than chest x-ray to identify these features, and also can detect other small nodules which may represent metastasis. If the CT scan findings are suspicious for malignancy, biopsy followed by an excision should be performed. Lesions that appear less likely to be malignant on CT scans can be followed with serial CT scans for a period of 2 years to monitor for growth.

SPN - 1st Previous X ray

Next - CT

Next Biopsy

Solitary Pulmonary Nodule |

Primary lung cancer	Squamous cell, adenocarcinoma, small cell, large cell, and carcinoid
Metastatic cancer	Melanoma, breast, head and neck, renal cell, colon, germ cell, and sarcoma
Benign infectious granulomas	Tuberculosis, histoplasmosis, atypical mycobacteria, coccidiomycosis, <i>Cryptococcus</i> , and blastomycosis
Benign neoplasms	Lipoma, hamartoma, and fibroma
Vascular	Arteriovenous malformations

Organ Transplantation