

CHAPTER 1

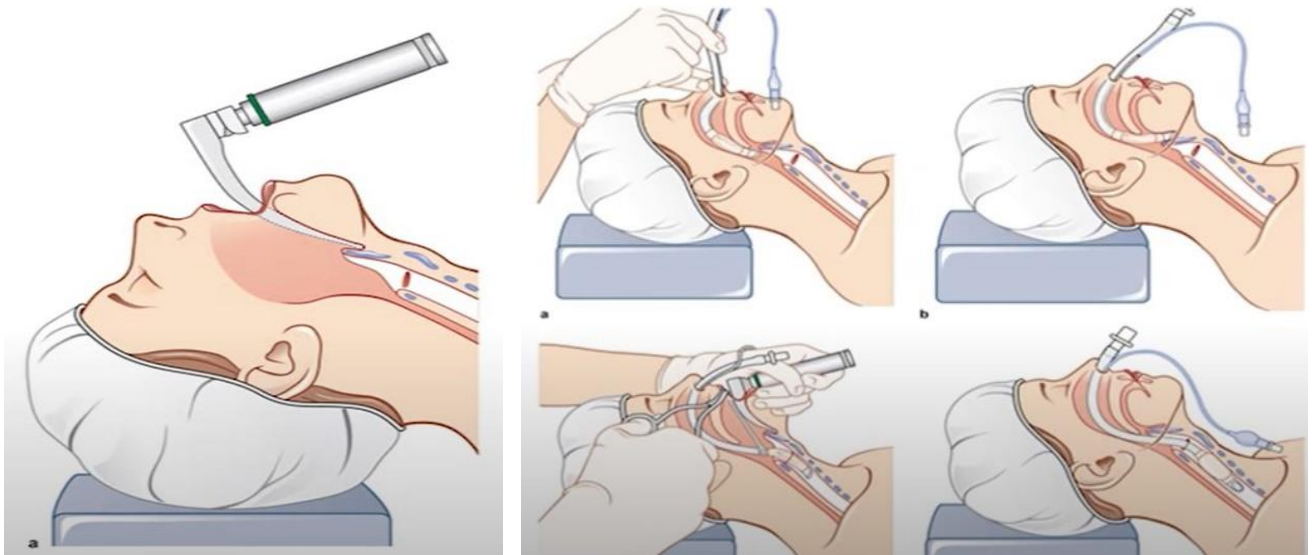
Trauma

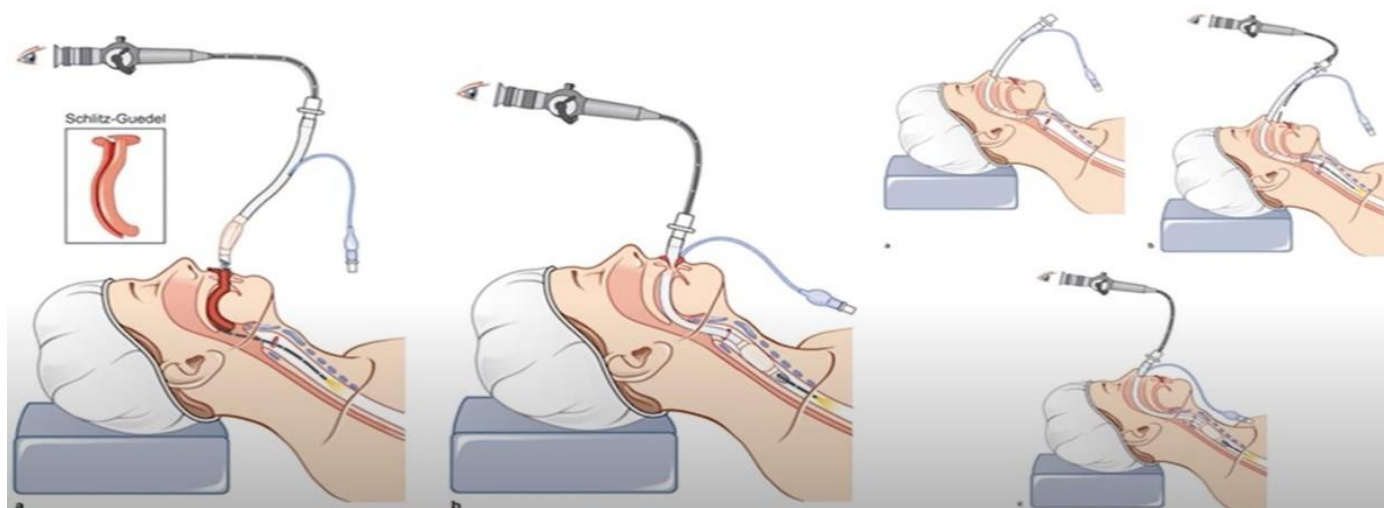
Trauma

Primary survey (the ABCs)

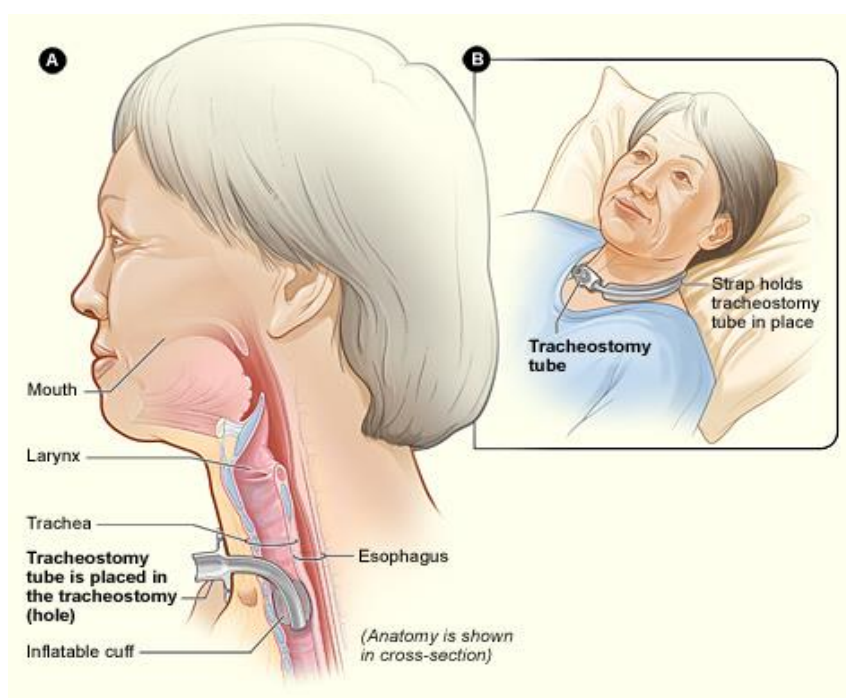
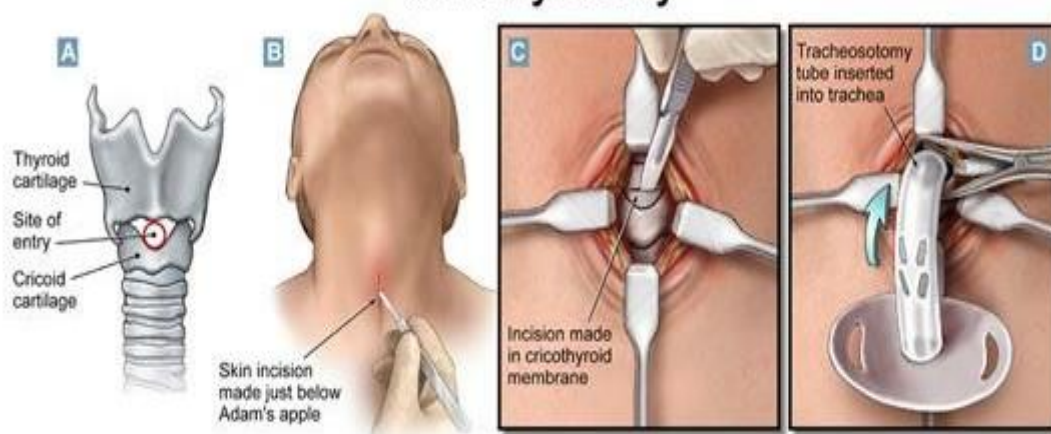
A. **Airway:**

- The first step in the evaluation of trauma is airway assessment and protection:
 - An airway is considered **protected** if the patient is **conscious and speaking in a normal tone of voice**.
 - An airway is considered **unprotected** if there is an **expanding hematoma or subcutaneous emphysema in the neck, noisy or “gurgly” breathing, or a Glasgow Coma Scale <8**.
- An airway should be secured before the situation becomes critical:
 1. **In the field:** an airway can be secured by **intubation or cricothyroidotomy**.
 2. In the **emergency** department: it is best done by **rapid sequence induction and orotracheal intubation**, with monitoring of pulse oximetry.
 3. In the presence of a **cervical spine injury**:
 - **Orotracheal intubation can still be done as long as the head is secured and in-line stabilization is maintained during the procedure.**
 - Another option in that setting is **nasotracheal intubation over a fiberoptic bronchoscope**.
 4. **If severe maxillofacial injuries** preclude the use of intubation or intubation is unsuccessful, **cricothyroidotomy may become necessary**.
 5. In the pediatric patient population (**age <12**): **Tracheostomy is preferred over cricothyroidotomy due to the high risk of airway stenosis (subglottic stenosis), as the cricoid is much smaller than in the adult.**



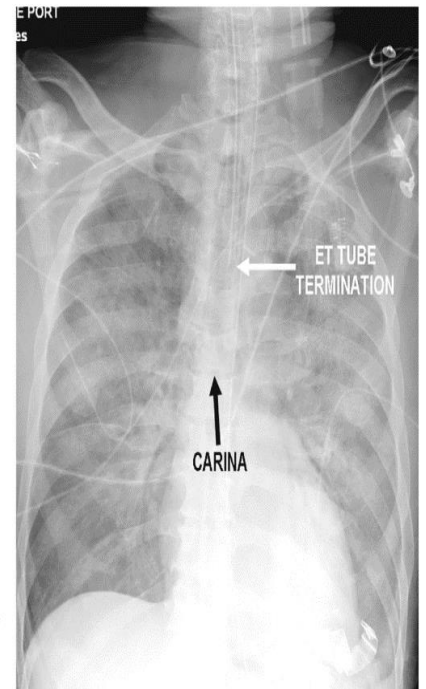
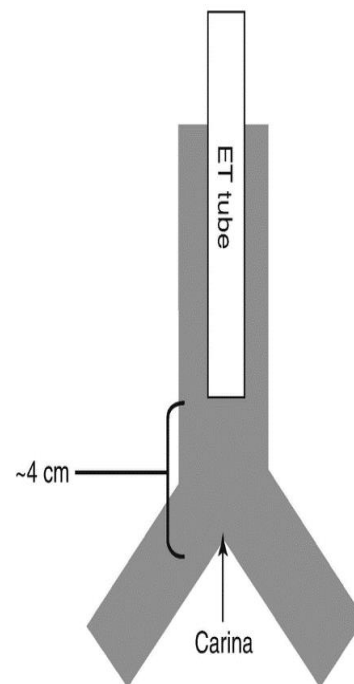
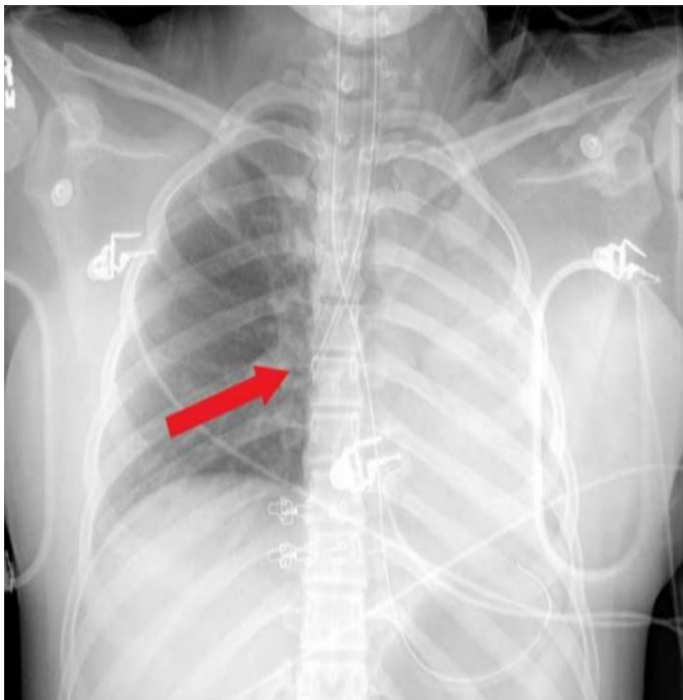


Cricothyrotomy



❖ N.B:

1. Right mainstem bronchus intubation is a relatively common complication of endotracheal intubation.
 - It causes **asymmetric chest expansion during inspiration and markedly decreased or absent breath sounds on the left side on auscultation**.
 - **The ideal location of the distal tip of the endotracheal tube (ETT) is 2-6 cm above the carina.**
 - Because the right mainstem bronchus diverges from the trachea at a relatively **non-acute angle**, an ETT advanced too far will preferentially enter into the right main bronchus.
 - This results in **overinflation of the right lung, underventilation of the left lung, and asymmetric chest expansion**. Auscultation will show markedly decreased or absent breath sounds. **Chest x-ray confirms the diagnosis.**
 - **Repositioning the endotracheal tube by pulling back slightly will move the tip between the carina and vocal cords and solve the problem.**

2. Management of cervical spine trauma:

- **The first step** in evaluating this type of patient in the field is to **stabilize the cervical spine and spinal column** with a backboard, rigid cervical collar, and lateral head supports until a spinal injury is excluded.
- **The next step is to assess the airway.** Unstable lesions above the third cervical vertebra level can cause immediate paralysis, and **lower cervical lesions can damage the phrenic nerve.**
- **Orotracheal intubation with rapid-sequence intubation is the preferred way to establish an airway unless there is significant facial trauma.**

Management of cervical spine trauma	
Prehospital	<ul style="list-style-type: none"> • Spinal immobilization (eg, backboard, rigid cervical collar, lateral head supports) • Careful helmet removal (eg, motorcycle helmet) • Airway oxygenation
Emergency department	<ul style="list-style-type: none"> • Orotracheal intubation preferred unless significant facial trauma present • Rapid-sequence intubation added for unconscious patients who are breathing but need ventilatory support • In-line cervical stabilization suggested unless it interferes with intubation • CT of entire cervical spine • Monitoring for neurogenic shock from spinal cord injury

B. Breathing:

- Breath sounds indicate satisfactory ventilation:
- Absence or decrease of breath sounds may indicate a **pneumothorax and/or hemothorax and necessitate chest tube placement.**
- Pulse oximetry indicates satisfactory oxygenation:
- Hypoxia may be secondary to airway compromise, pulmonary contusion, or neurological injury impairing respiratory drive and necessitate intubation.

❖ N.B:

- Positive pressure mechanical ventilation causes an acute increase in intrathoracic pressure, which, in a severely hypovolemic patient with low central venous pressure, can **collapse venous capacitance vessels (inferior vena cava) and cut off venous return.**
- **This sudden loss of right ventricular preload can cause acute circulatory failure and sudden cardiac death (SCD).**
- In addition, **sedatives** used prior to intubation cause **relaxation of venous capacitance vessels and can also contribute to decreased venous return.**

C. **Circulation:**

- Clinical signs of shock include the following:
 - Low BP (<90 mm Hg systolic).
 - Tachycardia (heart rate >100 bpm).
 - Low urinary output (<0.5 ml/kg/h).
- Patients in shock will be pale, cold, shivering, sweating, thirsty, and apprehensive.
- In the trauma setting:
 - In the trauma setting, shock is either hypovolemic (secondary to hemorrhage and the most common scenario) or cardiogenic (secondary to pericardial tamponade or tension pneumothorax due to chest trauma).
 - Hemorrhagic shock tends to cause collapsed neck veins due to low central venous pressure (CVP), while cardiogenic shock tends to cause elevated CVP with jugular venous distention. Both processes may occur simultaneously.
 - In pericardial tamponade, there is typically no respiratory distress, while in tension pneumothorax there is significant dyspnea, loss of unilateral breath sounds, and tracheal deviation.
- Treatment of hemorrhagic shock:
 - Treatment of hemorrhagic shock includes volume resuscitation and control of bleeding, in the OR or ED depending on the injury and available resources.
 - Volume resuscitation is initially with 2L of Lactated Ringer's solution unless blood products are immediately available.
 - In the setting of trauma, transfusion of blood products should be in a 1:1:1 ratio between packed RBCs, fresh frozen plasma, and platelets. Resuscitation should be continued until BP and heart rate normalize and urine output reaches 0.5-1.0 ml/kg/hr.
 - The preferred route of fluid resuscitation in the trauma setting is 2 large bore peripheral IV lines.
 - If this cannot be obtained, percutaneous subclavian or femoral vein catheters should be inserted.
 - In children age <6, intraosseous cannulation of the proximal tibia or femur is the alternate route.
- Pericardial tamponade:
 - Cardiac tamponade occurs acutely in trauma because of bleeding into a stiff pericardium that has no elasticity.
 - Cardiac tamponade is classified as acute or subacute based on the rate and volume of pericardial fluid accumulation.

A. **Acute cardiac tamponade:**

- 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 → obstructive shock with tachycardia and hypotension as well as muffled heart sounds (Beck's triad).
- 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.
- Pericardial tamponade is generally a clinical diagnosis and can be confirmed with U/S.
- 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. Fluid and blood administration while evacuation is being set up is helpful to maintain an adequate cardiac output.

B. **Subacute cardiac tamponade:**

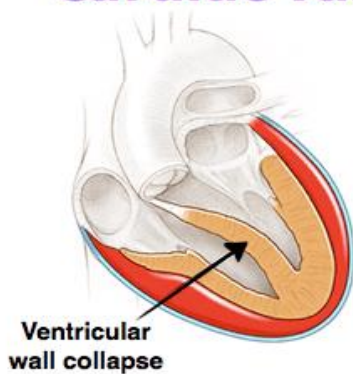
- In contrast, chronic processes (malignancy or renal failure) cause slow accumulation of pericardial fluid that gradually increases the intrapericardial pressure and allows the pericardial elasticity to adapt slowly (subacute cardiac tamponade). As a result, it may take 1-2 liters of fluid before the 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.

Characteristics of cardiac tamponade		
	Acute	Subacute
Rapidity of fluid accumulation	Minutes to hours	Days to weeks
Clinical signs	Beck triad: hypotension, JVD, muffled heart sounds Pulsus paradoxus (>10 mm Hg decrease in SBP with inspiration)	
Effusion volume	100-200 mL	1-2 L
Chest x-ray findings	Normal cardiac silhouette	Enlarged, globular cardiac silhouette



JVD = jugular venous distension; SBP = systolic blood pressure.

Cardiac Tamponade



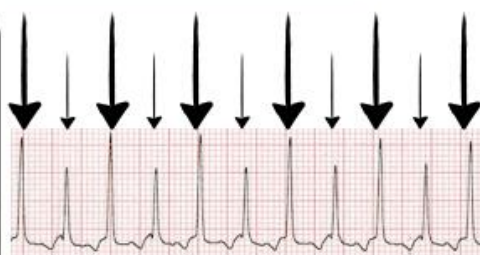
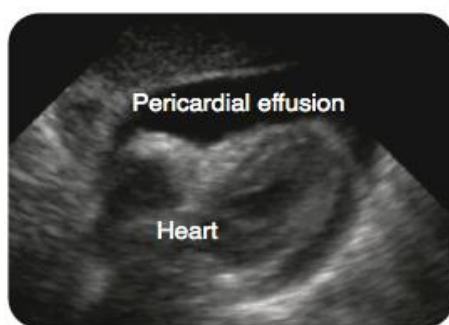
Beck's Triad

- 1 Hypotension
- 2 Jugular venous distension
- 3 Muffled heart sounds

Don't mix up with:

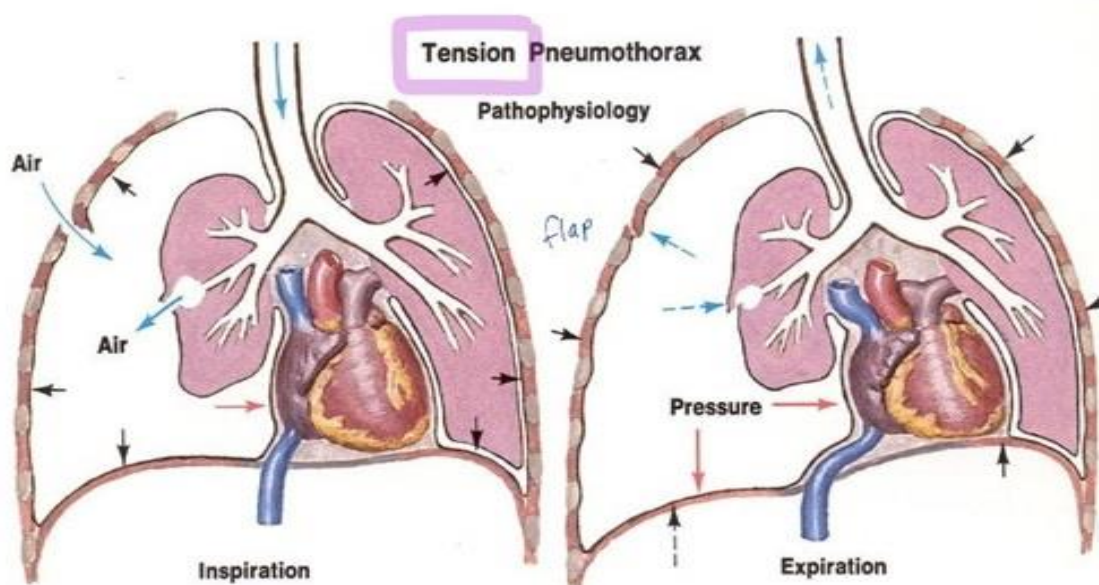
Tension pneumothorax

1. Hypotension
2. Jugular venous distension
3. Absent breath sounds



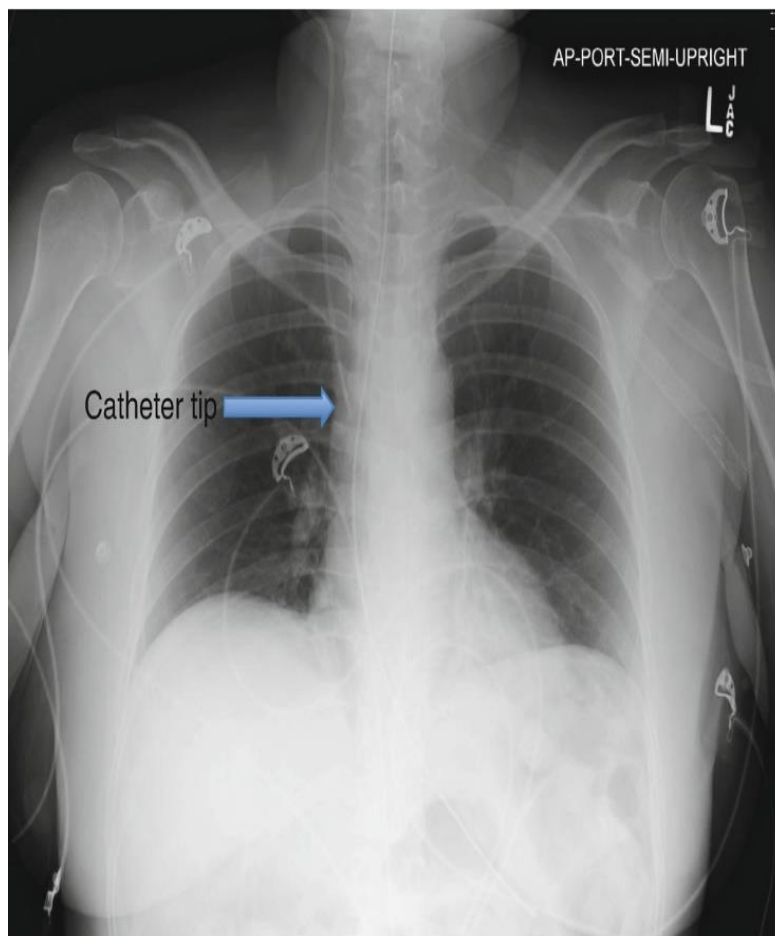
Electrical Alternans

- Tension pneumothorax:
- Tension pneumothorax is a clinical diagnosis based on physical exam.
- Management requires **immediate decompression of the pleural space**, initially with a **large-bore needle** which converts the tension to a simple pneumothorax and followed by chest tube placement.



❖ N.B:

- Central venous catheter (CVC) is commonly used for the administration of critical care medications (pressors, hypertonic saline) and in the setting of difficult vascular access or need for long-term medication (chemotherapy).
- The preferred points of central venous access are the internal jugular vein (typically by ultrasound guidance) or the subclavian vein (typically by anatomic landmark guidance).
- CVC complications occur in up to 15% of cases, and many are due to inappropriate catheter placement.
- The ideal placement of a CVC tip is in the lower superior vena cava. Tip placement in smaller veins (subclavian, jugular, azygous) predisposes to venous perforation.
- In addition, inappropriately placed catheter tips may cause lung puncture, leading to pneumothorax, or myocardial perforation, leading to pericardial tamponade. Arterial (subclavian) puncture is also a risk, particularly in the absence of ultrasound guidance.
- Confirmatory chest x-ray may be omitted in the setting of an uncomplicated (first needle insertion, no resistance to catheter advancement) ultrasound-guided CVC placement.
- Otherwise, a portable chest x-ray should be performed immediately following CVC placement to facilitate timely recognition of a misplaced catheter tip, identify possible injury (pneumothorax), and prevent exacerbation of injury (infusion of fluid into the pericardial space).
- Visualization of the catheter tip just proximal to the angle between the trachea and right mainstem bronchus confirms appropriate placement.

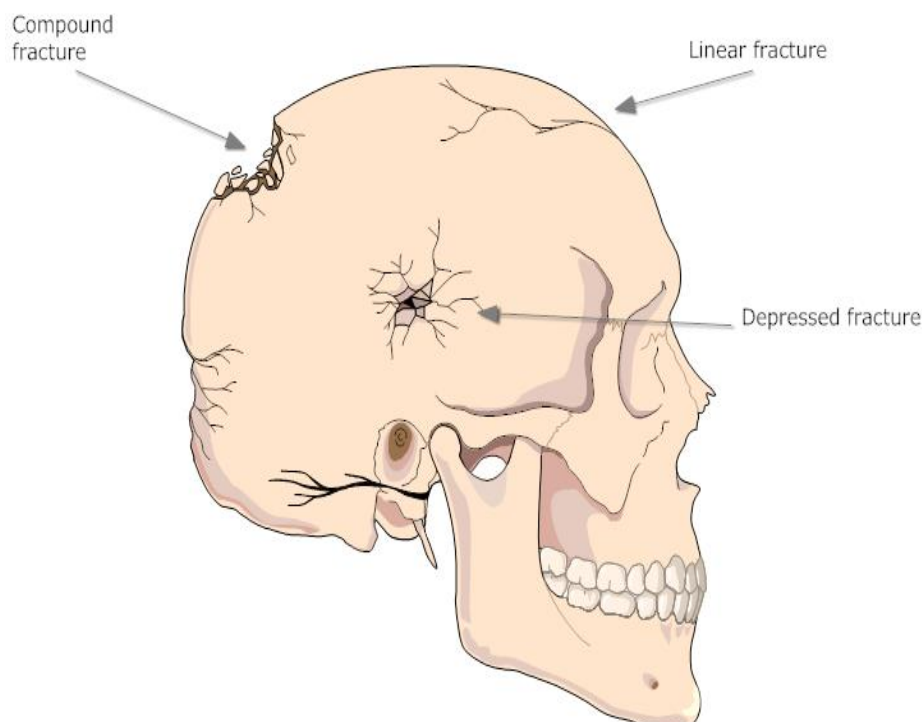


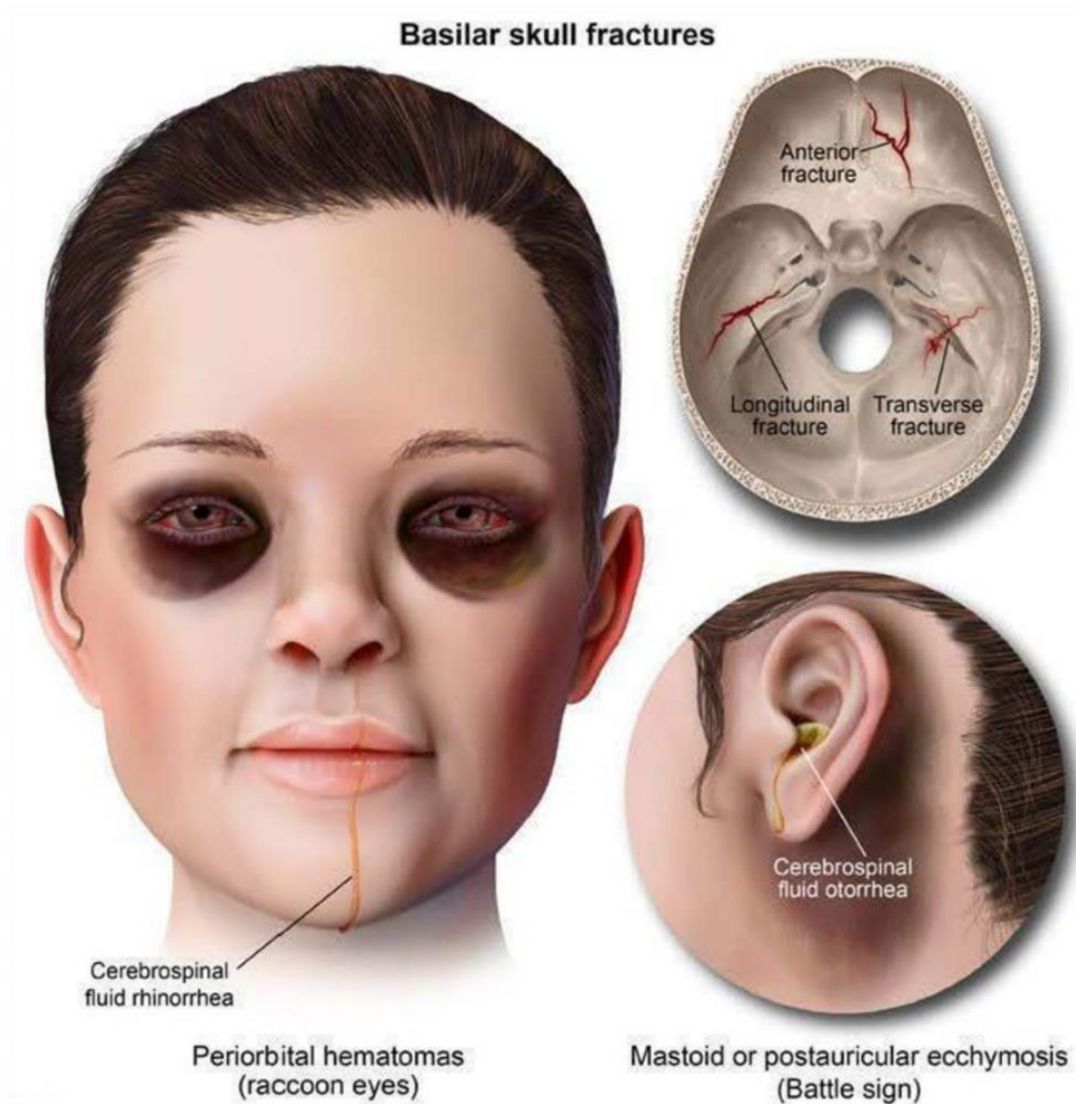
Secondary Survey (A review from head to toe)

- After the ABC's have been evaluated and any immediate life-threatening emergencies addressed, trauma evaluation continues with the secondary survey which is composed of a **complete physical exam to evaluate for occult injuries followed by chest x-ray and pelvic x-ray.**

A. Head Trauma:

- **Linear skull fractures are left alone if they are closed** (no overlying wound).
- **Open fractures** require **wound closure**. If comminuted or depressed, treat in the OR to **evaluate for any damage to the underlying brain tissue.**
- **Penetrating** head trauma as a rule **requires surgical intervention and repair of the damage.**
- **Anyone with head trauma who has become unconscious gets a CT scan to look for intracranial hematomas** → If negative and neurologically intact, they can go home if the family will awaken them frequently during the next 24 hours to make sure they are not going into coma.
- Signs of a fracture affecting the base of the skull include **raccoon eyes, rhinorrhea, otorrhea or ecchymosis behind the ear (Battle's sign)** → CT scan of the head is required to rule out intracranial bleeding and should be extended to include the neck to evaluate for a cervical spinal injury → **Expectant management is the rule and antibiotics are not usually indicated.**



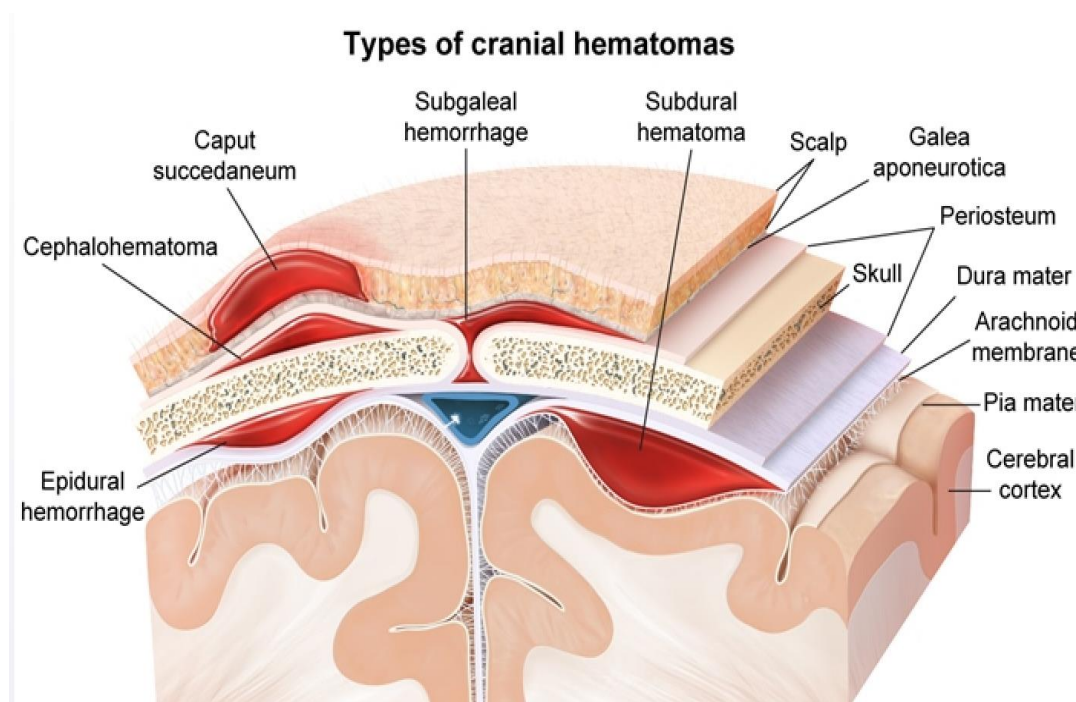


Clinical manifestations of basilar skull fractures

- Hematoma of the mastoid process or periauricular hematomas (Battle's sign)
- Bilateral peri-orbital hematomas (raccoon eyes)
- Hemotympanum
- Cerebrospinal fluid otorrhea
- Cranial nerve palsies (resulting in anosmia, vertigo, tinnitus, or hearing loss)

▪ Neurologic damage from trauma can be caused by 3 components:

1. **Initial blow.**
 2. Subsequent development of a **hematoma** that displaces the midline structures.
 3. Later development of **increased intracranial pressure (ICP)** due to cerebral edema.
- There is no treatment for the first (other than prevention), surgery can relieve the second, and medical measures can prevent or minimize the third.

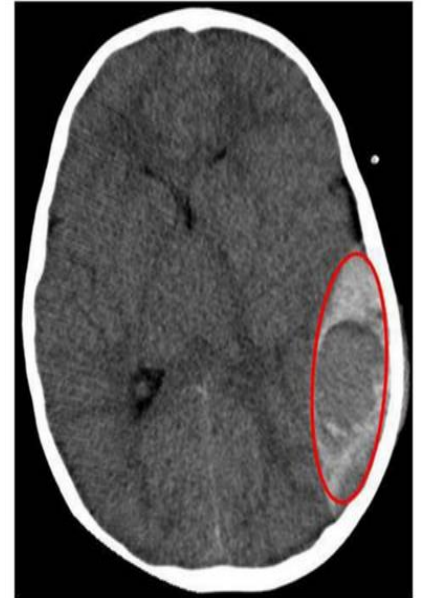


▪ Acute epidural hematoma:

- Acute epidural hematoma (EDH) is a condition resulting from **accumulation of blood in the potential space between the cranium and dura mater.**
- It most commonly occurs following traumatic injury to the sphenoid bone, resulting in **laceration of the middle meningeal artery** (higher arterial pressure can rapidly expand the hematoma and compress the temporal lobe).
- It has a **classic sequence of trauma, unconsciousness, a lucid interval (a completely asymptomatic patient who returns to his previous activity), gradual lapsing into coma again, fixed dilated pupil (90% of the time on the side of the hematoma), and contralateral hemiparesis with decerebrate posturing.**
- As the hematoma rapidly expands, deterioration of consciousness and symptoms of elevated intracranial pressure (nausea/vomiting, headache) arise.

- CT scan shows a **biconvex, lens-shaped hematoma**.
- **Symptomatic patients require emergent neurosurgical hematoma evacuation.**

Epidural hematoma	
Pathogenesis	Trauma to sphenoid bone with tearing of middle meningeal artery
Clinical features	<ul style="list-style-type: none"> • Brief loss of consciousness followed by lucid interval • Hematoma expansion leads to: <ul style="list-style-type: none"> ○ ↑ intracranial pressure (impaired consciousness, headache, nausea/vomiting) ○ Uncal herniation (ipsilateral CN III palsy & hemiparesis)
Diagnosis	Head CT: Biconvex (lens-shaped) hyperdensity that does not cross suture lines
Treatment	Urgent surgical evacuation for symptomatic patients



- Acute subdural hematoma:
- Acute subdural hematoma (SDH) occurs when traumatic shearing forces cause **tearing of the bridging veins**, leading to **slow** bleeding into the subdural space.
- Risk factors include **advanced age and chronic alcoholism** (due to brain atrophy and the bridging veins must traverse a longer distance, thereby becoming more susceptible to tears), as well as **anticoagulant use**.
- Acute subdural hematoma has the same sequence, but **the force of the trauma is typically much larger, and the patient is usually much sicker** (not fully awake and asymptomatic at any point), due to more severe neurologic damage.
- Symptoms of SDH develop **gradually** as they occur secondary to the rupture of low-pressure vessels. Acute SDH typically produces symptoms **1-2 days after onset**, whereas chronic SDH presents insidiously, **weeks after the initial injury**.
- Non-contrast head CT scan typically shows a **crescent-shaped hyperdensity** (red arrow) that crosses suture lines.
- **Small** hematomas are managed **conservatively** whereas **larger ones** causing neurologic deficits require **urgent surgical evacuation**.

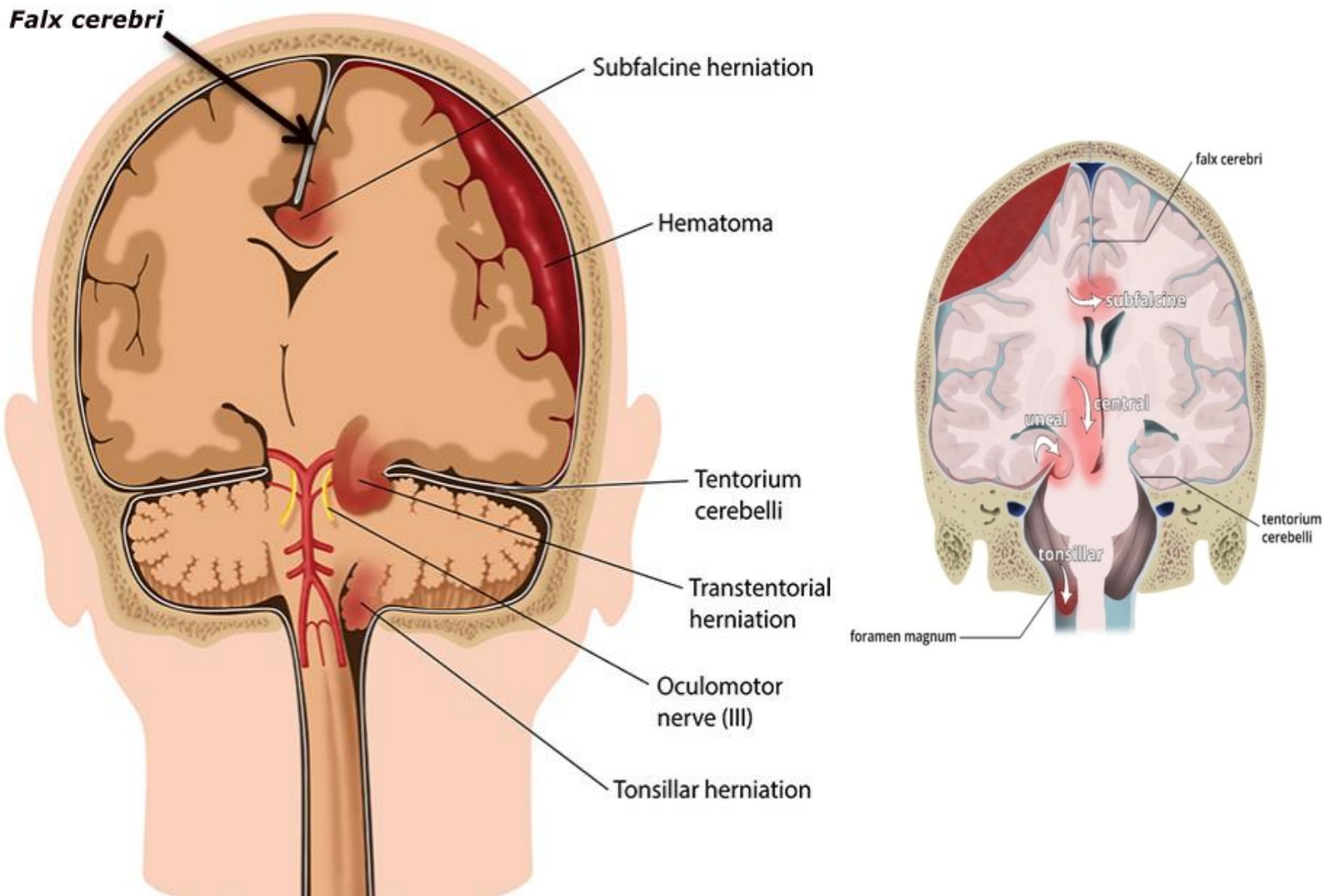
Subdural hematoma	
Pathogenesis	Rupture of bridging veins (head trauma)
Risk factors	<ul style="list-style-type: none"> • Elderly & alcoholics (cerebral atrophy, ↑ fall risk) • Infants (thin-walled vessels) • Anticoagulant use
Clinical features	<ul style="list-style-type: none"> • Acute: Gradual onset 1-2 days after injury <ul style="list-style-type: none"> ◦ Impaired consciousness (eg, coma), confusion ◦ Headache, nausea, & vomiting (↑ intracranial pressure) • Chronic: Insidious onset weeks after injury <ul style="list-style-type: none"> ◦ Headache, somnolence, confusion, lightheadedness ◦ Focal neurologic deficits
Diagnosis	Head CT: Crescent shaped hyperdensity (acute) or hypodensity (chronic) crossing suture lines
Treatment	<ul style="list-style-type: none"> • Reverse/discontinue anticoagulants • Surgical evacuation of symptomatic or large bleeds



- Chronic subdural hematoma:
 - Chronic subdural hematoma occurs in the **very old or in severe alcoholics**.
 - **Elderly patients are at higher risk for subdural hematoma due to increased fall risk and cerebral atrophy.**
 - Chronic subdural hematoma often **presents insidiously weeks after the initial injury** with headache, somnolence, confusion, and focal neurologic deficits.
 - Over several days or weeks, mental function deteriorates as hematoma forms.
 - CT scan is diagnostic, and surgical evacuation provides a dramatic cure.
- **Hypovolemic shock cannot happen from intracranial bleeding?! there isn't enough space inside the head for the amount of blood loss needed to produce shock. Look for another source**

Herniation syndromes

Major herniations of the brain



- The cranial vault is limited by the rigid cranial bones and **divided into compartments by the dural folds (falx cerebri and tentorium cerebelli)**.
- There is no room for brain expansion in the event of a brain edema, tumor, or hemorrhage. As a result, portions of the brain can protrude through the openings in the falx cerebri or through the foramen magnum. This process is called **herniation**.
- **Emergent craniotomy should be performed in patients with focal neurologic deficits to prevent brain herniation and death.**

1. **Cingulate (subfalcine) herniation under falx cerebri:**
 - The cingulate gyrus herniates under the falx cerebri, potentially **compressing the anterior cerebral artery (ischemic stroke)**.
2. **Downward transtentorial (central) herniation:**
 - Caudal displacement of brain stem → rupture of paramedian basilar artery branches → **Duret hemorrhages**.
 - Usually **fatal**.
3. **Transtentorial (Uncal) herniation:**
 - Occurs when **the innermost part of the temporal lobe (uncus) herniates through the gap between the crus cerebri and the tentorium**.
 - The most common cause of transtentorial herniation is an **ipsilateral mass lesion** (brain tumor, subdural or epidural hematoma, and intracerebral hemorrhage). This mass causes an increase in supratentorial pressure on the side of the lesion → forces the ipsilateral uncus to herniate through the gap between the crus cerebri and the tentorium (uncal herniation)
 - As a result, the following structures may become compressed:
 - A. **Ipsilateral oculomotor nerve (CN III) compression:**
 - Down and out position of the ipsilateral eye.
 - Dilated pupil.
 - Ptosis.
 - B. **Ipsilateral posterior cerebral artery compression:**
 - Contralateral homonymous hemianopia with macular sparing.
 - C. Contralateral cerebral peduncle compression against the tentorium may occur → contralateral corticocerebral tract lesion → **ipsilateral hemiparesis** (ipsilateral to the side of herniation).
 - D. Brain stem hemorrhage (Duret hemorrhage) may occur in the pons and midbrain **due to stretching and rupture of basilar artery, which is usually fatal**.
4. **Cerebellar tonsillar herniation into the foramen magnum:**
 - The cerebellar tonsils displace through the foramen magnum and compress the medulla → **cardiorespiratory arrest may occur**.

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

▪ Intervention for lowering Intracranial pressure:

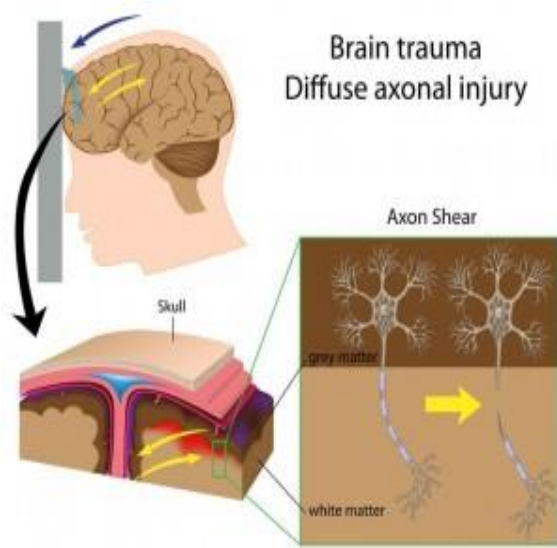
- Because the brain is an encased organ, relatively small changes in pressure can alter the intracranial pressure in important ways.
- The pressures in 3 distinct compartments of the brain: **the brain parenchyma, the cerebrospinal fluid (CSF), and cerebral blood flow (CBF) determine the overall intracranial pressure (ICP).**
- Both brain parenchyma and CSF have relatively constant pressures except in certain circumstances, **including space-occupying lesions and obstructions to CSF flow.**
- CBF is influenced by systemic blood pressure and is held constant over a fairly wide range of pressures by **cerebrovascular autoregulation**. However, the partial pressures of O₂ and CO₂ also play an important role in regulating CBF. **In conditions such as stroke or trauma, these systems are disrupted and interventions may be required to lower ICP.**
- CBF is an important target for therapy, and PaCO₂ is a potent regulator of CBF (much greater than pO₂). As levels of cerebral PaCO₂ rise, so does blood flow. **Lowering cerebral arterial PaCO₂ through hyperventilation results in rapid vasoconstriction and a consequent decrease in ICP.**

- Other interventions to lower ICP do so by lowering systemic pressures and **reducing metabolic demand** (sedation, Moderate hypothermia), or **increasing venous outflow** (head elevation), **reducing brain parenchyma water content/volume** (mannitol), or **reducing the volume of CSF** (therapeutic lumbar punctures).
- However, do not diurese the patient to the point of lowering systemic arterial pressure.

Cerebral perfusion pressure (CPP) = mean arterial pressure (MAP) - intracranial pressure (ICP)

Interventions for lowering ICP	
Intervention	Mechanism
Head elevation	Increased venous outflow from the brain
Sedation	Decreased metabolic demand & control of hypertension
Intravenous mannitol	Extraction of free water from brain tissue → osmotic diuresis
Hyperventilation	CO ₂ washout → cerebral vasoconstriction
Removal of CSF	Reduction of CSF volume/pressure

- Diffuse axonal injury:
- 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.













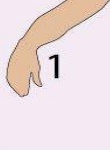


❖ N.B:

1. Glasgow coma scale (GCS):

- All trauma patients should be triaged using the Glasgow coma scale (GCS), which **can predict the severity and prognosis of coma, during the primary survey.**
- Maximum score 15 points** (full consciousness); **minimum score 3 points** (coma or death).
- Mild head injury: GCS score 13-15, Moderate head injury: GCS score 9-12, Severe head injury: GCS score ≤ 8 (Indication for endotracheal intubation)

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

Head Trauma - Glasgow Coma Scale					
Eye Opening					
Spontaneous	To speech	To pain	None		
<p>4</p> 	<p>3</p> 	<p>2</p> 	<p>1</p> 		
Verbal Response					
Oriented	Confused conversation	Inappropriate words	Incomprehensible sounds	None	
<p>5</p> 	<p>4</p> 	<p>3</p> <p>?@*%&! </p>	<p>2</p> <p>???????? </p>	<p>1</p> 	
<p>Best motor response - When there is right/left or upper/lower asymmetry use the best motor response to calculate the score as this is the best predictor of outcome.</p>					
Obeys commands	Localizes pain with purposeful movement	Flexion withdrawal to pain	Abnormal flexion (decorticate)	Extension (decorticate)	None (flaccid)
<p>6</p> 	<p>5</p> 	<p>4</p> 	<p>3</p> 	<p>2</p> 	<p>1</p> 

2. Traumatic brain injury can be categorized as mild, moderate, severe traumatic brain injury (TBI).

	GCS	LOSS of CONSCIOUSNESS	DURATION of AMNESIA	LEVEL of CONSCIOUSNESS
MILD	13 - 15	< 30 minutes	< 1 day	< 1 day
MODERATE	9 - 12	30 min - 24 hours	1 - 7 days	> 24 hours
SEVERE	≤ 8	>24 hours	>7 days	> 24 hours

*** IF GCS <8, TIME TO INTUBATE ***

Evaluation of head trauma in children age 2–18	
Clinical scenario	Recommendation
Any of the following: <ul style="list-style-type: none"> Focal neurologic findings Skull fracture, especially signs of basilar skull fracture Seizure Persistent altered mental status (eg, agitation, lethargy, slow response) Prolonged loss of consciousness 	Head CT without contrast
GCS = 15 with any of the following: <ul style="list-style-type: none"> Vomiting Headache Questionable or brief loss of consciousness Injury caused by high-risk mechanism of injury Severe mechanism of injury* 	Clinician preference: Observation for 4–6 hours OR Head CT without contrast
Minor head trauma (GCS = 15 with non-severe mechanism & no vomiting, headache, loss of consciousness, or signs of fracture)	No head CT

* Severe mechanism: car crash with patient ejection, passenger fatality, or rollover; pedestrian versus car; fall height >5 ft; or head hit by high-impact object

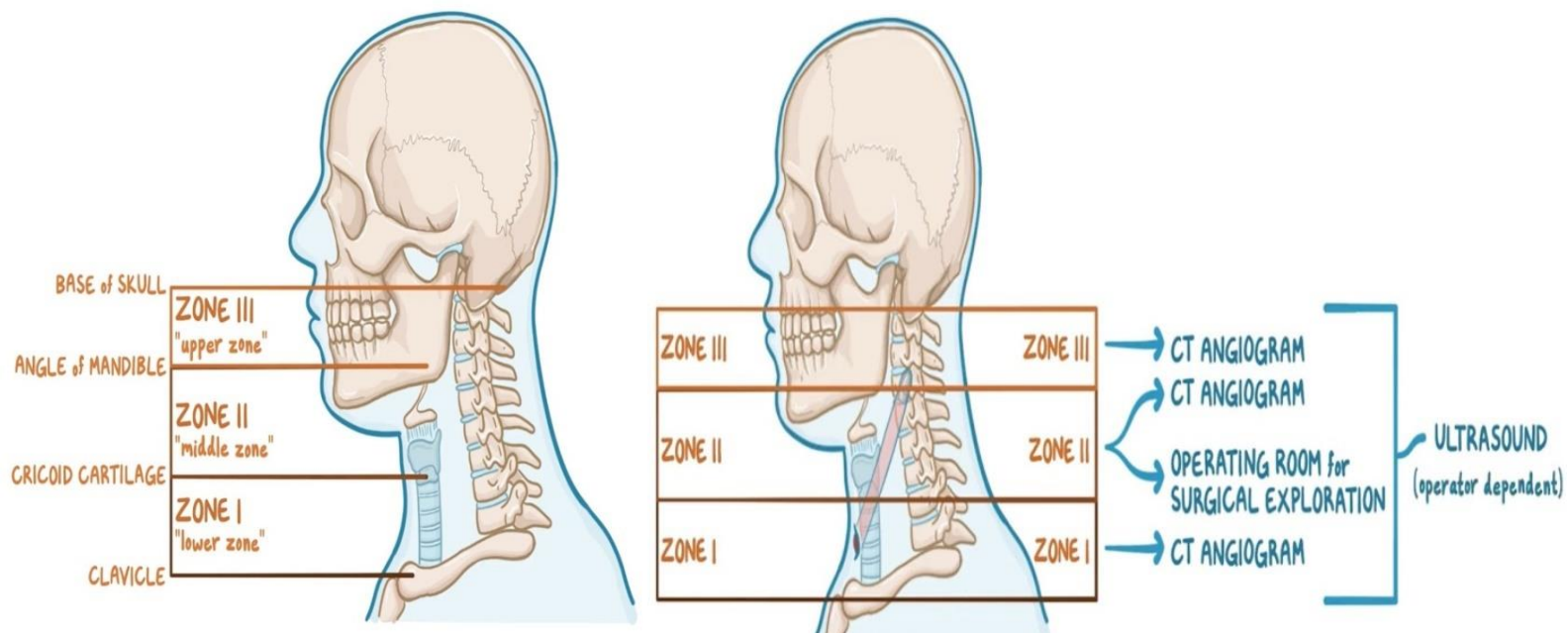
3. Post-concussive syndrome:

- Traumatic brain injury (TBI) of any severity can lead to (a few hours to days later) **post-concussive syndrome**, which is clinically described by the following constellation of symptoms: **headache, confusion, amnesia, difficulty concentrating or with multitasking, vertigo, mood alteration, sleep disturbance, and anxiety**.
- These symptoms typically resolve with **symptomatic treatment within a few weeks to months following TBI**; however, some patients may have persistent symptoms lasting >6 months.

B. Neck Trauma:

1. Penetrating trauma to the neck:

- For the purpose of evaluating penetrating neck trauma, the neck has been divided into 3 zones from caudad to cephalad:
 - Zone 1:** extends from the clavicles to the cricoid cartilage.
 - Zone 2:** from the cricoid cartilage to the angle of the mandible.
 - Zone 3:** from the angle of the mandible to the base of the skull.



- Penetrating trauma to the neck mandates surgical exploration in all cases where there is an expanding hematoma, deteriorating vital signs, or signs of esophageal or tracheal injury such as coughing or spitting up blood.
- For injuries to zone 1:
 - For injuries to zone 1, evaluate with angiography, esophagogram (water-soluble, followed by barium if negative), esophagoscopy, and bronchoscopy to help decide if surgical exploration is indicated and to determine the ideal surgical approach.
- For injuries to zone 2:
 - If the patient is stable with low index of suspicion of a significant injury, use the above diagnostic modalities to evaluate the situation and potentially avoid unnecessary surgical exploration.
 - If the patient's condition changes, however, urgent surgical exploration is indicated.

- For injuries to zone 3:

- For injuries to zone 3, **evaluate with angiography (both for diagnosis and potentially for embolization) for vascular injury.**

2. Blunt trauma to the neck:

- In all patients with severe blunt trauma to the neck, **the integrity of the cervical spine has to be ascertained.**
- Unconscious patients and conscious patients **with midline tenderness to palpation** should be evaluated initially with CT scan, and potentially followed with MRI depending on findings.

C. Spinal Cord Injury:

- Hemisection (Brown-Sequard):

- Hemisection (Brown-Sequard) is typically caused by a **clean-cut injury such as a knife blade.**
- More details in neurology section of internal medicine book.

- Anterior cord syndrome:

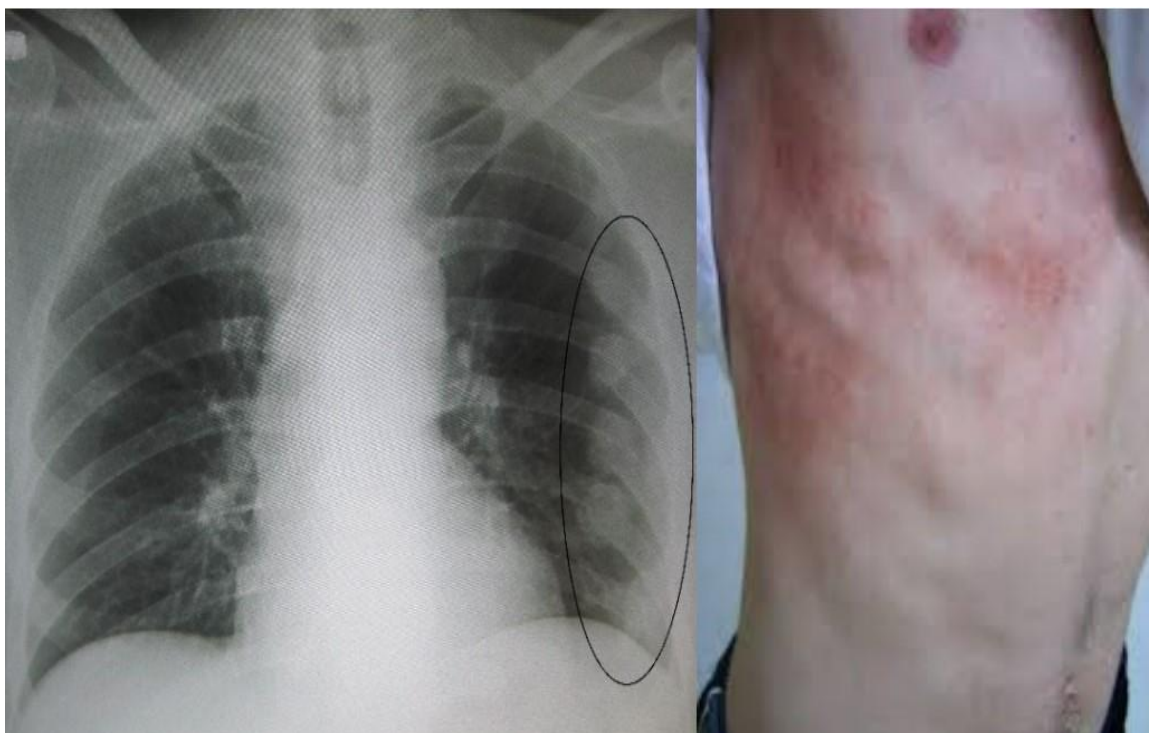
- Anterior cord syndrome is typically seen in **burst fractures of the vertebral bodies.**
- More details in neurology section of internal medicine book.

- Central cord syndrome:

- Central cord syndrome occurs in the elderly with **forced hyperextension of the neck, such as a rear-end collision.**
- More details in neurology section of internal medicine book.
- Management necessitates precise diagnosis of a cord injury, **best done with MRI.**

D. **Chest Trauma:**

- **Severe blunt trauma to the chest** may cause obvious injuries such as **rib fractures with a flail chest** or **sucking chest wound**, as well as less apparent injuries such as **pulmonary contusion**, **blunt cardiac injury**, **diaphragmatic injury**, and **aortic injury**.
- **Rib fractures:**
 - Up to half of rib fractures will not be evident on initial chest x-ray; therefore, the diagnosis **should be highly suspected in all patients with localized chest wall tenderness following trauma**.
 - Rib fractures are typically associated with **significant pain**, which causes **hypoventilation** that may lead to **atelectasis and pneumonia**.
 - **Maintaining adequate ventilation** is the main goal of rib fracture management.
 - **Pain control is essential to maintain deep breathing and adequate cough:**
 - For extensive rib fractures: managed as an inpatient, **epidural infusion is the preferred method of pain control**. Intercostal nerve blocks are also used but carry the risk of iatrogenic pneumothorax during administration.
 - Patients with less extensive rib fractures: managed with a **combination of nonsteroidal anti-inflammatory drugs (ketorolac, ibuprofen) and opioids**. Opioids have the disadvantage of central respiratory depression, but this disadvantage is typically outweighed by the benefits of adequate pain control.

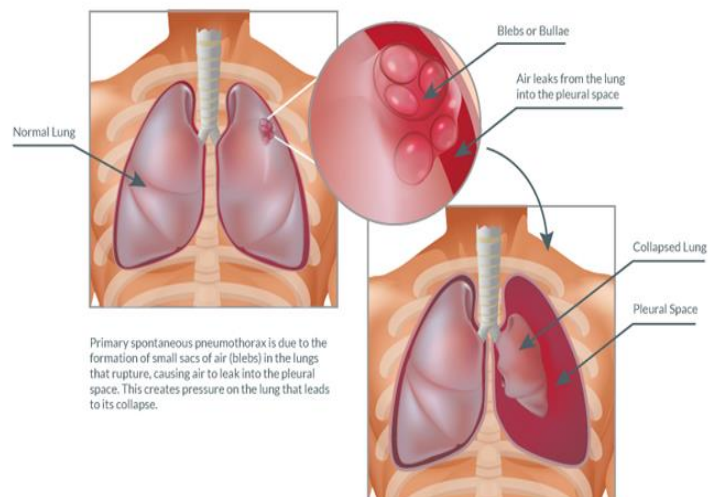
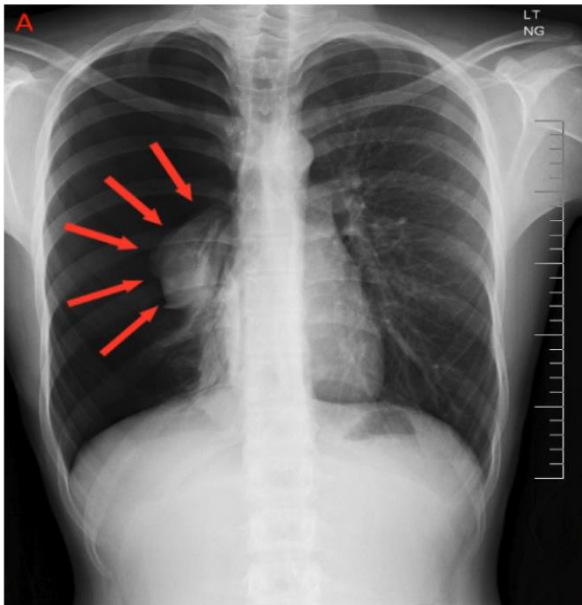


■ Pneumothorax:

- Accumulation of air in the pleural space.
- **Unilateral** chest pain and dyspnea, ↓ tactile fremitus, **hyperresonance**, diminished breath sounds, all on the affected side.

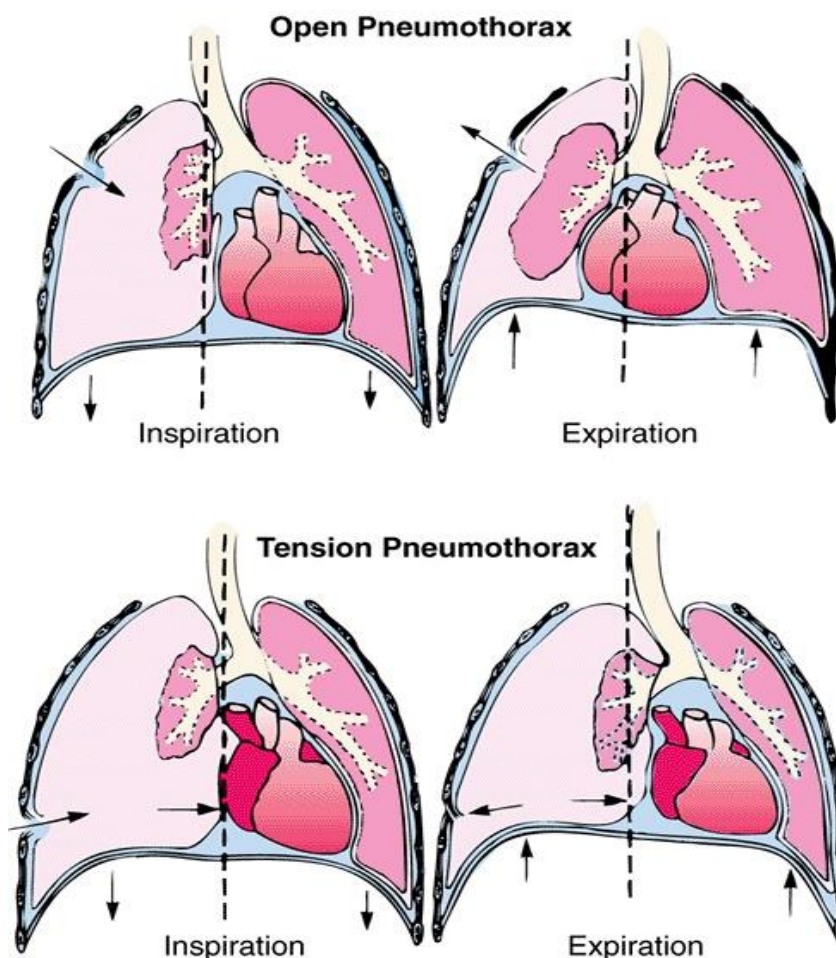
1. Primary spontaneous pneumothorax:

- Primary spontaneous pneumothorax is defined as a **pneumothorax in someone without pre-existing pulmonary disease and not caused by trauma or barotrauma**.
- **Rupture of apical subpleural blebs is the most common cause of primary spontaneous pneumothorax.**
- Occurs most frequently in **tall, thin, young males**.
- Results in collapse of a portion of the lung; **trachea shifts to the side of collapse**.



- Diagnosis is confirmed by identifying a visceral pleural line on chest x-ray with absent lung marking beyond the line.
- The management of PSP depends on the size of the lesion and the clinical status of the patient:
 - ✓ **Small pneumothoraces in clinically stable patients** can be **managed with observation and supplemental oxygen, which enhances the speed of resorption**.
 - ✓ **Large pneumothoraces in clinically stable patients** should undergo **decompression with a large-bore needle (needle thoracostomy)** inserted in the second or third intercostal space in the midclavicular line or at the fifth intercostal space in the mid or anterior axillary.

- ✓ Patients who are hemodynamically unstable should undergo emergent placement of a tube thoracostomy. If tube thoracostomy is not available or will be delayed, urgent needle decompression can be performed.
- 2. **Secondary spontaneous pneumothorax:**
 - Due to diseased lung (bullae in emphysema, COPD, cystic fibrosis), mechanical ventilation with use of high pressures → barotrauma.
 - Chronic destruction of alveolar sacs leads to the formation of large alveolar blebs, which can eventually rupture and leak air into the pleural space.
 - Diagnosis of pneumothorax is made by chest x-ray, which demonstrates a visceral pleural line beyond which no pulmonary markings are apparent.
 - Management depends on the size of the lesion and the clinical status of the patient, and ranges from observation with supplemental oxygen to emergency tube thoracotomy.
- 3. **Traumatic pneumothorax:**
 - Caused by blunt (rib fracture) or penetrating (gunshot) trauma.

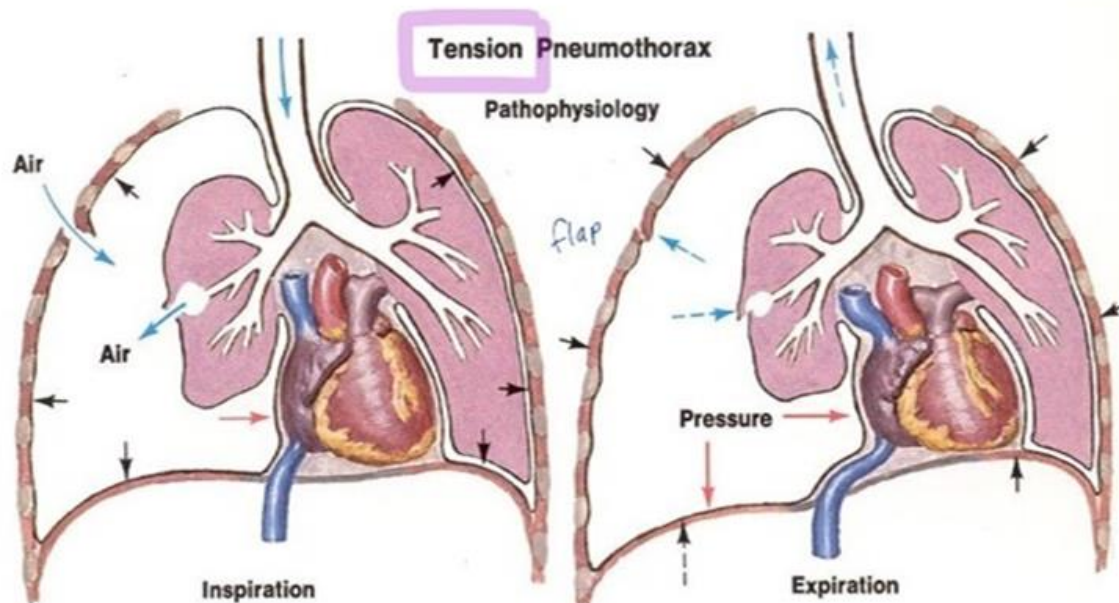


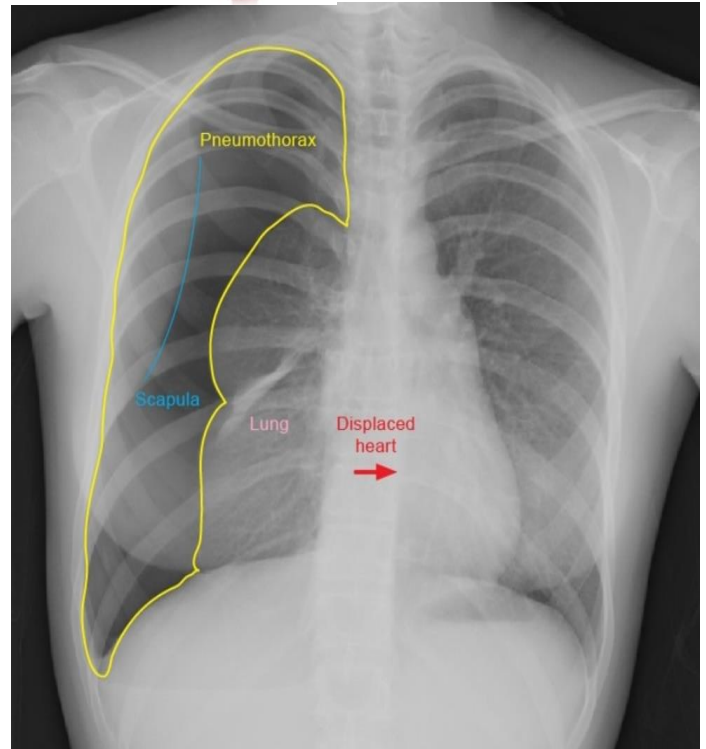
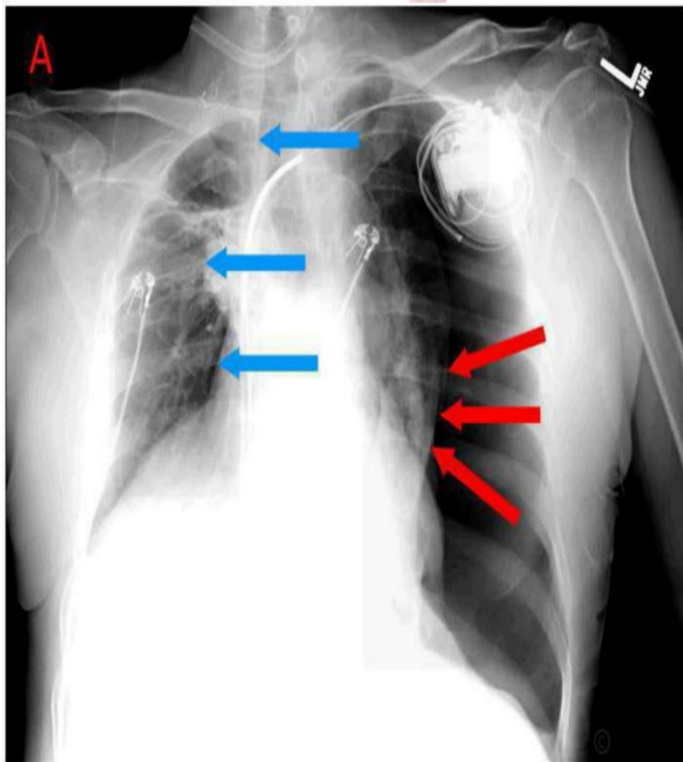
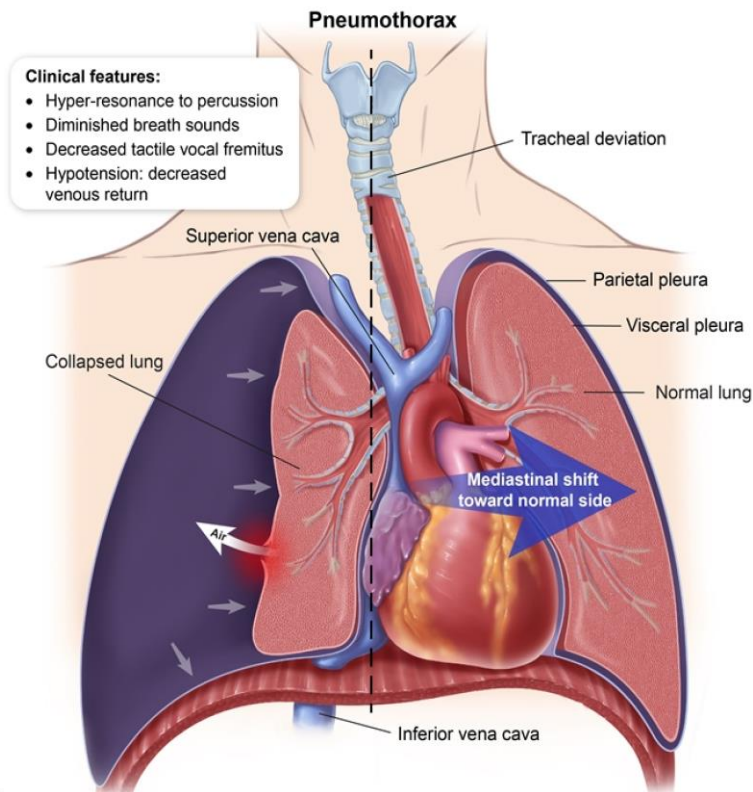
▪ Simple pneumothorax:

- Simple pneumothorax results from penetrating trauma such as a weapon or the jagged edge of a fractured rib.
- There is typically **moderate shortness of breath with absence of unilateral breath sounds and hyperresonance to percussion**.
- Diagnosis is **confirmed with chest x-ray**, and management consists of **chest tube placement**.

▪ Tension pneumothorax (TP):

- Can be any of the above.
- It develops when injured tissue forms a **one-way valve allowing air to enter the pleural space but preventing it from escaping naturally**.
- Tension pneumothorax (TP) is a **life-threatening condition** caused by air within the pleural space that **displaces mediastinal structures and compromises cardiopulmonary function**.
- TP is characterized by **rapid-onset severe shortness of breath, tachycardia, tachypnea, hypotension, and distension of the neck veins due to superior vena cava compression**.
- **TP is a clinical diagnosis, and decompression should be initiated immediately with needle thoracostomy (or, if available, direct emergency tube thoracostomy) in hemodynamically unstable patients.**
- Positive-pressure ventilation can worsen TP by increasing intrathoracic pressures and intensifying the one-way valve effect. As the pleural cavity fills with air, increased pressure is required to initiate inspiratory flow.





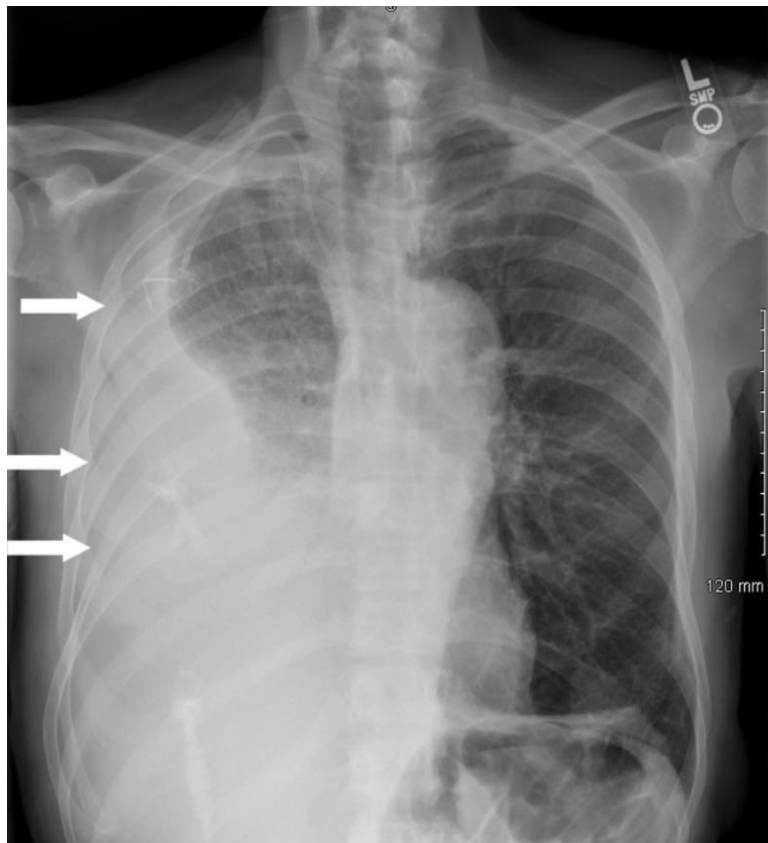
❖ N.B:

- Severe coughing paroxysms may result in **subcutaneous emphysema**, wherein air leaks from the chest wall into the subcutaneous tissues due to the high intra-alveolar pressure provoked by the cough. By a similar process, pneumothorax can occur; therefore, in such patients (those with apparent subcutaneous emphysema secondary to severe coughing paroxysms), **chest x-rays must be obtained emergency to rule out pneumothorax**.

Pneumothorax		
	Spontaneous pneumothorax	Tension pneumothorax
Associated features	<ul style="list-style-type: none"> Primary: no preceding event or lung disease; often thin, young men Secondary: underlying lung disease (eg, COPD, CF) 	<ul style="list-style-type: none"> Life-threatening Often due to trauma or mechanical ventilation
Signs & symptoms	<ul style="list-style-type: none"> Chest pain, dyspnea ↓ Breath sounds, ↓ chest movement Hyperresonant to percussion 	Same as spontaneous plus: <ul style="list-style-type: none"> Hemodynamic instability Tracheal deviation away from affected side
Imaging	<ul style="list-style-type: none"> Visceral pleural line Absent lung markings beyond pleural edge 	Same as spontaneous plus: <ul style="list-style-type: none"> Contralateral mediastinal shift Ipsilateral hemidiaphragm flattening
Management	<ul style="list-style-type: none"> Small (≤ 2 cm): observation & oxygen administration Large & stable: needle aspiration or chest tube 	<ul style="list-style-type: none"> Urgent needle decompression or chest tube placement

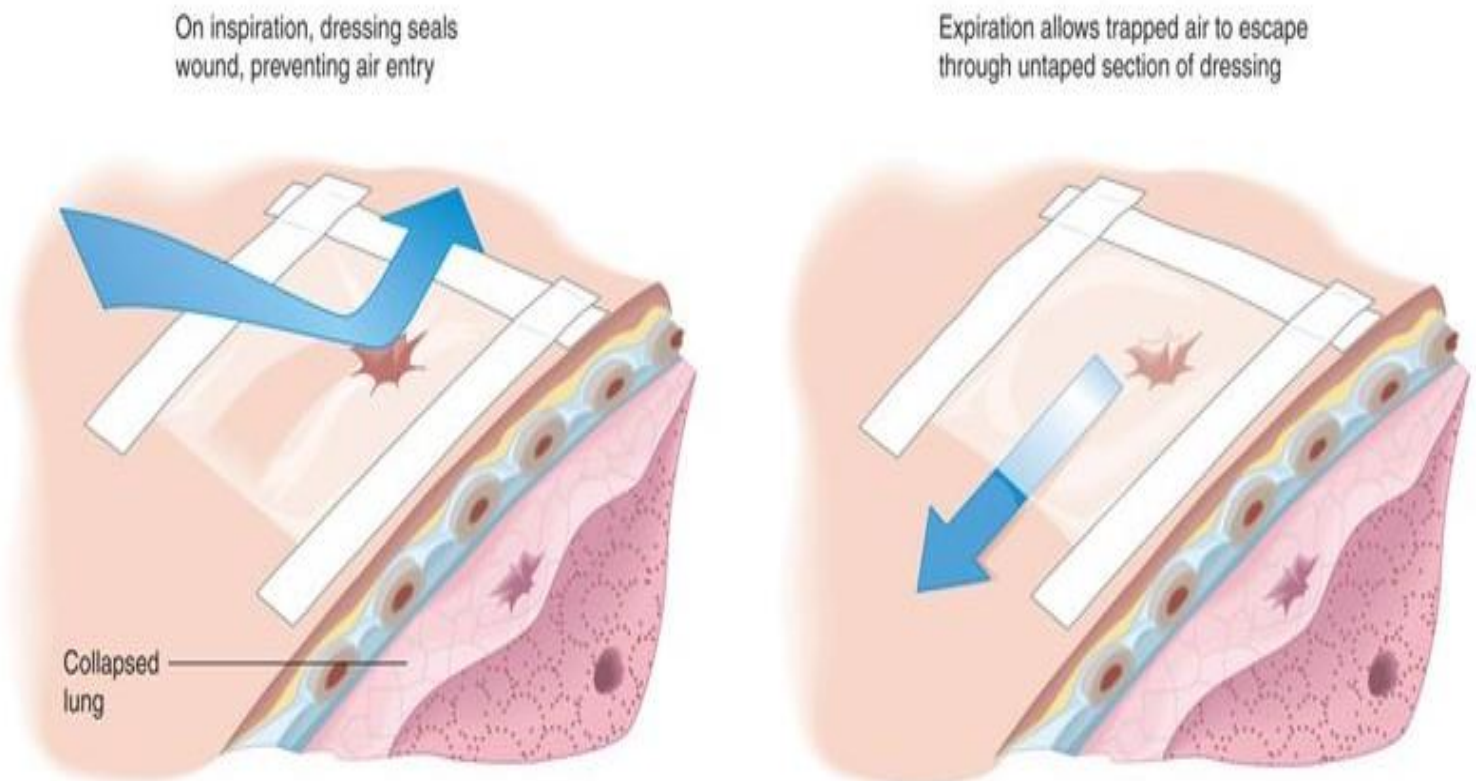
CF = cystic fibrosis; COPD = chronic obstructive pulmonary disease.

- Hemothorax:
 - Hemothorax happens the same way but the affected side will be **dull to percussion due to blood accumulation in the pleural space**.
 - The blood can originate directly from:
 - Lung parenchyma.
 - From the chest wall, such as an intercostal artery.
 - Diagnosis is **confirmed with chest x-ray**.
 - **Chest tube placement is necessary to enable evacuation of the accumulated blood to prevent late development of a fibrothorax or empyema**, but surgery to stop the bleeding is sometimes required.
 - If the lung is the source of bleeding, it usually **stops spontaneously** as it is a **low pressure system**.
 - In some cases where a systemic vessel such as an **intercostal artery** is the source of bleeding, **thoracotomy is needed to stop the hemorrhage**. Indications for thoracotomy include:
 - Evacuation of >1,500 mL when the chest tube is inserted.
 - Collecting drainage of >1 L of blood over 4 hours (250 mL/hr).
 - **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.**



▪ Sucking chest wounds:

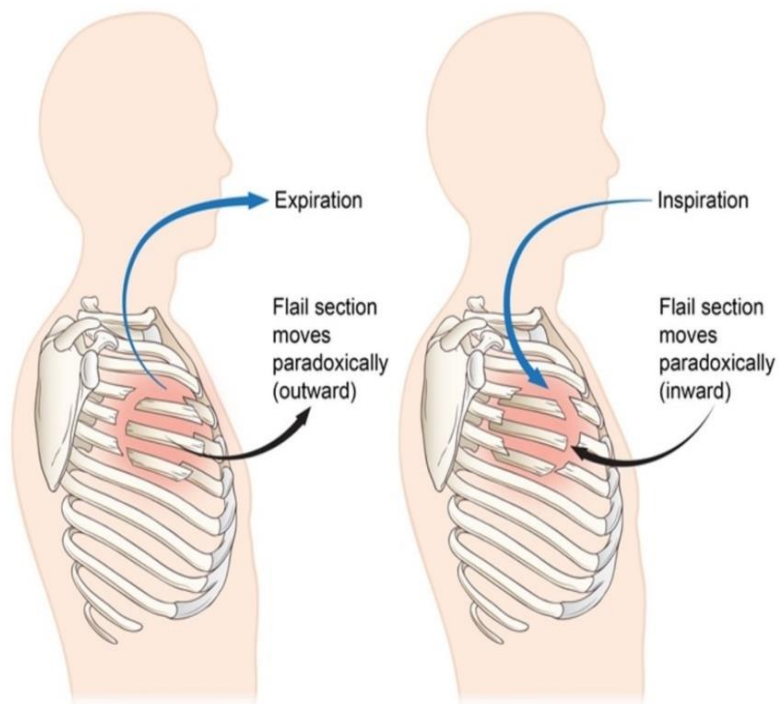
- Sucking chest wounds are obvious from physical exam, as there is a **flap that sucks air with inspiration**.
- **Untreated**, it will lead to a **deadly tension pneumothorax**.
- Initial management is with a **partially occlusive dressing secured on 3 sides, with one open side acting as a one-way valve**. This allows air to escape but not to enter the pleural cavity.



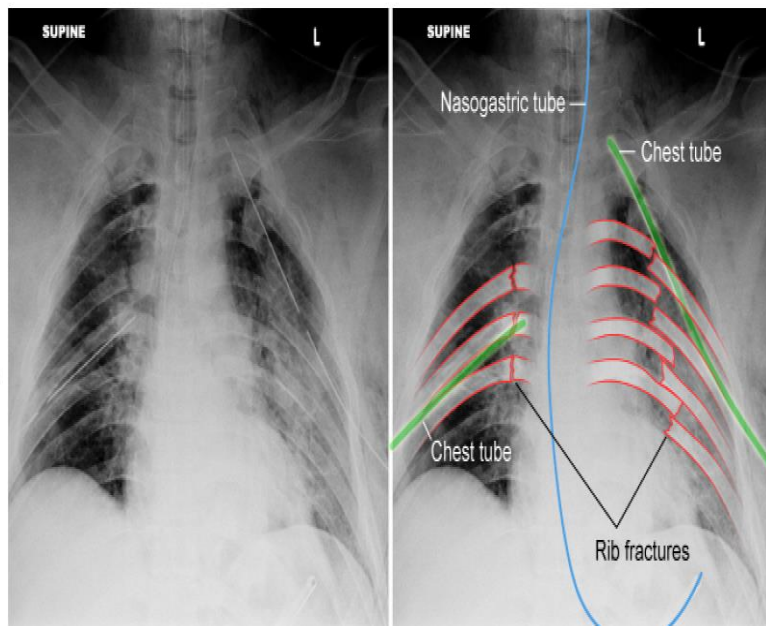
▪ Flail chest:

- Flail chest is usually caused by **blunt** thoracic trauma (steering wheel hitting the chest in a motor vehicle collision) and describes what occurs when **≥3 adjacent ribs fracture in ≥2 places**.
- The fractured portion of the rib cage (flail segment) separates from the rest of the chest wall and moves in a **paradoxical motion on respiration**.
- Due to negative intrathoracic pressure during inspiration and positive intrathoracic pressure during expiration, **the flail segment tends to retract inward during inspiration (versus moving outward with the chest wall) and bulges outward during expiration (versus retracting inward)**.

Flail chest



Flail chest



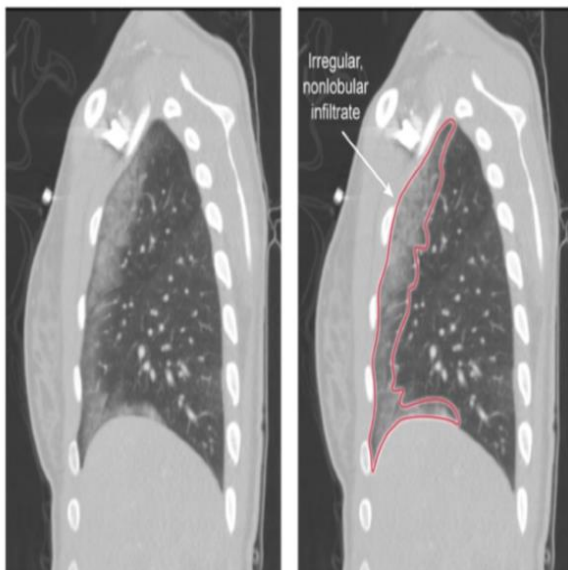
- Patients with flail chest present with **respiratory distress and tachypnea with shallow breaths**. Muscle splinting may attenuate the paradoxical motion of the flail segment and make the diagnosis difficult on physical examination alone.
- Due to the associated pain, patients with flail chest **take shallow breaths** (resulting in decreased bibasilar breath sounds) and compensate for the resulting hypoxemia with **tachypnea**.
- **Pain control and supplemental oxygen are early steps in management.**
- **Respiratory failure can occur, often due to associated pulmonary contusion and resultant collection of edema and blood in the alveoli.** Acute respiratory distress syndrome and pneumonia are other potential complications. **Intubation with mechanical positive-pressure ventilation and bilateral chest tube placement** (because lungs punctured by the broken ribs could leak air once positive pressure ventilation is started, which could lead to a tension pneumothorax) is often required.
- In addition to improving oxygenation, **mechanical positive pressure ventilation (MPPV) also corrects the paradoxical motion of the flail segment by replacing the normal negative intrapleural pressure with positive intrapleural pressure** and forcing the segment to move outward with the rest of the rib cage during inspiration.

Flail chest	
Pathophysiology	<ul style="list-style-type: none"> • ≥ 3 contiguous ribs fractured in ≥ 2 locations → flail chest segment
Findings	<ul style="list-style-type: none"> • Paradoxical chest wall motion with respiration • Chest pain, tachypnea, rapid shallow breaths • CXR: Rib fractures +/- contusion/hemothorax
Management	<ul style="list-style-type: none"> • Pain control, supplemental oxygen • PPV (+/- chest tube) if respiratory failure

CXR = chest x-ray; PPV = positive pressure ventilation.

- **Pulmonary contusion:**
 - Pulmonary contusion results in **intraalveolar hemorrhage and edema** and complicates 25%-35% of cases of blunt thoracic trauma (BTT).
 - Characteristic features include tachypnea, tachycardia, and hypoxia with rales and decreased breath sounds, all of which develop **<24 hours after BTT**.
 - Pulmonary contusion may not be clinically evident immediately following an injury, and initial chest x-ray may be negative. CT scans are more sensitive and can identify the **hallmark patchy nonlobular opacification (not restricted by anatomical landmarks)**.
 - Management involves pain control, pulmonary hygiene (nebulizer treatment, chest physiotherapy), and respiratory support, including mechanical ventilation in severe cases. **Administration of large volumes of intravenous fluid may worsen the underlying pulmonary edema.**

Pulmonary contusion



Pulmonary contusion	
Clinical features	<ul style="list-style-type: none"> • Present <24 hours after blunt thoracic trauma • Tachypnea, tachycardia, hypoxia
Diagnosis	<ul style="list-style-type: none"> • Rales or decreased breath sounds • CT scan (most sensitive) or CXR with patchy, alveolar infiltrate not restricted by anatomical borders
Management	<ul style="list-style-type: none"> • Pain control • Pulmonary hygiene (eg, incentive spirometry, chest PT) • Supplemental oxygen & ventilatory support

CXR = chest x-ray; PT = physiotherapy.

- **Blunt cardiac injury:**
 - Blunt cardiac injury should be suspected with the **presence of sternal fractures**.
 - Myocardial contusion is in the spectrum of potential cardiac injuries caused by blunt trauma to the chest, which also may include **asymptomatic cardiac arrhythmias; valvular, septal, or ventricular wall injuries; ventricular free wall rupture; pericardial tamponade; and death**.
 - ECG monitoring will detect any abnormalities (arrhythmia).
 - **An urgent echocardiogram should be obtained in patients with blunt chest trauma and signs of acute heart failure or shock.**

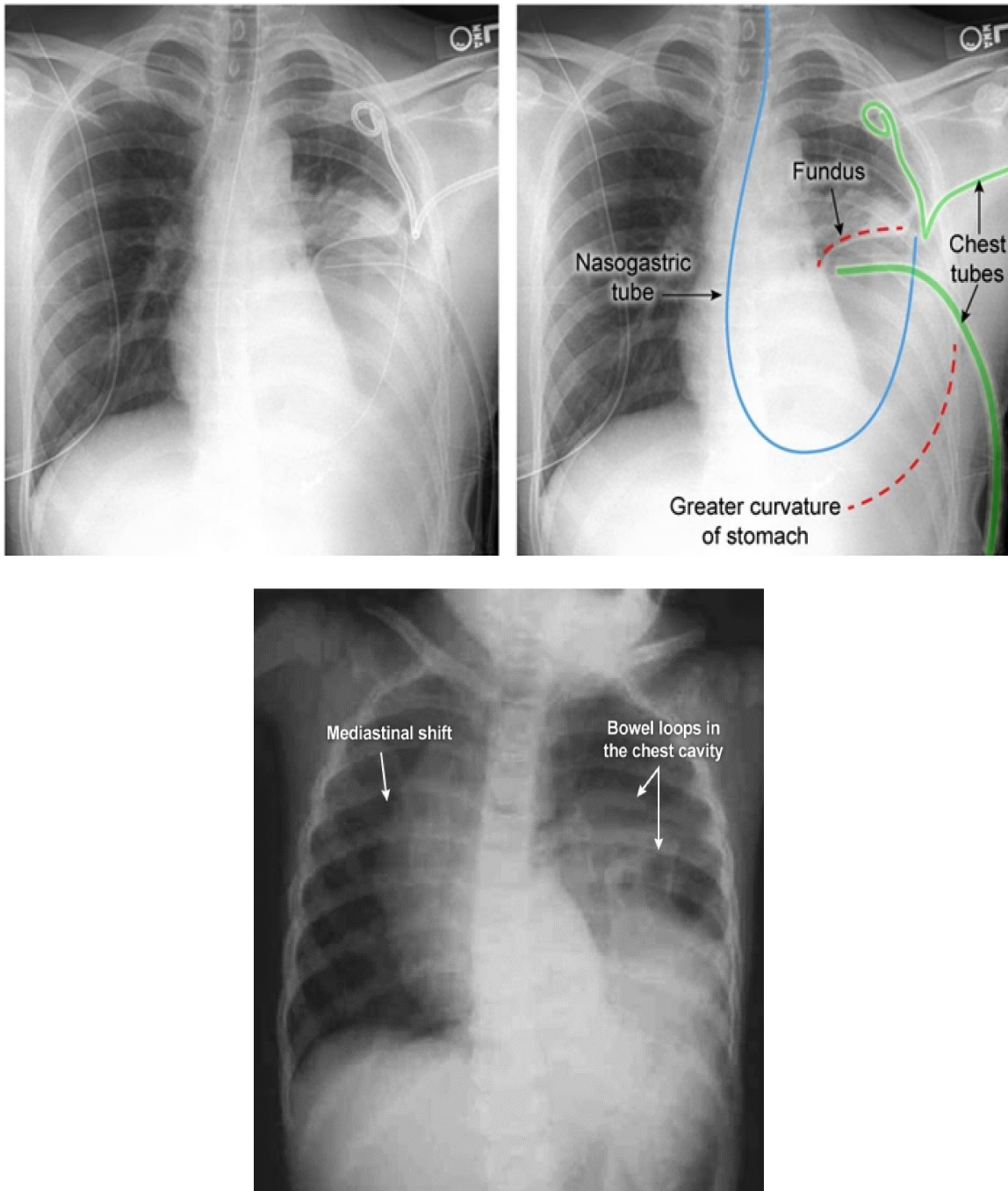
Blunt cardiac injury	
Pathophysiology	<ul style="list-style-type: none"> • Rapid deceleration or direct blow to the precordium → shearing, compression, abrupt pressure change
Clinical spectrum	<ul style="list-style-type: none"> • Arrhythmia ranging from asymptomatic (eg, PVCs) to fatal (eg, VFib) • Acute coronary syndrome from coronary dissection or thrombosis • Myocardial dysfunction ("myocardial contusion") • Ruptured valve, septum, or ventricular wall • Cardiac tamponade
Confirmatory testing	<ul style="list-style-type: none"> • ECG • Echocardiogram

PVCs = premature ventricular contractions; **VFib** = ventricular fibrillation.

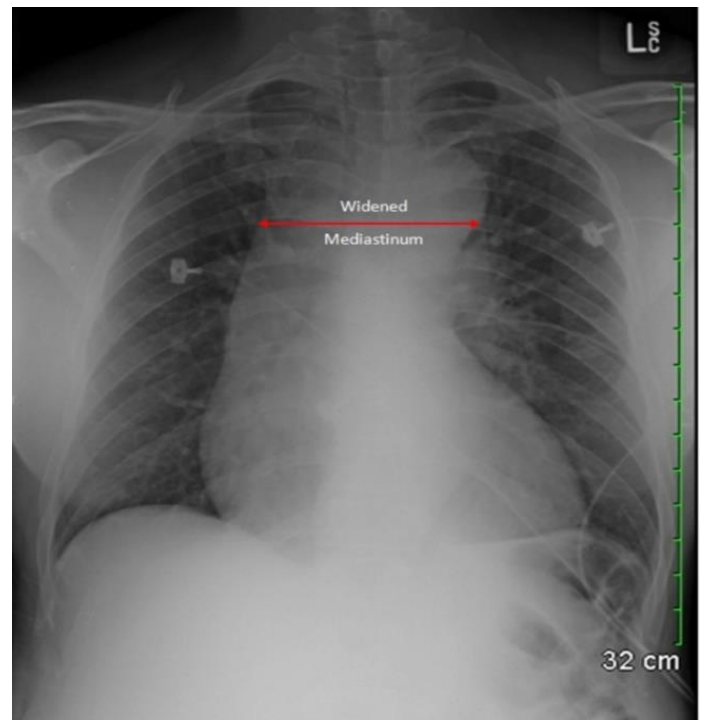
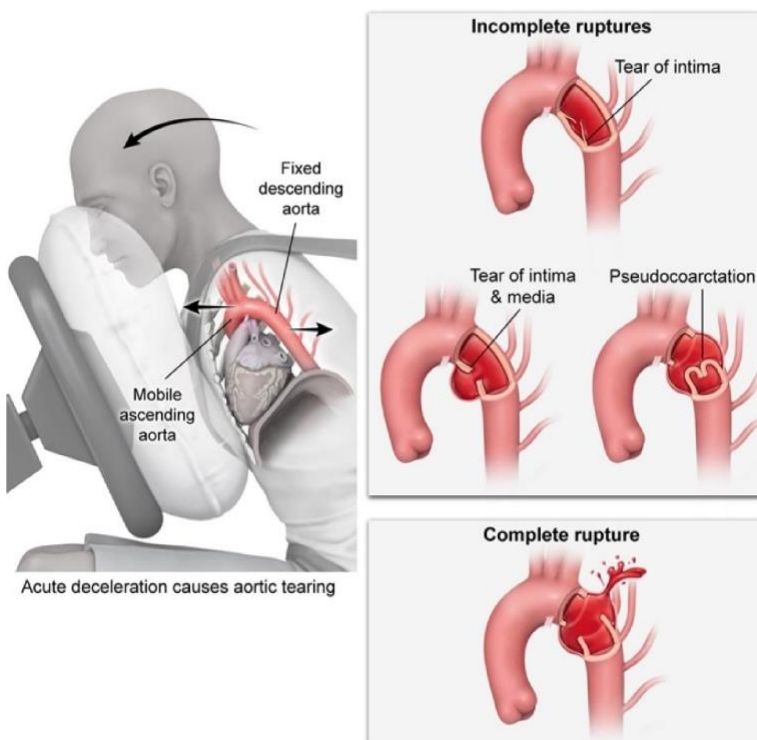
- **Traumatic rupture of the diaphragm:**
 - Blunt abdominal trauma can cause a **sudden increase in intraabdominal pressure that overcomes the muscular strength of the diaphragm** and leads to large radial tears in the muscle.
 - The resultant diaphragmatic rupture allows **leakage of intraabdominal contents into the chest**, causing compression of the lungs and mediastinal deviation.
 - **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 and abdomen is sometimes required if the chest x-ray does not visualize the area well**. The small bowel is sometimes present in the thoracic cavity.

- Chest x-ray showing a nasogastric tube in the pulmonary cavity is diagnostic.
- 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.

Diaphragmatic hernia

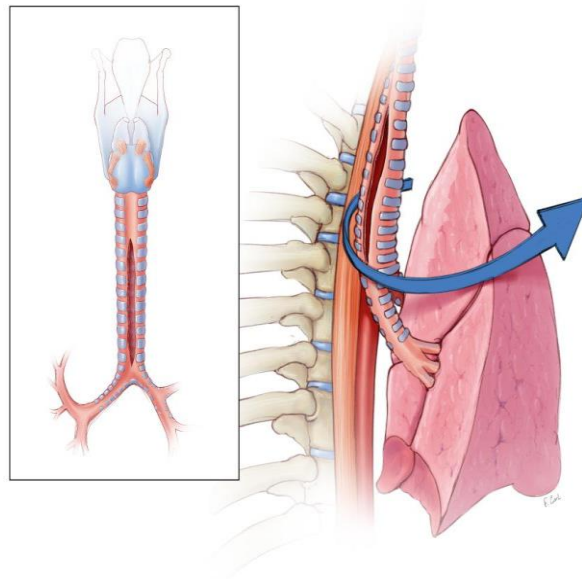


- Traumatic rupture of the aorta is the ultimate “hidden injury”:
- For patients involved in motor vehicle accidents or falls from > 10 feet (3 m), physicians must have a high suspicion for blunt aortic injury.
- Oftentimes in the setting of high energy aortic injury secondary to blunt chest trauma, aortic transection, circulatory collapse, and death are immediate sequelae. A minority of patients with aortic injury have an incomplete or contained rupture.
- Traumatic rupture of the aorta most commonly occurs at the junction of the arch and the descending aorta (aortic isthmus) where the relatively mobile aorta is tethered by the ligamentum arteriosum.
- Such an injury requires a significant deceleration injury and is totally asymptomatic until the hematoma contained by the adventitia ruptures resulting in rapid death.
- Suspicion should be triggered by one of the following:
 - Mechanism of injury.
 - Widened mediastinum on chest x-ray.
 - Presence of atypical fractures such as the first rib, scapula, or sternum, which requires great force to fracture.
- Chest x-ray is the initial screening test and widening of the mediastinum is the most sensitive finding. The diagnosis can be confirmed via CT angiogram.
- Management of patients with established aortic injury includes antihypertensive therapy where appropriate and immediate operative repair.



▪ Traumatic rupture of the trachea or major bronchus:

- Traumatic rupture of the trachea or major bronchus is suggested by developing **subcutaneous emphysema in the upper chest and lower neck**, or by a **large "air leak" from a chest tube**.
- Chest x-ray and CT scan confirm the presence of air outside the bronchopulmonary tree, and **fiberoptic bronchoscopy is necessary to identify the injury and allow intubation past the injury to secure an airway**.
- Surgical repair is indicated.
- Differential diagnosis of subcutaneous emphysema also includes **rupture of the esophagus and tension pneumothorax**.

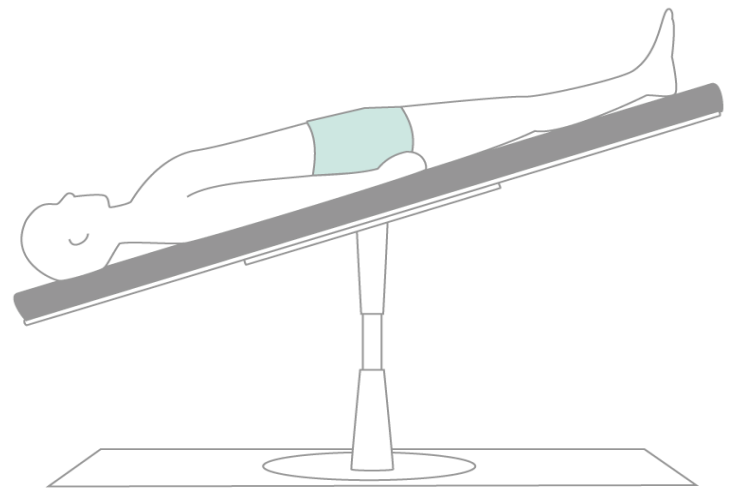
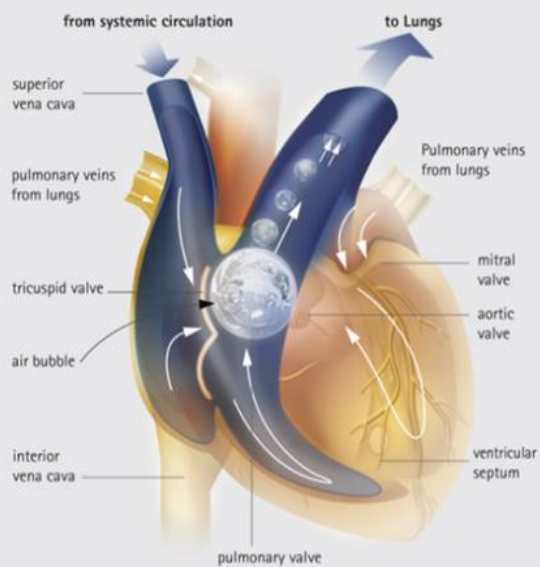


❖ N.B:

1. **Persistent pneumothorax and significant air leak following chest tube placement in a patient who has sustained blunt chest trauma suggests tracheobronchial rupture.**
 - Other findings include pneumomediastinum and subcutaneous emphysema (palpable crepitus below the skin).
 - **The right main bronchus is most commonly injured in these cases.**
 - **Bronchoscopy can confirm the diagnosis prior to operative repair.**
 - Operative repair is indicated.
2. Blunt thoracic trauma can cause a sudden increase in intraesophageal pressure sufficient to rupture the esophagus.
 - **If gastrointestinal contents leak from the esophagus into the pleural space, pleural effusion results, and fluid analysis typically reveals unusual color (green), low pH, and high amylase.**
 - **Water soluble contrast esophagography can confirm the diagnosis → leak from perforation.**
 - Management includes **NPO, prophylactic antibiotics, PPI, and surgical consultation.**

■ Air embolism:

- It also can occur when the subclavian vein is opened to the air (supraclavicular node biopsies, central venous line placement or lines that become disconnected), also leading to **sudden cardiovascular collapse and cardiac arrest**.
- Immediate management includes cardiac massage, with the patient positioned in Trendelenburg with the left side down.
- **Prevention includes the Trendelenburg position when the great veins at the base of the neck are to be accessed.**

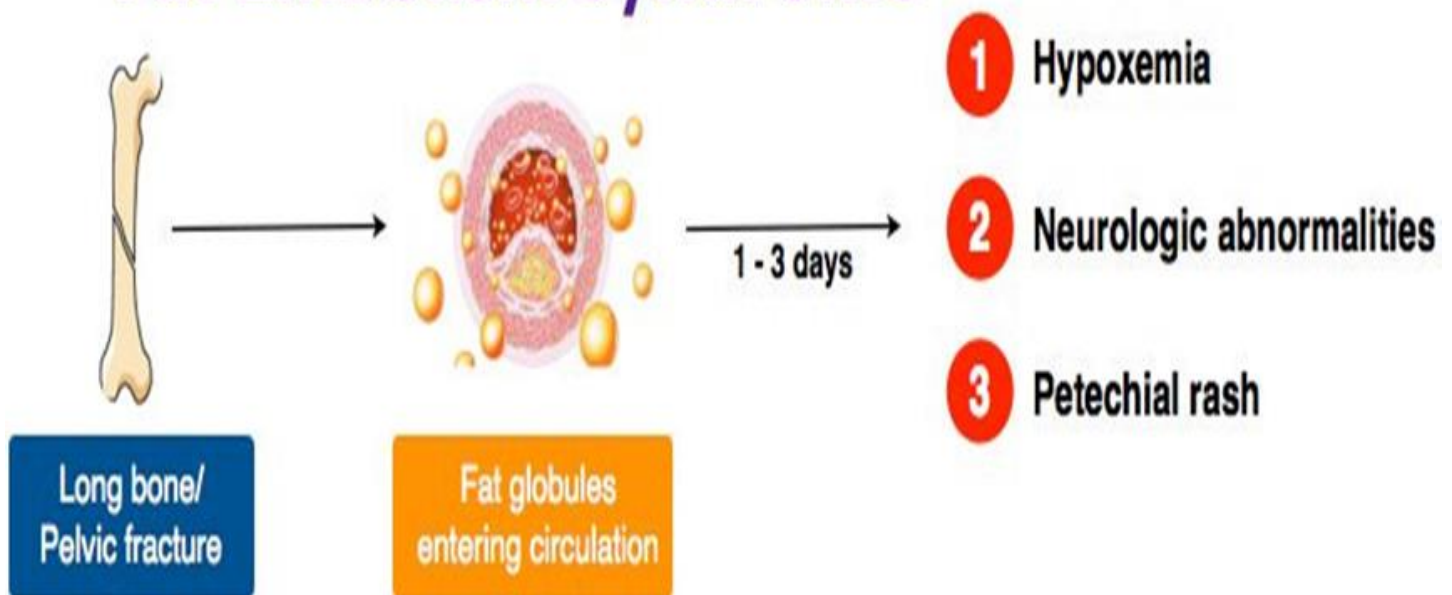


■ Fat embolism:

- Fat embolism may also produce **respiratory distress in a trauma patient who may not have necessarily suffered chest trauma**.
- The typical setting is the following:
 - The development of the classic triad **respiratory distress, diffuse neurological impairment (confusion), and an upper body petechial rash (due to thrombocytopenia)** within days of severe long bone fractures is characteristic of the fat embolism syndrome.
 - Pathophysiologically, the condition arises when a traumatic event dislodges fat globules from the bone marrow, allowing them to **travel through the marrow vascular sinusoids and into the pulmonary microvessels**.

- Diagnosis can be confirmed by **presence of fat droplets in urine or presence of intra-arterial fat globules on fundoscopy**. Serial x-ray shows increasing **diffuse bilateral pulmonary infiltrates within 24-48 hours of onset of clinical findings**.
- The mainstay of therapy is **respiratory support**. Use of heparin, steroids, and low molecular weight dextran is controversial.

Fat Embolism Syndrome



3. Abdominal Trauma:

- For the sake of evaluation and management, abdominal trauma is **divided into penetrating and blunt trauma based on the mechanism of injury**.
- Penetrating trauma is further differentiated into **gunshot wounds and stab wounds**.
- **Any penetrating injury in the thorax below the 4th intercostal space (level of the nipples) has potential to also involve the abdomen through the diaphragm and is assumed to involve both compartments until proven otherwise.**
- Gunshot wounds:
 - Gunshot wounds to the abdomen **require exploratory laparotomy for evaluation and possible repair of intra-abdominal injuries (almost always penetrate peritoneum → cause intraperitoneal organ injury), not to “remove the bullet”**.
- Stab wounds:
 - Following completion of the primary survey, the evaluation of patients with penetrating abdominal trauma (PAT) should **focus on identifying potentially life-threatening indications for urgent exploratory laparotomy to prevent sepsis or exsanguinating hemorrhage**.
 - The presence of any the following suggests significant injury and is an **indication for urgent exploratory laparotomy**:
 - Hemodynamic instability.
 - Peritonitis (rebound tenderness, guarding).
 - Evisceration (externally exposed intestines).
 - Signs of GI hemorrhage (Blood from a nasogastric tube or on rectal examination).
 - In the absence of the conditions above, **local wound exploration may be performed in the ED to assess whether or not the anterior rectus fascia has been penetrated (determine if there is penetration into the peritoneal space)**:
 - If the fascia is not violated → **the intra-abdominal cavity likely has not been penetrated and no further intervention is necessary**.
 - If the fascia has been violated → **surgical exploration is indicated to evaluate for bowel or vascular injury**, even in the setting of hemodynamic stability and lack of peritoneal findings on physical examination.
 - Patients without indications for urgent laparotomy should undergo further evaluation, including extended ultrasound examination (extended Focused Assessment with Sonography for Trauma [eFAST], which **evaluates for pneumothorax and hemothorax in addition to intraperitoneal injuries**).

- Blunt trauma to the abdomen:

- Blunt trauma to the abdomen with obvious signs of internal injury requires emergent surgical evaluation via exploratory laparotomy.
- Signs of internal injury include abdominal distention and significant abdominal pain with guarding or rigidity on physical examination consistent with peritonitis.
- The occurrence of blunt trauma even without obvious signs of internal injury requires further evaluation because internal hemorrhage or bowel injury can be slow and therefore present in a delayed fashion.

- Signs of internal bleeding:

- Signs of internal bleeding include a drop in BP, a fast and/or thready pulse, a low CVP, and low urinary output.
- Patients tend to be cold, pale, anxious, shivering, and thirsty.
- These signs of shock occur when 25-30% of blood volume is acutely lost, ~1,500 ml in the average-size adult.

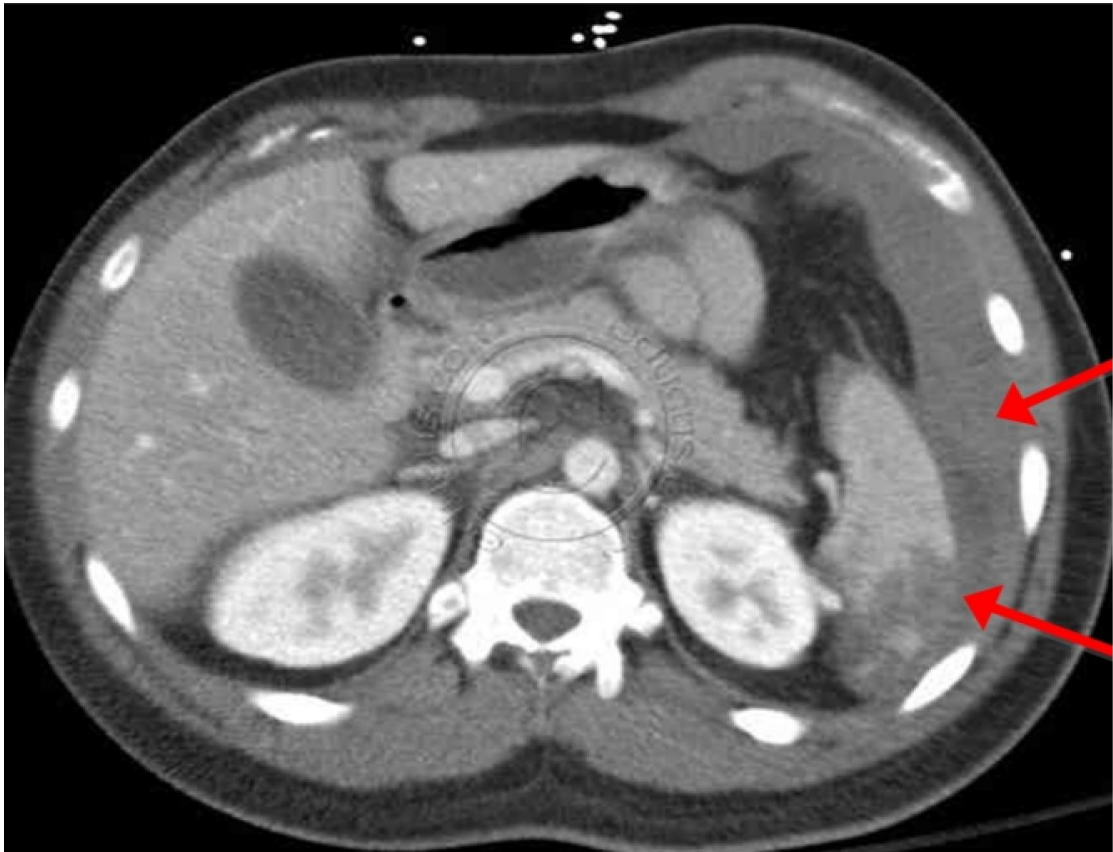
- There are few places in the body that this volume of blood can be lost without being obvious on physical or radiographic exam:

- The head is too small without causing a lethal degree of intracranial pressure.
- The neck could contain a significant amount of blood, but such a hematoma would be obvious on physical exam.
- The pericardial sac cannot contain a significant amount of blood loss without resulting in pericardial tamponade and rapid clinical deterioration.
- The pleural cavities could easily accommodate several liters of blood, with relatively few local symptoms, but that significant a hemothorax would be obvious on chest x-ray, which is routinely performed in the secondary survey of a trauma patient.
- The arms and legs would also be obviously deformed by a large hematoma if present.
- That leaves the abdomen, retroperitoneum, thighs (secondary to a femur fracture), and pelvis as the only places where a volume of blood significant enough to cause shock could “hide” in a blunt trauma patient that has become unstable. The femurs and pelvis are always checked for fractures in the initial survey of the trauma patient by physical exam and pelvic x-ray.
- So, a patient who has experienced blunt trauma who has become hemodynamically unstable with normal chest and pelvic x-rays likely has intra-abdominal bleeding.

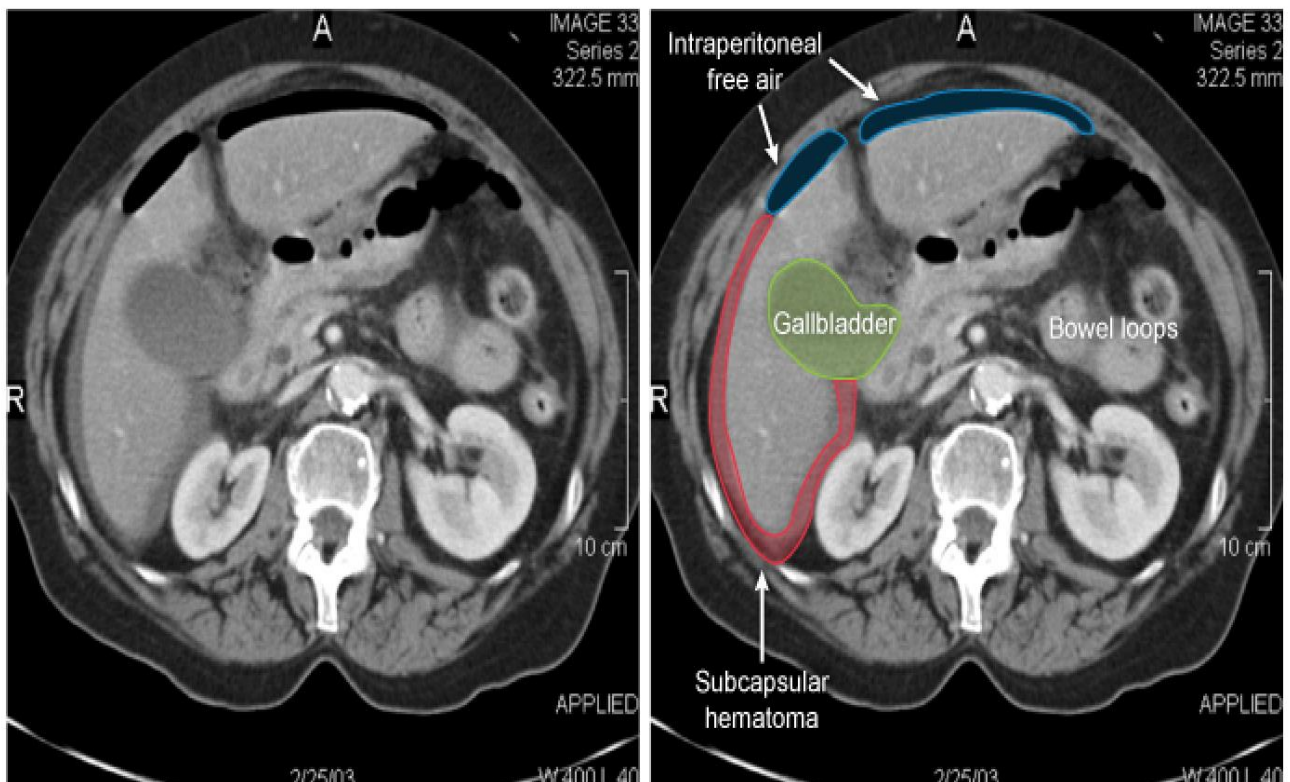
- Diagnosis can be quickly utilizing the “FAST” exam (Focused Abdominal Sonography for Trauma). FAST can be performed rapidly at the bedside and has good sensitivity and specificity for detecting hemoperitoneum, pericardial effusion, and intraperitoneal fluid.
- The evaluation and management of BAT depend on the patient's hemodynamic status and response to intravenous fluids:
 - o An unstable patient with these abnormal findings in FAST exam should be taken to the OR for immediate surgical exploration (urgent laparotomy).
 - o Patients who are hemodynamically stable (systolic blood pressure >90 mm Hg) and alert should undergo Focused Assessment with Sonography for Trauma (FAST). Abnormal FAST will need CT scan of the abdomen. Normal FAST will need serial abdominal exam. If FAST is limited or equivocal, a diagnostic peritoneal lavage (DPL) can be done to evaluate for hemoperitoneum.
 - o Those with a normal (negative) FAST (no intraperitoneal fluid) but high-risk features such as anemia or guarding should undergo subsequent CT scan of the abdomen. CT will show the presence of intra-abdominal fluid and can accurately delineate the source, typically the liver or spleen.
 - o Stable patients with altered mental status often proceed directly to CT imaging.
- If surgical exploration is indicated for penetrating or blunt trauma, certain principles must be employed:
 1. Prolonged surgical time and ongoing bleeding can lead to the “triad of death” → hypothermia, coagulopathy, and acidosis:
 - o The longer a patient is open, these components worsen and precipitate each other, resulting in a vicious cycle ultimately leading to death.
 - o Accordingly, the “damage control” approach has been adopted: immediate life-threatening injuries are addressed, less urgent injuries are temporized. Obviously repair of a major vascular structure with ongoing bleeding takes precedence. Next comes control of contamination from injury to the GI tract. If a bowel resection is necessary, reconstruction can be delayed as only the contamination is life-threatening, not the inability to digest food.
 - o If hypothermia, coagulopathy, or acidosis is setting in and injuries have been controlled, the operation is terminated, and the abdomen is packed with gauze pads and closed with a temporary closure. The patient is resuscitated in the ICU and returns to the OR at a later date when warm, not coagulopathic, and not acidotic for definitive reconstruction and abdominal closure.

2. If coagulopathy does develop during surgical exploration, it is objectively **treated with transfusion of RBCs, fresh frozen plasma, and platelets in equal quantities (1:1:1 ratio)**. This most realistically mimics the replacement of whole blood and enables not only adequate quantities of hemoglobin, but also adequate clotting factors to reverse the developing coagulopathy and enable control of hemorrhage.
- The abdominal compartment syndrome:
 - The abdominal compartment syndrome is when **the pressure in the peritoneal cavity is elevated and leads to end-organ injury**.
 - **This occurs when a significant amount of fluid is administered in an effort to resuscitate a patient in hypovolemic shock.**
 - Bowel edema develops, increasing intra-abdominal pressure, which is detrimental for several reasons:

 - First, the elevated pressure leads to decreased perfusion pressure to the viscera (Compression of IVC → ↓ VR → ↓ CO) contributing to **acute kidney injury** and possibly **bowel and hepatic ischemia**.
 - Second, the upward pressure of the viscera on the diaphragm prevents adequate expansion of the lungs and ventilation, contributing to **respiratory failure**.
 - Therefore, if bowel edema is observed or intra-abdominal pressure is elevated following surgical exploration, **the abdomen is not closed but rather left open**.
 - Similarly, if a patient is not surgically explored but undergoes a significant volume resuscitation and abdominal compartment syndrome develops, a **decompressive laparotomy may be indicated**.
 - Ruptured spleen:
 - The most common intra-abdominal organ injuries due to BAT are **hepatic and splenic lacerations**.
 - **Splenic injury is most likely in the setting of abdominal pain, tachycardia and left chest wall and shoulder pain without evidence of abnormalities of the shoulder (likely referred pain due to phrenic nerve irritation from splenic hemorrhage).**
 - If operative intervention is required, every effort is made to repair the spleen rather than remove it, especially in children. If removal is unavoidable, **postoperative immunization against encapsulated bacteria is mandatory**.

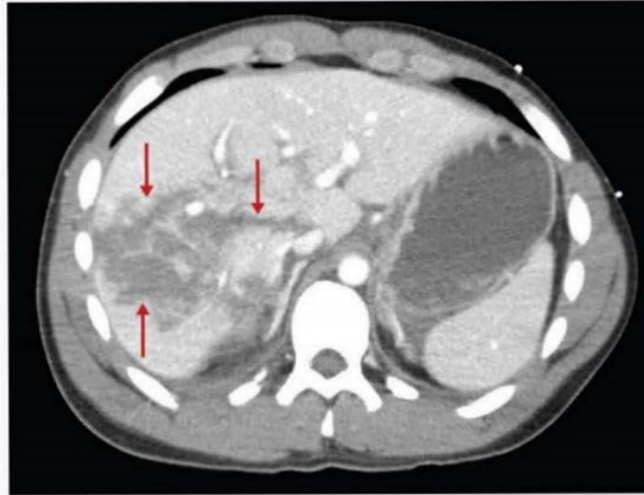


Intraperitoneal free air on abdominal CT scan



❖ N.B:

1. Hepatic laceration is one of the most common solid organ injuries due to blunt abdominal trauma.
 - Common manifestations include hypotension, free intraperitoneal fluid, right upper quadrant pain and bruising, and right shoulder pain due to phrenic nerve irritation.



2. Although gastrointestinal (GI) perforation is more frequently associated with penetrating abdominal trauma.
 - It can also result from BAT due to damage to the mesenteric blood supply, subsequent GI necrosis, and eventual perforation.
 - Consequently, although injury due to penetrating trauma may present acutely, signs and symptoms of perforation due to BAT may take several days to present.
 - Therefore, patients with any evidence of injury to the mesenteric vessels should be considered for longer periods of observation and monitoring.
 - When perforation does occur, the jejunum is most frequently involved, whereas the stomach and colon are less frequently injured.
 - Once perforation is identified, patients should be taken for urgent exploratory laparotomy.
3. Duodenal hematomas are more commonly seen in pediatric patients and most often occur following blunt abdominal trauma.
 - They are more commonly seen in children due to a number of anatomic differences, including thinner abdominal wall musculature, less abdominal adipose tissue and more pliable ribs (which absorb less force than the stiffer ribs of adults).
 - DH commonly occurs when a blunt force rapidly compresses the duodenum against the vertebral column. Following trauma, blood collects between the submucosal and muscular layers of the duodenum causing partial or complete obstruction.
 - Patients classically present 24-36 hours after the initial event with epigastric pain and vomiting due to failure to pass gastric contents beyond the obstructing hematoma.
 - Diagnosis is confirmed with CT imaging of the abdomen.
 - Most DHs will resolve in 1-2 weeks.
 - Management involves decompression by nasogastric tube and, in many patients, parenteral nutrition.
 - Surgery or percutaneous drainage may be considered to evacuate the hematoma if nonoperative management fails.

3. Pelvic Fracture:

- The pelvis is a complex ring in that it cannot be fractured in only one location; **multiple fractures are typically present**. These can range from minor to life-threatening.
- In pelvic fracture with ongoing significant bleeding causing **hemodynamic instability**, management is complex:
 - The first step for an obvious pelvic fracture in an unstable patient is **external pelvic wrapping for stabilization of the pelvis, which limits the potential space for ongoing blood loss**.
 - The next step is not surgical exploration but rather **angiography**:
 - This is because **it is incredibly difficult (often impossible) to identify the source of bleeding in the pelvis where a deep cavity contains significant organs and vessels including the complex sacral venous plexus**.
 - However, interventional radiologists can angiographically identify an arterial source of bleeding and potentially embolize the branch vessels and control hemorrhage.
 - **If no arterial bleeding is identified, the ongoing blood loss is presumed to be venous in origin**, and the internal iliac arteries are prophylactically embolized to prevent the inflow to these bleeding veins.
- **In any pelvic fracture, associated injuries have to be ruled out.** These include injuries to the:
 - **Rectum** → do a rectal exam and rigid proctoscopy.
 - **Vagina** in women → do a pelvic exam.
 - **Urethra** in men → do a retrograde urethrogram.
 - **Bladder** → do retrograde cystogram.



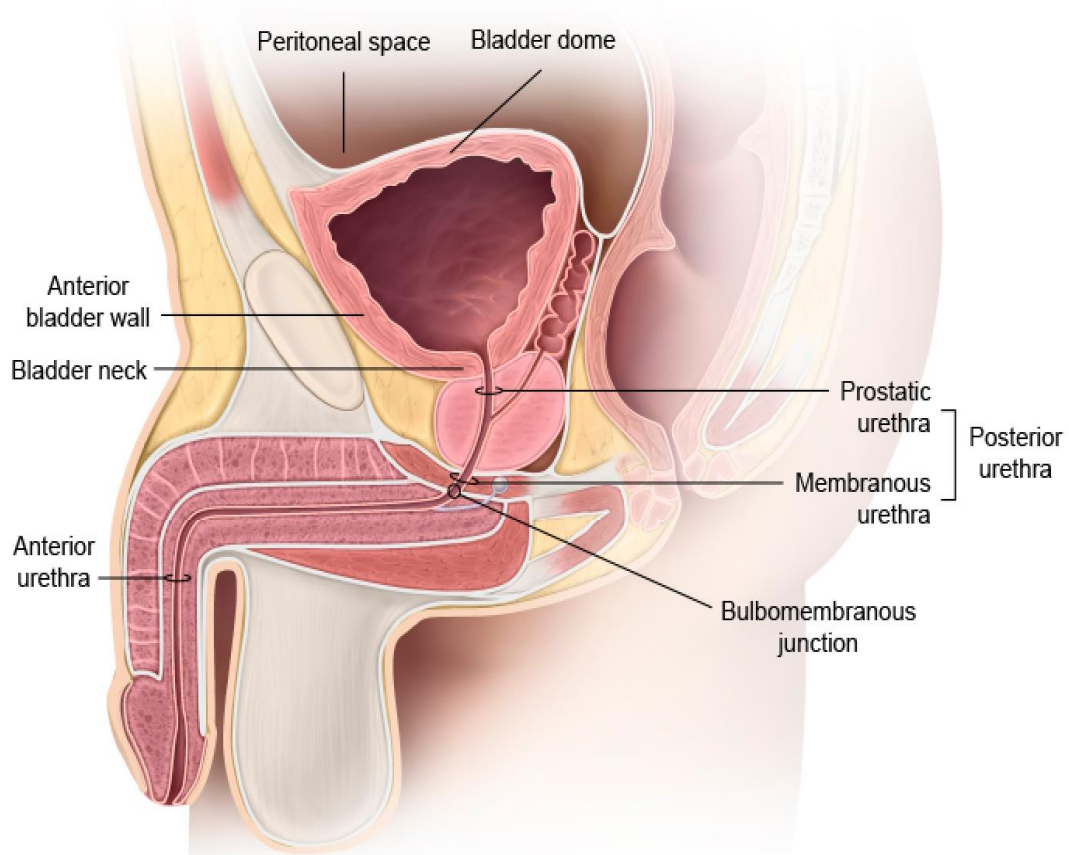
4. Urologic Injury:

- The hallmark of urologic injury is blood in the urine of someone who has sustained penetrating or blunt abdominal trauma. Gross hematuria in that setting must be investigated with appropriate studies.
- Penetrating urologic injuries as a rule are surgically explored and repaired.
- Blunt urologic injuries may affect the kidney, in which case the associated injuries tend to be lower rib fractures. If they affect the bladder or urethra, the usual associated injury is pelvic fracture.
- Urethral injuries:
 - Urethral injuries most commonly occur in men because of their longer urethral length and are divided into anterior and posterior urethral injuries.
 - Injury to the posterior urethra is associated with pelvic fractures, and the anterior urethra is most commonly damaged in straddle injuries.
 - The posterior urethra is located above the bulb of the penis, and the anterior urethra lies within the bulb and the remainder of the corpus spongiosum.
 - The posterior urethra is further divided into the prostatic and membranous segments; the anterior urethra is divided into bulbous and penile segments.
 - In contrast to the prostatic and bulbous segments, the membranous segment is relatively unsupported by the adjacent tissues and is the weakest point of the posterior urethra.
 - Trauma to the pelvis severe enough to cause fracture often results in disruption of the posterior urethra at the bulbomembranous junction (dividing point between the anterior and posterior urethra).
 - They may present with blood at the meatus. Other clinical findings include a scrotal hematoma, the sensation of wanting to void but inability to do so, and a “high-riding” prostate on rectal exam (it is not palpable on rectal exam).
 - The key issue in any of these is that a Foley catheter should not be inserted, as it might compound an existing injury; a retrograde urethrogram should be performed instead. If Foley catheter placement is attempted and resistance met, this should be a clue that a urethral injury may be present and attempt should be aborted.
 - Retrograde urethrogram: This diagnostic test involves an x-ray of the lower genitourinary tract obtained during the injection of radiopaque contrast into the urethra. A normal study demonstrates contrast entering the bladder uninterrupted. Extravasation of contrast from the urethra or inability of contrast to reach the bladder is diagnostic of urethral injury.

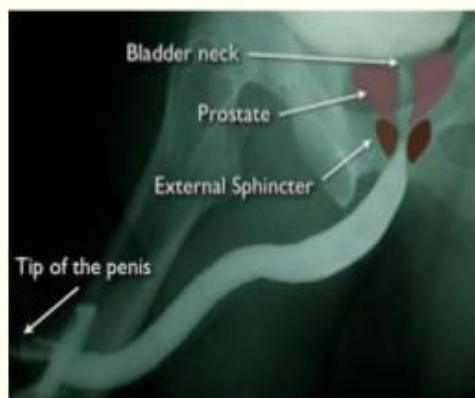
- **Indications for urethrogram include:**

- Blood at the meatus.
- Hematuria.
- Dysuria.
- Urinary retention.

Male urogenital anatomy



How does Retrograde Urethrogram look like ?



Normal Retrograde Urethrogram



Obstructed Retrograde Urethrogram

▪ **Bladder injuries:**

- Bladder injuries can occur in either sex, are usually associated with pelvic fracture, and are **diagnosed by retrograde cystogram or CT cystography**.

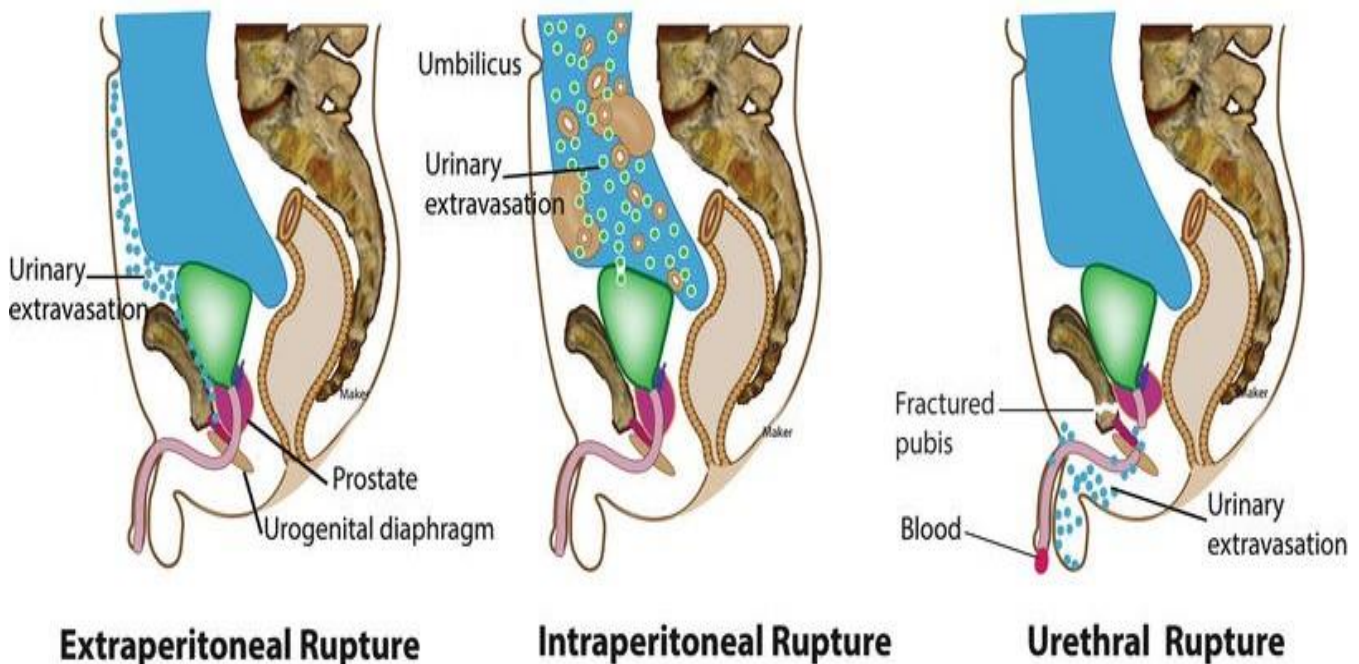
- Bladder rupture after blunt trauma is due to a sudden increase in intravesical pressure and most likely occurs following a **blow to the lower abdomen when the bladder is full and distended**.

- **Extraperitoneal bladder injury (EPBI):**

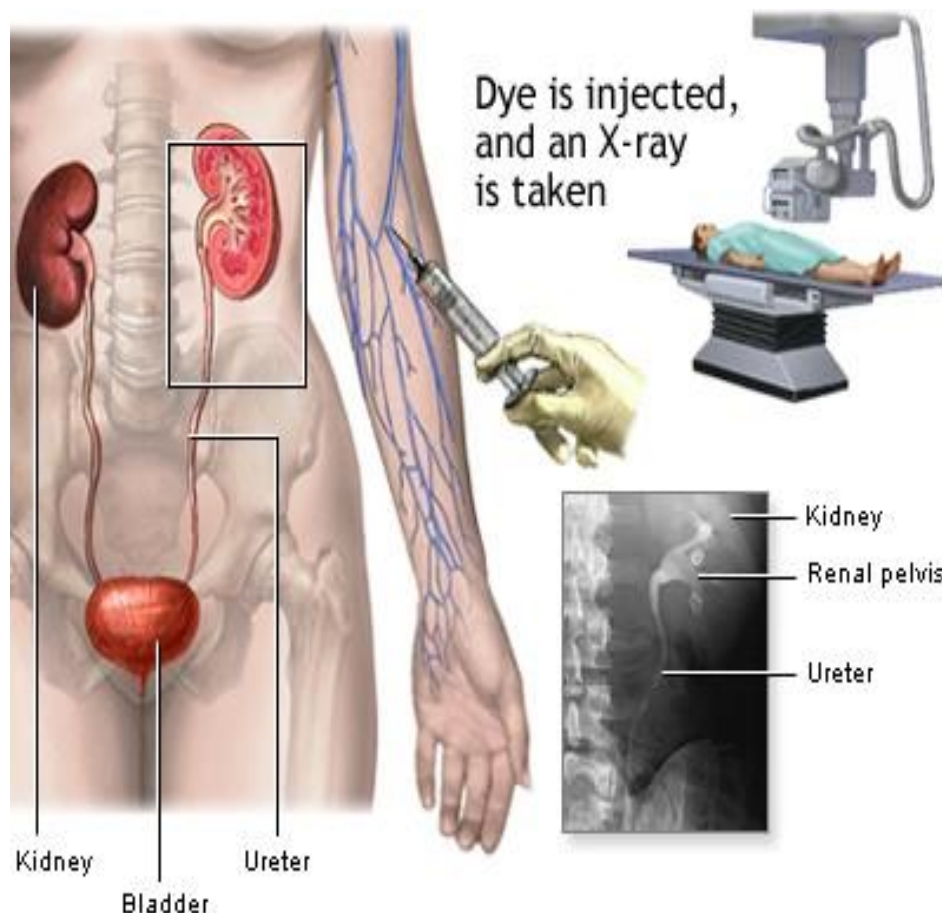
- Extraperitoneal bladder injury (EPBI) which may consist of either **contusion or rupture of the neck, anterior wall, or anterolateral wall of the bladder**.
- In the case of rupture, extravasation of urine into adjacent tissues causes **localized pain in the lower abdomen and pelvis**.
- **Pelvic fracture is almost always present in EPBI**, and sometimes a bony fragment can directly puncture and rupture the bladder.
- Gross hematuria is also usually present, and urinary retention (evidenced by suprapubic fullness) may occur, especially in the case of injury to the bladder neck.

- **Intraperitoneal bladder rupture:**

- Intraperitoneal bladder rupture describes **rupture of the dome of the bladder**; the dome is composed of the superior and lateral bladder walls.
- **The superior and lateral surfaces of the bladder compose the dome of the bladder and are bordered by the peritoneal cavity**.
- **Rupture of this area results in intraperitoneal urine leakage and typically presents with signs of chemical peritonitis** (diffuse abdominal tenderness, guarding, rebound).
- Pelvic fracture is often present but less commonly than in EPBI.

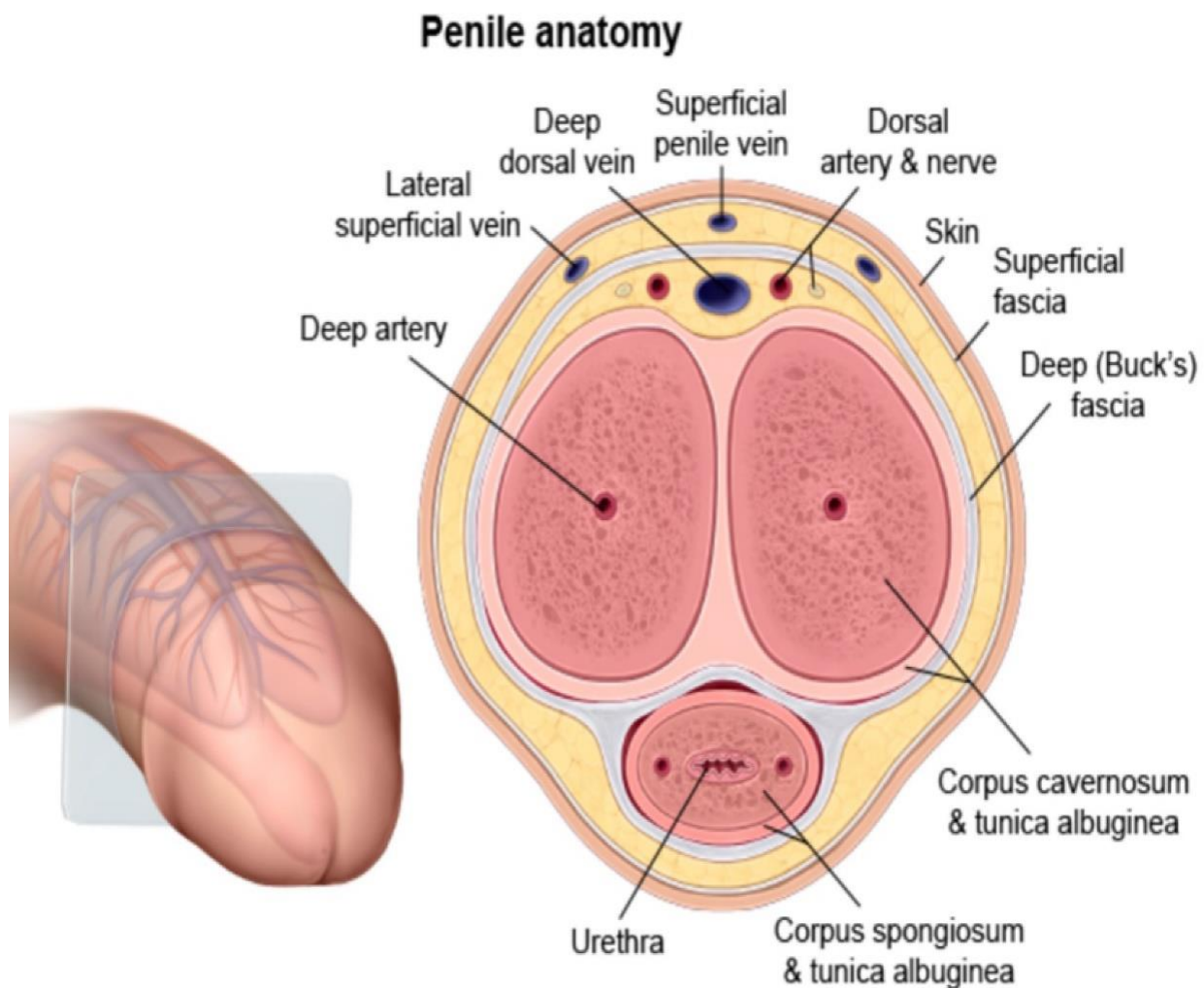


- In addition, irritation of the peritoneal lining of the right or left hemidiaphragm may cause referred pain to the ipsilateral shoulder (Kehr sign) as sensory innervation to the shoulder originates from the C3 to C5 spinal roots, these roots are also the origin of the phrenic nerve innervating the diaphragm.
- Management is **surgical repair**.
- **Renal injuries secondary to blunt trauma:**
 - Blunt genitourinary trauma (BGT) is **rarely life-threatening unless the kidneys or renal vasculature are involved**. However, **due to their retroperitoneal location and the protection afforded by the ribs, these structures are infrequently injured in BGT**.
 - When injury does occur, the most common renal lesions are contusions, lacerations, and renovascular injuries (pedicle avulsion, renal artery dissection).
 - All patients should undergo urinalysis, and **hemodynamically stable patients with evidence of hematuria should undergo further imaging with a contrast-enhanced CT scan of the abdomen and pelvis**.
 - **Hemodynamically unstable patients with evidence of renal trauma should undergo intravenous pyelography prior to surgical evaluation**.
 - Unless severe, **most renal injuries due to BGT can be managed nonoperatively**.



▪ Fracture of the penis:

- Fracture of the penis (fracture of the corpora cavernosa, fracture of the tunica albuginea) **occurs to an erect penis**, typically as an accident during vigorous intercourse (with woman on top).
- Patients typically experience an **audible snapping sensation**, detumescence, and minimal to severe pain (depending on severity of the injury); a hematoma forms rapidly, causing bending of the shaft of the penis at the fracture site.
- Frequently, the true history will be concealed by an embarrassed patient who concocts a cover story.
- Diagnosis is usually **clinical**. The only imaging test commonly used in evaluation is **retrograde urethrogram**, which is employed in cases of suspected urethral injury, a common complication.
- **Emergency surgical repair is required. If not done, impotence will ensue as either arteriovenous shunts or painful erections.**



❖ N.B:

1. Scrotal hematomas can attain alarming size, but typically do not need specific intervention unless the testicle is ruptured. The latter can be assessed with sonogram.
2. Peyronie disease is an acquired disorder characterized by fibrosis of the tunica albuginea of the penis, which restricts tissue expansion and flexibility during erections.
 - Manifestations often include penile pain, curvature, and/or dorsal nodules/plaques; distortion of the normal erectile shape of the penis may make sexual intercourse difficult.
5. Injury to the Extremities:
 - Penetrating injuries of the extremities:
 - In penetrating injuries of the extremities, the main issue is whether a vascular injury has occurred or not. Anatomic location provides the first clue:
 - When there are no major vessels in the vicinity of the injury, only tetanus prophylaxis and irrigation of the wound is required.
 - If the penetration is near a major vessel and the patient is asymptomatic, Doppler studies or CT angiogram is performed and will guide the need for a surgical intervention.
 - If there is an obvious vascular injury (absent distal pulses, expanding hematoma) surgical exploration and repair are required.
 - Simultaneous injuries of arteries and bone:
 - Simultaneous injuries of arteries and bone pose the challenge of the sequence of operative repair.
 - One perspective is to stabilize the bone first, then do the delicate vascular repair which could otherwise be disrupted by the bony reduction and fixation. However, during the orthopedic repair, ongoing ischemia is occurring as the arterial flow is disrupted.
 - A good solution, if proposed on the exam, is to place a vascular shunt, which allows temporary revascularization during the bony repair, with definitive vascular repair completed subsequently.
 - Crushing injuries of the extremities:
 - Crushing injuries of the extremities resulting in myonecrosis pose the hazard of hyperkalemia and renal failure as well as potential development of compartment syndrome.
 - Aggressive fluid administration, osmotic diuretics, and alkalinization of the urine with sodium bicarbonate are good preventive measures for the acute kidney injury, and a fasciotomy may be required to prevent or treat compartment syndrome.

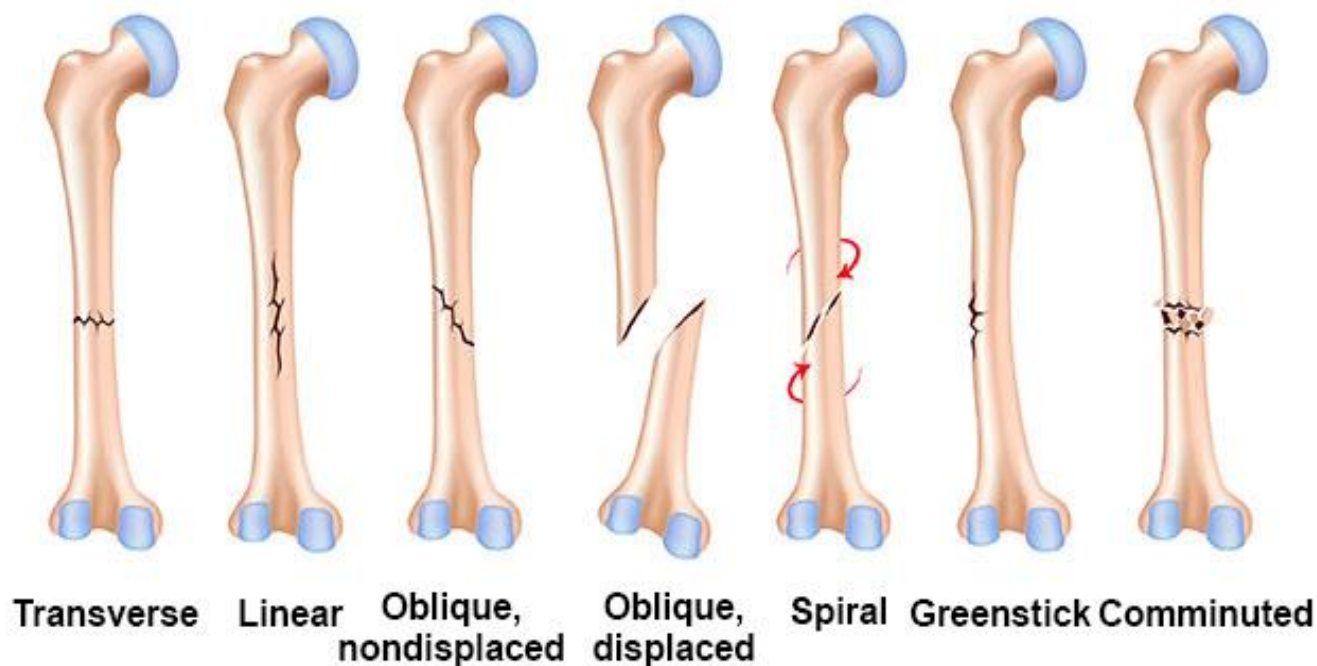
CHAPTER 2

Orthopedics and sport medicine

Orthopedics and sport medicine

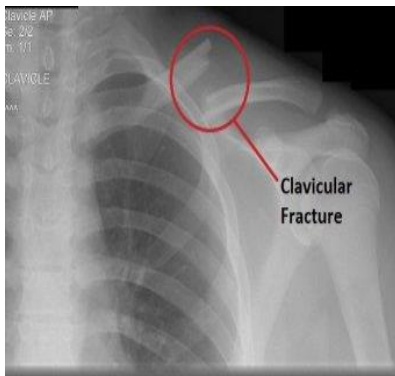
- X-rays for suspected fracture in adults should always include the following:
 - **Two views** at 90° to one another.
 - **Joints above and below** the broken bone.
 - If suggested by **the mechanism of injury**, bones that are in “the line of force,” which might also be broken (the lumbar spine must be evaluated for fracture following a fall from a significant height with foot fractures).
- As a general rule:
 - Broken bones that are **not badly displaced or angulated or that can be satisfactorily aligned by external manipulation can be immobilized in a cast (“closed reduction”).**
 - Broken bones that are **severely displaced or angulated or that cannot be aligned easily require surgical intervention to reduce and fix the fracture (“open reduction and internal fixation”).**

Types of Bone Fractures



■ Clavicular fracture:

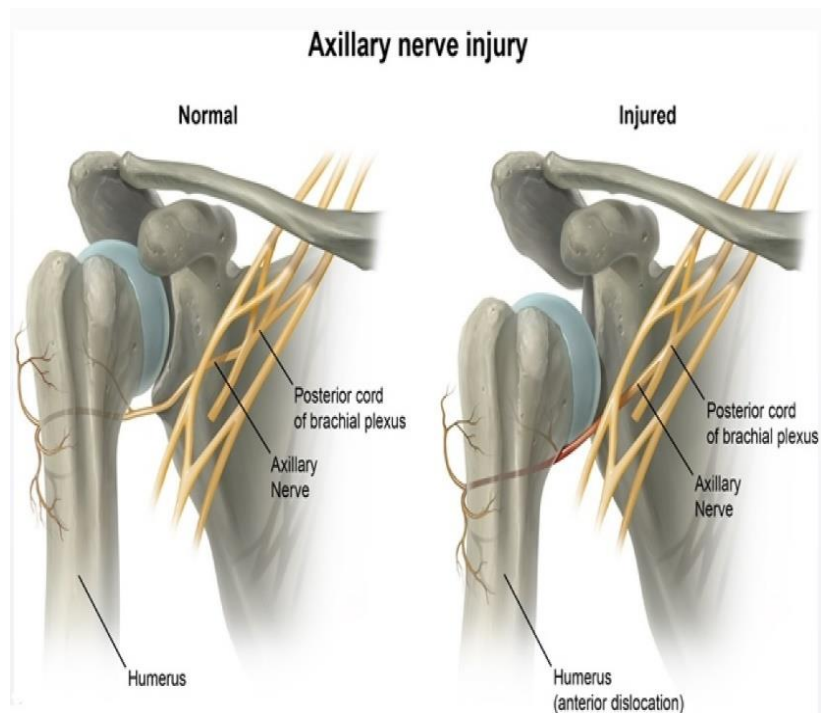
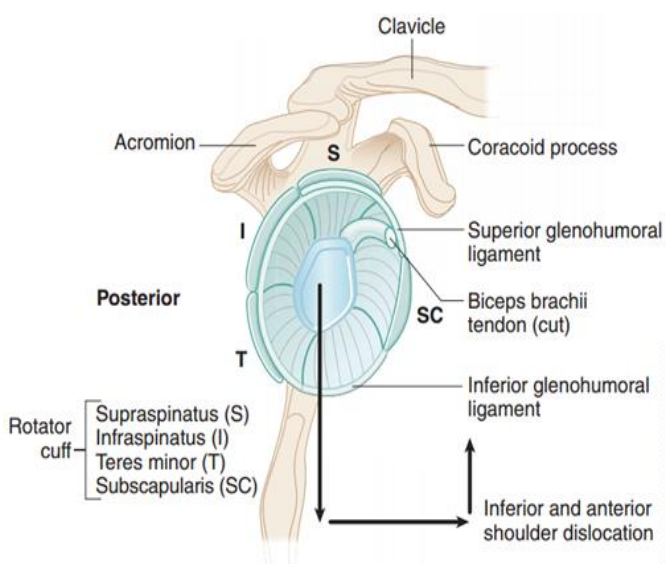
- The clavicle is one of the **most commonly injured bones in the body**. The majority of clavicular fractures occur in **the middle third of the bone (at the junction of middle and distal thirds)**.
- Injury to this bone classically occurs during athletic events and follows a **fall on an outstretched 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**.
- **All patients with a clavicular fracture should have a careful neurovascular examination to rule out injury to the underlying brachial plexus and subclavian artery and vein.**
- The presence of "**hard signs**" following a fracture indicate definite arterial injury and require **immediate surgical intervention**.
- However, the presence of "**soft signs**" also suggest an arterial injury may have occurred and **require vascular imaging to confirm and localize the site of injury**. **CT angiography is the diagnostic modality of choice due to its high sensitivity and specificity and rapid procedure time.**
- In absence of hard or soft signs → **placing the arm in a sling**. Figure-of-8 bandage treatment is now less popular.



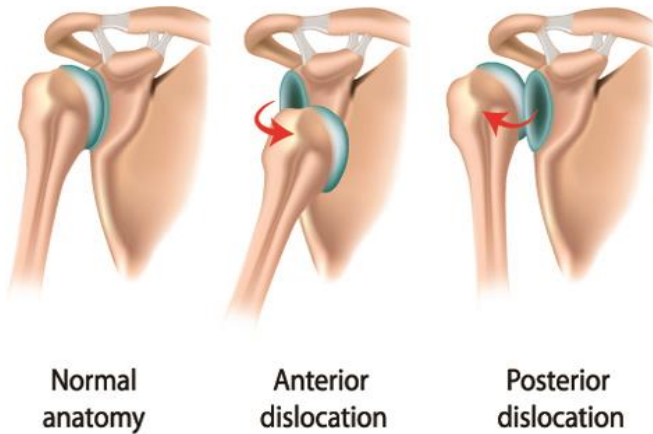
Signs of traumatic arterial injury	
Hard signs (require immediate surgery)	<ul style="list-style-type: none"> • Distal limb ischemia (eg, paralysis, pain, pallor, poikilothermia) • Absent distal pulses • Active hemorrhage or rapidly expanding hematoma • Bruit or thrill at site of injury
Soft signs (require further imaging)	<ul style="list-style-type: none"> • Diminished distal pulses • Unexplained hypotension • Stable hematoma • Documented hemorrhage at time of injury • Associated neurologic deficit

■ Anterior dislocation of the shoulder:

- The glenohumeral joint is the most commonly dislocated joint in the body due to the shallow articulation between the humeral head and the glenoid fossa of the scapula.
- The shoulder may dislocate anteriorly, inferiorly, or posteriorly, but anterior dislocations are by far the most common.
- Anterior dislocations are typically caused by a blow to an externally rotated and abducted arm.
- Patients hold the arm close to their body but rotated outward as if they were going to shake hands.
- When the head of the humerus is displaced anteriorly, there is flattening of the deltoid prominence, and anterior axillary fullness (due to the humeral head's displacement into this location).
- The axillary nerve is the nerve most commonly injured by anterior shoulder dislocations.
- It innervates the teres minor and deltoid (weakened shoulder abduction) muscles. It also provides sensory innervation to the skin overlying the lateral shoulder. Flattening of the deltoid muscle after a shoulder injury suggests anterior shoulder dislocation.
- AP and lateral x-rays are diagnostic.
- Some patients develop recurrent dislocations with minimal trauma.

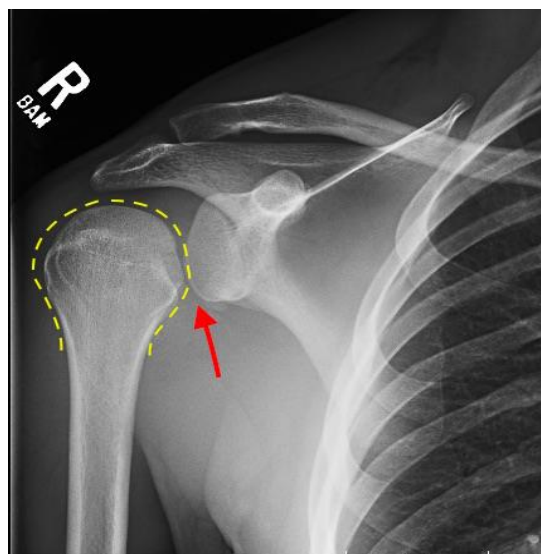


Shoulder Dislocation

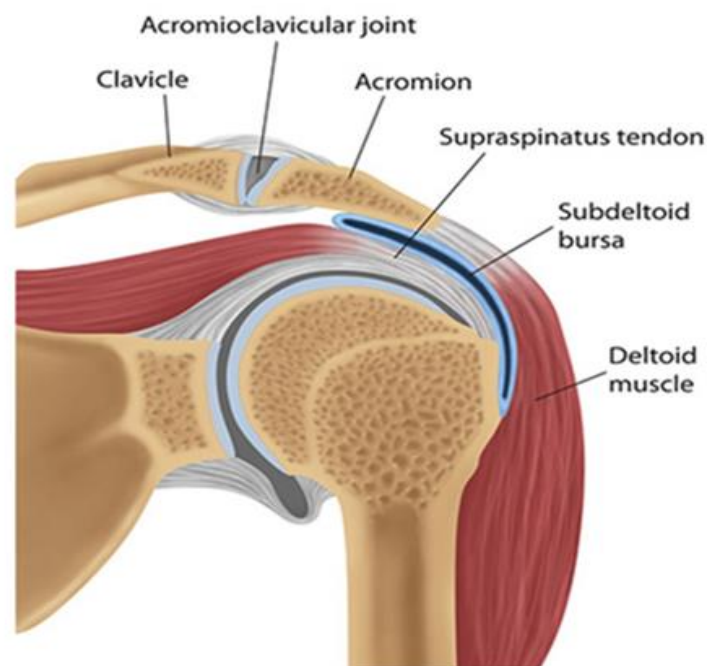
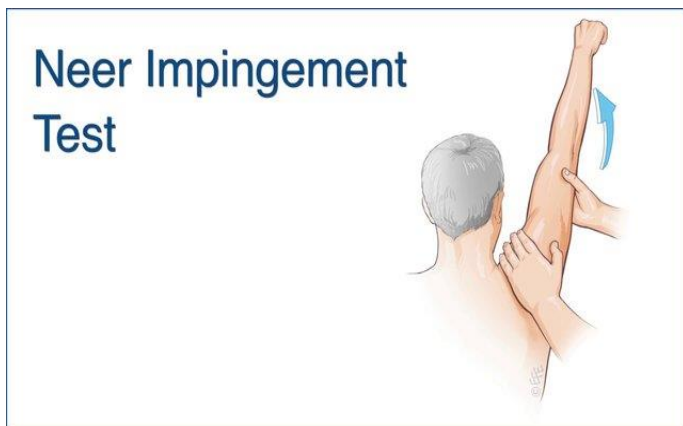


■ Posterior shoulder dislocation:

- **Posterior shoulder dislocation is rare** and occurs after massive uncoordinated muscle contractions, such as **epileptic seizure** or **electrical burn**.
- On examination, the arm is held **in adduction and internal rotation with impaired external rotation**, with flattening of the anterior aspect of the shoulder.
- X-rays show loss of the normal relation between the humeral head and glenoid and internal rotation of the humeral head.
- Most posterior dislocations are **managed with closed reduction**.

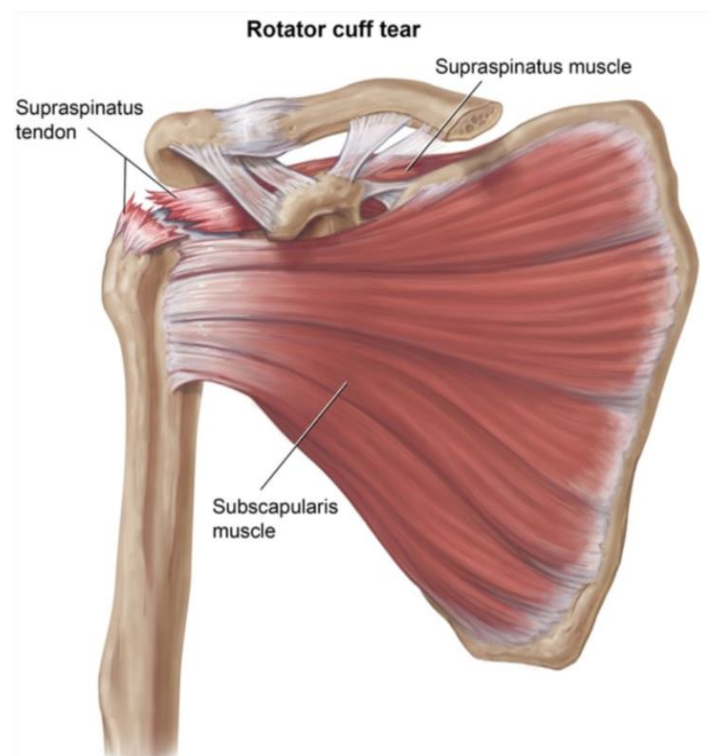


- Common causes of shoulder pain:
- A. Rotator cuff impingement or tendinopathy:
 - Rotator cuff tendinopathy results from repetitive activity above shoulder height (painting, ceilings) and is most common in middle-aged and older individuals.
 - In addition to the rotator cuff itself, pain may also emanate from the subacromial bursa and the tendon of the long head of the biceps.
 - On flexion or abduction of the humerus, the space between the humeral head and acromion is reduced, causing pressure on the supraspinatus tendon and subacromial bursa.
 - Impingement syndrome, a characteristic of RCT, refers to compression of these soft tissue structures.
 - Impingement can be demonstrated with the Neer test: With the patient's shoulder internally rotated and forearm pronated, the examiner stabilizes the scapula and flexes the humerus. Reproduction of the pain is considered a positive test.
 - Untreated, chronic RCT can increase the risk for rotator cuff tear.



- B. Rotator cuff tears:
 - The rotator cuff is formed by the tendons of the Supraspinatus, Infraspinatus, Teres minor, and Subscapularis muscles (SITS).
 - The supraspinatus is most commonly injured due to degeneration of the tendon with age and repeated ischemia induced by impingement between the humerus and the acromion during abduction.

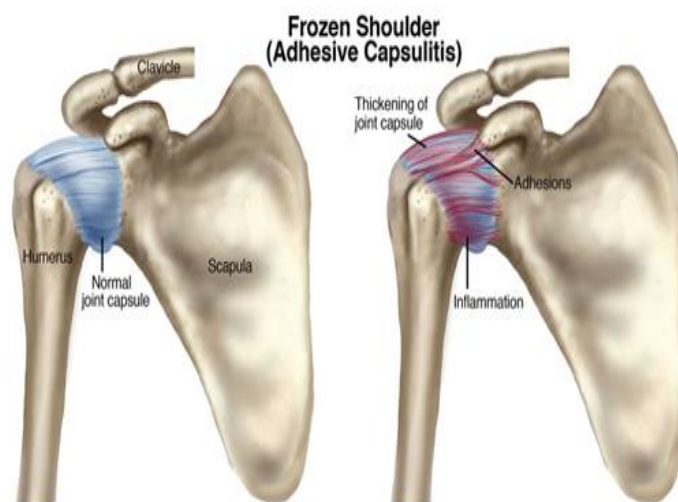
- A complete supraspinatus tear causes weakness of abduction, which can be appreciated in the drop arm test.
- In this test, the patient's arm is abducted above the head and the patient is asked to lower the arm slowly. With a complete tear, the patient will be unable to lower the arm smoothly and it will drop rapidly around mid-abduction.
- Although the supraspinatus is the primary muscle responsible for initiating the first 15 degrees of abduction, the loss of smooth adduction in the drop arm test typically occurs when the humerus is near the horizontal plane.
- MRI can confirm the diagnosis, and treatment usually requires surgery.



Rotator cuff tendinopathy & tear	
Rotator cuff impingement or tendinopathy	<ul style="list-style-type: none"> • Pain with abduction, external rotation • Subacromial tenderness • Normal range of motion with positive impingement tests (eg, Neer, Hawkins)
Rotator cuff tear	<ul style="list-style-type: none"> • Similar to rotator cuff tendinopathy • Weakness with abduction & external rotation • Age >40

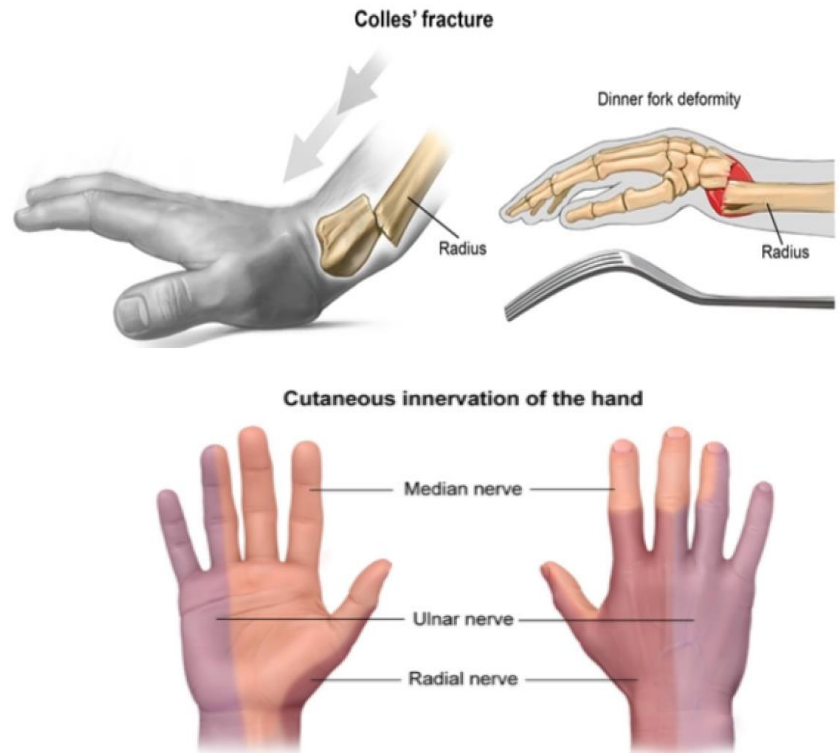
C. Adhesive capsulitis (AC):

- Adhesive capsulitis (frozen shoulder syndrome) is characterized by **markedly decreased range of motion (passive and active range of motion)**.
- The glenohumeral joint loses its normal distensibility due to **chronic inflammation, fibrosis, and contracture of the joint capsule**.
- Patients with AC typically report a gradual onset of shoulder stiffness, with or without mild pain, that limits their ability to flex, abduct (reach overhead), or rotate the humerus.
- AC can be idiopathic or secondary to underlying conditions such as rotator cuff tendinopathy (most common), subacromial bursitis, paralytic stroke, diabetes mellitus, or humeral head fracture.
- Diagnosis is confirmed on examination with **>50% reduction in both passive and active ROM**.



■ Colles' fracture:

- Colles' fracture results from **falling on an outstretched hand, particularly in athletes (high-impact falls) or elderly patients with osteoporosis (low- impact falls)**.
- Characteristic manifestations include **pain, swelling, and dinner fork deformity of the wrist**. In addition, severely displaced fractures may result in **neurovascular compromise**; therefore, careful assessment of pulse, capillary refill, and sensation is indicated.
- Dorsal displacement of the radius can result in **compression of the median nerve**, which enters the wrist through the carpal tunnel and **provides sensation to the lateral 3 and 1/2 digits and motor innervation to the thenar muscles** (opponens pollicis, abductor pollicis brevis).
- **Compression results in acute carpal tunnel syndrome symptoms, including paresthesia of the affected digits and impaired thumb abduction by abductor pollicis brevis.**
- Treatment is with **close reduction and long arm cast**.



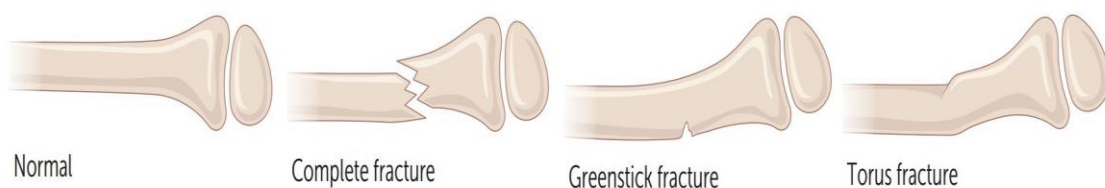
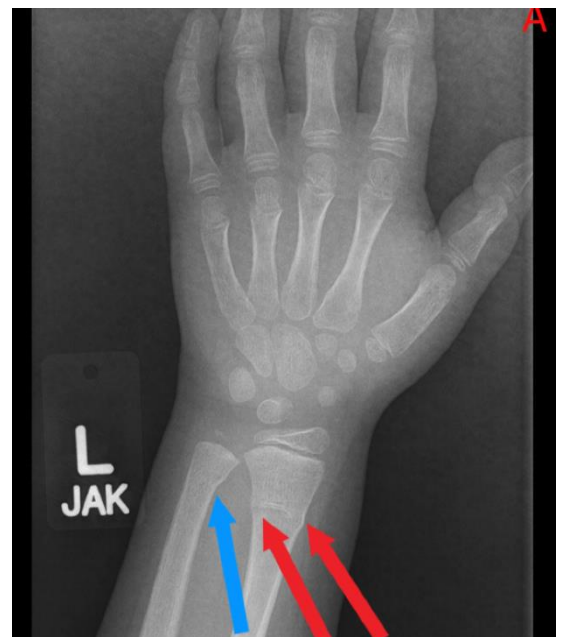
- Monteggia fracture:
- Monteggia fracture results from a **direct blow to the ulna (on a raised protective arm hit by a nightstick)**.
- There is **diaphyseal fracture of the proximal ulna, with anterior dislocation of the radial head**.
- Galeazzi fracture is the **mirror image**: the distal third of the radius gets the direct blow and has the fracture, and there is dorsal dislocation of the distal radioulnar joint.
- **MUGR** (Monteggia → Ulnar fracture, Galeazzi → Radial fracture).
- In both of these, **the broken bone often requires open reduction and internal fixation**, whereas the **dislocated one is typically handled with closed reduction**.



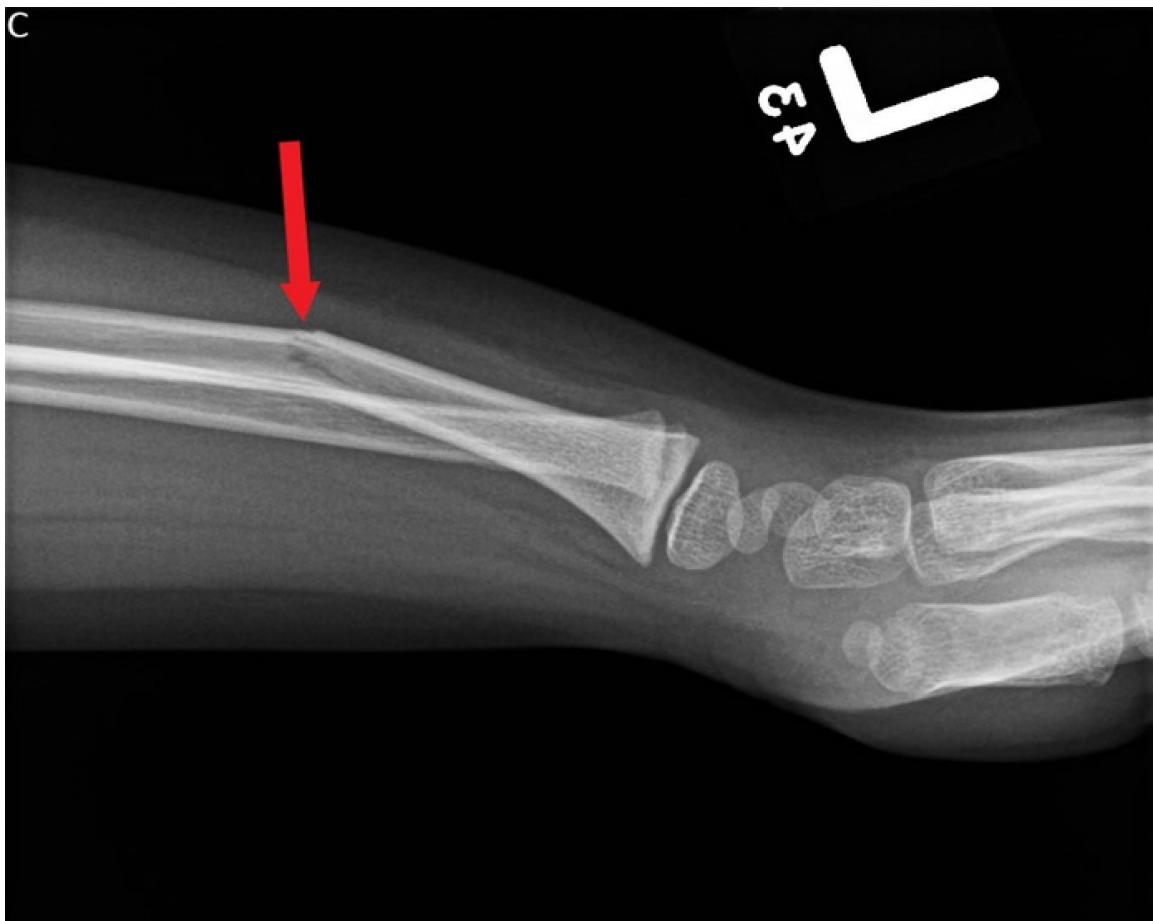
- Buckle (Torus) fracture:
 - Forearm fractures are common in children and most frequently involve the distal radius and/or ulna after a fall onto an outstretched hand.
 - Young children (age <10) have porous bone, which allows for compression of the distal metaphyseal cortex after an injury.
 - This results in a buckle fracture, which is considered a **stable and incomplete fracture** because it involves only one side of the bone.
 - Buckle fractures present with pain and tenderness over the fracture site. Wrist range of motion may be intact or slightly limited, and swelling is often absent.
 - X-ray is required to confirm the diagnosis and exclude a greenstick or complete fracture. Rather than a fracture line, a cortical bulge (blue and red arrows) is visualized.
 - Treatment involves pain control and prevention of reinjury (splint placement).
 - Most buckle fractures heal within a few weeks without complication.

Incomplete fracture

- Greenstick fracture
fracture through one cortex
- Torus fracture
buckling of the cortex
(*buckling fracture*)



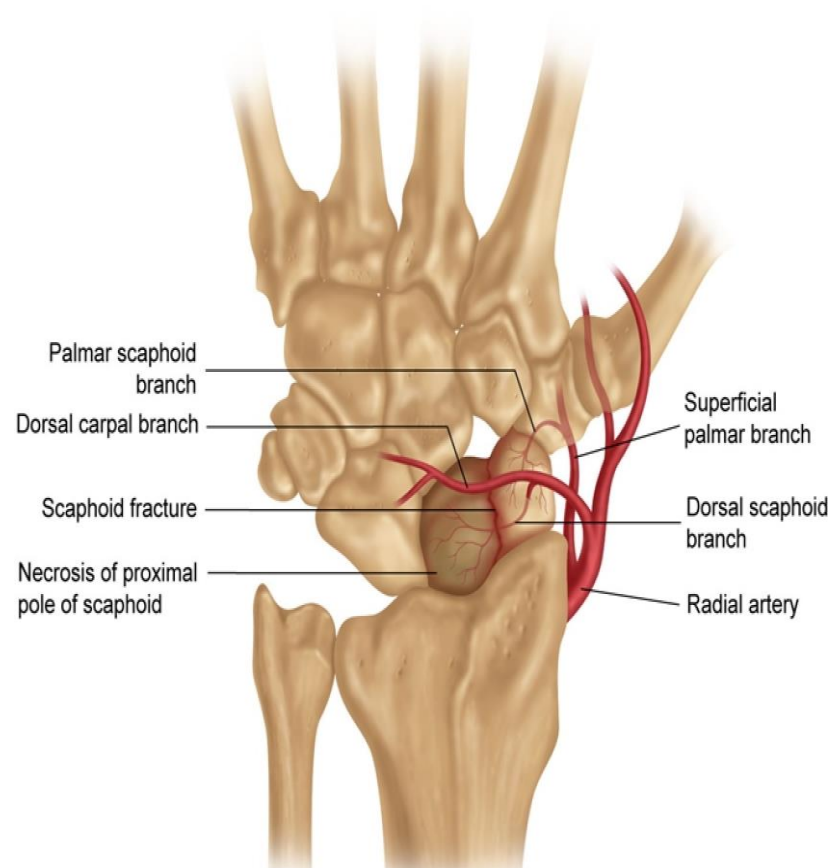
- Greenstick fracture:
 - Greenstick fractures of the forearm are common in children due to **relatively strong periosteum, which limits the fracture line from extending through the width of the bone.**
 - Because the periosteum surrounding the bone is thick and strong in children, the fracture may involve only one side of the bony cortex rather than extending through the width of the bone.
 - The opposite side appears to have a deformation or bend **without a break in the cortex.**
 - Prompt reduction (if displaced) and immobilization of the forearm is required for greenstick fractures because **they are considered unstable due to the potential for refracture or further displacement if improperly treated.**
 - Repeat x- rays should be performed prior to cast removal to confirm bony union.
 - **Once the fracture is fully healed, no long-term complications are expected.**



■ Fracture of the scaphoid:

- Scaphoid fractures are **the most common carpal bone fractures**.
- They frequently result from **falls onto an outstretched** hand that cause axial compression or wrist hyperextension.
- Chief complaint is **typically wrist pain, with physical exam revealing localized tenderness to palpation over the anatomic snuff box**.
- **Scaphoid fractures carry a significant risk of osteonecrosis** because the blood supply enters at the distal pole and flows proximally and can be disrupted by the fracture.
- In undisplaced fractures, x-rays are usually **negative, but thumb spica cast is indicated just with the history and physical findings**. If immediate diagnosis is needed, CT or MRI of the wrist can confirm the **fracture**. Wrist immobilization with a cast can be considered for nondisplaced fractures, **but patients should be monitored with serial x-ray to rule out osteonecrosis of the proximal segment and nonunion of the fracture**.
- **Displaced** scaphoid fractures may be visible on x-ray immediately following injury. Displaced fractures should be considered for **surgical intervention** (open reduction and internal fixation are needed).

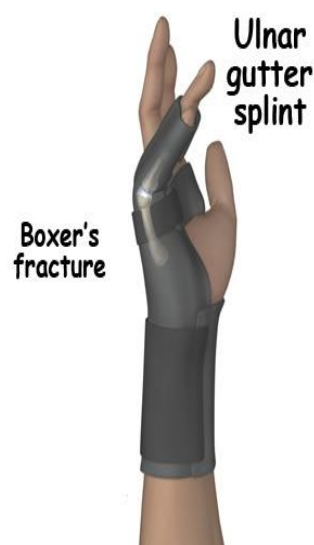
Scaphoid avascular necrosis



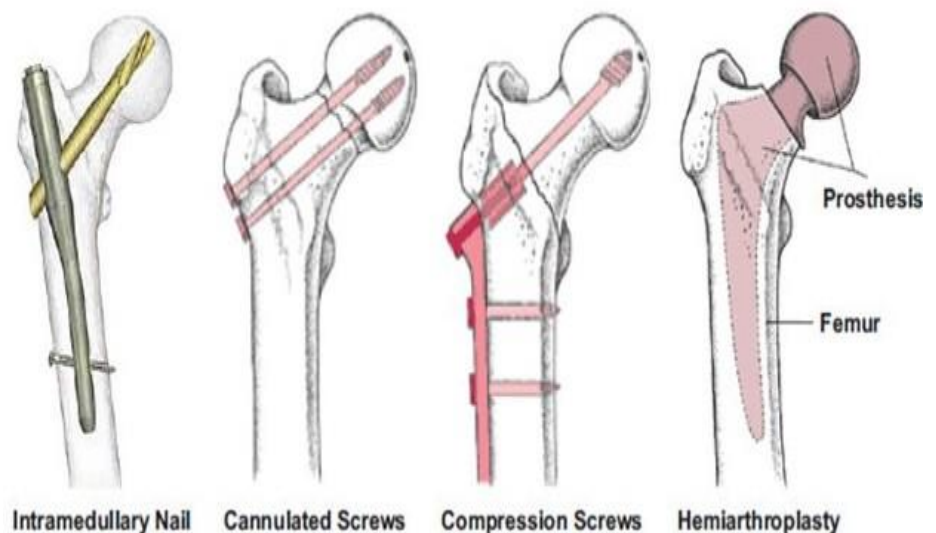
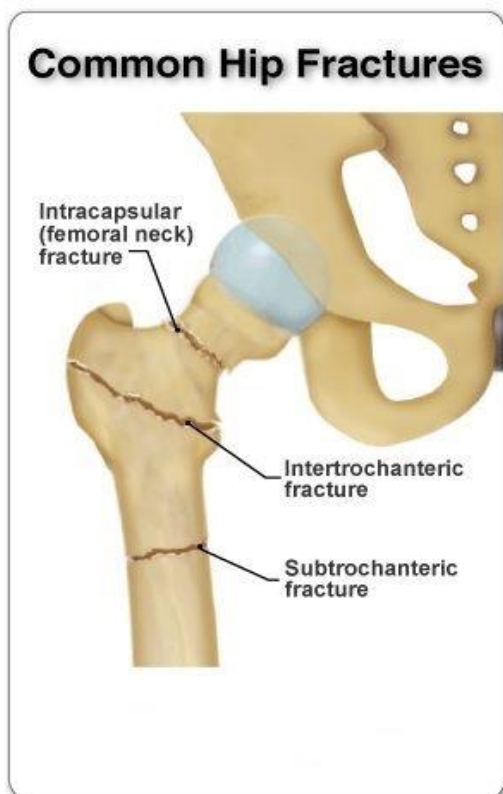


▪ Metacarpal neck fracture:

- Metacarpal neck fracture (typically the fourth or fifth, or both) happens when a **closed fist hits a hard surface (like a wall)**.
- The hand is swollen and tender, and x-rays are diagnostic.
- Treatment **depends on the degree of angulation, displacement, or rotary malalignment:**
 - **Mild** fractures → close reduction and ulnar gutter splint.
 - **Severe** fractures → K (Kirschner) wire or plate fixation.

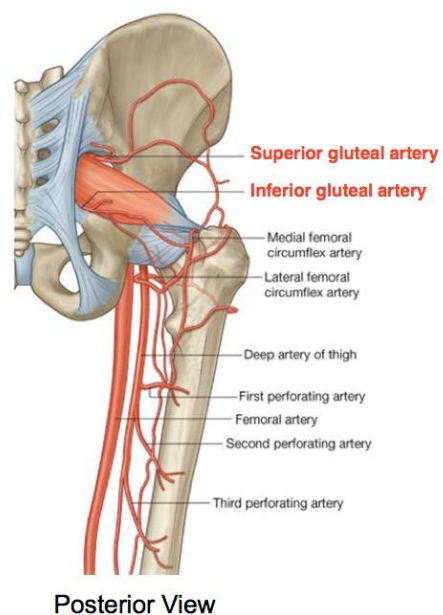
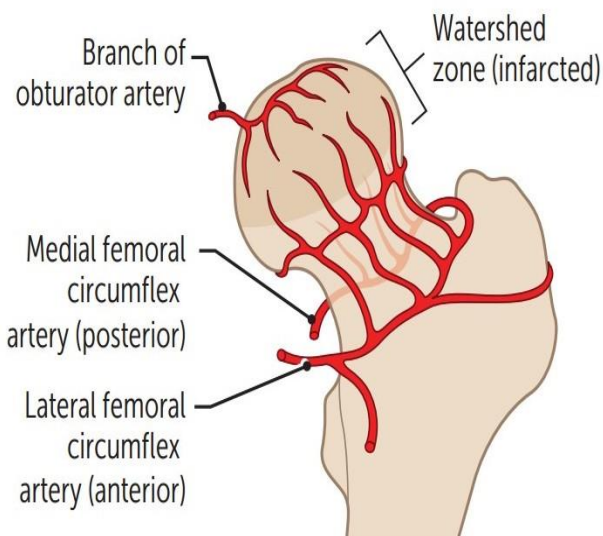
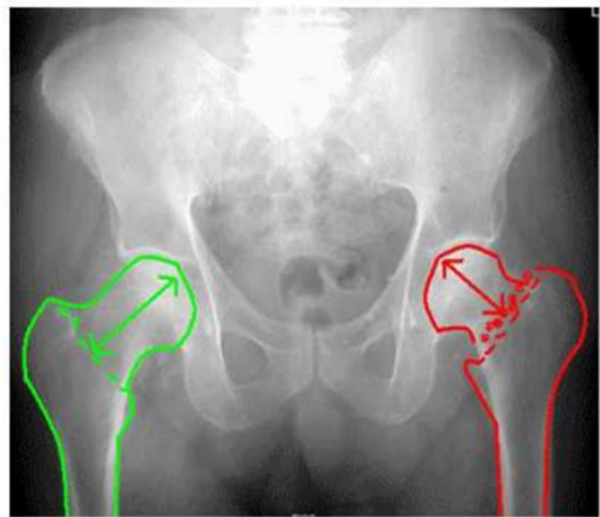


- Hip fracture:
 - Hip fracture typically occurs in the **elderly following a fall**.
 - Hip fractures are classified by anatomic location and fracture type into either **intracapsular** (femoral neck and head) or **extracapsular** (intertrochanteric, subtrochanteric).
 - **Intracapsular fractures have a higher chance of avascular necrosis**; extracapsular fractures have a greater need for implant devices (nails, rods). The specific surgical intervention required depends largely on the details of the individual case.
 - In general, **surgical repair should be done as soon as feasible to relieve pain, minimize complications, and reduce length of hospital stay**. **However, surgery may be delayed up to 72 hours to evaluate surgical risk and ensure medical stability**.
 - The unavoidable immobilization that ensues poses a **very high risk for deep venous thrombosis and pulmonary emboli**, thus post-op anticoagulation is recommended.
 - **If multiple fracture, fat embolism syndrome may develop**, in which severe respiratory distress occurs secondary to marrow fat entering the blood stream and embolizing to the pulmonary vasculature.



■ Femoral neck fracture:

- Femoral neck fracture, particularly if displaced, compromises the very tenuous blood supply of the femoral head.
- The femoral head and neck derive their blood supply from the **superior and inferior gluteal arteries** and **the medial and lateral femoral circumflex arteries**, vessels that together form the **trochanteric anastomosis**.
- The **medial femoral circumflex artery** makes the largest contribution to the blood supply of this region and is **vulnerable to damage from femoral neck fractures due to its close association with the posterior aspect of the femoral neck**. Injury of this vessel predisposes to avascular necrosis of the femoral head.
- Faster healing and earlier mobilization can be achieved by **replacing the femoral head with a prosthesis**.



- Avascular necrosis of femoral head:

- Etiology:

- Osteonecrosis is caused by occlusion of end arteries supplying the femoral head, leading to **necrosis and collapse of the periarticular bone and cartilage**.
- The femoral head has 2 main sources of blood: the ascending arteries and the foveal artery, which lies within the ligamentum teres. **The foveal artery is patent early in life, but may become obliterated in older patients**. For this reason, **aseptic necrosis of the femoral head is uncommon in children, but the risk rises in older patients**.
- Osteonecrosis can occur in **disorders that disrupt the circulation of bone through micro-occlusion, abnormal endothelial function, or increased intra-osseous pressure**.
- It is a common complication of **SLE**, and the risk is greatly increased in patients **treated with glucocorticoids**.
- Osteonecrosis is **common in patients with sickle cell disease** due to disruption of microcirculation in the bone by sickling as well as increased intraosseous pressure due to bone marrow hyperplasia.

- Clinical finding:

- Osteonecrosis of the femoral head is characterized by **pain in the groin, thigh, or buttock that is worsened by activity**.
- Early examination findings may be normal, but progression of the disease can lead to **reduced range of motion (particularly internal rotation and abduction) and joint instability**.

- Imaging:

- In the first few months, x-rays also will often be normal, and **MRI is a more sensitive test**.
- MRI can visualize the boundary between normal and ischemic bone, as well as the zone of hypervascularity. In advanced disease, plain x-rays may show subchondral lucency (crescent sign) and deformities of the femoral head.

- Management:

- Conservative treatment: indicated in early stages of disease to the reduce risk of femoral head collapse (Core decompression, Bone graft).
- Surgery: indicated in later stages of disease when bone structure is compromised (Hip replacement for advanced disease).

Avascular necrosis	
Etiology	<ul style="list-style-type: none"> • Steroid use • Alcohol abuse • Systemic lupus erythematosus • Antiphospholipid syndrome • Hemoglobinopathies (eg, sickle cell) • Infections (eg, osteomyelitis, HIV) • Renal transplantation • Decompression sickness
Clinical manifestations	<ul style="list-style-type: none"> • Groin pain on weight bearing • Pain on hip abduction & internal rotation • No erythema, swelling, or point tenderness
Laboratory findings	<ul style="list-style-type: none"> • Normal white blood cell count • Normal ESR & CRP
Radiologic imaging	<ul style="list-style-type: none"> • Crescent sign seen in advanced stage • MRI is most sensitive modality

CRP = C-reactive protein; ESR = erythrocyte sedimentation rate.

- Intertrochanteric fracture:
- Intertrochanteric fracture is **less likely to lead to avascular necrosis** and is usually treated with **open reduction and pinning**.
- The unavoidable immobilization that ensues poses a **very high risk for deep venous thrombosis and pulmonary emboli**, thus post-op anticoagulation is recommended.



▪ Femoral shaft fracture:

- Femoral shaft fracture is often treated with **intramedullary rod fixation**.
- If **bilateral and comminuted**, it may produce enough internal blood loss to lead to **shock** (external fixation may help while the patient is stabilized).
- If **open**, it is an orthopedic emergency, requiring **OR irrigation and closure within 6 hours**.
- If **multiple, fat embolism syndrome may develop**, in which severe respiratory distress occurs secondary to marrow fat entering the blood stream and embolizing to the pulmonary vasculature.



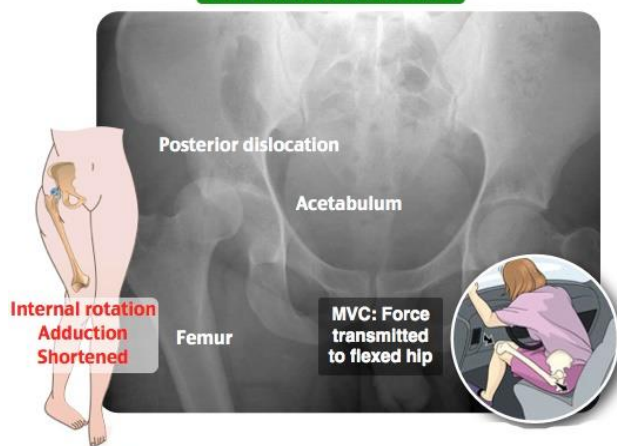
▪ Posterior dislocation of the hip:

- Posterior dislocation of the hip occurs when **the femur is driven backward**, such as in a head-on car collision where the **knees hit the dashboard**.
- The patient has hip pain and lies in the stretcher with **the leg shortened, adducted, and internally rotated** (in a broken hip the leg is also shortened, but it is **externally rotated**).
- Because of the tenuous blood supply of the femoral head, **emergency reduction is needed to avoid avascular necrosis** (closed reduction if there is no associated pathological fracture).



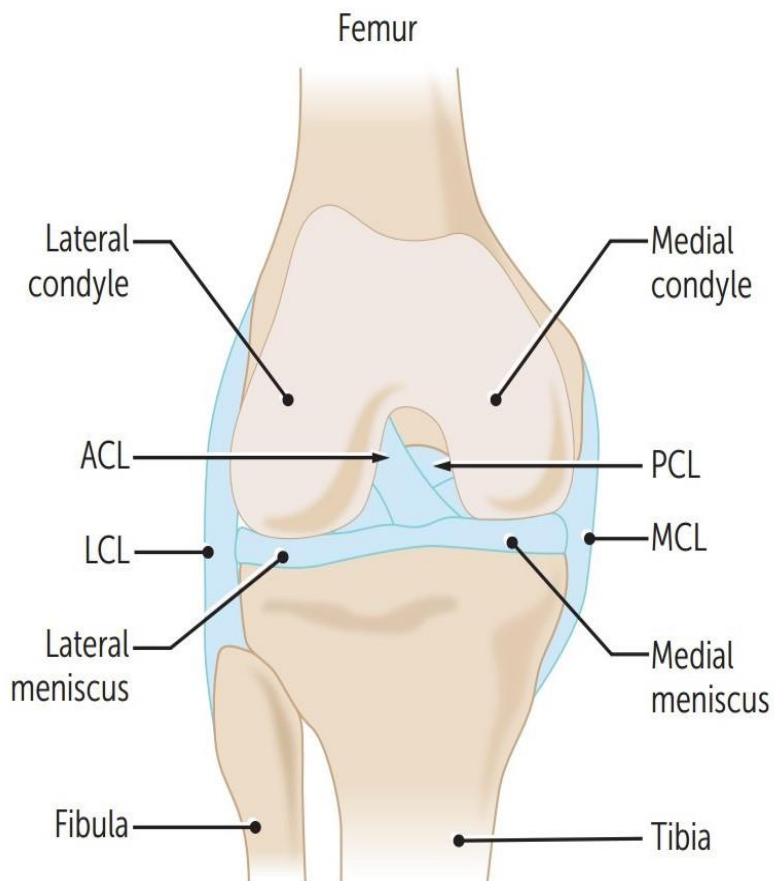
Posterior Hip Dislocation

Most common hip dislocation



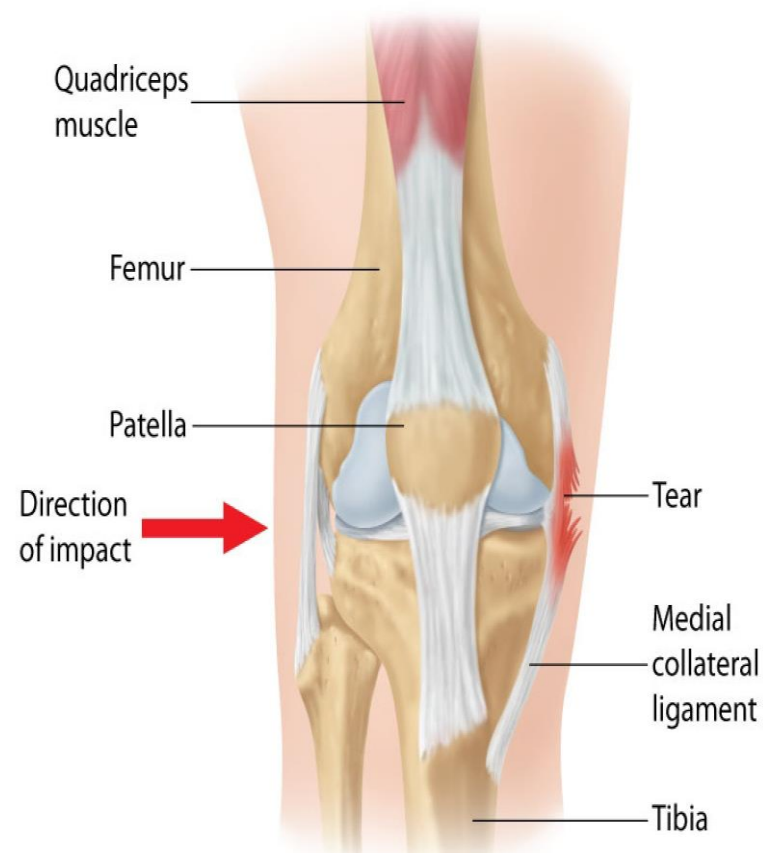
▪ Knee injury:

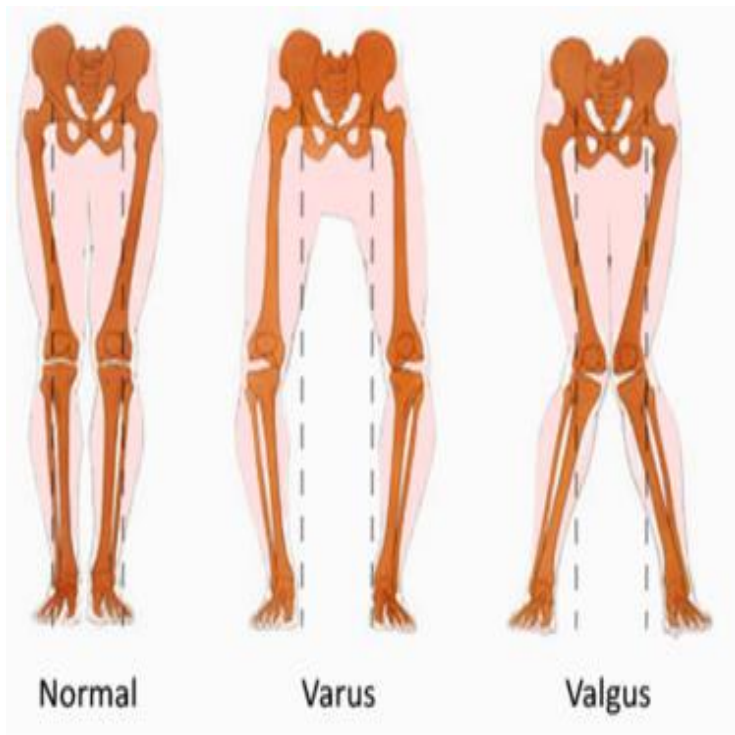
- Knee injury typically produces **swelling of the knee**; knee pain without swelling is unlikely to be a serious knee injury.
- Collateral ligament injury is usually sustained when the force of impact is at the side of the knee, a common sports injury. Medial blows disrupt the lateral ligament and vice versa.
- Isolated uncomplicated injuries are treated with a **conservatively** with **RICE** measures (**R**est, **I**ce, **C**ompression, and **E**levation and analgesics with progressive return to activity as tolerated).
- When several ligaments are torn, severe knee instability, or patients with physically demanding occupations **surgical repair is preferred**.



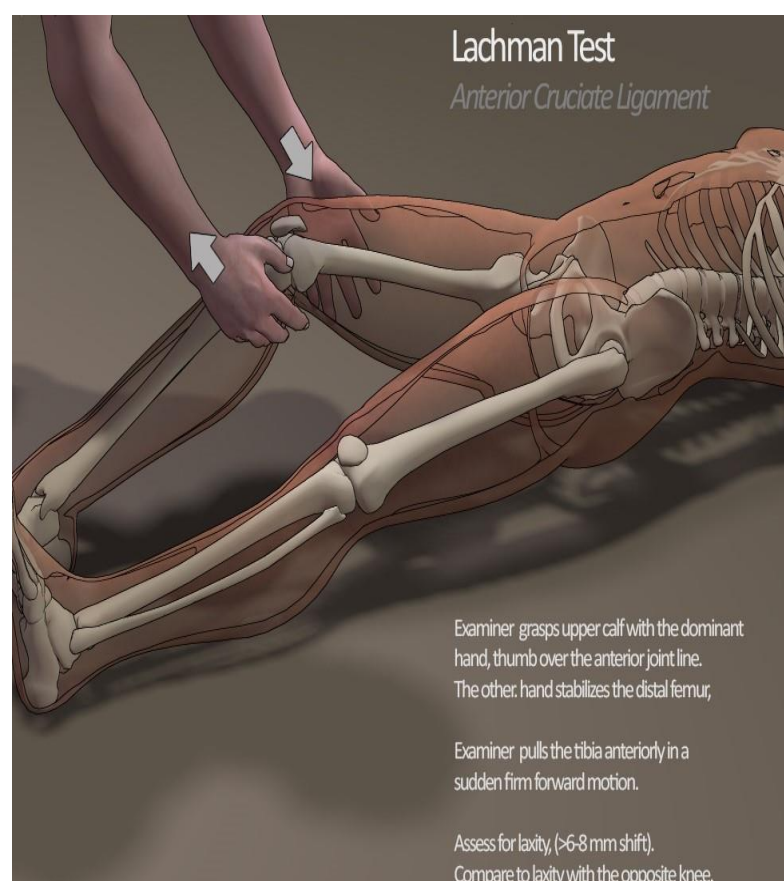
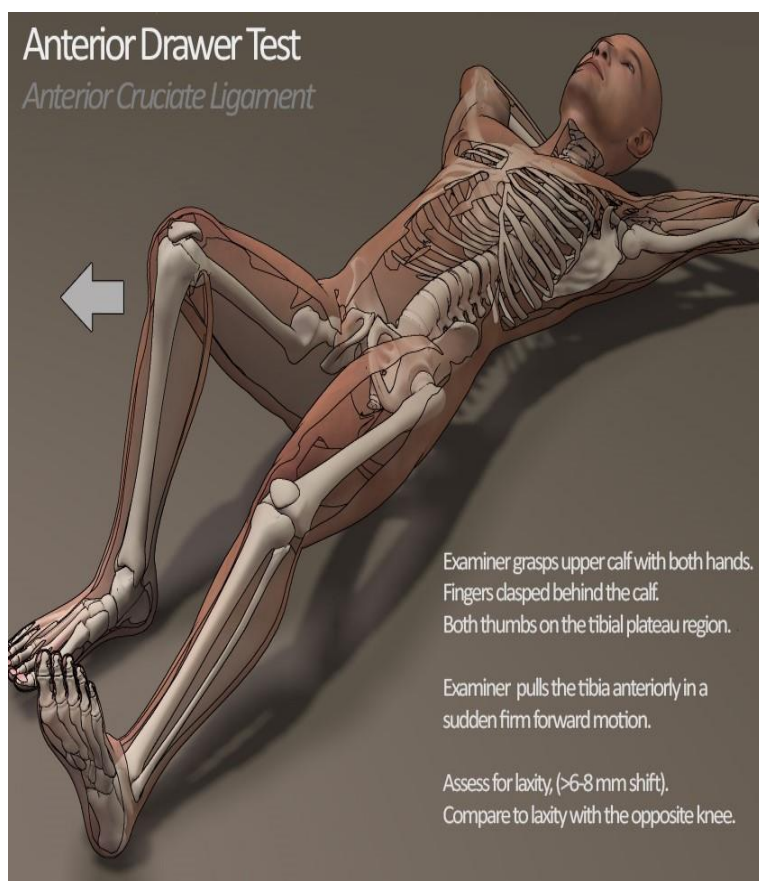
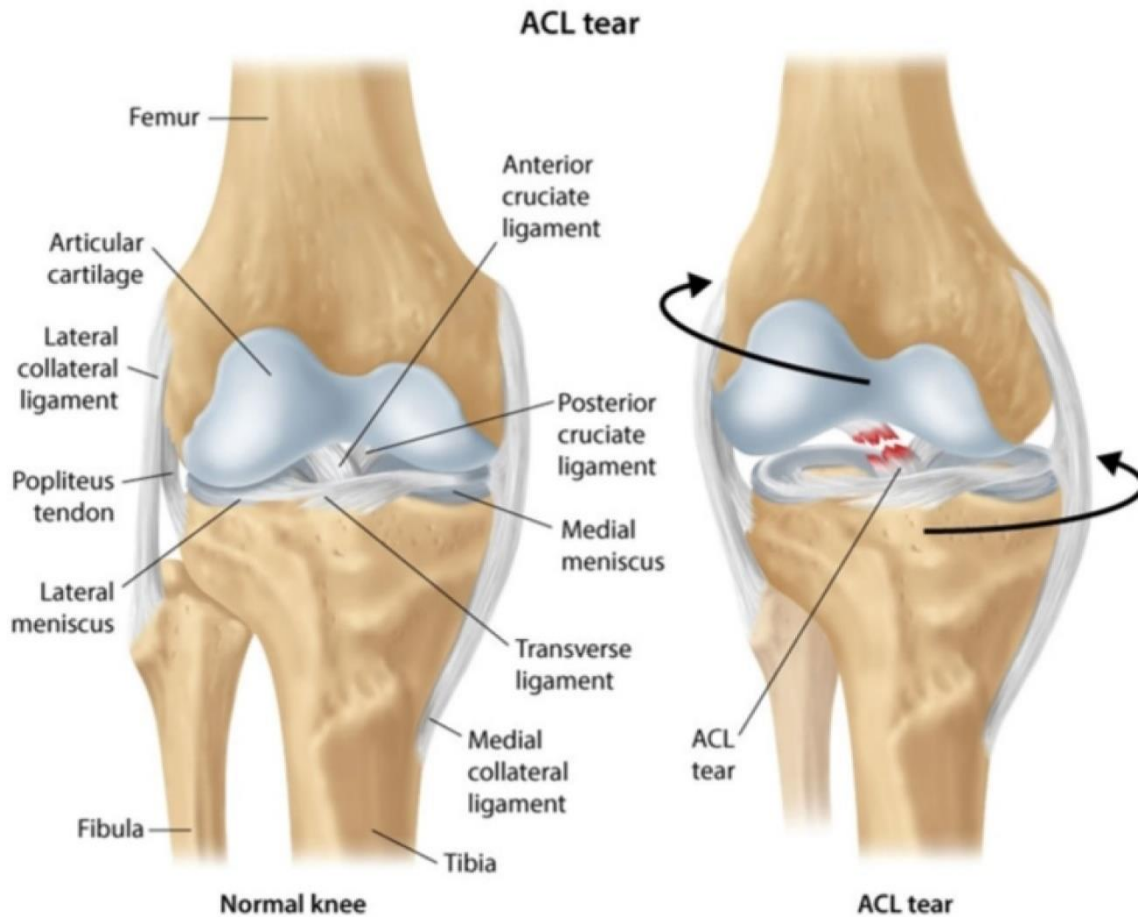
- Medial collateral ligament (MCL) tear:
- Medial collateral ligament (MCL) tear is a common knee injury caused by severe valgus stress (blow to the lateral knee) or twisting injury.
- Examination findings may include ecchymosis and joint line tenderness at the medial knee.
- Appreciable laxity when the leg is forced into abduction (valgus stress test) is helpful for diagnosis.
- Acute effusion/hemarthrosis is uncommon unless there is concurrent injury to the anterior cruciate ligament.
- MRI is the most sensitive test for diagnosis but is generally reserved for patients being considered for surgical intervention.
- Patients with uncomplicated MCL tears can be managed nonoperatively with Rest, Ice, Compression, and Elevation (RICE measures) and analgesics with progressive return to activity as tolerated.

Medial collateral ligament injury



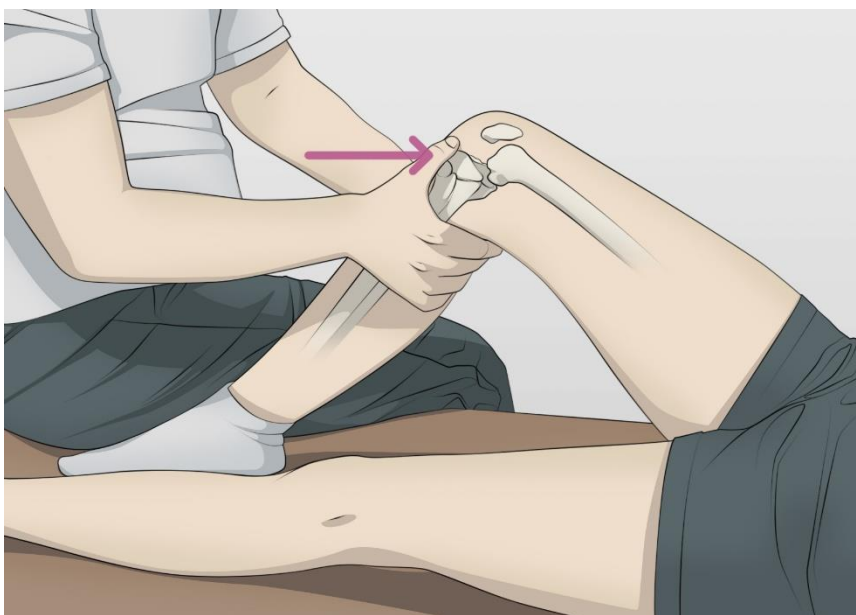


- Anterior cruciate ligament injury:
- Anterior cruciate ligament injury is more common than posterior injury.
- Injuries to the anterior cruciate ligament (ACL) are common in young athletes, especially in sports requiring rapid direction changes on the lower extremity (soccer, basketball, tennis).
- Patients with partial- or full-thickness ACL tears typically experience a "popping" sensation in their knee during the injury followed by rapid onset of hemarthrosis and a feeling of instability when bearing weight on the affected side.
- Patients with an ACL tear will show laxity at the knee with the tibia able to be pulled forward relative to the femur.
- Two such maneuvers - the Lachman test and the anterior drawer sign - are highly sensitive (>90%) and specific for ACL injuries. The diagnosis is usually confirmed on MRI.
- With the knee flexed 90°, the leg can be pulled anteriorly, like a drawer being opened (anterior drawer test).
- A similar finding can be elicited with the knee flexed at 20° by grasping the thigh with one hand, and pulling the leg with the other (Lachman test).



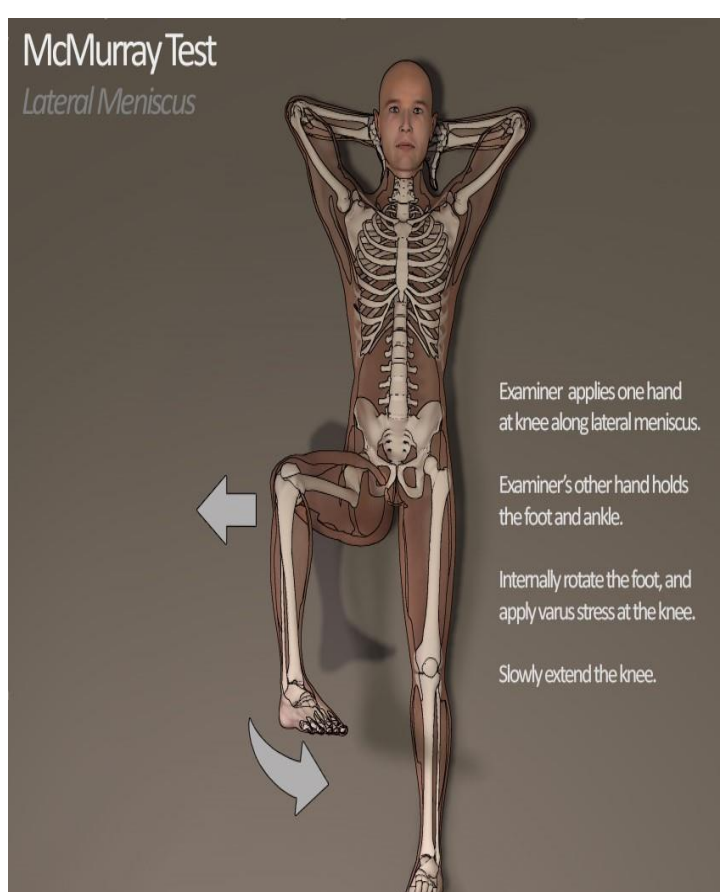
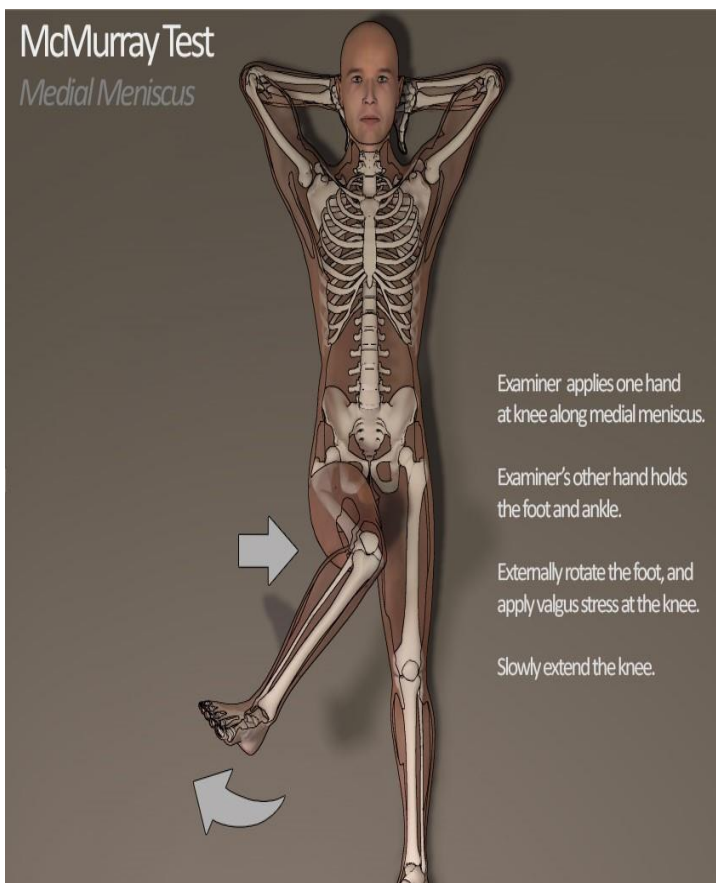
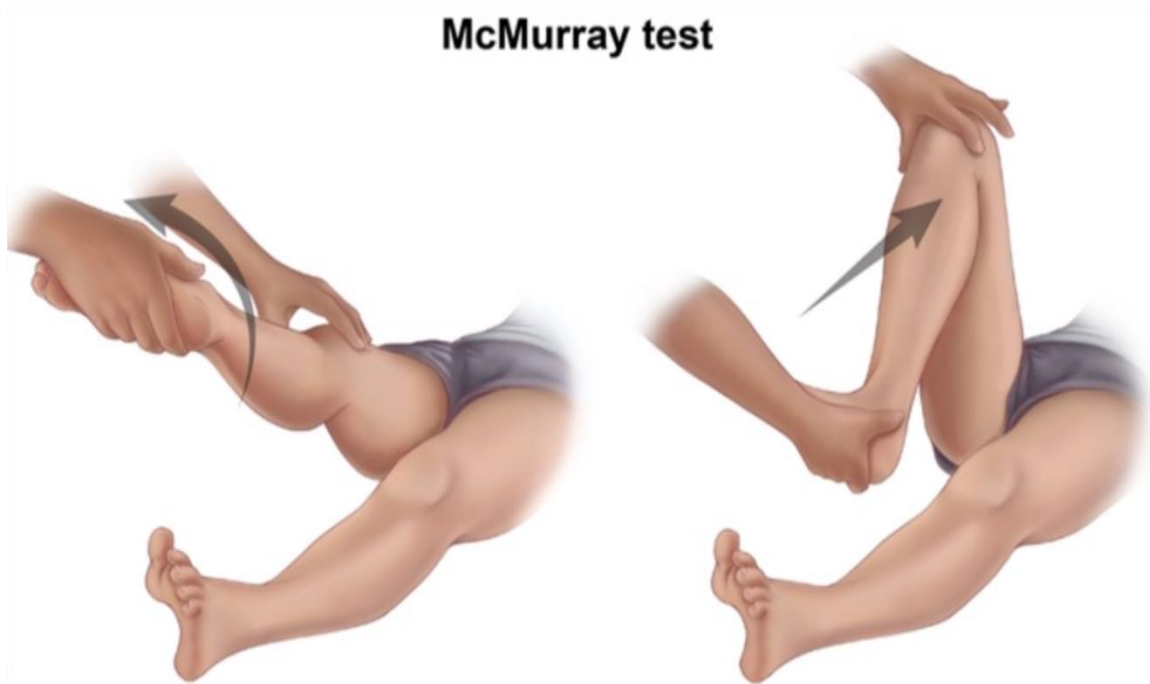
Features of anterior cruciate ligament injury	
Injury mechanisms	<ul style="list-style-type: none"> • Rapid deceleration or direction changes • Pivoting on lower extremity with foot planted
Symptoms	<ul style="list-style-type: none"> • Pain: rapid onset, may be severe • A "popping" sensation at the time of injury • Significant swelling (effusion/hemarthrosis) • Joint instability
Examination findings	<ul style="list-style-type: none"> • Anterior laxity of tibia relative to femur (anterior drawer test, Lachman test)
Diagnosis	<ul style="list-style-type: none"> • Magnetic resonance imaging
Treatment	<ul style="list-style-type: none"> • RICE (rest, ice, compression, elevation) measures • ± Surgery

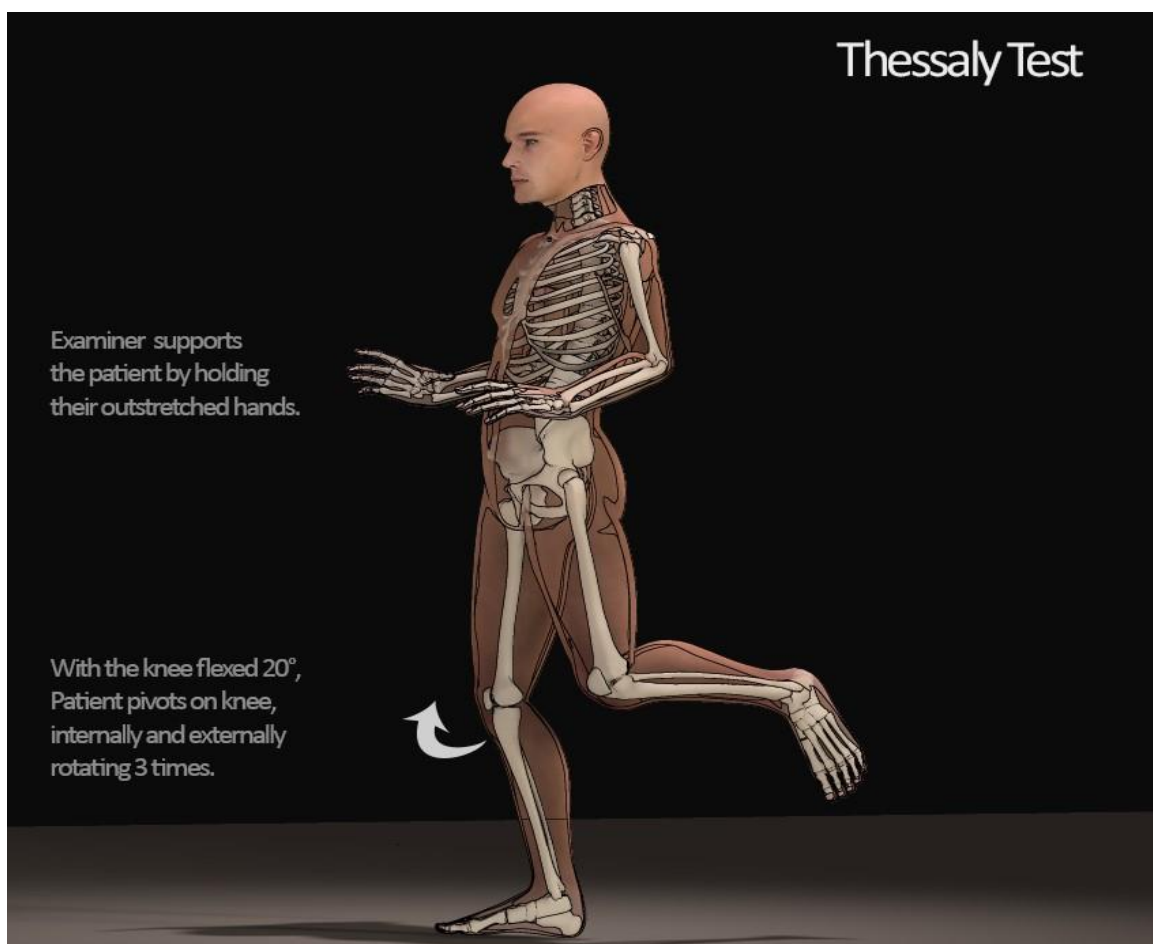
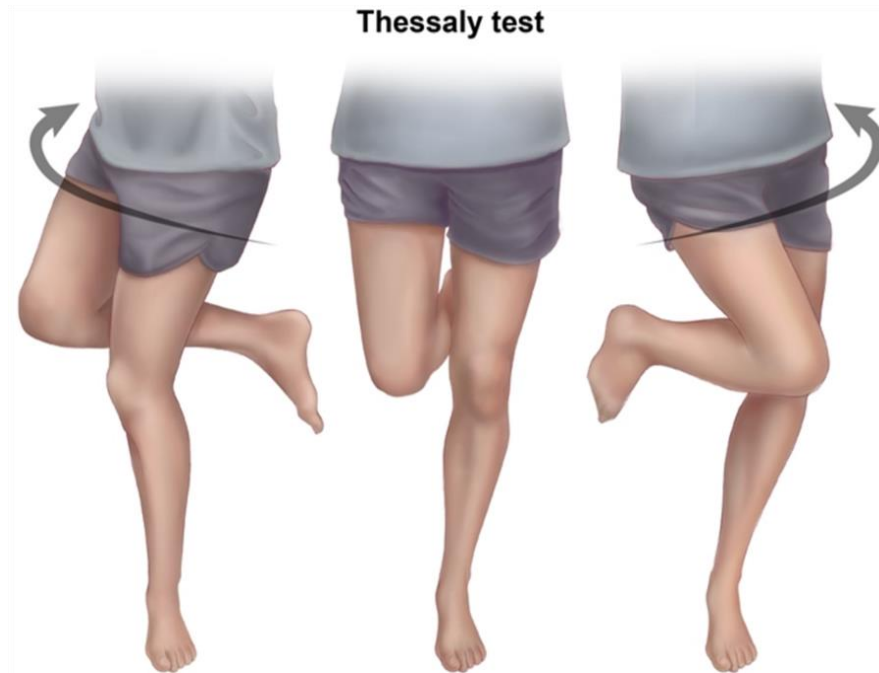
- Posterior cruciate ligament injury:
 - Posterior cruciate ligament injury produces **the opposite findings**.
 - MRI is diagnostic.
 - Sedentary patients may be treated with immobilization and rehabilitation, whereas athletes require arthroscopic reconstruction.



- Meniscal tear:
 - Tears of the medial meniscus often result from **twisting force with the foot fixed in young patients or due to degeneration of meniscal cartilage in older patients.**
 - **The medial meniscus is more commonly injured than the lateral.**
 - Injuries to the medial meniscus, medial collateral, and anterior cruciate often occur simultaneously (**Unhappy triad**).
 - Patients generally report a **popping sound followed by acute pain.**
 - Associated symptoms can include **catching, locking, reduced range of motion (a piece of torn meniscus get stuck between the condyles) and slow onset joint effusion.** Because the meniscus is not directly perfused, the effusion typically is not apparent for many hours.
 - Examination will show tenderness at the joint line on the affected side. Patients may also have **palpable locking or catching when the joint is rotated or extended while under load (Thessaly, McMurray tests).**
 - **Older** patients with tears due to chronic degeneration of the cartilage may show **osteoarthritic changes on x-ray**, but plain films are usually normal in young patients with traumatic tears.
 - **Diagnosis is confirmed with MRI or arthroscopy.**
 - Patients with short-term symptoms and minimal limitations in activity, or older patients with chronic tears due to degeneration of the cartilage, can be managed conservatively with rest and nonsteroidal anti-inflammatory drugs. **But young patients who have significant symptoms lasting >3-4 weeks should be considered for surgical intervention to relieve the symptoms and reduce the risk of further joint injury.**

Meniscal tears	
Etiology	<ul style="list-style-type: none"> • Younger patients: rotational force on planted foot • Older patients: degeneration of meniscal cartilage
Symptoms	<ul style="list-style-type: none"> • Acute "popping" sensation • Catching, locking, reduced range of motion • Slow-onset joint effusion
Examination	<ul style="list-style-type: none"> • Joint line tenderness • Pain or catching in provocative tests (Thessaly, McMurray)
Diagnosis	<ul style="list-style-type: none"> • MRI • Arthroscopy
Management	<ul style="list-style-type: none"> • Mild symptoms, older patients: rest, activity modification • Persistent symptoms, impaired activity: surgery

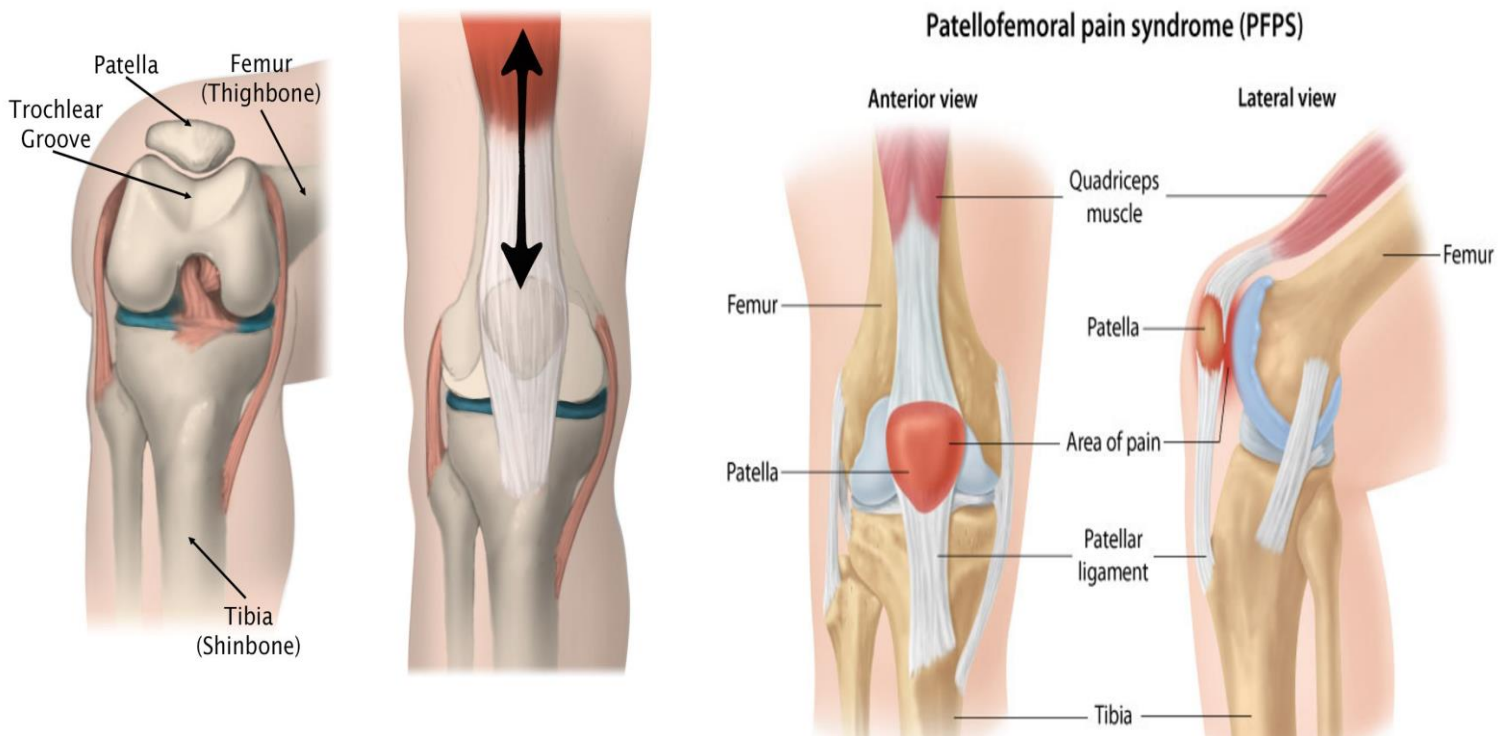




Special tests for knee examination	
MCL injury	Valgus stress test <ul style="list-style-type: none"> Stabilize lateral thigh; apply abduction force to lower leg Laxity indicates MCL injury
ACL injury	Anterior drawer test <ul style="list-style-type: none"> Patient supine with knee flexed Grip proximal tibia with both hands & pull anteriorly Lachman test <ul style="list-style-type: none"> Place knee at 30 degrees flexion Stabilize distal femur with 1 hand & pull proximal tibia anteriorly with the other Laxity of tibia indicates ACL injury
Meniscal tear	Thessaly test <ul style="list-style-type: none"> Patient stands on 1 leg with knee flexed 20 degrees Patient then internally & externally rotates on flexed knee McMurray test <ul style="list-style-type: none"> Passive knee flexion & extension while holding the knee in internal or external rotation Pain, clicking, or catching indicates meniscal tear

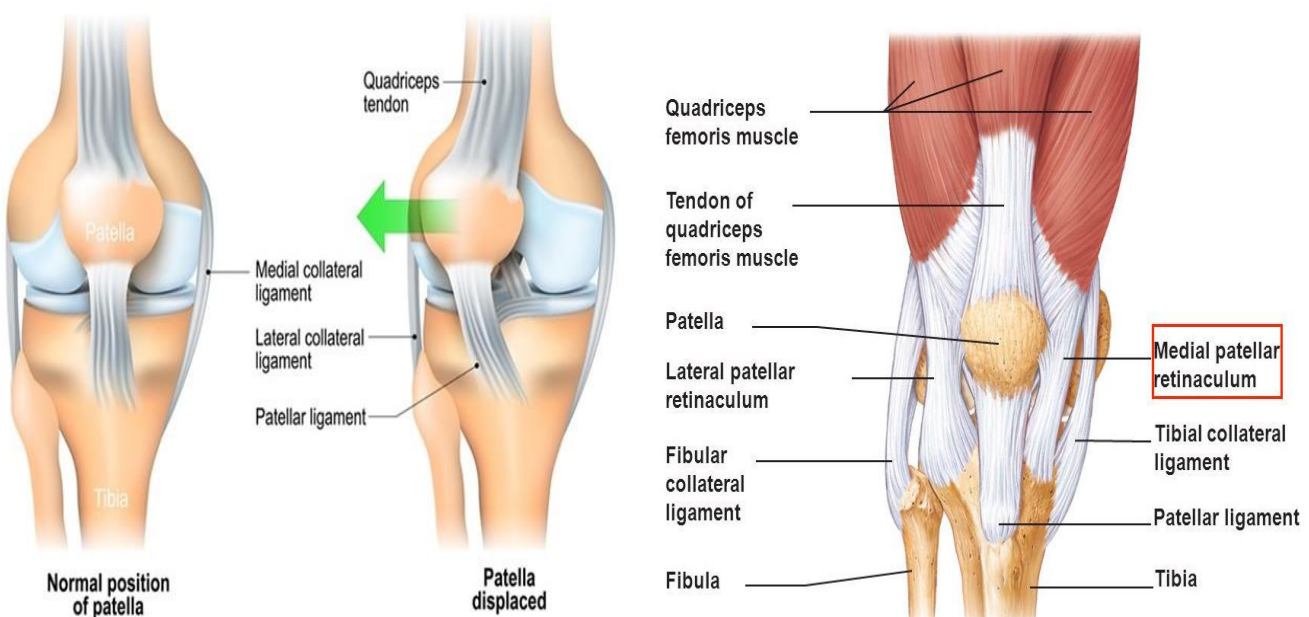
ACL = anterior cruciate ligament; **MCL** = medial collateral ligament.

- Patellofemoral pain syndrome (PFPS):
 - Patellofemoral pain syndrome (PFPS) is **one of the most common causes of chronic anterior knee pain in young athletes (more common in women)**.
 - It is usually related to **chronic overuse (runner's knee)** but can also occur acutely following trauma.
 - The precise source of pain is variable and not well understood, **but generally involves the track of the patella in the trochlear groove of the femur**.
 - Symptoms are typically worse during weight bearing with the knee flexed (**squatting, ascending/descending stairs**) or when running or sitting for extended periods.
 - The diagnosis of PFPS is primarily based **on characteristic history and examination findings**.
 - In general, the pain is provoked by maneuvers (squatting) that involve tonic contraction of the quadriceps with the knee in flexion.
 - **The patellofemoral compression test** (reproduction of pain when the patella is compressed into the trochlear groove) is often helpful but may generate significant discomfort for the patient.
 - The initial management of PFPS includes **activity modification** (reduced intensity of exercise) **and nonsteroidal anti-inflammatory drugs**. **Patients also should be counseled on stretching and strengthening exercises (quadriceps strengthening exercises)**.



▪ Patellar dislocation:

- Patellar dislocation usually occurs after quick, lateral movements on a flexed knee and is most common in young athletes (soccer or ice hockey players, gymnasts), dancers, and military trainees.
- Risk factors for patellar dislocation include age <20, joint laxity, lower extremity malalignment, and patellar subluxation (excess lateral movement within the trochlea).
- Patients may feel the knee giving way, followed by a popping noise and severe pain.
- The quadriceps muscles normally exert a lateral force on the patella, especially during quadriceps contraction; therefore, in cases of patellar dislocation, lateral displacement with associated tear of the medial patellofemoral ligament (which normally provides an opposing medial force) is most common. Superior and medial dislocations are rare.
- Examination shows a flexed knee with reduced range of motion and lateral displacement of the patella out of the trochlea, which may be palpable as a depression at the anterior knee.
- Other findings can include hemarthrosis and tenderness along the medial patella (reflecting tear of the medial patellofemoral ligament).
- X-rays may not be needed for diagnosis in clinically apparent dislocation but are performed after reduction to rule out additional injuries.
- Patellar reduction by gently extending the knee while applying a caudally and medially directed force on the lateral edge of the patella. Reduction should be followed by immobilization of the knee in extension for three weeks. Physiotherapy to strengthen the quadriceps femoris muscle: to prevent recurrent dislocation.



- Quadriceps tendon tears:

- At full activation, the quadriceps muscle (comprised of the rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius) generates extreme force across the knee joint.
- Sudden, forceful contraction, as can occur in deceleration from a fall or in certain athletic activities, can cause rupture of the quadriceps-patellar tendon complex.

- Tendon injuries can be categorized in relation to the patella:

A. Quadriceps tendon tears (proximal to the patella in the rectus femoris tendon): the patella rides low, indicating an intact connection to the tibia, with a palpable defect above the patella.

B. Patellar tendon tears (distal to the patella): the patella rides high, often with a palpable defect below the patella.

- Typical symptoms include an audible pop, rapid swelling, and inability to actively extend the knee against gravity.
- Risk factors include chronic kidney disease, hyperparathyroidism, and anabolic steroid abuse. Exposure to fluoroquinolone antibiotics may also increase the risk.
- The diagnosis and extent of the injury can be assessed on MRI.
- Management of complete tears of the quadriceps tendon requires surgical intervention.



- **Tibial stress fracture:**
 - Stress fractures are caused by repeated tension or compression without adequate rest and most commonly occur in athletes and military recruits who suddenly increase their activity.
 - These types of fractures are also common in women who have low bone density associated with low caloric intake and hypomenorrhea/amenorrhea (female athlete triad).
 - In addition to the tibia, stress fractures can also be seen in the metatarsals, tarsal bones (navicular, calcaneus), and, less commonly, the femur and pelvis.
 - The diagnosis is based primarily on clinical factors, including localized activity-related pain, swelling, and point tenderness on palpation.
 - X-rays are frequently normal (especially in the first few weeks) but may reveal periosteal reaction at the site of the fracture.
 - MRI and bone scan are more sensitive but not usually necessary.
 - Treat with a cast and repeat the x-rays in 2 weeks. Non-weight bearing with crutches is another option.

Stress fracture

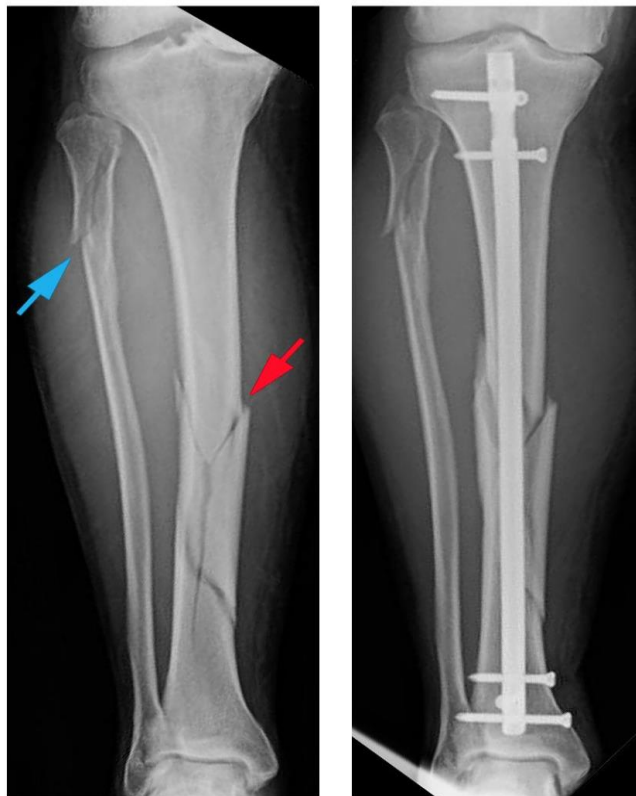
Risk factors	<ul style="list-style-type: none"> • Repetitive activities (eg, running, gymnastics) • Abrupt increase in physical activity • Inadequate calcium & vitamin D intake • Decreased caloric intake • Female athlete triad: low caloric intake, hypomenorrhea/amenorrhea, low bone density
Clinical presentation	<ul style="list-style-type: none"> • Insidious onset of localized pain • Point tenderness at fracture site • Possible negative x-ray in the first 6 weeks
Management	<ul style="list-style-type: none"> • Reduced weight bearing for 4-6 weeks • Referral to orthopedic surgeon for fracture at high risk for malunion (eg, anterior tibial cortex, 5th metatarsal)



- ❖ N.B:
 - The second metatarsal, which is subjected to significant extremes of loading during gait, is the most commonly involved metatarsal. Fractures of the second, third, and fourth metatarsals are managed conservatively as the surrounding metatarsals act as splints and nonunion is uncommon.
 - Initial interventions include rest and simple analgesics (acetaminophen).
 - Patients who continue to have pain despite activity restrictions may additionally be managed with a wide, hard-sole podiatric shoe.

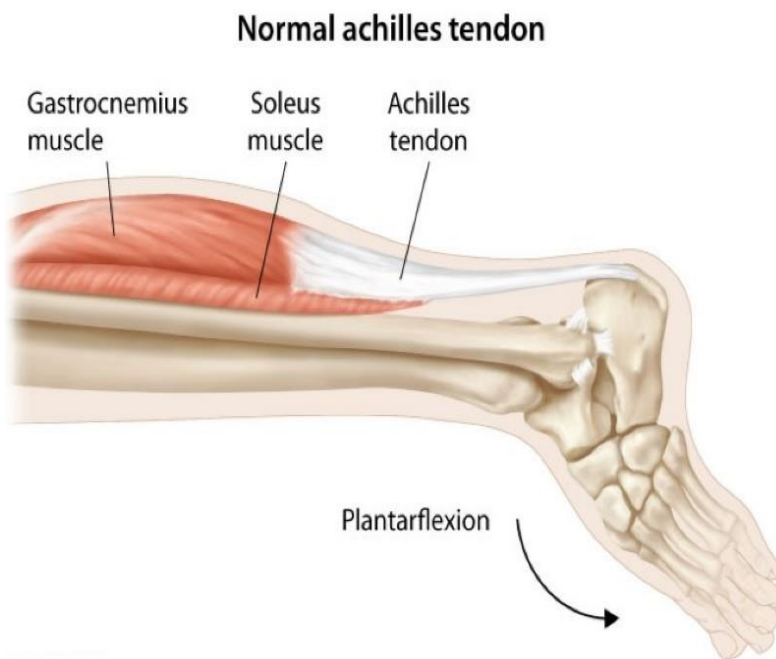
▪ Leg fracture:

- Leg fracture involving the tibia and fibula is often seen when a pedestrian is hit by a car.
- Physical exam shows angulation; x-rays are diagnostic.
- Casting takes care of the ones that are easily reduced; intramedullary nailing is needed for the ones that cannot be aligned.
- The lower leg (along with the forearm) is one of the most common locations for development of the compartment syndrome. Increasing pain after a long leg cast has been applied always requires immediate removal of the cast and appropriate assessment.



▪ Rupture of the Achilles tendon:

- Rupture of the Achilles tendon is seen in out-of-shape middle-aged men who subject themselves to severe strain (tennis, for instance). As they plant the foot and change direction, a loud popping noise is heard (like a rifle shot), and they fall clutching the ankle.
- Limited plantarflexion is still possible; but pain, swelling, and limping bring them to seek medical attention. Palpation of the tendon reveals a gap.
- Casting in equinus position allows healing in several months; surgery achieves a quicker cure.

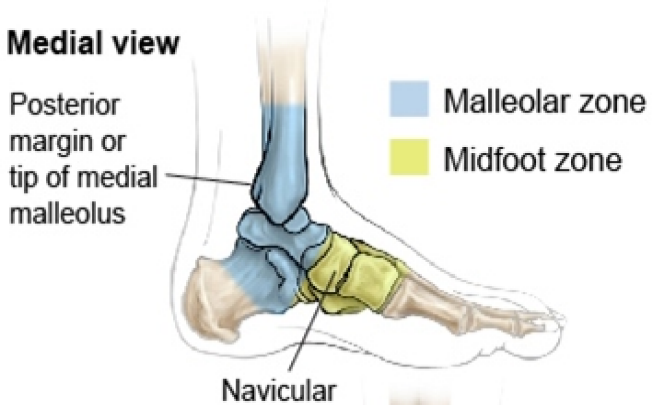



▪ Fracture of the ankle:

- Fracture of the ankle occurs **when falling on an inverted or everted foot**. In either case, both malleoli break.
- AP, lateral, and mortise x-rays are diagnostic.
- Open reduction and internal fixation are needed if the fragments are displaced.



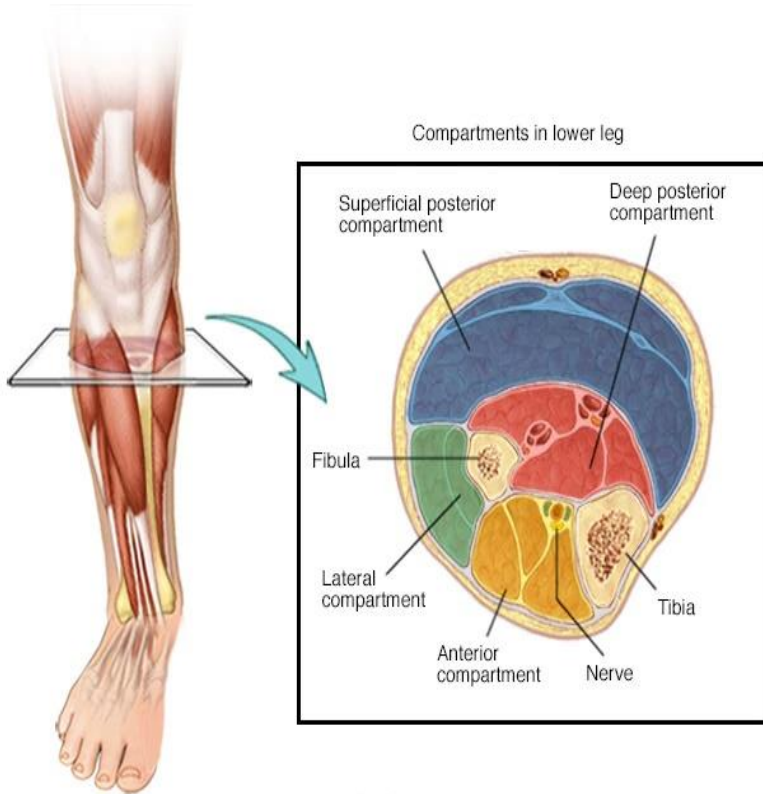
- Most ankle injuries are caused by sprains, in which the ligaments are injured but the bones remain intact. Uncomplicated sprains are typically managed conservatively (compression bandage or brace, ice packs, crutches to reduce weightbearing); however, orthopedic intervention may be required if a bony fracture is present.
- The Ottawa ankle rules were developed to determine which patients require imaging to rule out an ankle fracture. These rules have high sensitivity (up to 99%) for detecting fracture and can help reduce the number of unnecessary radiographs.
- Plain radiographs of the ankle are indicated in patients with pain in the area of the malleolus in association with either of the following:
 - Point tenderness over the posterior margin or tip of the malleolus
 - Inability to bear weight after the injury and for 4 steps during medical evaluation
- Further management depends on presentation and x-ray findings. Immediate orthopedics consult is indicated for any open fracture or in those with evidence of neurovascular impairment.

Ottawa ankle rules	
<p>X-ray of the ankle is required if:</p> <p>Pain at the <i>malleolar zone</i> and</p> <ul style="list-style-type: none"> • Tender at posterior margin/tip of medial malleolus OR • Tender at posterior margin/tip of lateral malleolus OR • Unable to bear weight 4 steps (2 on each foot) 	<p>Medial view</p>  <p>Posterior margin or tip of medial malleolus</p> <p>Navicular</p> <p>■ Malleolar zone ■ Midfoot zone</p>
<p>X-ray of the foot is required if:</p> <p>Pain at the <i>midfoot zone</i> and</p> <ul style="list-style-type: none"> • Tender at the navicular OR • Tender at the base of the 5th metatarsal OR • Unable to bear weight 4 steps (2 on each foot) 	<p>Lateral view</p>  <p>Posterior margin or tip of lateral malleolus</p> <p>Base of 5th metatarsal</p>

Compartment syndrome

- Acute compartment syndrome (ACS) occurs when **excessive fluid accumulation in a confined compartment of the body** (upper leg, **lower leg**, upper arm, **forearm**, hand, foot, abdomen) causes an increase in compartment pressure to the point that blood flow is severely impaired that leads to muscle and nerve ischemia.
- Compartment syndrome (CS) can be caused by:
 - Long bone fracture (**lower leg and forearm fractures are one of the most common locations for development of the compartment syndrome**).
 - Limb compression (crush injury, improperly fitted cast).
 - **After revascularization of an acutely ischemic limb.**
 - **The eschar that results from a circumferential, full thickness (third degree) burn often leads to constriction of venous and lymphatic drainage, fluid accumulation, and resulting distal ACS.**
- Compartment syndrome occurs **most frequently in the forearm or lower leg**.
- There is considerable variation in associated signs and symptoms, and a high index of suspicion is needed to make a diagnosis.
- **Patients typically have excruciating pain out of proportion to the injury that is worsened on passive range of motion and does not respond well to narcotics.**
- **Paresthesia from sensory nerve ischemia is usually an early finding.**
- **Pallor and pulselessness** are the result of arterial occlusion but is uncommon and not required for diagnosis.
- **Neurologic deficits** (sensory loss, motor weakness) may be present but develop later in the course of the disease.
- If elevated compartment pressure is allowed to persist, **tissue ischemia and eventual tissue death will occur.**
- **Diagnosis can be confirmed by measuring compartment pressures in the affected extremity** (compartment pressure >30 mm Hg indicates significant CS).
- However, in high-risk patients (those with limb revascularization) who develop symptoms suggestive of CS (pain, swelling, sensory loss), the diagnosis may be made on **clinical grounds alone**.

- **Emergency fasciotomy is required for treatment or, in the case of circumferential burns, escharotomy.** Time to fasciotomy is the most critical prognostic indicator and should be performed without delay.
- Pain under a cast is always handled **by removing the cast and examining the limb.**



SIGNS & SYMPTOMS

PAIN



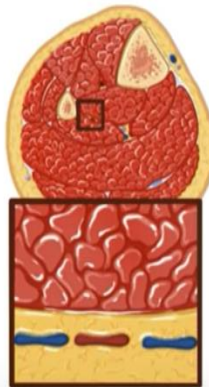
- * SHARP & DEEP
- * WORSENS w/ PASSIVE STRETCHING

PARESTHESIA



- * FEELING of PINS & NEEDLES
- ↓
- * ANESTHESIA

PULSELESSNESS



- * PRESSURE ↑↑↑
- ↓
- * ARTERIES COLLAPSE

LESS COMMON

PALLOR & POIKILOThERMIA

- * INABILITY to REGULATE BODY TEMPERATURE



PARALYSIS



* RARE

Open fracture

- Open fracture, in which a broken bone protrudes from the wound, **requires irrigation in the OR and suitable reduction within 6 hours from the time of the injury.**
- It is also called **compound fracture.**



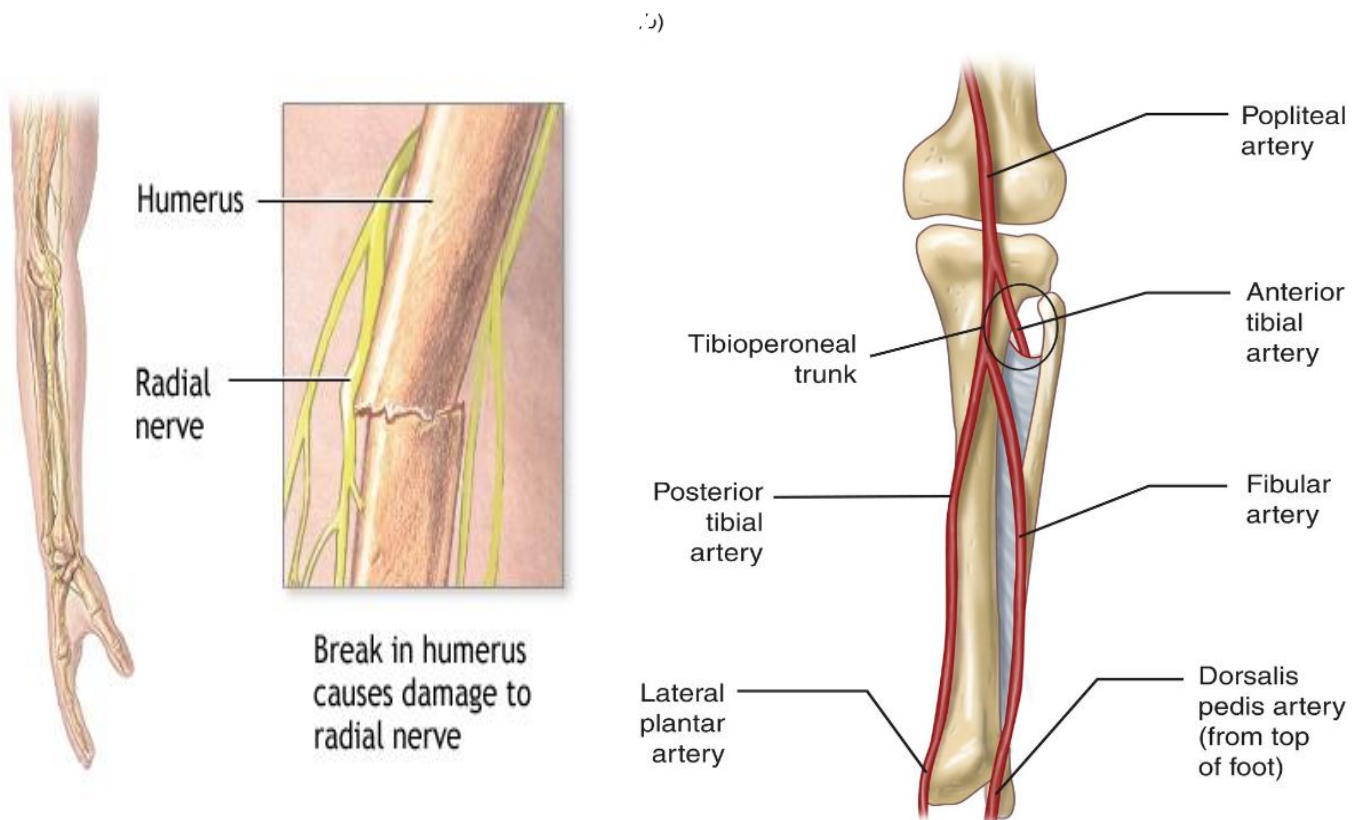
Associated neurovascular injuries

■ The radial nerve injury:

- The radial nerve can be injured in **oblique fractures of the middle to distal thirds of the humerus**.
- If a patient comes in unable to dorsiflex (extend) the wrist (wrist drop) and regains function when the fracture is reduced and the arm is placed on a hanging cast or coaptation sling, no surgical exploration is needed.
- However, if nerve paralysis develops or remains after reduction, the nerve is entrapped and surgery has to be done.

■ Popliteal artery injury:

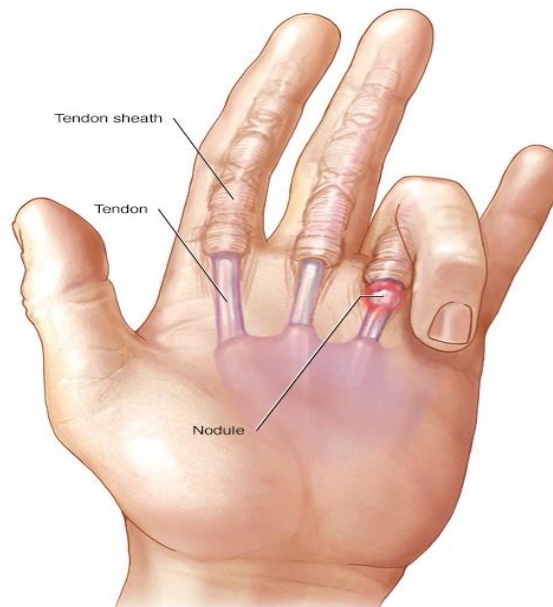
- Popliteal artery injury can occur in **posterior dislocations of the knee**.
- Following reduction of the dislocation, the popliteal artery must be evaluated with U/S, because even if distal pulses which had been absent return following reduction of the dislocation, there may be an intimal flap or local dissection that may need further evaluation with CT angiogram or surgical exploration.
- If pulses remain absent or an obvious injury is identified on U/S → surgical exploration is indicated.



Common Hand Problems

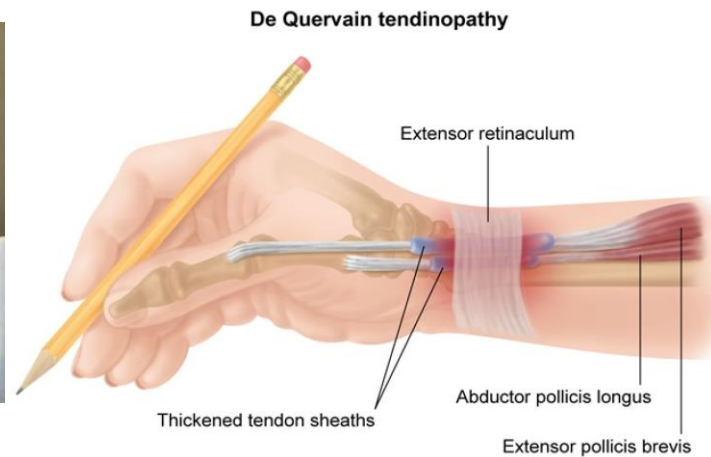
▪ Trigger finger:

- Trigger finger is more common in women and presents with acute finger flexion and the inability to extend it unless pulled with the other hand, which results in a "painful snap".
- Fibrocartilaginous metaplasia of the tendon sheath of the A1 annular pulley → loss of smooth gliding of the finger flexor tendons under the annular pulley → finger gets locked in flexed position
- Usually **idiopathic**. Mostly affects **thumbs and ring fingers**
- **Steroid injection is the first line of therapy**; surgery is the treatment of last resort.

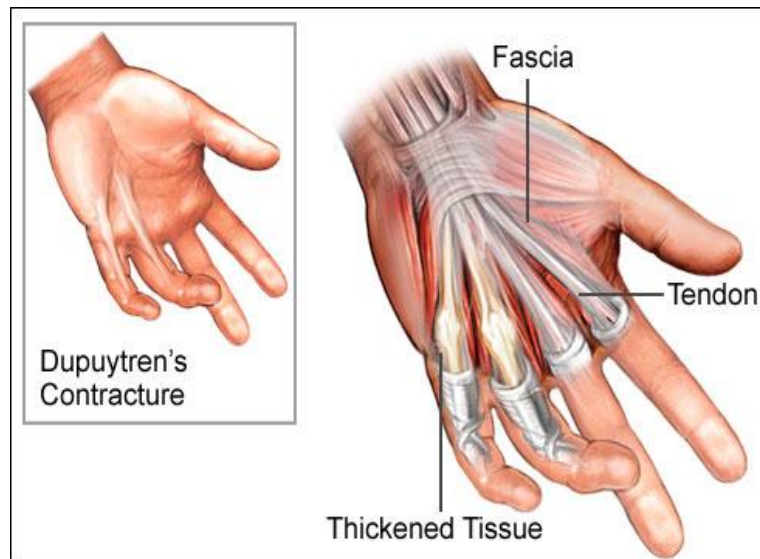


▪ De Quervain tenosynovitis:

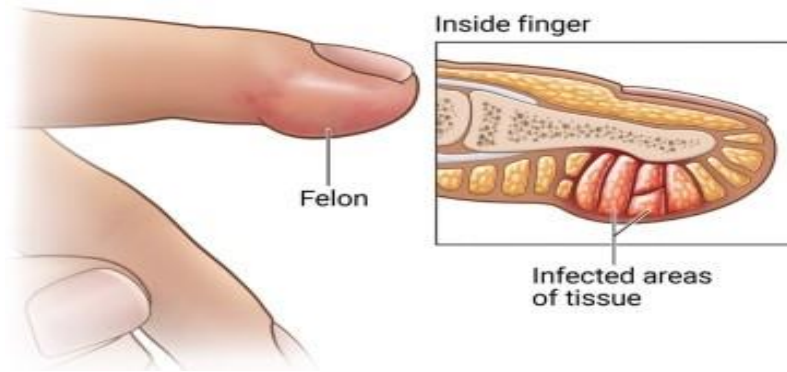
- De Quervain tenosynovitis is a condition that classically affects **new mothers who hold their infants with the thumb outstretched (abducted & extended)**.
- This condition is caused by inflammation of the abductor pollicis longus and extensor pollicis brevis tendons as they pass through a fibrous sheath at the radial styloid process.
- Tenderness can typically be elicited with direct palpation of the radial side of the wrist at the base of the hand. **Additionally, the Finkelstein test, which is conducted by passively stretching the affected tendons by grasping the flexed thumb into the palm with the fingers, elicits pain.**
- Splint and anti-inflammatory agents can help, but **steroid injection is most effective**. Surgery is rarely needed.



- Dupuytren contracture:
- Dupuytren contracture occurs in **older men of Norwegian ancestry and in alcoholics**.
- In Dupuytren contracture, **the palmar fascia within the hand becomes abnormally thick, which can cause the fingers to curl and can impair finger function**.
- There is contracture of the palm of the hand, and palmar fascial nodules can be felt.
- Surgery may be needed when the hand can no longer be placed flat on a table.



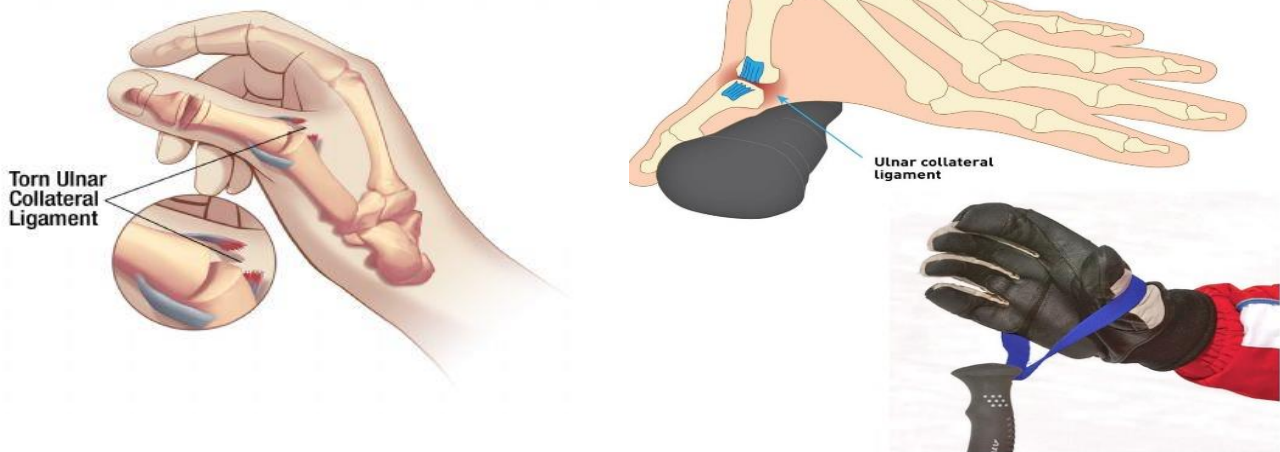
- Felon:
- A felon is **an abscess in the pulp of a fingertip**, caused by a neglected penetrating injury.
- Patients complain of throbbing pain, and have **all the classic findings of an abscess**, including fever.
- Because the pulp is a closed space with multiple fascial trabecula, pressure can build up and lead to tissue necrosis; thus **surgical drainage is urgently indicated**.



▪ Gamekeeper thumb:

- Gamekeeper thumb is an **injury of the ulnar collateral ligament sustained by forced hyperextension of the thumb** (historically suffered by gamekeepers when they killed rabbits by dislocating their necks with a violent blow with the extended thumb, nowadays seen as a skiing injury when the thumb gets stuck in the snow or the **ski strap during a fall**).
- On physical exam there is **collateral laxity at the thumb-metacarpophalangeal joint**, and if untreated it can be dysfunctional and painful, and lead to arthritis.
- **Casting with thumb spica** is usually effective in case of partial tear. Surgery is needed in cases of complete tear.

Game Keeper's Thumb



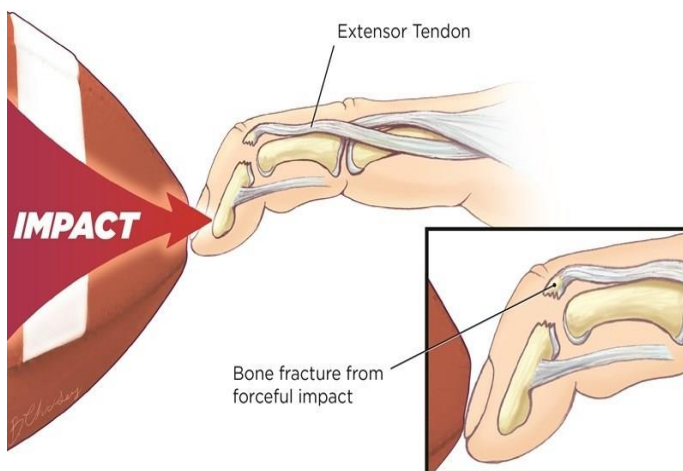
▪ Jersey finger:

- Jersey finger is **an injury to the flexor tendon sustained when the flexed finger is forcefully extended** (as in someone unsuccessfully grabbing a running person by the jersey).
- When making a fist, the distal phalanx of the injured finger does not flex with the others.
- Treatment is surgical repair.

Jersey Finger



- Mallet finger:
- Mallet finger is the opposite: **the extended finger is forcefully flexed** (a common volleyball injury), and the extensor tendon is ruptured.
- The tip of the affected finger remains flexed when the hand is extended, resembling a mallet.
- **Splinting is usually the first line of treatment.**

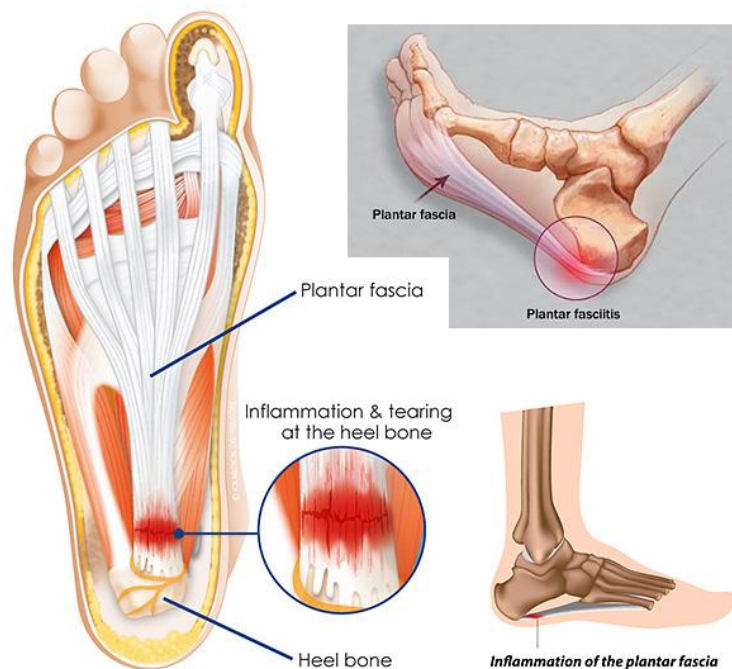


- **Traumatically amputated digits are surgically reattached whenever possible. The amputated digit should be cleaned with sterile saline, wrapped in a saline-moistened gauze, placed in a sealed plastic bag, and the bag placed on a bed of ice.** The digit should not be placed in antiseptic solutions or alcohol, should not be put on dry ice, and should not be allowed to freeze.

Foot Pain

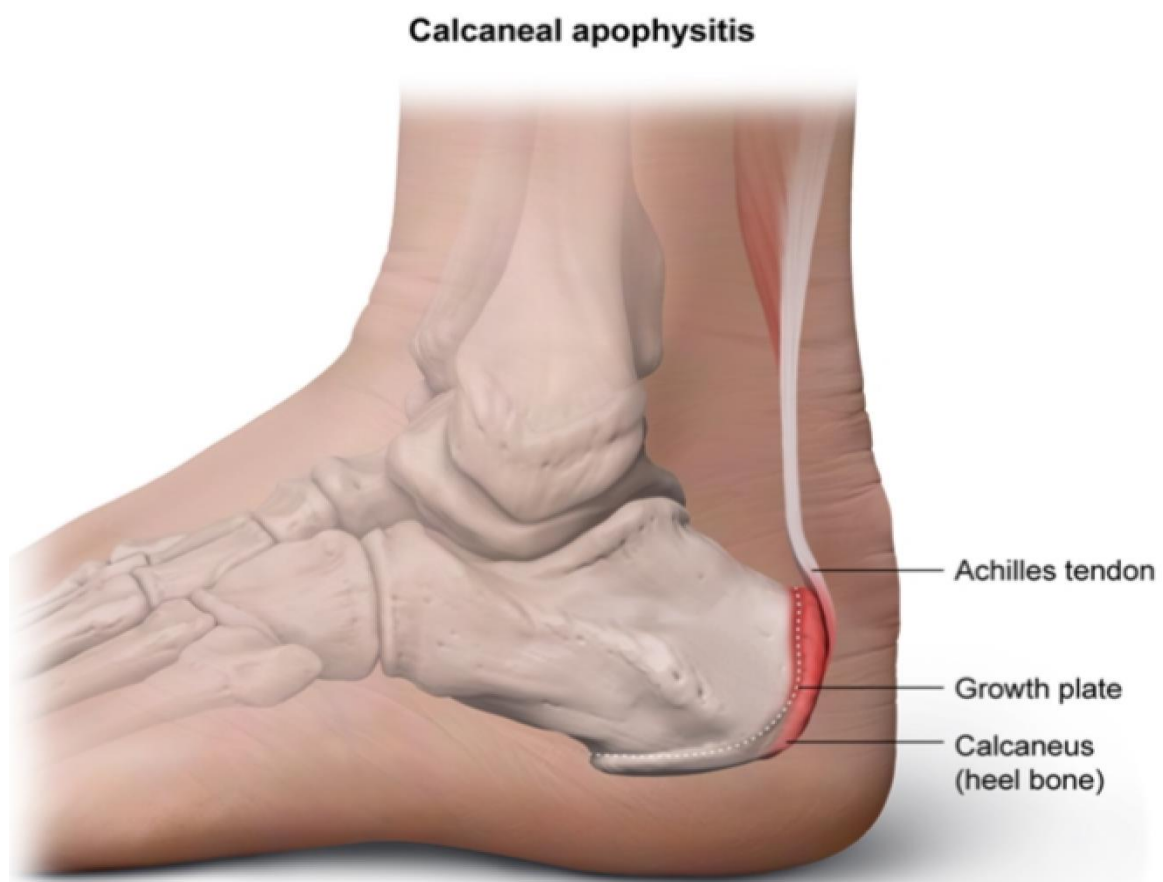
■ Plantar fasciitis:

- Plantar fasciitis is characterized by inflammation and degeneration of the plantar aponeurosis (deep plantar fascia), a thick, fibrous band that extends from the calcaneus to the toes and supports the longitudinal arch of the foot.
- Plantar fasciitis is a very common but poorly understood problem affecting older, overweight patients who complain of disabling, sharp heel pain every time their foot strikes the ground.
- It may also be seen in younger, well-conditioned individuals who participate in high-impact exercise or spend extended periods in bare feet.
- The pain is worse in the mornings.
- X-rays show a bony spur matching the location of the pain, and physical exam shows exquisite tenderness to palpation over the spur, although the bony spur is not likely the cause of the problem as many asymptomatic people have similar spurs.
- Initial management includes activity modification (avoiding walking barefoot, reducing high-impact exercise), stretching exercises, and padded heel inserts. However, the plantar aponeurosis recovers slowly; the long-term outcome is generally good, but symptoms may require months or even years for complete resolution.



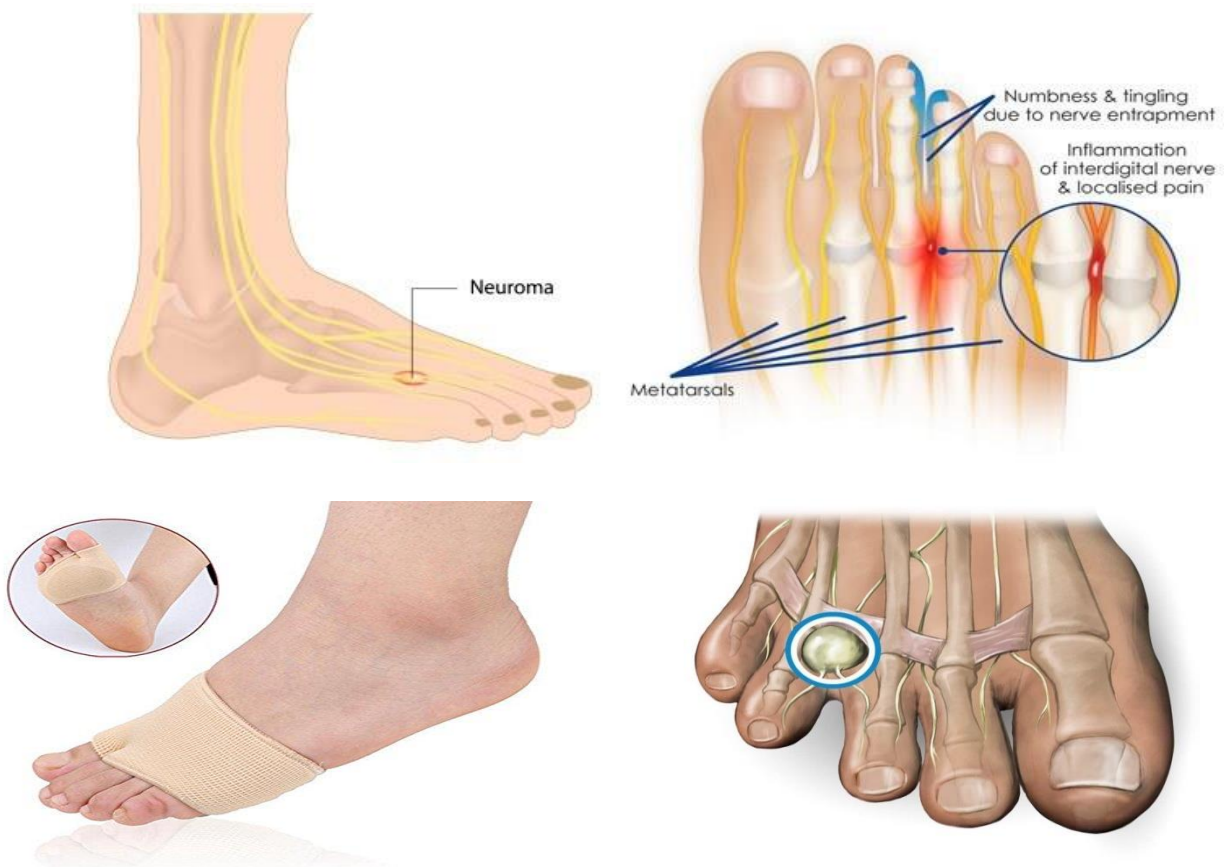
■ Calcaneal apophysitis (sever disease):

- Heel pain in an active boy is most commonly calcaneal apophysitis (Sever disease), an overuse injury caused by **repetitive microtrauma to the calcaneal apophysis (heel growth plate)**.
- The condition is typically seen in athletic children age 8-12 who participate in running or jumping sports (basketball) because the apophysis is most susceptible to stress during periods of rapid growth.
- Presentation involves chronic heel pain that is worse with activity, particularly when wearing shoes without heel support (flip flops, cleats).
- Diagnostic findings include tenderness with squeezing the heel (calcaneal compression test) and on palpation at the base of the heel over the apophysis. Heel swelling can occur with severe inflammation. In addition, **limited ankle dorsiflexion may be present due to an associated tight Achilles tendon and decreased gastrocnemius/soleus flexibility**.
- Treatment is generally **supportive** with stretching, ice, nonsteroidal anti-inflammatory drugs, and a heel cup insert for cushioning. Symptoms typically improve within a **couple of months**.



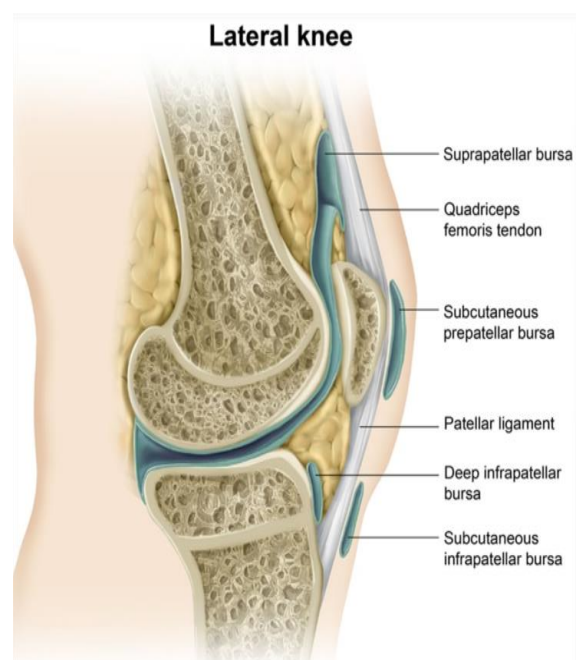
▪ Morton's neuroma:

- Morton neuroma is a mechanically induced degenerative neuropathy that causes numbness, aching, and burning in the distal forefoot from the metatarsal heads to the third and fourth toes which commonly occurs in **runners**.
- The symptoms are **worsened by walking on hard surfaces and wearing tight or high-heeled shoes**.
- The cause is typically **the use of pointed, high heel shoes** (or pointed cowboy boots) that force the toes to be bunched together.
- The diagnosis of Morton neuroma is primarily made **clinically**. On examination, squeezing the metatarsal joints will cause pain on the plantar surface of the foot along with crepitus between the third and fourth toes (Mulder sign).
- **Treatment involves metatarsal support with a bar or padded shoe inserts to decrease pressure on the metatarsal heads.**
- Surgery is usually reserved for patients who fail conservative treatment.



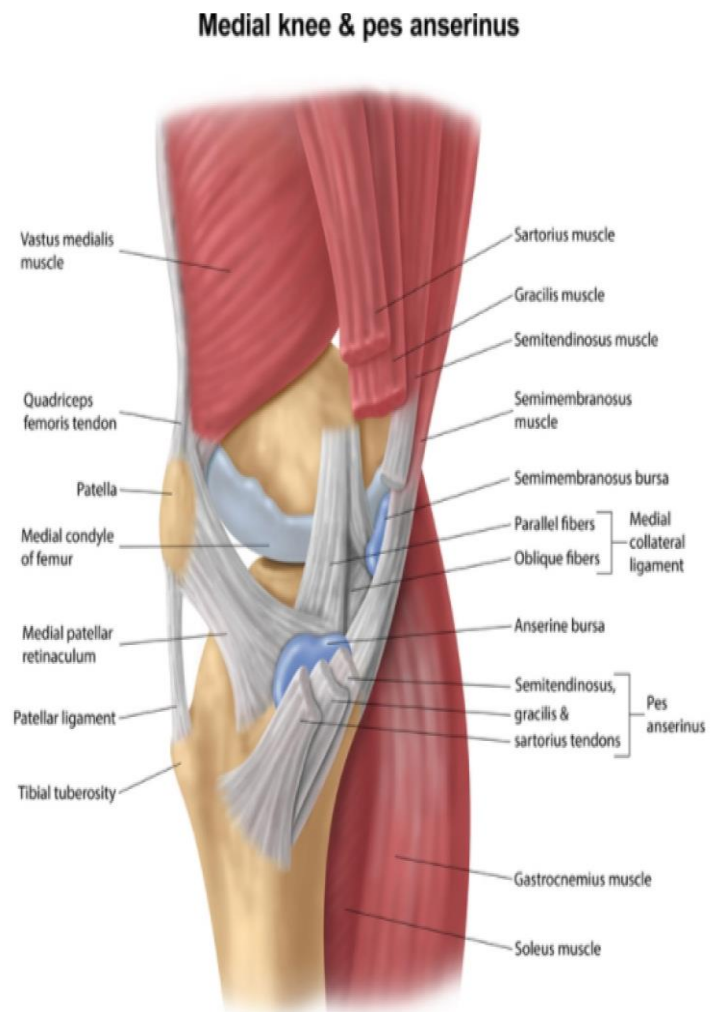
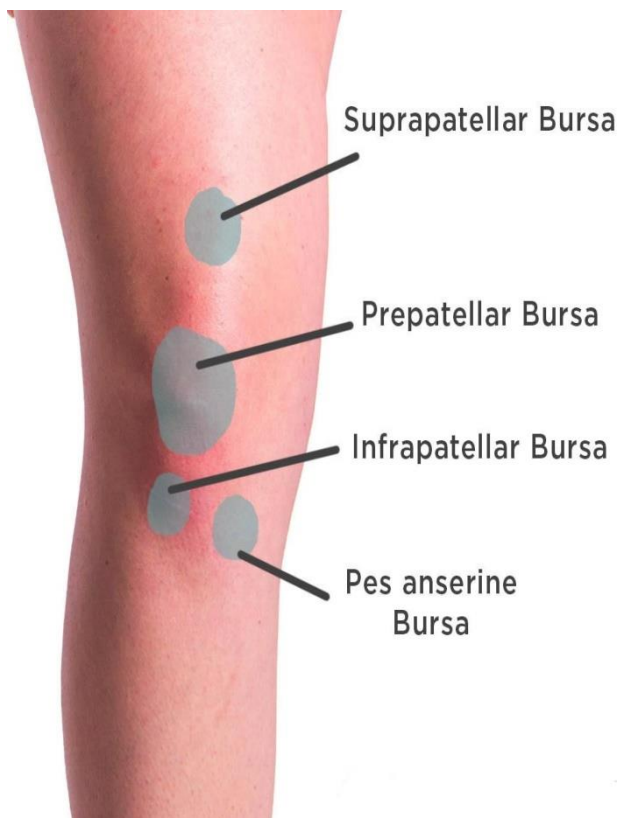
Bursitis

- A bursa is a synovial sac that **alleviates friction at bony prominences and ligamentous attachments**.
 - Bursae are vulnerable to acute injury or chronic repetitive pressure and may become inflamed due to **infection** (septic bursitis), **crystalline arthropathy** (gout), or **autoimmune conditions** (rheumatoid arthritis).
 - Because bursae are located in exposed positions, the pain and tenderness of bursitis may be exquisite. Other features may include **swelling and erythema, particularly with more superficial bursae**. Active range of motion is often decreased or painful, but passive motion is usually normal as it causes less pressure on the inflamed bursa.
1. **Prepatellar bursitis "housemaid's knee":**
 - Prepatellar bursitis is characterized by **anterior knee pain, tenderness, erythema, and localized swelling** and is common in occupations requiring repetitive kneeling.
 - **Prepatellar bursitis "housemaid's knee" is common in occupations requiring repetitive kneeling, such as concrete work, carpet laying, and plumbing.**
 - While bursitis in other locations is generally noninfectious, acute prepatellar bursitis is very commonly due to **Staphylococcus aureus**, which can infect the bursa via penetrating trauma, repetitive friction, or extension from local cellulitis.
 - **The diagnosis should be confirmed with aspiration of bursal fluid for cell count and Gram stain:**
 - If Gram stain and culture are negative, patients may be managed with activity modification and nonsteroidal anti-inflammatory drugs.
 - Otherwise, patients are treated with drainage and systemic antibiotics.



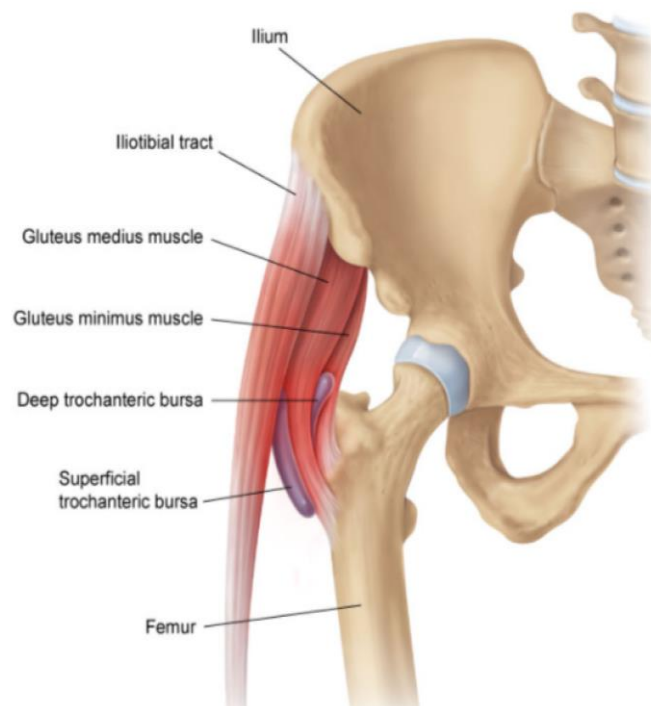
2. Pes anserinus pain syndrome (Anserine bursitis):

- Pes anserinus pain syndrome (PAPS) presents with **sharp, localized pain and tenderness over the anteromedial part of the tibial plateau just below the joint line of the knee and deep to the pes anserinus and is often exacerbated by pressure from the opposite knee while lying on the side.**
- This condition is often referred to as **anserine bursitis**, but most patients do not have true inflammation in the bursa, and multiple regional structures can contribute to the pain.
- The pes anserinus is formed by the **conjoined tendons of gracilis, sartorius, and semitendinosus.**
- PAPS can be caused by an **abnormal gait, overuse, or trauma.**
- Examination shows a well-defined area of tenderness over the medial tibial plateau just below the joint line. **A valgus stress test does not aggravate the pain, indicating no medial collateral ligament involvement.**
- **The diagnosis is primarily based on clinical features**, although x-ray can exclude concurrent osteoarthritis of the knee.



3. Greater trochanteric pain syndrome (Trochanteric bursitis):

- Greater trochanteric pain syndrome (GTPS) is an **overuse syndrome involving the tendons of the gluteus medius and minimus where they run over the greater trochanter.**
- GTPS is sometimes termed "trochanteric bursitis," although the bursae are not the source of pain in most patients.
- GTPS typically presents with **chronic lateral hip pain that is worsened with repetitive hip flexion (climbing stairs, walking uphill) or lying on the affected side.**
- This diagnosis of GTPS is based primarily on **clinical findings.** Physical examination shows local tenderness over the greater trochanter during flexion. Hip range of motion is normal, although **abduction may aggravate the pain.**
- Initial treatment of GTPS includes local heat, activity modification, and nonsteroidal anti-inflammatory drugs (NSAIDs). Physical therapy is often helpful as well. **Patients with persistent symptoms despite conservative therapy often benefit from local corticosteroid injection, which is generally safe and provides rapid pain relief.**



Popliteal (Baker) cyst

- A popliteal (Baker) cyst is due to **extrusion of synovial fluid from the knee joint into the gastrocnemius or semimembranosus bursa and is most common in patients with underlying arthritis.**
- Excessive synovial fluid formation (**due to osteoarthritis or rheumatoid arthritis**) and positive pressure in the knee during extension can cause passage of fluid into the bursa and gradual enlargement of the cyst.

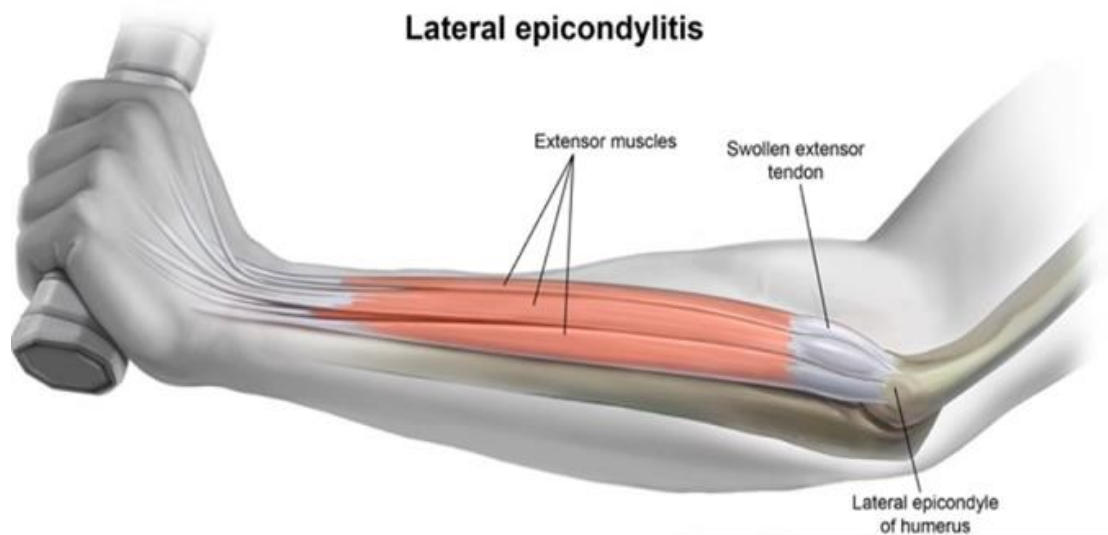
- Popliteal cysts are often **asymptomatic** and present as a **chronic, painless bulge behind the knee**.
- The diagnosis is usually apparent on examination, with a soft mass in **the medial popliteal space** that is **most noticeable with knee extension and less prominent with flexion**.
- Rupture of a popliteal cyst (following strenuous exercise) can cause posterior knee and calf pain, with tenderness and swelling of the calf **resembling deep venous thrombosis (DVT)**.
- An arc of ecchymosis is often visible distal to the medial malleolus "**crescent sign**".
- **Ultrasound can rule out DVT and confirm the popliteal cyst.**



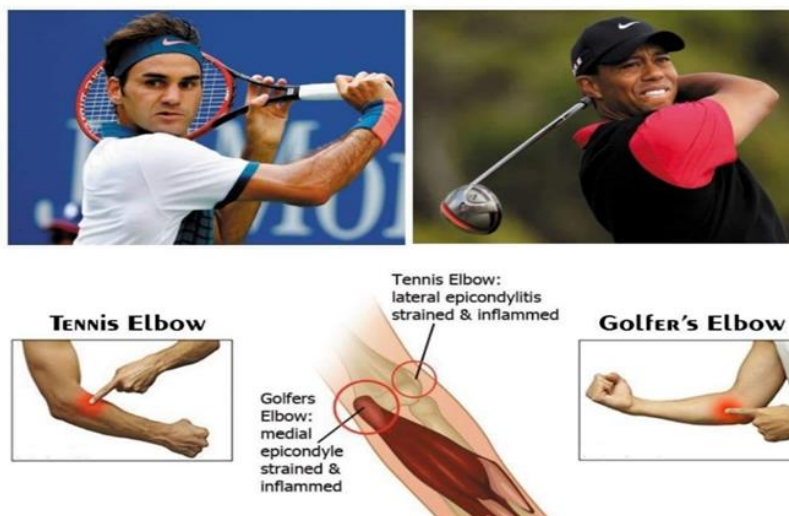
Popliteal (Baker) cyst	
Etiology	<ul style="list-style-type: none"> • Extrusion of fluid from knee joint space into semimembranosus/gastrocnemius bursa
Risk factors	<ul style="list-style-type: none"> • Trauma (eg, meniscal tear) • Underlying joint disease (eg, osteoarthritis, rheumatoid arthritis)
Clinical presentation	<ul style="list-style-type: none"> • Asymptomatic bulge behind knee that diminishes with flexion • Posterior knee pain, swelling, stiffness
Complications	<ul style="list-style-type: none"> • Venous compression (leg/ankle swelling) • Dissection into calf (erythema, edema, positive Homan sign) • Cyst rupture (acute calf pain, warmth, erythema, ecchymosis)

❖ N.B:

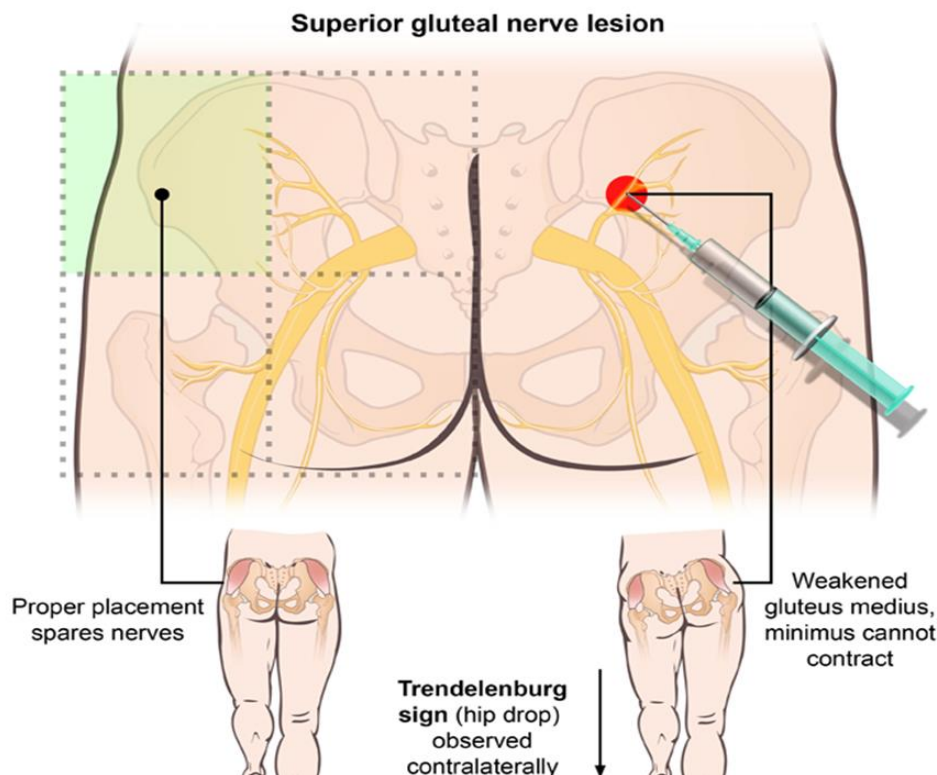
1. Lateral epicondylitis, often called "**tennis elbow**" is due to its frequent occurrence in casual tennis players.
 - Lateral epicondylitis is due to **overuse of the extensor muscles**.
 - The primary pathologic lesion is **noninflammatory angiofibroblastic tendinosis at the common extensor origin on the lateral epicondyle of the humerus**.
 - An analogous disorder (**medial epicondylitis**) can occur at the origin of the wrist flexors at the medial epicondyle (**golfers' elbow**).
 - Patients usually have a history of repetitive, forceful extension at the wrist.
 - Examination findings may include **tenderness at the lateral epicondyle and reproduction of pain with resisted extension or passive flexion at the wrist**.
 - Management includes **activity modification, nonsteroidal anti-inflammatory drugs, and counterforce bracing**.



TENNIS ELBOW vs. GOLFER'S ELBOW



2. Positive Trendelenburg sign is **drooping of the contralateral hemipelvis below its normal horizontal level during monopodal stance**.
 - 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, injections into the superomedial quadrant of the buttock, or inflammatory myopathies**, results in a positive Trendelenburg sign and gait.



3. A vertebral compression fracture (VCF) is a **common complication of advanced osteoporosis**.
 - Risk factors for VCF include trauma (falls), **osteoporosis/osteomalacia**, infection (osteomyelitis), malignancy with bone metastases, and metabolic abnormalities (hyperparathyroidism).
 - Patients with **gradual** onset VCF can be **asymptomatic**. However, an **acute** VCF can present with **low back pain and decreased spinal mobility after sudden bending, coughing, or lifting**.
 - The pain typically increases with standing, walking, or lying on the back.
 - Examination can show tenderness at the affected level.
 - **Each VCF can decrease a patient's height by >1 cm and lead to kyphosis, which is associated with decreased respiratory capacity and increased risk for atelectasis pneumonia.**
 - Kyphosis can also cause a protuberant abdomen, early satiety, and weight loss.
 - VCF is associated with a significantly increased risk for future vertebral and nonvertebral fractures.

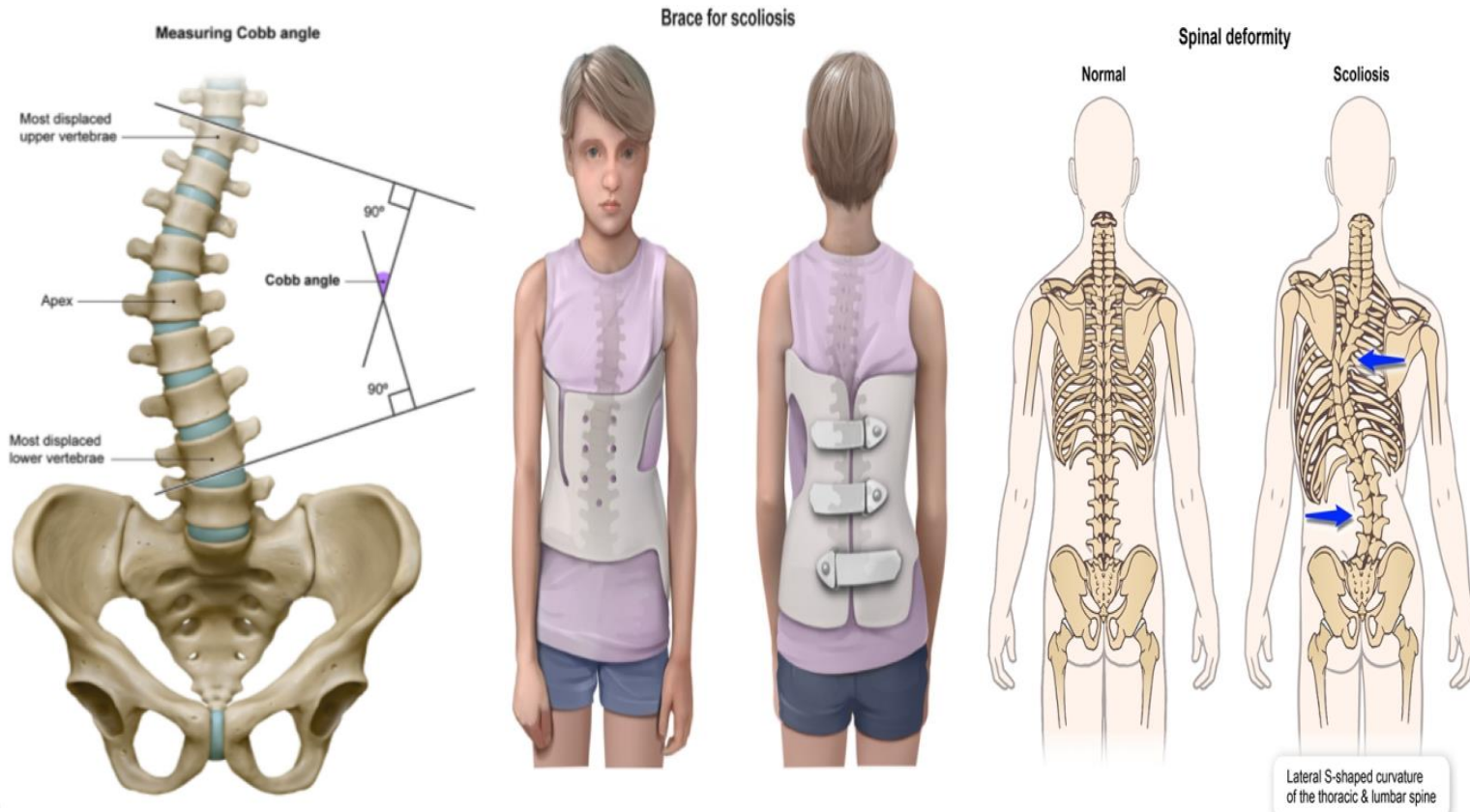


Clinical features of vertebral compression fracture

Etiologies	<ul style="list-style-type: none"> • Trauma • Osteoporosis, osteomalacia • Infection (eg, osteomyelitis) • Bone metastases • Metabolic (eg, hyperparathyroidism) • Paget disease
Clinical presentation	<p>Chronic/gradual VCF</p> <ul style="list-style-type: none"> • Painless • Progressive kyphosis • Loss of stature <p>Acute VCF</p> <ul style="list-style-type: none"> • Low back pain & decreased spinal mobility • Pain increasing with standing, walking, lying on back • Tenderness at affected level
Complications	<ul style="list-style-type: none"> • Increased risk for future fractures • Hyperkyphosis, possibly leading to protuberant abdomen, early satiety, weight loss, decreased respiratory capacity

VCF = vertebral compression fracture.

4. Adolescent idiopathic scoliosis (AIS) refers to **lateral curvature (Cobb angle >10 degrees) of the spine without a specified etiology**.
- The deformity typically presents in **children age >10 during periods of rapid growth**.
 - Although poor posture may be noted, **AIS is usually asymptomatic and identified during school scoliosis screening or annual physical examination**. Inspection of the back may reveal **asymmetry of the shoulders, scapulae, or iliac crest**.
 - **The first step in evaluating clinically evident spinal asymmetry is x-ray of the spine**. Full-back posteroanterior and lateral radiographs confirm the diagnosis, measure the severity of disease (Cobb angle), and assess skeletal maturity.
 - Management of AIS depends on risk of progression (degree of skeletal maturity) and severity (degree of curvature on x-ray):
 - Most curves are mild (Cobb angle 10-30 degrees) and can be **monitored clinically every 6 months**.
 - A thoracolumbosacral spinal **brace** is indicated in a child with growth potential remaining and Cobb angle >30 degrees to help **reduce curve progression**.
 - **Surgical** fixation is considered for **severe** curvature (Cobb angle >40-50 degrees).
 - Scoliosis is most commonly idiopathic in adolescents. **However, a pathologic cause (tumor, infection, trauma) should be suspected in a patient with scoliosis and any of the following red flag features:**
 - Back pain.
 - Neurologic symptoms.
 - Rapidly progressing curve (>10 degrees per year)
 - Vertebral anomalies on x-ray.



CHAPTER 3

Shock

Shock

- Inadequate organ perfusion and delivery of nutrients necessary for normal tissue and cellular function.
- Initially may be reversible but life-threatening if not treated promptly.

	Caused by	Skin	PCWP (Preload)	CO	SVR (Afterload)	Treatment
Hypovolemic	Hemorrhage, dehydration, Burns	Cold, clammy	↓↓	↓	↑	IV fluids
Cardiogenic	Acute MI, HF, valvular dysfunction, arrhythmia	Cold, Clammy	↑	↓↓	↑	Inotropes, diuresis
Obstructive	Cardiac tamponade, pulmonary embolism, Tension pneumothorax		↓			Relieve obstruction
Distributive	Sepsis, anaphylaxis	Warm	↓	↑	↓↓	IV fluids, pressors, epinephrine (anaphylaxis)
	CNS injury	Dry	↓	↓	↓↓	

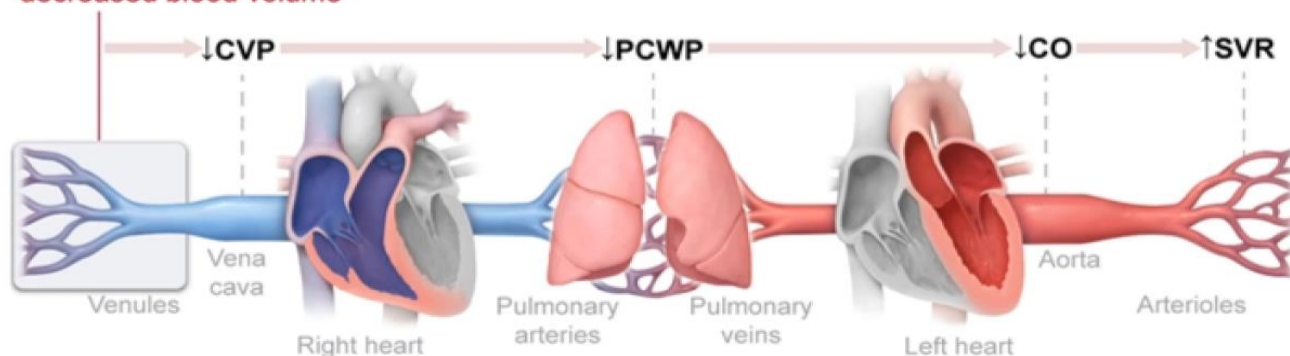
Central venous pressure in different types of shock

Shock	Hypovolemic	Distributive	Obstructive	Cardiogenic
CVP	↓	↓	↑	↑
Mechanism	↓ Intravascular volume	↓ Systemic vascular resistance	↑ Back pressure from obstructed cardiac filling	↑ Back pressure from forward pump failure

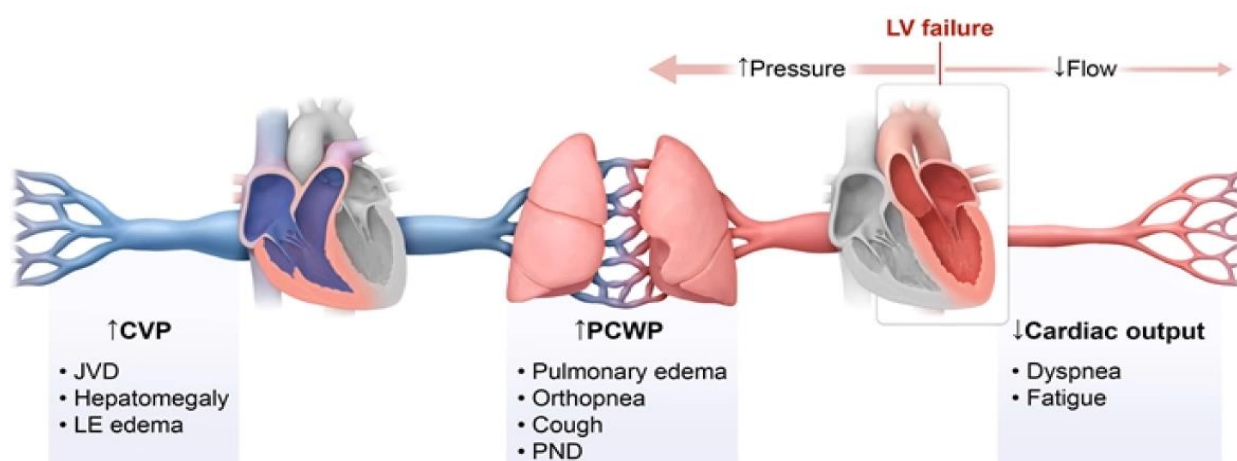
CVP = central venous pressure.

Hypovolemic shock

Primary disturbance:
decreased blood volume



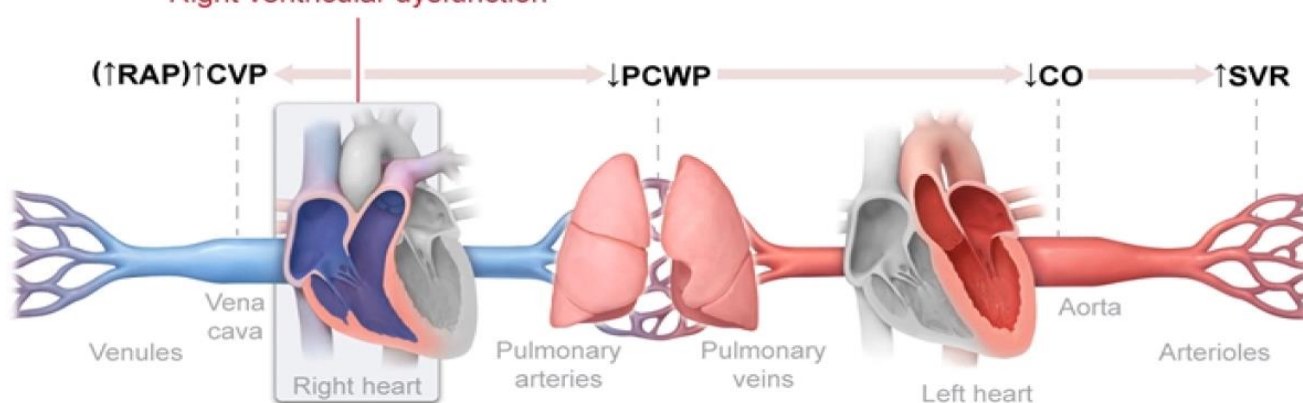
Left-sided heart failure



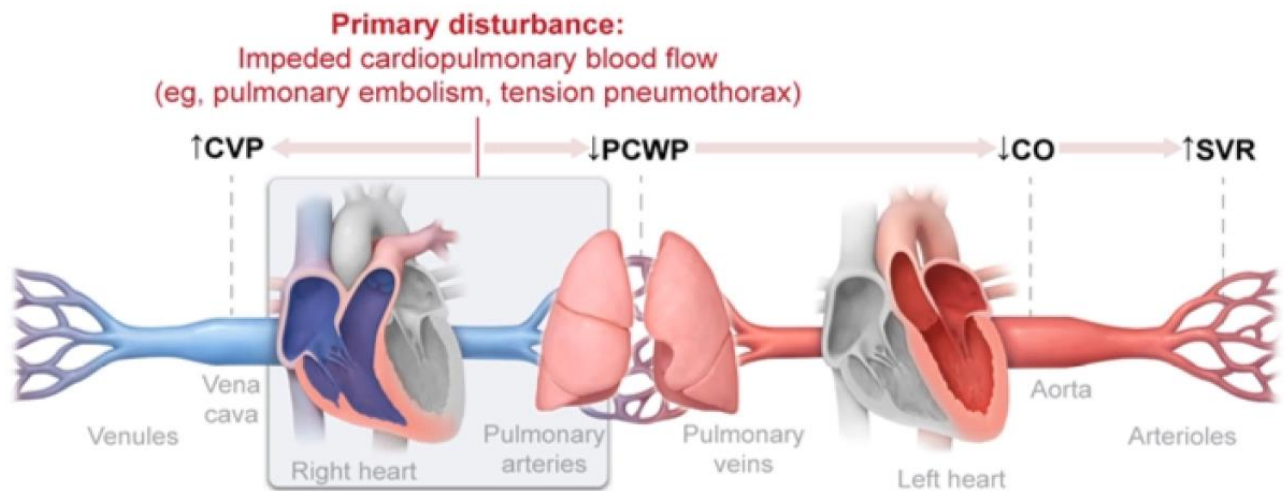
CVP = central venous pressure; JVD = jugular venous distension; LE = lower extremity; LV = left ventricular; PCWP = pulmonary capillary wedge pressure; PND = paroxysmal nocturnal dyspnea.

Right-sided heart failure

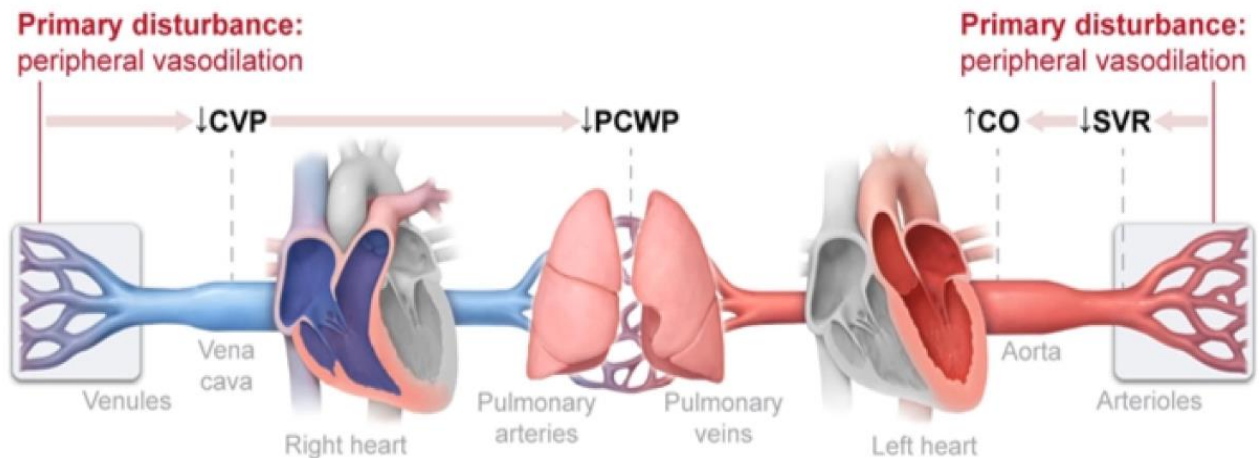
Primary disturbance:
Right ventricular dysfunction



Obstructive shock



Septic shock



CHAPTER 4

Pre-Op and Post-Op Care

Pre-Op and Post-Op Care

Preoperative assessment

- Patients undergoing surgery must be optimized prior to surgery in order to **decrease perioperative and postoperative complications**.
- A. **Cardiac Risk:**
 - **The number one limiting factor prior to surgery is a history of cardiovascular disease.**
 - If the patient is under the age of 35 and has no history of cardiac disease, **EKG is the only test needed.**
 - A patient who has a history of cardiac disease, regardless of age, must have:
 - EKG.
 - Stress testing to evaluate for ischemic coronary lesions.
 - Echocardiogram for structural disease and to assess ejection fraction.
 - Controlling systolic hypertension reduces perioperative cardiac complications, and systolic hypertension should be controlled prior to any elective surgery.
 - Ejection fraction:
 - **Ejection fraction <35% (normal 55%) poses prohibitive cardiac risk for elective non-cardiac operations.**
 - Incidence of peri-operative myocardial infarction (MI) could be as high as 75-85%, and mortality for such an event as high as 50-90%.
 - Jugular venous distention:
 - **Jugular venous distention, which indicates the presence of CHF, is the worst single finding predicting high cardiac risk.**
 - If at all possible, treatment with ACE inhibitors, beta-blockers, digitalis, and diuretics should precede surgery.
 - Recent MI is the next worse predictor of cardiac complications:
 - Operative mortality within 3 months of the infarct is 40% but drops to 6% after 6 months. **Therefore, delaying surgery longer than 6 months from MI is the best course of action.**
 - If surgery cannot be safely delayed, admission to the ICU before surgery is recommended to optimize cardiac performance.

- Goldman's index of cardiac risk assigns the following (Don't memorize):

- 11 points to jugular venous distention (evidence of CHF).
- 10 points to recent MI (within 6 months).
- 7 points each to either premature ventricular contractions (≥ 5 per min) or a rhythm other than sinus rhythm.
- 5 points to age >70 .
- 4 points to emergency nature of surgery.
- 3 points each to either aortic valve stenosis, poor medical condition, or surgery within the chest or abdomen.

- The risk of life-threatening cardiac complications is (Don't memorize):

- Only 1% with total score 0-5.
- The risk becomes 5% if the points 6-12.
- Increases to 11% with 13-25.
- Reaches 22% when the points >25 .

B. **Pulmonary Risk:**

- Smoking is by far the most common cause of increased pulmonary risk, and the problem is compromised ventilation (high PCO_2 , low forced expiratory volume in 1 second [FEV1]), rather than compromised oxygenation.
- The smoking history, or the presence of chronic obstructive pulmonary disease (COPD), should lead to evaluation.
- Start with pulmonary function tests, and, if abnormal, obtain an arterial blood gas.
- Cessation of smoking for 8 weeks and intensive respiratory therapy (physical therapy, expectorants, incentive spirometry, humidified air) should precede surgery.

C. **Hepatic Risk:**

- Predictors of mortality are stratified by the Child-Pugh classification system.
- The contributing factors can be remembered as Ascites, Bilirubin, Clotting (prothrombin time), Diet (serum albumin) and Encephalopathy (presence/absence).

D. **Renal Risk:**

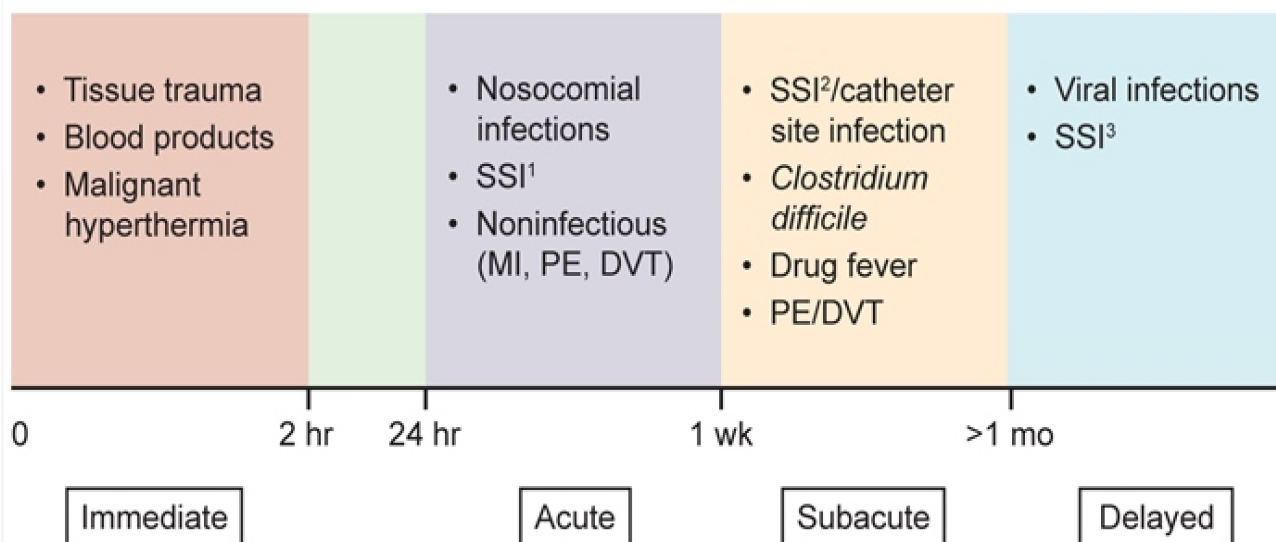
- Patients with known renal disease must be kept adequately hydrated; otherwise, hypoperfusion of the kidneys can lead to increased mortality.
- If a preexisting renal disease is present, volume loss during surgery will adversely and acutely affect renal function. Subsequent renin-angiotensin system activation will lead to further constriction of renal vasculature and make the creatinine clearance even lower.

- To ensure adequate kidney perfusion:
 - Give fluids before and during surgery.
 - If the patient is on dialysis, dialyze the patient 24 hours prior to surgery.
- E. **Nutritional Risk:**
 - Severe nutritional depletion is identified by one or more of the following:
 - Loss of 20% of body weight over 6 months.
 - Serum albumin <3 g/dL.
 - Low serum transferrin level.
 - Operative risk is multiplied significantly in those circumstances. Surprisingly, as few as 4-5 days of preoperative nutritional support (preferably via the gut) can make a big difference, and 7-10 days would be optimal if the surgery can be deferred for that long.
- F. **Metabolic Risk:**
 - Diabetic coma is an absolute contraindication to surgery.
 - Rehydration, return of urinary output, and at least partial correction of the acidosis and hyperglycemia must be achieved before surgery.

Postoperative Fever

- Postoperative fever (defined as $>38^{\circ}\text{C}$ [100.4°F]) commonly occurs after major surgery and can be **due to infectious or noninfectious causes**.
- The timing of the postoperative fever (immediate, acute, subacute, delayed) also suggests the likely diagnosis:
 - A. **Immediate fever (within a few hours)** in the operative or postoperative period is typically due to **prior infection or trauma, inflammation due to surgery, malignant hyperthermia, or medications (anesthetics) or blood products given during or prior to surgery**.
 - B. **Acute fever (within the first week after surgery)** is most often caused by **nosocomial infections (pneumonia, urinary tract infection) or other noninfectious causes such as pulmonary embolus (PE)**.
 - C. **Subacute fever (>1 week after surgery)** is usually due to **drug fever, clostridium difficile infection after long course of broad-spectrum antibiotics, surgical site infection, or PE**.
 - D. **Delayed postoperative fever (> 1 month after surgery)** is typically caused by an **infection (viral infections from blood products, infective endocarditis)**.

Timeline of cause of postoperative fever



SSI¹ = Due to group A *Streptococcus* (GAS) or *Clostridium perfringens*

SSI² = Due to other organisms (not GAS or *C perfringens*)

SSI³ = Due to indolent organisms

DVT = deep venous thrombosis; MI = myocardial infarction; PE = pulmonary embolism; SSI = surgical site infection.

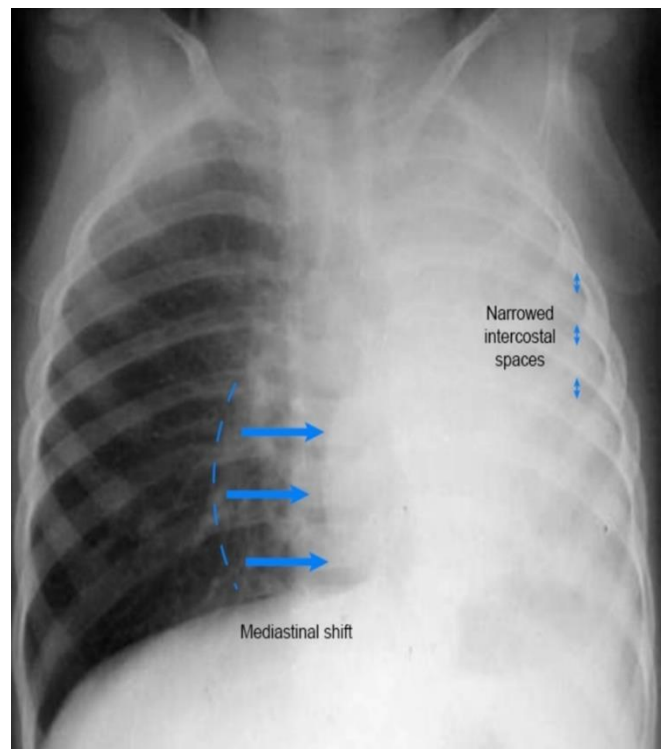
Causes of postoperative fever (the 5 Ws)	
Wind (lungs)	<ul style="list-style-type: none"> • Pulmonary embolus • Pneumonia • Aspiration
Wound	Surgical site infection
Water	Urinary tract infection
Walk	Deep venous thrombosis
Wonder drugs/ products	<ul style="list-style-type: none"> • Drug fever • Blood products • Intravenous lines

- Atelectasis (Wind):
 - Atelectasis is lobar or segmental collapse of the lung that causes decreased lung volume.
 - It is most common on **postoperative days 2 and 3 following abdominal or thoracoabdominal surgery.**
 - Atelectasis is one of the most common postoperative pulmonary complications and is usually **due to airway obstruction from retained airway secretions, decreased lung compliance, postoperative pain, and medications that interfere with deep breathing.**
 - Postoperative pain interferes with spontaneous deep breathing and coughing, which decreases the functional residual capacity and worsens the atelectasis. **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 (dyspnea, tachypnea)**.
- As compensation for the hypoxemia, patients usually **hyperventilate and develop respiratory alkalosis and decreased arterial partial pressure of carbon dioxide (PaCO_2)**.
- **Arterial blood gas levels typically show hypoxemia, hypocapnia, and respiratory alkalosis.**
- Chest x-ray demonstrates opacification of the affected lung area with mediastinal shifting toward the side of opacification. In addition, rib spacing becomes narrower in the affected hemithorax.
- **Preoperative patient education and a program of inspiratory muscle training (breathing exercises, forced expiration techniques, **incentive spirometry**) should be conducted to prevent postoperative atelectasis.**
- **Of these, incentive spirometry has been shown to be the most effective and is thus the first line preventive measure.**
- Additional postoperative strategies include adequate **pain control, continuous positive airway pressure, and chest physical therapy.**
- Chest physiotherapy is often useful in preventing mucus plugging and can be used to treat relatively minor atelectasis due to mucus plugging. Large-volume atelectasis typically requires bronchoscopy to remove the mucus plug.

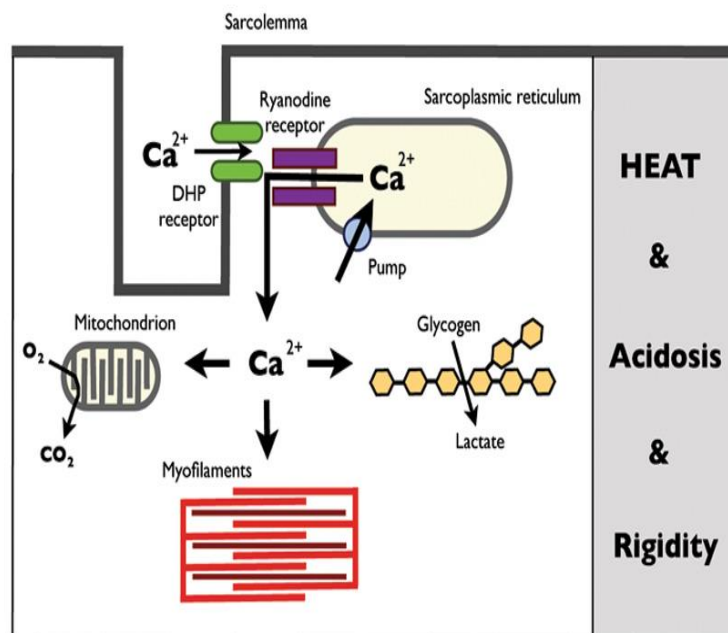


How to Use an Incentive Spirometer



- Pneumonia (Wind):
 - Pneumonia will happen in about 3 days if atelectasis is not resolved.
 - Fever will persist, leuko-cytosis will be present, and chest x-ray will demonstrate an infiltrate(s).
 - Obtain sputum cultures and treat with appropriate antibiotics.
- UTI (Water):
 - UTI typically produces fever starting on **post-operative day 3**.
 - Work up with a **urinalysis and urinary cultures** and treat with appropriate antibiotics.
- Deep thrombophlebitis (Walking):
 - Deep thrombophlebitis typically produces fever starting around **post-operative day 5**.
 - Physical exam is not sensitive for this pathology, so **obtain U/S with Doppler studies of the deep leg and pelvic veins**.
 - Treatment is systemic anticoagulation initially with heparin or unfractionated low molecular weight heparin and transitioned to a long-term anticoagulant, typically Warfarin.
- Wound infection (surgical site infection):
 - Wound infection typically begins to produce fever around **post-operative day 7**.
 - **Smoking is associated with an increased risk of surgical site infection and poor wound healing**. Current smoking is associated with greater risk than past smoking, but patients who quit are still at increased risk. **Smoking cessation is recommended prior to elective surgery, especially if cessation can be achieved at least 4-6 weeks before surgery**.
 - Physical exam will reveal **erythema, warmth, tenderness, and fluctuance**.
 - If only cellulitis is present → treat with antibiotics.
 - If an abscess is present or suspected → the wound must be opened and drained.
- Malignant hyperthermia (Wonder):
 - Occurs after administration of inhalation anesthetics especially **halothane** or **succinylcholine** to genetically susceptible individuals.
 - Genetic susceptibility may be related to **mutations in the genes encoding the ryanodine receptors** (calcium channel) of sarcoplasmic reticulum. It releases small amounts of calcium in the cytoplasm of the muscle fiber during muscle contraction.

- Abnormal ryanodine receptors release large amount of Ca after exposure to anesthetic → Excess of free Ca in the cytoplasm of muscle fibers stimulates its ATP-dependent reuptake by sarcoplasmic reticulum → **Excessive consumption of ATP generates heat, loss of ATP along with high temperature induces → muscle damage (Rhabdomyolysis) → release of potassium, myoglobin, and creatine kinase into circulation.**
- Clinically, malignant hyperthermia presents with **fever and muscle rigidity soon after surgery under general anesthesia**. Tachycardia, hypertension, hyperkalemia, acidosis and myoglobinuria are characteristic.
- Treatment:
 - o Malignant hyperthermia is a life-threatening condition and should be treated promptly.
 - o **Treatment is IV dantrolene**, 100% oxygen, correction of the acidosis, and cooling blankets.
 - o **Dantrolene is a muscle relaxant effective in malignant hyperthermia.** It acts on ryanodine receptor → prevents further release of Ca into the cytoplasm of muscle fibers.



- Deep abscesses:
 - Deep abscesses (intra-peritoneal: subphrenic, pelvic, or subhepatic) start producing fever around **post-operative days 10-15**.
 - CT scan of the appropriate body cavity is diagnostic.
 - Percutaneous image-guided drainage is therapeutic.

Wound dehiscence

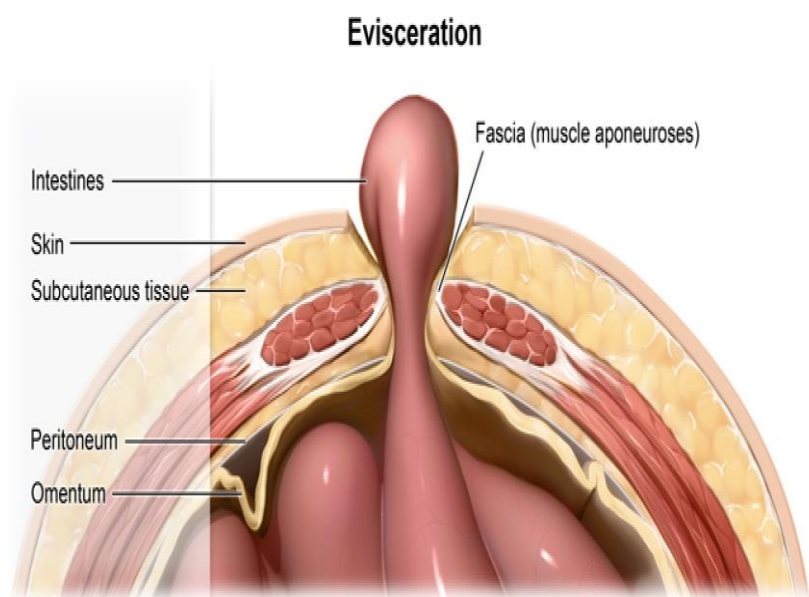
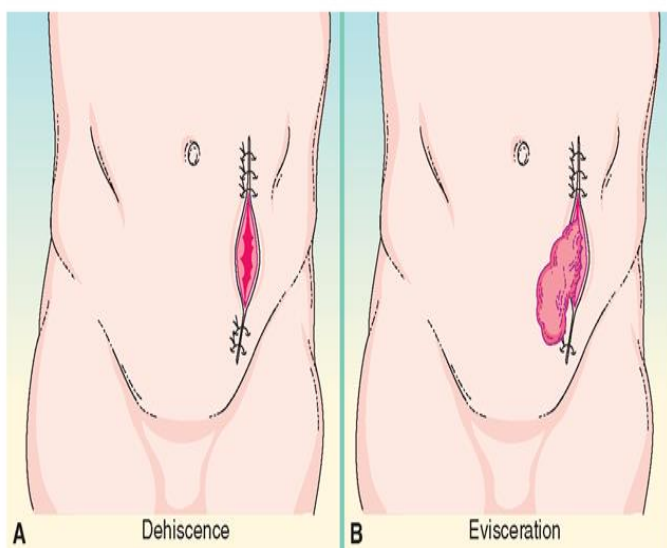
- Postoperative abdominal wounds can be categorized based on fascial involvement:

A. Superficial wound dehiscence:

- It is a separation of the skin and subcutaneous tissue with an **intact rectus fascia**.
- It typically develops **within the first postoperative week** and occurs secondary to an abnormal subcutaneous fluid buildup (seroma), resulting in a **scant serosanguineous fluid drainage**.
- Patients with a superficial dehiscence and **no signs of infection** (induration, erythema, purulent drainage) are **conservatively managed with regular dressing changes**.

B. Deep (fascial) wound dehiscence:

- It involves the rectus fascia (nonintact)** and results in exposure of the intraabdominal organs to the external environment.
- Fascial dehiscence is a surgical emergency **because of the risk of bowel evisceration and strangulation**.
- It typically happens when the patient (who may not have been recognized as having a dehiscence) coughs, strains, or gets out of bed.
- The patient must be kept in bed, and the bowel covered with large sterile dressings soaked with warm saline.
- Emergency abdominal closure is required.**



❖ N.B:

1. Return to consciousness after anesthesia (emergence) typically occurs within 15 minutes of extubation; at a minimum, patients should be responsive with intact protective (gag) reflexes within 30-60 minutes of the last administration of an anesthetic or adjuvant agent (opiate, muscle relaxant).
 - Delayed emergence occurs when a patient fails to regain consciousness within the expected window. The etiology is typically multifactorial but generally occurs due to 1 of 3 major causes:
 - A. **Drug effect:**
 - Preoperative drug ingestion (opiates, benzodiazepines, illicit drugs, anticholinergic drugs, antihistamines) may potentiate anesthetic effects.
 - Prolonged anesthesia duration or higher medication doses may also delay emergence.
 - B. **Metabolic disorder:**
 - Common etiologies include hyper- or hypoglycemia, hyper- or hypothermia, hyponatremia, and liver disease.
 - C. **Neurologic disorder:**
 - Intraoperative stroke, seizure (or postictal state), or elevation of intracranial pressure can cause prolonged alterations in mental status.
2. Dehydrated post-operative patients and the elderly are most prone to develop acute bacterial parotitis.
 - 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.

CHAPTER 5

Diseases of the Gastrointestinal System

General Surgery

Diseases of the gastrointestinal system

- **Mechanical intestinal obstruction:**
- Bowel obstruction is a **mechanical or functional** obstruction of the intestines due to various causes.

Causes of intestinal obstruction

Dynamic

- **Intraluminal**

Fecal impaction

Foreign body

Bezoars

Gall stones

- **Intramural**

Stricture

malignancy

- **Extramural**

Adhesions & bands

Hernias (internal – external)

Volvulus

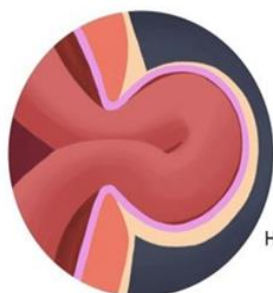
Intussusception

Adynamic

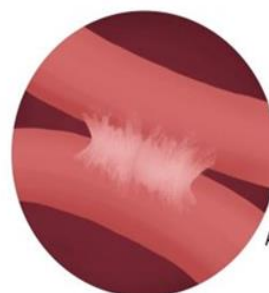
Paralytic ilus

Mesenteric vascular occlusion

Pseudo obstruction



Herniation



Adhesions



Intussusception



Volvulus

- There are 2 main types of obstruction:
 - Partial: A small amount of GI contents can pass.
 - Complete: No GI contents can pass.

- Small-bowel obstruction (SBO) is further categorized by **anatomic location** (proximal versus mid/distal) or simple versus strangulated:
 - **Proximal** obstructions are characterized by **early** vomiting, and abdominal discomfort.
 - **Mid or distal** obstructions typically present as colicky abdominal pain, **delayed** vomiting, prominent abdominal distension, constipation-obstipation, and **hyperactive bowel sounds**.

- Signs and Symptoms:
 - Severe waves of intermittent crampy abdominal pain.
 - Nausea and vomiting.
 - Hyperactive bowel sounds.
 - Early on, High-pitched “tinkling” sounds indicate that the intestinal fluid and air are under high pressure in the bowel (after a few days there is silence).

- Etiology:
 - **Adhesions from previous abdominal surgery (MCC).**
 - Incarcerated Hernias.
 - Crohn disease.
 - Neoplasms.
 - Intussusception.
 - Volvulus.
 - Foreign bodies.
 - Intestinal atresia.

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

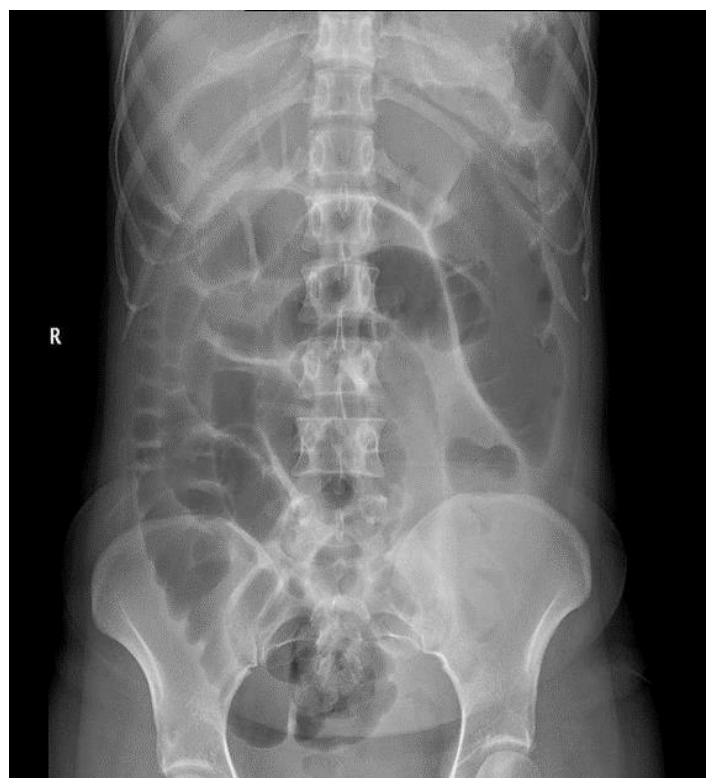
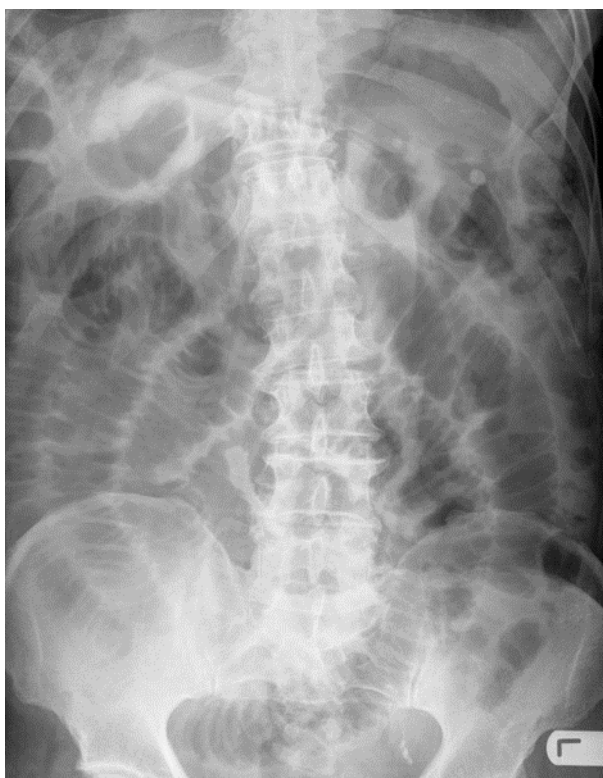
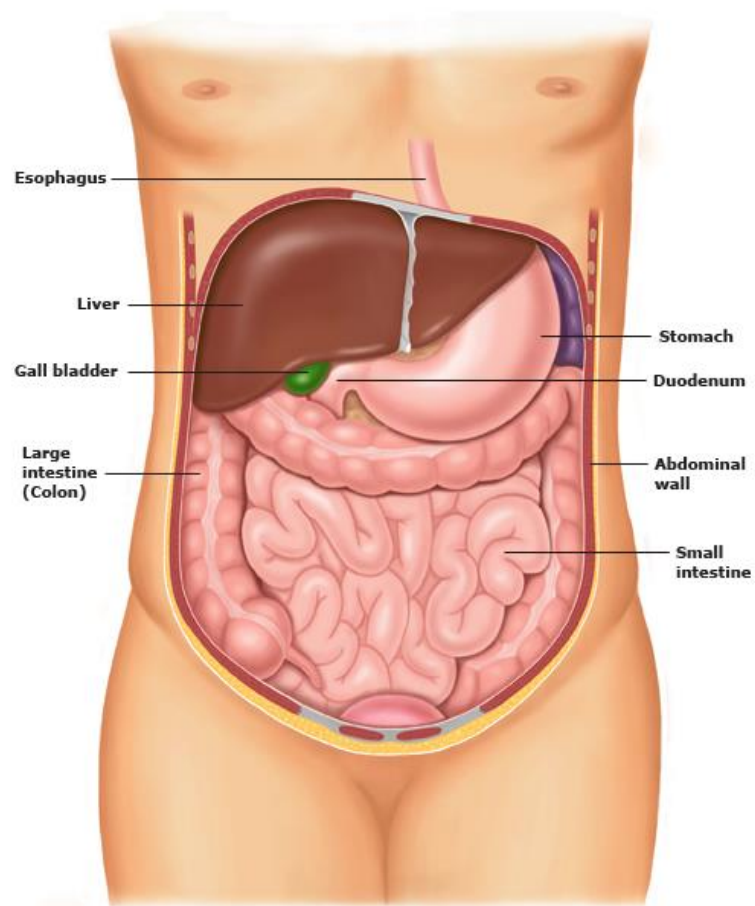
- Diagnostic Tests:
 - **The best initial test is abdominal x-ray**, which will show multiple air-fluid levels with dilated loops of small bowel.
 - **The most accurate test is a CT scan of the abdomen.** It will show a **transition zone** from dilated loops of bowel with contrast to an area of bowel with no contrast.

- Treatment:
 - A. Make patient NPO (Bowel Rest): Prevents further increase in bowel pressure.
 - B. Place NG tube with suction: Lowers bowel pressure proximal to obstruction.
 - C. Medical management: IV fluids to replace volume lost via third spacing.

D. Surgical decompression:

- Immediate surgical intervention is indicated for patients with intestinal obstruction who develop clinical or hemodynamic instability, fail to improve after initial conservative measures, and/or develop symptoms or signs of ischemia or necrosis.
- Delay in surgery may lead to perforation and significant risk of mortality.

Small-bowel obstruction	
Clinical presentation	<ul style="list-style-type: none"> • Colicky abdominal pain, vomiting • Inability to pass flatus or stool if complete (no obstipation if partial) • Hyperactive → absent bowel sounds • Distended & tympanitic abdomen
Diagnosis	<ul style="list-style-type: none"> • Dilated loops of bowel with air-fluid levels on plain film or CT scan • Partial: Air in colon • Complete: Transition point (abrupt cutoff), no air in colon
Complications	<ul style="list-style-type: none"> • Ischemia/necrosis (strangulation) • Bowel perforation
Management	<ul style="list-style-type: none"> • Bowel rest, nasogastric tube suction, intravenous fluids • Surgical exploration for signs of complications



Small bowel

- Located centrally
- Valvular markings, called valvulae conniventes, cross the entire width of the bowel and spaced close together
- “Stacked coin” appearance



Clinical pearl

Large Bowel

- Located peripherally
- Contains haustra which either...
 - ...do not connect from one wall to the other OR...
 - ...are spaced wider apart than the valvulae conniventes of the small bowel



■ **Paralytic (adynamic) ileus:**

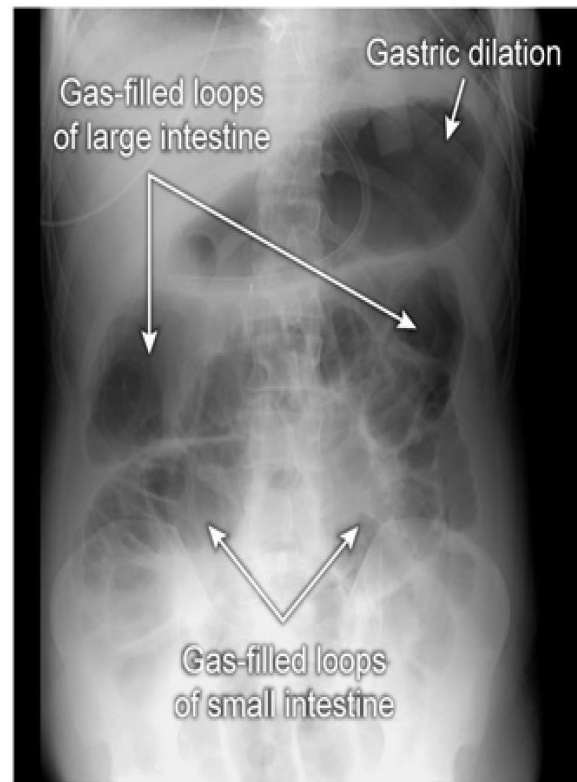
- Ileus is a **functional** defect in bowel motility without an associated physical obstruction.
- **Etiology:**
 - Ileus is most commonly due to abdominal surgery but can also be seen in other conditions such as retroperitoneal/abdominal hemorrhage or inflammation, intestinal ischemia and electrolyte abnormalities (**hypokalemia**).
 - Contributors to the pathophysiology of ileus include **increased splanchnic nerve sympathetic tone** following peritoneal instrumentation, local release of inflammatory mediators, and **postoperative opiate analgesic use (which causes decreased gastrointestinal motility and disordered peristalsis)**.
- **Signs and symptoms:**
 - Signs and symptoms of ileus include nausea, vomiting, abdominal distension, failure to pass flatus or stool (obstipation) and **hypoactive or absent bowel sounds**.
 - Some degree of ileus occurs following most abdominal procedures; however, persistence of the signs and symptoms (**>3-5 days postoperatively**) is termed prolonged (or "pathologic") postoperative ileus (PPI).
- **Management:**
 - The diagnosis is **clinical**, but abdominal x-rays (**classically revealing dilated gas-filled loops of bowel with no transition point**) can be helpful in confirmation.
 - Management is conservative and includes bowel rest, supportive care, and treatment of secondary causes.

Small bowel obstruction versus ileus		
	Small bowel obstruction	Ileus
Etiology	<ul style="list-style-type: none"> • Prior surgery (weeks to years) 	<ul style="list-style-type: none"> • Recent surgery (hours to days) • Metabolic (eg, hypokalemia) • Medication induced
Abdominal examination	<ul style="list-style-type: none"> • Distension • Increased bowel sounds 	<ul style="list-style-type: none"> • Possible distension • Reduced/absent bowel sounds
Small bowel dilation	Present	Present
Large bowel dilation	Absent	Present

Normal



Paralytic ileus



Abdominal x-ray revealing gas-filled loops of both the small and large intestines as well as gastric dilation.

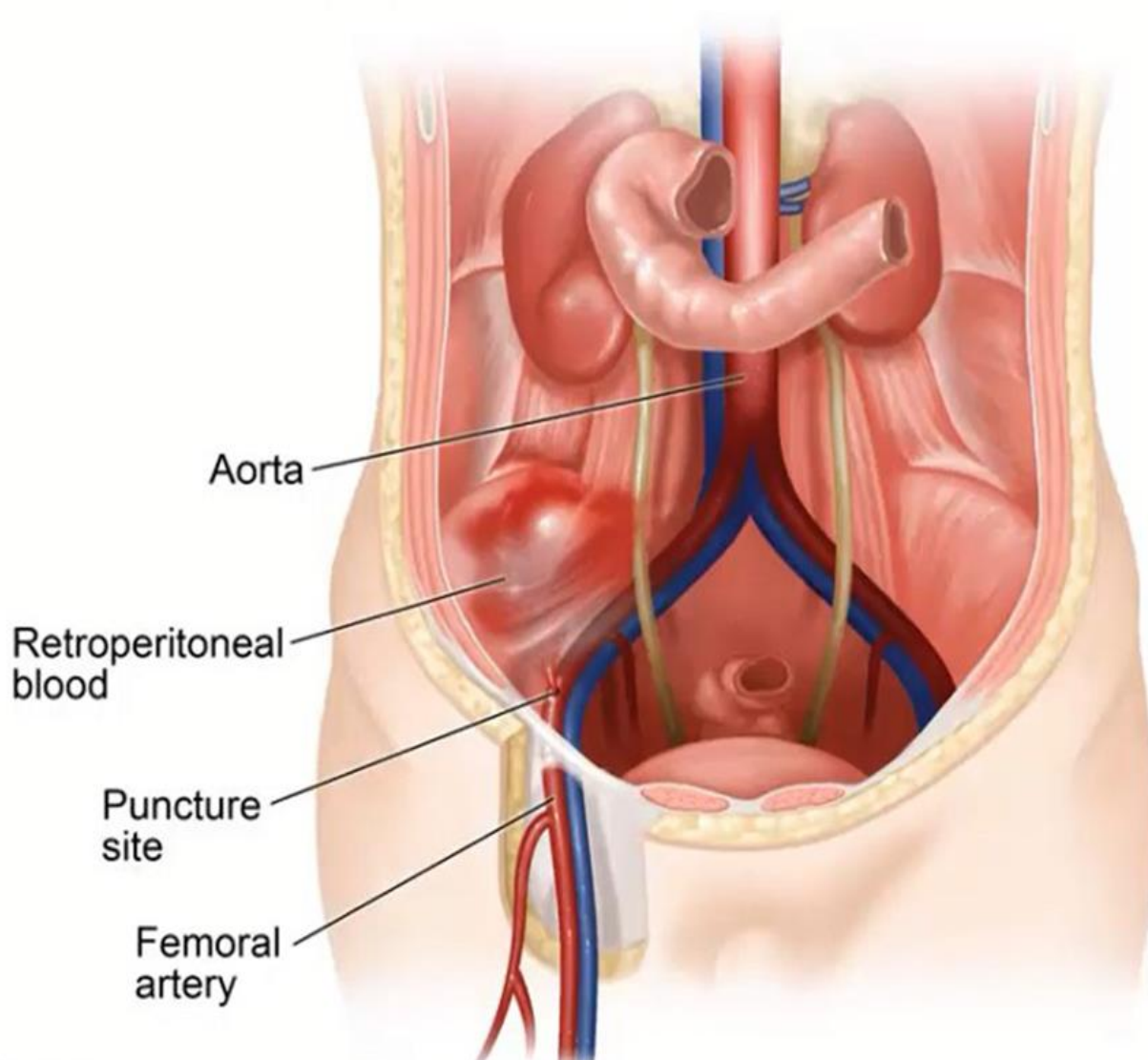
- **Acute colonic pseudo-obstruction (Ogilvie syndrome):**
 - Ogilvie syndrome is the **acute dilatation of the colon in the absence of any mechanical obstruction**.
 - **Etiology:**
Common causes include **electrolyte imbalance** (hypokalemia, hypomagnesemia) and factors that lead to **autonomic disruption of the colon** (major surgery, neurologic disease, anticholinergic medication).
 - Postoperative Ogilvie syndrome tends to present **3-7 days after surgery**.
 - **Finding:**
 - **Acute colonic pseudo-obstruction (Ogilvie syndrome) is an uncommon postoperative complication characterized by abdominal pain, distension, obstipation, and colonic dilation without radiographic evidence of an anatomic obstruction.**
 - Acute colonic pseudo-obstruction more commonly occurs in **men age >60**.
 - **Management:**
 - CT scan provides diagnostic confirmation **showing colonic dilation with no anatomic obstruction**.
 - Management involves **bowel rest (NPO) and placement of nasogastric and rectal tubes for colonic decompression**.
 - **Neostigmine** can be given intravenously if symptoms fail to improve after 48 hours or if the cecal diameter on imaging exceeds 12 cm (which is a strong predictor of impending perforation).

Acute colonic pseudoobstruction (Ogilvie syndrome)	
Etiologies	<ul style="list-style-type: none"> • Major surgery, traumatic injury, severe infection • Electrolyte derangement (↓ K, ↓ Mg, ↓ Ca) • Medications (eg, opiates, anticholinergics) • Neurologic disorders (eg, dementia, stroke)
Clinical findings	<ul style="list-style-type: none"> • Abdominal distension, pain, obstipation, vomiting • Tympanic to percussion, ↓ bowel sounds • If perforation: guarding, rigidity, rebound tenderness
Imaging	<ul style="list-style-type: none"> • X-ray: colonic dilation, normal haustra, nondilated small bowel • CT scan: colonic dilation without anatomic obstruction
Management	<ul style="list-style-type: none"> • NPO, nasogastric/rectal tube decompression • Neostigmine if no improvement within 48 hr

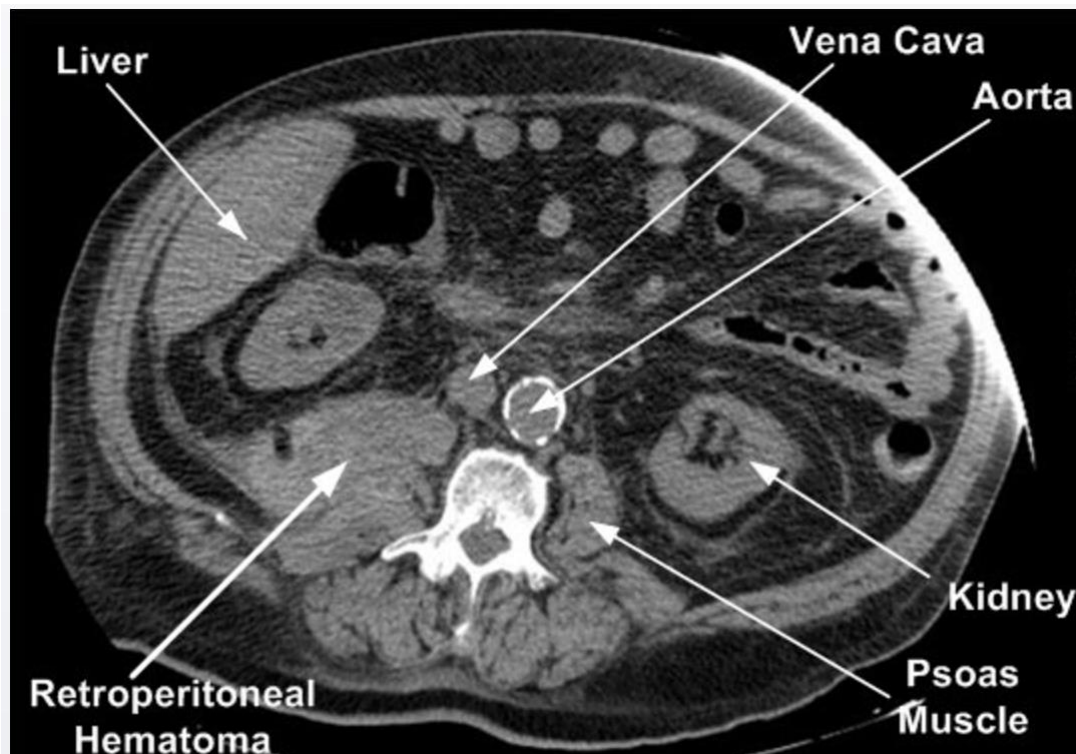
❖ N.B:

- Retroperitoneal hematoma can occur as a local vascular complication of cardiac catheterization, and often presents with sudden hemodynamic instability and ipsilateral flank or back pain.
- Cardiac catheterization requires vascular access through either the common femoral artery (CFA) or radial artery.
- When cannulating the CFA, the optimal entry site is the middle below the inguinal ligament.
- Arterial puncture above the inguinal ligament increases the risk of retroperitoneal hemorrhage as the area is directly over the retroperitoneal space.
- Bleeding in the retroperitoneal space cannot be controlled with manual compression and can lead to significant hemorrhage.
- Patients typically develop hemodynamic instability with significant hypotension, drop in hematocrit, and/or ipsilateral flank pain.

Retroperitoneal hemorrhage

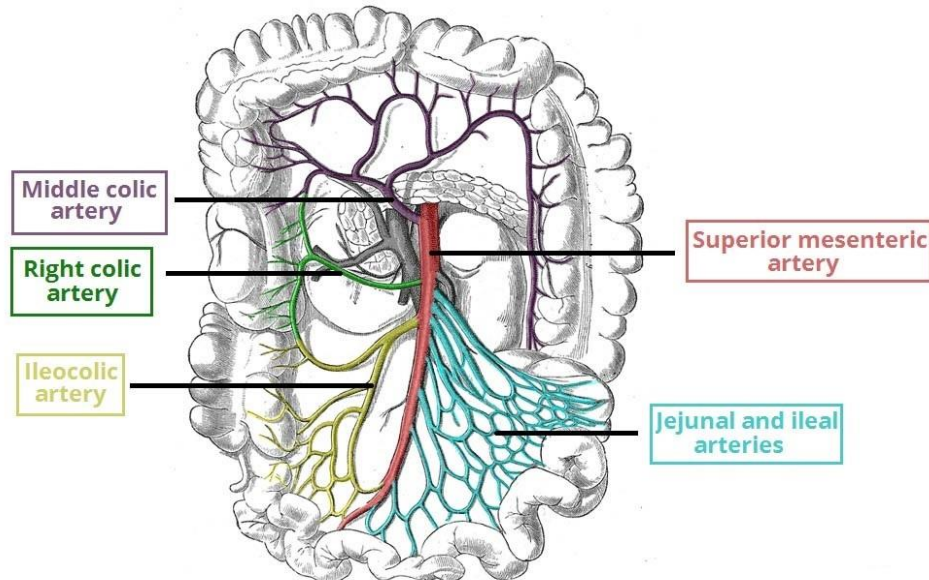


- Anticoagulation with warfarin places patients at risk for hemorrhage. Retroperitoneal hematoma may occur even without a supratherapeutic INR.
- **Diagnosis is confirmed with non-contrast CT scan of abdomen and pelvis or abdominal ultrasound.**
- Treatment is usually **supportive**, with intensive monitoring, bed rest, and intravenous fluids or blood transfusion.
- Surgical repair of hematomas or retroperitoneal hemorrhage is rarely required.



■ **Mesenteric Ischemia (MI):**

- Ischemic bowel disease is due to a **lack of blood flow to the mesentery of the bowel.**
- It is a progressive disease that begins with mild ischemia and progresses to full occlusion of blood flow.



A. **Acute mesenteric ischemia (AMI):**

- Acute mesenteric ischemia is the acute occlusion of mesenteric arteries, **most commonly the superior mesenteric artery.**
- AMI is most commonly due to abrupt arterial occlusion from either of the following:
 - Cardiac embolic events in the setting of **atrial fibrillation**, valvular disease (**infective endocarditis**), or cardiovascular aneurysms.
 - Acute thrombosis due to rupture of atheromatous plaque.
 - Low cardiac output states.
- **Acute mesenteric ischemia classically presents with acute-onset, severe, periumbilical pain out of proportion to physical examination findings.**
- **If bowel infarction occurs, patients may develop more focal abdominal tenderness (due to local inflammation/infarction), peritoneal signs (guarding, rebound tenderness), rectal bleeding (hematochezia), and sepsis.**
- Labs may show **increased lactic acid and leukocytosis.**
- The most common locations for infarction are **watershed areas.**
- **The best initial test is a CT scan of the abdomen.**
- **The most accurate test is mesenteric angiography.**

- Emergent laparotomy with resection of necrotic bowel is the most appropriate therapy.
- Endovascular therapy is indicated only if there is a clear reason to avoid surgery.

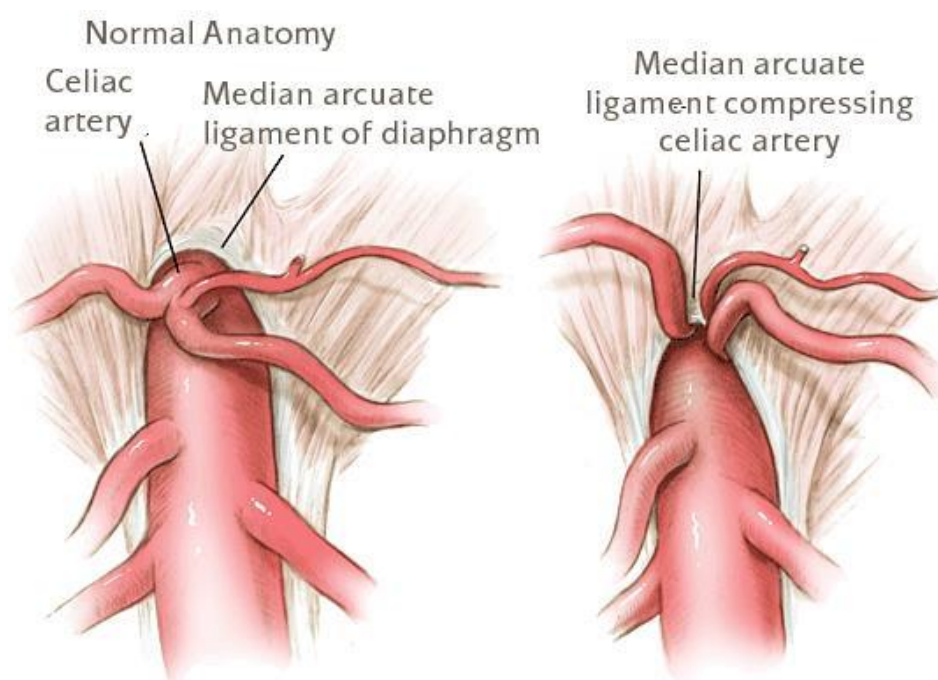
Acute mesenteric ischemia	
Presentation	<ul style="list-style-type: none"> • Rapid onset of periumbilical pain (often severe) • Pain out of proportion to examination findings • Hematochezia (late complication)
Risk factors	<ul style="list-style-type: none"> • Atherosclerosis (acute on chronic) • Embolic source (thrombus, vegetations) • Hypercoagulable disorders
Laboratory findings	<ul style="list-style-type: none"> • Leukocytosis • Elevated amylase & phosphate levels • Metabolic acidosis (elevated lactate)
Diagnosis	<ul style="list-style-type: none"> • CT (preferred) or MR angiography • Mesenteric angiography, if diagnosis is unclear

B. Chronic mesenteric ischemia (CMI):

- Chronic mesenteric ischemia results from atherosclerotic disease of 2 or more mesenteric vessels. **Most cases are due to atherosclerotic changes of the celiac or superior mesenteric arteries.**
- CMI commonly presents with **crampy postprandial epigastric pain (intestinal angina)**, food aversion, and weight loss. Patients may also report nausea, early satiety, and diarrhea.
- The anginal pain frequently starts within the first hour of eating and slowly resolves over the next 2 hours.
- The pathophysiology of the pain is most likely related to **shunting of blood away from the small intestine to meet the increased demand of the stomach.**
- It is analogous to angina of the heart but affects only the gut. In intestinal ischemia, eating is the equivalent of exertion in "chest pain with exertion".
- **The best diagnostic test is angiography.**
- Angiography is done first to delineate the location of the lesions; then **stenting or bypass reestablishes blood flow to allow surgical correction.**

Chronic mesenteric ischemia	
Etiology	<ul style="list-style-type: none"> • Atherosclerosis (smoking, dyslipidemia)
Clinical features	<ul style="list-style-type: none"> • Crampy, postprandial, epigastric pain • Food aversion & weight loss
Diagnosis	<ul style="list-style-type: none"> • Signs of malnutrition, abdominal bruit • CT angiography (preferred), Doppler ultrasonography
Management	<ul style="list-style-type: none"> • Risk reduction (eg, tobacco reduction), nutritional support • Endovascular or open surgical revascularization

- **Median Arcuate Ligament Syndrome (MALS):**
 - Patients with MALS present with severe postprandial abdominal pain, nausea, and weight loss.
 - The condition is caused by external compression of the celiac trunk by the median arcuate ligament.
 - MALS is a diagnosis of exclusion. Confirm with duplex ultrasonography to measure blood flow through the celiac artery.
 - The general approach to treatment of MALS is surgical decompression of the celiac artery.



- **Ischemic colitis (IC):**

- **Etiology:**

- Ischemic colitis is a common complication of vascular surgery (**Repair of an abdominal aortic aneurysm**), as patients are often older and have extensive underlying atherosclerosis. Contributing factors may include loss of collateral circulation, manipulation of vessels with surgical instruments, prolonged aortic clamping, and impaired blood flow through the inferior mesenteric artery.
- The most commonly involved segments of the colon include **the splenic flexure** at the "watershed" line between the territory of the superior and inferior mesenteric arteries and the **rectosigmoid junction** at the watershed between the sigmoid artery and superior rectal artery.

- **Finding:**

- Ischemic colitis is characterized by **acute abdominal pain and lower gastrointestinal bleeding**.
- **It typically follows an episode of hypotension and most commonly affects arterial watershed areas at the splenic flexure and rectosigmoid junction.**
- Fever, nausea, elevated lactic acid, and leukocytosis may also be seen.

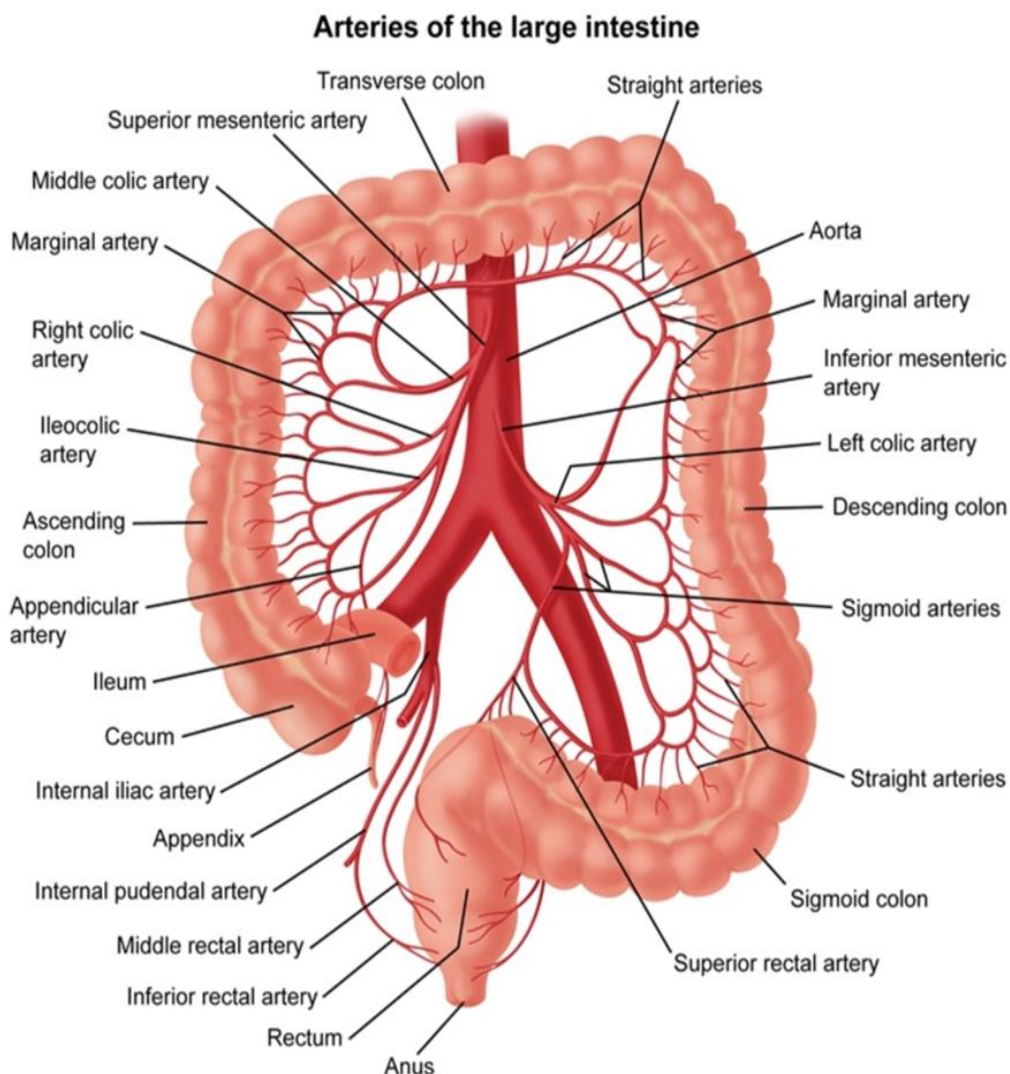
- **Diagnosis:**

- Abdominal CT scan with intravenous contrast may show thickened bowel wall, although it may demonstrate only nonspecific findings. **CT should be performed urgently to identify patients with ischemic colitis who need immediate surgical intervention** (extensive bowel damage, perforation).
- Colonoscopy shows segments of cyanotic mucosa and hemorrhagic ulcerations, with a sharp transition from affected to unaffected mucosa.



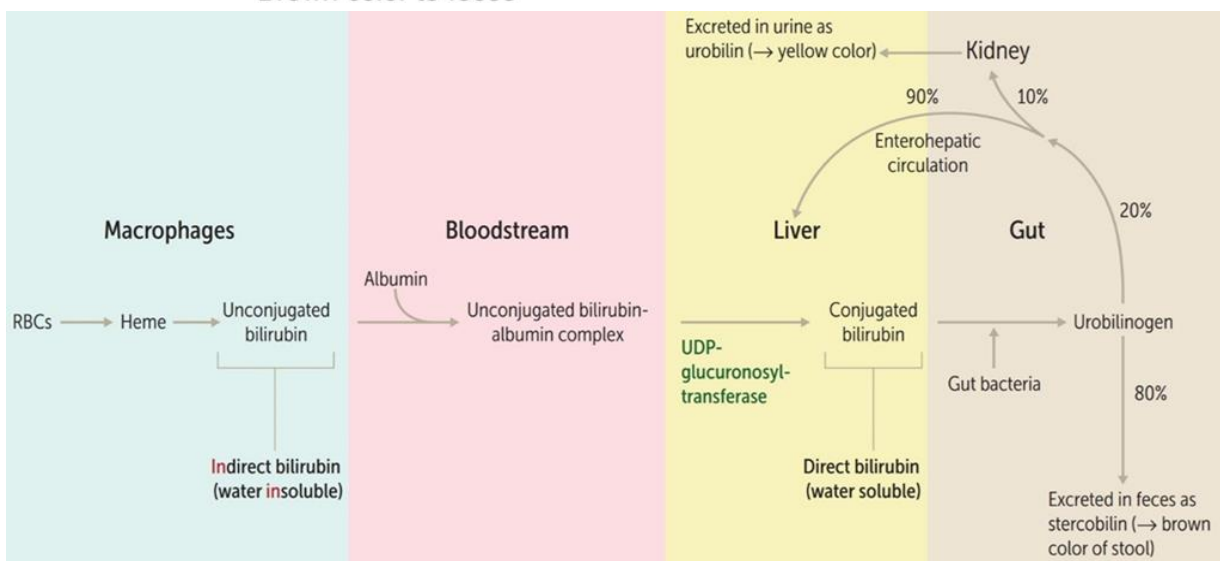
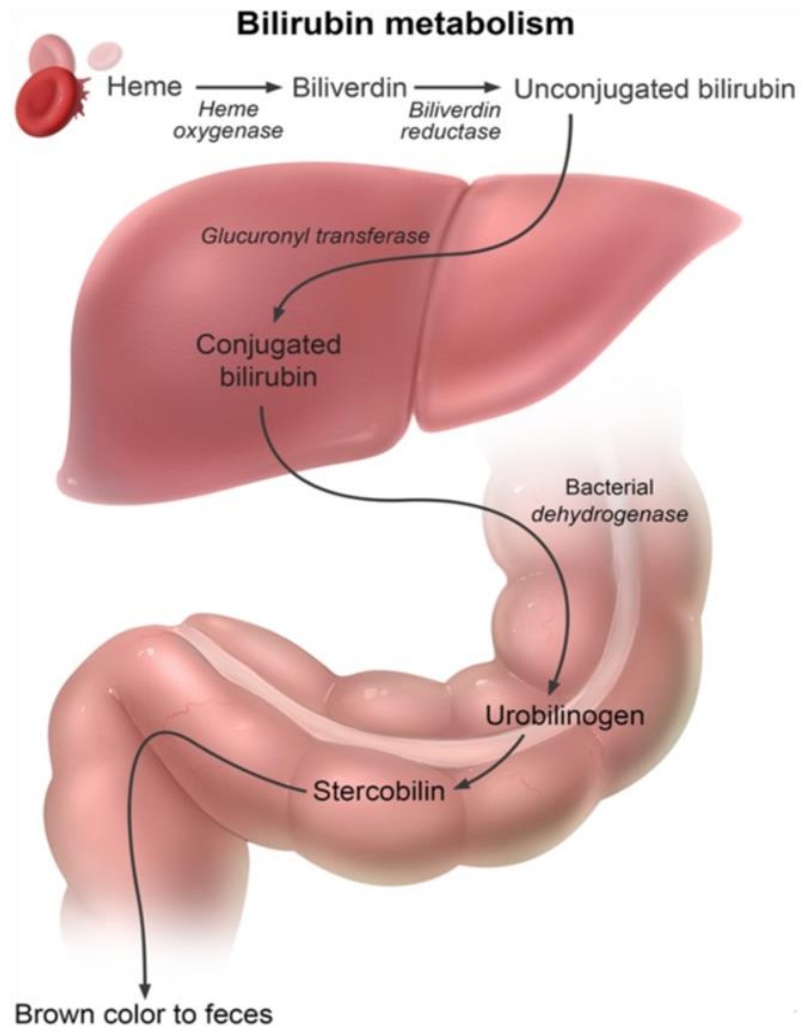
- **Treatment:**

- **Unless the patient has perforation or bowel gangrene, most cases are managed conservatively with intravenous fluids, bowel rest, and antibiotics.**
- **Colonic resection if necrosis develops.**



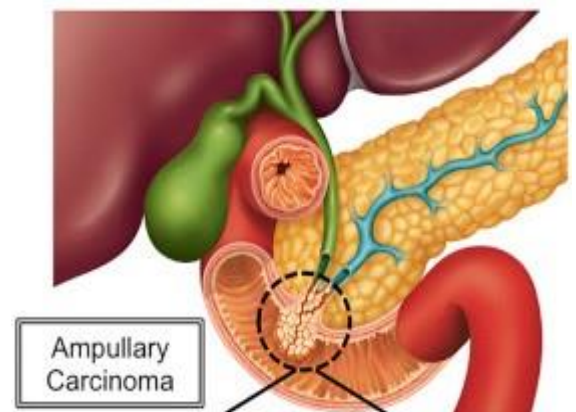
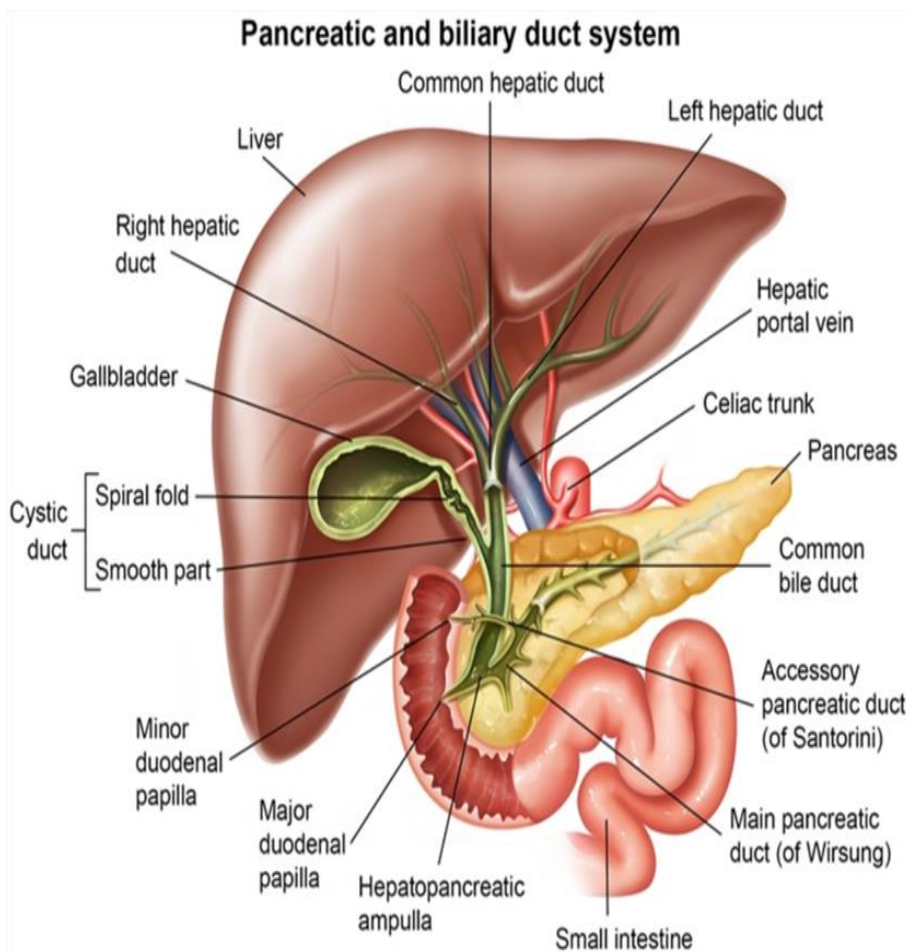
Colonic ischemia	
Pathophysiology	<ul style="list-style-type: none"> • Usually nonocclusive, “watershed” ischemia • Underlying atherosclerotic disease • State of low blood flow (eg, hypovolemia)
Clinical features	<ul style="list-style-type: none"> • Moderate abdominal pain & tenderness • Hematochezia, diarrhea • Leukocytosis, lactic acidosis
Diagnosis	<ul style="list-style-type: none"> • CT scan: Colonic wall thickening, fat stranding • Endoscopy: Edematous & friable mucosa
Management	<ul style="list-style-type: none"> • Intravenous fluids & bowel rest • Antibiotics with enteric coverage • Colonic resection if necrosis develops

Obstructive jaundice:



- Jaundice may be hemolytic, hepatocellular, or obstructive.
- Obstructive jaundice has **elevations of both fractions of bilirubin, modest elevation of transaminases, and very high levels of alkaline phosphatase.**
- Remember that bile's made up of conjugated bilirubin and **this blockage basically causes pressure to rise in the bile duct, which literally causes bile to leak through the tight junctions between hepatocytes, but that's not the only thing that leaks out though; bile salts, bile acids, and cholesterol all can get into the blood.** If they deposit in the skin, it could lead to **itchiness or pruritus**, but also lead to things like **hypercholesterolemia** and xanthomas.
- The excess CB is excreted in the urine → **dark urine.**
- Pale, **clay-colored (acholic) stool.** Fat malabsorption (steatorrhea, weight loss)
- **The first step in the workup is an U/S looking for dilatation of the biliary ducts,** as well as further clues as to the nature of the obstructive process.
- **In obstruction caused by stones, the stone that is obstructing the common duct is seldom seen, but stones are seen in the gallbladder, which because of chronic irritation cannot dilate.**
- **In malignant obstruction, a large, thin-walled, distended gallbladder is often identified (Courvoisier-Terrier sign).**
- Obstructive jaundice caused by stones should be suspected in the **female, fatty, fertile in her forties,** who has high alkaline phosphatase, dilated ducts on sonogram, and nondilated gallbladder full of stones. The next step in that case is an **endoscopic retrograde cholangiopancreatography (ERCP) to confirm the diagnosis, perform a sphincterotomy, and remove the common duct stone.** Cholecystectomy should usually follow during the same hospitalization.
- Obstructive jaundice caused by a tumor could be caused by **adenocarcinoma of the head of the pancreas, adenocarcinoma of the ampulla of Vater, or cholangiocarcinoma arising in the common duct itself.**
- **Once a tumor has been suspected by the presence of dilated gallbladder in the sonogram, the next test should be CT scan.** Pancreatic cancers that have produced obstructive jaundice are often big enough to be seen on CT. If the CT is negative, ERCP is the next step.

- Ampullary cancers or cancers of the common duct by virtue of their strategic location produce obstruction when they are very small, and therefore may not be seen on CT. However, **endoscopy will show ampullary cancers and the cholangiography will show intrinsic tumors arising from the duct.**
- **Ampullary cancer should be suspected when malignant obstructive jaundice coincides with anemia and positive blood in the stools.** Can bleed into the lumen like any other mucosal malignancy, at the same time that it can obstruct biliary flow by virtue of its location. Given that combination, endoscopy should be the first test.
- If cancer is suspected and a tumor is identified on CT or ERCP, it should be resected if no contraindications are present (evidence of metastatic disease).
- Pancreatic cancer is seldom cured, even when resectable by the Whipple operation (pancreatoduodenectomy).
- Ampullary cancer and cancer of the common duct have a much better prognosis (about 40% cure).

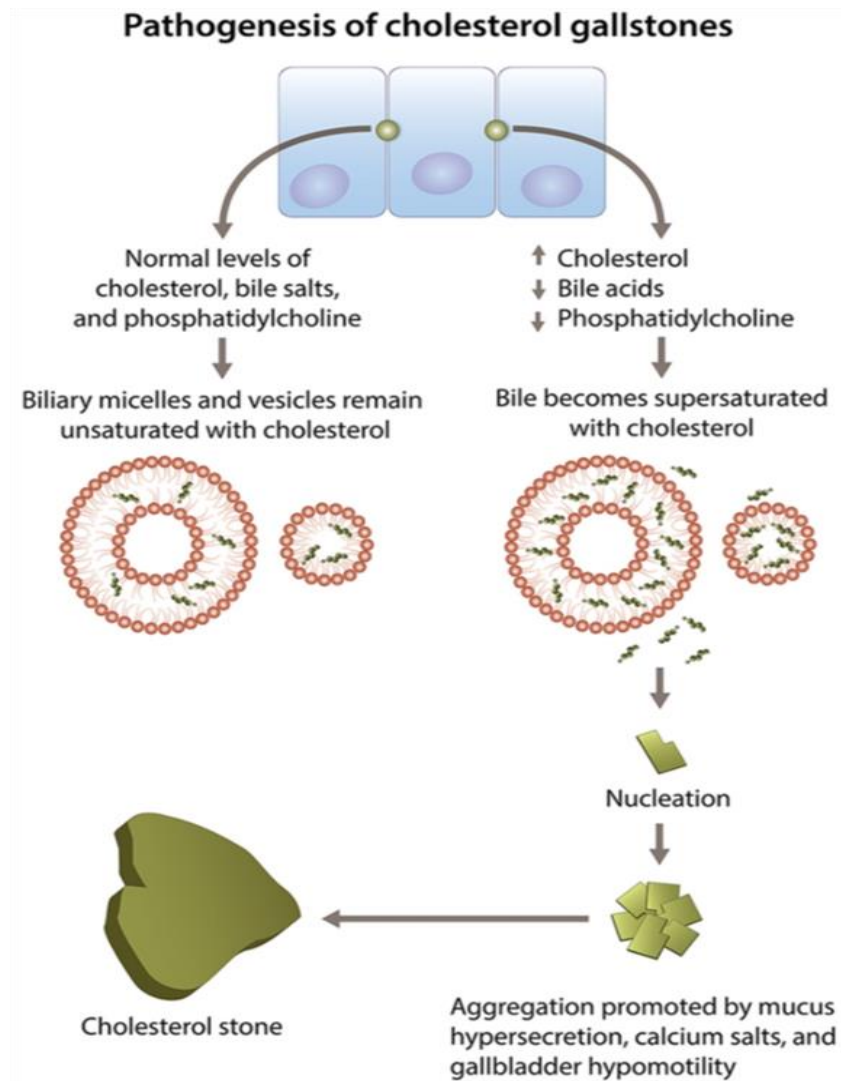


- Cholelithiasis (Gallstones):

- Solid, round stones in the gallbladder.
- Classically, cholelithiasis (gallstone disease) is most common in those who are "fat, fertile, female, and forty".
- The gallstones are formed by the aggregation of bile constituents and are categorized as **cholesterol stones, pigment stones, or mixed stones**.
- Due to precipitation of cholesterol (cholesterol stones) or bilirubin (bilirubin stones) in bile.
- Arises with:
 - Supersaturation of cholesterol or bilirubin.
 - Decreased phospholipids (lecithin) or bile acids (normally increase solubility).
 - Stasis.

- A. Cholesterol stones (yellow):

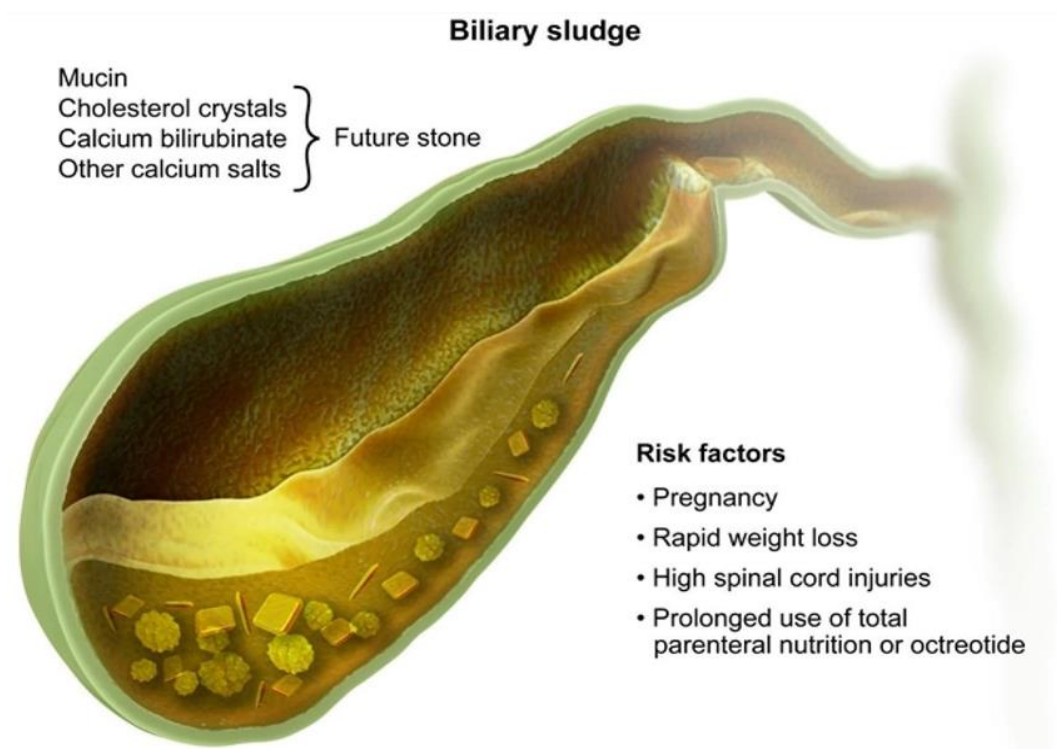
- **The most common type (90%)**, especially in the U.S.
- Usually **radiolucent** (10% are radiopaque due to associated calcium).
- Risk factors include **age (40s), estrogen (female gender, obesity, multiple pregnancies and oral contraceptives), fibrate, Native American ethnicity, Crohn disease, and cirrhosis**.
- Estrogenic influence **increases cholesterol synthesis by upregulating hepatic HMG-CoA reductase activity**, which causes the bile to become supersaturated with cholesterol.
- Progesterone reduces bile acid secretion and **slows gallbladder emptying**. When the gallbladder is hypomotile or there is more cholesterol than bile salts, the cholesterol precipitates into insoluble crystals that eventually form to make gallstones.
- Estrogen-induced cholesterol hypersecretion and progesterone-induced gallbladder hypomotility are responsible for the increased incidence of cholelithiasis in women who are pregnant or using oral contraceptives.
- Suppression of cholesterol 7 α -hydroxylase activity (through fibrate medications such as bezafibrate, fenofibrate, and ciprofibrate) **reduces the conversion of cholesterol into bile acids, resulting in an increased concentration of cholesterol within the bile**.
- When the gallbladder is hypomotile or there is more cholesterol than can dissolve into the bile salts, the cholesterol precipitates into insoluble crystals that eventually form to make gallstones.



B. **Bilirubin stones (pigmented):**

- Composed of bilirubin.
- Usually **radiopaque**.
- Risk factors include **extravascular hemolysis** (increased bilirubin in bile) and **biliary tract infection** (E coli, Ascaris lumbricoides, and Clonorchis sinensis).
- **Brown** pigment stones typically arise **secondary to infection of the biliary tract**, which results in the release of β -glucuronidase by injured hepatocytes and bacteria.
- **The presence of this enzyme contributes to the hydrolysis of bilirubin glucuronides and increases the amount of unconjugated bilirubin in bile.** Therefore, biliary infection with Escherichia coli, Ascaris lumbricoides, or clonorchis sinensis significantly elevates the risk of developing brown pigment stones.

- **Black** pigment stones form within the gallbladder when increased unconjugated bilirubin precipitates in bile as **calcium bilirubinate**.
 - Elevated levels of circulating unconjugated bilirubin are primarily associated with **chronic extravascular hemolysis** (sickle cell anemia, B-thalassemia, hereditary spherocytosis).
 - Diagnosis of cholelithiasis in all patients is **best confirmed with ultrasound**.
 - Gallstones are usually **asymptomatic**; complications include **biliary colic, acute and chronic cholecystitis, ascending cholangitis, gallstone ileus, and gallbladder cancer**.
 - **Asymptomatic** patients **do not require intervention**, but **symptomatic** cholelithiasis is usually treated with **laparoscopic cholecystectomy**.
- ❖ N.B:
- The presence of proteins and fatty acids in the duodenum acts as a stimulus for release of cholecystokinin (CCK) which in turn stimulates the contraction of the gallbladder.
 - **In patients on total parenteral nutrition or prolonged fasting, the normal stimulus for CCK release and gallbladder contraction is absent. This leads to biliary stasis and promotes the formation of bile sludge and gallstones.**
 - Small-bowel (ileal) resection also contributed to the formation of gallstones. Decreased enterohepatic circulation of bile acids results in altered hepatic bile composition, which becomes supersaturated with cholesterol and promotes gallstone formation.



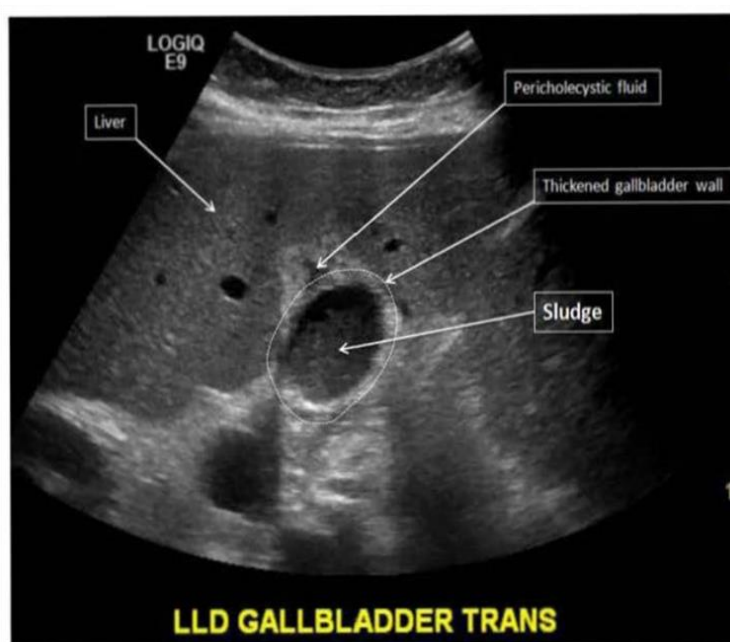
- Biliary colic:

- Biliary colic occurs when a stone temporarily occludes the cystic duct.
- This causes **colicky pain in the right upper quadrant radiating to the right shoulder and back, often triggered by ingestion of fatty food**, accompanied by nausea and vomiting, but without signs of peritoneal irritation or systemic signs of inflammatory process.
- **Biliary colic occurs due to increased intra-gallbladder pressure that is created when the gallbladder contracts against an obstructed cystic duct.**
- The episode is **self-limited** (10, 20, maybe 30 minutes), or easily aborted by anticholinergics.
- U/S establishes diagnosis of gallstones and elective laparoscopic cholecystectomy is indicated.

- Acute cholecystitis:

- Acute cholecystitis is characterized by **inflammation** and distension of the gallbladder due to obstruction of the cystic duct by a gallstone.
- Typical features include acute right upper quadrant pain and tenderness, fever, vomiting, and leukocytosis.
- The pain may **radiate to the right scapula** or be accompanied by **Murphy's sign**, described as **worsening of right upper quadrant pain with inspiration that sometimes causes the patient to suddenly hold their breath**.
- Uncomplicated cholecystitis can cause mild elevations in transaminases, total serum bilirubin from 1-4 mg/dL and serum amylase without obvious common bile duct or pancreatic disease. In these cases, the laboratory elevations are usually **due to passage of sludge or pus in the common bile duct**. However, alkaline phosphatase is usually not elevated without associated cholangitis or choledocholithiasis.
- Complicated cholecystitis: The tissue behind the duct obstruction becomes inflamed, from stasis leading to bacterial overgrowth. The subsequent ischemic changes most commonly lead to **gangrene and perforation, generalized peritonitis, or a well-circumscribed abscess**. Other potential complications include **cholangitis and chronic cholecystitis**.
- **U/S is diagnostic in most cases (gallstones, thick-walled gallbladder, and pericholecystic fluid).**
- **In equivocal cases, a radionuclide scan (HIDA) might be needed, and would show tracer uptake in the liver, common duct, and duodenum, but not in the occluded gallbladder.**

- Treatment for acute cholecystitis includes supportive care (nothing by mouth, intravenous antibiotics, and analgesics). However, early cholecystectomy (within 72 hours) reduces disease duration, duration of hospitalization, and mortality when compared to delayed cholecystectomy (>7 days after hospitalization).
- Early cholecystectomy is also advised for patients with other complications of gallstones, such as gallstone pancreatitis.
- Laparoscopic cholecystectomy is the surgical procedure of choice in patients without contraindications.



Management of gallstones	
Gallstones without symptoms	<ul style="list-style-type: none"> • No treatment necessary in most patients
Gallstones with typical biliary colic symptoms	<ul style="list-style-type: none"> • Elective laparoscopic cholecystectomy • Possible ursodeoxycholic acid in poor surgical candidates
Complicated gallstone disease (acute cholecystitis, choledocholithiasis, gallstone pancreatitis)	<ul style="list-style-type: none"> • Cholecystectomy within 72 hours

- Postcholecystectomy syndrome:

- Postcholecystectomy syndrome (PCS) refers to **persistent abdominal pain or dyspepsia (nausea) that occurs either postoperatively (early) or months to years (late) after a cholecystectomy.**
- PCS can be due to **biliary** (retained common bile duct or cystic duct stone, sphincter of Oddi dysfunction) or **extra-biliary** (pancreatitis, peptic ulcer disease, coronary artery disease) causes.
- Patients usually notice the same pain they had prior to surgery, new pain just after surgery, or the same pain that never went away.
- Laboratory findings can include elevated alkaline phosphatase, mildly abnormal serum aminotransferases, and dilated common bile duct on abdominal ultrasound. These findings usually suggest **common bile duct stones or biliary sphincter of Oddi dysfunction.**
- The next step involves endoscopic ultrasound **followed by direct visualization [endoscopic retrograde cholangiopancreatography (ERCP) or magnetic resonance cholangiopancreatography] for final diagnosis and guiding therapy.**
- Treatment for PCS is **directed at the causative factor.**

- Acalculous cholecystitis:

- **Acute acalculous cholecystitis is an acute inflammation of the gallbladder in the absence of gallstones that is most commonly seen in hospitalized and severely ill patients.**
- Common predisposing conditions include **recent surgery** (particularly cardiopulmonary, aortic, or abdominal), severe trauma, extensive burns, sepsis or shock, prolonged fasting or total parenteral nutrition, or critical illness requiring mechanical intubation.
- **Acalculous cholecystitis is likely due to cholestasis and gallbladder ischemia leading to secondary infection by enteric organisms and resultant edema and necrosis of the gallbladder.** Most patients affected by this condition have **no prior history of gallbladder disease.**
- Acalculous cholecystitis is a **serious condition that can lead to sepsis and death if undetected.**
- The clinical signs of disease (fever, leukocytosis) are vague, and patients most vulnerable to this condition are typically **non-communicative** due to their general medical condition.
- The best way to make the diagnosis is a **high degree of clinical suspicion and confirmation with imaging studies.** Complications include gangrene, perforation, and emphysematous cholecystitis.
- **Radiologic signs include gallbladder wall thickening and the presence of pericholecystic fluid without gall bladder stones.**
- Patients with unclear ultrasound findings may need abdominal CT scan or cholescintigraphy (hepatobiliary iminodiacetic acid [HIDA]) scans, which are more sensitive and specific for the diagnosis.

- The immediate treatment in critically ill patients includes antibiotics followed by drainage of any associated abscesses is the definitive therapy once the patient's medical condition improves.

Acalculous cholecystitis	
Risk factors	<ul style="list-style-type: none"> • Severe trauma or recent surgery • Prolonged fasting or TPN • Critical illness (eg, sepsis, ICU)
Clinical presentation	<ul style="list-style-type: none"> • Fever, leukocytosis, ↑ LFTs, RUQ pain • Jaundice & RUQ mass less common
Diagnosis	<ul style="list-style-type: none"> • Abdominal ultrasound (preferred) • HIDA or CT scan if needed
Treatment	<ul style="list-style-type: none"> • Enteric antibiotic coverage • Cholecystostomy for initial drainage • Cholecystectomy once clinically stable

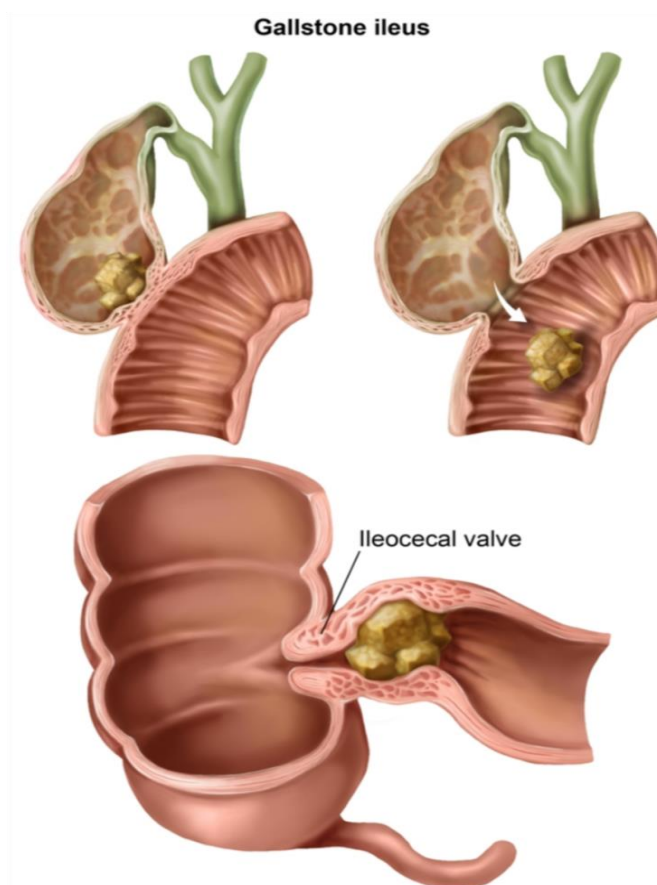
HIDA = hepatobiliary iminodiacetic acid; ICU = intensive care unit; LFTs = liver function tests; RUQ = right upper quadrant; TPN = total parenteral nutrition.

- Emphysematous cholecystitis:
 - Fever, right upper quadrant (RUQ) pain, and gas in the gallbladder wall is presenting with clinical manifestations of acute emphysematous cholecystitis, a life-threatening form of acute cholecystitis due to infection with gas-forming bacteria (Clostridium, some Escherichia coli strains).
 - Predisposing factors include vascular compromise (obstruction or stenosis of the cystic artery), immunosuppression (diabetes mellitus) and gallstones.
 - Crepitus in the abdominal wall adjacent to the gallbladder is occasionally detectable.
 - Complications include gangrene and perforation, the latter of which may transiently relieve pain but subsequently result in peritoneal signs
 - Diagnosis is confirmed with imaging demonstrating air-fluid levels in the gallbladder (as gas leaks into it), gas in the gallbladder wall, and occasionally pneumobilia (air within the hepatobiliary system).
 - Treatment requires emergent cholecystectomy and broad-spectrum parenteral antibiotic therapy (ampicillin-sulbactam).

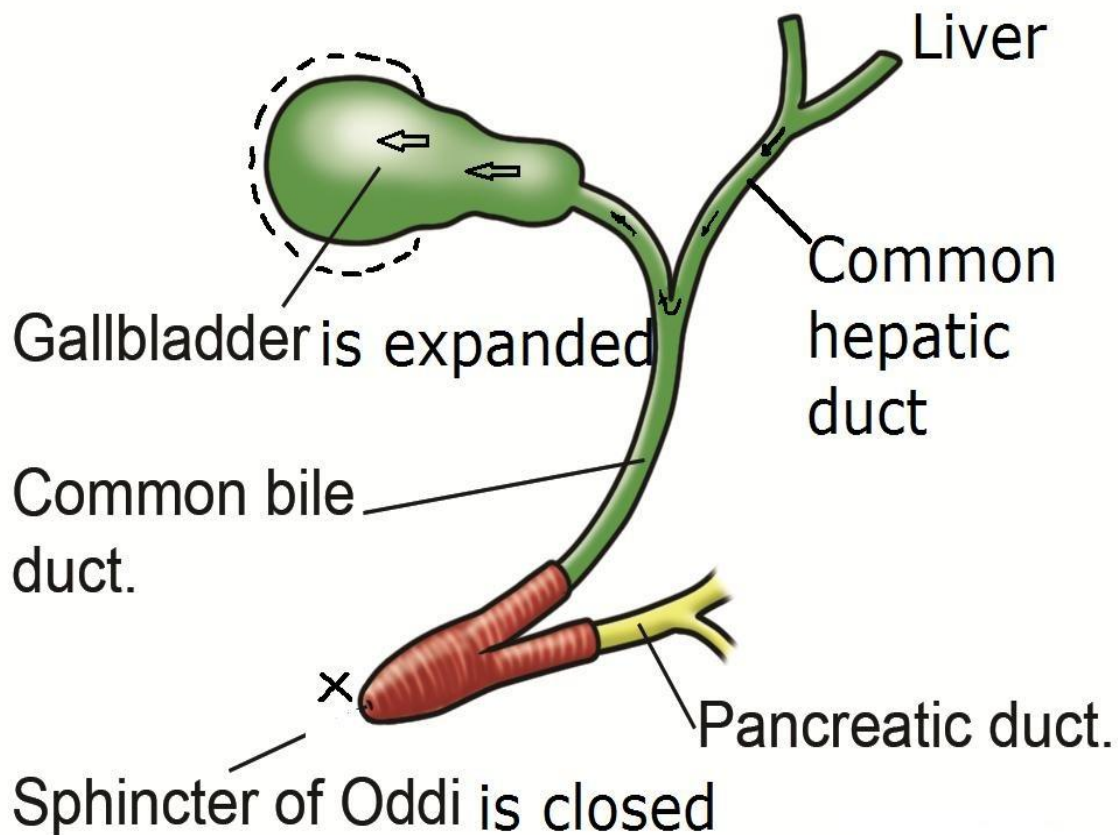
Emphysematous cholecystitis	
Risk factors	<ul style="list-style-type: none"> • Diabetes mellitus • Vascular compromise • Immunosuppression
Clinical presentation	<ul style="list-style-type: none"> • Fever, right upper quadrant pain, nausea/vomiting • Crepitus in abdominal wall adjacent to gallbladder
Diagnosis	<ul style="list-style-type: none"> • Air-fluid levels in gallbladder, gas in gallbladder wall • Cultures with gas-forming <i>Clostridium</i>, <i>Escherichia coli</i> • Unconjugated hyperbilirubinemia, mildly elevated aminotransferases
Treatment	<ul style="list-style-type: none"> • Emergency cholecystectomy • Broad-spectrum antibiotics with <i>Clostridium</i> coverage (eg, piperacillin-tazobactam)



- Gallstone ileus:
- Gallstone ileus results from small bowel obstruction due to a gallstone that has passed through a biliary-enteric fistula into the small bowel.
- Cholecystitis, which predisposes to biliary-enteric adhesions, is the most important risk factor, and patients are more commonly elderly women, which reflects their higher prevalence of gallstone disease.
- As the stone advances it may cause intermittent "tumbling" obstruction with diffuse abdominal pain and vomiting until finally lodging in the ileum, the narrowest section of the bowels, several days later.
- The patient with stuttering episodes of nausea and vomiting, pneumobilia (air in the biliary tree), hyperactive bowel sounds and dilated loops of bowels likely has a gallstone ileus.
- In addition to experiencing colicky pain and vomiting, patients may report distension and inability to pass flatus or stool and show signs of hypovolemia (hypotension, tachycardia).
- Diagnosis can be confirmed by abdominal CT scan, which may reveal gallbladder wall thickening, pneumobilia, and an obstructing stone.
- Treatment is surgical and involves removal of the stone and either simultaneous or delayed cholecystectomy.



- Sphincter of Oddi dysfunction (SOD):
- Sphincter of Oddi dysfunction is a functional biliary disorder due to dyskinesia or stenosis of the sphincter of Oddi.
- The sphincter of Oddi is a muscular valve controlling the flow of bile and pancreatic juice into the duodenum.
- SOD which can develop following any inflammatory process (surgery, pancreatitis), encompasses 2 separate physiologic entities dyskinesia and stenosis of the sphincter of Oddi.
- Obstruction of flow through the sphincter may result in retention of bile causing a functional biliary disorder that mimics a structural lesion.
- Recurrent episodic pain in the right upper quadrant or epigastric region, with corresponding aminotransferase and alkaline phosphatase elevations, is common; visualization of a dilated common bile duct in the absence of stones increases the likelihood of SOD.
- Sphincter of Oddi manometry is the gold standard for the diagnosis of SOD.
- Sphincterotomy is the treatment of choice in most cases.

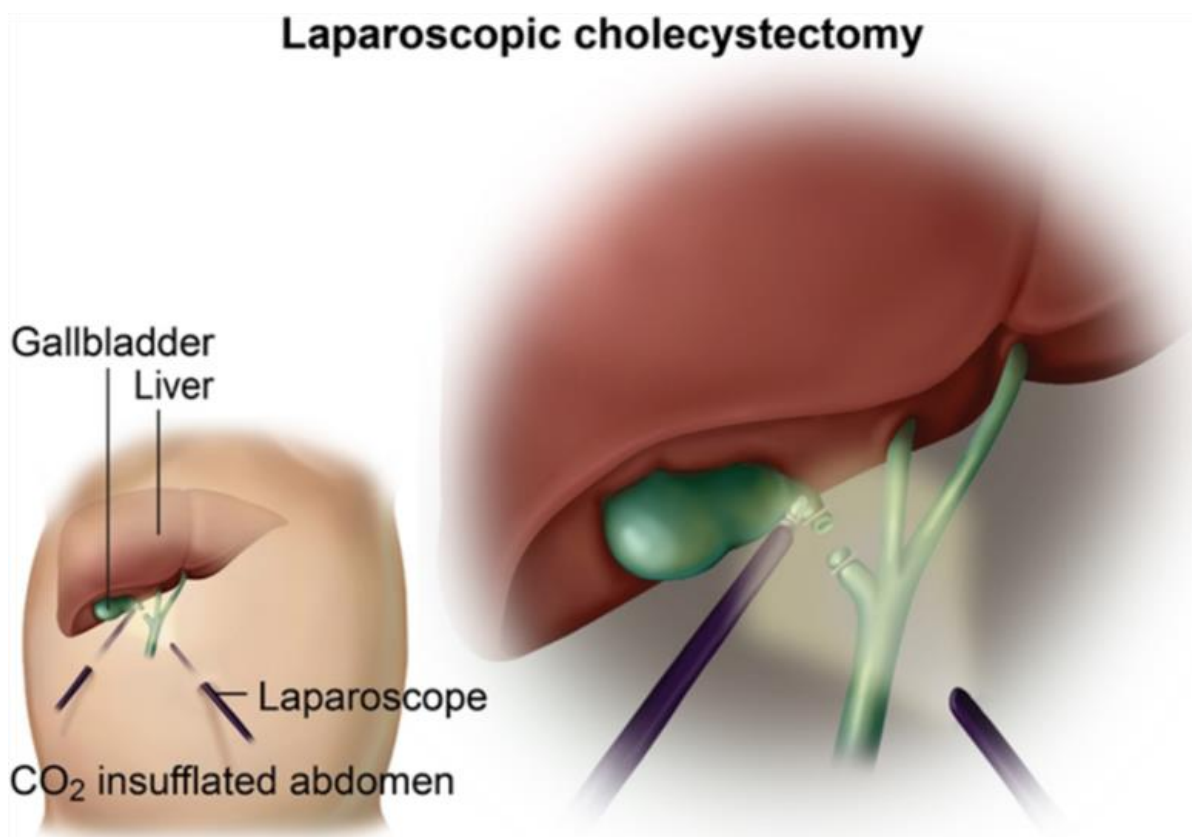


- Porcelain gallbladder:
- Porcelain gallbladder is a term used to describe the **calcium-laden gallbladder wall with bluish color and brittle consistency often associated with chronic cholecystitis**.
- The pathogenesis of the condition remains unclear, but it is thought that calcium salts are deposited intramurally due to the natural progression of **chronic inflammation or chronic irritation from gallstones**.
- Patients can be **asymptomatic**, have right upper quadrant pain, or have a firm and nontender right upper quadrant mass on examination.
- Plain x-rays can show a rimlike calcification in the area of the gallbladder, and CT scan typically reveals a **calcified rim in the gallbladder wall with a central bile-filled dark area**.
- Porcelain gallbladder has been associated with increased risk for **gallbladder adenocarcinoma (2%-5% in some studies)**.
- **Cholecystectomy is typically considered for patients with porcelain gallbladder**, particularly if they are symptomatic or have incomplete mural calcification.



- ❖ N.B:
1. Endoscopic retrograde cholangiopancreatography (ERCP), **a minimally invasive endoscopic technique** used to visualize and intervene upon the biliary and pancreatic ducts, is widely used for stone removal, tissue sampling, stent placement, and sphincterotomy.
 - In some patients, ERCP with manometry can confirm the diagnosis of sphincter of Oddi dysfunction (SOD), which typically causes biliary-type pain with no obvious etiology.
 - Post-ERCP complications can occur; **postprocedural abdominal pain is relatively common and may be due to infection (ascending cholangitis), perforation, or pancreatitis**.
 - **The most common complication is post-ERCP pancreatitis**, which occurs in up to 10% of patients and is particularly prevalent in those with SOD.

- Classic symptoms of pancreatitis include nausea, vomiting, and epigastric pain that radiates to the back and worsens with eating. Fever may occur due to inflammation.
 - Acute pancreatitis is diagnosed in patients meeting >2 of the following criteria:
 - Severe epigastric pain (often radiating to the back).
 - Amylase or lipase >3 times the upper limit of normal.
 - Characteristic findings of acute pancreatitis on imaging (CT, MRI, ultrasound).
 - Because the first 2 criteria are sufficient to confirm the diagnosis, serum amylase and lipase should be obtained as part of the initial workup in all patients with suspected pancreatitis.
 - These enzymes rise within several hours of the development of symptoms whereas CT findings may remain normal for up to 48 hours.
2. Laparoscopic intervention is a minimally invasive option for a number of intraabdominal surgeries (cholecystectomy, hysterectomy).
- Preparation for the surgery requires insufflation of CO₂ gas into the abdominal cavity to create space for surgical maneuvering and to facilitate adequate camera visibility during the procedure.
 - CO₂ is introduced into the abdomen via a needle or port, with continuous monitoring of intraabdominal pressure.
 - Peritoneal stretch receptors sense the increase in intraabdominal pressure and respond by triggering increased vagal tone. Close monitoring is required as patients may develop severe bradycardia, atrioventricular block, and sometimes asystole. The increased intraabdominal pressure can also cause a mechanical increase in systemic vascular resistance leading to elevations in blood pressure.



▪ Acute ascending cholangitis:

- Acute ascending cholangitis is a far more deadly disease, in which stones have reached the common duct producing partial obstruction and ascending infection.
- Biliary stasis predisposes to AC and the most common causes are due to bile duct obstruction from gallstones, malignancy, or stenosis. In the setting of stasis, the bile-blood barrier can be disrupted, allowing bacteria and toxins from the hepatobiliary system to translocate into the blood stream.
- Fever, jaundice, and right upper quadrant abdominal pain (Charcot triad) are consistent with acute cholangitis. Confusion and hypotension (Reynolds pentad) are also sometimes seen in severe AC If not treated promptly. AC can lead to septic shock.
- Laboratory results usually show leukocytosis and neutrophilia in addition to elevations in alkaline phosphatase, gamma-glutamyl transpeptidase, and direct bilirubin.
- Ultrasound or CT scan can be helpful in confirming the diagnosis and most frequently shows common bile duct dilation.
- Supportive care, broad-spectrum antibiotics, and biliary drainage, preferably by endoscopic retrograde cholangiopancreatography with sphincterotomy, are the mainstays of treatment. Other options for biliary decompression include percutaneous transhepatic cholangiography and open surgical decompression.

Acute cholangitis	
Clinical presentation	<ul style="list-style-type: none"> • Fever, jaundice, right upper quadrant pain (Charcot triad) • Mental status changes, hypotension (Reynolds pentad) • Liver failure • Acute kidney injury
Diagnosis	<ul style="list-style-type: none"> • Biliary dilation on ultrasound or CT scan • ↑ Alkaline phosphatase, gamma-glutamyl transpeptidase, direct bilirubin • Leukocytosis, ↑ C-reactive protein
Treatment	<ul style="list-style-type: none"> • Biliary drainage: Endoscopic retrograde cholangiopancreatography with sphincterotomy or percutaneous transhepatic cholangiography • Broad-spectrum antibiotics: Beta-lactam/beta-lactamase inhibitor, third-generation cephalosporin + metronidazole

- Hernias:

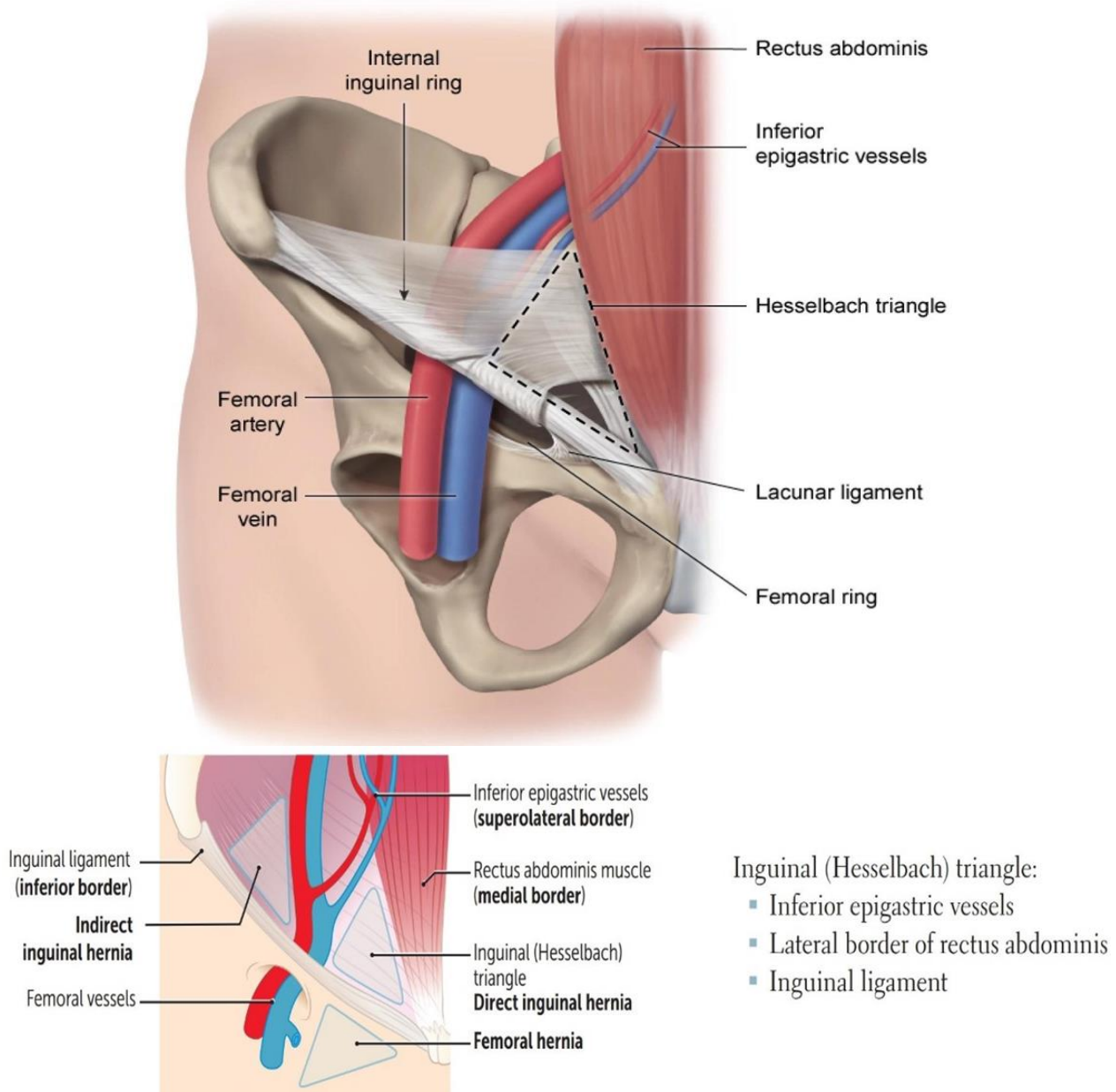
- Protrusion of peritoneum through an opening, usually **at a site of weakness**.
- Contents may be at risk for **incarceration** (not reducible back into abdomen/pelvis) and **strangulation** (ischemia and necrosis).
- Complicated hernias can present with tenderness, erythema, fever.

- A. Indirect inguinal hernia:

- In early fetal life, the testes are located in the peritoneal cavity and descend into the scrotum before birth.
- During the descent, they are **accompanied by an invagination of the peritoneum called the processus vaginalis**.
- This structure follows the testes into the scrotum and **obliterates after testicular descent is complete**.
- Remnants of the processus vaginalis form the tunica vaginalis of the testes.
- Failure of obliteration of the processus vaginalis leads to a **persistent connection between the scrotum and the peritoneal cavity through the inguinal canal**.
- If the opening is small and allows for fluid leakage only, hydrocele occurs. Diagnosis is by transillumination of the scrotum and scrotal ultrasound, which reveal fluid (only) in the tunica vaginalis sac.
- If the communication between the peritoneal cavity and the scrotum allows for the passage of abdominal organs, an indirect inguinal hernia develops.
- A bulge on the groin that increases during straining is the typical manifestation.
- Management:
 - Surgical hernia repair is recommended for the management of most abdominal hernias.
 - Elective surgery is indicated in reducible and incarcerated hernias.
 - Emergency surgery is indicated in obstructed or strangulated hernias.
 - Conservative management (observation) is indicated in asymptomatic wide-necked hernias in patients with high operative risk: A truss or corset may be considered in these patients to decrease the risk of obstruction and strangulation.

B. **Direct inguinal hernia:**

- Protrudes through **the inguinal (Hesselbach) triangle**. Bulges directly through abdominal wall **medial to inferior epigastric vessels**.
- Goes through the **external (superficial) inguinal ring only**.
- **Usually in older men due to an acquired weakness in the transversalis fascia.**
- **MDs don't Lie:**
 - o **M**edial to inferior epigastric vessels = **D**irect hernia.
 - o **L**ateral to inferior epigastric vessels = **I**ndirect hernia.

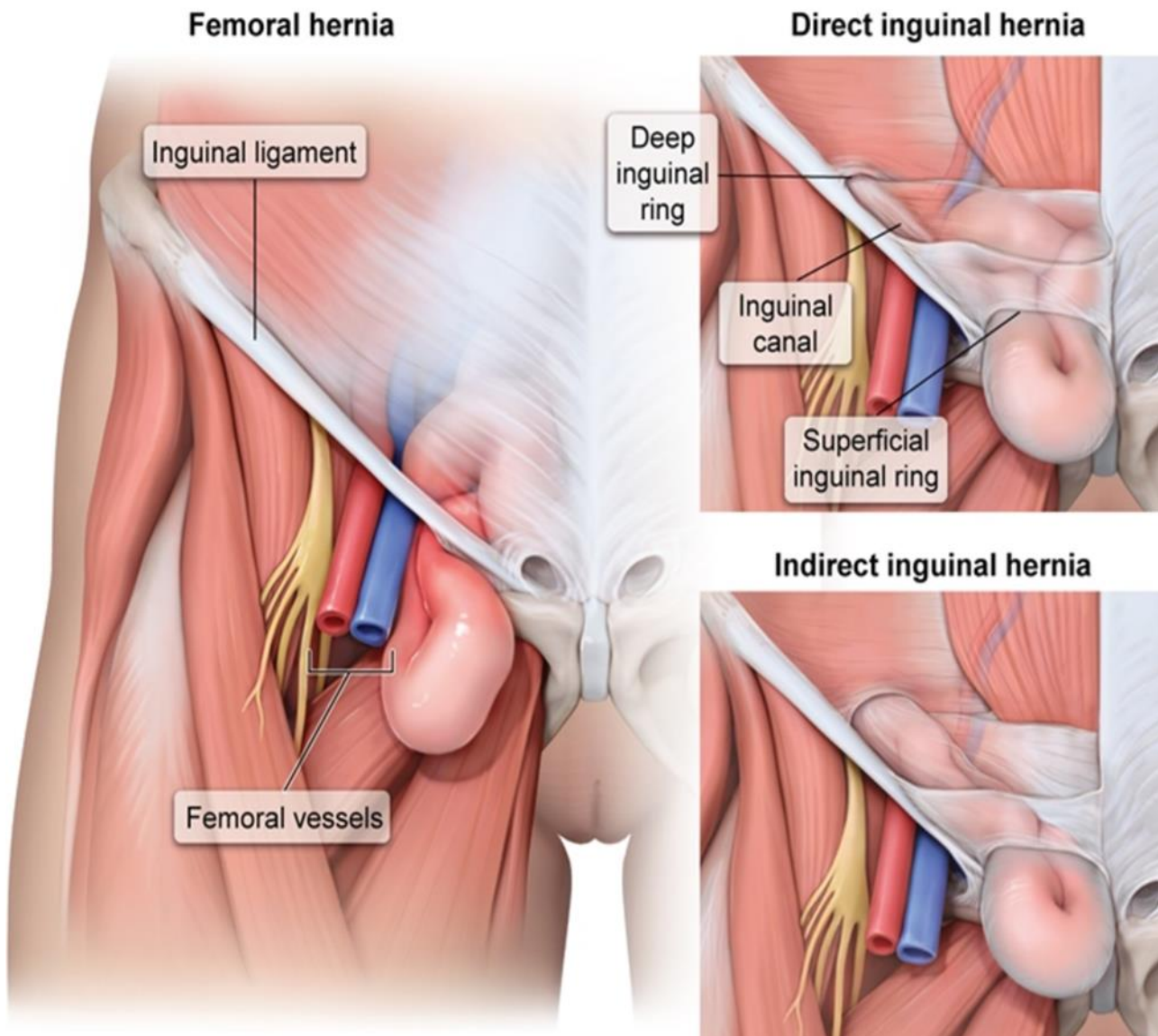
Groin hernias

C. **Femoral hernia:**

- More common in **females**.
- Femoral hernia (hernia located below inguinal ligament) is a displacement of abdominal or pelvic contents through the femoral ring (medial to the femoral artery and lateral to the inguinal ligament) and usually present with a **nontender, nonpulsatile bulge in the groin that grows in size with increased abdominal pressure**.
- Risk factors include chronic cough (chronic obstructive pulmonary disease), constipation, and smoking.
- The mass generally **worsens with increased abdominal pressure** (standing, Valsalva maneuver, coughing) and **improves with decreased abdominal pressure** (lying down).
- When a bowel loop is present within the hernia, it is often tympanitic to percussion.
- **Because femoral hernias pass through a narrow orifice, they are associated with a substantial risk of incarceration** (trapping of abdominal/pelvic contents within the hernia) **and strangulation** (constriction of blood flow with subsequent ischemia/necrosis).
- **Therefore, asymptomatic femoral hernias are generally referred for elective surgical repair to prevent potentially life-threatening complications and subsequent high-risk emergency surgery, which is associated with an increased risk of morbidity (bowel resection) and mortality.**
- In contrast, inguinal hernias (hernia above the inguinal ligament) are associated with a lower risk for incarceration and strangulation because hernia contents pass through a wider orifice. Therefore, **most asymptomatic inguinal hernias can be managed with reassurance and watchful waiting.**

Groin hernias			
Type	Classic presentation	Pathophysiology	Anatomy
Indirect inguinal	Male infants	Patent processus vaginalis	<ul style="list-style-type: none"> • Content protrudes through deep inguinal ring • Travels lateral to inferior epigastric vessels
Direct inguinal	Older men	Weakness of transversalis fascia	<ul style="list-style-type: none"> • Content protrudes through Hesselbach triangle • Travels medial to inferior epigastric vessels
Femoral	Women	Weakness of proximal femoral canal	<ul style="list-style-type: none"> • Content protrudes through femoral ring • Travels inferior to inguinal ligament

Groin hernias



D. Incisional hernia:

- Incisional hernias are **common in patients with obesity** (elevated intraabdominal pressure) and a **prior vertical or midline incision** (increased tissue tension). These 2 factors lead to **gradual fascial breakdown**; presentation **may be delayed for months to years**.
- Incisional hernias can be associated with acute gastrointestinal symptoms if the hernias become very large (mass effect) or bowel incarceration occurs (higher risk with smaller hernia size). However, **most patients have an asymptomatic abdominal mass (protruding abdominal contents) that slowly enlarges as the fascial defect expands**.
- Abdominal mass size **increases with Valsalva** because greater intraabdominal pressure causes more protrusion through the defect.

- **Palpable** fascial edges may be palpable in **nonobese patients**.
- **Management:**
 - Diagnosis is typically **clinical**. Abdominal CT scan may be used to identify unclear anatomy (surgical repair planning) or if there is concern for an acute complication (bowel incarceration).
 - **Conservative** management is indicated in:
 1. Asymptomatic incisional hernias, with a wide neck.
 2. Patients who are at a high anesthetic risk (advanced age, multiple comorbidities).
 - **Surgery** is indicated in **symptomatic/complicated** hernias or those with a **narrow neck**:
 1. Small incisional hernias (< 3 cm defect) → primary repair (Approximation of the edges of the fascial defect with sutures).
 2. Larger incisional hernias → hernioplasty (mesh repair).

Incisional hernia	
Pathogenesis	<ul style="list-style-type: none"> • Breakdown of prior fascial closure
Risk factors	<ul style="list-style-type: none"> • Obesity • Tobacco smoking • Poor wound healing (eg, immunosuppression, malnutrition) • Vertical or midline incision • Surgical site infection
Clinical features	<ul style="list-style-type: none"> • Abdominal mass that enlarges with Valsalva • Palpable fascial edges in nonobese patients • Possible delayed presentation (months-years)
Diagnosis	<ul style="list-style-type: none"> • Clinical • CT scan of abdomen

■ Appendicitis:

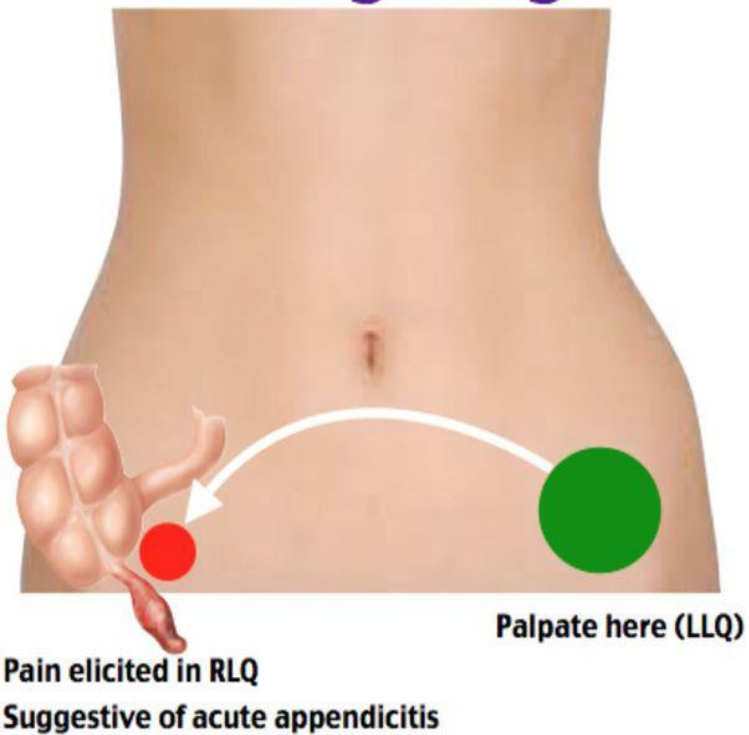
- **Acute inflammation of the appendix**, can be due to obstruction by **fecalith** (in adults) or **lymphoid hyperplasia** (in children).
- Obstruction by foreign bodies, nematodes, and carcinoids may also cause acute appendicitis.
- Retained mucus causes the appendicular wall to distend, which impairs venous outflow. The resulting hypoxia causes **ischemia and associated bacterial invasion**.
- Proximal obstruction of appendiceal lumen produces closed-loop obstruction → ↑ intraluminal pressure → stimulation of visceral afferent nerve fibers at T8-T10 → **initial diffuse periumbilical pain** → inflammation extends to serosa and irritates parietal peritoneum.
- Typical features include **migratory (vague periumbilical visceral pain caused by stretching of the appendiceal wall) abdominal pain, fever, nausea, vomiting, and anorexia**.
- Eventually the peritoneum becomes inflamed and the **pain becomes sharp and localizes to the RLQ**. Patients typically will have pain with palpation at **McBumey point** (1/3 the distance from right anterior superior iliac spine to umbilicus) and **Rovsing sign** (RLQ pain with deep palpation of the LLQ).
- **Acute appendicitis is a clinical diagnosis, and patients with a classic presentation (migratory pain, nausea, vomiting, fever, leukocytosis, McBumey point tenderness and Rovsing sign) should have an immediate appendectomy to prevent appendiceal rupture.**
- However, patients who do not have the typical features of appendicitis or those with other possible causes of RLQ pain (diverticulitis, ileitis, inflammatory bowel disease) **should have appropriate imaging with computed tomography or ultrasonography to confirm the diagnosis**.
- Inflammation and edema of the appendicular wall occur, causing further distention. Necrosis of the wall with **rupture** may follow. In this case, inflammatory fluid and bacterial contents spill into the peritoneal cavity, causing **peritonitis**.
- **Patients who have a delayed presentation with a longer duration of symptoms (>5 days) often have appendiceal rupture with a contained abscess.** These patients will generally have significant fever and leukocytosis, but findings on anterior palpation of the abdomen may be unrevealing.
- In such cases, **maneuvers that assess the deep abdominal spaces** (psoas sign, obturator sign, rectal examination) may be more informative.
- In particular, the psoas sign suggests the presence of an abscess posterior to the appendix adjacent to the psoas muscle. Computed tomography imaging can confirm the diagnosis in these cases.
- **Patients with a contained appendiceal abscess have a very high complication rate from immediate surgery due to the mass of inflamed, infected, and friable debris and adhesions.** If they are otherwise clinically stable, these patients should be managed with **intravenous antibiotics, bowel rest, and**

possibly percutaneous drainage of the abscess. They can return in 6-8 weeks for appendectomy on an elective basis ("interval appendectomy").

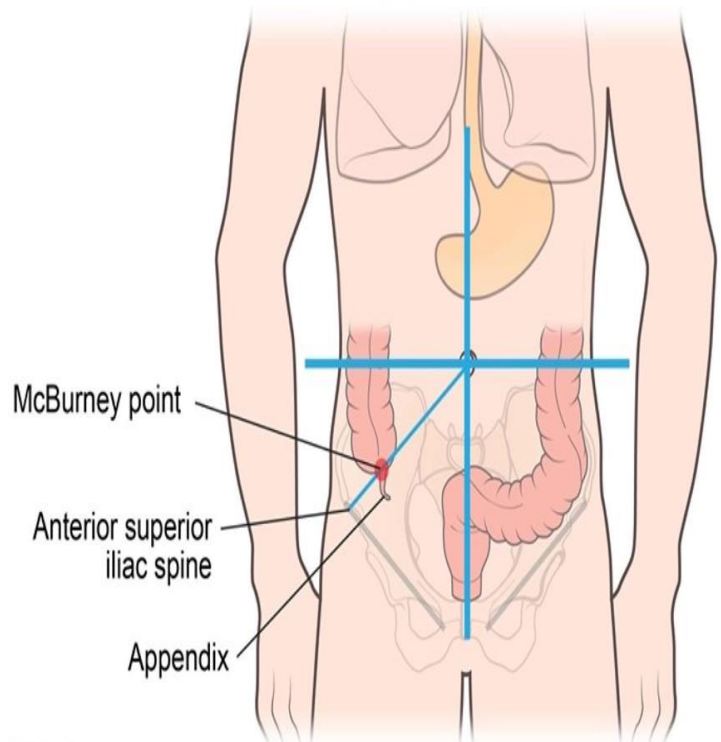
- Differential: diverticulitis (elderly), ectopic pregnancy (use hCG to rule out), pseudoappendicitis.

Examination signs in appendicitis		
Sign	Findings	Significance
Peritoneal signs <ul style="list-style-type: none"> • Rebound tenderness • Involuntary guarding • Abdominal rigidity 	<p>Acute increase in pain after removing the hand from applying pressure</p> <p>Tensing of abdominal wall muscles during palpation of abdomen</p> <p>Persistent tension of abdominal wall muscles</p>	Peritoneal irritation (rupture or impending rupture)
Psoas sign	RLQ pain with extension of right thigh	Abscess adjacent to psoas or retrocecal appendix
Obturator sign	RLQ pain with internal rotation of right thigh	Pelvic appendix or abscess
Rovsing's sign	RLQ pain with LLQ palpation & retropulsion of colonic contents	Acute appendicitis
Rectal tenderness	Right pelvic pain during rectal examination, especially with pressure on right rectal wall	Pelvic appendix or abscess

Rovsing's Sign



McBurney point

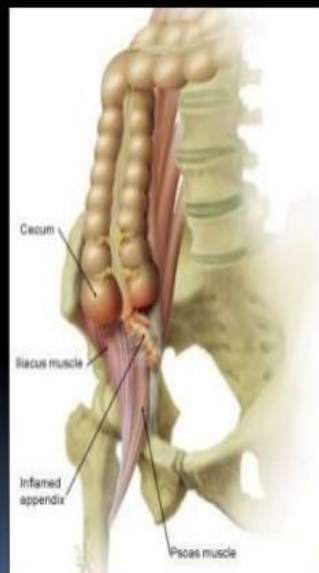


Psoas Sign

3

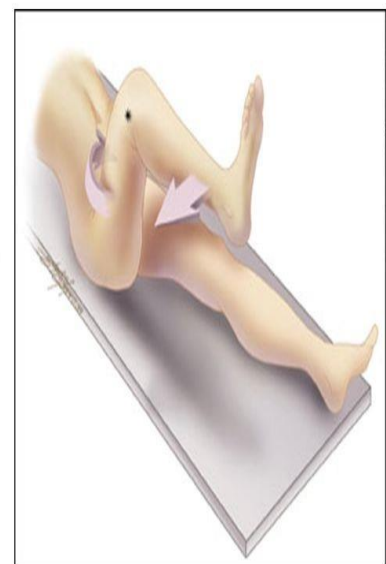
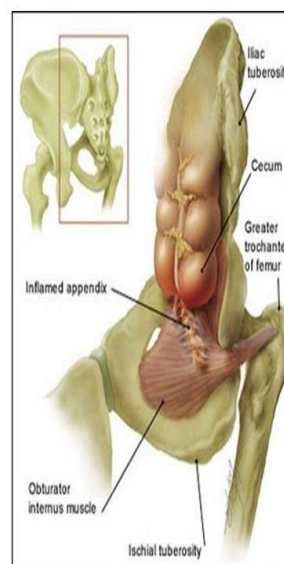


**Pain on hyperextension of right hip
While Patient is lying left lateral with knee extended**



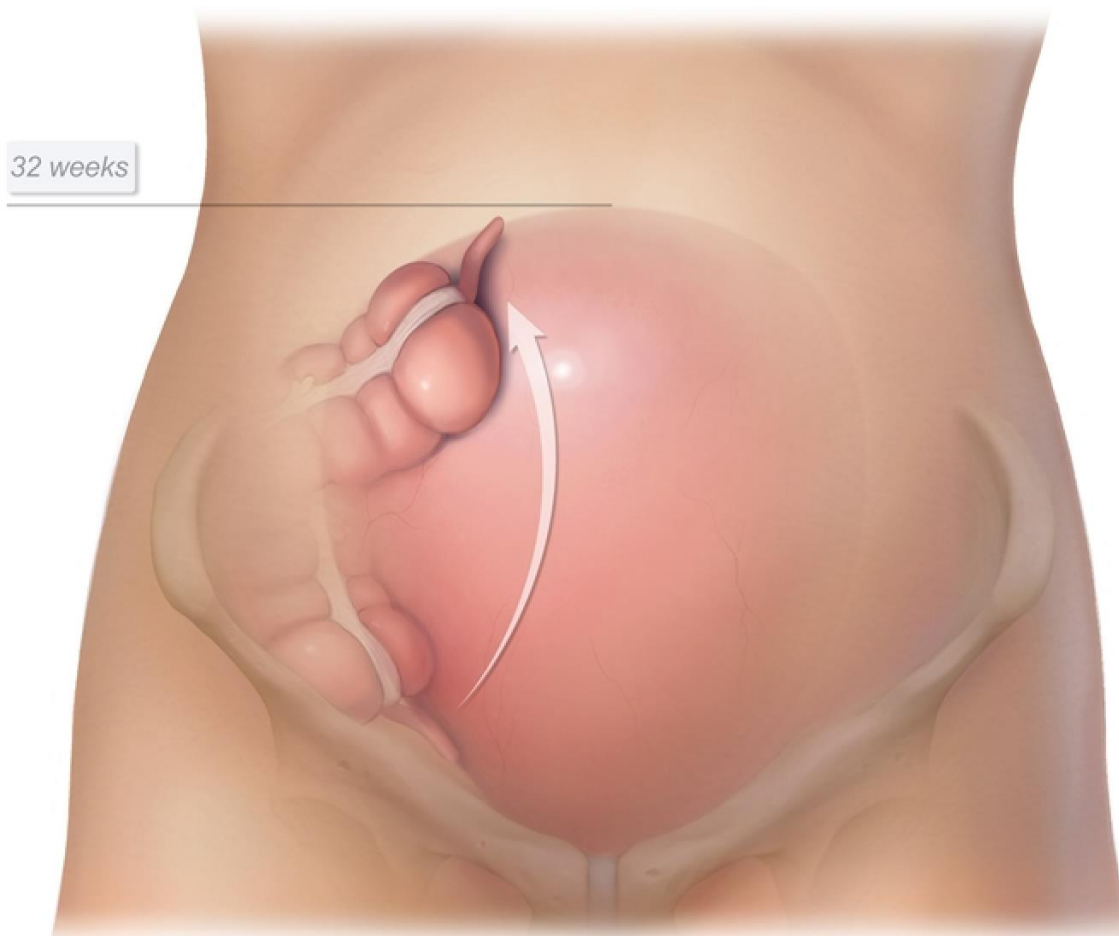
Also in Psoas abscess/ retroperitoneal bleed due to ruptured iliac vessel

Obturator Sign



❖ N.B:

- Acute appendicitis of pregnancy may result in a ruptured appendix if the diagnosis is delayed beyond 24-36 hours.
- Many of the symptoms, including nausea and vomiting, mimic symptoms of pregnancy.
- The most common symptom of appendicitis in pregnancy is right lower-quadrant pain.
- Depending on the gestational age of the pregnancy, the location of pain and tenderness may be higher than expected **due to displacement of the appendix upward by the gravid uterus.**
- The patient may or may not have a fever. An elevated leukocyte count may be present in appendicitis as well as in a normal pregnancy.
- **Ultrasound should be the first diagnostic test used to confirm the diagnosis of appendicitis in pregnancy and can also be helpful to rule out other potential diagnoses.**
- Nonvisualization of the appendix on ultrasound does not exclude the diagnosis of acute appendicitis. **If ultrasound is nondiagnostic, MRI can be performed in pregnant patients to assess further for possible appendicitis.**

Appendix during pregnancy

- Psoas abscess (PA):
 - PA occurs from either hematologic seeding from a distant infection or from direct extension of an intraabdominal infection (diverticulitis, vertebral osteomyelitis).
 - Psoas abscess commonly presents subacutely with fever and lower abdominal or flank pain radiating to the groin.
 - Risk factors include HIV, intravenous drug use, diabetes, and Crohn disease.
 - Psoas Abscess should be considered as part of the evaluation for fever of unknown origin. Deep abdominal palpation is required to elicit tenderness due to the location of the psoas on the posterior abdominal wall.
 - The "psoas sign" abdominal pain with hip extension can often be detected on examination.
 - Laboratory studies commonly show leukocytosis, and elevated Inflammatory markers.
 - CT scans are required to confirm the diagnosis.
 - Drainage is critical, and blood and abscess cultures should be obtained to guide antibiotic therapy.

Psoas abscess	
Clinical presentation	<ul style="list-style-type: none"> • Subacute fever, abdominal/flank pain radiating to groin • Anorexia, weight loss • Abdominal pain with hip extension (psoas sign)
Diagnosis	<ul style="list-style-type: none"> • CT scan of the abdomen & pelvis • Leukocytosis, elevated inflammatory markers • Blood & abscess cultures
Treatment	<ul style="list-style-type: none"> • Drainage • Broad-spectrum antibiotics



- Hemorrhoids:
 - Hemorrhoids are swollen veins in the lowest part of rectum and anus.
 - Hemorrhoids are caused by increased straining or intra-abdominal pressure (due to constipation, pregnancy, or extended periods of sitting).

A. **Internal hemorrhoids:**

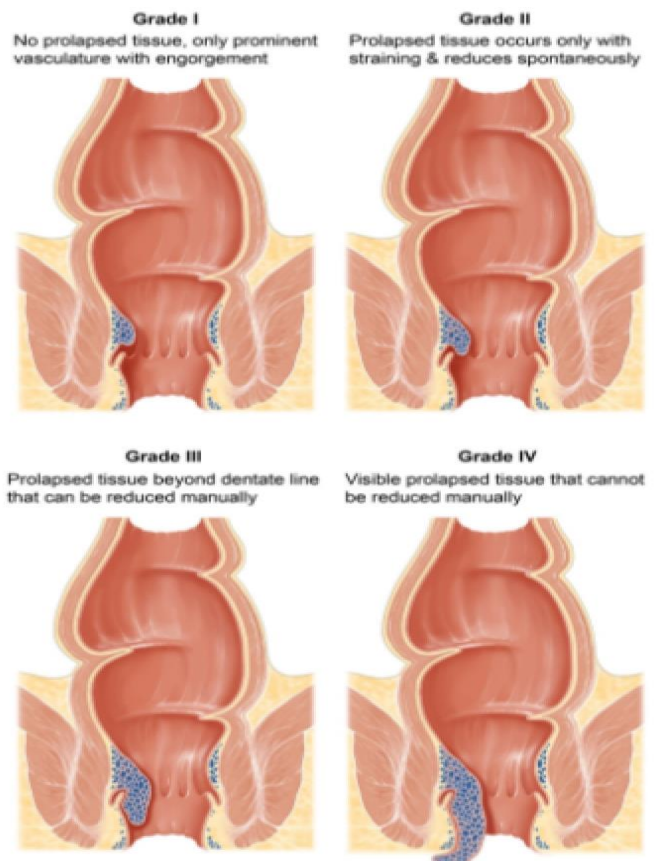
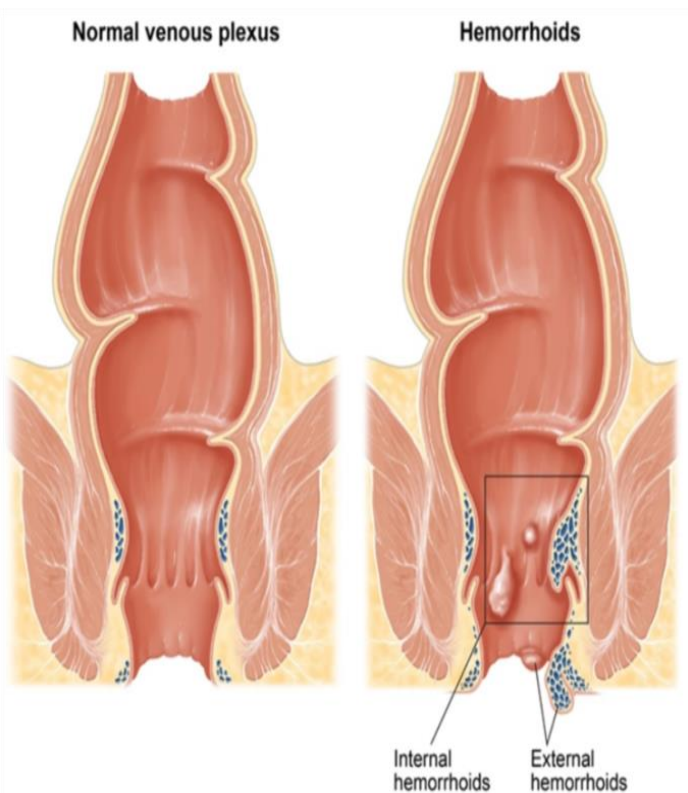
- Originate **above the dentate line** and can prolapse through the anal canal.
- They are often associated with **painless rectal bleeding** (can be treated with rubber band ligation).
- Internal hemorrhoids can become painful and produce itching if they are prolapsed.

B. **External hemorrhoids:**

- Originate **below the dentate line**.
- **Thrombosis can cause the surrounding skin to become inflamed and edematous, resulting in exquisite pain and tenderness.**

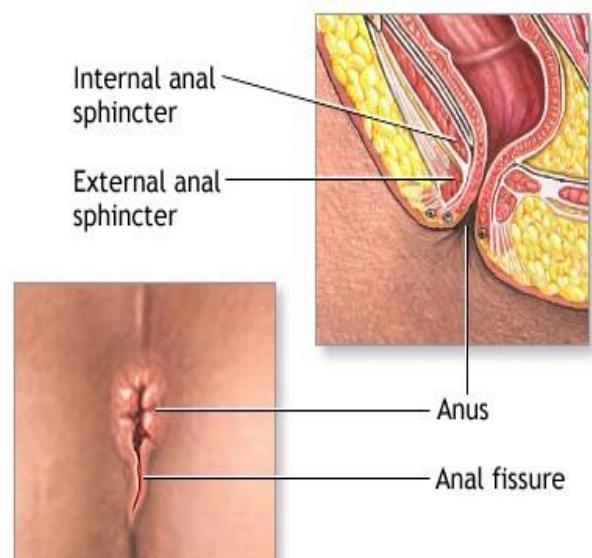
- **Management:**

- Initial management of **uncomplicated** hemorrhoids includes **increased intake of fluid and fiber**, reduction in fat and alcohol intake, and regular exercise.
- Additional measures may include phlebotonics (\uparrow venous tone and \downarrow hemorrhage), topical hydrocortisone, and local anesthetics (benzocaine, lidocaine).
- More aggressive management, including rubber band ligation and surgical hemorrhoidectomy, is generally advised only for patients with refractory symptoms or prolapsed hemorrhoids that cannot be reduced manually (grade IV hemorrhoids)



- Anal fissure:
 - Anal fissure happens to **young women**.
 - Anal fissures are characterized by **longitudinal tears in the anal canal distal to the dentate line and are most common at the posterior midline**.
 - Most fissures are related to **chronic constipation with high anal pressures and passage of hard stools**. They can also be seen with frequent diarrhea or anal sexual intercourse.
 - There is **exquisite pain with defecation and blood streaks covering the stools**.
 - The fear of pain is so intense that patients **avoid bowel movements** (and get constipated) and may even refuse proper physical examination of the area.
 - Examination may need to be **done under anesthesia** (the fissure is usually posterior, in the midline).
 - Initial treatment of anal fissures includes **dietary modification** (high-fiber diet, increased fluid intake), **stool softeners, and sitz baths** to increase blood flow to the injured mucosa.
 - A tight sphincter is believed to cause and perpetuate the problem, thus **therapy is directed at relaxing it**: stool softeners, topical nitroglycerin, local injection of botulinum toxin, steroid suppositories, or lateral internal sphincterotomy.
 - **Topical anesthetics (lidocaine) can enhance comfort. In addition, topical vasodilators (nifedipine, nitroglycerin) can be used to reduce pressure in, and increase blood flow to, the anal sphincter, facilitating healing.**

Anal fissures	
Etiology	<ul style="list-style-type: none"> Local trauma (eg, constipation, prolonged diarrhea, anal sex) Inflammatory bowel disease (eg, Crohn disease) Malignancy
Clinical presentation	<ul style="list-style-type: none"> Pain with bowel movements Bright red blood on toilet paper or stool surface Most common at posterior anal midline Chronic fissure may have skin tag at distal end
Treatment	<ul style="list-style-type: none"> High-fiber diet & adequate fluid intake Stool softeners Sitz baths Topical anesthetics & vasodilators (eg, nifedipine, nitroglycerin)



- **Anorectal abscess:**

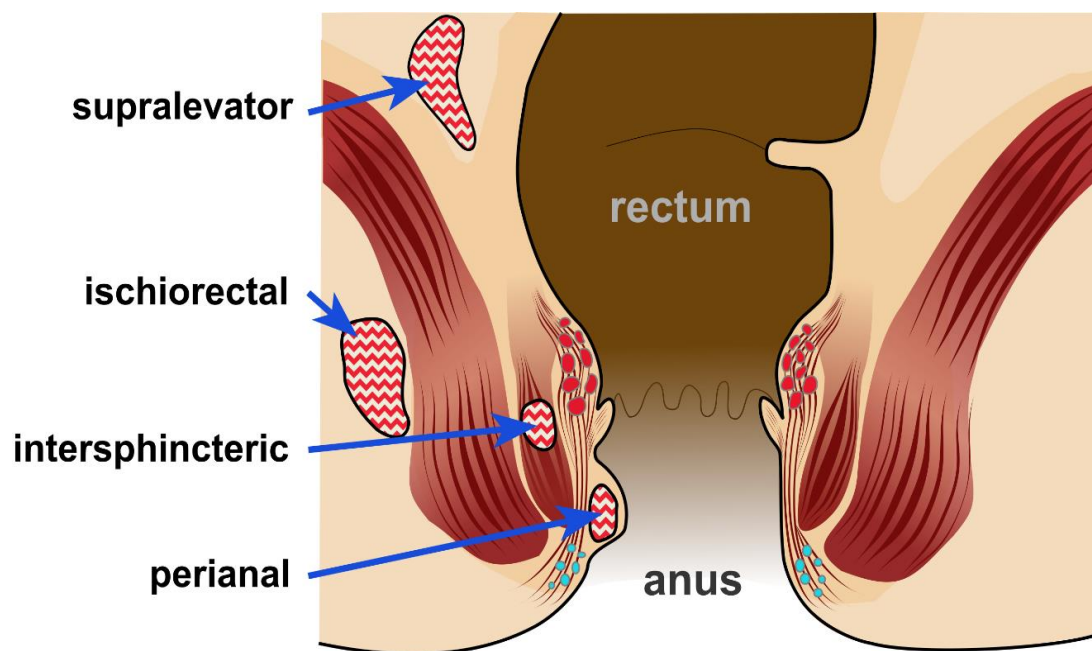
- It is due to occlusion of an anal crypt gland which allows for bacterial infection.
- Abscesses can form relatively acutely following gland obstruction due to the high levels of bacteria in the area.
- Anorectal abscesses are classified according to their anatomic location and the following are the most common types; Perianal abscess, Ischiorectal abscess, Intersphincteric abscess and Supralevator abscess.

- A. **Perianal abscess:**

- The most common type of anorectal abscesses.
- A perianal abscess is due to occlusion of an anal crypt gland which allows for bacterial infection.
- Initially, a perianal abscess may cause pain only with defecation and mild pruritus, but as the infection progresses → the pain becomes constant and can be associated with systemic manifestations such as fever.
- Untreated perianal abscesses often progress to form anorectal fistulae communications between the abscess and perirectal skin or nearby organs (fistula in ano).
- Early recognition followed by Incision and drainage of the perianal abscess is essential to avoid such progression.
- Examination of an anorectal fistula often reveals an external terminus and an indurated tract leading to the rectum. An internal terminus can sometimes be identified on anoscopy or by cautiously passing a probe through the fistula from the external opening.
- Management of an anorectal fistula requires surgical intervention (fistulotomy). Fistulas can often be assessed more fully (exploration with a soft probe) while patients are under anesthesia at the time of surgery to delineate the extent of the fistula.

- B. **Ischiorectal abscess (perirectal abscess):**

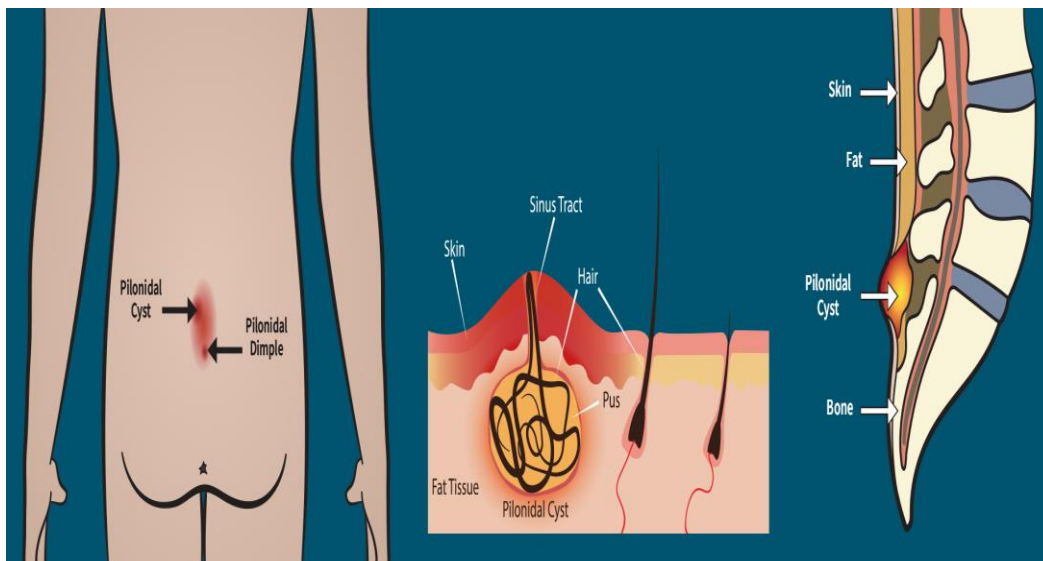
- The patient is febrile, with exquisite perirectal pain that does not let him sit down or have bowel movements.
- Physical exam shows all the classic findings of an abscess (rubor, dolor, calor, and fluctuance) lateral to the anus, between the rectum and the ischial tuberosity.
- Incision and drainage are needed, and cancer should be ruled out by proper examination during the procedure.



Anorectal fistula (fistula in ano)	
Causes	<ul style="list-style-type: none"> • Perianal abscess • Crohn disease • Malignancy, radiation proctitis • Infection (eg, lymphogranuloma venereum)
Clinical manifestations	<ul style="list-style-type: none"> • Perirectal pain, discharge • Inflammatory papule/pustule • Palpable fistula tract
Management	<ul style="list-style-type: none"> • Assess extent of fistula <ul style="list-style-type: none"> ◦ Gentle probe ◦ Imaging (endosonography, fistulogram, MRI) • Surgery (eg, fistulotomy)

C. Pilonidal disease (PD):

- Pilonidal disease most frequently affects males age 15-30 particularly obese individuals, those with sedentary lifestyles or occupations, and those with deep gluteal clefts.
- PD develops when an edematous, infected hair follicle in the intergluteal region becomes occluded. The infection spreads subcutaneously and forms an abscess, which can rupture and create a pilonidal sinus tract.
- As the patient sits or stands, hair and debris are forced into the sinus tract, resulting in recurrent infections and foreign-body reactions.
- The most common presenting manifestations include a painful, fluctuant mass 4-5 cm cephalad to the anus in the intergluteal region with associated mucoid, purulent, or bloody drainage.
- Pain is frequently worsened by activities that stretch the overlying skin (bending down).
- Treatment is drainage of abscesses and collected debris followed by excision of sinus tracts. Despite longer healing times, open closure is preferred due to decreased recurrence rates.



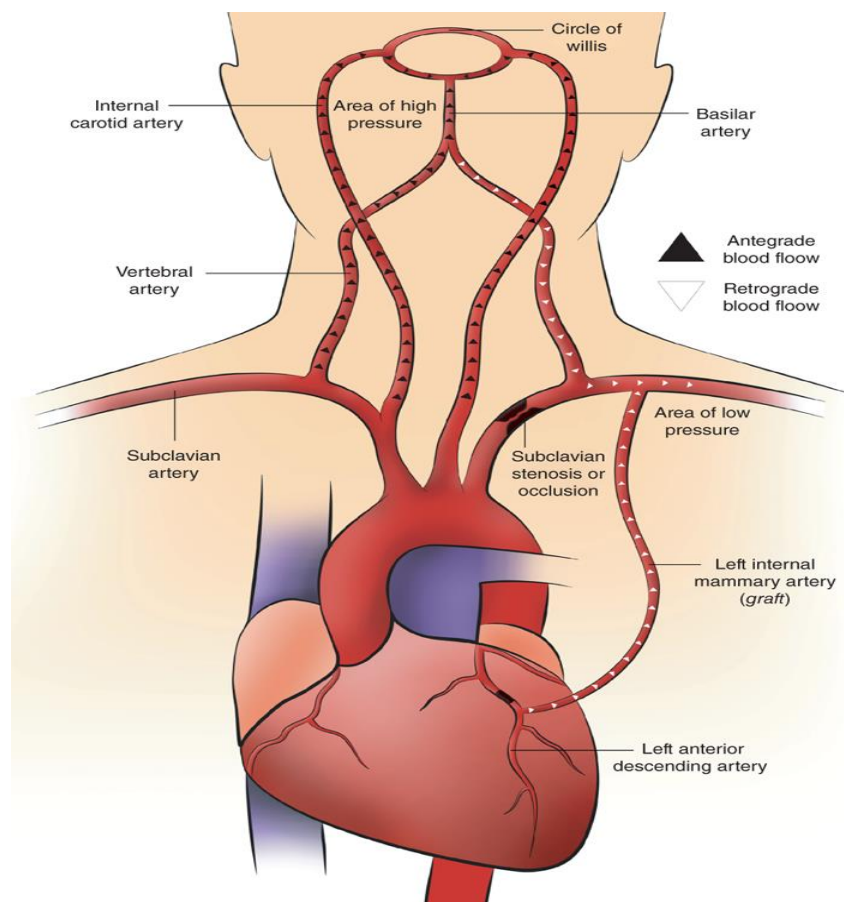
CHAPTER 6

Vascular Surgery

Vascular Surgery

■ Subclavian steal syndrome:

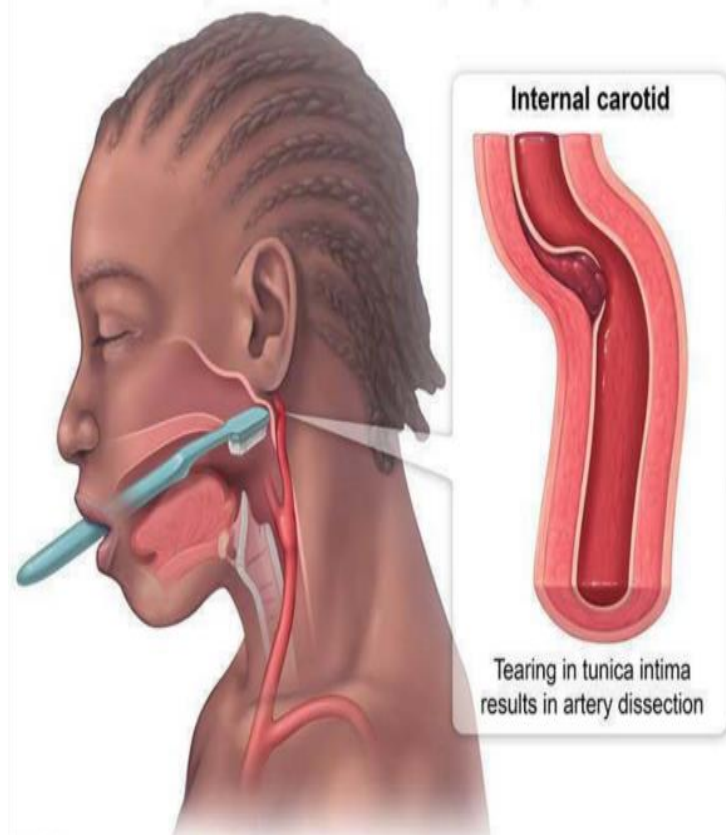
- Subclavian steal syndrome is **rare** but fascinating (medical school professors love it, thus it is likely to appear on exams).
- An arteriosclerotic stenotic plaque at the origin of the subclavian (proximal to the take-off of the vertebral arteries) **allows enough blood supply to reach the arm for normal activity, but does not allow enough to meet higher demands when the arm is exercised.**
- When that happens, **the arm sucks blood away from the brain by reversing the flow in the vertebral.**
- Clinically the patient describes **claudication of the arm** (coldness, tingling, muscle pain) and **posterior neurologic signs** (visual symptoms, equilibrium problems) when the arm is exercised.
- **Vascular symptoms alone would suggest thoracic outlet syndrome, but the combination with neurologic symptoms identifies the subclavian steal.**
- Duplex scanning is diagnostic when it shows **reversal of flow.**
- Bypass surgery is curative.



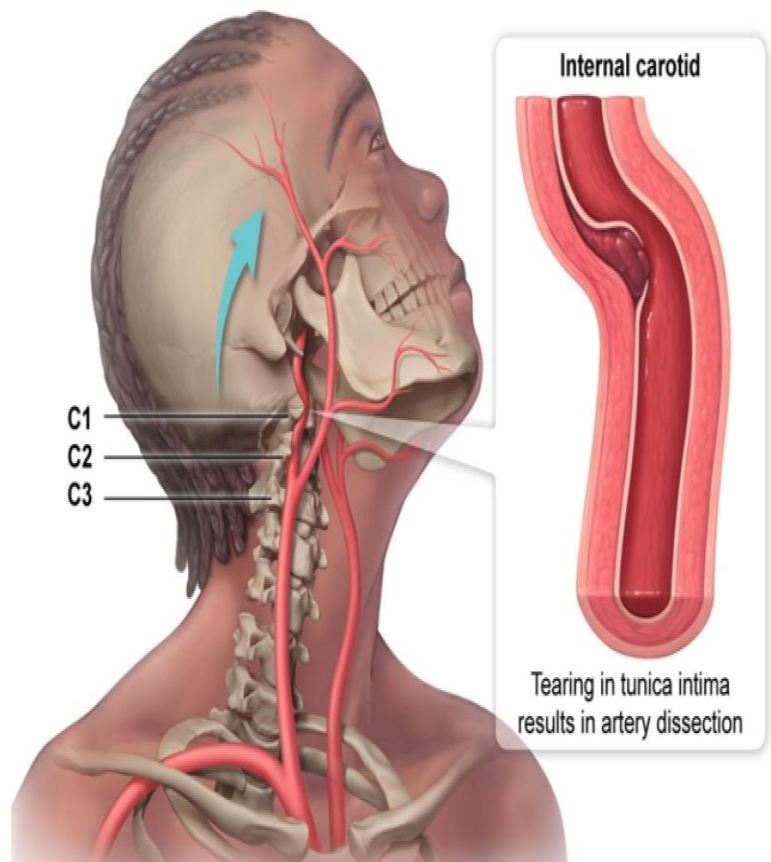
▪ Internal carotid artery injury:

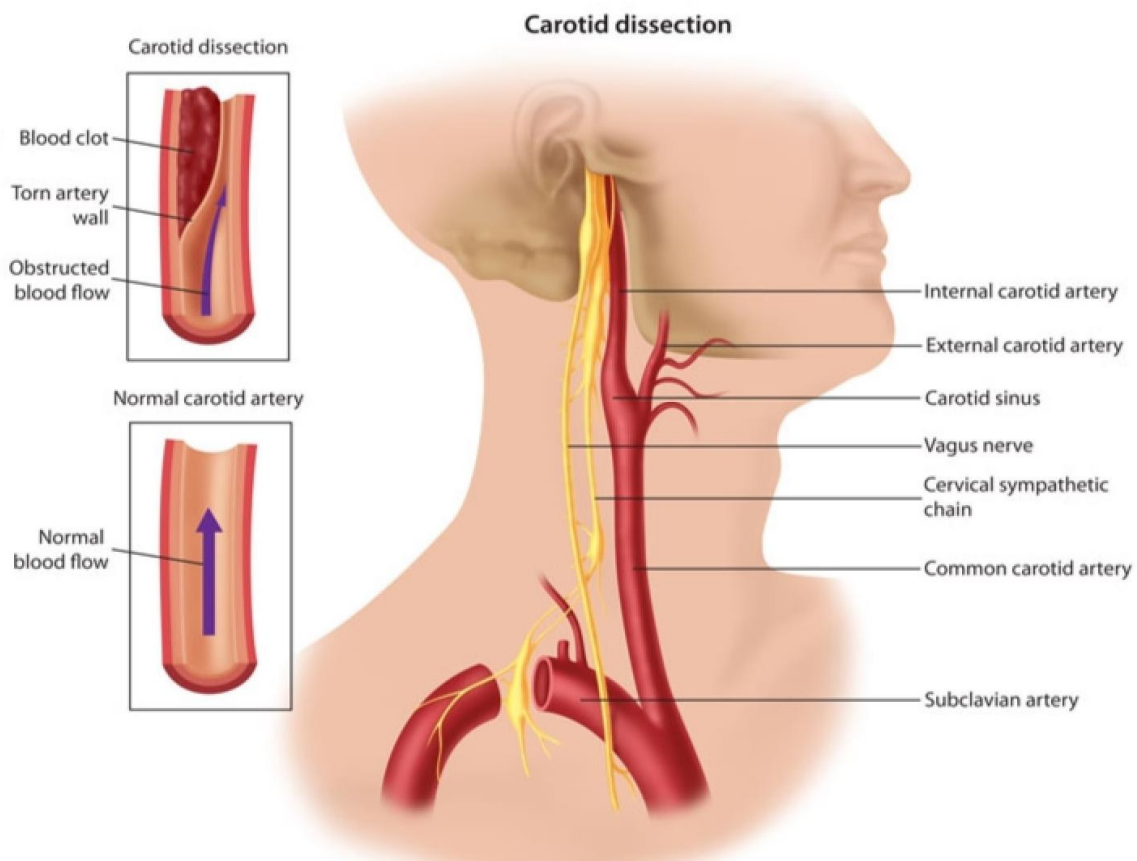
- Symptoms of hemiparesis and aphasia after injury to the posterior pharynx are suspicious for an injury to the cervical internal carotid artery, which is located directly lateral and posterior to the tonsillar pillars.
- Carotid artery dissection is a rare but potentially severe complication of penetrating trauma, seemingly minor oropharyngeal trauma (a fall with object in mouth), or neck strain/manipulation (yoga, sports).
- Intimal injury to the internal carotid artery can result in dissection or thrombus formation, which occurs over hours to days and can extend into the middle and anterior cerebral arteries.
- Patients may develop neck pain, "thunderclap" headache, and symptoms of an ischemic stroke (hemiparesis, facial droop, aphasia).
- The diagnosis of a carotid artery dissection is confirmed with CT or MR angiography.
- Treatment should be initiated after an intracerebral hemorrhage has been ruled out. Heparin therapy followed by oral anticoagulation for 3-6 months and/or Antiplatelet agents for 1 year.

Penetrating or blunt posterior pharyngeal trauma



Neck manipulation (eg, sports, chiropractic manipulation)





Traumatic carotid injuries

Mechanism

- Penetrating trauma
- Fall with object in mouth (eg, toothbrush, pencil)
- Neck manipulation (eg, yoga, sports)

Presentation

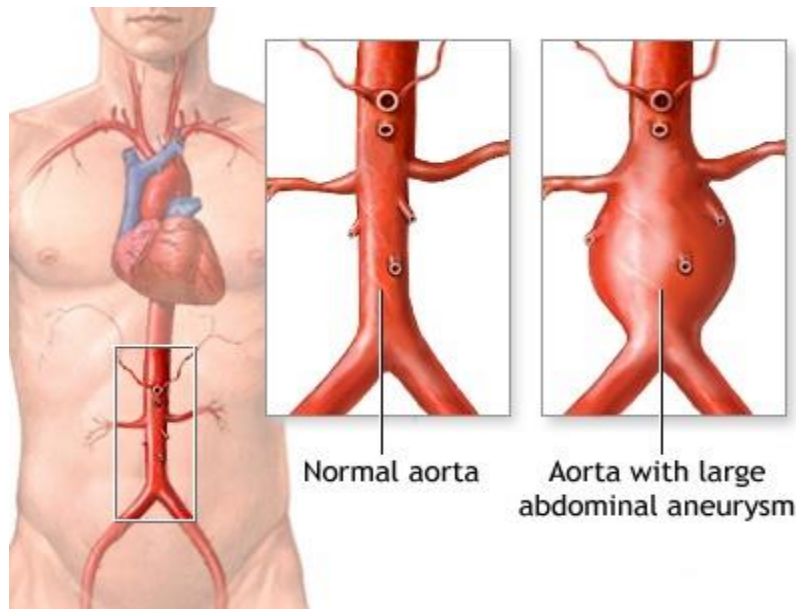
- Gradual-onset hemiplegia
- Aphasia
- Neck pain
- "Thunderclap" headache

Diagnosis

- CT or MR angiography

▪ Abdominal aortic aneurysm (AAA):

- Balloon-like dilation of the aorta.
- Abdominal aortic aneurysm (AAA) is typically **asymptomatic**, found as a **pulsatile abdominal mass on examination** (between the xiphoid and the umbilicus), or found on x-rays, U/S, or CT scans done for another diagnostic purpose, usually in an older man.
- 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**.
- 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**.
- In observational studies, the strongest predictors of abdominal aortic aneurysm expansion and rupture are **large aneurysm diameter, rapid rate of expansion, and current cigarette smoking**.

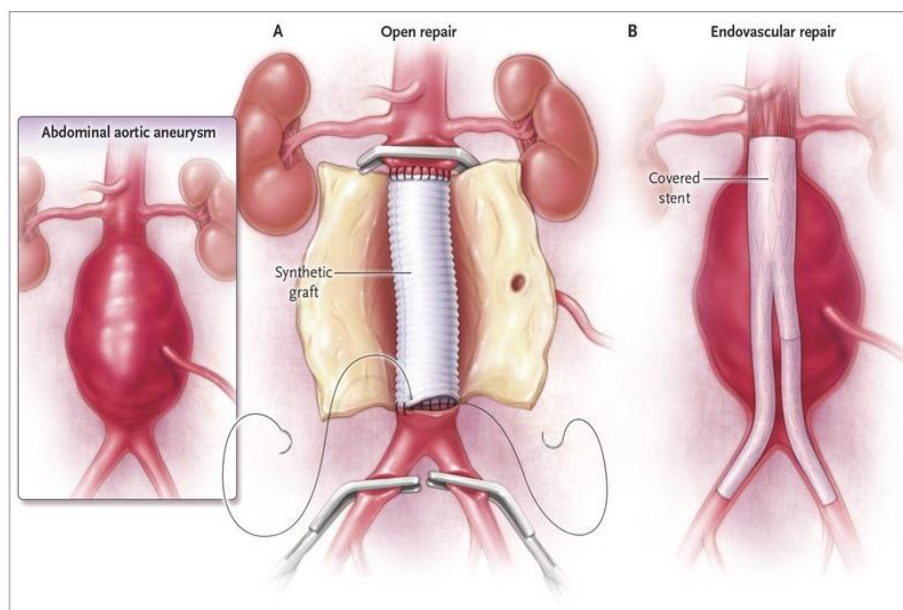


- **The imaging modality of choice for diagnosis and follow-up is abdominal ultrasound**, as it has nearly **100% sensitivity and specificity**, facilitates measurement of aneurysm size, and can show the presence of any associated thrombus. Ultrasound is relatively inexpensive compared to CT or MRI and has the benefit of not requiring contrast administration.
- **Size is the key to management**; if an aneurysm is found by physical exam, U/S or CT scan is needed to provide precise measurements:
 - If aneurysm is **≤4 cm**, it can be safely **observed**; chance of rupture is almost **zero**.
 - If aneurysm is **≥5.5 cm**, patient should have **elective repair** because chance of rupture is **very high**.
 - Aneurysms that grow **1 cm per year or faster** also need elective repair.

Management of AAA

- 3.0–4.0 cm: ultrasound every 2–3 years
- 4.0–5.4 cm: ultrasound or CT every 6–12 months
- ≥ 5.5 cm, asymptomatic: surgical repair

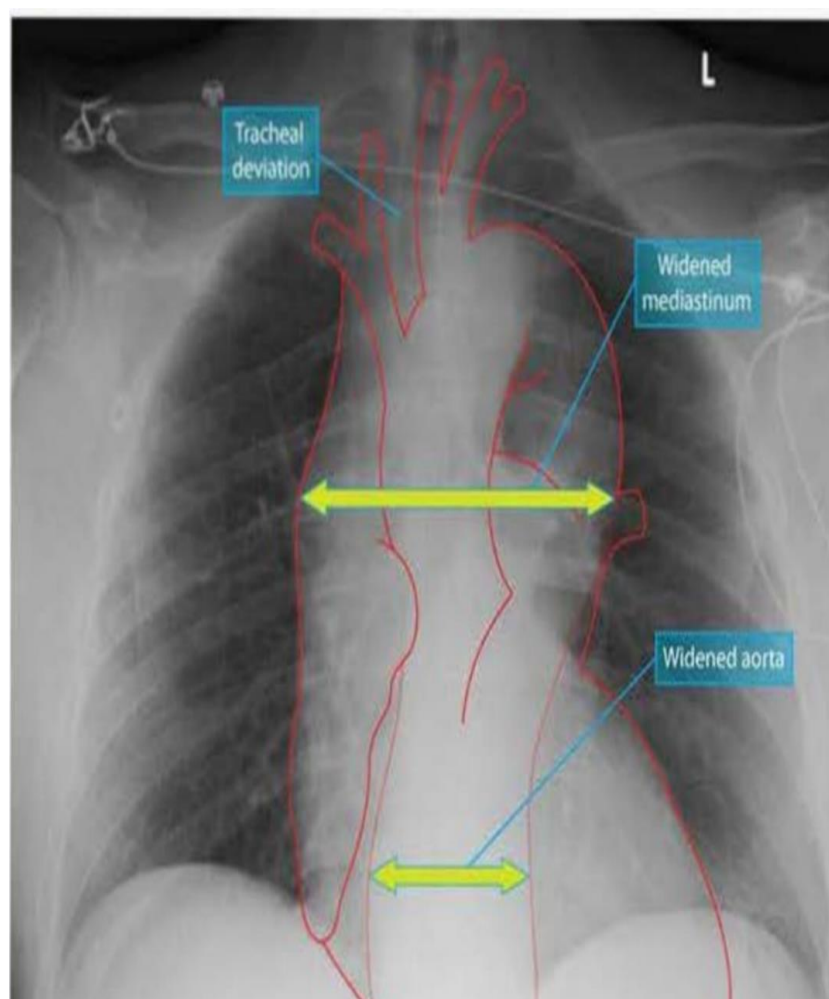
- Once the aneurysm ruptures, only about 50% of the patients survive to come to the hospital. They present with **profound hypotension, abdominal or back pain followed by syncope, and possible pulsatile mass on examination**.
- Patients who present with **acute onset of back pain and profound hypotension** should be evaluated for a presumptive diagnosis of ruptured abdominal aortic aneurysm and emergently taken to the operating room.
- The current indications for operative or endovascular repair include **aneurysm size >5.5 cm, rapid rate of aneurysm expansion (>0.5 cm in 6 months or >1 cm per year), and presence of symptoms (abdominal, back, or flank pain; limb ischemia) regardless of aneurysm size**.
- Most AAAs are now treated with endovascular stents inserted percutaneously. The 10-year outcome has been encouraging.
- Open AAA repair involves an interposition graft within the aneurysm sac and carries 10-15% peri-operative morbidity, with MI, renal failure, and bowel ischemia being the most severe culprits.



❖ N.B:

1. A tender AAA is at risk to rupture, so immediate repair is indicated.
 2. Excruciating back pain in a patient with a large AAA means that the aneurysm is already leaking. Retroperitoneal hematoma is already forming, and blowout into the peritoneal cavity is imminent; emergency surgery is required.
 3. Rupture of an abdominal aortic aneurysm (AAA) is a dramatic and often fatal process. As such, screening appropriate populations for this potentially lethal condition is advisable.
 - The United States Preventive Services Task Force (USPSTF) has identified men aged 65-75 who have smoked cigarettes as having the greatest benefit from screening and recommends a one-time abdominal ultrasound in such patients.
 - Screening and surgical repair of large AAAs (5.5 cm or greater) have been found to decrease AAA-specific mortality in this population.
 - Ultrasonography is the recommended screening modality due to its lower cost, non-invasive nature, and lack of ionizing radiation.
 - There are no recommendations for or against screening men aged 65-75 who have never smoked.
 4. Fluoroquinolones (levofloxacin) increase collagen degradation and are associated with adverse effects, including Achilles tendon rupture, retinal detachment, and aortic aneurysm rupture.
 - When possible, fluoroquinolone use should be avoided in patients with a known aortic aneurysm or substantial risk factors for aortic aneurysm.
- Thoracic aortic aneurysm (TAA):
- A. Ascending aortic aneurysms (60% of cases):
- Typically start anywhere from the aortic valve to the innominate artery.
 - These are most often due to cystic medial necrosis (which usually occurs with aging) or connective tissue disorders (Marfan syndrome, Ehlers-Danlos syndrome).
 - Also associated with tertiary syphilis.
- B. Descending aortic aneurysms (40% of cases):
- Arise distal to the left subclavian artery.
 - Descending aortic aneurysms, are usually due to atherosclerosis; risk factors include hypertension, hypercholesterolemia, and smoking.
- Presentation:
- Thoracic aortic aneurysm (TAA) can be asymptomatic or present with chest, back, flank, or abdominal pain depending on aneurysm location.
 - The most common symptomatic presentation of TAA is pain, which is typically localized to the chest and back.

- Major complication is dilation of the aortic valve root, resulting in **aortic valve regurgitation**.
- Other complications include **compression of mediastinal structures** (airway or esophagus) and thrombosis/embolism.
- If the TAA impinges upon the **esophagus**, it can also cause **dysphagia**. Similarly, compression of the **left recurrent laryngeal nerve or left vagus nerve** results in **hoarseness**, whereas compression of the **phrenic nerve** can cause **hemidiaphragmatic paralysis**.
- **Respiratory manifestations**, including **wheeze, cough, hemoptysis, and dyspnea** may occur due to **tracheobronchial obstruction**.
- **Management:**
 - Chest x-ray may suggest the diagnosis of TAA, demonstrating a **widened mediastinum, enlarged aortic knob, and tracheal deviation**.



- Peripheral artery aneurysm:

- Peripheral artery aneurysm manifests as a **pulsatile mass that can compress adjacent structures (nerves, veins), and can result in thrombosis and ischemia.**
- **Popliteal and femoral artery aneurysms are the most common** peripheral artery aneurysms. They are frequently associated with abdominal aortic aneurysms.
- **Femoral artery aneurysm presents with a pulsatile groin mass below the inguinal ligament; anterior thigh pain is due to the compression of the femoral nerve that runs lateral to the artery.**



- Peripheral Artery Disease:

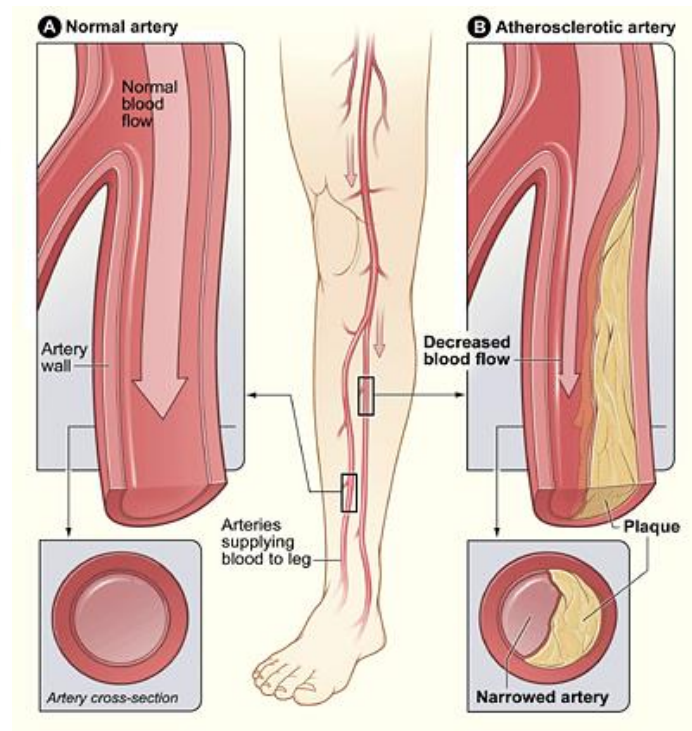
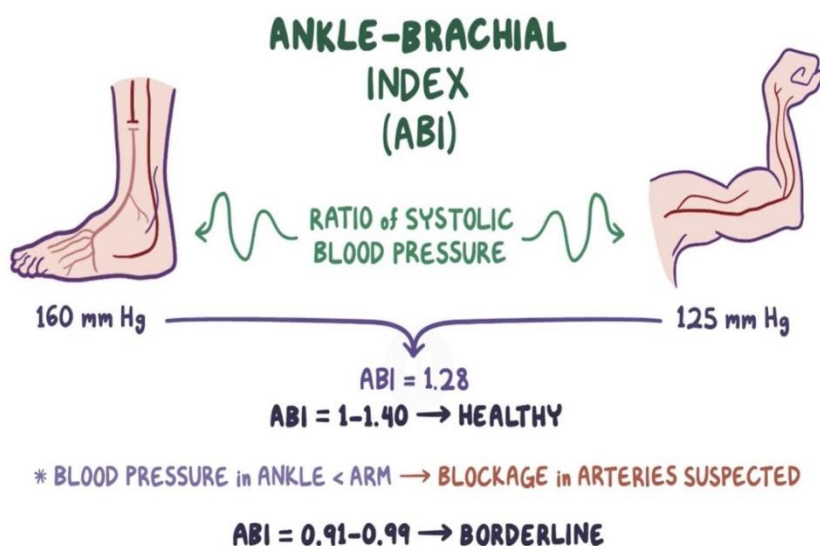
- Peripheral artery disease (PAD) is **the stenosis of peripheral arteries** with the same causative factors as coronary and carotid disease such as:
 - Diabetes mellitus.
 - Hyperlipidemia.
 - Hypertension.
 - Tobacco smoking.
- The key to this question is **leg pain in the calves on exertion, relieved by rest (Intermittent claudication).**
- Intermittent claudication is a muscle pain with exercise that remits with rest. The obstruction of blood flow **results from fixed stenotic atheromatous lesions.** These stenoses **prevent sufficient increase in blood flow to muscles during exercise, resulting in ischemic muscle pain.** This pain is rapidly relieved by rest because the residual blood flow is adequate to meet the metabolic demands of resting, but not exercising muscle.

- Severe disease is associated with loss of:

- Hair follicles.
- Sweat glands.
- Sebaceous glands.
- The skin becomes smooth and shiny.

- Diagnostic Tests:

- The best initial test is the ankle-brachial index (ABI).
- This is the ratio of the blood pressure in the ankles to the brachial arteries. Normally BP is equal between them, or slightly greater in the ankles because of gravity.
- If the difference between them is greater than 10% (ABI less than 0.9), then peripheral artery disease is present.
- The most accurate test is an angiogram, but this is not necessary unless specific revascularization will be done.



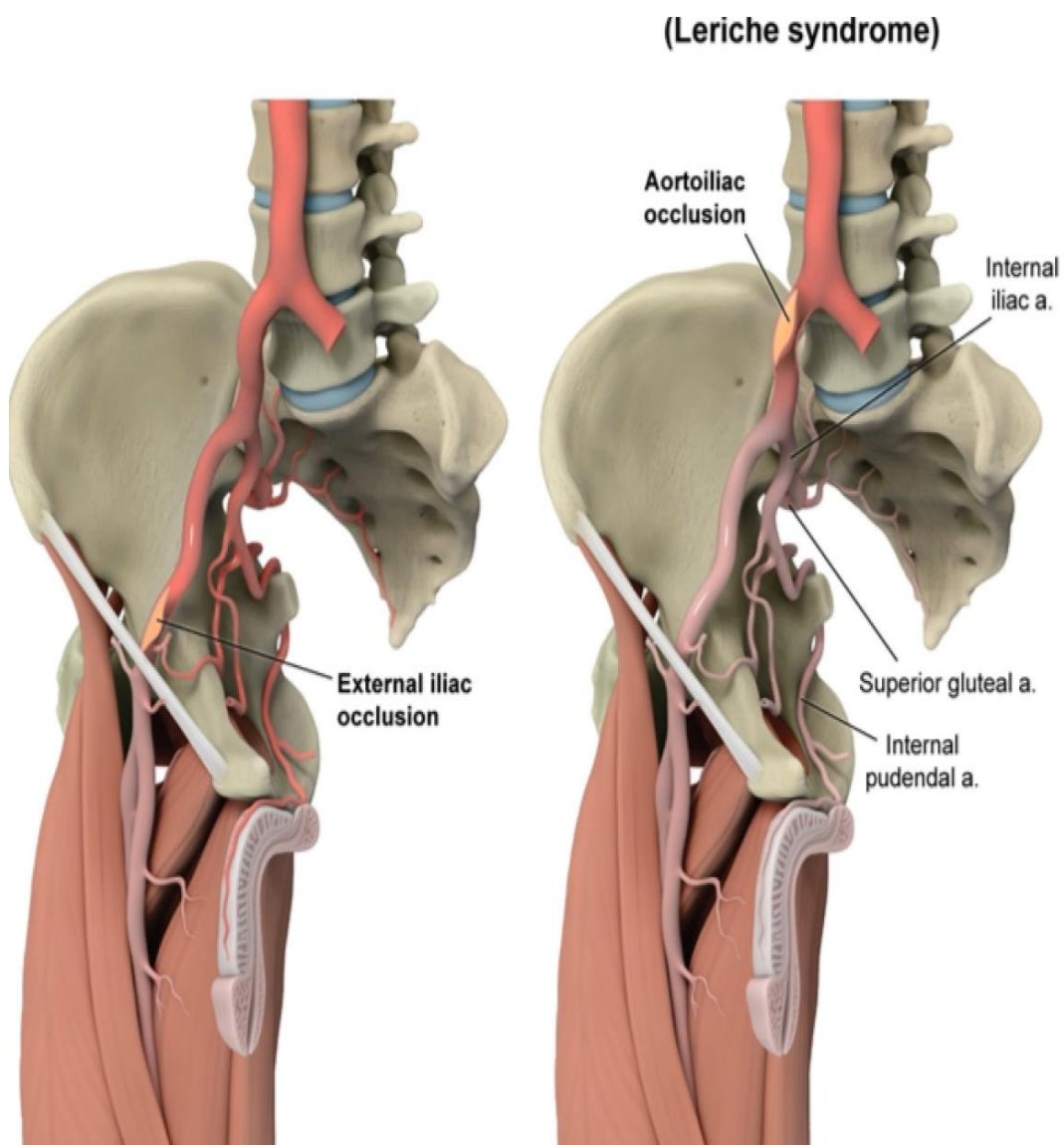
- Treatment:

- A supervised exercise program should be recommended as a part of an initial treatment regimen in all patients with intermittent claudication.
- Pharmacologic therapy with cilostazol and percutaneous or surgical revascularization should be reserved for those with persistent symptoms despite adequate supervised exercise therapy.

- The best initial medical therapy is:

- Antiplatelet medication: Aspirin or clopidogrel.
- Cilostazol.
- Stopping smoking.

- The single most effective medication is **cilostazol**. Surgery is done to bypass stenosis if these medical therapies are not effective.
- ❖ N.B:
 - Leriche syndrome is arterial occlusion at the bifurcation of the aorta into the common iliac arteries (**aortoiliac occlusion**) and is characterized by the triad of:
 - Bilateral hip, thigh, and buttock claudication.
 - Absent or diminished femoral pulses: from the groin distally, often with symmetric atrophy of the bilateral lower extremities due to chronic ischemia.
 - **Impotence: almost always present in men with this condition; in the absence of impotence, an alternate diagnosis should be sought.**

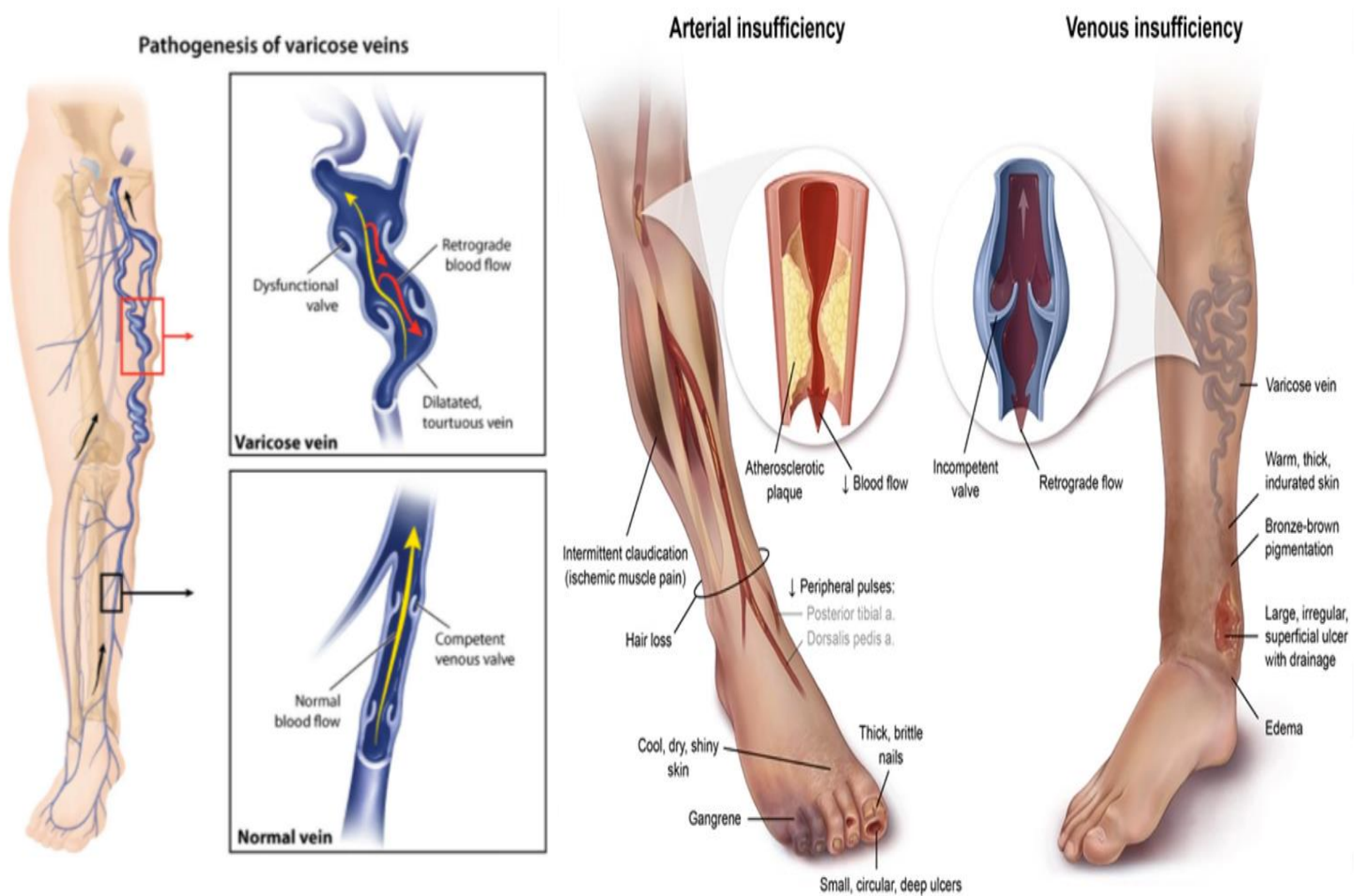


- Acute limb ischemia:
 - Acute arterial occlusion can result from 1 of 3 major causes:
 - Embolus from a cardiac or arterial source.
 - Arterial thrombosis.
 - Iatrogenic or direct blunt trauma to the artery.
 - The sudden development of symptoms in a previously asymptomatic patient is most consistent with an embolic occlusion.
 - The majority of arterial emboli originate from the heart, and lower extremities are affected much more commonly than upper extremities.
 - Potential cardiac sources of emboli include left atrial thrombus due to atrial fibrillation, left ventricular thrombus following an anterior myocardial infarction, infective endocarditis (septic emboli), and thrombus from prosthetic valves.
 - The patient suddenly develops the 6 Ps:
 - Pain.
 - Pallor.
 - Paresthesias.
 - Pulselessness.
 - Poikilothermia (cool extremity).
 - Paralysis (due to nerve ischemia).
- Urgent evaluation and treatment should be completed within 6 hours.
- Doppler studies will locate the point of obstruction.
- Patients with suspected acute arterial occlusion leading to an immediately threatened limb (sensory loss, rest pain, muscle weakness) should be immediately started on anticoagulation while further diagnostic procedures are performed. Heparin prevents further thrombus propagation and thrombosis in the distal arterial and venous circulation.
- Embolectomy with Fogarty catheters is effective for complete obstructions, and fasciotomy should be added if several hours have passed before revascularization to prevent compartment syndrome from reperfusion edema.

- Chronic venous insufficiency (CVI):

- Chronic venous insufficiency is a common cause of lower extremity edema that may be accompanied by **varicose veins, skin discoloration, and medial skin ulceration**.
- Chronic venous insufficiency is most commonly caused by **incompetence of venous valves leading to venous hypertension in the deep venous system of the legs**.
- Risk factors for CVI include **advancing age, obesity, family history, pregnancy, sedentary lifestyle, previous LE trauma, and previous LE venous thrombosis**.
- **Pathogenesis:**
 - Venous blood normally flows from superficial veins through perforating veins into the deep veins of the extremities.
 - Blood from the deep veins then travels upward and eventually drains into the inferior vena cava.
 - Valves located in the superficial, perforating, and deep veins **prevent backward flow from deep to superficial veins**.
 - Chronically elevated intraluminal pressure can lead to **dilation of the veins (varicose veins) and incompetence of the valves**.
 - This allows **retrograde flow** into superficial veins and results in a further increase in venous pressure.
- **Presentation:**
 - In relatively severe cases, redirection of blood from the deep venous system to the superficial venous system may lead to other physical examination findings, including **abnormal venous dilation (varicose veins), skin discoloration, lipodermatosclerosis, or skin ulceration (characteristically on the medial aspect of the lower leg)**.
 - **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**.
 - **Stasis dermatitis most classically involves the medial leg below the knee and above the medial malleolus**.
 - Pitting edema is the most common physical examination finding. Patients may present with leg discomfort, pain, or swelling that is **typically worse in the evening or following prolonged standing and improves after walking or leg elevation**.

- Inflammation of venules and capillaries as well as **fibrin deposition and platelet aggregation** cause **microvascular disease** and ultimately **ulcerations** will occur.
- **Treatment:**
- Initial treatment includes conservative measures with **leg elevation**, exercise, and compression therapy with compression stockings.



- Arteriovenous fistula (AVF):
- Symptomatic AVF **can be congenital or acquired** and creates an abnormal connection between the arterial and venous systems that **bypasses the capillary beds**.
- **Shunting of a large amount of blood through the fistula decreases systemic vascular resistance, increases cardiac preload, and increases cardiac output.**
- In patients with AVF and significant AV shunting, **there is a compensatory increase in the heart rate and stroke volume to meet the oxygen requirements of the peripheral tissues.**
- Clinical signs include **widened pulse pressure, strong peripheral arterial pulsation (brisk carotid upstroke), systolic flow murmur, tachycardia, and usually flushed extremities.** The left ventricle hypertrophies, and the point of maximal impulse is displaced to the left. An ECG usually shows **left ventricular hypertrophy.**
- Normal hearts are usually capable of increasing the stroke volume and cardiac output without significant problems. However, **cardiac function can occasionally decompensate over a period of time and result in cardiac failure.** Such patients are considered to have heart failure (despite their higher cardiac output) because the circulation is unable to meet the oxygen demand of the peripheral tissues.
- **Doppler ultrasonography** is the preferred test to diagnose an AVF in the extremity, and **surgical therapy** is indicated for a large AVF.
- ❖ N.B:
 - The femoral artery is the most common vascular access point in patients undergoing cardiac catheterization.
 - The femoral vein can be inadvertently punctured during needle insertion attempts to obtain femoral arterial access.
 - Following the procedure, inadequate hemostasis may allow persistent bleeding from the arterial puncture site to track in to the venous puncture site, creating an AVF.
 - Most patients are initially asymptomatic but gradually develop mild swelling and localized tenderness. As arterial pressure exceeds venous pressure throughout the cardiac cycle, a continuous bruit with a palpable thrill is typically present. In addition, distal pulses may be diminished in the affected extremity.
 - An untreated AVF can progressively enlarge and lead to **limb edema** (due to venous hypertension), **limb ischemia** (due to redirection of arterial blood flow), and **high-output heart failure** (due to blood returning to the right atrium without passing through peripheral resistance).
 - The diagnosis is typically confirmed by **duplex ultrasound.**
 - Management of **small** AVFs involves **observation** (sometimes resulting in spontaneous closure) or ultrasound-guided compression.
 - **Large** AVFs typically require **surgical repair.**

Local vascular complications of cardiac catheterization

Hematoma



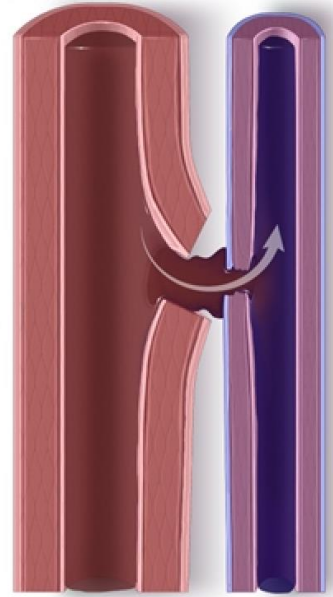
- \pm mass
- No bruit

Pseudoaneurysm

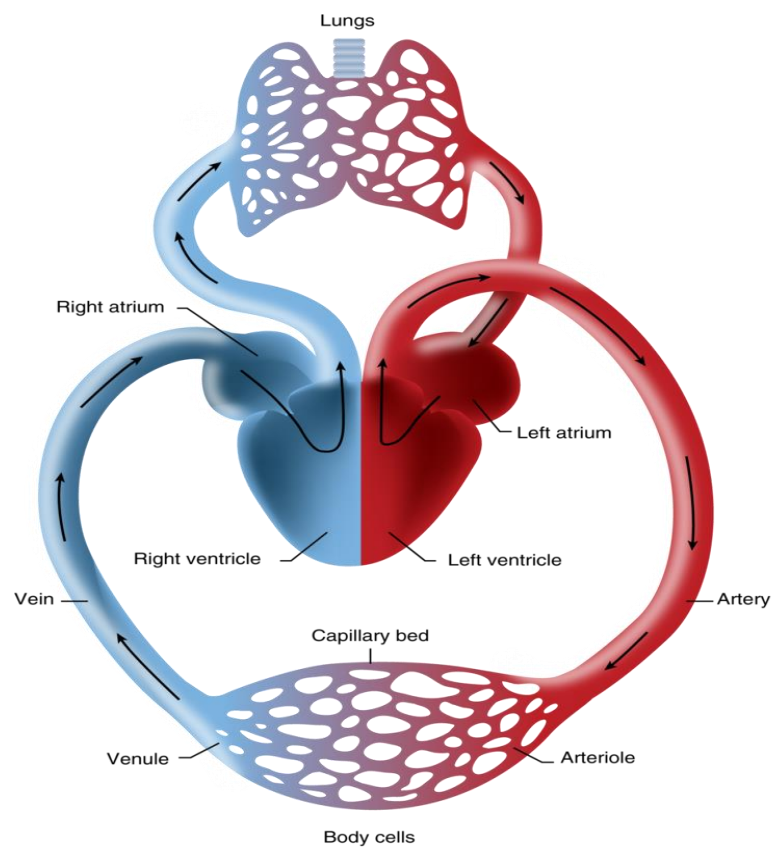


- Bulging, **pulsatile** mass
- **Systolic** bruit

Arteriovenous fistula



- No mass
- **Continuous** bruit



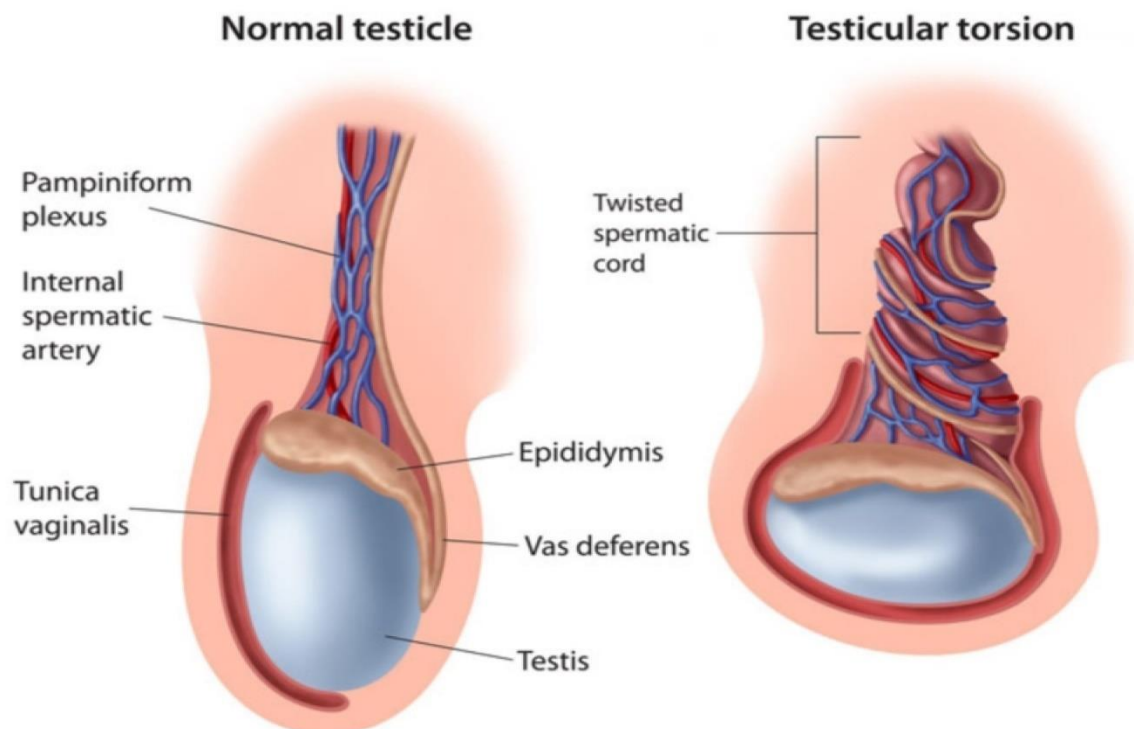
CHAPTER 7

Urology

Urology

▪ Testicular torsion:

- **Twisting of the spermatic cord**; thin-walled veins become obstructed leading to congestion and infarction.
- Usually due to congenital failure of testes to attach to the inner lining of the scrotum (via the processus vaginalis).
- Physical examination classically reveals a **profoundly tender, enlarged, high-riding testicle associated with marked scrotal swelling and erythema**. The cremasteric reflex (elevation of the ipsilateral testicle when the inner thigh is stroked) is **absent**.
- This is one of the few **urologic emergencies**, and time wasted doing any tests is tantamount to malpractice.
- The diagnosis is often made **clinically**. However, Doppler ultrasound of the scrotum can be used for confirmation or in case of equivocal findings. It may **demonstrate twisting of the spermatic cord or reduced blood flow**, and a reactive hydrocele may be present.
- **Immediate surgical intervention is indicated**. After the testis is untwisted, an orchiopexy is done to prevent recurrence; simultaneous contralateral orchiopexy is also indicated.

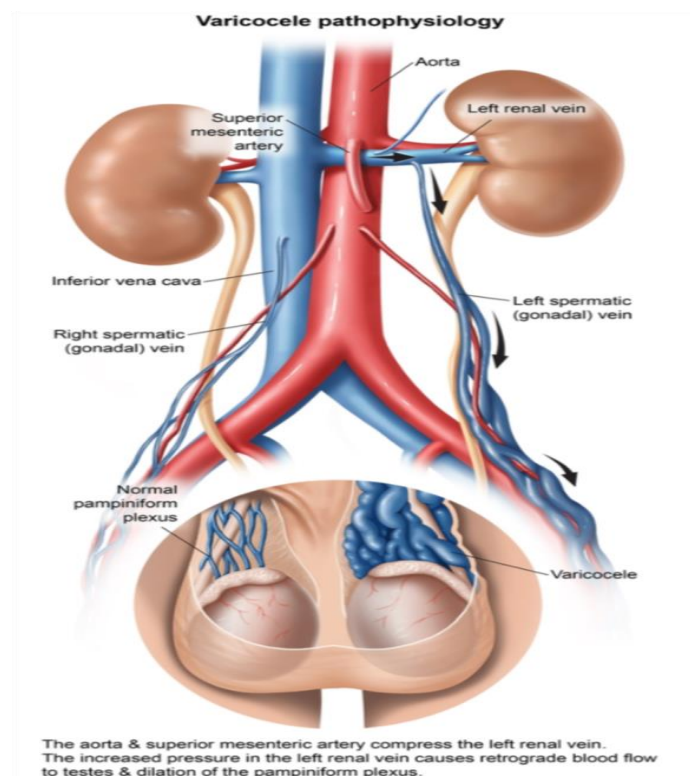
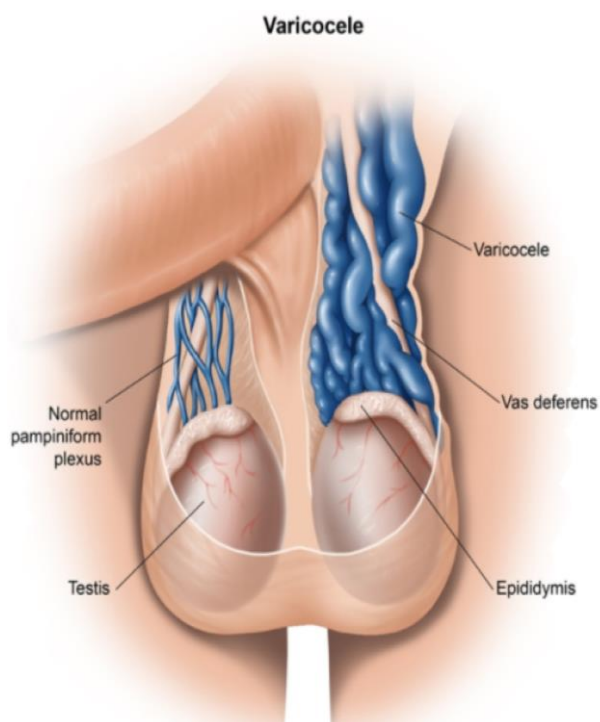


- Varicocele:

- A varicocele is a tortuous dilation of the pampiniform plexus of veins surrounding the spermatic cord and testis.
- It occurs in nearly 20% of postpubertal males and presents as a soft, irregular mass ("bag of worms") that increases in size with standing and Valsalva.
- Varicoceles can cause elevated scrotal temperatures, increasing the risk for infertility and testicular atrophy.
- Varicoceles are more common on the left side. The left spermatic (gonadal) vein drains to the left renal vein, which then passes between the superior mesenteric artery (SMA) and the aorta.
- The left renal vein is vulnerable to compression beneath the SMA ("nutcracker effect"), leading to increased pressure in the spermatic vein, incompetence of the valves, retrograde blood flow, and venous dilation.
- In contrast, the right spermatic vein drains directly into the inferior vena cava; right-sided varicoceles are relatively rare and can be a sign of malignant compression (renal cell carcinoma) or thrombosis.
- Management:
 - The diagnosis of varicocele can be confirmed on ultrasound, which shows dilation of the pampiniform plexus and retrograde venous flow.
 - Initial interventions for scrotal discomfort due to a varicocele include scrotal support and simple analgesics (nonsteroidal anti-inflammatory drugs).
 - Varicoceles are associated with increased risk for infertility and testicular atrophy, which may be due to slightly increased scrotal temperature.
 - For older men who do not have concerns about fertility, no intervention may be needed.
 - However, younger men should be followed for signs of testicular atrophy or changes in semen analysis; abnormal findings generally warrant surgical intervention. When intervention is indicated, surgical venous ligation can improve fertility.

Varicocele	
Clinical presentation	<ul style="list-style-type: none"> • Soft scrotal mass ("bag of worms") <ul style="list-style-type: none"> ◦ ↓ In supine position ◦ ↑ With standing/Valsalva maneuvers • Subfertility • Testicular atrophy
Ultrasound findings	<ul style="list-style-type: none"> • Retrograde venous flow • Tortuous, anechoic tubules adjacent to testis • Dilation of pampiniform plexus veins
Treatment	<ul style="list-style-type: none"> • Gonadal vein ligation (boys & young men with testicular atrophy) • Scrotal support & NSAIDs (older men who do not desire additional children)

NSAIDs = nonsteroidal anti-inflammatory drugs.



▪ **Epididymitis:**

- Epididymitis is often suspected when physical examination reveals **unilateral posterior testicular swelling and tenderness that improves with elevation of the testes** (Prehn sign).
- Most cases arise **when pathogens from the urethra travel in a retrograde fashion through the ejaculatory duct to the ductus deferens and epididymis**. The likely underlying pathogen differs **based on the age of the patient**:
 - Those age >35 most commonly develop epididymitis from bacteriuria related to bladder outlet obstruction (benign prostate hyperplasia). **Ascending coliform bacteria such as Escherichia coli are the most likely pathogens**, and urinary tract inflammatory symptoms (dysuria, frequency) are common.
 - Patients age <35 usually develop epididymitis due to **sexually transmitted infections with Chlamydia trachomatis or Neisseria gonorrhoeae**. These organisms often cause **asymptomatic urethritis**, so urinary symptoms occur less frequently.
- **Management:**
 - Confirmation requires **urinalysis/urine culture and nucleic acid amplification testing for N gonorrhoeae and C trachomatis**.
 - Treatment with **ceftriaxone plus doxycycline** (for N gonorrhoeae or C trachomatis) or **levofloxacin** (for enteric pathogens) is generally curative.
 - Nonsteroidal anti-inflammatory drugs and testicular elevation may provide **symptomatic** relief but must be accompanied by antibiotic therapy.

Acute epididymitis	
Etiology	<ul style="list-style-type: none"> • Age <35: sexually transmitted (chlamydia, gonorrhea) • Age >35: bladder outlet obstruction (coliform bacteria)
Manifestations	<ul style="list-style-type: none"> • Unilateral, posterior testicular pain • Epididymal edema • Pain improved with testicular elevation • Dysuria, frequency (with coliform infection)
Diagnosis	<ul style="list-style-type: none"> • NAAT for chlamydia & gonorrhea • Urinalysis/culture

NAAT = nucleic acid amplification test.

▪ **Hematuria:**

- Hematuria can be due to glomerular or non-glomerular causes:

A. **Glomerular hematuria:**

- Glomerular hematuria usually causes **microscopic hematuria**, though gross hematuria may be present in some cases.
- It is usually due to **glomerulonephritis (GN) or basement membrane disease**.
- Symptoms are usually absent or nonspecific (low back pain), and patients may present after developing features of **nephritic syndrome** (hematuria, hypertension, oliguria, elevated creatinine).
- Urinalysis usually shows **proteinuria with dysmorphic RBCs or RBC casts**.

B. **Non-glomerular hematuria:**

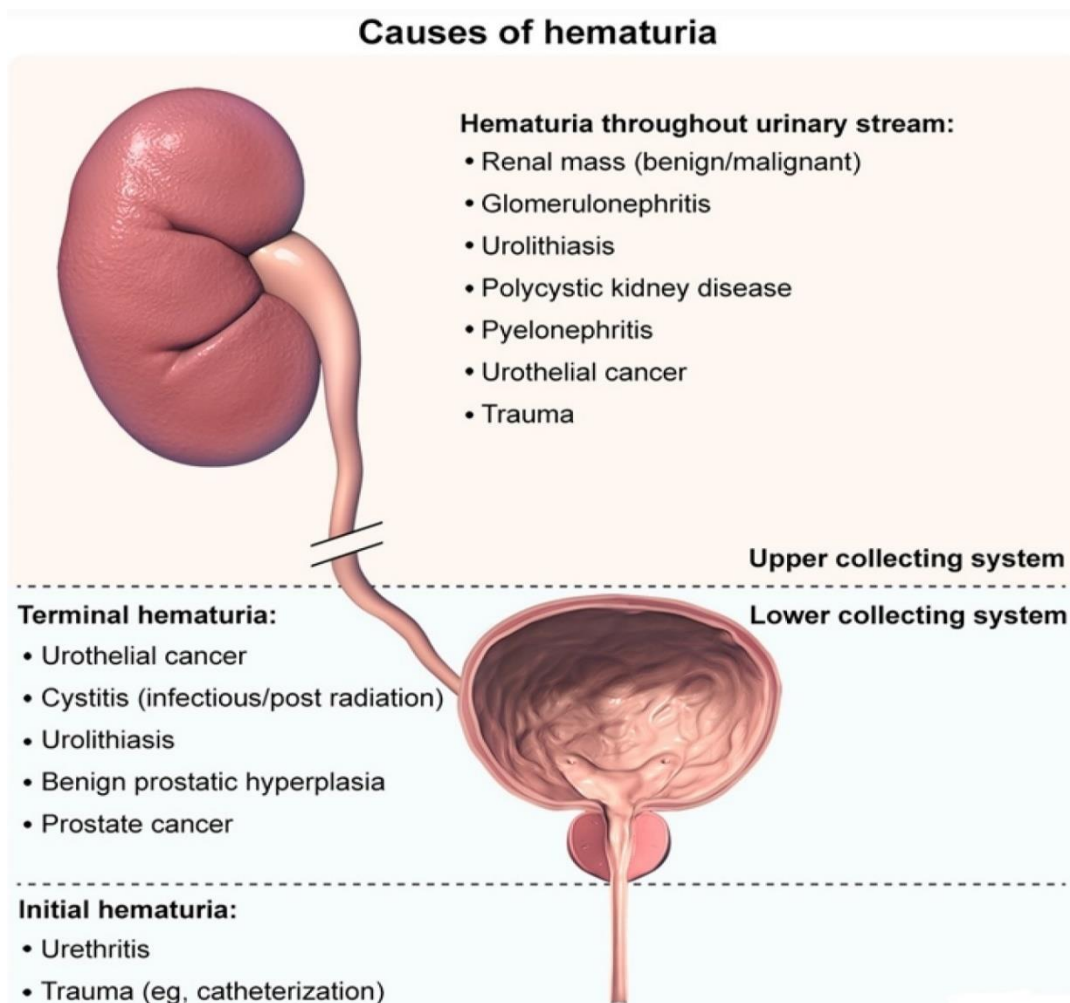
- Non-glomerular hematuria is **more common**.
- It is characterized by **gross hematuria with normal-appearing red blood cells (RBCs) but without significant proteinuria**.
- Etiologies include **cancer** (renal cell, prostate), **infections** (cystitis), **polycystic kidney disease**, and **nephrolithiasis**.
- Patients frequently have **specific urinary symptoms** (dysuria, flank pain, renal colic) that help to clarify

	Glomerular hematuria	Non-glomerular hematuria
Type of hematuria	Microscopic > gross hematuria	Gross > microscopic hematuria
Common etiologies	<ul style="list-style-type: none"> • Glomerulonephritis • Basement membrane disorders (eg, Alport syndrome) 	<ul style="list-style-type: none"> • Nephrolithiasis • Cancer (eg, renal cell, prostate) • Polycystic kidney disease • Infections (eg, cystitis) • Papillary necrosis, renal infarction
Clinical presentation	<ul style="list-style-type: none"> • Nonspecific or no symptoms • Nephritic syndrome (hypertension, oliguria, elevated creatinine) 	<ul style="list-style-type: none"> • Dysuria or symptoms of urinary obstruction (flank pain, renal or ureteral colic, anuria)
Urinalysis	<ul style="list-style-type: none"> • Blood and protein • RBC casts, dysmorphic RBCs 	<ul style="list-style-type: none"> • Blood but no protein • Normal appearing RBCs

RBC = red blood cell.

the diagnosis.

- Gross (visible or macroscopic) hematuria can be classified based on the stage of voiding at which bleeding predominates:
 - o **Initial** hematuria is characterized by blood at the **beginning** of the voiding cycle (Initial hematuria suggests **urethral damage**).
 - o **Terminal** hematuria is characterized by blood at the **end** of voiding cycle (terminal hematuria indicates **bladder or prostatic damage**).
 - o **Total** hematuria is characterized by blood during the entire voiding cycle (total hematuria reflects damage in **the kidney or ureters**).

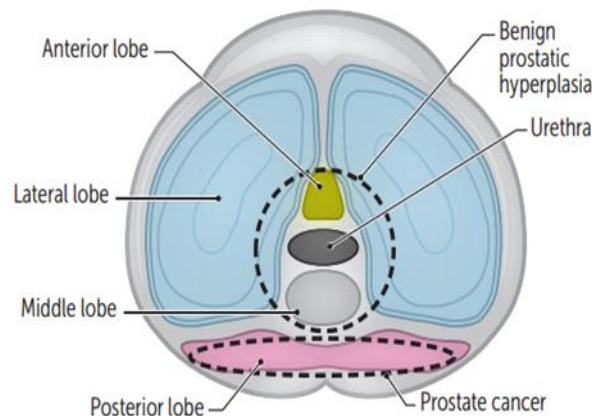


❖ N.B:

- Rifampin causes red to orange discoloration of body fluids. 'Red urine' in a patient taking rifampin is usually a benign drug effect.

▪ Benign prostatic hyperplasia (BPH):

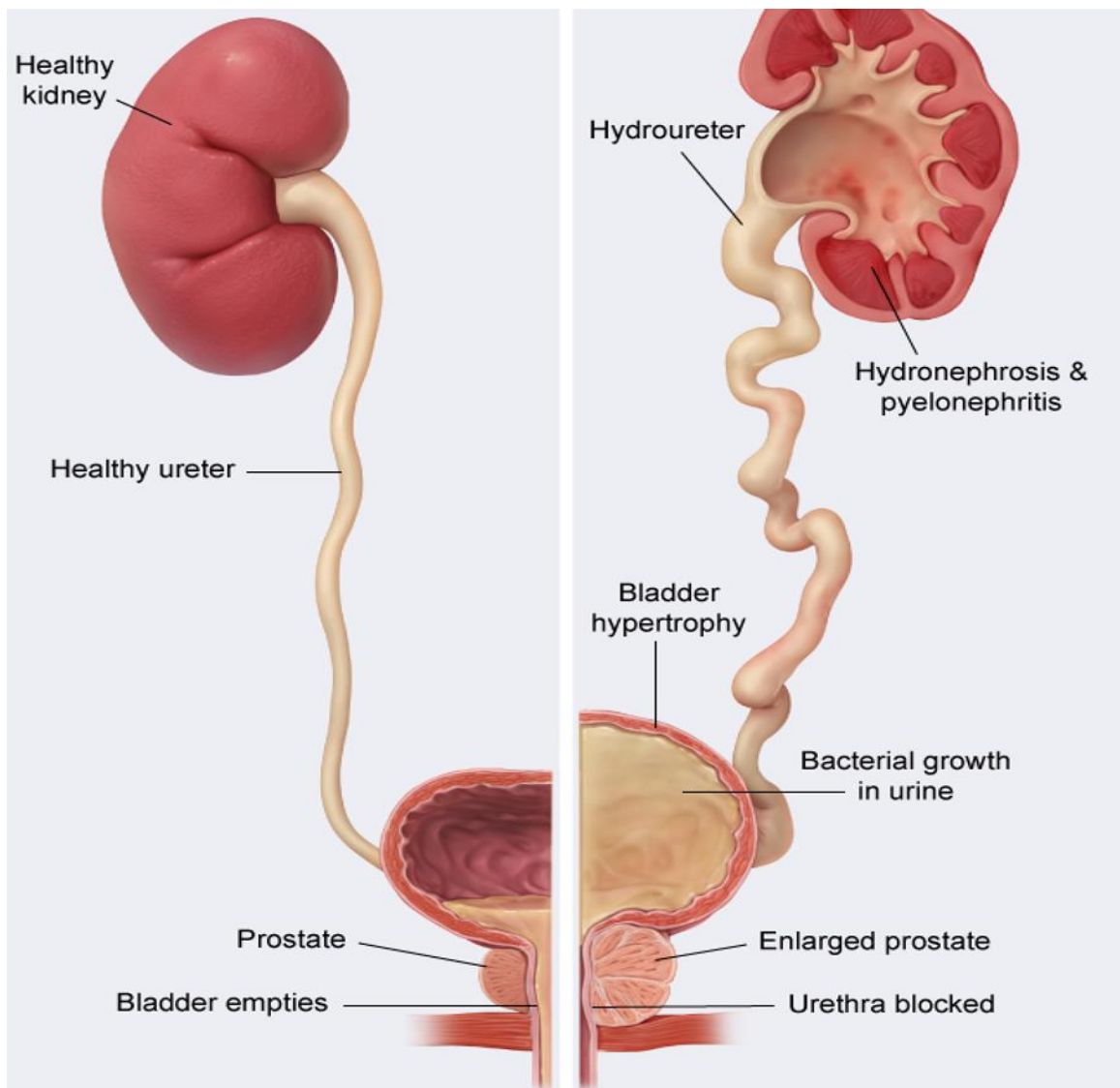
- Hyperplasia of prostatic stroma and glands.
- Age-related change (present in most men by the age of 60 years); no increased risk for cancer.
- Related to dihydrotestosterone (DHT):
 - Testosterone is converted to DHT by 5 α -reductase in stromal cells.
 - DHT acts on the androgen receptor of stromal and epithelial cells resulting in hyperplasia.
- Occurs in the central periurethral and transition zones of the prostate (lateral and middle lobes), which compress the urethra into a vertical slit.



- Clinical features:

- Urinary complaints are the primary symptoms in BPH, including urinary hesitancy, urgency, frequency, incomplete voiding, post-void leakage of urine, and nocturia.
- Urinary retention leads to increased pressure in the urinary tract, causing characteristic morphological changes. The bladder wall hypertrophies, to increase its contractile force. As urinary retention progresses, the ureters, renal pelvis, and calyces dilate and deform, leading to hydronephrosis.
- The renal parenchyma ultimately becomes atrophic and scarred due to reflux of urine and damage of renal tissue.
- The condition should be promptly treated, as prolonged obstruction can cause permanent damage and chronic renal failure.

- **Management:**
- Diagnosis is made from the history and physical, including the digital rectal exam. On palpation, the prostate has a **rubbery, smooth consistency**, in contrast to prostate cancer, where the gland is **nodular and very firm**.
- Prostate-specific antigen (PSA) is often **slightly elevated** due to the increased number of glands; PSA is made by prostatic glands and liquefies semen.
- **α 1-antagonists** (terazosin, **tamsulosin**), which cause relaxation of smooth muscle; **5 α -reductase inhibitors** (finasteride) which reduce hormonal influence on the prostate by preventing the conversion of testosterone to dihydrotestosterone.



- Prostate adenocarcinoma:
 - Malignant proliferation of prostatic glands.
 - **Most common cancer in men**; 2nd most common cause of cancer-related death.
 - Risk factors include age, race (**African Americans** > Caucasians > Asians), and diet high in saturated fats.
 - Prostatic carcinoma is most often **clinically silent**.
 - **Usually arises in the peripheral, posterior region of the prostate**, hence, does not produce urinary symptoms early on.
 - Because the tumor is located on the periphery of the prostate, it is easily detected on digital rectal examination **as an asymmetric nodular enlargement of the prostate**.
 - **Significant elevation** of prostate-specific antigen (PSA) support the diagnosis but are not present in all cases. Confirmation generally requires **transrectal biopsy**.
 - **Spread to lumbar spine or pelvis is common; results in osteoblastic metastases that present as low back pain and increased serum alkaline phosphatase (due to bone metastases), PSA, and prostatic acid phosphatase (PAP).**
 - Prostatectomy is performed for localized disease; advanced disease is treated with hormone suppression to reduce testosterone and DHT. Continuous GnRH analogs (**leuprolide**) shut down the hypothalamus (LH and FSH are reduced). **Flutamide** acts as a competitive inhibitor at the androgen receptor.
 - Prostatectomy may have a slight benefit over radiation in terms of survival. The most common complications of prostatectomy are:
 - **Erectile dysfunction.**
 - **Urinary incontinence.**

Comparison of benign prostatic hyperplasia & prostate cancer		
	BPH	Prostate cancer
Risk factors	• Age >50	• Age >40, African American & family history
Affected part	• Central portion (transitional zone)	• Usually peripheral zone of prostate but can be anywhere
Examination	• Symmetrically enlarged & smooth prostate • Can have elevated PSA	• Asymmetrically enlarged, nodules & firm prostate • Markedly elevated PSA

BPH = benign prostatic hyperplasia; PSA = prostate-specific antigen.

▪ **Testicular Cancer:**

- Testicular cancer presents with a **solid, hard, painless testicular mass that does not transilluminate. Increased with history of cryptorchidism.**
- Arise from **germ cells or sex cord-stroma.**
- **Germ cell tumors are the most common type** of testicular tumor (> 95% of cases).

Malignant testicular neoplasms		
Germ cell (95%)	Seminoma	<ul style="list-style-type: none"> • Retain features of spermatogenesis • β-hCG, AFP usually negative
	Nonseminoma	<ul style="list-style-type: none"> • ≥ 1 partially differentiated cells: yolk sac, embryonal carcinoma, teratoma, and/or choriocarcinoma • β-hCG, AFP usually positive
Stromal (5%)	Leydig	<ul style="list-style-type: none"> • Often produces excessive estrogen (gynecomastia) or testosterone (acne) • Can cause precocious puberty
	Sertoli	<ul style="list-style-type: none"> • Rare • Occasionally associated with excessive estrogen secretion (eg, gynecomastia)

AFP = alpha-fetoprotein.

- **Diagnostic Testing:**
 - Scrotal ultrasound, which usually reveals a solid, hypoechoic lesion (seminoma) or a lesion with cystic areas and calcifications (nonseminomatous germ cell tumor [NSGCT]).
 - Serum tumor markers such as β -hCG, alpha-fetoprotein, and lactate dehydrogenase, which are often elevated (particularly with NSGCTs).
 - **Radical inguinal orchiectomy, which is performed to confirm the diagnosis histologically and provide definitive treatment.**
 - Do not cut the scrotum, which can spread the disease. **Needle biopsy of the testicle is always a wrong answer.**
 - **After orchiectomy, radiation is used for local disease and chemotherapy is used for widespread disease.**

