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Shock

<table>
<thead>
<tr>
<th></th>
<th>Hypovolemic Shock</th>
<th>Cardiogenic Shock</th>
<th>Obstructive Shock</th>
<th>Distributive Shock (Septic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Output</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Increased</td>
</tr>
<tr>
<td>Vascular resistance</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Mixed venous O2 saturation (Mvo2)</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Normal or High</td>
</tr>
<tr>
<td>Base deficit</td>
<td>High</td>
<td>High</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>PCWP</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>LVEDP</td>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Cardiac index (Normal: 2.8-4.2)</td>
<td>Decreased</td>
<td>Very Decreased</td>
<td></td>
<td>Increased</td>
</tr>
</tbody>
</table>

Neurogenic shock:
- It is also a type of distributive shock but in this, the MVO2 will be low because: hypoperfused tissue extracts more oxygen.
- Whenever there is hypotension, there is always tachycardia, BUT in neurogenic shock is special that it has: **HYPOTENSION + BRADYCARDIA** → In exam whenever you see hypotension + bradycardia always think of these possibilities:
  1. Neurogenic shock
  2. Heart block
  3. Right ventricular infarction

Notes:
1- MVO2 is the best indicator of tissue hypoxia.
   - In hypovolemic shock, decreased blood flow through tissue leads to the increased extraction of oxygen hence MvO2 decreases
   - In septic shock, as blood flow is increased, tissue can’t extract oxygen so Mvo2 increases
2- In septic shock, decreased LVEDP is due to neutrophil transmission through pulmonary capillaries into alveoli → produces non-cardiogenic pulmonary edema

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

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

**Class I hemorrhage:** The patient compensates for blood loss through Sympathetic response that induces:
1- mild tachycardia
2- peripheral vascular constriction. Capillary refill is maintained (< 2 seconds).

**Class II hemorrhage:** All of the manifestations can be attributed to further increases in sympathetic discharge and shunting of blood from less critical vascular beds such as the skin, leading to skin vasoconstriction → skin is cold & moist.

**Class III hemorrhage:** Decompensation=Hypotension (can no longer maintain their blood pressure at normal levels) despite further increases in heart rate and peripheral vascular constriction. Leading to:
1- decreased level of consciousness
2- further decrease in urine output due to poor cerebral and renal perfusion, respectively.

**Class IV hemorrhage:** Patient appears lethargic, tachypneic and has markedly decreased urine output. Circulatory failure & death are imminent → without therapeutic intervention.

First physiological changes after hemorrhage: tachycardia and peripheral vascular constriction are the first physiological changes.
Trauma

Glasgow Coma Scale

It's used to:
1- Predict the prognosis of coma
2- Predict prognosis of other medical conditions, such as:
   - bacterial meningitis,
   - traumatic brain injury,
   - subarachnoid hemorrhage.
3- Diagnose coma \( \rightarrow \) Findings used to diagnose coma include:
   a- Impaired brainstem activity, e.g. disruption of :
      - Papillary light
      - Extraocular muscles
      - Corneal reflexes
   b- Motor dysfunction (e.g., decorticate or decerebrate posturing),
   c- Impaired level of consciousness (GCS < 8)

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
- Obey: 6
- Localizes: 5
- Withdraws: 4
- Flexion posturing (Decorticate): 3
- Extension posturing (Decerebrate): 2
- None: 1

Blunt Head Trauma

- After blunt head trauma:
  1- Look for \( \rightarrow \) Scalp lacerations, skull depression
  2- Look for \( \rightarrow \) Signs/symptoms worrisome for transtentorial (uncal) herniation secondary to an Epidural hematoma

Epidural Hematoma:

- Cause: from rupture of the middle meningeal artery \( \rightarrow \) arterial pressure can rapidly expand hematoma & compress temporal lobe.
  The fluid resuscitation in such patient likely increased the rate at which the epidural hematoma expanded.
- Cushing's triad: 1- Hypertension, 2- Bradycardia, 3- Respiratory despression \( \rightarrow \) Elevated intracranial pressure (ICP)
- CT scan: Biconvex hematoma

Uncus is the inner most part of the temporal lobe and herniates through the tentorium to cause pressure on:
1- Contralateral cerebral peduncle against the edge of the tentorium
2- Ipsilateral oculomotor nerve
3- Ipsilateral posterior cerebral artery

Transtentorial (Uncal) Herniation

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Neurologic Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compression of the contralateral crus cerebri against the tentorial edge</td>
<td>Ipsilateral hemiparesis</td>
</tr>
<tr>
<td>Compression of the ipsilateral oculomotor nerve (i.e., CN III) by the herniated uncus</td>
<td>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 abducens (i.e., CN VI) action (occurs late)</td>
</tr>
<tr>
<td>Compression of the ipsilateral posterior cerebral artery (i.e., ischemia of visual cortex)</td>
<td>Contralateral homonymous hemianopsia</td>
</tr>
<tr>
<td>Compression of the reticular formation</td>
<td>Altered level of consciousness; coma</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Minor head trauma (most)</th>
<th>Traumatic brain injury (TBI)</th>
<th>Severe TBI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GCS = 15</strong></td>
<td><strong>Mild TBI</strong></td>
<td><strong>Moderate TBI</strong></td>
</tr>
<tr>
<td>- Normal mental status exam.</td>
<td>- GCS= 13-15</td>
<td>- GCS= 12-9</td>
</tr>
<tr>
<td>- No abnormal neurologic exam.</td>
<td>Patients with mild-to-moderate TBI</td>
<td></td>
</tr>
<tr>
<td>- No abnormal Fundus exam.</td>
<td>- If patient has <strong>vomiting, headache, or brief loss of consciousness</strong>:</td>
<td></td>
</tr>
<tr>
<td>- no physical evidence of skull fracture</td>
<td>→ should also have a <strong>CT scan</strong> of the head.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- If CT scan is normal → <strong>discharge</strong> with a reliable caretaker and printed instructions (with a list of symptoms) that describe when they should return to the hospital.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>2- If patients doesn’t have any of the above symptoms:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>→ <strong>Do not need neuroimaging</strong> and can be observed for <strong>4-6 h</strong> in the emergency department.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>with neuroimaging reserved for those who worsen</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Discharged with NO further imaging or studies</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>if a reliable individual can monitor them for <strong>24 hours</strong> following the injury.</td>
<td></td>
</tr>
</tbody>
</table>

**Diffuse axonal injury**

Most significant cause of morbidity in patients with traumatic brain injuries (TBI)

**Cause:**
- It is frequently due to **traumatic deceleration**: Sudden acceleration-deceleration impact produces rotational forces affect brain areas

**Site:**
- Where the density difference is the maximum, thus most of the diffuse axonal injury occur at **gray white matter junction**.

**Clinical features:**
- Patient loses consciousness instantaneously and later develops **persistent vegetative state**.

**Diagnostic imaging:**
1- **CT scan** characteristically **shows numerous minute punctate hemorrhages with blurring of grey white interface**.
2- **MRI** is more sensitive than **CT scan** for diagnosing diffuse axonal injury

**Intracranial pressure:**
- The **Monro-Kellie principle** states that the volume within the skull is fixed and normally contains three components: **brain, blood, and CSF**.

  - Increase in the amount of these components (e.g., cerebral edema, hematoma, hydrocephalus) or
  - Addition of other components (e.g., tumor)

results in **increased ICP**.

Using this principle, treatment is aimed at either:
1- Reducing the volume of the components or
2- Expanding the volume available through surgical decompression.

**Cerebral blood flow (CBF)** determines the volume of blood and increases with:
1- **Hypercapnia**
2- Increased metabolic demand
3- **Hypoxia**

through **Cerebral vasodilation & Elevated blood pressure** → increase CBF → increase ICP

**Treatment of increased ICP** focuses on maintaining Cerebral Perfusion Pressure:
( The goal is to keep CPP > 70–80 mm Hg )

**CPP** = mean arterial pressure (MAP) - ICP

**Short-term hyperventilation** lowers the ICP by lowering the vascular CO2 concentration in the brain (i.e., CO2 washout), leading to cerebral vasoconstriction.

However, this should be closely monitored because extreme hyperventilation can decrease CBF too much and lead to marked iatrogenic brain ischemia.
Airway Assessment (A)

1. Determine the patient's level of consciousness and
2. Note the presence of any respiratory effort. (Dyspnea – cyanosis – respiratory movement)
3. In known or suspected cervical-spine (C-spine) injury, all assessments should be undertaken with the C-spine immobilized.

- **First step** in evaluating this 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.
- **Next step** is to assess the airway because unstable lesions:
  - Above C3 level → can cause immediate paralysis
  - Below C3 level → can damage the phrenic nerve
- Cervical spine injuries can be associated with:
  1. Oral maxillofacial trauma,
  2. Hemorrhage in the retropharyngeal space,
  3. Airway and neck edema.

<table>
<thead>
<tr>
<th>Apneic, Unconscious Patients</th>
<th>Patients with Respiratory Effort</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open the airway</td>
<td></td>
</tr>
<tr>
<td>1) If C-spine is not injured</td>
<td>Signs: wheezing, sonorous respirations, stridor, cough, and dysphonia.</td>
</tr>
<tr>
<td>2) If C-spine is injured</td>
<td>- Administer high-flow oxygen.</td>
</tr>
<tr>
<td>Place the head in the sniffing position with chin lift maneuver</td>
<td>- Clear and position the airway as described.</td>
</tr>
<tr>
<td>Jaw thrust maneuver</td>
<td>- Prolapse of the tongue and accumulation of secretions, blood, or vomitus are common causes of obstruction.</td>
</tr>
<tr>
<td>Clearing the airway</td>
<td>- Back blows or the Heimlich maneuver may clear the obstruction. If not, use suction or direct visualization and a Magill forceps or finger.</td>
</tr>
<tr>
<td>- 1) Clear the airway of obstructions using a rigid suction catheter to remove any blood, vomitus, or secretions from the oropharynx.</td>
<td>Obstructions that persist require intubation:</td>
</tr>
<tr>
<td>- 2) Remove any large obstructing foreign bodies from the oropharynx 1) manually or with 2) Magill forceps</td>
<td>1. Oropharyngeal or Nasopharyngeal tube.</td>
</tr>
<tr>
<td>If the patient remains apneic</td>
<td>2. Orotracheal or Nasotracheal tube</td>
</tr>
<tr>
<td>Assist ventilation using:</td>
<td>3. Cricothyroidotomy,</td>
</tr>
<tr>
<td>1) Equipments are available</td>
<td>4. Tracheostomy, or percutaneous transtracheal jet ventilation (PTTJV).</td>
</tr>
<tr>
<td>2) Equipment aren’t available</td>
<td></td>
</tr>
<tr>
<td>1- Endotracheal intubation.</td>
<td>1. Oropharyngeal or Nasopharyngeal tube.</td>
</tr>
<tr>
<td>2- Administer high-flow O2</td>
<td>2. Orotracheal or Nasotracheal tube</td>
</tr>
</tbody>
</table>

Orotracheal intubation with rapid sequence intubation (RSI):
- It is the preferred route to manage unstable and apneic patients to protect the airway and provide oxygenation.
- Four people are involved in RSI:
  - one manually stabilizes the patient
  - one induces the patient with anesthesia
  - one applies cricoid pressure to prevent passive regurgitation until endotracheal tube placement is confirmed
  - one places the endotracheal tube.

Manual stabilization requires firmly holding either side of the patient’s head with the neck midline and on a firm surface, without applying traction. This prevents any flexion or rotation of the neck during intubation.

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

Surgical cricothyroidotomy is preferred over surgical tracheostomy.
- But should be converted to formal tracheostomy in 5-7 days if prolonged airway control is needed.
- Prolonged use of cricothyroidotomy has a high incidence of: tracheal stenosis.

Tracheostomy is no longer a first option to establish an airway because of its complications.
## Chest Trauma

**Immediately Life-Threatening Thoracic Injuries Identified on the Primary Survey**

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tension Pneumothorax</strong></td>
<td>Diagnosis is made clinically because the patients need immediate treatment &amp; should NOT wait for X-ray. Symptoms: Pneumothorax is characterized by Dyspnea, Tachypnea, Hypoxia, Hypotension. Inspection: 1. Jugular venous distention (but this may be absent if the patient is hypovolemic) 2. Diminished of chest movement. Palpation: tracheal shift away from the affected side. Percussion: the chest is hyperresonant. Auscultation: decreased or absent breath sounds on the affected side. Treatment: Needle thoracostomy should be done emergently; followed by tube thoracostomy (chest tube).</td>
</tr>
<tr>
<td><strong>Open pneumothorax</strong></td>
<td>There is equilibration between intrathoracic and atmospheric pressure and so negative intrathoracic pressure cannot be generated → thus, effective ventilation is impaired since air goes through the chest wall rather than into the lung resulting in severe hypoxia. Treatment: Needle thoracostomy should be done emergently; followed by tube thoracostomy &amp; closure of the wound.</td>
</tr>
<tr>
<td><strong>Massive hemorthorax</strong></td>
<td>Injury to the chest wall, great vessels, or lung can result in intrapleural bleeding or hemorthorax. Symptoms: Dyspnea, Tachypnea, Hypoxia. Blood loss leads to: ↓ Hypotension, ↓ Narrow pulse, Neck Veins are collapsed. Palpation: contralateral tracheal deviation. Percussion: dullness to percussion. Auscultation: decreased breath sounds on the affected side. Treatment: 1. Tube thoracostomy should be performed immediately. 2. Surgical thoracostomy if: 1. Initial blood loss &gt; 1.0 or 2. Ongoing continuing loss 150-200 mL/h for 2-4 h.</td>
</tr>
<tr>
<td><strong>Flail Chest</strong></td>
<td>caused by three or more adjacent rib fractures that break in two places → unstable chest wall segment that moves in a paradoxical motion with respiration → the flail segment moves inward with inspiration, thus ↓ tidal volume. Symptoms: 1. Pain 2. Dyspnea. 3. Tachypnea (the patient may be able to compensate initially for the reduced tidal volume by hyperventilating) with shallow respirations secondary to pain. 4. Paradoxical chest wall movement. 5. When fatigue or underlying pulmonary injury develops → frank respiratory failure. Treatment: 1. Early steps: A) Supplemental oxygen (1st line of ttt)/ B) Pain control with IV morphine or fentanyl 2. Mechanical positive pressure ventilation: Replaces the normal negative intrapleural pressure during spontaneous ventilation with positive intrapleural pressure.</td>
</tr>
<tr>
<td><strong>Cardiac Tamponade</strong></td>
<td>→ lead to Cardiogenic Shock. - pericardium is not very distensible and tamponade can occur even with a small amount (200 mL) of blood. Symptoms: 1. Pulsus Paradoxus (more than 10 mmHg decrease in Systolic BP during inspiration. 2. Beck's triad: 1) Hptension (despite aggressive fluid) 2) JVD 3) Muffled heart sounds. ECG: 1- Electrical alternans 2- Sinus tachycardia 3- Low voltage QRS. CXR: Normal without a change in cardiac silhouette size due to the small amount of pericardial fluid. Treatment: 1. Volume resuscitation. 2. Immediately with decompression: Emergent pericardiocentesis or surgical pericardiotomy.</td>
</tr>
<tr>
<td><strong>Blunt Aortic Injury</strong></td>
<td>Suspcion: 1- Motor vehicle accidents 2- Falls from &gt; 10 feet. Symptoms: Highly variable, but common are: 1- Anxiety 2- Tachycardia 3- Hypertension. Screening: Chest x-ray is appropriate initial screening study: 1. Mediastinal widening is the most sensitive finding for blunt aortic injury. 2. Deviation of the trachea or nasogastric tube to the right. 3. Depression of the left mainstem bronchus. → Where the history and chest x-ray findings are equivocal, chest CT and angiography are appropriate.</td>
</tr>
<tr>
<td><strong>Tracheobronchial Perforation</strong></td>
<td>These patients suffer rapid deceleration chest trauma. The right main bronchus is most commonly injured in these cases. Most important initial diagnostic study in all stabilized patients (airway, breathing and circulation secure) following blunt chest trauma → Chest radiography. If chest x-ray shows: 1. Persistent pneumothorax despite chest tube placement 2. Pneumomediastinum. And on examination: 3. Subcutaneous emphysema → then diagnosis of: Tracheobronchial Perforation. Diagnosis confirmed with: high-resolution CT scanning, bronchoscopy or surgical exploration. Operative repair.</td>
</tr>
</tbody>
</table>
## Miscellaneous Chest Traumas

<table>
<thead>
<tr>
<th>Pulmonary contusion</th>
<th>The forces associated with blunt thoracic trauma can be transmitted to lung parenchyma.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms:</strong></td>
<td>- Usually develop usually in the <strong>first 24 hours</strong> (often within few minutes);</td>
</tr>
<tr>
<td></td>
<td>- **↑**Tachypnea, **↑**Tachycardia, **↓**Hypoxia ... are characteristic.</td>
</tr>
<tr>
<td><strong>Examination:</strong></td>
<td>- Chest wall bruising</td>
</tr>
<tr>
<td></td>
<td>- <strong>Unilateral</strong> decreased breath sounds on side of pulmonary contusion.</td>
</tr>
<tr>
<td><strong>Investigations:</strong></td>
<td>- Chest x-ray → reveals patchy irregular alveolar infiltrate</td>
</tr>
<tr>
<td></td>
<td>- CT scan → may be employed to make an early diagnosis.</td>
</tr>
<tr>
<td></td>
<td>- ABG → typically shows hypoxemia.</td>
</tr>
</tbody>
</table>

**ARDS:**
- Manifests **24-48 hours** from the trauma
- **Bilateral** lung involvement

**ARDS:** 1. Manifests **24-48 hours** from the trauma 2. **Bilateral** lung involvement

**Treatment:**
- Administration of large volumes of IV fluid may hasten this process.

### Rib fractures

- Should be suspected in all patients with localized chest wall tenderness following trauma;
- Significant pain, which causes hypoventilation that may ultimately result inatelectasis and pneumonia

**Investigations:**
- Chest x-ray: BUT up to half of rib fractures will not be evident on initial chest x-ray.

**Treatment:**
- Pain management and respiratory support are the priorities in the management of rib fractures:
  1. Oral agents → as: opiates and/or NSAIDS are most commonly utilized
  2. BUT an intercostal nerve block with a long-acting local anesthetic:
     - can be used if oral or systemic analgesics are not sufficiently effective.
     - Provide pain relief without affecting respiratory function (as opiate analgesics) but there is risk of pneumothorax.

### Cardiac Contusion

**Myocardial contusion** should be suspected in patients with evidence of injury to the anterior chest and

- In the setting of trauma (blunt chest trauma) → low PCWP → Hypovolemic shock from blood loss.
  - Elevated CVP/PCWP or failure of hypotension to resolve after a bolus of intravenous fluids → suggest an alternative diagnosis.

**Investigations:**
- can be confirmed with positive cardiac markers and EKG changes.  

Question from medscape: (look at Chest x-ray)
Blunt cardiac trauma can cause myocardial rupture as a result of cardiac compression between the sternum and the spine, direct impact, or deceleration injury → right atrium is the most commonly involved chamber.

This patient also have tension pneumothorax → after placement the chest tube → the next most appropriate step is doing ECHO

### Iatrogenic Tension Pneumothorax

- **Placement of subclavian central venous catheters** accounts for about 1/4 of iatrogenic tension pneumothorax.
- **Clinical features & management as the same as traumatic tension pneumothorax**

- Patients who continue to remain hemodynamically unstable after needle decompression → should have a FAST (Focused Assessment with Sonography for Trauma) examination to look for pericardial tamponade.

### Cardiac Contusion

**Myocardial contusion** should be suspected in patients with evidence of injury to the anterior chest and
Abdominal Trauma

Sharp Abdominal Trauma

- **On expiration**, diaphragm can rise as high as:
  - 4th thoracic dermatome on right
  - 5th thoracic dermatome on the left (i.e., level of nipples)
- Any penetrating injury in the thorax **below the level of the nipples** has great potential to involve *abdomen* through the diaphragm and is assumed to involve both compartments until proven otherwise.
- **On inspiration**, diaphragm can go down to the 12th thoracic dermatome on both sides.

**First step:** *Fluid resuscitation*  
Then, see if the patient is hemodynamically **STABLE** /or **UNSTABLE** & needs exploratory laparotomy.

1. **If the patient is UNSTABLE:** → Doing FAST → If negative → Directly go for Exploratory Laparotomy
2. **If the patient is STABLE:** → doing FAST → If negative → Do Diagnostic Peritoneal Lavage (DPL)

Penetrating Sharp Abdominal Trauma (Gun Shot Wound (GSW))

- Indication of **immediate exploratory laparotomy**:
  1. Hemodynamically unstable patient
  2. Evidence of peritonitis
  3. Evisceration of any organ

- Would be more likely to occur in penetrating (rather than blunt) trauma.
- Initial presentation as an acute abdomen.
- Upright abdominal x-ray studies (or CT scan): free air under the diaphragm

**Spleen Injury**

Most commonly injured organ following blunt abdominal trauma

<table>
<thead>
<tr>
<th>Symptoms:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- symptoms can occur hours later, indicating ongoing splenic injury.</td>
</tr>
<tr>
<td>1. <strong>Left upper quadrant abdominal pain</strong></td>
</tr>
<tr>
<td>2. <strong>abdominal wall contusion</strong></td>
</tr>
<tr>
<td>3. <strong>left shoulder pain</strong> referred from splenic hemorrhage irritating phrenic nerve and diaphragm (Kehr sign).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- The initial examination after Blunt Abdominal Trauma can be <strong>unremarkable</strong></td>
</tr>
<tr>
<td>1. <strong>Left lower chest wall tenderness</strong></td>
</tr>
<tr>
<td>2. <strong>Hypotension</strong></td>
</tr>
</tbody>
</table>

**Investigations:**
- Initial CT can diagnose the injury.
### Duodenal hematomas

- Most Commonly occur following direct blunt abdominal trauma
- More commonly seen in children.
  - Fallowing trauma, blood collects between the submucosal and muscular layers of the duodenum causing obstruction.

#### Symptoms:
- Patients classically present with epigastric pain and vomiting due to the failure to pass gastric secretions past the obstructing hematoma.

#### Treatment:
- Most hematomas will resolve spontaneously in 1-2 weeks
- Conservative: Intervention of choice is nasogastric suction and parenteral nutrition.
- Surgery: may be considered to evacuate the hematoma if this more conservative method fails.

#### Investigations:
- Blunt abdominal trauma can compress the neck and/or body of the pancreas against the vertebral column resulting in: 1- Pancreatic contusion 2- Crush injury 3- Laceration 4- Transaction

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasogastric suction + parenteral nutrition</td>
<td>Conservative treatment for hematomas</td>
</tr>
<tr>
<td>Nasogastric suction and parenteral nutrition</td>
<td>If conservative method fails</td>
</tr>
</tbody>
</table>

---

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### Pediatric Surgery: Congenital Diaphragmatic Hernia

- The primary abnormality is the hypoplastic lung with fetal type circulation
- **Symptoms:** Dyspnea is noted at birth
- **X-ray:** Loops of bowel in left chest are seen on x-ray.
- **Treatment:** 1. By performing: endotracheal intubation, low-pressure ventilation, sedation and NG suction.
  2. Delay repair 3-4 days to allow lung maturation.
Traumatic Amputation:
- All patients suffering traumatic amputations should be treated as candidates for reimplantation while in the field.
- As such, their amputated limb or digit should be wrapped in sterile gauze, moistened with sterile saline and placed in a plastic bag.
  ➔ Packaging of the amputated part in this manner prolongs the viability of the part for up to 24 hours.
- Younger patients suffering sharp amputations with no crush injury or avulsion are the best candidates for amputation reimplantation.

Traumatic Lower Spinal Cord injury:
- It is most commonly caused by motor vehicle accidents.
- The trauma causes:
  1. Primary injury to the spinal cord through: mechanical compression, contusion, or shear injury.
  2. Secondary injury follows (within minutes to hours) causes: spinal cord edema that eventually leads to central hemorrhagic necrosis.
- Management:
  1. All trauma patients with suspected spinal injuries should first be hemodynamically stabilized and have a secure airway (possibly requiring intubation). The neck should be immobilized until spinal injury has been ruled out.
  2. Then, Investigations: IMAGING: such as CT scans and x-rays, might then be required to:
     a. Diagnose the injury and
     b. Evaluate the extent of spinal cord damage.
- Management:
  1. High-dose intravenous steroids (e.g., methylprednisolone): is somewhat controversial, with conflicting evidence on benefit in spinal cord injury patients.
  2. Surgical decompression is indicated in patients who need acute spine stabilization.

Hypovolemic shock & Positive Pressure Ventilation:
- Hypovolemia due to hemorrhage → decreased venous return and therefore decreased end diastolic volume and cardiac output.
  ➔ Hypovolemic shock due to hemorrhage → decreased venous return→compensating for the hypovolemia by improving venous return.
- Positive pressure mechanical ventilation:
  1. Acutely increases intrathoracic pressure, increasing right atrial pressure and decreasing systemic venous return. This sudden loss of venous return may cause acute circulatory failure and death.
  2. Sedative medications used prior to intubation relax venous capacitance vessels and may themselves cause circulatory failure.

Tetanus Immunization
- All patients with traumatic wounds should be assessed for the need of tetanus prophylaxis.
  • Tetanus immune globulin (TIG): provides passive, temporary, and immediate immunity.
  • Tetanus-diphtheria toxoid (Td): provides active, prolonged, and delayed immunity.

Td booster should be administered to:
1. Individuals with more severe or dirty wounds who received their latest dose more than 5 years ago and
2. Individuals with unimmunized minor wounds who received their latest dose more than 10 years ago.

TIG injection should be administered to:
1. Individuals who have received less than three doses of tetanus vaccine and
2. Individuals with more severe or dirty wounds who have an unknown immunization status.

Wounds at high risk for vegetative Clostridium tetani growth are those that provide an anaerobic environment for growth, such as
1. Puncture wounds
2. Projectile wounds
3. Wounds containing foreign bodies
4. Sites of active infection by other organisms
5. Wounds containing necrotic tissue.

Tetanus prophylaxis

<table>
<thead>
<tr>
<th></th>
<th>Clean or minor wound</th>
<th>Dirty or severe wound</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥3 tetanus toxoid doses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetanus toxoid-containing vaccine* only if last dose was &gt;10 years ago</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No TIG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TIG</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Unimmunized, uncertain, or <3 tetanus toxoid doses

<table>
<thead>
<tr>
<th></th>
<th>Clean or minor wound</th>
<th>Dirty or severe wound</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tetanus toxoid-containing vaccine* only if last booster given &gt;5 years ago</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No TIG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetanus toxoid-containing vaccine* PLUS TIG</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Booster given as tetanus/diphtheria toxoids adsorbed (Td) or tetanus toxoid/acellular pertussis (Tdap)

TIG = Tetanus immune globulin.
## Orthopedics

### Dislocations

#### Shoulder

<table>
<thead>
<tr>
<th>Anterior (Most common)</th>
<th>Force</th>
<th>Examination &amp; Deformity</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Posteriorly directed force on distal humerus or forearm during abduction causes <strong>cantilever effect that drives humeral head forward and tears anterior shoulder capsule</strong></td>
<td><strong>Physical exam:</strong>&lt;br&gt;1- Prominence of the acromion&lt;br&gt;2- with an abnormal subacromial space where the humeral head normally resides.</td>
<td><strong>Defomity:</strong>&lt;br&gt;Patient holds the arm slightly abducted and internally rotated&lt;br&gt;<strong>Anteriorly directed force on proximal humeral epiphysis</strong>&lt;br&gt;Anterior sag test: &lt;br&gt;Reverse pivot shift test&lt;br&gt;<strong>Dashboard injury:</strong>&lt;br&gt;Causes humeral head dislocation during abduction causes <strong>rotation deformity</strong>&lt;br&gt;<strong>Neurovascular injury:</strong> Femoral Artery/Nerve&lt;br&gt;<strong>Closed reduction:</strong> (Kocher’s method)</td>
</tr>
</tbody>
</table>

| Posterior | Force on: Flexed, Abducted and externally rotated hip<br>Nerve injury: Sciatic Nerve<br>**Neurovascular injury:** Femoral Artery/Nerve | LL is :<br>**Posterior drawer test**<br>**Reverse pivot shift**<br>**Varus stress test**<br>Upper limb is apparently longer |

| Posterior | Force on: Flexed, Abducted and externally rotated hip<br>Nerve injury: Sciatic Nerve<br>**Neurovascular injury:** Femoral Artery/Nerve | LL is :<br>**Posterior drawer test**<br>**Reverse pivot shift**<br>**Varus stress test**<br>Upper limb is apparently longer |

### Ligament Tears

#### Cruciate Ligaments

<table>
<thead>
<tr>
<th>Anterior cruciate ligament injury</th>
<th>Posterior cruciate ligament injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History of :</strong>&lt;br&gt;1- Forceful hyperextension injury to knee&lt;br&gt;2- Noncontact torsional injury of the knee during deceleration</td>
<td><strong>Classically seen in the dashboard injury :</strong>&lt;br&gt;Forceful posterior-directed force on the tibia with the knee flexed at 90 degrees</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tests are used for clinical diagnosis :</th>
<th>Tests are used for clinical diagnosis :</th>
</tr>
</thead>
<tbody>
<tr>
<td>1- Anterior drawer test&lt;br&gt;2- Lachman’s test&lt;br&gt;3- Pivot shift test</td>
<td>1- Posterior drawer&lt;br&gt;2- Posterior sag tests&lt;br&gt;3- Reverse pivot shift</td>
</tr>
</tbody>
</table>

### Collateral Ligaments

<table>
<thead>
<tr>
<th>Medial collateral ligament injury</th>
<th>Lateral collateral ligament injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Abduction injury</strong> to the knee</td>
<td><strong>Adduction injury</strong> to the knee</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tests are used for clinical diagnosis :</th>
<th>Tests are used for clinical diagnosis :</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valgus stress test</td>
<td>Varus stress test</td>
</tr>
</tbody>
</table>
Meniscal Ligament

- Meniscal injuries often result from **twisting injuries** with the foot fixed.
- Medial meniscus is more commonly injured than the lateral meniscus.

### Medial Meniscal Tear

**Symptoms:**
- Patients generally complain of a **popping** or **snapping sensation** followed by severe pain at the time of injury.
- **Swelling and pain** (usually gradually occurs within **24 hours** after the injury)
  - Because the meniscus is not directly perfused, effusion following injury typically is not clinically apparent for many hours.

**Examination:**

<table>
<thead>
<tr>
<th>Knee function</th>
<th>Provocative tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint line tenderness</td>
<td>Popping, catching, catching</td>
</tr>
<tr>
<td>Loss of smooth flex or extension</td>
<td>Knee giving out</td>
</tr>
<tr>
<td>Inability to move forward and backward while squatting</td>
<td>Locking of the knee (i.e., inability to extend the knee) → generally seen in &quot;bucket handle&quot; tears</td>
</tr>
<tr>
<td>Effusion</td>
<td></td>
</tr>
</tbody>
</table>

**Complication:**
- Untreated patients can develop symptoms weeks later consisting of:
  1. Popping, catching
  2. Knee giving out
  3. Locking of the knee (i.e., inability to extend the knee) → is generally seen in "bucket handle" tears

**Differential Diagnosis:**
- Anserine bursitis
  - Typically affects athletes and obese middle-aged to elderly women.
  - Anserine bursitis causes tenderness over the medial aspect of the knee, A popping sensation is NOT typically reported.
  - The anserine bursae underlie the conjoined tendons of the gracilis and semitendinosus muscles and separate them from the head of the tibia.

**Ligamentous Injuries cause rapid joint swelling due to hemarthrosis:**
- Ligaments have much greater vascular supply than menisci (menisci which rely on diffusion for nourishment)
- 
  - Ligamentous injuries cause rapid joint swelling due to hemarthrosis
  - Meniscal injuries usually gradually occur within **24 hours** after the injury

---

**Features of anterior cruciate ligament injury**

<table>
<thead>
<tr>
<th>Injury mechanisms</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Rapid deceleration or direction changes</td>
<td>• Pain: rapid onset, may be severe</td>
</tr>
<tr>
<td>• Pivoting on lower extremity with foot plant</td>
<td>• &quot;Popping&quot; sensation at the time of injury</td>
</tr>
<tr>
<td>• Significant swelling (effusion/hemarthrosis)</td>
<td>• Joint instability</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Examination findings</th>
<th>Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Anterior laxity of tibia relative to femur (anterior drawer test, Lachman test)</td>
<td>• Magnetic resonance imaging</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• RICE (rest, ice, compression, elevation) measures</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• +/- Surgery</td>
</tr>
</tbody>
</table>

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### Common Fractures

<table>
<thead>
<tr>
<th>Type</th>
<th>Bones Involved</th>
<th>History and Physical</th>
<th>Treatment</th>
<th>Clinical Pearls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boxer</td>
<td>Fifth-metacarpal neck</td>
<td>Punching hard object or surface with a strong force applied to fifth metacarpal</td>
<td>1. Closed reduction&lt;br&gt;2. Ulnar gutter splint&lt;br&gt;3. Surgical pinning</td>
<td>Beware the &quot;fight bite&quot;: open wounds from teeth will need surgical Scaphoid exploration to rule out tendon involvement</td>
</tr>
<tr>
<td>Scaphoid</td>
<td>Scaphoid</td>
<td>1. Fall on radially deviated outstretched hand with a dorsiflexed wrist &gt;95&lt;br&gt;2. &quot;Snuffbox&quot; tenderness</td>
<td>Second, if suspected: Thumb spica cast for 7-10d followed by repeating x-rays&lt;br&gt;2. Possible surgery</td>
<td>Increased risk of proximal fracture fragment AVN&lt;br&gt;First, confirm by X-ray: Not seen on X-ray for 1–2 weeks after injury</td>
</tr>
<tr>
<td>Smith</td>
<td>Distal radius</td>
<td>Fall on flexed wrists distal radius is anteriorly displaced</td>
<td>1. Cast&lt;br&gt;2. Closed reduction&lt;br&gt;3. Possible surgery</td>
<td>Much less common than Colles fracture</td>
</tr>
<tr>
<td>Colles</td>
<td>Distal radius</td>
<td>Fall on ulnar deviation with angulation</td>
<td>Closed reduction&lt;br&gt;Cast forearm in supination&lt;br&gt;Surgical repair of ulna</td>
<td>Increased risk of avascular necrosis&lt;br&gt;DVT prophylaxis.</td>
</tr>
<tr>
<td>Fracture of distal radius</td>
<td>Trauma (direct blow or fall)</td>
<td>Surgical repair&lt;br&gt;External rotation of ulna&lt;br&gt;Cast in supination&lt;br&gt;Surgical repair of ulna</td>
<td>Surgical repair&lt;br&gt;External rotation of ulna&lt;br&gt;Cast in supination&lt;br&gt;Surgical repair of ulna</td>
<td>Increased risk of avascular necrosis&lt;br&gt;DVT prophylaxis.</td>
</tr>
<tr>
<td>Humerus</td>
<td>Fracture of proximal one third of ulna with humeral head</td>
<td>Fracture of proximal one third of ulna with humeral head</td>
<td>Closed reduction of radial head&lt;br&gt;2. Surgical repair of ulna</td>
<td>Increased risk of avascular necrosis&lt;br&gt;DVT prophylaxis.</td>
</tr>
<tr>
<td>Pelvic</td>
<td>Pelvis</td>
<td>Major trauma</td>
<td>Pain control&lt;br&gt;surgical repair if in weightbearing portion</td>
<td>High risk of compartment syndrome</td>
</tr>
</tbody>
</table>

### Stress Fractures

The fractures occur due to a sudden increase in repeated tension or compression without adequate rest that eventually breaks the bone.

- It most commonly occurs in athletes (up to 15% incidence in runners) or nonathletes who suddenly increase their activity.
- Causes: are categorized as: 1. Activity related (e.g., excessive training and improper footwear) 2. Biomechanical (e.g., weak calf muscles, high arched feet) 3. Metabolic (e.g., demineralized bone from hormonal or nutritional diseases)

#### Metatarsal stress fractures

- Typically occur in athletes and military recruits, due to the sudden and drastic increase in activity by the latter.
- **Symptoms & Signs:**
  1. Complaining of slow onset foot pain that initially only occurs with activity but later is present during rest as well.
  2. Point tenderness over the affected metatarsal is present on examination.
- **Treatment:**
  1. Fractures of the second, third and fourth metatarsals are managed conservatively because the surrounding metatarsals act as splints and nonunion is uncommon.
  2. **Rest and pain control are the most appropriate treatment.**

#### Medial tibial stress syndrome

("shin splints" with NO tibial tenderness on palpation)

- Tibia is the major weight-bearing bone in the leg, and patients usually develop medial tibial stress syndrome. This can progress with further activity to a complete or incomplete fracture, resulting in pain to palpation of the tibia.
- **Diagnosis clinically made:**
  1. Pain at a specific area that increases with jumping or running
  2. Is associated with local swelling and point tenderness to palpation
- **Investigations:**
  1. X-rays: frequently normal BUT can reveal periosteal reaction in the site of the fracture.
  2. Injury is best defined radiographically using MRI or bone scan.
- **Treatment:** involves rest and healing of the stress fracture.

---

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### Miscellaneous Disorders

<table>
<thead>
<tr>
<th>Osteomyelitis</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bone infection via</strong></td>
<td></td>
</tr>
<tr>
<td>1. Hematogenous spread or 2. Local extension</td>
<td></td>
</tr>
</tbody>
</table>

- **Clinical features**: Bone pain, tenderness, fever, chills; possible skin involvement with a draining sinus
- **Lab**: X-rays are NOT helpful initially and only show signs of infection after 10 days; MRI: demonstrates bone edema early in disease. Bone scan: will show increased uptake after 72 hr.
- **Treatment**: 1. IV antibiotics for 4 to 6 weeks (empiric initially, pathogen specific following culture); 2. Incision & Drainage: must be performed for an abscess inside the bone (i.e., sequestrum) or in surrounding tissue
- **Complications**: Inadequately treated infection can lead to chronic osteomyelitis

<table>
<thead>
<tr>
<th>Septic arthritis</th>
<th></th>
</tr>
</thead>
</table>

- **Cause**: The disorder is a mechanically induced neuropathic degeneration.
- **Symptoms**: Numbness and burning between 3rd & 4th toes worsened by walking on hard surfaces & wearing tight shoes.
- **Examination**: Clicking sensation when palpatting space between 3rd & 4th toes (Mulder sign) while squeezing metatarsal joints
- **Treatment**: Using metatarsal support with a bar or padded shoe inserts to decrease pressure on the metatarsal head.

→ If conservative treatment fails: surgical treatment

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- **Organisms**: 1. In both infants and children → *Staphylococcus aureus* is the most common.
  2. Other common organisms include: 1) Group B streptococcus & Escherichia coli in infants,
  2) Streptococcus pyogenes in children.
- **Clinical features**: Bone pain, tenderness, fever, chills; possible skin involvement with a draining sinus
- **Lab**: X-rays are NOT helpful initially and only show signs of infection after 10 days; MRI: demonstrates bone edema early in disease. Bone scan: will show increased uptake after 72 hr.
- **Treatment**: 1. IV antibiotics for 4 to 6 weeks (empiric initially, pathogen specific following culture); 2. Incision & Drainage: must be performed for an abscess inside the bone (i.e., sequestrum) or in surrounding tissue
- **Complications**: Inadequately treated infection can lead to chronic osteomyelitis

---

### Trochanteric Bursitis

- It is inflammation of the bursa surrounding the insertion of the gluteus medius onto femur’s greater trochanter.
- **Cause**: Excessive frictional forces secondary to overuse, trauma, joint crystals, or infection.
- **Symptoms**: Complaining of hip pain when: 1. Pressure is applied (as when sleeping on the effected side) and 2. External rotation or resisted abduction

### Anserine Bursitis

- Anserine bursa is located: anteromedially over the tibial plateau just below the joint line of the knee.
- **Cause**: result of an abnormal gait, overuse or trauma.
- **Symptoms**: Patients typically complaining of:
  - Localized pain over the anteromedial tibia
  - Pain is often present overnight as pressure from knees making contact with one another while patient lies on their side can exacerbate pain.
- **Examination**: Reveals a well-defined area of tenderness over the medial tibial plateau just below the joint line
- **D.D.:** Medial collateral ligament injury → "Valgus stress test" does NOT aggravate the pain indicating that MCL is absent.
- **Treatment**: 1. Rest, Ice and maneuvers to reduce pressure on bursa. 2. Corticosteroid injections into the bursa are also helpful.

### Morton neuroma

- Occurs in runners (not a true neuroma)
- **Symptoms**: Numbness and burning between 3rd & 4th toes worsened by walking on hard surfaces & wearing tight shoes.
- **Examination**: Clicking sensation when palpatting space between 3rd & 4th toes (Mulder sign) while squeezing metatarsal joints
- **Treatment**: Using metatarsal support with a bar or padded shoe inserts to decrease pressure on the metatarsal head.

→ If conservative treatment fails: surgical treatment

### Tarsal tunnel syndrome

- Involves compression of the *tibial nerve* as it passes through the ankle.
- **Cause**: it is usually caused by a fracture of the bones around the ankle.
- **Symptoms**: Burning, numbness, and aching of the distal plantar surface of the foot or toes that radiate up to the calf.
Risk factors:
- Adhesive capsulitis
- Head of the biceps
- Rotator Cuff Tear
- Osteoarthritis
- Acromegaly
- Thoracic outlet syndrome
- Rupture of the biceps
- Epicondylitis
- De Quervain tenosynovitis
- Subacromial bursitis
- Spondylosis
- Thoracic outlet syndrome
- Adhesive capsulitis

Symptoms:
- Typically 1. Pain of the neck & arm → Movement at the neck exacerbates symptoms
- 2. Paresthesias of the arm (present in 80% of patients)
- 3. Weakness may affect the shoulder, elbow or wrist, depending on the cervical root(s) involved.

History of chronic neck pain is typical.

Symptoms:
1. Motor: Limited neck rotation and lateral bending is due to osteoarthritis and Secondary muscle spasm.
2. Sensory deficit: decreased pinprick sensation on the posterior aspect of the right forearm is due to osteophyte-induced radiculopathy.

Other findings during cervical spondylosis may include: narrowing of the disk spaces and hypertrophic vertebral bodies.

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Serious Complications of fractures:

1. **Fat embolism**: may occur from 12 to 72 hours after the injury. Common in patients with polytrauma, especially with multiple fractures of long bones.

   - **Characterized by**:
     1. CNS dysfunction: initially as confusion & agitation, may progress to stupor, seizures, or coma.
     2. Severe respiratory distress + tachycardia, tachypnea, FEVER
     3. Petechial rash
     4. Subconjunctival hemorrhage

   - **Diagnosis is confirmed by**:
     1. Presence of fat droplets in urine or
     2. Presence of intra-arterial fat globules on fundoscopy.

   - **Other manifestations**:
     1. Thrombocytopenia, anemia
     2. Hypofibrinogenemia

   - **Serial X-ray**: shows increasing diffuse bilateral pulmonary infiltrates within 24-48 hours of onset of clinical findings.

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

2. **Compartment syndrome**:

   - **Causes**:
     1. Direct trauma (hemorrhage)
     2. Prolonged compression of extremity
     3. After revascularization of acutely ischemic limb

   - **Muscles of the extremity are encased in fascial compartments** that do not allow for expansion of tissue.

   - Blood flow at the capillary level relies on the pressure difference between the arterial and venous systems.

   - **Hemorrhage or edema within muscle causes increased pressure** within the non-distensible fascial compartment.

   - **This increased pressure interferes with perfusion** by disallowing passage of blood from the arterial system into the capillary beds of the affected muscles. This eventually leads to **muscle necrosis**.

   - **Symptoms**: Injury to limb + severe pain that is worsened on Passive Motion = Compartment Syndrome.

     - Aesthenias, Pallor, Paralysis, Pulseless of the affected limb.

   - **Investigations**: Pressure in the compartments can be measured directly using a needle and pressure transducing catheter system.

     - **Pressures over 30 mmHg** may result in cessation of blood flow through the capillaries.

     - **should be treated emergently by fasciotomy**. Then the leg is always elevated as well.

   - **Complications**: Volkmann’s ischemic contracture final sequel of compartment syndrome in which dead muscle has been replaced with fibrosis.

   - If compartment syndrome has been ruled out and the leg swelling is due to edema → then elevation & ice.
Pediatrics Orthopedics

Slipped Capital Femoral Epiphysis (SCFE)
- It is commonly seen in obese adolescent boys
- Risk factors:
  - Multiple gestation
  - White ethnicity
  - Family history
- Symptoms:
  - Typically present with insidious onset of:
    1. Hip pain
    2. Knee pain (referred pain)
- Treatment:
  - Surgical pinning of the slipped epiphysis
- Investigations:
  - Hip X-ray: Frog-leg, lateral is diagnostic imaging of choice

Acetabular dysplasia of the Hip (DDH)
- Physical examination:
  1. Inspection (A/P in-Ex)
    - Normally : there is smooth abduction to almost 90 degrees.
    - May be limited abduction (acetabular dysplasia) or absence of abduction.
  2. Palpation
    - Vascular sign: No femoral pulsation (because femoral artery is normally felt over the femoral head).
- Treatment:
  - Pavlik harness for 1-2 months: a splint that holds the hip in flexion and abduction while preventing extension and adduction, which can exacerbate dislocation.
- Complications:
  - 1. Acetabular dysplasia
  - 2. Leg length discrepancy
  - 3. Trendelenburg gait
  - 4. Scoliosis
  - 5. Arthritis
  - 6. AVN

Legg-Calve-Perthes Disease
- It is characterized by displacement of the femoral head on the femoral neck due to disruption of the proximal femoral growth plate.
- Mechanism:
  - The physis (i.e. physical junction between the femoral head and neck) weakens during early adolescence because it is rapidly expanding and primarily composed of cartilage, which does not possess strength of bone.
  - When exposed to excessive shear stress (which is magnified by obesity) → The physis fractures and the femoral head slips posteriorly and medially relative to the femoral neck.
- Symptoms:
  - Typically present with insidious onset of:
    1. Hip pain
    2. Knee pain (referred pain)
- Treatment:
  - Idiopathic AVN of capital femoral epiphysis most common: 3 - 8 years of age, more in males (20% bilateral)
  - X-ray: shows 1) Asymmetric hips
  - Treatment: 1. Containment of hip within acetabulum via bracing or surgical means

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Transient Synovitis

- Most common cause of hip pain in children
- Typically occurring in boys age 2-10 years

Clinical Features

- Leg Pain ONLY in night +
- Typical:
  - Patellar tendinitis
  - Patellofemoral syndrome (overuse injury)
  - Osteochondritis dissecans
  - Patellofemoral compression test

Diagnosis

- Normal physical & laboratory findings
- Decreased inflammation
- Decrease in range of motion
- Pending if the physical exam is unremarkable.
- X-rays may reveal mild adduction of the metatarsals at the tarsometatarsal articulation,
  reveal mild adduction of the metatarsals at the tarsometatarsal articulation,
  demonstrate separation at the
  reveal mild adduction of the metatarsals at the tarsometatarsal articulation,
  demonstrate separation at the
  reveal mild adduction of the metatarsals at the tarsometatarsal articulation,
  demonstrate separation at the

Differential Diagnosis of anterior knee pain in young patient

- Patellar tendinitis
- Patellofemoral syndrome
- Osteochondritis dissecans
- Patellofemoral compression test

Radiographic

- DO NOT demonstrate separation at the tibial tubercle.

Treatment

- Traction symptoms of tibial tubercle
- Fragmentation of tibial tubercle

Metatarsus Adductus

- Congenital foot deformity
- Normal
- Metatarsus Adductus

- Interestingly, deformity is most frequent in first-born infants; this is attributed to molding effect of the primigravid uterus
- Examination of the newborn: reveals deformity of the feet : Adduction of the anterior aspect of the foot with a convex lateral border and concave medial border
- Ankle movements are normal, and passive and active movement of the foot overcorrects the deformity into abduction. About 10% of patients also have an associated acetabular dysplasia
- AP radiographs: reveal mild adduction of the metatarsals at the tarsometatarsal articulation, and an increased angle between the 1st and 2nd metatarsals.
- Metatarsus adductus is subdivided into three types:

<table>
<thead>
<tr>
<th>Type I</th>
<th>Type II</th>
<th>Type III</th>
</tr>
</thead>
<tbody>
<tr>
<td>feet that overcorrect both passively and actively into abduction</td>
<td>feet that correct to the neutral position with passive and active movements</td>
<td>feet and do not correct</td>
</tr>
<tr>
<td>Managed with:</td>
<td>Managed with: serial casts</td>
<td>Surgical treatment may be required if there is significant residual metatarsus adductus in children 4 years of age.</td>
</tr>
<tr>
<td>1- Orthosis or corrective shoes</td>
<td>1- Orthosis or corrective shoes</td>
<td>1- Orthosis or corrective shoes</td>
</tr>
<tr>
<td>2- If no results ⇒ plaster casts</td>
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</tr>
</tbody>
</table>

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### Nursemaid's elbow

- **Definition:** 1. Equinus and Varus of the calcaneum  
  2. Talus, varus of the midfoot  
  3. Adduction of the foot  

- **Causes:** 1. Congenital: are usually isolated, idiopathic cases  
  2. Teratologic: are associated with a neuromuscular disorder or a complex syndrome  
  3. Positional: occur due to abnormal positioning of the affected foot in utero.

- **Treatment:**
  1. Initially, **Conservative treatment:** stretching and manipulation of the foot, followed by serial plaster casts  
  2. **Operative management:** is indicated if conservative management gives unsatisfactory results:
     - It is preferably performed between 3 and 6 months of age, but always before 12 months.

- **Complications:**
  - Untreated cases result in further deformation, abnormal gait, and development of ulcerations.

### Common Brachial Plexus Disorders

<table>
<thead>
<tr>
<th>Condition</th>
<th>Cause of Injury</th>
<th>Clinical Features</th>
</tr>
</thead>
</table>
| Complete brachial plexus injury | Injury causes damage to all nerves | Motor: affects all muscles of upper limb except trapezius  
Sensory: loss of deep sensation of whole upper limb except face  
Medical side of arm: supplied by intercosto-brachial n.(T2)  
Skin over upper deltoid muscles: supplied Supraclavicular is (C4) |
| Erb-Duchenne palsy | Superior trunk (C5 – C6) | Hyperadduction of arm causing widening of the humeral-glenoid gap (e.g., birth, shoulder dystocia)  
Waiter's tip deformity: Arm extended and adducted with pronated forearm |
| Klumpke palsy | Inferior trunk (C8 – T1) | Hyperabduction of arm places excess tension on lower cords and nearby sympathetic chain  
Affects muscles innervated by the ulnar nerve:  
1. **Claw hand** deformity  
2. Atrophy of the hypothenar & interosseous muscles  
3. Association with Horner syndrome due to sympathetic paralysis |
| Deltoid paralysis | Axillary nerve (C5,C6) | Anterior shoulder dislocation causes axillary nerve impingement or stretching  
Motor: Impaired shoulder abduction (15-90 degree) or elevation  
Sensory: Small patch of numbness over deltoid(C5 dermatome) |
| Claw hand | Ulnar nerve (C8, T1) | Epiphysial separation of medial epicondyle of humerus  
Motor:  
1. Atrophy of hypothenar  
2. Atrophy interoseous  
3. Paralysis of medial 2 lumbricals, with intact lateral 2 lumbricals (because they are supplied by median nerve)  
4. Paralysis of adductor pollicis  
Sensory loss of:  
- Autonomo zone: little finger(palmar surface)  
- Medical 1/3 of palm  
- Medial 1 & 1/2 fingers |
| Wrist drop | Radial nerve (C5- C8) | Mid-humerus fracture causes nerve impingement or tear  
Motor:  
- Flexed elbow - Pronated forearm  
- Wrist drop  
Sensory loss of:  
- Autonomo zone: Dorsal skin of 1st web space (coin area) |
Injury to "long thoracic nerve":
- leads to winged scapula due to paralysis of Serratus anterior muscle.
- Most common cause is iatrogenic injury during axillary lymphadenectomy.

Important Nerves of Lower Extremities

### Sciatic Nerve

**Motor:**
1. Paralysis of Hamstrings → weak flexion of knee (some flexion is preserved by Sartorius & Gracilis).
2. Paralysis of all leg muscles → foot drop.
3. Paralysis of intrinsic muscles of foot → clawing of toes.

**Sensory:**
- Loss of sensation below knee (EXCEPT medical side of leg, foot & big toe) supplied by Saphenous nerve "a branch from femoral n."

### Femoral Nerve

**Motor:**
4. Innervates the muscles of the anterior compartment of the thigh, responsible for knee extension & hip flexion.

**Sensory:**
- Provides sensation to:
  1. Anterior thigh
  2. Lower medial leg via the saphenous branch

### Obturator Nerve

**Motor:**
1. Medial compartment of the thigh:
   - gracilis adductor
   - adductor brevis
   - anterior portion of adductor magnus
   - responsible for adduction of the thigh

**Sensory:**
- Provides sensation over the middle medial thigh

### Tibial Nerve

**Motor:**
1. Posterior compartment of the thigh
2. Posterior compartment of the leg
3. Plantar muscles of the foot
   - responsible for:
     1. Flexion of the knee and digits
     2. Plantar flexion of the foot

**Sensory:**
- Provides sensation to:
  1. Anterolateral leg
  2. Dorsum of the foot

### Common Peroneal Nerve

- Injury: Loss of dorsiflexion & eversion.
- These two nerves supply:
  1. Muscles of anterior
  2. Muscles of lateral leg.

**Sensory:**
- Provides sensation to:
  1. Anterolateral leg
  2. Dorsum of the foot

### Damage to Superior Gluteal Nerve

**Normal Physiology:**
- Normally, the gluteus medius & 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.

**Causes of damage:**
- Weakness of these muscles, as can occur in:
  1. Neuromuscular disease
  2. Impingement of or trauma to the superior gluteal nerve
  3. Inflammatory myopathies

- **Results in:**
  1. Positive Trendelenburg sign:
     - a drooping of the contralateral pelvis that occurs when the patient stands on one foot.
  2. Trendelenburg gait:
     - Waddling in quality

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Hemoptysis is defined as any expectoration of blood, with a wide spectrum from minimal blood streaking in sputum to the presence of frank blood and/or clots. Despite the many causes of hemoptysis, pulmonary airway disease ranks as the most common cause.

- **Massive Hemoptysis** — It is defined as:
  - >600 ml of expectorated blood over a 24-hour period or a bleeding rate >100 ml/hour.
  - The greatest danger in massive hemoptysis is asphyxiation due to the airway flooding with blood.

  Initial management involves:
  1. Airway: Establishing an adequate patent airway
  2. Breathing: Maintaining adequate ventilation and gas exchange
  4. The patient should be placed with the bleeding lung in the dependent position (lateral position) to avoid blood collection in the airways of the opposite lung.

Upper GIT bleeding manifest as:

1. **HEMATEMESIS**: (excluding hemoptysis or swallowed blood from epistaxis) is observed during upper gastrointestinal bleeding.
   - Blood or material in the nasogastric lavage tests positive for blood.
   - The aspirate will be negative in about 10% of patients with duodenal source of GI hemorrhage.
   - A duodenal source cannot be excluded unless gastric lavage contents reveal bile.
   - Even if bile is returned, the bleeding may have resolved spontaneously prior to arrival.
   - If a patient reports unwitnessed hematemesis & gastric lavage is inconclusive → consultation with a gastroenterologist for early endoscopy.

2. **MELENA AND HEMATOCHIEZA**:
   - Melena is usually due to bleeding from an upper GI source above "ligament of Treitz"
   - Hematochezia from an upper source usually indicates severe hemorrhage and corresponds with significant increases in mortality.

3. **ABSENCE OF BLEEDING**: If nasogastric lavage reveals BILE and NO blood, then active bleeding from an upper GI source is less likely.

---

**Esophageal Varices**

<table>
<thead>
<tr>
<th>Non bleeding varices</th>
<th>Managed with nonselective B-Blockers: as Propranolol. Such therapy can reduce the risk of bleeding by up to half.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding varices</td>
<td>A) Endoscopic: 1. Band ligation 2. Sclerotherapy</td>
</tr>
<tr>
<td></td>
<td>B) TIPS ➔ Portosystemic shunt connects the portal venous system to the systemic venous system.</td>
</tr>
<tr>
<td></td>
<td>C) Surgically ➔ Portosystemic shunt</td>
</tr>
<tr>
<td></td>
<td>These procedures are considered a last resort in variceal bleeding unresponsive to medical and endoscopic interventions and may worsen the encephalopathy in patient.</td>
</tr>
</tbody>
</table>
Lower GI bleeding manifest as:

1. **HEMATOCEZIA:**
   - Hematochezia due to:
     1. Lower GI bleeding (distal to Ligament of Treitz) “Usually”
     2. Most common causes of acute lower GI bleeding (LGIB) in patients over 50 years old are:
        1. Diverticulosis
        2. Vascular (angiodysplasia, ischemia)
        3. Infectious
        4. Neoplasms

2. Upper GI source in up to 15% of patients presenting with hematochezia. In these instances, consider:
   - 1. Aortoenteric fistula (in patients with abdominal aortic aneurysm repair) or
   - 2. Duodenal ulcer

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

**Colon cancer**
- Tends to present with chronic occult blood loss. Gross bleeding is less likely.
## Esophagus

**Boerhaave Syndrome**

- **Spontaneous rupture (perforation) of the esophagus** can occur during episodes of vomiting, particularly when the patient is resisting the vomiting reflex.
- **Cause:** This results because high intraabdominal pressures are transmitted into the mediastinal esophagus where the transmural difference in pressure is large due to negative intrathoracic pressure.
- **Site:** Esophageal rupture occurs a few centimeters above the gastroesophageal junction.
- **Symptoms:** **Pneumomediastinum**, leads to: 1. Severe retrosternal pain + Crepitus in the suprasternal notch.
- **Examination:** 1. subcutaneous emphysema in the neck 2. Characteristic crunching sound on auscultation of heart due to mediastinal emphysema (i.e. Hamman sign)
- **Investigations:**
  A) Chest X-ray: 1. Air in the paraspinous muscles 2. widened mediastinum, 3. pneumomediastinum, or 4. pleural effusions with or without pneumothorax (usually hours to days later).

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### Esophageal Achalasia

- Most common motility disorder

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Widening of the gastric outlet or hiatal hernia</td>
<td>Antacids as per GERD</td>
</tr>
<tr>
<td>Type II</td>
<td>Herniation of the stomach fundus through the diaphragm parallel to the esophagus</td>
<td>Surgical repair due to risk of strangulation</td>
</tr>
<tr>
<td>Type III</td>
<td>Herniation of the stomach fundus as well as sliding of the gastroesophageal junction above the diaphragm</td>
<td>Surgical reduction and repair</td>
</tr>
<tr>
<td>Type IV</td>
<td>Herniation of abdominal organs (e.g. spleen, intestines, liver, pancreas) above the diaphragm</td>
<td>Surgical reduction and repair</td>
</tr>
</tbody>
</table>

- **Loss of esophageal motility & failure of lower esophageal sphincter (LES) relaxation** → results in the dilatation of the proximal esophagus
- **Causes:** may be caused by 1. Ganglionic degeneration or 2. Chagas disease
- **Symptoms:** **Dysphagia of liquids first** then to both solids & liquids + weight loss + repulsion of undigested foodstuffs.
- **Risk of:** Esophageal Cancer because stasis promotes development of Barrett esophagus
- **Investigations:**
  1. Barium swallow: 1) Dilatation of the proximal esophagus with subsequent narrowing of the distal esophagus 2) may esophageal diverticula
  2. Manometry: ↑ LES pressure and failure to relax
- **Treatment:**

### Esophageal Diverticula

- **Pulsion diverticula involving only the mucosa, located between the thyropharyngeal and cricopharyngeus muscle fibers** (condition associated with muscle dysfunction/spasms)
- **Symptoms:** **Dysphagia, Regurgitation of solid foods,** Choking, Left sided neck mass, and bad breath
- **Diagnosis:** Clinically + Barium swallow
- **Treatment:** Myotomy of cricopharyngeus muscle and removal of diverticulum

### Esophageal Tumors

- **Squamous Cell CA** → Most common / alcohol and tobacco synergistically ↑ risk of development/ seen in men in 6th decade
- **Adenocarcinoma** → Seen in patients with chronic reflux → Barrett’s esophagus → squamous to columnar metaplasia
- **Symptoms:** Dysphagia, weight loss, hoarseness, tracheoesophageal fistula, recurrent aspiration
- **Investigation:** Barium study demonstrates classic **apple-core lesion** / Confirmed with endoscopy with biopsy, CT of abdomen
- **Treatment:** Esophagectomy with gastric pull-up or colonic interposition with or without chemotherapy/radiation
### Stomach

#### Gastric Atresia
- Failure of recanalization of foregut → esophageal atresia
- Failure of separation between esophagus & trachea → TrechoEsophageal fistula.

##### Symptoms:
1. Suction of neonate → Excessive frothy saliva.
2. Reflux of contents into the trachea through the fistula → aspiration → bronchopneumonia → Cyanosed with Choking
3. Arrest of Ryle tube 10 cm from the nostril (due to atresia).

##### Investigations:
1. Plain X-ray (lateral view):
   - Keep the Ryle tube placed and take x-ray film, it will shows arrest of the metallic end of the tube due to blind pouch, with air in pouch.
   - IF fundic air bubble → fistula of lower pouch
   - IF absent air bubble → blind lower pouch
2. Lipidol study: Will determine the type of the esophageal atresia.

##### Treatment:
- Excision of the fistula between esophagus & trachea, and then restore the continuity of the fistula (end to end anastomosis) either by 3-layer or 2-stage procedure.

#### Gastric Tumors
- Benign tumors
- Malignant Tumors

##### Risk factors:
- H. pylori: 2
- Alcohol intake: 2
- Smoking: 2
- Manic depressive illness: 3
- Urologic: 1
- Abdominal scars: 1
- Crohn disease: 1

##### Associated syndromes:
- Familial adenomatous polyposis: 1
- Turcot's syndrome: 1
- Peutz-Jeghers syndrome: 1
- Cronkite-Newton syndrome: 1

##### Several classic physical findings in metastatic gastric cancer:
1. Virchow's node = large rock-hard supraclavicular node
2. Krukenberg tumor: mucinous, signet-ring cells that metastasize from gastric CA to bilateral ovaries, palpate for ovarian masses in women
3. Sister Mary Joseph sign: metastasis to umbilicus, feel for hard nodule there, associated with poor Px
4. Blumer's shelf = palpable nodule superiorly on rectal exam.

##### History:
- Weight loss, anemia, anorexia, GI upset

##### Treatment:
- Upper GIT endoscopy with biopsy
- Mostly palliative: combination surgery and chemotherapy when tolerated

##### Prognosis:
- About 5% survival at 5 yrs

### Dumping syndrome

- It is a common *postgastrectomy complication*.
- Pathophysiology:
  - Involves rapid emptying of hypertonic gastric content into the duodenum and small intestine.
  - This process leads to:
    1. Fluid shift from intravascular space to the small intestine
    2. Release of intestinal vasoactive polypeptides
    3. Stimulation of autonomic reflexes
  - This leads to *postprandial (25-30 minutes after eating)*:
    1. Abdominal cramps
    2. Weakness
    3. Light-headedness
    4. Diaphoresis

- The diagnosis is made clinically / 1- Provocative tests & 2- Contrast x-ray demonstrates rapid gastric emptying

##### Treatment:
- *Dietary changes are helpful* to control the symptoms (Symptoms usually diminish over time)
- In resistant cases, *Octreotide* should be tried.
- In intractable cases, *Reconstructive surgery*
**Gastric Outlet Obstruction in Pediatrics**

**Definition:**
Thickening of muscles at the pyloric canal > 4 mm (n: 4 mm) resulting in lumen collapse.

**Clinical picture:**

- **Symptoms:**
  1. Neonate, Female patient (female to male ratio 4:1)
  2. Aged from 2 to 4 weeks (extreme is from 1 to 8 weeks)
  3. With projectile non-bilious stained vomiting (only milk) of nearly every day.
  4. Failure to thrive: progressive weight loss & dehydration.

- **Signs:**
  1. General → Weight loss (Failure to thrive) + dehydration + Aspiration (bad chest)
  2. Local → may palpable "Olive like lump" in Rt. Hypochondrium + visible peristaltic waves in the upper abdomen

**Investigation:**

1. **Abdominal Ultrasound:** (sensitivity > 98%) → MOST useful tool for confirming the diagnosis
   - Muscle thickness > 4 mm - Pyloric canal Length > 16 mm
   - If U/S (Abdominal) not diagnostic → Barium meal, Gastrographin: → dilated stomach
   - Lab: Hypochloremic Metabolic Alkalosis

2. Surgery is performed once the electrolyte levels normalize → Ramstedt's Pyloromyotomy
3. If U/S is equivocal → Barium Study: → MOST diagnostic
   - Barium Meal: → passes of blood stained stools
   - Abdominal Ultrasound: → Gastric & Duodenal distention.
   - Ladd's bands (peritoneal bands) attach caecum & duodenum & liver

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### Differential Diagnosis

**Intussusception**
- **Cause:** Jejunal atresia is thought to occur due to an intrauterine vascular accident that causes necrosis and resorption of a segment of bowel.
- **Severity:** Obstruction varies from membranous web to full atresia and loss of bowel length.
- **Prenatal diagnosis:** Prenatal ultrasound can diagnose intestinal atresia.
- **Symptoms:** If not discovered prenatally, the infant will develop shortly after birth:
  - Vomiting & abdominal distention along with feeding intolerance
- **Investigations:** Radiography can usually confirm the diagnosis by showing:
  - Air-fluid levels with a gasless lower abdomen
  - Triplet bubble signs on abdominal radiographs, indicating dilation of a larger amount of small bowel than that seen in duodenal atresia.
- There is only 10% incidence of associated anomalies.
- **Treatment:** 1. Initially be focused on resuscitation and stabilization of the patient, 2. Followed by surgical correction.
- **Prognosis:** A surgical repair must be considered in the length of affected bowel as well as the birth age and weight of the infant.
- **Ileocolic junction** is most involved with invagination of ileum into the colon.
- **Complications:** Heterotropic gastric tissue may be present in the diverticulum, which results in ulcerations & bleeding.
- **Investigation:** Diagnosis is best made with 99m pertechnetate scanning (uptake by heterotopic gastric mucosa).

**Meckel’s diverticulum**
- **Cause:** It is present in 2-3% of the population and is the most common anomaly of the gastrointestinal tract.
- **Symptoms:** Typically presents with hematochezia in 2 to 3 year-old children.
- **Complications:** Heterotopic gastric tissue may be present in the diverticulum, which results in ulcerations & bleeding.
- **Investigation:** Diagnosis is best made with technetium-99m pertechnetate scanning (uptake by heterotopic gastric mucosa).

### Luminal causes:

**Meconium ileus**
- Meconium is as thick as glue and difficult to propel, resulting in impaction in the ileum and a narrow, underused colon (microcolon).
- It occurs in 10% in patients with Cystic Fibrosis.
  - Meconium ileus can be the earliest manifestation of cystic fibrosis due to abnormal chloride transport that results in tenacious secretions in multiple organs, including the intestines.
- **Symptoms:** Persistent vomitings. Don’t confuse this with duodenal atresia.
- **Examination:** reveals a crying infant with a distended abdomen.
- **Radiographic features:**
  - X-ray: multiple dilated loops of small bowel and a granular, ground glass appearance in lower abdomen
  - Contrast enema: showing microcolon
- **Treatment:** Ileostomy may be needed & may life saving.
Large Intestine

Mural causes:
- **Cause:** Results from failed development of the enteric nervous system of a variable portion of the distal gut
- Most commonly involves the rectosigmoid. The affected colonic segment cannot relax and therefore is chronically contracted.
- Hirschsprung disease should be suspected in any neonate who has delayed passage of meconium within 48 hours of birth.
- It is strongly associated with Down syndrome.

**Symptoms:**
1. Should be suspected in any neonate who has delayed passage of meconium within 48 hours of birth
2. Poor feeding, even biliary emesis

**Signs:**
1. Signs: (of neonatal intestinal obstruction due to meconium impaction):
   - Inspection → abdominal distention
   - DRE → produce an explosive expulsion of gas and stool ("squirt sign") from temporary relief from the obstruction

**Complications:**
1. General → Delayed growth & development (failure to thrive)
2. Local → Obstructive toxic enterocolitis (fever, diarrhea & abdominal distention)

**Investigations:**
1. Imaging:
   - Abdominal x-ray: Large bowel obstruction (haustrations don’t reach the other side of lumen).
   - In toxic enterocolitis perforation → air under diaphragm.
   - Unprepared Barium enema: Narrow aganglionic segment → transition zone → with marked proximal colon dilatation (megacolon).
   - Rectal biopsy: (Gold standard for diagnosis) which demonstrates the absence of ganglion cells.

**Treatment:**
- Surgical resection of the aganglionic segment followed by anastomoses of the normal bowel to the anus.

Meconium plug syndrome
- Distal colonic obstruction associated with narrow left colon & the failure of meconium in the distal colon to pass through the pelvic floor → not a similar presentation to Meconium ileus.
- Has a similar presentation to Meconium ileus, but occurs in patients with Meconium ileus.
- Gastroschisis entity is usually corrected.

Anus

**Etiology:** Failure of fusion between proctodeum & posterior allantoic duct

**Types:** According to termination of bowel:
1. High anomalies (more common) → above levator ani → No sphincter + Fistula (worse prognosis)
   - Failure of the rectum to pass through the pelvic floor (proximal to pelvic floor)
   - Associated with: 1. Urinary tract fistula 2. Defective anal sphincter 3. Other anomalies (VACTREL)
   - Types: 1. Anorectal agenesis 2. Rectal agenesis 3. Persistent cloaca
   - Treatment: Delay repair until further growth (But before toilet training time)
2. Low anomalies (less common) → Below levator ani → No Fistula
   - Distal to the pelvic floor.
   - Not Associated with: Fistula, Defective anal sphincter, other anomalies.
   - Types: 1. Covered anus 2. Membranous anus
   - Treatment: a colostomy needs to be done for high rectal pouches.

**Differential diagnosis of delayed passage of meconium**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Level of obstruction</th>
<th>Meconium consistency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hirschsprung</td>
<td>Rectosigmoid</td>
<td>Normal</td>
</tr>
<tr>
<td>Meconium ileus</td>
<td>Ileum</td>
<td>Inspissated</td>
</tr>
</tbody>
</table>

**Differentiating features of Hirschsprung disease & meconium ileus**

<table>
<thead>
<tr>
<th>Associated Disorder</th>
<th>Hirschsprung disease</th>
<th>Meconium ileus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Down syndrome</td>
<td></td>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td>Typical level of obstruction</td>
<td>Rectosigmoid</td>
<td>Ileum</td>
</tr>
<tr>
<td>Meconium consistency</td>
<td>Normal</td>
<td>Inspissated</td>
</tr>
<tr>
<td>&quot;Squirt sign&quot;</td>
<td>Positive</td>
<td>Negative</td>
</tr>
</tbody>
</table>

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Gastroschisis
- Defect is to the right of the cord
- Covering: No protective membrane
- Umbilical cord: normal (it reaches the baby)
- Contains: Bowel
- Basic Science: (reading)
  1. Cause: Incomplete fusion during the fourth week of development results in a defect that allows abdominal viscera to protrude through the anterior abdominal wall, which is made when the lateral body folds move ventrally and fuse in midline.
  2. Gastroschisis occurs when the neural crest fails to migrate, resulting in the absence of enteric neurons within the myenteric plexus & submucosal plexus.

Omphalocele
- Defect: Midline abdominal wall defect
- Covered by: Peritoneum
- Umbilical cord: Inserts at the apex of the defect
- Contains: Multiple abdominal organs
- Occurs with: 1. Edward’s Syndrome 2. Patau Syndrome

Treatment:
1. Small defects: Close small defects primarily
2. Large defects:
   - These require silastic "siloh" to protect the bowel and manual replacement of the bowel daily until complete closure (in about 1 week)
   - In the mean time give: 1. Parenteral nutrition (bowel will not work in gastroschisis) 2. and IV antibiotics

Umbilical Hernia
- Appears as soft swelling covered by skin that protrudes during crying, coughing or straining, and can be reduced easily
- Defect: at the linea alba (Due to imperfect closure or weakness of the umbilical ring)
- Covered by: Skin
- Umbilical cord: Inserts at the apex of the defect
- Contains: Omentum or portions of the small intestine

Treatment:
- Most disappear within 1 year.

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Choledochal Cyst

A Choledochal Cyst is a congenital abnormality of the biliary ducts characterized by the dilatation of intra or extra-hepatic biliary ducts or both.

Origin: It has a multifactorial origin, but most of the cases are related to the anomalous pancreaticobiliary junction, which leads to:
- weakness and dilatation of the biliary wall due to the flux of alkaline pancreatic secretions into the biliary tree.

Types:
- Type 1 (Most common type) features the dilatation of the entire common hepatic and common bile ducts or segments of each.
- Type 2: Relative isolated protrusions or diverticulae from the common bile duct wall.
- Type 3: Cyst found in the intraduodenal part of common bile duct.
- Type 4: Multiple dilatations in the intra and extra hepatic biliary tree.
- Type 5: Isolated dilatation of intrahepatic bile ducts.

Clinical presentations: varies with the age
1. In infants, it may present with jaundice and the passage of acholic stools.
2. In children, it causes abdominal pain, jaundice, and attacks of recurrent pancreatitis (the amylase & lipase levels).
3. In adults, it can present with vague epigastric or right upper quadrant abdominal pain or cholangitis.

Complications: Choledochal cysts can degenerate into cholangiocarcinoma.

The initial investigation of choice is an ultrasonogram followed by CT scan or MRI as needed.

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The 4 main causes of acute abdomen are:
1. Perforation
2. Obstruction
3. Inflammation/infection
4. Ischemia

When "Surgical Treatment" is the answer:
1. Peritonitis (excluding primary peritonitis)
2. Abdominal pain/tenderness + signs of sepsis
3. Acute intestinal Ischemia
4. Penumoperitoneum

When "Medical Treatment" is the answer:
1. Primary peritonitis
2. Pancreatitis
3. Cholangitis \(\rightarrow\) is Medical Emergency, and ERCP intervention is treatment of choice
4. Urinary Stones
5. Ruptured ovarian cyst
6. Non-Surgical Causes:
   - Lower Lobal Pneumonia
   - Inferior MI
   - Pulmonary Embolism

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