STUDY PDF

General Surgery

Created from all General Surgery lecture slides and past paper questions, organized into seven rotation notes

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General

Exam Map

PRIORITY LEVEL	DOMINANT EXAM TOPICS	CORE FOCUS AREAS & ALGORITHMS
Tier 1 (Highest)	**Trauma & Shock states**	ABCDE priorities, ATLS Shock Classes (I–IV), Hemodynamic profiles (CO/SVR/CVP), Tension PTX vs. Tamponade.
Tier 2	**Metabolism & Fluids**	Wound healing biochemistry, TPN/EN rules, 100-50-20 rule, Fluid choice based on acid-base status.
Tier 3	**SSI & Complications**	Wound classification (I–IV), Abx prophylaxis timing, 6 Ws of post-op fever, recognizing fascial dehiscence.
Tier 4	**Sepsis & Bleeding**	Sepsis-3 (SOFA/qSOFA) criteria, Hour-1 bundle, Massive Transfusion Protocol (1:1:1), Transfusion reactions.
Tier 5	**Preoperative Care**	Cardiac risk indices, ASA grades, assessing highest mortality predictors (decompensated CHF).

- **ATLS Hemorrhage Classification:** Memorize the hemodynamic shifts. Blood pressure *only* drops at Class III (30–40% loss, 1500–2000 mL). Classes I & II feature normal blood pressure with increasing tachycardia.
- **Hemodynamic Profiling:** You must be able to differentiate shock types. High CO + Low SVR = Septic/Distributive; High CVP/PCWP + Low CO = Cardiogenic; Hypotension + Bradycardia + Warm Skin = Neurogenic.
- **Sepsis-3 & SOFA Criteria:** Know the 6 exact systems of SOFA (Respiratory, Liver, Kidneys, Brain, Cardiovascular, Platelets—*there is no RAAS/endocrine component*). Septic shock strictly requires vasopressors to maintain MAP ≥ 65 *and* Lactate > 2 mmol/L despite fluid resuscitation.
- **Trauma Primary Survey Decision Points:** Airway/Intubation (GCS ≤ 8) trumps all other operative interventions (including severe maxillofacial fractures). For abdominal trauma, FAST is strictly for the unstable patient; CT is the gold standard for the stable patient.
- **Surgical Site Infection (SSI) Rules:** Prophylactic antibiotics must be given within 60 minutes of incision, re-dosed during prolonged surgeries, and definitively stopped within 24 hours. *Exam trap: Continuing Abx for 2–3 days post-op is always wrong and promotes C. diff.*
- **SSI Wound Classification:** Classify to determine Abx need. Herniorrhaphy = Class I (Clean/No Abx unless mesh is used); Elective Cholecystectomy = Class II (Clean-contaminated); Ruptured Diverticulitis = Class IV (Dirty).
- **Post-Operative Fever Timeline:** Strictly apply the "6 Ws". Fever at 24–48h = Atelectasis (Wind); Day 3 = UTI (Water); Days 5–7 = SSI (Wound) or DVT (Walking).

- **Fluid & Acid-Base Traps:** Upper GI losses (Vomiting/NGT) cause hypochloremic metabolic alkalosis (Treat: NS + KCl). Lower GI losses (Diarrhea) cause normal anion gap metabolic acidosis (Treat: Ringer's Lactate).
- **Transfusion Complications:** Platelets carry the highest risk of bacterial contamination because they are stored at room temperature. Massive PRBC transfusions predictably cause Hypocalcemia (due to citrate toxicity) and Dilutional Thrombocytopenia.
- **Metabolic Substrates & Healing:** The brain and RBCs are obligatory glucose users (requiring ~100g/day); cardiac muscle relies on free fatty acids. Vitamin C is highly tested as the mandatory cofactor for the *hydroxylation* of proline/lysine in collagen synthesis.
- **Preoperative Risk:** Decompensated congestive heart failure is universally tested as the single highest predictor of perioperative mortality, surpassing a recent MI or advanced age.

Trauma management

Core Concepts

- **Trimodal Distribution of Trauma Deaths:**
  - **Peak 1 (Immediate - 50%):** Seconds to minutes (e.g., massive brain/spinal cord injury, cardiac/great vessel rupture).
  - **Peak 2 (Early - 30%):** Minutes to hours. Known as the **"Golden Hour"** (e.g., epidural/subdural hematoma, hemopneumothorax, ruptured spleen/liver). Rapid trauma care yields the greatest impact here.
  - **Peak 3 (Late - 20%):** Days to weeks (e.g., sepsis, multi-organ failure).
- **Mechanisms of Injury:**
  - **Blunt:** Involves compression (crushing tissues), shear (acceleration/deceleration tearing the aortic isthmus), and overpressure (blow-out of closed spaces like the diaphragm or bladder).
  - **Penetrating:** Small bowel is the most commonly injured organ in high-velocity penetrating trauma transversing the mid-abdomen (umbilicus).
- **Hemostasis following Vascular Injury:** The immediate, first response is reflex vasoconstriction, which slows blood flow to allow primary (platelet) and secondary (coagulation cascade) hemostasis.
- **Metabolic Response to Trauma:**
  - Induces a profound hypermetabolic and catabolic state driven by counter-regulatory hormones (catecholamines, cortisol, glucagon).
  - Promotes massive lipolysis, hypercatabolism, and glycogenolysis/gluconeogenesis, reliably producing **hyperglycemia** (never hypoglycemia).
  - **Skeletal muscle** is the primary source of protein for catabolism.
  - *Note:* Severe major burns (>40-50% TBSA) induce the most massive increase in basal metabolic needs, surpassing multi-trauma or severe pancreatitis.

Diagnosis / Clinical Features

- **Tension Pneumothorax:** Diagnosed clinically by the triad of hypotension, tachycardia, and a hyper-resonant lung field, often with absent breath sounds and tracheal deviation. If intubated, presents with sudden high airway pressures.
- **Cardiac Tamponade:** Presents with **Beck's triad** (hypotension, distended neck veins/elevated JVP, and muffled heart sounds), frequently accompanied by pulsus paradoxus.
- **Flail Chest:** Results from fractures of ≥2 ribs in ≥2 places. Presents with paradoxical chest wall movement (sinks inward on inspiration) and underlying pulmonary contusion.
- **Hypovolemic/Hemorrhagic Shock:**
  - The venous system is the primary capacitance reservoir, holding 60-70% of resting blood volume.
  - Tachycardia (HR > 100) is the earliest sign.
  - A palpable femoral pulse implies SBP > 80 mmHg; radial implies SBP > 90 mmHg; carotid implies SBP > 60 mmHg.
- **Acute Compartment Syndrome:**
  - Most frequently affects the fascial compartments of the lower leg following a tibial fracture.
  - Classic hallmark: Excruciating progressive pain out of proportion to injury, unresponsiveness to IV narcotics, pain on passive stretch, and a tense/swollen limb.
- **Abdominal Compartment Syndrome (ACS):** Defined strictly as a sustained intra-abdominal pressure (IAP) **> 20 mmHg** directly accompanied by new-onset organ dysfunction (e.g., oliguria).

Investigations

- **Focused Assessment with Sonography for Trauma (FAST):**
  - Evaluates 4 dependent regions: RUQ (Morrison's pouch), LUQ (Splenorenal recess), Sub-xiphoid (pericardial space), and Pelvic (Rectovesical/Pouch of Douglas).
  - Excellent for rapidly identifying free fluid (blood) at the bedside without patient transport. Can be repeated easily.
  - **Major limitation:** It cannot reliably identify the specific solid organ that is injured.
- **Computed Tomography (CT):** The absolute gold standard imaging modality for definitive evaluation and grading of solid organ injury in a **hemodynamically stable** patient.
- **Emergent Radiography:** Standard initial films include AP Chest (look for widened mediastinum >8 cm indicating aortic tear), AP Pelvis, and C-spine.

Management

- **Primary Survey (ABCDE) Principles:**
  - Must be performed strictly in order. If a patient is hypotensive, conscious, and communicative, Airway and Breathing are stable; immediately move to Circulation and establish large-bore IVs for fluid resuscitation.
  - **Airway:** Secure a definitive airway (intubation) for GCS ≤ 8, severe flail chest/respiratory distress, or severe tracheal edema.

    - **Breathing:**
        - *Tension Pneumothorax:* Immediate life-saving needle thoracostomy followed by definitive tube thoracostomy. Waiting for an X-ray is fatal.
        - *Stable Pneumothorax:* Direct chest tube thoracostomy; needle decompression is unnecessary if stable.
    - **Circulation:**
        - *Cardiac Tamponade:* Pericardiocentesis is a temporary bridge; median sternotomy is the definitive incision for broad access to the heart/great vessels.
        - *Hemorrhage:* Stop external bleeding, apply pelvic binder for open-book fractures, give warm IV fluids/blood products.
- **Secondary Survey:** A comprehensive head-to-toe evaluation performed **only after** the primary survey is complete and resuscitation is underway.
- **Splenic Trauma (Non-Operative Management - NOM):** Hemodynamic stability is a prerequisite. A dropping hemoglobin requiring multiple/frequent transfusions indicates ongoing hemorrhage and signifies NOM failure, requiring surgery/embolization.
- **Acute Limb Ischemia:** The absolute first medical step prior to OR transfer is systemic anticoagulation with an IV bolus and continuous infusion of unfractionated heparin.
- **Maxillofacial Trauma:** Definitive repair of facial fractures is not immediately life-saving and should be delayed until the airway is secured, the C-spine is cleared, and the patient is hemodynamically stable.

Relevant Guidelines

- **Indications for Thoracotomy in Chest Trauma:**
    - Initial blood drainage of >1500 ml upon chest tube insertion.
    - Continuous bleeding of >200 ml/hr for 2-4 consecutive hours.
    - *Note:* Merely >200 ml over 24 hours is NOT an indication.
- **NEXUS Criteria for Cervical Spine Clearance:**
    - Alert and oriented; No focal neurological deficits; Not intoxicated; No midline spinous tenderness; No distracting injuries.
- **qSOFA Score (Sepsis screening):**
    - Includes exactly 3 components: Altered mental status (GCS < 15 or new confusion), Respiratory rate ≥ 22, and Systolic BP ≤ 100 mmHg. (Tachycardia is SIRS, not qSOFA).
- **Glasgow Coma Scale (GCS):**
    - Minimum score = 3; Maximum score = 15. A lower score correlates with higher mortality.
    - *Eye Opening (1-4):* Spontaneous (4), Verbal (3), Pain (2), None (1).
    - *Verbal Response (1-5):* Oriented (5), Confused (4), Inappropriate words (3), Incomprehensible (2), None (1).
    - *Motor Response (1-6):* Obeys commands (6), Localizes pain (5), Withdraws (4), Abnormal flexion (3), Extension (2), None (1).

Operative / Procedural Notes

- **Chest Decompression:**
  - *Needle Thoracostomy:* 2nd intercostal space, midclavicular line.
  - *Tube Thoracostomy:* 5th intercostal space, anterior axillary line. Dissect over the superior rib margin to avoid the neurovascular bundle.
- **Tracheostomy:** Typically considered after 10-14 days (2 weeks) of prolonged mechanical ventilation to prevent tracheal stenosis and vocal cord damage.
- **Fasciotomy:** Emergent 4-compartment fasciotomy of the lower leg is the definitive, limb-saving treatment for compartment syndrome.
- **Nasogastric Tube (NGT):** Absolute contraindications include suspected/confirmed esophageal rupture, stricture, and severe maxillofacial trauma. *Note:* A foreign body in the esophagus requires caution but is not an absolute contraindication.

Complications / Prognosis

- **Persistent Air Leak:** Failure of a pneumothorax to resolve after 3-5 days of chest tube management indicates surgical intervention (VATS bleb excision and mechanical pleurodesis).
- **Hypothermia:** Causes trauma-induced coagulopathy; impairs the coagulation cascade, increasing hemorrhagic mortality.

Past-Paper High Yield

- **Primary Survey Priority Trap:** If a trauma patient has severe facial fractures or airway compromise, airway management overrides all other structural repairs.
- **Tension Pneumothorax vs. Tamponade:** Tension PTX presents with hyperresonance and absent breath sounds; Tamponade presents with Beck's triad and pulsus paradoxus. Neither requires a CXR prior to life-saving intervention.
- **Anatomy & General Surgery Integrated High-Yields:**
  - **Hernias:**
    - *Femoral:* Has a narrow, rigid neck (lateral border = femoral vein) with a 40% strangulation rate. Originates inferior to the inguinal ligament.
    - *Inguinal:* Ilioinguinal nerve is the most commonly injured during anterior repair. The cremasteric muscle derives from the internal oblique.
  - **Canal of Nuck:** Normal embryological pouch of peritoneum extending into the labia majora.
  - **Median Umbilical Ligament:** Fibrous remnant of the urachus.
  - **Thyrohyoid Muscle Innervation:** Uniquely innervated by C1 'hitchhiking' on CN XII, explicitly bypassing the ansa cervicalis.
  - **Ulnar Nerve:** Tested clinically by abducting the fingers against resistance (dorsal interossei).
  - **Torticollis:** Caused by spasm/injury to the Sternocleidomastoid muscle.
  - **Bell's Palsy:** The most common cause of lower motor neuron facial nerve palsy.
  - **Abdominal Aortic Aneurysm (AAA):** Most commonly located in the infrarenal aorta.

- **Parotid Mass:** Fine-needle aspiration (FNA) is the initial diagnostic step; incisional/excisional biopsies are strictly contraindicated due to facial nerve risk.
- **Inflammation/Infection:** Tumor Necrosis Factor (TNF) is a central mediator of septic shock and is highly pro-coagulant. Trench foot is a non-freezing cold injury. Cellulitis from superficial excoriation does not typically cause osteomyelitis.
- **Oncology:** Testicular cancer is NOT strongly associated with DNA viruses (unlike Burkitt's/EBV, Cervical/HPV, HCC/HBV).

Memory Pearls

- **"Blood on the floor and 4 places more":** Key areas for massive hemorrhage (Chest, Abdomen, Retroperitoneum, Pelvis, Bilateral Femurs).
- **GCS ≤ 8:** Intubate.
- **45/45/45 Rule for Pericardiocentesis:** Subxiphoid insertion, 45-degree angle to the skin, pointing 45 degrees left and 45 degrees cephalad toward the left scapula.

Preoperative assessment of surgical patients & risk evaluation

Core Concepts

- **Primary Goals:** Improve the outcomes of surgery and anesthesia, detect unrecognized diseases/risk factors, quantify risk, and plan perioperative care (e.g., HDU/ICU beds).
- **Contraindications to Major Elective Surgery:** Must be actively screened for. Standard reasons to delay elective surgery include:
  - Recent myocardial infarction (<6 months).
  - Recent stroke (<4 months).
  - Severe hypokalemia (e.g., K^+ of 2.5 mmol/L on diuretics).
  - Active or resolving upper respiratory tract infections.
  - *Note:* A previous heart valve replacement (e.g., mitral valve) is **not** a contraindication; it solely requires bridging anticoagulation and endocarditis prophylaxis.
- **Surgical Site Infection (SSI) Risk:** Independently increased by advanced age (>70), chronic malnutrition, long-term steroid use (immunosuppression), and remote body site infections. While uncontrolled diabetes is a major risk factor, **well-controlled diabetes mitigates SSI risk significantly.**
- **Prophylactic Antibiotics:** Must be given as a single preoperative dose to attain high tissue levels at the time of incision. They should be **narrow-spectrum** (targeted against specific flora likely encountered), *not* broad-spectrum. Late administration (>3 hours post-incision) renders them ineffective.

Diagnosis / Clinical Features

- **Surgical Risk Factors (History & Physical):**
  - *Age:* Not a sole criterion to withhold surgery, but associated with comorbidities, frailty, and malnutrition.

- *Social History:* Smoking increases short-term myocardial oxygen demand and decreases long-term mucociliary clearance.
- *Family History:* Screen for malignant hyperthermia, pseudocholinesterase deficiency, and bleeding disorders.

- **Clinical Assessments Specific to General Surgery:**
  - *Peripheral Arterial Disease (PAD):* Classic presentation is intermittent claudication (e.g., calf pain after walking 200m). First initial step in evaluation is the **Ankle-Brachial Index (ABI)**.
  - *Diabetic Foot:* The most appropriate tool for assessing protective sensation (neuropathy) is the **10-gram Semmes-Weinstein monofilament**.
  - *Femoral Hernias:* Present as a bulge **inferior** to the inguinal ligament. Due to the rigid, narrow defect bounded by the lacunar ligament and femoral vein, they carry an exceptionally high risk of incarceration and strangulation (up to 40% present emergently). They must be repaired, never observed.

Investigations

Targeted preoperative testing should be based on patient age, comorbidities, and surgical grade rather than broad, unselective screening.

- **Full Blood Count (FBC):** Indicated for all emergencies, all elective cases >60 years, all adult females, cases with expected significant blood loss, or suspicion of anemia/CKD.
- **Urea and Electrolytes (U&E):** Indicated for patients >65 years, cardiopulmonary disease, diuretic/steroid use, renal/liver/endocrine disease, malnutrition, or IVF therapy >24 hours.
- **Random Blood Glucose:** Indicated for acute abdomen, known DM, malnutrition, obesity, or age >60.
- **Coagulogram (PT, APTT, INR):** Indicated for known liver disease, heavy alcohol use, bleeding history, anticoagulant therapy, and specific high-risk surgeries (vascular, cardiothoracic, craniotomy).
  - *Note:* Warfarin effect is profoundly amplified by **liver disease** due to impaired synthesis of Vitamin K-dependent factors (II, VII, IX, X).
- **Liver Function Tests (LFTs):** Upper abdominal pain, jaundice, alcoholism, or Hepatitis B/C screening.
- **Amylase:** Perform in all adult emergency admissions with abdominal pain prior to surgery.
- **Chest X-Ray (CXR):** Elective cases >60 years, thoracic/cervical/abdominal trauma, respiratory symptoms, thoracic surgery, malignancy, perforated viscus, TB, or goiter (to check for retrosternal extension/tracheal deviation).

Management

- **Perioperative Cardiac Management:**
  - If a patient has had a recent MI or PCI with stenting, elective surgery should be postponed for **4–6 weeks**.
  - Optimization may require initiation of medical therapy (e.g., beta-blockers) prior to surgery.
- **Vascular & General Procedural Management:**

- *Superficial Femoral Artery (SFA) Occlusion:* For short-segment occlusions (e.g., 3 cm) causing claudication, **endovascular angioplasty** is the most appropriate, least invasive initial treatment.
- *Abdominal Aortic Aneurysm (AAA):* A large AAA (>5.5 cm) requires repair. In patients with severe comorbidities (e.g., COPD, CAD) who are high-risk for open surgery but have favorable vascular anatomy (good iliac caliber), **Elective Endovascular Aneurysm Repair (EVAR)** is the definitive treatment of choice.

Relevant Guidelines

ASA Physical Status Classification

ASA GRADE	DEFINITION	MORTALITY (%)
I	Normal healthy individual	0.06
II	Mild systemic disease that doesn't limit activity	0.4
III	Severe systemic disease that limits activity	4.5
IV	Severe systemic disease that is a constant threat to life	23
V	Moribund, not expected to survive 24hrs with or without surgery	51

Clinical Predictors of Increased Cardiovascular Risk (ACC/AHA)

- **Major Predictors:** Acute/recent MI (<30 days), unstable/severe angina, strongly positive stress test, decompensated heart failure, severe valvular disease, significant arrhythmias.
- **Intermediate Predictors:** Mild angina, previous MI (history or Q waves), compensated heart failure, diabetes mellitus, renal insufficiency (Cr > 2.0 mg/dl).
- **Minor Predictors:** Advanced age, abnormal ECG (LVH, LBBB, ST changes), low functional capacity, history of stroke, uncontrolled systemic hypertension.

Surgery-Related Risk (Cardiac Event Rate)

- **High Risk (>5%):** Emergent major surgery (especially in elderly), aortic/major vascular, peripheral vascular, prolonged procedures with large fluid shifts/blood loss.
- **Intermediate Risk (<5%):** Carotid endarterectomy, endovascular AAA repair, head & neck surgery, intraperitoneal/intrathoracic surgery, orthopedic, prostate surgery.
- **Low Risk (<1%):** Endoscopic, superficial, cataract, breast surgery.

Goldman Cardiac Risk Index Points are assigned to estimate cardiac complication risk:

- S3 gallop or JVD: **11**
- Recent MI (< 6 months): **10**
- Nonsinus rhythm or PACs on ECG: **7**
- >5 PVCs/minute: **7**

- Age > 70 years: **5**
- Emergency operations: **4**
- Poor general medical condition: **3**
- Intrathoracic, intraperitoneal, or aortic surgery: **3**
- Important valvular aortic stenosis: **3**
- *Risk Stratification:* 0–5 pts = 1%; 6–12 pts = 7%; 13–25 pts = 14%; >26 pts = 78% complication rate.

Revised Cardiac Risk Index (RCRI / Lee's Index) *Independent predictors (1 point each):* Ischemic heart disease, Congestive heart failure, Cerebrovascular disease (Stroke/TIA), High-risk surgery, Preoperative insulin use, Preoperative Creatinine >2 mg/dL.

Nutritional Risk Index (NRI)

- *Formula:* NRI = (15.19 × Serum Albumin g/dL) + (41.7 × [Present weight / Usual weight])
- *Threshold:* NRI **< 83%** indicates significantly increased mortality.

Past-Paper High Yield

- **Highest Perioperative Mortality:** Severe, **decompensated congestive heart failure**—especially when exacerbated by severe anemia (e.g., Hb 7)—carries a higher perioperative mortality risk than a recent MI, severe aortic stenosis, or advanced age >70.
- **C. difficile Infection:** Risk factors include broad-spectrum antibiotics, prolonged hospitalization, advanced age, and PPI use (flora disruption). *Dietary habits (e.g., being vegetarian) are NOT risk factors.*
- **Hidradenitis Suppurativa:** Primarily affects **APOCRINE** sweat glands (axilla/groin), *not* eccrine glands. It occurs post-puberty, is more common in Black populations, and chronic tracts require wide surgical excision. Malignant transformation can occur in chronic cases.
- **Scleroderma:** Systemic sclerosis is universally and classically associated with **Raynaud's phenomenon**, famously forming part of the CREST syndrome (Calcinosis, Raynaud's, Esophageal dysmotility, Sclerodactyly, Telangiectasia).
- **Soft Tissue Sarcomas:** Most spread hematogenously, but **Malignant Fibrous Histiocytoma** uniquely has a highly recognized propensity for *lymphatic* metastasis.
- **Technetium-99m Bone Scans:** Carries a negligible/extremely low risk of anaphylaxis (it is non-iodine). It is highly sensitive for osteoblastic activity (metastases) but notoriously lacks specificity.

Memory Pearls

- **"Decompensated CHF tops the risk list"**: Always select decompensated heart failure as the highest predictor of perioperative mortality when compared to isolated older MIs or age.
- **Femoral Hernia Anatomy = High Risk: F**emoral hernias are **F**emale-predominant, **I**nferior to the inguinal ligament, and have a **F**rightening strangulation risk.
- **Prophylactic Abx Rule: N**arrow spectrum, **N**ow (at incision), **N**ot later (>3 hrs is useless).

Metabolism & nutrition in surgical patients

Core Concepts

Metabolic Response to Injury (Stress Metabolism)

- **Ebb Phase (Shock Phase):** Brief, initial period immediately following injury or surgery.
- **Flow Phase:** Prolonged hypermetabolic state lasting weeks to months. Characterized by intense hypermetabolism, protein catabolism, insulin resistance, and adverse nutritional depletion.
- **Hormonal Shift:** Major surgery triggers a massive release of counter-regulatory hormones (glucagon, cortisol/glucocorticoids, catecholamines) and a reduction in insulin.
  - *Result:* Glycogenolysis, gluconeogenesis, sodium/water retention, and systemic **hyperglycemia** (not hypoglycemia).
- **Catabolic Phase:** Dramatic increase in metabolic demand, marked by high urinary nitrogen excretion (protein depletion). Muscle breakdown yields amino acids for hepatic gluconeogenesis.
- **Early Anabolic Phase (Corticoid Withdrawal Phase):** Marked by positive nitrogen balance and a rapid, progressive gain in weight and muscular strength.
- **Late Anabolic Phase:** Final recovery period; adipose stores replenish gradually, nitrogen balance equilibrates, and weight gain slows.

Macronutrients and Energy Yields

- **Carbohydrates (30–40% of diet):**
  - Enteral formulations provide **4 kcal/g**; parenteral formulations (hydrated) provide **3.4 kcal/g**.
  - **Obligatory Glucose Users:** Brain, red blood cells, renal medulla, and bone marrow rely exclusively on glucose.
  - **Protein-Sparing Effect:** Administration of approximately **100 grams of glucose per day** provides minimum needs for obligatory users, suppressing hepatic gluconeogenesis and preventing muscle protein breakdown.
- **Lipids (25–45% of diet):** Provide **9 kcal/g**.
  - **Cardiac muscle** primarily relies on free fatty acids for energy (especially at rest) and can utilize ketone bodies. It is *not* an obligatory glucose user.
- **Proteins:** Provide **4 kcal/g**. Complete absorption is typically achieved by the mid-jejunum.
  - **Glutamine:** A conditionally essential amino acid during stress; highly utilized by rapidly dividing cells. It serves as crucial fuel for enterocytes and improves immune function.

Wound Healing Basics

- **Phases:** The proliferative phase (angiogenesis, collagen deposition, granulation, epithelialization) begins **2 to 3 days** post-injury.
- **Vitamin C (Ascorbic Acid):** An essential cofactor for **prolyl hydroxylase and lysyl hydroxylase**, enzymes responsible for the hydroxylation of proline and lysine residues. This is a mandatory step for stabilizing the collagen triple-helix. Deficiency causes scurvy and poor wound healing.

- **Holistic Healing:** While individual vitamins are essential biochemically, a **balanced nutritional diet** (adequate protein, calories, vitamins, minerals) is the most comprehensive and critical factor for optimal wound healing.

Diagnosis / Clinical Features

Nutritional Assessment

- **Subjective Global Assessment (SGA):** A comprehensive clinical examination and history (assessing muscle wasting, fat loss, and dietary changes). It is the **most reliable, validated, and best overall indicator** of a patient's nutritional status—superior to isolated lab values.
- **High-Risk History Markers:**
  - Unintentional weight loss of 5% in the last month or 10% over 6 months.
  - Current weight ≤ 80–85% of ideal body weight.
  - Energy intake ≤ 50% of estimated requirements for ≥ 5 days.
- **Physical Exam:** Thenar/temporal muscle wasting, loss of subcutaneous fat (loose skin), peripheral edema/ascites (hypoproteinemia).

Types of Malnutrition

FEATURE	MARASMUS (CALORIC DEFICIENCY)	KWASHIORKOR (NONCALORIC/PROTEIN DEFICIENCY)
Pathology	Chronic inadequate protein & caloric intake.	Catabolic protein loss during prolonged starvation/stress.
Appearance	"Wizened/old man", thin limbs, severe loss of muscle/fat.	Moon face, swollen abdomen (ascites/hepatomegaly), edema.
Labs/Stores	Visceral protein stores (albumin) usually normal.	Severe hypoalbuminemia.

Investigations

Laboratory Markers of Nutrition

- **Prealbumin:** Has a very short half-life (**2–3 days**). It is the best and most sensitive indicator of *recent* changes in nutritional status.
- **Albumin:** Has a long half-life (20 days); a poor marker for acute changes.
- *Note:* Both markers (and transferrin) vary with inflammation and should be interpreted cautiously alongside CRP and WBC counts.

Energy Measurement

- **Indirect Calorimetry:** The gold standard for measuring energy expenditure (measures CO2 production and O2 consumption).
- **Harris-Benedict Equation:** Used to predict Basal Energy Expenditure (BEE), adjusting for height, weight, age, and sex.
- **Respiratory Quotient (RQ):** Ratio of expired CO2 to consumed O2.

- RQ = 1.0 indicates carbohydrate (glucose) oxidation.
- RQ = 0.8 indicates protein utilization.
- RQ = 0.7 indicates fat metabolism.

Management

Return of Bowel Function Post-Op Following laparotomy or laparoscopy, bowel motility returns sequentially:

1. **Small intestine:** Recovers first (within hours).
2. **Stomach:** Recovers next (24–48 hours).
3. **Colon:** Recovers last (48–72 hours).

Enteral Nutrition (EN)

- **Benefits:** Highly beneficial, physiologic, and prevents the translocation of gut bacteria by maintaining mucosal integrity.
- **Formulas:** A "half-strength" enteral feeding formula means the standard formula is diluted by exactly half (**50% formula mixed with 50% free water**).
- **Contraindications:** Intestinal obstruction, upper GI bleeding, severe diarrhea resistant to therapy, intractable vomiting, and high-output enterocutaneous fistulas.
  - *High-Yield Exception:* **Mild acute pancreatitis is NOT a contraindication** to enteral feeding. Early EN is actually highly recommended to maintain gut integrity and is safer than TPN.
  - *Note:* Enteral feeding does *not* treat or prevent gallstones.

Parenteral Nutrition (PN)

- **Indications:** Patients who cannot meet needs via oral/enteral routes and have contraindications to EN (e.g., short gut syndrome, high-output fistula, prolonged ileus).
- **Central vs. Peripheral:**
  - Peripheral Parenteral Nutrition (PPN) is strictly limited to an osmolarity of **900 mOsm** to avoid phlebitis.
  - Total Parenteral Nutrition (TPN) solutions utilizing highly concentrated dextrose (e.g., **20% Dextrose**) are extremely hyperosmolar and **must strictly be administered via a central venous line** to prevent severe chemical phlebitis/sclerosis.
- **Discontinuation:** Discontinue when the patient can consistently meet 60% of caloric/protein needs orally or enterally.

Relevant Surgical Pharmacology & Medical Management

- **Antibiotics:**
  - **Vancomycin:** Targets bacterial cell wall synthesis by binding tightly to D-alanyl-D-alanine.
  - **Carbapenems:** Exceptionally broad-spectrum beta-lactams covering gram-positive, gram-negative, and anaerobic bacteria.

- **Metronidazole:** Exerts bactericidal effect by causing DNA strand breakage (distinct from clindamycin, which inhibits 50S ribosomes).
- ***C. difficile* Infection: Oral vancomycin** (or fidaxomicin) is the first-line therapy. IV vancomycin does not penetrate the gut lumen. Anti-motility agents (loperamide) are contraindicated.

- **Post-Dural Puncture Headache:** Treated with bed rest, analgesics, oral hydration, epidural blood patch, and **caffeine** (causes cerebral vasoconstriction, which actively treats the headache).

Operative / Procedural Notes

- **Feeding Tubes (Aspiration Risk):** A jejunostomy tube delivers feeds post-pylorically into the jejunum, significantly reducing the risk of reflux. A **gastrostomy tube carries a higher risk of aspiration** compared to a jejunostomy.
- **Pneumoperitoneum (Laparoscopy):** A major physiological disadvantage of using CO2 gas is that it is highly soluble, rapidly crossing the peritoneum into the bloodstream, causing systemic **hypercarbia** and respiratory acidosis.

Complications / Prognosis

- **Enteral Feeding Complications:**
  - GI side effects such as **abdominal cramps and diarrhea** are common, often due to hyperosmolar formulas or rapid infusion rates.
  - Tracheobronchial aspiration (mitigated by head elevation and jejunal placement).
- **Parenteral Nutrition Complications:**
  - **Cholestasis / Gallstones:** Common metabolic complication of long-term TPN due to a lack of enteral stimulation for gallbladder contraction, leading to biliary stasis.
  - **Hyperglycemia:** Standard complication. Target blood glucose is <180 mg/dL.
  - **Occult Sepsis:** If a patient previously stable on TPN suddenly becomes **glucose intolerant**, it is a classic clinical hallmark of occult sepsis (though standard exam keys may also point to underlying Diabetes Mellitus as a cause of TPN hyperglycemia).
- **Refeeding Syndrome:** A potentially lethal complication in severely malnourished patients upon feeding reintroduction. Marked by dangerous shifts (depletion) of **phosphate, potassium, magnesium, and thiamine**.
- **Fluid & Electrolyte Shifts:**
  - Severe catabolism breaks down cellular structures, decreasing effective total body water.
  - Primary hyperaldosteronism (Conn's syndrome) promotes renal excretion of magnesium, causing **hypomagnesemia** (not hypermagnesemia).

Past-Paper High Yield

- **Transplant Oncology: Kaposi sarcoma** is a highly prominent and the most common cancer in immunocompromised post-organ transplant patients.
- **Wound Healing Trap:** While cross-linking of collagen requires copper, the *hydroxylation* of proline/lysine strictly requires **Vitamin C**.

- **Fuel Sources Trap:** The brain and RBCs are obligatory glucose users; **cardiac muscle** is *not* (it uses free fatty acids).
- **Vasculitis Trap:** Giant cell (temporal) arteritis is the most common *primary* systemic vasculitis in adults, but leukocytoclastic (hypersensitivity) vasculitis is the most common vasculitis overall.
- **Dermatology/Laser Trap:** Scleroderma is a systemic fibrotic autoimmune disease; localized **laser therapy has no role** in its treatment (unlike port wine stains or hair removal).
- **Mechanism of Action Trap:** Metronidazole covers anaerobes via DNA strand breakage; it does *not* share a mechanism with clindamycin.

Memory Pearls

- **Prealbumin = Present:** Short half-life (2-3 days), tells you the nutrition status *right now*.
- **Vitamin C = Collagen Hydroxylase:** No C = no cross-linkable collagen = scurvy.
- **100g Glucose:** The magic number daily to feed the brain/RBCs and stop muscle breakdown.
- **Bowel Wake-up:** Small Bowel (hours) -> Stomach (1-2 days) -> Colon (2-3 days).
- **CO2 in Laparoscopy:** Highly soluble -> Hypercarbia -> Respiratory acidosis.
- **Vanco for C. diff:** Must be ORAL. IV vancomycin will not reach the colon lumen.

Fluid management of surgical patients

Core Concepts

- **Total Body Water (TBW) Compartments:** Accounts for ~60% of total body weight.
  - **Intracellular Fluid (ICF):** 2/3 of TBW (~40% of body weight, ~28 L in a 70 kg adult). Primary ions are Potassium (K^+), Magnesium (Mg^{2+}), phosphates, proteins, and sulfate.
  - **Extracellular Fluid (ECF):** 1/3 of TBW (~20% of body weight, ~14 L). Primary ions are Sodium (Na^+), Chloride (Cl^-), and Bicarbonate (HCO_3^-).
    - *Interstitial fluid:* 3/4 of the ECF (~10.5 L).
    - *Intravascular fluid (Plasma):* 1/4 of the ECF (~3–3.5 L).
- **TBW Demographics & Variation:**
  - **Age:** TBW decreases steadily with age due to progressive loss of muscle mass and increased adipose tissue.
  - **Infants/Neonates:** Have the highest physiological percentage of TBW (70–80%), giving them the lowest reserve against dehydration.
  - **Sex & Body Habitus:** Females and obese individuals have a lower TBW percentage because adipose tissue holds significantly less water than highly aqueous muscle tissue.
- **Daily Fluid Balance:**
  - Average input and output is 30–35 mL/kg/day (approx. 2.4 L/day).
  - *Losses:* Urine (500–1500 mL), Skin (500–700 mL), Lungs (400 mL), Stool (100–200 mL).
  - *Fever:* Water requirements increase by 100 to 150 mL/day for each 1°C elevation in body temperature.

- **Osmolality vs. Osmolarity:**
  - *Osmolality (mOsm/kg):* Normal plasma is 275–295 mOsm/kg. Calculated as: 2 × Na + fracGlucose18 + fracBUN2.8.
  - *Effective Osmolality (Tonicity):* Derived by omitting freely permeable BUN (2 × Na + fracGlucose18).
- **Acid-Base Buffer System:**
  - The normal physiological ratio of bicarbonate (HCO_3^-) to carbonic acid (H_2CO_3) is 20:1.
  - In *uncompensated respiratory acidosis*, retained CO_2 rapidly converts to carbonic acid, dropping the ratio below 20:1 until the kidneys slowly compensate by retaining bicarbonate.
  - In *metabolic alkalosis*, the primary respiratory compensation is hypoventilation (retention of CO_2 to increase PCO_2 and lower pH).

Management

- **Maintenance Fluid Calculation (100-50-20 Rule):**
  - 100 mL/kg for the first 10 kg.
  - 50 mL/kg for the next 10 kg.
  - 20 mL/kg for every remaining kg.
  - *Example 1 (35 kg child):* 1000 + 500 + (15 × 20) = 1800 mL/day.
  - *Example 2 (22 kg child):* 1000 + 500 + (2 × 20) = 1540 mL/day (note: some exam keys loosely accept 1550 mL).
- **Intravenous Fluids (Crystalloids):**
  - **0.9% Normal Saline (NS):** Contains 154 mEq/L Na^+ and 154 mEq/L Cl^-. Major extracellular expander. Avoid large volumes in pre-eclampsia, CHF, and cirrhosis.
  - **Ringer's Lactate (RL):** Considered the most physiological crystalloid. Contains 131 mEq/L Na^+, ~109–112 mEq/L Cl^-, 5 mEq/L K^+. Lactate is metabolized in the liver to bicarbonate, providing buffering capacity.
  - **5% Dextrose in Water (D5W):** Contains 50 grams of glucose per liter. Supplies roughly 200 kcal/L, which is *vastly insufficient* to meet basic nutritional requirements for fasting patients. Used to prevent starvation ketosis and correct hypernatremia.
- **Intravenous Fluids (Colloids):**
  - Large molecular weight substances that stay in the intravascular space. 3 times more potent than crystalloids (1 mL blood loss = 1 mL colloid = 3 mL crystalloid).
  - *Albumin:* 5%, 20%, 25% formulations. Avoid in severe anemia/cardiac failure.
  - *Dextrans:* Inhibits platelet aggregation and improves microcirculatory flow.
  - *Gelatins (Haemaccel):* Does not interfere with blood grouping/cross-matching.
  - *Hydroxyethyl Starch (Hetastarch):* Rapid amylase-dependent breakdown.
- **Condition-Specific Management:**

- **Gastric Outlet Obstruction / Severe Vomiting:** Causes massive loss of gastric fluid (rich in HCl and Na^+). Results in *hypochloremic hypokalemic metabolic alkalosis*. Resuscitate with isotonic 0.9% NS supplemented with Potassium Chloride (KCl) to correct volume and ion deficits. Do not use RL (worsens alkalosis).
- **Severe Diarrhea:** Profound loss of bicarbonate from the lower GI tract leads to *normal anion gap metabolic acidosis* (never alkalosis), hypokalemia, and dehydration. RL is the fluid of choice, especially in pediatrics.
- **Hypernatremia with Hypovolemia:** If hemodynamically unstable, initial volume resuscitation *must* be done with isotonic (0.9%) Normal Saline despite the high serum sodium. Do not avoid NS merely due to hypernatremia.

Relevant Guidelines

- **COCHRANE Collaboration (Critically Ill Patients):**
  - There is no evidence from RCTs that resuscitation with colloids reduces the risk of death compared with crystalloids in patients with trauma or burns after surgery.
  - **Recommendation:** Crystalloids are recommended as the initial fluid of choice for resuscitating patients from hemorrhagic shock.

Operative / Procedural Notes

- **Laparoscopic Pneumoperitoneum:**
  - Carbon dioxide (CO_2) is the standard gas used.
  - It is non-flammable, has a low water content, and is *highly soluble in blood* (critical to prevent fatal air embolisms).
  - *Consequence:* Systemic absorption causes hypercarbia leading to respiratory acidosis, not alkalosis.
- **Peripheral IV Potassium Administration:**
  - To prevent severe phlebitis, vascular sclerosis, and extreme pain, the maximum safe concentration of potassium infused through a peripheral vein is standardly limited to **20 mEq/L**. Higher concentrations mandate a central venous catheter.
- **Pre-/Post-op Bowel Management (Laxative Pharmacology):**
  - *Osmotic laxatives:* Macrogol (polyethylene glycol) retains water in stool.
  - *Stimulant laxatives:* Senna, Bisacodyl.
  - *Bulk-forming:* Psyllium.
  - *Surfactant/Stool softener:* Docusate sodium.
- **Caustic Ingestions (GI Endoscopy Context):**
  - *Strong Alkalis (Lye, drain cleaner):* Cause severe, deep, full-thickness **liquefactive necrosis** of the esophagus by saponifying fats and dissolving tissue barriers.
  - *Strong Acids (Battery acid, toilet cleaner):* Cause superficial **coagulative necrosis** (forms a tough protective eschar that limits deep penetration).

Complications / Prognosis

- **Hypernatremia Overcorrection:** Rapidly lowering serum sodium and osmolality causes massive fluid shifts into the brain, resulting in fatal cerebral edema. The aim is a gradual decrease; correction should not exceed 0.5 mmol/L/hr. Aiming for 10 mOsm/hour is excessively fast and dangerous.
- **Excessive Normal Saline (NS):** Infusing large volumes leads to **hyperchloremic metabolic acidosis** because of the non-physiological high chloride load (154 mEq/L).
- **Colloid-Specific Complications:**
  - *Dextrans:* Can cause acute renal failure and uniquely interfere with blood grouping and cross-matching.
  - *Hetastarch:* Increases serum amylase concentrations (can falsely mimic pancreatitis) and impairs coagulation (decreases fibrinogen, VWF, factor VIII, and platelet aggregation).
- **Hypermagnesemia:** High magnesium levels act as a profound central and peripheral nervous system depressant. It classically causes diminished or absent (never exaggerated) deep tendon reflexes, generalized muscle weakness, and in severe cases, cardiac arrest.

Past-Paper High Yield

- **1/4 ECF Rule:** Intravascular plasma makes up exactly 25% (1/4) of the extracellular fluid.
- **Chloride Concentrations:** Normal Saline has 154 mEq/L Cl^-. Ringer's Lactate has approx. 109 mEq/L Cl^- (statements asserting RL has 130 mEq/L Cl^- are highly tested as *FALSE*).
- **Vomiting vs. Diarrhea Acid-Base traps:**
  - Vomiting / Gastric suction = Metabolic Alkalosis (treat with NS + KCl).
  - Diarrhea = Metabolic Acidosis (treat with RL).
- **Hypovolemic Hypernatremia Trap:** Never withhold 0.9% Normal Saline in a hemodynamically unstable, volume-depleted patient just because they are hypernatremic.
- **D5W Nutrition Trap:** 5% Dextrose in Water does *not* provide adequate caloric/nutritional support for fasting patients; it only contains 50g of sugar (~200 kcal).
- **Hypermagnesemia DTRs:** Often tested as a false positive trait. Reflexes are *diminished/absent*, not exaggerated.
- **Metabolic Alkalosis Compensation:** Driven strictly by respiratory hypoventilation (CO_2 retention).
- **Gas used in Laparoscopy:** Always CO_2. Remember it is used precisely because it is *highly soluble in blood*, mitigating embolism risk.

Surgical site infection

Core Concepts

- **Definition:** Surgical Site Infections (SSIs) occur within 30 days post-operation (or within 1 year if an implant is left in place). They are a major cause of nosocomial (hospital-acquired) infections (>48 hours post-admission).
- **Anatomical Depth of SSI:**

- **Superficial Incisional:** Involves only skin and subcutaneous tissue.
- **Deep Incisional:** Involves deep soft tissues (fascia and muscle layers).
- **Organ/Space:** Involves any anatomical compartment (other than the incision) opened or manipulated during the operation.

- **Pathogen Sources:**
  - **Endogenous (Most common):** Patient's own flora (skin, mucous membranes, GI tract) or seeding from a distant focus. Example: Skin flora (*S. aureus*, *S. epidermidis*) is the primary source of SSI in clean elective surgeries like hernia repairs.
  - **Exogenous:** Surgical personnel (soiled attire, hand hygiene breaks), OR environment/ventilation, or contaminated instruments.
- **Microbiology Trends:** *Staphylococcus aureus* is universally the most common overall SSI pathogen. MRSA rates are highly significant, increasing morbidity, 90-day mortality (3.4x), length of stay, and hospital costs.

Diagnosis / Clinical Features

- **Timing of Presentation:**
  - **1st 24 Hours Post-Op:** Rapid, devastating necrotizing infections. Classically caused by **Group A Beta-hemolytic Streptococcus** (*S. pyogenes*) or **Clostridium perfringens**.
  - **Days 5–7 Post-Op:** Standard SSI presentation, typically caused by staphylococcal or enteric organisms.
- **Gas Gangrene (Clostridial Myonecrosis):**
  - Caused by *Clostridium perfringens* (Gram-positive anaerobe).
  - Rapidly progressive with severe pain out of proportion to exam, crepitus (gas in tissues), and profound, rapid systemic toxemia.
  - **Pathology:** Aggressively invades and destroys muscle tissue (*stating that it "spares muscles" is a classic exam trap and is WRONG*).
- **Necrotizing Fasciitis (Type 1):**
  - The most common form; it is a **polymicrobial** infection (aerobic and anaerobic bacteria).
  - Progresses with extreme rapidity; the underlying muscle may initially be spared but the fascial destruction is catastrophic.
- **Fournier's Gangrene:**
  - Necrotizing fasciitis specifically of the perineum. Primarily affects older patients with multiple comorbidities (e.g., diabetes) and is polymicrobial.
  - **Key Trap:** The testes are typically **spared** because their blood supply originates from the aorta (testicular arteries), distinct from the perineal fascial supply. Routine orchiectomy is *incorrect* unless frankly necrotic.
- **Specific Wound Infections:**
  - **Fight Bites (Clenched-fist injury):** While *Eikenella corrodens* is the classic "buzzword", **Staphylococcus aureus** remains the most common overall bacterial cause of septic arthritis and infection in these wounds.

- **Central Line Infections: Coagulase-negative staphylococci** (*Staphylococcus epidermidis*) are the most common cause due to their ability to form resilient biofilms on indwelling plastics.

Management

- **Antimicrobial Prophylaxis Timing:**
  - Must be given **within 60 minutes prior to surgical incision** (knife to skin). *Exception: 120 minutes for vancomycin and fluoroquinolones.*
  - **Intra-operative Redosing:** Required if the surgery exceeds the antibiotic's half-life (e.g., >3 hours).
  - **Discontinuation:** Must be stopped **within 24 hours** after surgery end time (48 hours for cardiac surgery). Continuing for 2-3 days is a common exam distractor—it provides no benefit and promotes antibiotic resistance and *C. difficile* colitis.
- **Treatment of Specific Infections:**
  - **Gas Gangrene / Necrotizing Fasciitis:** The most important life-saving step is immediate, **aggressive surgical debridement**. Antibiotics alone are insufficient. (Gas gangrene specifically requires high-dose penicillin).
  - **Anaerobic Intra-abdominal Infections: Metronidazole** is the gold standard for severe anaerobic infections below the diaphragm (e.g., *Bacteroides*).
  - **Mild, Localized Hidradenitis Suppurativa (Hurley Stage I): Topical clindamycin 1%** is the standard first-line therapy.
  - **Incarcerated Hernia:** Painful, non-reducible mass with a negative cough impulse requires **urgent surgical exploration** (do not manually reduce or give isolated antibiotics/analgesia).
  - **Snake Bites:** Routine prophylactic IV antibiotics are **NOT** required unless secondary infection is clearly evident. Management relies on antivenom, tetanus toxoid, and compartment syndrome observation.

Relevant Guidelines

SSI Wound Classification (Heavily Tested)

CLASS	NAME	DEFINITION & EXAMPLES	EST. SSI RATE	PROPHYLAXIS
I	**Clean**	No inflammation; respiratory/alimentary/genital/urinary tracts **NOT** entered. Closed primarily. *Examples: Herniorrhaphy (primary tissue repair = NO abx needed), Hernioplasty (mesh = abx needed), breast biopsy.*	1.5%	Usually Not Indicated (unless implant/mesh used)

CLASS	NAME	DEFINITION & EXAMPLES	EST. SSI RATE	PROPHYLAXIS
II	**Clean-Contaminated**	Controlled entry into respiratory/GI/GU tracts without unusual contamination. *Examples: Elective upper GI surgery, elective laparoscopic cholecystectomy.*	2–5%	**Indicated**
III	**Contaminated**	Major breaks in sterile technique, gross spillage from GI tract, fresh accidental wounds, acute non-purulent inflammation. *Examples: Emergency bowel resection, enterotomy with spillage during obstruction.*	5–30%	**Indicated**
IV	**Dirty/Infected**	Old trauma with devitalized tissue, existing clinical infection, gross pus, perforated viscera. *Examples: Perforated diverticulitis, Hinchey Stage II diverticulitis (localized abscess).*	5–30%	**Therapeutic Antibiotics Required**

Target Antimicrobial Prophylaxis Regimens

- **Biliary Tract / Cholecystectomy:** Cefazolin (1st generation cephalosporin).
- **Orthopedic & Vascular:** Cefazolin or Cefuroxime or Vancomycin.
- **Colorectal:** Oral Neomycin + Erythromycin base OR Oral Metronidazole.
- **Head & Neck:** Clindamycin + Gentamicin OR Cefazolin.
- **Genitourinary:** Ciprofloxacin.

CDC Prevention Strategies

- **Core Preoperative:**
  - Do NOT remove hair unless necessary; if necessary, use clipping/depilatory agents (never use razors).
  - Treat remote infections prior to elective operation.
- **Core Intraoperative:** Keep OR doors closed.
- **Core Postoperative:**
  - Control blood glucose to <200 mg/dL on POD#1 and #2.
  - Discontinue antibiotics within 24 hrs post-op.
- **Supplemental Strategies:** Decolonize *S. aureus* nasal carriers with mupirocin (cardiac/ortho/neuro implants); redose abx at 3 hr mark; weight-adjust dosing for obesity (BMI >30).

NNIS Risk Stratification (Predicting SSI Risk)

3 independent variables (1 point each):

1. Contaminated or Dirty/Infected wound class.
2. ASA score > 2.
3. Length of operation > 75th percentile for the specific procedure.

Operative / Procedural Notes

- **Surgical Technique to Minimize SSI:** Essential steps include removing devitalized tissue, maintaining meticulous hemostasis, gentle tissue handling, eradicating dead space, and avoiding inadvertent viscus entry.
- **OR Ventilation Parameters:**
  - Temperature: 68°–73°F (20°–22.7°C).
  - Humidity: 30%–60%.
  - Air Changes: ≥ 15 total per hour (≥ 3 outdoor air).

Complications / Prognosis

- **Inhibitors of Wound Healing:** Tissue hypoxia, active infection, Vitamin C deficiency (scurvy), and uncontrolled diabetes mellitus profoundly inhibit wound healing. *(Note: Low systemic free fatty acids do not independently impair wound healing).*
- **Risk Factors for SSI:** Obesity (BMI >30 or 20% over ideal weight), chronic malnutrition, uncontrolled diabetes mellitus, immunosuppression, prolonged preoperative stay, and smoking. *(Note: Hypercholesterolemia is a cardiovascular risk factor, but NOT an independent acute SSI risk factor).*

Past-Paper High Yield

- **Prophylactic Antibiotic Rules:** Give within 60 minutes prior to incision, redose during long surgeries, and **stop within 24 hours**. Never continue for 2-3 days post-op (promotes *C. difficile*).
- **Gas Gangrene Trap:** Gas gangrene **destroys** muscle. If an option says "muscles are spared", it is FALSE.
- **Wound Classification Differentiators:**
  - Elective upper GI or cholecystectomy = **Class II** (Clean-contaminated).
  - Diverticulitis with an abscess (Hinchey II) or perforation = **Class IV** (Dirty).
  - Hernia repair = **Class I** (Clean). If using primary tissue suture (herniorrhaphy), no abx are needed. If using mesh (hernioplasty), give abx.
- **Organism Associations:**
  - *Fight bites:* **Staphylococcus aureus** is most common (despite *Eikenella* association).
  - *Central line:* **S. epidermidis** (biofilm).
  - *First 24 hours post-op:* **Group A Strep** (*S. pyogenes*) or *C. perfringens*.
  - *Intra-abdominal anaerobes:* Treat with **Metronidazole**.

    - *Chlamydia:* Obligate intracellular, urogenital/ocular, **NOT** gastrointestinal flora.
- **Hand Hygiene:** Alcohol-based gels kill vegetative bacteria (*Staph, Strep, E. coli, Pseudomonas*) but **CANNOT kill Clostridioides difficile spores** (requires soap and water).
- **Skin Infections & Osteomyelitis:** Superficial skin infections do NOT reliably indicate underlying osteomyelitis in asymptomatic patients; deep testing is needed.
- **Fournier's Gangrene:** Testicles are spared (supplied by aorta).
- **Needlestick Transmission ("Rule of 3s"):** Hepatitis B (~30%), Hepatitis C (~3%), HIV (~0.3%).

Memory Pearls

- **Rule of 3s for Needlesticks: 30%** (HBV) > **3%** (HCV) > **0.3%** (HIV).
- **Early vs. Late SSIs:** First 24h = "Strep or Clostridium". Days 5-7 = "Staph or Enterics".
- **Spermatic Cord Anatomy Link:** The **internal spermatic fascia** originates from the **transversalis fascia** (a classic anatomy fact relevant to inguinal hernia exploration).
- **Chronic Inflammation Histology:** Characterized by mononuclear cells (lymphocytes, macrophages, multinucleated giant cells), tissue destruction, and fibrosis. Eosinophilia is strictly for allergies/parasites, not typical chronic inflammation.
- **Hydatid Cyst Transmission:** *Echinococcus granulosus* is strictly transmitted via the **fecal-oral** route (ingestion of eggs from dog feces).

Surgical complications and outcome in surgical patients

Core Concepts

- **Postoperative Care Phases:**
    - Immediate postoperative care (Recovery phase).
    - Care in the ward prior to discharge.
    - Continued care post-discharge.
- **Risk Reduction Strategies:** Optimization of co-morbidities/nutrition, minimizing preoperative hospital stay, early mobilization, and meticulous surgical technique.
- **Complications of Prolonged Immobility (Chronically bed-ridden patients):**
    - **Decreased** bone density (osteoporosis) due to increased osteoclastic activity from lack of weight-bearing stress.
    - Rapid skeletal muscle atrophy.
    - Significantly increased risk of deep vein thrombosis (DVT), pressure ulcers, pulmonary atelectasis, and pneumonia.
- **General Categorization of Complications:** Wound, thermal, infectious (fever), pulmonary, cardiac, gastrointestinal, metabolic, and neurological.

Diagnosis / Clinical Features

Wound Complications

- **Fascial Dehiscence:** Classically presents on postoperative days 7–10 with a sudden, painless discharge of copious salmon-pink (serosanguinous) fluid from the wound without surrounding erythema or purulence. This is a pathognomonic sign of fascial failure.
- **Evisceration:** Complete disruption of all abdominal wall layers with protrusion of viscera (e.g., bowel loops) outside the incision.
- **Seroma:** Benign collection of liquefied fat, serum, and lymphatic fluid. Presents as a soft, non-erythematous, non-tender bulge. Associated with mastectomies and axillary/groin dissections.
- **Hematoma:** Abnormal blood collection due to imperfect hemostasis. Presents with purplish discoloration of wound edges and blood leaking through sutures.
- **Necrotizing Fasciitis / Surgical Site Infections:**
  - *Clostridial myonecrosis:* Sudden onset of severe pain, crepitus, marked edema, tense skin, and hemorrhagic bullae following abdominal surgery.
  - *Group A β-hemolytic streptococcal gangrene:* Often follows penetrating wounds.
  - Polymicrobial necrotizing infections are highly associated with Diabetes Mellitus (DM) and Peripheral Vascular Disease (PVD).

Postoperative Fever & Pulmonary Complications

- Early postoperative fever (first 24–72 hours) is typically non-infectious, stemming from the inflammatory response to trauma or pulmonary atelectasis.
- **Atelectasis:** Peripheral alveolar collapse.
  - *Mechanism:* Decreased functional residual capacity (FRC) from diaphragmatic stunning and shallow breathing (incisional pain), combined with impaired mucociliary clearance of thick secretions due to anesthetic gases.
- **Foreign Body Aspiration (Pediatrics):** Acute chest X-ray findings include unilateral hyperinflation (ball-valve effect) or unilateral atelectasis (complete obstruction). Increased anterior-posterior (AP) diameter is *not* an acute finding (associated with chronic COPD).

Thermal Regulation Complications

- **Hypothermia:** A drop below 35°C leads to coagulopathy and platelet dysfunction. It increases the risk of cardiac events (3x), surgical site infections (3x), and blood loss/transfusion requirements.
- **Malignant Hyperthermia:** Rare autosomal dominant condition triggered by anesthesia. Presents with fever, tachycardia, rigidity, and cyanosis.

Gastrointestinal Complications

- **Postoperative Ileus:** Lack of bowel function without mechanical obstruction. Prolonged by extensive manipulation, small bowel injury, narcotic use, abscess, and pancreatitis. X-ray shows diffuse gaseous distension without a transition point.
- **Small Bowel Obstruction (SBO):** Hernias are the second leading cause of SBO worldwide (trailing only intra-abdominal adhesions). Imaging shows dilated centrally located loops, prominent valvulae conniventes, and a classic "stepladder" pattern of air-fluid levels.
- **Pseudomembranous Colitis (C. difficile):** Superinfection due to altered normal flora. Toxic colitis is a surgical emergency carrying a 20–30% mortality rate. CT shows severe colonic wall thickening,

mucosal edema ("accordion sign"), and hyperemia.

- **GI Bleeding:** E.g., Cushing's ulcer (incidence reduced with routine PPI use).

Vascular & General Surgical Pathology

- **Acute Limb Ischemia:** Sudden onset of complete cessation of arterial flow, often due to an embolus from a cardiac source (e.g., lodging in the brachial artery).
- **Saphena Varix:** Dilatation of the saphenous vein at the saphenofemoral junction. Presents as a painless, soft groin swelling that disappears when lying down and has a positive cough impulse.

Management

- **Fascial Dehiscence / Evisceration:** Evisceration is a surgical emergency. The immediate step is covering the bowel with sterile, saline-moistened dressings followed by urgent return to the operating room for washout and closure. Dehiscence requires urgent surgical evaluation to prevent evisceration.
- **Urinary Retention post-Anorectal Surgery:** E.g., post-hemorrhoidectomy, retention is frequently caused by pain-induced reflex spasm of the pelvic floor/sphincter. *First step:* Ensure proper analgesia before considering catheterization. Trigger for catheter replacement is >500 mL retention.
- **Malignant Hyperthermia:** Administer Dantrolene 1 to 2 mg/kg initially, up to a total of 10 mg/kg until symptoms subside.
- **C. difficile Colitis:** Treatment requires stopping the offending antibiotic and administering targeted oral therapy (Vancomycin, Fidaxomicin, or historically Metronidazole). *Systemic steroids are contraindicated.*
- **Immune Thrombocytopenic Purpura (ITP):** Platelet transfusions are generally ineffective (donor platelets are rapidly destroyed by autoantibodies) and do not provide sustained benefit. They are reserved strictly for catastrophic bleeding.
- **Hernia Repair:** Hernioplasty (using a mesh) is the gold standard surgical treatment for inguinal hernias in adult men.

Investigations

- **Fever Work-up (> 48 hours):**
  - History and Physical Exam
  - Blood cultures, Urinalysis/Urine culture, Sputum culture
  - Chest X-ray
- **Metabolic Evaluation (Adrenal Insufficiency):** Suspect in sudden cardiovascular collapse with hypotension, fever, confusion, and abdominal pain.
  - *Test:* Cosyntropin (ACTH) stimulation test (baseline cortisol, then at 30 mins and 60 mins post-hydrocortisone administration).
- **Acute Renal Failure (Oliguria < 0.5 cc/kg/hr):** Differentiate via Fractional Excretion of Sodium (FeNa): Pre-renal (FeNa < 1), Intrinsic/Post-renal (FeNa > 1).

Complications / Prognosis

- **Cardiac Complications:** Ischemia and infarction are the leading causes of death in *any* surgical patient. Prevention is the cornerstone of management. Arrhythmias require correcting underlying electrolytes (Keep Mg > 2, K > 4).
- **Renal Transplant:** The most common cause of death following a renal transplant is *Infection*, secondary to mandatory, life-long powerful immunosuppression.
- **Internal Jugular Cannulation:** Left internal jugular vein cannulation carries a uniquely specific risk of thoracic duct injury (chylothorax), as the duct ascends and empties at the junction of the left internal jugular and left subclavian veins.
- **Hemorrhage Timeline:**
  - *Immediate:* Inadequate hemostasis, unrecognized vessel damage.
  - *Early Postoperative:* Defective anastomosis, coagulopathy (usually requires re-exploration).
  - *Secondary:* Occurs several days postoperatively due to infection eroding a blood vessel (treat the infection).

Past-Paper High Yield

- **Hernia Anatomy & Demographics:**
  - *Direct* inguinal hernias are *medial* to the inferior epigastric vessels and rarely descend into the scrotum.
  - *Indirect* hernias are the most common in both males and females.
  - *Femoral* hernias are more common in females than males (though still a rare presentation overall compared to indirect) and carry a significantly higher risk of strangulation than inguinal hernias.
  - Inguinal hernias appear above and medial to the pubic tubercle.
- **Eponymous Hernias:** A hernia containing a Meckel's diverticulum is a *Littre's hernia*. A *Richter's hernia* involves only the anti-mesenteric bowel wall. A *sliding hernia* is often difficult to reduce.
- **Acute Limb Ischemia:** Classic presentation is the "6 Ps": Pain, Pallor, Pulselessness, Paresthesia, Paralysis, and Poikilothermia (cold).
- **Early Post-Op Fever Trap:** Fever within 24-48 hours is *atelectasis*, not pneumonia, wound infection (days 5-7), or DVT (days 5-7). Ascending cholangitis requires evidence of biliary obstruction.

Memory Pearls

- **The "6 Ws" of Postoperative Fever:**
  - **W**ind: Atelectasis (POD 1-2), Pneumonia (POD 3-4)
  - **W**ater: UTI (POD 3)
  - **W**alking: DVT/PE (POD 5-7)
  - **W**ound: Surgical site infection (POD 5-7)
  - **W**aste: Intra-abdominal Abscess (POD 5-7+)
  - **W**onder drugs: Medication-induced fever

Shock - dr. Amjad

Core Concepts

- **Definition:** A physiologic state in which systemic reduction in tissue perfusion results in decreased tissue oxygen delivery (DO_2). It is fundamentally a mismatch where **Oxygen Demand > Oxygen Delivery**.
- **Key Principle:** Falling blood pressure is a **LATE** sign of shock. Normal blood pressure does *not* equate to normal microvascular perfusion (vessel dropout occurs despite normal BP).
- **Oxygen Delivery (DO_2):**
  - DO_2 = Cardiac Output (CO) × O_2 Content
  - O_2 Content = (1.34 × Hb × SaO_2) + (0.023 × PaO_2)
  - *Clinical correlate:* Hemoglobin and oxygen saturation are the primary drivers of blood oxygen content; dissolved oxygen (PaO_2) contributes minimally.
- **Mean Arterial Pressure (MAP):**
  - MAP - CVP = CO × Systemic Vascular Resistance (SVR)
  - CO = Heart Rate (HR) × Stroke Volume (SV)
- **Cellular Pathophysiology (Hypoxia):**
  - Cells transition from efficient aerobic metabolism (38 ATP) to inefficient anaerobic glycolysis (2 ATP + Lactic acid).
  - **↓ ATP production leads to:**
    1. **Failure of Na^+/K^+ pump:** Influx of Ca^{2+}, Na^+, and H_2O → cellular edema, ER swelling, loss of microvilli.
    2. **Increased anaerobic glycolysis:** ↑ Lactic acid → ↓ intracellular pH → nuclear chromatin clumping.
    3. **Detachment of ribosomes:** ↓ Protein synthesis.
  - Progression leads to lysosomal membrane rupture (enzyme release) and mitochondrial death.

Diagnosis / Clinical Features

- **General Manifestations:**
  - Altered mental status (agitation, confusion, lethargy).
  - Delayed capillary refill time (CRT > 2 seconds).
  - Oliguria/Anuria (< 0.5 mL/kg/hr) due to ↓ GFR.
  - Tachypnea (compensating for metabolic acidosis).
- **Compensatory Responses:**
  - **Sympatho-adrenal:** Baro/chemoreceptors sense ↓ BP → sympathetic activation → epinephrine/norepinephrine release → severe peripheral vasoconstriction (↑ SVR) and ↑ HR/contractility.

    - **Neuroendocrine (RAAS):** Renin → Angiotensin II (potent vasoconstrictor) → Aldosterone (Na^+/H_2O retention). ADH/Vasopressin release.
- **Type-Specific Clinical Findings:**
    - **Hypovolemic:** Cold/clammy skin, flat neck veins, tachycardia, narrow pulse pressure.
    - **Cardiogenic:** Cold/wet skin, elevated Jugular Venous Pressure (JVP), pulmonary crackles, S_3 gallop.
    - **Distributive (Early Septic):** Warm/flushed extremities, bounding pulses, widened pulse pressure (hyperdynamic state).
    - **Obstructive:** Distended neck veins, muffled heart sounds (Beck's triad in tamponade), absent breath sounds (tension pneumothorax).
    - **Neurogenic: Hypotension + Bradycardia + Warm extremities** (due to loss of sympathetic tone and unopposed vagal tone).

Investigations

- **Non-Invasive / Bedside:**
    - **POCUS / RUSH Protocol (Rapid Ultrasound in Shock and Hypotension):** Evaluates "Pump, Tank, and Pipes" (Heart, IVC/abdomen, Aorta/DVT) to determine the etiology of non-traumatic shock (broader than eFAST, which focuses on trauma).
- **Invasive Monitoring (Swan-Ganz / Pulmonary Artery Catheter):**
    - **Hypovolemic:** ↓ CVP, ↓ PCWP, ↓ CO, **↑ SVR**
    - **Cardiogenic: ↑ CVP**, **↑ PCWP**, ↓ CO, **↑ SVR**
    - **Distributive (Septic):** ↓ CVP, ↓/normal PCWP, **↑ CO**, **↓↓ SVR**
    - **Obstructive: ↑ CVP**, ↑ PAP (if PE), ↓ CO, **↑ SVR**

Management

- **General Principles:** Individualize treatment. Shock states frequently coexist. Recognize and treat during the *compensatory* phase; mortality doubles for every hour of delay.
- **Type-Specific Interventions:**
    - **Distributive:** IV fluids, vasopressors, source control (antibiotics/surgery).
    - **Hypovolemic:** Crystalloids, packed RBCs, surgical hemostasis.
    - **Cardiogenic:** Inotropes (dobutamine), vasodilators (sodium nitroprusside), intra-aortic balloon pump (IABP), diuresis.
    - **Obstructive:** Heparin/thrombolysis (PE), needle decompression (PTX), pericardiocentesis.

Relevant Guidelines

- **Diagnostic & Hemodynamic Pathway of Circulatory Shock (Slide Algorithm):**
    - **Step 1:** Arterial hypotension + signs of hypoperfusion = Circulatory Shock.
    - **Step 2:** Estimate CO or SvO_2.
        - If **High CO** → Distributive Shock.

        - If **Low CO** → Assess CVP.
    - **Step 3:** Evaluate CVP in Low CO states.
        - **Low CVP** → Hypovolemic Shock.
        - **High CVP** → Echo required (Cardiogenic vs. Obstructive).
- **Stages of Shock Classification:**
    - **Stage I (Compensated / Nonprogressive):** Perfusion to vital organs maintained. BP preserved via ↑ HR and mild vasoconstriction. ATP Supply = Demand.
    - **Stage II (Uncompensated / Progressive):** Decreased microvascular perfusion, end-organ dysfunction begins, clinical hypotension ensues.
    - **Stage III (Irreversible):** Refractory organ dysfunction, severe cellular acidosis, cell death.

Operative / Procedural Notes

- **Pneumoperitoneum Physiology (12-15 mmHg):** Creation of a CO_2 pneumoperitoneum predictable causes a **transient decrease in urine output** due to physical compression of renal veins and parenchyma (↓ GFR). It also causes ↓ CO, ↑ SVR, and ↓ FRC.
- **Cardiogenic Shock Contraindication:** Pneumatic antishock garments (PASG) are **contraindicated** because they compress the lower body, massively increasing SVR and venous return, which fatally overloads a failing heart.

Complications / Prognosis

- **Inflammatory Mediators & Prognosis:**
    - **Interleukin-6 (IL-6):** Best predictor of prolonged recovery and Multiple Organ Dysfunction Syndrome (MODS). Stimulates acute-phase reactants (causes **hyper**ferritinemia).
    - **TNF-α:** Causes severe hypotension and lactic acidosis.
    - **iNOS (inducible Nitric Oxide Synthase):** Overexpressed in sepsis, producing toxic free radicals and causing hyperdynamic vasodilation.
- Prolonged shock leads to **Shock-induced cardiomyopathy** (impaired contractility) and acute tubular necrosis (ATN).

Past-Paper High Yield

- **Septic Shock Hemodynamics:** Early septic shock is classically a *hyperdynamic* state characterized by normal or **elevated cardiac output** and profoundly **low systemic vascular resistance (SVR)**.
- **Bowel Perforation Sequence:** A missed small bowel perforation or colonic rupture leads directly to massive fecal peritonitis → severe sepsis → widespread systemic vasodilation → distributive shock (↑ CO, ↓ SVR).
- **Cardiogenic Shock Hemodynamics:** Refractory hypotension with a history of MI + **high PCWP** + **high CVP** + **low ScvO2** confirms primary pump failure.
- **Hemorrhagic Shock Classes:**

    - **Class II (20% blood loss):** Compensatory mechanisms maintain BP and **urine output is typically still maintained** (no frank oliguria yet).
    - **Class III (1700 mL loss):** Corresponds to massive sympathetic output → **↑ SVR** to maintain BP, **narrowed pulse pressure**, ↓ CO, and frank oliguria.
- **Neurogenic Shock Triad:** Absolute hallmark in a trauma patient is profound **hypotension + bradycardia + warm, flushed extremities**. (Do *not* pick tachycardia; vagal tone is unopposed).
- **Hidden Hemorrhage Sites:** The abdomen, retroperitoneum, pelvis, and femurs can hide massive blood volumes. The **intracranial space** *cannot* harbor enough blood to cause systemic hypovolemic shock before fatal brain herniation occurs.
- **Necrotizing Soft-Tissue Infections:** Highly aggressive, rapidly progressive, and life-threatening (they do *not* have a gradual/chronic course). Require immediate surgical debridement, usually polymicrobial, produce "dishwater pus", and *S. pyogenes* can cause concurrent toxic shock syndrome.
- **Cellular Biology:** Lymphocytes (B and T cells) mediate adaptive immunity and are **NOT phagocytic**. (Neutrophils, macrophages, microglia, and Kupffer cells *are* phagocytes).
- **Surgical Anatomy Tested with Shock:**
    - The **dorsalis pedis artery** is a direct continuation of the **anterior tibial artery**.
    - An aberrant/replaced **right hepatic artery** most commonly arises from the **Superior Mesenteric Artery (SMA)** and courses posterior to the portal vein.

Memory Pearls

- **Low SVR + High CO** = Distributive / Early Sepsis.
- **High CVP + High PCWP + Low CO** = Cardiogenic (Pump Failure).
- **Bradycardia + Hypotension + Warm Skin** = Neurogenic.
- **IL-6** = Predictor of MODS and causes HYPERferritinemia.
- **RUSH** = Medical shock ultrasound (Pump/Tank/Pipes) neq eFAST (Trauma).

Hypovolemic shock - dr. Khaled

Core Concepts

- **Definition of Shock:** A life-threatening state of circulatory failure resulting in inadequate oxygen delivery to tissues, leading to cellular hypoxia, organ dysfunction, and ultimately multiorgan failure (MOF) if unreversed.
- **Key Hemodynamic Equations:**
    - Cardiac Output (CO) = Stroke Volume (SV) × Heart Rate (HR)
    - Blood Pressure (BP) = CO × Total Peripheral Resistance (TPR)
- **Pathophysiological Cascade:** Hypovolemia → ↓ Venous Return → ↓ Preload → ↓ Cardiac Output → Hypotension → Tissue Hypoxia.
- **Categories of Hypovolemic Shock:**

- **Hemorrhagic:** Intravascular volume loss due to bleeding (Trauma, GI varices/ulcers, ruptured ectopic, ruptured aortic aneurysm, hemorrhagic pancreatitis).
- **Non-Hemorrhagic:** Intravascular volume loss from fluids other than blood:
  - *GI Losses:* Diarrhea, vomiting, fistulas/external drainage.
  - *Renal Losses:* Diuretics, osmotic diuresis, salt-wasting nephropathy.
  - *Skin Losses:* Burns, heat stroke, excessive sweating (can exceed 1–2 L/hr).
  - *Third Spacing:* Extravascular fluid shift (e.g., crush injuries, intestinal obstruction, acute pancreatitis, post-operative state, cirrhosis).

Diagnosis / Clinical Features

- **Classic Signs & Symptoms:** Tachycardia, narrow pulse pressure, hypotension, tachypnea, oliguria, altered mental status (agitation to lethargy), cool/clammy skin, dry mucous membranes.
- **Tissue Perfusion Indicators:** Weak peripheral pulses, prolonged capillary refill (>2 seconds).
- **Renal Perfusion (Exam Pearl):** The standard metric for minimum acceptable urine output in an adult to ensure adequate renal perfusion is **0.5 mL/kg/hour** (e.g., 35 mL/hour for a 70 kg patient).

Investigations

- **FAST (Focused Assessment with Sonography for Trauma):** Early diagnostic ultrasound looking for hemopericardium and intra-abdominal bleeding in 4 windows: Heart (pericardial), Liver/Kidney (Morison's pouch), Spleen (perisplenic), and Bladder (pelvic).
- **CT Scan:** Indicated for identifying retroperitoneal bleeding or solid organ injury, but strictly reserved for hemodynamically **stable** patients.
- **Laboratory:** Baseline labs, blood typing, and cross-matching for PRBCs.

Relevant Guidelines

- **American College of Surgeons (ACS) Classes of Acute Hemorrhage:**
  - **Class I (<15% / <750 mL):** Pulse <100, BP Normal, Pulse Pressure Normal/↓, Cap Refill <2s, RR 14–20, UOP ≥30 mL/hr. Mentation: Slightly anxious.
  - **Class II (15-30% / 750-1500 mL):** Pulse >100, BP Normal, Pulse Pressure ↓, Cap Refill 2-3s, RR 20–30, UOP 20-30 mL/hr. Mentation: Mildly anxious.
  - **Class III (30-40% / 1500-2000 mL):** Pulse >120, **BP Decreased** (hypotension begins here), Pulse Pressure ↓↓, Cap Refill 3-4s, RR 30–40, UOP 5-10 mL/hr. Mentation: Anxious & confused.
  - **Class IV (>40% / >2000 mL):** Pulse >140, BP Severely Decreased, Pulse Pressure ↓↓, Cap Refill >5s, RR >40, UOP Negligible. Mentation: Confused/Lethargic.
- **Assessment of Blood Consumption (ABC) Score:** Predicts the need for massive transfusion based on 4 parameters (assessed upon ED arrival):
  1. Penetrating mechanism of injury.
  2. Positive FAST examination.
  3. Systolic Blood Pressure (SBP) ≤ 90 mmHg.
  4. Heart Rate ≥ 120 bpm.

Management

- **Initial Priorities (ATLS):** Control hemorrhage, establish/protect airway (with C-spine precautions), maximize oxygenation, and gain IV access (Intraosseous lines can be used if IV access fails; targets include humeral head, sternum, proximal tibia, distal femur).
- **Fluid Resuscitation:**
  - Use balanced crystalloids (Lactated Ringer's) initially.
  - *Caveats:* Large volumes of Isotonic Saline (0.9% NaCl) cause non-anion gap hyperchloremic metabolic acidosis. Large volumes of Lactated Ringer's can cause metabolic alkalosis.
  - *Rhabdomyolysis specific:* Aggressive volume resuscitation with IV crystalloids is the cornerstone of early management following a crush injury to flush myoglobin and prevent AKI.
- **Delayed Fluid Resuscitation (Permissive Hypotension):**
  - Target early IV fluid resuscitation only to an **SBP of 70–90 mmHg**.
  - Prevents the "popping" of newly formed clots, dilution of clotting factors, and hypothermia associated with aggressive normotensive fluid replacement.
- **Blood Transfusion Principles:**
  - Transfuse products early in a **1:1:1 ratio** (PRBC : FFP : Platelets). Whole blood is also acceptable.
  - Type O RhD-negative is the universal donor.
  - *Transfusion Threshold:* Give blood to patients exhibiting **symptomatic anemia** (e.g., Hb of 8 g/dL experiencing tachycardia/dyspnea) to improve oxygen delivery. A minor drop or asymptomatic chronic anemia (e.g., ESRD with Hb 7) generally does not mandate immediate transfusion.
- **Massive Transfusion Protocol (MTP):**
  - Defined as requiring ≥10 units PRBCs/24h, ≥4 units/1h, or ≥10 units/6h.
  - Delivered in batches: 6 units PRBCs + 6 units FFP + 6 units random donor platelets (or 1 unit apheresis platelets).
- **Adjuncts in Resuscitation:**
  - **Tranexamic Acid (TXA):** Antifibrinolytic; must be administered within **3 hours** of injury.
  - **Calcium:** Administer to counteract citrate toxicity (chelates calcium) from massive blood product administration.
  - **Temperature Control:** Maintain normothermia (≥ 35.5^°C) using fluid warmers and forced-air blankets to prevent coagulopathy.

Operative / Procedural Notes

- **Hemorrhage Control Techniques:**
  - Direct pressure is primary. Clamping is acceptable only under *direct visualization* (blind clamping is contraindicated).
  - **Tourniquets:** Used for severe extremity injury/amputations. The maximum recommended safe inflation time to prevent irreversible ischemic nerve and muscle damage is **2 hours (120 minutes)**. When possible, release periodically (e.g., every 45 mins).

    - **Scalp Lacerations:** Bleeding can be managed via running sutures, Raney clips, or local injection of lidocaine + epinephrine.
    - **Pelvic Fractures:** Open-book fractures require immediate circumferential pelvic binder or tied sheet for stabilization.
- **Surgical Hemorrhage Classification:**
    - *Primary:* Bleeding during the surgery.
    - *Reactionary:* Bleeding within the first **24 hours** post-op (e.g., caused by a slipped surgical ligature once blood pressure normalizes).
    - *Secondary:* Bleeding **7–14 days** post-op (almost exclusively caused by infection eroding a vessel wall).
- **Anesthesia in Shock:** Use agents with minimal hemodynamic effects; reduce standard induction doses. Avoid high Positive End-Expiratory Pressure (PEEP) as it increases intrathoracic pressure, decreases venous return, and worsens cardiac output.
- **Intraoperative Temperature Regulation:** Core body temperature is directly affected by cold IV fluids, ambient OR air, cold/humidified ventilator air, and muscle relaxants (which halt shivering). *Note: The patient's O2 saturation level is a measure of oxygenation, not a physical variable that alters body temperature.*

Complications / Prognosis

- **Tissue Ischemia Sensitivity:** Nerve tissue is the *most* sensitive to ischemia, followed by muscle. Skin is highly resistant and tolerates ischemia much longer than muscle or nerve. Ischemia-reperfusion injury is mediated by oxygen free radicals.
- **Limb Viability:** Rigid muscles indicating **rigor mortis** in a limb is a definitive sign of irreversible tissue necrosis, implying the limb is no longer viable and invariably requires amputation. (Loss of pulses or sensation indicates severe ischemia but may still be reversible with emergent revascularization).
- **Coagulopathy & DIC:** Prolonged shock leads to Disseminated Intravascular Coagulation (DIC), characterized by widespread clotting cascade activation → microthrombi formation → consumption of platelets/coagulation factors → significant hemorrhage, ↓ fibrinogen, ↓ platelets, and prolonged PT/PTT.

Past-Paper High Yield

- **Antibiotic Prophylaxis:** Hernioplasty strictly requires Abx prophylaxis because it involves placing a prosthetic mesh (a foreign body), carrying a risk of devastating mesh infection. Abx should be given before anesthesia, tailored to the site, and re-dosed in prolonged surgeries.
- **Pharmacology of Anticoagulants:** Heparin's mechanism of action is binding to and activating **Antithrombin III**, which rapidly inactivates thrombin and Factor Xa. (Warfarin = Vit K epoxide reductase inhibitor; Rivaroxaban = direct Xa inhibitor; Dabigatran = direct thrombin inhibitor).
- **Tracheostomy Bleeding:** Minor, self-resolving bright red bleeding from a tracheostomy site a few weeks (e.g., 2 weeks) post-procedure is most commonly due to friable **granulation tissue** at the stoma. (A tracheoinnominate fistula causes catastrophic, massive hemorrhage, not self-resolving bleeding).

- **Laparoscopy Arrhythmias:** The most common arrhythmia during laparoscopy is **sinus bradycardia** (caused by rapid CO2 insufflation stretching the peritoneal membrane, triggering a vagovagal reflex).
- **Dermis Anatomy:** The dermis is the thick, highly vascularized inner layer of skin that contains sensory nerve endings and appendages (hair follicles, sweat glands, sebaceous glands). The epidermis is avascular.
- **Hypercalcemia Management:** Calcitonin quickly lowers serum calcium, but patients rapidly develop **tachyphylaxis (tolerance)** within 48–72 hours. It is for acute, short-term management only, *not* life-long treatment.
- **Soft Tissue Sarcoma Metastasis:** Retroperitoneal and abdominal soft tissue sarcomas primarily metastasize via the hematogenous route to the **LUNGS** (not the liver). Lymphatic spread is rare.
- **Cadaveric Organ Donation:** Strictly requires that the donor has suffered **irreversible** brain injury meeting legal and clinical criteria for brain death.
- **Surgical Lasers: Not all lasers cause superficial corneal damage**. Safety and depth of tissue/corneal interaction depend strictly on the specific wavelength (e.g., CO2 lasers damage the cornea, but Nd:YAG lasers pass through the cornea and damage the retina). Appropriate wavelength-specific safety glasses must be worn.
- **Diabetic Foot Infections:** The **'probe-to-bone' test** is the most reliable bedside method to diagnose osteomyelitis in a diabetic foot ulcer. If a sterile probe touches hard, gritty bone, osteomyelitis is highly likely.

Memory Pearls

- **Shock Classes by BP Drop:** BP does not drop until **Class III** (30-40% blood loss).
- **MTP Ratio:** 1 : 1 : 1 (PRBC : FFP : Platelets).
- **DIC Labs:** ↓ Platelets, ↓ Fibrinogen, ↑ PT/PTT, Bleeding + Clotting.
- **Urine Output Target:** 0.5 mL/kg/hr.
- **Reactionary Bleeding:** <24 hrs (slipped tie); **Secondary Bleeding:** 7-14 days (infection).

Sepsis

Core Concepts

- **Sepsis (Sepsis-3 Definition):** Life-threatening organ dysfunction caused by a dysregulated host response to infection.
- **Septic Shock:** A subset of sepsis where circulatory, cellular, and metabolic abnormalities are profound enough to significantly increase mortality (often >40%).
- **Pathophysiology:** Sepsis represents a loss of homeostasis triggered by an infection:
  - **Pro-inflammatory State:** Cytokine storm (TNF, IL-1, IL-6) leads to massive inflammation and endothelial dysfunction.
  - **Endothelial Dysfunction:** Causes excessive vasodilation, relative hypovolemia, and capillary leakage (tissue edema).

    - **Pro-coagulant State:** Inflammation and endothelial injury stimulate tissue factor expression and inhibit fibrinolysis (e.g., via inactivation of Protein C). This leads to microvascular thrombosis, impaired tissue perfusion, and potential Disseminated Intravascular Coagulation (DIC).
    - **Metabolic Impact:** Severe systemic inflammatory response syndrome (SIRS), such as that caused by peritonitis, throws the body into a severe hypermetabolic state, drastically **increasing** caloric and energy requirements.
- **Tumor Necrosis Factor (TNF):** A highly **pro-thrombotic** mediator in sepsis; it promotes coagulation and inhibits natural anticoagulant pathways. It is *not* an anticoagulant.

Diagnosis / Clinical Features

- **General Presentation:**
    - **Tachypnea** is the most common and earliest clinical sign (present in ~99% of cases). Do not rely solely on the presence of a fever to suspect sepsis.
    - Tachycardia, altered mental status, and oliguria are also highly prevalent.
- **Body's Response to Shock:**
    - **Sympathetic Activation:** Release of epinephrine/norepinephrine causes tachycardia and vasoconstriction.
    - **RAAS and ADH Activation:** Promotes sodium and water retention to preserve intravascular volume.
    - **Blood Flow Redistribution:** Shunts blood away from the skin, GI tract, and kidneys to preserve the brain and heart, leading to pallor, delayed capillary refill, and oliguria.
- **Common Sources & Pathogens:**
    - **Respiratory (35%)** and **Urinary Tract (35%)** are the most common primary sources.
    - **Gram-Negative Sepsis:** The urinary tract is the most common source of Gram-negative sepsis in the elderly. *Escherichia coli* is overwhelmingly the most common causative organism for septic shock originating from the GI or urinary tracts.
- **Surgical Risk Factors:** Advanced age, immunosuppression (steroids, chemotherapy), emergency operations, prolonged procedures, contaminated fields (bowel perforation), and indwelling devices (central lines, catheters).

Investigations

- **Serum Lactate:** A critical marker of tissue hypoperfusion and anaerobic metabolism.
    - < 1 mmol/L: Normal
    - > 2 mmol/L: Abnormal / Diagnostic threshold for shock
    - ≥ 4 mmol/L: Severe tissue dysoxia; triggers immediate aggressive resuscitation bundle
- **Blood Cultures:** Must be obtained **before** starting antimicrobial therapy, provided this does not delay antibiotics by more than 45 minutes. *Note:* Cultures are frequently **negative** in 30-50% of patients with severe sepsis or septic shock.

- **ScvO2 (Central Venous Oxygen Saturation):** Reflects the balance between oxygen delivery and oxygen consumption. Target > 70%. If low, interventions include fluids, packed red blood cells (target Hct > 30%), or inotropes (dobutamine).

Management

- **Hour-1 Resuscitation Bundle:** ("Time Zero" = time of presentation/recognition)
  1. **Measure lactate level** (re-measure if initial is > 2 mmol/L).
  2. **Obtain blood cultures** prior to administering antibiotics.
  3. **Administer broad-spectrum IV antibiotics** (ideally within 1 hour; delaying significantly increases mortality).
  4. **Rapid IV fluid resuscitation:** Give **30 mL/kg of crystalloid fluids** for hypotension or if lactate is ≥ 4 mmol/L.
     - *Fluid note:* Ringer's Lactate contains Sodium, Chloride, Potassium, Calcium, and Lactate. It does **NOT** contain Magnesium.
  5. **Apply vasopressors** if hypotensive during or after fluid resuscitation to maintain a MAP ≥ 65 mmHg.
- **Source Control:** Anatomic source control (e.g., draining an abscess, resecting ischemic bowel, removing an infected device) must be achieved **as rapidly as possible**. It must *never* be delayed to "stabilize the patient."
- **Vasopressor Therapy: Norepinephrine** is the first-line vasopressor for fluid-refractory septic shock. Dopamine is not recommended as first-line due to arrhythmia risks.
- **Corticosteroids:** Administer IV hydrocortisone *only* if adequate fluid resuscitation and vasopressor therapy are unable to restore hemodynamic stability.
- **Glucose Control:** Target an upper blood glucose level of ≤ 180 mg/dL to prevent hypoglycemia while avoiding severe hyperglycemia.

Relevant Guidelines

- **Sepsis-3 Definitions & Pathways:**
  - **Clinical Criteria for Sepsis:** Suspected or documented infection plus an acute change in the **SOFA score of ≥ 2 points**. (A SOFA score of ≥ 2 reflects an overall mortality risk of ~10% in a general hospital population).
  - **qSOFA (Quick SOFA):** A prompt used outside the ICU to identify patients at high risk of poor outcomes (Score ≥ 2 is positive). Includes:
    1. Systolic BP < 100 mmHg
    2. Altered Mental Status (GCS < 15)
    3. Tachypnea (Respiratory Rate ≥ 22 breaths/min)
  - **Clinical Criteria for Septic Shock:**
    1. Persisting hypotension requiring **vasopressors to maintain a MAP ≥ 65 mmHg** AND
    2. **Serum lactate level > 2 mmol/L**
    3. *Despite adequate fluid resuscitation.*

- **The SOFA (Sequential Organ Failure Assessment) Score:** Evaluates exactly **six** organ systems.
  1. **Respiratory:** PaO2/FiO2 ratio
  2. **Coagulation:** Platelet count
  3. **Liver:** Bilirubin levels
  4. **Cardiovascular:** MAP or dose of vasopressors required
  5. **Central Nervous System:** Glasgow Coma Scale (GCS)
  6. **Renal:** Creatinine levels or Urine Output
  - *Crucial Distinction:* The **Renin-Angiotensin System** is NOT part of the SOFA score.

Complications / Prognosis

- **Multiple Organ Dysfunction Syndrome (MODS):** Represents a continuum of progressive physiological derangements. The mortality rate is **extremely high** and directly correlates with the number of organ systems failing (e.g., 1 organ = ~22% mortality; 4+ organs = >70% mortality). Aggressive antibiotic use does *not* make this a "low mortality" condition.
- **DIC (Disseminated Intravascular Coagulation):** Widespread microvascular clotting consumes platelets and clotting factors, drastically increasing bleeding risk.

Past-Paper High Yield

- **Diagnostic Thresholds:** Under Sepsis-3, organ dysfunction is clinically identified by an acute change in the total SOFA score of **≥ 2 points** (NOT ≥ 4 points).
- **SOFA Components:** The SOFA score explicitly evaluates the respiratory, coagulation, liver, cardiovascular, central nervous system, and renal systems. It **does NOT evaluate the renin-angiotensin system**.
- **Definition of Septic Shock:** True septic shock strictly requires *both* the need for vasopressors to maintain MAP ≥ 65 mmHg *and* a serum lactate > 2 mmol/L despite adequate fluid resuscitation.
  - *Legacy Classification Trap:* If a patient presents with severe hypotension that normalizes completely and remains stable after a rapid fluid bolus, they have **Severe Sepsis** (under historical Sepsis-2 criteria) but do *not* meet the criteria for Septic Shock, as they did not require vasopressors.
- **First-line Pressor:** If a patient receives 30 ml/kg of crystalloid fluids and remains hypotensive (e.g., BP 80/60 mmHg), they have fluid-refractory shock. The immediate next step is to **initiate a norepinephrine infusion**. (Dopamine is incorrect).
- **Antibiotic Timing:** Broad-spectrum antibiotics MUST be administered within **1 hour** of recognition. Delaying them up to 6 hours to obtain cultures is completely incorrect and universally increases mortality.
- **Source Control:** Source control must be prioritized alongside resuscitation and should **NOT be delayed** to stabilize the patient.
- **Microbiology:** *Escherichia coli* is the most overwhelmingly common causative organism for septic shock originating from the GI or urinary tract, particularly in the elderly. Furthermore, it is false to claim that 90% of patients have positive blood cultures; up to 30-50% will have negative cultures.

- **Fluid Composition:** Ringer's lactate solution contains Sodium, Chloride, Potassium, Calcium, and Lactate. It does **NOT** contain Magnesium.
- **Metabolism:** SIRS/Sepsis (e.g., from peritonitis) causes a hypermetabolic state that drastically **increases** energy requirements. (Do not select answers suggesting energy requirements decrease).
- **Cytokines:** TNF is highly **pro-thrombotic** (it is NOT a systemic anticoagulant).
- **Miscellaneous General Surgery Exam Trivia (Mapped to this block):**
  - **Therapeutic Radiation:** Toxic to rapidly dividing bone marrow cells, causing **lymphopenia** (NOT lymphocytosis). Chronic effects include tissue fibrosis, necrosis, telangiectasia, and secondary malignancies.
  - **Hemorrhoids:** Second-degree hemorrhoids (prolapse with straining but reduce spontaneously) should initially be managed **conservatively** with diet (fiber/water) and topical agents.

Memory Pearls

- **Sepsis-3 Septic Shock = MAP < 65 (requiring pressors) + Lactate > 2 (despite fluids).**
- **Hour-1 Sepsis Bundle = Lactate → Cultures → Antibiotics → 30ml/kg Fluids → Vasopressors.**
- **SOFA = 6 Systems (Lungs, Liver, Kidneys, Brain, Heart/Vessels, Platelets).** No endocrine or RAAS components.

Bleeding and blood transfusion

Core Concepts

- **Rationale for Transfusion:** Improving oxygen-carrying capacity of the blood to prevent tissue hypoxia (organs most sensitive are the heart and brain).
- **Oxygen Delivery (O2 Flux):** Cardiac Output × (Hemoglobin × Oxygen Saturation).
- **Blood Components and Preparation:** Prepared via differential centrifugation in a closed system.
  - **First Centrifugation (Soft Spin):** Separates heavier RBCs from platelet-rich plasma (PRP).
  - **Second Centrifugation (Hard Spin):** Separates PRP into Platelet Concentrate and Platelet-Poor Plasma (FFP).
- **Storage and Composition:**
  - **Whole Blood:** Stored at 4°C for up to 35 days. Platelets and coagulation factors become inactive after 3–5 days. Must be ABO identical.
  - **Packed Red Blood Cells (PRBCs):** Stored at 4°C for up to 42 days. One unit (10 cc/kg) increases Hb by ~2.5 g/dL. Must be ABO compatible.
  - **Platelets:** Stored at 20–24°C (room temperature) for up to 5 days. Contains donor leukocytes and cytokines. One unit/10 kg increases platelet count by 50,000/µL. Donor/recipient must be ABO identical.
  - **Fresh Frozen Plasma (FFP):** Contains all coagulation factors. Stored at -18°C for up to 12 months. Requires ABO compatibility, but **Rh compatibility is not required** (lacks RBCs).

- **Cryoprecipitate:** Precipitate formed when FFP is thawed at 4°C; refrozen and stored up to 1 year. Rich in Fibrinogen, von Willebrand Factor, Factor VIII, and Factor XIII.

Diagnosis / Clinical Features

- **Acute Hemolytic Transfusion Reaction (AHTR):**
  - Triggered by ABO/Rh incompatibility (usually due to a labeling or clerical error).
  - Pathophysiology: Pre-formed recipient antibodies activate complement, causing rapid intravascular hemolysis. Can occur with just 1–2 cc of RBCs.
  - **Awake patient:** Fever, chills, severe back/flank pain, dyspnea, hypotension, dark urine (hemoglobinuria), and pallor.
  - **Anesthetized patient (in the OR):** Classic symptoms are masked. The most reliable signs of AHTR under general anesthesia are **unexplained hypotension** and **sudden, diffuse microvascular bleeding/oozing** from the surgical field (due to acute DIC).
- **Febrile Non-Hemolytic Transfusion Reaction (FNHTR):**
  - Defined by a temperature rise of >1°C.
  - Caused by recipient alloantibodies directed against donor HLA antigens/leukocytes and accumulated cytokines.
- **Allergic Reaction:** Ranges from mild urticaria/wheezing to anaphylaxis. Classically occurs in IgA-deficient patients receiving blood products containing IgA.
- **Transfusion-Related Acute Lung Injury (TRALI):**
  - ARDS-like pulmonary syndrome occurring 1–6 hours post-transfusion.
  - Caused by donor WBC antibodies inducing pulmonary leukostasis. Carries high mortality.

Investigations

- **Blood Bank Workup for Hemolytic Reactions:**
  - Direct Antiglobulin Test (DAT/Coombs test) – positive in AHTR.
  - Visual check of plasma for free hemoglobin (hemoglobinemia).
  - Urine analysis for hemoglobinuria.
  - Repeat crossmatch and blood group typing.
  - Baseline coagulation panel (PT, PTT, Fibrinogen, D-dimer) to assess for DIC.
- **Viscoelastic Whole-Blood Assays (TEG® / ROTEM®):**
  - Provides continuous graphic display of clot initiation, strength, and lysis.
  - Allows targeted transfusion:
    - **Prolonged R-time (clotting time):** Indicates coagulation factor deficiency → Give FFP.
    - **Decreased Alpha-angle/K-time:** Indicates poor fibrinogen/fibrin polymerization → Give Cryoprecipitate.
    - **Decreased MA (Maximum Amplitude):** Indicates poor clot strength → Give Platelets (or Cryoprecipitate).
    - **Elevated Ly30:** Indicates hyperfibrinolysis → Give Tranexamic Acid (TXA).

Management

- **AHTR Management:**
  - **IMMEDIATELY STOP the transfusion.**
  - Assess ABCs, maintain IV access, and vigorously hydrate with normal saline or Lactated Ringer's to maintain urine output.
  - Administer diuretics to protect renal tubules from free hemoglobin.
  - Return remaining blood and tubing to the blood bank.
- **FNHTR Management:** Stop transfusion; administer antipyretics (and corticosteroids for severe reactions). Future transfusions should use leukoreduced products.
- **Management of Massive Hemorrhage:**
  - **Hemostatic Resuscitation:** Early use of balanced ratios (1:1:1 of PRBC : FFP : Platelets). High FFP-to-RBC and Platelet-to-RBC ratios significantly reduce mortality and multi-organ failure.
  - Do not rely solely on hemoglobin triggers during active hemorrhagic shock; initiate rapid replacement to prevent dilutional coagulopathy.
  - Administer Tranexamic Acid (TXA) early (1 g loading dose, followed by 1 g over 8 hours).
- **Specific Reversals & Scenarios:**
  - **Warfarin Toxicity with Active Bleeding:** Administer FFP (or Prothrombin Complex Concentrate) + Vitamin K.
  - **Hemophilia A:** FFP is contraindicated for volume or routine replacement; specific recombinant Factor VIII or Cryoprecipitate is required.
  - **Upper GI Bleed in Cirrhosis:** Target restrictive Hb (7–8 g/dL). Over-transfusion (>9 g/dL) directly increases portal venous pressure and exacerbates variceal rebleeding.

Relevant Guidelines

- **American Association of Blood Banks (AABB) Transfusion Triggers:**
  - **Hb < 7 g/dL:** Transfusion recommended.
  - **Hb 7–8 g/dL:** Restrictive strategy; consider transfusion only if symptomatic (chest pain, orthostatic hypotension, tachycardia unresponsive to fluid, CHF).
  - **Hb 8–10 g/dL:** Transfusion generally not indicated (unless specific active ischemia or massive ongoing bleeding).
  - **Hb > 10 g/dL:** Transfusion NOT indicated.
- **Massive Transfusion Protocol (MTP) Activation & Targets:**
  - **Activation Criteria:** Actual or anticipated need for ≥ 4 units RBC in < 4 hours + hemodynamic instability; OR replacement of >50% blood volume within 3 hours; OR loss of one total blood volume (~70 mL/kg) in 24 hours.
  - **Laboratory Targets during MTP:**
    - Hemoglobin > 8–10 g/dL
    - Platelets > 50,000/µL (>100,000/µL for head trauma)
    - PT / PTT < 1.5 × control

    - Fibrinogen > 1.0 g/L (treat with Cryoprecipitate if lower)
- **ATLS Classification of Hemorrhage (Adults):**
    - **Class I:** Blood loss up to 15% (<750 mL). Normal HR, BP, Pulse Pressure.
    - **Class II:** Blood loss 15–30% (750–1,500 mL). Tachycardia (>100), Normal BP, Decreased Pulse Pressure.
    - **Class III:** Blood loss 30–40% (1,500–2,000 mL). Tachycardia (>120), Decreased BP, Confused mental status.
    - **Class IV:** Blood loss >40% (≥ 2,000 mL). HR >140, Decreased BP, Lethargic/negligible urine output.

Complications / Prognosis

- **The Lethal Triad of Trauma:** A self-reinforcing, deadly cycle comprising:
    1. **Hypothermia (<35°C):** Decreases enzymatic activity of coagulation factors (by 50% at 34°C) and prevents platelet activation (almost eliminated at 30°C).
    2. **Acidosis (Base Deficit ≥ 6):** Interferes with the assembly of coagulation factor (Xa/Va) complexes. Contributes *more* to coagulopathy than hypothermia and is harder to reverse.
    3. **Coagulopathy (ACOT):** Driven by hemodilution (crystalloids/PRBCs replacing whole blood) and consumption (DIC).
- **Massive Transfusion Metabolic Complications:**
    - **Hypocalcemia:** Citrate anticoagulant in stored blood rapidly chelates serum calcium. Presents with hypotension, narrow pulse pressure, and prolonged QT interval. Treat with slow IV 10% calcium gluconate.
    - **Hyperkalemia:** Potassium leaks from stored RBCs. Exacerbated by concurrent hypothermia and acidosis.
    - **Dilutional Thrombocytopenia:** Massive PRBC transfusion (which lacks platelets) drastically dilutes circulating platelets.
- **Transfusion-Transmitted Infections:**
    - **Bacterial Contamination:** Platelets are the most common source because they are stored at room temperature (20–24°C), favoring Gram-positive (Staph/Strep) growth.
    - **Cytomegalovirus (CMV):** Intracellular virus residing in donor leukocytes. Risk is virtually eliminated by leukoreduction filters.
    - Viral risks are now exceptionally low (HIV 1:1.8M, HCV 1:1.6M) due to Nucleic Acid Testing (NAT). The most common serious hazard (53%) is administrative labeling errors (wrong blood).
- **Heparin-Induced Thrombocytopenia (HIT):**
    - Immune-mediated IgG response against Heparin-PF4 complexes.
    - Causes paradoxical severe arterial and venous thrombosis.
    - Platelet counts drop by ~50% from baseline but **rarely drop below 20,000/µL**.
    - Stop all heparin immediately and initiate a direct thrombin inhibitor.

Past-Paper High Yield

- **Most common reaction:** Febrile non-hemolytic transfusion reaction (FNHTR) is the single most common adverse reaction, caused by recipient antibodies against donor leukocytes/cytokines.
- **Platelet storage & infection:** Because platelets are stored at room temperature, they are highly conducive to bacterial growth and are the *most common* source of transfusion-transmitted bacterial infections.
- **FFP indications & limitations:** FFP is indicated for rapid reversal of warfarin in active bleeding (provides all coagulation factors). It is *never* indicated for routine volume repletion. FFP contains no platelets; thus, it is completely useless for a patient with a prolonged bleeding time (which indicates a primary hemostasis/platelet defect).
- **Massive Transfusion definition traps:** Massive transfusion is defined quantitatively (volume/speed of RBC replacement). Although FFP is needed to treat the resulting coagulopathy, the *need for FFP* is not part of the diagnostic definition of massive transfusion.
- **Citrate Toxicity:** Causes **HYPOcalcemia** (not hypercalcemia) due to calcium chelation.
- **Dilutional effect:** Transfusion of PRBCs *decreases* platelet count via dilutional thrombocytopenia.
- **Chemotherapy limits:** Severe hematological suppression (myelosuppression causing neutropenia/thrombocytopenia) is the most frequent condition forcing alteration or delay of chemotherapy regimens.
- **Transplant prerequisites:** ABO blood group compatibility is the most absolute, critical initial barrier for cadaveric organ transplants to prevent hyperacute rejection.
- **CMV transmission:** Occurs specifically via donor WBCs.
- **Hemolytic reaction in the OR:** Classic signs (flank pain, chills, fever) are masked under anesthesia. Sudden, unexplained hypotension and diffuse microvascular oozing (DIC) are the classic presentations.

Memory Pearls

- **B**acteria love **P**latelets (Stored at Room Temp).
- **C**MV lives in the **C**ells (White blood cells).
- **C**itrate **C**helates **C**alcium → Hypocalcemia (Long QT).
- Transfusing **P**RBCs massively drops **P**latelets (Dilutional Thrombocytopenia).
- **Lethal Triad: H**ypothermia, **A**cidosis, **C**oagulopathy (HACk). Acidosis is the most detrimental to the coagulation cascade.

Gastrointestinal

Exam Map

YIELD TIER	TOPIC WEIGHT	PRIMARY REVISION TARGETS
Tier 1 (15+ Qs)	Gallstones, IBD, Gastric Cancer, Bowel Obstruction, Diverticular Disease	Biliary obstruction/infection, UC/Crohn's surgical indications, EUS staging, Strangulation & Volvulus, Hinchey classification.
Tier 2 (8-14 Qs)	Liver Tumors, Spleen, GERD, Obesity, Pancreatitis, Appendicitis, Colorectal Ca	HCC biopsy/transplant criteria, Vaccine timing & OPSI, Manometry & pH impedance, RYGB vs. Sleeve, Ranson's criteria, Alvarado score, Polyp genetics.
Tier 3 (1-7 Qs)	Pancreatic Ca/Cysts, Ischemia, Anorectal, NETs, Hydatid Cysts	Resectability criteria, Cyst fluid CEA, SMA embolism, Dentate line anatomy, Gastrinoma/Insulinoma diagnosis, PAIR protocol.

- **Gold Standard Imaging Paradigms:** Endoscopic Ultrasound (EUS) is universally tested as the most accurate modality for T-stage (mural depth) and N-stage in GI malignancies (Esophageal, Gastric). CT Abdomen/Pelvis with contrast is the definitive next step for acute mechanical obstructions, diverticulitis, and equivocal appendicitis.
- **Strict Clinical Contraindications:** Diagnostic colonoscopy and barium enema are absolutely contraindicated during *acute diverticulitis* (perforation risk). Percutaneous drainage without scolicidal agents is contraindicated in *hydatid cysts* (anaphylaxis/seeding), and routine drainage is avoided in *amebic liver abscesses* (managed medically).
- **Abscess & Peritonitis Algorithms:** Localized collections (Hinchey II diverticulitis, appendiceal/psoas abscesses) mandate **image-guided percutaneous drainage + IV antibiotics**. Diffuse purulent/fecal peritonitis (Hinchey III/IV) requires emergent surgery (Hartmann’s procedure).
- **IBD Operative Decision Points:** Acute severe Ulcerative Colitis management hinges on the "Day 5 Rule" (failure of IV steroids by day 5 mandates Subtotal Colectomy or rescue biologics). Crohn's disease surgery is explicitly reserved for *complications* (strictureplasty, fistulas) to preserve bowel length.
- **Obstruction & Volvulus Management:** Sigmoid volvulus allows initial endoscopic decompression (flexible sigmoidoscopy) followed by elective resection. Cecal volvulus explicitly mandates primary surgical resection (Right Hemicolectomy).
- **Scoring Systems & Criteria:** High-yield exam traps involve exclusions in common scores. Ranson's criteria (Pancreatitis) *excludes* Amylase/Lipase. Child-Pugh score (Cirrhosis) uses PT/INR, *not* PTT or AST/ALT. Alvarado score guides appendicitis management (CT imaging for equivocal scores of 4-6).
- **Perioperative Priorities:** Elective splenectomy requires vaccinations against encapsulated organisms (SHiN) **2 to 4 weeks prior** to surgery. Elective colorectal surgery prophylaxis must cover *both* Gram-negative bacilli and anaerobes (never monotherapy with a cephalosporin).

- **Bariatric Surgical Physiology:** Roux-en-Y gastric bypass causes dumping syndrome (useful for "sweet eaters"), cures GERD, but uniquely risks internal hernias. Sleeve gastrectomy removes ghrelin, cannot cause internal hernias, but *worsens* GERD.
- **Lower GI Bleed Distinctions:** Massive, acute, painless arterial bleeding = Diverticulosis or Angiodysplasia (not uncomplicated CRC). Painless, bright red dripping blood = Internal hemorrhoids (visceral innervation). Severe tearing pain with minimal blood = Anal fissure.
- **Surgical Oncology Genetics:** FAP (APC gene mutation) mandates prophylactic total proctocolectomy. Diffuse gastric cancer (linitis plastica) is linked to CDH1/E-cadherin and blood type A. MEN-1 differentiates a standard Gastrinoma (ZES) from one presenting with concurrent hypercalcemia.

GERD, hiatal hernia & achalasia

Core Concepts

- **Gastroesophageal Reflux Disease (GERD) Definition:** A condition developing when the retrograde flow of gastric contents into the esophagus causes troublesome symptoms and/or complications. It represents an imbalance between injurious refluxed material and physiological defensive mechanisms.
- **Anti-Reflux Defensive Mechanisms:**
  - **Lower Esophageal Sphincter (LES):** The primary barrier to reflux. It is a high-pressure zone created by:
    - **Intrinsic Smooth Muscle Tone:** The competence of the LES is primarily maintained by the *intrinsic myogenic resting tone* of the sphincter's smooth muscle (not merely by extrinsic intra-abdominal pressure).
    - **Extrinsic Factors:** The diaphragmatic crura act synergistically as an external sphincter (the 'pinchcock' effect) to increase pressure during inspiration.
    - **Anatomical Factors:** Angle of His, clasp and sling fibers, and an adequate length of intra-abdominal esophagus.
  - **Clearance & Buffering:** Esophageal peristalsis, gravity, salivary production (buffering acid), and epithelial barrier function (tight junctions).
  - **Gastric Factors:** Normal fundic compliance and proper gastric emptying.
- **Pathophysiology of Reflux:**
  - **Transient LES Relaxations (TLESRs):** The primary physiological mechanism for GERD. TLESRs are triggered by gastric fundus distention (e.g., after eating large meals), which actually *increases* the likelihood of acid reflux.
  - **Mechanically Defective LES:** Defined manometrically by one or more of the following:
    - Resting LES pressure < 6 mmHg
    - Overall sphincter length < 2 cm
    - Intra-abdominal esophageal length < 1 cm
- **Physiological Modulators of LES Tone:**

- *Increases tone:* Gastrin (prevents reflux during gastric digestion), upright posture, weight loss.
- *Decreases tone:* Alcohol, high-fat meals, peppermint, calcium channel blockers.

- **GERD Phenotypes:**
  - Non-Erosive Reflux Disease (NERD): Most common (60-70%).
  - Erosive Esophagitis (EE): Occurs in ~30%.
  - Barrett's Esophagus (BE): Occurs in 6-15%.

Diagnosis / Clinical Features

- **Typical GERD Symptoms:** Heartburn, acid regurgitation, dysphagia, odynophagia, hypersalivation (water brash).
- **Atypical GERD Symptoms:** Chest pain, cough, adult-onset asthma (50% of adult asthma patients have esophagitis or abnormal acid exposure), chest infections/lung fibrosis, otitis, laryngitis, sinusitis, dental erosions.
- **Alarming Symptoms:** Anemia, weight loss, dysphagia/odynophagia.
- **Achalasia Presentation:** Progressive, severe dysphagia to *both* solids and liquids, frequent regurgitation of undigested food, and weight loss.
- **Esophageal Cancer Presentation:** Progressive dysphagia to solid foods is the hallmark symptom. The vast majority present with advanced, late-stage disease (Stage III or IV) because the esophagus lacks a serosa and has a rich lymphatic supply.

Investigations

- **Proton Pump Inhibitor (PPI) Trial:** Standard first-line diagnostic and therapeutic approach for typical GERD symptoms. Routine 24-hour pH monitoring is *not* required before initiating empiric PPI therapy.
- **Endoscopy (EGD) with Biopsy:**
  - Initial diagnostic evaluation tool for structural abnormalities; mandatory before anti-reflux surgery.
  - Most patients (~60-70%) with classic GERD symptoms will have entirely normal esophageal mucosa on endoscopy (NERD).
  - Required in suspected achalasia strictly to rule out pseudoachalasia (malignancy).
- **24-Hour pH Impedance Study:**
  - The most precise test to quantify reflux (measures total time, supine/upright time, number of episodes).
  - Probe is placed 5 cm above and 5 cm below the LES.
  - *Indications:* Atypical symptoms, absence of esophagitis on EGD, atypical/refractory response to medical treatment.
  - *Preparation:* Stop H2 blockers 3 days prior and PPIs 14 days prior.
- **High-Resolution Esophageal Manometry:**
  - *GERD:* Mandatory preoperatively to assess sphincter status and tailor the wrap according to esophageal pump function.

- *Achalasia:* The definitive, gold standard, and most sensitive diagnostic test. Definitively shows absence of peristalsis (aperistalsis) in the esophageal body and incomplete relaxation of a hypertensive LES.

- **Barium Swallow:**
  - Used to assess hiatal hernia size/type and esophageal peristalsis.
  - In GERD, it demonstrates reflux in only 40% of cases (a negative test does not exclude GERD).
  - In Achalasia, shows a dilated esophagus with classic tapering "bird's beak" appearance at the GE junction (suggestive, but manometry is still required for confirmation).

Management

- **Medical Therapy:**
  - Intensive PPI therapy (2-3 months) plus lifestyle modifications (avoiding large/fatty meals, weight loss). PPIs reduce gastric acidity by 80-90%.
  - *Limitation:* 80% of patients experience recurrent symptoms 6 months after stopping treatment.
- **Surgical Indications for GERD:**
  - Complications of reflux not responding to medical treatment.
  - Symptoms interfering with lifestyle despite medical therapy.
  - Presence of a hiatal hernia.
  - Patient preference to avoid lifelong medical therapy (e.g., young age).
  - Markedly hypotensive LES on manometry.
- **Pre-Operative Prerequisites:** All patients must receive an intensive medical trial (2-3 months), and both endoscopy and esophageal manometry are *mandatory* before considering surgery.

Relevant Guidelines

- **Diagnostic Pathway Based on Symptoms:**
  - *No Alarm Symptoms:* Initiate a 6-week empiric trial of PPIs + lifestyle modifications. If no improvement, proceed to endoscopy.
  - *Alarm Symptoms Present (Anemia, Weight Loss, Dysphagia):* Immediate endoscopy is indicated.
- **Los Angeles (LA) Grading System of Esophagitis:**
  - **LA-A:** ≥ 1 mucosal break, ≤ 5 mm, does not extend between mucosal folds.
  - **LA-B:** ≥ 1 mucosal break, > 5 mm, does not extend between mucosal folds.
  - **LA-C:** Mucosal breaks extending between folds, involving < 75% of the circumference.
  - **LA-D:** Mucosal break(s) involving > 75% of the esophageal circumference.
- **Hill Grading System of Hiatal Hernia (Endoscopic Retroflexion):**
  - **Hill I:** Normal tissue fold tightly approximated around the endoscope.
  - **Hill II:** Slight opening and mild laxity of the tissue fold.
  - **Hill III:** Marked reduction in muscular collar with a clear opening around the endoscope.
  - **Hill IV:** Absolute absence of a muscular fold; wide open GE junction; endoscope sits in a large hiatal hernia cavity.

Operative / Procedural Notes

- **Principles of Anti-Reflux Surgery:**
  - The primary goal is to preserve the anti-reflux valve while maintaining the patient's ability to swallow normally and belch (to relieve gaseous distension).
  - Ensure the wrap is placed over the sphincter, not the gastric body.
  - Only use the *gastric fundus* for the wrap (normal swallowing triggers vagally mediated relaxation of both the LES and fundus; using the fundus ensures the wrap relaxes appropriately).
  - Avoid injury to the vagus nerve (can cause failure of the sphincter to relax).
- **Basic Surgical Steps:**
  1. Restore intra-abdominal esophageal length.
  2. Excise the hernial sac.
  3. Posterior Cruroplasty (suturing the diaphragmatic crura posterior to the esophagus).
  4. Wrap the intra-abdominal esophagus with the fundus (tension-free).
- **Types of Fundoplication:**
  - **Nissen:** Complete 360^° wrap (associated with 90% long-term success but higher risk of post-op dysphagia/gas bloat).
  - **Toupet:** Partial 270^° posterior wrap.
  - **Dor:** Partial 180^°-200^° anterior wrap.
- **Collis-Nissen:** An esophageal lengthening procedure utilized if the patient has a foreshortened esophagus.

Complications / Prognosis

- **Barrett's Esophagus (BE):** Replacement of normal squamous epithelium with columnar epithelium (intestinal metaplasia). Premalignant condition requiring regular endoscopic follow-up.
  - Risk of dysplasia/cancer is 0.2%–0.5% per year (40x higher than the general population).
  - Virtually all esophageal adenocarcinomas arise from BE. 1/3 of patients with BE present with malignancy.
- **Esophageal Cancer Prognosis:** Overall survival remains exceedingly poor. The 5-year survival rate generally hovers around **20%** (not 60%). Lymph node metastasis frequently involves the celiac nodes for distal tumors.
- **Complications of Fundoplication:**
  - **Dysphagia:** Very common early post-op (19-90%), but persists long-term in only 5-10%.
  - **Gas Bloating Syndrome:** Inability to belch. Occurs if the surgical valve is too tight, preventing retrograde venting during physiological gastric fundus distention.
  - Intrathoracic migration of the wrap (herniation).
  - Failure of the procedure (5-10%).

Past-Paper High Yield

- **LES Pathophysiology:** The intrinsic myogenic tone of the smooth muscle is the primary mechanism for resting LES competence.
- **Gastric Distension Trap:** Gastric fundus distension (eating large meals) does *not* decrease reflux; it actively INCREASES it by triggering TLESRs.
- **Medical Management of GERD:** Routine 24-hour pH monitoring is FALSELY claimed to be required before starting PPIs. An empiric PPI trial is standard care.
- **Endoscopy Findings in GERD:** It is FALSE that esophagitis is found in >90% of GERD endoscopies. Approximately 60-70% have NERD with a completely normal mucosal appearance.
- **Achalasia Diagnostics:** While a barium swallow showing a 'bird beak' is classic, **high-resolution esophageal manometry** is the definitive, gold standard, and most sensitive diagnostic test. You *must* select manometry if asked for the diagnostic confirmation.
- **Esophageal Cancer Survival:** Do not be tricked by optimistic numbers; 5-year survival is ~20% (poor prognosis), usually presenting late with solid food dysphagia.
- **Substances Modulating Tone:** Gastrin *increases* LES tone. Alcohol, fatty foods, peppermint, and CCBs *decrease* LES tone.

Memory Pearls

- **Endoscopy pre-op:** Can't cut without an EGD and a Manometry first.
- **Typical GERD without alarm signs:** Pop a PPI for 6 weeks, no scope needed yet.
- **Achalasia:** "Bird's beak" on barium = think it. Manometry (aperistalsis + failed LES relaxation) = confirm it. Endoscopy = rule out cancer.
- **The Wrap:** Nissen = 360^° (full circle). Toupet = 270^° (two-thirds, posterior). Dor = 180^° (half, anterior door). Use the *fundus* only!

Esophageal cancer

Core Concepts

- **Pathophysiology of Spread:** Local lymph node invasion occurs exceptionally early and rapidly because esophageal lymphatics are located within the **lamina propria** (unlike the rest of the GI tract where they are deep in the submucosa).
- **Squamous Cell Carcinoma (SCC):**
  - **Location:** Predominantly in the midportion of the esophagus.
  - **Epidemiology:** Dominates (90% of cases) in the high-risk "esophageal cancer belt" (Northern Iran to North-Central China) and Southern/Eastern Africa. Male-to-female ratio is 3:2.
  - **Risk Factors:** Smoking, excessive alcohol, dietary factors (high-temperature beverages, red meat, low folate/zinc, N-nitrous compounds, aflatoxin, pickled vegetables), achalasia, caustic strictures, atrophic gastritis, tylosis, and poor oral hygiene.
- **Adenocarcinoma (ACA):**
  - **Location:** Majority located near the gastroesophageal junction (GEJ).

- **Epidemiology:** Male-to-female ratio is 10:1.
- **Risk Factors:** GERD, Barrett's metaplasia, smoking, alcohol, obesity, and *Helicobacter pylori* infection.

Diagnosis / Clinical Features

- **Presentation:** Early intramucosal cancers are often asymptomatic or present with subtle, transient "sticking" of solid foods or retrosternal burning.
- **Advanced Symptoms:**
  - Progressive solid food dysphagia.
  - Weight loss (due to dysphagia, diet changes, and tumor-related anorexia).
  - Regurgitation of saliva/food uncontaminated by gastric acid.
  - Hoarseness (recurrent laryngeal nerve involvement).
  - Chronic GI blood loss and aspiration pneumonia.
- **Red Flag:** Tracheobronchial fistula formation is a terminal complication; life expectancy is less than 4 weeks following its development. Massive upper GI hemorrhage indicates erosion into the aorta.

Investigations

- **Upper Endoscopy (EGD) & Biopsy:** Gold standard for diagnosis. Detects subtle plaques/nodules (early) or ulcerated/circumferential masses (advanced). Note: 17% of endoscopically "benign" looking lesions prove malignant on biopsy.
- **Chromoendoscopy:** Uses stains during EGD to highlight abnormal mucosa.
  - **Lugol's Iodine:** Normal squamous mucosa (rich in glycogen) stains dark brown. Neoplastic/dysplastic tissue remains unstained ("Lugol-voiding" areas).
- **Barium Esophagram:** Demonstrates mucosal lumen narrowing, stricture length, and classic "apple-core" filling defects in advanced circumferential carcinoma. Cannot assess mural depth.
- **Endoscopic Ultrasound (EUS):**
  - **Gold standard for T-staging (mural depth) and local N-staging.**
  - Capable of fine needle aspiration (FNA) of suspicious regional lymph nodes.
  - Visualizes 5 distinct sonographic layers: (1) Balloon/mucosa interface [hyperechoic], (2) Mucosa/lamina propria/muscularis mucosae [hypoechoic], (3) Submucosa [hyperechoic], (4) Muscularis propria [hypoechoic], (5) Adventitia [hyperechoic].
  - *Limitations:* Poor at assessing tracheobronchial invasion, unable to pass through tight stenoses, cannot detect distant metastases.
- **PET/CT Scan:** Recommended for staging systemic disease. 78% sensitivity for identifying nodal disease and distant metastasis.

Management

- **Nutritional Optimization:** Required prior to treatment.
  - **Crucial Pre-operative Caveat:** Percutaneous Endoscopic Gastrostomy (PEG) tubes **must be avoided** if the stomach is planned as a surgical conduit. PEG placement risks injuring the right

gastroepiploic artery, which is the vital vascular pedicle for the gastric pull-up. Use NG or jejunal feeding tubes instead.

- **Endoscopic Resection (EMR/ESD):** For highly selected superficial lesions (e.g., ≤ 15 mm, well-differentiated, < 500 µm submucosal invasion, negative margins, N0).
- **Primary Surgery:** Indicated for strictly early-stage disease (Stage 0, I, IIa / T1-T2, N0, M0).
- **Neoadjuvant Therapy + Surgery:** Standard of care for locally advanced, resectable disease (Stage IIB and III / T3 or node-positive, selected T4a).
- **Primary/Definitive Chemoradiation:** For Stage IV disease, unresectable tumors (T4b), or patients who are poor surgical candidates/refuse surgery.
- **Endoscopic Palliation:**
  - **Stents:** Self-expanding metal stents (SEMS); covered stents (silicone/polyurethane) are specifically beneficial for sealing tracheoesophageal fistulas. 95% effective for dysphagia.
  - **Photodynamic Therapy (PDT):** Injection of a photosensitizing agent activated by targeted laser light, causing selective tumor necrosis.
  - **Other:** Nd-YAG laser photoablation, thermal ablation, dilation, sclerotherapy.

Relevant Guidelines

- **TNM Staging (Slide-derived criteria):**
  - **T1:** Invades lamina propria or submucosa.
  - **T2:** Invades muscularis propria.
  - **T3:** Invades adventitia (beyond muscularis propria).
  - **T4a (Resectable):** Invades adjacent structures (pleura, pericardium, diaphragm).
  - **T4b (Unresectable):** Invades adjacent structures (trachea, bone, aorta).
  - **N1 / N2 / N3:** 1-2 nodes / 3-6 nodes / ≥ 7 nodes.
- **Stage-Directed Treatment Algorithm:**
  - **Stage 0, I, IIA:** Primary surgery.
  - **Stage IIB, III:** Trimodality therapy (Neoadjuvant chemoradiation followed by surgery).
  - **Stage IV:** Primary chemoradiotherapy.
- **Siewert Classification (GEJ Adenocarcinomas):**

TYPE	LOCATION EPICENTER	REQUIRED SURGICAL APPROACH	LYMPH NODE SPREAD
I (Distal esophagus)	1 cm to 5 cm above cardia	Esophagectomy (negative gastric margins)	Mediastinal & celiac
II (True cardia)	1 cm above to 2 cm below cardia	Esophagectomy (negative gastric margins)	Abdominal
III (Subcardial)	2 cm to 5 cm below cardia	Total gastrectomy (+/- esophagectomy)	Abdominal

Operative / Procedural Notes

- **Esophagectomy Principles:** Outcomes are similar between transthoracic (with thoracotomy) and transhiatal (without thoracotomy) approaches.
- **Conduit Options for Reconstruction:**
  - **Stomach (Most common):** Relies on the **right gastric** and **right gastroepiploic** artery pedicles.
  - **Left colon:** Ascending branch of left colic artery pedicle.
  - **Right colon:** Middle colic artery pedicle.
  - **Jejunal free graft:** Requires microvascular anastomosis.
- **Route of Reconstruction:** The new conduit is translocated most commonly via the posterior mediastinum, but can also be routed substernally, subcutaneously (anterior to sternum), or via the pleural spaces.
- **Endoscopic Mucosal Resection (EMR) Technique:** "Cap and snare" or injection method. A saline cushion is injected into the submucosal space to elevate the lesion away from the muscularis propria, preventing perforation during electrocautery snare resection.

Complications / Prognosis

- Unresectability criteria: Metastatic disease (peritoneal, lung, bone, adrenal, brain, liver), extraregional lymph node spread (paraaortic/mesenteric), or T4b local invasion (aorta, trachea, spine).
- 50% "cure rate" limit for locally advanced tumors undergoing trimodality therapy due to inadequate pathologic response and occult distant metastases.

Past-Paper High Yield

- **Locally Advanced Management (T3N0):** The definitive next step in management for T3N0 esophageal cancer is **neoadjuvant chemoradiation therapy followed by surgical resection**. Primary esophagectomy alone has unacceptably high recurrence rates for T3 tumors. Gastrectomies are reserved for gastric or Siewert III tumors.
- **Staging Accuracy (EUS): Endoscopic Ultrasound (EUS)** is tested heavily as the *single most accurate modality* for determining the exact T-stage (mural depth) and N-stage in esophageal cancer. It uniquely delineates the mucosa, submucosa, muscularis, and adventitia. CT is for distant/gross invasion; PET is for metabolic/systemic spread; Barium evaluates stricture length/luminal diameter, not mural depth.

Memory Pearls

- **Lymphatic mapping:** "Esophagus breaks the GI rules" – lymphatics start in the *lamina propria*, leading to incredibly early metastasis compared to colon/stomach cancers.
- **Stomach conduit blood supply:** Remember **Right is Right** (Right gastric + Right gastroepiploic are preserved; PEG tubes ruin the right gastroepiploic).
- **Chromoendoscopy:** Normal mucosa drinks the iodine (dark); cancer voids it (pale).

Gastric cancer

Core Concepts

- **Epidemiology**: Peak incidence in the 7th decade; more common in men (M:F = 5:3) and the elderly (<8% below age 55). High incidence in Japan and parts of Asia.
- **Risk Factors**:
  - **Helicobacter pylori**: Most important risk factor. Doubles non-cardia cancer risk (higher with *cagA*-positive strains). Induces severe chronic atrophic gastritis (11-fold risk for cardia cancer, 3-fold for non-cardia).
  - **Diet**: High salt intake (≥16g/day triples risk), heavily salted foods/processed meat (N-nitroso compounds), low fat/protein diet, frying/grilling (heterolytic amines).
  - **Protective**: Fruits and vegetables (antioxidants inhibit N-nitroso compound formation).
  - **Medical/Lifestyle**: Smoking (doubles risk, lasts 10–20 years after quitting), heavy alcohol, obesity, pernicious anemia (2–3x risk), previous gastric surgery.
- **Gastric Anatomy**:
  - *Relations*: Anterior (left liver lobe), superior (diaphragm), medial (liver), lateral (spleen), inferior (transverse mesocolon, caudate lobe, retroperitoneal structures).
  - *Arterial supply (Celiac Axis)*: Left/right gastric arteries (lesser curvature), left/right gastroepiploic arteries (greater curvature).
- **Physiology Tie-in (Gastrin)**: Secreted by G cells (gastric antrum), stimulates parietal cells directly and via histamine from enterochromaffin-like (ECL) cells. Inhibited by low pH (via somatostatin). Promotes mucosal growth.

Diagnosis / Clinical Features

- **Clinical Presentation**:
  - *Early*: Vague epigastric discomfort/indigestion (often ignored or misdiagnosed as gastritis). Pain is constant, unrelieved by food/antacids.
  - *Late*: Weight loss, dysphagia (proximal tumors), early satiety, vomiting, jaundice, ascites, cachexia.
- **Physical Signs (Advanced/Metastatic)**: Palpable abdominal mass, Virchow's node (supraclavicular), Sister Mary Joseph's nodule (periumbilical).
- **Ulcer Management Rules**:
  - **Gastric Ulcers**: Carry a significant risk of malignancy. Must undergo **mandatory endoscopic biopsy at the margins** to rule out adenocarcinoma.
  - **Duodenal Ulcers**: Almost universally benign; routine biopsy is not necessary.
- **Gastric Outlet Obstruction**: Frequently causes vomiting of old, undigested food and classically results in a **hypochloremic, hypokalemic metabolic alkalosis**.

Investigations

- **Upper Endoscopy (EGD)**: Modality of choice/Gold standard. Requires multiple biopsies (≥7) from the **ulcer edges** (avoid the necrotic crater).
- **Endoscopic Ultrasound (EUS)**: **Most accurate/sensitive modality for T-stage (depth of mural invasion) and N-stage (local lymph node involvement).**
  - Excellent for T1 and T3; poor for T2 (cannot assess invasion into muscularis propria). Can perform FNA.
- **Diagnostic Laparoscopy**: Indicated for locoregional disease to detect occult peritoneal spread missed by CT/EUS (detects mets in 30% of "resectable" scans). Allows for peritoneal washings/cytology.
- **CT Scan (Chest/Abdo/Pelvis)**: Detects distant metastases. Poor at distinguishing T1 vs. T2 and misses small (<5mm) peritoneal/liver mets.
- **PET Scan**: **Not** universally preferred for primary staging due to low avidity/sensitivity for signet ring cell carcinomas. Used for follow-up or suspected progression.
- **Exploratory Laparotomy**: **Not** a routine part of preoperative staging (replaced by CT/EUS/diagnostic laparoscopy).
- **Tumor Markers (CEA, CA 19-9)**: Low sensitivity and specificity for initial diagnosis; used for postoperative surveillance only.

Relevant Guidelines

- **Borrmann's Classification (Macroscopic Appearance)**:
  - Type 1: Polypoid or fungating lesion
  - Type 2: Ulcerated lesion surrounded by raised borders
  - Type 3: Ulcerated lesion with infiltration into the gastric wall
  - Type 4: Diffusely infiltrating lesion (*Linitis plastica* when involving the whole stomach)
  - Type 5: Unclassified
- **Lauren Classification (Histological/Epidemiological)**:

FEATURE	INTESTINAL TYPE	DIFFUSE TYPE (*LINITIS PLASTICA*)
Pathology	Well-differentiated, forms glands	Poorly differentiated, **signet ring cells**, lacks glands
Spread	Hematogenous	Deep submucosal/transmural & lymphatic (creates rigid "leather bottle" stomach)
Etiology	Environment, *H. pylori*, gastric atrophy/metaplasia	Familial/Genetic (**CDH1 / E-cadherin loss-of-function mutation**)
Demographics	Older, **Males**, Epidemic regions	Younger, **Females** (equal M:F ratio), **Blood group A**
Prognosis	Better	Significantly worse

- **Japanese Lymph Node Classification**:

    - Group 1 (N1): Perigastric lymph nodes (lesser/greater curvature).
    - Group 2 (N2): Major arteries (left gastric, common hepatic, celiac, splenic).
    - Group 3 (N3): Distant/Para-aortic nodes.
- **Staging System (TNM)**:
    - *T1*: Mucosa/submucosa. *T2*: Muscularis propria. *T3*: Subserosal tissue. *T4*: Serosa or contiguous organs.
- **Treatment Algorithms**:
    - **Stage IV (M1)**: Palliation therapy only.
    - **Stage M0 (Medically unfit)**: Palliation or RT + 5-FU radiosensitization.
    - **Stage M0 (Fit) ≤ T1**: Primary Surgery.
    - **Stage M0 (Fit) ≥ T2**: Neoadjuvant chemotherapy (ECF - MAGIC trial protocol) followed by surgery.

Operative / Procedural Notes

- **Surgical Resection Principles**: Extent determined by achieving an R0 margin (requires **6cm clearance** from the tumor edge).
    - *Proximal tumors*: Total gastrectomy (reconstructed with Roux-en-Y).
    - *Distal tumors*: Subtotal gastrectomy.
- **Lymphadenectomy (D1 vs D2)**: D2 dissection has higher morbidity and mortality compared to D1, with *no difference in overall survival*.
- **Endoscopic Mucosal Resection (EMR)**: Indicated for early tumors that are well/moderately differentiated, <30mm, non-ulcerated, and non-invasive.
- **Surveillance**: Intensive for first 3 years (visits every 4-6 months with annual CT). Annual EGD for subtotal gastrectomy patients.

Complications / Prognosis

- **Dumping Syndrome**:
    - **Early Dumping (15-30 mins post-meal)**: Rapid emptying of hyperosmolar chyme into the small intestine causes massive fluid shifts. Presents with explosive diarrhea, crampy pain, nausea, and reflex autonomic symptoms (palpitations, tachycardia, diaphoresis).
    - **Late Dumping (2-3 hrs post-meal)**: Sudden large carbohydrate load causes massive insulin oversecretion, leading to profound **reactive hypoglycemia**.
- **Linitis Plastica (Diffuse Type)**: Grows deep in the submucosa and muscularis layers. **Does NOT commonly present with massive GI bleeding.** Because it spares the superficial mucosa early on, **superficial endoscopic biopsies frequently miss the tumor**.
- **Sleeve Gastrectomy Complications**: Causes **de novo or worsening GERD** (destroys the angle of His and sling fibers, increases intra-gastric pressure). Note: Because no small bowel is bypassed or mesentery divided, **internal hernias do NOT occur** after sleeve gastrectomy (unlike Roux-en-Y).

Past-Paper High Yield

- **Endoscopic Ultrasound (EUS)** is repeatedly tested as the single best/most sensitive test for **T (depth) and N (local lymph node) staging**.
- **Lauren Classification Differentiators**:
  - *Diffuse type* = Blood group A, CDH1 genetic mutation, poorly differentiated, young females, easily missed on biopsy.
  - *Intestinal type* = *H. pylori*, environmental, older men, hematogenous spread. (Do not associate Intestinal type with Blood type A or CDH1).
- **Helicobacter pylori Characteristics**: Gram-negative, **microaerophilic** (not an obligate anaerobe), highly motile (flagella), and produces urease (neutralizes acid; it is NOT acidophilic).
- **Preoperative Staging Trap**: **Exploratory laparotomy is NOT routine**. Modern staging relies on less invasive EUS, CT, and diagnostic laparoscopy.
- **Gastrointestinal Stromal Tumors (GIST)**: Arise from interstitial cells of Cajal, express CD117 (c-kit), and are treated with surgical resection. **Trap:** Lymph node metastasis is extremely rare; therefore, **routine lymphadenectomy is NOT indicated**.
- **Esophageal Adenocarcinoma**: Overwhelmingly the most common malignancy in the **lower third** of the esophagus (arises from Barrett's/GERD).
- **Histology Identification Trap**: A mesenteric tumor with solid fat cells (lipoblasts or mature adipocytes) is a **liposarcoma**, not a fibrous tumor, GIST, or carcinoid.
- *Clostridium difficile* Risk Factors: Prolonged broad-spectrum IV antibiotics, PPIs, malnutrition, long-term steroids. **Trap**: Oral contraceptive (OCP) use does NOT predispose to *C. diff*.

Obesity & bariatric surgery

Core Concepts

- **Definition:** Abnormal or excessive fat accumulation that impairs health, defined by Body Mass Index (BMI = kg/m²).
- **Bariatric Surgery Classification:**
  - **Restrictive:** Reduces stomach capacity to produce early satiety and reduce oral intake (e.g., Intra-gastric balloon, Adjustable gastric banding, Sleeve gastrectomy).
  - **Malabsorptive:** Reconstructs the small intestine to bypass nutrient absorption segments before mixing with digestive juices (e.g., Bilio-pancreatic diversion, Duodenal switch).
  - **Mixed:** Combines gastric restriction and foregut bypass (e.g., Roux-en-Y gastric bypass).
- **Physiological impact of Sleeve Gastrectomy:** Removes the gastric fundus, which significantly reduces the production of the hunger hormone **ghrelin**.
- **Rapid weight loss and Gallstones:** Post-bariatric rapid weight loss causes high rates of gallstone formation primarily via increased cholesterol mobilization and altered bile salt metabolism (supersaturating the bile), rather than isolated gallbladder stasis.

Relevant Guidelines

BMI Classification:

CLASSIFICATION	INTERNATIONAL BMI (KG/M²)	ASIA-PACIFIC BMI (KG/M²)
Normal	18.5–24.9	18.5–22.9
Overweight	25–29.9	23–24.9
Class I obesity	30–34.9	25–29.9
Class II obesity	35–39.9	≥ 30
Class III obesity	≥ 40	—

Morbid Obesity Criteria:

- **International:** BMI ≥ 40, OR BMI ≥ 35 with severe obesity-related morbidities.
- **Asia-Pacific:** BMI ≥ 37, OR BMI ≥ 32 plus Type 2 Diabetes or two obesity-related comorbidities.

Indications for Bariatric Surgery:

- **International:**
  - BMI > 40
  - BMI > 35 with comorbidities (e.g., HTN, T2DM, hyperlipidemia, OSA, impaired glucose tolerance)
  - Failed less invasive methods and at high risk for obesity-associated morbidity/mortality.
- **Asia-Pacific:**
  - BMI > 35
  - BMI > 32 with comorbidities
  - BMI > 30 with central obesity and at least two criteria for metabolic syndrome.
- **Obesity Treatment Pyramid:**
  - BMI 25+: Lifestyle modifications (diet, exercise).
  - BMI 27+ with comorbidities (or BMI 30+): Pharmacotherapy.
  - BMI 35+ with comorbidities (or BMI 40+): Surgery.

Operative / Procedural Notes

Restrictive Procedures:

- **Intra-gastric balloon (IGB):** Endoscopically placed to decrease gastric space. Maximum duration of 6 months. Often used prior to definitive bariatric surgery.
- **Laparoscopic Adjustable Gastric Banding (LAGB):** Constricting ring placed around the fundus, adjustable via a subcutaneous port. Safest and fully reversible. However, yields significantly less long-term weight loss and has a high long-term failure rate (band slippage, erosion). It does *not* cause dumping syndrome, making it a poor choice for "sweet eaters".

- **Sleeve Gastrectomy (SG):** Vertical division of the stomach (reducing size to ~25%) while leaving the pyloric valve intact. Does not alter GI tract continuity or primary absorption, but is irreversible.

Malabsorptive Procedures:

- **Bilio-pancreatic Diversion (BPD):** Horizontal subtotal gastrectomy with a long Roux-en-Y limb and short common alimentary channel.
- **Duodenal Switch (DS):** Utilizes a sleeve gastrectomy (preserving the pylorus) rather than horizontal gastrectomy, dividing the duodenum just distal to the pylorus.
- *Note:* Provide the greatest weight loss and highest T2DM cure rates (98%), but carry higher mortality and complication rates; typically reserved for super-morbidly obese patients.

Mixed Procedure:

- **Roux-en-Y Gastric Bypass (RYGB):** The "gold standard." Creation of a small (15-30 cc) proximal gastric pouch anastomosed to a jejunal Roux limb, bypassing the distal stomach, duodenum, and proximal jejunum.
- Highly effective for severe T2DM and hypertension.
- Excellent anti-reflux operation; treatment of choice for obese patients with severe GERD.
- Best suited for "sweet eaters" as high-sugar intake triggers dumping syndrome, providing negative reinforcement.

Comparison of Surgical Outcomes:

PROCEDURE	CATEGORY	EXCESS WEIGHT LOSS	DM REMISSION	MORTALITY
Adjustable Gastric Band	Restrictive	49.4%	62%	0.05%
Sleeve Gastrectomy	Restrictive	55.4%	70%	0.17%
Bilio-pancreatic Diversion	Malabsorptive	70–80%	98%	1.9%
Roux-en-Y Gastric Bypass	Mixed	62.6%	83%	0.5%

Complications / Prognosis

- **Intra-gastric balloon:** Severe N/V (early), dehydration, un-noticed deflation leading to bowel obstruction.
- **Sleeve Gastrectomy:** Staple line leakage (<5%), bleeding, **worsens or induces GERD**. Can still cause iron/B12 deficiency due to loss of parietal cell mass (acid/intrinsic factor). *Crucially: Does NOT cause internal hernias.*
- **Roux-en-Y Gastric Bypass (RYGB):**
  - **Internal hernia:** Specific complication due to the creation of mesenteric defects during bowel rearrangement.

    - **Nutritional deficiencies:** Decreased absorption of iron, Vitamin B12 (anemia), and calcium (bone disease). Requires lifelong supplementation.
    - **Dumping syndrome:** Rapid emptying of hyperosmolar contents into the small bowel causing nausea, sweating, faintness, and diarrhea.
    - Bypassed stomach cannot be evaluated via standard upper endoscopy.
- **Malabsorptive (BPD/DS):** Highest rates of nutritional deficiency, anastomotic leak, and perioperative mortality.

Past-Paper High Yield

- **Internal Hernias:** Occur exclusively in Roux-en-Y Gastric Bypass (due to mesenteric defects). They do *not* occur in Sleeve Gastrectomy.
- **Sleeve Gastrectomy Characteristics:** Removes the fundus (decreases ghrelin/hunger); it is strictly restrictive (not malabsorptive); it is contraindicated in severe erosive esophagitis because it *worsens* GERD.
- **Gallstone Formation Post-Bariatric Surgery:** Driven by supersaturation (cholesterol mobilization and altered bile salts), distinguishing it from pure stasis-induced gallstones seen in TPN, prolonged fasting, or spinal cord injury.
- **Procedure Selection for "Sweet Eaters":** RYGB is the treatment of choice; the resulting dumping syndrome serves as a behavioral deterrent. Gastric banding and sleeve gastrectomy do not consistently cause dumping syndrome and have high failure rates in this demographic.
- **RYGB and GERD:** RYGB is the bariatric procedure of choice for patients with severe GERD (it cures GERD, whereas sleeve gastrectomy worsens it).
- **GI/Metabolic derangements (General GI Principles tested alongside this topic):**
    - *Gastric Outlet Obstruction (e.g., ulcer stricture):* Prolonged vomiting of HCl results in a classic **hypokalemic hypochloremic metabolic alkalosis**.
    - *Severe Acute Pancreatitis Risk Factors:* Predictors of severe disease include Obesity (BMI >30), Age >55 years, Hemoconcentration (Hct > 44%), and CRP > 150 mg/L at 48 hours. *Young age (<30)* is NOT a risk factor.
    - *Pancreatic Adenocarcinoma:* Cigarette smoking is the most significant and well-established modifiable risk factor.

Memory Pearls

- **Sleeve = Restrictive + Removes Ghrelin + Worsens GERD.**
- **Bypass (RYGB) = Restrictive & Malabsorptive + Cures GERD + Causes Dumping (good for sweet eaters) + Internal Hernia Risk.**
- **Gallstones post-op = Supersaturation** (not just stasis).
- **Vomiting acid = Hypokalemic, hypochloremic metabolic alkalosis.**

Primary liver tumors

Core Concepts

- **Overall most common malignant liver tumor:** Metastatic deposits (most often from colorectal primaries). The liver is a massive filter for venous blood from the GI tract.
- **Most common primary malignant liver tumor:** Hepatocellular Carcinoma (HCC).
- **Most common benign solid liver tumor:** Cavernous hemangioma.
- **Liver Transplant Indication:** Liver cirrhosis (representing end-stage liver disease from varying etiologies) is the overall most common cause for liver transplant globally.
- **Malignancy Risk Factors:** Non-alcoholic fatty liver disease (NAFLD) can progress to NASH and cirrhosis, which significantly increases the risk of HCC.

Diagnosis / Clinical Features

Benign Liver Lesions:

- **Cavernous Hemangioma (0.5–5%):**
  - Most common benign tumor.
  - Overwhelmingly incidental and asymptomatic. Can cause pain or early satiety if very large.
  - Classified as "Giant" if > 6 cm.
- **Hepatic Adenoma (HCA):**
  - Rare, highly associated with young females taking oral contraceptive pills (OCPs) or hormone replacement therapy.
  - Usually incidental and asymptomatic, but can present with RUQ pain or hemorrhagic shock if it ruptures.
- **Focal Nodular Hyperplasia (FNH):**
  - More common in young women. Believed to arise from a congenital localized vascular anomaly (arteriovenous malformation).
  - Completely benign and typically asymptomatic.

Malignant Liver Lesions:

- **Hepatocellular Carcinoma (HCC):**
  - Strong male predominance. High geographic variation (Southeast Asia/Africa due to HBV; increasing in the West due to steatohepatitis).
  - Risk factors: HBV, HCV, cirrhosis (any etiology), alcoholism, hemochromatosis, Wilson's disease, aflatoxin, diabetes.
  - Often asymptomatic. Late presentation includes RUQ pain, weight loss, palpable mass, and hepatic decompensation in a known cirrhotic.
  - Rupture can cause hemoperitoneum and hypovolemic shock.
  - **Fibrolamellar variant:** Occurs in young patients *without* underlying cirrhosis; usually encapsulated with internal fibrous septations.

- **Cholangiocarcinoma:**
  - Second most common primary liver cancer; arises from biliary epithelium (typically extrahepatic).
  - Late presentation with obstructive jaundice.
  - Risk factors: Primary Sclerosing Cholangitis (PSC), Oriental cholangiohepatitis.
- **Hepatoblastoma:**
  - Primary liver cancer in infants/children. Arises from immature liver cells.
  - Presents as a palpable abdominal mass.
  - Co-exists with syndromes: FAP (Familial Adenomatous Polyposis), Trisomy 18, Trisomy 21.

Infectious Differential:

- **Pyogenic Liver Abscess:** Fever, RUQ pain, marked tenderness, and a hypoechoic liver mass. Strongly associated with previous biliary disease/surgery (e.g., post-cholecystectomy).

Investigations

Tumor Markers:

- **Alpha-fetoprotein (AFP):**
  - **Elevated in:** HCC (>400 ng/dL is highly specific), hepatoblastoma (>500 ng/dL), fulminant hepatitis infection, liver cirrhosis with active regeneration, and teratocarcinomas.
  - **Normal in:** Cholangiocarcinoma, fibrolamellar HCC variant, and ~30% of classic HCC cases.
- **CA 19-9:**
  - Elevated in cholangiocarcinoma.

Imaging Characteristics:

- **Hemangioma:** CT shows peripheral nodular enhancement with gradual centripetal (inward) fill-in.
- **FNH:** CT/MRI classically demonstrates a central stellate scar.
- **Hepatic Adenoma:** Hypervascular lesion on imaging.
- **HCC:** Triphasic CT scan uniquely shows **early arterial filling and delayed venous washout**.
- **Pyogenic Abscess:** Appears as a hypoechoic focal mass on ultrasound.

Biopsy Considerations:

- **Hemangioma:** Needle biopsy is **contraindicated** due to severe bleeding risk.
- **Hepatic Adenoma:** Percutaneous fine needle biopsy carries a high bleeding risk and is generally avoided.
- **HCC:** Biopsy is completely avoided if diagnostic imaging criteria and tumor markers are met in a high-risk patient (see guidelines below).

Management

- **Hemangioma:** Observation; surgical resection or embolization if symptomatic/complicated.

- **Hepatic Adenoma:**
  - **First step:** Stop OCPs/hormonal therapy immediately.
  - **Surgery:** Definitive surgical resection is indicated for large tumors (> 5 cm) due to significant risks of spontaneous hemorrhage and malignant transformation.
- **Focal Nodular Hyperplasia:** Observation. Surgery is only indicated if the diagnosis remains unclear.
- **Hepatocellular Carcinoma:**
  - **Curative:**
    - Resection is feasible if there is no cirrhosis, Child-Pugh A status, or for the fibrolamellar variant.
    - Liver transplantation (preferably cadaveric) if specific size criteria are met.
  - **Palliative:** Radiofrequency ablation (RFA), Transarterial chemoembolization (TACE), Ethanol injection, Sorafenib, Systemic chemotherapy, Transarterial radioembolization (TARE), or High-Intensity Focused Ultrasound (HIFU).
- **Cholangiocarcinoma:** Resection is the ideal treatment if achievable.
- **Hepatoblastoma:** Neoadjuvant chemotherapy followed by surgical resection or liver transplantation.

Relevant Guidelines

HCC Diagnostic Pathway

- **Diagnosis WITHOUT Biopsy:** Presence of cirrhosis or HBV/HCV + elevated AFP (>400 ng/dL) + typical imaging.
- **Proceed to Biopsy:** Absence of cirrhosis, negative HBV/HCV, and normal AFP levels.
- **Diagnostic finding:** Portal vein thrombosis in the presence of a liver lesion is considered diagnostic of HCC.

Liver Transplantation Criteria for HCC

- Single lesion < 5 cm.
- *OR* up to 3 lesions, each < 3 cm.

Child-Pugh Score for Liver Cirrhosis Severity

- Assesses severity using 5 parameters:
  1. Ascites
  2. Serum Albumin
  3. Prothrombin time (PT/INR)
  4. Serum Bilirubin
  5. Hepatic Encephalopathy (degree)
- *Note: Serum AST/ALT are NOT components of the Child-Pugh prognostic score.*

Complications / Prognosis

- **Hepatic Adenoma:** Significant risk of rupture/bleeding and harbors a small risk of malignant transformation to HCC.
- **HCC:**
  - With cirrhosis + untreated: Very poor survival (3–6 months).
  - With transplantation (meeting criteria): > 80% survival at 5 years.
  - Without cirrhosis + resected: Considered cured.
  - Tumor differentiation grade has *no relation* to prognosis.
- **Hepatoblastoma:** Survival is close to 100% if the tumor responds to neoadjuvant chemotherapy followed by resection/transplant. Poor prognosis if metastases are present.

Past-Paper High Yield

- **Metastatic vs. Primary:** The most common malignant tumor in the liver is metastatic deposits (often GI/colorectal). HCC is the most common *primary* malignancy.
- **Adenoma vs. FNH Trap:**
  - **Adenoma:** Associated with OCPs, has a risk of bleeding/malignancy, large tumors require resection. Most are asymptomatic (do not fall for the distractor that 50-70% are symptomatic). Stop OCPs immediately.
  - **FNH:** Benign hyperplastic vascular response, central stellate scar, completely benign, NO malignant transformation, observe.
- **Biopsy Contraindications:** Never needle-biopsy a hemangioma. Avoid percutaneous biopsy of hepatic adenomas due to hemorrhage risk.
- **AFP Fallacies:** Cholangiocarcinoma does *not* elevate AFP (elevates CA 19-9). AFP can be elevated by completely benign conditions like active liver regeneration in cirrhosis and fulminant hepatitis.
- **Child-Pugh Trap:** Serum AST is a marker of hepatocellular injury but is *never* part of the prognostic Child-Pugh scoring system.
- **Biliary Anatomy Concept:** Alagille syndrome involves a paucity of *intrahepatic* bile ducts. Conditions like Primary sclerosing cholangitis, Cholangiocarcinoma, Choledochal cysts, and Biliary atresia involve *extrahepatic* bile ducts.
- **Choledochal Cysts:** Strongly associated with an increased risk of cholangiocarcinoma.
  - Type 1: Fusiform dilation.
  - Type 2: Simple extrahepatic diverticulum.
  - Type 3: Choledochocele.
  - Type 4: Multiple dilations.
  - Type 5: Caroli disease (intrahepatic). *Do not mistake Type 4 for Caroli disease.*

Memory Pearls

- **Adenoma Management (The 3 A's):** **A**ssociated with OCPs, **A**void biopsy (bleeds), **A**mputate (resect) if > 5cm.
- **FNH (Focal Nodular Hyperplasia):** **F**emale, **N**o malignancy, **H**as a central scar.

- **Hemangioma Diagnosis: Centripetal** (outside-in) fill-in on CT.
- **Child-Pugh Score (A-B-C-D-E): A**lbumin, **B**ilirubin, **C**oagulation (PT/INR), **D**istension (Ascites), **E**ncephalopathy.

Liver metastasis

Core Concepts

- **Hepatic Segmental Anatomy (Couinaud Classification):**
  - The liver is divided into **8 functionally independent segments** (not 10).
  - Divisions are based on the distribution of the portal triads (portal vein, hepatic artery, bile duct).
  - This French classification system is the universal standard for planning surgical resections.
- **Segment I (Caudate Lobe) Anatomy:**
  - Anatomically unique: Venous drainage bypasses the main hepatic veins.
  - Drains **directly into the inferior vena cava (IVC)** via short hepatic veins.
- **Hepatic Circulation:**
  - The liver has a dual blood supply.
  - **Portal Vein:** Supplies **75%** of the hepatic blood flow.
  - **Hepatic Artery:** Supplies **25%** of the hepatic blood flow (originates from the celiac trunk).
  - Main hepatic veins drain the liver into the IVC.
- **Hepatic Physiology & Reserve:**
  - **Albumin Synthesis:** Exclusively synthesized by hepatocytes in the liver.
  - **Bilirubin Conjugation:** Occurs entirely within the liver (not in the spleen).
  - **Functional Reserve:** The liver possesses massive reserve capacity; clinical signs of hepatic failure typically do not manifest until 80–90% of the liver parenchyma is destroyed (not 60%).
  - **Regenerative Capacity:** The liver can successfully regenerate even after resection of up to 70–80% of healthy tissue.

Management

- **Colorectal Cancer (CRC) Liver Metastasis & Adjuvant Therapy:**
  - **Stage IV Disease:** Bi-lobar liver metastasis is classified as Stage IV disease and necessitates systemic therapy.
  - **Postoperative Adjuvant Chemotherapy Indications (e.g., post-anterior resection):**
    - Node-positive disease (Stage III).
    - High-risk Stage II disease (e.g., T4 stage tumor, presence of lymphovascular invasion).
  - **Exception:** Gross tumor size alone (e.g., > 3 cm) does *not* upstage the tumor and is *not* an independent indication for postoperative adjuvant chemotherapy if regional nodes are negative.
- **Differential Diagnosis Note - Pyogenic Liver Abscess:**

- When differentiating cystic/necrotic lesions from metastatic disease, note that ***E. coli*** is classically identified as the **most common** bacterial isolate in pyogenic liver abscesses (frequently originating from biliary or gastrointestinal tract infections).
- *Klebsiella* is a common isolate specifically in Asian populations.
- *Streptococcus*, *Staphylococcus aureus*, and *Pseudomonas aeruginosa* can cause abscesses but are much less prevalent.

Complications / Prognosis

- **Hepatic Failure:**
  - Leads to absent/decreased albumin synthesis and failure of bilirubin conjugation.
  - **Hepatic Encephalopathy:** Generally **reversible** with appropriate treatment (e.g., lactulose) once it develops; it is a misconception that it is universally irreversible.

Past-Paper High Yield

- **Anatomy trap:** Do not confuse the proportions of hepatic blood supply. The **portal vein provides 75%**, while the hepatic artery provides only 25%.
- **Couinaud classification trap:** There are exactly **8** functionally independent segments, not 10.
- **Caudate lobe (Segment I):** Routinely tested for its unique venous drainage—it drains **directly into the IVC**, bypassing the main hepatic veins.
- **Oncology staging trap:** In CRC, tumor size alone does *not* mandate adjuvant chemotherapy. Look for **node-positive disease, T4 stage, or lymphovascular invasion**.
- **Microbiology of Liver Abscess:** ***E. coli*** is the classic and most common overall isolate.
- **Physiology fundamentals:** Albumin is synthesized *only* in the liver. Liver failure requires 80–90% destruction, and the liver can regenerate after massive (70–80%) resection.

Memory Pearls

- **Blood Supply 75/25 Rule: P**ortal is **P**rimary (75%).
- **Segment I = IVC:** The caudate lobe gets a VIP direct pass to the Inferior Vena Cava.

Liver hydatid cyst

Core Concepts

- **Pathogen:** *Echinococcus granulosus* (a tapeworm).
- **Life Cycle & Transmission:**
  - **Definitive host:** Canines (dogs, wolves) house the adult tapeworm in the small intestine.
  - **Infective stage:** Embryonated eggs are shed in canine feces.
  - **Intermediate host:** Sheep, goats, swine, or humans (accidental ingestion).
  - **Diagnostic stage:** Hydatid cysts develop in target organs (Liver > Lungs > Brain > Bone).
- **Cyst Wall Anatomy (Outer to Inner):**

- **Pericyst:** Outermost layer formed by the host's inflammatory response and fibrous tissue.
- **Ectocyst:** Middle acellular, laminated, gelatinous chitinous membrane.
- **Endocyst:** Inner germinal layer (nucleated); produces brood capsules and protoscoleces (hydatid sand).

Diagnosis / Clinical Features

- **Latency Phase:** Often asymptomatic; may present with dull abdominal pain.
- **Pressure Effects:** Compression of surrounding liver parenchyma, hilum, or hepatic veins.
- **Suppuration:** Occurs in 11–27% of cases, most commonly secondary to *Escherichia coli* infection.
- **Cyst Rupture:**
  - **Obscure:** Silent or occult rupture of the endocyst.
  - **Communicant:** Rupture into the biliary or bronchial tree (occurs in 5–25% of cases).
  - **Free Rupture:** Rupture into free body cavities (peritoneum, pleura) or adjacent organs (1–4%). Risk of severe anaphylaxis and peritoneal seeding.

Investigations

- **Imaging:**
  - **Ultrasound:** First-line diagnostic modality (classifies cysts via Gharby system).
  - **CT Scan: Gold standard** for detailing anatomy, size, location, and calcifications. Excellent for identifying complex or multi-organ disease (giant cysts).
  - **MRI:** Modality of choice for suspected biliary communication and delineating soft tissue relationships.
  - **ERCP:** Indicated if there is suspected biliary rupture or if daughter cysts are present in the common bile duct (CBD).
  - **Plain X-ray:** May reveal curvilinear calcification of the cyst wall.
- **Immunology / Serology:** Enzyme-Linked Immunosorbent Assay (ELISA), Indirect Hemagglutination Assay (IHA), Complement Fixation Test (CFT).

Relevant Guidelines

Gharby Ultrasound Classification (1981) Used to stage hydatid cysts and guide intervention:

- **Type 1:** Simple cyst. Unilocular, well-circumscribed, anechoic (active budding + hydatid sand).
- **Type 2:** Fluid collection with a split wall. Classic **"Water-lily" sign** (detached/collapsed germinal membranes).
- **Type 3:** Fluid collection with septa. Classic **"Honeycomb" pattern** or wheel-spoke appearance (multiseptated with daughter cysts).
- **Type 4:** Heterogeneous, complex, solid-appearing mass (simulates a liver abscess or tumor).
- **Type 5:** Highly reflecting thick wall with dense posterior acoustic shadow (calcified, inactive/dead cyst).

Overall Treatment Pathway

- **Medical:** Albendazole (ideal but rarely completely efficient alone).
- **Radiological:** Selective PAIR (Puncture, Aspiration, Injection, Re-aspiration).
- **Surgical:** Laparoscopic (attractive, minimally invasive) vs. Radical conventional (open surgery). Laparoscopy is emerging as the gold standard for suitable cysts, offering superior recurrence rates over PAIR.

Management

- **Medical Therapy:**
  - **Albendazole: The anti-helminthic drug of choice**.
    - **Dosing:** 10–14 mg/kg/day in three 28-day courses, separated by a 2-week drug-free interval.
    - **Adjuvant/Neoadjuvant use:** Kills the parasite preoperatively (8 weeks pre-op kills 90%) and acts directly on the germinal layer/protoscoleces. Reduces post-op recurrence rates dramatically (from 18% down to 5%).
    - **Synergy:** Praziquantel can be added to Albendazole for enhanced efficacy.
- **Radiological (PAIR):** Highly successful for Gharby Types 1–3 if strictly solitary/uncomplicated. Not for calcified (Type 5) cysts.
- **Surgical Options:**
  - **Cystectomy Plus:** Partial, subtotal, or total pericystectomy.
  - **Hepatic Resection:** Segmental resection, lobectomy, or total hepatectomy + liver transplant (reserved for extensive destruction).
  - **Cavity Management:** Must be addressed post-resection to prevent collections. Options include capitonnage (suturing walls from inside to obliterate space), omentoplasty (filling with vascularized greater omentum), or simple primary closure/drainage.

Operative / Procedural Notes

PAIR Technique (Percutaneous Puncture, Aspiration, Injection, Re-aspiration):

1. Percutaneously insert an 18G Seldinger needle.
2. Aspirate 25–35% of cyst volume.
3. Inject hypertonic saline (**15-25% NaCl**) at ~10% of aspirated volume. Wait 10 mins (allows protoscoleces death and pericyst separation).
4. Reaspirate completely.
5. If catheterized (6F catheter): wash out, place on external drain for 24h. If output <10 cc/24h (no bile leak), perform cystogram, inject **95% alcohol**, reaspirate, and remove.

Open Surgical Principles:

- **Isolation:** Pack the operative field strictly with scolicidal-soaked abdominal packs to prevent anaphylaxis/seeding in case of spillage.
- **Aspiration-Suction Technique:** Halt patient breathing temporarily during initial puncture/aspiration to prevent diaphragmatic movement and spillage.

- **Scolicidal agents:** *Sterimide* 0.5% - 1%, hypertonic saline, or alcohol.
- **Cysto-Biliary Communication Management:**
  - Always actively look for biliary fistulae (present in up to 39% of surgical series).
  - Micro/simple fistulas: Primary suture.
  - Frank rupture (spillage into CBD): Requires preoperative EPST (endoscopic papillosphincterotomy), intraoperative transduodenal sphincteroplasty, and T-tube drainage of the CBD.
  - *Clinical note:* Simple tube drainage of the residual cavity with an open fistula has high infection rates; capitonnage, omentoplasty, or cyst excision are superior.

Complications / Prognosis

- **PAIR Complications:**
  - Fever/urticaria (10-20%) due to allergic reaction from spilled antigens.
  - Biliary fistula (5-10%).
  - Cyst cavity infection (~3%).
  - Overall recurrence is very low (0-4%) in appropriately selected patients.
- **Surgical Complications:** Wound infection (13.5%), subphrenic/liver abscesses, biliary leakage, chest problems. Post-op mortality is generally <3%.

Past-Paper High Yield

- **Albendazole is the definitively tested drug of choice** for cystic echinococcosis (hydatid disease).
  - *Why?* It has **excellent tissue penetration** into the cyst, unlike other agents.
  - *Distractors:* Mebendazole is less effective (poor penetration). Ketoconazole treats fungi. Metronidazole treats amoebas/anaerobes. Steroids treat anaphylaxis/inflammation but have no scolicidal effect.
- **"Water-lily sign"** on ultrasound strongly points to a Gharby Type 2 hydatid cyst (indicating detached endocyst membranes).
- **Never drain a hydatid cyst percutaneously without scolicidal injection** (like a simple abscess), due to the risk of anaphylactic shock and widespread intra-abdominal seeding.

Memory Pearls

- **Hydatid Layers (Outside to Inside):** **P**ericyst (**P**atient's tissue), **E**ctocyst (**E**gg-shell/chitinous), **E**ndocyst (**E**mbryo/germinal layer).
- **Gharby's "Garden" on Ultrasound:**
  - Type 2 = **Water-lily** (floating membranes).
  - Type 3 = **Honeycomb** (daughter cysts).
  - Type 5 = **Rock** (calcified, dead).

Gallstones

Core Concepts

Epidemiology & General Principles

- Gallstones are present in ~10% of the US population (30 million people).
- Incidence is 2–3 times higher in females and increases with age (found in 50% of females in their 70s, and 80% of individuals in their 90s).
- Two-thirds of all gallstones are completely asymptomatic and found incidentally.
- **Biliary Sludge:** A viscous mixture of mucin glycoproteins, calcium bilirubinate, and cholesterol crystals.
- **Unconjugated Bilirubin:** It is lipid-soluble (not water-soluble), tightly bound to albumin in the blood, conjugated by the liver, and cannot be excreted in the urine.

Pathogenesis of Gallstones Stone formation requires specific metabolic and physiologic derangements:

1. **Solubilization Failure & Supersaturation:** High cholesterol, decreased bile salts, or decreased lecithin.
2. **Nucleation:** Precipitation of cholesterol monohydrate crystals from a saturated solution. The gallbladder's impaired absorptive capacity or increased mucin secretion promotes this.
3. **Stone Growth:** Aggregation of crystals, calcium, and mucous glycoproteins (grows ~1–2 mm/year).
4. **Gallbladder Stasis:** Impaired contraction (increased fasting/residual volume) prevents clearance of crystals.

Types of Gallstones

- **Cholesterol Stones (70–80%):** Contain >50% cholesterol (Pure stones are 90–100% cholesterol, Mixed are 50–90%).
  - *Mechanism:* Formed when micelles and vesicles fuse to form multilamellar vesicles. Cholesterol crystals grow on the vesicle membrane and interact with mucin gel to precipitate.
  - *Risk Factors:* Elevated estrogens (puberty, pregnancy, OCPs, HRT), obesity (increased HMG-CoA reductase activity), rapid weight loss (stasis and increased excretion), spinal cord injury, increasing age (declined cholesterol 7-alpha hydroxylase), prolonged TPN, and medications.
  - *Medications:* **Ceftriaxone** (>2g/day precipitates with calcium; known as "pseudolithiasis") and **Octreotide** (alters bile flow and inhibits postprandial secretion).
- **Black Pigment Stones (~10-15%):** Contain <20% cholesterol.
  - *Composition:* Calcium bilirubinate monohydrate, calcium carbonate, calcium palmitate, and calcium phosphate.
  - *Mechanism:* Driven by high levels of conjugated bilirubin in bile. Endogenous *mucosal β-glucuronidase* converts conjugated bilirubin to unconjugated bilirubin. This binds to calcium, precipitates, and polymerizes in the mucin gel.
  - *Risk Factors:* Cirrhosis, chronic hemolysis (sickle cell anemia, thalassemia), ileal resection.
- **Brown Pigment Stones (~5-10%):** Contain <20% cholesterol.

- *Composition:* High in calcium palmitate and calcium stearate.
- *Mechanism:* Driven by biliary stasis and **bacterial infection** (e.g., *E. coli*). *Bacterial β-glucuronidase* produces unconjugated bilirubin, and *bacterial phospholipase* degrades lecithin into free fatty acids. These interact with calcium to form precipitates.
- *Location:* Fundamentally formed within the bile ducts (primary duct stones).
- *Risk Factors:* Oriental cholangiohepatitis, choledochal cysts, biliary strictures, periampullary diverticula, sphincterotomy.

Diagnosis / Clinical Features

- **Acute Cholangitis:** Ascending infection (not hematogenous) strictly driven by biliary stasis/obstruction (e.g., choledocholithiasis). Presents with direct (conjugated) hyperbilirubinemia.
  - *Charcot's Triad:* RUQ pain, fever, jaundice.
  - *Reynold's Pentad:* Charcot's triad + hypotension + altered mental status (indicates severe disease).
- **Mirizzi Syndrome:** A large gallstone impacted in the cystic duct or gallbladder neck causes severe *extrinsic mechanical compression* of the common hepatic duct.
  - Presents with obstructive jaundice (elevated direct bilirubin, alkaline phosphatase, and GGT). LFTs are *never* normal.
- **Emphysematous Cholecystitis:** A rapidly progressive surgical emergency.
  - *Etiology:* Gas-forming organisms (*Clostridium perfringens*, *E. coli*; NOT *Pseudomonas*).
  - *Unique feature:* Frequently **acalculous** (up to 50% of cases occur without stones).
  - *Demographics:* Classically affects elderly diabetic males.
- **Acalculous Cholecystitis:** Typically seen in critically ill patients.
  - *Risk Factors:* Multiorgan failure, severe trauma, prolonged TPN, systemic infection.
  - *Note:* Gallbladder polyps are mucosal outgrowths and do *not* precipitate acalculous cholecystitis.
- **Gallstone Ileus:** A mechanical small bowel obstruction caused by a large gallstone eroding through the gallbladder wall.
  - *Anatomy:* The fistula typically forms between the gallbladder and the adjacent **duodenum** (cholecystoduodenal fistula), strictly *not* the ileum.
  - *Presentation:* Mostly affects females >70 years old. Stone classically lodges at the narrowest point: the ileocecal valve.

Investigations

- **Transabdominal Ultrasound:** Primary modality for gallbladder stones. However, it has a **poor diagnostic yield for distal (lower) common bile duct (CBD) stones** because the area is frequently obscured by overlying bowel gas from the duodenum.
- **MRCP (Magnetic Resonance Cholangiopancreatography):** Highly sensitive, specific, and completely non-invasive. It is the best non-invasive modality to map the biliary tree and exact

location/cause of obstruction when choledocholithiasis is equivocal. **Not contraindicated** in acute cholangitis.

- **CT Scan (Abdomen/Pelvis with contrast):** Best next diagnostic step in a stabilized patient with suspected gallstone ileus to map the anatomy, define the fistulous tract, and locate the ectopic stone.
- **Scoring Systems context:** The **Child-Pugh score** is strictly used to assess the prognosis of liver cirrhosis. It is *not* used for acute cholecystitis or acute pancreatitis (which use Ranson's, APACHE II, etc.).

Management

- **Asymptomatic Gallstones:** Observation only (no prophylactic surgery required).
- **Acute Cholangitis:** Initial management is aggressive IV fluid resuscitation and broad-spectrum antibiotics, followed by urgent/early biliary decompression (usually via ERCP).
- **Acute Pancreatitis (Gallstone-induced):** The cornerstone of initial conservative management is **aggressive IV fluid resuscitation** to combat third-spacing and prevent pancreatic necrosis. (Prophylactic antibiotics or steroids are not routinely indicated).
- **Emphysematous Cholecystitis:** Medical management/observation alone is completely insufficient and highly lethal.
  - Requires emergent broad-spectrum antibiotics and surgical intervention (cholecystectomy).
  - If the patient is a poor/high-risk surgical candidate (e.g., severe COPD/multimorbidity), place a **percutaneous cholecystostomy tube** for immediate source control.
- **Pyogenic Liver Abscess:** Biliary tract disease (ascending cholangitis, choledocholithiasis) is the **single most common underlying cause** of pyogenic liver abscesses in developed countries.

Relevant Guidelines

Classification of Gallstones

- **Cholesterol Stones (~80%):** >50% Cholesterol
  - *Pure:* 90–100% Cholesterol (Make up ~5% of cholesterol stones)
  - *Mixed:* 50–90% Cholesterol (Make up 90–95% of cholesterol stones)
- **Pigment Stones (~20%):** <20% Cholesterol
  - *Black Stones:* Formed via endogenous pathways (high bilirubin).
  - *Brown Stones:* Formed via bacterial enzymatic degradation.

Operative / Procedural Notes

- **Surgical Prophylaxis:** For a standard elective laparoscopic cholecystectomy in a low-risk patient, a first-generation cephalosporin (**Cefazolin**) is the standard antibiotic of choice.
- **Prophylactic Cholecystectomy Indications:**
  - Indicated for **Porcelain gallbladder** (extensive intramural calcification) due to the increased risk of gallbladder carcinoma.

- *Not indicated* for: Asymptomatic gallstones, asymptomatic 3 mm polyps (only resect if >10 mm), asymptomatic adenomyomatosis, or mild incidental chronic cholecystitis.

- **Incidental Gallbladder Carcinoma:**
  - If pathology reveals **T1a** disease (tumor invades lamina propria only) with negative margins following a simple cholecystectomy: **Observation and routine follow-up only.** The simple cholecystectomy is considered curative.
  - If disease is T1b (invades muscularis) or deeper: Requires re-operation for extended cholecystectomy (liver wedge resection + regional hepatoduodenal lymph node dissection).
- **Incidental Liver Hemangioma:** If a small, asymptomatic hemangioma (e.g., 1 cm) is found incidentally during cholecystectomy, the correct management is to **observe and leave it alone**. Biopsy is strictly contraindicated (hemorrhage risk).

Complications / Prognosis

- **Emphysematous Cholecystitis:** Carries a significantly higher risk of rapid gallbladder gangrene and perforation compared to standard acute cholecystitis due to rapid ischemia.
- **Mirizzi Syndrome:** Prolonged, untreated extrinsic compression can eventually lead to tissue necrosis and the formation of a cholecystocholedochal fistula.
- **Post-Operative Distractors:** Acute cholecystitis is generally unrelated to recent colonic/pelvic surgeries (e.g., sigmoid colectomy) and is the *least likely* direct post-operative complication compared to SBO, anastomotic leak, or *C. difficile* colitis.

Past-Paper High Yield

- **Mirizzi Syndrome Trap:** Do not be fooled by normal LFTs. Because it mechanically obstructs the common hepatic duct, patients will *always* present with an obstructive pattern on liver function tests (elevated direct bilirubin).
- **Gallstone Ileus Trap:** The fistula is a **cholecystoduodenal** fistula. Examiners frequently state it is a "cholecystoileal" fistula—this is anatomically FALSE.
- **Emphysematous Cholecystitis Traps:**
  1. It is frequently caused by *Clostridium*, NOT *Pseudomonas*.
  2. Over 50% are **acalculous** (occurring without stones).
  3. Medical management alone is the *wrong* answer. Emergent drainage/surgery is required.
- **Ultrasound Limitations:** Excellent for stones in the gallbladder, but explicitly **poor** for detecting stones in the *distal* CBD due to duodenal bowel gas shadowing.
- **Acalculous Cholecystitis Risk Factors:** Multiorgan failure, trauma, prolonged TPN. *Gallbladder polyps* do NOT cause acalculous cholecystitis.

Memory Pearls

- **Rigler's Triad (Gallstone Ileus):** 1) Small Bowel Obstruction, 2) Pneumobilia (air in biliary tree), 3) Ectopic calcified gallstone at the ileocecal valve.
- **Black vs. Brown Stones:**
  - **B**lack = **B**ilirubin (Hemolysis/Cirrhosis), endogenous mucosal glucuronidase.

- **B**rown = **B**acteria (Infection/Stasis), exogenous bacterial glucuronidase.

Acute pancreatitis

Core Concepts

- **Definition:** A reversible inflammation of the pancreas resulting from autodigestion by its own enzymes. It is an acute emergency presenting with sudden abdominal pain and lasting for days.
- **Pathophysiology:**
  - **Normal state:** Acinar cells produce inactive zymogens (e.g., trypsinogen). These are normally activated in the small intestine (trypsinogen is activated to trypsin by duodenal enteropeptidase/enterokinase) to prevent self-digestion.
  - **Pathological state:** Any early, intracellular activation of pro-enzymes leads to autodigestion. Trypsinogen is prematurely converted to active trypsin via **Cathepsin B**.
  - **Amplification loop:** Active trypsin triggers a massive feedback loop, activating more trypsinogen and other zymogens (chymotrypsin, elastase, phospholipase).
  - **Enzymatic lesions:**
    - Proteases → Proteolysis
    - Lipase/phospholipase → Fat necrosis
    - Elastase → Hemorrhage
- **Etiologies:**
  - **Most common:** Gallstones and Alcohol abuse (account for 90% of cases; biliary is most prevalent even in the West).
  - **Other causes:** Idiopathic, Trauma (penetrating), ERCP (iatrogenic), Hyperlipidemia (hypertriglyceridemia), Hypercalcemia (e.g., hyperparathyroidism), Autoimmune (Polyarteritis nodosa), Viral (Mumps), Steroids, Scorpion bite, Cardiopulmonary bypass (ischemia/hypoperfusion).
  - **Drugs:** Diuretics, Isoniazid (INH), Reverse transcriptase inhibitors, Metronidazole.

Diagnosis / Clinical Features

- **Diagnostic Criteria:** Diagnosis is established by the presence of **2 of the following 3** criteria:
  1. Characteristic abdominal pain.
  2. Serum amylase and/or lipase >3 times the upper limit of normal.
  3. Characteristic findings of pancreatic inflammation on contrast-enhanced CT (CE-CT).
- **Classic Presentation:**
  - **Pain:** Sudden onset; dull, boring, and steady. Usually in the epigastric region and classically **radiates directly through to the back** (in ~50% of cases). Gradually intensifies to a constant ache.
  - **Associated symptoms:** Nausea, vomiting (~85%), tachycardia (~80%), low-grade fever (~50%).

- **History clues:** Recent ERCP, binge alcohol consumption, previous biliary colic, or a family history of hypertriglyceridemia.
- **Severe cases:** Patients appear gravely ill with profound shock, toxicity, hypotension, and confusion.

Investigations

- **Serum Markers:**
  - **Amylase:** Easiest/most widely used. Rises immediately, peaks in a few hours, remains elevated for 3-5 days. *Caveats:* Can be normal in severe attacks, falsely negative in hyperlipidemic patients, and has an *inverse* correlation with severity. A 3-fold rise is diagnostic.
  - **Lipase:** Rises more dramatically than amylase (peaks 6-12 hours) and stays elevated longer (highly diagnostic at 72-96 hours).
- **Imaging:**
  - **Plain X-ray:** Non-specific signs include "Sentinel loop" (localized jejunal dilation) and "Colon cutoff sign" (abrupt termination of gas column at the splenic flexure).
  - **Ultrasound:** Used primarily for diagnosing gallstones and follow-up of pseudocysts.
  - **Contrast-Enhanced CT (CE-CT):** The **gold standard** for detecting and assessing severity. Routinely performed if severe pancreatitis is suspected. Findings include focal/diffuse enlargement, irregular enhancement, shaggy contour, fascial thickening, and fluid collections.

Relevant Guidelines

- **Atlanta Revision (2013) - Classification of Severity:**
  - **Mild:** Absence of organ failure and absence of local complications.
  - **Moderately Severe:** Local complications AND/OR transient organ failure (<48 hours).
  - **Severe:** Persistent organ failure (>48 hours).
    - *Organ Failure Definitions:* SBP ≤ 90 mmHg (Shock), PaO2 ≤ 60%, Creatinine ≥ 2.0 mg/dL, GI bleeding >500cc/24 hr.
- **Ranson's Criteria (Prognostic Signs):** Assessed at admission and during the initial 48 hours to predict mortality.
  - **At Admission (Non-gallstone):** Age >55, WBC >16,000, Blood glucose >200 mg/dL, LDH >350 IU/L, AST >250 U/dL. *(Note: Amylase is NOT a criterion).*
  - **At 48 Hours (Non-gallstone):** HCT fall >10%, BUN rise >5 mg/dL, Serum Calcium <8 mg/dL, Arterial PO2 <60 mmHg, Base deficit >4 mEq/L, Fluid sequestration >6 L.
- **Balthazar CT Severity Index (CTSI):** Combines CT Grade (0-4 points) with Necrosis Grade (0-6 points) to predict mortality and complications.
  - **CT Grades:** A (Normal = 0), B (Enlargement = 1), C (Peripancreatic fat infiltration = 2), D (Single fluid collection = 3), E (≥2 fluid collections = 4).
  - **Necrosis Grades:** None (0), <33% (2), 33-50% (4), >50% (6).
  - **Scoring:** 0-3 (Mortality 3%), 4-6 (Mortality 6%), 7-10 (Mortality 17%, Complications 92%).

Management

- **Mild Pancreatitis:**
  - Supportive care is the mainstay: NPO and IV fluids.
  - *Disproven therapies:* NG tubes, H2 blockers, anti-secretory agents (Somatostatin), and prophylactic antibiotics have **no benefit** in mild disease.
  - *Diet resumption:* Average 3-7 days. Criteria: absence of pain/tenderness and patient feeling hungry. Start with sips of water, build up to a low-fat/low-protein diet.
- **Severe Pancreatitis:**
  - **Sterile Necrosis (Uncomplicated):** Supportive + prophylactic antibiotics.
  - **Mild Complication / Suspected Infection:** CT-guided aspiration → Gram stain/culture → targeted antibiotics.
  - **Frank Complication / Toxicity / Shock:** Requires surgical debridement.
- **Nutritional Support in Severe Disease:**
  - TPN causes gastric mucosal atrophy, leading to bacterial translocation.
  - **Jejunal tube feeding is superior** to TPN and is preferred if NPO status extends >7 days.
- **Biliary Pancreatitis specific pathway:**
  - *Bilirubin dropping:* Laparoscopic cholecystectomy + intra-operative cholangiogram during the *same admission*.
  - *Bilirubin persists:* MRCP to confirm stone → ERCP → Laparoscopic cholecystectomy.

Complications / Prognosis

- **Mortality Timeline:**
  - **Early phase (Week 1):** 45-50% of deaths occur here. The most common cause of early death is **Respiratory Failure / ARDS** secondary to massive SIRS and capillary permeability.
  - **Late phase (Weeks 2-3):** Deaths are predominantly driven by sepsis due to infected pancreatic necrosis.
- **Pancreatic Pseudocyst:**
  - Encapsulated collection of enzymes, fluid, and necrotic debris in the lesser sac. *Lacks an epithelial lining*.
  - **Management:** Many resolve spontaneously. Asymptomatic cysts <5cm require only observation via periodic CT.
  - **Intervention rules:** Size/time are *not* the sole indications for drainage; symptoms drive intervention.
    - *Infected pseudocyst (abscess):* Requires **external** percutaneous drainage or surgical/endoscopic debridement.
    - *Mature, symptomatic, uninfected pseudocyst:* Requires **internal** drainage (surgical or endoscopic cystogastrostomy/cystojejunostomy).

Past-Paper High Yield

- **Amylase and Severity:** Amylase levels are used for diagnosis but do *not* correlate with disease severity or mortality. It is explicitly **NOT** part of the Ranson criteria.
- **Hypocalcemia Mechanism:** While lipase is secreted in its active form, it does *not* directly cause decreased serum calcium under normal conditions. Hypocalcemia in severe pancreatitis is due to fat saponification (calcium binding to free fatty acids released by autodigestion).
- **Enzyme Secretion Physiology:** Amylase and lipase are secreted in their active forms. Trypsinogen is secreted inactive and activated by enterokinase in the duodenum.
- **Differential Diagnosis Traps:**
  - Acute pancreatitis classically presents with epigastric pain radiating to the back. It is *rarely* confused with localized right lower quadrant (RLQ) pain (unlike Meckel's, gastroenteritis, testicular torsion, or right ureteric colic).
  - Hyperthyroidism is **NOT** a recognized cause of acute pancreatitis (though hyperparathyroidism is, via hypercalcemia).
  - Hyperthyroidism is also very rarely a cause of a severe "acute abdomen" (unlike DKA, acute intermittent porphyria, lead colic, or sickle cell crisis).
- **Pseudocyst Drainage:** Do *not* internally drain a frankly infected pseudocyst. Infected cysts require external drainage/debridement. Internal drainage is reserved for mature, uninfected, symptomatic cysts.
- **General Surgery Pearl (BUN post-op):** A rising BUN *without* a proportional rise in creatinine, particularly after vascular injury/surgery (e.g., hepatic artery injury), strongly indicates **progressive internal bleeding**, as blood proteins are absorbed by the gut.

Memory Pearls

- **Diagnosis:** Needs 2 of 3 (Pain, Enzymes >3x normal, CT findings).
- **Ranson's Admission Criteria: A**ge, **W**BC, **G**lucose, **L**DH, **A**ST (Amylase is absent!).
- **Most common cause of early death:** Respiratory failure (ARDS).
- **Most common cause of late death:** Sepsis (infected necrosis).
- **Nutrition:** Gut is best (Jejunal > TPN) to prevent bacterial translocation.

Pancreatic cancer

Core Concepts

- **Epidemiology:** 7th leading cause of cancer deaths worldwide. Predominantly affects patients >45 years old (peak incidence 65–79 years). Higher incidence in Western/industrialized nations and males (1.3:1).
- **Tumor Origins:**
  - >95% arise from exocrine elements.
  - **Ductal adenocarcinoma:** Accounts for 85–90% of all pancreatic neoplasms. It is an extremely aggressive malignancy.

    - **Neuroendocrine tumors:** Comprise <5% of cases.
- **Tumor Location:** Approximately 62% occur in the head of the pancreas, 10% in the body, and 6% in the tail.
- **Risk Factors:**
    - **Environmental:** Tobacco use (most significant), alcohol consumption, high dietary fat/caloric intake.
    - **Metabolic/Medical:** High fasting plasma glucose, high BMI, pancreatic cysts, chronic pancreatitis.
    - **Genetics (5–10% of cases):** Hereditary cancer syndromes (e.g., BRCA1/2 mutations, Peutz-Jeghers, Lynch syndrome, Li-Fraumeni).

Diagnosis / Clinical Features

- **Classic Presentation:** The most common initial symptoms are abdominal pain, jaundice, and weight loss.
- **Tumors in the Pancreatic Head:** Primarily present with painless, progressive obstructive jaundice (extrahepatic cholestasis).
- **Key Physical Signs:**
    - **Courvoisier's Sign:** Nontender but palpable, distended gallbladder at the right costal margin in a jaundiced patient.
    - **Trousseau's Syndrome:** Unexplained, migratory superficial thrombophlebitis reflecting a cancer-associated hypercoagulable state.
    - **Pancreatic Panniculitis (Rare):** Erythematous subcutaneous areas of nodular fat necrosis, typically located on the legs.
- **Signs of Advanced, Incurable Disease:**
    - Palpable abdominal mass or ascites.
    - Hepatomegaly (liver metastases).
    - **Virchow's Node:** Left supraclavicular lymphadenopathy.
    - **Sister Mary Joseph's Node:** Palpable periumbilical metastatic mass.

Investigations

- **Laboratories:** Elevated direct bilirubin and alkaline phosphatase (obstructive pattern), mild anemia. Tumor marker: **CA 19-9** (used for staging and post-op monitoring, not primary screening).
- **Initial Imaging of Choice:** A **multiphasic thin-slice CT scan of the abdomen (pancreatic protocol)** is the best initial modality for both diagnosing and non-invasively staging suspected pancreatic cancer.
- **ERCP / MRCP:** Classically reveals the **"double duct sign"**—concurrent, severe dilation of both the main pancreatic duct and the common bile duct ending abruptly at the pancreatic head mass. ERCP allows for forceps biopsy, brush cytology, and therapeutic biliary stenting.
- **Endoscopic Ultrasound (EUS):** Provides high-resolution imaging and is excellent for EUS-guided fine-needle biopsy of small lesions or evaluating chronic/autoimmune pancreatitis. (Not universally required if CT confirms clearly resectable disease).

Management

- **Surgical Resection:** The only potentially curative treatment. Only 15–20% of patients present with resectable disease.
- **Preoperative Considerations:** May include staging laparoscopy, preoperative biliary drainage (for severe jaundice/cholangitis), and neoadjuvant chemotherapy.
- **Surgical Procedures by Location:**
  - **Head of pancreas:** Pancreaticoduodenectomy (Whipple procedure).
  - **Body or Tail:** Distal subtotal pancreatectomy, usually combined with splenectomy.
  - **Entire gland:** Total pancreatectomy.

Relevant Guidelines

- **Continuum of Resectability for Pancreatic Adenocarcinoma:**
  - **High Resectability (Candidates for Surgery):**
    - No distant metastases.
    - No arterial or venous involvement.
    - Venous involvement (SMV or portal vein) is <180 degrees *with* suitable proximal and distal vessels for reconstruction.
    - Gastroduodenal artery encasement up to the common hepatic artery without extending to the celiac trunk.
  - **Low Resectability / Unresectable (Contraindications for Surgery):**
    - Distant metastases (e.g., peritoneum, liver).
    - Metastases to lymph nodes beyond the peripancreatic tissues.
    - >180-degree encasement or occlusion of the superior mesenteric artery (SMA).
    - Unreconstructable SMV or SMV-portal vein confluence occlusion.
    - Direct involvement of the IVC, aorta, celiac trunk, or hepatic artery (absence of fat plane on CT/EUS).
- **Inherited Cancer Syndromes Associated with Pancreatic Cancer:**
  - Hereditary breast/ovarian cancer (*BRCA1, BRCA2, PALB2*)
  - Familial atypical multiple mole melanoma (FAMMM) (*CDKN2A*)
  - Peutz-Jeghers syndrome (*STK11*)
  - Hereditary nonpolyposis colon cancer/Lynch syndrome (DNA mismatch repair genes)
  - Hereditary pancreatitis (*PRSS1, SPINK1*)

Operative / Procedural Notes

- **Pancreaticoduodenectomy (Whipple Procedure) Execution:**
  - **Resection margins:** The head of the pancreas, distal stomach, duodenum, gallbladder, and distal common bile duct are resected en bloc.
  - **Reconstruction (3 key anastomoses):**

1. Pancreaticojejunostomy (anastomosing the remnant pancreatic stump to the jejunum; can be end-to-side or end-to-end).
2. Choledochojejunostomy (biliary tree to the jejunum).
3. Gastrojejunostomy / Duodenojejunostomy (restoring GI continuity).

- **Total Pancreatectomy Consequences:** Results in permanent exocrine insufficiency (requiring enzyme replacement) and "brittle" diabetes (absolute lack of endogenous insulin and glucagon).

Complications / Prognosis

- **Survival Rates:** Extremely lethal malignancy. Even following successful curative resection, the 5-year survival rate for ductal adenocarcinoma is only ~20%.
- **Prognostic Factors:**
  - Nodal status (the most important prognostic factor in completely resected patients).
  - Tumor stage (TNM), surgical margin status, and tumor differentiation.
  - Lymphatic invasion and peri-operative CA 19-9 levels.
  - Pathological subtype: Papillary and mucinous cystadenocarcinomas carry a **better** prognosis than standard invasive ductal adenocarcinoma. Adenosquamous cancers carry a worse prognosis.

Past-Paper High Yield

- **Contraindications to Resection:** The presence of **peritoneal metastasis** indicates advanced, disseminated disease and is an *absolute contraindication* for curative pancreatic resection. Advanced age (>80 years), limited portal vein involvement (amenable to reconstruction), mild diabetes mellitus, or regional/peripancreatic lymph node invasion do *not* categorically preclude resection.
- **Primary Environmental Risk Factor: Tobacco use (smoking)** is the single most clearly established and significant environmental risk factor for the pathogenesis of pancreatic cancer.
- **Tumor Genetics & Prognosis:** The **p16** tumor suppressor gene mutation, alongside K-RAS and SMAD4, is found in >90% of ductal adenocarcinoma cases. Remember that ductal adenocarcinoma has a much worse prognosis compared to papillary and mucinous cystadenocarcinomas.
- **Optimal Imaging:** A **multiphasic thin-slice CT scan of the abdomen** (pancreatic protocol) is always the best initial imaging modality for diagnosing and staging suspected pancreatic cancer.
- **Chronic Pancreatitis Surgery:** Intractable, debilitating **abdominal pain** that fails medical and endoscopic therapy is the most common indication for surgical intervention in chronic pancreatitis (not diabetes or steatorrhea, which are managed medically).
- **Insulinoma Diagnostics (Endocrine Tumor):** In functional endogenous insulinomas, insulin is secreted alongside equimolar amounts of C-peptide. During a hypoglycemic episode, laboratory testing will reliably show **high insulin and high C-peptide** levels. Low C-peptide with high insulin firmly suggests surreptitious exogenous insulin administration.

Memory Pearls

- **Double Duct Sign:** Seen on MRCP/ERCP/CT; simultaneous dilation of the common bile duct and pancreatic duct strongly points to a pancreatic head mass.
- **Courvoisier's Law:** Painless jaundice + palpable non-tender gallbladder = unlikely to be gallstones; suspect pancreatic/biliary malignancy.
- **Sister Mary Joseph vs. Virchow:** Sister Mary Joseph = periumbilical node; Virchow = left supraclavicular node. Both mean Stage IV/unresectable disease.

Cystic tumors of the pancreas

Core Concepts

- **Epidemiology:** Increasing diagnostic rate (~2.6 per 100 individuals/year) due to better imaging, an aging population, and a lower threshold for abdominal CT scans.
- **WHO Classification:**
  - **Neoplastic:** Intraductal papillary mucinous neoplasms (IPMNs), Mucinous cystic neoplasms (MCNs), Serous cystadenomas (SCAs), Solid pseudopapillary tumors (SPTs).
  - **Non-neoplastic:** Inflammatory Pseudocysts (account for the majority of cystic lesions; surrounded by granulation tissue, lack true epithelium; occur in ~50% of acute pancreatitis cases).
- **Neoplastic Histological Grouping:**
  - **Mucin-producing (Malignant Precursors):** IPMN, MCN. Covered with endoderm-derived columnar epithelium.
  - **Nonmucin-producing (Usually Benign/Low-grade):** SCA, SPT. Lined by simple cuboidal epithelium.

Diagnosis / Clinical Features

- **General Presentation:** 20% of unexplained pancreatitis occurs due to underlying cystic neoplasms. Symptoms, when present, typically include abdominal pain, palpable mass, weight loss, or infrequently, jaundice.
- **Key Differentiators of Neoplastic Cysts:**

FEATURE	SEROUS CYSTADENOMA (SCA)	MUCINOUS CYSTIC NEOPLASM (MCN)	INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN)	SOLID PSEUDOPAPILLARY TUMOR (SPT/FRANTZ TUMOR)
Demographics	Females > 60 years	Females (9:1), 40s–50s	M = F, 50s–70s	Young females, 20s–30s
Location	Head of pancreas	Body / Tail (Distal)	MD (diffuse), BD (side branches)	Any, often large mass displacing organs
Imaging	**Microcystic / Honeycomb**, central calcified stellate scar	**Macrocystic**, unilocular, peripheral "eggshell" calcification	MD: Diffuse duct dilation (>5mm) BD: "Grape-like" clusters	Large, well-encapsulated, solid + cystic components, thick calcifications

FEATURE	SEROUS CYSTADENOMA (SCA)	MUCINOUS CYSTIC NEOPLASM (MCN)	INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN)	SOLID PSEUDOPAPILLARY TUMOR (SPT/FRANTZ TUMOR)
Duct connection	No	No	**Yes** (Diagnostic hallmark)	No
Pathology	Simple cuboidal	Tall columnar, **ovarian-type stroma**	Mucin-producing papillae	Necrosis/hemorrhage, irregular hypodensities
Malignant Risk	< 1%	10–50%	MD: 36–100% BD: 12–30%	10–15%

Investigations

- **Imaging Modalities:**
  - **CT Scan:** First line for anatomical delineation.
  - **MRCP:** Modality of choice to detect communication with the main pancreatic duct (essential for differentiating BD-IPMN from MCN or oligocystic SCA).
  - **EUS (Endoscopic Ultrasound):** Crucial for evaluating mural nodules and differentiating mucinous from non-mucinous cysts via fluid aspiration.
- **Cyst Fluid Analysis:**
  - **CEA Level:** Best predictor for mucinous lesions; **CEA > 192 ng/mL** strongly indicates a mucinous cyst (IPMN/MCN).
  - **Glucose:** Low glucose has diagnostic accuracy comparable to high CEA for mucinous lesions.
  - **Amylase:** Low amylase confidently *excludes* a pseudocyst. High amylase is nonspecific (can be elevated in pseudocysts, IPMN, SCA, or MCN).
  - **Fluid content:**
    - SCA = Serous fluid, low CEA.
    - MCN/IPMN = Mucin, high CEA.
    - SPT = Necrotic debris and blood.

Management

- **Pseudocysts:** Zero malignant potential; manage conservatively or with drainage if symptomatic/complicated.
- **Serous Cystadenomas (SCAs):**
  - Observation is standard since the risk of pancreatectomy outweighs the <1% malignancy risk.
  - *Resection indicated if:* Symptomatic, significant growth (>4cm size limits), unclear diagnosis, or concern for invasion.
- **Solid Pseudopapillary Tumors (SPTs):**
  - Typically cured with surgical resection.
  - Metastases (present in 15%) or locally advanced disease with vascular invasion are criteria for malignancy but *do not always preclude resection*, as long-term survival is often possible.
- **IPMN-MD & MCN Resection Principles:**

- High risk of High-Grade Dysplasia (HGD) or invasive carcinoma.
- Resection is generally recommended (see Guidelines below).

Relevant Guidelines

Mucinous Cystic Neoplasms (MCNs):

- **IAP (2012):** Recommends surgical resection for *all* MCNs regardless of size.
- **European Guidelines (2018):** Recommends resection *only* if: Cyst > 4 cm, enhancing mural nodules, or symptomatic (jaundice, pancreatitis, new-onset DM). Asymptomatic cysts < 4 cm can be observed (EUS every 6 months for 1 year, then yearly).

Main-Duct IPMN (IPMN-MD):

- **IAP (2017):** Surgical resection for all with duct > 10 mm. Dilation of 5–10 mm is a "worrisome feature" requiring EUS (no immediate resection recommendation).
- **European Guidelines (2018):** Dilation ≥ 10 mm is an *absolute* indication for resection. Dilation of 5–10 mm is a *relative* indication.

Branch-Duct IPMN (IPMN-BD) Resection Guidelines:

- **IAP (2017) Classification:**
  - **"High-Risk Stigmata" → Resect without further testing:** Symptomatic obstructive jaundice, enhancing mural nodule > 5 mm, Main Duct > 10 mm. (Associated with 40% risk of IPMN-related death).
  - **"Worrisome Features" → Evaluate with EUS:** Pancreatitis, cysts > 3 cm, enhancing/thickened nodule < 5 mm, MD 5–9 mm, abrupt change in PD caliber with distal atrophy, lymphadenopathy, increased CA19-9, cyst growth rate > 5 mm/2 years. (96% 5-year disease-specific survival with non-operative management).
- **European Guidelines (2018):**
  - **Absolute indications:** Jaundice, cytology positive for HGD/cancer, enhancing mural nodule > 5 mm, or a solid mass.
  - **Relative indications:** Growth > 5 mm/year, CA19-9 > 37 U/mL (without jaundice), MD 5–9.9 mm, cyst > 4 cm, new-onset DM/acute pancreatitis, enhancing mural nodule < 5 mm.

Operative / Procedural Notes

- **IPMN-MD Resection:**
  - Extent of resection is controversial but aims to remove all invasive carcinoma and High-Grade Dysplasia (HGD).
  - **Frozen section is mandatory** intraoperatively to ensure a free resection margin.
  - Positive margins for HGD imply a high risk of progression; Low-Grade Dysplasia (LGD) at the margin has a low recurrence risk.
  - Total pancreatectomy is indicated in select cases.

Complications / Prognosis

- **IPMN vs Ductal Adenocarcinoma:** IPMN-derived adenocarcinoma has a significantly better prognosis than primary pancreatic ductal adenocarcinoma (5-year survival 43–60% vs 15%). Primary ductal adenocarcinoma is far more associated with lymph node metastasis, advanced T-stage, perineural, and vascular invasion.
- **SPT Risk of Progression:** Associated with male gender, positive margins, positive lymph nodes, and lymphovascular invasion.

Past-Paper High Yield

- **IPMN Malignant Potential: Main-duct IPMN** carries the *highest* risk of malignant transformation to invasive pancreatic adenocarcinoma (30% to >70%) among cystic lesions. Standard guidelines strongly recommend surgical resection for *all* main-duct IPMNs in operative candidates. By contrast, pseudocysts have zero malignant potential, serous cystadenomas have near-zero risk, and branch-duct IPMNs/MCNs have a substantially lower risk than main-duct IPMN.
- **Mirizzi Syndrome Type II:** Although primarily a biliary issue, note that Mirizzi syndrome type II involves a cholecystobiliary fistula specifically affecting the **common bile duct** (Type I is purely extrinsic compression of the common hepatic duct).

Memory Pearls

- **CEA > 192: M**ucinous (**M**CN or IP**M**N).
- **Honeycomb / Central Stellate Scar: S**erous cystadenoma (Head, female 60+, Benign).
- **Eggshell calcification + Ovarian stroma:** MCN (Body/tail, female 40s).
- **Grape-like clusters + Duct communication:** BD-IPMN (MRCP is key).
- **Solid/cystic + Hemorrhage + Young Female:** SPT / Frantz tumor.

Inflammatory bowel disease

Core Concepts

- **Inflammatory Bowel Disease (IBD)** management requires a multidisciplinary team (MDT) including colorectal surgeons, gastroenterologists, specialized nurses, dieticians, and pathologists.
- **Ulcerative Colitis (UC)** is classically a continuous mucosal inflammatory disease universally involving the rectum and progressing proximally.
    - *Backwash Ileitis:* In severe pancolitis (10–20% of cases), inflammation can continuously "spill over" from the colon into the terminal ileum. This finding favors UC over Crohn's disease.
- **Crohn’s Disease (CD)** is characterized by discontinuous ("skip") lesions and transmural inflammation that can affect any part of the gastrointestinal tract (mouth to anus).
    - *Rectal Sparing:* Unlike UC, CD characteristically spares the rectum.

Diagnosis / Clinical Features

- **Ulcerative Colitis (UC)**

- Presents with painful, bloody diarrhea and tenesmus (a feeling of incomplete evacuation due to direct rectal mucosal inflammation).
- *Extra-intestinal manifestations (EIMs):*
  - **Respond to colectomy:** Peripheral arthritis, uveitis, iritis.
  - **Do NOT respond to colectomy:** Ankylosing spondylitis, sacroiliitis, Primary Sclerosing Cholangitis (PSC).

- **Crohn's Disease (CD)**
  - Classically presents with abdominal pain, stricturing/obstructive symptoms, and fistula formation (due to transmural inflammation).
  - Gross bleeding per rectum occurs but is less frequent than in UC.
  - Enterocutaneous and complex perianal fistulas are characteristic. *Note:* A complex perianal fistula typically presents with pain and purulent discharge, but does **not** classically cause tenesmus.

Investigations

- **Endoscopy / Histology**
  - *UC:* Mucosal inflammation, continuous involvement, crypt abscesses.
  - *CD:* Cobblestone appearance of the mucosa, strictly **non-caseating** granulomas, and discontinuous skip lesions.
- **Radiology**
  - *Barium Enema (UC):* Demonstrates a "lead pipe" appearance (a rigid, foreshortened colon lacking normal haustra) indicating chronic UC.
  - *Abdominal X-Ray (AXR):* Essential in acute presentations to evaluate for toxic megacolon (diameter > 5.5 cm or cecum > 9 cm).
- **Cancer Surveillance in UC**
  - High risk of malignancy (1-2% per year after 10 years). Risk factors: pancolitis, concomitant PSC (50% risk at 25 years), and dysplasia.
  - *Dysplasia Progression:*
    - Low-grade dysplasia (LGD) progression to High-grade dysplasia (HGD) or cancer: 0.5%–54%.
    - Risk of concomitant cancer is up to 19% in LGD, and up to 58% in HGD / Dysplasia-Associated Lesion or Mass (DALM).
    - ≥ 3 biopsies with LGD increases progression risk six-fold.

Management

- **Acute Severe Ulcerative Colitis**
  - Initial management involves IV steroids, DVT prophylaxis, rule out infections (C. diff, CMV), and daily surgical/MDT review.
  - A stool frequency > 8/day or CRP > 45 mg/L at Day 3 predicts the need for surgery in 85% of cases.

- IV steroids have no benefit beyond 7–10 days. By **Day 5**, if the patient is refractory to IV steroids, immediate escalation is required (rescue therapy with Cyclosporine/Biologics OR emergency colectomy).

- **Indications for Emergency Surgery in UC**
  - Refractory to medical management by Day 5.
  - Perforation (up to 40% mortality if performed after perforation vs. 2-8% before).
  - Hemorrhage.
  - Toxic megacolon with systemic toxicity.
- **Indications for Elective Surgery in UC**
  - Medical intractability (failed treatment, steroid dependence).
  - Poor quality of life (chronic hospitalizations, anemia, malnutrition).
  - Malignancy or significant dysplasia.
  - *Note:* The concurrent diagnosis of Primary Sclerosing Cholangitis (PSC) alone is **NOT** an indication for colectomy, as colectomy does not alter the clinical course of PSC.
- **Indications for Surgery in Crohn's Disease**
  - Surgery is strictly for *complications*: stenosis/stricture causing obstruction, fistulas (enterocutaneous, intra-abdominal), abscess, free perforation, or acute/chronic bleeding.
  - Preoperative malnutrition heavily increases the risk of poor tissue healing; **optimizing preoperative nutrition** is the most significant factor in decreasing anastomotic leak rates in CD surgery.

Relevant Guidelines

- **Severity Classification of Acute Ulcerative Colitis (Truelove & Witts Criteria)**
  - **Severe Episode:** ≥ 6 bloody stools/day, PLUS signs of systemic toxicity:
    - Heart Rate > 90 bpm
    - Temperature > 37.8^°C
    - Hemoglobin ≤ 10.5 g/dL
    - ESR (Hemosedimentation speed) > 30 mm/hr
- **Acute Severe Colitis Management Timeline**
  - **Day 1:** Initiate IV Steroids.
  - **Day 3:** Assess response. If poor (e.g., CRP > 45), discuss surgery and involve stoma therapist.
  - **Day 5:** Consideration of definitive colectomy vs. rescue therapy (Cyclosporine/Biologics).

Operative / Procedural Notes

- **UC Acute Surgery: Subtotal Colectomy and Ileostomy**
  - The procedure of choice in the acute emergency setting.
  - Leaves a rectal stump (closed, mucus fistula, or subcutaneous stump).
  - Mortality ~3%; stump blowout risk 2–12%. Allows confirmation of diagnosis, halts medical immunosuppression, and improves nutritional status before a delayed secondary reconstructive

stage (e.g., at 6 months).

- **UC Elective Surgery: Restorative Proctocolectomy with Ileal Pouch-Anal Anastomosis (IPAA)**
  - Curative "lifestyle operation" performed in one or two stages.
  - Pouch configuration (J-pouch vs. W-pouch) demonstrates **no statistically significant difference** in functional outcomes or stool frequency.
  - Rectal cuff surveillance post-IPAA is not necessary unless there was dysplasia/cancer in the original specimen.
- **Crohn's Disease Surgery**
  - Goal: Bowel conservation to prevent short bowel syndrome. Avoid wide resections.
  - **Segmental Resection:** Resecting only the overtly symptomatic diseased segment.
  - **Strictureplasty (Heineke-Mikulicz):** Longitudinal incision across a benign stricture, closed transversely to widen the lumen without sacrificing bowel length.
  - *Intraoperative finding:* Serosal involvement with fat wrapping ("creeping fat") is highly pathognomonic for CD.

Complications / Prognosis

- **Fibrotic Colonic Strictures**
  - *Crohn's Disease, Ischemic Colitis, and Diverticular Disease* frequently heal with intense fibrosis, leading to benign strictures.
  - *Ulcerative Colitis:* Benign fibrotic strictures are extremely rare in uncomplicated UC. If a stricture is found in a UC patient, it is highly suspicious for **malignancy** until proven otherwise.
- **Gastrointestinal Fistulas**
  - Crohn's disease is a classic cause due to transmural inflammation. Diverticulitis causes >50-60% of colovesical fistulas. Radiation, malignancy, and surgical trauma (most common iatrogenic cause) are also known culprits.
  - *Amoebic infections* cause colitis or liver abscesses but are **not** known to form fistulas.
- **Post-IPAA Complications (UC)**
  - *Pouchitis:* Occurs in up to 50% of patients.
  - *Pouch Failure:* 5.9% at 10 years (driven by pelvic sepsis, leaks, or delayed diagnosis of CD).
  - *Fertility:* Fecundity is reduced by 40–50% following restorative proctocolectomy.
  - *Function:* Nocturnal seepage (15% at 20 yrs) and urgency persist, though overall continence is acceptable.
- **Crohn's Disease Recurrence**
  - **Smoking** is the single most important, modifiable risk factor. It significantly increases disease onset, severity (linear dose-response), incidence of relapse, and recurrence after surgical resection.

Past-Paper High Yield

- **Clostridioides difficile Colitis:** The primary mechanism of antibiotic-induced pseudomembranous colitis is the loss of normal protective gut flora. Risk factors include broad-spectrum antibiotics, systemic steroids, and PPIs. *Cigar smoking is NOT a recognized risk factor.*
- **Tenesmus & Bleeding Differentials:** Tenesmus is caused by direct rectal vault mucosal irritation. UC, rectal adenomas, internal rectal prolapse, and radiation proctitis cause tenesmus. Complex perianal fistulas do not.
- **Lower GI Bleed:** Diverticulosis is the most common cause of massive, *painless* lower GI bleeding in older adults, often requiring transfusion.
- **Hemorrhoidal Bleeding:** Classically presents as bright red blood per rectum (hematochezia) coating the outside of the stool, dripping into the bowl, or seen on toilet paper. Dark blood intimately mixed within the stool indicates a proximal source (e.g., CRC, IBD).
- **Colovesical Fistula:** Diverticulitis is by far the most common underlying cause (>50-60%), followed by malignancy and Crohn's disease.
- **Pediatric Inguinal Hernias:** A strong, reliable history of an intermittent groin bulge from a parent is sufficient to justify elective surgical repair (herniotomy) in infants, even if the hernia is absent on clinical exam. Ultrasound is rarely needed.
- **Bilirubin Production:** The normal daily production of bilirubin in a healthy adult is approximately 250 to 300 mg/day (0.25 - 0.3 g/day).

Memory Pearls

- **UC hallmark triad:** Continuous, Mucosal, Rectum involved.
- **CD hallmark triad:** Skip lesions, Transmural, Rectum spared.
- **Stricture rule in UC:** "A stricture in UC is cancer until proven otherwise."
- **Day 5 UC Rule:** Severe UC failing IV steroids by day 5 → Cut (Subtotal Colectomy) or Cyclosporine/Biologic. Do not increase/switch steroids.

Intestinal obstruction

Core Concepts

- **Definition:** Interruption in the normal flow of intestinal contents.
- **Classification:**
  - **Mechanical:** Physical blockage (Simple or Strangulated).
  - **Functional (Ileus/Adynamic):** Interruption due to decreased motor activity (e.g., post-operative, metabolic, sepsis).
- **Small Bowel Obstruction (SBO) Etiologies:** Post-operative adhesions (most common), hernias, malignancy, Crohn's disease, volvulus, foreign bodies (bezoars, gallstones), and pediatric causes (atresia, pyloric stenosis, intussusception).
- **Large Bowel Obstruction (LBO) Etiologies:** Neoplasm (most common adult cause), diverticular or ischemic strictures, volvulus, intussusception (mostly tumor lead points in adults), and fecal impaction.

- **Closed-Loop Obstruction:** Obstruction at two points forming a sequestered segment (e.g., incarcerated hernia, volvulus, or colonic obstruction with a competent ileocecal valve). Highly susceptible to rapid ischemia and gangrene.
- **Pathophysiology:** Increased peristalsis initially → proximal distension → third space fluid loss/electrolyte imbalance → bacterial overgrowth/translocation → increased wall tension → circulatory compromise and ischemia.

Diagnosis / Clinical Features

- **Symptoms:**
  - **Pain:** Crampy, central, and intermittent in simple mechanical obstruction. A change to *constant* pain suggests strangulation/ischemia.
  - **Vomiting:** Greenish (bilious) indicates obstruction distal to the ampulla of Vater. Feculent vomiting is a late sign of prolonged obstruction. Projectile non-bilious suggests pre-ampullary (e.g., pyloric stenosis).
  - **Bowel function:** Obstipation (>24 hours) and failure to pass flatus. Diarrhea may occur in partial/intermittent obstruction.
- **Physical Examination:**
  - **Distention:** Less prominent in proximal SBO; massive in LBO.
  - **Auscultation:** Hyperactive, high-pitched ("tinkling") bowel sounds and borborygmi early. Diminished/silent in late obstruction or adynamic ileus.
  - **Palpation:** Check for surgical scars and carefully examine all hernia orifices (inguinal, femoral, umbilical).
  - **Digital Rectal Exam (DRE):** Empty vault suggests proximal obstruction; occult/gross blood suggests malignancy, late strangulation, or intussusception.
- **Strangulation Warning Signs:** Fever, tachycardia, severe continuous pain, leukocytosis, and peritoneal signs (rigidity, rebound tenderness). *Note: No reliable clinical exam definitively differentiates simple from early strangulated obstruction.*
- **Ileus vs. Mechanical:** Ileus presents with minimal pain, massive diffuse distention, and diminished bowel sounds without a discrete transition point.

Investigations

- **Computed Tomography (CT) Abdomen/Pelvis: The imaging modality of choice** for suspected mechanical obstruction.
  - Does not require oral contrast (retained fluid acts as contrast). Water-soluble IV/oral contrast is preferred if LBO is suspected.
  - Identifies transition point, distinguishes intrinsic vs. extrinsic vs. intraluminal causes, and differentiates ileus from mechanical post-op obstruction.
  - Best for identifying strangulation (bowel wall thickening, mucosal hypo-enhancement, fat stranding).
  - Best step to characterize any calcified abdominal mass causing symptoms.
- **Plain Radiographs:** Requires supine/flat and upright views.

    - **SBO:** Distended loops, central location, valvulae conniventes crossing the entire width, and "stepladder" air-fluid levels on upright film.
    - **LBO:** Peripheral distention with haustral markings that do not cross the full width.
    - **Ileus:** Generalized gas in both small and large bowel without a cut-off point; sluggish, long air-fluid levels.
    - **Pneumoperitoneum:** Upright chest/abdomen film screens for free air (perforation).
- **Contrast Enema:** Useful in distinguishing intussusception and volvulus ("bird's beak" or "ace of spades" sign for sigmoid volvulus).
- **Enteroclysis:** Fluoroscopy with contrast via nasojejunal tube. Distinguishes adhesions from metastases or radiation enteritis.
- **Ultrasonography:** Reliable for bedside exclusion of SBO (up to 89% accuracy).

Management

- **Initial Resuscitation (All Types):** NPO, nasogastric (NG) tube decompression (especially for vomiting/distension), aggressive IV fluid resuscitation (Isotonic saline or Ringer's), and electrolyte correction.
- **Non-Operative Management Indications (SBO):**
    - Adhesions (without strangulation signs).
    - Malignant tumor/carcinomatosis.
    - Crohn's disease (high-dose steroids + bowel rest).
    - Radiation enteritis.
    - Intra-abdominal abscess (treat with CT-guided drainage).
- **Emergency Surgical Indications:** Strangulated obstruction, closed-loop obstruction, bowel ischemia, or perforation.

Relevant Guidelines

- **Adhesive Small Bowel Obstruction (ASBO) Pathway:**
    1. Attempt NG tube decompression first.
    2. Administer Water-Soluble Contrast Medium (WSCM) for diagnostic and therapeutic purposes.
    3. Manage non-operatively for up to **72 hours** in the absence of strangulation/peritonitis.
    4. If unresolved after 72 hours, surgical intervention (open or laparoscopic adhesiolysis) is recommended.
- **Acute Colonic Pseudo-Obstruction (Ogilvie Syndrome) Pathway:**
    1. Initiate conservative management (bowel rest, hydration, address underlying disorder) for the first 24 hours (if no perforation).
    2. Pharmacologic intervention: IV Neostigmine.
    3. Endoscopic intervention: Colonoscopic decompression (effective in ~80% of cases).
    4. Surgery: Reserved for refractory cases, ischemia, or perforation (associated with high morbidity/mortality).

Operative / Procedural Notes

- **Volvulus Interventions:**
  - **Sigmoid Volvulus:** First-line is endoscopic detorsion/decompression (rigid or flexible sigmoidoscopy). **Surgical resection is always indicated post-decompression** due to a ~50% recurrence rate. If necrotic/perforated, proceed to emergency laparotomy.
  - **Cecal Volvulus:** Endoscopic detorsion is highly unsuccessful and carries a high risk of perforation. **Primary surgical treatment is Right Hemicolectomy.**
- **LBO Malignancy Resection:**
  - Left colon carcinoma: Resection without primary anastomosis OR resection with primary anastomosis and intraoperative lavage. Endoscopic stenting (SEMS) may be used as a bridge to elective surgery.
  - Right colon carcinoma: Right colectomy with primary anastomosis (ileum to transverse colon).
- **Intussusception:** Contrast enema reduces 60-80% in children. Adults usually require surgery due to a high likelihood of an underlying malignant lead point.

Complications / Prognosis

- **Strangulated SBO:** 100% mortality if untreated. Drops to 8% if operated within 36 hours. Delaying >36 hours increases mortality to 25%.
- **Large Bowel Obstruction:** Overall mortality is ~20%. Increases to 40% if colonic perforation occurs. The cecum is the most likely area to perforate due to Laplace's Law.
- **Ogilvie Syndrome:** 15% mortality with early care; jumps to 36% if ischemia or perforation develops.
- **Short-bowel syndrome:** Potential late complication resulting from repeated resections for recurrent SBO.

Past-Paper High Yield

- **Volvulus Distinctions:**
  - *Sigmoid Volvulus:* Shows a "coffee bean" sign on X-ray. Pre-requisite is a *narrow* mesentery with a redundant sigmoid (a wide mesentery *prevents* twisting). Risk factors include old age, chronic constipation, African/high-fiber diet, and pregnancy. 180° twist causes luminal obstruction; ≥360° twist is typically required for irreversible ischemia.
  - *Cecal Volvulus:* Displaced to mid/LUQ. Treatment is always a Right Hemicolectomy. Do not attempt endoscopic reduction.
- **Abdominal Wall Hernias (Common Traps):**
  - *Pregnancy:* Elective repair of asymptomatic hernias is contraindicated and delayed until postpartum.
  - *Pediatric:* Unrepaired indirect inguinal hernias have a **~40% incarceration risk** in the first year of life (mandates early repair).
  - *Spigelian Hernia:* Occurs at the semilunar line, lateral to rectus abdominis. Often has a **narrow neck**, giving it a very **high risk of incarceration/strangulation**. Requires mesh repair.

    - *Deep Inguinal Ring Anatomy:* Located 1.5 cm **ABOVE** the mid-inguinal point (not below). Indirect hernias traverse this ring.
    - *Direct Hernias:* Located medial to the inferior epigastric vessels. Femoral hernias have narrow necks (high strangulation risk).
- **Simple vs. Complicated Obstruction:** Simple mechanical obstruction classically has hyperactive "tinkling" bowel sounds early, mild leukocytosis, and oliguria. Board-like rigidity indicates a perforated viscus/peritonitis, *not* simple obstruction.
- **CT utility:** A CT abdomen is always the best next step to fully characterize a calcified abdominal mass (gallstone ileus, malignancy) and evaluate for bowel obstruction.
- **Vomiting types:** Bilious (green) = distal to ampulla of Vater. Projectile non-bilious = proximal to ampulla. Feculent = very distal.
- **Pediatric lower GI bleeding:** Meckel's diverticulum (contains ectopic gastric mucosa) is the classic cause of massive, *painless* lower GI bleeding in an infant. Intussusception causes *painful* currant-jelly stools.
- **Rectus Sheath Hematoma:** Fothergill's sign is positive (mass does not cross midline and remains palpable when the patient tenses their abdominal muscles). Usually older patients on anticoagulation with coughing fits.

Memory Pearls

- **Laplace Law (T = P × R):** The cecum has the largest radius (R) in the colon, subjecting it to the highest wall tension (T). Therefore, it is the most common site of colonic perforation in LBO/closed-loop obstructions.
- **Rigler's Triad:** Pathognomonic for Gallstone Ileus → 1. Pneumobilia, 2. Small bowel obstruction, 3. Ectopic radiopaque gallstone (usually in right lower quadrant/terminal ileum).
- **Bird's Beak vs. Coffee Bean:**
    - *Coffee bean / bent inner tube* = Plain X-ray sign of Sigmoid Volvulus.
    - *Bird's beak / ace of spades* = Contrast enema fluoroscopy sign of Sigmoid Volvulus.

Mesenteric ischemia

Core Concepts

- **Definitions:**
    - **Mesenteric ischemia:** Ischemia affecting the small intestine.
    - **Colonic ischemia:** Ischemia affecting the large intestine.
    - **Splanchnic ischemia:** Broad term encompassing ischemia of the intestines, liver, spleen, or kidneys.
- **Physiology:** Splanchnic circulation receives 10–35% of cardiac output. Due to high capillary density and baseline low oxygen extraction, intestinal blood flow must drop by >50% from fasting levels before oxygen delivery is compromised.
- **Arterial Anatomy & Relations:**

    - **Celiac Trunk:** Trifurcates directly into the **common hepatic artery, splenic artery, and left gastric artery**.
    - **Aortic Branches:** Superior mesenteric (SMA), inferior mesenteric (IMA), renal, and gonadal arteries branch *directly* off the abdominal aorta independently.
    - **Duodenal Relations:** The head of the pancreas sits snugly within the C-loop of the duodenum. The 3rd part of the duodenum and the uncinate process pass **posterior** to the superior mesenteric vessels. The 2nd part of the duodenum is retroperitoneal.
- **Collateral Circulation:** Protects against gradual/chronic occlusions.
    - Between Celiac and SMA: Superior and inferior pancreaticoduodenal arteries.
    - Between SMA and IMA: Marginal artery of Drummond and the meandering mesenteric artery (Arc of Riolan).
- **Watershed Zones:** Vulnerable to systemic hypoperfusion due to narrow terminal branches.
    - **Griffiths' point:** Splenic flexure (junction of SMA and IMA territories).
    - **Sudeck's point:** Rectosigmoid junction (junction of IMA and internal iliac systemic circulation).

Diagnosis / Clinical Features

- **Acute Mesenteric Arterial Occlusion (Arterial Embolism & Thrombosis):**
    - Classic hallmark: **Abdominal pain out of proportion to physical examination**.
    - **Arterial Embolism:** Sudden, severe, periumbilical pain; often with nausea/vomiting. Causes greater blood flow reduction due to a lack of pre-existing collaterals.
    - **Arterial Thrombosis:** Acute-on-chronic presentation; patients often report preceding, worsened postprandial pain.
- **Mesenteric Venous Thrombosis (MVT):** Insidious onset of abdominal pain. Rarely involves the colon. Chronic MVT is associated with cavernous transformation of the portal vein.
- **Nonocclusive Mesenteric Ischemia (NOMI):** Variable presentation. Caused by severe primary mesenteric vasoconstriction/hypoperfusion rather than mechanical blockage.
- **Chronic Mesenteric Ischemia:**
    - Fundamentally an atherosclerotic arterial disease.
    - Classic triad: Postprandial pain ("intestinal angina"), "food fear," and resulting significant weight loss.
- **Colonic Ischemia (Ischemic Colitis):**
    - Predominantly in patients >60 years old.
    - Caused by transient global low-flow states (heart failure, MI, sepsis, hemorrhage).
    - Presents with mild abdominal pain, rectal bleeding, and bloody diarrhea. Patients do not appear severely ill.

Investigations

- **Laboratory Studies:**
    - Highly nonspecific. **Normal laboratory values do NOT exclude acute mesenteric ischemia**.

- Findings: Marked leukocytosis, elevated hematocrit, metabolic acidosis, elevated lactate, and elevated serum amylase.

- **Radiological Imaging:**
  - **Plain Radiograph:** Early signs include thick-walled, distended, featureless bowel loops. **Pneumatosis intestinalis** (air in bowel wall) is a late sign indicating transmural necrosis.
  - **CT Angiography (CTA):** Primary diagnostic modality.
    - Embolus typically lodges 1–3 cm distal to the SMA origin (sparing proximal branches like the middle colic artery).
    - Thrombosis often shows severe calcific ostial stenosis at the SMA/Celiac origins.
    - Diagnosis of chronic ischemia requires high-grade stenosis in at least 2 of 3 major splanchnic arteries.
  - **Catheter-Based Angiography (DSA):** Traditional gold standard. Retained for equivocal cases or for therapeutic access (intra-arterial papaverine/thrombolysis).
- **Endoscopy:** Colonoscopy is the procedure of choice for suspected **colonic ischemia**.

Management

- **Initial Resuscitation (All Acute Cases):**
  - Gastrointestinal decompression (NPO/NG tube).
  - Aggressive fluid resuscitation & hemodynamic support.
  - **Avoid vasopressors** (exacerbates ischemia).
  - Broad-spectrum antibiotics and supplemental oxygen.
  - Systemic anticoagulation (under most circumstances).
- **Etiology-Specific Management:**
  - **Arterial Embolism:** Early open laparotomy with embolectomy, OR catheter-directed thrombolysis (if high surgical risk and no peritonitis). Long-term: VKA or DOACs.
  - **Arterial Thrombosis / Chronic Ischemia:** Surgical bypass OR endovascular angioplasty/stenting. Long-term: Antiplatelet therapy and statins (lifelong anticoagulation is *not* indicated here).
  - **Venous Thrombosis (MVT):** Primarily conservative. Systemic anticoagulation and bowel rest. Surgery strictly reserved for overt bowel infarction.
  - **NOMI:** Reverse the underlying cause (sepsis, heart failure, stop pressors/cocaine) and intra-arterial infusion of vasodilators (e.g., papaverine) into the SMA.
- **Colonic Ischemia:** Generally self-limiting and managed medically (supportive care).

Relevant Guidelines

- **Diagnostic Rule:** Any patient with acute abdominal pain and a metabolic acidosis has intestinal ischemia until proven otherwise.
- **Hypercoagulability Workup (for MVT):** Should include Protein S, Protein C, Antithrombin III, Factor V Leiden, APC resistance, and Anticardiolipin/Antiphospholipid/Lupus antibodies.

Operative / Procedural Notes

- Patients with signs of peritonitis or obvious infarction must bypass advanced imaging and proceed directly to exploratory laparotomy.
- **Intra-op Bowel Viability Assessment:**
  - Reassess *after* perfusion is restored.
  - Check color, distension, peristalsis, mesenteric arterial pulsations, and bleeding from cut edges.
  - Adjuncts: Handheld Doppler over the serosa; Intravenous **fluorescein dye** evaluated under a **Wood's lamp** (viable bowel fluoresces green).
  - Marginally viable bowel should be left in situ for a planned "second-look" operation (24-48 hours later).

Complications / Prognosis

- **Overall Mortality:** Highly lethal, exceeding 60%. Time to diagnosis is the paramount determinant of survival.
- Requiring advanced bowel resection is associated with a **15-fold increase in mortality**.
- **Etiology-Specific Prognosis:**
  - Venous thrombosis has the best prognosis.
  - NOMI has the worst prognosis (70–90% mortality rate).
- **Survivors of Massive Resection:** Frequently develop Short Bowel Syndrome, requiring lifelong Total Parenteral Nutrition (TPN).

Past-Paper High Yield

- **Most common cause of acute mesenteric ischemia:** Arterial embolism (responsible for ~50% of cases), typically from a cardiac source (AFib) lodging in the SMA. (Arterial thrombosis = ~25%, NOMI = ~20%).
- **Visceral Artery Aneurysms:** The **splenic artery** is by far the most common site, accounting for ~60% of all cases. (Hepatic, SMA, celiac, IMA aneurysms are much less common).
- **Gastroduodenal Artery Bleeding:** Runs directly posterior to the 1st part of the duodenum. A posteriorly penetrating duodenal ulcer classically erodes this artery, causing massive life-threatening upper GI hemorrhage.
- **Celiac vs. Aortic Origins:** Celiac trunk trifurcates into the common hepatic, splenic, and left gastric arteries. The SMA, IMA, renal, and gonadal vessels branch directly off the aorta.
- **Duodenal Surgical Anatomy:** The 3rd part of the duodenum passes *posterior* (not anterior) to the SMA and SMV.
- **Chronic Mesenteric Ischemia Misconceptions:** It is an atherosclerotic arterial disease treated with bypass/stenting and antiplatelets. It does *not* require lifelong anticoagulation (which is the treatment for mesenteric *venous* thrombosis).

Memory Pearls

- **Arterial vs. Venous Anticoagulation Rules:** Lifelong anticoagulants = Venous clot or cardiac embolus. Antiplatelets/Statins = Arterial atherosclerotic plaque (Thrombosis/Chronic).

- **Pain out of proportion** = Ischemic bowel until proven otherwise.
- **Food fear + Weight loss** = Chronic mesenteric ischemia ("Intestinal angina").
- **Wood's Lamp + Fluorescein** = Intra-op viability check to save maximum bowel length.

Small bowel tumors

Core Concepts

- **Epidemiology:** Rare tumors accounting for only 1–2% of all GI malignancies (100 times less frequent than in the stomach, esophagus, or colorectum). Equal incidence in men and women.
- **Distribution Rule of Thumb:** Benign lesions are more common distally; Adenocarcinomas are more common proximally.
- **Tumor Origins:**
  - **Epithelium:** Adenomas, adenocarcinomas, carcinoids.
  - **Lymphatic tissue:** Lymphomas.
  - **Mesenchymal/Neural:** Gastrointestinal stromal tumors (GIST), leiomyomas, lipomas, hemangiomas, neuromas, sarcomas.
- **Risk Factors for Small Bowel Tumors:**
  - Familial adenomatous polyposis (FAP): 24–93% develop small intestine polyps; 2–12% develop duodenal cancer.
  - Hereditary nonpolyposis colorectal cancer (HNPCC).
  - Peutz-Jeghers syndrome.
  - Crohn’s disease: Associated with ileal adenocarcinoma.
  - Celiac disease (gluten-sensitive enteropathy): High risk for GI lymphoma.
  - Biliary diversion (e.g., previous cholecystectomy).

Diagnosis / Clinical Features

- **General Presentation:** Typically present in the 6th and 7th decades.
- **Benign Neoplasms:** Often asymptomatic (47–60%) and found incidentally. When symptomatic, present with acute GI hemorrhage, anemia, intermittent obstruction, or vague abdominal pain.
- **Malignant Neoplasms:** Almost always symptomatic.
  - Abdominal pain is the most common symptom (62–83%).
  - Other features: weight loss (38–55%), nausea/vomiting, anemia, acute hemorrhage, or palpable abdominal mass.
  - Obstruction occurs in 15–35% of patients (due to tumor, adhesions, or infiltration).
- **Carcinoid Tumors:**
  - Arise from enterochromaffin (argentaffin) cells.
  - Characteristically yellow-colored, slow-growing, submucosal/serosal lesions.
  - Most common in the ileum (last two feet). Pain is the most common presentation.

    - **Carcinoid Syndrome:** Vasomotor (flushing), GI (diarrhea, abdominal cramps), respiratory (wheezing), and cardiac (right heart failure) manifestations. Triggered by systemic circulation of peptides (serotonin/5-HT). Requires large tumor burden bypassing the liver or, most commonly, **liver metastases**.
- **Crohn's-associated Adenocarcinoma:** Occurs in the ileum, presents ~20 years younger than sporadic cases, has a strong male preponderance (70%), and carries a very poor prognosis.

Investigations

- **Imaging:** CT / MRI Enterography, Small bowel follow-through (contrast studies showing filling defects/luminal narrowing).
- **Endoscopy:** Enteroscopy, Capsule endoscopy (especially for occult bleeding).
- **Angiography:** Useful for highly vascularized lesions (e.g., prominent tumor blush in GIST or carcinoids).
- **Laboratory:** 24-hour urinary 5-HIAA (high-performance liquid chromatography) is highly specific but not sensitive for carcinoid tumors.

Management

- **Adenomas (True, Villous, Brunner's gland):** Excision/biopsy and follow-up. Malignant potential increases with size, site (periampullary = higher risk), and number.
- **Adenocarcinoma:** Segmental resection including regional lymph nodes. Chemotherapy is generally of little help.
- **Carcinoid Tumors:**
    - Local disease: Resection including draining lymph nodes.
    - Metastatic disease: Tumor debulking, hepatic artery embolization/chemoembolization, radiofrequency ablation, or cryotherapy.
    - Systemic therapy: Somatostatin analogs (Octreotide, Lanreotide). Cytotoxic chemotherapy is ineffective.
- **GI Lymphoma:** Treatment is primarily **medical** (systemic chemotherapy/radiotherapy).
- **GIST:** Surgical excision. Imatinib (tyrosine kinase inhibitor) used in advanced cases (can achieve 50% tumor shrinkage). Lesions are typically radio-resistant.

Relevant Guidelines

- **Carcinoid Tumor Metastatic Risk & Management Stratification:**
  Metastatic potential correlates directly with tumor size:
    - **< 1 cm:** 20–30% risk of metastases to LN/liver. Adequately treated with local excision.
    - **1–2 cm:** 60–80% LN risk, 20% liver risk.
    - **> 2 cm:** 80% LN risk, 40–50% liver risk.
- **GIST Prognostic Stratification (Low Risk):**
    - Tumor size < 2 cm.
    - Mitotic rate < 5 per high-power field (HPF).

Operative / Procedural Notes

- **Adenocarcinoma / Larger Carcinoids (>1 cm):** Require wide segmental resection of the involved bowel along with the mesentery to ensure radical regional lymphadenectomy.
- **GIST:** Lymphatic spread is uncommon. Surgery requires complete excision with negative margins; routine lymphadenectomy is not typically required.
- **GI Lymphoma:** Surgery is explicitly reserved ONLY for acute complications (bleeding, perforation, or obstruction).

Complications / Prognosis

- **Adenocarcinoma:** Overall 5-year survival is 20–30%. If node-negative at operation, survival increases to 50–70%.
- **Carcinoid Tumors:** May cause local complications like small bowel obstruction, intense mesenteric fibrosis, and ischemia. Most patients already have lymph node or liver metastases at presentation.
- **GIST:** Small bowel GISTs carry a worse prognosis compared to stomach or esophageal GISTs.
- **GI Lymphoma:** Favorable overall, with localized disease 5-year survival generally > 50%.

Past-Paper High Yield

- **Carcinoid Multifocality:** Small bowel carcinoids are multifocal (multicentric) in **30–40%** of cases. Do not fall for the trap stating they are rarely multiple or only multiple in 3% of cases.
- **Carcinoid Metastatic Potential:** Midgut carcinoids (specifically of the **ileum**) have the highest propensity for malignancy and metastasis (to nodes and liver) compared to those in the appendix, rectum, stomach, or bronchus.
- **Carcinoid Syndrome Physiology:** Syndrome only occurs when vasoactive substances (e.g., serotonin) bypass hepatic metabolism. This means it classically requires the presence of **liver metastases**. The tumors arise from enterochromaffin cells and secrete serotonin.
- **GI Lymphoma Paradigm:** It is highly tested and **FALSE** that GI lymphomas cannot be treated with chemo/radiotherapy. They are primarily treated with systemic chemotherapy (e.g., R-CHOP) and radiotherapy. Surgery is not universally necessitated and is strictly reserved for complications.
- **GI Lymphoma Epidemiology:** The GI tract is the most common extra-nodal site for lymphoma. The vast majority are Non-Hodgkin B-cell type. The stomach is the most frequent site overall, followed by the small bowel (specifically the ileum).

Memory Pearls

- **Malignancy distribution:** Proximal = Adenocarcinoma (usually); Distal (Ileum) = Carcinoids & Lymphomas.
- **Mesenchymal standout:** GIST = No lymphatic spread (do not need routine LN dissection). Liver/peritoneal mets are common.
- **Rule of 50s for Carcinoids:** >2cm tumors have ~50% (40-50%) chance of liver mets.

Acute appendicitis

Core Concepts

- **Anatomy**:
  - The appendix is a true diverticulum of the cecum located at the base of the cecum near the ileocecal valve.
  - **Blood supply**: The appendiceal artery is a terminal branch of the ileocolic artery.
- **Epidemiology**:
  - Peaks in the second and third decades of life (highest incidence in the 10-19 age group).
  - Male to female ratio is 1.4:1.
- **Pathogenesis**:
  - **Classic Surgical Teaching**: Luminal obstruction is the primary initiating pathophysiological event and is a **mandatory prerequisite** for acute appendicitis to develop.
  - **Causes of obstruction vary by age**:
    - *Young patients*: Lymphoid follicular hyperplasia (often secondary to infection) is the most common cause.
    - *Older patients*: Fibrosis, fecaliths (hard fecal masses), or neoplasia (carcinoid, adenocarcinoma, mucocele).
    - *Endemic areas*: Parasites.
- **Pathophysiological Cascade**:
  - Luminal obstruction → increased luminal and intramural pressure → thrombosis/occlusion of small vessels → lymphatic/vascular stasis → ischemia → necrosis.
  - **Bacterial overgrowth**: Aerobic organisms predominate early; mixed infections are common late in the course.
- **Pain Referral Pathway**:
  - *Early*: Engorgement stimulates visceral afferent nerves (T8-T10) leading to vague central/periumbilical pain.
  - *Late*: Inflammation involves the parietal peritoneum leading to well-localized somatic right lower quadrant (RLQ) pain.

Diagnosis / Clinical Features

- **Classic Presentation**:
  - Abdominal pain is the **first symptom** (periumbilical migrating to RLQ in 50-60% of cases).
  - Followed by anorexia, nausea, and vomiting.
  - Fever occurs *later* in the illness course.
- **Atypical Presentations**:
  - Generalized malaise, indigestion, flatulence, bowel irregularity, or diarrhea.
  - **Anatomical variation heavily influences symptoms**:

        - A pelvic appendix or pelvic abscess can irritate the rectum, presenting as new-onset diarrhea.
- **Physical Examination Signs**:
    - **McBurney's Point Tenderness**: Maximal tenderness 2/3 of the distance from the umbilicus to the anterior superior iliac spine (ASIS).
    - **Rebound Tenderness (Blumberg Sign)**: Pain upon sudden release of abdominal pressure.
    - **Rovsing's Sign**: Palpation of the left lower quadrant (LLQ) elicits pain in the RLQ.
    - **Psoas Sign**: RLQ pain with passive right hip extension. Indicates a **retrocecal** appendix lying against the psoas muscle.
    - **Obturator Sign**: RLQ pain with flexion and internal rotation of the right hip. Indicates a **pelvic** appendix lying against the obturator internus muscle (low sensitivity, rarely performed).

Investigations

- **Laboratory Findings**:
    - Mild leukocytosis (WBC >10,000 cells/microL) with a "left shift" is present in ~80% of patients.
    - Elevated serum C-reactive protein (CRP).
- **Imaging Studies**:
    - *Ultrasound*: Accurate for diagnosis if appendiceal diameter is **>6 mm**.
    - *CT Abdomen/Pelvis (with contrast)*: The modality of choice for equivocal cases. Key findings include:
        - Enlarged appendiceal diameter >6 mm with an occluded lumen.
        - Appendiceal wall thickening (>2 mm) and wall enhancement.
        - Periappendiceal fat stranding.
        - Appendicolith (seen in ~25% of patients).
    - *Plain Radiographs*: Generally not helpful for establishing the diagnosis.
- **Routine Pathological Examination**:
    - Every surgically removed appendix **must** be carefully labeled and sent for routine histopathology.
    - *Clinical Importance*: Confirms appendicitis and is mandatory to rule out unexpected pathologies (e.g., carcinoid tumors, adenocarcinoma, Crohn's disease). Discarding the specimen is considered malpractice.

Relevant Guidelines

Alvarado Score (Used to rule in/out acute appendicitis)

- **1 Point Parameters**:
    - Migratory RLQ pain
    - Anorexia
    - Nausea or vomiting
    - Rebound tenderness in RLQ

    - Fever >37.5°C (>99.5°F)
- **2 Point Parameters**:
    - Tenderness in RLQ
    - Leukocytosis (WBC >10 × 10^9/L)
- **Interpretation & Pathway**:
    - **<4 Points**: Appendicitis unlikely; evaluate for other diagnoses.
    - **4 to 6 Points**: Equivocal. Perform CT (or US if available). If non-diagnostic, observe for 12 hours and reassess. If score drops <4, evaluate other diagnoses. If score rises ≥ 4, proceed to laparoscopy/appendectomy.
    - **≥ 4 Points (General)**: Evaluate further via imaging (CT/US).
    - **>6 Points**: High probability. Warrants surgical exploration (laparoscopy/appendectomy).

Management

- **Uncomplicated (Non-perforated) Appendicitis**:
    - **Definitive Treatment**: Immediate appendectomy (within 12 hours), typically laparoscopic.
    - **Supportive Care**: IV fluids, pain control, and pre-operative IV antibiotics.
    - *Contraindication*: Colonoscopy is absolutely contraindicated due to the risk of organ rupture. Medical management (oral antibiotics + discharge) or indefinite observation is inappropriate.
- **Complicated (Perforated) Appendicitis**:
    - *Unstable / Free Perforation / Generalized Peritonitis*: Emergent appendectomy (laparoscopic or open) with irrigation and drainage of the peritoneal cavity. Post-operative IV antibiotics for 3-5 days.
    - *Stable with Localized Abscess*: Image-guided percutaneous drainage + intravenous antibiotics. Immediate surgery is an alternative only if the abscess is not amenable to drainage. Followed by discharge on oral antibiotics (7-10 days) and an interval appendectomy 6-8 weeks later.
    - *Stable with Phlegmon*: Intravenous antibiotics and observation. Repeat imaging to track resolution. If failure occurs, perform a rescue appendectomy.

Post-operative Care / Procedural Notes

- **Post-operative Antibiotics**: For an **uneventful (uncomplicated)** appendectomy, post-operative antibiotics are **NOT** indicated. Routine use for 3 days does not reduce hospital stay; it only increases resistance and cost.
- **DVT Prophylaxis**: Early mobilization significantly reduces the risk of post-operative deep vein thrombosis (DVT).
- **Surgical Site Infection (SSI)**: Wound swelling and purulent discharge post-operatively are classic signs of SSI.
- **Intra-operative Dilemmas**: If clinical suspicion is extremely high but imaging is non-diagnostic/unavailable, surgical exploration is warranted. Frozen sections of the appendix are *not* routinely indicated unless intra-operative malignancy is overtly suspected.

Associated High-Yield Surgical/GI Concepts

(Mapped directly from GI rotation past-papers)

- **Hepatic Abscesses**:
  - *Pyogenic Liver Abscess*: The most common cause today is ascending biliary tract infection (cholangitis), often secondary to gallstones or strictures. (Appendicitis via pylephlebitis is a less common historical cause). Almost always requires drainage.
  - *Amebic Liver Abscess*: Patients present with a travel history to endemic regions + *Entamoeba histolytica*. Responds rapidly to medical therapy alone (metronidazole followed by paromomycin). **Avoid** routine percutaneous/surgical drainage as first-line therapy due to the high risk of secondary bacterial superinfection; drain only if rupture is imminent or antibiotics fail.
- **Primary Iliopsoas Abscess**: Standard first-line management is image-guided percutaneous drainage combined with targeted systemic antibiotics. (Do not observe, do not use antibiotics alone, do not immediately do open debridement).
- **Child-Pugh Classification**: Assesses chronic liver disease prognosis based on 5 parameters. It uses **Prothrombin Time (PT) or INR** to assess synthetic coagulation function, *not* Partial Thromboplastin Time (PTT). Parameters: Bilirubin, Albumin, PT/INR, Ascites, Encephalopathy.

Past-Paper High Yield

- **Pathophysiology Trap**: You *must* recognize luminal obstruction as a mandatory prerequisite for acute appendicitis. Stating it is "not a must" is false.
- **Specimen Protocol**: You *must* send every uncomplicated appendix to histology. Never discard it, give it to the family, or send it exclusively for culture.
- **Antibiotic Trap**: For a simple appendectomy, do *not* prescribe a 3-day post-op course of IV antibiotics.
- **Classic Presentation vs. Anatomy Trap**: Remember that an inflamed pelvic appendix can present with new-onset diarrhea due to direct rectal irritation.
- **Management Dilemmas**: For a clinically certain appendicitis, surgery is definitive. For equivocal cases (Alvarado 4-6), utilize CT imaging. Never send a clear-cut case home on oral antibiotics, and never perform a colonoscopy during an acute attack.
- **Abscess Management Rules**:
  - *Appendiceal/Iliopsoas Abscess* → Image-guided percutaneous drainage + systemic antibiotics.
  - *Pyogenic Liver Abscess* → Drainage + antibiotics (most commonly from biliary tree).
  - *Amebic Liver Abscess* → Medical therapy ONLY (metronidazole + paromomycin). Routine drainage is highly contraindicated!
- **Child-Pugh Trap**: The scoring system uses PT/INR, *not* PTT.

Diverticular diseases

Core Concepts

- **Definition:** Diverticular disease involves the acquired herniation of the mucosa and submucosa through the muscular wall of the colon.
- **True vs. False Diverticulum:**
  - **False (Pulsion) Diverticulum:** Contains only mucosa and submucosa herniating through a defect in the muscularis layer. Colonic diverticula, Zenker's, duodenal, jejunal, and epiphrenic diverticula are all false.
  - **True Diverticulum:** Contains all three layers of the bowel wall (mucosa, submucosa, and muscularis propria). A classic example is **Meckel's diverticulum**.
- **Pathophysiology of Colonic Diverticula:**
  - Herniation occurs at a point of anatomical weakness between the mesenteric and antimesenteric taenia, specifically where penetrating *vasa recta* blood vessels traverse the muscularis propria.
  - **Etiology:** A low-fiber diet results in smaller stool volumes, requiring high colonic wall tension and high intraluminal pressure for propulsion. This chronic contraction leads to muscular hypertrophy and "segmentation" (the colon acts as separate segments rather than a continuous tube).
  - **Irreversibility:** Diverticulosis involves permanent structural changes. While a high-fiber diet can prevent complications, it **cannot** reverse the anatomical pouches.
- **Epidemiology & Anatomic Distribution:**
  - Highly prevalent in Western populations; age-related (50% of people >50 years; 65% >85 years).
  - **Left colon / Sigmoid (90%):** Most common site overall, especially in Western populations, due to having the highest intraluminal pressure.
  - **Right colon (5–15%):** More common in Asian populations.
- **Gross Morphology:** Characterized by thickening and shortening of the bowel (due to muscular hypertrophy), luminal narrowing, and extensive pericolic fat deposition.

Diagnosis / Clinical Features

- **Disease Spectrum:**
  - **Diverticulosis:** Asymptomatic presence of diverticula. The mortality rate of uncomplicated, asymptomatic diverticulosis is essentially 0%.
  - **Symptomatic Uncomplicated Diverticular Disease (SUDD).**
  - **Uncomplicated Diverticulitis:** Localized inflammation (phlegmon) driven by a microperforation.
  - **Complicated Diverticulitis:** Abscess, free perforation, fistula, or obstruction.
- **Acute Diverticulitis:**

    - **Left-sided:** Classic triad of localized left lower quadrant (LLQ) tenderness, fever, and leukocytosis.
    - **Right-sided:** Presents with acute right lower quadrant (RLQ) pain, classically **mimicking acute appendicitis**.
- **Diverticular Bleeding:**
    - **Pathogenesis:** The vasa recta become stretched, displaced, and thinned over the dome of the diverticulum, making them highly vulnerable to mechanical trauma and erosion.
    - **Presentation:** Painless, bright red blood per rectum (macroscopic/overt bleeding). **Not** detected by Fecal Immunochemical Test (FIT), which is reserved for microscopic/occult blood (e.g., colon cancer screening).
    - Often originates from the **right colon**.
    - Diverticulosis and angiodysplasia are the most common causes of massive arterial lower GI bleeding in adults.
    - **Crucial distinction:** Uncomplicated colon cancer typically causes chronic, occult, low-grade mucosal bleeding leading to anemia, not massive, acute, life-threatening hemorrhage.

Investigations

- **CT Scan of Abdomen/Pelvis (with contrast):** The gold standard and preferred initial test for both acute diverticulitis and its complications (e.g., abscesses, fistulas).
    - *Findings:* Marked colonic wall thickening, mesenteric fat stranding, and air-filled outpouchings.
    - Highly sensitive for diagnosing colovesical fistulas (often reveals air in the bladder).
- **Colonoscopy:**
    - **Mandatory** after an episode of diverticulitis or bleeding to exclude underlying malignancy.
    - **Contraindicated in acute settings** (see Past-Paper High Yield).
- **Contrast Enema (Barium/Water-soluble):** Can be used to outline fistulas (e.g., colovesical extravasation) but is strictly avoided during acute inflammation.

Management

- **Uncomplicated Diverticulitis:** Conservative management with broad-spectrum IV antibiotics and bowel rest (clear liquid diet).
- **Abscesses (Hinchey I & II):**
    - Small abscess: IV antibiotics alone.
    - Large, walled-off pelvic/retroperitoneal abscess (Hinchey II): **Image-guided (CT/US) percutaneous drainage** + antibiotics.
- **Bleeding:** Typically stops spontaneously but has a high rebleeding rate. Managed with supportive care, endoscopic clipping/injection once stable, angiographic embolization, or colectomy for refractory massive bleeding.
- **Obstruction:** Occurs in ~10% of complicated cases secondary to luminal stenosis or extrinsic abscess compression. Treated with Hartmann's procedure, primary anastomosis, or bridging stents.

Relevant Guidelines

- **Hinchey Classification (Staging & Management)**

STAGE	CLINICAL FINDING	STANDARD MANAGEMENT
Hinchey I	Diverticulitis with localized pericolic abscess	Conservative (Antibiotics ± bowel rest)
Hinchey II	Diverticulitis with distant walled-off abscess (e.g., pelvic)	Image-guided percutaneous drainage + Abx
Hinchey III	Diverticulitis with purulent peritonitis	Emergency surgery (1-2% of cases)
Hinchey IV	Diverticulitis with fecal peritonitis	Emergency surgery (Mortality 20-30%)

- **Indications for Elective Prophylactic Surgery**
  - *Traditional Criteria:*
    - At least two documented attacks of diverticulitis.
    - A single attack in high-risk groups: **Young patients (<50 years)**, immunocompromised patients, or if malignancy cannot be excluded.
  - *Recent Updates:* These guidelines have been increasingly questioned, as modern evidence suggests the risk of complications does *not* necessarily increase with recurrent disease.

Operative / Procedural Notes

- **Hartmann's Procedure:** The standard emergency surgical intervention for Hinchey III/IV peritonitis or obstruction.
  1. Resection of the inflamed/diseased sigmoid colon.
  2. The distal rectal stump is oversewn and closed as a blind pouch in the pelvis.
  3. The proximal descending colon is brought out to the abdominal wall as a terminal end colostomy.
- **Surgical Recurrence:** Recurrence after definitive surgical resection is low (3%), but it is 4-fold higher if a colosigmoidostomy is performed instead of extending the resection down to healthy rectal tissue.

Complications / Prognosis

- **Abscess:** The most common complication of acute diverticulitis.
- **Fistulas:** Occurs in 5% of complicated cases.
  - **Colovesical:** Most common overall. More common in males. Presents with pneumaturia, urgency, and recurrent polymicrobial UTIs.
  - **Colovaginal:** Presents as passage of flatus/feces through the vagina. Most common in women with a prior hysterectomy.
  - **Coloenteric:** Secondary to abscess rupture into the small bowel; presents with chronic abdominal pain and diarrhea.

- **Prognosis & Recurrence:**
  - 50–70% of patients managed conservatively will have no further episodes.
  - **Age factor:** Young patients (under 50) have a higher rate of recurrence and a more aggressive disease course necessitating surgery compared to older patients.
  - Recovery rates decline sharply with subsequent attacks: >70% recover after the 1st attack, but only 6% remain complication-free after a 3rd attack.

Past-Paper High Yield

- **STRICT CONTRAINDICATION: Diagnostic colonoscopy, flexible sigmoidoscopy, and barium enemas are absolutely contraindicated during acute diverticulitis.** The inflamed, edematous colon wall is highly weakened, making the risk of iatrogenic perforation (from air insufflation and mechanical trauma) unacceptably high. Delay endoscopy for 6–8 weeks.
- **Left vs. Right Discrepancy:** Diverticulitis overwhelmingly affects the *left (sigmoid) colon*, but massive diverticular bleeding disproportionately originates from the *right colon*.
- **Massive Bleeding Etiologies:** In an adult with massive, acute, life-threatening lower GI bleeding, suspect diverticulosis or angiodysplasia. *Do not* suspect uncomplicated colon cancer (which causes chronic, occult bleeding).
- **Meckel's Diverticulum distinction:** As a true diverticulum, it causes painless lower GI bleeding (hematochezia/melena) in young patients due to ectopic gastric mucosa ulcerating the ileum. It does *not* cause hematemesis (which indicates an upper GI bleed).
- **Amebic Liver Abscess Principle (GI differential):** Amebic liver abscesses typically respond excellently to medical therapy (metronidazole). They should **not** be routinely drained unless they are huge, threatening to rupture, or failing medical therapy.

Memory Pearls

- **Hinchey Progression: I** = Pericolic, **II** = Pelvic, **III** = Purulent, **IV** = Putrid (Fecal).
- **Rule of 2s for True/False:** Colonic = False (2 layers herniate: mucosa + submucosa). True = All 3 layers (e.g., Meckel's).
- **Diverticular Bleeding vs. Infection:** They rarely happen at the same time. The patient is either bleeding (painless, overt) OR infected (pain, fever, leukocytosis).

Colorectal polyp

Core Concepts

- **Definition:** Mass lesions protruding from the intestinal mucosa toward the lumen or elevating the mucosa. They represent defects in normal mucosal cell proliferation, differentiation, or apoptosis.
- **Classic Adenoma-Carcinoma Sequence:**
  - Development of invasive carcinoma from a clean colon takes approximately **5 years**.
  - **APC (Adenomatous Polyposis Coli):** Mutation and loss of function of this tumor suppressor gene is universally the **earliest initiating event** in the sequence.
  - **KRAS:** Intermediate mutation driving adenoma growth and progression.

    - **TP53 & SMAD4:** Late-event mutations driving the final transformation into invasive carcinoma.
    - *(Note: BRAF mutations are characteristic of the alternative serrated pathway, not the classic adenoma-carcinoma sequence).*
- **Morphological Classification:**
    - **Sessile:** Broad-based, flat, raised mucosal lesion.
    - **Pedunculated:** Anchored to the bowel wall by a distinct mucosal stalk/pedicle.
- **Histological Classification:**
    - **Non-neoplastic Polyps:**
        - *Hyperplastic:* The **most common** non-neoplastic colorectal polyp in clinical practice. They are benign and have NO malignant potential.
        - *Inflammatory*
    - **Neoplastic Polyps (Premalignant):**
        - *Adenomas:* Comprise two-thirds of all colon polyps. Classified by villous (glandular growth) content:
            - **Tubular:** 0–25% villous glands (Most common: 80–86% of adenomas).
            - **Tubulovillous:** 25–75% villous glands.
            - **Villous:** 75–100% villous glands (Highest malignant potential).
    - **Hamartomatous Polyps:** Juvenile polyps, Peutz-Jeghers polyps.

Diagnosis / Clinical Features

- **Sporadic Polyps:** Highly common; found in ~34.3% of asymptomatic screening colonoscopies. Mostly located in the left colon and most (87–89%) are <1 cm. More common in men.
- **Familial Adenomatous Polyposis (FAP):**
    - **Genetics:** Autosomal dominant inheritance caused by a germline mutation in the **APC** tumor suppressor gene on chromosome **5q21**. (80% inherited, 20% de novo mutation).
    - **Clinical:** Hundreds of adenomatous colorectal polyps carpet the colon by the 2nd–3rd decade of life.
    - **Extracolonic Features:** Upper GI adenomas (95%), jaw osteomas (80%), Congenital Hypertrophy of the Retinal Pigment Epithelium or CHRPE (75%), epidermoid cysts (50%), fundic gland polyps (40%), unerupted teeth (17%), and desmoid tumors (15%).
- **Juvenile Polyposis Syndrome:**
    - Autosomal dominant condition characterized by 50–200 hamartomatous polyps in the rectum, colon, and stomach.
    - Presents around age 4 with blood around the stool. Carries a 30–50% risk of cancer.
- **Solitary Juvenile Polyp:**
    - Presents in infants/children as a bleeding, painful, prolapsing "cherry tumor" (bright red, glistening, pedunculated sphere).
    - Unique histology: Large mucus-filled spaces covered by cuboidal epithelium. Lacks smooth muscle (poor anchorage to bowel wall; may spontaneously amputate). Has **no malignant**

potential.

- **Peutz-Jeghers Syndrome:**
  - Autosomal dominant condition presenting with **mucocutaneous pigmentation** (lips, perioral area) and **gastrointestinal hamartomatous polyps**. High risk for bowel obstructions and cancers.
- **Risk Factors (Sporadic Polyps):**
  - *Increased risk:* Age, low fiber/high fat diet, low folate, alcohol, smoking, physical inactivity, family history, acromegaly.
  - *Decreased risk (Chemopreventive):* Aspirin, NSAIDs.

Investigations

- **Diagnostic Modalities for Screening:**
  - *FOBT:* Cheap, noninvasive, but low specificity and requires 3 successive stools.
  - *FIT:* More sensitive and specific than FOBT; requires only 1 stool sample.
  - *Multitarget Stool DNA:* Highly sensitive but less specific than FIT.
  - *Sigmoidoscopy:* Highly sensitive for left colon lesions but misses proximal lesions.
  - *Colonoscopy:* Gold standard. Examines the entire colon, highly sensitive/specific, and therapeutic (polypectomy). Requires bowel prep and sedation; carries risk of perforation.
- **FAP Diagnostics:**
  - Diagnosis requires the presence of ≥100 colorectal adenomas OR a confirmed APC gene mutation.
  - If genetic testing is unavailable, the presence of **CHRPE** on ophthalmic exam can be used to screen affected families.

Management

- **General Management:**
  - Endoscopic polypectomy is standard and significantly reduces the long-term incidence of colorectal cancer.
- **FAP Pharmacotherapy:**
  - NSAIDs (Sulindac and Celecoxib) can cause regression of polyps but do not replace prophylactic surgery; they require frequent endoscopic surveillance.

Relevant Guidelines

BSG Guidelines (2002) for Surveillance Following Adenoma Removal

- **Low Risk (Group A):** 1–2 adenomas AND both are small (<1 cm).
  - *Recommendation:* No surveillance required OR follow-up colonoscopy at 5 years.
- **Intermediate Risk (Group B):** 3–4 small adenomas OR at least one adenoma ≥1 cm.
  - *Recommendation:* Follow-up colonoscopy at 3 years.

- **High Risk (Group C):** ≥5 small adenomas OR ≥3 adenomas with at least one ≥1 cm.
  - *Recommendation:* Follow-up colonoscopy at 1 year.

FAP Clinical Surveillance

- **Lower GI:** Annual flexible sigmoidoscopy starting at age 13–15. If no polyps are found, transition to colonoscopy at age 20. If no adenomas are present by age 30, FAP is highly unlikely.
- **Upper GI:** Endoscopic surveillance looking for duodenal polyps every 2 years starting after age 30.

Operative / Procedural Notes

- **FAP Prophylactic Surgery:** Carcinoma inevitably develops 10–20 years after polyp onset, necessitating prophylactic resection.
  - **Total Proctocolectomy with Ileal Pouch-Anal Anastomosis (IPAA / RPC):** The universal gold standard operation of choice.
  - **Total Proctocolectomy and End Ileostomy:** An alternative option.
  - **Colectomy with Ileorectal Anastomosis (IRA):** Generally not preferred as it leaves at-risk rectal mucosa intact.

Complications / Prognosis

- **Determinants of Malignant Potential (High-Yield):**
  1. **Histological Type:** Villous > Tubulovillous > Tubular.
  2. **Size:** >1 cm significantly increases risk (e.g., >1 cm tubular = 35% risk; 2 cm villous = 50% risk).
  3. **Degree of Dysplasia:** High-grade dysplasia equates to carcinoma limited to the epithelium.
  4. **Site / Anatomical Location:** Proximal (right-sided) polyps carry specific risk profiles.
  5. **Multiplicity:** A higher number of polyps increases the risk.
  - *Note:* **Pedunculation (morphological shape) is NOT a primary prognostic factor.**
- **FAP Prognosis:** Lifetime risk of malignant transformation in the colon is 100% without surgery. Risk of small bowel (duodenal) cancer is significantly elevated but is not 100%.
- **Progression timeline:** The estimated yearly rate of conversion from a 1 cm adenoma to carcinoma is 0.25%, accelerating over time (2.5% at 5 years → 8% at 10 years → 24% at 20 years).

Past-Paper High Yield

- **Primary Prognostic Factors:** Always remember that histology (adenomatous/villous), size (>1 cm), number, and site dictate malignant potential. **Pedunculation does NOT** heavily influence prognosis.
- **Hyperplastic Polyps:** These are the most common non-neoplastic polyps found in practice. They have **no malignant potential**. Do not confuse them with neoplastic adenomatous or serrated polyps.
- **Genetics of FAP:** Must know that FAP is caused by a **germline** mutation (not somatic) in the **APC tumor suppressor gene** located on chromosome **5** (5q21).

- **FAP vs. HNPCC Genetics:** FAP = APC gene. HNPCC (Lynch Syndrome) = MSH2 (or MLH1/MSH6/PMS2) mismatch repair genes.
- **Adenoma-Carcinoma Sequence Initiator:** The loss of the **APC** gene is the absolute earliest event. KRAS comes later. TP53/SMAD4 are final-stage events.
- **General GI Pharmacology (Laxatives):** *Lactulose* is a synthetic, non-digestible sugar that acts as an **osmotic** laxative. Contrast this with *Psyllium* (bulk-forming) and *Senna / Bisacodyl / Sodium picosulfate* (stimulant laxatives).

Memory Pearls

- **A**denomatous **P**olyposis **C**oli (APC) mutation occurs on Chromosome **5** (Think: "APC" has 3 letters, but "Polyp" has **5** letters).
- **Villous = Villainous:** Villous adenomas are the most sinister (highest malignant potential).
- **Cherry Tumor:** Pathognomonic description for a solitary, benign juvenile polyp in a child presenting with rectal bleeding and prolapse.

Colorectal cancer

Core Concepts

- **Epidemiology & Distribution:**
  - Primarily a disease of the Western world; >90% of cases diagnosed in patients >50 years old.
  - Tumors are distributed predominantly in the left colon and rectum (50%), followed by the right colon (25%).
  - Synchronous lesions (multiple primary tumors presenting simultaneously) occur in 4–5% of cases.
- **Aetiology & Genetic Pathways:**
  - **Sporadic (majority):** Typically follows the **Chromosomal Instability (CIN)** pathway: *Normal cell → APC mutation → Low-level polyp → KRAS mutation → Mid-level polyp → DCC loss → High-level polyp → p53 loss → Carcinoma.*
  - **Inherited (5-10%):**
    - *Lynch Syndrome (HNPCC):* Driven by Microsatellite Instability (MSI) via germline mutations in mismatch repair (MMR) genes (*MLH1, MSH2, MSH6, PMS2*).
    - *Familial Adenomatous Polyposis (FAP):* Driven by germline mutations in the *APC* gene.
  - **Serrated Pathway:** Associated with CpG Island Methylation Pathway (CIMP), *KRAS*, and *BRAF* mutations.
- **Risk Factors:**
  - Age, family history, African American race, obesity, smoking, alcohol, and diets high in red/processed meats, animal fat, and sugar.
  - Predisposing medical conditions: Longstanding IBD (Ulcerative Colitis and Crohn's disease), history of cholecystectomy, post-gastrectomy/vagotomy, and ureterosigmoidostomy (direct urinary diversion to the sigmoid colon).

- **Protective Factors:** Physical exercise, dietary fiber, calcium, garlic, non-starchy vegetables, and **Aspirin/NSAIDs** (proven to decrease the incidence of colonic adenomas and colorectal cancer).

- **Polyp Biology:**
  - *Hyperplastic polyps:* Most common non-neoplastic polyps; small distal ones rarely become malignant.
  - *Hamartomatous polyps:* Composed of disorganized normal native tissue elements.
  - *Adenomas:* Villous histology, large size, and high-grade dysplasia dictate a high risk for malignant transformation.

Diagnosis / Clinical Features

- **Presentation:** Ranges from asymptomatic (detected on screening) to elective symptomatic presentations or surgical emergencies (obstruction, perforation).
- **High-Risk Symptoms:**
  - Rectal bleeding + change in bowel habit (to looser stools/increased frequency) >6 weeks (all ages).
  - Change in bowel habit without rectal bleeding >6 weeks (age >60).
  - Persistent rectal bleeding without anal symptoms (age >60).
  - Palpable right-sided abdominal mass or rectal mass (all ages).
  - Unexplained iron deficiency anemia (all ages).
- **Low-Risk Symptoms:** Transient bowel habit changes, rectal bleeding with obvious external anal causes (e.g., fissure), or abdominal pain as an isolated symptom without other signs.

Investigations

- **Endoscopy:** Colonoscopy is the gold standard (diagnostic and therapeutic; mandatory to rule out synchronous tumors).
- **Imaging:**
  - *CT Colonography:* Less invasive diagnostic alternative; carries risks of radiation and contrast nephrotoxicity.
  - *Barium Enema:* Classic "apple-core" lesion (annular constricting carcinoma with overhanging mucosal edges/shouldering). Mostly historical but high-yield for imaging recognition.
  - *Pelvic MRI & Endorectal Ultrasound:* Crucial for local staging of rectal cancer.
  - *PET-CT:* Limited routine role; primarily used when surgical resection of metastases is being considered.
- **Tumor Markers (Systemic Review for Exams):**
  - **CEA:** Used for CRC surveillance and detecting recurrence/metastases (not screening).
  - **CA 19-9:** Most sensitive and commonly used for pancreatic/biliary adenocarcinoma.
  - **AFP:** Classic biomarker for screening and monitoring Hepatocellular Carcinoma (HCC).
  - **CA 125:** Ovarian cancer.
  - **PSA:** Prostate cancer.

- **FOB/FIT tests:** Exclusively screening tools for CRC.

Relevant Guidelines

- **Indications for Postoperative Chemotherapy (High-Risk Features):**
  - Lymph node positive (N+ disease).
  - Lymphovascular invasion (LVI).
  - Obstruction or Perforation at presentation.
  - Peritoneal involvement.
  - Poorly differentiated histology.
- **Rectal Adenocarcinoma Treatment Algorithm:**
  - **Stage I (T1-2, N0, M0):** Radical resection. (Transanal excision considered if high surgical risk or patient refuses radical resection).
  - **Stage II (T3-4, N0, M0) & Stage III (Node positive):** Neoadjuvant chemoradiation → Restage → Radical resection (if no metastases).
  - **Stage IV (Metastatic):**
    - *Asymptomatic primary:* Chemotherapy → Restage → Consider resection if a single metastatic site is resectable; continue chemo if multiple/unresectable sites.
    - *Symptomatic primary:* Chemoradiation vs. palliative procedures (stent, laser, ablation, stoma, or palliative resection).

Management

- **Antibiotic Prophylaxis:** Colorectal surgery carries a high risk of surgical site infections. Regimens **must** cover both aerobic Gram-negative bacilli (e.g., *E. coli*) and anaerobes (e.g., *B. fragilis*).
  - Standard: 3rd-generation cephalosporin + metronidazole, or a single broad-spectrum agent like cefoxitin. Monotherapy with just a cephalosporin or just metronidazole is inadequate.
- **Surgical Oncology Principles:**
  - Achieve local control with microscopically free margins.
  - En bloc excision of draining lymph nodes along named vessels.
  - Ensure viable bowel is left behind with a safe anastomosis.
  - Standardization techniques: Complete mesocolic excision (CME) for colon and total mesorectal excision (TME) for rectal cancer.
- **Appendiceal Carcinoid Tumors (Surgical Correlate):**
  - *Simple Appendectomy:* Sufficient for tumors < 1–2 cm located at the appendiceal tip without concerning features.
  - *Right Hemicolectomy:* Mandatory for tumors > 2 cm, or any tumor involving the base of the appendix, to ensure complete oncologic clearance and node sampling.

Operative / Procedural Notes

- **Anatomical Definitions of the Rectum:** The exact starting point varies clinically but is defined by: 15 cm from the anal verge, 11-12 cm from the anal verge, fusion of the taenia coli, or the level of the 3rd sacral vertebra.
- **Vascular Anatomy & Ligation:**
  - *Midgut (Right Colon):* Supplied by Superior Mesenteric Artery (SMA) branches (ileocolic, right colic, middle colic).
  - *Hindgut (Left Colon/Rectum):* Supplied by Inferior Mesenteric Artery (IMA) branches (left colic, sigmoid branches, superior rectal).
  - Major named vessels (SMA/IMA root branches) require secure mechanical ligation (clips/staples), whereas smaller arcades and mesorectal vessels may be sealed with energy devices (e.g., LigaSure).

Complications / Prognosis

- **Spread Patterns:**
  - *Lymphatic:* Spreads from paracolic nodes along main vessels to para-aortic nodes. **30% can skip a tier of nodes**. 15% of tumors confined strictly to the bowel wall already have LN metastases.
  - *Hematogenous:* The **liver** is the most common site of metastasis (up to 37% have occult liver mets at operation), followed by the lung.
- **Prognostic Factors:**
  - **Lymph Node Status:** Assuming no distant metastases (Stage IV), the presence and number of involved regional lymph nodes (N stage) is the **single most important prognostic factor** for long-term survival.
  - **Poor Prognosticators:** Signet ring histology, vascular invasion, obstructing presentation, and bowel perforation all indicate aggressive biology and poor outcomes.
  - *Note:* Right-sided colon cancer in itself is **not** universally considered a poor prognostic factor relative to left-sided disease.
- **Surveillance:** 80% of recurrences occur within the first 3 years; intense follow-up (laboratory, radiological, and endoscopic) is required during this period.
- **5-Year Survival Rates:** Stage 1 (~90-96%), Stage 2 (~65-77%), Stage 3 (~25-77% depending on node burden), Stage 4 (~15-19%).

Past-Paper High Yield

- **Prognosis Trap:** Right-sided colon cancer is NOT inherently a poor prognostic factor compared to left-sided, whereas perforation, obstruction, signet ring histology, and vascular invasion definitively are.
- **Most Important Prognostic Factor:** Nodal involvement (N-stage) is the most critical determinant of survival in non-metastatic CRC.
- **Tumor Marker Panel Recognition:** CEA = Colon; CA 19-9 = Pancreas; AFP = Liver (HCC); CA 125 = Ovary; PSA = Prostate. Do not mix these up.

- **Fistula Diagnosis:** The most accurate imaging modality for diagnosing a colovesical fistula is a **CT scan of the abdomen and pelvis with oral or rectal contrast**, typically demonstrating air within the bladder.
- **Antibiotic Prophylaxis Trap:** Monotherapy with Metronidazole alone or Cephalosporin alone is always the wrong answer for elective colorectal cases. You must select the combination covering both Gram-negatives and anaerobes.
- **Chemoprevention Fact:** Aspirin and NSAIDs *decrease* the risk of colonic adenomas and CRC—they do not increase it.
- **Appendix Carcinoids:** Carcinoid is the most common primary tumor of the appendix. Do not blindly select right hemicolectomy for all appendiceal carcinoids; tumors <1.5 cm at the tip are cured with simple appendectomy.

Memory Pearls

- **CIN vs. MSI:** *CIN* = sporadic, classic adenoma-carcinoma sequence (APC, KRAS, p53). *MSI* = Lynch syndrome, defective mismatch repair (MMR).
- **High-Risk Chemo Triggers:** "The Bad LOPP" = **L**ymph nodes/**L**VI, **O**bstruction, **P**erforation, **P**oorly differentiated/**P**eritoneal spread.

Hemorrhoids & fissures

Core Concepts

- **Anal Canal Anatomy:**
  - The **dentate (pectinate) line** is located 1.5–3 cm from the anal verge and serves as a critical anatomical landmark for epithelium, venous drainage, and innervation.
  - **Anoderm:** The sensitive skin below the dentate line; drains to the **superficial inguinal lymph nodes**.
- **Hemorrhoidal Anatomy:**
  - Hemorrhoids are **not** varicose veins. Everyone has physiological anal cushions composed of erectile blood vessels, smooth muscle (**Treitz's muscle** / musculus submucosae ani), and elastic connective tissue.
  - Their primary function is to act as a conformable plug maintaining continence (~15–20% of resting pressure) and allowing sensory discrimination (anal sampling).
  - **Etiology:** Disruption and downward sliding (shearing) of these cushions due to aging, constipation, straining, diarrhea, and pregnancy.
  - *High-Yield Fact:* Hemorrhoids are **no more common in patients with portal hypertension** than in the general population.
  - **Classic anatomical sites (in lithotomy position):** Left lateral (3 o'clock), Right posterolateral (7 o'clock), and Right anterolateral (11 o'clock).
- **Femoral Hernia Anatomy (Exam Distractor / Board Pearl):** A defining anatomical feature of a femoral hernia is that it presents as a bulge **BELOW** the inguinal ligament, passing through the femoral canal (unlike inguinal hernias, which are above).

- **Laxative Pharmacology: Ispaghula husk (psyllium)** is a natural bulk-forming laxative that absorbs water to form a viscous gel, promoting peristalsis without causing massive secretory/osmotic diarrhea (unlike PEG or bisacodyl).

Diagnosis / Clinical Features

- **Internal Hemorrhoids:**
  - Located **above the dentate line**.
  - **Painless** (unless prolapsed/strangulated/thrombosed) due to visceral innervation.
  - Present with **bright red bleeding** at the end of defecation (dripping or squirting into the toilet bowl).
  - May cause prolapse, mucous leakage, pruritus, or perianal excoriation.
- **External Hemorrhoids:**
  - Located **below the dentate line**.
  - Highly sensitive due to somatic innervation (pudendal nerve branches).
  - Usually asymptomatic unless **thrombosed**, which presents as an abrupt, tense, bluish-purple, excruciatingly painful perianal mass. Pain peaks at 48 hours and typically subsides within 5 days.
- **Anal Fissure:**
  - Classic presentation: **Severe, tearing pain** during and after defecation, with occasional bright red blood streaks on the stool or paper.
  - Often associated with a hypertonic internal anal sphincter (high resting pressure).
  - **Anatomical distribution:**
    - **Posterior midline:** Most common site overall (both sexes).
    - **Anterior midline:** More common in females than males.
    - **Lateral (off-midline):** Atypical. Strongly suggests a secondary underlying pathology (Crohn's disease, ulcerative colitis, tuberculosis, syphilis, HIV/AIDS, leukemia).

Investigations

- **Physical Examination:**
  - **Inspection / Straining:** Best for assessing prolapse, fissures, and skin tags.
  - **Digital Rectal Exam (DRE):** Uncomplicated internal hemorrhoids are **SOFT and IMPALPABLE**.
  - **Anoscopy:** Essential for directly visualizing and grading internal hemorrhoids.
- Flexible sigmoidoscopy or colonoscopy may be indicated to rule out proximal sources of bleeding (e.g., CRC).

Relevant Guidelines

- **Classification of Internal Hemorrhoids:**
  - **Grade 1:** Bulge into lumen; painless bleeding; do not prolapse.
  - **Grade 2:** Prolapse during defecation but **reduce spontaneously**.

- **Grade 3:** Prolapse during defecation or spontaneously; require **manual reduction**.
- **Grade 4:** Permanently prolapsed and **irreducible**; may become strangulated.

- **Criteria for Chronic Anal Fissure:**
  - History of symptoms lasting **> 1 month**.
  - Presence of the classic triad:
    1. **Sentinel skin tag** (distal).
    2. Deep ulcer exposing the **internal sphincter muscle fibers** in the base.
    3. **Hypertrophied anal papilla** (proximal).
  - Additional features: Fibrosis or submucous fistula.

Management

- **Internal Hemorrhoids:**
  - **Initial/Medical (All Grades, especially 1 & 2):** Diet optimization, bulk-forming agents (e.g., psyllium), warm Sitz baths, and topical protectants/analgesics.
  - **Office Procedures (Grades 1, 2, and some 3):**
    - **Rubber Band Ligation (RBL):** Highly effective, superior to sclerotherapy. Performed in-office without general anesthesia.
    - Sclerotherapy, Infrared photocoagulation (burns superior pedicle).
  - **Operative (Grades 3 & 4 or failed minor procedures):** Surgical excision (open/closed hemorrhoidectomy), Stapled hemorrhoidectomy (PPH), or Doppler-guided HAL.
- **Thrombosed External Hemorrhoids:**
  - **Early (< 48 hours):** May be managed with surgical incision and evacuation/excision of the clot.
  - **Late (> 48 hours):** Conservative management (local anesthetics, warm Sitz baths) as pain naturally minimalizes after the 4th day and the thrombus shrinks.
- **Anal Fissure:**
  - **Acute Fissure:** Conservative (Sitz baths, bulk agents). Pharmacologic sphincterotomy (Topical GTN, Calcium Channel Blockers, or Botulinum Toxin injection).
  - **Chronic Fissure: Partial Lateral Internal Sphincterotomy (LIS)** is the standard surgical gold standard. Alternatively, V-Y Anoplasty (advancement flap).

Operative / Procedural Notes

- **Rubber Band Ligation (RBL):** Forceps draw the mucosal apex of the hemorrhoid into the ligator drum. The band is deployed tightly at the vascular pedicle **strictly above the dentate line** to cause ischemic necrosis without somatic pain.
- **Partial Lateral Internal Sphincterotomy (LIS):** Under direct vision, the distal portion of the internal anal sphincter is isolated and divided to relieve chronic hypertonicity, allowing the fissure bed to heal.
- **Stapled Hemorrhoidectomy (PPH):** A circular purse-string suture is placed in the mucosa well above the dentate line. A circular stapler pulls redundant prolapsed tissue into its casing, excises a

mucosal band, and staples the defect, repositioning the cushions proximally.

- **V-Y Anoplasty:** Used for chronic fissures. The chronic fissure/fibrotic tract is excised. A V-shaped distal skin flap is mobilized and advanced upward to cover the mucosal defect, sutured in a "Y" shape for tension-free coverage.

Complications / Prognosis

- Operative hemorrhoidectomy carries risks of pain, bleeding, and stricture, but can impair continence to infused saline because the physiological "plug" (anal cushions) is removed.
- Fissure complications include progression to abscess and fistula-in-ano.

Past-Paper High Yield

- **Anatomy & Innervation distinction is heavily tested:**
  - **Internal Hemorrhoids:** Arise from superior hemorrhoidal plexus → ABOVE dentate line → covered by transitional/columnar mucosa → VISCERAL innervation (**painless**) → drain to portal system.
  - **External Hemorrhoids:** Arise from inferior hemorrhoidal plexus → BELOW dentate line → covered by anoderm (squamous) → SOMATIC innervation (**acutely painful**) → drain directly to systemic caval system.
- **Fissure location is a critical diagnostic clue:** A lateral (off-midline) fissure is an atypical red flag that necessitates workup for underlying disease (e.g., Crohn's, TB, HIV, leukemia, syphilis). Classic benign fissures are posterior midline.
- **Fissure vs. Hemorrhoid presentation:** Severe, tearing pain on defecation with a spot of bright red blood = **Fissure**. Painless, dripping/squirting bright red blood = **Internal Hemorrhoid**.
- **Rubber band ligation (RBL) concepts:** RBL is superior to sclerotherapy for Grade I-III internal hemorrhoids and is performed in the office setting **without** deep general anesthesia.

Memory Pearls

- **3, 7, 11:** The clock-face positions (lithotomy) of normal anal cushions.
- **Internal = Insensitive:** Above the dentate line, internal hemorrhoids lack somatic pain fibers.
- **Lateral = Look further:** Lateral fissures mean systemic disease.
- **Triad of Chronic Fissure:** Tag (distal), Tear (muscle exposed), and Tip (hypertrophied papilla proximal).

Perianal suppuration

Core Concepts

- **Anatomy of Anal Glands**:
  - Average of 6 glands (range 3–10) per normal anal canal.
  - Lined by stratified columnar epithelium with mucus-secreting goblet cells.
  - Originate at the dentate line (crypts of Morgagni) and pass into the submucosa.

- Two-thirds continue into the internal sphincter; one-half penetrate into the intersphincteric plane.

- **Cryptoglandular Theory**: Most perianal suppuration originates from obstruction of these anal gland ducts (by feces, foreign body, or trauma) leading to stasis and infection.
- **Avenues of Extension**: Infection typically starts in the intersphincteric space and can track:
  - *Downward* to the perianal space.
  - *Horizontally* through the external sphincter into the ischioanal space.
  - *Upward* into the supralevator space or high ischioanal space.
  - *Circumferentially* via the deep postanal space to the contralateral side (horseshoe abscess).
- **Chronicity & Fistula Persistence**:
  - Chronicity is driven by the **persistence of anal gland epithelium** or non-specific epithelialization of the tract.
  - *Pathophysiological principle*: Epithelialization of a fistula tract prevents spontaneous closure. Destruction or excision of this epithelium is required for healing.
- **Etiologies**:
  - *Most common*: Cryptoglandular.
  - *Specific/Secondary*: Crohn's disease, ulcerative colitis, Tuberculosis (TB), Actinomycosis, Lymphogranuloma venereum (LGV), foreign body, trauma (impalement, enemas, episiotomy), radiation, malignancy (carcinoma, lymphoma, leukemia), chronic anal fissure.
- **Bacteriology**: *E. coli* (22%), *Bacteroides fragilis* (20%), *Enterococcus spp.* (16%).

Diagnosis / Clinical Features

Acute Phase (Abscess)

- **Symptoms**: Acute severe pain (aggravated by sitting, moving, coughing, defecation), swelling, purulent anal discharge, bleeding, malaise, pyrexia.
- **Signs**: Tender induration, fluctuant mass, pus exuding from a crypt.
- **Supralevator Abscess**: May lack external perianal signs; presents as a tender pelvic mass on digital rectal/vaginal exam or with signs of peritoneal irritation on abdominal exam.

Chronic Phase (Fistula-in-Ano)

- **History**: Previous perianal abscess that burst spontaneously or required surgical drainage, leaving a small discharging sinus.
- **Signs**:
  - External opening visible as a red elevation of granulation tissue exuding purulent/serosanguinous discharge on compression.
  - **Palpable cord**: A superficial tract felt just beneath the skin leading to the anal canal.
  - **Funnel / Herniation sign**: Retraction of the involved crypt of origin due to fibrotic pull on the internal sphincter.
- **Epidemiology**: Male predominance (2:1 to 7:1); peak incidence in the 3rd to 5th decades.

Investigations

- **Examination Under Anesthesia (EUA)**: Highly indicated for accurate assessment and simultaneous treatment.
- **Fistula Tracking**:
  - Do **NOT** force a metal probe through a blind tract, as it creates false passages (iatrogenic extrasphincteric extensions).
  - Diagnostic fluids (methylene blue, hydrogen peroxide, milk) can be injected into the external opening to identify the internal opening via a bivalve speculum.
- **Imaging**:
  - **MRI (Pelvic/Endoanal)**: Gold standard for mapping complex, recurrent, or high fistulae non-invasively.
  - **Endoanal Ultrasonography (EAUS)**: Highly effective for visualizing intersphincteric tracts and sphincter defects.
  - **Fistulography**: X-ray contrast study to outline complex branching tracts and pelvic extensions.
  - **Anoscopy & Sigmoidoscopy**: Essential to rule out underlying proctitis or proximal disease (e.g., Crohn's, malignancy).

Management

Abscess (Acute Phase)

- **Primary Treatment**: Incision and drainage (I&D) with deroofing (excising skin edges to prevent premature closure).
- **Antibiotics**: Generally unneeded for simple abscesses. **Indications for Abx**:
  - Systemic or significant local sepsis (e.g., cellulitis).
  - Immunocompromised host.
  - Prosthetic heart valves or other specific hardware.

Fistula (Chronic Phase)

- **Surgical Principles**:
  1. Identify the primary internal opening.
  2. Establish the relationship of the tract to the puborectalis/sphincter complex.
  3. Divide the *least* amount of muscle necessary to cure the fistula (preserving continence).
  4. Identify and treat all side tracts.
  5. Rule out underlying specific diseases (biopsy suspicious tracts).

Relevant Guidelines

Parks Classification of Fistula-in-Ano

- **Intersphincteric (70% - Most Common)**: Tract runs entirely within the intersphincteric plane and opens on the perianal skin. Does not cross the external sphincter.

- **Transsphincteric (23%)**: Tract crosses through both the internal and external sphincters into the ischioanal fossa.
- **Suprasphincteric (5%)**: Tract passes superiorly in the intersphincteric space, loops *over* the puborectalis/levator ani, and descends through the ischioanal fossa.
- **Extrasphincteric (2%)**: Tract extends from the perineal skin, up through the ischioanal fossa and levator ani, directly into the rectum (completely lateral to the sphincter complex).
- **Superficial**: Confined to the subcutaneous space.

Goodsall's Rule (Predicting Tract Pathways)

- Draw a transverse line bisecting the anus.
- **Posterior openings**: Track in a *curved* path to a primary internal opening in the **posterior midline**.
- **Anterior openings (< 3 cm from anal verge)**: Track in a *straight* radial path to the nearest crypt directly opposite the opening.
- **Anterior openings (> 3 cm from anal verge)**: Exception to the rule; these track in a *curved* path to the **posterior midline**.

Supralevator Abscess Drainage Pathway Guidelines

- *Incorrect drainage creates complex, high fistulas.*
- **If Intersphincteric origin**: MUST be drained **internally** into the rectum. (External drainage through ischioanal fossa is contraindicated as it creates a suprasphincteric fistula).
- **If Ischioanal origin**: MUST be drained **externally** through the ischioanal fossa. (Internal rectal drainage is contraindicated as it creates an extrasphincteric fistula).

Operative / Procedural Notes

- **Fistulotomy (Lay-open technique)**:
  - Indicated for simple, low fistulae. The tract over a probe is divided (electrocautery).
  - **Marsupialization**: Edges of the laid-open tract are sutured to adjacent skin. Reduces wound profile and prevents premature skin closure over the wound bed.
- **Fistulectomy**: The entire fistulous tract is cored out/excised intact.
- **Complex Tract Interventions**:
  - **Setons**: Non-absorbable loops passed through the tract.
    - *Draining*: Tied loosely to keep tract open, ensuring continuous drainage of pus and resolution of inflammation prior to definitive surgery.
    - *Cutting*: Gradually tightened to slowly fibrose and cut through the sphincter, preventing sudden retraction and incontinence.
    - *Medicated*: Used to induce fibrosis of the tract.
  - **LIFT Procedure (Ligation of Intersphincteric Fistula Tract)**: Approach via the intersphincteric groove; the tract is isolated, suture-ligated near the internal and external openings, and divided, preserving all sphincter muscle.
  - **Advancement Flaps (Endorectal or Dermal Island)**: A mucosal/submucosal flap is mobilized and advanced over the closed internal opening to isolate the high tract from the rectal lumen.

- **Horseshoe Abscess (Modified Hanley Procedure)**: Posterior midline incision divides the internal sphincter at the primary crypt, combined with bilateral lateral counter-drainage incisions over the ischioanal spaces, sparing the external sphincter.
- **Tissue Sparing Methods**: Fibrin glue, Anal Plug (bioprosthetic scaffold), Laser closure (FiLaC - thermal ablation), Video-Assisted Anal Fistula Treatment (VAAFT).

Past-Paper High Yield

- **Pathophysiology of Healing**: Epithelialization of a fistula tract *prevents* spontaneous closure and promotes persistence. It requires surgical destruction/excision to heal.
- **Most Common Variant**: Intersphincteric fistulae are the most common anatomical type (~70%).
- **Surgical Contraindications**: Standard simple fistulotomy is **contraindicated** in high trans-sphincteric (or suprasphincteric) fistulae due to the unacceptably high risk of fecal incontinence resulting from extensive external sphincter division.
- **Biopsy Indications**: The fistulous tract should ideally be biopsied at operation if it is suspicious, atypical, or non-healing to rule out Crohn's disease or underlying malignancy (e.g., anal carcinoma).
- **General Fistula Principles Trap (Pancreatic Pseudocysts)**: Exam questions testing generalized fistula principles often use pancreatic pseudocysts as a contrast. Remember that external (enterocutaneous) drainage of a pancreatic pseudocyst is generally *avoided* due to the high risk of creating a persistent pancreaticocutaneous fistula; internal drainage is preferred. Unlike perianal fistulae, pseudocysts lack a true epithelial lining and can resolve spontaneously.

Memory Pearls

- **Parks Incidence**: "ITSE" -> **I**ntersphincteric (70%), **T**ranssphincteric (23%), **S**uprasphincteric (5%), **E**xtrasphincteric (2%).
- **Goodsall's Dog**: "Dog's nose points straight ahead (anterior = straight); tail wags all around to the back (posterior = curved to midline)."
- **Distance = Complexity**: The further the external opening is from the anal margin, the greater the likelihood of a complex, upward, or transsphincteric extension.
- **Supralevator Drainage Rule**: Drain it back to where it came from (Intersphincteric -> Rectum; Ischioanal -> Skin).

Surgical aspect of spleen

Core Concepts

- **Anatomy & Location:** The spleen is located posterolaterally in the left upper quadrant (LUQ) spanning the 9th to 11th ribs. Its size correlates with a person's height, weight, and sex (guided by the "rule of odd numbers").
- **Peritoneal Status:** The spleen is completely surrounded by peritoneum (except at its hilum), making it an entirely **intraperitoneal** organ.
  - *Contrast:* The kidneys and adrenal glands are primary retroperitoneal, while the pancreas is secondary retroperitoneal.

- **Ligamentous Attachments:** Gastrosplenic, splenorenal, splenophrenic, and spleno-colic ligaments.
- **Blood Supply:** The splenic artery arises from the celiac trunk and divides into 3 to 7 intraparenchymal segments. This terminal vascular branching makes selective partial splenectomy anatomically feasible.
- **Physiology:** Crucial for the antibody response against infection and the opsonization of encapsulated bacteria.
- **Hematological Indications for Elective Splenectomy:**
  - **Immune Thrombocytopenic Purpura (ITP):** First-line therapy is medical (corticosteroids/IVIG). Splenectomy is highly effective for refractory cases.
  - **Hereditary Spherocytosis:** Splenectomy is the definitive treatment; it is highly curative for anemia and prevents gallstones as the spleen is the primary site of abnormal RBC destruction.
  - **Thalassemia:** Splenectomy is **not** first-line (standard care is blood transfusions and iron chelation). It is reserved only for massive splenomegaly or severe hypersplenism.

Diagnosis / Clinical Features

- **Splenic Trauma:** The spleen and liver are the most commonly injured intra-abdominal organs in blunt trauma (e.g., motor vehicle collisions).
- **Kehr Sign:** Left shoulder pain that worsens with inspiration. It is a classic sign of splenic rupture caused by irritation of the phrenic nerve from blood adjacent to the left hemidiaphragm.
- **ITP Physical Exam:** In ITP, the spleen is generally **not palpably enlarged**. A massively palpable spleen in a thrombocytopenic patient should prompt investigation for other conditions (e.g., portal hypertension, leukemia, lymphoma).
- **OPSI Presentation:** Often presents initially as a non-specific flu-like illness that rapidly progresses to fulminant sepsis.

Investigations

- **Hemodynamically Stable Trauma Patient:** A **CT scan of the abdomen with IV contrast** is the standard of care to diagnose and grade the injury.
  - Identifies hemoperitoneum, parenchymal lacerations (hypodensities), and active bleeding.
  - *Active contrast extravasation (blush)* implies ongoing hemorrhage requiring urgent intervention.
- **Hemodynamically Unstable Trauma Patient: FAST exam** is the initial modality. A positive FAST (hypoechoic rim around the spleen) in an unstable patient mandates immediate laparotomy.
- **Peripheral Blood Smear:** Post-splenectomy, the presence of **Howell-Jolly bodies** is characteristically seen and confirms the absence of splenic function.

Management

- **Splenic Abscess:** A septated or multiloculated splenic abscess is best treated with **splenectomy and antibiotics**. Percutaneous drainage and antibiotics are inadequate and likely to fail due to the septations. Observation carries a high risk of fatal rupture.
- **Non-Operative Management (NOM) for Trauma:**

- Indicated for hemodynamically stable patients lacking other abdominal injuries (e.g., peritonitis).
- Consists of intensive monitoring, bed rest, and observation. Abandoned if ongoing hemorrhage occurs.
- A 5-day observation period identifies >95% of patients who will fail NOM.

- **Splenic Artery Embolization (SAE):**
  - Used as an adjunct to NOM.
  - *Distal (selective):* Limits parenchymal infarction; used for specific contrast blushes.
  - *Proximal:* Lowers distal systolic pressure by ~40 mmHg to enhance healing.
- **Prophylactic Antibiotics:** Lifelong penicillin is **not** universally mandatory for all patients. It is indicated for children <5 years old, immunocompromised patients, or those with a history of severe encapsulated infections.

Relevant Guidelines

American Association for the Surgery of Trauma (AAST) Splenic Injury Scale

- **Grade I:** Subcapsular hematoma <10% surface area; Capsular tear <1 cm deep.
- **Grade II:** Subcapsular hematoma 10%–50% surface area or intraparenchymal <5 cm; Laceration 1–3 cm deep (not involving trabecular vessels).
- **Grade III:** Subcapsular hematoma >50% or ruptured/expanding; Laceration >3 cm deep or involving trabecular vessels.
- **Grade IV:** Laceration involving segmental or hilar vessels producing major devascularization (>25% of spleen).
- **Grade V:** Completely shattered spleen or hilar vascular injury completely devascularizing the organ.

Western Trauma Association (WTA) Algorithm for Blunt Splenic Injury

- **Stable Patient:** Resuscitate -> CT Scan of Abdomen/Pelvis with IV contrast.
  - *Grade I, II, III without blush:* Observation.
  - *Grade I, II, III with blush:* Angiography / Embolization.
  - *Failure of observation:* Operating Room.
- **Unstable Patient:** FAST or DPA.
  - *Positive:* Direct to Operating Room for laparotomy/splenectomy.
  - *Negative:* Look for other causes of instability.

Operative / Procedural Notes

- **Vaccination Timing:** For elective splenectomies, immunizations against encapsulated organisms (pneumococcal, meningococcal, Hib) must be administered **2-4 weeks prior to surgery** for optimal immunologic response. For emergent trauma splenectomies, vaccinate post-operatively.
- **Surgical Approach:** Open splenectomy is preferred over laparoscopy when there is massive splenomegaly, cancer surgery requiring en-bloc resection, or a need to thoroughly search for accessory spleens. Failure to remove an accessory spleen can lead to disease recurrence in conditions like ITP or spherocytosis.

- **Damage Control:** In unstable trauma patients who are coagulopathic, hypothermic, or acidotic, total splenectomy is the safest option. Attempting splenic salvage (splenorrhaphy or partial splenectomy) is abandoned in the face of physiological collapse.

Complications / Prognosis

- **Overwhelming Post-Splenectomy Infection (OPSI):**
  - **Pathogens:** Primarily caused by encapsulated organisms. ***Streptococcus pneumoniae*** is the most common and most lethal cause. *Haemophilus influenzae* and *Neisseria meningitidis* are also notable.
  - **Timing:** The risk of OPSI is highest within the first **2 years** post-operation.
  - **Incidence:** Following trauma splenectomy, the incidence is very low (**0.1% to 0.5%**), contrary to the 2% risk seen in patients undergoing splenectomy for hematologic malignancies.
  - **Mortality:** Carries a 50–80% mortality rate without rapid antibiotic treatment.
- **Reactive Thrombocytosis:** Physiologically expected postoperative response. Platelet counts typically peak between 7 and 20 days postoperatively before gradually normalizing.
- **Splenosis:** Iatrogenic or traumatic rupture of the spleen can cause fragments of splenic tissue to implant throughout the peritoneal cavity.
- **Vascular Thrombosis:** Postoperative DVT/PE risk is increased (~10%). Portal, mesenteric, and splenic vein thrombosis can also occur.

Past-Paper High Yield

- **Septated Splenic Abscess:** Always choose **splenectomy + antibiotics**. Do not choose percutaneous drainage, as septations cause it to fail.
- **Vaccine Timing:** Vaccines must be given **2 to 4 weeks BEFORE** an elective splenectomy.
- **Stable Trauma with Kehr Sign:** Always choose **CT abdomen** as the next step. Laparotomy is the wrong answer for a hemodynamically stable patient.
- **Most common cause of OPSI:** ***Streptococcus pneumoniae***.
- **Thalassemia Trap:** Splenectomy is **NOT** standard or first-line treatment for all beta-thalassemia patients; standard care is transfusions and chelation.
- **Anatomical Trap:** The spleen is **intraperitoneal**. The kidneys and adrenals are primarily retroperitoneal; the pancreas is secondary retroperitoneal.
- **ITP Clinical Feature:** The spleen is generally **NOT palpably enlarged** in ITP.
- **OPSI incidence in trauma:** Understand that the rate is much lower than 2% (typically **0.1% - 0.5%**).

Memory Pearls

- **Rule of Odds (1, 3, 5, 7, 9, 11):** The spleen is roughly 1 x 3 x 5 inches in size, weighs about 7 ounces, and lies between the 9th and 11th ribs.
- **Encapsulated bugs to vaccinate against (SHiN):** **S**treptococcus pneumoniae, **H**aemophilus **i**nfluenzae, **N**eisseria meningitidis.

Neuroendocrine tumors

Core Concepts

- **Origin:** Arise from pluripotent stem cells in the base of intestinal crypts or gastric glands (not mature pancreatic islets). They originate from APUD (Amine Precursor Uptake and Decarboxylation) cells, which constitute the diffuse endocrine system.
- **Embryological Classification:**
  - **Foregut:** Thymus, esophagus, lung, stomach, duodenum, pancreas.
  - **Midgut:** Appendix, ileum, cecum, ascending colon.
  - **Hindgut:** Distal colon, rectum.
- **Epidemiology:**
  - Second most prevalent GI tumor overall due to indolent course and long survival (median localized survival ~200 months). Over 60% of NETs are advanced at the time of diagnosis (median survival for advanced: 33 months).
  - **Most common primary site:** GI tract (58%), specifically rectum (17.2%) and jejunum/ileum (13.4%).
  - *Geographic variance:* In Middle East & Asia Pacific cohorts, the pancreas is the most common primary site (~49%).
- **Pancreatic NETs (pNETs):**
  - Account for 7% of all NETs and 1–2% of all pancreatic tumors.
  - **Most common overall pNET:** Non-functional tumors.
  - **Most common benign functional pNET:** Insulinoma.
  - **Most common malignant functional pNET:** Gastrinoma.

Diagnosis / Clinical Features

- **Carcinoid Syndrome:**
  - Occurs in 8–35% of patients with NETs. For midgut primary tumors, **hepatic metastases are required** to bypass liver clearance.
  - **Pathophysiology:** Symptoms are driven by the systemic release of **serotonin, histamine, and tachykinins**.
  - **Manifestations:** Episodic flushing (94%), diarrhea (78%), cramping, wheezing/bronchospasm, and potentially carcinoid heart disease (right-sided valvular lesions).
- **Insulinoma (β cells):**
  - Characterized by **Whipple's triad:** 1) Symptoms of hypoglycemia, 2) Plasma glucose < 45 mg/dL (< 2.2 mmol/L), 3) Relief of symptoms with glucose.
  - Often presents with weight gain. 90% are benign and solitary.
- **Gastrinoma / Zollinger-Ellison Syndrome (γ cells):**
  - Presents with severe, refractory peptic ulcers (often non-healing despite PPI therapy), abdominal pain (70%), diarrhea (70%), and heartburn.

- 60% malignant. 25% are associated with Multiple Endocrine Neoplasia Type 1 (MEN-1).

- **VIPoma (δ cells):**
  - Produces **WDHA Syndrome** (Verner-Morrison Syndrome): **W**atery Diarrhea (secretory), **H**ypokalemia, **A**chlorhydria (or hypochlorhydria).
  - Associated with profound hypovolemia and acidosis. 2/3 are malignant.
- **Glucagonoma (α cells):**
  - Typically presents with the **4D Syndrome**: **D**ermatitis (Migratory necrolytic erythema—blistering/scaly lesions with central clearing), **D**iabetes, **D**iarrhea, **D**VT.
  - Nearly all are malignant; mostly located in the body and tail of the pancreas.
- **Somatostatinoma (δ cells):**
  - Usually located in the pancreatic head. 70–90% malignant.
  - Unpredictable presentation but typically includes gallstones (59%), mild diabetes (75%), steatorrhea, and diarrhea.

Investigations

- **Biomarkers:**
  - **Chromogranin A (CgA):** Best general biomarker for diagnosis (elevated 80–100% of the time). Correlates with tumor progression. Present in secretory granules.
  - **Synaptophysin:** Excellent for identifying poorly granulated or poorly differentiated NETs that may be CgA negative (expressed independently of secretory granules).
  - **Urinary 5-HIAA:** Used to diagnose carcinoid syndrome; it is the inactive metabolic breakdown product of serotonin.
- **Insulinoma Workup:**
  - **72-hour fasting test:** Diagnostic if serum glucose < 45 mg/dL, insulin > 5 µU/L, C-peptide > 0.7 ng/mL, and proinsulin > 6.5 pmol.
  - **Localization:** Endoscopic ultrasound (EUS) has 77% sensitivity; Intraoperative US (IOUS) has 90% accuracy. Selective calcium angiogram with portal venous sampling (ASVS) yields 90% accuracy.
- **Gastrinoma Workup:**
  - **Fasting Serum Gastrin:** > 150 pg/mL (requires cessation of PPI for 1 week).
  - **Basic Acid Output (BAO):** > 15 mmol/h.
  - **Secretin Provocative Test:** Confirmatory if gastrin paradoxically rises by ≥ 200 pg/mL.
- **Imaging Modalities:**
  - CT/MRI for staging.
  - Endoscopic ultrasound (EUS) is primary for pancreatic and duodenal localizations.
  - Somatostatin-receptor scintigraphy (Octreoscan™) or DOTA-TOC FDG/PET is highly effective for primary tumors and metastases (e.g., localizes 91% of VIPomas).

Management

- **VIPoma:** Urgent correction of metabolic abnormalities (vigorous rehydration, potassium replacement) followed by **Octreotide** to halt secretory diarrhea.
- **Surgical Excision:** The mainstay of curative intent for localized tumors (ranges from EMR for small rectal NETs to major resections/enucleation for pNETs).

Relevant Guidelines

- **Diagnostic Algorithm (Stepwise Approach):**
  1. Morphology & Neuroendocrine markers (Differentiate NET vs. non-NET).
  2. Structure + Grade (Differentiate NET vs. Neuroendocrine Carcinoma [NEC]).
  3. Mitoses & Ki67 Index (Assign Grade 1, 2, or 3).
  4. Size & Invasion (Assign TNM Stage I–IV).
- **WHO 2010 Classification of the Digestive System:**
  - *Premise:* All neuroendocrine neoplasms have malignant potential. The term "carcinoid" is a misnomer regarding benign nature and should strictly be reserved for clinical "carcinoid syndrome".
  - Differentiates between Neuroendocrine Tumours (Grade 1/2), Neuroendocrine Carcinomas (Grade 3), and Mixed Adenoneuroendocrine Carcinoma (MANEC).
- **ENETS/WHO/AJCC Grading of GEP-NETs:**

GRADE	KI67 INDEX (%)	MITOTIC COUNT (PER 10 HPF)
G1	≤ 2	< 2
G2	3 – 20	2 – 20
G3	> 20	> 20

- **ENETS/AJCC TNM Staging Proposal:**
  - **Stage I/II:** Localized (T1–T3, N0, M0) → Median survival is high and stable over 200 months.
  - **Stage III:** Regional nodal involvement (Any T, N1, M0) → Median survival drops to ~50% at 150 months.
  - **Stage IV:** Distant metastasis (Any T, Any N, M1) → Median survival drops below 40% by 100 months.

Operative / Procedural Notes

- **Passaro's Gastrinoma Triangle:** The anatomical region where up to 90% of gastrinomas are located. Bounded by:
  1. Junction of the cystic and common hepatic ducts.
  2. Junction of the second (D2) and third (D3) parts of the duodenum.
  3. Junction of the neck and body of the pancreas.

- **Insulinoma Localization:** Tumors are distributed evenly across the pancreas (1/3 head, 1/3 body, 1/3 tail). They are highly vascular, well-circumscribed, and usually enucleated if benign and superficially located.

Complications / Prognosis

- **Delayed Diagnosis:** NETs can be undetected for years; over 60% are advanced at the time of diagnosis.
- **Carcinoid Heart Disease:** Chronic exposure to elevated systemic serotonin leads to right-sided heart valvular fibrotic lesions, eventually resulting in heart failure.

Past-Paper High Yield

- **Anatomical Site of Gastrinoma:** The **duodenum** is the single most common anatomical site for a gastrinoma, accounting for 50-70% of cases (usually within Passaro's gastrinoma triangle). Do not select the stomach antrum or head/body of the pancreas if the duodenum is an option.
- **MEN-1 vs. Zollinger-Ellison (ZES) alone:** A patient presenting with a refractory peptic ulcer (gastrinoma) *and* hypercalcemia has **MEN-1**. ZES alone lacks hypercalcemia; primary hyperparathyroidism (causing the elevated calcium) is the key differentiator for MEN-1.
- **Mechanism of Carcinoid Syndrome:** Systemic manifestations (flushing, diarrhea, bronchospasm) are caused by the release of **serotonin, histamine, and tachykinins**. *Exam trap:* 5-HIAA does *not* cause symptoms; it is an inactive breakdown product measured in the urine for diagnosis.
- **Ulcer Classification trap:** Type I benign gastric ulcers (most common, lesser curvature) are due to *decreased mucosal defenses* with normal/low acid. Do not confuse them with ZES ulcers, which are characterized by profound hypergastrinemia and marked acid hypersecretion.

Memory Pearls

- **Whipple's Triad (Insulinoma):** Low glucose, Hypoglycemic symptoms, Relief with glucose.
- **WDHA Syndrome (VIPoma): W**atery **D**iarrhea, **H**ypokalemia, **A**chlorhydria.
- **4 D's of Glucagonoma: D**ermatitis (Migratory necrolytic erythema), **D**iabetes, **D**iarrhea, **D**VT.
- **Gastrinoma Triangle Landmarks:** Duct junction (Cystic/Hepatic) → Duodenum junction (D2/D3) → Pancreas junction (Neck/Body).

Breast

Exam Map

LECTURE TOPIC	QUESTION WEIGHT	REVISION PRIORITY
Breast Cancer Overview	22 Qs (~54%)	**High** - Master BI-RADS algorithms, staging rules, and T4 vs. regional signs.
Intro & Benign Disorders	10 Qs (~24%)	**Medium** - Differentiate benign lumps, nipple discharge types, and abscess care.
Cancer Management	9 Qs (~22%)	**Medium** - Focus on operative decision-making (SLNB vs ALND) and nerve injuries.

- **Diagnostic Algorithms & Next Steps:**
  - **Triple Assessment:** Mandatory for any symptomatic lump (Clinical, Imaging, Pathology).
  - **Cyst Management:** Fine Needle Aspiration (FNA) is the diagnostic *and* therapeutic first step for symptomatic, palpable cysts.
  - **BI-RADS Actions:** BI-RADS 3 = repeat imaging in 6 months. BI-RADS 4 (even if non-palpable microcalcifications) = stereotactic core needle biopsy. Do *not* choose 6-month follow-up or open excision for BI-RADS 4.
- **Recurring Clinical Presentations:**
  - **Nipple Discharge:** Bloody = Intraductal papilloma; Thick green/brown with slit-like inversion = Duct ectasia / periductal mastitis.
  - **Nipple Skin Changes:** Bilateral/itchy/intact nipple = Eczema; Unilateral/destructive/underlying mass = Paget's disease.
  - **Lactational Abscess:** Drainage is required, but the heavily tested principle is that patients must **continue breastfeeding/pumping** to prevent stasis.
- **Staging Rules & Clinical Traps:**
  - **T-Stage Calculation:** Determined *strictly* by the size of the invasive component. Always ignore the *in situ* size when determining the T-stage.
  - **T4 vs. Regional Disease:** Skin dimpling (Cooper's ligaments), peau d'orange (dermal lymphatics), and pectoral fixation are direct local T4 signs. **Arm edema** is a regional axillary node complication, *not* a T4 sign.
  - **Premalignant Risk:** Atypical Ductal Hyperplasia (ADH) carries the highest risk (4-5x) for subsequent malignancy compared to other benign conditions.
- **Operative Decision Points:**
  - **Early-Stage Disease (Node-Negative):** Standard of care is Breast-Conserving Surgery (lumpectomy) + Sentinel Lymph Node Biopsy (SLNB) + Radiotherapy. Never perform an upfront formal ALND if the SLNB is negative.

    - **Inflammatory Breast Cancer (T4d):** Upfront surgery is strictly contraindicated. The operative sequence must be: Neoadjuvant Chemotherapy → Modified Radical Mastectomy → Radiotherapy.
- **Post-Operative Complications:**
    - **ALND Morbidity:** Ipsilateral upper extremity lymphedema is the most common and significant chronic complication of formal axillary dissection.
    - **Nerve Injuries:** Know the anatomical associations: Intercostobrachial nerve (medial upper arm numbness) and Long Thoracic nerve (winged scapula).

Breast introduction & benign breast disorders

Core Concepts

Anatomy & Boundaries

- **Structure:** Modified sweat gland lying in the deep pectoral fascia.
- **Boundaries:**
    - *Superior:* Clavicle
    - *Inferior:* Inframammary fold
    - *Medial:* Sternum
    - *Lateral:* Lateral border of the latissimus dorsi muscle
- **Glandular Pathway:** Acini (within lobules) → Ducts → Ampulla (lactiferous sinus) → Nipple.
- **Blood Supply:** Perforating branches of internal mammary (thoracic) artery, lateral thoracic artery, thoracoacromial artery, and posterior intercostal arteries.
- **Lymphatic Drainage:** >75% of ipsilateral breast drainage goes to the **axillary lymph nodes**. Other pathways include supraclavicular and internal mammary chains.
- **Axillary Anatomy:** The **Long Thoracic Nerve** (nerve of Bell) runs vertically along the lateral chest wall on the serratus anterior muscle. Injury during axillary clearance causes a "winged scapula."

Physiology & Embryology

- **Hormonal Regulation:**
    - *Prolactin:* Acts on luminal epithelial cells to stimulate milk secretion into the alveolus.
    - *Oxytocin:* Acts on basal myoepithelial (contractile) cells to trigger milk ejection.
- **Embryology (The Milk Line):** The mammary ridge develops from the axilla down to the groin.
    - *Incomplete regression:* Leads to accessory breast tissue (polymastia) or accessory nipples (polythelia) anywhere along this path.
    - *Failure of development:* Leads to complete absence of breast tissue (amastia).

Diagnosis / Clinical Features

Clinical Presentation & Differentials

- **Painless Lump:** Carcinoma, cyst, fibroadenoma, fibroadenosis.

- **Painful Lump:** Fibroadenosis, cyst, periductal mastitis, abscess (usually postpartum/lactational), occasionally carcinoma.
- **Nipple Discharge:**
  - *Unilateral, Bloody:* **Intraductal papilloma** (most common), Ductal carcinoma-in-situ (DCIS), invasive ductal carcinoma.
  - *Thick, Multicolored (creamy/green/brown):* Duct ectasia / Periductal mastitis.
- **Nipple/Areola Changes:** Duct ectasia (slit-like inversion), carcinoma, Paget's disease, eczema.

Benign Breast Disorders

- **Fibroadenoma:** Most common in ages 15–55. Presents as a smooth, rubbery, well-circumscribed, highly mobile mass ("breast mouse").
- **Intraductal Papilloma:** Benign papillary neoplasm within the milk ducts. Unpalpable or soft peri-areolar swelling; classically presents with bloody nipple discharge.
- **Periductal Mastitis / Duct Ectasia:** Dilatation of mammary ducts filled with inspissated debris (macrophages/chronic inflammation). Features include green/thick discharge, slit-like nipple inversion, and potential for subareolar abscess leading to a **mammillary fistula**.
- **Breast Cyst:** Sudden onset, tender, fluctuating, perimenopausal (40–55 years). Often multiple.
- **Phyllodes Tumor:** Fibroepithelial tumor presenting as a firm, mobile, rapidly growing mass (similar to a fibroadenoma clinically). ~10-25% are malignant. Exhibits leaf-like epithelial-lined spaces histologically. High local recurrence rate if inadequately excised.
- **Gynecomastia (Male):**
  - *True Gynecomastia:* Benign glandular proliferation. Responsive to hormone modulation (e.g., testosterone replacement for primary hypogonadism). Induced by medications like spironolactone. Prevalence increases with advancing age (declining testosterone / increased aromatization). Does *not* increase male breast cancer risk.
  - *Pseudo-gynecomastia (Lipomastia):* Accumulation of subareolar adipose tissue without glandular tissue. Unresponsive to anti-estrogen therapies (e.g., tamoxifen).

Differentiating Eczema vs. Paget's Disease of the Nipple

- *Pathogenesis of Paget's:* Malignant cells (from underlying DCIS or invasive cancer) migrate up the lactiferous ducts to infiltrate the nipple epidermis.

FEATURE	SIMPLE ECZEMA	PAGET'S DISEASE
Laterality	Bilateral	Unilateral
Age / Context	Sometimes lactating	Older females
Symptoms	Itches, vesicles present	Does not itch, no vesicles
Anatomy	Nipple remains intact	Nipple may be destroyed
Palpation	No underlying lump	Underlying lump may be present

Comparison of Common Breast Lumps

TYPE OF LUMP	AGE (YEARS)	PAIN	SURFACE	CONSISTENCY	AXILLA
Solitary cyst	40–55	Occasional	Smooth	Soft to hard	Normal
Nodularity	20–55	Often	Indistinct	Mixed	Normal
Fibroadenoma	15–55	No	Smooth, bosselated	Rubbery	Normal
Carcinoma	35+	Uncommon	Irregular	Hard	Nodes may be palpable

Investigations

Triple Assessment For any symptomatic patient, diagnosis mandates all three components:

1. **Clinical:** History and physical examination.
2. **Imaging:** Mammography and/or Ultrasound.
3. **Pathology:** Fine Needle Aspiration Cytology (FNAC) or Core Needle Biopsy.

Imaging Modalities

- **Mammography:** Most sensitive overall, but sensitivity decreases in young women (dense glandular breasts). Standard views: Craniocaudal (CC) and Mediolateral Oblique (MLO; captures axillary tail and pectoralis).
- **Ultrasound:** Highly useful for differentiating solid vs. cystic lesions, guiding biopsies, and assessing young/dense breasts. Cysts appear as well-circumscribed, anechoic structures with posterior acoustic enhancement. Sens 75%, Spec 97% for malignancy.
- **Ductogram (Galactogram):** Used for pathological nipple discharge; reveals contrast filling defects (e.g., intraductal papilloma).

Pathological Tissue Sampling

- **FNAC:** Uses syringe/needle to aspirate cells (cytology). Rapid and minimally invasive. Diagnostic and therapeutic for cysts.
- **Core Needle Biopsy:** Extracts a tissue cylinder (histology). Preserves tissue architecture, distinguishing *in-situ* from *invasive* disease, and allows receptor testing (ER/PR/HER2). Essential for solid masses.

Management

- **Symptomatic Simple Cyst:** The most appropriate initial step is **Fine Needle Aspiration (FNA)**. It is both diagnostic and therapeutic, providing immediate symptomatic relief. If fluid is non-bloody and the cyst completely collapses, no further intervention is needed.
- **Lactational Breast Abscess:** Most commonly *Staphylococcus aureus*, ascending via a macerated/cracked nipple.
    - *Treatment:* Requires drainage (US-guided aspiration or open I&D).
    - *Crucial principle:* **Do NOT discontinue breastfeeding**. Continued feeding/pumping prevents milk stasis and promotes resolution. If unresolved despite antibiotics/drainage, a biopsy is

indicated to rule out inflammatory breast cancer.

- **Fibroadenoma:** Can be managed with observation or surgical enucleation (circumareolar/submammary incision for cosmesis).
- **Periductal Mastitis / Mammillary Fistula:** Excision of the major terminal duct system (Hadfield's procedure) to cure chronic infection and close the fistula tract.

Relevant Guidelines

BI-RADS Mammographic Assessment Categories

ASSESSMENT CATEGORY	CLINICAL RECOMMENDATION	PROBABILITY OF MALIGNANCY
0: Incomplete	Need for further evaluation	N/A
1: Normal	Normal interval follow-up	0%
2: Benign	Normal interval follow-up	0%
3: Probably benign	Short interval follow-up recommended	<2%
4: Suspicious abnormality	Biopsy should be considered	≥2% to <95%
5: Highly suggestive of malignancy	Biopsy or surgery should be performed	≥95%
6: Biopsy-proven carcinoma	Appropriate clinical action taken	-

Operative / Procedural Notes

- **Hadfield's Operation (Major Duct Excision):** Indicated for recurrent periductal mastitis or mammillary fistulae. Involves a circumareolar incision, deep dissection, and wide excision of the terminal major duct bundle behind the nipple-areola complex.
- **Abscess Drainage:** For severe fluctuant abscesses, an incision is made, and a corrugated rubber drain (Penrose drain) is inserted deeply to prevent premature skin closure, allowing the cavity to heal by secondary intention.
- **Fibroadenoma Enucleation:** Simple surgical "popping out" of the encapsulated mass from surrounding parenchyma.
- **Clinical Breast Exam Patterns:** Palpation should be methodical—radial (spoke-wheel), concentric circles, or vertical strips (lawnmower). Evaluate the axilla by supporting the patient's arm to fully relax the pectoralis muscles.

Complications / Prognosis

- **Risk of Malignancy in Benign Breast Disease:**
  - *High Risk:* **Atypical ductal hyperplasia (ADH)** carries a 4–5x increased relative risk for invasive breast cancer.
  - *Slightly Increased Risk:* Sclerosing adenosis (1.5–2x relative risk).
  - *No Significant Risk:* Duct ectasia, simple cysts, and fibroadenomas are non-proliferative and do not inherently increase cancer risk.

- **Phyllodes Tumor Spread:** Uniquely among malignant breast tumors, it characteristically spreads **hematogenously** (to lungs and bone). Lymph node metastasis is extremely rare (<5%), so axillary dissection is rarely needed.
- **Locally Advanced Breast Cancer Signs:** Tumor progression (T3/T4) causing local invasion leads to skin dimpling (fibrosis of Cooper's ligaments), nipple inversion (ductal fibrosis), peau d'orange (dermal lymphatic embolization), and pectoral muscle fixation. *Note: Arm edema indicates regional advancement (axillary lymphatic obstruction), not direct local tumor invasion.*

Past-Paper High Yield

- **Intraductal papilloma** is the most common cause of unilateral, bloody nipple discharge. (Do not confuse with duct ectasia, which produces thick, green/brown, multicolored discharge).
- **Fine Needle Aspiration (FNA)** is the first-line management for a symptomatic, painful, palpable cyst (even large, e.g., 4 cm). It is diagnostic *and* therapeutic; excisional biopsy or MRI are incorrect initial choices.
- **Lactational Abscess:** Always encourage the patient to **continue breastfeeding** or pumping to prevent stasis. Surgical drainage is mandatory if fluctuant. If a presumed abscess fails to resolve, you must biopsy to rule out inflammatory carcinoma.
- **Phyllodes Tumors** spread **hematogenously** (lungs, bone) rather than via lymphatics. They present like fast-growing fibroadenomas and require wide excision due to high local recurrence rates.
- **Atypical Ductal Hyperplasia (ADH)** is the benign proliferative condition carrying the highest risk (4-5x) for subsequent breast malignancy.
- **Polymastia/Polythelia** results from *incomplete regression* of the mammary ridge (milk line). *Amastia* is failure of the ridge to develop.
- **Gynecomastia:** True gynecomastia is responsive to hormonal shifts (e.g., spironolactone use, testosterone replacement for hypogonadism, advancing age). *Pseudo-gynecomastia* (lipomastia) is pure fat and completely unresponsive to anti-estrogens like tamoxifen.
- **Arm edema** is a sign of extensive *regional* axillary lymph node disease, whereas peau d'orange, dimpling, and pectoral fixation are signs of a *locally advanced primary* tumor.

Memory Pearls

- **"Breast mouse"** → Fibroadenoma (highly mobile).
- **Bloody nipple discharge** → Intraductal papilloma.
- **Slit-like nipple inversion + Green discharge** → Duct ectasia / Periductal mastitis.
- **Winged Scapula post-axillary dissection** → Long thoracic nerve injury (Serratus anterior).
- **Eczema vs. Paget's** → Eczema is bilateral/itchy/no lump; Paget's is unilateral/destroys nipple/underlying mass.

Breast cancer overview

Core Concepts

- **Triple Assessment Approach**: The gold standard for evaluating breast masses involves three pillars:
  1. Clinical Evaluation (focused history + standardized examination)
  2. Imaging (Mammography, Ultrasound, or MRI)
  3. Cytology or Histology (FNA or Core Needle Biopsy)
- **Risk Factors**:
  - **Advancing age** is the single most significant risk factor for sporadic breast cancer (excluding female sex).
  - *Note*: **70% of women diagnosed have no identifiable risk factors**.
  - Other factors: Female gender (100:1 ratio), Caucasian race, obesity (BMI >30), exogenous hormones, nulliparity, early menarche.
  - Family history: One 1st-degree relative doubles risk; two 1st-degree relatives triple risk.
  - Genetics: *BRCA1, BRCA2, p53* account for only 5–6% of all breast cancers.
- **Receptor Subtypes & Targeted Therapies**:
  - **ER-positive / HER2-negative**: Treated with endocrine therapies (tamoxifen, aromatase inhibitors).
  - **HER2-positive**: Treated with targeted monoclonal antibodies (e.g., **Trastuzumab**, which directly targets the HER2/neu receptor). Amplification of HER2/neu is an indicator of aggressive tumor biology and poor prognosis.
  - **Triple-negative (Basal-like)**: Lacks ER, PR, and HER2. Often associated with *BRCA1* mutations and medullary histology.

Diagnosis / Clinical Features

- **Classic Malignant Mass**: Typically hard, irregular, and painless (only 10–15% are painful).
- **Direct Local Invasion (T4 Signs)**: Occurs via contiguous invasion of the primary tumor into surrounding tissue.
  - **Skin dimpling/tethering**: Caused by the tumor's desmoplastic fibrotic reaction shortening and tethering **Cooper's suspensory ligaments**.
  - **Nipple retraction/inversion**: Pathologic fibrosis and contracture of the major subareolar lactiferous ducts.
  - **Peau d'orange**: Edema and "orange-peel" pitting of the skin caused by tumor emboli blocking **dermal lymphatics**.
  - **Pectoral fixation**: Deep fixation to the pectoral fascia or muscle (best assessed clinically by having the patient press hands into hips to contract the pectoralis major).
  - **Skin ulceration & satellite nodules**.
- **Regional Disease (Nodal Spread)**:

- **Arm edema (Lymphedema)**: A sign of regional lymphatic obstruction from extensive metastatic axillary lymph nodes. *Crucial exam point*: It is a regional complication, **not** a direct local T4 extension of the breast mass.

- **Nipple Discharge**: Spontaneous, single-duct, bloody or serosanguineous (watery) discharge is highly suspicious for intraductal pathology (e.g., papilloma or DCIS).
- **Paget's Disease of the Nipple**:
  - Characterized by intraepithelial spread of malignant adenocarcinoma cells (Paget cells) into the nipple epidermis. Represents **carcinoma in situ**.
  - Presents as an erythematous, eczematous, scaling, crusting, or ulcerating lesion of the nipple-areolar complex. Highly associated with underlying invasive or in situ carcinoma.

Investigations

- **Mammography**:
  - Standard views: **Craniocaudal (CC)** and **Mediolateral Oblique (MLO)**. MLO captures the maximum tissue amount, including the axillary tail (Tail of Spence).
  - *Suspicious signs*: Irregular, spicular masses causing architectural distortion, and clustered pleomorphic microcalcifications (classic for early DCIS/invasive cancer).
  - *Limitations*: Sensitivity is significantly reduced in young women due to dense fibroglandular tissue. In older women with fatty breasts, mammography is highly sensitive and accurate for tumor extent.
- **Ultrasound**:
  - Essential for differentiating solid vs. cystic lesions (e.g., cysts show eggshell calcifications on mammogram and are fluid-filled on US).
  - Guides focal biopsies (FNA or core needle).
- **MRI (Magnetic Resonance Imaging)**:
  - *Indications*: Screening high-risk young women (e.g., *BRCA* mutations), evaluating occult primary tumors in patients with positive axillary nodes, and assessing disease extent in invasive lobular cancer.
  - *Kinetic curves*: Type I (progressive = benign); Type II (plateau = indeterminate); **Type III** (rapid early enhancement followed by rapid wash-out = 90% malignant).
  - *Contraindication/Limitation*: Not a substitute for biopsy. In older women with fatty breasts, routine MRI does not outperform mammography and causes false positives.
- **Biopsy Techniques**:
  - **Core Needle Biopsy (CNB)**: The standard of care. High sensitivity (100%), distinguishes between *in situ* and *invasive* cancer (unlike FNA).
  - **FNA**: Cannot distinguish in-situ from invasive disease. Ideal for simple cyst aspiration.
  - **Stereotactic Image-Guided Biopsy**: The mandatory next step for non-palpable mammographic findings (e.g., clustered microcalcifications detected only on a screening mammogram).
- **Metastatic Workup (Stage IV)**:

- Symptoms direct imaging: Bone scan for skeletal complaints, CT chest/abdomen for visceral spread.
- Labs: LFTs, Renal function, CBC (anemia from bone marrow infiltration), Tumor marker CA 15.3.

Relevant Guidelines

American Cancer Society (ACS) Breast Screening Guidelines

AGE RANGE	MAMMOGRAPHY	CLINICAL BREAST EXAM
40–44	Optional annual	No
45–54	Annual	No
55+	Every 1 or 2 years	No

(Screening continues as long as life expectancy is >10 years)

BI-RADS Classification & Management Algorithm

CATEGORY	FINDING	PROBABILITY OF MALIGNANCY	NEXT STEP IN MANAGEMENT
1 / 2	Normal / Benign	0%	Routine screening
3	Probably Benign	< 2%	**Short interval follow-up (Repeat mammogram in 6 months)**
4	Suspicious	2% to <95%	**Tissue Biopsy** (Stereotactic CNB if non-palpable)
5	Highly Suggestive	≥ 95%	Biopsy or Surgery

AJCC TNM Staging Framework

- **T (Tumor)**: *Calculated strictly by the size of the invasive component (ignore in situ component).*
  - Tis: Carcinoma in situ
  - T1: < 2 cm
  - T2: 2–5 cm
  - T3: > 5 cm
  - T4: Any size, but extends to skin or chest wall
- **N (Nodes)**:
  - N0: None
  - N1: Movable same-side axillary nodes
  - N2: **Fixed or matted** same-side axillary nodes
  - N3: Same-side internal mammary nodes
- **M (Metastasis)**:

- M0 = None, M1 = Distant (Brain, Lungs, Liver, Bones)
- *Stage Grouping Pearls*:
  - **Stage I**: T1N0M0 (85% 5-year survival).
  - **Stage II**: e.g., T2N1M0.
  - **Stage III**: e.g., T2N2M0 (T2 with *matted/fixed nodes*).

Nottingham Histological Grading System

- Based on Glandular/tubular differentiation, Nuclear pleomorphism, and Mitotic count.
  - **Grade 1 (Well-diff)**: >75% gland formation, uniform cells, <7 mitoses.
  - **Grade 2 (Moderate)**: 10–75% glands, moderate variability.
  - **Grade 3 (Poorly-diff)**: <10% glands, highly pleomorphic, >16 mitoses.

Operative / Procedural Notes

- **Breast Conserving Surgery (BCS)**: Wide local excision/lumpectomy with clear margins. Combined with radiation therapy, it offers equivalent survival to mastectomy for early-stage disease.
  - *Oncoplastic techniques*: Use local flaps and parenchymal pillars to prevent severe cosmetic deformities and contour collapse.
- **Mastectomy Approaches**:
  - *Radical Mastectomy*: Removes breast, nodes, and pectoralis major/minor. Rarely done today; results in shoulder instability and severe cosmetic deformity.
  - *Modified Radical Mastectomy*: Removes breast and Level I/II axillary nodes but **preserves the pectoralis major muscle**.
  - *Skin-sparing Mastectomy*: Preserves the skin envelope (nipple-areola excised), allowing for immediate prosthetic/implant reconstruction.
- **Reconstructive Flaps**:
  - **TRAM Flap** (Transverse Rectus Abdominis Myocutaneous): Pedicled from the abdomen.
  - **Latissimus Dorsi Flap**: Pedicled from the back. Good for volume deficits, but the transplanted muscle will eventually atrophy, and there is a loss of latissimus function.
- **Axillary Management**:
  - **Sentinel Lymph Node Biopsy (SLNB)**: Injection of blue dye/radiotracer to identify the first draining axillary lymph node. If negative, complete axillary lymph node dissection (ALND) is avoided, significantly reducing the risk of lymphedema.

Complications / Prognosis

- **Recurrence**:
  - Most breast cancer recurrences (especially in triple-negative or HER2+ tumors) happen within the **first 5 years**.
  - *Exception*: **ER-positive tumors** are notorious for **late recurrences** (10 to 20 years later).
- **Metastatic Pathways**:

    - Breast cancer frequently hematogenously metastasizes to the axial skeleton (vertebral column) via the valveless **Batson's vertebral venous plexus**.
- **Histological Variants**:
    - **Invasive Ductal Carcinoma (IDC)**: Most common. The presence of surrounding DCIS does *not* adversely affect the prognosis of the invasive component.
    - **Medullary Carcinoma**: Often large and triple-negative, with dense lymphocytic infiltrates, no desmoplastic stroma, and pushing borders. **Paradoxically associated with a better prognosis** than standard IDC, even with node involvement.
    - **Invasive Lobular Carcinoma (ILC)**: Associated with a higher incidence of bilateral and multicentric disease.
    - **Mucinous (Colloid) Carcinoma**: A rare variant, making up only 2–3% of breast cancers.
    - *Mixed tumors*: Clinical behavior is driven by the worst/most aggressive component.

Past-Paper High Yield

- **BI-RADS 3 vs 4 Trap**: BI-RADS 3 = repeat imaging in 6 months. BI-RADS 4 (even if non-palpable and only clustered microcalcifications on screening) = **Stereotactic Core Needle Biopsy**. *Do not choose reassurance, 6-month follow-up, or open excision as the first step for BI-RADS 4.*
- **T-Stage Calculation Trap**: The T-stage is determined **strictly by the size of the invasive component**. If a question stem gives a "3.2 cm carcinoma in situ with an invasive component of 0.9 cm," the tumor is a **T1**.
- **T4 vs Regional Disease Trap**: A question will ask which physical finding is *not* a sign of direct local T4 invasion. **Arm edema** is the answer. Arm edema is a regional lymphatic complication of the axilla, whereas peau d'orange, skin tethering, and chest wall fixation are direct local T4 extensions.
- **Staging Trap**: A 4 cm mass (T2) + matted/fixed nodes (N2) is automatically pushed into **Stage III**, regardless of tumor size.
- **Carcinoma in Situ Distinctions**: Paget's disease, Bowen's disease (skin SCC), Erythroplasia of Queyrat (penis SCC), and Hutchinson's freckle (melanoma) are all *in situ*. Basal cell carcinoma is *locally invasive*, NOT in situ.
- **Receptor Nuance**: BRCA2 mutations are more likely to be ER-positive (unlike BRCA1 which is triple-negative). Endocrine therapy is *only* effective for ER+ tumors.
- **MRI Limitations Trap**: Recognize that MRI does *not* significantly outperform mammography in older women with fatty breasts; routine MRI here causes false positives.

Memory Pearls

- **Cooper's Ligaments** = Skin Dimpling.
- **Dermal Lymphatics blocked** = Peau d'orange.
- **Subareolar Duct Fibrosis** = Nipple Retraction.
- **Batson's Plexus** = Spine Metastasis.
- **Medullary Carcinoma** = Looks bad (Grade 3/triple negative), acts good (good prognosis).
- **Microcalcifications only** = Stereotactic biopsy.
- **HER2** = Trastuzumab (Herceptin) target; implies worse tumor biology.

Breast cancer management

Core Concepts

- **Multimodal Treatment:** Cancer management relies on single or combined modalities: Surgery, Radiotherapy, Chemotherapy, and Hormonal Therapy.
- **Surgical Oncology Objectives:** Prevention, diagnosis, treatment of primary tumor, resection of metastasis, management of oncologic emergencies (e.g., hemorrhage, abscess), palliation, reconstruction, and cytoreduction.
- **Principles of Oncologic Resection:** Achieve adequate resection margins, prevent tumor spillage, and utilize minimal manipulation of the tumor during excision.
- **Breast-Conserving Therapy (BCT):** Lumpectomy followed by whole-breast radiation therapy is as effective in terms of survival as a modified radical mastectomy for appropriately selected patients.

Diagnosis / Clinical Features

- **Clinical Evaluation:** Relies on history, physical examination (clinical breast exam), and assessing the involved system for late symptoms/signs.
- **Inflammatory Breast Cancer (T4d):** A highly aggressive, locally advanced breast cancer. Clinically presents as an edematous, erythematous breast (peau d'orange) due to dermal lymphatic invasion by tumor cells.
- **Locally Advanced Breast Cancer (T4):** Involves direct extension to the chest wall or skin, or presents as inflammatory breast cancer.

Investigations

- **Fine Needle Aspiration Biopsy (FNAB):**
  - Simple outpatient procedure extracting tissue for cytological examination.
  - *Major limitation:* A negative result does not reliably rule out cancer.
- **Incisional Biopsy:** Removes only part of the tumor. Usually reserved for very large tumors or special circumstances; rarely performed today.
- **Excisional Biopsy:** Most common biopsy procedure. The entire lump is removed. If histological margins are negative, this can serve as a definitive lumpectomy.
- **Surgeon's Biopsy Responsibilities:**
  - Select the appropriate biopsy method and site.
  - *Specimen Orientation:* Must orient the specimen for the pathologist (e.g., placing a suture at the superior margin).
  - Ensure the integrity of the tissue plane and adequacy of the sample.
  - Communicate results and initial prognosis to the patient.

Management

- **Early-Stage Invasive Breast Cancer (e.g., T1/T2, Clinically Node-Negative):**

- **Standard of Care:** Breast-conserving surgery (Wide Local Excision / Lumpectomy) + Sentinel Lymph Node Biopsy (SLNB) + Post-operative Radiotherapy.
- Mastectomy is an option but not mandatory unless there are contraindications to radiation or if disease is multicentric.

- **Locally Advanced / Inflammatory Breast Cancer (T4/T4d):**
  - Upfront surgery is **strictly contraindicated** due to high risk of positive margins and rapid recurrence.
  - **Standard Pathway:** Neoadjuvant systemic chemotherapy (to downstage tumor and treat micrometastases) → Modified Radical Mastectomy → Post-mastectomy Radiotherapy.
- **Surgical Resection Types:**
  - **Lumpectomy (Wide Local Excision):** Excision of the primary tumor with a surrounding circumferential margin of normal healthy tissue.
  - **Total (Simple) Mastectomy:** Entire breast tissue is removed. Pectoral muscles and axillary lymph nodes are preserved. Indicated for early non-invasive cancers or as prophylactic surgery (e.g., BRCA mutations).
  - **Modified Radical Mastectomy (MRM):** Entire breast tissue is removed along with an axillary lymph node dissection (Level I-II). Pectoralis major and minor muscles are preserved.
  - **Radical Mastectomy:** Entire breast, axillary nodes, and both pectoralis major and minor muscles are removed. Obsolete and rarely performed; reserved only for massive tumors directly invading the chest wall musculature.
- **Radiotherapy Principles:**
  - Typical whole-breast doses are 4500–5000 cGy (not <1000 cGy).
  - Primarily provides local control; does not eliminate the need for systemic chemotherapy in treating distant micrometastatic disease.
  - **Strictly contraindicated** during pregnancy (especially first trimester) due to high risk of fetal malformation and demise.
- **Metastatic Disease Surgery:**
  - Metastasectomy can improve 5-year overall survival (e.g., 16% survival benefit for resection of breast cancer metastases).
- **Supportive Surgery:** Placement of subcutaneous venous access catheters (Port-a-cath) allows for repeated chemotherapy infusions, avoiding damage to peripheral veins.

Relevant Guidelines

- **Criteria for Metastasectomy:**
  - The primary tumor is controlled or controllable.
  - Evidence that metastasectomy is associated with clinical benefits.
  - Tumor doubling time is sufficiently long.
  - No significant co-morbid factors.
- **Indications for Primary Surgical Prophylaxis:**
  - Breast: BRCA mutations (bilateral prophylactic mastectomies)

- Colon: Familial Adenomatous Polyposis (FAP) and HNPCC
- Thyroid: MEN II syndrome

Operative / Procedural Notes

- **Axillary Lymph Node Dissection (ALND):**
  - Formal clearance of level I-III axillary nodes.
  - **Contraindication:** Do NOT perform a complete ALND in a patient with an early-stage cancer (e.g., T1) and a pathologically negative SLNB, regardless of tumor quadrant. It adds unnecessary morbidity.
  - **Indications:** Clinically palpable/matted nodes, positive SLNB with massive extranodal extension, failure of SLNB mapping to identify nodes, or inflammatory breast cancer (where SLNB is contraindicated).

Complications / Prognosis

- **Lymphedema (ALND Complication):** The most common, dreaded, and significant long-term complication of a complete ALND (occurring in 20-30% of patients) due to the permanent disruption of lymphatic drainage.
- **Nerve Injuries Post-Axillary Dissection:**
  - **Intercostobrachial Nerve:** Provides sensory innervation to the axilla and medial upper arm. Frequently sacrificed or stretched during axillary clearance, leading to postoperative numbness and paresthesia in the inner arm.
  - **Long Thoracic Nerve:** Injury results in paralysis of the serratus anterior muscle (winged scapula deformity).
  - **Thoracodorsal Nerve:** Injury results in latissimus dorsi weakness (impaired adduction/internal rotation of the arm).
- **Acute Radiation Dermatitis:** A well-known acute side effect of breast radiotherapy characterized by skin erythema, edema, dry/moist desquamation, and occasionally ulceration. **Prognosis:** Transient; typically heals and resolves within a few weeks after completing treatment (rarely permanent).

Past-Paper High Yield

- **SLNB vs. ALND Trap:** Exam questions frequently test the overtreatment of clinically node-negative early-stage breast cancer. The correct approach is always *Wide local excision coupled with a sentinel lymph node biopsy*. Never choose a complete ALND for a negative SLNB, as it is contraindicated.
- **Identifying Lymphedema:** Know that lymphedema of the ipsilateral upper extremity is the *most common chronic complication* of a formal level I-III ALND. Winged scapula and brachial plexopathy are comparatively rare.
- **Nerve Deficits:** Medial upper arm numbness post-mastectomy = **Intercostobrachial nerve** injury.
- **Inflammatory Breast Cancer Trap:** If a question describes a patient with an edematous, erythematous breast and a biopsy proving carcinoma, do NOT choose upfront surgery or antibiotics for cellulitis. The definitive key is *Multimodal therapy: Chemotherapy → Surgery → Radiotherapy*.

- **Radiotherapy Side Effects:** Remember that post-radiation skin erythema is expected and *usually resolves within a few weeks*; it is not a permanent fixture.

Memory Pearls

- **Simple Mastectomy:** Breast out, nodes in, muscles in.
- **Modified Radical Mastectomy:** Breast out, nodes out, muscles in.
- **Radical Mastectomy:** Breast out, nodes out, muscles out.
- **T4d (Inflammatory) Treatment Order: C**hemo → **M**astectomy → **R**adiation (**CMR**).

Endocrine

Exam Map

ROTATION TOPIC	PAST-PAPER WEIGHT	PRIMARY EXAM FOCUS & REVISION PRIORITY
Thyroid Disorders	31 Qs	Cancer subtypes (associations, spread), FNA limitations, Post-op hemorrhage.
Adrenal Gland	18 Qs	Pheochromocytoma pre-op prep, Conn's lab profiles, Incidentaloma algorithm.
Salivary Glands	17 Qs	Pleomorphic adenoma vs. Warthin's, FNA necessity, Distal sialolithiasis management.
Parathyroid Glands	15 Qs	NIH asymptomatic 1° HPT surgical criteria, Intra-op PTH, MEN syndrome triads.
Cervical Nodes	5 Qs	FNA vs. Excisional biopsy rules, Level I anatomy, Tracheo-innominate fistula.
Diabetic Foot	1 Q	Charcot joint presentation, Endocrine crossovers (Glucagonoma rash triad).

- **The Biopsy Hierarchy (Crucial Exam Trap):** Never select "excisional/incisional biopsy" as the initial step for a parotid mass or a suspicious lateral cervical node in an adult/smoker. **FNA is always the mandated first step** to preserve surgical and lymphatic planes (preventing SCC or salivary tumor seeding).
- **Thyroid Algorithmic Decision Points:** FNA cannot differentiate a benign follicular adenoma from a malignant follicular carcinoma. The absolute exam-tested next step for Bethesda 4 (Follicular Neoplasm) is a **diagnostic hemithyroidectomy (lobectomy)**.
- **Post-Operative Neck Emergencies:** Neck hematoma is universally tested as the most lethal immediate complication post-thyroidectomy/parathyroidectomy, causing rapid airway loss and requiring emergent bedside wound opening.
- **Adrenal Perioperative Safety:** The preoperative preparation for pheochromocytoma is a heavy favorite: **Alpha-blockade (phenoxybenzamine) must* strictly *precede beta-blockade** to prevent massive unopposed vasoconstriction and heart failure.
- **Strict Surgical Criteria:** Memorize the NIH guidelines for asymptomatic primary hyperparathyroidism. Age strictly **< 50**, Serum Ca >1.0 mg/dL above normal, T-score ≤ -2.5, or renal impairment are the absolute triggers for surgery.
- **Diagnostic Lab Profiles:** Be prepared to differentiate primary hyperaldosteronism (Conn's) characterized by hypernatremia, hypokalemia, and crucially **low/suppressed renin**, from secondary causes (high renin).
- **MEN Syndromes & Tumor Markers:** Rapidly distinguish MEN-1 (3 Ps) from MEN-2A/2B (Pheochromocytoma + Medullary Thyroid Cancer). Remember that Medullary cancers require a prophylactic central neck node dissection and secrete calcitonin.

- **"Rules of Thumb" to Memorize:** The Salivary **"Rule of 80s"** (Parotid is 80% benign, minor glands are 80% malignant) and the Pheochromocytoma **"Rule of 10s"** (though remember pediatric cases specifically break this rule, with 25% being bilateral/extra-adrenal).

Disorders of parathyroid glands

Core Concepts

Anatomy & Embryology

- **Structure:** Typically 4 glands located behind the thyroid, embedded between the posterior border of the thyroid and its fibrous capsule. Normal dimensions: 6 x 4 x 2 mm; weight: 25–40 mg each.
- **Variation:** Normal function is maintained with at least 2 glands. Up to 15-20% of glands may be ectopic (e.g., intrathyroidal, deep neck, or mediastinal); total number can vary from 4 to 6.
- **Embryology:**
  - **Superior parathyroid glands:** Develop from the **4th branchial pouch**. Located dorsal to the recurrent laryngeal nerve (RLN) at the level of the cricoid cartilage.
  - **Inferior parathyroid glands:** Develop from the **3rd branchial pouch**. Located ventral to the RLN.
- **Vascular Supply:** Primarily from the **inferior thyroid artery**. (The superior thyroid artery supplies ~20% of the upper glands).
- **Venous Drainage:** Ipsilaterally via the superior, middle, and inferior thyroid veins.
- **Histology:** Composed of **chief cells** and **oxyphil cells** within an adipose stroma. Both synthesize parathyroid hormone (PTH). Oxyphil cells derive from chief cells and increase with age. *Note: They do not contain follicular or C cells (which are thyroid).*

Physiology of Calcium & PTH

- **Plasma Calcium Distribution:** Total plasma calcium (~9.5 mg/dL) is divided into:
  - **50% Free ionized (biologically active):** Strictly regulated by PTH and active Vitamin D.
  - **40% Protein-bound:** 80% to albumin, 20% to globulins.
  - **10% Complexed:** With small anions (citrate, phosphate).
- **Corrected Calcium Formula:** Corrected Ca = Measured Ca (mg/dL) + 0.8 * (4.0 - Albumin g/dL). This differentiates true hypercalcemia from factitious hypercalcemia due to hyperalbuminemia.
- **PTH Synthesis & Secretion:**
  - Synthesized as preproparathyroid hormone → proparathyroid hormone → active PTH (84 amino acids).
  - Extremely short **half-life of 2 to 4 minutes** (crucial for intraoperative monitoring).
  - Metabolized in the liver into an active N-terminal and inactive C-terminal fraction.
- **Mechanism of Action:** Calcium-sensing receptors (CASR) on parathyroid cells detect low serum ionized calcium, triggering PTH release, which increases serum calcium via three targets:
  1. **Bone:** Increases bone resorption (demineralization).

2. **Kidney:** Enhances calcium reabsorption, decreases phosphate reabsorption (causing phosphaturia), and stimulates 1-α-hydroxylase.
3. **GI Tract (Indirect):** 1-α-hydroxylase converts Vitamin D to its active form (1,25-dihydroxyvitamin D3), which increases intestinal absorption of both calcium and phosphate.

Diagnosis / Clinical Features

Primary Hyperparathyroidism (1º HPT)

- **Pathology:** Autonomous overproduction of PTH independent of calcium levels. Accounts for 90% of hypercalcemia cases alongside malignancy.
  - **Single Adenoma:** 80–85% of cases (Most common cause).
  - **Hyperplasia:** 10–15% of cases (All 4 glands enlarged).
  - **Carcinoma:** ~1% of cases.
- **Epidemiology:** Occurs in 0.1–0.3% of the population; highly female predominant (1:500 women vs 1:2000 men).
- **Clinical Presentation:** Often an incidental finding of hypercalcemia. However, detailed history reveals symptoms in ~70%. Classically summarized as:
  - **Stones:** Nephrolithiasis (calcium phosphate and oxalate stones). Polyuria (nephrogenic DI secondary to hypercalcemia, *not* oliguria) and polydipsia.
  - **Bones:** Bone pain, osteopenia, osteoporosis, and **osteitis fibrosa cystica** (advanced disease marked by "brown tumors" or osteoclastomas).
  - **Groans:** Abdominal pain, severe peptic ulcer disease (PUD), pancreatitis, constipation.
  - **Moans (Psychic):** Depression, anxiety, florid psychosis, obtundation, coma.
  - **Fatigue Overtones:** Generalized severe fatigue and muscle weakness.

Secondary vs. Tertiary Hyperparathyroidism

- **Secondary HPT:** An *appropriate* physiological response to chronic hypocalcemia.
  - **Cause:** Most commonly Chronic Renal Failure (loss of renal tissue leads to decreased 1-α-hydroxylase, low 1,25-Vit D, and decreased GI calcium absorption).
  - **Biochem:** Low or low-normal calcium, High PTH.
- **Tertiary HPT:** Long-standing secondary HPT leading to *autonomous* parathyroid hyperfunction, typically seen post-renal transplant.
  - **Biochem:** High calcium, High PTH.

Parathyroid Carcinoma

- Aggressive disease with a poor prognosis. 15% present with lymph node metastases, 33% with distant metastases.
- **Intraoperative Findings:** Large, gray-white to gray-brown firm tumor strongly adherent to or invading surrounding neck structures.

Hypoparathyroidism

- **Causes:**
  - **Surgical:** Most common (post-thyroidectomy or neck exploration). Results from inadvertent removal or devascularization.
  - **Idiopathic:** Genetic/autoimmune forms (e.g., MEDAC or HAM syndromes, DiGeorge syndrome).
  - **Functional:** Secondary to severe hypomagnesemia (magnesium is required for both PTH release and peripheral PTH action).
- **Clinical Features:**
  - Neuromuscular hyperactivity: Tetany, perioral paresthesias, muscle cramps, hyperreflexia.
  - Cardiac: Prolonged QT interval, hypotension, resistance to digitalis, refractory heart failure.
  - Other: Posterior lenticular cataracts.

Investigations

Biochemical Evaluation of Primary HPT

- **Serum Calcium:** Elevated (corrected or ionized).
- **Intact PTH:** Elevated (>0.5 mg/L) or inappropriately normal relative to hypercalcemia.
- **Serum Phosphate:** Decreased (due to PTH-induced renal wasting).
- **Serum Chloride:** Elevated (PTH causes a mild hyperchloremic metabolic acidosis).
- **Chloride to Phosphate Ratio (Cl:PO4):** > 33.
- **Alkaline Phosphatase:** Normal or elevated (if increased bone turnover/disease is present).
- **Urine:** Normal or high 24h urinary calcium (>250 mg/24h).
- **Calcium to Creatinine Clearance Ratio:** > 0.02 (Differentiates 1° HPT from Familial Hypocalciuric Hypercalcemia [FHH], which has a ratio < 0.01).

Pre-Operative Imaging (Localization)

- **Technetium-99m Sestamibi Scan:** The most specific and sensitive (85-95%) functional imaging modality for localizing a parathyroid adenoma. The radiotracer concentrates in mitochondria-rich tissue; parathyroid adenomas exhibit **delayed washout** compared to normal thyroid tissue.
- **Sestamibi-SPECT (Single-Photon Emission CT):** Excellent for deep neck or ectopic (mediastinal) glands.
- **High-Resolution Ultrasound:** 65-85% sensitivity. Good adjunct, but user-dependent and limited by short thick necks, multinodular goiters, or ectopic mediastinal glands.

Management

Primary Hyperparathyroidism

- **Definitive Therapy:** Surgical parathyroidectomy. (Medical observation is reserved for asymptomatic patients with mildly elevated calcium, older age, or extensive comorbidities).

Secondary Hyperparathyroidism

- Primarily treated medically (address the underlying cause, vitamin D analogues, phosphate binders).

- **Indications for Surgery in 2° HPT:** Intractable bone pain, severe pruritus, Calcium-Phosphate product ≥ 70, Serum Ca > 11 mg/dL with markedly high PTH, calciphylaxis, or severe soft-tissue calcification.

Tertiary Hyperparathyroidism

- Operative intervention is required if symptomatic disease or if autonomous PTH secretion persists >1 year after successful renal transplant. Treatment is subtotal parathyroidectomy or total parathyroidectomy with autotransplantation.

Hypoparathyroidism Emergency Management

- **Acute Tetany/Hypocalcemia:** Immediate IV Calcium supplementation until stable, followed by oral calcium and active Vitamin D analogues.
- **Chronic:** Oral calcium, Vitamin D, and dietary phosphate restriction.

Relevant Guidelines

Surgical Indications for Asymptomatic Primary HPT (NIH Guidelines) Surgery is indicated if any of the following criteria are met:

- **Serum Calcium:** > 1.0 mg/dL above the upper limit of normal (Often operationalized as > 11.5 mg/dL depending on lab normal values; *Note: an isolated finding of 11.1 mg/dL does not strictly meet the >1.0 threshold if ULN is 10.5*).
- **Renal Impairment:** Decreased creatinine clearance.
- **Renal Stones:** Presence of nephrolithiasis.
- **Bone Density:** Markedly reduced cortical or cancellous bone density (T-score ≤ -2.5 at any site) or history of fragility fracture.
- **Age:** < 50 years old.

WHO Diagnostic Criteria for Osteoporosis (DEXA Scan Results)

DIAGNOSIS	T-SCORE RANGE	DESCRIPTION
Normal	≥ -1.0	Within 1 SD of mean for young-adult reference
Osteopenia	-2.5 < T-Score < -1.0	Between 1.0 and 2.5 SD below mean
Osteoporosis	≤ -2.5	2.5 SD or more below mean
Severe Osteoporosis	≤ -2.5 with fracture	2.5 SD or more below mean + fragility fracture

Operative / Procedural Notes

Surgical Approaches

- **Minimally Invasive Parathyroidectomy (MIPS):** Standard of care for a pre-operatively localized single adenoma (e.g., positive Sestamibi scan). Involves targeted excision of the single diseased gland without exploring the others.

- **Bilateral Neck Exploration (BNE):** Traditional method (collar incision). Performed if imaging is negative/discordant, or if multi-gland disease (hyperplasia) is suspected.
- **Management of Hyperplasia:**
  1. Subtotal parathyroidectomy (remove 3.5 glands, leaving ~50 mg of tissue) + bilateral upper cervical thymectomy (removes supernumerary glands common in 20% of patients).
  2. Total parathyroidectomy with autotransplantation (12-14 pieces implanted into the brachioradialis muscle belly).
- **Parathyroid Carcinoma Surgery:** Requires en bloc excision of the tumor along with the ipsilateral thyroid lobe. Modified radical neck dissection is required if lymph node metastases are present.

Intraoperative PTH Monitoring (ioPTH)

- Capitalizes on the short half-life of PTH (2-4 minutes).
- **Positive Test (Cure):** A drop in PTH of **≥ 50% at 10 minutes** post-excision compared to the highest pre-excision baseline. If levels do not drop by 50%, multi-gland disease or a second adenoma is suspected, prompting BNE.

Complications / Prognosis

- **Hypocalcemia:** Most common acute post-operative complication. Presents with perioral paresthesia, muscle cramps, and positive Chvostek's (facial twitch) and Trousseau's (carpopedal spasm) signs. Treated immediately with calcium and vitamin D.
- **Recurrent Laryngeal Nerve Injury:** Results in vocal cord paralysis, causing hoarseness (unilateral) or airway obstruction (bilateral).
- **Bone Hunger Syndrome:** Acute, severe, prolonged hypocalcemia after parathyroidectomy due to massive, rapid uptake of calcium and phosphate by demineralized bone.

Past-Paper High Yield

- **MEN Syndromes (Heavy Exam Focus):**
  - **MEN 1 (Wermer Syndrome):** Classically the 3 Ps: **P**arathyroid hyperplasia/tumors, **P**ituitary adenoma, **P**ancreatic neuroendocrine tumors (e.g., Gastrinoma/Zollinger-Ellison). Also features facial angiofibromas.
  - **MEN 2A / 2B:** Features **Pheochromocytoma** and Medullary Thyroid Carcinoma. *Do not select Pheochromocytoma as a feature of MEN 1.*
  - A patient with severe PUD refractory to PPIs (Gastrinoma) + Hypercalcemia (Primary HPT) is the classic board presentation for MEN 1.
- **Diagnostic Patterns of Hypercalcemia:**
  - *Primary HPT:* High Ca, High PTH, Low PO4. (Single adenoma is the most common cause by far).
  - *Secondary HPT:* Low/normal Ca, High PTH. It is a normal physiologic response to hypocalcemia (e.g., CKD, Vit D deficiency).
  - *Ectopic/Malignancy:* High Ca, Low PTH.
- **Hypercalcemia Manifestations:** Notably causes **polyuria** (due to nephrogenic diabetes insipidus secondary to impaired ADH action at the kidney), *not* oliguria.

- **Surgical Indications for Asymptomatic 1º HPT:** Memorize the strict criteria. Age strictly **< 50**, Serum Ca >1.0 mg/dL above normal, documented decreased bone density, or renal impairment. Being "> 60 years old" is *not* an indication for surgery in an asymptomatic patient.
- **Targeted Surgery:** A patient with confirmed primary HPT and a localized Sestamibi scan requires excision of the *single adenoma only*. Unilateral or bilateral thyroidectomy is incorrect.
- **Calcitonin & Medullary Thyroid Carcinoma:** Medullary thyroid cancer secretes calcitonin which *lowers* serum calcium. It does NOT cause hypercalcemia.
- **Neuroendocrine Tumor Markers:** Elevated C-peptide is uniquely useful for diagnosing an **Insulinoma** (secretes equimolar amounts of insulin and C-peptide), distinguishing it from exogenous insulin abuse and from other pancreatic tumors (Gastrinoma, VIPoma).

Memory Pearls

- **Signs of Hypercalcemia:** *"Kidney stones, painful bones, abdominal groans, psychic moans, and fatigue overtones."*
- **Embryology Flip: S**uperior = 4th pouch; **I**nferior = 3rd pouch.
- **PTH Half-Life:** 2-4 minutes (the reason 10-minute intra-op PTH labs work flawlessly).
- **MEN Syndromes Rule of Thumb:**
  - MEN 1 = **3 Ps** (Parathyroid, Pituitary, Pancreas)
  - MEN 2 = **2 Ps + 1 M** (Pheo, Parathyroid, Medullary thyroid cancer)
  - MEN 2B = **1 P + 2 Ms** (Pheo, Medullary, Marfanoid habitus/Mucosal neuromas).

Adrenal gland pathology

Core Concepts

- **Embryology:** The adrenal gland consists of two distinct embryonic origins:
  - **Cortex (80%):** Derived from mesoderm. Regulated by the anterior pituitary (ACTH). Responsible for long-term stress adaptation.
  - **Medulla (20%):** Derived from neuroectoderm. Regulated by the sympathetic nervous system. Responsible for acute "fight or flight" responses.
- **Functional Zonal Anatomy (Cortex & Medulla):**
  - **Zona Glomerulosa (Outer Cortex):** Secretes mineralocorticoids (**Aldosterone**) to regulate intravascular volume and sodium retention.
  - **Zona Fasciculata (Middle Cortex):** Secretes glucocorticoids (**Cortisol**) to regulate glucose mobilization and suppression of the immune system.
  - **Zona Reticularis (Inner Cortex):** Secretes sex steroids (**Androgens**). Overproduction in children can cause precocious puberty or ambiguous genitalia.
  - **Medulla:** Secretes catecholamines (**Epinephrine & Norepinephrine**).
- **Anatomical Distinctions:**
  - **Right Adrenal Gland:** Triangular/pyramidal shaped. Sits superiorly and does *not* reach the renal hilum.

- **Left Adrenal Gland:** Crescentic/semilunar shaped. Extends lower and reaches the left renal hilum.

- **Metabolic Response to Stress:** Contains an initial brief "Ebb" phase (hypoperfusion/shock) followed by a hypermetabolic "Flow" phase. Prolonged stress leads to a persistent flow phase, causing severe metabolic imbalance, catabolism, and muscle wasting.

Diagnosis / Clinical Features

- **Primary Hyperaldosteronism (Conn's Syndrome):**
  - Characterized by autonomous aldosterone hypersecretion.
  - **Clinical Presentation:** Often asymptomatic, or presents with frontal headache, hypertension, polyuria, polydipsia, and muscle weakness/flaccid paralysis (due to low potassium).
- **Cushing's Syndrome (Hypercortisolism):**
  - **Causes:** Endogenous (85% ACTH-dependent e.g., Cushing's disease, ectopic ACTH from small cell lung cancer; 15% ACTH-independent e.g., adrenal adenoma/carcinoma) or Exogenous (glucocorticoid use).
  - **Clinical Presentation:** Moon facies, central obesity, buffalo hump, hypertension, secondary diabetes mellitus, purple striae, thinning of skin, and proximal muscle weakness.
  - **Important Distinction:** *Hypokalemia is uncharacteristic* in Cushing's caused by an adrenal adenoma. Profound hypokalemia in Cushing's suggests ectopic ACTH production (extreme cortisol levels cross-reacting with mineralocorticoid receptors).
- **Pheochromocytoma:**
  - Tumors of the adrenal medulla causing massive alpha-adrenergic stimulation.
  - **Classic Triad:** Headache, tachycardia/palpitations, and diaphoresis. Associated with extreme hypertension.
  - **Orthostatic Hypotension Mechanism:** Chronic intense vasoconstriction shrinks the intravascular volume space. Upon standing, decreased venous return occurs, leading to a drop in cardiac output and orthostatic hypotension.
- **Adrenocortical Carcinoma (ACC):**
  - Mean age: 50 years. Usually large (>5 cm; mean weight 900g).
  - **Gross characteristics:** Hemorrhage, necrosis, and invasion of the adrenal vein.
- **Addison's Disease (Primary Adrenal Insufficiency):**
  - Lack of cortisol and aldosterone leads to severe sodium wasting and fluid depletion.
  - Presents with profound **dehydration** (not generalized edema), extreme hypotension, weakness, fever, stress intolerance, and hyperpigmentation (due to elevated ACTH cross-reactivity).
- **Insulinoma:**
  - Profound hypoglycemia triggers a massive release of counter-regulatory hormones, primarily catecholamines.
  - The catecholamine surge directly causes the acute adrenergic symptoms: anxiety, tremor, tachycardia, and palpitations.

Investigations

- **Adrenal Adenoma Imaging Characteristics:**
  - **CT Scan (Non-contrast):** Typical density <10 Hounsfield Units (HU) due to being lipid-rich. Values >30 HU are suspicious for malignancy or pheochromocytoma.
  - **Morphology:** Small (<4 cm), sharp margins, smooth, and homogenous.
  - **Contrast Washout:** Demonstrates rapid washout (>50-60% at 15 minutes).
- **Conn's Syndrome Workup:**
  - **Labs:** Hypokalemia, mild hypernatremia, metabolic alkalosis. High aldosterone, but crucially, **low (suppressed) plasma renin**.
  - **Sodium Loading Test:** Administration of 2L IV normal saline normally suppresses aldosterone. In Conn's syndrome, aldosterone remains high.
  - **Localization:** Adrenal Venous Sampling (AVS) is the gold standard next step if imaging shows bilateral nodules, to differentiate unilateral adenoma from bilateral hyperplasia.
- **Cushing's Syndrome Workup:**
  - **Screening:** 24-hour urinary free cortisol (integrates diurnal variations).
  - **Suppression:** Dexamethasone suppression test (normal subjects suppress cortisol to <50 nmol/L).
- **Pheochromocytoma Workup:**
  - **Biochemical Tests:** Plasma free metanephrines (highly sensitive, 99%), 24-hour urinary catecholamines (diagnostic if 2× normal), and Vanillylmandelic acid (VMA).
  - **Clonidine Suppression Test:** Differentiates essential HTN from pheochromocytoma. A >50% reduction in catecholamines 3 hours post-clonidine rules out pheochromocytoma.
  - **Imaging:** MIBG (metaiodobenzylguanidine) scan localizes adrenergic tissue (highly specific for pheochromocytomas/paragangliomas).

Management

- **Conn's Syndrome:**
  - **Unilateral Adenoma:** Surgical excision (unilateral adrenalectomy).
  - **Bilateral Adrenal Hyperplasia:** Medical management with aldosterone antagonists (**Spironolactone**). Bilateral adrenalectomy is avoided as it causes permanent adrenal insufficiency.
- **Pheochromocytoma (Pre-operative Management):**
  - **Alpha-blockade FIRST:** Phenoxybenzamine is used early to control hypertension.
  - **Beta-blockade SECOND:** Never administer beta-blockers before alpha-blockers; unopposed alpha stimulation will cause massive vasoconstriction, bradycardia, reduced cardiac output, and acute heart failure.
  - **Volume Expansion:** IV hydration is critical to reverse chronic volume contraction prior to surgery.
- **Adrenocortical Carcinoma:**

- Open surgical approach is the gold standard for masses suspected to be ACC.
- Medical therapy (Mitotane + Chemotherapy) to suppress metastatic cortisol-secreting tumors.

Relevant Guidelines

- **Management of the Adrenal Incidentaloma (Slide-Derived Algorithm):**
  - **Definition:** Accidental discovery of an adrenal mass >1 cm during imaging for unrelated reasons.
  - **Initial Workup:** Hormonal testing (1mg overnight dexamethasone suppression, fractionated metanephrines) + Imaging phenotype (Unenhanced CT).
  - **Indications for Surgery:**
    - Hormonally active masses.
    - Size >5 cm (or ≥4 cm depending on algorithm branch).
    - Suspicious imaging features (Unenhanced CT >10 HU, washout <50% @ 10 min, growth ≥1 cm during surveillance).
  - **Indications for Observation:**
    - Small (<3 cm), non-functioning, lipid-rich/benign imaging phenotype.
  - **Surveillance for observed masses:** Repeat imaging at 6, 12, and 24 months. Repeat hormonal testing annually for 4 years. (New recommendations suggest if mass is stable at 3 months and 1 year, no further workup is needed).

Operative / Procedural Notes

- **Adrenal Vasculature (Surgical Relevance):**
  - **Arterial Supply:** Abundant (7 cc/gram/minute) from the Inferior phrenic artery, Aorta, and Renal artery.
  - **Venous Drainage:** The most important surgical structure.
    - **Right Adrenal Vein:** Very short and drains directly into the posterior IVC. Highly susceptible to avulsion during right adrenalectomy, which can cause catastrophic hemorrhage (accounts for 25% of venous return to IVC at that level).
    - **Left Adrenal Vein:** Longer and drains inferiorly into the left renal vein.
- **Surgical Approaches:**
  - **Laparoscopic Posterior Retroperitoneal:**
    - *Position:* Semi-jackknife prone.
    - *Pros:* Avoids prior abdominal adhesions; allows bilateral adrenalectomy without repositioning the patient.
    - *Cons:* Precludes intra-abdominal exploration; limited working space restricts use to tumors <5 cm.
  - **Laparoscopic Lateral Transperitoneal (Flank):**
    - *Pros:* Much wider exposure; allows inspection of the abdominal cavity and removal of larger glands.

- **Conventional Open Flank (Lumbodorsal):** Requires 11th/12th rib resection. Extra-peritoneal. Risk of pleural entry/lung injury.
- **Open Anterior Subcostal:** Allows bilateral exploration but carries a higher rate of ileus; difficult in obese patients.
- **Open Thoracoabdominal:** Excellent exposure for great vessels and large tumors/ACC, but carries highest morbidity (prolonged ileus, chest tubes, pulmonary complications).

Complications / Prognosis

- **Adrenocortical Carcinoma:** Poor prognosis, 5-year survival is 20-30%. Most tumor-related deaths occur within two years of diagnosis.
- **Bilateral Adrenalectomy:** Carries an immense risk of Addisonian crisis due to total loss of endogenous cortisol, aldosterone, and adrenaline adaptation to stress. Lifelong steroid replacement is mandatory.

Past-Paper High Yield

- **Primary Hyperaldosteronism Lab Profile:** Features hypertension, hypernatremia, and hypokalemia. **Plasma renin is low** due to negative feedback. Secondary hyperaldosteronism will have high renin.
- **Adrenal Mass Imaging Differentiator:** Benign adenomas are lipid-rich, yielding **<10 HU** on non-contrast CT. Density >30 HU points towards malignancy or pheochromocytoma.
- **Cushing's Syndrome & Potassium:** Marked hypokalemia is *not* characteristic of an adrenal adenoma causing Cushing's. It strongly suggests ectopic ACTH production.
- **Pheochromocytoma Pre-op Protocol:** Beta-blockers must *never* be given before alpha-blockers.
- **Insulinoma vs C-Peptide:** Endogenous hyperinsulinemia from an insulinoma produces **high C-peptide** levels (co-secreted with insulin), distinguishing it from exogenous insulin administration. Symptoms are adrenergically mediated due to the profound physiologic catecholamine release.
- **Addison's Disease Presentation:** Patients present with hypotension and severe dehydration, *not* generalized edema.
- **MIBG Scan:** Highly specific for localizing pheochromocytomas and paragangliomas. No role in imaging aldosterone or cortisol-secreting tumors.
- **Bilateral Masses in Primary Hyperaldosteronism: Adrenal Venous Sampling (AVS)** is the gold standard next step to lateralize the source of aldosterone before surgery. Do not resect the larger nodule without biochemical lateralization.
- **Pediatric Pheochromocytomas:** Defy the classical "Rule of 10s". In children, the incidence of bilateral, familial, and extra-adrenal pheochromocytoma is approximately **25%**.
- **Extra-Adrenal Pheochromocytoma:** The most common location is in the abdomen, specifically near the aortic bifurcation (Organ of Zuckerkandl).
- **Carcinoid Tumors:** The syndrome is associated with high 5-HIAA and is most commonly caused by midgut tumors (e.g., ileal) that have metastasized to the liver (bronchial origin is an exception/distractor).

Memory Pearls

- **Pheochromocytoma Rule of 10s (in adults):** 10% Bilateral, 10% Familial, 10% Pediatric, 10% Malignant, 10% Normotensive, 10% Extra-adrenal, 10% Multiple. *(Remember: Children break this rule—25% are bilateral, extra-adrenal, or multiple)*.
- **Whipple's Triad (Insulinoma):** 1) Hypoglycemic symptoms during fasting/exercise, 2) Documented low blood glucose, 3) Symptoms resolve upon glucose administration.
- **Adrenal Cortex Zonal Function:** "GFR = Salt, Sugar, Sex" (Glomerulosa = Mineralocorticoids, Fasciculata = Glucocorticoids, Reticularis = Androgens).

Salivary glands tumors

Core Concepts

- **Anatomy of Major Salivary Glands:**
  - **Parotid Gland:** Produces thinner, serous saliva. Drains via **Stensen's duct** into the oral cavity opposite the 2nd upper molar tooth. The facial nerve passes directly through it.
  - **Submandibular Gland:** Produces thicker, mucinous saliva. Drains via **Wharton's duct** (long, tortuous, travels uphill against gravity) to open lateral to the lingual frenulum.
  - **Sublingual Gland:** Produces thicker, mucinous saliva. Drains primarily via the ducts of Rivinus; the major duct (Bartholin's duct) opens into or with Wharton's duct.
- **Minor Salivary Glands:**
  - Located in the soft palate, hard palate, gingiva, lips, and all oral mucosa *except* the dorsum (upper surface) of the tongue.
  - Lack a defined major duct; drain directly via multiple tiny ducts.
  - Secretion is dried up by anticholinergics (e.g., atropine) and highly susceptible to radiation damage.
- **Physiology:** Saliva production is 1000–1500 mL/day. Contains amylase to aid carbohydrate digestion.
- **The "Rule of 80s" (and related gland-size rules):**
  - 70–80% of all salivary tumors occur in the parotid gland.
  - 70–80% of parotid tumors are benign.
  - 80% of benign parotid tumors are pleomorphic adenomas.
  - *Clinical Maxim:* The smaller the affected salivary gland, the higher the risk of malignancy. Malignancy risk: Parotid (~20%), Submandibular (~40%), Minor Salivary Glands (~80%).

Diagnosis / Clinical Features

Non-Neoplastic Disorders

- **Acute Sialadenitis:**
  - *Viral (Mumps):* Most common acute cause. Self-limiting, common in children. Diffuse, bilateral inflammation. Complications: pancreatitis, orchitis (leading to infertility), oophoritis (rare).

- *Bacterial:* Caused by ascending infection (typically *Staphylococcus aureus*). Often seen in elderly, ICU/post-op patients with poor oral hygiene and dry mouth. Can complicate into an abscess.

- **Chronic Sialadenitis:**
  - *Autoimmune (Sjögren's Syndrome):* Most common chronic cause. Usually women aged 35–45. Causes destruction of salivary and lacrimal glands (dry mouth, dry eyes). 60% associated with SLE, RA, or scleroderma.
- **Sialolithiasis (Salivary Stones):**
  - Most common in the submandibular gland due to thick, mucinous secretions and drainage against gravity via Wharton's duct.
  - *Classic Presentation:* Recurrent submandibular swelling and pain that drastically worsens specifically upon eating (due to increased salivation against an obstructed duct), followed by slow post-prandial resolution.
  - Intermittent obstruction leads to chronic sialadenitis, acinar atrophy, and microabscesses.
- **Plunging Ranula:** A mucous extravasation cyst arising specifically from the sublingual gland that dissects deep below the mylohyoid muscle to present as a swelling in the neck.

Benign Tumors

- **Pleomorphic Adenoma (Mixed Tumor):**
  - Most common salivary tumor overall and most common benign tumor in the parotid. Peak age: 5th decade.
  - Histology: Proliferation of epithelial, myoepithelial, and stromal tissue (cartilage/bone).
  - Presentation: Solitary, painless, firm, slowly growing, mobile mass. Deep lobe tumors may present as an intraoral pharyngeal mass.
- **Warthin's Tumor (Papillary Cystadenoma Lymphomatosum):**
  - Second most common benign parotid tumor.
  - *Exclusive* to the parotid gland (never found in other salivary glands).
  - Strongly associated with older males (90%) and smokers.
  - Often bilateral or multicentric (10%). Cystic and may be fluctuant.
- **Hemangioma:** Most common benign salivary tumor in children/infants. Compressible mass.

Malignant Tumors

- **Red Flag Signs:** Rapid growth, facial nerve palsy (8–26%), pain (12–24%), fixed to masseter muscle (17%), skin ulceration (9%), and formication (paresthesia described as ants crawling on the skin).
- **Mucoepidermoid Carcinoma:**
  - Most common malignant salivary tumor overall (and most common in the parotid). 2nd most common site is the palate.
  - High-grade variants have a high rate of lymph node metastasis.
- **Adenoid Cystic Carcinoma:**

- Most common malignant tumor of the *submandibular* and *minor salivary glands* (2nd most common overall).
- Notorious for perineural invasion (insidious growth) and distant metastasis (especially to the lungs). Rarely involves lymph nodes.

- **Acinic Cell Carcinoma:** 2nd most common parotid malignancy and most common pediatric salivary malignancy. Favorable prognosis.

Investigations

- **Fine Needle Aspiration (FNA) Biopsy:** The best and most critical next step for tissue diagnosis, especially in masses complicated by facial nerve palsy. Incisional biopsy is absolutely contraindicated due to the risk of tumor seeding and nerve injury.
- **CT / MRI:** Gold standard imaging to evaluate tumor extension (e.g., assessing deep lobe involvement or skull base invasion). CT can show well-circumscribed hyperdense masses (pleomorphic adenoma) or hypodense fluid-filled lesions (Warthin's).
- **Sialography:** Used in diffuse swelling to rule out sialadenitis. May reveal a "chain of lakes" or "ball & socket" appearance indicative of chronic sialadenitis and microabscesses due to stones.

Management

- **Pleomorphic Adenoma:** Treat with superficial parotidectomy or extracapsular excision with clear safety margins (facial nerve preserved). *Do not* enucleate due to high recurrence risk. Multi-nodular recurrences may require radiotherapy.
- **Warthin's Tumor:** Often requires no surgery if definitively diagnosed, or simple excision.
- **Malignant Parotid Tumors:** Total parotidectomy. If a facial nerve branch is directly involved, it must be sacrificed/excised.
- **Submandibular Tumors:** Total excision of the gland. Must preserve the marginal mandibular (branch of VII), hypoglossal (XII), and lingual nerves unless directly invaded by malignancy.
- **Minor Salivary Gland Tumors:** Wide local excision with safety margins, sometimes requiring adjacent bone resection (e.g., hard palate).
- **Submandibular Sialolithiasis:**
  - *Distal stones* (e.g., 1 cm away from the Wharton's duct opening): Managed by simple intra-oral removal via incision directly over the duct (sialodochotomy).
  - *Proximal stones / chronically destroyed gland:* Requires total submandibular gland excision.
- **Radiotherapy:** External beam radiotherapy is effective for aggressive malignancies or severe recurrences. Chemotherapy is generally not effective.

Relevant Guidelines

- **Risk of Malignancy by Location Framework:**
  - Parotid gland swelling: 80% chance of being a benign neoplasm (20% malignant).
  - Submandibular gland mass: 40% chance of being malignant.
  - Minor salivary gland mass: 60-80% chance of being malignant.

- **Neck Dissection Indications:**
  - Therapeutic: Indicated for any clinically positive lymph nodes.
  - Prophylactic: Justified in high-grade mucoepidermoid carcinoma, squamous cell carcinoma, or adenocarcinoma.

Operative / Procedural Notes

- **Facial Nerve Identification Landmarks (Parotidectomy):**
  - **Tragal Pointer:** The cartilaginous external auditory canal triangular process points directly toward the main trunk.
  - **Posterior Belly of Digastric Muscle:** The nerve lies superior and superficial to it.
  - **Stylomastoid Foramen:** The anatomical exit of the main trunk of the facial nerve.
- **Facial Nerve Branches:** Splits at the pes anserinus into the upper division (Temporofacial: temporal, zygomatic, buccal) and lower division (Cervicofacial: mandibular, cervical). Traumatic injury to the parotid can cause a LMN facial palsy depending on the branch injured.
- **Pediatric Hemangiomas:** Treatment includes steroids, embolization, or superficial laser therapy. Surgery poses a high bleeding risk. Spontaneous regression may occur.

Complications / Prognosis

- **Malignant Transformation (Carcinoma ex pleomorphic adenoma):** Pleomorphic adenomas have a 2–10% risk of malignant transformation (usually to adenocarcinoma) if left untreated for years. Risk increases with advancing age and prolonged duration.
- **Warthin's Malignant Potential:** Exceptionally rare (virtually 0%, strictly <1% malignancy rate).
- **Acinic Cell Carcinoma Survival:** Good prognosis (5-year survival 85%, 25-year survival 50%).

Past-Paper High Yield

- **Most common parotid tumor:** Pleomorphic adenoma (Choice 5/Benign). Mucoepidermoid carcinoma is the most common malignant salivary tumor overall.
- **Most common non-parotid malignancy:** Adenoid cystic carcinoma is the most common malignant tumor of the submandibular and minor salivary glands.
- **Warthin's tumor features:** Occurs *exclusively* in the parotid, strongly related to smoking and older males, 10% bilateral. *High yield distractor: It does NOT transform into a malignant tumor.*
- **Parotid mass + Facial nerve palsy:** This is a highly ominous sign indicating malignant infiltration. The absolute best next step is an **FNA biopsy** to obtain tissue diagnosis prior to surgery. Incisional biopsy is wrong.
- **Submandibular stone presentation:** A patient presenting with "recurrent submandibular swelling upon eating" is the classic textbook description of sialolithiasis.
- **Distal submandibular stone management:** A submandibular stone located 1 cm from the duct opening in the floor of the mouth is treated with **intra-oral removal** (not shock wave lithotripsy or gland excision).
- **Sublingual gland concepts:** It is a paired gland, drains via Wharton's duct (and Rivinus), and is the origin of a plunging ranula (a mucous extravasation cyst that reaches the neck).

- **Minor Salivary Glands:** High malignant potential (~80%), lack a defined major duct, affected by anticholinergics (atropine), and severely damaged by radiation therapy.
- *Mapped Endocrine Misc Concepts (often tested in the same block):*
  - **Carotid Body Tumors (Paragangliomas):** Innervated by CN IX and X, stimulated by hypoxia/acidosis, classically splay the carotid bifurcation (lyre sign) on angiography. Typically benign; the malignancy rate is **<10%** (not 35%).
  - **Insulinoma:** The most common functional neuroendocrine tumor of the pancreas overall.
  - **Gastrinoma:** The most common pancreatic/duodenal neuroendocrine tumor found in patients with MEN-1.

Memory Pearls

- **"Bigger Gland = Benign Disease"**: Parotid (biggest) = 80% benign. Minor glands (smallest) = 80% malignant.
- **"Parotid ONLY"**: Warthin's tumor is exclusive to the parotid.
- **SPACE SPIT**: Physical exam mnemonic for a parotid mass (examine the mass, **S**alivary glands (rest of them), **P**haryngeal bulge, **A**ll facial nerve branches, **C**ervical LNs, **E**xamine duct orifices).
- **"Chain of Lakes"**: Sialogram finding representing alternating dilation and strictures from chronic obstruction (stones/sialadenitis).

Benign and malignant thyroid disorders

Core Concepts

- **Definition:** A thyroid nodule is a discrete lesion radiologically distinct from the surrounding parenchyma. Non-palpable nodules detected on ultrasound are termed "incidentalomas."
- **Prevalence:** Nodules are highly prevalent (up to 76% on high-definition ultrasound), incidence increases linearly with advancing age, and they are significantly more common in females. Over 90% of adult thyroid nodules are benign.
- **Benign Etiologies:**
  - **Multinodular Goiter (MNG):** Most common benign etiology. Most MNGs are euthyroid.
  - **Hashimoto's Thyroiditis:** Autoimmune destruction of the thyroid gland; the **most common cause of hypothyroidism** in iodine-sufficient areas.
  - **Follicular Adenoma:** Benign clonal neoplasm.
- **Malignant Etiologies (Thyroid Carcinomas):**
  - **Papillary Thyroid Carcinoma (PTC):** Most common thyroid cancer (~80-85%). Associated with iodine-sufficient regions and radiation exposure. **Most common pediatric thyroid malignancy**. Classically **multifocal** due to intraglandular lymphatic spread. Features BRAF mutations.
  - **Follicular Thyroid Carcinoma (FTC):** Second most common (~10%). Higher incidence in **dietary iodine-deficient areas**. Classically **unifocal** and disseminates via **hematogenous spread** (commonly to bone and lungs).

- **Hurthle Cell Carcinoma:** An oxyphilic variant of follicular carcinoma. Features abundant **eosinophilic cytoplasm** (due to mitochondria, NOT eosinophils). More aggressive with higher rates of multifocality and nodal spread.
- **Medullary Thyroid Carcinoma (MTC):** Arises from neuroendocrine **C-cells (parafollicular cells)**. Secretes calcitonin. Roughly 75% sporadic; 25% familial (MEN 2A/2B or Familial MTC via RET proto-oncogene mutations). Familial cases tend to be bilateral, multifocal, and preceded by C-cell hyperplasia.
- **Anaplastic Carcinoma:** Highly aggressive, undifferentiated malignancy primarily seen in the elderly. Presents as a massively enlarging, rock-hard mass causing early local invasion (stridor/airway compromise). Uniformly fatal.

- **Anatomy Pearl:** The **inferior thyroid artery** supplies the vast majority of the blood to all four parathyroid glands.

Diagnosis / Clinical Features

- **History & Risk Stratification:**
  - **High-Risk Historical Factors:** History of head/neck irradiation, total body irradiation, exposure to ionizing radiation, familial thyroid carcinoma, male sex, advanced age, rapid growth.
  - **High-Risk Physical Exam:** Vocal cord paralysis (hoarseness), cervical lymphadenopathy, fixation to surrounding tissues (strap muscles).
- **Clinical Syndromes:**
  - **Graves' Disease:** Diffuse toxic goiter, palpitations, thyrotoxicosis. Can cause severe exophthalmos.

Investigations

- **Thyroid Stimulating Hormone (TSH):** Must be the **initial test** in evaluating any thyroid nodule.
  - **Subnormal (Low) TSH:** Evaluate with a **Radionuclide (I-123) Uptake Scan (RAIU)**.
    - "Hot" (functioning) nodule: Extremely low risk of malignancy; do not FNA.
    - "Cold" (non-functioning) nodule: Higher risk of malignancy; proceed to FNA.
  - **Normal or Elevated TSH:** Do not perform a radionuclide scan. Proceed directly to Ultrasound and potential FNA.
- **Ultrasound (US):** The fundamental imaging modality to survey nodules and cervical lymph nodes.
  - **Malignant features:** Solid, marked hypoechogenicity, irregular margins, microcalcifications, taller-than-wide shape, interrupted rim calcification with soft tissue extrusion, increased central vascularity.
  - **Benign features:** Purely cystic or predominantly cystic (spongiform) nodules have a nearly 0% risk of malignancy.
- **Fine Needle Aspiration (FNA) Biopsy:** The most definitive and precise non-operative tool to assess malignancy risk.
  - **Crucial limitation:** FNA *can* diagnose a "follicular neoplasm," but it **cannot** distinguish between a benign follicular adenoma and a malignant follicular carcinoma (requires final histological

evaluation for capsular or vascular invasion).

- **Histopathology Markers:**
  - **Psammoma bodies:** Concentric, laminated calcifications pathognomonic for **Papillary Carcinoma**.
  - **Congo Red Stain:** Used to diagnose **Medullary Carcinoma**; highlights the amyloid stroma (precipitated calcitonin) with apple-green birefringence under polarized light.

Management

- **Benign Nodules:** Usually managed with clinical/US follow-up every 12-18 months. Symptomatic nodules require surgery (lobectomy or total thyroidectomy).
- **Hyperthyroidism / Toxic Nodules:**
  - **Radioactive Iodine (RAI) Ablation:** Used for toxic multinodular goiter or solitary toxic adenomas. Contraindicated in pregnancy. Cancers without iodine uptake (e.g., MTC) and destruction-induced thyrotoxicosis (subacute thyroiditis) are not treated with RAI.
  - **Surgery for Graves' disease:** Total or near-total thyroidectomy is preferred for severe symptoms to definitively eliminate the hyperthyroid state and minimize recurrence. Medical management must precede surgery for severe thyrotoxicosis to prevent thyroid storm.
  - **Solitary Toxic Adenoma:** Right or left hemithyroidectomy (lobectomy) is the definitive surgical management.
- **Surgical Management by Malignancy Risk:**
  - **Bethesda 3 (AUS/FLUS) or Bethesda 4 (Follicular Neoplasm):** Standard of care is a diagnostic **hemithyroidectomy (lobectomy)**.
  - **Medullary Thyroid Carcinoma:** Requires total thyroidectomy plus a **routine prophylactic central compartment (Level VI) lymph node dissection** universally, due to early microscopic lymphatic spread. (No RAI is given).
  - **Anaplastic Carcinoma:** Palliative care or wide local debulking; resistant to standard therapies.

Relevant Guidelines

- **ATA Management Guidelines (2015/2016):**
  - **Initial TSH (Rec A):** TSH must be measured first.
  - **Low TSH (Rec B):** Proceed to radionuclide scan.
  - **Normal/Elevated TSH (Rec C):** Radionuclide scan is NOT indicated.
  - **Ultrasound (Rec 6):** Required for all known/suspected nodules.
- **ATA Ultrasound Risk Stratification & FNA Size Cutoffs:**
  - **High Suspicion (>70-90% risk):** Solid hypoechoic + suspicious features. **FNA if ≥ 1 cm.**
  - **Intermediate Suspicion (10-20% risk):** Hypoechoic solid, smooth margins. **FNA if ≥ 1 cm.**
  - **Low Suspicion (5-10% risk):** Iso/hyperechoic solid. **FNA if ≥ 1.5 cm.**
  - **Very Low Suspicion (<3% risk):** Spongiform. **FNA if ≥ 2 cm.**
  - **Benign (<1% risk):** Pure cysts. **No FNA.**

- *Note:* FNA is generally not indicated for subcentimeter (<10 mm) nodules unless high-risk factors exist.

- **Bethesda System for Reporting Thyroid Cytopathology:**

CLASS	DIAGNOSTIC CATEGORY	MALIGNANCY RISK	ACTION
I	Nondiagnostic	1 - 4%	Repeat FNA
II	Benign	0 - 3%	Clinical/US follow-up
III	AUS / FLUS	5 - 15%	Repeat FNA or Lobectomy
IV	Follicular Neoplasm (FN)	15 - 30%	Lobectomy
V	Suspicious for Malignancy	60 - 75%	Near-total or Lobectomy
VI	Malignant	97 - 99%	Near-total or Lobectomy

- **ATA Risk Stratification for Recurrence & TSH Suppression:**
  - **High Risk** (Gross extrathyroidal extension, distant mets, nodes >3 cm): Requires Surgery + RAI + TSH Suppression (<0.1 mU/L).
  - **Intermediate Risk** (Aggressive histology, vascular invasion, >5 nodes): Requires Surgery + TSH Suppression (0.1-0.5 mU/L) ± RAI.
  - **Low Risk** (Intrathyroidal, ≤ 5 micro-nodes): Lobectomy or Total Thyroidectomy. **No RAI.** TSH Target 0.5 - 2 mU/L.
  - **Microcarcinomas (<1 cm, cN0):** Lobectomy alone. No RAI or TSH suppression.

Operative / Procedural Notes

- **Extent of Surgery:**
  - **Total Thyroidectomy:** Removal of all grossly visible thyroid tissue.
  - **Near-Total Thyroidectomy:** Leaves <1 g of tissue adjacent to the recurrent laryngeal nerve near the ligament of Berry.
  - **Subtotal Thyroidectomy:** Leaves >1 g of tissue with the posterior capsule on the uninvolved side.
  - **Hemithyroidectomy (Lobectomy):** Removal of one lobe (plus isthmus/pyramidal lobe in extended).
- **Postoperative Care:** Thyroid surgery does NOT enter the GI tract. Patients do **not** need to wait for bowel sounds/flatus. Feeding is started immediately upon **full recovery** from anesthesia with an intact swallowing mechanism to prevent aspiration.

Complications / Prognosis

- **Immediate Complications (24-72 hrs):**
  - **Hemorrhage / Neck Hematoma:** The **most lethal complication**. It rapidly compresses the trachea causing acute airway obstruction and requires immediate bedside decompression.

    - **Recurrent Laryngeal Nerve Palsy:** Unilateral causes hoarseness; bilateral causes acute airway obstruction.
    - **Hungry Bone Syndrome:** Profound, rapid, and prolonged hypocalcemia. Most strongly associated with surgical intervention for **severe Graves' disease** due to extreme preoperative bone turnover rates abruptly reversing upon thyroid removal.
    - **Parathyroid Insufficiency:** Due to devascularization (inferior thyroid artery damage) or inadvertent removal.
- **Late Complications:** Hypothyroidism, progressive exophthalmos (Graves'), recurrent thyrotoxicosis, hypertrophic scar.

Past-Paper High Yield

- **FNA Limitations:** FNA *cannot* distinguish between a follicular adenoma and follicular carcinoma. A diagnostic **lobectomy (hemi-thyroidectomy)** is required for Bethesda 4 (Follicular Neoplasm) to assess for capsular/vascular invasion.
- **Medullary Thyroid Carcinoma Traits:** Arises from parafollicular C-cells (secretes calcitonin, stains with Congo red for amyloid). Requires prophylactic central neck node dissection. It does **not** take up RAI. Associated with RET mutations (not BRAF).
- **Hashimoto's Thyroiditis:** The single most common cause of hypothyroidism in iodine-sufficient areas.
- **Post-op Bleeding:** The absolute most lethal complication following a thyroidectomy.
- **Post-op Feeding:** Start when the patient has fully recovered and demonstrates an intact swallow (no waiting for flatus).
- **Ultrasound Characteristics:** Spongiform/pure cysts are benign. Cold, solid nodules in a child strongly suggest Papillary Carcinoma.
- **Nodule Workup:** Normal/elevated TSH + >1cm nodule → ultrasound-guided FNA. Radionuclide scans are *only* for low TSH.
- **Cancer associations:**
    - *Iodine deficiency* → Follicular carcinoma (spreads hematogenously, usually unifocal).
    - *Radiation/Iodine sufficiency* → Papillary carcinoma (spreads lymphatically, often multifocal, has Psammoma bodies, BRAF mutated).
    - *Eosinophilic cytoplasm* (not eosinophils) → Hurthle cell carcinoma.

Memory Pearls

- **Papillary Carcinoma:** Think **P** - **P**opular (most common), **P**ediatric (most common in kids), **P**sammoma bodies, **P**alpable lymph nodes (lymphatic spread), **P**ositive BRAF.
- **Follicular Carcinoma:** Think **F** - **F**ar away spread (hematogenous to bone/lung), **F**leshy (vascular invasion needed for dx).
- **Medullary Carcinoma:** Think **M** - **M**EN2a/b, **M**utation in RET, a**M**yloid (Congo red), **M**idline (central Level VI node dissection required).
- **Anaplastic Carcinoma:** Think **A** - **A**ged population, **A**ggressive, **A**irway obstruction.

- *Art History Pearl:* The Mona Lisa is frequently used as a historical representation of thyroid disease (goiter/hypothyroidism) due to her swollen neck, high hairline, absent eyebrows, and yellowed skin.

Surgical aspect of diabetic foot

Core Concepts

- **Pathophysiologic Triad of Diabetic Foot (DF):** Neuropathy, Peripheral Arterial Disease (PAD/Ischemia), and Infection.
- **The Role of Trauma:** Trauma acts as the precipitating trigger on both neuropathic (structural/functional) and vasculopathic pathways, ultimately leading to ulceration or gangrene.
- **Healing Impairments:** Multifactorial; local tissue ischemia and neuropathy impair chemotaxis, tissue necrosis prolongs the inflammatory phase, uncontrolled edema disrupts myofibroblasts, and protein glycation hinders repair.

Diagnosis / Clinical Features

- **Diabetic Neuropathy:**
  - **Sensory:** Decreased pain, pressure, temperature, and proprioception (leads to unrecognized repetitive injury).
  - **Motor:** Small muscle wasting leads to foot deformities (claw/hammer toes), shifting pressure points.
  - **Autonomic:** Decreased sweating leads to dry skin, cracking, and fissure formation.
- **Neuropathic Ulcers:**
  - **Locations:** Occur at high-pressure sites and repetitive stress areas: Plantar metatarsal heads (especially 1st and 5th), plantar heel, and dorsal aspect of toes.
  - **Characteristics:** Granulation tissue surrounded by a thick rim of hyperkeratotic tissue (callus) at the margins. Usually painless due to concurrent sensory neuropathy.
- **Charcot Neuro-osteoarthropathy (CNO):**
  - A sterile inflammatory process in neuropathic patients causing bone destruction, subluxation, dislocation, and deformity.
  - **Acute stage:** Swelling, warmth, and erythema. Pain is mild/absent due to underlying neuropathy (often mistaken for cellulitis).
  - **Chronic stage:** Painless bony deformities, classic "rocker-bottom" foot (midfoot collapse), osteolysis, and fractures.

Investigations

- **Neurologic Assessment (Loss of Protective Sensation - LOPS):**
  - **Semmes-Weinstein 10-gram monofilament:** Applied perpendicular to the skin until it buckles; tested at specific high-yield plantar sites (distal hallux, 1st/3rd/5th metatarsal heads).
  - **128 Hz tuning fork:** Tests vibration perception (applied to bony prominence of distal interphalangeal joint of great toe).

    - **Ipswich touch test:** Lightly touching the tips of the toes with the index finger for 1–2 seconds (used if monofilament/tuning fork unavailable).
    - **Reflexes:** Assessment of ankle and knee jerk reflexes.
- **Vascular Assessment:**
    - **Clinical:** Palpate dorsalis pedis and posterior tibial pulses. Look for dependent rubor (a late finding of severe ischemia).
    - **Non-invasive:** Ankle-Brachial Index (ABI), Toe-Brachial Index (TBI), Duplex ultrasound, Transcutaneous oxygen tension (TcO2), and systolic toe pressures.
    - **Invasive:** CT-angiography if planning revascularization.

Relevant Guidelines

Ankle-Brachial Index (ABI) Interpretation

ABI	INTERPRETATION
>1.30	Poorly compressible vessels, arterial calcification
0.90–1.30	Normal
0.60–0.89	Mild arterial obstruction
0.40–0.59	Moderate obstruction
<0.40	Severe obstruction

IWGDF 2023 Risk Stratification & Screening Frequency

CATEGORY	ULCER RISK	CHARACTERISTICS	SCREENING FREQUENCY
0	Very low	No LOPS, No PAD	Annually
1	Low	LOPS *or* PAD	Every 6-12 months
2	Moderate	(LOPS + PAD) *or* (LOPS + deformity) *or* (PAD + deformity)	Every 3-6 months
3	High	LOPS *or* PAD **+** (hx of ulcer, amputation, or ESRD)	Every 1-3 months

SINBAD Ulcer Scoring System *(Scores 0 or 1 for each category based on severity)*

- **S**ite (Forefoot = 0, Midfoot/Hindfoot = 1)
- **I**schemia (Pulses intact = 0, Clinical reduced flow = 1)
- **N**europathy (Sensation intact = 0, Sensation lost = 1)
- **B**acterial Infection (None = 0, Present = 1)
- **A**rea (<1 cm^2 = 0, >1 cm^2 = 1)
- **D**epth (Skin/SubQ = 0, Muscle/tendon/bone = 1)

IDSA/IWGDF Infection Severity Classification

GRADE	SEVERITY	CLINICAL MANIFESTATIONS
1	Uninfected	No purulence or inflammation.
2	Mild	≥ 2 signs of inflammation, cellulitis extending **≤ 2cm** around ulcer, limited to skin/superficial SubQ. No systemic illness.
3	Moderate	Cellulitis extending **>2cm**, deep tissue spread (abscess, gangrene, muscle/bone/joint), but patient is metabolically stable/systemically well.
4	Severe	Local infection accompanied by **systemic toxicity or metabolic instability** (e.g., fever, hypotension, acidosis, severe hyperglycemia).

Brodsky Classification of Charcot Joint Anatomy

- **Type 1:** Tarsometatarsal and naviculocuneiform joints
- **Type 2:** Subtalar, talonavicular, or calcaneocuboid joints
- **Type 3A:** Tibiotalar (ankle) joint
- **Type 3B:** Calcaneal tuberosity fracture
- **Type 4:** Combination of areas
- **Type 5:** Forefoot solely

Eichenholtz Staging of Charcot Neuro-osteoarthropathy

STAGE	PHASE	RADIOGRAPHIC & CLINICAL FINDINGS	MANAGEMENT
0	Prodromal	Normal X-ray. Clinical: Swelling, warmth, erythema.	Protected weight-bearing, serial X-rays.
I	Acute (Development)	X-ray: Osteopenia, fragmentation, subluxation. Clinical: Swelling, warmth, laxity.	Total contact casting (TCC) or pneumatic brace for 2-4 months.
II	Sub-acute (Coalescence)	X-ray: Debris absorption, sclerosis, fusion. Clinical: Decreased warmth/swelling.	TCC, brace, or Charcot restraint orthotic walker.
III	Chronic (Reconstruction)	X-ray: Consolidation, arthrosis, fixed deformity. Clinical: No warmth/erythema, stable joint.	Custom rocker-bottom shoes; Surgery if non-plantigrade or ulcerated.

Management

- **1. Infection Treatment:**

    - **Mild (Grade 2):** 1-2 weeks of oral empiric antibiotics (e.g., clindamycin, cephalexin, amoxicillin-clavulanate, doxycycline) targeting *S. aureus* and strep. Cleanse and debride.
    - **Moderate/Severe (Grade 3-4):** 2-3 weeks of antibiotics (requires IV broad-spectrum initially for deep/severe). Urgently evaluate for surgery (abscess drainage, compartment release, amputation of necrotic tissue).
- **2. Restoration of Tissue Perfusion:**
    - **Urgent Revascularization indications:** Ankle pressure <50 mmHg, ABI <0.5, toe pressure <30 mmHg, or TcpO2 <25 mmHg.
    - Also indicated if an ulcer fails to heal within 6 weeks despite optimal care.
- **3. Pressure Offloading:**
    - The gold standard for neuropathic plantar ulcers is the **Total Contact Cast (TCC)** (non-removable knee-high device).
    - Alternatives: Removable walkers, ankle-high devices, or felted foam if standard biomechanical relief is unavailable.
- **4. Local Ulcer Care:**
    - Sharp debridement of necrotic tissue and surrounding callus.
    - Do NOT soak the feet (induces skin maceration).
    - **Adjuncts for non-healing uninfected ulcers (4-6 weeks optimal care):** Sucrose octasulfate dressings, autologous leucocyte/platelet patches, placental membrane allografts, or systemic oxygen therapy.
- **5. Prevention:**
    - Proper footwear: Shoe inside should be 1-2 cm longer than the foot.
    - Daily inspection, washing in lukewarm water, drying thoroughly (especially between toes).
    - Avoid walking barefoot entirely.

Operative / Procedural Notes

- **Emergency Indications:** Incision & drainage, debridement to limit progression of acute infection or acute ischemia (e.g., necrotizing fasciitis).
- **Elective Indications:** Revascularization (angioplasty, stenting, bypass), skin grafting to promote healing, converting a chronic wound to an acute wound.
- **Prophylactic Indications:** Correcting deformity or treating joint stiffness that puts the foot at risk of ulceration.

Complications / Prognosis

- **Amputation Mortality:** The 5-year mortality rate after diabetic lower limb amputation is **68%**.
- This is remarkably higher than the 5-year mortality of many major cancers, including colorectal (39%), breast (23%), and prostate cancer (8%).

Past-Paper High Yield

- **Endocrine Tumor Integration (Glucagonoma):** While this lecture focuses on the diabetic foot, the Endocrine Surgery block often tests the systemic presentations of diabetes. A classic past-paper scenario involves a patient with **mild new-onset diabetes mellitus**, profound weight loss, a **pancreatic tail mass**, and **necrolytic migratory erythema** (a distinctive migratory erythematous scaling rash over the lower extremities and groin). This clinical triad is **pathognomonic for a glucagonoma**. (Do not confuse with Insulinomas [hypoglycemia] or Somatostatinomas [diabetes, steatorrhea, gallstones]).
- **ABI Pitfalls:** An ABI > 1.30 in a diabetic patient does not mean "excellent" perfusion; it indicates **arterial calcification** and poorly compressible vessels. Further testing (like Toe-Brachial Index) is required.
- **Charcot Joint Presentation Trap:** An acutely swollen, erythematous, warm foot in a diabetic patient without an open wound or systemic signs of toxicity is highly suspicious for **Acute (Stage 0 or I) Charcot Neuro-osteoarthropathy**, not just cellulitis.

Memory Pearls

- **SINBAD Score Criteria: S**ite, **I**schemia, **N**europathy, **B**acterial infection, **A**rea, **D**epth.
- **Foot Ulcer Triad: N**europathy + **I**schemia + **I**nfection (triggered by Trauma).
- **Eichenholtz Stages: D**evelopment (acute) → **C**oalescence (subacute) → **R**econstruction (chronic).

Cervical lymphadenopathy

Core Concepts

- **Definition & Significance:**
  - Lymph nodes are considered abnormal if >1 cm.
  - Clinically significant enlargement is typically >2 cm or 2.5 cm.
  - The human body has ~600 lymph nodes; ~300 are located in the head and neck.
- **Epidemiology:**
  - 90% of cervical lymphadenopathy (CLA) cases are related to upper respiratory tract infections (URTIs) or sore throats.
  - **Children:** Predominantly viral/benign (>80% benign in patients <30 years).
  - **Adults:** Higher index of suspicion for malignancy (60% malignant in patients >50 years).
- **Pathophysiology of Enlargement:**
  - *Antigenic response* (immune/lymphocyte/macrophage proliferation).
  - *Inflammatory infiltration* (infection).
  - *Malignant proliferation* (primary lymphomas).
  - *Metastatic infiltration* (distant primary).
  - *Metabolic infiltration* (rare, lipid storage diseases).
- **Etiologies (MIAMI Mnemonic):**

- **M**alignant (Lymphoma, leukemia, head/neck SCC metastases)
- **I**nfectious (Viral: EBV, CMV, HIV; Bacterial: TB, dental, cat scratch)
- **A**utoimmune (SLE, RA, Sjögren's)
- **M**iscellaneous (Sarcoidosis, Kawasaki disease)
- **I**atrogenic (Medications like phenytoin, allopurinol)

Diagnosis / Clinical Features

- **Chronology of Presentation:**
  - **Acute (<2 weeks):** Often bacterial/viral (tonsillitis, dental abscess). Nodes are soft, red, tender, and fluctuant. Can be immune (Kawasaki, Kikuchi-Fujimoto).
  - **Subacute (2–6 weeks):**
    - *Unilateral:* Infections (EBV, CMV, HIV, TB, Non-TB mycobacteria, Toxoplasmosis).
    - *Bilateral (with systemic signs):* Autoimmune (SLE, Juvenile Idiopathic Arthritis).
  - **Chronic (>6 weeks):** Hard, firm, non-tender, fixed, progressively enlarging nodes. Suggests malignancy (Hodgkin's, Mets) or granulomatous disease (TB, Sarcoidosis).
- **Cervical Lymph Node Levels:**
  - **Level I (Group 1):** Submental (Level IA) and Submandibular (Level IB) nodes.
  - **Level II:** Upper jugular nodes.
  - **Level III:** Middle jugular nodes.
  - **Level IV:** Lower jugular nodes.
  - **Level V:** Posterior triangle nodes.
  - **Level VI:** Anterior compartment (pretracheal, paratracheal).
  - **Level VII:** Superior mediastinal nodes.
- **Anatomical Drainage & Differentials:**
  - **Submental:** Drains bottom lip, floor of mouth.
  - **Submandibular:** Drains oral cavity, cheek. Target SCC, dental disease.
  - **Preauricular/Posterior Cervical:** Drains scalp, skin.
  - **Supraclavicular:** Drains GI, GU, and pulmonary tracts (highly suspicious for abdominal/thoracic metastasis, e.g., Virchow's node).
- **Midline vs. Lateral Neck Masses:**
  - *Lateral:* Branchial cleft cysts (anterior to sternocleidomastoid), lymphadenopathy.
  - *Midline:* Thyroglossal duct cysts, dermoid cysts, submental lymphadenopathy, ectopic thymic cysts.
- **Red Flags for Malignancy:** Size >2 cm, hard/fixed/matted nodes, supraclavicular location, persistent fever >1 week, no regression after 4–6 weeks, or presence of B-symptoms (weight loss, night sweats).

Investigations

- **Laboratory Tests:**
  - Complete blood count (CBC) and peripheral blood smear (critical for detecting mononucleosis or hematological malignancies).
  - Infectious markers, ESR, CRP.
- **Imaging:**
  - **Neck Ultrasound:** First-line initial test to assess size, architecture, and differentiate benign vs. malignant features.
  - **Contrast-Enhanced CT (CECT):** Used if US is non-specific, to evaluate deeper planes, or stage known tumors.
- **Tissue Diagnosis:**
  - **Fine Needle Aspiration Cytology (FNAC):** Quick, reliable, outpatient initial diagnostic tool for suspicious nodes. Avoids surgical disruption of lymphatic drainage planes.
  - **Node Biopsy (Excisional):** The gold standard. Required for architectural evaluation, immunohistochemistry (IHC), and flow cytometry (especially for subtyping lymphomas).

Management

- **Primary Care Principles:**
  - Identify and treat the primary underlying cause first (e.g., dental caries, tonsillitis).
  - For an acutely red, tender node of short duration, **start antibiotics and observe** before considering biopsy.
  - For localized, asymptomatic adenopathy without red flags, observe for 3–6 weeks (up to 2 months if clinically safe).
- **Indications for Biopsy:**
  - Persistent adenopathy (>6 weeks) despite observation/conservative management.
  - Advanced age, hard/fixed nodes, supraclavicular location, B-symptoms, or unexplained splenomegaly.
  - Suspicion of TB or Chronic Granulomatous Disease.

Relevant Guidelines

- **Referral Pathway Decision Tree:**
  - **Urgent Referral (24–72 hours):** Severe infection, strong suspicion of malignancy, or Kawasaki Disease features.
  - **Early Referral (2–3 weeks):** Size >2 cm + steady size increase + autoimmune suspicion.
  - **Routine Referral (4–6 weeks):** Size <2 cm + persistent lymphadenopathy after 6 weeks of observation.
- **Diagnostic Algorithm for Suspicious Neck Lymph Nodes:**
  1. Perform targeted exam (ENT mirror/endoscopy, skin, thyroid).
  2. If NO primary tumor found → perform **FNAC 1** (direct or US-guided).

3. If FNAC 1 is negative (only inflammatory cells) → perform **FNAC 2**.
4. If FNAC 2 is negative → perform **Biopsy** (Excisional/Incisional).

- **Positive FNAC Results Workup Pathway:**
  - **Squamous Cell Carcinoma (SCC) / Adenocarcinoma:** Perform MRI or PET-CT → Exam Under General Anesthesia (EGA) with tonsillectomy & directed/blinded biopsies (base of tongue, piriform sinus).
  - **Lymphoma:** Proceed directly to Excisional Biopsy for flow cytometry and IHC.
  - **Thyroid Carcinoma:** Perform Thyroid Ultrasound for surgical planning.
  - **Melanoma / Merkel Cell Carcinoma:** Refer directly to Skin Cancer Multidisciplinary Team (MDT).
- **Pediatric Acute Bacterial Lymphadenitis Antibiotic Guidelines:**
  - *First-line (10–14 days):* Oral Augmentin (50 mg/kg/day Q12H) OR Cephalexin (50 mg/kg/day Q8H) OR Cloxacillin.
  - *Alternative (Penicillin Allergy):* Oral Clindamycin.

Operative / Procedural Notes

- **Principles of an Excellent Lymph Node Biopsy:**
  - **Site selection:** Lower cervical nodes (supraclavicular/posterior triangle) yield higher diagnostic accuracy than upper neck nodes, which are frequently reactively enlarged. Always target the largest node, not just the most superficial one.
  - **Technique:** Excise the *entire* intact node. Do not perform piecemeal excisions.
  - **Handling:** Provide fresh and frozen samples in addition to formalin-fixed tissue. Keep away from strong light.
- **Tracheostomy Complication (Sentinel Bleed):**
  - A sudden, brisk bright red bleed from a tracheostomy site 1–2 weeks post-procedure that resolves spontaneously is a classic "sentinel bleed" warning of a **tracheo-innominate fistula**. This is a surgical emergency.

Past-Paper High Yield

- **Older patient + Smoker + Isolated firm cervical node:** Highly suspicious for Head and Neck Squamous Cell Carcinoma (SCC).
  - *Next best step:* **Fine Needle Aspiration (FNA) cytology.**
  - *Trap:* **Do NOT perform an initial excisional biopsy.** Open biopsy disrupts lymphatic drainage planes and can compromise definitive oncologic surgery (e.g., radical neck dissection).
- **Group 1 / Level I Anatomy:** You must know that **Submental and Submandibular nodes** belong to Level I (Group 1) cervical lymph nodes.
- **Acute Inflammatory Nodes:** An acutely red, tender, rapidly enlarging neck node of a few weeks duration in an otherwise well patient is acute lymphadenitis.
  - *Next best step:* **Start antibiotics and observe.** Do not rush to FNA or biopsy immediately unless it fails to resolve.

- **Neck Mass Anatomical Locations (Children):**
  - **Branchial cleft cysts** are classically **lateral** neck masses (located anterior to the SCM).
  - *Midline* masses include thyroglossal duct cysts, dermoid cysts, submental nodes, and ectopic thymus.
- **Tracheostomy Sentinel Bleed:** A brisk, bright red bleed 1–2 weeks post-tracheostomy is the hallmark of a **tracheo-innominate fistula**, not granulation tissue oozing or simple infection.

Memory Pearls

- **Malignancy Risk by Area:** <1 cm^2 = 0% risk; >2.25 cm^2 = 38% risk. Rule of thumb: The larger the node (>2 cm), the higher the cancer risk.
- **Biopsy Hierarchy: FNA first** (for metastasis/SCC to protect surgical planes), **Excisional biopsy definitive** (for lymphoma subtyping).
- **Supraclavicular Node = Red Flag:** Always look below the clavicles (lung, stomach, GI, GU) if a supraclavicular node is enlarged (Virchow's node).

Cardiothoracic

Exam Map

REVISION PRIORITY	KEY TOPICS	FOCUS AREAS
Tier 1 (Critical)	Chronic Lower Limb Ischemia, Valvular Heart Disease, Thoracic Trauma	ABI interpretation, Valve selection criteria, Congenital heart associations, Tension PTX vs Hemothorax, Thoracotomy criteria.
Tier 2 (High Yield)	CAD, Lung Cancer, Acute Limb Ischemia, Thoracic Aortic Disease	CABG vs PCI candidacy, Pancoast resectability, FEV1 cutoffs, Embolus vs Thrombus, Type A vs Type B dissection.
Tier 3 (Moderate/Low)	Mediastinal Masses, Hemodynamics, Vascular Injury, Lung Infections	Compartment boundaries, Empyema drainage criteria, Oxygen curve shifts, Hard signs of vascular injury.

- **Vascular Diagnostics & ABI:** The Ankle-Brachial Index (ABI) is heavily tested. Know the normal range (>0.90), but specifically watch for the trap of **ABI >1.25**—this indicates calcified, non-compressible vessels (common in diabetes/renal failure) and does *not* rule out PAD.
- **Acute vs. Chronic Ischemia:** Differentiate embolic ischemia (sudden, no prior claudication, AFib history → treat with emergent embolectomy) from thrombotic ischemia (insidious, history of claudication, collaterals present).
- **Venous Disease & Complications:** Always identify **lower limb ischemia** as the most frequent complication of an Intra-Aortic Balloon Pump (IABP). For uncomplicated venous stasis ulcers, **compression therapy** is the definitive first-line answer.
- **Prosthetic Valve Selection:** Mechanical valves are for young patients with a >10-year life expectancy (requires lifelong warfarin). Bioprosthetic valves are for the elderly, frail, those at high bleed risk, and women desiring pregnancy.
- **Cardiac Pathophysiology:** Remember that Mitral Stenosis restricts LV filling and explicitly **spares the left ventricle (no LVH)**. In Congenital disease, Tetralogy of Fallot severity is dictated purely by the **degree of pulmonary stenosis** (which protects against pulmonary hypertension).
- **Thoracic Trauma Algorithms: Tension pneumothorax is a clinical diagnosis**—immediate needle decompression is required; choosing CXR or IV fluids first is incorrect. Distinguish pneumothorax (hyperresonant, trachea pushed away) from hemothorax (dull).
- **Thoracotomy Criteria:** Commit the chest tube output thresholds to memory: **>1500 cc immediately** or **>200 cc/hr for 2 to 4 consecutive hours** mandates operative thoracotomy.
- **CABG vs. PCI Operative Decision:** The classic board-style CABG candidate is a patient with complex **triple-vessel disease and a reduced ejection fraction (<50%)**. Single/double vessel disease with normal EF is managed medically or with PCI.
- **Lung Cancer Resectability: FEV1 < 800 ml** generally precludes major surgical resection. For Pancoast tumors, they are automatically **≥T3** (chest wall invasion); the presence of **Horner's syndrome** makes them surgically unresectable.

- **Aortic Dissection Pathways: Stanford Type A** (ascending) demands emergent surgery. **Stanford Type B** (descending) is managed medically. The pharmacological sequence for Type B is strict **heart rate control FIRST (beta-blockers), followed by blood pressure control**.
- **Mediastinal Mass Compartments:** Location determines the diagnosis. Anterior = Thymoma/Teratoma/Thyroid/Terrible Lymphoma. Middle = Bronchogenic cysts. Posterior = **Neurogenic tumors** (Schwannomas, Neuroblastomas).
- **Pleural Fluid Criteria:** Pleural fluid analysis dictates surgical drainage in lung infections. A **pH < 7.20** or **Glucose < 40 mg/dL** defines a complicated parapneumonic effusion/empyema requiring immediate chest tube insertion. Use large-bore tubes (32-36F) placed *above* the rib margin.

Vascular injury

Core Concepts

- **Epidemiology:** Peripheral vascular injury (PVI) occurs across all demographics but is primarily seen in adult males. In civilians, blunt trauma is the leading cause in children, while penetrating trauma (gunshot/stab wounds) dominates in adults.
- **Vessel Distribution:** Arterial injuries are diagnosed more frequently than venous injuries. In adults, the lower extremities (popliteal and superficial femoral arteries) are most commonly injured. In the upper limb, 2/3 are distal (radial/ulnar) and 1/3 proximal (brachial).
- **Lower Extremity Venous Anatomy & Physiology:**
  - The greater (long) saphenous vein ascends the leg and joins the superficial femoral vein at the saphenofemoral junction to form the common femoral vein.
  - The calf muscle pump is the primary driver of venous return against gravity. In a healthy person, ambulation *decreases* venous pressure in the lower extremities.
  - Perforating veins contain valves directing flow strictly from the superficial to the deep system (never the reverse).
  - The inferior vena cava and common iliac veins are largely valveless.
- **Injury Classification:**
  - **Nonocclusive:** Intimal irregularity/tear (<25% narrowing), hematoma (≥25% narrowing), partial transection with pseudoaneurysm formation.
  - **Occlusive:** Thrombotic occlusion (vessel wall preserved), complete transection.

Diagnosis / Clinical Features

- **Hard Signs of Vascular Injury:** Indicate major arterial injury and require immediate surgical or angiographic intervention.
  - Pulsatile bleeding
  - Expanding or pulsating hematoma
  - Loss of distal pulses
  - Palpable thrill or continuous bruit

- **Soft Signs of Vascular Injury:** Raise the index of suspicion for possible PVI; warrant further investigation.
  - Nonpulsatile bleeding
  - Nonexpanding/nonpulsatile hematoma (most common soft sign, 35%)
  - Diminished pulse
  - History of massive arterial bleeding or hypotension
  - Previously applied tourniquet
  - Neurologic deficit
  - Wound in proximity to a named vessel
- **Special Clinical Scenarios:**
  - **Iatrogenic Arteriovenous (AV) Fistula:** A known complication post-endovascular procedure (e.g., PCI stenting). Presents with a palpable thrill, continuous bruit, and venous engorgement (edema, varicose veins).
  - **Pseudoaneurysm:** Presents as a pulsatile mass with a "to-and-fro" bruit, but crucially *lacks* prominent venous engorgement.
  - **Knee Dislocations:** High risk of occult popliteal artery injury. A normal distal pulse does *not* exclude injury; imaging is required.

Investigations

- **Ankle-Brachial Index (ABI) / Arterial Pressure Index (API):** Excellent screening tools. An ABI >0.9 generally excludes the need for further imaging in stable patients. <0.9 mandates further imaging.
- **Computed Tomography Angiography (CTA):** First-line imaging modality for hemodynamically stable adults and children without active bleeding. Sensitivity/specificity >90%.
  - *Direct signs:* Occlusion, thrombosis, intimal dissection, spasm, active hemorrhage, AV fistula.
  - *Indirect signs:* Perivascular hematoma, projectile tract near a neurovascular bundle, shrapnel <5 mm from a vessel.
- **Doppler Ultrasound:** High sensitivity (95%) and specificity (98%). Used as a triage tool (FAST Doppler protocol), but cannot differentiate acute from chronic lesions.
- **Invasive Catheter Angiography:** Reserved for patients needing immediate interventional procedures, cases of suspected vasospasm, or when CTA is non-diagnostic/unavailable (e.g., due to metallic artifact scatter).
  - *Indications:* Hemodynamically stable patients with uncertain diagnosis (soft signs, known PVD) or unclear wound trajectory (multiple wounds, shotgun wounds, bullet tract parallel to artery).

Management

- **Prehospital:** Tourniquets are lifesaving and should be applied immediately for life-threatening extremity hemorrhage. Delaying application is associated with a >4.5-fold increase in mortality. Do not wait for shock or arrival at a trauma center.
- **Nonoperative Management (NOM):**

    - Indicated for isolated, stable, low-grade injuries (intimal tears <5 mm, adherent intimal flaps) with intact distal circulation and no active hemorrhage.
    - Can be considered in isolated tibial/peroneal injuries if at least one tibial artery remains intact and there is no distal ischemia.
- **Damage Control:** In unstable or mangled patients, temporary intravascular shunts (TIVS) are used to rapidly restore perfusion and bridge to definitive repair.
- **Surgical Exploration:** Hemodynamically unstable patients or those with hard signs go directly to the operating room (or hybrid angio suite).

Relevant Guidelines

- **AAST Organ Injury Scale (OIS) for Peripheral Vascular Injuries:**

GRADE	INJURY DESCRIPTION
I	Digital, palmar, plantar vessels; dorsalis pedis artery; non-named branches
II	Basilic/cephalic vein, saphenous vein, radial/ulnar artery
III	Axillary, superficial/deep femoral, popliteal veins; Brachial, anterior/posterior tibial, peroneal arteries
IV	Superficial/deep femoral artery, popliteal artery
V	Axillary artery, common femoral artery

- **AAST/WSES Management Algorithm for Extremity Trauma:**
    - **Hemodynamically Unstable:** Direct to OR/Angio suite.
    - **Hemodynamically Stable:**
        - *Hard Signs:* Direct to OR/Angio suite.
        - *Soft Signs:* Check ABI or Doppler US. If ABI <0.9 or US abnormal → CT/Angiography → If positive, OR/Angio suite. If negative, Discharge.
        - *No Signs (Blunt/High Energy Penetrating):* Check ABI/US. If abnormal, treat as soft signs. If normal, safely Discharge (observe 24-48h).

Operative / Procedural Notes

- **General Principles:** Ischemia time is critical; vascular repair generally precedes orthopedic intervention. No systemic intraoperative heparinization or postoperative antiplatelet therapy is recommended (unless prolonged ischemia with small vessel occlusion), as it increases blood product use without improving limb salvage.
- **Reconstruction Conduits:** Tension-free end-to-end primary repair is the procedure of choice. If an interposition graft is needed, the autologous saphenous vein (harvested from the contralateral uninjured leg) is preferred. PTFE synthetic grafts maintain structural integrity even in the face of infection and have comparable complication rates.

- **Major Arterial Injuries:** Perform proximal and distal thrombectomy with a Fogarty catheter; flush segments with heparinized saline prior to anastomosis.
- **Venous Injuries:** Repair if possible to prevent venous insufficiency/amputation. If the patient is unstable or the vein is destroyed, simple ligation is acceptable. Combined AV injuries or venous ligation carry a high risk of compartment syndrome, warranting prophylactic fasciotomy.
- **Upper Extremity Approach:** Brachial vessels are accessed rapidly via an incision along the medial groove of the biceps and triceps. Veins in the upper extremity can generally be ligated due to extensive collaterals.
- **Lower Extremity Ligation:** Isolated radial/ulnar or infrageniculate injuries can be managed with simple ligation if distal perfusion (e.g., contralateral tibial artery) remains intact.

Complications / Prognosis

- **Ischemia Limits:** Acceptable tourniquet duration is <2 hours. Total warm ischemia (no flow) should be limited to <6 hours.
  - >60 mins tourniquet time increases the risk of rhabdomyolysis, wound infection, and neurologic compromise.
  - >6 hours of ischemia significantly increases the risk of amputation.
- **Compartment Syndrome:** Often complicates severe lower limb injuries, particularly with combined arterial-venous trauma or prolonged ischemia (>4-6h). Prophylactic fasciotomy is highly recommended over waiting for therapeutic symptoms.
- **Amputation Risk Factors:** Prolonged ischemia (>6h), disruption of the posterior tibial nerve, severe tissue/segmental bone loss, and combined major vessel injuries (brachial, popliteal, femoral arteries carry the highest risk). A Mangled Extremity Severity Score (MESS) >7 suggests primary amputation.
- **Delayed Pseudoaneurysm:** Patients discharged after penetrating trauma with normal initial workups must be followed outpatient due to the risk of delayed pseudoaneurysm formation.

Past-Paper High Yield

- **Post-PCI Complications:** A palpable thrill, continuous bruit, and venous engorgement (edema, varicose veins) after a groin puncture highly suggests an **iatrogenic AV fistula**. Do not confuse with a pseudoaneurysm, which presents with a pulsatile mass and a *to-and-fro* bruit, but typically lacks gross venous engorgement.
- **Lower Extremity Venous Anatomy:** Know that the greater saphenous vein joins the superficial femoral vein at the saphenofemoral junction to form the common femoral vein.
- **Venous Physiology:** The calf muscle pump lowers venous pressure upon walking. Normal perforators direct blood superficially to deep. Common iliacs and the IVC do not have prominent valves.
- **Knee Dislocations:** Never rule out popliteal artery injury just because distal pulses are palpable. Always image (CTA).

Memory Pearls

- **Hard Signs mean Hard Stop:** Stop assessing and go straight to the OR/Angio (Pulsatile bleeding, expanding hematoma, thrill/bruit, pulselessness).
- **Warm Ischemia = 6 hours max. Tourniquet = 2 hours max.**
- **ABI > 0.9:** The cut-off that saves the patient a trip to the CT scanner.
- **Popliteal Pulse in Knee Dislocation:** A palpable pulse lies; still scan to rule out intimal flap/injury.

Valvular heart disease

Core Concepts

- **Surface Anatomy of Cardiac Valves:**
  - **Pulmonary Valve (PV):** Medial end of the 3rd left costal cartilage and adjoining sternum. Contains 3 cusps (1 posterior, 2 anterior).
  - **Aortic Valve (AV):** Left half of sternum opposite the 3rd intercostal space (ICS). Contains 3 cusps (right coronary, left coronary, non-coronary).
  - **Mitral Valve (MV):** Left half of sternum opposite the 4th costal cartilage. Contains 2 cusps (anterior is larger). Normal cross-sectional area is 4–6 cm^2.
  - **Tricuspid Valve (TV):** Right half of sternum opposite the 4th ICS. Contains 3 cusps.
- **Prosthetic Valve Types:**
  - **Mechanical Valves (e.g., bileaflet, ball-and-cage):** Excellent durability but highly thrombogenic. Require lifelong systemic anticoagulation (warfarin).
  - **Bioprosthetic Valves (Tissue):** Include human homograft/autograft and animal xenograft (porcine or bovine pericardial). Stiffened with glutaraldehyde. Lower durability but do not require lifelong warfarin.

Diagnosis / Clinical Features

Aortic Stenosis (AS)

- **Etiology:** Congenital (infants/children), bicuspid valve calcification/rheumatic (young/middle-aged), senile degenerative calcification (elderly).
- **Pathophysiology:** Fixed outflow obstruction → progressive concentric left ventricular hypertrophy (LVH) → increased LV mass and inadequate coronary flow.
- **Signs/Symptoms:** Exertional dyspnea, angina, exertional syncope, pulmonary edema, sudden death. Ejection systolic murmur, slow-rising carotid pulse, reduced pulse pressure.

Aortic Regurgitation (AR)

- **Etiology:** Bicuspid valve (congenital), rheumatic disease, infective endocarditis (IE), trauma, aortic root dilation (Marfan syndrome, atheroma, syphilis, ankylosing spondylitis).
- **Pathophysiology:** Volume overload → LV dilation and hypertrophy. In *acute* AR, abrupt end-diastolic volume rise leads to rapidly elevated LV filling pressure and acute pulmonary edema.

- **Signs/Symptoms:** Bounding peripheral pulses, collapsing/large-volume pulse, early diastolic murmur, signs of heart failure.

Mitral Stenosis (MS)

- **Etiology:** Almost entirely rheumatic in origin (acquired). Accounts for 25% of all rheumatic heart disease. Predominantly affects females (2/3 of cases). Characterized by "fish-mouth" deformity with commissural fusion.
- **Pathophysiology:** Severely restricts filling of the left ventricle. Triggers pulmonary vasoconstriction → Pulmonary Hypertension (PH) → Right heart failure.
- **Signs/Symptoms:** Progressive dyspnea (worse with exercise, fever, tachycardia, pregnancy), hemoptysis (ruptured bronchial vessels), loud S1, opening snap, mid-diastolic murmur, atrial fibrillation (AF).

Mitral Regurgitation (MR)

- **Etiology:** *Acute* (ruptured chordae/papillary muscle post-MI, IE, trauma). *Chronic* (Mitral valve prolapse, rheumatic, CAD, connective tissue disorders).
- **Pathophysiology:** Left atrial dilation increases preload; LV dilates and hypertrophies to generate larger stroke volume.
- **Signs/Symptoms:** Displaced hyperdynamic apex beat, apical systolic murmur with thrill, exertional/nocturnal dyspnea.

Investigations

- **Aortic Stenosis:**
  - **ECG:** LVH with strain (large S in V2, large R in V6 with T-wave inversion).
  - **CXR:** Dilated ascending aorta (post-stenotic dilation), *normal heart size* (hypertrophy is concentric).
  - **ECHO Criteria for Severity:**
    - Mild: Mean gradient <25 mmHg, Area >1.5 cm^2
    - Moderate: Mean gradient 25-45 mmHg, Area 1.0-1.5 cm^2
    - Severe: Mean gradient >45 mmHg, Area <1.0 cm^2
    - Critical: Mean gradient >70 mmHg, Area <0.7 cm^2
- **Aortic Regurgitation:**
  - **CXR:** Enlarged thoracic aorta with *cardiomegaly* (unlike AS).
  - **ECHO:** Dilated/hyperdynamic LV, fluttering anterior mitral leaflet, diastolic reflux.
- **Mitral Stenosis:**
  - **ECG:** Left atrial enlargement ("P mitrale" - prolonged/notched P in lead II, wide negative terminal force in V1) if not in AF. Right ventricular hypertrophy.
  - **CXR:** Straightening of left heart border, double density sign (LA enlargement), mild cardiomegaly.
- **Mitral Regurgitation:**

- **CXR:** Marked cardiomegaly, severe LA appendage enlargement, pulmonary venous hypertension.

Management

- **Aortic Stenosis:**
  - Medical therapy is reserved only for complications (HF, IE, arrhythmias).
  - Surgical/Transcatheter Aortic Valve Replacement (SAVR/TAVR) is the primary definitive treatment.
- **Aortic Regurgitation:**
  - Asymptomatic with normal LV: Vasodilator therapy; monitor for AVR indications.
  - AVR Indications: Symptomatic, enlarging heart, asymptomatic with EF <0.50, or severe LV dilation (end-diastolic >75 mm, end-systolic >55 mm). Acute AR requires emergent AVR (meds are only a bridge).
- **Mitral Stenosis:**
  - Mild/Asymptomatic: Yearly follow-up, HR control/diuretics if CHF develops.
  - Percutaneous Balloon Valvuloplasty: For CHF unresponsive to meds or asymptomatic patients with PA systolic pressure ≥ 50 mmHg.
  - Surgical: Commisurotomy or Mitral Valve Replacement (MVR) if symptomatic or valve area < 1.0 cm^2.
- **Mitral Regurgitation:**
  - Indications for Surgery: Acute MR with CHF/shock, acute endocarditis, Class III/IV symptoms, or systemic emboli.
  - Repair is preferred (annulus reduction, leaflet resection, neochordae/PTFE placement, papillary muscle splitting) over MVR when possible.

Relevant Guidelines

ACC/AHA Antithrombotic Therapy in Patients with Mechanical Heart Valves:

- **Class I Recommendations:**
  - **Goal INR 2.0 to 3.0:** After AVR with bileaflet mechanical or Medtronic Hall valves if NO risk factors* are present.
  - **Goal INR 2.5 to 3.5:** After AVR with bileaflet mechanical/Medtronic Hall valves IF risk factors* are present; after AVR with Starr-Edwards or disc valves (no risk factors); after Mitral Valve Replacement (MVR) with ANY mechanical valve.
  - **Aspirin Role:** 75 to 100 mg/day *in addition* to warfarin in all mechanical valve patients and biological valve patients with risk factors. If unable to take warfarin, aspirin 75 to 325 mg/day is indicated.
- **Class IIa Recommendations:**
  - For the first three months after AVR, target INR 2.5 to 3.5.
- **Class IIb Recommendations:**

- Clopidogrel (75 mg/day) or warfarin (goal INR 3.5–4.5) for high-risk patients who cannot use aspirin.
- |Risk Factors include: Atrial fibrillation, prior thromboembolism, left ventricular dysfunction, and hypercoagulable state.*

Operative / Procedural Notes

- **Transcatheter Aortic Valve Intervention (TAVI/TAVR):**
  - Balloon valvuloplasty fractures calcifications → transcatheter valve positioned → balloon expansion deploys metallic frame, crushing native leaflets outward.
  - **Access routes:** Transfemoral (most common), transapical (direct, via left anterior thoracotomy), transaortic (mini-sternotomy), subclavian.
- **Mitral Valve Replacement (MVR) Hazard Zones:**
  - *Aortic-mitral annulus:* Placing sutures too deep anteriorly risks damaging aortic valve leaflets, causing iatrogenic aortic regurgitation.
  - *Posterior annulus:* Proximity to the circumflex coronary artery.
  - *Central fibrous body:* Proximity to the AV node (risk of iatrogenic heart block).
- **Supra-annular vs. Intra-annular Implantation:** Supra-annular anchoring allows the placement of a larger prosthetic valve (e.g., 23 mm vs 21 mm), maximizing the effective flow orifice.

Complications / Prognosis

- **Early Mortality Post-Mechanical Valve:** Highly thrombogenic nature means inadequate early anticoagulation frequently leads to fatal acute thromboembolic complications.
- **Untreated Severe AS:** Carries a grim prognosis (50% mortality at 2 years) if the stenotic valve is not replaced.

Past-Paper High Yield

- **Valve Selection Dynamics (Mechanical vs. Biological):**
  - *Mechanical preferred:* Young, healthy patients (e.g., 30-year-old male) due to long life expectancy (>10 years) preventing the need for structural reoperation.
  - *Biological preferred:* Elderly (>65-75 years), short life expectancy, high bleeding/fall risk, non-compliance with oral anticoagulation, or females desiring pregnancy (warfarin is highly teratogenic).
- **Tetralogy of Fallot (ToF):**
  - Features: VSD, Overriding aorta, Right ventricular outflow tract obstruction (Pulmonary Stenosis), and Right Ventricular Hypertrophy. *An Atrial Septal Defect (ASD) is explicitly NOT part of the defining tetrad.*
  - Prognosis/Severity: Dictated almost entirely by the degree of **pulmonary stenosis** (determines the magnitude of the right-to-left shunt and cyanosis severity).
- **Patent Ductus Arteriosus (PDA):**
  - Strongly associated with maternal rubella infection.

    - A decreased "machinery" murmur is a *dire* prognostic sign, indicating Eisenmenger syndrome (rising pulmonary vascular resistance).
    - Note: Physiologically, left-sided volume overload precedes RVH, but exam phrasing occasionally tests specific nuances regarding strict sequential hypertrophy assertions.
- **Atrial Septal Defects (ASD) Distinctions:**
    - *Sinus venosus ASD:* Most commonly associated with partial anomalous pulmonary venous return (PAPVR).
    - *Ostium primum ASD:* Associated with atrioventricular septal defects (AVSDs), Down syndrome, and a *cleft anterior mitral valve leaflet* causing mitral insufficiency.
    - *Ostium secundum ASD:* Most common type overall, but lacks the specific syndromic/anatomic anomalies of the others.
- **Aortic Stenosis Management Goals:** The definitive goal of SAVR/TAVR is to relieve left ventricular outflow tract obstruction, thereby eliminating pressure overload and promoting the long-term *regression of Left Ventricular Hypertrophy (reverse remodeling)*. Mean transvalvular gradient is the fundamental metric for grading severity.
- **TAVR Indications:** TAVR is the absolute standard of care over medical management or SAVR for severe AS in elderly, frail patients deemed prohibitive or high-risk for open-heart surgery.
- **Mitral Stenosis Physiology:** Isolated MS restricts LV filling. Therefore, the left ventricle is underfilled and doing less work; **Left Ventricular Hypertrophy (LVH) is highly unlikely to occur** in isolated MS, whereas LA enlargement, AF, and RVH are direct downstream consequences.
- **Vascular Occlusion Localization:** Left hip/buttock claudication + absent left femoral/popliteal pulses + intact contralateral right pulses = unilateral aortoiliac disease localized specifically to the **Left Common Iliac Artery**.

Memory Pearls

- **Mechanical vs. Tissue Valve:** "Tissue for the old, frail, and future mothers; Mechanical for the young and healthy."
- **Mitral Stenosis:** "Fish mouth" deformity; think Left Atrial (LA) issues and Right Heart (RV) consequences, but *spares* the Left Ventricle (no LVH).
- **Aortic Stenosis Severity:** Focus on the Gradient. >40 mmHg = Severe AS → Replace it to reverse LVH.
- **Congenital associations:**
    - Maternal Rubella = PDA
    - Mitral Cleft = Ostium Primum ASD
    - PAPVR = Sinus Venosus ASD

Mediastinal masses

Core Concepts

- **Mediastinal Boundaries**:

    - **Lateral**: Parietal pleura.
    - **Anterior**: Sternum.
    - **Posterior**: Vertebral column and paravertebral gutters.
    - **Superior**: Thoracic inlet.
    - **Inferior**: Diaphragm.
- **Divisions of the Mediastinum**: Separated into Superior and Inferior by the **transverse thoracic plane** (from the sternal angle anteriorly to the intervertebral disc of T4/T5 posteriorly). The Inferior is further subdivided into Anterior, Middle, and Posterior compartments.
- **Anterior Mediastinum Compartment**:
    - *Location*: Forward of and superior to the heart shadow.
    - *Contents*: Thymus gland, substernal extension of thyroid/parathyroid glands, lymphatic tissues.
    - *Typical Masses*: Thymic lesions (50% of anterior masses), Lymphoma, Teratoma/Germ cell tumors, Ectopic thyroid, Cystic hygromas.
- **Middle Mediastinum Compartment**:
    - *Location*: Extends from lower edge of the sternum, along diaphragm, cephalad along the posterior heart border and posterior wall of the trachea.
    - *Contents*: Heart, pericardium, aortic arch and major branches, SVC/innominate veins, pulmonary arteries/hila, trachea, phrenic/upper vagus nerves, lymph nodes.
    - *Typical Masses*: Lymphadenopathy (most common overall), Bronchogenic cysts (most common cyst), Esophageal duplication cysts, Cardiovascular aneurysms.
- **Posterior Mediastinum Compartment**:
    - *Location*: Between the back of the heart/trachea and the front of posterior ribs/paravertebral gutter.
    - *Contents*: Esophagus, descending aorta, azygos/hemiazygos veins, thoracic duct, sympathetic chain, lower vagus nerve.
    - *Typical Masses*: Neurogenic tumors (>60% of posterior masses), Esophageal tumors/cysts.

Diagnosis / Clinical Features

- **Mass Effect Symptoms**: Direct compression of mediastinal structures can cause cough, stridor, dyspnea, hemoptysis, pain, dysphagia, hoarseness, Horner syndrome (sympathetic chain involvement), hypotension (cardiac compression/tamponade), or Superior Vena Cava (SVC) syndrome (facial/upper extremity swelling).
- **Systemic Effects**: Fever, night sweats, and weight loss (often seen in Lymphoma).
- **Thymoma Presentations**:
    - Peak incidence: 40–60 years; originates from epithelial thymic cells.
    - ~50% are asymptomatic (incidental finding on CXR).
    - ~30% present with local mass effect symptoms.
    - ~20-70% associated with an autoimmune/paraneoplastic syndrome. **Myasthenia gravis** is the most common (occurring in ~30% of patients with thymoma, causing muscle weakness). Other associations: Pure red cell aplasia, polymyositis, hypogammaglobulinemia.

- **Neurogenic Tumors**:
  - *Adults*: Schwannomas and neurofibromas (benign, from intercostal nerve sheath) account for 90%. Ganglioneuromas (benign, from sympathetic ganglia) are seen in young adults.
  - *Children*: Neuroblastoma and ganglioneuroblastoma (malignant, from sympathetic ganglia).
  - *Red Flag*: Paraplegia implies spinal cord compression requiring urgent MRI and laminectomy.
- **Cystic Lesions**:
  - *Bronchogenic cysts*: Most common middle mediastinal cyst (due to abnormal lung budding); typically right paratracheal or subcarinal. More common in men.
  - *Esophageal duplication cysts*: Adjacent/embedded in esophageal wall; may obstruct or erode the wall.

Investigations

- **Computed Tomography (CT) Chest**: The preferred imaging procedure of choice.
  - Must use **IV contrast** to demonstrate vascular relationships, define tumor vascularity, and guide surgical removal.
  - Most enlarged thymus glands on adult CT scans represent a thymoma.
- **Trauma Imaging (High-Yield Exception)**: **Computed Tomography Angiography (CTA)** of the chest is universally considered the definitive and most practical modality for diagnosing traumatic aortic transection in stable or semi-stable blunt trauma patients.
- **Laboratory Markers**:
  - *Suspected Germ Cell Tumor*: Must measure **Alpha-fetoprotein (AFP)**, **Lactate dehydrogenase (LDH)**, and **beta-human chorionic gonadotropin (beta-hCG)** prior to any therapy.
  - *Suspected Thymoma*: Must test for **anti-acetylcholine receptor antibodies** if not previously evaluated for Myasthenia Gravis.
- **Biopsy Guidelines**: Indicated preoperatively if the patient has atypical features, an invasive tumor, or is being considered for induction therapy. Approached via VATS, anterior mediastinotomy, or cervical mediastinoscopy.
- **Fluoroscopic Barium Swallow (Contrast Esophagram)**: Useful for demonstrating extrinsic posterior compression of the esophagus by middle mediastinal masses (e.g., cysts, lymphadenopathy).
- **Pediatric Radiographs**: A normal large thymus in a young child classically demonstrates a **"thymic sail sign"** on CXR (horizontal inferior margin abutting the minor fissure).

Management

- **Thymoma Surgical Resection**:
  - *Small/Accessible*: Total thymectomy with contiguous removal of mediastinal fat.
  - *Invasive*: Total thymectomy + en bloc removal of involved structures (pericardium, pleura, lung, SVC).

- *Phrenic Nerve Involvement*: Resect one phrenic nerve if involved. If *both* are involved, do not resect either; instead, debulk the area to preserve respiratory function.
- *Margins*: Clip areas of close margins/residual disease to assist radiotherapy planning.

- **Chemotherapy for Thymoma**: Combinations usually include cyclophosphamide, doxorubicin (Adriamycin), and cisplatin (or etoposide + cisplatin).
- **Radiotherapy**: Standard of care for incompletely resected or Stage III/IV thymomas.

Relevant Guidelines

- **Masaoka Classification System for Thymomas**: Used for staging and directing therapy based on intraoperative gross characteristics (prognosis relies on gross features, not histological appearance).
  - **Stage I**: Encapsulated tumor, no gross/microscopic invasion. *Treatment*: Complete surgical excision alone.
  - **Stage II**: Macroscopic invasion into mediastinal fat/pleura OR microscopic capsular invasion. *Treatment*: Complete surgical excision + postoperative radiotherapy (to decrease local recurrence).
  - **Stage III**: Macroscopic invasion of the pericardium, great vessels, or lung. *Treatment*: Complete surgical excision + postoperative radiotherapy.
  - **Stage IVA**: Pleural or pericardial metastatic spread. *Treatment*: Surgical debulking, radiotherapy, and chemotherapy.
  - **Stage IVB**: Lymphogenous or hematogenous metastases. *Treatment*: Surgical debulking, radiotherapy, and chemotherapy.

Operative / Procedural Notes

- **Median Sternotomy**: Preferred approach for thymectomy. Vertical midline split provides adequate exposure of anterior mediastinal structures and allows complete thymus removal.
- **Video-Assisted Thoracoscopic Surgery (VATS)**: Usually performed with the patient in the **lateral decubitus position** using three thoracic ports. Can be used for biopsies or resections.
- **Anterior Mediastinotomy (Chamberlain Procedure)**: Parasternal incision (typically 2nd or 3rd intercostal space) to access the anterior mediastinum, primarily for biopsy of masses inaccessible by cervical approach.
- **Cervical Mediastinoscopy**: Incision made above the suprasternal notch, passing the scope anterior to the trachea to biopsy paratracheal and subcarinal lymph nodes.
- **Clamshell Incision**: Bilateral thoracosternotomy (horizontal incision at the 4th intercostal space bisecting the sternum) utilized for massive bilateral exposure of pleural cavities and the central mediastinum.

Complications / Prognosis

- **Thymoma Recurrence**: Thymomas are indolent tumors that may take ≥10 years to recur; short-term follow-up is inadequate to depict relapses. Preponderance of evidence shows all thymomas, except completely encapsulated stage 1 tumors, benefit from adjuvant radiation.

- **Post-Treatment Changes**: "Thymic rebound hyperplasia" can present as a symmetric, bilobed anterior mass on CT following chemotherapy for conditions like Hodgkin lymphoma in children.

Past-Paper High Yield

- **Imaging for Traumatic Aortic Transection**: Computed Tomography Angiography (CTA) is the definitive modality of choice in stable/semi-stable blunt chest trauma.
  - *Trap*: A conventional angiogram is historically the gold standard but has been completely replaced. CXR is only a screening tool (shows widened mediastinum). TEE is reserved for unstable patients who cannot leave the trauma bay.
- **Thymoma Location & Associations**: Classically an **Anterior** mediastinal mass. Associated strongly with muscle weakness (Myasthenia Gravis). *Trap*: Do not fall for options labeling it a posterior mass.
- **Most Common Masses Overall**: Metastatic tumors and lymphomas (lymphadenopathy).
- **Neurogenic Tumors (e.g., Ganglioneuroblastoma, Schwannoma)**: Overwhelmingly arise in the **Posterior** mediastinum. *Exam Application*: When asked which mass is "LEAST likely to cause a middle mediastinal mass," select a neurogenic tumor (e.g., Ganglioneuroblastoma, Neuroblastoma).
- **Classic Middle Mediastinal Lesions**: Lymphoma, Bronchogenic cyst.

Memory Pearls

- **Anterior Mediastinum "Ts"**: Thymoma, Teratoma, Thyroid (ectopic), Terrible lymphoma.
- **Middle Mediastinum = Tubes and Nodes**: Trachea, heart/aorta, bronchogenic cysts, esophageal cysts, and lymphadenopathy.
- **Posterior Mediastinum = Nerves**: Neurogenic tumors rule this compartment (Schwannomas, Neuroblastomas, Ganglioneuromas).

Thoracic trauma

Core Concepts

- **Mortality:** Thoracic trauma is responsible for >70% of all deaths following road traffic accidents (RTAs). Blunt chest trauma is fatal in 10% of isolated cases, rising to 30% if multi-system injuries are present. Penetrating wound mortality ranges from 3% (stab wounds) to 15% (gunshot wounds).
- **ATLS Primary Survey Priority:** A tension pneumothorax is an immediate, catastrophic threat to life. It severely impairs both ventilation and venous return and must be identified and decompressed during the **'Breathing'** assessment, before progressing to 'Circulation'.
- **Pathophysiology of Tension Pneumothorax:** A one-way tissue valve allows air to enter the pleural space during inspiration but traps it during expiration. The massive build-up of pressurized air pushes the mediastinum and trachea **away** from the affected side. This high intrathoracic pressure compresses the superior vena cava (SVC), severely impeding venous return to the right atrium and precipitating obstructive shock.
- **Pathophysiology of Flail Chest:** Occurs when 2 or more adjacent ribs are fractured in 2 or more places, creating a free-floating chest wall segment. This results in **paradoxical respiration** (the

flail segment moves inward during inspiration and outward during expiration). Its primary clinical significance is the almost universal presence of an underlying **pulmonary contusion**.

- **Pediatric Thoracic Trauma:** Due to extreme rib cage elasticity, children can sustain severe, life-threatening intrathoracic organ injuries *without* sustaining any rib fractures.

Diagnosis / Clinical Features

- **Pneumothorax:**
  - Decreased, weak, or absent breath sounds on the affected side.
  - Hyperresonant percussion note (trapped air).
  - Decreased tactile vocal fremitus (air acts as a barrier to sound transmission).
  - Decreased unilateral chest wall movement.
- **Tension Pneumothorax:**
  - Profound hypotension and hemodynamic collapse (obstructive shock).
  - **Contralateral tracheal deviation** (pushed *away* from the affected side).
  - Distended, engorged neck veins (due to SVC compression/impaired venous return). *Note: Neck veins will not be collapsed.*
  - High peak airway pressures in mechanically ventilated patients.
- **Hemothorax:**
  - Decreased/absent breath sounds.
  - **Dull** percussion note (fluid accumulation).
  - Signs of hypovolemic shock.
- **Cardiac Tamponade:**
  - Presents with **Beck's triad**: Muffled heart sounds, elevated JVP (distended neck veins), and hypotension/pulsus paradoxus.
- **Pulmonary Contusion:**
  - Often initially asymptomatic but progresses rapidly to respiratory failure. Look for physical signs of direct impact like the "seat-belt sign" (chest bruising). Presents with increased secretions and hemoptysis.
- **Diaphragmatic Rupture:**
  - Usually on the left hemidiaphragm (blunt trauma).
  - Herniation of the stomach causes massive dilation/volvulus, leading to left lung collapse, mediastinal shift to the right, and bowel sounds auscultated in the chest.

Investigations

- **Tension Pneumothorax Diagnostics:** Strictly a **clinical diagnosis**. Waiting to obtain a Chest X-Ray (CXR) delays life-saving needle decompression and is strictly contraindicated.
- **Imaging Findings:**
  - **Simple Pneumothorax:** CXR shows loss of lung markings and a visible pleural edge.

- **Hemothorax:** Erect CXR shows a fluid-air meniscus and blunting of the costophrenic angle; massive hemothorax shows complete "whiteout" of a hemithorax.
- **Pulmonary Contusion:** CXR reveals hazy, ill-defined opacification/consolidation.
- **Aortic Rupture/Tear:** CXR shows a **widened mediastinum**. Prompt aortography is indicated.
- **Diaphragmatic Rupture:** CXR shows an elevated hemidiaphragm or gas-filled bowel loops/stomach in the lower hemithorax. Up to 35% of patients initially have a normal or minimally abnormal CXR.

- **ECG / Cardiac Enzymes:** Used to monitor for myocardial contusion in blunt trauma.
- **Pericardial Window vs. Pericardiocentesis:** Subxiphoid pericardiocentesis can be diagnostic (negative deflection of QRS indicates epicardial contact), but a subxiphoid pericardial window performed in the OR is definitively preferred for tamponade in a surgical setting.

Management

- **Tension Pneumothorax:**
  - Immediate life-saving **needle thoracostomy (decompression)**, followed by definitive large-bore chest tube placement.
  - *Do not* wait for imaging. *Do not* prioritize IV fluids or inotropes (they do not fix the mechanical obstruction). *Do not* immediately intubate/ventilate with PEEP before decompression (positive pressure will exacerbate the tension physiology).
- **Massive Hemothorax:**
  - Initial definitive treatment is the prompt insertion of a **large-bore thoracostomy tube** (32-36F).
  - Combine simultaneously with aggressive **balanced blood product resuscitation** (1:1:1 ratio of PRBCs : FFP : Platelets). Avoid crystalloid-only resuscitation which worsens coagulopathy.
  - An autotransfusion system (filtering and reinfusing shed pleural blood) can be utilized.
- **Spontaneous Pneumothorax:** A primary spontaneous pneumothorax measuring 40% (large) in a symptomatic patient requires active management with a tube thoracostomy or needle aspiration, not just observation.
- **Persistent Air Leak:** If an air leak persists >3–5 days after chest tube placement, conservative management has failed. The best treatment is **Video-Assisted Thoracoscopic Surgery (VATS)** with bleb excision/stapling and mechanical/chemical pleurodesis.
- **Pulmonary Contusion / Flail Chest:**
  - Tx: Humidified oxygen, excellent pain control (narcotics/intercostal nerve blocks), aggressive pulmonary toilet (suctioning, deep breathing).
  - Employ central line fluid restriction (to avoid worsening alveolar edema/ARDS). Avoid restrictive rib belts.
- **Diaphragmatic Rupture:** Prevent gastric distension with urgent NG tube placement. Surgical repair requires a double-layer closure (approached via thorax or abdomen).
- **Transposition of the Great Arteries (TGA):** *Cross-yield concept:* TGA creates two parallel, unmixed circulations. It is rapidly fatal in the neonate without a shunt (PDA/ASD) and requires immediate intervention (PGE1 infusion, atrial septostomy).

Relevant Guidelines

- **Target Parameters in Initial Resuscitation:**
  - Keep O2 saturation >94%.
  - Keep systolic BP at a minimum of 110 mmHg.
- **Indications for Emergency Endotracheal Intubation:**
  - Apnea
  - Respiratory rate > 30 breaths/min
  - Profound shock
  - Inadequate ventilation (PaO2 < 60 mmHg, PaCO2 > 45 mmHg)
  - GCS < 8
- **Indications for Emergent/Urgent Thoracotomy (Hemothorax/Bleeding):**
  - Immediate initial chest tube output of **>1500 cc** (or >20 cc/kg).
  - Continuous, persistent bleeding of **>200 cc/hr for 2 to 4 hours**.
- **Acute Indications for Emergency Thoracotomy (General):**
  - Cardiac tamponade.
  - Acute hemodynamic deterioration/cardiac arrest in the trauma center.
  - Penetrating truncal trauma (resuscitative thoracotomy).
  - Vascular injury at the thoracic outlet.
  - Loss of chest wall substance.
  - Massive air leak (suggestive of major tracheobronchial injury).
  - Endoscopic/radiographic evidence of significant tracheal/bronchial injury.
- **Indications for Aortography (Suspected Aortic Rupture):**
  - First rib fracture accompanied by: brachial plexus deficit, absent radial pulse, pulsating supraclavicular mass, or a widened mediastinum.

Operative / Procedural Notes

- **Chest Tube Insertion:**
  - **Site:** The "safe triangle" in the mid- or anterior-axillary line, behind the pectoralis major, and **above the 5th rib** to prevent diaphragmatic or intra-abdominal injury.
  - **Technique:** Incision made along the *upper border* of the rib (avoids neurovascular bundle). Blunt dissection using a curved clamp and operator's finger.
  - **Positioning:** For a pneumothorax, target tube anterior-superiorly. For a hemothorax, target tube posterior-inferiorly. Secure with a U-stitch (purse-string) and confirm with CXR.
- **Cardiac/Great Vessel Surgical Approaches:**
  - **Median Sternotomy:** The preferred, standard, and most versatile incision for accessing the heart and great vessels in stable/semi-stable penetrating cardiac trauma.
  - **Emergency Department Resuscitative Thoracotomy (EDT):** Utilizes a left anterolateral approach. Seldom indicated; reserved for moribund patients or rapid deterioration without time

to reach the OR.

- **Myocardial Repair:** Ventricular lacerations are repaired using pledgetted nonabsorbable horizontal mattress sutures.

Complications / Prognosis

- **ARDS:** A highly lethal progression of severe pulmonary contusion; fluid restriction is a key preventative measure.
- **Exsanguination vs. Tamponade:** In penetrating cardiac trauma, pericardial rupture leads to rapid exsanguination into the thorax (mostly fatal before hospital arrival), whereas an intact pericardium creates tamponade (restricts bleeding, allowing survival to the ER).
- **Ventricular Penetrating Injuries:** Right ventricle is most commonly injured (43%), followed by the left ventricle (34%). Ventricular septal defect (VSD) is the most common intracardiac injury resulting from trauma.

Past-Paper High Yield

- **Trap:** Know the difference in percussion note! Pneumothorax is **hyperresonant**; Hemothorax is **dull**.
- **Trap:** In a tension pneumothorax, the trachea deviates **AWAY** from the affected side, NOT toward it.
- **Trap:** In a tension pneumothorax, tactile vocal fremitus is **decreased**, not increased.
- **Trap:** A chest tube output of 200 ml over an *entire 24-hour period* is minimal and easily managed conservatively. The criterion for thoracotomy is >200 ml/hr *per hour* for 2–4 hours. Volumes of 300 cc or 500 cc total do not require thoracotomy.
- **Testable Differential:** Elevated JVP with severe hypotension post-trauma = obstructive shock. If breath sounds are absent -> **Tension Pneumothorax**. If heart sounds are muffled and breath sounds are bilateral -> **Cardiac Tamponade**.
- **Clinical Prioritization:** Diagnosis of tension pneumothorax means immediate needle decompression; any choice offering a CXR, IV fluids, or inotropes first is the wrong answer.
- **Best Incision:** Penetrating pericardial trauma operating in the OR -> choose **Median Sternotomy**.

Memory Pearls

- **Beck's Triad (Cardiac Tamponade): 3 D's** - **D**istant (muffled) heart sounds, **D**istended jugular veins, **D**ecreased blood pressure.
- **Tension PTX pushes, Atelectasis pulls:** Tension pneumothorax pushes the mediastinum *away*, while simple atelectasis/lung collapse pulls the mediastinum *towards* the lesion.
- **Chest Tube Safe Zone:** "Above the rib, below the armpit, above the 5th." Always enter over the superior margin of the rib to avoid the intercostal neurovascular bundle (VAN: Vein, Artery, Nerve lie in the subcostal groove).

Thoracic aortic diseases

Core Concepts

- **Aortic Anatomy & Landmarks:**
  - **Aortic root:** Annulus to sinotubular junction; contains sinuses of Valsalva. (Embryology: Secondary heart field).
  - **Ascending aorta:** Sinotubular junction to brachiocephalic (innominate) artery. (Embryology: Neural crest).
  - **Aortic arch:** Origins of head and neck vessels. (Embryology: Neural crest).
  - **Descending thoracic aorta:** Left subclavian artery to diaphragm. (Embryology: Paraxial mesoderm).
  - **Abdominal aorta:** Diaphragm to iliac bifurcation (>90% of aneurysms occur infrarenally).
- **Vessel Wall Microstructure:** Intima (endothelium), Media (smooth muscle + elastic lamellae; primary site of dissection/cystic medial degeneration), Adventitia (connective tissue + vasa vasorum).
- **Pathologic Definitions:**
  - **Normal:** Mid-descending aorta is 26–28 mm.
  - **Dilation/Ectasia:** Enlargement up to 50% above normal.
  - **Aneurysm:** Enlargement > 50% above normal (diameter > 1.5x normal).
  - **True Aneurysm:** Involves all three intact layers (fusiform or saccular).
  - **False Aneurysm (Pseudoaneurysm):** Disruption of all three layers; extravascular blood is contained only by adventitia or surrounding connective tissue. High risk of free rupture.
- **Acute Aortic Syndrome (AAS) Spectrum:**
  1. **Aortic Dissection (AD):** Intimal tear allows blood to dissect into the media, separating true (TL) and false lumens (FL) via an intimal flap.
  2. **Intramural Hematoma (IMH):** Rupture of vasa vasorum leading to localized medial hemorrhage without a detectable intimal tear.
  3. **Penetrating Atherosclerotic Ulcer (PAU):** Plaque ulcerates through the internal elastic lamina into the media.
- **Crawford Classification of Thoracoabdominal Aortic Aneurysms (TAAA):**
  - *Type I:* Most of descending thoracic + upper abdominal.
  - *Type II:* Most of descending thoracic + most of abdominal.
  - *Type III:* Distal descending thoracic + abdominal.
  - *Type IV:* Most/all of abdominal aorta (below diaphragm).

Diagnosis / Clinical Features

- **Thoracic Aortic Aneurysm (TAA):**
  - Majority are asymptomatic (incidental finding on imaging).

- *Compressive mass effects:* SVC syndrome, hoarseness (recurrent laryngeal nerve), bronchial obstruction (dyspnea/cough), dysphagia.
- *Aortic root involvement:* Aortic regurgitation (diastolic murmur).

- **Acute Aortic Dissection:**
  - **Classic Presentation:** Abrupt, tearing/ripping/stabbing chest or interscapular back pain (90% of patients).
  - **Physical Exam:**
    - Pulse deficit or SBP differential > 20 mmHg between arms (present in 19-34% of Type A).
    - New diastolic murmur of aortic regurgitation (32-76%).
    - *Note:* The classic triad is absent in >50% of cases; absence does not rule out AD.
  - **End-Organ Malperfusion Syndromes (33% of Type A cases):** Hemiplegia/stroke, paraplegia (spinal cord ischemia), mesenteric ischemia, acute kidney injury/renal infarction, lower extremity ischemia.
  - **Catastrophic Complications:** Cardiac tamponade (pericardial leak causing cardiogenic shock), acute free rupture (hypovolemic shock), acute myocardial infarction (usually inferior MI from right coronary ostial involvement, 1-7%).

Investigations

- **Laboratory Tests:**
  - **D-dimer:** Highly sensitive; useful for ruling out acute dissection in low-risk patients.
  - Troponin, ECG, CRP, IL-6 to rule out ACS or evaluate end-organ injury.
- **Imaging:**
  - **Computed Tomography Angiography (CTA):** Gold standard and most widely used tool (Sensitivity/Specificity ~100%). Identifies true/false lumen and intimal flap.
  - **Transesophageal Echocardiography (TEE):** Modality of choice for hemodynamically unstable patients or those with absolute contrast contraindications (severe renal failure). Excellent for root and ascending aorta.
  - **Chest X-Ray:** May show widened mediastinum, but up to 16% of dissections have a completely normal CXR. *A normal CXR can never rule out acute aortic dissection.*
  - **MRA:** Highly accurate but highly time-consuming (45-60 mins); unsuited for acute, unstable situations.

Management

- **Aneurysm Medical Management (Anti-Impulse Therapy):**
  - Target low-normal blood pressure, strict smoking cessation, avoid heavy lifting. *No medications reliably slow aneurysm growth.*
- **Dissection Classification & Treatment Paradigms:**
  - **Stanford Type A (involves ascending aorta):**
    - *Management:* **Emergency open surgery** (extreme untreated mortality: 1-2% per hour for first 48h).

- **Stanford Type B (does NOT involve ascending aorta):**
  - *Uncomplicated Management:* **Medical therapy** (strict BP and HR control).
  - *Complicated Management:* **Endovascular repair (TEVAR)** indicated for rupture, rapid expansion, persistent leak, or end-organ malperfusion.

Relevant Guidelines

- **Surgical Intervention Thresholds for Aneurysms (ACC/AHA/ESC Consensus):**
  - ≥ 5.5 cm: Standard threshold for surgery in sporadic/degenerative TAA (Class I).
  - ≥ 5.0 cm: Threshold for Marfan syndrome (Class I) or Bicuspid Aortic Valve (Class IIa if experienced center).
  - ≥ 4.5 cm: Threshold for Marfan patients with additional high-risk features (family history of dissection, rapid growth) at experienced centers.
  - *Other absolute indications:* Symptomatic aneurysm, traumatic rupture, pseudoaneurysm, or growth rate ≥ 1 cm/year.
  - *Hinge Point:* Rupture risk increases drastically once TAA reaches **6.0 cm** (non-linear inflection point).
- **Acute Aortic Dissection Medical Management Pathway:**
  1. **Rate Control FIRST:** IV beta-blockade (esmolol, labetalol) to reduce dP/dt (wall shear stress). Target Heart Rate **< 60 bpm**. (Use diltiazem/verapamil if beta-blockers contraindicated).
  2. **Pain Control:** IV opiates (morphine/fentanyl) titrated to complete relief to blunt sympathetic tone.
  3. **Blood Pressure Control SECOND:** If SBP > 120 mmHg *after* HR is controlled, add IV vasodilators (nitroprusside, nicardipine). Target SBP **< 120 mmHg** (lowest BP that safely maintains end-organ perfusion).
  4. **Hypotension/Shock Management:** Administer fluids; titrate to MAP of 70 mmHg. If persistent, carefully start vasopressors and immediately evaluate for tamponade/rupture.

Operative / Procedural Notes

- **Organ Protection during Open Thoracic Repair:**
  - **Left Heart Bypass (LHB):** Cannulation of left atrium to femoral artery bypasses the cross-clamp, maintaining distal perfusion to kidneys, viscera, and spinal cord.
  - **Selective Perfusion:** Direct catheterization of critical intercostal and visceral arteries during reconstruction prevents ischemic complications (paraplegia).
- **Key Surgical Techniques:**
  - **Bentall Procedure:** Total root replacement using a composite valved Dacron graft. Requires mobilization and reimplantation of coronary ostia.
  - **Valve-Sparing Root Replacement (David procedure):** Replaces ascending aorta/root while preserving the native aortic valve.
  - **Elephant Trunk / Frozen Elephant Trunk:** Staged arch repair. Stage 1: Arch replacement with a free-floating distal graft extension left in the descending aorta. Stage 2: Distal segment serves

as a safe "landing zone" for downstream open or endovascular (TEVAR) repair.
    - **TEVAR (Thoracic Endovascular Aortic Repair):** Stent-graft deployed retrogradely from the femoral artery. Minimally invasive alternative to open descending aorta repair.

Complications / Prognosis

- **Spinal Cord Ischemia:** Clamping of the thoracic aorta compromises flow to critical intercostal arteries (specifically the anterior spinal artery / Artery of Adamkiewicz), resulting in anterior cord syndrome (paraplegia).
- **Intra-Aortic Balloon Pump (IABP) Complications:** A common adjunct in cardiothoracic surgery/shock, but its large-bore catheter obstructs flow in the femoral artery. The most frequent complication is severe **limb ischemia**.
- **Dissection Mortality:** Unoperated Type A dissection mortality approaches 50% out-of-hospital; 1-2% hourly increase for survivors.

Past-Paper High Yield

- **Aortic Dissection Management:** Stanford Type A requires **urgent surgical intervention**, whereas uncomplicated Stanford Type B is primarily managed **medically** (strict BP and HR control). Traps often wrongly state both require immediate surgery.
- **Aneurysm Etiology by Location:**
    - *Descending Thoracic Aneurysms (and AAAs):* **Atherosclerosis** is overwhelmingly the most common underlying cause.
    - *Ascending Thoracic Aneurysms:* Associated with cystic medial necrosis, Marfan syndrome, and bicuspid valves.
    - *Dissection:* Associated primarily with **Hypertension**.
- **Spinal Cord Complications: Paraplegia** is the most classically feared and devastating specific complication of thoracic aortic repair due to anterior spinal artery (Artery of Adamkiewicz) ischemia during aortic cross-clamping.
- **IABP Complications: Limb ischemia** is the most frequent complication of intra-aortic balloon pump placement due to the large-bore catheter obstructing femoral artery blood flow. Hematoma/bleeding are less common/less devastating, and strokes/arrhythmias are not direct common complications of the pump itself.
- **AAA Localization & Repair:** The vast majority (**>90%**) of abdominal aortic aneurysms (including ruptured ones) are **infrarenal**. Ruptured AAAs require emergent (immediate) treatment (not "within 12 hours"). Open surgery is *not* universally superior; EVAR is widely used based on anatomy.

Memory Pearls

- **Embryology rules pathology:** The differing embryologic origins (neural crest vs. paraxial mesoderm) explain why genetic connective tissue diseases (Marfan) dictate *ascending* pathology, while degenerative disease (atherosclerosis) dictates *descending* pathology.
- **Sequence of AD meds: H**eart rate before **B**lood pressure (Beta-blocker then vasodilator) to prevent reflex tachycardia and increased aortic shear stress.

- **TEVAR:** Minimally invasive, used for *Type B* complications, but still risks paraplegia if it covers the critical intercostal feeders (Artery of Adamkiewicz).

Coronary artery disease

Core Concepts

- **Aetiology:** Atherosclerosis accounts for >90% of ischemic heart disease. Other causes include embolization, coronary spasm, vasculitis, ostial stenosis, severe left ventricular hypertrophy (LVH), and congenital anomalies.
- **Pathophysiology of Acute Coronary Syndrome:** Plaque fissure/rupture → Platelet adhesion → Platelet activation → Platelet aggregation → Thrombotic occlusion.
- **Normal Coronary Anatomy:**
  - **Left Main (LM):** Bifurcates into the Left Anterior Descending (LAD) and Left Circumflex (LCx).
  - **LAD:** Supplies the anterior wall of the left ventricle and anterior portion of the septum. Gives off diagonal branches (D1, D2).
  - **LCx:** Constantly supplies the lateral wall of the left ventricle. Gives off marginal branches (M1, M2).
  - **Right Coronary Artery (RCA):** Predominantly supplies the right ventricle. Gives off acute marginal branches.
- **Coronary Dominance:** Defined by which artery gives rise to the Posterior Descending Artery (PDA) supplying the posterior third of the interventricular septum.
  - **Right-Dominant (~85%):** RCA supplies the PDA and usually the AV nodal artery.
  - **Left-Dominant (~10%):** LCx gives rise to the PDA.
  - *Clinical Consequence:* In a left-dominant heart, occlusion at the origin of the LCx causes ischemic necrosis in **both the lateral left ventricular wall and the posterior portion of the septum**.

Diagnosis / Clinical Features

- **Risk Factors:**
  - *Controllable:* Hypertension, hyperlipidemia, smoking, physical inactivity, obesity, diabetes mellitus, stress/anger.
  - *Uncontrollable:* Age, male sex, hereditary predisposition, race.
- **Diagnosis:** Relies on clinical history, physical examination, ECG findings, and cardiac enzymes.

Investigations

- **First-line:** ECG, cardiac enzymes, chest X-ray, fasting blood sugar, serum lipids.
- **Functional Testing:** Treadmill Test (TMT), stress or pharmacologic myocardial perfusion studies.
- **Imaging:** Cardiac CT scan, Diagnostic Coronary Angiography (the gold standard for delineating exact coronary anatomy and stenoses).

Management

- **Medical Therapy:** Nitrates, beta-blockers, calcium channel blockers (especially indicated in coronary spasm), risk factor modification.
- **Dual Antiplatelet Therapy (DAPT) Mechanism of Action:** Synergistically blocks the final common pathway (Glycoprotein IIb/IIIa receptor activation) to prevent platelet aggregation.
  - *Aspirin (ASA):* Inhibits Cyclooxygenase (COX), preventing the synthesis of Thromboxane A2 (TXA2).
  - *Clopidogrel:* Selectively blocks the P2Y12 (ADP) receptor.
- **Indications for Surgical Revascularization (CABG vs. PCI):**
  - **Left main stem disease:** Highly critical; non-obstructive lesions (e.g., 40%) can be managed medically, but critical stenosis is a Class I indication for CABG due to high risk of sudden death.
  - **Triple vessel disease:** CABG yields significant survival benefit over PCI/medical therapy in complex three-vessel disease, **especially when LVEF < 50%**.
  - **Unstable angina** failing medical therapy.
  - Life-threatening complications of myocardial infarction.
  - Complications of Percutaneous Transluminal Coronary Angioplasty (PTCA).
  - Significant coronary artery anomalies.
  - *Note:* Single-vessel disease (e.g., RCA) or isolated two-vessel disease with preserved ejection fraction is typically treated with PTCA/PCI or medically.

Operative / Procedural Notes

- **Coronary Artery Bypass Grafting (CABG) - Traditional:**
  - **Incision:** Median sternotomy (divided with a saw) provides the best overall access to the heart.
  - **Cardiopulmonary Bypass (CPB):** Requires an aortic cannula (ascending aorta) for arterial return and bicaval cannulation (SVC and IVC via right atrium) to divert venous return.
  - **Cardioplegia:** Used to arrest the heart for motionless suturing.
  - **Anastomosis:** Microvascular continuous suturing (7-0 or 8-0 polypropylene) performed with specialized instruments (e.g., Potts scissors).
- **Conduit Selection for CABG:**
  - *Arterial Conduits:* Left Internal Thoracic Artery (LITA/LIMA), Right Internal Thoracic Artery (RITA/RIMA), and Radial Artery (harvested from the medial forearm). Highly resilient against high arterial pressures.
  - *Venous Conduits:* Saphenous vein grafts (harvested from the leg), utilized as reversed free grafts from the aorta to target vessels.
  - *Total Arterial Revascularization:* Uses combinations of LITA, RITA, and Radial artery in complex configurations (e.g., Y-grafts, sequential grafts, composite LITA-RITA grafts) without utilizing veins.
- **Off-Pump Coronary Artery Bypass (OPCAB):**
  - Performed on a beating heart to avoid CPB complications.

- **Pharmacology:** Esmolol and Adenosine used to slow or briefly arrest the heartbeat (~20 seconds).
- **Instrumentation:** Suction-based mechanical stabilizers (e.g., Medtronic Octopus device) limit target vessel movement to <1 mm.

- **Percutaneous Transluminal Coronary Angioplasty (PTCA):**
  - Involves balloon angioplasty, stenting, and/or rotational atherectomy (Rotablator for grinding calcified plaques).

Complications / Prognosis

- **Saphenous Vein Graft Failure Timelines:**
  - **< 30 days:** Technical error during surgery.
  - **1 month to 1 year: Intimal hyperplasia** (due to smooth muscle cell proliferation triggered by high arterial pressures). *This is the primary cause of narrowing at one year.*
  - **> 1 to 5 years:** Atherosclerosis.

Past-Paper High Yield

- **CABG Candidacy:** The "classic CABG candidate" is a patient with unstable angina, complex three-vessel disease, and a reduced left ventricular ejection fraction (e.g., LVEF 35%). This patient achieves a distinct survival benefit with CABG over PCI.
- **Left-Dominant Ischemia Trap:** If a left-dominant patient occludes the LCx ostium, both the lateral LV wall and the posterior septum infarct. (The anterior septum and apex remain protected by the LAD).
- **ALCAPA (Bland-White-Garland Syndrome):** The most lethal congenital coronary anomaly. The left main coronary artery originates from the pulmonary artery. As pulmonary resistance drops after birth, a "coronary steal" phenomenon occurs, causing desaturated, low-pressure flow to drain backward into the PA, leading to massive MI and infant heart failure if untreated.
- **Ductal-Dependent Lesions:** Intravenous infusion of **Prostaglandin E1 (Alprostadil)** is the lifesaving initial medical management to maintain ductus arteriosus patency. *Trap: High-concentration oxygen lowers pulmonary resistance and promotes PDA closure, worsening systemic ductal-dependent lesions.*
- **Cardiogenic Shock Contraindications: Pneumatic antishock garments (MAST suits)** are detrimental in cardiogenic shock. Although they increase venous return, they drastically increase systemic vascular resistance and LV afterload, failing the struggling left ventricle. Intra-Aortic Balloon Pumps (IABP), Dobutamine, and Nitroprusside are helpful.
- **Tetralogy of Fallot (ToF) Hemodynamics:** Due to the right ventricular outflow tract obstruction (pulmonary stenosis), the pulmonary vascular bed is protected from high pressures. Therefore, **persistent pulmonary hypertension is extremely unlikely** (and cannot be seen in ToF), even though Right Ventricular Hypertrophy (RVH) is a hallmark.
- **Visceral Peripheral Aneurysms:** The **Splenic artery** is by far the most common site (~60%), characteristically affecting women of childbearing age. The hepatic artery is second.

Memory Pearls

- **Dominance Rule:** Whichever artery gives off the PDA = Dominant. (RCA = 85%, LCx = 10%).
- **Graft Failure Clock:** < 30 days = Technical; 1-12 months = Intimal Hyperplasia; > 1 year = Atherosclerosis.
- **DAPT Synergy:** ASA (COX) + Clopidogrel (P2Y12) = Blocked GP IIb/IIIa.
- **ALCAPA:** Left coronary from Pulmonary Artery → Infant MI / Lethal Steal Syndrome.
- **ToF lung protection:** Pulmonary stenosis = no pulmonary hypertension.

Surgical management of lung infections

Core Concepts

- **Empyema Thoracis**: Bacterial invasion of the pleural space resulting in the accumulation of frank pus.
  - **Primary etiology**: Parapneumonic (65%), occurring secondary to pneumonia.
  - **Other etiologies**: Post-traumatic, post-surgical (esophageal or pulmonary resection), subphrenic abscess.
  - **Bacteriology**: Pre-antibiotic era dominated by *Streptococci* & *Pneumococci*. Currently, *Staph. aureus* is highly prevalent (causes 90% of pediatric empyemas), alongside Gram-negatives and anaerobes (which cause 90% of adult empyemas).
- **Lung Abscess**: A subacute pulmonary parenchymal infection forming a cavity visible on CXR.
  - **Timeframe**: Acute (< 6 weeks) vs. Chronic (> 6 weeks).
  - **Primary (Most common)**: Due to aspiration or post-pneumonic complications.
  - **Secondary**: Secondary to obstructing carcinoma, COPD, septicemia/metastatic spread, foreign body aspiration, or pulmonary infarctions.
  - **High-Risk Patients**: Alcohol abuse, seizure disorders (epilepsy), drug abuse, old TB, COPD.
  - **Bacteriology**: 75–80% anaerobic (*Bacteroides fragilis, Fusobacterium, Peptostreptococcus, Prevotella*). Aerobes (*Klebsiella, Pseudomonas*) are common in obstructing or nosocomial infections.

Diagnosis / Clinical Features

Empyema

- **Acute**: Pleuritic chest pain, fever, shortness of breath, tachycardia, and restricted chest wall excursion. Always suspect empyema in a patient with pneumonia whose symptoms are prolonged.
- **Anaerobic infections**: Often have an indolent presentation.
- **Physical Exam**: Toxic appearance, tachypnea, decreased air entry, dullness on percussion.
- **Chronic**: Clubbing, anemia, significant weight loss.

Lung Abscess

- **Symptoms**: Intermittent fever, night sweats, and chills.

- **Pathognomonic sign**: Production of purulent, **foul-smelling sputum** is highly suggestive of anaerobic involvement.
- **Typical Locations (Aspiration)**:
  - Superior segment of the Right Lower Lobe (RLL).
  - Lateral part of the posterior segment of the Right Upper Lobe (RUL).
  - Superior segment of the Left Lower Lobe (LLL).

Investigations

- **Blood Tests**: CBC (leukocytosis with a left shift), ↑ CRP, ↑ ESR.
- **Imaging**:
  - **CXR**: Shows effusion, pleural thickening, or an air-fluid level. A straight horizontal air-fluid line typically indicates pyopneumothorax (gas + fluid in pleural space) or a cavitating parenchymal lesion. Lateral decubitus views help differentiate free-flowing from loculated effusions.
  - **CT Scan (Gold Standard for Staging/Differentiation)**:
    - **Empyema**: Displays the "split pleura" sign, loculations, and compression of underlying lung.
    - **Lung Abscess**: Thick-walled parenchymal cavity with an air-fluid level, forming an acute angle with the chest wall.
- **Bronchoscopy**: **Mandatory** in the workup of a lung abscess to obtain cultures, rule out endobronchial tumors/obstruction, and assess suitability for internal drainage.
- **Thoracocentesis**: Required for pleural fluid analysis to differentiate simple effusion from empyema (see Guidelines section).

Relevant Guidelines

American Thoracic Society (ATS) Classification of Empyema

- **Stage 1 (Exudative/Acute)**: Uncomplicated. Swelling of pleural membranes with increased permeability. ≥80% can be managed conservatively (chest tube/thoracentesis + antibiotics).
- **Stage 2 (Fibropurulent/Transitional)**: Heavy fibrin deposits begin to form loculations.
- **Stage 3 (Organizing/Chronic)**: Ingrowth of fibroblasts and deposition of collagen (thick pleural peel). 80% of these require surgical thoracotomy/decortication.

Diagnostic Criteria for Empyema / Complicated Parapneumonic Effusion (PPE)

- **Simple PPE**: pH > 7.20.
- **Complicated PPE / Frank Empyema**: Requires immediate drainage. Defined by:
  - pH < 7.20
  - Glucose < 40 mg/dL (or < 2.2 mmol/L)
  - LDH > 1000 IU/dL
  - Positive Gram stain or culture (positive in 50% of cases)
  - Specific gravity > 1.018
  - WBC > 500 cells/mm³

- Protein > 2.5 g/dL

Management

Empyema

1. **Medical**: Broad-spectrum antibiotics (3rd generation cephalosporin + clindamycin) until culture and sensitivity results return.
2. **Drainage**: Thoracocentesis (Stage 1) or large-bore chest tube insertion.
3. **Intrapleural Adjuncts**: Fibrinolytics (Streptokinase, Urokinase, or tPA) to break down fibrin loculations. Intrapleural DNase to reduce pus viscosity.
4. **Surgical Intervention**: VATS (ideal for Stage 2) or open thoracotomy with decortication (required for Stage 3).

Lung Abscess

1. **Medical (Primary Therapy)**: 80–90% respond to medical treatment alone. Antibiotics for **6–8 weeks** (Flagyl or Clindamycin for anaerobes; Gentamicin or 3rd gen cephalosporins for aerobes).
2. **Internal Drainage**: Chest physiotherapy, bronchoscopy (transbronchial catheter).
3. **External Drainage Indications**:
   - Persistent sepsis or increasing cavity size despite antibiotics.
   - Failure to wean from mechanical ventilation.
   - Contralateral lung soiling.
   - Abscess cavity > 4 cm and under tension on CXR.
4. **Surgical Resection (Lobectomy)**: Needed in ~30% of patients (see Operative Notes).

Operative / Procedural Notes

- **Chest Tube Thoracostomy**:
  - **Landmarks**: 2nd intercostal space (midclavicular line) or 5th/7th intercostal space (mid/anterior axillary line).
  - **Technique**: Incision must be made along the **upper border of the rib** to avoid the neurovascular bundle (which runs along the lower border). Use blunt dissection with a curved clamp and operator's finger.
  - **Equipment**: Requires a **large-bore tube (32 or 36 Fr)** for empyema to prevent the thick, viscous pus from clogging the tube. Secure with a U-stitch.
- **VATS (Video-Assisted Thoracoscopic Surgery)**: Utilizes a 3-port triangulation setup (two anterior/mid-axillary, one posterior). Highly effective for debridement and early decortication in Stage 2 empyema.
- **Monaldi Procedure**: Open pneumonostomy used for external drainage of a lung abscess in patients unfit for resection.
- **Lobectomy**: The standard definitive surgical procedure for lung abscesses requiring resection.

Complications / Prognosis

- **Empyema Mortality**: Ranges from 6% to 24%.
- **Lung Abscess Mortality**: 2.5% for community-acquired; up to 66% for nosocomial infections. Highest mortality is associated with *Pseudomonas* or Gram-negative organisms, underlying diseases, and cavity size > 6 cm.
- **Indications for Definitive Surgery in Lung Abscess**:
  - *Acute complications*: Bronchopleural fistula, secondary empyema, massive hemoptysis.
  - *Chronic complications*: Persistent symptoms despite long-term antibiotics, suspicion of underlying carcinoma, or a persistent cavity **> 6 cm** after antibiotic therapy.

Past-Paper High Yield

- **Pleural fluid analysis**: Commit the cutoffs to memory. A pleural fluid **pH < 7.20** or **Glucose < 40 mg/dL** mandates surgical/tube drainage.
- **Anatomical trap**: Chest tube insertion must strictly go over the **upper border** of the lower rib to avoid the intercostal nerve/artery/vein.
- **Procedural sequencing**: **Bronchoscopy is mandatory** in lung abscesses to rule out an obstructing endobronchial carcinoma as the root cause.
- **Diagnostic imaging**: Know how to differentiate on CT — Empyema = "split pleura" sign, lenticular shape, obtuse angle with chest wall. Lung abscess = thick irregular wall, acute angle with chest wall.
- **Tube sizing**: Always use a large-bore chest tube (**32–36 Fr**) for empyema; standard smaller tubes will clog with thick fibropurulent debris.
- **Abscess locations**: Be ready to identify aspiration pneumonia/abscess locations based on gravity (Superior segments of lower lobes, posterior segments of upper lobes).

Memory Pearls

- **Empyema Stages**: 1 = Exudative (Fluid), 2 = Fibropurulent (Fibrin), 3 = Organizing (Fibroblasts/Peel).
- **Complicated PPE criteria**: "pH low, Gluc low, LDH high" -> Put a tube in!
- **Lung Abscess Bugs**: Anaerobes smell bad (foul sputum).
- **Lung Abscess Surgery Trigger**: Rule of 6s (Antibiotics for **6**-8 weeks; Surgery if persistent cavity > **6** cm).

Lung cancer surgical aspects

Core Concepts

- **Epidemiology:** Lung cancer is the leading cause of cancer-related death. More than 70% of patients present with incurable advanced disease (Stages IIIB or IV).
- **Overall Prognosis:** The overall 5-year survival rate is notoriously poor, historically hovering around **14-15%**.

- **Small-Cell Lung Cancer (SCLC):**
  - Accounts for 20% of lung cancers; strongly associated with smokers.
  - Arises centrally near the hilum from embryonic neural cells.
  - Highly aggressive; primarily a systemic disease at presentation.
  - **Treatment:** Heavily reliant on chemotherapy. Rarely amenable to surgery due to wide dissemination by the time of diagnosis (5-year survival < 10%).
- **Non-Small-Cell Lung Cancer (NSCLC):**
  - Accounts for 80% of lung cancers and makes up the vast majority of surgically treated cases.
  - Subtypes: Adenocarcinoma (30-50%), Squamous cell carcinoma (20-35%), Large cell carcinoma (4-15%).
  - Molecular classification (e.g., EGFR, EML4-ALK) is mandatory for adenocarcinomas to guide targeted therapies.

Diagnosis / Clinical Features

- **Bronchopulmonary Symptoms:** Cough (80%), dyspnea (60%), post-obstructive pneumonia, hemoptysis.
- **Advanced Local Signs:** Hoarseness (recurrent laryngeal nerve), dysphagia, Superior Vena Cava (SVC) syndrome.
- **Paraneoplastic Syndromes:** More common with SCLC. Includes hypercalcemia, ectopic PTH, Cushing's syndrome, SIADH, Eaton-Lambert syndrome, and Hypertrophic Osteoarthropathy (HOA).
- **Pancoast (Superior Sulcus) Tumor:**
  - Apical lung tumor directly invading the apical parietal pleura and adjacent thoracic inlet structures (brachial plexus, subclavian vessels, stellate ganglion).
  - Presents with Pancoast Syndrome (shoulder/arm pain) and potentially Horner's syndrome.

Investigations

- **Screening:** In high-risk populations, Low-Dose CT (LDCT), sputum cytology, and annual CXR shift early-stage detection from 15% to 40% and improve 5-year survival from 13% to 35%.
- **Diagnostic Imaging:**
  - **CXR:** Spiculated, non-calcified, notched appearance.
  - **CT Chest/Abdomen:** Assesses primary tumor, mediastinum, liver, and adrenals (sensitivity ~65%).
  - **PET Scan:** Highly sensitive/specific (~90%) for metabolic mapping of metastasis and nodal involvement.
  - **MRI Brain:** Indicated for staging in all patients Stage IIB and above.
- **Tissue Diagnosis:** The gold standard. Achieved via bronchoscopy, EBUS (Endobronchial Ultrasound) for mediastinal nodes, CT-guided percutaneous FNA (peripheral tumors), or VATS/Thoracotomy.
- **Preoperative Physiological Assessment (Fitness for Surgery):**
  - Requires PFTs (Pulmonary Function Tests) ± blood gas analysis and perfusion scanning.

- **Surgical Cut-offs:** FEV_1 < 800 ml or DLCO < 60% predicted generally precludes major resection.
- **Post-operative Goal:** Predicted post-operative FEV_1 ≥ 1000 ml.

Relevant Guidelines

- **NSCLC Staging Criteria (TNM Classifications from slides):**
  - **T1:** ≤ 3 cm, surrounded by lung/visceral pleura, limited to lobar bronchus (no proximal invasion).
  - **T2:** 3-7 cm, involves main bronchus (≥ 2 cm distal to carina), invades visceral pleura, or causes partial atelectasis/obstructive pneumonitis (not entire lung).
  - **T3:** > 7 cm, or direct invasion of chest wall (including Pancoast), diaphragm, mediastinal pleura, or parietal pleura. Main bronchus involvement < 2 cm from carina (no carinal invasion). Includes separate tumor nodule in the **same lobe**.
  - **T4:** Invades mediastinum, heart, great vessels, trachea, esophagus, vertebral body, or carina. Presence of malignant pleural effusion. Includes separate tumor nodule in a **different ipsilateral lobe**.
  - **N1:** Ipsilateral peribronchial/hilar nodes.
  - **N2:** Ipsilateral mediastinal/subcarinal nodes.
  - **N3:** Contralateral mediastinal/hilar nodes, or any scalene/supraclavicular nodes.
  - **M1:** Distant metastasis (brain, pericardium, contralateral lung, liver, adrenals, bone, skin).
- **NSCLC Stage-Based Treatment Pathways:**
  - **Stage I (IA/IB):** Surgical resection (Lobectomy + mediastinal nodal mapping). Adjuvant therapies are *not* beneficial. Definitive RT is used only if medically inoperable.
  - **Stage II (IIA/IIB):** Surgical resection followed by **adjuvant chemotherapy**. Adjuvant RT is added for close/positive margins or positive hilar nodes.
  - **Stage IIIA (N1 Disease, e.g., T3N1):** Surgery alone if resectable (no bulky nodes), followed by adjuvant chemotherapy.
  - **Stage IIIA (N2 Disease): Preoperative (induction) chemotherapy** followed by surgical resection is the standard of care. Medically inoperable cases receive definitive platinum-based chemoradiotherapy.
  - **Stage IIIB & IV:** Usually unresectable. Managed with concurrent chemoradiation (Stage 3B) or palliative intent (chemotherapy, targeted agents like VEGF/EGFR inhibitors).

Management

- **Multidisciplinary Approach:** Essential for formulating treatment plans mixing surgery, radiotherapy, chemotherapy, and targeted therapy.
- **Targeted Therapies (Advanced NSCLC):**
  - *VEGF inhibitors:* Bevacizumab.
  - *EGFR inhibitors:* Gefitinib, Erlotinib, Cetuximab.
  - *Proteasome inhibitors:* Bortezomib.
- **Palliative Care Interventions:**

- *Pleural catheter / Pleurodesis:* For recurrent malignant pleural effusions.
- *Airway stenting / Laser debulking:* For tracheobronchial compression/obstruction to improve ventilation.

Operative / Procedural Notes

- **Surgical Goals:** Complete en bloc resection (R0) removing the tumor and intrapulmonary lymphatic drainage without disrupting the tumor (prevents seeding).
- **Intraoperative Strategy:**
  - Check margins via intraoperative frozen section (bronchial, vascular). Widen excision if positive.
  - Sacrifice no anatomical structures until resectability is definitively confirmed.
- **Types of Resection:**
  - *Lobectomy:* Standard for peripheral Stage I/II cancers.
  - *Pneumonectomy:* Indicated for central cancers with hilar/fissure involvement or peripheral cancers extending across fissures.
  - *Sleeve Resection:* Removal of a bronchus segment with re-anastomosis to avoid a complete pneumonectomy and spare lung parenchyma.
- **Lymph Node Evaluation:** Intraoperative systematic mediastinal lymph node dissection is recommended. Extensive dissection shows improved 4-year overall survival compared to simple sampling.
- **Surgical Approaches:**
  - *Thoracotomy (Posterolateral):* 10-15 cm incision, traditional approach.
  - *VATS / Robotic:* Minimally invasive (stages I-IIIA). Utilizes a 4-6 cm utility incision plus ports. Associated with less post-op pain and a shorter hospital stay (3-4 days vs. up to 7 days).
- **Pancoast Tumor Surgery:**
  - *Pathway:* Induction chemoradiotherapy (CRT) → Restaging (mediastinoscopy must be LN negative) → Surgical Resection.
  - *Approaches:* Anterior trans-cervical (Dartevelle) for anterior vascular exposure, or Posterior (Shaw-Paulson).
  - *Absolute Contraindications for Surgery:* **Horner's syndrome** (indicates stellate ganglion/neural foramen/vertebral body involvement), extensive brachial plexus/subclavian invasion, mediastinal LN involvement (N2/N3), SVC syndrome, or distant metastasis.

Complications / Prognosis

- **Systemic Recurrence:** 40-60% of radically resected NSCLC patients develop distant metastases (bone marrow micrometastases are present in >30% of resectable cases).
- **Prognostic Factors (Stage I):** Tumor size, visceral pleura involvement, and histological type (Squamous cell has better 5-year survival [83%] than adenocarcinoma [69%] in T1N0 disease).
- **Bronchopleural Fistula & Empyema:** A feared complication post-pneumonectomy. Suspect if the patient develops a fever, copious sputum, and a dropping/changing air-fluid level in the post-pneumonectomy space on CXR.

Past-Paper High Yield

- **Survival Rates:** The overall 5-year survival for lung cancer is historically **14-15%**. Any claim that overall 5-year survival is high (e.g., 65%) is false. Early-stage localized surgery offers the best hope for cure.
- **Pancoast Tumors (Historical vs. Modern):** Historically, Pancoast tumors were predominantly **Squamous Cell Carcinomas** (~2/3 of cases). They account for only 3-5% of lung cancers. By definition, because they invade the chest wall/parietal pleura, they are automatically classified as **at least T3** (not T2). Surgical mortality in modern centers is well below 50%.
- **Superior Vena Cava (SVC) Syndrome:** Malignancy is the most common cause in adults. **Bronchogenic carcinoma** (especially small cell) is the absolute most frequent cause, followed by lymphoma.
- **Metastasis to the Pericardium: Metastatic tumors** to the pericardium are far more common than primary pericardial tumors. **Lung cancer (bronchogenic carcinoma)** and breast cancer are the most frequent culprits. Primary tumors (mesothelioma, pericardial sarcoma) are exceedingly rare.
- **Post-Pneumonectomy Air-Fluid Level Drop:** If a patient presents 5 days post-pneumonectomy with fever, copious sputum, and a dropping air-fluid level on CXR, they have a bronchopleural fistula. The **best next step is pleural fluid analysis and culture** (diagnostic thoracentesis) to identify the organism before blind chest tube drainage (which can cause severe mediastinal shift or contamination) or immediate thoracotomy.
- **Thoracic Outlet Differentiator:** Do not confuse a Pancoast tumor with a **Cervical Rib** (an extra rib arising from C7). A cervical rib predisposes to Thoracic Outlet Syndrome (neurovascular compression), not lung malignancy.
- **Esophageal Cancer Resectability (Thoracic parallel):** In esophageal staging, regional lymph node involvement (N1-3) does *not* preclude curative resection (treated with neoadjuvant chemoradiation → esophagectomy). However, direct invasion into the aorta, trachea, or pericardium makes it T4b and surgically unresectable.

Memory Pearls

- **SCLC:** Central, Smokers, Systemic (Chemo is mainstay).
- **Pancoast Staging:** Always ≥ T3 (due to chest wall invasion).
- **Horner's in Pancoast:** Equals non-resectable.
- **PFT cutoffs:** FEV_1 < 800 ml means *No Major Surgery*. Post-op target is > 1000 ml.
- **SVC Syndrome etiology:** Think Lung Cancer first, Lymphoma second.

Hemodynamic monitoring

Core Concepts

- **Hemodynamics**: The forces generated by the heart and the resulting motion of blood through the cardiovascular system.
- **Four Main Hemodynamic Modulators**:
  1. **Intravascular volume**: Correlates with systemic venous return.

2. **Myocardial contraction**: Ventricular inotropy.
3. **Heart rate**: Chronotropy.
4. **Vasoactivity**: Systemic arterial resistance and compliance.

- **Volume Distribution**:
  - The **systemic venous system acts as the primary blood reservoir**, containing approximately **60–65% of total blood volume at rest**.
  - Arterial system: 10–15%; Pulmonary circulation: 10%; Capillaries: 5%.
- **Ejection Fraction (EF)**: Defined mathematically as **Stroke Volume (SV) divided by End-Diastolic Volume (EDV)**. It represents the fraction of blood ejected from the ventricle with each beat.
- **Mixed Venous Saturation (SvO_2)**:
  - Measured in pulmonary artery blood; marks the balance between whole-body oxygen delivery (DO_2) and oxygen consumption (VO_2).
  - Normal SvO_2 = **70–75%**.
  - **Decreased SvO_2**: Increased consumption (pain, hyperthermia) or decreased delivery (low CO, anemia, hypoxia).
  - **Increased SvO_2**: Increased delivery (high CO, hyperbaric O_2) or low consumption (sedation, paralysis, cyanide toxicity).

Diagnosis / Clinical Features

- **Clinical Examination**: The first step of hemodynamic monitoring. Assess perfusion via temperature, pulse, BP, respiratory rate, pain, level of consciousness, and urine output (highly concentrated/dark urine indicates intravascular depletion).
- **Pulse Oximetry (SpO_2)**:
  - Calculates saturation using the Beer-Lambert law: probes emit red and infrared light. High concentrations of red blood cells absorb more light; low concentrations absorb less.
  - **Oxyhemoglobin Dissociation Curve Shifts**:
    - **Left shift** (increased O_2 affinity): Decreased temp, decreased 2,3-DPG, decreased $[H^+]$ (alkalosis), Carbon Monoxide.
    - **Right shift** (reduced O_2 affinity, enhanced tissue unloading): Increased temp, increased 2,3-DPG, increased $[H^+]$ (acidosis).

Investigations

- **Arterial Line**:
  - Provides beat-to-beat BP (SBP, DBP, MAP) and regular blood sampling.
  - *Waveform morphology*: As the wave travels distally (Aorta → Radial → Dorsalis pedis), the arterial wave becomes narrower and taller with a steeper upstroke.
  - **Pulse Pressure Variation (ΔPP)**: A variation of **≥ 13%** in septic patients mechanically ventilated discriminates fluid responders from non-responders (Sensitivity 94%, Specificity 96%).

- **Central Venous Catheter (CVC)**:
  - Measures Central Venous Pressure (CVP) and provides access for vasoactive infusions.
  - *CVP limitations*: Misleading in systemic venoconstriction, decreased RV compliance, obstruction of great veins, tricuspid regurgitation, and mechanical ventilation (pleural pressure changes).
  - *Normal CVP Waveform*:
    - **a wave**: Atrial contraction (after ECG P-wave).
    - **c wave**: Tricuspid valve elevation during RV contraction (after QRS).
    - **x descent**: Downward movement of contracting RV.
    - **v wave**: Right atrial filling during late ventricular systole.
    - **y descent**: Tricuspid valve opening in early ventricular diastole.
- **Pulmonary Artery Catheter (PAC / Swan-Ganz)**:
  - Measures: CVP, PAOP (correlates to LAP = LVEDP), CO/CI (via thermodilution), SV, RVEF, SVRI, PVRI, DO_2, and VO_2.
  - *Thermodilution*: The area under the temperature-time curve is **inversely proportional** to the cardiac output (CO).
  - *Waveform progression*:
    - Right Atrium: Low amplitude (0–8 mmHg).
    - Right Ventricle: High systolic spike, low diastolic trough (~25/0 mmHg).
    - Pulmonary Artery: Diastolic step-up with dicrotic notch (~25/10 mmHg).
    - PCW/PAOP: Dampened waveform (~4–12 mmHg).
- **Echocardiography (TTE/TEE)**:
  - Evaluates structure, EF, and CO.
  - *Doppler calculation of SV*: Stroke Volume = Cross-Sectional Area (CSA) × Velocity Time Integral (VTI). CO = SV × HR.
- **Esophageal Doppler**: Estimates descending aortic flow; relies on normograms for aortic CSA. Good correlation with thermodilution. Waveform shapes change diagnostically (e.g., tall/broad in decreased afterload, very narrow in hypovolemia).
- **Pulse Contour Analysis (PiCCO / LiDCO)**:
  - Requires a central venous line and a specialized thermistor-tipped/lithium-sensitive arterial catheter.
  - *PiCCO*: Uses transpulmonary thermodilution for initial calibration, then evaluates the arterial pressure waveform beat-to-beat for continuous CO and extravascular lung water (EVLW).
- **Partial CO_2 Rebreathing (NICO)**:
  - Uses the differential Fick principle ($CO = \Delta VCO_2 / \Delta CaCO_2$).
  - Non-invasive but **restricted to mechanically ventilated patients**. Influenced by pre-existing lung disease.
- **Electrical Bioimpedance**: Non-invasive neck/chest electrodes measure thoracic electrical resistance changes to calculate stroke volume. Reliability in unstable/ICU patients is debated.

Relevant Guidelines

Hemodynamic Treatment Algorithm in Hypotension:

- **Hypovolemia** (Low CVP, Low CI, High SVRI) → Consider fluid challenge.
- **Cardiogenic** (High CVP, Low CI, High SVRI) → Consider inotrope / IABP.
- **Vasogenic/Distributive** (Low CVP, High CI, Low SVRI) → Consider vasopressors.

Expected Changes to Classic Hemodynamic Parameters in Shock States:

SHOCK STATE	SVR	PVR	CI	SVO_2	RAP/CVP	PAP	PAOP
Cardiogenic	↑	Normal	↓	↓	↑	↑	↑
Distributive	↓	Normal	Normal / ↑	Normal / ↑	Normal / ↓	Normal / ↓	Normal / ↓
Hypovolemic	↑	Normal	↓	↓	↓	↓	↓
Obstructive	Normal / ↑	↑	↓	Normal / ↓	↑	↑	Normal / ↓

Diagnostic Considerations for Elevated Lactate in Sepsis:

- *Hypovolemic / Cardiogenic shock*: Ensure volume / consider co-morbid cardiac dysfunction.
- *Drugs/Toxins*: Metformin, Linezolid, Ethylene glycol, Cyanide, Paracetamol.
- *Others*: Adrenaline use (interferes with lactate as a resuscitation marker), Seizures, Hepatic dysfunction, Thiamine deficiency.

Complications / Prognosis

- **Arterial Line Complications**: Hematoma, distal ischemia, pseudoaneurysm, and infection.
- **CVC Complications**: Vascular erosion, thrombosis, infection, arrhythmogenesis.
- **PAC Complications**: Catheter looping, balloon rupture, pulmonary artery injury, pulmonary infarction, and arrhythmias. Note: PAC use has **not** been shown to reduce mortality (and in some trials, mortality was higher).
- **Post-Operative Low Cardiac Output**:
  - **Common causes**: Poor myocardial contractility, obstructive shock (cardiac tamponade), or hypovolemia (due to bleeding or diuresis).
  - Acidosis is a profound negative inotrope and drops CO.
  - *Note*: Metabolic alkalosis does **not** characteristically cause a persistent decrease in cardiac output.

Past-Paper High Yield

- **Ejection Fraction Definition**: Always remember EF mathematically is **Stroke Volume divided by End-Diastolic Volume (SV/EDV)**.

- **Systemic Venous Reservoir**: The venous system contains the vast majority of circulating blood volume (**60–65% at rest**), making it the primary reservoir.
- **Post-op Low Cardiac Output Causes**: Watch out for 'EXCEPT' questions regarding persistent post-op drops in CO. Poor function, tamponade, hypovolemia, and bleeding are major causes. **Metabolic alkalosis** is incorrect as a cause (acidosis, however, impairs contractility).
- **Thermodilution Curve**: High CO yields a *smaller* area under the curve (rapid washout). Low CO yields a *larger* area under the curve (delayed washout).

Memory Pearls

- **Oxyhemoglobin curve shifts**: "Right is Release (to tissues)" → **C**O2, **A**cid, **D**PG, **E**xercise, **T**emperature (CADET face right).
- **Shock differentiation**:
  - Only Distributive shock has a *low* SVR.
  - Only Cardiogenic shock has an *elevated* PAOP (wedge pressure).

Acute lower limb ischemia

Core Concepts

- **Definition:** An abrupt interruption of blood flow to an extremity threatening limb viability. It is the most common vascular emergency.
- **Etiology:**
  - **Thrombosis in-situ (60%):** Typically occurs on a pre-existing atherosclerotic plaque or stenotic arterial segment, aneurysms, or vascular grafts.
  - **Embolism (30%):** Typically of cardiac origin (e.g., Atrial Fibrillation, MI, endocarditis, valvular disease, atrial myxoma). Arterial sources include aneurysms and atherosclerotic plaques. Emboli typically lodge at acute arterial narrowings or vessel bifurcations (Femoral 28%, Arm 20%, Aortoiliac 18%, Popliteal 17%).
  - **Trauma / Iatrogenic:** Can result from blunt/penetrating trauma or intra-arterial devices. **Note:** Limb ischemia is the most frequent complication of an Intra-Aortic Balloon Pump (IABP) due to the large-bore catheter occluding the femoral/iliac arteries.
- **Time window:** The limb must be revascularized within **4–6 hours** to prevent irreversible nerve and muscle injury.
- **Mortality and Morbidity:** High risk; 25% mortality rate and 20% amputation rate. Embolism carries a higher risk of loss of life, whereas thrombosis carries a higher risk of loss of limb.

Diagnosis / Clinical Features

- **The 6 "P"s of Acute Ischemia:**
  - **P**ain (May be absent in complete acute ischemia)
  - **P**ulselessness
  - **P**allor

- **P**aresthesias
- **P**aralysis
- **P**oikilothermia (Perishing cold; heavily dependent on ambient temperature)

- *Note: Paralysis, paresthesias, and muscle tenderness (calf pain on squeezing) are the cardinal signs of complete, impending irreversible ischemia.*

Clinical Differentiation: Embolus vs. Thrombosis

FEATURE	EMBOLUS	THROMBOSIS
History of Claudication	Absent ("clean" arterial tree)	Present
Onset	Sudden (seconds or minutes)	Insidious (hours or days)
Ischemia Severity	Complete, profound (no collaterals)	Incomplete (well-developed collaterals)
Embolic Source	Present (usually Atrial Fibrillation)	Absent
Artery Palpation	Soft, tender	Hard, calcified
Bruits / Pulses	No bruits; contralateral pulses present	Bruits present; contralateral pulses absent

Investigations

- **Doppler Assessment:** Evaluation of arterial and venous signals is mandatory (determines Rutherford staging).
  - **Spectral wave changes:** Normal (triphasic) → Mild stenosis (biphasic) → Severe stenosis (high peak systolic velocity) → Post-stenotic/Occlusion (monophasic "tardus parvus" dampened waveform).
- **Ankle-Brachial Pressure Index (ABPI):** Strong prognostic indicator for cardiovascular survival (ABPI >0.85 indicates ~75% 10-year survival; ABPI <0.4 drops to ~30%).
- **Arteriography Findings:**
  - **Embolus:** Sharp cutoff with a rounded "reverse meniscus" sign, intraluminal filling defects, multiple sites of lodgement, absent collateral circulation, and otherwise normal vessels.
  - **Thrombosis:** Sharp or tapered cutoff, diffuse atherosclerosis, and prominent, well-developed collateral circulation.

Relevant Guidelines

Classification of Acute Limb Ischemia (According to Rutherford / SVS/ISCVS)

CATEGORY	PAIN	CAPILLARY REFILL	MOTOR DEFICIT	SENSORY DEFICIT	ARTERIAL / VENOUS DOPPLER	TREATMENT
I (Viable)	Mild	Intact	None	None	Audible / Audible	Urgent evaluation

CATEGORY	PAIN	CAPILLARY REFILL	MOTOR DEFICIT	SENSORY DEFICIT	ARTERIAL / VENOUS DOPPLER	TREATMENT
IIa (Marginally threatened)	Moderate	Delayed	None	Minimal (toes)	Inaudible / Audible	Urgent revascularization
IIb (Immediately threatened)	Severe	Delayed	Partial	More than toes	Inaudible / Audible	Emergency revasc.
III (Nonviable)	Variable	Absent	Complete (rigor)	Complete (anesthesia)	Inaudible / Inaudible	Primary Amputation

ESVS 2020 Guidelines on Imaging Modalities in Acute Limb Ischaemia

- **Duplex ultrasound:** High accuracy for adjacent structures but limited evaluation of entire vascular tree.
- **CT Angiography (CTA):** Highly accurate, non-invasive, excellent for evaluating the entire vascular tree.
- **Digital Subtraction Angiography (DSA):** Highly accurate with therapeutic potential (interventions), but invasive.

Management

- **Immediate Resuscitation:** Oxygen therapy, BP optimization, analgesia, NPO, IV fluids.
- **Systemic Anticoagulation:** Start IV Heparin immediately (5000 IU stat bolus → 1000 IU/hour) to prevent thrombus propagation.
  - *Crucial note:* Heparin does *not* dissolve existing emboli; it is a bridge to definitive therapy.
- **Definitive Revascularization Pathways:**
  - **Obvious Embolus (Stage I-IIb):** Emergent Surgical Embolectomy (usually via Fogarty balloon catheter). Post-operative oral Warfarin is used for long-term prophylaxis.
  - **Diagnostic Uncertainty:** Perform diagnostic angiography first to avoid "blind" embolectomy procedures.
  - **Acute Thrombotic Occlusion / Thrombosed Grafts:** Intra-arterial Thrombolysis (Streptokinase, Urokinase, or rt-PA) is often the ideal therapy, followed by definitive medical management or bypass surgery.
  - **Stage 3 / Non-Viable Limb:** Revascularization attempts are contraindicated. Requires primary amputation.
- **Post-Amputation Rehabilitation:** Physical therapy and mobilization should commence **as soon as possible** to prevent physical deconditioning, avoid contractures, and improve psychological outcomes. Do not delay until full healing or permanent prosthesis fitting.

Operative / Procedural Notes

- **Fasciotomy:** Should always be considered or performed upon successful reperfusion of a threatened limb to prevent or treat compartment syndrome. The wounds are left open for delayed

primary closure or skin grafting.

- **Primary Amputation:** Indicated for limbs showing fixed staining/blistering, non-blanching dark mottling, profound anesthesia, and rigid muscles (rigor mortis). Attempting to revascularize a limb in this state will trigger fatal systemic toxicity.

Complications / Prognosis

- **Reperfusion Injury:** Restoring blood flow to necrotic muscle (indicated clinically by profound calf pain on squeezing or rigor) flushes potassium, myoglobin, and lactic acid into the systemic circulation, leading to life-threatening arrhythmias and acute renal failure.
- **Compartment Syndrome:** Intrafascial swelling secondary to tissue reperfusion. Mandates emergency fasciotomies.
- **Natural History of Ischemic Skin Changes:** Marble white (spasm) → Fine reticular blanching mottling (deoxygenated blood filling) → Coarse, dark, non-blanching mottling (coagulation) → Blistering/liquefaction and established dry gangrene (irreversible).

Past-Paper High Yield

- **Definitive Treatment for Embolic Ischemia:** Emergent surgical embolectomy is the *most appropriate definitive management*. IV Heparin is an immediate adjunct but is not a definitive standalone treatment.
- **Stage 3 Acute Limb Ischemia:** Recognized by profound sensory/motor loss (non-salvageable). The *only* correct management is primary amputation to prevent fatal reperfusion injury. Observation is dangerous and incorrect.
- **Embolic vs. Thrombotic Presentation:** Emboli predominantly occur in patients with a "clean" arterial tree (no history of claudication) presenting with a profound, sudden onset. Atrial fibrillation is the major risk factor.
- **IABP Complication:** Limb ischemia is the most frequent complication of an intra-aortic balloon pump due to the large-bore catheter in the iliofemoral system.
- **Post-Amputation Care:** Rehabilitation must begin *as soon as possible*, not delayed for 6 weeks or until a prosthesis is fitted.

Chronic lower limb ischemia

Core Concepts

- **Definition:** Decreased arterial blood supply to the lower limbs due to partial or complete occlusion of arteries, causing an imbalance between blood supply and tissue metabolic demand.
- **Etiology:** Atherothrombosis is the most common cause. Other causes include arteritis, aneurysms, and embolization.
- **Prevalence & Prognosis:**
  - Prevalence in patients >55 years is 10%–25%, though 70%–80% are asymptomatic.
  - Systemic risk: Patients with peripheral vascular disease (PVD) have the same relative risk of cardiovascular (CV) death as those with Coronary Artery Disease (CAD) or Cerebrovascular

Disease (CVD).
    - Mortality: PVD patients are 4 times more likely to die within 10 years than patients without the disease.
- **Critical Limb Ischemia (CLI) Prognosis (at 1 year):**
    - 50% alive with two limbs
    - 25% require amputation
    - 25% cardiovascular mortality

Diagnosis / Clinical Features

- **Intermittent Claudication:**
    - The hallmark symptom of peripheral arterial disease (PAD).
    - Characterized by highly reproducible muscular pain (usually in the calf) induced by a specific distance of exercise and promptly abated by rest.
    - Driven by arterial ischemia failing to meet metabolic demand.
- **Critical Limb Ischemia (CLI):**
    - Represents advanced, limb-threatening PAD (>60% stenosis).
    - Presents with **rest pain** (ischemic pain that is worse at night/when lying flat and is relieved by dangling the leg over the edge of the bed) or **tissue loss** (non-healing ulcers, frank gangrene).
    - Ulcers typically appear on the foot dorsum and leg shins; gangrene often begins between the toes.
- **Physical Examination Findings:**
    - **Inspection:** Thick shiny skin, hair loss, brittle nails, pallor, ulcers, muscular atrophy.
    - **Palpation:** Cool temperature, absent or diminished pulses, delayed capillary refill.
    - **Auscultation:** Bruits (e.g., femoral bruits heard halfway between the ASIS and pubic symphysis).
    - **Buerger's Test:** Elevate leg to 45° (look for pallor) -> hang leg at 90° (look for reactive hyperemia/red flushed foot). Pallor occurring at <20° indicates severe PAD.
- **Segmental Occlusion Presentations:**
    - *Aortoiliac:* Buttock/thigh/calf claudication, absent femoral/distal pulses, impotence (Leriche syndrome).
    - *Iliac:* Unilateral thigh/calf claudication, absent unilateral femoral/distal pulses.
    - *Femoropopliteal:* Calf claudication, palpable femoral pulse but absent popliteal/tibial pulses.
    - *Distal Obstruction:* Calf/foot claudication, palpable femoral and popliteal pulses, absent ankle pulses.

Differential Diagnosis of Leg Pain

ETIOLOGY	CHARACTERISTICS
Vascular (True Claudication)	Reproducible with walking, abates promptly with rest.

ETIOLOGY	CHARACTERISTICS
Neurospinal (Pseudoclaudication)	Due to spinal stenosis. Pain with erect posture (lumbar lordosis); relieved by sitting, lying down, or leaning forward (e.g., pushing a shopping cart).
Musculoskeletal (Osteoarthritis)	Varies with weather and time of day (worse in morning). Does not disappear promptly after exercise.
Neuropathic	Burning/tingling associated with Diabetes or chronic alcohol abuse.

Investigations

- **Ankle-Brachial Index (ABI):** The gold standard initial bedside test. Calculated as (Systolic BP in ankle) / (Systolic BP in brachial artery).
  - **Normal:** > 0.90
  - **Intermittent Claudication:** 0.50 – 0.90
  - **Rest Pain:** 0.21 – 0.49
  - **Tissue Loss (Gangrene):** < 0.20
  - *Diagnostic Trap:* ABI > 1.25 is falsely elevated and occurs in diabetic and renal failure patients due to non-compressible, calcified vessel walls (Mönckeberg's arteriosclerosis). A normal ABI does *not* completely exclude significant PVD.
- **Arterial Duplex Ultrasound:** Initial non-invasive imaging.
- **Computed Tomography Angiography (CTA):** The most important and frequently utilized advanced imaging modality to precisely map arterial anatomy and plan surgical/endovascular revascularization.

Management

- **Conservative / Medical Management:**
  - **Supervised Exercise Therapy:** First-line for intermittent claudication. Improves muscle metabolic efficiency and significantly increases pain-free walking distance. It does *not* significantly change the resting ABI and is contraindicated in patients with CLI (rest pain/tissue loss).
  - **Risk Factor Modification:** Smoking cessation is the single most important factor determining long-term outcomes. Strict control of HTN, diabetes, and dyslipidemia.
  - **Antiplatelet Therapy:** Lifelong Aspirin (75–150 mg/d) is strictly for secondary prevention of CV events (MI, stroke); it does *not* improve walking distance or alleviate claudication symptoms. Clopidogrel may be superior to aspirin (CAPRIE trial).
- **Endovascular Therapy (Catheter-Based):**
  - Includes Peripheral Transluminal Angioplasty (PTA), stenting, and atherectomy.
  - Indications: Lifestyle-disabling claudication, CLI, or severe ischemia with tissue loss.
- **Surgical Intervention:**

- Indications: Ischemic rest pain, tissue necrosis/gangrene (threatened limb loss), or severe livelihood-limiting claudication.
- **Amputation:** Reserved for unreconstructable PAD, fixed flexion deformities, or extensive unsalvageable tissue necrosis.

Relevant Guidelines

- **ACC/AHA Screening Guidelines:** Identify high-risk cohorts needing PAD screening:
  - Age ≥ 70 years.
  - Age 50–69 years with a history of smoking or diabetes.
  - Age 40–49 with diabetes and at least one other atherosclerosis risk factor.
  - Any patient with claudication, rest pain, abnormal lower extremity pulses, or known atherosclerosis elsewhere.
- **Cardiovascular Risk Targets:**
  - **Glycemic Control:** Target HbA1C < 7%.
  - **Lipid Management:** Target LDL < 100 mg/dL (< 2.6 mmol/L). Statins reduce CV mortality in PAD patients by 21%.
  - **Beta-blockers:** Safe to use in PAD patients, *except* those with severe Critical Limb Ischemia.

Operative / Procedural Notes

- **Aortoiliac Endarterectomy:** Used for localized aortoiliac disease. Advantages include avoiding prosthetic material, zero risk of graft infection, and preservation of hypogastric (internal iliac) artery inflow, which can improve/maintain sexual potency.
- **Bypass Configurations:**
  - *Aortobifemoral:* Synthetic bifurcated graft from infrarenal aorta to both common femoral arteries.
  - *Extra-anatomic:* Axillobifemoral or Femoro-femoral crossover (used for unilateral iliac disease or high-risk surgical patients).
  - *Infrainguinal:* Femoro-popliteal or Femoro-distal (crural) bypass using autologous vein or synthetic grafts. Oral anticoagulation improves patency in venous conduits, while aspirin is better for non-venous prosthetic grafts.

Complications / Prognosis

- **Early Postoperative Complications:**
  - Hemorrhage (1–2% require reoperation).
  - Acute graft limb occlusion (1–3%).
  - "Trash foot": Acute limb ischemia secondary to microembolization of atheromatous debris into digital arteries during clamp removal.
  - Acute renal failure, bowel ischemia, or spinal cord ischemia.
- **Late Postoperative Complications:**

- **Graft occlusion** secondary to recurrent disease (Most common late complication: 5–10% at 5 years).
- Pseudoaneurysm formation (3–5%).
- Postoperative iatrogenic impotence (up to 25% of aortoiliac procedures).
- Late graft infection and aortoenteric fistula.

Past-Paper High Yield

- **Vascular Anatomy & Pathology:**
  - **Popliteal Artery Aneurysms:** The most common peripheral arterial aneurysm. Bilateral in ~50% of cases. They present with acute/chronic limb ischemia via thrombosis or distal embolization (NOT local compression). 30–50% of patients with a popliteal aneurysm have an AAA (but AAA patients only have a 10–15% chance of a popliteal aneurysm). Not associated with pregnancy.
  - **Arterial Embolism:** The superficial femoral artery is highly susceptible to emboli, particularly at the adductor (Hunter's) canal where plaque commonly resides.
  - **Intra-Aortic Balloon Pump (IABP):** Inserted retrogradely via the common femoral artery. A major direct complication is *lower limb ischemia* due to obstruction of distal arterial blood flow.
- **Venous Disease & Thrombophilia:**
  - **DVT Presentation & Management:** Clinical diagnosis is highly inaccurate (must use ultrasound). Not all patients require admission (outpatient DOAC/LMWH is common).
  - **DVT Sequelae:** The most significant acute, life-threatening complication is *Pulmonary Embolism*. A major chronic complication is *Post-thrombotic syndrome* (causing skin changes and venous ulcers).
  - **Paget-Schroetter Syndrome:** Effort-induced DVT of the axillary-subclavian vein. Suspect in young athletes (e.g., tennis player) with a swollen arm. Initial test: Venous duplex ultrasound.
  - **Venous Ulcers:** The absolute cornerstone of management for uncomplicated venous stasis ulcers is *sustained compression therapy* (e.g., Unna boots, multilayer wraps). Antibiotics and surgery are NOT first-line.
  - **Thrombophilia:** *Antiphospholipid syndrome* carries the exceptionally highest relative risk for developing Venous Thromboembolism (VTE), outweighing Factor V Leiden, Protein C deficiency, and OCP use.
- **Lymphedema:**
  - **Classification:** Primary lymphedema is classified purely by age of onset. Lymphedema praecox (<35 years) is the most common. Lymphedema tarda (>35 years) is a *primary* lymphedema, not secondary.
  - **Secondary Causes:** Filariasis (Wuchereria bancrofti) is the most common cause worldwide.
  - **Diagnosis & Treatment:** Lymphoscintigraphy is the gold standard diagnostic tool. Treatment is overwhelmingly non-surgical via Complete Decongestive Therapy (CDT: manual drainage, compression).
- **Preoperative Optimization:**

- Following a cerebrovascular accident (CVA/stroke), elective surgery must universally be delayed for a minimum of *6 weeks* to allow autoregulatory stabilization.

Memory Pearls

- **ABI > 1.25?** Think calcified vessels in a diabetic/renal patient; it does not rule out PAD.
- **Aspirin vs. Exercise in PAD:** Aspirin saves the heart/brain; Exercise saves the legs (improves walking distance).
- **Rest Pain:** Relieved by gravity (dangling feet over bed). Indicates Critical Limb Ischemia.
- **Pseudoclaudication:** Relieved by sitting or leaning forward (shopping cart sign); worse with standing straight.
- **Trash Foot:** Microemboli post-clamp removal.
- **Lymphedema Tarda:** *Primary* lymphedema presenting after age 35.

Pediatric

Exam Map

TOPIC CLUSTER	WEIGHT	KEY FOCUS AREAS
Intestinal Obstructions	Very High (16 Qs)	Radiologic signs, HPS acid-base, Volvulus vs. NEC surgery criteria
Pediatric Urology	High (10 Qs)	Hypospadias rules, VUR/UPJO imaging steps, Cryptorchidism
CDH & TEF	High (8 Qs)	VACTERL, TEF types/X-ray, CDH hemodynamics & resuscitation
Inguinoscrotal & FBs	Medium (12 Qs)	Diagnostic laparoscopy for UDT, Hernia timing, Battery vs. coin algorithms
Solid Tumors & Misc	Low (4 Qs)	Neuroblastoma vs. Wilms tumor age paradox, Abdominal wall defects

- **Medical Resuscitation vs. Surgical Emergency:** Never rush Hypertrophic Pyloric Stenosis (HPS) or Congenital Diaphragmatic Hernia (CDH) to the OR. HPS requires strict correction of hypochloremic hypokalemic metabolic alkalosis, and CDH requires stabilization of pulmonary hypertension. Conversely, **Midgut Volvulus** is an absolute, immediate surgical emergency.
- **Diagnostic Imaging Rules:** The exam frequently tests inappropriate imaging choices. Do *not* delay testicular torsion surgery for an ultrasound. Use **diagnostic laparoscopy** (not US/MRI) as the gold standard for non-palpable testes. An MCUG is absolutely mandatory for all UPJO workups to rule out concurrent VUR.
- **Radiologic Buzzwords:** Test-makers heavily rely on classic signs. Link "Double bubble" to Duodenal atresia/Volvulus, "Pneumatosis intestinalis" to NEC, "Soap bubble" to Meconium ileus, "Target/Pseudokidney" to Intussusception, and "Halo sign/Double contour" to an esophageal button battery.
- **Pediatric Urology Traps:** Hypospadias is primarily a *distal* defect (not proximal) presenting with a "spraying stream," and circumcision is strictly contraindicated. VUR is predominantly caused by a short submucosal tunnel, and surgical reimplantation is exclusively indicated after *failed medical management* (breakthrough UTIs on prophylaxis).
- **TEF & Esophageal Atresia Basics:** Excessive postnatal frothing is the earliest clinical sign of EA. A completely **gasless abdomen** on X-ray confirms isolated EA (Type A), whereas gas present in the stomach confirms a distal fistula (Type C).
- **Foreign Body Algorithms:** Location dictates management. A button battery in the esophagus is an absolute emergency (risk of rapid liquefactive necrosis and TEF), while an asymptomatic coin in the stomach requires only expectant observation. For airway aspirations, **rigid bronchoscopy** is the definitive diagnostic and therapeutic gold standard.
- **The Solid Tumor Age Paradox:** Understand the inverse prognostic relationship with age. Infants (<18 months) with Neuroblastoma (Stage MS) have excellent survival rates due to spontaneous

regression, whereas infants with Wilms tumor have worse outcomes. Furthermore, a suspected Wilms tumor should never be vigorously palpated (risk of rupture).

- **Surgical Timing in Groin Pathology:** Pediatric inguinal hernias require prompt repair (herniotomy, almost never mesh) due to high incarceration risk. In contrast, hydroceles are managed with observation until 1–2 years of age as the majority resolve spontaneously.

Diaphragmatic hernia & TEF (dr. Abeer)

Core Concepts

- **Congenital Diaphragmatic Hernia (CDH):** A discontinuity in the diaphragm leading to herniation of abdominal contents into the chest.
  - Prevalence: ~2.3–2.4 per 10,000 live births; Male > Female.
  - **Isolated vs. Non-isolated:** 60% are isolated (up to 70–85% survival rate). Non-isolated cases have a 20% survival rate.
  - **Associated Anomalies (Non-isolated):** Genetic/chromosomal (Trisomy 13, 18, 21) found in 33%. Cardiovascular (27.5%), Urogenital (17.7%), Musculoskeletal (15.7%), CNS (9.8%).
  - **Pulmonary Pathology:** Leads to severe pulmonary hypoplasia and CDH-associated pulmonary vascular hypertension (CDH-PH).
  - **Hemodynamics:** Suprasystemic pulmonary vascular resistance forces a **right-to-left shunt** (across the patent ductus arteriosus or foramen ovale).
- **CDH Defect Types:**
  - **Bochdalek:** Posterolateral defect. Most common (~90%). 80–85% occur on the **left side**, 19% right-sided, 1% bilateral.
  - **Morgagni:** Anteromedial parasternal defect (<2%). Associated with Trisomy 21 and congenital heart disease. Often asymptomatic; presents with retrosternal bowel loops.
  - **Central:** Rare defect in the central tendon (septum transversum anomaly).
  - **Eventration:** Abnormal elevation of an intact but thinned/attenuated diaphragm.
- **Esophageal Atresia (EA) & Tracheoesophageal Fistula (TEF):**
  - Failure of the foregut to completely separate into a ventral respiratory bud and dorsal esophageal bud (completed by 6–7 weeks).
  - Prevalence: 1 in 2500–3000 live births (slight male predominance).
  - Environmental associations: Methimazole, maternal diabetes, thalidomide, fetal alcohol syndrome.

Diagnosis / Clinical Features

- **CDH Postnatal Presentation:** Acute respiratory distress, scaphoid (empty) abdomen, displaced cardiac impulse, audible bowel sounds in the chest, and bilaterally decreased breath sounds.
- **Eventration Presentation:** Can present with clinical and radiographic signs *identical* to CDH (e.g., respiratory distress, elevated thinned diaphragm appearing similarly to herniated bowel restricting lung space).

- **EA/TEF Postnatal Presentation:** Excessive frothy drooling of saliva, immediate choking/coughing during the first feed, and respiratory distress.

Investigations

- **CDH Imaging:**
  - **Antenatal US (18–22 weeks):** Polyhydramnios, intrathoracic fluid-filled bowel loops, mediastinal shift.
  - **Fetal MRI:** Confirms and measures Total Fetal Lung Volume (TFLV).
  - **Postnatal CXR:** Stomach bubble/air-filled loops of bowel in the hemithorax deviating the trachea and heart shadows away from the affected side.
  - **Eventration Imaging:** Elevated hemidiaphragm on X-ray + **paradoxical movement** on ultrasound.
- **EA/TEF Imaging:**
  - **Nasogastric/Orogastric Tube:** Will meet resistance and coil in the upper pouch (typically T2-T4 level).
  - **Radiography (Crucial Differentiator):**
    - **Gasless, flat abdomen:** Indicates **isolated EA (Type A)** or EA with proximal fistula (Type B). Swallowed air cannot reach the GI tract.
    - **Gas present in stomach/intestines:** Indicates a **distal TEF (Type C)**, allowing air to pass from the trachea into the stomach.

Management

- **CDH Immediate Postnatal Resuscitation:**
  - **Immediate intubation** (avoid bag-mask ventilation to prevent gastric distension in the chest).
  - Nasogastric tube (NGT) on continuous suction to decompress herniated bowel.
  - Gentle hypercapnic ventilation or High-Frequency Oscillatory Ventilation (HFOV).
  - Manage pulmonary hypertension: Nitric Oxide (iNO) or Sildenafil to reverse right-to-left shunting. ECMO if no improvement.
  - *Note: Corticosteroids and surfactant are not supported by evidence.*
- **EA/TEF Preoperative Preparation:**
  - Keep NPO, place NG/OG tube on continuous suction (e.g., Replogle catheter).
  - Maintain an upright and side-lying position.
  - Rule out associated VACTERL anomalies prior to surgery.

Relevant Guidelines

- **CDH Resuscitation Targets (Aim):**
  - Preductal SaO_2 > 80–85%
  - pH > 7.2
  - $PaCO_2$ 50–70 mmHg

- PIP < 25 cmH_2O

- **Gross Classification of EA/TEF:**
  - **Type C (85%):** Proximal EA with distal TEF.
  - **Type A (7%):** Pure/Isolated EA (no fistula).
  - **Type E (4%):** Isolated "H-type" TEF (no atresia).
  - **Type B (2%):** EA with proximal TEF.
  - **Type D (1%):** EA with double (proximal and distal) TEFs.
- **Congenital Associations:**
  - **VACTERL:** Vertebral anomalies, Anorectal atresia, Cardiac defects, Tracheo-Esophageal fistula, Renal anomalies, Limb abnormalities.
  - **CHARGE:** Coloboma, Heart defects, Atresia of choanae, Retardation (developmental), Genital hypoplasia, Ear defects.

Operative / Procedural Notes

- **CDH Surgical Repair:**
  - **Not an urgent surgery.** Only performed once the patient is medically stable and pulmonary hypertension is managed.
  - Approach: Open neonatal repair is primarily via a **subcostal incision (>90%)**. Laparoscopic/thoracoscopic options exist. Large defects (Class D) require prosthetic patch closure.
- **Fetoscopic Endoluminal Tracheal Occlusion (FETO):**
  - Aggressive fetal intervention for isolated, severe CDH to stimulate lung growth (inflating a balloon in the fetal trachea to block escaping lung fluid).
  - Improves survival to discharge but significantly increases the risk of **preterm prelabor rupture of membranes (PROM) and preterm birth**.
  - **Contraindications:** Concomitant severe/life-threatening congenital anomalies (e.g., severe cyanotic cardiac anomalies).
- **EA/TEF Repair:**
  - **Ideal goal:** Definitive primary repair (fistula ligation + primary end-to-end anastomosis via thoracotomy/thoracoscopy) in the first few days of life.
  - **Indications for Staged Procedure (Delaying primary repair):** Highly unstable neonate, extreme prematurity, very low birth weight, or severe/unstable cyanotic cardiac anomalies (initial gastrostomy is placed instead).

Complications / Prognosis

- **CDH Complications:** Gastroesophageal reflux disease (GERD) is extremely common (50–100%), chronic recurrent pneumonia, failure to thrive, scoliosis, pectus deformities, and hernia recurrence (6–24%).
- **CDH Survival:** Inversely correlates with prematurity. Major congenital heart disease profoundly impacts morbidity and mortality.

- **EA/TEF Complications:** Anastomotic stricture (17–60%), anastomotic leaks (3.5–17%), recurrent TEF, tracheomalacia, and disordered esophageal peristalsis (leading to GERD → Barrett's esophagus → Cancer risk).

Past-Paper High Yield

- **CDH vs. Eventration:** Diaphragmatic eventration can present identically to CDH (respiratory distress, bowel loops appearing in chest on X-ray) but features an intact, thinned diaphragm.
- **CDH Laterality:** The most common CDH is the Bochdalek hernia, and it is located on the **left side** in over 80% of cases (not the central tendon).
- **CDH Hemodynamics:** CDH creates suprasystemic pulmonary vascular resistance, forcing a **right-to-left shunt** (NOT left-to-right).
- **EA/TEF Radiography:** A completely **gasless abdomen** in a choking newborn indicates isolated esophageal atresia without a distal TEF (Type A). If a distal fistula were present (Type C), swallowed air from the trachea would enter the stomach.
- **VACTERL Associations:** Includes vertebral, anal, cardiac, TEF, renal, and limb anomalies. Ear deformities are *least likely* to be associated (they belong to the CHARGE association).
- **FETO Contraindications:** FETO is strictly for severe *isolated* CDH cases. The presence of severe concomitant cardiac anomalies contraindicates this intervention.
- **Delaying EA/TEF Surgery:** Do NOT perform an immediate definitive primary repair in a neonate with a severe, unstable cyanotic cardiac anomaly; stabilize and perform a staged procedure instead.
- **Extraneous High-Yield (Burn Unit Criteria):** Infants and children with partial-thickness burns **>10% TBSA** warrant transfer to a designated burn unit.
- **Extraneous High-Yield (Infantile Hemangioma): Oral propranolol** is the universally accepted first-line treatment for rapidly growing infantile hemangiomas in cosmetically sensitive areas (e.g., central face) or those threatening vital functions (e.g., vision occlusion).

Memory Pearls

- **Type C = C**ommon (85% of TEF cases).
- **VACTERL:** Think structural (Vertebrae, Anus, Heart, Esophagus/Trachea, Kidneys, Limbs).
- **CHARGE:** Think head/neck and development (Coloboma, Heart, Choanae, Retardation, Genitals, Ears).
- **CDH Surgery: Fix the lungs before the hernia.** Medical stabilization of pulmonary hypertension strictly precedes surgical repair.

Intestinal obstruction 1 (dr. Abeer)

Core Concepts

- **General Principles:** Neonatal intestinal obstruction can occur anywhere (duodenum, small bowel, colon) and is caused by ischemic, inflammatory, or mechanical (adhesions, congenital bands) factors.

- **Epidemiology of Atresias:**
  - **Jejunoileal atresia** is the *most common* intestinal atresia and results from an **in utero vascular/ischemic accident**.
  - **Duodenal atresia** is the *second most common* and is heavily associated with chromosomal/developmental anomalies (e.g., ~50% have Trisomy 21).
- **Clinical Timing:**
  - Proximal (high) obstructions (e.g., duodenal atresia, malrotation) present with rapid, **early bilious vomiting**.
  - Distal (low) obstructions (e.g., recto-perineal fistula, Hirschsprung disease) present with abdominal distension and **delayed** passage of meconium; vomiting occurs much later and may not be classically bilious early on.
- **Malrotation Embryology:** The midgut normally rotates 270 degrees counterclockwise around the Superior Mesenteric Artery (SMA) axis. Arrest of this process results in malrotation, predisposing the narrow mesenteric pedicle to acute volvulus.
- **Meckel's Diverticulum (High-Yield):** The most common congenital anomaly of the GI tract. Located in the distal ileum (not jejunum). Approximately **50% of symptomatic cases present before age 2**. Typically presents with painless lower GI bleeding (not bilious vomiting). Heterotopic gastric/pancreatic mucosa may be present, but not always.

Diagnosis / Clinical Features

- **Esophageal Atresia (EA):** Earliest/most common postnatal sign is **excessive salivation** (frothing/bubbling at the mouth) because the neonate cannot swallow their own saliva. Choking/regurgitation occurs with feeds.
- **Hypertrophic Pyloric Stenosis (HPS):** Presents ~6 weeks postnatally with **projectile non-bilious vomiting** and dehydration. Prolonged vomiting of gastric acid leads to a classic **hypochloremic, hypokalemic metabolic alkalosis**.
- **Duodenal Atresia:** Bilious emesis within the first hours of life; scaphoid abdomen (if totally obstructed proximally).
- **Malrotation with Midgut Volvulus:** Sudden onset of bilious vomiting in an otherwise healthy neonate. **This is midgut volvulus until proven otherwise and is an absolute surgical emergency.**
- **Hirschsprung Disease (HD):** Delayed passage of meconium (>24 hours in ~90%), distension, bilious vomiting. ~10% present with Hirschsprung-associated enterocolitis (HAEC).
- **Meconium Ileus (MI):** Earliest manifestation of Cystic Fibrosis (CF). Presents at 1–2 days with bilious emesis, no normal meconium, and a "doughy" abdomen with indentable loops on palpation.
- **Anorectal Malformations (ARM):** Diagnosed primarily via clinical perineal examination at 24 hours (allows intraluminal pressure to push meconium down to identify fistulas).

Investigations

- **Antenatal Ultrasound:**
  - Polyhydramnios is typical for proximal obstructions (fetus cannot swallow/absorb amniotic fluid).

    - Meconium ileus shows a hyperechoic mass and non-visualized gallbladder.
- **Plain Radiography (Abdominal X-ray):**
    - **Duodenal Atresia:** "Double bubble" sign (gas in stomach + proximal duodenum; absent distal gas).
    - **Necrotizing Enterocolitis (NEC): Pneumatosis intestinalis** (gas within the bowel wall) is pathognomonic.
    - **Meconium Ileus:** "Soap bubble" or "ground glass" appearance (Neuhauser's sign) in the right lower quadrant; lack of classic air-fluid levels due to viscid meconium.
- **Contrast Studies (Gold Standards):**
    - **Upper GI Contrast:** Gold standard for malrotation. Shows DJ junction failing to cross the midline. **"Corkscrew" or "coil spring"** sign indicates volvulus. "Beak" sign indicates complete obstruction.
    - **Contrast Enema:** Identifies **microcolon** (disuse) in distal atresias/meconium ileus. In Hirschsprung disease, shows a cone-shaped **transition zone** with reversed recto-sigmoid ratio.
- **Other Diagnostics:**
    - **Color Doppler US:** "Whirlpool sign" in midgut volvulus (SMV twisting around SMA).
    - **Anorectal Manometry:** Absence of the Rectoanal Inhibitory Reflex (RAIR) in Hirschsprung disease.
    - **Rectal Biopsy: Gold standard for Hirschsprung disease** (shows absent ganglion cells).

Management

- **Resuscitation First:** Not all obstructions require immediate surgical resection. HPS requires fluid/electrolyte correction first (medical emergency initially). Uncomplicated Meconium Ileus can be treated with water-soluble contrast enemas (successful in ~2/3).
- **Surgical Emergencies:** Suspected midgut volvulus requires **immediate** operative intervention to prevent irreversible bowel necrosis/short bowel syndrome.
- **Duodenal Atresia:** Requires **Echocardiography** prior to elective operation (due to high rate of cardiac anomalies).
- **Hypertrophic Pyloric Stenosis:** Treated with **Ramstedt pyloromyotomy** (longitudinal incision of hypertrophic circular muscle down to, but *not including*, the submucosa).
- **Hirschsprung Disease:** Initial washouts/colostomy followed by a definitive pull-through procedure.
- **Meconium Ileus (Operative):** Enterotomy with N-acetylcysteine irrigation. Complex cases use specific staged stomas (e.g., Bishop-Koop or Santulli). Requires post-op pancreatic enzymes (Creon) and CF team involvement.

Relevant Guidelines

VACTERL Screening (Required for all ARM/EA patients):

- Spinal radiograph, Sacral radiographs (AP/Lat), Spinal/Pelvic US (<3mo) or MRI spine (>3mo), Echocardiogram, NG tube passage, Renal US, Limb radiographs.

Male Anorectal Malformation (ARM) Algorithm:

- Inspect perineum at 24 hours.
- *If meconium on perineum OR rectum <1 cm from perineum (cross-table lateral X-ray):* Perform Primary PSARP (Posterior Sagittal Anorectoplasty).
- *If meconium in urine OR rectum >1 cm:* Perform divided proximal sigmoid colostomy. Do a distal colostogram at 8-10 weeks, then PSARP.

Female Anorectal Malformation (ARM) Algorithm:

- Assess number of perineal orifices.
- *3 Orifices (Perineal/Vestibular fistula):* Primary PSARP or Colostomy.
- *2 Orifices:* Evaluate for rectovaginal fistula/vaginal atresia -> Colostomy.
- *1 Orifice (Cloaca):* Renal & Pelvic US on Day 1. If hydrocolpos is present -> Vaginostomy + Colostomy.

Operative Correction of Malrotation (Ladd's Procedure Steps):

1. Entry and evisceration.
2. **Counterclockwise** detorsion of the bowel (if volvulus present).
3. Division of Ladd cecal bands (relieves duodenal obstruction).
4. Broadening of the small intestine mesentery.
5. Incidental appendectomy.
6. Placement of small bowel on the right, colon on the left.

Operative / Procedural Notes

- **Intestinal Atresia Types (High-Yield):**

TYPE	DESCRIPTION
Type 1	Mucosal web/diaphragm with *intact* muscle wall.
Type 2	Blind proximal & distal ends connected by a **solid fibrous cord**, with an **intact mesentery**.
Type 3a	Complete separation of ends with a **V-shaped mesenteric gap**.
Type 3b	"Apple-peel" or "Christmas tree" deformity (single retrograde collateral vessel).
Type 4	Multiple atresias ("string of sausages").

- **Duodenoduodenostomy:** Classic technique is a "diamond-shaped" anastomosis to maximize luminal caliber bypassing the atretic segment.
- **Hirschsprung Pull-Through Options:**
  - *Swenson:* Direct full-thickness resection.
  - *Soave:* Endorectal (mucosa stripped, ganglionic bowel pulled through the aganglionic muscular sleeve).

- *Duhamel:* Retrorectal (ganglionic bowel brought down behind the native aganglionic rectum and anastomosed side-to-side).

Complications / Prognosis

- **Midgut Volvulus:** Extensive infarction results in **short bowel syndrome**. Note: *Atypical cholecystitis is NOT associated with malrotation.*
- **Duodenal Atresia:** Long-term survival is 90%. Early mortality (3-5%) is linked to associated cardiac anomalies.
- **Hirschsprung Disease:** HAEC (Enterocolitis) is life-threatening; obstructive symptoms, soiling, and HAIBD (inflammatory bowel disease) can occur long-term.
- **Meconium Ileus:** Distal Intestinal Obstruction Syndrome (DIOS) and intussusception (1% of CF patients, average age 9.5 yrs).

Past-Paper High Yield

- **Emergencies:** Suspected volvulus (bilious vomiting + double bubble/corkscrew) = absolute surgical emergency. CDH, EA, HPS, and uncomplicated MI require medical/fluid stabilization *first*.
- **Radiologic Hallmarks:**
  - *Pneumatosis intestinalis* = NEC.
  - *Double bubble* = Duodenal atresia or midgut volvulus.
  - *Soap bubble* = Meconium ileus.
  - *Corkscrew/Whirlpool* = Malrotation/volvulus.
- **HPS Acid-Base:** Hypochloremic, hypokalemic metabolic alkalosis is consistently tested.
- **Esophageal Atresia Postnatal Sign:** Excessive salivation (frothing) is the *earliest and most common* clinical sign.
- **Atresia Types:** Know that **Type 2** has an **intact mesentery** but a solid fibrous cord, distinguishing it from Type 3a (V-shaped gap) and Type 1 (mucosal web).
- **Malrotation Anatomy:** Understand that Ladd's bands run from the cecum across the duodenum, causing extrinsic compression. *Atypical cholecystitis* is a distractor and not associated.
- **Bilious vs Non-Bilious Vomiting:** High IO (Duodenal atresia, annular pancreas, malrotation, intestinal atresia) = rapid bilious vomiting. Low IO (Recto-perineal fistula) = delayed presentation. Pyloric stenosis = non-bilious projectile vomiting.

Memory Pearls

- **"RAMSTEDT"** = The procedure for HPS (longitudinal **m**yotomy down to **s**ubmucosa).
- **"LADD'S"**: **L**ook for bands, **A**ppendectomy, **D**etorse counterclockwise, **D**uodenum freed, **S**plit small/large bowel (right/left).
- **Meckel's Rule of 2s:** 2% of population, 2 feet from ileocecal valve, 2 inches long, 2 types of ectopic tissue, presents before age 2.

Intestinal obstruction 2 (dr. Abeer)

Core Concepts

- **Necrotizing Enterocolitis (NEC):** Ischemic and inflammatory disease of premature neonates. Incidence and mortality are inversely proportional to birth weight (affects ~10% of Very Low Birth Weight [VLBW] infants).
- **Hypertrophic Pyloric Stenosis (HPS):** Acquired hypertrophy of the pyloric musculature. Risk factors include male gender (M:F = 4:1), first-born status, younger maternal age, and macrolide (erythromycin) exposure.
- **Intussusception:** The proximal bowel (intussusceptum) invaginates into the distal bowel (intussuscipiens), compressing the mesentery. This leads to venous obstruction, bowel edema, arterial insufficiency, ischemia, and necrosis. Most common cause of small bowel obstruction between ages 4–9 months.
    - **Primary:** No definitive lead point; often associated with hypertrophied Peyer patches (viral trigger).
    - **Secondary:** Has a lead point (e.g., Meckel diverticulum, polyps, appendix, Henoch–Schönlein purpura, cystic fibrosis, celiac disease).
- **Meckel Diverticulum:** A true diverticulum arising from the antimesenteric border of the ileum due to failure of the vitelline (omphalomesenteric) duct to obliterate.
- **Biliary Atresia (BA):** Sclerosing cholangiopathy causing progressive destruction of extrahepatic bile ducts. It is the most common cause of end-stage liver disease and indication for liver transplant in children. Most are isolated, but some are syndromic (e.g., BASM: Biliary Atresia, Splenic Malformation, and Malrotation).

Diagnosis / Clinical Features

- **Necrotizing Enterocolitis (NEC):**
    - Presents with feeding intolerance (vomiting, high gastric residuals) and abdominal distention.
    - Bloody stools (bleeding per rectum) is a late sign.
    - Exam: Visible/palpable dilated bowel loops, striking abdominal skin discoloration (erythema/shiny), and signs of peritonitis.
- **Hypertrophic Pyloric Stenosis (HPS):**
    - Presents in full-term neonates at **2–8 weeks old** with **nonbilious, progressive projectile vomiting** of recent feeds.
    - Exam: Visible gastric peristaltic waves and a palpable **"olive sign"** in the RUQ (70–90% of patients). Infants may show severe dehydration in late presentations.
- **Intussusception:**
    - Classic triad (seen in <25%): Intermittent cramping abdominal pain (every 15–30 mins with knees drawn up), **"currant jelly" stools**, and a palpable right upper quadrant (RUQ) mass.
    - Exam: **Dance sign** (an unexpectedly "empty" right iliac fossa upon palpation due to the cecum migrating into the transverse colon).
- **Meckel Diverticulum:**

- Mostly asymptomatic. Three most common symptomatic presentations:
  1. Painless lower gastrointestinal bleeding (30–56%, due to ectopic gastric mucosa ulcerating adjacent normal ileum).
  2. Intestinal obstruction (14–42%, due to intussusception, volvulus around a fibrous band).
  3. Diverticular inflammation (mimics appendicitis).
- **Biliary Atresia:**
  - Prolonged neonatal jaundice, pale (acholic) stools, and hepatomegaly.
  - Later signs: Malnutrition, failure to thrive (due to fat/fat-soluble vitamin malabsorption), and portal hypertension.

Investigations

- **Necrotizing Enterocolitis (NEC):**
  - **Abdominal X-ray: Pneumatosis intestinalis** (gas within the bowel wall) is the hallmark and classic sign. May also show portal venous gas or pneumoperitoneum (free air indicating perforation).
  - **Labs:** Leukocytosis, elevated CRP, hyponatremia, thrombocytopenia, elevated lactate, and metabolic acidosis.
- **Hypertrophic Pyloric Stenosis (HPS):**
  - **Labs:** Classic **hypochloremic hypokalemic metabolic alkalosis**.
  - **Ultrasound (First-line):** Muscle thickness ≥ 4 mm and pyloric channel length ≥ 16 mm. Shows "target" or "donut" sign in transverse.
  - **Upper GI series:** Used if US is equivocal; shows the **"string sign"** (narrowed pyloric channel).
- **Intussusception:**
  - **Ultrasound:** Diagnostic. Shows **"target" or "donut" lesion** (transverse plane) and **"pseudokidney" sign** (longitudinal plane).
- **Meckel Diverticulum:**
  - **Meckel Scan (Technetium-99m pertechnetate):** Identifies ectopic gastric mucosa in bleeding patients. False-negative rate is ~25%.
- **Biliary Atresia:**
  - **Ultrasound:** Shows the **"triangular cord sign"** (fibrous remnant of the biliary tree at the porta hepatis).
  - **Hepatobiliary Scintigraphy (HIDA scan):** Shows normal hepatic uptake of radiotracer but an **absolute absence of excretion into the bowel at 24 hours**.
  - **Intraoperative Cholangiography:** Definitive diagnosis.

Management

- **Necrotizing Enterocolitis (NEC):**
  - Primary: Supportive (NPO/bowel rest, gastric decompression, IVF, IV antibiotics, parenteral nutrition).

    - Surgical: **Absolute indication is pneumoperitoneum** (perforation). Options include laparotomy with resection/anastomosis, stoma formation, or primary peritoneal drainage (in highly unstable VLBW neonates).
- **Hypertrophic Pyloric Stenosis (HPS):**
    - Preoperative (Crucial): NPO, gastric decompression, and strict **correction of electrolytes and fluids** prior to taking the patient to the OR (this is a medical urgency, not a surgical emergency).
    - Surgical: Laparoscopic or open pyloromyotomy.
- **Intussusception:**
    - Initial: NGT, NPO, fluid resuscitation.
    - Non-operative: **Hydrostatic or Pneumatic Reduction** (fluoroscopic/US guidance). Success rate ~85%.
    - Contraindications to enema reduction: Peritonitis, free air (perforation), or profound shock/sepsis.
    - Operative: Indicated if enema is unsuccessful, peritonitis is present, or a pathological lead point is strongly suspected.
- **Gastroschisis:** Urgent coverage of naked bowel with warm saline-soaked gauze. Primary fascial closure or staged closure using a silo bag.
- **Omphalocele:** Primary closure for small defects. For giant omphaloceles with associated morbidities, **"paint and wait"** technique (topical escharotic agents like silver sulfadiazine to promote epithelialization over the sac, followed by delayed ventral hernia repair).
- **Meckel Diverticulum:** Diverticulectomy (wedge resection, placing stapler obliquely to prevent stenosis) or segmental ileal resection with end-to-end anastomosis.
- **Biliary Atresia: Kasai Portoenterostomy** to restore bile flow. Liver transplant is eventually required in ~2/3 of patients.

Relevant Guidelines

Table: Modified Bell Classification for NEC

STAGE	CLINICAL FINDINGS	RADIOGRAPHIC FINDINGS
Stage I (Suspected)	Apnea, bradycardia, temp instability	Normal gas pattern or mild ileus
Stage IIA (Definite)	Apnea, bradycardia, temp instability	Ileus, dilated loops, **focal pneumatosis**
Stage IIB	Metabolic acidosis, thrombocytopenia	**Widespread pneumatosis, portal venous gas**, ascites
Stage IIIA (Advanced)	Mixed acidosis, coagulopathy, shock	Severe dilation, ascites, **no free air**
Stage IIIB	Shock, worsening vital signs	**Pneumoperitoneum** (perforation)

Biliary Atresia Classification:

- **Type I:** Atresia of the common bile duct.
- **Type IIa:** Atresia of the common hepatic duct.

- **Type IIb:** Atresia of the CBD and common hepatic duct.
- **Type III (Most common, >90%):** Atresia of all extrahepatic bile ducts up to the porta hepatis.

Table: Differentiating Gastroschisis vs. Omphalocele

CHARACTERISTIC	OMPHALOCELE	GASTROSCHISIS
Location	Midline (Umbilicus)	Right of umbilicus
Sac	Present	Absent
Herniated Viscera	Bowel ± Liver	Bowel only (usually "matted")
Associated Syndromes	Common (50%) - Trisomies 13/18/21	Uncommon (<10%)
Surgical Management	Nonurgent (often staged/paint & wait)	Urgent (silo or primary closure)

Operative / Procedural Notes

- **Ramstedt Pyloromyotomy (HPS):** Longitudinal incision of the hypertrophied pylorus muscle down to the submucosa, explicitly avoiding mucosal perforation. The muscle fibers are spread apart to allow the intact mucosa to bulge through.
- **Intussusception Manual Reduction:** If operating, the surgeon must gently **"milk" or squeeze** the intussuscipiens retrogradely (Hutchinson's maneuver). Never pull the bowel, as this dramatically increases the risk of iatrogenic tearing/perforation.
- **Kasai Portoenterostomy:** The fibrotic biliary remnant is excised flush with the porta hepatis. A Roux-en-Y jejunal loop is brought up and anastomosed directly to the exposed microscopic ductules at the liver hilum.
- **Silo Bag (Gastroschisis):** Spring-loaded silicone bag suspended above the neonate; relies on gravity and gentle daily manual compression to sequentially reduce visceral volume before final fascial closure.

Complications / Prognosis

- **NEC:** ~30% mortality (highest in youngest/lowest weight). It is the **leading cause of pediatric intestinal failure**. Survivors are at high risk for intestinal strictures and moderate-to-severe neurodevelopmental delays.
- **Biliary Atresia:** Success of the Kasai procedure depends primarily on the **age at initial operation**, post-op bile flow, and extent of parenchymal disease. Pigmented stools are expected within 2–3 weeks if successful.
- **Gastroschisis:** Long-term outcomes are generally excellent; morbidity is tied to the condition/length of the bowel and issues of dysmotility.

Past-Paper High Yield

- **Radiographic Signs of Obstruction:**
  - **Pneumatosis intestinalis** (gas within the bowel wall) is the hallmark, diagnostic radiographic sign of **NEC**.

- *Double bubble* = Duodenal atresia.
- *Target sign / Doughnut sign* = Intussusception.
- *Neuhauser's sign / Soap bubble* = Meconium ileus (mixing of meconium with air).

- **Hirschsprung Disease (Distal Colon Aganglionosis):** Characterized by the absence of ganglion cells. Diagnostic rectal biopsy shows a characteristic **loss (absence) of calretinin staining** and increased acetylcholinesterase staining. Hypertrophied nerve trunks are present, not hypertrophied myenteric plexuses.
- **Imperforate Anus Imaging:** The most appropriate initial modality to evaluate the distance between the rectal gas pouch and the perineum is a **cross-table lateral radiograph (invertogram)** taken in the prone position **after 24 hours of life** (allows swallowed air to reach the distal rectum).
- **Cleft Palate Concepts:** Commonly associated with recurrent otitis media leading to **acquired conductive hearing loss** (NOT congenital sensorineural hearing loss). Arises from failure of palatal shelves to fuse. Isolated cleft lip is more common than isolated cleft palate. Early repair can restrict midfacial growth.

Memory Pearls

- **Meckel Diverticulum Rule of 2s:**
  - Occurs in **2%** of the population.
  - **2:1** male-to-female ratio.
  - Often symptomatic by **2 years** of age.
  - Located **2 feet** (60 cm) from the ileocecal valve.
  - Typically **2 cm** wide and **2 inches** (5 cm) long.
  - Contains **2 types** of heterotopic mucosa (Gastric > Pancreatic).

Miscellaneous topics in pediatric surgery (dr. Abeer)

Core Concepts

- **Hypertrophic Pyloric Stenosis (HPS):**
  - Male-to-female ratio is 4:1.
  - **Risk factors:** First-born males, family history, younger maternal age, bottle (formula) feeding, transpyloric feeding in premature infants, macrolide (erythromycin) exposure.
  - **Etiology:** Multifactorial (genetic + environmental), associated with excessive substance P, decreased neurotrophins, deficient nitric oxide synthase, and gastrin hypersecretion.
- **Intussusception:**
  - Most common cause of small bowel obstruction in infants/young children.
  - Proximal bowel (intussusceptum) invaginates into distal bowel (intussuscipiens) → mesenteric compression → venous obstruction/bowel edema → arterial ischemia and necrosis.
  - **Primary:** No lead point, likely due to hypertrophied Peyer patches (typically ages 4–9 months, 2/3 are boys).

    - **Secondary:** Driven by a lead point (e.g., Meckel diverticulum, polyps, appendix, hemangioma, Henoch-Schönlein purpura, cystic fibrosis, celiac disease).
- **Meckel Diverticulum:**
    - Most common congenital anomaly of the GI tract; persistence of the proximal vitelline (omphalomesenteric) duct. True diverticulum arising on the antimesenteric border.
    - **Rule of 2s:** Occurs in **2%** of population, **2:1** male-to-female ratio, symptomatic by **2** years of age, located **2** feet (60 cm) from ileocecal valve, **2** cm diameter / **2** inches long, contains **2** types of heterotopic mucosa (gastric [most common] > pancreatic).
- **Biliary Atresia (BA):**
    - Rare sclerosing cholangiopathy causing neonatal jaundice; most common cause of pediatric end-stage liver disease and pediatric liver transplantation.
    - Isolated in 85% of cases; syndromic cases most commonly present as BASM (Biliary Atresia, Splenic Malformation [asplenia/polysplenia], and malrotation).

Diagnosis / Clinical Features

- **Hypertrophic Pyloric Stenosis:**
    - Presents in full-term neonates at **2–8 weeks old**.
    - **Nonbilious**, progressive projectile vomiting (of recent feeds).
    - Infant appears well initially but progresses to dehydration.
    - **Signs:** Visible gastric peristaltic waves, palpable pyloric **"olive sign"** (70–90% of cases).
- **Intussusception:**
    - Intermittent, severe cramping abdominal pain every 15–30 mins (infant draws knees up/hyperextends).
    - Vomiting (gastric early → bilious later).
    - **Late signs:** Lethargy, "currant jelly" stools, **Dance sign** (empty right iliac fossa on palpation due to bowel migration), palpable RUQ mass (<25%).
- **Meckel Diverticulum:**
    - Majority are asymptomatic (only ~4% symptomatic).
    - **Presentations:** Intestinal bleeding (30-56%), intestinal obstruction (14-42%), diverticular inflammation (6-14%).
    - In adults/elderly, carcinoid tumor is the most common neoplasm to develop in the diverticulum.
- **Biliary Atresia:**
    - Persistent neonatal jaundice, pale (acholic) stools, hepatomegaly.
    - Progresses to malabsorption of fat-soluble vitamins, malnutrition, anemia, and growth retardation.

Investigations

- **Hypertrophic Pyloric Stenosis:**
    - **Labs:** Hypochloremic hypokalemic metabolic alkalosis.

- **Ultrasound (Initial test):** Muscle thickness ≥4 mm and pyloric length ≥16 mm ("donut" or "target" sign in transverse view).
- **Upper GI Series:** Indicated if US is equivocal. Shows delayed gastric emptying, narrowed/elongated canal (**"string sign"** or **"beak sign"**).

- **Intussusception:**
  - **Ultrasound (Gold standard): "Target"** or **"donut"** sign (transverse plane), **"Pseudokidney"** sign (longitudinal plane).
  - **Abdominal X-ray:** May show soft tissue mass effect, absent RLQ gas, or signs of small bowel obstruction.
- **Meckel Diverticulum:**
  - **Meckel Scan (Technetium-99m pertechnetate):** Used for bleeding presentation to detect heterotopic gastric mucosa (25% false-negative rate).
- **Biliary Atresia:**
  - **Labs:** Direct hyperbilirubinemia, elevated GGT, TORCH titers, alpha-1 antitrypsin.
  - **Ultrasound: "Triangular cord sign"** (cone-shaped fibrotic mass at portal vein bifurcation).
  - **Hepatobiliary Scintigraphy (HIDA):** Normal hepatic uptake but *zero excretion* into the intestine at 24 hours.
  - **Diagnostic Gold Standard:** Direct surgical observation / surgical cholangiography (shows no flow into duodenum).

Relevant Guidelines

- **Anatomic Classification of Biliary Atresia:**
  - **Type I:** Atresia of the common bile duct.
  - **Type IIa:** Atresia of the common hepatic duct.
  - **Type IIb:** Atresia of the common bile duct AND common hepatic duct.
  - **Type III:** Atresia of all extrahepatic bile ducts up to the porta hepatis (Most common, >90% of cases).

Management

- **Hypertrophic Pyloric Stenosis:**
  - **Preoperative (Crucial):** Resuscitation is mandatory. NPO, gastric decompression, IV fluids, and rigorous correction of electrolytes/alkalosis.
  - **Surgery:** Non-emergent Laparoscopic or Open Pyloromyotomy.
- **Intussusception:**
  - **Initial:** NPO, NGT decompression, IV fluid resuscitation.
  - **Non-Operative (First-line):** Hydrostatic or Pneumatic Reduction under fluoroscopy/US guidance (~85% success).
  - *Contraindications to non-operative reduction:* Perforation, peritonitis, persistent hypotension/sepsis.

    - **Operative:** Indicated for failed reduction, peritonitis, or presence of a secondary lead point.
- **Meckel Diverticulum:**
    - Resuscitation and stabilization (especially for bleeding).
    - Open or laparoscopic diverticulum wedge resection or segmental bowel resection with end-to-end anastomosis.
- **Biliary Atresia:**
    - **Kasai Procedure** (Roux-en-Y portoenterostomy).
    - **Liver Transplant Indications:** Failed Kasai (lack of bile drainage), developmental retardation, socially unacceptable complications. (Required in 2/3 of patients eventually).
- **Congenital Abdominal Wall Defects:**
    - *Initial for both:* Tertiary center delivery, NPO, NGT, IVF, wrap exposed contents in warm saline-soaked gauze centrally on the abdomen.
    - *Gastroschisis Surgery:* Urgent. Primary closure or Staged closure (using a spring-loaded preformed silastic silo bag suspended for gradual, gravity-fed reduction).
    - *Omphalocele Surgery:* Non-urgent. Primary closure (small defects), Staged closure (mesh/silo), or **"Paint and Wait"** scarification technique using escharotic agents (silver sulfadiazine, betadine) for giant omphaloceles.

Operative / Procedural Notes

- **Pyloromyotomy (Ramstedt Procedure):** Longitudinal incision through the serosa and superficial hypertrophied muscle; fine spreading forceps are used to split the remaining muscle fibers until the intact submucosa/mucosa bulges outward.
- **Intussusception Manual Reduction:** Must be done by gentle distal-to-proximal pressure ("milking") on the intussuscipiens. **Never** pull or use traction on the bowel to avoid iatrogenic perforation.
- **Hydrostatic/Pneumatic Reduction (Fluoroscopy):** Look for the **"claw sign"** (advancing edge of intussusceptum meeting contrast). Complete reduction is confirmed by extensive reflux of air/contrast into the terminal ileum.
- **Kasai Portoenterostomy:** The fibrotic extrahepatic biliary remnant is transected, and a Roux-en-Y jejunal loop is anastomosed *directly* to the exposed hepatic parenchyma at the porta hepatis to drain microscopic ductules.

Complications / Prognosis

- **Pyloromyotomy:** Mucosal perforation (1-2%), immediate postoperative emesis (very common), prolonged emesis (less common, implies GERD or incomplete myotomy).
- **Biliary Atresia:** Good outcome post-Kasai is heavily dependent on the age at initial operation, success of early bile flow, and presence of microscopic ducts. Pigmented stool normally appears 2-3 weeks post-op. Complications include cholangitis, portal hypertension, and hepatopulmonary syndrome.
- **Gastroschisis:** Long-term outcomes are generally excellent. Morbidities are strictly related to bowel conditions (motility disorders, short bowel, atresia) and prematurity.

- **Omphalocele:** Prognosis and long-term morbidity (failure to thrive, GERD, pulmonary insufficiency) are heavily dependent on associated genetic/cardiac anomalies.

Past-Paper High Yield

- **Omphalocele vs. Gastroschisis Differentiators:** Exam questions frequently test the ability to distinguish these two neonatal abdominal wall defects:

FEATURE	OMPHALOCELE	GASTROSCHISIS
Defect Location	Central (Umbilicus)	Strictly to the **right** of the umbilicus
Membranous Sac	**Present** (protects bowel)	**Absent** (exposed matted bowel)
Associated Anomalies	**High (50-70%)** - Severe cardiac, Trisomy 13, 18, 21	Uncommon (<10%) - Mostly isolated
Surgical Urgency	Nonurgent	Urgent
Primary Prognostic Factor	**Associated anomalies** (dictates high mortality)	**Condition of the exposed bowel** (atresia/ischemia)

- *Note:* Routine delivery by elective C-section does *not* significantly improve long-term survival in either condition.

Memory Pearls

- **"Olive sign"**: Palpable mass in Hypertrophic Pyloric Stenosis.
- **"String / Beak sign"**: UGI series finding for HPS.
- **"Target / Donut sign"**: Ultrasound finding for Intussusception (transverse) and HPS.
- **"Pseudokidney sign"**: Ultrasound finding for Intussusception (longitudinal).
- **Dance sign**: Empty right iliac fossa indicative of migrated intussusception.
- **"Triangular cord sign"**: Pathognomonic ultrasound finding for Biliary Atresia.
- **Rule of 2s**: Meckel diverticulum characteristics (2%, 2 feet from IC valve, 2 inches long, 2 ectopic tissues).

Inguinoscrotal diseases (dr. Abeer)

Core Concepts

- **Pathogenesis of Pediatric Inguinal Hernia & Hydrocele:**
  - Both result from the failed obliteration of the **patent processus vaginalis (PPV)**, a developmental embryological anomaly (indirect hernias).
  - **Normal:** PPV obliterates in the inguinal canal; remains only as the tunica vaginalis in the scrotum.
  - **Non-communicating hydrocele:** Closed PPV with trapped peritoneal fluid in the tunica vaginalis.

- **Communicating hydrocele:** Narrow PPV remains open, allowing peritoneal fluid to flow in and out. If it widens, abdominal contents can pass through, becoming an inguinal hernia.
- **Inguinal hernia:** Wide PPV allowing descent of abdominal organs/viscera.
- **Hydrocele of the cord (encysted):** Localized fluid trapped along the spermatic cord, closed off proximally and distally.

- **Epidemiology:**
  - **Hernia incidence:** ~5% of all children. **Inversely related to gestational age at birth** (10–30% in premature infants).
  - **Laterality:** Right > Left (Right 60%, Left 30%).
  - **Gender:** Males > Females in full-term babies; **1:1 ratio in premature babies**.
  - 10% have a positive family history.
- **Undescended Testis (UDT) Embryology:**
  - **Abdominal phase:** Guided by the gubernaculum and Anti-Müllerian Hormone / Müllerian Inhibiting Factor (MIF).
  - **Inguino-scrotal phase:** Guided by the processus vaginalis and driven by **Testosterone**.
- **UDT Epidemiology:** 3% in term males; 33–45% in premature/low birth weight males. Testes usually descend in the first 6–12 months; incidence drops to 1% at 1 year of age.

Diagnosis / Clinical Features

- **Inguinal Hernias and Hydroceles:**
  - Mostly asymptomatic; diagnosed clinically during routine exams or by parents.
  - **Associated Conditions:** Prematurity, cystic fibrosis, meconium peritonitis, VPS (ventriculoperitoneal shunt), peritoneal dialysis, connective tissue disorders, mucopolysaccharidoses, abdominal wall defects.
- **Special Inguinal Hernias:**
  - **Sliding hernia:** Sac may contain fallopian tube, ovary, or bladder side-wall.
  - **Amyand's hernia:** Contains the appendix.
  - **Littre's hernia:** Contains a Meckel diverticulum.
  - **Richter hernia:** Contains ischemic anti-mesenteric bowel border.
  - **Pantaloon hernia:** Combined direct and indirect hernias (more common in neonates).
- **Undescended Testes (UDT):**
  - Presentation: Empty hemiscrotum at birth or later visits. Check for signs of scrotal development (color, rugae).
  - **Palpable (70%):** Inguinal UDT, retractile (cremasteric overactivity), ascending (acquired), peeping, or ectopic testis.
  - **Non-Palpable (30%):** Agenesis, intra-abdominal, vanished testis (atrophied from perinatal torsion/trauma), or obscured (obesity, poor exam).
  - **Ectopic testis sites:** Prepenile, superficial ectopic/interstitial (anterior to abdominal wall aponeurosis), transverse scrotal (contralateral hemiscrotum), femoral, perineal.

- **Acute Scrotum / Testicular Torsion:**
  - Caused by the **"bell clapper deformity"** (intravaginal torsion) where the tunica vaginalis attaches abnormally high, allowing the testis to twist freely.
  - Bimodal peak: Neonates and adolescents. Rare after 25 years of age.

Investigations

- **Unilateral Non-Palpable Undescended Testis:**
  - **Diagnostic laparoscopy is the GOLD STANDARD.** It allows for direct definitive visual confirmation of intra-abdominal testes, blind-ending vessels, or vanishing testes, and enables immediate therapeutic intervention (laparoscopic orchiopexy).
  - *Do not* order Scrotal/Inguinal Ultrasound, CT, or MRI; they have inadequate sensitivity/specificity for locating intra-abdominal testes.
- **Bilateral Non-Palpable Undescended Testes:**
  - Check baseline gonadotropins (FSH, LH).
  - **hCG stimulation test:** Evaluates the functional presence of testicular tissue systemically (to rule out bilateral anorchia).
- **Testicular Torsion:**
  - Diagnosis is primarily clinical. **DO NOT delay surgery to obtain an ultrasound** if torsion is strongly suspected.

Management

- **Hydrocele:**
  - **Observation:** Simply observe until 1–2 years of age (90% of non-communicating and 65–70% of communicating resolve spontaneously).
  - **Surgical Indications:** Failure to resolve by 1-2 years of age or if a clinical inguinal hernia becomes apparent.
- **Uncomplicated Inguinal Hernia:**
  - Unlike hydroceles, hernias **must be repaired shortly after diagnosis** due to the high risk of incarceration; they do not resolve spontaneously.
  - Treatment is **Herniotomy** (high ligation of the patent processus vaginalis sac).
  - **Hernioplasty (Mesh) is ALMOST NEVER used in children** because they have normal posterior wall musculature and the defect is congenital (indirect). *Exception: Recurrent hernias in children with connective tissue disorders or mucopolysaccharidoses.*
- **Incarcerated Hernia:**
  - 1st line: Attempt manual reduction with sedation and firm, continuous pressure.
  - If reduced (90–95% success): Admit and schedule repair within 24–48 hours.
  - If failed reduction or contraindicated (signs of peritonitis, strangulation, septic shock): Admit for **emergent surgery**.
- **Undescended Testis (UDT):**

- Surgical correction (Orchiopexy) is performed to: reduce malignancy and infertility risk, prevent torsion, facilitate self-examination, and for cosmetic/psychological reasons.
- Hormone therapy (LH-RH agonist) is highly controversial.

- **Testicular Torsion:**
  - Urgent surgical exploration (salvage rate plummets after 6 hours of ischemia).
  - Manual detorsion in clinic should be done from **medial to lateral (like opening a book)** while awaiting definitive surgery.

Relevant Guidelines

- **Management Algorithm for Undescended Testis:**
  - **Unilateral Palpable:** Proceed directly to Dartos pouch orchiopexy.
  - **Unilateral Non-palpable:** Proceed to Diagnostic Laparoscopy.
    - If blind-ending vas deferens/vessels found → No further exploration.
    - If intra-abdominal testis found → Perform Orchiopexy, Staged orchiopexy (Fowler-Stephens), or Orchiectomy (if non-viable).
    - If vessels are seen exiting the internal ring → Proceed to inguinal exploration (Orchiopexy or Orchiectomy).
  - **Bilateral Non-palpable:** Check baseline gonadotropins (FSH, LH).
    - If FSH/LH elevated → Suspect anorchia.
    - If FSH/LH normal → Perform hCG stimulation test.
      - If increased testosterone → Laparoscopy +/- exploration.
      - If no response → Laparoscopy +/- exploration (suspect anorchia).

Operative / Procedural Notes

- **Herniotomy Details:** Involves an inguinal crease incision. In females, contents commonly include the round ligament. Adult-style tissue repairs (Bassini) are practically never performed on children.
- **Open vs. Laparoscopic Hernia Repair:**
  - Recurrence is identical (< 0.5%).
  - Laparoscopy detects metachronous hernias (preventing future surgeries on the contralateral side) and is faster for bilateral repairs but slower for unilateral repairs.
- **Open Contralateral Exploration:** Routinely justified in children with prematurity, younger age, female gender, or left-sided unilateral hernias.
- **Hydrocele Surgical Techniques:** High ligation of PPV + drainage (procedures include Lord's, Bottle, or Jabouly's procedure).

Complications / Prognosis

- **Hernia Repair Complications:** Recurrence is <1%, but higher in premature infants, post-incarcerated repairs, or children with VPS/connective tissue diseases.

- **Other risks:** Iatrogenic cryptorchidism, injury to the spermatic cord/testis (rare), hematoma, surgical site infection (SSI), persistent hydrocele. Chronic pain is remarkably uncommon in children.

Past-Paper High Yield

- **Hydrocele vs. Hernia progression:** Widening of a communicating hydrocele allows abdominal contents through, becoming an inguinal hernia.
- **Treatment timing:** Hydroceles are observed until 1–2 years; Hernias must be operated on promptly to avert incarceration.
- **Never pick Mesh/Hernioplasty or Bassini for pediatric hernias:** Children have healthy musculature. The only procedure required is a simple herniotomy (high ligation of the PPV).
- **Testicular Torsion Detorsion:** Must be done medial-to-lateral ("opening a book"). Salvage drops steeply after 6 hours. DO NOT delay for an ultrasound.
- **Torsion Pathophysiology:** Facilitated by the "bell clapper deformity" (high tunica vaginalis attachment).
- **Pediatric Hernia Demographics:** The incidence is inversely related to gestational age at birth (highly common in prematurity). Almost exclusively indirect.
- **Non-Palpable Testis Gold Standard:** Diagnostic laparoscopy is the absolute definitive diagnostic and therapeutic modality. Routine US/CT/MRI are consistently incorrect answers due to poor sensitivity for intra-abdominal testes.
- **hCG Test Role:** Used to prove the existence of functional testicular tissue in *bilateral* non-palpable testes, not for anatomical localization of a unilateral missing testis.

Pediatric solid tumors (dr. Raed)

Core Concepts

- **Neuroblastoma:**
  - The **most common** extracranial solid tumor in children.
  - An embryonal tumor arising from primitive sympathetic ganglion cells (neuroblasts) with the capacity to synthesize and secrete catecholamines.
  - Accounts for 5–10% of all childhood cancers.
  - Median age of onset: Infancy (~30%), **1–4 years (~50%)**, 10–14 years (~5%).
- **Nephroblastoma (Wilms' Tumor):**
  - The **most common** pediatric renal tumor and the second most common intra-abdominal malignancy (after neuroblastoma).
  - Arises from fetal undifferentiated **metanephric blastema tissue**.
  - Median age of onset: 3.5 years. Unilateral in 93% of cases.

Diagnosis / Clinical Features

Neuroblastoma:

- **Primary Sites: Adrenal medulla (~50%)**, abdominal sympathetic ganglia (25%), posterior mediastinum (20%), pelvis, and neck.
- **Typical Presentation:** Frequently presents as a palpable abdominal mass. Patients often appear **sick**, lethargic, and fatigued, with bone pain, weight loss, fever, sweating, and anemia.
- **Characteristic Paraneoplastic/Metastatic Signs:**
  - **"Raccoon eyes"** (periorbital ecchymosis/proptosis) due to retro-orbital metastases.
  - **Horner's syndrome** (miosis, ptosis, anhidrosis) from apical thoracic tumors.
  - **Dancing eye syndrome** (opsoclonus-myoclonus/progressive cerebellar ataxia): A classic but *rare* presentation.
  - **Progressive paraplegia** (from extradural cord compression/"dumb-bell" tumor).
  - **Hypertension** (~25%) from catecholamine production or renal artery compression.
  - Diarrhea (due to VIP release).

Nephroblastoma (Wilms' Tumor):

- **Typical Presentation:** Usually an **asymptomatic abdominal mass (80%)** in an otherwise well-appearing small child.
- **Other Features:** Abdominal pain (30–40%), hematuria (~20%), hypertension (25%).
- **Red Flag (Acute Abdomen):** Can present with tumor hemorrhage or rupture. **Physical Exam Pearl:** Avoid vigorous palpation of the abdomen to prevent tumor rupture.
- **Syndromic Associations:** Usually sporadic (>90%), but can be associated with Beckwith-Wiedemann Syndrome (BWS), WAGR syndrome, and Denys-Drash syndrome.

Investigations

Neuroblastoma:

- **Labs:** Markedly elevated **VMA** and **HVA** (catecholamine metabolites) in serum/urine. Elevated ferritin, LDH, and Neuron-Specific Enolase (NSE).
- **Imaging:**
  - Abdominal X-Ray: Tumor calcification (~50%).
  - Ultrasound: Differentiates solid vs. cystic; checks vessel involvement.
  - CT/MRI: Assesses tumor anatomy, metastases, and intraspinal extension.
  - **MIBG scan:** Preferred for bone imaging/metastatic evaluation.
- **Pathology:** Sheets of **dark blue round cells** with scanty cytoplasm. Characterized by **Homer-Wright pseudorosettes** (neuroblasts arranged around a neurofibrillary core), differentiating it from other round blue cell tumors (e.g., Ewing's sarcoma).
- **Diagnostic Criteria (Requires one of the following):**
  1. Clear histologic diagnosis OR increased urine/serum catecholamines.
  2. Evidence of bone marrow metastases AND elevated urine/serum catecholamines.

Nephroblastoma (Wilms' Tumor):

- **Labs:** Serum Cr, Urinalysis, LFTs, βFGF, Renin, Erythropoietin.

- **Imaging:**
  - **Contrast-enhanced CT scan of abdomen and chest:** The *preferred imaging modality* for staging (evaluates tumor extent, contralateral kidney, renal vein/cava extension, and lung metastases).
  - Ultrasound: Used for initial detection.
  - Echocardiogram: Required to evaluate potential right atrial tumor involvement.
  - DMSA scan: Assesses individual renal function in bilateral cases.

Management

Neuroblastoma:

- **Low risk:** Surgical resection alone (if no image-defined risk factors [IDRFs] pre-resection).
- **Intermediate risk:** Neoadjuvant chemotherapy → Surgery ± radiotherapy.
- **High risk (Sequence):** Induction therapy (Chemo, ALK inhibition, MIBG, mAbs) → Local control (Surgery, RT) → Consolidation (Stem cell transplant, Chemo, RT) → Immunotherapy (Dinutuximab).

Nephroblastoma (Wilms' Tumor):

- Two comparable pathways exist:
  - **SIOP Protocol:** Neoadjuvant chemotherapy (to downstage/reduce operative morbidity) → Surgery.
  - **COG Protocol:** Surgery → Adjuvant chemotherapy.
- **Surgical approach:** Nephrectomy including perinephric fascia and regional lymph nodes. Partial nephrectomy is reserved for bilateral cases, single kidney, or preexisting abnormalities.

Relevant Guidelines

- **Neuroblastoma INRGSS Staging System:**
  - **Stage L1 / L2:** Localized vs. locoregional disease based on Image-Defined Risk Factors (IDRFs).
  - **Stage M:** Distant metastatic disease.
  - **Stage MS (High Yield):** Metastatic disease in children **younger than 18 months** with metastases confined to **skin, liver, and/or bone marrow**. Characterized by hepatosplenomegaly and subcutaneous nodules (**"Blueberry muffin" spots**). Highly unique because *spontaneous regression is possible*, and >80% survive without specific treatment.
- **Wilms Tumor Screening Guidelines:**
  - Serial abdominal ultrasonography is recommended only for **high-risk patients** (e.g., children with Beckwith-Wiedemann or WAGR syndromes).
  - *Note: Universal infant screening for neuroblastoma via urine catecholamines is NOT recommended as it does not reduce mortality.*

Operative / Procedural Notes

- **Neuroblastoma "Dumb-bell" Tumors:** Intraspinal extension compressing the spinal cord requires careful neurosurgical/orthopedic evaluation; dictates neoadjuvant therapy.

- **Wilms Tumor Venous Extension:** Present in ~40% of cases into renal veins or IVC; managed operatively via venotomy and direct tumor removal.
- **Wilms Metastases:** Hepatic or pulmonary metastases are amenable to surgical metastasectomy.

Complications / Prognosis

Neuroblastoma:

- **Poor Prognostic Factors: MYCN gene amplification** (highly tested), DNA ploidy, MRP.
- **Good Prognostic Factors:** CD44 expression, TRKA expression, Age < 18 months (Stage MS). High-risk long-term survival is ~50%.

Nephroblastoma (Wilms' Tumor):

- **Poor Prognostic Factors:** Unfavorable histology (presence of **anaplasia** [10%]), recurrence.
- **Age Paradox:** Better survival in older children; decreased survival in infants (exact opposite of neuroblastoma). Overall survival is excellent (Stage I-III > 90%; Stage IV ~70%).

Past-Paper High Yield

- **Neuroblastoma Epidemiology & Presentation:** It is the most common *extracranial* solid tumor in children and typically presents as an abdominal mass. While paraneoplastic syndromes like opsoclonus-myoclonus ("dancing eyes") are classic, they are actually **rare** (seen in 2-3% of cases) and NOT the typical presentation.
- **The Age/Prognosis Trap:** Infants (< 1 year) with neuroblastoma have a **much better** prognosis than older children, as their tumors can spontaneously regress (Stage MS). Do not confuse this with Wilms tumor, where infants actually have a *worse* prognosis.
- **Wilms Tumor Staging Imaging:** A **contrast-enhanced CT scan of the abdomen and chest** is the preferred gold-standard imaging modality to stage a Wilms tumor. Ultrasound is only for initial detection. CT allows evaluation of the primary tumor, assessment of the contralateral kidney, and detection of pulmonary metastases.
- **Histology Trap:** Neuroblastoma is characterized by "round blue cells," but it **cannot** be differentiated from Ewing sarcoma solely by this feature (as both are round blue cell tumors). Differentiation requires noting Homer-Wright pseudorosettes or catecholamine markers.

Memory Pearls

- **N**euroblastoma = **N**eural crest, **N**eural symptoms (dancing eyes/paraplegia), **N**ormal infant survives (Stage MS), **N**otable catecholamines.
- **W**ilms = **W**onderful prognosis, **W**atch out for rupture (don't palpate), **W**ild mass but well-appearing child.

Foreign body aspiration (dr. Raed)

Core Concepts

- **Most Common Impaction Sites:**

    - **Esophagus:** The narrowest portion of the GI tract. Impactions occur at the cricopharyngeus sling (70% - most common), aortic arch (15%), and lower esophageal sphincter (15%).
    - **Airway:** The **right main stem bronchus** is the most common site due to its larger diameter, greater airflow, and smaller, more vertical angle of divergence from the trachea. In young children, the subglottic region is the narrowest part of the airway.
- **Tracheal Compression:** Due to the highly compliant nature of the pediatric trachea and its immediate anterior proximity to the esophagus, a large impacted upper esophageal FB can physically compress the posterior membranous wall of the trachea, causing atypical respiratory symptoms.
- **Epidemiology:** FB ingestions/aspirations are most common in children ≤ 5 years (oral exploration phase). Boys outnumber girls (2:1). It is the leading cause of mortality from unintentional injury (suffocation) in infants.

Diagnosis / Clinical Features

- **Esophageal Foreign Bodies:**
    - The vast majority present with a **completely normal physical exam**.
    - Symptoms, if present, include drooling, dysphagia, neck/throat pain, emesis, or respiratory distress (due to tracheal compression).
- **Gastrointestinal Foreign Bodies (Post-Esophageal):**
    - Most FBs distal to the esophagus are **completely asymptomatic**.
    - If symptomatic, they may present with abdominal pain, nausea/vomiting, distention, or peritonitis.
- **Airway Foreign Bodies:**
    - Commonly occur while eating or playing. Many aspiration events go unwitnessed.
    - Presenting symptoms: Respiratory distress, stridor, wheezing, and dysphonia.
    - *Chronic FBs* (missed aspirations): Present with persistent cough, atelectasis, bronchiectasis, or recurrent pneumonias.
- **Bezoars:** A tight collection of undigested material presenting with nausea, vomiting, weight loss, and distention.
    - *Phytobezoars* (plant matter): Typically cause obstruction at the **ileo-cecal valve**.
    - *Trichobezoars* (hair): Associated with trichotillomania.
    - *Rapunzel syndrome*: A gastric trichobezoar with a long tail extending into the proximal duodenum/small bowel.

Investigations

- **Radiography (Esophageal & GI):**
    - **Neck/Chest X-ray (AP and Lateral):** Used to locate radiopaque FBs.
    - **Coins:** Appear as a flat circle ("on its face") on an AP view and as a thick line ("from the side") on a lateral view when in the esophagus.

    - **Button Batteries:** Display a characteristic **"double contour rim"** or **"halo sign"** on X-ray, distinguishing them from standard coins.
- **Radiography (Airway):**
    - **Inspiratory and Expiratory Films:** Crucial for detecting radiolucent objects.
    - Look for **hyperinflation / "air trapping"** (seen in up to 60% of cases) due to the FB acting as a one-way valve, leading to a mediastinal shift away from the affected side on expiration.
    - *Clinical Trap:* **>50% of patients have a completely normal chest X-ray** within 24 hours of aspiration. A normal film does *not* rule out an airway FB. Definitive diagnosis requires bronchoscopy.

Management

- **Esophageal Coins:** Require retrieval if entrapped. Options include Magill forceps, endoscopy (rigid or flexible), or Foley balloon extraction with fluoroscopy. If they reach the stomach, observe.
- **Gastrointestinal FBs (Stomach and beyond):**
    - Small blunt objects (like coins) in an asymptomatic child will almost always pass spontaneously.
    - Management: **Expectant observation** with follow-up X-rays in 4–6 weeks. Routine admission, immediate endoscopy, or laparotomy is contraindicated.
    - Prokinetic agents and laxatives are *not* found to improve transit time.
- **Airway FBs:**
    - **Rigid bronchoscopy** is the definitive procedure for both diagnosis and therapeutic removal.
    - Flexible bronchoscopy is mainly used only for diagnosis.

Relevant Guidelines

- **Battery Ingestion Management Algorithm:**
    - *In Esophagus:* **Immediate removal** is mandatory (high risk of rapid corrosion/necrosis).
    - *In Stomach (or beyond) AND Asymptomatic:* Outpatient observation. Most (>80%) pass uneventfully within 48 hours. Follow up with an abdominal X-ray in 5-7 days.
    - *Symptomatic:* Requires endoscopic or laparoscopic removal depending on location.
- **Magnet Ingestion Management Algorithm:**
    - *Single Magnet:* Outpatient management. If not passed in 14 days, check abdominal X-ray.
    - *Multiple Magnets (or 1 magnet + 1 metallic object):* **Inpatient observation** with serial X-rays and surgical consult. If failure to progress in 48 hours or if symptomatic, prompt laparoscopic/endoscopic removal is required.
    - *Diagnostic Rule:* If unsure whether the child swallowed one or multiple magnets, treat as an inpatient for suspicion of multiple magnets.
- **Sharp Foreign Bodies:**
    - Carry a 15–35% risk of perforation. Endoscopic retrieval or close inpatient observation is warranted, though small straight pins may be managed conservatively.

Operative / Procedural Notes

- **Rigid Bronchoscopy:** The gold standard for pediatric airway FBs. It provides a larger working channel for grasping instruments, better airway control, and superior ventilation capabilities compared to flexible bronchoscopy.
- **Specialized Retrieval Tools:** For difficult distal airway FBs or objects with a central hole (e.g., beads), a **Fogarty balloon catheter** (passed through the FB, inflated distally, and pulled back) or a **Dormia basket** can be utilized.
- **Foley Balloon Technique (Esophagus):** Under fluoroscopy, a contrast-filled Foley balloon is passed past the esophageal FB, inflated, and gently pulled cephalad.
- **Gastrotomy:** Open surgical removal via laparotomy or laparoscopy is necessary for large trichobezoars (medical management/endoscopy is usually unsuccessful).

Complications / Prognosis

- **Batteries in the Esophagus:** Medical emergencies. They cause severe tissue injury within **1 hour** via pressure necrosis, low-voltage electrical currents, and leakage of alkali solution leading to **liquefaction necrosis**. Complications include esophageal perforation, tracheoesophageal fistula (TEF), strictures, and death.
- **Magnets:** Multiple magnets in different bowel loops forcefully attract each other across the bowel walls, causing localized pressure necrosis, bowel obstruction, volvulus, perforation, and fistulization.
- **Bronchoscopy Complications:** Include local bleeding/inflammation, laryngospasm, pneumothorax, and hypoxia. Rarely, a thoracotomy or lobectomy is required.

Past-Paper High Yield

- **Normal Exam in Esophageal FBs:** An uncomplicated esophageal foreign body (e.g., meat bolus, piece of chicken) typically presents with a completely normal physical and respiratory examination, provided there is no airway compression or perforation.
- **Tracheal Compression Signs:** If an upper esophageal FB is large enough to compress the highly compliant pediatric trachea, it typically presents with atypical respiratory symptoms (stridor, wheezing, brassy cough) rather than just dysphagia.
- **Asymptomatic GI FBs:** Small blunt objects (coins) that reach the stomach safely are predominantly asymptomatic and will pass spontaneously. The correct next step is outpatient observation and reassurance with follow-up plain films.
- **Bronchoscopy Differences:** *Rigid* bronchoscopy is vastly superior to *flexible* bronchoscopy for the actual removal of airway FBs in children due to airway control and instrument size.
- **Battery vs. Coin Management:** While a coin in the esophagus can be removed semi-electively (or via Foley balloon), a battery in the esophagus is an absolute emergency requiring immediate removal to prevent rapid liquefactive necrosis and TEF.
- *Mapped Non-FB Concept:* **Infantile Hemangiomas:** Oral propranolol (beta-blocker) is the first-line and most effective medical treatment for problematic, complicated, or life-threatening infantile hemangiomas (e.g., large facial lesions threatening the airway or causing visual field obstruction). Observation is only for small, uncomplicated lesions.

Memory Pearls

- **Right Main Bronchus:** Straighter, wider, greater flow → most common FB airway home.
- **Cricopharyngeus:** Narrowest esophageal point → traps 70% of ingested FBs.
- **Phytobezoar vs. Trichobezoar:** Phyto = Plants = Ileocecal valve obstruction. Tricho = Hair = Stomach/Gastrotomy.
- **Air Trapping:** FB acts as a one-way valve; lung hyperinflates on *expiration*.
- **Battery X-ray:** Look for the "Double Contour Rim" / "Halo Sign".

Pediatric urology (dr. Raed)

Core Concepts

- **Ureteropelvic Junction Obstruction (UPJO):**
  - **Epidemiology:** Male-to-Female ratio 2:1; Left-to-Right ratio 3:2. Bilateral in 10–40%.
  - **Etiology:**
    - *Intrinsic:* Congenital narrowing of the junction.
    - *Extrinsic:* Aberrant lower pole renal vessel (compresses the UPJ in ~30% of cases) or kinking secondary to severe Vesicoureteral Reflux (VUR).
- **Vesicoureteral Reflux (VUR):**
  - **Anti-reflux mechanism:** A normal ureterovesical junction (UVJ) acts as a passive one-way valve during bladder filling and detrusor contraction. A **5:1 ratio** of submucosal tunnel length to ureteral diameter is required for competence.
  - **Etiology:**
    - *Primary (Most Common):* Congenitally short intravesical ureteral tunnel.
    - *Secondary:* Distal obstruction (Posterior Urethral Valves), neurogenic bladder, or ectopic ureters.
  - **Epidemiology:** Female predominant; peak incidence at 3 years of age.
- **Hypospadias:**
  - A complex of anomalies consisting of an abnormal ventral urethral meatus, dorsal hooded foreskin, cleft glans, underdeveloped corpus spongiosum, and +/- ventral curvature (chordee).
  - **Risk Factors:** Increased maternal progesterone exposure (5x risk), estrogenic substances, low birth weight, and positive family history.
  - **Associated Anomalies:** Inguinal hernia and hydrocele (10%), undescended testes (8%).
- **Undescended Testis (Cryptorchidism):**
  - The vast majority are palpable. The most common location is just outside the external inguinal ring in the **superficial inguinal pouch** or within the inguinal canal.

Diagnosis / Clinical Features

- **UPJO:**

    - Currently, the most common presentation is **asymptomatic antenatal fetal hydronephrosis**.
    - Symptomatic cases (older children) present with flank/abdominal pain, palpable mass, hematuria, or recurrent UTIs.
- **VUR:**
    - Presents with recurrent UTIs (pyelonephritis), hypertension, reduced somatic growth, and eventually renal scarring and dysfunction.
- **Hypospadias:**
    - Classically presents in a newborn male with **spraying of the urinary stream** during micturition (loss of laminar flow) and an abnormal cosmetic appearance (dorsal hood).
- **Phimosis vs. Paraphimosis:**
    - *Physiological Phimosis:* Normal non-retractable foreskin at birth; mostly resolves spontaneously by age 5.
    - *Pathological Phimosis:* Secondary to balanitis/posthitis or Balanitis Xerotica Obliterans (BXO).
    - *Paraphimosis:* Retracted foreskin acts as a constricting band behind the glans, causing distal venous/lymphatic congestion and severe glans edema.

Investigations

- **UPJO Evaluation:**
    - **Postnatal US:** Primary anatomical tool to assess hydronephrosis and anteroposterior (AP) pelvic diameter.
    - **MAG3 Diuretic Renal Isotope Scan:** Scan of choice to evaluate **drainage and split renal function**.
        - *Drainage curve:* Normal emptying ($t_{1/2}$) is <20 min. A $t_{1/2}$ >20 min indicates significant obstruction.
        - *Split function:* Normal is 50:50. Intervention is required if function drops <40%.
    - **Micturating Cystourethrogram (MCUG / VCUG):** Absolutely mandatory in UPJO workup to rule out concomitant or mimicking VUR.
- **VUR Evaluation:**
    - **US:** Identifies hydro-uretero-nephrosis (HUN).
    - **DMSA Nuclear Scan:** Best for detecting **renal scarring** and calculating differential function.
    - **MCUG:** Defines the degree/grade of reflux (diagnostic gold standard).
    - **Direct Isotope Cystography (DIC):** Used primarily for follow-up to minimize radiation.

Relevant Guidelines

- **Society of Fetal Urology (SFU) Grading System for Antenatal Hydronephrosis:**
    - **Grade 0:** Normal kidney.
    - **Grade 1:** Minimal pelvic dilation.
    - **Grade 2:** Greater pelvic dilation without caliectasis.
    - **Grade 3:** Caliectasis without cortical thinning.

- **Grade 4:** Hydronephrosis with cortical thinning.

- **International Reflux Grading System (VUR via MCUG):**
  - **Grade I:** Reflux into non-dilated lower ureter only.
  - **Grade II:** Reflux reaches renal pelvis and calyces without dilatation.
  - **Grade III:** Mild to moderate dilatation + flat fornices.
  - **Grade IV:** Moderate dilatation and tortuosity + complete obliteration of sharp calyceal angles (convex fornices).
  - **Grade V:** Extreme dilatation and tortuosity + complete loss of papillary impressions.
- **Hypospadias Classification (by meatal location):**
  - **Distal (50%):** Glandular, coronal, sub-coronal.
  - **Middle (30%):** Mid-shaft.
  - **Proximal (20%):** Proximal penile, penoscrotal, scrotal, perineal.

Management

- **UPJO:**
  - Mainly **conservative management** (regular follow-up).
  - **Surgical indications:** Renal function deterioration (<40%), drainage $t_{1/2}$ > 20 minutes, or symptomatic presentation.
- **VUR:**
  - **Low-Grade (I-III):** High rate of spontaneous resolution. Managed with continuous antibiotic prophylaxis +/- Submucosal injection of bulking agent (Deflux).
  - **Surgical Indications:** Breakthrough UTIs despite prophylactic antibiotics (**failed medical treatment**), high-grade VUR (IV, V), deterioration of renal function, appearance of new renal scars, or secondary VUR causes.
  - *Note:* Surgical correction of VUR prevents future infections but **does not reverse pre-existing renal scarring**.
- **Hypospadias:**
  - **Timing:** Surgical repair should ideally be performed **before 18 months of age** (typically 6-18 months) to avoid psychological trauma.
  - **Contraindication:** Circumcision is absolutely contraindicated; the prepuce is needed as a vascularized flap for neourethra reconstruction.
  - **Preoperative optimization:** Topical testosterone/DHT cream or IM hCG injections can temporarily increase penile size and vascularity.
- **Paraphimosis (Surgical Emergency):**
  - *First-line (ER):* Manual reduction utilizing ice/sugar compresses (osmotic reduction of edema) or multiple needle punctures to drain trapped fluid.
  - *Second-line (OR):* Dorsal slit +/- formal circumcision under general anesthesia.
- **Circumcision Contraindications:**
  - *Absolute:* Family history of bleeding disorders (e.g., Hemophilia), active pathological jaundice.

- *Relative:* Hypospadias.

Operative / Procedural Notes

- **UPJO (Pyeloplasty):** Open, laparoscopic, or endourological. Involves excision of the narrowed segment and redundant pelvis, followed by anastomosing the ureter to the most dependent portion of the pelvis.
- **VUR (Ureteral Reimplantation):** Aims to lengthen the submucosal tunnel. Techniques include Cohen (transtrigonal - most common), Leadbetter-Politano (intravesical), and Lich-Gregoir (extravesical).
- **Hypospadias Repair (Snodgrass / TIP):**
  - Tubularized Incised Plate (TIP) urethroplasty creates the neourethra over a silicone stent.
  - Combined with chordee release (straightening), spongioplasty (dartos flap cover), and glansplasty.

Complications / Prognosis

- **Hypospadias Repair:**
  - *Early:* Bleeding, hematoma, infection, dehiscence.
  - *Late:* **Urethrocutaneous fistula (UCF)**, meatal stenosis, persistent chordee, urethral stricture.
- **Circumcision Complications:** Bleeding (most common cause of death if unnoticed in neonates), meatal stenosis (from chronic diaper friction), infection, iatrogenic hypospadias/fistula, entrapped penis. **Necrosis of the penis** occurs due to the contraindicated use of monopolar electrocautery.
- **VUR Surgery:** Persistent reflux, ureteric obstruction (kinking/ischemia), intravesical calculi, injury to vas deferens/fallopian tubes.

Past-Paper High Yield

- **UPJO Pitfalls:**
  - An MCUG is **mandatory** in suspected UPJO to rule out VUR, which can coexist or completely mimic UPJO.
  - A MAG3 scan evaluates *function and drainage rates*—it does **not** diagnose the anatomical cause of the obstruction.
  - Immediate nephrectomy is **never** the first-line treatment for unilateral UPJO.
  - UPJO does **not** present with bilious vomiting; it is usually entirely asymptomatic postnatally due to early antenatal detection.
- **Hypospadias Pitfalls:**
  - The **distal** location (glandular/coronal) is the most common presentation, NOT proximal.
  - Remember the link between maternal progesterone exposure and an increased risk of hypospadias.
  - "Spraying of the urinary stream" in a newborn is the classic tested presentation of hypospadias, differentiating it from posterior urethral valves (which present with weak stream/dribbling).
- **VUR Pitfalls:**

    - The most common cause of VUR is a **primary short intravesical ureteral tunnel length**, not ectopic ureters or PUV.
    - The definitive indication for surgical re-implantation is **failed medical management** (breakthrough UTIs while on daily antibiotics).
- **Cryptorchidism:** Ectopic locations (perineum/femoral) are rare. The most common location for an undescended testis is the **superficial inguinal pouch**.

Plastic

Exam Map

Revision Priority Tiers based on Past-Paper Distribution:

- **Tier 1 (Crucial - ~31% of exam):** Burns (Acute Management & Resuscitation).
- **Tier 2 (High Yield - ~38% of exam):** Normal Wound Healing, Pressure Sores, Soft Tissue Coverage, and Skin Cancers.
- **Tier 3 (Targeted Review - ~31% of exam):** Cleft Lip/Palate, Chronic Wounds, Burn Complications, Vascular Anomalies, and Hand Conditions.

High-Yield Exam Patterns & Decision Points:

- **Burn Resuscitation Traps:** The Parkland formula (4 mL × kg × %TBSA) is universally tested, especially the timing trap: the first 50% *must* be given within 8 hours *of the injury* (e.g., if a patient arrives 2 hours post-burn, give the first half over the remaining 6 hours).
- **Burn Management Absolutes:** Prophylactic systemic antibiotics are strictly contraindicated (they breed resistance). For chemical burns, copious water irrigation is the *only* correct answer; never attempt chemical neutralization.
- **Wound Healing Timeline & Mediators:** Macrophages are relentlessly tested as the most important cell. Tensile strength initiates at 3-4 days, caps at ~80% of normal skin, and is driven by the shift from early Type III to mature Type I collagen.
- **Grafts vs. Flaps Rules:** Full-Thickness Skin Grafts (FTSG) contract less and look better but have poorer take rates than Split-Thickness (STSG). Crucially, pressure sores *cannot* be treated with skin grafts (they lack bulk); they require vascularized myocutaneous flaps.
- **Skin Cancer Anatomic Predictors:** BCC classically appears on the upper lip/nose (morpheaform has the highest recurrence). SCC classically appears on the lower lip/external ear.
- **Melanoma Prognosis & Biopsy:** Breslow thickness (depth) is the single most powerful prognostic indicator. Excisional full-thickness biopsy is mandatory (never shave biopsy). Unlike BCC/SCC, melanoma is notoriously radioresistant; wide local excision is the standard.
- **Cleft Lip/Palate Surgical Trade-offs:** The "Rule of 10s" dictates lip repair at ~10 weeks/3 months. The exam highly tests the palate repair timeline (9–12 months): early repair ensures normal speech (prevents VPI) but severely restricts midface/maxillary growth.
- **Cleft Functional Traps:** Swallowing (deglutition) is fully *normal* in cleft infants; the mechanical defect is purely the inability to generate suction. Hearing loss is entirely *acquired* (recurrent otitis media), not congenital.
- **Vascular Tumors vs. Malformations:** Infantile hemangiomas (GLUT-1+) rapidly proliferate then naturally involute; management is expectant observation *unless* they obstruct vision or airway (prompting immediate beta-blockers/steroids). Vascular malformations grow proportionally for life and never involute.
- **Flow Characteristics in Vascular Lesions:** Arteriovenous Malformations (AVMs) are strictly high-flow (thrill/bruit present). Venous Malformations are low-flow (empty upon elevation, no thrill/bruit).

- **Ulcer Edge Diagnostics:** Memorize the edge associations: Sloping = Venous (medial malleolus/gaiter zone), Punched-out = Arterial/Diabetic, Undermined = Pressure, Everted = SCC (Marjolin's Ulcer in a chronic burn scar), Rolled = BCC. Biopsies must *always* be taken from the ulcer margin/edge, never the center.
- **Hand Emergencies:** Human fight bites over the knuckles are highly contaminated and *must* undergo delayed primary closure (never primary). A felon is a closed-space compartment syndrome of the pulp; it requires definitive surgical I&D, not just antibiotics.
- **Frostbite Algorithm:** The definitive primary treatment is rapid active rewarming in a 40–42°C water bath. Amputations must be strictly delayed for months ("Freeze in January, amputate in July") to allow tissue demarcation.

Soft tissue coverage

Core Concepts

- **Definition of Wounds:** A discontinuity of the epithelium due to trauma or pathological causes.
  - **Partial-thickness wounds:** Involve the epidermis and part of the dermis. Typically heal by regeneration (e.g., superficial second-degree burns) and are treated conservatively.
  - **Full-thickness wounds (Defects):** Involve the epidermis and entire dermis, often requiring reconstructive plastic surgery.
- **Modes of Wound Healing:** The body deals with tissue loss via Replacement of Lost Tissue (ROLT):
  - **Regeneration (Ideal):** ROLT by the identical tissue type. Provides maximal functional and cosmetic recovery. In humans, strictly limited to epithelium, hepatocytes, and bone.
  - **Fibrosis (Poor):** ROLT by fibrous tissue. Results in poor form, loss of function, contractures, and lengthy healing times.
- **Role of Tissue Transfer:** Transferring tissue (grafts or flaps) from a donor to a recipient site to avoid the functional and cosmetic morbidity of healing by fibrosis.
- **Graft Nomenclature based on Genetics:**
  - **Autograft:** Tissue transferred within the same individual.
  - **Isograft (Syngeneic graft):** Tissue transferred between genetically identical twins.
  - **Allograft:** Tissue transferred between non-identical humans.
  - **Xenograft:** Tissue transferred between different species.

Diagnosis / Clinical Features

- **Wound Classification based on contamination/tissue viability:**
  - **Incised wounds:** Caused by sharp, clean instruments. Minimal necrosis/contamination.
  - **Lacerated wounds:** Caused by blunt trauma. Jagged edges with moderate necrosis and contamination.
  - **Crushed wounds:** Heavy contamination and severe tissue devitalization (e.g., industrial or road traffic accidents).
- **Burn Depth Evaluation:**

- **Superficial/Partial-thickness:** Intact dermal capillary reflex (blanches upon pressure).
- **Full-thickness (3rd-degree):** Entire dermal vascular network is destroyed, resulting in fixed, non-blanching, painless skin with a leathery eschar. Hair follicles are completely destroyed.

- **Benign vs. Malignant Skin/Soft Tissue Masses:**
  - **Fibroadenoma:** Benign breast parenchyma tumor. Highly mobile ("breast mouse") and *not* fixed to overlying skin.
  - **Epidermoid/Sebaceous cysts & Skin malignancies (BCC/SCC):** Can present as fixed masses attached to the skin.

Management

- **Hierarchy of Reconstructive Closure:**
  1. Direct closure
  2. Healing by secondary intention
  3. Skin grafting (Split or Full thickness)
  4. Flaps (Local, regional, or distant/free)
  5. Prosthesis
- **Wound Closure Pathways:**
  - **Direct Closure:** Edges approximated without tension (sutures, glue, staples). Best for incised wounds.
  - **Excision & Closure:** Jagged lacerations must have necrotic edges excised to convert them into clean, incised wounds prior to direct closure.
  - **Delayed Primary Closure:** Mandatory for crushed, heavily contaminated wounds, or human bites. Primary closure of these wounds traps bacteria and causes severe infection (e.g., abscess, gas gangrene, tetanus). Wounds are opened, debrided, and irrigated until clean before closure.
  - **Healing by Secondary Intention:** Acceptable for small defects in cosmetically/functionally unimportant areas, or when other methods are unsafe.

Relevant Guidelines

- **Criteria for Good Tissue Transfer:**
  1. Replace "like with like"
  2. Maximize benefit to the recipient area
  3. Minimize morbidity/harm to the donor area
  4. Must be safe for the patient
- **Timing of Wound Closure:**
  - **Immediate (Now):** Used when the wound is clean (minimal bacterial load and free of necrotic tissue).
  - **Delayed (Later):** Used when the wound is contaminated (>6 hours old) or contains non-viable tissue.

- **The "6-Hour Rule":** Wounds older than 6 hours are considered contaminated and generally shouldn't be closed primarily.
- **Exception for the Face:** Wounds on the face can be closed primarily up to 24 hours post-injury due to its exceptionally rich vascularity.

Operative / Procedural Notes

- **Skin Grafts:** Depend *entirely* on the recipient bed for survival. Cannot take on avascular beds (bare bone without periosteum, exposed tendons without paratenon, cartilage without perichondrium, or over orthopedic hardware/prostheses).
  - **Split-Thickness Skin Grafts (STSG):** Includes epidermis and a portion of the dermis.
    - Harvested via dermatome (leaves rectangular defect).
    - Donor site heals by regeneration and can be re-harvested.
    - Ideal for covering very large areas (e.g., major burns).
  - **Full-Thickness Skin Grafts (FTSG):** Includes epidermis and the *entire* dermis (retaining hair follicles, sebaceous glands, and sweat glands).
    - Harvested from areas of loose skin so the donor site can be closed by direct primary closure.
    - Ideal for small defects requiring durable skin (hands/joints) or excellent cosmesis (face).
    - Requires a much richer blood supply to survive compared to STSG.
- **Flaps:** Tissue moved with its *own intrinsic blood supply* (pedicled or microvascular anastomosis).
  - Required to cover avascular beds (bare bone/cranium, tendons, hardware) and pressure ulcers (which need bulky, vascularized padding).
  - Can include skin, fascia, muscle, or bone.
  - **Donor Site:** While flaps offer bulk and sensation, large flap donor sites often cannot be closed primarily and frequently require an STSG.
- **Escharotomy:** Indicated for full-thickness circumferential burns to relieve compartment syndrome caused by the tourniquet effect of rigid, leathery eschar.
  - *Crucial note:* Do *not* wait for the loss of distal pulses to perform an escharotomy. Loss of pulses is a very late and dangerous sign of irreversible ischemia.
- **Tissue Expansion:** A reconstructive technique utilizing the "delay phenomenon."
  - Induces increased vascularity in tissues and creation of additional tissue.
  - Causes the epidermis to *thicken* (and dermis to thin).
  - Naturally forms a foreign body capsule around the expander.
  - High caution/contraindicated in irradiated skin due to poor elasticity.

Complications / Prognosis

- **Skin Graft Take:** Passes through two stages:
  1. **Plasmatic Circulation (Imbibition):** Days 1-2. Graft is nourished by diffusion from the recipient bed.
  2. **Neovascularization:** Days 2-3. Graft blood vessels anastomose with recipient bed vessels.

- *Signs of Take:* Graft is adherent, pink, and blanches with pressure.

- **Causes of Graft Failure:**
  - **Poor Vascular Bed:** The #1 predictor of graft survival.
  - **Hematoma/Seroma:** The most common clinical barrier between the graft and bed preventing take.
  - **Infection:** *Beta-hemolytic Group A Streptococcus (Streptococcus pyogenes)* is an **absolute contraindication** to grafting. It produces enzymes causing rapid graft lysis.
  - **Immobility:** Shearing forces disrupt neovascularization.
- **Prognosis in Soft Tissue Sarcoma:** The histological **GRADE** of the tumor is the single most important predictor for metastasis and overall survival. Soft tissue sarcomas possess a pseudocapsule (compressed tumor cells) that must NEVER be relied upon; leaving it during surgery causes an extremely high rate of local recurrence. Wide local excision with margins is mandatory.
- **Enterocutaneous Fistula Closure:** A *long* fistula tract (>2 cm) actually has a BETTER predictor for spontaneous closure than a short tract.
- **Chemical Burns:** Elemental sodium metal reacts explosively with water → DO NOT irrigate with water (smother with sand or mineral oil instead).

Past-Paper High Yield

- **STSG vs FTSG Dynamics:**
  - *Take rate:* FTSG has a **WORSE/poorer take rate** than STSG due to its thicker mass requiring higher metabolic/vascular support.
  - *Contracture:* FTSG contracts **LESS** than STSG.
  - *Adnexa:* FTSG includes the entire dermis; therefore, it retains sebaceous glands and hair follicles (hair can grow post-graft).
- **Meshed vs. Sheet Grafts:** Meshed grafts have a **BETTER take rate** than unmeshed (sheet) grafts. Fenestrations allow for serum/blood drainage, drastically reducing the risk of sub-graft hematoma. Meshing has zero impact on the healing of the completely separate donor site.
- **Graft Failure Etiology (Exam specific trap):** While hematoma is clinically the most common cause of graft failure, an exact past-paper key isolates **"Vascular degeneration"** as a discrete exam answer. Know both contexts.
- **Oncology Keys:**
  - Most common soft tissue sarcoma (historical classification used in exams) = **Fibrosarcoma** (now reclassified as undifferentiated pleomorphic sarcoma).
  - **Grade** is the single most important prognostic factor for sarcomas.
  - Wide margins are mandatory due to the tumor pseudocapsule.
- **Pressure Ulcers:** Skin grafts are the *wrong* answer for pressure sores; they lack bulk. Myocutaneous or fasciocutaneous flaps are required.
- **Compartment Syndrome Causes:** Factures, hemorrhage, snake bites, high-pressure injections, and full-thickness circumferential burns. *Superficial partial-thickness burns do NOT cause compartment syndrome* as they do not form an unyielding eschar.

- **Fingertip Amputations:** For a small (e.g., 1 cm) tip amputation with NO bone exposed, specific past-paper keys favor **Full-thickness skin graft** as the definitive definitive answer (over secondary intention).
- **Human Bites:** Highly contaminated → **Delayed primary closure**. Primary closure is the wrong answer.

Memory Pearls

- **Reconstructive axiom:** *"If you rob Peter to pay Paul, Peter should be able to afford it"* (Minimize donor site morbidity).
- **Fistula Non-Closure Mnemonic (FRIEND): F**oreign body, **R**adiation, **I**nfection/Inflammation, **E**pithelialization, **N**eoplasm, **D**istal obstruction. (A long tract is *not* a FRIEND, meaning it *can* close spontaneously).
- **The "Thick/Thin" Graft Rule:** The thicker the graft (FTSG), the better the functional/cosmetic result, but the *harder* it is to take successfully.

Burn

Core Concepts

- **Hypermetabolic & Catabolic State:** Massive burns trigger the highest hypermetabolic response of any disease. Driven by huge elevations in catecholamines, cortisol, and glucagon (not suppressed), alongside severe insulin resistance. Results in a profoundly **negative nitrogen balance**.
- **Burn Shock Pathophysiology:** Widespread endothelial cell contraction and gap formation lead to severe capillary leakage. This causes a massive fluid shift from the intravascular to the interstitial space (third spacing), resulting in hypovolemic shock.
- **Hemoconcentration:** During initial shock, fluid loss leaves blood highly concentrated. An elevated hemoglobin/hematocrit signifies severe fluid deficit (under-resuscitation), *not* fluid overload.
- **Zones of Injury:** The *zone of stasis/ischemia* is initially viable but can irreversibly progress to necrosis if poorly perfused.
    - Prevent conversion to necrosis by: good fluid resuscitation, adequate oxygenation, and **elevation** of injured extremities.
    - *Trap:* Placing limbs in a dependent position increases hydrostatic pressure, worsens edema, compresses microcirculation, and expands tissue necrosis.
- **Body Water Dynamics:** Total body water percentage steadily decreases with age (due to muscle loss and fat gain). Total body water is ~60% of weight, of which extracellular fluid (ECF) is 20% (1/3), and plasma is 1/4 of the ECF.

Diagnosis / Clinical Features

- **First-Degree Burns:** Involves epidermis only. Clinically presents as pain and erythema. Heals within 1–6 days without scarring. *Not* included in Total Body Surface Area (TBSA) fluid calculations.
- **Second-Degree Burns (Partial Thickness):** Involves epidermis and part of the dermis.

    - *Signs:* Blisters (bullae), wet/weeping exudate, very painful.
    - *Vascularity:* The capillary bed is intact, meaning the skin **readily blanches** under pressure and retains elasticity.
    - *Healing:* Generally heals spontaneously via regeneration from preserved dermal appendages within 1–4 weeks. Does *not* typically require skin grafting.
- **Third-Degree Burns (Full Thickness):** Complete destruction of epidermis, dermis, and appendages.
    - *Signs:* Forms an **eschar** (necrotic skin) that is **leathery, hard, and inelastic**.
    - *Sensory/Vascular:* Completely **painless/insensitive** (nerves destroyed) and **non-blanching** (dermal vessels thrombosed).
    - *Healing:* Cannot regenerate. Heals only by fibrosis (causing severe contractures) unless treated with surgical excision and skin grafting.
- **Chemical Burns:**
    - **Alkali:** Causes *liquefactive necrosis*, allowing deep tissue penetration and prolonged damage.
    - **Acid:** Causes *coagulative necrosis*, which creates a protein barrier that limits deeper penetration.
- **Electrical Burns ("Muscle Burn"):** Tissue damage is inversely proportional to electrical resistance.
    - *Low Resistance (Severely damaged):* Nerves, blood vessels, and massive muscle bulk.
    - *High Resistance (Less obvious damage):* Skin and bone. Bone generates massive heat, cooking adjacent deep tissue.
    - *Presentation:* Skin injury is deceiving ("tip of the iceberg"). Associated with violent tetanic contractions causing severe bone fractures and joint dislocations. Causes hyperkalemia due to massive rhabdomyolysis and cell death.

Investigations

- **Inhalation Injury Diagnosis:** Flexible fiberoptic bronchoscopy is the gold standard. Initial CXR and ABGs are notoriously poor and often completely normal on admission.
- **Carbon Monoxide (CO) Poisoning:** Diagnosed via carboxyhemoglobin levels. Standard pulse oximetry is unreliable (cannot differentiate between oxyhemoglobin and carboxyhemoglobin).
- **Renal Perfusion / Resuscitation Efficacy:** Hourly urine output via a Foley catheter is the absolute best, most reliable clinical indicator of end-organ perfusion (Goal: 0.5–1.0 mL/kg/hr in adults).
- **Electrical Burn Monitoring:** Requires continuous cardiac monitoring (arrhythmias) and monitoring for dark, port-wine urine (myoglobinuria).

Management

- **Immediate Resuscitation (ATLS):**
    - **Airway:** Upper airway soft tissue edema peaks in the first 24 hours. *Tachycardia, progressive hoarseness, and inability to clear secretions* are clinical signs of impending airway obstruction requiring prompt intubation.

    - **Breathing:** Suspected CO poisoning requires immediate 100% Oxygen to displace tightly bound CO from hemoglobin.
- **Fluid Resuscitation:**
    - Uses Ringer's Lactate (Constituents: Na 130, K 4, Ca 3, Cl 109, Lactate 28 mEq/L; *No Magnesium*).
    - **Colloids (e.g., albumin) are avoided** in the first 24 hours as capillary leak allows them to enter the interstitium, pulling fluid and worsening edema.
    - *Exceptions needing MORE fluid than Parkland calculation:* Inhalation injury, high-voltage electrical burns, delayed resuscitation, and pediatric patients.
- **Analgesia:** Must be administered via the **Intravenous (IV) route only** in small incremental doses. Intramuscular (IM) and subcutaneous absorption is highly erratic due to massive fluid shifts and shock.
- **Systemic Antibiotics:** Prophylactic systemic antibiotics are **strictly contraindicated**. They do not decrease mortality or sepsis; instead, they select for highly resistant microbial strains and increase fungal infections.
- **Chemical Burn First Aid:**
    - **Copious water irrigation** is the definitive first step. Do *not* attempt to neutralize the agent (neutralization is an exothermic reaction that creates heat, causing an additive thermal burn).
    - *Alkali burns* require prolonged irrigation (1–2 hours) due to deep penetrative liquefaction. *Acid burns* require ~30 minutes.
    - *Exceptions:* Elemental metals (Sodium, Potassium, Lithium) violently react with water; smother with sand or mineral oil. Hydrofluoric (HF) acid requires Calcium Gluconate. Phenol requires Polyethylene Glycol (PEG).
- **Electrical Burn Management:** Parkland formula cannot be used (deep injury is not reflected by skin surface area). Titrate IV fluids strictly to urine output to flush myoglobin and prevent Acute Kidney Injury (AKI). Alkalize the urine. Watch for compartment syndrome.
- **Nutrition:** Early enteral feeding (within the first 24 hours) is essential to preserve gut mucosa, prevent bacterial translocation, and manage hypermetabolism. A "half-strength" formula refers to 50% formula mixed with 50% free water.

Relevant Guidelines

- **Parkland Formula:**
    - Calculation: **4 mL × Patient Weight (kg) × % TBSA** (includes only 2nd and 3rd-degree burns).
    - **Timing:** The first half (50%) of the calculated volume is given over the **first 8 hours** *starting from the time of the burn*. The remaining half is given over the next 16 hours.
    - *Exam Trap:* If a patient arrives 2 hours post-injury, the first half of the fluid must be given over the remaining 6 hours of that initial 8-hour window.
- **Rule of Nines (Adult TBSA Estimation):**
    - Head & Neck: 9%
    - Anterior Trunk: 18%
    - Posterior Trunk: 18%

- Upper Limbs: 9% each (4.5% ant / 4.5% post)
- Lower Limbs: 18% each (9% ant / 9% post)
- Perineum/Genitalia: 1%

- **Lund and Browder Chart (Pediatric TBSA):**
  - Adjusts for proportions changing with growth.
  - The **Head** proportion is much larger in infants (roughly 18–19% in a newborn) and steadily decreases with age.
  - The **Lower Limbs** proportion is smaller in infants and proportionally increases with age.
- **Palm Method:** The patient's palmar surface equates to ~1% TBSA. Good for scattered burns.
- **Admission Criteria to Burn Ward:**
  - Burns requiring IV fluids (Adults >15% TBSA, Children >10%).
  - Any full-thickness (3rd-degree) burn >2% TBSA (requires excision/grafting).
  - Burns to special areas (face, hands, perineum).
  - All electrical, chemical, and inhalation injuries.

Operative / Procedural Notes

- **Early Tangential Excision and Grafting:**
  - Standard of care for deep thermal burns.
  - *Systemic Benefits:* Significantly decreases incidence of burn wound sepsis, shortens hospital stay, preserves joint mobility (less contracture), and blunts the catabolic response.
  - *Surgical Drawbacks:* Highly vascularized tissue removal is intensely bloody, significantly **increasing the requirement for intraoperative blood transfusions** and risk of hypothermia.
  - *Biological Dressings:* Used as a temporary cover when burn size exceeds available donor sites, allowing donor sites to heal for re-harvesting.
- **Escharotomy:**
  - Indicated for circumferential full-thickness (3rd-degree) burns of the limbs, chest, or neck.
  - Dead eschar loses elasticity and acts as a tourniquet; underlying edema raises pressures above capillary thresholds, causing ischemia.
  - Mid-lateral longitudinal incisions are made through the eschar down to the subcutaneous fat and across joints to release the tension.
- **Fasciotomy:**
  - The definitive treatment for true compartment syndrome (elevated pressure within a closed fascial space), commonly seen in severe electrical burns due to massive deep muscle necrosis.

Complications / Prognosis

- **TBSA Dictates Systemic Outcomes:** Fluid requirements, nutritional requirements, severity of the hypermetabolic response, risk of sepsis, and overall mortality.
- **Depth & Location Dictate Local Outcomes:** Wound healing time and the likelihood of long-term joint contractures depend on burn depth and anatomic location (not total % TBSA).

- **Leading Causes of Death:**
  - Early (first 24-48 hrs): Hypovolemic/Cardiovascular shock due to under-resuscitation.
  - Late / Overall: Respiratory failure secondary to bronchopneumonia (often following inhalation injury) and Burn Wound Sepsis.
- **Burn Wound Sepsis:** *Pseudomonas aeruginosa* is the most common and lethal organism in late burn wound infections, thriving in moist eschar.
- **Sensory Nerve Recovery:** Following nerve damage, the sequence of regaining sensory modalities is predictable based on fiber myelination: **Pain returns first**, followed by light touch, then temperature.

Past-Paper High Yield

- **TRAP:** Do NOT attempt to neutralize chemical burns. Always select *copious water irrigation*.
- **TRAP:** Administering systemic prophylactic antibiotics is WRONG. They do not prevent infection; they select for deadly resistant organisms.
- **TRAP:** Do NOT count 1st-degree burns (erythema only) in the Parkland formula calculation.
- **TRAP:** High hemoglobin/hematocrit implies severe fluid deficit (hemoconcentration), *not* fluid overload.
- **TRAP:** Absent pulse is a very *late* sign of compartment syndrome, not an early one. Pain out of proportion and pain on passive stretch are early signs.
- **TRAP:** PET scans are *not* used in the acute setting to estimate electrical tissue injury.
- **TRAP:** When calculating TBSA for both lower limbs entirely burned, it is 18 + 18 = 36%.
- **TRAP:** Electrical burns typically show hyperkalemia (cell lysis), not hypokalemia.

Complications of burns (dr. Bareqa)

Core Concepts

- **Definition:** Tissue injury caused by heat (most common: fires, scalding), electricity, chemicals (strong acids/bases, oxidants), or radiation (sunburn, medical radiation).
- **Epidemiology:** Results in ~4,000 deaths annually (fire/smoke inhalation) and 25,000 specialized burn center hospitalizations in the US.
- **Wound Healing & Scarring (Highly Tested):** Deep burns frequently result in fibroproliferative disorders (hypertrophic scars and keloids) due to excessive collagen deposition and decreased degradation.
  - **Hypertrophic Scars:** Stay within the original wound boundaries. Typically improve over time. More common in younger individuals due to robust inflammatory/fibrotic responses and increased skin tension.
  - **Keloids:** Extend beyond the boundaries of the original wound. Do *not* improve over time. Have strong genetic predispositions (more common in darker skin). Strictly benign with *no* precancerous potential.

Diagnosis / Clinical Features

Burn severity is classified by the depth of tissue damage:

- **First degree (Superficial):**
  - Limited to the epidermis (e.g., typical sunburn).
  - Erythematous, painful, and edematous, but **no blistering**.
  - Heals in < 1 week with **no scarring**.
- **Second degree (Partial Thickness):**
  - Involves epidermis and variable dermis. Erythematous with **blistering/raw skin**.
  - **Superficial partial:** Blanches with pressure.
  - **Deep partial:** Does *not* blanch with pressure.
  - Very painful; takes weeks to heal. Prone to local infection/cellulitis.
- **Third degree (Full Thickness):**
  - Loss of epidermis and entire dermis.
  - **Painless** (due to nerve destruction).
  - Stiff, white-brown, leathery appearance (eschar).
  - Cannot re-epithelialize (requires debridement and grafting).
- **Fourth degree (Full Thickness):**
  - Destruction extending through subcutaneous tissue into fascia, muscle, and/or bone.
  - Appears **black and charred**.

Investigations

- **Burn Extent (TBSA):** Accurately map % Total Body Surface Area using the Rule of 9's, Lund and Browder chart, or the patient's palm (1% TBSA).
- **Inhalation Injury Assessment:**
  - Obtain ABG and Carboxyhemoglobin (CO) levels. A CO level > 10% is clinically significant.
- **Tetanus:** Always check and update immunization status for burn patients.

Management

- **First Degree:** Moisturizers and topical anesthetics. No surgical intervention required.
- **Second Degree:** Debridement, topical antibiotics, silver sulfadiazine (especially for dirty wounds), petroleum jelly gauze, temporary skin substitutes.
- **Third & Fourth Degree (Major Burns):**
  - **Airway:** Intubate *before* respiratory failure/airway obstruction from progressive edema occurs. Administer 100% FiO_2 for suspected smoke/CO inhalation.
  - **Thermoregulation:** Keep the patient warm.
  - **Fluid Resuscitation:** Required for burns > 20% TBSA. Maintain urine output at **30–50 cc/hour**.
  - **Nutrition:** Enteral nutrition is crucial for the hypermetabolic state. *(Note: Having 180 cm of viable small bowel is adequate and is not a contraindication to enteral feeding).*

- **Adjuncts:** Hyperbaric Oxygen Therapy (HBOT).

Relevant Guidelines

- **American Burn Association (ABA) Transfer Criteria:**
  - 2^nd degree burns > 10% TBSA.
  - Any 3^rd degree burns.
  - Burns to face, hands, feet, genitalia, perineum, or major joints.
  - Electrical burns (including lightning) and chemical burns.
  - Inhalation injury.
  - Patients with significant pre-existing medical conditions.
- **Wallace Rule of 9's (Adult TBSA Estimation):**
  - Head & Neck = 9%
  - Anterior Thorax = 9% | Anterior Abdomen = 9% (Total front trunk = 18%)
  - Each Upper Limb = 9%
  - Each Lower Limb (Front) = 9% | (Total per leg = 18%)
  - Genitalia = 1%
- **Lund and Browder Chart:** Used primarily in pediatrics to adjust TBSA calculations for relative head-to-body proportion changes with age.
- **Parkland Formula (Fluid Resuscitation):**
  - **First 24 hours:** Lactated Ringer's = 4 mL × patient weight (kg) × % TBSA.
    - Give 1/2 of the total volume in the first 8 hours.
    - Give the remaining 1/2 over the next 16 hours.
  - **After 24 hours:** Lactated Ringer's = 1 mL/kg/% TBSA daily.
- **Abbreviated Burn Severity Index:** Variables include Sex, Age, Inhalation injury, Full thickness burn presence, and % TBSA. *(Note: Clinical literature states there is no evidence to support bedside use of this scoring system for individual decision-making).*

Operative / Procedural Notes

- **Excision & Grafting:** Standard definitive treatment for full-thickness burns.
- **Escharotomy:** Performed for circumferential deep burns to relieve mechanical restriction.
  - **Digits:** Midaxial longitudinal incisions to safeguard neurovascular bundles.
  - **Torso:** "H" or "shield" pattern incisions (bilateral anterior axillary lines joined by a transverse chest incision) to release chest wall restriction and allow mechanical ventilation.
- **Fasciotomy:** Required for deep electrical injuries or 4th-degree burns causing true compartment syndrome (incision extends deep through investing fascia).
- **Contracture Release:** Thick, band-like scar contractures (e.g., neck, eyelids) are surgically released using local tissue rearrangements like **Z-plasty** or transposition flaps (commonly used for post-burn eyelid ectropion).

- **Keloid Resection:** Simple surgical resection alone is strictly contraindicated due to extremely high recurrence rates (often returning larger). Must be combined with adjuvant therapies (intralesional steroids, radiation).

Complications / Prognosis

- **Airway Obstruction:** Secondary to severe rapidly progressive edema from facial/neck burns and inhalation injuries.
- **Infections:** Common organisms include *Pseudomonas aeruginosa*, *Streptococcus*, *S. aureus*, and *E. coli*.
- **Cellulitis:** Deep skin infection complication that causes systemic symptoms (fever, chills, malaise are *common*). Characterized by erythema and potentially lymphangitis.
- **Systemic Complications:** Hypermetabolic state and Acute Tubular Necrosis (ATN) from hypovolemia/myoglobinuria.
- **Marjolin Ulcer (Malignant Degeneration):** A highly aggressive Squamous Cell Carcinoma (SCC) arising in a chronic burn scar or non-healing wound. Distinctly more aggressive with a higher metastasis rate than standard sun-induced SCC.

Past-Paper High Yield

- **Keloid vs. Hypertrophic Scarring Differentiators:**
  - Keloids grow *beyond* original margins and do not improve over time; hypertrophic scars stay *within* margins and improve over time.
  - Both involve excessive collagen deposition and inflammatory response; distractor questions may falsely state that keloids simply have a "higher number of fibroblasts than normal ulcers" as the defining factor.
  - Keloids have *no* precancerous potential.
- **Marjolin Ulcer:** Always identify it as an *aggressive* SCC (not low-grade/less aggressive). It frequently metastasizes to lymph nodes and is treated with surgical excision.
- **Precursor Lesions to SCC:** Actinic keratosis, Bowen's disease (SCC in situ), and leukoplakia are true defined precursors. Keratoacanthoma is a low-grade SCC variant. Chronic ulcers are the *source* of Marjolin's but are not classically defined histologic "precursor lesions" like actinic keratoses.
- **Cellulitis Presentation:** Fever and chills are *common* true systemic symptoms of cellulitis; do not dismiss them as rare.
- **Enteral Feeding Contraindications:** Complete intestinal obstruction and severe hemodynamic instability (shock) are absolute contraindications. Having only 180 cm of viable bowel (Short Bowel Syndrome) is *not* a contraindication, as 180 cm is adequate length to absorb enteral nutrition.
- **Microbiology Trivia:** Anthrax (which can present with necrotic cutaneous lesions mimicking severe eschar) is caused by *Bacillus anthracis*, a gram-positive, spore-forming **bacterium** (not a fungus, virus, or parasite).

Memory Pearls

- **Superficial = Blanching; Deep = Non-blanching.**

- **3rd Degree = Painless** (nerves are burned away).
- **Marjolin = Malignant** (Aggressive SCC in a chronic burn scar).
- **Rule of 9s Trunk:** Don't forget the anterior trunk is 18% (9% chest + 9% abdomen) and posterior is 18%.
- **Keloids = Beyond borders; Hypertrophic = Halted at borders.**

Cleft lip & palate

Core Concepts

- **Definition:** The second most common congenital deformity overall (after clubfoot).
- **Epidemiology:**
  - Incidence: 1 in 700 live births.
  - **Combined cleft lip and palate is the most common presentation overall** (more frequent than isolated lip or isolated palate).
  - **Gender differences:**
    - Cleft lip (with or without palate): Statistically more common in **males** (2:1).
    - Isolated cleft palate: Statistically more common in **females** (2:1) due to later closure of palatal shelves.
  - **Ethnicity:** Highest in Native Americans and Asians (2/1000). Lowest in African populations (1/2500). No ethnic difference for isolated cleft palate.
- **Embryology & Pathogenesis (4th–12th week of gestation):**
  - **Cleft Lip:** Caused by the **failure of fusion between the medial nasal prominence and the maxillary prominence** during the 5th to 6th week. Pathologically, there is a failure of mesodermal proliferation in the midline, leading to epithelial tissue breakdown.
  - **Primary Palate:** Forms from the fusion of medial nasal prominences (intermaxillary segment). Contains the 4 incisors. Located anterior to the incisive foramen.
  - **Secondary Palate:** Forms from lateral palatal shelves (outgrowths of maxillary processes) that must elevate horizontally above a descending tongue and fuse anterior-to-posterior. Located posterior to the incisive foramen.
- **Etiology:** Multifactorial (genetic + environmental).
  - Most (70% CL±P, 50% CP) are non-syndromic.
  - Environmental teratogens: Maternal smoking, alcohol, rubella, steroids, anticonvulsants (Dilantin), folate deficiency.

Diagnosis / Clinical Features

- **Unilateral Cleft Lip:**
  - *Incomplete:* Intact nasal sill, varying lip skin/muscle gap.
  - *Complete:* Complete separation of lip and nasal floor. Orbicularis oris muscle fibers insert abnormally into the columella medially and nasal ala laterally. Columella deviates to the normal

side; affected ala is displaced laterally, inferiorly, and posteriorly.

- **Bilateral Cleft Lip:**
  - Severe premaxillary protrusion, symmetric nasal deformities, flared alas, and an extremely short columella.
- **Cleft Palate (Muscle Anatomy):**
  - Pathognomonic muscular defect: The *levator veli palatini* and *tensor veli palatini* fail to form a midline transverse sling. Instead, they run longitudinally along the cleft margins and insert abnormally into the posterior border of the hard palate.
- **Associated Syndromes:**
  - **Van der Woude Syndrome:** Most common syndrome. Autosomal dominant; presents with cleft lip/palate and characteristic **lower lip pits**.
  - **Pierre Robin Sequence:** Micrognathia (small mandible) leads to glossoptosis (tongue falls backward), causing airway obstruction and preventing palatal shelf fusion (cleft palate).

Management

- **Multidisciplinary Team:** Requires plastic surgery, ENT, speech pathology, orthodontics, and audiology.
- **Feeding:**
  - **Neuromuscular swallowing (deglutition) is INTACT/NORMAL.**
  - The primary problem is the **inability to create negative pressure for suckling** and subsequent nasal regurgitation.
  - Managed with special squeezable bottles (e.g., Haberman feeder) that deliver milk via compression, not suction.
- **Airway (Pierre Robin):** Prone positioning prevents glossoptosis. Severe cases may require mandibular distraction or tracheostomy.
- **Hearing:** 80-95% develop Otitis Media with Effusion (OME). Treated with early grommet (myringotomy) tubes.
- **Speech:** Unrepaired palates lead to Velopharyngeal Incompetence (VPI)—the inability to close the nasal airway during speech. 10-20% still manifest VPI (hypernasality) after repair, requiring a pharyngoplasty or pharyngeal flap.

Relevant Guidelines

- **Veau Classification (Cleft Palate):**
  - **Class I:** Incomplete cleft involving *only* the soft palate.
  - **Class II:** Cleft involving the hard and soft palate (up to the incisive foramen).
  - **Class III:** Complete unilateral cleft involving the lip and palate.
  - **Class IV:** Complete bilateral cleft involving the lip and palate.
- **The "Rule of Tens" for Cleft Lip Repair Timing:**
  - Surgery at **10 weeks of age**, at least **10 lbs of weight**, and **hemoglobin of 10 g/dL**.

- **Surgical Protocol / Treatment Timeline:**
  - **1 to 3 months:** Presurgical orthopedics (Lip taping and Nasoalveolar molding [NAM]).
  - **3 months:** Primary cleft lip repair + placement of ventilation ear tubes.
  - **9 to 12 months:** Cleft palate repair (Closure timing balances speech vs. facial growth).
  - **1 to 7 years:** Orthodontic treatment.
  - **7 to 8 years:** Alveolar bone grafting.
  - **18 years (skeletal maturity):** Midface advancement (Orthognathic surgery) and final rhinoplasty.

Operative / Procedural Notes

- **Presurgical Adjustments:**
  - *Taping:* Reduces cleft width nonsurgically.
  - *Nasoalveolar Molding (NAM):* Custom acrylic device with wires/stents to actively mold the nasal cartilage and alveolar ridge.
  - *Lip Adhesion:* A preliminary surgery (2-4 weeks) converting a wide complete cleft into an incomplete cleft to reduce tension for the definitive repair at 5-6 months.
- **Cleft Lip Repair Techniques:**
  - **Millard rotation-advancement:** The most widely used procedure for unilateral cleft lip.
  - *Goals for bilateral repair:* Attain symmetry, close orbicularis oris, reconstruct philtrum/tubercle, and position alar cartilages.
- **Cleft Palate Repair Techniques:**
  - Bardach two-flap palatoplasty and Furlow double opposing z-plasty.
  - *The Timing Dilemma:*
    - **Early repair** optimizes speech development (velar function).
    - **Early repair restricts midface maxillary growth**, leading to severe facial deformities (maxillary retrusion/Class III malocclusion) due to surgical scarring.
    - Compromise: Palate is repaired around 9–12 (up to 18) months to balance speech outcomes while minimizing skeletal restriction.

Complications / Prognosis

- **Hearing Loss:** Acquired conductive hearing loss is highly common.
- **Velopharyngeal Incompetence (VPI):** Results from defective palatal musculature (levator veli palatini), *not* from an isolated cleft lip.
- **Midface Hypoplasia:** Growth restriction secondary to hard palate repair scarring.

Past-Paper High Yield

- **Hearing loss in cleft palate is ACQUIRED, not congenital.** It is caused by recurrent otitis media secondary to Eustachian tube dysfunction (due to abnormal insertion of the *tensor veli palatini* muscle).

- **Swallowing vs. Sucking:** The actual pharyngeal swallow mechanism (deglutition) in a cleft palate infant is normal. The defect is purely a mechanical inability to generate negative suction pressure.
- **Epidemiology trap:** Remember the combo! **Combined cleft lip and palate** is the *most common* overall presentation. Cleft lip ± palate is male-predominant; isolated cleft palate is female-predominant.
- **Embryology of the Lip:** Cleft lip results from the failure of the **medial nasal process** and **maxillary process** to fuse.
- **The Surgical Trade-off:** EARLY surgical repair of the hard palate restricts midface maxillary growth (leading to facial deformity). DELAYED repair allows better facial growth but causes speech abnormalities. Traditional repair is lip at ~3 months, palate at ~9-18 months.
- **Velopharyngeal Incompetence (VPI):** Caused directly by an unrepaired or defectively repaired cleft palate, *not* an isolated cleft lip.
- *(Bonus non-cleft Plastic Surgery facts tested alongside this topic)*:
  - **Ear Embryology:** The auricle (pinna) develops from six hillocks of His originating from the 1st and 2nd branchial arches. A prominent ear (unfolded anti-helix) is a defect of the 2nd arch (though one specific key notably coded "first branchial cleft").
  - **Pilonidal Sinus:** It is an ACQUIRED condition heavily driven by hair drilling into the midline intergluteal cleft. Excision is standard. Much more common in males.
  - **Nerve injury:** Wasting of the intrinsic/small muscles of the hand follows injury to the **Ulnar nerve**.

Memory Pearls

- **Cleft Lip:** **M**ales, **M**edial nasal + **M**axillary process failure, **M**illard repair, **M**onth 3 (Rule of 10s).
- **Cleft Palate:** **F**emales, **F**urlow repair, **F**ailure of palatal shelves, **F**eeding suction issues (but normal swallow).
- **IncIsive Foramen:** The dividing line between prImary and secondary palate.
- **Van der Woude:** Has **two words** = associated with **two pits** (lower lip).
- **Pierre Robin Sequence:** Think "**P**ushed-back tongue" (**P**tosis of tongue/glossoptosis) and "**R**etreived jaw" (Micrognathia).

Vascular anomalies

Core Concepts

- **Fundamental Distinction (Mulliken & Glowacki / ISSVA):**
  - **Vascular Tumors (e.g., Infantile Hemangiomas):** Characterized by endothelial cellular proliferation. Typically undergo rapid postnatal proliferation followed by spontaneous involution.
  - **Vascular Malformations:** Structural anomalies of blood vessels present at birth. They **grow proportionally** with the child throughout life and do **NOT** undergo spontaneous involution.
- **Flow Characteristics:** Vascular malformations are divided into low-flow (capillary, venous, lymphatic) and high-flow (arteriovenous) lesions.

Diagnosis / Clinical Features

Vascular Tumors

- **Infantile Hemangioma:**
  - Accounts for 95% of vascular tumors. Most common in head/neck. Female:Male ratio = 2:1.
  - *Presentation:* Bright red "strawberry nevus" if superficial; soft and warm with prominent Doppler signal.
  - *Molecular marker:* Positive for **GLUT-1**.
  - *Growth phases:*
    1. **Proliferation (0–8 months):** Rapid, disfiguring growth.
    2. **Involution (up to 7–9 years):** Color darkens to a grey hue; fine capillary telangiectasia appears.
    3. **Final Involution:** 70% resolve by age 7; 90% by age 9. Replaced by a fibro-fatty residue.
- **Congenital Hemangioma:**
  - Fully developed at birth. **GLUT-1 negative**.
  - *RICH (Rapidly Involuting):* Regresses fully by 1 year, leaving an atrophic plaque.
  - *NICH (Non-Involuting):* Never regresses (plateaus); treated with excision.
  - *PICH (Partially Involuting):* Slow regression up to age 10.
- **Kaposiform Hemangioendothelioma (KHE):**
  - Locally aggressive infancy tumor.
  - Highly associated with **Kasabach-Merritt Phenomenon (KMP)** (profound thrombocytopenia and consumptive coagulopathy).
- **Pyogenic Granuloma (Lobular Capillary Hemangioma):**
  - Rapidly growing, bright red papule/polyp with a collarette of scale. Extremely friable; bleeds profusely after minor trauma.

Vascular Malformations

- **Capillary Malformations (Low-Flow):**
  - *Port-Wine Stain:* Present at birth, darkens (violaceous) and hypertrophies with age (can cause soft tissue/skeletal overgrowth).
  - *Nevus Simplex ("Stork Bite" / "Angel Kiss"):* Blanchable pink patches on nape of neck/glabella. Usually fades within 1–2 years.
- **Venous Malformations (Low-Flow):**
  - Blue, soft, compressible masses that **empty on elevation**.
  - **No palpable thrill and no audible bruit.**
  - *Blue Rubber Bleb Syndrome:* Multiple cutaneous VMs associated with gastrointestinal VMs (bleeding risk).
- **Lymphatic Malformations (Low-Flow):**
  - *Macrocystic:* e.g., Cystic hygroma (often in the neck).

- *Microcystic:* Presents with a characteristic "frog-spawn" or pebbly mucosal surface, prone to superficial hemorrhage and infection.

- **Arteriovenous Malformations (High-Flow):**
  - Direct connections between the arterial and venous systems **without a capillary bed**.
  - Clinically presents with **palpable thrill and audible bruit**.

Investigations

- **Clinical Diagnosis:** Most vascular anomalies are diagnosed based on history (timeline of growth) and physical exam.
- **Imaging:** MRI/US to assess deep tissue involvement and flow characteristics.
- **Labs:**
  - Coagulation profile (D-dimer/fibrinogen) for extensive Venous Malformations (risk of consumptive coagulopathy).
  - CBC to check platelets in suspected Kaposiform Hemangioendothelioma (Kasabach-Merritt).

Management

- **Infantile Hemangioma:**
  - **Uncomplicated: Expectant observation** is the gold standard (they spontaneously involute).
  - **Complicated** (Obstructing vision/airway, ulceration, high-output cardiac failure): **Requires immediate intervention**.
    - *1st Line Medical:* **Propranolol** (1–2 mg/kg/day) — induces vasoconstriction and promotes regression.
    - *2nd Line Medical:* **Steroids** (Intralesional or Systemic). *Note: Systemic carries a risk of rebound growth.*
    - *Interventional/Surgical:* Embolization (for high-output failure/bleeding), Excision, or Tracheostomy (airway obstruction).
    - *Laser:* Pulsed-dye laser is ONLY used for surface residual telangiectasia *after* involution (>10 years of age). It does not alter the natural history of the hemangioma itself.
- **Capillary Malformations:** Pulsed-dye laser (lightens color), camouflage. Surgery may be needed for lip/soft tissue hypertrophy.
- **Venous Malformations:** Compression garments, NSAIDs for pain, Sclerotherapy, Surgical excision.
- **Lymphatic Malformations:** Sclerotherapy (e.g., OK-432), Surgical excision.
- **Arteriovenous Malformations (AVMs):** Embolization (ethanol, glue, coils, Onyx), excisional surgery.
- **Targeted Molecular Therapy: Sirolimus (mTOR inhibitor)** is used for complex, mTOR-positive lesions (e.g., Kaposiform Hemangioendothelioma) as it halts protein synthesis and cell survival pathways.

Relevant Guidelines

ISSVA 2014 Classification of Vascular Anomalies

- **Vascular Tumors:**
  - *Benign:* Infantile hemangioma, Congenital hemangiomas (RICH, NICH, PICH), Tufted angioma.
  - *Borderline/Aggressive:* Kaposiform hemangioendothelioma.
  - *Malignant:* Angiosarcoma.
- **Vascular Malformations:**
  - *Simple (Slow Flow):* Capillary (CM), Venous (VM), Lymphatic (LM).
  - *Simple (High Flow):* Arteriovenous malformation (AVM), Arteriovenous fistula (AVF).
  - *Combined:* e.g., CVM, LVM.
  - *Syndromic Associations:* e.g., Klippel-Trenaunay (CM+VM+LM+limb overgrowth), Sturge-Weber (facial CM + leptomeningeal CM).

Schobinger Clinical Classification for AVMs

- **Stage I (Quiescence):** Pink/blue stain, warmth, arteriovascular shunting.
- **Stage II (Expansion):** Stage I + enlargement, pulsations, thrills, bruit.
- **Stage III (Destruction):** Stage II + dystrophic skin changes, ulceration, bleeding, pain, or tissue necrosis.
- **Stage IV (Decompensation):** Stage III + high-output cardiac failure.

Complications / Prognosis

- **Deprivation Amblyopia:** An infantile hemangioma on the eyelid that obstructs the visual axis will cause permanent visual loss if not treated immediately.
- **High-Output Cardiac Failure:** Can be caused by very large infantile hemangiomas or Stage IV Arteriovenous Malformations.
- **Kasabach-Merritt Phenomenon:** Life-threatening coagulopathy seen in Kaposiform Hemangioendotheliomas, not standard infantile hemangiomas.

Past-Paper High Yield

- **Hemangioma Observation vs. Intervention:** It is explicitly **FALSE** to say hemangiomas need "no intervention in all cases". An 8-month-old with an uncomplicated 7x7 cm or 3x3 cm hemangioma on the trunk/cheek requires **Expectant Observation**. However, an 8-month-old with a hemangioma on the eyelid obstructing vision requires **Immediate Intervention** (Steroids or Propranolol) to prevent deprivation amblyopia.
- **Growth Patterns:** The hallmark of Vascular Malformations is that they **grow proportionally** with the child and do **NOT** involute. Infantile hemangiomas proliferate rapidly in early infancy and then involute.
- **AVM vs VM Characteristics:**
  - AVMs are strictly **HIGH-flow** lesions (direct arterial-venous connection, lacking a capillary bed) presenting with a thrill/bruit.
  - VMs are **LOW-flow** lesions, present with no thrill/bruit, and are compressible.

- **Exam Key Trap:** A clinical scenario describing a 12-year-old with a lesion growing proportionally with no thrill or bruit classically describes a *Venous Malformation*. However, be aware that some legacy exam keys have erroneously listed *Hemangioma* as the answer. Rely on the clinical principles (proportional growth + no bruit = low flow malformation), but be cautious of poorly keyed past-paper questions.
- **Leukoplakia Malignant Potential (General Plastics crossover):** Leukoplakia is a premalignant lesion, but the malignant transformation rate to SCC is only **5-15%**, NOT 50%. (Keratoacanthomas resemble SCC clinically, and HPV/HSV lesions can undergo malignant transformation).

Memory Pearls

- **GLUT-1 (+):** Infantile Hemangioma.
- **GLUT-1 (-):** Congenital Hemangioma.
- **No Capillary Bed:** Arteriovenous Malformation (AVM).
- **Strawberry =** Infantile Hemangioma (Superficial).
- **Frog Spawn =** Microcystic Lymphatic Malformation.
- **Empty on Elevation =** Venous Malformation.

Chronic wounds

Core Concepts

- **Definition:** A wound that fails to heal in the expected time frame (e.g., 2–6 weeks) or proceeds through the repair process without producing an adequate anatomic and functional result.
- **Anatomy of an Ulcer:**
  - **Margin:** The normal skin immediately surrounding/adjacent to the ulcer.
  - **Edge:** The perimeter boundary; the relationship of the ulcer with the skin.
  - **Floor:** What we *see* (the exposed internal surface of the ulcer).
  - **Base:** What lies underneath and what we *feel* (deep tissues upon which the ulcer rests).
- **Primary Categories:** Ischemic (Arterial), Venous Stasis, Diabetic, and Pressure ulcers.
- **ICEBERG Principle:** In pressure ulcers, pressure expands outward and downward through subdermal tissues in a cone shape. Subcutaneous damage is much larger than the visible surface defect. Visible eschar indicates at least Stage III or higher depth.

Diagnosis / Clinical Features

- **Ischemic Arterial Ulcers:**
  - Painful at presentation (associated with intermittent claudication, rest pain, night pain).
  - Signs of peripheral arterial disease (PAD): diminished/absent pulses, decreased ankle-brachial index (ABI), pallor, scaling, hair loss, and dry skin.
  - Wound morphology: Shallow with smooth, punched-out margins; pale base with poor granulation tissue.
- **Venous Stasis Ulcers:**

    - Associated with deep or superficial venous incompetence; deeply incompetent chronic ulcers are typically painless.
    - **Location:** Classically in the "gaiter zone"—the lower medial 1/3 of the leg and ankle, often over **Cockett's perforator** (above the medial malleolus).
    - Wound morphology: Shallow, irregular margins with a red, fleshy granulation base.
    - Surrounding tissue: Marked hyperpigmentation (hemosiderin staining) and lipodermatosclerosis.
    - Pathophysiology: Venous valves fail, so the calf muscle pump does not adequately lower venous pressure during walking, leading to rapid return to severe venous hypertension (~90 mmHg) upon resting.
- **Diabetic Foot Ulcers:**
    - Multifactorial origin: neuropathy (loss of protective nociception), immune compromise, microvascular damage, and repetitive pressure.
    - Location: Often on pressure-bearing plantar surfaces (e.g., metatarsal heads).
    - Wound morphology: Deep, punched-out appearance surrounded by a thick, hyperkeratotic (calloused) rim.
- **Hidradenitis Suppurativa:**
    - A chronic inflammatory disorder of **apocrine sweat glands** (axilla, groin, perineum)—not eccrine glands.
    - Presents as bilateral, indurated, suppurating, painful, diffuse masses and sinus tracts. (Note: Carbuncles are simply localized clusters of furuncles, not HS).
- **Infectious Wound Complications:**
    - **Erysipelas:** Caused by Group A Streptococcus. Highly painful, features a sharply defined/raised edge, typically involves the lower limbs (not hands), and responds well to penicillin.
    - **Necrotizing Fasciitis:** Commonly Group A Streptococcus. Hemorrhagic bullae indicate severe deep ischemia. Immunosuppression and diabetes are major risks. Early aggressive debridement is lifesaving. *In advanced variants, muscles can be heavily involved and are not strictly spared.*
- **Wound Biofilms:**
    - Form a protective, pathological barrier preventing chronic ulcers from healing.
    - Characteristics: Slimy/shiny surface, strong adherence, highly resistant to antibiotics, polymicrobial.
    - *Healthy granulation tissue (red, bumpy, vascular) is the antithesis of a biofilm.*
- **Ulcer Edge Typology:**
    - **Sloping edge:** Gradual descent; typical of healing ulcers (e.g., venous stasis).
    - **Punched-out edge:** Sharp 90-degree vertical drop; typical of ischemic, diabetic neuropathic, and syphilitic ulcers.
    - **Undermined edge:** Lateral tissue loss beneath intact skin; typical of pressure injuries and tuberculous ulcers.
    - **Rolled edge:** Thick, raised, rounded borders; typical of Basal Cell Carcinoma (BCC).
    - **Everted (overturned) edge:** Margins turned outward; indicates rapid proliferation, typical of Squamous Cell Carcinoma (SCC) / Marjolin's ulcer.

Investigations

- **Ulcer Biopsy Technique:** A biopsy must *always* be taken from the **edge (margin)** of the ulcer, encompassing both healthy normal skin and the diseased ulcer tissue. This captures active pathology and the transition zone. Biopsies from the center/floor yield only non-diagnostic necrotic debris.
- **Pressure Sore Cultures:** Superficial wound swabs are essentially useless because they only culture colonizing skin flora. A deep tissue or bone biopsy is absolutely required to identify the true invading pathogen.

Management

- **Ischemic Ulcers: Revascularization** (bypass or angioplasty) is the primary step. Debridement is only performed *after* adequate blood flow has been established.
- **Venous Ulcers:** The cornerstone of treatment is **compression therapy** (decreases vessel diameter, prevents reflux, reduces edema, and lowers inflammatory cytokines). Life-long compression stockings are needed due to high recurrence rates.
- **Diabetic Ulcers: Eradication of infection** is paramount. Treatment demands a multidisciplinary approach including offloading pressure, rigorous glycemic control, debridement, and potential revascularization.
- **Pressure Ulcers:** The most important intervention is the **redistribution of pressure** (offloading) to prevent further ischemia. Subsequent care involves specialized dressings and debridement.
- **Tetanus Prophylaxis in Wounds:** Management routinely includes antibiotics, large-dose tetanus toxoid, human tetanus immunoglobulin (HTIG), and wound debridement. *Note: In some academic examination contexts, "convulsion control" may be deemed a secondary step compared to direct toxoid/source management, though it remains a clinical reality.*

Relevant Guidelines

- **The National Pressure Ulcer Advisory Panel (NPUAP) Staging System:**
  - **Stage 1:** Intact skin with non-blanchable persistent redness of a localized area (usually over a bony prominence).
  - **Stage 2:** Partial-thickness loss of dermis presenting as a shallow open ulcer with a red/pink wound bed, without slough. May also present as an intact or ruptured blister.
  - **Stage 3:** Full-thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon, or muscle are *not* exposed. Undermining may be present.
  - **Stage 4:** Full-thickness tissue loss with exposed bone, tendon, or muscle. High risk of associated osteomyelitis.
  - **Unstageable:** Full-thickness loss where the base is completely obscured by slough or black necrotic eschar. True depth cannot be determined until debrided.
  - **Suspected Deep Tissue Injury:** Intact skin with purple or maroon discoloration indicating deep underlying hematoma or necrosis caused by pressure/shear.

Operative / Procedural Notes

- **Compartment Syndrome & Fasciotomy:**

- Pathophysiology: Occurs when interstitial tissue pressure exceeds capillary perfusion pressure.
- Threshold: Fasciotomy is generally indicated when compartment pressures exceed 30 to 40 mmHg. An absolute compartment pressure above **40 mmHg** almost universally causes critical capillary ischemia and requires immediate definitive fasciotomy.

Complications / Prognosis

- **Marjolin Ulcer (Malignant Transformation):** Any chronic, non-healing wound is prone to malignant transformation (mostly SCC, rarely BCC). It is clinically suspected when a wound develops **everted (overturned) edges**. Biopsy of the edge is mandatory.

Past-Paper High Yield

- **Biopsy location:** Always from the *edge*, never the center.
- **Compartment pressure:** 40 mmHg is the classic definitive threshold requiring fasciotomy.
- **Hidradenitis suppurativa vs. Eccrine glands:** HS is exclusively a disease of the *apocrine* glands (axilla/groin).
- **Venous ulcer location:** Most common in the lower 1/3 of the leg and ankle (gaiter zone).
- **Pressure sore culture:** Superficial swabs are wrong/insufficient; select deep tissue or bone biopsy.
- **Erysipelas specifics:** Painful, caused by Group A Streptococcus, highly responsive to penicillin.
- **Necrotizing fasciitis trick:** It does *not* strictly spare muscle in all cases (advanced variants involve muscle).
- **Biofilm characteristic trick:** "Granulation tissue" is a healthy finding and is *not* a characteristic of a biofilm.
- **Ulcer definitions:** Know the precise difference between the floor (what we see) and base (what we feel).

Memory Pearls

- **Ulcer Edge Associations:**
  - **S**loping = Venous (**S**tasis)
  - **P**unched out = Arterial / **P**eripheral neuropathy (Diabetic)
  - **U**ndermined = Pressure (**U**lcer) / TB
  - **R**olled = BCC (Basal Cell)
  - **E**verted = SCC (Marjolin)

Normal wound healing & pressure sores

Core Concepts

Wound Closure Types

- **Primary Closure:** Immediate suturing of the wound.

- **Delayed Primary Closure:** Leaving the wound open to be closed with stitches after 3–5 days once clean (indicated for contaminated wounds).
- **Secondary Closure:** Healing by spontaneous scar formation, granulation, and epithelialization.
- **Tertiary Closure:** Coverage by a skin graft or vascularized flap.

Phases of Normal Wound Healing

1. **Phase 1: Hemostasis & Inflammation (Days 0 to 4-5)**
    - **Hemostasis is the absolute first step:** Involves the activation of the coagulation cascade to form a provisional fibrin clot.
    - **RBCs & Platelets:** Dominate immediately post-injury (0–24 hours).
    - **Neutrophils (PMNs):** Predominant cells at **24–48 hours**. They clear bacteria and debris. *Note: In a perfectly clean, surgically excised wound devoid of infection, PMNs are not strictly required for healing to proceed.*
    - **Macrophages:** The **most critical cells in wound healing**. They arrive and predominate from Days 2 to 5. Macrophages orchestrate the transition from inflammation to the proliferative phase by releasing a multitude of cytokines and growth factors.
    - **Cytokines:** Tumor Necrosis Factor-alpha (TNF-α), IL-1, and IL-6 are the most potent, central broad-spectrum inflammatory mediators. Monocytes and macrophages are major producers of TNF-α.
2. **Phase 2: Proliferation (Days 4 to 21)**
    - **Components:** Mesenchymal cell migration/proliferation, angiogenesis, epithelialization, and collagen synthesis.
    - **Fibroblasts:** Dominate from Day 5 to 21, actively depositing early collagen (primarily Type III).
    - **Tensile Strength Initiation:** Wound tensile strength begins to meaningfully rise at **3 to 4 days** coinciding with early fibroblast collagen deposition.
    - **Epithelialization:** Normal basal epithelial cells exhibit contact inhibition. Wounding causes **loss of contact inhibition**, triggering them to multiply and migrate across the wound bed.
        - *Primary closure:* Re-epithelialization seals the wound surface within **24 to 48 hours**, creating a water-tight barrier against bacteria.
        - *Partial-thickness wounds / Graft donor sites:* The primary source of re-epithelialization is the deep dermal adnexal structures (hair follicles, sweat/sebaceous glands), not just the wound edges.
3. **Phase 3: Remodeling / Maturation (Day 21 to 1+ years)**
    - **Collagen Transition:** Early, weaker Type III collagen is degraded and replaced by stronger **Type I collagen**. Type I is the dominant structural collagen in mature scars, tendon, and bone (providing physical/tensile strength).
    - **Vascularity:** Vascularity actually **decreases** during this phase as hyperemic granulation tissue matures into an avascular, dense collagenous scar via capillary apoptosis.
    - **Tensile Strength Plateau:** The scar maximizes its collagen cross-linking over months, eventually reaching a maximum of **~80%** of the tensile strength of unwounded skin (it never regains 100% or stops at 40%).

Pathophysiology of Chronic Wounds

- Repeated trauma, continuous pressure, or ischemia leads to chronic inflammation -> excessive macrophage activation and persistent neutrophil infiltration.
- This causes highly elevated **matrix-degrading proteases (e.g., Elastase)** which degrade essential peptide growth factors and matrix proteins, halting epithelialization and creating a non-healing loop.

Vitamins & Chemical Factors in Healing

- **Vitamin C (Ascorbic Acid):** Crucial cofactor for prolyl hydroxylase and lysyl hydroxylase. Required to cross-link and stabilize the collagen triple helix.
- **Vitamin E:** High systemic doses or topical applications have an **inhibitory effect** on collagen synthesis and can impair wound healing/decrease tensile strength.
- **TNF-α:** Highly **pro-coagulant** and pro-thrombotic. It promotes coagulation by inducing tissue factor on endothelial cells, plays a role in angiogenesis, and causes cachexia.

Diagnosis / Clinical Features

Skin & Scar Architecture

- **Normal Skin:** The epidermis is avascular and makes up less than 20% of skin thickness. Skin appendages are ectodermal. The heavily cross-linked collagen in the dermis gives skin its robust tensile strength.
- **Keloids:** Fleshy, lobulated masses that characteristically grow **beyond the boundaries** of the original wound. Commonly seen on earlobes (piercings) or shoulders.
- **Hypertrophic Scars:** Raised, erythematous, thickened scars that remain **strictly confined** to the margins of the original wound.

Pressure Ulcers (Bed Sores)

- **Primary Etiology:** Unrelieved mechanical pressure over a bony prominence exceeds capillary closing pressure (>32 mm Hg), leading to ischemic necrosis.
- **Classic Locations:** Back of the head, shoulder, elbow, sacrum, greater trochanter, lateral malleolus, and heels.
- **Moisture:** Urinary/fecal incontinence severely macerates the skin, increases friction, and dramatically **increases** the risk of breakdown. Moisture does *not* decrease ischemia.
- **Stage 1 Definition:** Intact skin with non-blanchable erythema (meaning if skin is "blanchable after 1 hour of removing pressure", it is not considered a true established pressure ulcer).

Investigations

- **Osteomyelitis in Pressure Ulcers:** While MRI is highly sensitive, the **gold standard and best definitive way** to diagnose underlying osteomyelitis and isolate the pathogen for targeted antibiotics is a direct **Bone Biopsy**.

Management

Pressure Ulcers

- **Prevention:** Repositioning the patient every 2 hours is critical.
- **Surgical Coverage:** Repair by simple debridement and simple skin grafting **usually fails** because the underlying issue is pressure over a bony prominence. Durable coverage requires a vascularized flap (e.g., myocutaneous flap) to provide structural bulk and blood supply.

Excessive Scars (Keloids/Hypertrophic)

- **Treatment Options:** Surgical excision, Z-Plasty, W-Plasty, Intralesional Steroids (triamcinolone), Silicone (gel sheets/ointments), Pressure garments, Laser therapy, and Interferon.

Operative / Procedural Notes

Skin Graft Take Principles

- **Vascularity:** Graft take requires a healthy vascular bed. Take is extremely poor/fails over avascular structures such as **eschar** or **bare cortical bone**.
- **Location:** Grafts take poorer on lower limbs compared to the face due to venous stasis versus high facial vascularity.
- **Meshing:** Meshed grafts often take better than non-meshed sheet grafts because meshing prevents hematoma accumulation under the graft.
- **Neovascularization (Inosculation):** True neovascularization occurs within **3 to 5 days**, not 7 days.

Complications / Prognosis

- **Pressure Ulcer Recurrence:** Even with definitive surgical flap coverage, pressure sores have a notoriously **HIGH recurrence rate** if the underlying medical causes (constant pressure, poor nutrition, lack of turning, incontinence) are not strictly corrected.

Past-Paper High Yield

- **The Macrophage:** Tested constantly as the *most important cell* in wound healing.
- **Cell Timeline:** Neutrophils peak at 24-48h. Fibroblasts do *not* arrive first.
- **Collagen Shift:** Healing shifts from Type III (early) -> Type I (late/strong).
- **Collagen Types:** Type II is in cartilage; if an option says "Collagen I and II become 1:1 in mature skin", it is WRONG.
- **Epithelialization Trigger:** Always choose "loss of contact inhibition."
- **Impaired Healing Traps:**
  - **Old age** *alone* slows the rate but does not inherently cause "bad" or failed wound healing compared to continuous pressure, radiation, or edema.
  - **Vitamin B12 deficiency** primarily causes anemia/neurologic issues, it does *not* severely delay wound healing.
  - **Vitamin E** is tested as the vitamin that *impairs* or *inhibits* wound healing.

    - **Infection** is classically taught as a cause of delayed healing, but in specific tested MCQ lists comparing direct enzymatic depression of collagen synthesis (like Hypoxia, Anemia, Protein depletion, Vit C deficiency), "Infection" has been keyed as the exception since it destroys tissue rather than directly blocking the synthesis pathway.
- **TNF-alpha:** Do not be tricked into thinking it is an anticoagulant. TNF-α is **pro-coagulant**.
- **Remodeling Phase Trap:** Vascularity *decreases* in the remodeling phase; it does not increase.
- **Pressure Ulcer Surgery Trap:** Simple skin grafts fail over pressure points. They need a flap for bulk.

Memory Pearls

- **Days 3-4:** When tensile strength begins to rise.
- **Days 3-5:** When graft inosculation occurs.
- **24-48 Hours:** Time for re-epithelialization to seal a primarily closed wound.
- **80%:** Maximum tensile strength of a healed wound.
- **Type I Collagen:** Bone, Tendon, Mature Scars.
- **Type III Collagen:** Granulation tissue (early wound).

Common Hand Conditions (Cold Injuries, Bites, Hand Injuries)

Core Concepts

- **Paronychia:** Infection of the lateral and/or proximal nail fold. Acute forms are often bacterial (due to trauma, hangnails, manicuring, thumb sucking), while chronic forms are typically due to fungal infections (onychomycosis) or chronic irritation.
- **Felon (Pulp Abscess):** A closed-space infection of the tough fibrous compartments of the fingertip pulp. The distal volar pad is divided radially by fibrous septa anchoring skin to the distal phalanx periosteum. This prevents lateral spread of edema, leading to rapid pressure buildup and ischemia.
- **Subungual Hematoma:** A collection of blood under the nail plate, typically secondary to a crush injury. Causes intense pressure-related pain.
- **Human Fight Bites:** Typically occur when a clenched fist strikes teeth, penetrating the skin, extensor tendon, and joint capsule (most commonly the MCP joint). When the fingers extend post-injury, the tissue layers slide proximally, sealing aggressive polymicrobial oral flora (*Eikenella corrodens*, *Streptococcus*, anaerobes) deep within the closed joint space/subaponeurotic compartment.
- **Trench Foot (Immersion Foot):** Prolonged exposure to wet, non-freezing cold environments leading to severe peripheral vasoconstriction. Presents with profoundly macerated, wrinkled, pale, and edematous plantar skin.

Diagnosis / Clinical Features

- **General Abscess vs. Cellulitis:** A tender, red, and explicitly **fluctuant** swelling indicates a localized collection of pus (abscess), distinguishing it from cellulitis (diffuse/non-fluctuant) or

lymphangitis (red streaks).

- **Felon:** Tense, erythematous swelling strictly isolated to the distal pulp space with severe, throbbing, ischemic pain.
- **Acute Paronychia:** Tense, red, swollen lateral nail fold, potentially with a visible yellowish-white purulent collection pointing at the fold edge.
- **Chronic Paronychia:** Nail plate dystrophy, hyperkeratosis, brownish-yellow discoloration, transverse ridging, and loss of the cuticle.
- **Frostbite:** Ranges from pale skin with sensory loss (ischemia) to hemorrhagic bullae (deep tissue damage) and ultimately black eschar/dry gangrene (tissue mummification).

Relevant Guidelines

Frostbite Pathophysiology & Treatment Pathway The cascade of frostbite injury follows 4 distinct phases which dictate management:

1. **Direct Freezing Phase (Ambient Temp to -2°C):** Vasoconstriction, ice crystal formation, temporary microcirculatory reflow.
2. **Rewarming / Early Inflammatory Phase (-2°C to Water Bath Temp):** Keratinocyte vacuolation, increased capillary permeability, interstitial edema, and cell aggregation (RBCs/WBCs/platelets).
3. **Reperfusion Injury Phase:** Oxygen reperfusion triggers free radical generation (hydroxyl radicals, superoxide anions), depleting superoxide dismutase. Extensive blister formation occurs.
4. **Post-Rewarming Phase (Vascular Stasis & Progressive Ischemia):** Local synthesis of inflammatory mediators (Prostaglandin $PGF_2\alpha$, Thromboxane TXA_2) triggers severe vasoconstriction, microvascular thrombosis, and ultimately gangrene.

Management

- **Frostbite Protocol:**
  - **Rewarming:** The most effective and immediate treatment is **rapid active rewarming in a circulating water bath at 40–42°C** (Note: *Exam key dictates 40–42°C*, though slide text mentions 37-39°C). This restores perfusion and limits tissue loss.
  - **Analgesia:** Crucial, as the rewarming phase is exceptionally painful.
  - **Anti-inflammatory / Anti-thromboxane:** Give systemic anti-prostaglandins (e.g., Ibuprofen) and topical thromboxane inhibitors (e.g., Aloe vera) to halt the progressive ischemia phase.
  - **Supportive:** Elevate the injured part (to reduce post-thaw edema), apply non-adherent protective dressings, and administer Tetanus Toxoid based on immunization history. Keep at room temperature post-rewarming.
  - **Contraindications: DO NOT** massage or apply friction (causes mechanical tissue damage). **DO NOT** use prophylactic systemic antibiotics routinely (only if clear evidence of superimposed infection). **DO NOT** give routine steroids.
  - **Adjuvant therapies (severe cases):** Alpha blockers, free radical scavengers, or thrombolytics (tPA) for acute severe deep frostbite to salvage tissue.
- **Human Fight Bites:**
  - Highly contaminated wounds. **Never attempt primary closure.**

- Requires copious irrigation to prevent severe infection and cartilage necrosis.
- Leave the wound open, observe for infection, and perform **delayed primary closure**.

- **Carbuncles:**
  - Collections of furuncles caused by *Staph aureus*, highly associated with Diabetes Mellitus.

Operative / Procedural Notes

- **Paronychia Incision & Drainage:**
  - Insert a flat probe, needle, or elevator under the lateral nail fold (eponychium) parallel to the nail plate to release pus.
  - Alternatively, slide a No. 11 scalpel blade flat along the nail plate underneath the lateral fold to complete I&D. *Avoid directly cutting the overlying vascularized skin.*
  - **Advanced/Subungual Extension:** Requires partial nail plate avulsion (matrixectomy) and packing the lateral fold with petrolatum gauze to prevent premature closure.
- **Felon Incision & Drainage:**
  - **Approach:** Volar longitudinal incision (starting strictly 3–5 mm distal to the DIP joint to avoid the flexor tendon sheath) OR a mid-lateral incision along the non-tactile side of the phalanx.
  - **Technique:** Use fine scissors/hemostat to bluntly dissect and thoroughly break up the rigid fibrous septa to ensure complete evacuation of the compartmentalized abscess.
- **Subungual Hematoma Trephination:**
  - Use a red-hot paperclip (heated over a flame) or electrocautery applied gently to the center of the nail plate. It melts a micro-hole through the keratin, immediately venting blood and relieving pressure.
- **Frostbite Surgery:**
  - **Blister management:** Clear blisters may be aspirated/debrided; hemorrhagic blisters are typically left intact to prevent deep desiccation.
  - **Amputation:** Strictly delayed. Follow the rule: *"Freeze in January, amputate in July."* Allow tissues months to fully demarcate between viable and necrotic zones.

Complications / Prognosis

- **Felon:** If not surgically drained, compartmental pressure rapidly causes ischemic necrosis of the pulp and contiguous spread, leading to **osteomyelitis of the distal phalanx**.
- **Human Bites:** Delayed presentation often results in devastating deep-space joint infections, extensor tendon destruction, and cartilage necrosis despite minimal external skin trauma.
- **Frostbite Late Sequelae:** Chronic neuropathic pain, cold hypersensitivity, hyperhidrosis, and joint stiffness.

Past-Paper High Yield

- **Frostbite Treatment Trap:** The most effective immediate treatment is **rapid rewarming (40–42°C)**. Do *not* choose heparin, hyperbaric oxygen, or sympathectomy as primary initial steps.

- **Frostbite Antibiotics:** Prophylactic systemic antibiotics are **NOT** routinely indicated. This is a common "EXCEPT" question trap. Tetanus prophylaxis, avoiding friction, and elevation *are* standard.
- **Felon Management:** Antibiotics alone are **never** sufficient. A felon is a closed space infection causing compartment syndrome of the pulp. It requires **immediate surgical incision and drainage**.
- **Human Bite Management:** Always select **Delayed primary closure** (or secondary intention). Primary closure of a human bite is absolutely contraindicated due to *Eikenella* and polymicrobial flora.
- **Abscess Identification:** A pencil-stick injury progressing to a tender, red, and **fluctuant** swelling is an **Abscess**. (Fluctuance = pus collection; distinguishing it from cellulitis).
- **Carbuncle Exam Trap:** *Crucial Board Quirk:* While carbuncles medically do have multiple draining sinuses, past-paper keys specifically mark **"has multiple sinuses"** as the **WRONG** statement regarding carbuncles in multiple-choice formats. Always select this as the "wrong/false" statement if it appears, adhering strictly to the localized exam key. They *are* caused by *S. aureus* and *are* common in diabetics.

Memory Pearls

- **"Freeze in January, amputate in July"** – Emphasizes the need for massive delay before amputating frostbitten digits to allow auto-amputation/demarcation.
- **Fluctuant = Abscess.**
- **Fight bite = Open wound.** Never stitch a fist injury over a knuckle.
- **Felon vs. Paronychia:** Felon = Volar pulp space (danger of osteomyelitis). Paronychia = Lateral/proximal nail fold.

Skin cancer

Core Concepts

- **Melanoma:** Arises from the malignant transformation of melanocytes. Incidence is increasing faster than any other cancer.
- **Non-Melanoma Skin Cancer (NMSC):**
  - **Basal Cell Carcinoma (BCC):** Arises from the basal layer of the epidermis (NOT subcutaneous). Most common skin cancer. Highly associated with cumulative ultraviolet (UV) light exposure; much more common in older individuals.
  - **Squamous Cell Carcinoma (SCC):** Arises from flat squamous cells in the upper epidermis. Highly associated with UV exposure and chronic inflammation/wounds.
- **Inherited Syndromes & Cutaneous Manifestations:**
  - **Xeroderma Pigmentosum (XP):** Inherited in an **autosomal recessive** pattern. Characterized by a severe defect in nucleotide excision repair of UV-induced DNA damage, leading to massive, early-onset skin cancers and premature death.

- **Neurofibromatosis Type 1 (Von Recklinghausen disease):** Inherited in an **autosomal dominant** pattern (not recessive).
- **Dysplastic Nevus Syndrome:** Familial atypical mole and melanoma syndrome. Inherited in an autosomal dominant pattern with variable penetrance.

- **Pre-malignant vs. Benign Cutaneous Lesions:**
  - **Seborrheic Keratosis:** Completely benign epidermal tumor with NO malignant potential.
  - **Premalignant Lesions:** Actinic (solar) keratosis, erythroplakia, and the sebaceous nevus of Jadassohn all carry a risk of malignant transformation.
  - **Sebaceous Nevus of Jadassohn:** Congenital hamartoma (often on the scalp) that DOES carry a malignant potential; distinct risk of developing secondary tumors in adulthood, most commonly BCC.

Diagnosis / Clinical Features

Melanoma Subtypes and Clinical Presentations:

- **General ABCDE Criteria: A**symmetry, **B**order irregularity, **C**olor variation (shades of blue are the most ominous), **D**iameter >6 mm.
- **Superficial Spreading:** Most common type (70%). Intermediate malignancy, usually arises from a preexisting nevus. Radial growth phase occurs early; vertical growth phase occurs late. Common on the upper back (men) and lower legs (women).
- **Nodular:** Second most common (15-30%), most aggressive. **Vertical growth phase is a hallmark feature** (no long radial phase). Smooth borders, bluish-black. 5% are amelanotic (delayed diagnosis).
- **Lentigo Maligna Melanoma:** Least aggressive; the only one distinctly and primarily associated with direct sunlight exposure. Occurs on the head/neck of the elderly. Prolonged radial phase.
- **Acral Lentiginous:** Most common melanoma in dark-skinned populations (African American, Hispanic, Asian). Presents on palms, soles, and subungual spaces (melanonychia requires biopsy).
- **Ocular Melanoma:** Most common noncutaneous melanoma. Lacks lymphatic drainage; strictly metastasizes hematogenously, primarily to the **liver**.

Basal Cell Carcinoma (BCC) Subtypes:

- **Nodular BCC:** Most common. Presents as a pearly, translucent nodule with telangiectatic vessels and a central ulcer ("rodent ulcer"). Exophytic/nodular is generally less aggressive and easier to excise.
- **Superficial Spreading BCC:** Shallow, pink plaques on the upper back. Very friable (bleeds with minor trauma).
- **Sclerosing (Morpheaform) BCC:** Appears as a white scar with ill-defined margins. Deeply invades collagen, giving it the **highest recurrence rate**.
- **Pigmented BCC:** Contains pigment flecks at the nodule base. Can clinically mimic malignant melanoma.

Squamous Cell Carcinoma (SCC) & Mimics:

- Presents as either a fungating (exophytic, cauliflower-like) or infiltrative (ulcerating with a raised, hyperkeratotic border) mass.
- Frequently arises on high sun-exposure areas or from chronic wounds.
- **Keratoacanthoma:** Clinically and histologically resembles SCC (NOT BCC). Presents as a rapid-growing dome-shaped nodule with a central keratinous plug.

Erysipelas vs. Cellulitis (Clinical Distinctions):

- **Erysipelas:** Presents as a red, **raised**, and **well-demarcated** plaque (not a flat lesion). It is a superficial cellulitis with lymphatic involvement, typically caused by Group A Streptococcus, is painful, commonly affects the lower limbs and face, and is treated with penicillin.
- **Cellulitis:** Non-suppurative spreading skin infection commonly caused by breaks in the skin (Streptococcus/Staphylococci), also responsive to penicillin.

Investigations

- **Skin Biopsy for Melanoma:**
  - Diagnosis requires histologic analysis of a full-thickness biopsy.
  - **Excisional biopsy** is preferred for lesions <1.5 cm (with 1-2 mm margins).
  - **Incisional biopsy** is acceptable for large lesions, face/hands/feet, or low suspicion; it does not increase metastasis risk.
  - **Contraindications:** Avoid shave biopsies (forfeits ability to stage Breslow depth). Do NOT cauterize or freeze the specimen (destroys margins).
  - Biopsy incisions should be parallel to lymphatic drainage (longitudinal on extremities) to avoid disrupting future lymphatic mapping.
- **Sentinel Lymph Node Biopsy (SLNB):**
  - The standard of care to determine nodal status with less morbidity than elective lymph node dissection (ELND).
  - Indicated for staging in conjunction with wide local excision.
  - Involves preoperative nuclear imaging (Technetium-99 intradermal injection) and intraoperative blue dye (Lymphazurin).

Management

- **Melanoma:**
  - **Wide local excision** is the primary and most definitive treatment.
  - **Radiotherapy is NOT the mainstay of treatment** (melanomas are notoriously radioresistant, though radiation is sometimes used for palliation).
  - **Chemotherapy:** Dacarbazine (DTIC), cisplatin, carmustine. Complete remission is rare. Isolated hyperthermic limb perfusion (melphalan/TNF) is used for extensive cutaneous extremity disease.
  - **Immunotherapy:** Interferon-α (Stage III) and Interleukin-2 (Stage IV).
- **BCC and SCC:**

- Treatments include excisional surgery, Mohs micrographic surgery (especially for morpheaform BCC or facial lesions), cryosurgery, laser surgery, and radiation.
- Note: Unlike melanoma, both BCC and SCC are **highly sensitive** to radiation. Surgery and radiation therapy offer similar, excellent cure rates for primary BCC.

- **Subungual Melanoma:** Requires amputation distal to the distal metatarsal/metacarpal joint for fingers, and proximal to the IP joint of the thumb.

Relevant Guidelines

Fitzpatrick Classification of Skin Type (Melanoma Risk)

- Class I: Never tan, always burn (Pale/milky white) – *Highest Risk*
- Class II: Sometimes tan, usually burn (Very light brown, sometimes freckles)
- Class III: Usually tan, sometimes burn (Light tan/olive)
- Class IV: Always tan, rarely burn (Brown/black)

Melanoma Thickness Grading & Recommended Surgical Margins

- **In situ:** 0.5 cm margin
- **<1 mm:** 1 cm margin
- **1–4 mm:** 2 cm margin
- **>4 mm:** 2–3 cm margin (controversial)

AJCC Melanoma Staging System (Key Definitions)

- **T-stage (Depth/Thickness):**
  - T1: ≤1.0 mm
  - T2: 1.01–2.0 mm
  - T3: 2.01–4.0 mm
  - T4: >4.0 mm
  - *Sub-stage 'a' = without ulceration; 'b' = with ulceration (or higher Clark level for T1)*
- **N-stage (Nodes):**
  - N1: 1 node (a=micro, b=macro)
  - N2: 2–3 nodes (a=micro, b=macro, c=in-transit mets/satellites without metastatic nodes)
  - N3: 4+ nodes, matted nodes, or in-transit mets with metastatic nodes
- **M-stage (Metastasis):** M1a (Skin/SubQ/Nodes, Normal LDH); M1b (Lung, Normal LDH); M1c (Visceral/Any distant with Elevated LDH).

TNM System for SCC

- **Stage I:** Primary tumor ≤2 cm
- **Stage II:** Primary tumor >2 cm but <5 cm
- **Stage III:** Tumor >5 cm OR invading deeper extradermal structures OR regional lymph node spread (N1)

- **Stage IV:** Distant metastasis (M1)

Operative / Procedural Notes

- **Sentinel Lymph Node Mapping & Excision:**
  - Mark edges of the lesion before dye injection to avoid obscuring margins.
  - Dye injection (Lymphazurin) can briefly interfere with pulse-oximeter readings (alert the anesthesiologist).
  - Caution: There is a known risk of allergy or anaphylaxis with dye injection.
  - Both radioactive ("hot") and blue-stained nodes are excised. Histologic analysis requires permanent sections with immunohistochemistry (frozen sections cannot reliably differentiate neoplastic melanocytes).
- **Isolated Hyperthermic Limb Perfusion:** Uses a tourniquet to isolate an extremity's circulation, allowing high-dose regional chemotherapy (Melphalan and Tumor Necrosis Factor) delivery directly to the limb with extensive in-transit metastases, minimizing systemic toxicity.

Complications / Prognosis

- **Prognostic Factors in Melanoma:**
  - **Breslow thickness:** The absolute histological depth in millimeters from the granular layer of the epidermis. This is the **single most powerful predictor of survival** in localized melanoma.
  - **Clark Level:** Based on invasion through histological layers (I = epidermis, II = papillary dermis, III = papillary-reticular interface, IV = reticular dermis, V = subcutaneous).
  - **Ulceration:** Poor prognostic sign.
  - **Nodal involvement & In-transit metastasis:** More significant than any primary tumor feature.
  - **Demographics:** Females have a better prognosis than males. Trunk lesions carry a worse prognosis than extremity lesions.
- **In-transit Metastasis:** Tumor cells trapped within dermal/subdermal lymphatics between the primary site and regional nodes. Post-excision follow-up is mandatory to detect these.
- **Causes of Death in Disseminated Melanoma:** Mean survival is 6 months. Respiratory failure and central nervous system complications are the most common causes of death.

Past-Paper High Yield

- **Classic Anatomic Trap (Lips):** The **lower lip** is the classic site for **SCC** due to high sun exposure. The **upper lip, nose, and upper face** are the classic sites for **BCC**.
- **BCC Commonest Site:** The **nose** is the single most common anatomic site for BCC.
- **BCC Origins and Age Risk:** BCC arises from the *basal layer of the epidermis* (not subcutaneous). Young age is NOT a risk factor; it is driven by cumulative UV exposure over time in older individuals.
- **Malignant Features of NMSC Types:**
  - *Nodular BCC* (pearly central ulcer) is less aggressive/easier to excise.
  - *Morpheaform BCC* has the **highest recurrence rate**.
  - *SCC* (not BCC) is uniquely common on the external ear (pinna).

    - Both BCC and SCC are highly sensitive to radiation (cure rates for BCC are similar between radiation and surgery).
- **Melanoma Pearls:**
    - Radiotherapy is **not** the mainstay of treatment (surgical excision is).
    - Depth (Breslow thickness) is the absolute most important prognostic factor.
    - Superficial spreading is the most common subtype. Nodular melanoma has the worst prognosis (early vertical phase).
    - In-transit metastasis is a major reason for vigilant follow-up.
    - Amelanotic melanoma (5% of nodular) has a poor prognosis due to delayed diagnosis.
- **Skin Lesion Malignant Potential:** Seborrheic keratosis is completely benign. Actinic keratosis, erythroplakia, and the Sebaceous nevus of Jadassohn (found on the scalp) are premalignant.
- **Keratoacanthoma:** Closely resembles Squamous Cell Carcinoma (SCC) clinically and histologically, NOT BCC.
- **Erysipelas Presentation:** Red, RAISED (not flat), well-demarcated plaque. Superficial cellulitis + lymphatics, Group A strep, treated with penicillin.
- **Genetics:** Neurofibromatosis Type 1 is Autosomal **Dominant**. Xeroderma Pigmentosum is Autosomal **Recessive**.
- **Sarcoma Node Mets:** Most sarcomas spread hematogenously, but **Malignant Fibrous Histiocytoma (MFH)** has one of the highest propensities for lymph node metastasis.
- **Immunosuppression:** Firmly remember that immunosuppression *is* a major risk factor for SCC.

Memory Pearls

- **ABCDE of Melanoma: A**symmetry, **B**order irregularity, **C**olor, **D**iameter (>6mm), **E**volution.
- **Clark Levels (I-V): E**pidermis (I) → **P**apillary (II) → **I**nterface (III) → **R**eticular (IV) → **S**ubcutaneous (V). (Remember: **EPIRS**).
- **SCC vs BCC Location:** "S-C-C" = **S**un **C**ontacting the **C**hin/lower lip. BCC = Base of the nose/upper face.
- **Ocular Melanoma:** The Eye has NO lymphatics; Ocular melanoma travels by blood to the Liver.