Hernias:

The mortality associated with bowel strangulation is over 10%.

indirect hernias account for 60-75% of all hernia -most common type in both genders-/ femoral hernias are more likely to affect females.

The femoral canal is the medial compartment of the femoral sheath and lies medial to the femoral vein, serving as a potential space that allows for venous expansion and contains lymphatic vessels, including the lymph node of Cloquet. Its anterior boundary is formed by the inguinal ligament, while the posterior boundary is the pectineal ligament (also known as Cooper's ligament), which lies over the pectineus muscle. Medially, the canal is bordered by the lacunar ligament, and laterally by the femoral vein. A common misconception is that the lacunar ligament forms the lateral border, but this is incorrect—it actually forms the medial edge.

A saphena varix is a venous dilation at the saphenofemoral junction, presenting as a painless, compressible groin mass with a cough impulse that reduces when supine, often seen with chronic venous insufficiency. An arteriovenous malformation causes a pulsatile groin mass with a palpable thrill or audible bruit due to abnormal high-flow vascular connections, irreducible and unrelated to positioning. A false femoral artery aneurysm, typically post-traumatic or iatrogenic, manifests as a tender, pulsatile mass without reducibility, often with a hematoma or bruit. Femoral hernias, more common in women, appear below the inguinal ligament as firm, often irreducible masses with high strangulation risk, lacking the cough impulse seen in venous lesions.

The least likely differential diagnosis of a groin lump in a supine patient is?

- A. Irreduicable inguinal hernia.
- B. Psoas abscess.
- C. Hodgkin lymphoma.
- D. Saphenavarix.
- E. Femoral artery aneurysm

answer is D: even though Hodgkin is very rare to be isolated in the groin <5%, if it was there it will be palpable while supine unlike saphena

Inguinal hernia repair is typically a clean, outpatient procedure, even under local anesthesia, contradicting the false claim that it requires hospitalization. While irreducibility significantly elevates strangulation risk (demanding urgent intervention), elective repairs are classified as clean surgeries unless bowel compromise occurs. Postoperative complications include chronic pain (affecting up to 20% of patients, often mesh or nerve-related) and testicular atrophy (1-5% from spermatic cord injury).

inguinal hernia is the seconds most common cause of obstruction after adhesions.

Direct inguinal hernias are exceptionally rare in women due to anatomic protections: a narrow inguinal canal and reinforced posterior wall (Hesselbach's triangle), making weakness here unlikely. Instead, women predominantly develop indirect inguinal hernias (from congenital defects) or femoral hernias (pelvic anatomy favors femoral canal laxity). Incisional and umbilical hernias occur equally in both sexes.

HY Point 1: Female hernias:

Most common = indirect inguinal.

Most dangerous = femoral (high strangulation risk).

HY Point 2: Direct inguinal hernia = Male-dominated (10:1 ratio).

this is according to DeepSeek

in the past papers there was a Q asking what is the least likely hernia in a female it was answered femoral.

IV fluids and blood transfusion and electrolytes:

in hypovolemic hypernatremia use Normal saline / ringer lactate even at the cost of initial rise in Na.

Hypernatremia is a life-threatening condition that can occur in hypovolemic, euvolemic, or hypervolemic states, and is often seen in settings like major burns or diabetes insipidus. The primary issue is a water deficit, not sodium overload, and its untreated form has a high mortality rate, particularly in vulnerable populations. Management involves hypotonic fluid replacement, which can be administered orally, rectally (in rare or palliative cases), or intravenously. The key principle in treatment is slow correction of serum sodium, aiming for a reduction of about 0.5 mEq/L per hour, to avoid cerebral complications. Thus, the statement that fluid therapy should decrease osmolality by 10 mOsm/hour is incorrect, as it suggests a dangerously fast rate.

Blood transfusions, particularly packed red cells, can lead to a variety of complications. They can transmit infections like cytomegalovirus, especially through white cells in non-leukoreduced products. Allergic reactions are common, ranging from mild rash to bronchospasm in severe anaphylaxis. In critically ill or septic patients, transfusions may even contribute to microcirculation thrombosis due to inflammatory and procoagulant effects. However, PRBC transfusions do not increase platelet count

a massive transfusion is either:

10 unites in 24h

whole blood volume in 24h

50% of blood volume in 3h

more than 3 units in 1 hour with active bleeding

when to give PRBCs?

if Hb less than 7

if Hb less than 8 with symptoms -tachycardia/SOB

if there is CAD/ MI/ TIA/ angina/ CHF/ syncope then Hb less than 10.

patients on hemodialysis can withstand Hb as low as 7 if asymptomatic,

in the cases of bleeding we transfuse based on symptoms not Hb change.

The most fatal transfusion reaction is ABO incompatibility, causing acute hemolysis, DIC, and shock. TRALI is serious but less lethal. Allergic/febrile reactions are uncomfortable but rarely dangerous. Circulatory overload is manageable with volume control.

allergic transfusion response is more common in IgA deficient individuals

TRALI is a non-cardiogenic pulmonary edema caused by donor antibodies (often in FFP), presenting within 6 hours with hypoxemia and bilateral infiltrates. Unlike TACO, PAWP is normal. Supportive care is mainstay; steroids are ineffective. While serious, it's not the most common transfusion reaction.

Key for Shelf High-Yield Points

Definition: TRALI = Acute lung injury ($PaO_2/FiO_2 \leq 300$) within 6 hours of transfusion, no cardiac overload (PAWP ≤18 mmHg).

Mechanism: Donor anti-HLA/anti-neutrophil antibodies → neutrophil activation → capillary leak.

High-risk product: FFP > platelets > RBCs (due to antibody content).

Diagnosis: Clinical + exclusion of TACO (BNP/NT-proBNP may help differentiate).

Treatment: Supportive only (oxygen, mechanical ventilation if needed).

Mortality: ~10% (lower than ABO incompatibility but higher than other reactions).

Hypercalcemia most frequently stems from malignancy (e.g., breast cancer metastases). Severe hypercalcemia (>12 mg/dL) induces extracellular volume depletion through nephrogenic DI and GI losses, necessitating aggressive NS hydration to restore volume and enhance renal calcium excretion.

it may present with similar sign and symptoms of hyperglycemia including: polyuria polydypsia and dehydration

Normal saline contains 154 mEq/L of both sodium and chloride, making it strongly hyperchloremic. Ringer's lactate is more physiologically balanced with electrolyte levels closer to plasma. Hypotonic fluids like half-normal saline can dangerously lower serum osmolality, potentially causing cerebral edema as water shifts into cells. Colloid solutions, while effective volume expanders, carry risks of fluid overload in vulnerable patients. D5W provides nutrition- its minimal 170 kcal/L can't meet basic metabolic needs and lacks essential proteins, fats, and electrolytes required to prevent catabolism during fasting. so it is not enough alone.

Hypermagnesemia causes neuromuscular depression, leading to reduced or absent deep tendon reflexes—the opposite of exaggeration. While ECG changes (peaked T waves, bradycardia) mirror hyperkalemia due to shared membrane effects, and elevated magnesium often accompanies hyperkalemia.-espicailly in renal failure-

the ECF is rish in sodium and chloride while ICF is rish in pattsium and phosphate

ECF has less protein and is more alkaline

while ICF is acidic

ICF has larger volume.

ECF is the volume of distribution of cystalloids.

while colloids stay intravscular.

129- A 24-year-old healthy male, undergoing hernia repair at 12mid-day, he started fasting at midnight (12 hours fasting), what is the maintenance fluid therapy?

- a. 1250 ml NS+ 500 ml D5W
- b. 1250 ML D5W+ 500 ml NS
- c. 1000 ml D5W+ 2500ml RL
- d. 1000 ml RL+ 2500 ml D5W

the question should give the weight of the pateint

DeepSeek calculated according to Na equivalence needs and osmolarity

the naswer was A.

in the file it was answered B.

Nutrition:

Parenteral nutrition (PN) is less likely to cause diarrhea than enteral feeding but is more expensive, increases infection risk, impairs gut immunity, and causes metabolic bone disease. Always prefer enteral nutrition unless the gut is nonfunctional.

Key for Shelf:

Enteral nutrition is superior (cheaper, preserves gut immunity, fewer infections)

PN indications: Short bowel syndrome, complete obstruction

PN complications: Line infections, liver dysfunction, metabolic bone disease

Only PN advantage: Lower diarrhea risk (due to bypassing gut)

Enteral feeding via a nasogastric (NG) tube is a widely used method for patients who cannot swallow but have a functional GI tract. A common side effect is abdominal cramping and diarrhea, especially with improper feeding rates or formulas. NG feeding is not thrombogenic, unlike parenteral routes. It is not suitable for short gut syndrome, where the reduced absorptive surface may necessitate parenteral nutrition. Contrary to misconception, NG feeding has a lower risk of septic complications compared to parenteral nutrition and is a standard approach in post-stroke patients with swallowing issues, not a contraindication.

While esophageal stricture complicates NGT insertion and requires careful techniques (e.g., dilation or fluoroscopic guidance), it is not an absolute contraindication—unlike confirmed or suspected esophageal rupture, where NGT insertion risks lethal mediastinitis, or esophageal foreign bodies, which may perforate if displaced.

other contraindications include:

basilar skull fracture or facial fracture -facial is relative contraindication according to DeepSeek-

and bleeding diathesis -also relative according to DeepSeek-

Gastric fluid contains low potassium (5–10 mmol/L), contrary to the false claim of high potassium concentration. Pancreatic secretions are strongly alkaline (pH ~8) to neutralize gastric acid, while bile is mildly alkaline (pH 7.2). Gastric fluid chloride matches plasma levels (~110 mmol/L) due to HCl dissociation. Most GI losses can be replaced with normal saline plus potassium, except gastric losses which also require hydrogen ion replacement.

Key for Shelf:

Gastric vs. intestinal secretions:

Gastric: High H^+/Cl^-, low K^+

Small bowel: High K^+ (20–30 mmol/L) and HCO_3^-

Pancreatic/biliary: Alkaline (neutralizers)

Replacement fluids:

Gastric: Half-normal saline + KCl + acid (e.g., proton pump inhibitors)

Intestinal: Normal saline + KCl

The flow phase is the prolonged, catabolic stage of the hypermetabolic response that can last weeks or months in critically ill patients. It results in muscle breakdown, negative nitrogen balance, and worsening nutritional status. The ebb phase is brief and marked by metabolic suppression, while the healing phase represents recovery and anabolic rebuilding.

carbs+protein have 4kCal per gram while fat has 9 while alcohol has 7.

but this is enteral

parenteral : carbs have 3.4 Kcal per gram

peripheral Parenteral Nutrition (PPN)

Glucose Concentration: Limited to ≤10% dextrose to avoid phlebitis and vein sclerosis. Higher concentrations (>10%) are hypertonic and damage peripheral veins.

Caloric Delivery: Inadequate for long-term or high-calorie needs due to glucose and lipid restrictions. Typically used for short-term (<2 weeks) or supplemental support.

Amino Acids: Can be administered peripherally if the solution's osmolarity is <900 mOsm/L, though central delivery is preferred for full nutritional support.

Central Parenteral Nutrition (CPN or TPN)

Access: Requires a central venous catheter (e.g., subclavian or internal jugular vein) terminating in the superior vena cava to tolerate hypertonic solutions (e.g., 20–50% dextrose).

Caloric Delivery: Provides complete nutrition, including high glucose concentrations, lipids, and amino acids, making it suitable for long-term use.

Complications:

Thrombosis: Common with long-term central catheters; subclavian access has the lowest risk.

Infection: Catheter-related bloodstream infections (e.g., from S. aureus, Candida) require prompt line removal.

Metabolic: Hyperglycemia, refeeding syndrome, and liver dysfunction.

HY Point 1: Peripheral PN → Max 10% glucose; central PN → 25–50% glucose.

HY Point 2: Subclavian > internal jugular > external jugular for central access.

HY Point 3: Thrombosis risk is high with long-term central lines (anticoagulation may be needed).

Wound Healing and Infections:

Surgical wounds classify by bacterial load:

Class I (Clean): Sterile, no hollow viscus entry (e.g., thyroidectomy).

Class II (Clean/Contaminated): Controlled entry into non-infected hollow organs (elective upper GI surgery, cholecystectomy). Needs prophylactic antibiotics.

Class III (Contaminated): Spillage from non-purulent inflammation (bowel obstruction, open fractures). Needs prophylactic antibiotics.

Class IV (Dirty/Infected): Established infection (perforated diverticulitis, penetrating trauma). Requires therapeutic antibiotics + drainage.

Key Distinctions for Shelf:

Class II vs. III: Controlled vs. uncontrolled spillage.

Antibiotics: Prophylactic (Class I/II/III) vs. therapeutic (IV).
some sources say no need for antibiotic is class1

Prophylactic just given before surgery

Therapeutic given before and after

Surgical Site Infections (SSI) – Risk Factors Breakdown

Patient-Specific Risk Factors

1-Age

2-Diabetes

3-Nicotine Use (Smoking)

Vasoconstriction → Reduced tissue oxygenation → Delayed wound healing.

Best practice: Smoking cessation ≥4 weeks pre-op reduces risk.

4-Steroid Use

5-Malnutrition

5-Obesity (≥20% over ideal body weight)

6-Prolonged Preoperative Hospital Stay

7-Staphylococcus aureus Nasal Colonization

8-Perioperative Blood Transfusion

9-Remote Infections at Time of Surgery

1-Active infections (e.g., UTI, pneumonia) increase bacteremia risk → Higher SSI.

11-Immunosuppression (e.g., HIV, chemotherapy)

Nutritional risk is best identified by:

Weight loss >15% (most objective).

Albumin/transferrin (lab markers of protein status).

Physical exam (direct evidence of muscle/fat loss).

we need vitC and zinc for proper wound closure.

Wound healing relies on collagen production, which depends critically on vitamin C as a cofactor for collagen-forming enzymes. While other factors like calories, protein, and vitamin D contribute, vitamin C deficiency alone can halt proper wound repair, as seen in scurvy. Without adequate vitamin C, even well-nourished patients fail to form stable collagen, leading to weak scar tissue and wound breakdown. This makes vitamin C the most indispensable nutrient for healing, surpassing general dietary balance or energy intake in importance.

HY Point 1: Vitamin C enables collagen cross-linking → #1 for wound healing.

HY Point 2: Protein deficiency is the #2 factor (needed for collagen substrate).

HY Point 3: Zinc is also key (cofactor for DNA/RNA polymerases in cell proliferation).

there was a Q that asked most important factor for wound healing: and the answer was balanced diet -i think it is wrong- but the doctors might want it as the correct answer -it is up to U-

VAC Therapy (Mechanisms, Indications, & Contraindications)

1. Mechanism of Action (HOW IT WORKS)

Vacuum-assisted closure (VAC) therapy applies negative pressure to wounds, achieving:

Granulation tissue formation: Stimulates fibroblasts and collagen deposition.

Angiogenesis: Increases localized blood flow via microvascular stretch.

Infection control: Reduces bacterial colonization by removing exudate/debris.

Edema reduction: Sucks out interstitial fluid, decreasing swelling.

Wound contraction: Uniformly draws edges together (macrodeformation).

Moist environment: Maintains ideal healing conditions (prevents desiccation).

Epithelial migration: Accelerates skin regrowth over granulation tissue.

2. Key Indications (WHEN TO USE IT)

VAC is ideal for large, complex, or chronic wounds, including:

Traumatic wounds: Degloving injuries, avulsions.

Post-surgical: Dehiscence, open abdomen/thorax (temporary closure).

Chronic ulcers: Diabetic, pressure, or venous stasis ulcers.

Infected wounds: After debridement (e.g., necrotizing fasciitis).

Graft bolstering: Secures skin grafts/myocutaneous flaps.

Compartment syndrome: Post-fasciotomy (reduces edema recurrence).

3. Critical Contraindications (WHEN TO AVOID IT)

Exposed vessels/nerves/organs: Negative pressure can cause rupture or erosion (e.g., carotid artery, bowel).

Malignancy in wound: Risk of spreading tumor cells.

Untreated osteomyelitis: Requires antibiotics/debridement first.

Non-enteric fistulas: May worsen output (e.g., tracheoesophageal fistula).

surgical site infection typically appears 3-5 days post op.

Surgical site infections (SSIs) are classified into three main types based on anatomic involvement and timing:

Superficial Incisional SSI: Occurs within 30 days post-operation and involves only the skin and subcutaneous tissue at the incision site. This is the most superficial type, presenting with erythema, pain, or purulent drainage but not extending deeper.

Deep Incisional SSI: Also occurs within 30 days (or up to 1 year if an implant is present) and affects deeper soft tissues, including fascia and muscle layers. These infections often require surgical debridement and broader-spectrum antibiotics due to their severity.

Organ/Space SSI: The most severe type, involving internal organs or body cavities (e.g., peritoneum, joint space) that were manipulated during surgery. Like deep SSIs, the timeframe extends to 1 year if an implant is involved. These infections may present with abscess formation or systemic sepsis and demand aggressive intervention.

Key Points:

Implants (e.g., prostheses, mesh) extend the surveillance period to 1 year due to delayed infection risks.

Diagnosis requires infection to be clearly linked to the surgical procedure, excluding unrelated infections.

Treatment varies by depth: Superficial SSIs may need only wound care, while deep/organ-space infections often require reoperation.

another classification is

minor vs major:

minor is a superficial infection that involves only the skin and subcutaneous tissue it has no systemic symptoms.

major is more extensive infection that involves either:

Deep tissues (fascia or muscle),

Or the organ/space (e.g., intra-abdominal abscess, infected prosthetic joint).

and it shows systemic signs and symptoms.

Sepsis and Shock:

Systemic Inflammatory Response Syndrome (SIRS) is defined by meeting two out of four criteria, representing an acute change from baseline:

Temperature > 38°C or < 36°C

Heart rate > 90 beats per minute

Respiratory rate > 20 breaths per minute or $PaCO_2$ < 32 mmHg

White blood cell count > 12,000/mm^3 or < 4,000/mm^3 or bands > 10%

Sepsis is the systemic inflammatory response to infection.

Severe Sepsis involves organ dysfunction secondary to sepsis, such as hypoperfusion, hypotension, acute lung injury, encephalopathy, acute kidney injury, or coagulopathy.

Septic Shock is characterized by sepsis-induced hypotension that persists despite adequate fluid resuscitation and is associated with hypoperfusion

in recent years we dont use the terms SIRS and severe sepsis

we only use sepsis and septic shock

what is septic shock?

sepsis that requires vasopressor to keep MAP over 65 despite proper fluid rescusitation

and

lacatet more than 2mmol/L

this is from the lecture "sepsis" given by doctor Amjad bani hani.

now there is something called sofa assessment :

it has many points according to different parameters.

RS/CVS/LIVER/RENAL.

what is important is that U know:

Sepsis requires SOFA score increase ≥2 (not 4) to define organ dysfunction. Mortality risk rises with SOFA ≥2 (~10%), and antibiotics should be given urgently. Baseline SOFA = 0 is assumed in healthy patients.

Key for Shelf

HY Point 1: Sepsis-3 organ dysfunction = SOFA increase ≥2 (not 4!).

HY Point 2: SOFA ≥2 ≈ 10% mortality; ≥4 suggests septic shock (>40% mortality).

HY Point 3: Hour-1 bundle: antibiotics + fluids + lactate within 1 hour.-these things should be given within the first hour thus the name hour 1 bundle-.

there is something called quick sofa -qSOFA- which is a screening tool (not diagnostic) for sepsis risk in non-ICU settings.

Criteria (≥2 of 3):

Respiratory rate ≥22/min

Altered mentation (GCS <15)

SBP ≤100 mmHg

Purpose: Rapidly identify patients who may need further workup (e.g., SOFA score, lactate levels).

Evidence: qSOFA ≥2 predicts worse outcomes (e.g., ICU stay, mortality) but has low sensitivity (misses early sepsis).

Thorough Dissection of Key Points

vs. SOFA:

SOFA: Gold standard for defining sepsis (requires labs, e.g., PaO_2/FiO_2, Cr, platelets).

qSOFA: No labs needed; faster but less accurate.

so it is used in a non ICU patient which doesnt have all the monitors that can be used to give parameters used in SOFA assessment.

Key for Shelf : cardiogenic shock.

HY Point 1: Nitroprusside contraindicated in most cardiogenic shock (↓ BP → collapse).

HY Point 2: Norepinephrine = 1st-line vasopressor (↑ SVR, preserves coronary perfusion).

HY Point 3: Dobutamine may be added if CO remains low (β1-agonist ↑ contractility).

HY Point 4: Mechanical support (e.g., IABP, Impella) for refractory cases.

Trauma:

FAST exam is a rapid, bedside ultrasound used in blunt abdominal trauma to detect free fluid (e.g., hemoperitoneum), offering advantages like speed (performed in minutes), repeatability, and no need for patient transport. However, it cannot specify the injured organ—this is a key limitation compared to CT, which provides detailed anatomic localization (e.g., liver vs. spleen injury). While FAST is ideal for unstable patients requiring immediate intervention, CT remains superior for definitive diagnosis, retroperitoneal evaluation, and assessing solid organ damage. Remember: FAST answers "Is there blood?" but not "Where exactly is it from?"—a critical distinction in trauma management.

The primary survey in trauma adheres strictly to the ABCDE algorithm, prioritizing rapid identification of life-threatening conditions through targeted imaging. While CXR (pneumothorax), pelvic X-ray (fracture-related hemorrhage), FAST exam (hemoperitoneum), and cervical spine assessment (spinal cord protection) are essential components, plain abdomen X-ray has no role in this initial phase due to its poor sensitivity for acute injuries

remember in ACUTE TRAUMA

ABCDE:

if the patients airway are patent and he is breathing

we move on to circulation

a Q gave an abdominal trauma patient who is hypotensive but breathing and airways are normal the best next step was establishing IV lines

A child who was involved in a road traffic accident has a bleeding open femur fracture. What is the first step in fluid resuscitation in the emergency room?

- A. Bolus 10ml/kg of normal saline
- B. Bolus 20ml/kg of normal saline
- C. Bolus 20ml/kg of colloid
- D. Transfuse 20ml/kg of packed red blood cells
- E. Transfuse 20ml/kg of packed red blood cells, fresh frozen plasma, and platelets

In pediatric hemorrhagic shock, the first intervention is a 20 mL/kg bolus of normal saline to restore perfusion. This aligns with ATLS protocols and addresses hypovolemia while blood products are prepared. Colloids lack evidence for routine use, and transfusion is reserved for refractory shock. Key priorities: Control bleeding (e.g., tourniquet for femur fracture) and reassess after each bolus.

Key for Shelf:

HY Point 1: 20 mL/kg crystalloid ×2 before blood (exceptions: active hemorrhage with unstable vitals).

HY Point 2: Transfuse 1:1:1 PRBC:FFP:platelets only if ongoing blood loss (e.g., trauma with coagulopathy).

HY Point 3: Avoid colloids—they're expensive and don't improve outcomes in trauma.

if it was an adult:

we dont use weigh based approach

we give 1-2 liters of saline after accessing the IV line in this time we will get the blood cross matched to know their type

after these 2 liters if we need more resuscitation we give blood products

but if we dont have blood products we can use up to 5 liters NS or LR.

In acute trauma, the neuroendocrine stress response triggers a catabolic state dominated by catecholamines, cortisol, and glucagon, driving glycogenolysis and proteolysis to mobilize substrates for survival. This results in hyperglycemia from hepatic gluconeogenesis (using lactate, alanine, and glycerol), while muscle breakdown creates a negative nitrogen balance, depleting glutamine critical for immune and gut function. Lipolysis releases fatty acids as fuel, but insulin and growth hormone resistance prevent anabolic recovery, exacerbating muscle wasting

During trauma, skeletal muscle is the primary protein reservoir broken down to supply amino acids for metabolic demands. The liver utilizes these amino acids to synthesize acute-phase proteins, while plasma proteins are depleted but not actively mobilized

a question was about a trauma pateint, what is the source of protein the answer was liver

i think it is wrong the answer is skeletal muscle

while the liver is the source of glucose.

Major trauma/shock induces hyperkalemia due to cellular breakdown, acidosis, and renal dysfunction, while triggering Na^+/water retention (not secretion) via aldosterone. Renal perfusion drops as blood is shunted to vital organs, and cortisol surges to mobilize glucose (causing hyperglycemia, not hypoglycemia).

Other Surgical Topics:

Fournier gangrene is a rapidly progressive, necrotizing fasciitis of the perineum, genitalia, and lower abdominal wall, most commonly affecting elderly males and those with significant comorbidities such as diabetes mellitus, immunosuppression, or chronic kidney disease. It is typically caused by polymicrobial infections, involving a mix of aerobic and anaerobic organisms such as E. coli, Streptococcus, and Bacteroides species. The disease has a high mortality rate, often between 20–40%, particularly if there is a delay in diagnosis or inadequate surgical intervention. While aggressive debridement of necrotic tissue is critical for survival, orchidectomy is not routinely required even when the scrotum is involved, because the testes have a separate blood supply from the testicular arteries and are often spared. Removal of the testes is only necessary if they are directly affected by the infection or have become necrotic.

The management of Ludwig's angina begins with securing the airway due to the high risk of respiratory compromise from neck swelling and infection. Once the airway is protected, treatment progresses with IV antibiotics to address the infection, followed by incision and drainage if necessary. Addressing the underlying cause, such as treating a tooth abscess or underlying comorbidities like diabetes, is important for long-term care. Finally, supportive measures like IV fluids are used, but they do not take precedence over ensuring the patient can breathe.

C.diff is caused by antibiotics/ steroids/ malnutrition and PPI.

when treating it stop the offending antibiotic and start oral Vancomycin.

vaccines (pneumococcal, meningococcal, Haemophilus influenzae) should be given 1–2 weeks after spleen removal to prevent overwhelming postsplenectomy sepsis. The spleen is frequently injured in blunt trauma, and preservation depends on patient stability—not just associated injuries. Non-operative management can be used for any injury grade in stable patients, and the risk of overwhelming postsplenectomy sepsis is less than 1% in adults (not more than 10%).

Operative Antibiotic Prophylaxis

Reduces bacterial counts at the surgical site.

Should be administered within 60 minutes before the surgery begins (knife to skin).

A repeat dose may be required for longer surgeries, depending on the antibiotic's half-life.

Prophylaxis should not extend beyond 24 hours.

Key factors include prevailing pathogens, antibiotic resistance patterns, and the type of surgery.

Note: Antibiotic prophylaxis is not a replacement for aseptic techniques or proper surgical practices.

now most importantly the antibiotics should be stopped within 24h after surgery - the doctors like this- except cardio 48h

also it should be given WITHIN 1h before surgery meaning it can be given 30 minutes before - there is a Q on this one-

Lymphedema praecox refers to primary lymphedema with onset before age 35 (not exclusive to primary vs. secondary).

Lymphedema tarda describes primary lymphedema with onset after age 35 (also unrelated to secondary causes).

Primary lymphedema predominantly affects women, while filariasis stands as the leading global cause of secondary lymphedema. Lymphoscintigraphy reliably diagnoses the condition, and treatment combines manual lymphatic drainage, compression therapy, and surgical interventions for refractory cases.

PEG stands for **Percutaneous Endoscopic Gastrostomy**.

It's a **minimally invasive procedure** used to place a **feeding tube directly into the stomach** through the abdominal wall, using **endoscopic guidance**.

jejunostomy is a procedure to place a **feeding tube directly into the jejunum** (part of the small intestine), bypassing the stomach. It's used when **gastric feeding is unsafe or ineffective**.

There are a few types, depending on how it's placed:

- **Surgical jejunostomy** (open or laparoscopic)
- **Percutaneous endoscopic jejunostomy (PEJ)** – less common
- **Jejunal extension via PEG** (PEG-J) – feeding tube passed from the stomach into the jejunum

Gastrostomy (PEG) vs. Jejunostomy:

PEG: Easier to place (endoscopic), but higher aspiration risk (stomach feeds).

Jejunostomy: Lower aspiration risk (post-pyloric feeds), but harder to place (requires fluoroscopy/surgery).

Key Facts:

PEG is not surgically closed (tract closes on its own).

Endoscopic placement is standard (not open surgery).

Safer than TPN (gut > IV nutrition).

Shelf Tip:

PEG contraindications: Gastric outlet obstruction, ascites, coagulopathy.

Aspiration risk: Jejunostomy < Gastrostomy < Nasogastric tube.

majority of absorption takes place in the jejunum.

A myocardial infarction within the past 4 months now carries the highest perioperative mortality risk (5-15%), surpassing even severe aortic stenosis (5-10%). This is due to the heightened vulnerability of recently infarcted myocardium to ischemic stress during surgery, with reinfarction rates dramatically elevated within 6 months post-MI.

If the patient has a stroke wait 6 months at least before doing any operations

some weird anastheisa Q

Post-dural puncture headache (PDPH) arises from CSF hypovolemia due to dural tear during epidural anesthesia. Caffeine intake is therapeutic (not avoided), as it vasoconstricts intracranial vessels and boosts CSF pressure, directly opposing the headache mechanism. First-line measures include bed rest (to reduce CSF leak), oral hydration (to stimulate CSF production), and analgesics. For persistent symptoms, an epidural blood patch is definitive

any antibiotic prophylaxis for GI surgery choose cefazolin.- it is a 1st generation cephalosporin-.

Prophylactic antibiotics must be narrow-spectrum (e.g., cefazolin) to target likely pathogens without promoting resistance. They are given as a single pre-incision dose to achieve high tissue levels during surgery-can be redosed only if surgery >4h or major blood loss.-, become ineffective if delayed beyond 3 hours, and do not prevent non-surgical infections (e.g., pneumonia).

there was a stupid question about tetanus im not writing it.

the answers were stupid and it had more than 1 choice as an answer.

Prophylactic antibiotics are unnecessary for duct ectasia procedures, as this benign condition lacks infection risk. In contrast, they are indicated for colectomy (high SSI risk due to bowel exposure) and hernia repairs with mesh (implant-related infection risk)

if there was herniorraphy without mesh it would also not need prophylaxis

there was a question about this and had both duct ectasia and hernioraphy as choices and the answer was herniorraphy.

Erysipelas, caused by Group A Streptococcus, presents with painful, raised, well-demarcated erythema (not flat lesions), typically on the face or legs. Its sharp borders distinguish it from cellulitis, and penicillin remains the gold standard treatment.

HY Point 1: "Streptococcal raised redness" = Erysipelas.

HY Point 2: Cellulitis vs. Erysipelas:

Erysipelas: Sharper borders, more superficial.

Cellulitis: Deeper, indistinct margins.

Erysipelas is a streptococcal infection (not staphylococcal) presenting with painful, sharply demarcated erythema, most often on the legs or face. Penicillin is curative.

erysipalis cannot be caused by staph..

Klebsiella pneumoniae now surpasses E. coli as the predominant cause of pyogenic liver abscesses, particularly among diabetic patients and those of Asian descent. This gram-negative pathogen is notable for causing solitary abscesses that may metastasize to distant sites like the eyes or CNS, a hallmark of hypervirulent K1/K2 strains. While E. coli remains important (especially in polymicrobial infections of biliary origin), and Staphylococcus aureus should be considered in IV drug users.

Diverticulitis:

The Hinchey classification system categorizes diverticulitis severity into four progressive stages based on anatomical spread and contamination. Stage I involves localized inflammation with a pericolic or mesenteric abscess confined near the colon wall, presenting with characteristic left lower quadrant pain and fever. The infection progresses in Stage II to form a pelvic abscess extending into the retroperitoneal space, marked by more severe systemic symptoms and clear abscess formation visible on imaging.

Stage III signifies a critical transition to purulent peritonitis as infected material breaches containment and fills the peritoneal cavity, producing diffuse abdominal findings and systemic toxicity. The final and most severe Stage IV occurs when fecal matter escapes into the peritoneal space, creating fecal peritonitis with its characteristically grave prognosis and high mortality risk.

stage 1 can be considered a phlegmon

Clinically, the Hinchey stages guide both urgency and type of intervention. Stages I-II may sometimes be managed non-operatively with antibiotics and drainage, while Stages III-IV are surgical emergencies. The classification also correlates with wound contamination status: localized inflammation (Stage I) may be clean-contaminated if no abscess forms, but any abscess (Stage II) upgrades the wound to contaminated, and peritonitis (Stages III-IV) is considered dirty, requiring distinct surgical approaches

there is a question bellow

says diverticular stage 2 is contaminated that is why i wrote this paragraph. :)

Amputations:

Major lower limb amputations show better functional outcomes with below-knee (BKA) vs above-knee (AKA) due to preserved knee mechanics - AKA requires 60-100% more energy for ambulation. Diabetics have 10-20x higher amputation risk from PAD/neuropathy, accounting for 70% of non-traumatic cases. Emergency amputations (trauma/sepsis) carry worse prognoses than elective procedures due to unoptimized patients. Specialist surgical teams improve outcomes by reducing complications like wound dehiscence and phantom pain. Post-op, DVT risk jumps to 5-15% (mandate prophylaxis) and mortality is higher for AKA (25-50% at 1 year vs 15-20% for BKA). Always attempt BKA when viable - it doubles the chance of prosthetic use and halves energy expenditure compared to AKA.

Postoperative Care:

Postoperative atelectasis is most often caused by mucus plugs and presents with exam findings like dullness to percussion. It is managed with pulmonary hygienemand analgesia. Fever from atelectasis occurs early (days 1–2),

- ✅ "Atelectasis = Day 1-2 fever, mucus plugs, incentive spirometry."
- ✅ "Day 4 fever? Infection or clot—not atelectasis."
- ✅ "No breath sounds + dullness = atelectasis (not effusion)."
- ✅ "Pain control lets them cough → prevents atelectasis."

Haemorrhage

Immediate:

Inadequate haemostasis or unrecognized damage to blood vessels can lead to immediate bleeding.

Early postoperative:

Defective vascular anastomosis, clotting factor deficiency, or the use of intraoperative anticoagulants may cause early postoperative hemorrhage. Surgical re-exploration is often necessary in such cases.

Secondary hemorrhage:

This type of hemorrhage occurs several days after surgery and is typically related to infection that erodes blood vessels. Treatment focuses on addressing the underlying infection.

At which day post-op will

the protein start to be

metabolized?

- a. 5
- b. 7 NOT SURE
- c. 10
- d. 13
- e. 15

the answer in the file was B

but it is actullay A:

Protein catabolism begins by Day 3 post-op as glycogen depletes, becoming significant by Day 5 when muscle-derived amino acids fuel gluconeogenesis and acute-phase responses. This aligns with the transition from the "flow phase" (Days 1-3) to the "catabolic phase" (Days 3-7) of surgical stress. By Day 7, protein loss peaks, making Day 5 the most accurate start point for clinically relevant protein metabolism.

Key for Shelf

HY Point 1: Glycogen lasts ~48h → Then protein/fat catabolism.

HY Point 2: Negative nitrogen balance = Hallmark of catabolism (peaks Days 5-7).

Refeeding Syndrome:

Re-feeding syndrome is characterized by severe hypophosphatemia, hypokalemia, and hypomagnesemia due to insulin-driven intracellular shifts during nutritional repletion. These abnormalities can lead to cardiac arrhythmias, respiratory failure, and neurologic complications (e.g., seizures). Prevention involves gradual caloric advancement

Key for Shelf:

HY Point 1: Hypophosphatemia is the hallmark (check levels before starting nutrition).

HY Point 2: Replace electrolytes aggressively (IV phosphate if <2.0 mg/dL).

HY Point 3: Start nutrition slowly (≤20 kcal/kg/day in high-risk patients).

These are 1 line questions in the last few pages of the file.
eeeeasy.

• Not to do in sepsis?

Infection source control during 1st hour, stabilize the patient first

• Wrong about fluids:

130mml/L of Cl in ringer lactate (112)

• Regarding wounds which is true?

Diverticulitis stage 2 is considered a contaminated wound

• 1st in hemostasis >>> Vasoconstriction

• Vit. C.>>> Hydroxyaltion of procollagen

• True about abx: carabapenems have good coverage for gram +ve and anaerobes

• True about gas gangrene?? pain, crepitus and toxemia

• True>>> Canal of Nuck opens in labia majora

• True about hypovolemic shock? Increased SVR

• MCC in septic shock? staph/ pseudomonas/ Ecoli

• True about septic shock:

Persisting hypotension requiring vasopressors to maintain a MAP of 65

• Management of gastric outlet obstruction with hypochloremic hypokalemic metabolic alkalosis?

0.9 NS infusion with KCl

• Most common cause of death after blood transfusion?

TRALI - i think it is ABO incombatibilty-

• Which of the following is true about body fluids?

It might be affected by wide range of physiological variation

• Which of the following is true about body fluids?

The concentration of sodium in the intravascular and the interstitial compartment is almost equal.

• Amino acid most important in improving immunity:

Glutamine

• Limit for K in peripheral line: 20 mEq

• Pt with crush injury, in respiratory distress, multiple rib fractures,

life saving measure is:

Intubation and mechanical ventilation

• Plain AXR in SBO, what is the finding?

Valvulae conniventes (plica circularis)

• Not complication of TPN: - Hypoglycemia (mentioned in past papers, but both HYPO/HYPER

glycemia are possible complications of TPN)