Peripheral Vascular Disease (PVD)

Claudication affects only 10-35% of PVD patients—most are asymptomatic or have atypical symptoms-so in pateints with PVD almost 70% will be asymptomatic-. Asymptomatic PVD isn't benign; it doubles cardiovascular mortality and requires aggressive risk modification (statin, antiplatelet). Rest pain signals critical limb ischemia (50% amputation risk at 1 year), while claudication is often stable with medical management.

ABI is imperfect—---normal values miss non-compressible arteries (diabetics need toe-brachial index) and proximal disease. so the BPI is imperfect bcs DM can cause calcification of arteries raising the ABPI falsely

also if the disease was in proximal arteries it will also miss it.

intermittent claudication is exercise induced pain. it disappears at night -U can say improves at night but there is no improvement just the pain goes away-.

other HY points:

"Pain resolves within 2–5 minutes of rest."

"Symptoms are reproducible with consistent walking distance."

exercise program help to increase the walking distance -which is the distance walked until there is pain- and it is the first step in management not medications.

HY Point 1: IC-intermittent claudication-= pain with walking, relief with standing.

HY Point 2: Night pain = CLI -critical limb ischemia-

HY Point 3: Ankle-brachial index (ABI) <0.9 confirms PAD.

in CLI:

patients undergo revascularization first (endovascular > bypass)

However, CLI has a far worse prognosis than claudication (5-year mortality 50-70% vs. 10-30%)

When arteries are non-reconstructable or infection is life-threatening, amputation becomes the only option

we try to delay amputation as much as we can and the majority of cases undergo revascularization.

Revascularization in PAD is prioritized for critical limb ischemia (rest pain, ulcers, gangrene) to prevent limb loss, while claudication is managed conservatively unless disabling. Ischemic neuropathy, though rare, reflects advanced ischemia.

Revasculirization surgery is indicated in all of the following cases except:

- A) Claudication
- B) ischemic neuropathy
- c) rest pain
- D) ulcer
- e) gangrene

the answer file said B

DeepSeek:

Revascularization in PAD is prioritized for critical limb ischemia (rest pain, ulcers, gangrene) to prevent limb loss, while claudication is managed conservatively unless disabling. Ischemic neuropathy, though rare, reflects advanced ischemia

Claudication is managed with exercise, smoking cessation, and statins/antiplatelets. Revascularization is considered only if symptoms are refractory to conservative measures and severely impair function.

DM/ HTN and smoking

all are risk factors for PVD + hypercholesterolemia or history ischemic cardiac disease. but the 3 above have more risk.

from the 3

DM is the most one associated with symptomatic PVD.

HY Point 1: DM > HTN for symptomatic PVD

HY Point 2: Smoking is the #1 modifiable risk factor for PVD incidence (but DM wins for symptom severity).

Popliteal artery aneurysms (PAAs) are the most common peripheral aneurysms, accounting for ~70% of cases (femoral aneurysms are second).

They exhibit a strong systemic vascular link:

Bilaterality (50-70%): Always assess the contralateral artery.

30-50% of PAA patients have an AAA (so PAA should prompt AAA screening).

Only ~10-20% of AAA patients have a PAA.

Clinical Presentation & Complications:

Most are asymptomatic but may present with:

Acute limb ischemia (from thrombosis, the most dreaded complication).

Chronic claudication (if gradual thrombosis occurs).

Compression symptoms (rare; e.g., tibial nerve palsy, DVT from popliteal vein compression).

Gender disparity: 95% male, typically >60 years old.

Claudication management hinges on lifestyle modification (exercise, smoking cessation) and cardiovascular risk reduction (e.g., statins). While aspirin is vital for preventing thrombotic events, it lacks symptomatic benefit. Revascularization (bypass/angioplasty) is reserved for refractory cases

-Aspirin prevents thrombosis but doesn't directly improve blood flow to relieve claudication-

so it is used in the treatment but not due to its symptomatic benifit.

In symptomatic PAD (intermittent claudication), CTA is the most important diagnostic test because it precisely maps arterial lesions, guiding revascularization decisions. While ABI is the initial screening tool, it cannot identify stenosis location.

Deep Vein Thrombosis (DVT)

DVT management prioritizes prompt outpatient anticoagulation for stable patients, with admission reserved for high-risk cases.

Nearly half of DVT patients harbor silent PEs, necessitating cardiorespiratory assessment regardless of symptoms.

Anatomic location dictates urgency—proximal DVTs always require treatment, while select calf DVTs may be monitored.

Provoked vs. unprovoked status determines therapy duration, with unprovoked cases warranting extended treatment or cancer screening.

Crucially, anticoagulation should never be delayed for imaging when clinical probability is high.

Key for Shelf:

HY Point 1: DOACs first, hospitalize never (unless high bleeding/instability).

HY Point 2: No dyspnea ≠ no PE—check vitals in all DVT diagnoses.

from slides:

when to admit or stay outpatient

DVT Admission Criteria (Rare):

Absolute:

Active bleeding or very high bleeding risk (e.g., recent brain hemorrhage).

Severe renal dysfunction (CrCl <30 mL/min) → limits LMWH/DOAC use.

Relative:

Iliofemoral DVT (if considering catheter-directed thrombolysis).

PE Admission Criteria (More Common):

Hemodynamic instability (SBP <90 mmHg, shock).

Hypoxemia (requires supplemental O_2).

Severe pain (needs IV narcotics).

Same bleeding/renal contraindications as DVT.

Massive PE (RV strain on echo/CT → thrombolysis candidate).

Key for Shelf:

✅ HY Point 1: "BLEEDING + KIDNEYS" = Admission triggers for both DVT/PE.

✅ HY Point 2: PE > DVT for hospitalization (due to RV failure risk).

✅ HY Point 3: Iliofemoral = "leg PE" → thrombolysis considered like massive PE.

Identify the condition with the highest relative risk (RR) of DVT among genetic/acquired thrombophilias:

Highest RR (>10x)up to 40 times more risk: Antiphospholipid syndrome (APS).

Moderate RR (3-8x): Factor V Leiden (heterozygous), prothrombin G20210A.

Lower RR (2-5x): Protein C/S deficiency (heterozygous).

note: factor V Leiden is the most common inherited genetic thrombophilia

all the genetic thrombophilia's have an autosomal dominant inheritance

however:

Homozygous Factor V Leiden

RR: 50-100x (vs. general population)

Lifetime VTE risk: >80%

Homozygous Prothrombin G20210A

RR: 20-50x.

1. Phlegmasia Alba Dolens ("Milk Leg" or "White Leg")

Definition:

Acute, near-total occlusion of the deep venous system (iliofemoral DVT) with preserved superficial venous drainage.

Pathophysiology:

Massive DVT → venous outflow obstruction → reflex arterial spasm → pale, pulseless leg.

Superficial veins remain patent (collateral drainage).

Classic Triad:

Pale/white limb ("alba" = white).

Absent pulses (spasm, not arterial occlusion).

Severe pain + edema.

Key Point:

Reversible with urgent anticoagulation (LMWH).

No tissue necrosis (distinguishes from PCD).

2. Phlegmasia Cerulea Dolens ("Blue Leg")

Definition:

Total venous occlusion (deep + superficial systems) → venous gangrene.

Pathophysiology:

DVT progression → capillary rupture → hemorrhagic ischemia → blue/purple discoloration.

No venous outflow → compartment syndrome → limb necrosis.

Classic Triad:

Blue/purple limb ("cerulea" = blue).

Hemorrhagic bullae (late sign).

Gangrene (without intervention).

Key Point:

Surgical emergency → 30-50% mortality.

Requires thrombolysis/thrombectomy ± fasciotomy/amputation.

venous system:

Valves: are not Present in iliac veins but in the femoral and popliteal.

Muscle pump: Calf contraction drives >60% venous return → critical for flow.

Perforators: Direct blood superficial → deep → failure causes varicose veins.

Walking: Drops venous pressure in healthy people (rises in disease).

great saphenous vein joins the femoral vein but doesn't form the common femoral vein.

Now to get the last point straight if U look at images they join together and they become the common femoral DeepSeek has problem with the wording FORM.

HY Point 1: Muscle pumps + valves = venous return engines (calf contraction contributes ~60% of flow).

HY Point 2: Perforators flow superficial→deep—reversal indicates pathology (e.g., chronic venous insufficiency).

HY Point 3: Venous pressure drops with walking (if it rises, think venous obstruction/insufficiency).

.which of the following is true regarding venous circulation:

- A)Muscle contraction play no role in venous return
- B) The common iliac veins has valves
- C) The greater saphenous vein joins the femoral vein to become the common femoral vein
- D) In healthy person, venous pressure increases with walking
- e) The perforating veins in the leg directs the flow from the deep to superficial system

the file says it is D

but according to DeepSeek it is C but the sentence is not completely true

patients with chronic venous insufficiency -medial malleolar ulcer, varicosities, and symptoms relieved by elevation-

the primary cause or the diagnosis is:

Perforator incompetence:

it Allows retrograde flow from deep to superficial veins → venous hypertension → ulcer.

-this is more plastic and present in the lecture -chronic wounds-

Lymphedema management prioritizes conservative measures (compression, skin care, weight loss), with surgery as a last resort.

While filariasis dominates globally, lymphedema praecox is the leading primary form. -most common as a whole is filariasis put the most common primary form is praecox-.

Diagnosis relies on clinical assessment and lymphoscintigraphy -U can use lymphogram but it is outdated-.

weight loss helps : Obesity exacerbates lymphatic dysfunction; weight loss reduces fluid load.

Sign of hemolytic reaction after blood transfusion:

Answer: burning at site of injection

Congenital Heart Defects & Fetal Circulation

At birth, the fetal circulation undergoes critical changes:

the foramen ovale functionally closes within hours due to rising left atrial pressure (though anatomic closure takes months),

while the ductus arteriosus—which shunted right-to-left blood in the fetus—typically closes within 24–72 hours post-birth due to increased oxygen tension (PaO_2 >50 mmHg) and decreased prostaglandins, reversing any persistent flow to left-to-right.so even if it remained patent the shunt is left to right bcs the left side has higher pressures.

The umbilical arteries, branches of the internal iliac arteries (not common iliac), atrophy after clamping.

Crucially, pulmonary vascular resistance (PVR) drops immediately at birth due to lung expansion and oxygen exposure, independent of ductus closure, facilitating pulmonary blood flow.

Low oxygen tension (e.g., in cyanotic heart disease) paradoxically maintains ductus patency (PDA), while high oxygen promotes its closure.

know these facts about fetal circulation.

HY Point 1: O_2 closes DA, pressure closes foramen ovale.

HY Point 2: Umbilical arteries = internal iliac branches (testable anatomy).

HY Point 3: PVR drops first → then DA/foramen respond.

Tetralogy of Fallot (TOF) - The Classic Tetrad

Ventricular Septal Defect (VSD)

Right Ventricular Outflow Tract Obstruction (RVOTO)

Right Ventricular Hypertrophy (RVH)

Secondary to RV pressure overload (due to VSD + RVOTO).

"Boot-shaped heart" on CXR from prominent RV.

Overriding Aorta

Contributes to right-to-left shunting and cyanosis.

most common congenital cardiac defect:

membranous VSD.// more than the muscular type.

Tetralogy of Fallot is caused by malalignment or anterosuperior displacement of the infundibular septum, resulting in pulmonary stenosis, RVH, overriding aorta, and a VSD. It is not related to aorticopulmonary membrane defects, which are seen in truncus arteriosus.

During atrial development, the foramen primum closes via fusion of the septum primum with the AV cushions.

Septum secundum with septum primum fusion leads to closure of the foramen ovale.

In TOF, pulmonary stenosis severity is the primary physiologic driver, dictating shunt direction (right-to-left → cyanosis)

Anomalous Left Coronary Artery from Pulmonary Artery (ALCAPA):

Pathophysiology:

Left coronary artery (LCA) arises from PA.

Postnatal PA pressure drop → LCA perfusion ceases → LV ischemia → cardiogenic shock.

Lethality: Untreated ALCAPA has 90% mortality by age 1 due to LV infarction

Origin of the right coronary artery from the pulmonary artery:RCA supplies less myocardium; often asymptomatic or presents in adulthood.

PDA all true except:

- a) Associated with maternal rubella
- B) Decreased murmur sound is a bad prognostic factor
- C) LV hypertrophy precedes RV hypertrophy
- D) Most cases closes spontaneously in the neonatal period
- e) One third die at age of 40 if not corrected

answer in the file was C

DeepSeek says it is D

chat gpt says it is C.

1. Partial Anomalous Pulmonary Venous Return (PAPVR)

Definition: Some (1–3) pulmonary veins drain into the right atrium (RA) or systemic veins (e.g., SVC) instead of the left atrium (LA).

2. Total Anomalous Pulmonary Venous Return (TAPVR)

Definition: All pulmonary veins drain into the RA/systemic veins (none connect to LA).

Sinus venosus defect (SVD) is the ASD most tied to PAPVR due to shared embryologic origin near the sinoatrial junction.

Data: >90% of SVD cases have PAPVR

Mediastinum

mediastinal masses:

Mediastinal masses are compartment-specific: thymomas (anterior) link to myasthenia gravis, while neurogenic tumors (posterior) are often asymptomatic. Though primary masses (e.g., lymphomas, thymomas) are classic, metastases (e.g., from lung/breast cancer) are statistically more common.

which of the following least likely to cause middle mediastinal mass:

- a) Neuroblastoma
- B) Lymphoma
- C) Thymoma
- D) Ganglionblastoma

the answer in the file was D

but DeepSeek and chat gpt:

Correct Answer: C ("Thymoma")

Thymomas are exclusively anterior mediastinal masses (thymus location).

Never occur in the middle mediastinum.

Why Others Are More Likely (or Possible):

A ("Neuroblastoma") & D ("Ganglioneuroblastoma"):

Typically posterior mediastinal (neural crest origin).

Rarely, large tumors may extend into middle compartment (but origin is posterior).

B ("Lymphoma"):

Common in middle mediastinum (hilar/paratracheal lymph nodes).

Also occurs in anterior mediastinum (especially Hodgkin's).

Metastatic disease (particularly bronchogenic carcinoma) dominates pericardial tumors, overshadowing primary tumors like mesothelioma. Lung cancer's propensity for direct extension and hematogenous spread makes it the leading cause, while lymphoma and mesothelioma are rarer.

Metastatic tumors are 30-50x more common than primary pericardial tumors.

Lung cancer accounts for 36% of pericardial metastases.

Lymphoma involves pericardium in ~10% of advanced cases.

Mesothelioma of pericardium is exceptionally rare (<0.002%).

Lung and Thorax

an air fluid level post-pneumonectomy is bronhopleural fistula until proven otherwise

the other signs will be fever and cough with lots of sputum.

the first step is drainage.

HY factoids:

A febrile post-pneumonectomy patient with an air-fluid level likely has empyema with BPF, requiring urgent chest tube drainage for both diagnosis and treatment. While cultures and antibiotics are adjuncts, they're futile without source control. Thoracotomy is reserved for complex cases. Delaying drainage risks sepsis or tension physiology.

Key for Shelf:

HY Point 1: Air-fluid level post-pneumonectomy = BPF until proven otherwise.

HY Point 2: Drain first, culture later (emergent vs. elective diagnostics).

HY Point 3: Thoracotomy only if drainage fails or necrotic tissue exists.

in the file the first step was fluid analysis and culture

the things i wrote are from DeepSeek.

choose what U like.

Chest trauma contributes to 25% of trauma deaths, primarily from immediately lethal vascular injuries or rapidly correctable conditions like tension pneumothorax.

now in cases of hemothorax when do we do thoracostomy ?

if there is rapid accumulation of more than 1500 ml-roughly 3rd of the body's blood volume- in the pleural cavity/ or the rate of more than 200ml per hour for at least 3 to 4 hours.

Tracheal/bronchial repairs are done using posterolateral thoracotomy --median sternotomy is for cardiac/vascular injuries).

Persistent low CO post-cardiac surgery typically stems from preload reduction (tamponade, hypovolemia, bleeding) or impaired contractility (myocardial dysfunction)

now blood PH : alkalosis doesn't effect cardiac function but acidosis does

it decreases Cardiac output.

Key for Shelf:

HY Point 1: "Tamponade, bleeding, and pump failure" are the CO killers.

HY Point 2: Acidosis ↓ CO, but alkalosis is benign/helpful.

HY Point 3: Bleeding → ↓ Preload → ↓ CO (treat with volume, not inotropes).

now how does acidosis decrease CO well it has many effects look it up if U like

Key for Shelf

✅ HY Point 1: pH <7.2 = Critical threshold for myocardial dysfunction.

✅ HY Point 2: Lactic acidosis is both cause and effect of low CO.

pneumothorax:

Tension PTX is a clinical diagnosis → treat based on:

Hypotension + respiratory distress

Unilateral absent breath sounds

Tracheal deviation (late sign)

CXR delays definitive care (needle thoracostomy comes first).-"Needle first, ask later" (no CXR).-

Other False Statements:

"Tracheal deviation is the first sign" → Incorrect (hypotension occurs earlier).

"All pneumothoraces require chest tube" → Incorrect (small simple PTX may resolve).

Pneumothorax most reliably presents with decreased/absent breath sounds due to lung collapse, while hyperresonance on percussion further supports the diagnosis. Mediastinal shift, if present, is contralateral (key for tension pneumothorax). Dullness suggests effusion/consolidation, and crepitations indicate alternative diagnoses.

Spiculated margins on imaging are highly predictive of lung cancer due to invasive tumor behavior, warranting prompt biopsy or resection

HY Point 1: Spiculation = malignancy until proven otherwise (93% PPV).

HY Point 2: Volume doubling time 30-400 days = typical malignant growth.

HY Point 3: Adenocarcinoma is most associated with spiculated/GGO nodules.

most common lung tumors are from metastesis

most common primary tumor is adenocarcinoma then squamous cell carcinoma.

pan coast tumor what is correct:

- a) Pancoast tumor account for 25% of all bronchogenic carcinomas
- b) Squamous cell carcinomas account for two third of all pancoast tumours
- C) Pancoast tumours are by definition T2 tumors
- D) Induction chemo-radiotherapy is the standard of care for any potentially resectable Pancoast tumor
- e) Surgery for pancoast tumour is associated with 50% mortality rate

A: Incorrect → Pancoast tumors are 3-5% of lung cancers.

B: Incorrect → Adenocarcinoma (60%) > squamous (30%).

C: Incorrect → Pancoast tumors are T3/T4 by definition.

D: Incorrect terminology → "Induction" is for SCLC; "neoadjuvant" is correct for NSCLC.

E: Incorrect → Surgery mortality is <5%.

so according to the new epidemiological studies adenocarcinoma is more common than squamous

the answer in the file was B

lung CA:

Surgery is curative for early-stage NSCLC (I-II).

Neoadjuvant chemo helps in advanced resectable (IIIA) NSCLC.

Chemoradiation is standard for unresectable IIIB.

IIIA (resectable) vs. IIIB (unresectable).

SCLC = chemo-responsive, rarely surgical.

Overall 5-year survival is ~20%).

Hypertension is the most common etiology of thoracic aortic aneurysms.

the file says it is atherosclerosis but i watched the lecture the doctor did not mention it

the hypertension is most common cause

While atherosclerosis predominates in abdominal aneurysms, it plays a lesser role in TAAs.

thoracic aortic repair complications:

Most feared complication is:

paraplegia

Mechanism: Interruption of spinal cord blood flow (e.g., during aortic clamping or coverage of critical segmental arteries in endovascular repair) → ischemic injury to the anterior spinal artery → paraplegia (lower limb paralysis).

Other Complications (Why They're Less Feared):

Stroke (from emboli during aortic manipulation).: Significant but often manageable with rehab; mortality depends on severity.

Renal Failure (due to ischemia or contrast nephropathy).: May require temporary dialysis but is often reversible.

Bleeding (aortic tissue fragility).: Controllable with surgical re-exploration or transfusions.

best imaging modality to reveal vascular disease is CT angio

AAA repair requires direct exclusion of the aneurysm sac via open graft placement or endovascular stenting. Axillo-bifemoral bypass, while useful for aortic occlusive disease or infected grafts, merely creates an alternative blood flow path and leaves the aneurysm untreated—making it inappropriate for AAA management.

Miscellaneous

Most common cause of death at site of accident is:

- a) Vascular injury
- B) Thoracicinjury
- c) CNS injury

the answer is A

classes of hypovolemic hemorrhage :

class 1 lost less than 750ml of blood

class 2 lost 750-1500 ml of blood.

treat both with crystalloids

class 3 1500-2000

class 4 more than 2000 ml

we treat these two with PRBCs.

in a normal person with normal hemoglobin

p50: 25mmhg

p75: 40mmhg

p90: 60mmhg

P50 Definition:

Partial pressure of oxygen (PaO_2) at which Hb is 50% saturated.

compartment syndrome:

a limb-threatening condition caused by increased pressure within a fascial compartment.

Classic 5 P's:

Pain (out of proportion, passive stretch pain)

Paresthesia (early nerve compression)

Pallor/Pulselessness (late signs; unreliable for early diagnosis)

Paralysis (very late)

Pulselessness is a LATE sign (indicating arterial occlusion or irreversible damage).

Early signs: Severe pain, paresthesia, and tense swelling.

Clinical Impact: Waiting for pulselessness delays life-saving fasciotomy.

Diagnosis:

Clinical suspicion + compartment pressure measurement (>30 mmHg or ΔP <30 mmHg [diastolic BP − compartment pressure]).

Treatment:

Emergent fasciotomy of all affected compartments to prevent necrosis.Fasciotomy must include all involved compartments (e.g., 4 in forearm, 4 in leg) to prevent Volkmann's contracture or amputation.

In aortic stenosis, CHF portends the worst survival due to irreversible LV failure, while syncope and angina reflect less advanced disease. The onset of CHF signals urgent need for valve replacement, whereas angina may allow delayed intervention

HY Point 1: CHF = worst prognosis (50% 1-year mortality untreated).

HY Point 2: Syncope > Angina in severity (3 vs. 5-year median survival).

HY Point 3: Valve replacement is lifesaving for symptomatic AS (especially CHF).

Intra-aortic balloon pump (IABP):

IABP Mechanics:

Diastolic inflation: Augments coronary perfusion.

Systolic deflation: Reduces afterload.

Femoral artery access: Primary insertion site.

Limb ischemia dominates IABP complications due to femoral artery catheterization, occurring in 20-30% of patients. While aortic dissection and thrombosis are serious, they are far less frequent

Stroke and arrhythmias are rare/unrelated to IABP.

Mechanical Valve Advantages:

Durability: Lasts decades (lifelong in young patients).

Indications: Preferred for patients with long life expectancy (e.g., age <50–60) or high risk of bioprosthesis degeneration.

but it needs lifelong warfarin therapy a

Bioprosthetic Valve Advantages:

No lifelong anticoagulation: Preferred for older patients (>65–70) or those with contraindications to warfarin (e.g., pregnancy, bleeding risk).

Valve selection balances durability vs. anticoagulation burden. Mechanical valves suit younger patients (e.g., 30-year-old male) who can tolerate warfarin, while bioprostheses favor older adults or those avoiding anticoagulation (e.g., pregnancy). -warfarin is teratogenic-.

HY Point 1: <50–60 years old → Mechanical valve (if no anticoagulation contraindications).

HY Point 2: >65–70 years old → Bioprosthetic valve (avoids warfarin; lifespan matches valve durability).

HY Point 3: Pregnancy = Bioprosthetic valve (warfarin is teratogenic).

CABG Indications - Concise Guide

1-Triple Vessel Disease -RCA/LAD/LCX. typically with EF less than 40%.

Blockages in all 3 major coronary arteries → CABG preferred over stents (better long-term survival).

2-Left Main Coronary Artery Disease

50% blockage in left main artery → CABG mandatory (high mortality if untreated).

3-Unstable Angina (Failed on maximum doses of Meds)

Chest pain persists despite drugs → needs surgery.

4-PTCA Complications

Failed angioplasty/stent (e.g., artery dissection) → emergency CABG.

5-MI Complications

Ruptured heart muscle/VSD → immediate repair.-ruptured papillary muscle leading to severe acute mitral regurg.

6-Coronary Anomalies

Birth defects causing ischemia → may need CABG.

know the anatomical supply of each coronary artery and the concept of left dominant or right dominant PDA.

I did not include 2020 questions
try and test Urself. ☺