



## Chapter 87 – Peripheral Arteriovascular Disease

### Episode Overview:

1. What is an atheroma and how is it formed?
2. What are the classic symptoms of arterial insufficiency?
3. Provide a differential diagnosis for chronic arterial insufficiency.
4. What is blue toe syndrome? What is its significance?
5. Differentiate between thrombotic and embolic limb ischemia based on clinical features.
6. What is the management of an acutely ischemic limb?
7. List three disorders characterized by abnormal vasomotor response.
8. Describe Raynaud's disease and how it's treated?
9. What is the most common site for an arterial aneurysm in the leg?
10. List four potential sites for upper extremity aneurysms, and their associated underlying causes.
11. Name three types of visceral aneurysms and their associated conditions.
12. List 6 differential diagnosis of an occluded indwelling catheter and describe the management of a suspected line infection.
13. What are the two types of arteriovenous (AV) fistulae used for dialysis?
14. How do you access an AV fistula?
15. List 5 complications of dialysis fistulas and treatment.
16. List the 3 types of thoracic outlet syndrome. What are the typical symptoms of thoracic outlet syndrome? What is a simple bedside test for this condition?
17. List 4 anatomic abnormalities associated with thoracic outlet syndrome.

### Wisecracks:

1. Describe Buerger's sign and the ankle brachial index.
2. List the clinical criteria for Buerger's Disease (5).
3. What is Leriche's syndrome?
4. List 4 types of infectious aneurysms.
5. Differentiate between arterial insufficiency ulcers and venous stasis ulcers.

### Rosen's in Perspective

This chapter covers the diseases that affect the medium and small arteries in the body (ie. not the aorta, innominate, common carotid, subclavian, or pulmonary arteries)

While not featured in the podcast, we're going to take a deeper dive into the 3 layers of an arterial wall here in the shownotes...because this is going to be a beast of a podcast!

Describe the 3 wall layers of an artery:

- Tunica:
  - Intima: the inner endothelial lining that protects the subendothelium. The thin endothelium is thrombosis-resistant. Endothelial injury leads to cascades of atherosclerosis and pro-thrombotic activity.



- Media: made of circular and spiral smooth muscle cells. This contains the outer elastic membrane which is replaced with fibrous tissue in the aging process.
- Adventitia: contains nerve fibers and nutrient vessels. Medium sized arteries have connections with the autonomic nervous system to allow control of vascular tone and blood flow.

Pathophysiology of the EIGHT disease processes that affect arteries:

Disease process	Pathophysiology	Notes
<i>Atherosclerosis</i>	<ul style="list-style-type: none"> <li>• Begins with the formation of an atheroma = a fibrofatty plaque. Usually extends into the intima.</li> <li>• These lipid-cores with fibrous caps usually calcify - leading to brittle crystalline vessels.</li> <li>• These can ulcerate and produce cholesterol emboli and/or lead to in-situ thrombosis.</li> </ul>	<ul style="list-style-type: none"> <li>• Commonly affects large and medium sized vessels, especially around branch points.</li> <li>• Aortoiliac, femoral, popliteal, coronary, internal carotid, circle of Willis.</li> <li>• Can lead to weakness of the media and subsequent aneurysmal dilation.</li> </ul>
<i>Aneurysm</i>	<ul style="list-style-type: none"> <li>• True aneurysm: localized dilation of intact vessel wall.</li> <li>• Pseudoaneurysm: the entire wall perforates or ruptures - the extravasated blood is contained by the surrounding tissue (forming a fibrous sac that communicates with the artery).</li> </ul>	<ul style="list-style-type: none"> <li>• Can also have intra-aneurysmal ulcers (leading to atheroembolism) or intramural thrombosis</li> </ul>
<i>Embolism</i> (includes thromboembolism, atheroembolism)	<ul style="list-style-type: none"> <li>• A blood clot or foreign body that is carried to a distant site from its point of origin.</li> <li>• Most are arterial thromboembolisms and originate in the heart. (e.g. post MI, AFib).</li> <li>• Other sources include bacterial emboli, tumour material, foreign bodies, etc.</li> </ul>	<ul style="list-style-type: none"> <li>• Sudden occlusion of an artery that doesn't have collateral circulation = bad</li> <li>• Embolic occlusion occurs commonly at femoral and popliteal branch points.</li> <li>• Watch for life threatening reperfusion injury.</li> <li>• Atheroembolism: cholesterol, calcium, platelets that lodge in distal small arteries. (Ischemic strokes, cool painful cyanotic toes).</li> </ul>
<i>Thrombosis</i> (arterial thrombosis)	<ul style="list-style-type: none"> <li>• In-situ formation of blood clots in the arteriovascular system.</li> <li>• Usually due to atherosclerosis</li> </ul>	<ul style="list-style-type: none"> <li>• Often caused by atherosclerotic plaque rupture or endothelial injury due to trauma or vasculitis</li> </ul>
<i>Inflammation</i>	<ul style="list-style-type: none"> <li>• Can be due to drugs, irradiation, mechanical trauma, bacterial invasion, IVDU, etc.</li> </ul>	<ul style="list-style-type: none"> <li>• Noninfectious systemic: necrotizing vasculitis ( look for macules, papules, vesicles, bullae).</li> <li>• Aspergillosis or mucormycosis.</li> </ul>
<i>Trauma</i>	<ul style="list-style-type: none"> <li>• Partial wall lacerations form expanding hematomas</li> <li>• Complete wall lacerations cause arterial spasm and retraction = less bleeding</li> <li>• Blunt trauma may cause a dissection, which forms a site</li> </ul>	<ul style="list-style-type: none"> <li>• Can get delayed bleeding when the completely lacerated artery relaxes.</li> </ul>



	for complete thrombosis days later.	
<i>Vasospasm</i>	<ul style="list-style-type: none"> <li>Abnormal vasospasm response in distal arteries</li> <li>Unknown etiology - never leads to tissue loss.</li> </ul>	<ul style="list-style-type: none"> <li>e.g. Raynaud's, livedo reticularis, acrocyanosis, erythromelalgia</li> </ul>
<i>Arteriovenous fistula</i>	<ul style="list-style-type: none"> <li>Due to congenital disease, arterial erosion or aneurysm, vasculitis, trauma, inflammation.</li> </ul>	<ul style="list-style-type: none"> <li>Leads to chronic venous hypertension, dermatitis, ulceration.</li> <li>Most AV fistulas are associated with a false aneurysm.</li> </ul>

### 1) What is an atheroma and how is it formed?

“The basic lesion, the atheroma, or fibrofatty plaque, is a raised focal plaque within the intima; it has a lipid core (mainly cholesterol, usually complexed to proteins and cholesterol esters) covered by a fibrous cap. As the plaques increase in size and number, they progressively encroach on the lumen of the artery and the adjacent media. Atheromas compromise arterial blood flow and weaken the walls of the affected arteries.”

- From Rosen’s 8th Edition.

### 2) What are the classic symptoms of arterial insufficiency?

- This is a chronic disease which produces two different pain syndromes:
  - Intermittent claudication
  - Ischemic pain at rest

Intermittent claudication can occur anywhere, but usually is in the calf (femoral or popliteal disease). Described as cramping pain *reproduced by the same degree of exercise and relieved by 1-5 minutes of rest.*

- Can also occur in the aortoiliac vessels causing buttocks/hip pain, aching, discomfort, weakness, or “giving out” with exercise
- See the discussion below on Leriche’s syndrome

When the disease progresses, ischemic symptoms can occur at rest. It usually starts at the foot. The classic scenario would be a person being awoken from sleep complaining of severe un-relenting pain distal to the midfoot. This pain is NOT relieved by analgesics and WORSENERD with elevation. Ask about the “dangling over the bed” or sleeping in a recliner chair. Their pain should improve with standing up.

### 3) Provide a differential diagnosis for chronic arterial insufficiency.

**NB: Less than 5% of lower extremity ulcers are caused by arterial insufficiency**

- Chronic venous insufficiency (much more common than arterial insufficiency)
- Neuropathic ulceration (from uncontrolled diabetes mellitus)
- Nocturnal muscle cramps
- Aortoiliac occlusive disease leading to atheromatous emboli (causing multiple ulcerations)



- Vasculitis
- Chronic subcutaneous intravenous drug use
- Malignant ulcers (look for thickened, rolled, elevated edges with a central depression containing granulation tissue)
- Osteoarthritis from associated joints
- Spinal stenosis
- Cauda equina syndrome (disc, cancer, infection)
- Hypertensive ulcers (lateral ankle - blue-red areas of infarcted skin)

#### 4) What is blue toe syndrome and what is its significance?

Blue toe syndrome is caused by acute arterial occlusion due to microemboli (atheroemboli) made of cholesterol, calcium, platelets, and other debris that break off from proximal aneurysms/plaques and lodge distally

- AKA: “trash foot”
- In the CNS, these phenomena lead to TIA’s and Strokes
- In the lower extremities these form painful cyanotic areas on the feet (usually toes), i.e. Blue toes

The key is to try and identify the proximal source of the atheroemboli, which may include AAA, iliac/femoral/carotid/popliteal artery atheromas, etc.

Most of these patients should have good peripheral pulses, asymmetric distribution of these lesions, and no evidence of systemic vasculitis.

Treatment includes identification and removal of the proximal source. Usually CT-angiography is the test of choice to identify the proximal lesion. Definitive treatment includes local endarterectomy, vascular bypass, angioplasty, etc. Medical management is with antiplatelets and anticoagulant to prevent further thrombosis.

#### 5) Differentiate between thrombotic and embolic limb ischemia based on clinical features.

See table 87-1 in Rosen’s:

	<b>Thrombotic</b>	<b>Embolic</b> (clots lodge at arterial bifurcations)
<b>History</b>	<ul style="list-style-type: none"> <li>● History of claudication or rest pain</li> <li>● Atherosclerosis and traditional cardiovascular risk factors</li> </ul>	<ul style="list-style-type: none"> <li>● History of AFib/arrhythmias or thrombotic disease</li> <li>● PFO’s or congenital heart disease</li> <li>● Look for a cause such as an LV thrombus (recent MI)</li> </ul>



<p><b>Physical Exam</b></p> <p>Remember check bilateral pulses, scored from 0-4+. NB: 10% of the population doesn't have a dorsalis pedis pulse.</p> <p>If you can't feel pulses, use doppler US</p>	<ul style="list-style-type: none"> <li>● <b>Diffuse demarcation of ischemia</b></li> <li>● Physical findings of PVD, proximal, contralateral limbs showing diminished or absent pulses indicate chronicity</li> <li>● Often have well developed collaterals</li> </ul>	<ul style="list-style-type: none"> <li>● <b>Sudden loss of a pulse</b></li> <li>● Few physical findings of long-standing PVD</li> <li>● Proximal and contralateral Limb pulses normal</li> <li>● SHARP demarcation of Ischemia on inspection (chalky white limb distal to the clot)             <ul style="list-style-type: none"> <li>○ This may progress to cyanosis</li> <li>○ Paralysis or paresthesias = Limb threatening ischemia</li> </ul> </li> </ul> <p>Fundoscopy exam may show Roth's spots (IE) or Hollenhorst plaques (atheromatous emboli)</p> <p>PEARL: if the Limb still has sensitivity to light touch it probably still is viable</p> <p>If the limb is paralysed or with NO sensation, it may not be viable or needs STAT surgery!.</p> <p>A contracted muscle with woody hardness = irreversible ischemia.</p>
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**Additional differential of acute arterial insufficiency:**

- Phlegmasia cerulea dolens (cyanotic appearing leg, with swelling)
- Phlegmasia alba dolens
- Aortic dissection
- Acute spinal subarachnoid hemorrhage

**6) What is the management of an acutely ischemic limb?**

Usually presents with some of the 6 P's: pain, pallor, pulselessness, paresthesias, paralysis, poikilothermia (cold). See Fig. 87-1 in Rosen's.



**\*\*\*paresthesias or paralysis represents an acute limb-threatening ischemic event\*\*\***

- **These patients need immediate surgical assessment and intervention (limb salvage decreases after 4-6 hours):**
  - *If embolic disease = fogarty catheter embolectomy urgently*
    - *CT angiography or ultrasound usually prolong the limb's ischemic time!*
    - *Start IV heparin immediately!*
  - *If thrombotic disease = the in-situ thrombosis needs aggressive thrombectomy with bypass grafting, amputation, or revascularization with intra-arterial tPA or heparinization*
- In patients whom you can't differentiate between the two - angiography usually helps!

For people without these limb-threatening features, we still want to determine if it is an embolus or an in-situ thrombosis

- Emboli usually happen in people who haven't developed collateral circulation. They present with a sudden "shock" sensation to their extremity causing paralysis or an arm/leg "stroke", These patients need immediate removal of this clot. (Just like the limb-threatening scenario)
  - People with atherosclerotic disease usually have longstanding atherosclerosis and collateral circulation. They benefit from systemic heparinization and intra-arterial fibrinolysis.
- Therapies to manage peripheral arterial thrombosis can be divided into:
    - Non-invasive approaches:
      - Acute anticoagulation with heparin
        - The "Go to" for acute arterial embolism, acute arterial thrombosis, subclavian vein thrombosis (difficult to access).
        - Give 80U/kg bolus, followed by an infusion of 18u/kg/hr
        - Heparin minimizes clot propagation.
        - There may be some relative and absolute contraindications.
      - Fibrinolytic therapy:
        - Low dose tPA, often catheter directed.
        - The choice for non-limb threatening ischemia. It usually takes 6-72 hrs for the clot to lyse.
        - Most patients require subsequent secondary bypass grafting and percutaneous transluminal angioplasty.
      - Prevention: Lifestyle changes! Smoking cessation, exercise!
      - Hyperbaric therapy for chronic diabetic ischemic ulcers, skin graft salvage, etc.
    - Invasive approaches:
      - Fogarty catheter thrombectomy:
        - "Fogarty arterial embolectomy catheter is a device developed in 1961 by Dr. Thomas J. Fogarty to remove fresh emboli in the arterial system. It consists of a hollow tube with an inflatable balloon attached to its tip. The catheter is inserted into the blood vessel through a clot. The balloon is then inflated to extract it from the vessel." - Wikipedia.



- Usually used for lower extremity arterial thrombosis. (Not used in veins because of valves).
- Peripheral percutaneous transluminal angioplasty
  - Often performed with stent placement.
  - Can be performed with various recanalization devices (lasers, drills, rotating wires).
- Grafting
  - Usually autologous vein grafts are used.
  - Prosthetic grafts are used for larger vessels but have risks of: erosion into adjacent structures, infection, pseudoaneurysm, endoleaks, thrombosis, distal embolisms).
  - Often a palliative treatment.

## 7) List three disorders characterized by abnormal vasomotor response.

Abnormal vasomotor response usually occurs in the small arteries - where blood flow is controlled by local, autonomic, and humoral mechanisms. These diseases, classically, are benign and NOT associated with skin/tissue loss (however, they can cause troubling symptoms).

Because we love giving you more than just three, we'll list five, and you'll remember three!

### 1. Raynaud's disease

- See next question!

### 2. Raynaud's phenomenon

- Raynaud's disease, when there is a secondary cause:
  - i. Connective tissue disease, scleroderma, RA, SLE
  - ii. Treatment = the underlying disease.

### 3. Benign Livedo reticularis

- Similar to Raynaud's disease, but affecting the dermal arterioles of the extremities and trunk.

### 4. Acrocyanosis

- Persistent, painless, symmetrical cyanosis of the fingers, hands, feet.
- Benign disease, unlike central cyanosis.
- Supportive care: keep the body warm!

### 5. Primary erythromelalgia

- Rare. Can occur in a primary or secondary form (SLE, HTN, diabetes, PVD)
- Paroxysmal vasodilation with burning pain, increased skin temp, and redness to feet and hands.
- Attacks occur in modest ambient temperatures.
- No tissue loss occurs, but attacks can be disabling .
- No definitive treatment - supportive ice/cold baths, elevation, rest.

## 8) Describe Raynaud's Disease and how it is treated.

Remember, this is classically a vasospastic disorder with no known underlying cause.

Diagnostic criteria:



1. Precipitated episodes after cold or emotional upset
2. Bilateral symptoms
3. No or minimal gangrene (tissue loss)
4. No disease condition that could cause secondary Raynaud's phenomenon is present
5. Symptoms have occurred for at least 2 years

The classic attack is tri-phasic: Pallor (chalk white). → cyanosis → rubor/red

- 1) Complete closure of the palmar/digital arteries
- 2) Slight flow of blood
- 3) Arterial flow returns to baseline - with hyperemia

Usually has a benign course. True Raynaud's disease can be managed supportively. (occasionally CCB's are used for symptomatic relief).

## 9) What is the most common site for arterial aneurysm in the leg?

- Popliteal aneurysms
  - Often bilateral! And associated with an AAA
- Second most common = femoral aneurysms

Diagnosed with CT / ultrasound

## 1) List four potential sites for upper extremity aneurysms, and their associated underlying causes.

- Upper extremity aneurysms are a rare occurrence, unless there has been localised trauma
  - Proximal subclavian artery aneurysms
    - Thoracic outlet obstruction
    - Trauma
    - Atherosclerosis
  - Axillary artery aneurysms
    - Post-blunt trauma
    - Prolonged use of crutches
    - Anterior shoulder dislocation / humerus fracture
  - Ulnar artery aneurysm
    - Carpenters (hypothenar hammer syndrome)
  - Radial artery aneurysm
    - Post-cardiac angiography

## 11) Name three types of visceral aneurysms and their associated conditions.

- **Splenic artery aneurysm**
  - 60% of all visceral aneurysms



- More common in women than men
  - Portal hypertension, pregnancy related changes, fibrodysplasia
- Rarely symptomatic, rarely rupture.
- If it ruptures: 95% of the time it is in young women during pregnancy
  - \*rupture can be confused as an ectopic pregnancy rupture/placental abruption leading to intra-peritoneal bleeding in pregnant women\*
- Every symptomatic aneurysm needs intervention (70% risk of mortality if it ruptures in a woman of child-bearing age)
- Asymptomatic aneurysms should be coiled
- **Hepatic artery aneurysm**
  - 2nd most common type
  - Stem from atherosclerosis, infection, post-trauma, polyarteritis nodosa.
    - Usually men > 60 years old
  - Usually asymptomatic - if symptomatic usually present with symptoms similar to cholecystitis or pancreatitis.
  - High mortality rate with rupture - therefore usually should be intervened on when found
- **Superior mesenteric artery aneurysm**
  - 60% are caused by non-hemolytic streptococci from left sided infective endocarditis.
  - Other etiologies: atherosclerosis, trauma.
  - Usually a younger patient < 50 years old.
  - Symptoms = upper abdominal angina.
  - Treatment: treat infective endocarditis as applicable, difficult to coil due to bowel viability issues.

**12) List 6 differential diagnoses of an occluded indwelling catheter and describe the management of a suspected line infection.**

From Rosen’s Box 87-1:

<b>Complete occlusion - “nothing moves”</b>	<ul style="list-style-type: none"> <li>● Clot in catheter lumen</li> <li>● Precipitate in catheter lumen (crystal clog)</li> <li>● Mechanical obstruction/kink/twist</li> </ul>
<b>Withdrawal occlusion - “no suck”</b>	<ul style="list-style-type: none"> <li>● Catheter against vessel wall</li> <li>● Fibrin sheath</li> <li>● Ball valve/mural line thrombosis</li> <li>● Subclavian vein thrombosis!</li> </ul>
<b>Intermittent complete occlusion/withdrawal occlusion - “finicky line”</b>	<ul style="list-style-type: none"> <li>● Pinch off syndrome: catheter lumen is pinched by mechanical forces (first rib and clavicle/muscles)</li> </ul>

**Key points on troubleshooting a poorly functioning line:**

- 1) Get an x-ray to confirm proper placement and position
- 2) Assess for signs/symptoms of vein thrombosis (there may be no obvious symptoms)
- 3) See if changes in body position/arms/valsalva assists in flushing/withdrawal



- 4) Consider intracatheter tPA (lysis) (5000 U urokinase, clamp for 30 mins, then aspirate)

#### Management of suspected line infection:

- Think Local vs Systemic
- **Local** - signs of infection with no sepsis
  - Usually no catheter removal needed; antimicrobials usually adequate
  - Tunneled catheters may need to be removed regardless
  - Thrombophlebitis (erythema and swelling with no purulence/fever may respond to supportive care)
- **Systemic** - look for sites of infection outside the catheter and manage appropriately
  - UTI
  - Anorectal SSTI's
  - Respiratory tract
  - Catheter
    - Septic central venous thrombosis, progressing to perivascular infection or abscess
  - Two to three blood cultures:
    - At least one from a peripheral blood vessel and one from the catheter itself
  - IV Antibiotics
  - Consider CT-with contrast to look for perivascular infection/abscess
  - May need surgical excision.

### 13) What are the two types of arteriovenous (AV) fistulae used for dialysis?

- 1) Cimino-Brescia fistula
  - Preferred fistula for long term hemodialysis.
  - Usually radial artery and cephalic forearm vein anastomosed
  - Longest functional AV fistula
- 2) Prosthetic bridge fistula
  - "H"-shaped, with a prosthetic conduit connecting vein and artery.

### 14) How do you access an AV fistula?

- Ask for help! (read online/call a friend!)
- If you are the most qualified person to do it:
  - Try to choose a different peripheral venipuncture site if possible!
  - Maintain complete sterility
    - Clean hands
    - Complete sterile attire
    - Good skin prep.
    - Sterile gauze and supplies
    - **\*\*NO tourniquet allowed\*\***
      - **Access the venous side of the fistula for venipuncture**



- Apply gentle sterile pressure for 5 mins post-puncture (don't fully occlude the fistula)

### 15) What are the potential complications of AV fistula creation?

- Thrombosis
- Infection
- Steal phenomenon
  - Atherosclerotic disease distal to the shunt (e.g. the vein) leading to stealing of blood through the ulnar artery via the palmar arch - can lead to finger tip ischemia
- Venous hypertension\*\*
  - Acute venous HTN is a surgical emergency that occurs in the first few weeks post-op AV fistula creation.
  - Leads to acute, severe venous stasis in the extremity
  - Can lead to increased distal venous dilation and life threatening rupture if not recognized and treated surgically ASAP (vein ligation)
- Bleeding

### 16) List the 3 types of thoracic outlet syndrome. What are the typical symptoms of thoracic outlet syndrome? What is a simple bedside test for this condition?

The subclavian artery exits above the first cervical rib – in between two muscles (scalenus medius and anticus) and then passes under the clavicle enroute to the axilla. The brachial plexus follows this course as does the subclavian vein.

#### Types:

- Compression of the brachial plexus, subclavian vein, subclavian artery, or a combination of all at the “superior aperture of the thorax”
  - Everyone has some sort of anatomic abnormality that predisposes them to symptoms
- Categorized by predominant structure compressed: Vein/Artery/Nerve
- 95% of cases involve compression of the brachial plexus
- Usually all types occur in people aged 20-50 yrs.
- The arterial type is the most rare, but is the most serious

#### Symptoms:

- Irritation of the brachial plexus affects the lower nerve roots:
  - C8/T1: pain and paresthesias in the ulnar nerve distribution
- Upper 3 nerve roots being compressed: neck, ear, upper chest, upper back, and in the radial nerve distribution.
- Venous compression = often leads to a thrombosis of the subclavian vein producing obstructive symptoms
- Artery compression = creation of a post-stenotic aneurysm.



### Beside tests:

- **Beside tests for the arterial type are insensitive and unreliable**
  - Adson, costoclavicular, hyperabduction tests = poor
- Best screening test is the EAST
  - Elevated arm stress test
    - Patient sitting, arms in 90 deg. Abduction, elbows at 90 degrees of flexion, shoulder blades squeezed together, THEN ask the patient to slowly open and close their fists for 3 minutes.
    - You should expect the normal patient to have some fatigue
    - +ve test = early heaviness and fatigue, numbness of the hand, and progressive aching to through the arm and the top of the shoulder
      - Key finding: progressive, crescendoing symptoms that become intolerable! Symptoms should affect the hand AND the shoulder/arm.
    - The EAST assesses for all three types of thoracic outlet syndrome.
- Other test for arterial variant:
  - Blood pressures in upper arms, if > 20mmHg systolic difference in the asymptomatic limb can suggest arterial stenosis.

### Differential diagnosis and Management:

- Herniated cervical disk, cervical spondylitis, spinal cord tumors, peripheral nerve compression, MSK shoulder disease, peripheral neuropathies, MS, ETOH abuse, diabetes, and many others!
- Management depends on the severity and type:
  - Nerve compression only = physio, shoulder rehab, then maybe surgery
  - Arterial compression is a BIG deal:
    - Risk for thrombosis, thromboembolism, acute ischemia, rupture, etc.
    - Acute thrombosis = see the first few questions above.

### 1) List four anatomic abnormalities associated with thoracic outlet syndrome.

See Rosen's Fig 87-3.

Think muscle, ligament, and bone abnormalities that compress the nerves/arteries/veins in-between the clavicle and the first rib:

- Scalenus muscle / pectoralis minor muscle / subclavius muscle hypertrophy
- Costoclavicular membrane / costocoracoid ligament anomaly
- Accessory cervical rib / First rib anomaly / Long transverse process / clavicle abnormalities

The four categories:

- 1) Cervical rib syndrome = an extra rib (70% bilateral)
- 2) Scalenus-anticus syndrome = the neurovascular Bundle is pinched by the anterior scalene muscle.



- 3) Costoclavicular syndrome = shoulders moved back/down due to muscle hypertrophy/trauma
- 4) Hyperabduction syndrome = when the arms are positioned in a hyper-abducted position and the pectoralis minor muscle compresses the neurovascular Bundle.

## Wisecracks:

### 1) Describe Buerger's sign and the Ankle-Brachial Index (ABI)

#### Buerger's Sign

- Provides evidence of severe advanced arterial ischemia:
  - Place the patient supine
  - Raise their legs to 45 degrees
  - Then lift their feet > 35 cm above the right atrium.
  - IF their foot becomes pale/ chalk white = arterial insufficiency
  - IF not try these provocative tests:
    - Have them dorsiflex 5-6 times
  - Next get them to sit up with their feet hanging over the edge of the bed
    - Look for a rapid colour change from pallor → cyanosis → hyperemia
- **Ankle-Brachial Index**
  - Compares the systolic BP of the ankle with the systolic BP of the brachial artery. Ankle SBP / Brachial SBP = ABI
  - Remember that >0.9 is "a pass"
  - To measure it:
    - Have the patient supine.
    - Measure their systolic brachial BP
    - Then measure their systolic ankle BP (cuff just at the distal calf) - you'll need to use the doppler U/S to find the DP or PT pulse
    - Mild arterial insufficiency is 0.5-0.7

### 2) List the clinical criteria for Buerger's Disease (5).

It would be easier to say this is a disease from eating too many burgers....but unlike arteriosclerosis obliterans, this is a disease known as **thromboangiitis obliterans**

- It's an idiopathic inflammatory occlusive disease involving the medium-small arteries of the hands and feet.
  - Typical patient - male 20-40 years old, who smokes tobacco
  - Unknown pathogenesis
  - Often spreads from artery inflammation to inflammation of the veins/nerves
- **Clinical syndrome/criteria**
  - Foot claudication, hand claudication, fingertip/toe ulcers, Raynaud-type cold response plus
  - 1) History of smoking
  - 2) Onset before age 50
  - 3) Infrapopliteal arterial occlusive lesions



- 4) Upper limb involvement / phlebitis migrans
- 5) Absence of atherosclerotic risk factors other than smoking
- **Management**
  - Stop smoking cigarettes!
  - If they don't stop - they will eventually need amputation
  - Those with severe symptoms at risk for tissue loss:
    - Prostaglandin E1 infusion, antithrombotic/antiplatelets

### 3) What is Leriche's syndrome?

- A type of chronic arterial insufficiency - wherein there is aortoiliac occlusive atherosclerotic disease.
- Causes bilateral claudication symptoms in the buttocks/hips/thighs AND associated with impotence in men

### 4) List four types of infective aneurysms.

See Rosen's table 87-2.

#### 1) Mycotic aneurysms

- NOT caused by a fungus! Osler defined it as an infected aneurysm as a result of infectious endocarditis
- Typical causes:
  - Streptococci viridans
  - Staphylococcus aureus
- Develops via septic emboli implantation
  - Haematogenous seeding to already damaged arteries (from atherosclerosis)
  - Lodgement of septic emboli in the vasa vasorum of larger vessel walls/arterial bifurcations / AV fistula / arterial stenosis sites.
- Sites: aorta, SMA, intracranial, femoral

#### 2) Atherosclerotic arteries (most common type of infected aneurysms)

- Hematogenous seeding of bacteria to already atherosclerotic arteries
- Salmonella, E. coli, etc.
- Most common sites are LARGE vessels (aortoiliac).
- If they rupture, 75% mortality rate!

#### 3) Pre-existing aneurysms

- Occur from bacteremia, in people > 50 years of age
- **\*\*anyone with a pre-existing aneurysm and +ve blood cultures, should be treated as though they have an infected aneurysm.\*\***
- Unusual incidence
- Staphylococcus and other gram +ves
- >90% mortality rate if they rupture (usually large AAAs)
- Treatment = antibiotics for >6 weeks (or longer), and then surgical repair.

#### 4) Post-traumatic pseudoaneurysms

- Occur due to hemodynamic monitoring access, angiography, IVDU



- Femoral, carotid, brachial
- Usually patients <30 years old. Very common incidence.
- Staph. Aureus, polymicrobial
- <5% risk of mortality if it ruptures.

**5) Differentiate between arterial insufficiency ulcers and venous stasis ulcers.**

	<b>Arterial insufficiency ulcers</b>	<b>Venous insufficiency - 90% of lower extremity ulcers</b>
<b>Signs on Physical Exam</b>	<ul style="list-style-type: none"> <li>● Muscular atrophy</li> <li>● Hair loss over the toes and feet (thick toenails)</li> <li>● Shiny/scaly skin</li> <li>● Skeletonized due to atrophy of skin, fat and muscle</li> <li>● Deep penetrating ulcers</li> <li>● Ulcer base is <u>gray, yellow, black, with minimal granulation tissue</u></li> <li>● <u>Ulcer rim is sharply demarcated</u>, with no signs of cellular proliferation or epithelialization.</li> </ul>	<ul style="list-style-type: none"> <li>● Edema</li> <li>● Prominent superficial veins</li> <li>● Stasis dermatitis</li> <li>● Ulcers with a weeping base and extensive granulation tissue</li> </ul>
	<ul style="list-style-type: none"> <li>● Usually distal to the ankle - areas of trauma (toes, heels, metatarsal heads)</li> <li>● Watch for suppurative drainage (chronic osteomyelitis, infection).</li> <li>● <b>Usually painful</b>, and symptoms improve in a dependent position</li> <li>● Look for co-existent signs of chronic arterial insufficiency</li> </ul>	<ul style="list-style-type: none"> <li>● Usually proximal to the ankle - near the medial malleolus</li> <li>● Usually mildly painful, and improve with elevation</li> <li>● Usually develop more rapidly</li> </ul>