**VASCULAR SURGERY NOTES**

* **Topics to cover:**
  + ***Arterial disease***
    - Peripheral artery/vascular disease
      * Acute limb (acute ischemic limb)
      * Chronic limb ischemia
    - Aneurysms
    - Carotid artery disease
  + ***Diabetic foot***
    - Ischemic component
    - Neuropathic component
    - Infectious component
  + ***Peripheral venous disease***
    - Complications of chronic venous insufficiency
      * Varicose veins
      * Lipodermatosclerosis/stasis dermatitis
      * Venous ulcers
    - Deep vein thrombosis
  + ***Lymphatic disease***
    - Lymphedema

**ARTERIAL DISEASE**

* **General considerations**
  + Ischemia = diminished blood supply; may be acute or chronic depending on the speed of arterial occlusion
  + Effect of ischemia depends on:
    - Type of artery involved
    - Rate of occlusion (acute is more serious than chronic)
    - The state of collateral vessels (most arteries have some sort of collateral circulation, but it needs time to be efficient and if the vessels are unhealthy it may be even less efficient)
    - General condition of the patient (that big push is by the heart, so if the heart is failing, the distal circulation is already weak)
* **Acute ischemic limb**
  + Sudden interference of arterial flow to a limb
  + **Surgical emergency** 
    - LL and skeletal muscle can generally tolerate **6 hours of ischemia** before irreversible damage occurs
  + This should be on the top of your differential if a patient presents with symptoms of limb (especially LL) ischemia with signs of ischemic changes, that is of recent onset (i.e. it is **ACUTE; < 2 weeks**)
  + Etiology (most important are first 3):
    - **1= Embolism**
    - **2= Thrombosis**
    - **3= Arterial trauma**
    - Others (compartment syndrome, etc.)
  + **Embolism**
    - **Cardiac source (MC)**
      * **Atrial fibrillation-related mural thrombus** (particularly in left atrial auricle)
      * **Valvular lesions** including **bacterial endocarditis** (particularly on already abnormal valves such as in rheumatic heart disease)
      * **Prosthetic heart valves** (particularly the bovine type, when patients forget to take anti-coagulants for days)
      * **True aneurysms** (in the heart or other places)
    - Other sites:
      * **Aneurysms** (thrombus within them can embolize)
    - **Sites of embolization:**
      * Usually at sites of **bifurcation of arteries**
      * **LL > upper limb**
      * **Bifurcation of common femoral** into deep and superficial femoral arteries (40%) > aortic bifurcation > popliteal artery bifurcation > brachial artery bifurcation > common carotid artery bifurcation
  + **Thrombosis:**
    - **Atherosclerosis** 
      * **Atheromatous plaque may rupture** resulting in endothelial injury and resulting in an **overlying thrombosis** (i.e. similar to acute coronary syndrome)
      * MC site of LL atherosclerosis is **superficial femoral artery (SFA) near hunter’s canal**
    - May also occur in the setting of Beurger’s disease (vasculitis)
  + **Arterial trauma:** 
    - In the setting of a **limb trauma**, a **fracture** of a limb (particularly around the distal femur for popliteal artery injury and distal humerus for brachial artery injury) can result in arterial damage/avulsion
    - It may also result secondary to injection of IV drugs (and IA drugs) into the artery
  + Embolism vs thrombosis (not important)
    - Books will often compare both causes
    - Embolism patients may demonstrate a history of arrhythmias/AF or recent MI and the affected limb may show no signs of chronic limb ischemia; because there is no time for collaterals, loss of limb function may be present; the contralateral limb is normal (pulses felt)
    - Patients with thrombosis often have an underlying history of peripheral vascular disease (chronic ischemia) resulting in long term complaints of claudications (left unRxed), trophic factors such as hair loss and nail abnormalities; because of collaterals, there may be some function; contralateral limb has weak pulses to indicate bad peripheral vascularity
  + **Consequence:**
    - There may be **complete recovery** or **progression** of the acute limb into gangrene (wet kind) or into chronic ischemia with its resultant complications
    - In the picture of trauma, patients may develop ischemic contractures (e.g. Volkmann’s contracture in supracondylar fracture of the humerus)
* **Clinical features:**
  + ***Symptom:***
    - Sudden onset of severe lower limb pain
  + ***6Ps:***
    - **P**ain (acute onset)
    - **P**ulselessness
    - **P**allor
    - **P**aralysis/power loss (indicates impending gangrene!)
    - **P**arasthesia
    - **P**oikilothermia (Perishing cold)
  + ***P/E:***
    - **Inspection:**
      * Limb is pale early on and then becomes cyanotic/mottled later
    - **Palpation:**
      * Delayed capillary refill
      * Non-palpable distal pulses (dorsalis pedis, posterior tibial, popliteal and femoral pulses must all be examined)
      * You would also feel for tenderness and temperature (will be cold)
    - **Auscultation** (if feasible, for bruits)
  + What you should think is:
    - Patient with acute limb pain and absent distal pulses, think of acute ischemic limb!
    - Because this occurs suddenly, there is not enough time for collaterals to help supply the blocked off distal segment
    - **Ischemic necrosis and gangrene can develop rapidly**, the limb needs to be salvaged by **prompt investigation** that is relevant for **rapid management**
      * **Aseptic wet gangrene** develops in **acute limb** necrosis because the supply is cut off suddenly
      * FYI, **chronic ischemia** results in **dry gangrene** (unless infected, in which case it would be a septic wet gangrene)
* **Investigations:**
  + **Is it always indicated?**
    - In a high degree of clinical suspicion, especially in the delayed/severe cases, one might skip investigations and go directly to management, however nowadays, US can be done very rapidly
  + **Lab and imaging:**
    - Lab: CBC, Coagulation profile
    - **Imaging: Duplex, Doppler US; ECG and Echocardio**
  + Duplex and Doppler US to identify the point of blockage = BEST THING TO DO
  + Echocardiography may be done in order to identify the source of embolus; ECG to check for atrial fibrillation
  + **DO NOT DELAY MANAGEMENT WITH INVESTIGATIONS!**
* Management:
  + **General measures:**
    - IV fluids, pain management
  + **Embolism = embolectomy** 
    - Done using a **FOGARTY balloon catheter**
    - Start with a femoral arteriotomy
    - The catheter is advanced past the embolus and then the balloon is inflated
    - The catheter is then pulled out, sweeping the embolus with it
  + **Thrombosis = revascularization** (**thrombectomy or catheter-directed thrombolysis** [less severe cases])
    - Thrombolysis is achieved using a catheter whose tip is dug into the thrombus and thrombolytic drugs are given: **t-PA (alteplase)** > **streptokinase** 
      * You **can only give streptokinase once**, because patients **develop Ab against it** (it is antigenic) and patients can also **develop hypersensitivity reactions to it**
      * t-PA (tissue plasminogen activator) is associated with less bleeding risk and is not antigenic
    - **All severe cases require surgical intervention (surgical thrombectomy)**
  + **COMPLICATION OF REPERFUSION = COMPARTMENT SYNDROME** (monitor the affected LL for 4 – 6 hours!)
  + **Thrombolytic use:**
    - ***Absolute CI:***
      * Prior intracerebral hemorrhage
      * Known structural IC vascular malformation or neoplasm
      * Significant head trauma
      * Ischemic stroke within the last 3 months
      * Suspected aortic dissection
      * Active bleeding (excluding menses) or bleeding diasthesis
    - ***Relative CI:***
      * Severe uncontrolled HTN
      * Hx of ischemic stroke >3 months ago
      * Traumatic/prolonged CPR and major surgery >3 months
      * Prior streptokinase exposure
      * Pregnancy
      * Active peptic ulcer
      * Age > 75
      * DM retinopathy
  + **Additional notes on arterial injury/trauma:**
    - Causes:
      * **Penetrating** (low velocity [knives], high velocity [bullet])
      * **Blunt injuries** (RTA, fall from heights 🡪 direct injury or secondary to fractures and dislocations: suprachondylar fracture of humerus and femur)
      * **Iatrogenic injuries** (arterial cannulation, catheterization)
      * **Intra-arterial drug injection**
    - **Examination of signs:**
      * **Hard signs** = must **surgically explore** without tests
* External arterial bleeding (especially if pulsatile)
* Loss of distal pulses
* Expanding/pulsating hematoma
* 6Ps
* Palpable thrill or audible bruit at or distal to site of injury
* **Soft signs** = **urgent investigations** needed (duplex, XR)
* Small hematoma not expanding or pulsating
* Proximity of penetrating wound to major vascular structure
* Adjacent nerve injury resulting in neurological deficit
* Pre-hospital bleeding that stopped
* **CHRONIC LIMB ISCHEMIA**
  + Chronic ischemia due to **inability of arterial supply to meet cellular metabolic demands**
  + Slowly developing arterial obstruction that **gives time for collaterals to develop**, **complications develop gradually**
    - **Arterial supply of lower limb:**
      * Abdominal aorta bifurcates into the left and right **common iliac arteries**, which then divides into the **internal and external iliac arteries**; the external iliac arteries continue past the inguinal ligament to **become the common femoral artery**, which divides into the **deep and superficial femoral artery**; the superficial one continues into the popliteal fossa as the **popliteal artery**; the popliteal artery divides to give you a **posterior tibial artery** (behind medial malleolus) and **anterior tibial artery**, which gives rise to the **dorsalis pedis artery** (in between both malleoli or lateral to the extensor hallucis longus tendon)
  + **Etiology:**
    - **Atherosclerosis (MCC)**
      * Risk factors: **non**-**modifiable** (old age, male, family history) and **modifiable** (DM, HTN, hyperlipidemia, smoking, physical inactivity)
      * In DM 🡪 **ischemic component of the diabetic foot**
      * Which vessels are affected the most? **Coronaries > cerebral > carotid > lower limb vessels** > renal > SMA > UL

**Leriche syndrome:**

Aortoiliac diease affecting internal iliac arteries as well; classic **TRIAD:**

1. **Claudications in whole LL** (particularly **gluteal muscles**)
2. **Absent bilateral femoral pulses**
3. **Impotence** (IIA involvement)
   * + - **LOWER LIMB LOCATIONS:** 
         * ***Aortoiliac*** (classically Leriche syndrome)
         * ***Femoropopliteal***
         * ***Tibial***
       - Complications of atherosclerosis:
         * **Coronary heart disease** (e.g. angina, ACS), **CVA** (e.g. stroke, TIA)
         * **Chronic and acute (on top of chronic) limb** ischemia resulting in **ischemic ulcers**, **gangrene**
         * **Aneurysm** (the wall becomes weak)
   * **Symptoms:**
     + **Intermittent claudications:**
       - Pain in a **group of muscles** occurring **with exertion/exercise** and **relieved by rest** (but not immediately, **takes a few minutes**)
       - Aortoiliac disease 🡪 gluteal IC > thigh and calf IC
       - Superficial femoral 🡪 calf claudications
       - Popliteal disease 🡪 foot claudications
       - Pain is **reproducible** in that the patient develops the pain in a fixed distance (“**claudication distance**”) that over time **gradually reduces**, indicating **increased severity**
       - So, claudication distance decreases with time and the recovery time required increases as well
       - **DDx of intermittent claudications**
         * **Osteoarthrosis** (patient appears overweight, will **point to the knees**, there is always **resting background pain**; i.e. pain never really 100% goes away; **pain worse at the end of the day**; **peripheral pulses may be normal**)
         * Neurogenic claudications (sciatica and spinal stenosis; the pain classically RADIATES from the back down to the limbs; peripheral pulses are normal)
       - **When taking history,** you must ask about:
         * **Claudication distance?**
         * How it **affects lifestyle?**
         * **Occupation?** (patients with active jobs [e.g. farmers] may require more aggressive Rx than sedentary jobs)
     + **Rest pain:**
       - This represents a much **more advanced level of ischemia**, in which patient is in pain even at rest
       - Classically, **must be >2 weeks** (because if <2 weeks, consider acute limb ischemia!)
       - The pain is **classically severe enough to wake patients from sleep** (**lying down makes the pain worse**)

If someone is found to have chronic limb ischemia, especially in severe stages with rest pain and ulceration**, it would be wise to investigate for the cardiovascular system**, because there may be a high risk of MI (and CVA). In **diabetic foot patients with ischemic ulcers, no 1 = heart, but also kidney and eyes need to be checked.**

* + - * Partially relieved by **hanging their legs off the side of the bed**
      * Patients tend to adapt by **sleeping on rocking chairs** or with legs on side of bed
    - **Ischemic ulceration or gangrene:** 
      * This represents the **most severe stage** of involvement
      * **Arterial ulcers** are **punched out** (well-demarcated and deep, small), **painful**, and located in **most distal areas of foot** where there is the **least blood supply** (i.e. **tips of toes**, over the **lower leg/shin**, but also in the **heel**; there is no strict rule of course)

**Ulcer = break in continuity of the epithelium**

History Questions to ask include:

**- When did you notice it?**

- **What made you notice it?** (is it pain, bleeding, discharge?)

**- Progression** (did it change in size, shape, symptoms)

**- Does it interfere with activity?**

- **Hx of injury** or **walking barefoot**, **type of shoes worn**

- Previous history?

* + - * The **floor/base** of the ulcer is **necrotic** (dark colored, black) with **slough tissue** (vs uninfected neuropathic ulcers); this means that **healing is unlikely** to occur spontaneously, and **patients need to be Rx** using revascularization (see later)
      * They are accompanied by **trophic changes** in the skin (**hair loss, nail hyperkeratosis**) and **impalpable distal pulses**
      * Gangrene here would be **dry gangrene** (unless infected), affecting **particularly the toes** of the foot, resulting in a black mummification, which can **spontaneously amputate**; don’t get surprised to see gangrene and missing toes in these patients
    - **Stratification:**
      * ***Early:***
        + **Stage I: asymptomatic**
        + **Stage II: intermittent claudications**
      * ***Advanced*** (**critical** limb ischemia):
        + **Stage III: rest pain**
        + **Stage IV: ulceration or gangrene**
  + **Signs/Examination:**
    - General exam of patient (well or sick-looking)
    - **Inspection**
      * Exposure should be of **BOTH LL**, ideally **up to the umbilicus or groin to study the whole LL**
      * In chronic ischemia, comment on **trophic factors** (hair loss, abnormal nails, muscle atrophy), **skin color** (pale, cyanosed/mottled, erythematous if cellulitis present), gangrenous areas (comment on the stump) and any digit loss

Ulcers with necrotic/slough bases are ischemic and will not heal spontaneously. Ulcers with red (granulation tissue) bases (as in an uninfected neuropathic ulcer) are well perfused and will heal and only require patient education, good wound care and dressing, risk factor modification and precautions to be done.

* + - * **Comment on the ulcers:** **site, size, shape, depth, edges, base** (can you see granulation tissue or slough/necrosis, MSK tissue), **discharge, surrounding skin**
      * Always **look in between toes and at the heel**
      * In DM, you would **comment about deformities** (**claw foot**, contractures, Charcot joints)
    - **Palpation**
      * Ask for pain before touching
      * Feel for **temperature** (they would be cold) and compare both limbs
      * Feel for the **peripheral pulses** (all of them), in most cases, the DP and PT pulses may be impalpable while the femoral pulse would be palpable (unless Leriche)
      * You may also test for capillary refill, edema
    - **Auscultation**
      * You may find a bruit/murmur over site of blockage
    - **Special tests:**
      * **Vascular angle (Buerger’s angle)**
        + Normal patients can have their LL flexed to 90 degrees at the hip (the hip vs the bed) *without* any change in LL/toe color (remains pink)
        + Patients with PVD develop blanching of the LL/toe, the degree at which blanching occurs is known as the **vascular angle**
        + The **smaller the angle at which blanching occurs, the more severe the ischemia**
        + **30 degrees or less** represents severe ischemia
  + **Ideal investigations:**
    - The patient may have undiagnosed DM, so investigate for that (but if you’re investigating specifically for chronic limb ischemia, then US)
    - **Lab:** 
      * CBC
      * **Blood glucose, HbA1C**
      * **Lipid profile**
      * **RFTs (BUN, Cr)**
    - **Imaging/procedures:**
      * **Doppler and Duplex US**
      * **ECG**
      * Role arteriography and CTA: be careful, contrast induced nephropathy and anaphylactic reaction; not always helpful
    - **Non-invasive tools useful for stratifying:**
      * **Doppler and duplex US** (find site of blockage)
      * **Ankle brachial index (ABI)**
  + **ABI:**
    - Traditionally done using BP monitor on UL and LL
    - Using the US probe, the compressibility of the arteries provides you with a systolic BP reading
    - We compare the BP at the **ankle** to that of the **brachial artery**
    - **Normal ratio is 0.9 – 1.2**
    - In **chronic ischemia, the ABI is LOWERED** (numerator becomes lower) and this can be used to **stratify the severity of ischemia:**
      * **0.5 – 0.9** ~ **intermittent claudication** (moderate)
      * **0.3 – 0.5 ~ rest pain** (severe)
      * 0.3 and less ~ **ulceration and impending gangrene** (very severe!)
      * Others: 0.8 – 0.9 mild; 0.5 – 0.8 moderate; <0.5 severe
      * >1.2 ~ FN
    - FALSE NEGATIVE:
      * Patients with **long-standing DM** have **arterial wall calcifications** that resist BP cuff inflation, resulting in a falsely elevated BP
      * This results in a normal or high ABI, but it is NOT true
* **Conservative management (for all patients):**
  + **Patient education**
    - Patient should be aware about the cause of his condition, possible complications, need for proper RF modification (stop smoking, diet and DM control)
    - **Lifestyle modification** for chronic limb ischemia includes:
      * **Exercises** (3 times a week, every other day), patient attempts to exercise with a stopwatch until the claudication occurs, then rests and then repeats until ½ hour or 45 minutes
      * **Smoking cessation**
      * **Foot care** (**daily inspection** [have someone else look at it for you, if you have DM], **avoiding walking barefoot**, **wearing appropriate sized shoes**, **foot hygiene**)
  + **Risk factor modification (medical Rx)**
    - Tight **glycemic control**
    - **Cardiovascular disease screening and management** (i.e. control hyperlipidemia, heart disease, etc.):
      * Aspirin, statins and ACEI
* **Surgical management = Revascularization**
  + Divide into **critical** and **non-critical** ischemia:
    - **Critical = need for revascularization**, now
      * Rest pain
      * Ischemic ulcer
      * Gangrene
      * **You DO NOT debride and amputate these cases first**, because there is **no proper healing and recurrence is high**, instead, you **prepare for revascularization** (gangrenous areas **auto-amputate** themselves)
    - **Non-critical = if impacts lifestyle, consider revascularization**, otherwise **conservative management is tried first**
      * Asymptomatic patients
      * Intermittent claudications
  + **Revascularization methods:**
    - **Endovascular** technique:
      * **PCTA:** percutaneous transluminal angioplasty **with or without stenting** (balloon inflated) using a catheterization technique (similar to that of the heart)
      * The **need of using angiography** means the risk of contrast nephropathy and anaphylaxis
    - **Open surgical techniques:**
      * **Bypass grafting:** long saphenous vein is grafted to bypass the blockage in the case of femoral artery disease (in aorto-iliac, a synthetic graft is used: PTFE)
  + **Role of amputation**
    - **Spreading or massive gangrene; infected gangrene**; patient desire (most patients do not!)
    - **We generally wait for spontaneous separation in the case of dry gangrene** [in ischemic limbs] besides trying revascularization techniques (however, if it gets infected or spreads rapidly, we could opt for a major amputation)
    - Amputating at the exact site of gangrene does not help, because the area right above it will eventually get gangrene too, so find the level of arterial involvement and use that as your reference point
    - **MC indication for amputation in GCC is diabetic foot**; MC indication in developing countries = trauma
      * Other indications and causes include cancerous bone and soft tissue tumors, massive osteomyelitis, and frostbite, mines/war
      * Ligate major vessels, use an oscillating saw to cut through limb, flaps are made to cover the stump (some are left open: guillotine), when enough time has passed, a prosthesis is offered
    - Levels of lower limb amputation:
      * Toe/digits
      * Partial foot (Chopart)
      * Ankle disarticulation (Syme’s)
      * Below knee amputation (BKA)
      * Knee disarticulation
      * Above knee amputation (AKA)
      * Hip disarticulation

NOTE: most of the topics discussed in chronic limb ischemia apply to the ischemic component of the diabetic foot

**ANEURYSMS**

* **Definition:**
  + **Localized dilation of an artery** that is **>50% of its expected size** (**1.5x bigger than the normal diameter** of the given segment)
    - Normal size of aortic lumen is **about 2 cm**
  + **RF: M>F**; **old age** (>60), family history
  + May be:
    - True or false (pseudoaneurysm)
    - Fusiform (whole), saccular (part of wall) or dissecting
    - May be pathological, traumatic and congenital
* **MC site is the abdominal aorta (AAA or triple A)**
  + MC specific site is the **INFRA-RENAL abdominal aorta**
* **Causes:**
  + **Pathological:**
    - **Atherosclerosis (MCC)**
      * Common in AA and popliteal artery
    - **Collagen vascular diseases** (Ehler Danlos, Marfan, vasculitides)
    - **Septic emboli** secondary to BE and **mycotic aneurysms** (salmonella, staph)
    - Cystic median sclerosis
    - Syphilis (results in aortic root aneurysm)
  + **Traumatic:**
    - Blunt trauma
    - Note: pseudoaneurysm = hole in artery resulting in hematoma around artery whose wall becomes fibrosed and appears as though an aneurysm is present
  + **Congenital:**
    - Berry aneurysm (in circle of willis) – occurs alone, with ADPKD
* **Complications:**
  + **Rupture** (our biggest fear) – results in **fatal hemorrhage**
    - 95% of patient with AAA rupture die before reaching the hospital
    - 50% of those who do reach, die in the hospital ☹
    - **RF = SIZE,** HTN, smoking, COPD
  + Compression on adjacent structures
  + **Thromboembolism** (distally as blue toe syndrome, acute limb ischemia)
* **Clinical features/presentation:**
  + **Asymptomatic (75%)**
    - Found incidentally on imaging
  + **Symptomatic:**
    - **Acute expansion**
    - **Rupture**
    - **Mass effect**
  + **Abdominal mass**
    - Pulsatile abdominal mass (umbilical**, epigastric**) on inspection
    - Palpation:
      * Size can be approximated
      * Expansile (expanding in all directions, not just upwards)
      * Proximal pressure 🡪 reduced pulsations; distal pressure 🡪 more tense
      * Thrill maybe felt
    - Auscultation: bruit heard
  + **Rupture:**
    - Syncope, hypotension, severe pain at site (e.g. abdominal pain), palpable pulsatile abdominal mass above umbilicus (if abdominal), airway obstructive symptoms if in thorax
    - **Triad of AAA rupture:** 
      * **Hypotension**/syncope
      * **Abdominal pain** (periumbilical) or back pain
      * **Palpable, pulsatile abdominal mass**
    - **DDx =** myocardial infarction (inferior wall), acute pancreatitis, duodenal ulcer perforation, acute mesenteric ischemia +/- hepatobiliary or renal
* **Investigations (if not ruptured):**
  + **Lab:**
    - CBC, coagulation profile, RFTs
  + **Imaging:**
    - **Abdominal US**
    - **CT with contrast** (especially in patient who are stable)
    - If in ER, they do a AXR 🡪 egg-shell calcification of wall
* **Management:** 
  + **Decision to repair:** 
    - **Size**
      * **If >5.5 cm in males or >5 cm in females 🡪 must repair** (regardless of symptomatic or not)
    - **Symptomatic**
      * **If <5 cm and symptomatic 🡪 consider repair**
      * If <5 cm and asymptomatic, but larger than 50% of diameter 🡪 monitor by US yearly
    - **Rupture** (unstable patient with Hx or has triad):
      * **Straight to OT** for emergency repair
  + **Conservative management:**
    - Risk factor modification and surveillance (US)
  + **Surgical repair:**
    - **Open surgery** (laparotomy approach with graft replacement)
      * Patient with iliac involvement require a bifurcated graft (looks like pantaloons)
      * Early complications: **MI** (MCC of postop death in elective surgeries) **AKI**, **thromboembolism** from atheroma (acute limb), **colonic ischemia** (IMA is in the site of repair), anterior spinal syndrome, impotence
      * Graft infection/thrombosis/fistula (aortoenteric or aortovenous)
    - **Endovascular repair** (Endovascular aortic repair = **EVAR**)
      * Preferred, decreased procedure time, need for transfusion, length of hospitalization and recovery time
      * **Risk of endoleak** and **device failure or migration**, thrombosis
  + **AAA rupture:**
    - ABCs (collect blood for Hct, BT and Cx for PRBCs)
    - Emergency laparotomy for surgical repair

**AORTIC DISSECTION** (BRIEFLY)

* Tear in the aortic intima with blood entering in between the intima and media layer, resulting in a false lumen
* Types:
  + **Stanford classification**
    - **Type A = ascending aorta** 🡪 **surgical** management
    - **Type B = descending aorta** 🡪 **medical** +/- surgical
  + Debakey classification
    - Type I = ascending + arch + descending
    - Type II = ascending only
    - Type III = descending only (aorta after the left subclavian a)
* Causes:
  + **HTN (MCC)**
    - **Malignant HTN** as blood pressures out of heart it damages the aorta resulting in weakness between the layers
  + **CTD** (e.g. **Marfan’s, Ehler Danlos**)
  + **Arteritis** (e.g. takayasu arteritis)
  + Infections (e.g. syphilis)
* **Clinical features:**
  + Sudden onset of **severe tearing chest pain** that **radiates to the back**
    - **DDx:** acute pancreatitis, **MI**, esophageal rupture
    - The pain may run downwards as the tear progresses
  + **Asymmetrical BP and pulses between arms**
  + **Ischemic syndromes** due to occlusion of branches of aorta
    - Ascending aorta 🡪 back up to **coronaries 🡪 MI** with **new aortic regurgitation** (new diastolic murmur)
    - Carotids 🡪 ischemic stroke, horner’s
    - SMA, IMA 🡪 mesenteric ischemia
    - Renal arteries 🡪 AKI 🡪 anuria
    - Peripheral vessels 🡪 limb ischemia
  + Syncope
* Investigations:
  + Lab:
    - CBC (Hct), Cx+BT
    - Coagulation profile
    - BUN, Cr, electrolytes, BG
    - Lactate, amylase, cardiac markers
  + Imaging/procedures:
    - Ideally, if you suspect it, **CT angiography w/ contrast = GOLD STANDARD**
    - But you will most likely assume it is an MI, so you would do:
      * ECG (assume MI?): left axis deviation, LVH voltage criteria, new heart block
      * CXR may be done at any given point, and if it were done, CXR findings include a **widened mediastinum (>6 cm)**, pleural cap (pleural effusion in lung apices)
* **Management:**
  + Don’t waste too much time in investigating, as it is a life-threatening condition
  + Type A = SURGICAL; type B = MEDICAL +/- vascular
    - ABCs first
    - **Medical Rx = beta blockers** (reduce BP, as the high pressure opens up the lumen)
      * With surveillance, it may be enough for type B
      * Alternative include CCBs (nondihydropyridine) for COPD, asthmatics
    - **Type A requires emergency SURGERY:**
      * Intimal tear segment is resected, reconstitution of flow through true lumen
      * May replace affected aorta with prosthetic graft
      * **2/3rd of patients die of operative or post-operative complications** (especially since blood loss means no flow to vital organs)
      * Aortic valve replacement may be necessary

**CAROTID ARTERY DISEASE**

* Dr. Rani did not mention much about it, but it’s important in real life ☺
* **MCC = atherosclerosis**
  + RF as for all: modifiable and non-modifiable
* MC location:
  + Near **bifurcation of the common carotid artery**
* Clinical features:
  + Hx:
    - TIA (<24 hours by rule, but usually <1 hour)
      * Motor, amaurosis fugax, etc.
    - Stroke
  + P/E:
    - Bruit heard over carotid artery (does NOT correlate with degree of stenosis)
* **Investigations:**
  + **Lab:**
    - CBC
    - Coagulation profile
  + **Imaging:**
    - **Carotid duplex US**
    - **Angiography is the gold standard**, but rarely done because it is invasive
    - **CT angiography**
* Don’t forget about **ABCD2 score for TIA**
  + Risk of stroke is high if the score is high, so evaluate and manage ASAP
* **Management:**
  + **Conservative/medical**
    - **Patient education**
    - **Lifestyle modification**
    - **Risk factor modification**
      * Anti-platelet drugs (aspirin)
  + **Surgical Rx:**
    - Open: **carotid endarterectomy (Classical)**
      * Indicated if **>70% stenosed** and **symptomatic**
      * It involves **stripping away the intima layer**
      * Not recommended if actually has a stroke, stenosis is much >>>70% and if patient has many comorbidities or a short life expectancy
    - Endovascular angioplasty and stenting is not preferred

**Diabetic Foot**

* **Any patient with DM that has a lower limb disease** consisting of **3 components** that are **not mutually exclusive**:
  + **Ischemia** (chronic limb ischemia)
  + **Neuropathy (MC)**
  + **Infection**
  + **Mixed type** (any of the above in conjunction)
* Diabetic patients often develop these complications in **10 – 15 years of disease activity, often when uncontrolled**
  + If it occurs **<10 years**, suspect **non-compliance** or **late diagnosis** (patient had DM for years but it was not diagnosed)
  + So ask patients when taking history about:
    - **How long ago was the diagnosis of DM made?**
    - Whether they are **compliant or not**
    - About the **modifiable risk factors** of CV disease (e.g. smoking, hyperlipidemia, HTN, sedentary lifestyle, physical inactivity)
    - Ask about **other complications of DM/CVS**, as patients with diabetic foot often have concurrent morbidities like:
      * **Coronary artery disease** (angina, Hx of ACS)
      * **Cerebrovascular disease** (stroke)
      * **Nephropathy/chronic renal failure**
      * **Retinopathy**
    - **Ask about foot care** (this will also be discussed in the management)
      * Whether they see a podiatrist or not
      * The type of shoes they wear, foot hygiene or not
      * If they walk around barefoot; ask about past history of cuts in the feet (or if this is possible)
    - **Ask about symptoms of ischemia, neuropathy and infection**
      * **Intermittent claudication, rest pain, about the ulcer** (in detail)
      * **Loss of sensation** (they might actually tell you)
      * **Feature of local and systemic infection** (fever, redness, swelling, discharge)
* **Ischemic component:**
  + Patients with DM develop vascular changes that often classified as:
    - **Macroangiopathic** (atherosclerosis)
    - **Microangiopathic**
  + Peripheral vessels may be heavily calcified and ischemic, resulting in a picture of **chronic limb ischemia**
    - Remember, though, if patient present with acute symptoms (<2 weeks) of rest pain in a limb, you must rule out an acute limb
  + This is exactly like that of chronic limb ischemia
    - **Intermittent claudication** (ask about claudication distance and how it affects lifestyle)
    - **Rest pain** (worsens when lying down, awakens them from sleep, improve with legs dangling on side of bed)
    - **Ischemic ulcer**
      * Site is typically on **distal digits** (but also anterior shin and medial malleolus, but it’s classically for venous ulcers)
      * It is usually punched out, well demarcated, painful and has a necrotic/slough base
      * They don’t heal well on their own; don’t debride
* **Neuropathic component (MC presentation)**
  + **Three main entities:**
    - **Sensory neuropathy**
      * This manifests as gradual loss of sensation in the extremities in the **glove & stocking pattern**, with the most distal areas first
      * **The first sensation to be lost is VIBRATION**, then follows the others (**pressure, proprioception, pain and temperature**)
      * They develop **neuropathic pain** (altered sensation)
      * Patients often complain of **pins and needles** (parasthesias), numbness, **BURNING** (like **walking on hot coal**), **eventually no sensation will be felt**
      * The problem with this is that they will not be able to detect even minor and major injuries to the feet (minor injuries will go unnoticed and get complicated; they will not be able to tell if their feet is being burned or if their shoes are too tight or lose or if there is pressure)
      * They should always practice foot care (as mentioned above) IN ADDITION to visits to the HC for foot exam and **MONOFILAMENT testing** (**10 mg force** is applied when the **monofilament is bent**, and if the patient doesn’t feel this, this means they have neuropathy)
      * **Neuropathic ulcers** can develop secondary to improper foot care
        + Occur over **pressure points** (**medial and plantar aspect of big toe**, plantar foot, **heel and medial malleolus**)
        + Or over **areas of pressure by tight shoes or slippers** (which may produce **a “mirror imaging”** effect seen over BOTH lower limbs; e.g. slipper arch)
        + The pressure areas can vary depending on whether they have **deformities** (e.g. **Charcot joints, claw foot**, flat foot)
        + The ulcers are **painless**, have **slopping borders**, with the edges showing a **callus formation**, the **base is red indicating granulation tissue** (**good sign**, indicates **good perfusion** and can **heal if uninfected** or not too necrotic); there may be some necrotic tissue, **which can be debrided by the physician**
        + The distal pulses are palpable if they only have neuropathy (but concurrent ischemia is not uncommon)
        + Neuropathic ulcers usually **heal well unless secondarily infected** (signs of foul smelling discharge and toxemia should be noted) or there is concurrent severe ischemia (which needs to be addressed first)
    - **Motor neuropathy**
      * DM motor neuropathy can result in **muscle weakness**, paralysis and the **end picture is deformity**
      * The main deformity is the **claw foot** in which all the toes are curled down forming a claw (the toes will be have a much higher risk of developing ulcers)
      * Repetitive damage to joints (because of patient not feeling when they hit it) can result in an arthropathic deformity known as **charcot foot/joint**
        + Acute charcot arthropathy presents with erythema and warmth over the foot with intact skin and x-ray findings of degenerative changes
    - **Autonomic neuropathy**
      * Autonomic neuropathy in DM in general causes many symptoms (**orthostatic hypotension, ED, bladder incontinence, gastroparesis, silent MI**), but in the foot we primarily focus on the **function of the sweat gland in the skin**
      * Patients develop **drying and cracking of the skin**, **predisposing to injury and infection**
      * Patients are often told to **keep their feet moist** (but **drying in between their toes** to prevent **fungal infections**)
* **Infection & mixed type**
  + DM are more prone to infections owing to the impaired function of the immune system and their increased risk of getting injuries which may go unnoticed and untreated
  + **Infections in DM can be devastating** (some are fatal), but be aware of:
    - **Tinea pedis** (in between toes)
      * Erythema and whitish material between toes
    - **Infected ischemic or neuropathic ulcer** (mixed type)
      * The ulcer will be **dirty, discharge will be foul smelling** (can be detected even before wound is exposed)
      * Infection **can impede healing** and can erode the tissue to produce **cellulitis, foot abscess, osteomyelitis, septicemia**
    - Cellulitis
    - **Foot abscess**
    - **Osteomyelitis** 
      * Exposed bone in ulcers is treated as osteomyelitis
    - **Necrotizing fasciitis**
      * Fatal and spreads quickly, often misdiagnosed
      * When this occurs in the perineum, it is known as Fournier’s gangrene, and it is rapid spreading and fatal
  + The commonest causes of foot infections are **POLYMICROBIAL** 
    - You must empirically cover gram +ve, gram –ve and anaerobes
    - As with the general population, **MCC of osteomyelitis is staph aureus**
  + Make sure you know that the infection may be limited to the site of infection or may become systemic, so ask about:
    - **Local symptoms** (redness, warmth, swelling, discharge, pain, etc.)
    - **Systemic symptoms** (fever, chills, altered mentation, etc.)
* **EXAMINATION**
  + **General examination** (state of wellbeing)
    - Did they come in a wheelchair? Who brought them?
    - Note whether the patient is toxic/sick-looking or not
    - Vitals should be recorded (special note on fever, HR, BP)
  + **Inspection:**
    - Expose the LL, comment on obvious findings (amputation and its level)
    - **Color of limb** (pale, cyanosed; erythematous; gangrene)
    - **Trophic factors** (**hair, nails, swelling, rashes** such as **necrobiosis diabeticorum**, **dryness and cracks**, muscle atrophy)
    - **Deformities** (claw-foot or toe, deviated toes, missing toes)
    - **Between toes** for tinea pedis
    - **Inspect the whole foot** (including heel and malleoli) for ulcers, which you should comment on:
      * Site (very important)
      * Size (LxW)
      * Shape
      * Depth
      * Base (GT or slough/necrosis)
      * Edges (rolled/punched-out/slopping/over-riding
      * Discharge (color, smell)
      * Surrounding skin (callus; erythematous?)
      * Q: in what condition non-healing ulcer? Infected or heavily ischemic
    - Inspect the leg for scars of previous surgery (i.e. CABG for long saphenous vein)
    - If they have an amputation, describe the stump (like an ulcer)
  + **Palpation**
    - Ask for pain first, palpate for **peripheral pulses** (impalpable in ischemia, but find where it is palpable to assess; femoral pulses should be palpable if it is femoropopliteal involvement), **temperature** (cold = ischemic; hot = infected), **tenderness**, **capillary refill** (should be <2 seconds), **edema** and masses
  + **Auscultation over arteries** (for bruits)
  + Special tests:
    - **Buerger’s vascular angle**
    - **Monofilament testing for sensation**
    - Ophthalmoscopy for retinopathy
    - **Ankle-brachial index (ABI)**
* **Investigations:**
  + Most patients do not require a referral to the ER (family physicians should be able to manage) unless:
    - **Severe infections are suspected** (significant cellulitis, suspected foot abscess, signs of toxemia)
    - **Infected ulcer** with foul smelling discharge
    - **Signs of critical ischemia** (rest pain, ischemic ulcer and gangrene)
  + Investigations (if in ER and requires admission):
    - **Lab:**
      * CBC (look for leukocytosis)
      * RBG and HbA1C
      * BUN&Cr, GFR, electrolytes (assess kidney function) + urinalysis
      * If appears toxic, blood culture & blood gases
      * Lipid profile (LDL, HDL, cholesterol)
      * **If discharge: swab C&S** (will take 3 or more days, so empirical antibiotics are indicated)
    - Imaging/procedures:
      * **Duplex and Doppler US**
      * **Foot imaging** (MRI is the best; don’t forget osteomyelitis may not be obvious until 7 – 10 days; R/O abscess, study charcot joint)
      * **ECG** (cardiovascular screen)
* **Management (stable patients):**
  + **1 = Patient education**
    - **Complications of DM**
    - **Importance of compliance** and **risk factor modification** (i.e. take **medications, stop smoking, exercise**, F/U)
    - **Daily foot inspection, hygiene, foot care**, etc.
      * **Inspect between toes** for fungal infection
      * **Ask someone else to inspect it for them** because DM patients may have retinopathy and not see properly!
      * **Moisten the leg** (because of dryness and scaling) but keep the **space between the toes try**
      * **Don’t wear tight shoes**, **don’t walk barefoot**
      * **Special shoes** for neuropathic ulcers (**off-loading shoes**/cushions)
  + **2 = Risk factor modification**
    - Stop smoking, exercise until claudication distance (see up)
    - Rx for hyperlipidemia, cardiovascular (coronary artery disease, HTN) and renal disease (e.g. aspirin, statins, ACEI)
  + **3 = specific management:**
    - In all cases, 1&2 is done
    - **Ischemic component:**
      * Non-critical 🡪 1 & 2 (above) is enough, emphasis on special exercises
      * Critical (rest pain, ischemic ulcer, gangrene; ABI <0.5) 🡪 **REVASCULARIZATION**
      * **Don’t amputate or debride**, it doesn’t help
      * Revascularization can be open surgical (bypass grafting) or endovascular (PTCA w/ or w/o stenting)
    - **Neuropathic component**:
      * Ulcers are **debrided carefully and dressing** is done periodically **until they heal**
      * They are asked to wear **OFF-LOADING shoes** and pads which prevent contact with surfaces that can promote neuropathic pressure ulcers from forming and healing
      * Their feet are assessed for the pressure points, and pads/shoes are made specifically for them
      * **Medications may be used for neuropathic pain**
    - **Infection:**
      * Broad spectrum antibiotics (cefuroxime + metronidazole) are given empirically until C&S results are obtained
      * **PO if local infection**; **IV if aggressive local infection and osteomyelitis** and if there are systemic symptoms (toxic patient)
      * Foot abscess 🡪 incision and drainage required
      * Necrotizing fasciitis 🡪 surgical debridement
  + If doctors ask you what else do you want to evaluate if you are presented with a DM patient with a diabetic foot?
    - Coronary heart disease > nephropathy > retinopathy etc

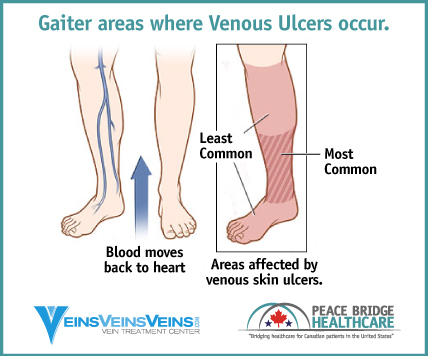
**PERIPHERAL VENOUS DISEASE**

* **Veins of the lower limb**
  + 3 sets of veins you should know about:
    - **Superficial veins**
      * They are **SQ veins** that lie superficial to the deep fascia
      * They become engorged in varicose veins, and their tributaries are evident in telangiectasias of the LL
      * Consists of the long saphenous vein and short saphenous vein
      * **Long saphenous vein**
        + **Longest vein in the body**
        + Runs on the **MEDIAL side of the LL**
        + Starts as the **continuation of the medial marginal vein of the foot**
        + Runs upwards and **ANTERIOR to the medial malleolus** (don’t confuse it with the posterior tibial artery which runs POSTERIOR to the medial malleolus and this is where you’d feel the pulse!)
        + **Runs on medial aspect of leg** and then **1 hand-breadth medial to the patella (landmark!)**
        + It then joins the **femoral vein** at the **saphenofemoral junction (SFJ)**, which is 3.5 cm below and lateral to the pubic tubercle (the pubic tubercle is felt by feeling the thigh adducted and follow the adductor longus muscle up)
        + The **saphenous NERVE** runs closely with the saphenous vein **in the LEG**, but separate in the THIGH (this is the **basis of stripping of the long vein down to the knee and not beyond**)
      * **Short saphenous vein**
        + Originate in the **lateral side** of the dorsal venous arch and continue on the **lateral side of the foot**
        + It runs **BEHIND the lateral malleolus**
        + Runs on the midline on posterior aspect of the leg and **drains into the POPLITEAL VEIN**
        + It is followed closely by the **SURAL NERVE**
    - **Deep veins of the LL**
      * They are named along with their arterial counterpart and run next to each other; **femoral vein, popliteal vein, posterior tibial vein**
      * They have **unidirectional valves** that help propagate venous blood upwards
      * They lie within the **deep fascial compartment** and are surrounded by **muscles and strong arterial pulsations**, which all **aid in venous return**
    - **Perforator veins**
      * There are set of veins that connect the superficial vein to the deep veins
      * They also **contain valves** that also help **prevent reflux of blood into the superficial veins**
      * There are **3 main perforator veins** (6, 12, 18 cm from sole of foot)
      * Damage to the valves of these veins (by **recurrent DVT** or high backpressure related to other risk factors) is a **prime factor in producing varicose veins**
    - **FACTORS THAT HELP VENOUS RETURN FROM LOWER LIMBS:**
      * **Muscle pump** (especially the gastrocnemius **“2nd heart”**)
      * **Transmitted arterial pulsations** to the deep veins
      * **Unidirectional valves**
      * **Negative intrathoracic pressure**
* **Disease of the venous system** in the lower limb can be classified into:
  + **Superficial vein problems:**
    - Telangiectasia
    - Varicose veins
    - **Superficial thrombophlebitis**
    - **Lipodermatosclerosis (stasis dermatitis)**
    - **Venous ulcers** and bleeding
  + **Deep vein thrombosis**
* Risk factors of **venous thrombosis = VIRCHOW triad**
  + **1 = venous stasis** 
    - **Prolonged immobilization**
      * Bed-bound (old age)
      * Pelvic and hip fractures
      * Peri-operative
      * Pregnancy
      * Intra-abdominal tumors (or high IAP)
      * Prolonged airplane or car rides
    - Varicose veins
  + **2 = endothelial injury**
    - Trauma to vein wall (**pelvic operations** [especially gynecological and obstetric operations], accidents)
    - Atherosclerosis
    - **Prosthetic valves**
    - Venepunctures/cannulation/**indwelling catheters** (YES! Especially central lines!)
    - Inflammatory processes
  + **3 = hypercoagulability or turbulence**
    - ***Thrombophilias***
      * Protein C&S deficiency
      * Factor 5 leiden
      * Polycythemia, MM
      * Malignancies
      * Pregnancy (post-partum up to 6 weeks)
      * Collagen vascular diseases
    - Turbulence in AV fistula
    - Medications (e.g. **OCP**s, tamoxifen)
* **Chronic Venous insufficiency**
  + Includes:
    - Varicose veins دوالي
    - Telangiectasias
    - Lipodermatosclerosis (stasis dermatitis)
    - Superficial thrombophlebitis
    - Venous ulcers
  + **Special considerations: varicose veins**
    - **Dilated, tortuous** superficial veins
    - Causes:
      * **PRIMARY VV:**
        + ***Idiopathic*** (theories postulate either weak wall or congenital valvular incompetence that manifests later in life when compounded by other factors)
        + Age: **20s – 30s**
        + **Female > males** (probably because females seek medical attention for cosmetic reasons + pregnancy)
        + **OCCUPATION: jobs that involve standing** (shawarma cooks, cafeteria workers)
        + You **MUST** ask about occupation in VV!
        + Other factors:

**High parity**

**Marked obesity**

**Use of OCPs**

* + - **SECONDARY VV:**
      * **Past DVTs** (**damage to perforator veins** from high pressure)
      * **Pregnancy**
      * **Vascular malformation** (AV fistula or purely venous)
      * **Pelvic tumors/large masses** (large fibroid, ovarian cyst/tumor, IVC compression by retroperitoneal mass)
      * **Congenital** (**Klippel-Trenalaundy**)
  + **SITE:**
    - Varicose veins can happen virtually anywhere in the LL
    - Spectrum of CHRONIC VENOUS INSUFFICIENCY prefers the GAITER area
    - The **GAITER area** includes the **ankle and lower leg**, particularly on the **medial aspect**, **near the medial malleolus**
    - If you see the spectrum occurring on LATERAL side of leg, think of secondary VV and Klippel
  + **Presentation:**
    - **Obvious dilated veins** (cosmetic reasons)
      * Early on can be visible under skin as **telangiectasia**
    - **Pain**
      * **Dull, dragging/heaviness/discomfort in LL**, particularly around calf muscles
      * **Worse at the end of the day**
      * **Better when lying down,** **raising legs and with walking** (opposite of chronic ischemia)
      * **BIG DDx:** is this chronic ischemia? DOES NOT worsen when lying down, and does NOT awaken patient from sleep
    - **Bleeding**
      * The overlying skin may **ulcerate and bleed** (appropriate thing to do is to apply pressure/compression and wrap)
    - **RASH: Lipodermatosclerosis** (**stasis dermatitis**)
      * **Eczematous rash** over the **gaiter area** that is **hyperpigmented/brown**
      * Represents **hemosiderin deposits** in the skin from **extravasated blood**
      * The patient complains of itchiness and pigmentation
    - **Superficial thrombophlebitis**
      * Thrombosis and inflammation of the superficial veins
      * Present with **pain and redness over the affected vein**
      * There is a **palpable tender cord like structure** over affected area
      * Typically a **self-limiting/benign disorder**
      * However, migrating thrombophlebitis can be a feature of pancreatic cancer known as Trosseau syndrome
    - **Venous ulcer**
      * Always compared with ischemic ulcer, because they must be distinguished for Rx (if you put compression stockings for a person with ischemia, good luck…)
      * They occur over the **gaiter area**, tend to be **larger** and **more irregular** in shape, **shallow**, with **slopping borders**
      * They are much more wet and will probably have a **GT floor** with variegations
      * They **can be painful**, but **not as painful as an ischemic ulcer**
      * The **surrounding skin may show signs of stasis** **dermatitis**, telangiectasia and varicose veins as well as edema
* **P/E:**
  + General exam
  + **Inspection**
    - Optimal position = STANDING
    - The varicose veins classically reduce in size when lying down
    - It classically occurs over the gaiter area (medial lower leg)
    - Comment on **leg skin color, trophic factors, deformities, ulcers** as in diabetic foot
    - Leg is described to look like an **inverted champagne bottle**
  + **Palpation:**
    - Temperature, tenderness, peripheral pulses, edema
    - If there is erythema, tenderness and a palpable cord: superficial thrombophlebitis
    - If you feel a thrill 🡪 possible AVM or AV fistula
  + **Auscultation**
    - Bruit (AVM/AV fistula)
  + **Special tests**
    - **Trendelenburg tourniquet test** for **varicose veins**
    - Aim of this test is to **identify the level of valvular insufficiency**
    - The patient **is lying down with the leg raised to drain out the veins**
    - **A tourniquet is applied to the SFJ** and the **patient is told to stand**
    - **Two possible outcomes:**
      * **No refilling:** the insufficiency is at the level of SFJ or above
      * **Filling:** the insufficiency is below the level of the SFJ
        + You will need to repeat it at the possible levels of the perforator to find out which is insufficient
  + **Investigations:**
    - For DVT (possibly):
      * Duplex and doppler US
  + **Management**
    - **1 = patient education (all cases)**
      * Patient should know that the condition can be related to their occupation
      * They should **avoid long standing** and **try to raise their legs up whenever possible**
      * Use of **graduated compression stockings** is recommended
      * That once it is present, it is difficult to reverse the process, but easy to prevent it from progressing
    - **Telangiectasia** (cosmetic reasons) **🡪 sclerotherapy** 
      * Sclerotherapy involves injecting a sclerosant that will **damage the endothelial wall of the vein and cause it to collapse**
      * ADR: if sclerosant goes under the skin, it can result in **skin damage and sloughing** (which is undesirable); **rarely: DVT** if it gets into the deep veins
    - **Lipodermatosclerosis** 🡪 1 + surgical options
      * **SURGICAL MANAGEMENT INVOLVES:**
        + **Long saphenous vein is ligated at the SFJ**
        + **The vein is removed by STRIPPING** down to **the KNEE LEVEL** (Why? We don’t remove it completely because of **possible injury to the saphenous nerve in the leg**, where it runs close to the long saphenous vein)
        + **Avulsion of the vein**
      * **Endovenous laser ablation therapy (EVLT)** under US guidance
        + Positive = avoid groin incision and stripping
      * For patients with short saphenous vein disease, we do a ligation at the saphenopopliteal junction **WITHOUT stripping of the vein** (to **prevent injury to the sural nerve**)
    - **Venous ulcers**
      * BEFORE TREATING, **MAKE SURE YOU EXCLUDE**:
        + **Diabetic foot**
        + **Ischemic ulcer**
        + **DVT**
      * **Dressing, graduated compression therapy**
        + Compression therapy involves **graduated layers of cloth is wrapped in sequential order** (most layers in the distally, least layer proximally) to **promote venous return**
        + We have the **4 layer graduated compression** (done at the clinic) or patients can be told to wear **Una boots**
        + The ulcer takes 4 weeks or more to heal completely
      * After, they **may continue conservative Rx** or **do the above surgery if patient wills**
* **Deep vein thrombosis (DVT)**
  + RF
    - Basically virchow triad, but we highlight:

Things I need to know about my patient with LL swelling:

Is it **unilateral or bilateral**? Is it anywhere else?

Is there **pain or skin changes**?

**ROM ok or not?**

**Any history of trauma** or sport event?

**Is she pregnant?** If not, is she on **OCPs**?

Is he/she on any drugs?

Does he/she have any **medical conditions** that can cause edema?

**Recent surgery or immobilizing situation** (long flight)?

Any history of bites?

**If young, any family history?**

---

PE: is it pitting or not?

* + - * **Immobility** (peri-operative)
      * **Certain operations** (gynecological, obstetric, colorectal, orthopedic surgery)
      * **History of previous DVTs** (VERY IMPORTANT)
      * **Puerperal** (up to 6 weeks post-partum)
      * **Malignancies**
      * **Certain fractures** (especially pelvic/hip)
      * **Hypercoagulable states** (Thrombophilias, CTD, APL)
      * **OCP use**
  + Occur in 25 – 50% of surgical patients and many non-surgical patients
    - All inpatients must be assessed for DVT risk and offered prophylaxis if appropriate
  + **Presentation:**
    - **Majority occur silently** (i.e. asymptomatic occurrence)
      * May pass unnoticed until complications develop
    - Some present with the **classical symptoms and sign of DVT**
      * Symptoms include PAIN, REDNESS, SWELLING in the leg
      * The hallmark symptom is **acute unilateral limb swelling** (1 – 3 days history)
        + Patients can have limb pain and redness
      * SIGNS include ERYTHEMA, WARMTH, TENDERNESS and SWELLING (pitting edema); **patients may have low-grade fever**
      * Pain is described as **aching discomfort or tightness**, usually in the involved calf or thigh, **aggravated by movement**
      * **Swelling occurs depending on the involved vein;** popliteal vein thrombosis 🡪 foot and ankle; femoral thrombosis 🡪 calf and lower leg; ilio-femoral thrombosis 🡪 whole lower limb
      * Tenderness is felt on palpation of the calf (or thigh)
      * **HOMAN’S SIGN (UNRELIABLE):** dorsiflexion of the foot results in pain in the affected area, causing the patient to resist dorsiflexion
    - **Some present with the complications of thromboembolism** (i.e. **pulmonary embolism**; chronic venous insufficiency and if severe, phlegmasia dolens alba)
      * PE 🡪 dyspnea, chest pain, hemoptysis, tachypnea and tachycardia
  + **DDx according to Hx & PE:**
    - **Baker’s cyst rupture**
      * Patient may have a history of arthritis or a mass behind the knee
      * There is particular pain, bruising and a palpable swelling in the **posterior knee**
    - **Cellulitis (and deeper infections)**
      * Very difficult to distinguish from DVT (since it is also acute)
      * You Rx based on your clinical suspicion of DVT
      * If a fever is present, I assume it would be of a higher grade vs DVT (fever can be present in DVT)
      * If the pain is very severe (out of proportion) and signs grow too rapidly, please consider necrotizing fasciitis
    - **Lymphedema**
      * Often **CHRONIC**, large, **involves the dorsum of the foot** and toes
      * **Non-pitting edema**
    - **Tendon strain/tear/rupture** (E.g. Achilles tendon)
      * Inciting injury or trigger in Hx; bruising at the ankle
    - **Insect bites**
      * Hx of bite or going to the woods or whatever
    - If following trauma, consider fracture and compartment syndrome
  + **WELL’S SCORE**
    - There is one for DVT alone and one for PE
    - The one for DVT is **1 point for any of the below**:
      * **Active cancer**
      * **Immobilization** of lower extremity
      * **Recently bedridden** for 3 or more days or **recent surgery within the last 3 months**
      * **Localized tenderness** along the distribution of the deep vein system
      * **Entire leg is swollen**
      * Calf swelling at least 3 cm larger than the asymptomatic side
      * **PITTING edema** confined to the symptomatic leg
      * Collateral superficial veins
      * **Previously documented DVT**
    - If alternative Dx is at least as likely as, REMOVE 2 points
    - **The simplified version is:** 
      * **<2 points = unlikely** (**D-dimer can be used** to R/O, do US if high)
      * **2 or more points = likely (go directly to US)**
  + **Investigations:**
    - **Lab:**
      * **D-dimer:**
        + If **normal, you essentially R/O DVT**
        + SENSITIVE (SNOUT: rules out) but NOT specific (if raised, you don’t know if it’s from DVT or something else: pregnancy, malignancy, infection, recent surgery)
        + If **raised, proceed to imaging (US)**
        + If **normal, think of other causes**
      * **Coagulation profile** (**baseline monitoring for Rx**)
      * Others based on your suspicion of cause or you’re not sure of what it is (thrombophilia screen if young patient or has family history; labs for possible malignancies)
    - **Imaging:**
      * **Venous ultrasound (duplex, doppler)**
        + Findings include a **dilated affected vein** that is **non-compressible**, poor blood flow, echogenic material in lumen, poor augmentation with distal compression of vein
      * The classical **GOLD standard is VENOGRAPHY**
      * Alternatives include CT venography
  + **Management:**
    - **Objectives:**
      * **Prevention of new thrombi**
      * **Prevent PE**
      * To **minimize venous valve damage**
      * Note: our goal is NOT to dissolve the clot, but nowadays there may be a role in doing so with thrombolytics
    - **Medical Rx:**
      * If bed resting then with **LL elevation** (reduces edema and pain), but promote **early ambulation**
      * **Graduated compression stockings** (debate on whether it can help with something called post-phlebitic syndrome)
      * **Anticoagulation:**
        + **HEPARIN:**

Classically **IV unfractioned heparin**

**Works in 3 minutes** (immediately, but not instantly)

**Takes 4-6 hours to PEAK**

**Needs APTT monitoring** (every 4 – 6 hours)

**Goal is 2x baseline APTT** (so if baseline is 30 s, goal is 60 s)

**Begun with warfarin** because warfarin takes 3 days to work and because initially warfarin is pro-thrombotic (“bridge therapy”)

**Heparin may be stopped at 3 days when INR is therapeutic**

**LMWH** **SQ**

E.g. Enoxaprin

Lower risk of bleeding

No need for APTT monitoring

Patient can do it at home

**Direct factor Xa inhibitor, SQ**

E.g. Fondaparinux

**It is SAFE in pregnancy**

**ADR of heparin:**

***Bleeding risk*** (intra-cerebral bleeding is our major concern)

Heparin induced ***thrombocytopenia (HIT)*** – they have risk of **THROMBOSIS** (because actually the thrombi are being used up!)

Antidote = **protamine sulfate**

* + - * + **Long-term anticoagulation** (classically oral warfarin)

**Oral warfarin**

Given immediately with heparin as bridge therapy: it is **pro-thrombotic initially for 48 hours** (will cause **skin necrosis**)

**Takes about 3 days to peak** (because of its MOA)

MOA = blocks vitamin K epoxide reductase in liver in producing vitamin K dependent coagulation **factors 2, 7, 9, 10** (but also C &S, which have the shortest half life and are the first be affected, hence the initial prothrombotic state)

Needs **INR monitoring** beginning on **day 3**

Goal is 2x normal (so INR 1 is normal, **INR 2-3 is goal**)

Duration is about **3 – 6 months** depending on patient risk

**ADR of warfarin:**

***Bleeding risk***

***Teratogenic*** (CI in pregnancy)

Reversal agent:

Immediate: FFP (e.g. pre-OP reversal)

Vitamin K

**Direct thrombin (IIa) inhibitors**

E.g. Dabigatran

NO need for INR monitoring

* + - * **Considerations:**
        + **Only use heparin in pregnancy** for short and long term (warfarin is avoided)
        + If has massive disease**, catheter directed thrombolytic therapy may be considered**
        + Avoid ANY forms of heparin if patient develops HIT
        + **Indications of IVC filter:**

***CI*** to anticoagulation

***Complicated*** anticoagulation (e.g. developed IC bleeding)

***Refractory*** to anticoagulation

* + **Complications of DVT**:
    - Pulmonary embolism
    - Recurrent DVTs
    - Chronic venous insufficiency
    - **Prevention/prophylaxis:**
      * ***Pre-OP:***
        + **Smoking cessation**
      * ***Intra-OP:***
        + If **high risk**, **SQ heparin** (LMWH or others)

Elderly patient, long operation, history of DVT, on OCP or pregnant

* + - * + **Pneumatic compression device (PCD)**
      * ***Post-OP:***
        + **Early mobilization**
        + Good post-op hydration
        + **Graduated compression stockings (GCS)**
      * ***Out of hospital:***
        + **Patient education:**

**Avoid prolong immobilization** or get up every once in a while

Promote exercise

* + - * + **Compression stockings**
    - Additional information that is good to know:
      * Upper limb DVT?
        + Axillary-subclavian DVT occurs with central venous catheters, TPN and IV chemotherapy

**LYMPHATIC DISORDERS**

* **Lymphedema = chronic lymphatic obstruction** 
  + Raised **interstitial fluid** that **drains poorly**
    - The job of the lymphatic system is to **return all the fluid materials that has crossed the capillary membranes into the interstitial space back into the venous circulation** (through the thoracic duct > right lymphatic duct); it also propagates foreign substances, materials to lymph node to **mount an appropriate immune response**; chylomicrons also travel in lymphatic system to get to the systemic circulation
  + Lymphedema causes **massive LL swelling that is non-pitting** (vs edema) and **involves the dorsum of the foot** (dorsal hump and squaring of the toes) unlike DVT
  + Causes
    - **PRIMARY lymphedema = congenital lymphedema**
      * May present at birth or infancy = **lymphedema congenital**
      * May present at adolescence = **lymphedema precox**
      * May present in adulthood (mid 30s) = **lymphedema tarda**
    - **SECONDARY lymphedema**
      * **IATROGENIC (MCC)**:
        + **Axillary clearance** in **breast surgery** (axillary LN dissection)
        + **RADIOTHERAPY**
      * **INFECTION – filariasis** (by nematodes: wucheria bancrofti and brugia malayi) and TB
      * TUMORS (metastasis and lymphomas)
  + **Pathological changes:**
    - Swelling (accumulation of fluid (pitting), then fibrous tissue replacing SQ fat (non-pitting)) 🡪 **skin changes** (skin thickening, hyperkeratosis, vesicles) 🡪 **bulges** (elephantiasis)
  + **Presentation:**
    - In secondary lymphedema, a big DDx is DVT, however, lymphedema is:
      * **Longer in course** (takes weeks to months to become very obvious)
      * **Involves the dorsum of the foot**
  + **Investigations:**
    - Lab:
      * For filariasis: midnight blood film/peripheral blood smear for micofilaria, intradermal skin testing
    - Imaging:
      * **Lymphoscintigraphy**
  + **Management:**
    - **1 = patient education and conservative steps**
      * If primary, that it is congenital and will take a lot of effort to figure out
      * **Raising legs and exercise helps**
    - Primary lymphedema = 1 + **compressive therapy** to aid in drainage
    - Secondary lymphedema = Rx 1+ Rx underlying cause + compressive therapy
      * **Filariasis Rx = diethylcarbamazine**
    - Some operative techniques have been done, but don’t bother yourself with that
  + **Complications**
    - **Disfiguring**, really affects lifestyle ☹
    - **Recurrent infections** (cellulitis, lymphangitis, lymphadenitis)
      * The stagnant lymph acts as a good infection medium
    - Chronic lymphedema 🡪 **LYMPHANGIOSARCOMA**
      * In this case, called **STEWART-TREVES syndrome**