Arterial Disorders

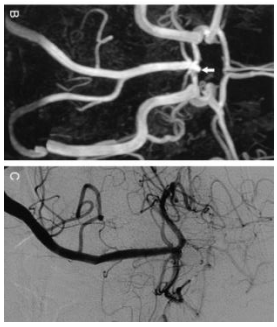
Arterial Ischemia

- Definition: arterial supply is not enough to meet tissue requirements.
- Types:
  - **Acute or Chronic:** according to speed of arterial flow reduction
  - The effects of ischemia depend upon:
    1. The type of artery:
       - e.g., The subclavian artery very efficient collateral circulation.
       - The popliteal artery has poor collateral circulation
    2. The rate of occlusion of the artery:
       - Acute ischemia is much more serious than chronic ischemia as there is no enough time for collateral circulation to open.
    3. The state of the collateral vessels:
       - Healthy collateral vessels can compensate to some extent the ill effects of ischemia.
    4. The general condition of the patient:
       - presence of myocardial insufficiency or severe anemia exacerbate effects of ischemia.

✓ Arteries are characterized by the presence of collateral circulation, which is normally collapsed. Circulation opens whenever the blood flow in the main artery is inadequate for the tissue requirements.

- Vascular Laboratory:
  - **Doppler ultrasound:**
    - Noninvasive
    - Painless
    - Portable
    - inexpensive
  - **Multidetector computed tomographic angiography: (CTA)**
    - Done by injecting an iodine rich contrast material through an arm vein (not an artery) and so it is less invasive than catheter angiography. urea and creatinine must be estimated because the contrast material is **nephrotoxic**.

  - **Magnetic resonance angiography (MRA):**
    - MRA can visualize blood vessels **without** injection of any contrast.
  - **Digital subtraction arteriography (DSA):**
    - allows visualization of arterial tree **without** arterial cannulation to avoid its possible side effects
    - An x-ray picture is taken & image is introduced to the computer.



  - **Direct arteriography:**
    - **Invasive** only performed in need for endovascular procedures

a) Direct femoral arteriography:
   - direct puncture of the artery is done and injection of the contrast material will allow visualization of the whole arterial tree.
   - This technique is losing popularity nowadays in favor of the more informative aortography.

b) Trans-femoral aortography:
   - A cannula is introduced into the femoral artery (the pulse is felt on this side), through it a guide wire is introduced. The cannula is removed and then a long catheter is introduced along the guide wire till it reaches the desired level and the contrast material is injected **(Seldinger technique)**.
   - Trans-axillary aortography is done in occluded whole distal aorta
   - Trans-lumbar aortography by direct aorta puncture is no longer done

Acute Limb Ischemia

- Definition: sudden interruption of the flow to a limb → limb loss or death.
- Etiology:
  1. Arterial embolism
  2. Acute arterial thrombosis
  3. Arterial trauma
  4. Aortic dissection
  5. External compression, e.g., tight tourniquet
  6. Compartment syndrome
  7. Extensive iliofemoral DVT.

Arterial embolism

- Definition: Embolus is originally a Greek word that means a plug or stopper. Embolism is the passage of a matter from one part of the circulation to another through a vascular lumen.
- Sources:
  A. **Cardiac emboli:** (about **80-90**%) secondary to:
     - **Atrial fibrillation:** is the commonest cause as it commonly results in stasis and the formation of a left atrial thrombus.
     - **Myocardial infarction:** → formation of a mural thrombosis.
     - **Left ventricular aneurysm**, **Endocarditis** on top of rheumatic or congenital heart diseases.

  B. **non-cardiac emboli:** (about **20%**):
     - Extensive atherosclerotic lesions or aneurysms of major arteries as aortic arch or descending aorta
- Sites:
  1) Common Femoral Artery bifurcation 40%
  2) Aortic bifurcation (saddle embolus).
  3) Popliteal Artery bifurcation.
  4) Brachial Artery bifurcation.
  5) Common Carotid Artery bifurcation.

Acute Arterial Thrombosis

- Etiology:
  - Atherosclerotic obstruction: Acute thrombotic occlusion on top of atherosclerotic narrowing is the most common cause.
  - Pump failure: sudden ↓↓ in cardiac output → acute limb ischemia in patients with extensive atherosclerotic peripheral vascular disease.
  - Hypercoagulable states: usually in small arteries.
  - Febrile illness: or gastroenteritis especially in children may lead to hemoconcentration with subsequent thrombosis.
  - Acute graft thrombosis: may occur early due to a technical factor or late due to progression of the original disease.
  - Inadvertent intra-arterial injection: which may occur in addicts causes intensive vasospasm followed by thrombosis.
- Pathophysiology:
  i. Propagation of thrombus: After circulatory arrest, widespread distal intravascular thrombosis occurs (Collateral circulation also markedly impaired)
  ii. Associated venous thrombosis: This is due to a combination of sluggish flow and ischemic injury to the intima of the involved veins.
  iii. Compartment syndrome: ischemic muscles get swollen, which in turn exaggerates the effects of ischemia.
    - After revascularization more edema of muscles occurs →more circulatory interruption (can be avoided by **early fasciotomy**).
  iv. Systemic and metabolic sequalae:
    - Muscle ischemia leads to rhabdomyolysis with liberation of potassium, lactic acid and myoglobin. The sudden release of these metabolites into the systemic circulation will lead to **"reperfusion syndrome"** characterized by metabolic acidosis, hyperkalemia & myoglobinuria
    - Myoglobinuria and acidic urine lead to acute renal failure.
- Clinical picture: 6 p's
  - Symptoms:
    a) Pain: is usually the first symptom.
    b) Paresthesia.
    c) Paralysis: due to ischemic myopathy more than nerve hypoperfusion (function of intrinsic muscles of the foot is lost 1st) If ischemia continues, patients develop calf tenderness due to swelling of gastrocnemius & soleus muscles → indicates muscle infarction.
  - Signs:
    a) Pallor: The color of skin is marble white with absent capillary refill. The veins recollapsed and there is venous guttering. The leg may even appear cadaveric. Fixed mottling and fixed cyanosed or purple areas of skin that fail to blanch on press.
    b) Pulselessness is the absolute prerequisite of acute ischemia.
    c) Poikilothermia (coldness)

✓ **sensitivity of various tissues to ischemia is variable**

1. Sensory nerves responsible for light touch & proprioception are very sensitive followed by fibers for pain, temp. & pressure.
2. Motor nerves →muscle weakness.
3. The skin. At 1ST pale, then dusky blue due to capillary V.D. lastly extravasation of blood due to capillary disintegration skin isn't viable

❖ Types:

Types	Embolic	Thrombotic
History	Arrhythmia	Claudication
Source emboli	Present	Absent
Radial pulse	Irregular (A.F.)	Regular
Skin color	White	Dusky
Limb nutrition	Normal	Pic. Chronic ischemia
Angiography	Sharp cut-off	Tapering stenosis
Collaterals	Minimal	Extensive

✓ **Limb Viability:**

a. Viable: No sensory loss or muscle weakness, with audible arterial & venous Doppler signal
b. Immediate threatened: (Salvageable with immediate revascularization). Sensory loss is marked with rest pain, moderate muscle weakness, usually inaudible arterial Doppler signals, but audible venous Doppler signals.
c. Irreversible: Profound sensory loss, profound muscle weakness or paralysis and inaudible arterial or venous Doppler signals. This stage is non-salvageable

❖ Investigations:

- Duplex scan: localizes and identifies embolism or thrombosis.
- Arteriography: may cause a delay of 2-3 hours. → not done in threatened limb CT angiography or transfemoral arteriography are better
  - **In embolism:** proximal arteries are normal and there is abrupt occlusion (classic meniscus sign) & no collateral arteries.
  - **In acute thrombosis:** the proximal arteries are unhealthy and there is extensive collateral circulation. Angiography will show the distal run-off.
- Echocardiography: detects cardiac sources of embolism. The trans-esophageal route is four times more informative than the trans-thoracic.
- CBC: raised hemoglobin, blood urea nitrogen & creatinine indicate intravascular hypovolemia due to fluid sequestration in limb.
- ABG: Acidosis and raised creatine phosphokinase and WBCs indicate extensive muscle necrosis.

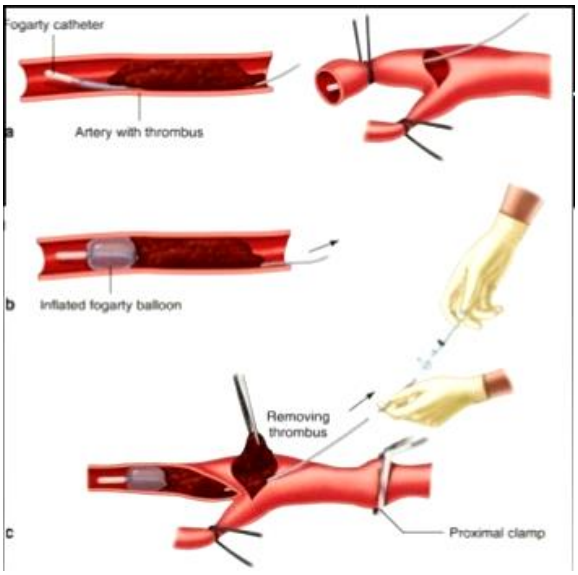
❖ Treatment:

- General measurers:
  - Control of arrhythmias or heart failure and cardiac monitoring.
  - Correction of dehydration.
  - Control of pain.
  - Oxygen administration.

- **Immediate heparinization**: prevents proximal and distal thrombus formation. start with a loading dose of unfractionated heparin **80** IU/Kg followed by maintenance dose of **18 IU/ Kg/hour**. The dose is controlled by checking activated partial thromboplastin time **(APTT)** every 12 hours which should be maintained at **2.5-3 times** the baseline level.

➢ Embolism:

1. Urgent embolectomy using **Fogarty balloon catheter** is the standard method for removal of arterial emboli.
2. The operation should be done as long as the limb is viable. As delay leads to obstruction of collaterals by propagation of thrombosis.
3. The operation can be done under local, general or epidural anesthesia.
4. **Femoral artery** embolectomy is done via common femoral transverse arteriotomy.
5. **Aortic bifurcation** embolectomy is done via bilateral femoral arteriotomies.
6. **Brachial artery** embolectomy is done via a brachial artery exposure.
7. **Arteriography** done on table to confirm clearance of the arterial tree.



8. **Heparin** should be continued postoperatively until cardiac condition is assessed & need for more heparin therapy is determined.
9. **Fasciotomy:** Embolectomy is followed by compartment syndrome. Therefore, it should be decompressed by full-length skin & fascial incisions from the knee to the ankle.
10. The source of arterial emboli should be corrected.

➢ Acute Thrombosis:

- **Severe ischemia:** Urgent arteriography is performed to plan for emergency revascularization surgery.
- **Moderate ischemia:** Urgent arteriography is performed.
  - There is time for the use of thrombolytic therapy.
  - After thrombus dissolution → Elective revascularization surgery.
  - Elective surgery carries a better prognosis that emergency surgery.

✓ **Embolic acute ischemia**: urgent surgery
✓ **Acute thrombosis:** may produce less severe ischemia because previous chronic ischemia opens collaterals.

Arterial Injuries

❖ Etiology:

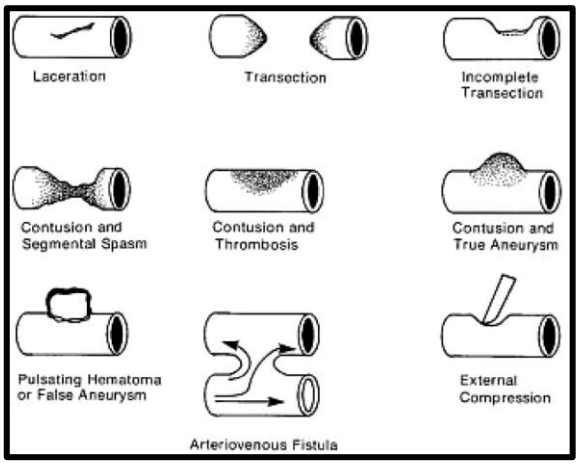
➢ Penetrating trauma
- Low-velocity agents: e.g., knives damage structures in their pathway.
- High-velocity missiles widespread: damage can affect remote vessel.
- Close range shotgun widespread damage that penetrates the tissues.

➢ Blunt trauma road traffic accident.
- Direct injury to major vessels.

- Fractures and dislocations, e.g.: supracondylar fractures of humerus.

➢ Intra-arterial drug injection.
➢ Iatrogenic
- pelvic operations.
- Arterial cannulation in arteriography.

❖ Types:
➢ Arterial tear:
a) Complete tear (transection): transected ends retract, constrict & thrombose → ischemia rather than hemorrhage.
b) Partial tear:
- The coats retract → widening of the gap → presents with hemorrhage.
- If bleeding remain within the tissues → pulsating hematomas → traumatic false aneurysm (pseudoaneurysm).
- If bleeding finds its way to the exterior (ext. wound, peritoneal or pleural cavities) → life threatening hemorrhage may occur.
- Concomitant injury to adjacent arteries & veins → A-V fistula.



➢ Arterial contusion & thrombosis:
- Commonest type in closed injuries.
- Presents with ischemia & thrombosis occurs over damaged area.

➢ Arterial spasm: with ischemia & diagnosed after exploration to exclude contusion & thrombosis.

❖ Clinical picture:
➢ Hard signs: **Sure signs of arterial injury:**
1. **External arterial bleeding.**
2. **Loss of distal pulses.**
3. **Classic manifestations of acute ischemia (6Ps).**
4. **Pulsating or expanding hematoma.**
5. **A palpable thrill or an audible bruit.**

➢ Soft signs: These are less specific signs:
1. Adjacent nerve injury →neurological deficit.
2. Proximity of penetrating wound to a major vascular structure.
3. Moderate sized hematoma not pulsating & not expanding.
4. Diminished pulse compared to the contralateral site.
5. History of prehospital hemorrhage that has stopped, or presentation with shock that cannot be explained by other injuries.

OT: operation theater

API: arterial pressure index

❖ Investigations:
➢ **No need in hard signs:** Immediate surgical exploration is life-saving!
➢ **In patients with soft signs:** urgent investigations are needed.
- **Plain X-ray:** to detect foreign bodies or fractures.
- **Arteriography:** only for patients with either an abnormal extremity pulse examination or a Doppler index less than 1.0.
- **CT angiography:** for arterial injury & adjacent structures.

❖ Treatment:
➢ Immediate treatment
1. Control of external bleeding:
- Direct local compression of the site of injury is the most effective *method* at the scene of accident or in the emergency room.

- Blind placement of clamps into a bleeding wound should be avoided.
- Tourniquets should be avoided since they can occlude collateral inflow.
- Embedded knives or hematomas should not be disturbed.

2. Resuscitation: by blood and IV fluids.
3. Heparinization: Systemic heparin is recommended ONLY in patients with isolated vascular injury presenting with ischemia. Avoid in patients with multiple injuries especially those involving CNS, eyes & bone.
4. Prophylactic antibiotics: should be given early.

➢ Definitive treatment:
- Time: within **6 to 8** hours of injury for limb salvage
- Methods:
  - Clean-cut arterial injuries:
    - **Longitudinal tears** are closed with vein patch to avoid lumen narrowing
    - In **transverse incision <½** the circumference, the injury is repaired.
    - If **>½ the circumference** is injured, complete division is performed then end to end repair.
    - In **completely divided**, direct end to end anastomosis is done.
  - If the artery is contused or divided:
    - **Damaged edges** or segments are excised.
    - **Patch Angioplasty**: Reconstruction with interposition graft (vein or synthetic).
  - If the artery is found spastic:
    - Local application of **papaverine** is tried.
    - **forcible dilatation** using Fogarty catheter or vessel dilator.
    - If spasm still persists, **excision** of spastic segment with a saphenous vein graft.

➢ Adjuvants:
- **Local heparin** should be used routinely.
- Injured main veins repaired **before arteries**.
- Adequate **skin covering** is essential.
- **Fasciotomy** is done in:
  - Late cases or muscle edema.
  - Combined arterial and venous injuries.
  - Swelling or paralysis after revascularization.

✓ In displaced **fractures** pulses frequently return after reduction (within **1h**).
✓ If pulses don't return within this time → Artery should be explored.

Any associated fractures should be immobilized by metal fixation prior to performance of vascular anastomosis.

One should never assume before exploration that a spasm is present & that it will be relieved with time. This serious error resulted in many limb losses. If vascular injury is found it should be repaired.

Intraarterial Drug Injection

- Etiology:
  - Drug addicts wrongly inject drugs into arteries.
  - Anesthetists wrongly inject thiopentone into an artery during induction of anesthesia.
- Sites: most common *brachial & radial* arteries.
- Clinical Picture:
  - Burning discomfort extending from point of injection to tips of fingers → occurs immediately after injection.
  - This is rapidly followed by severe pain & blanching.
  - Oedema develops rapidly and may be severe.
  - The hand is typically held in a claw like position.
  - Sensory loss is usually present. Whole wrist & hand might be involved, but changes are most severe in the distal digits, The fingers are usually cool & deeply cyanotic.
  - Digital gangrene & less commonly total extremity gangrene.
  - Signs Digital pulses are often felt and normal.
- Treatment:
  1) Heparin at dose of 10.000/I.U. intravenously, followed by infusion to keep PTT at 1.5-2 times.
     - In intra-arterial thiopentone injection → Anesthetist shouldn't remove needle from artery before injecting heparin intraarterially, Heparin prevents thrombosis of small vessels.
  2) Dexamethasone 4mg I.V every 6h→↓↓edema & tissue pressure elevation.
  3) Low molecular weight dextran (dextran 40) minimizes platelet aggregation.
  4) Strong analgesics.
  5) Extremity elevation to reduce edema.
  6) Early passive and active exercises.

Chronic Limb Ischemia

- Etiology:
  a) **Atherosclerosis** (most common cause specially >45 y)
     N.B. if patient < 45 y → Diabetic presenile atherosclerosis.
  b) Arteritis: in both upper & lower limbs.
  c) Raynaud's disease (females, upper limb)
  d) Buerger's disease (males, lower limb)
- Risk Factors:
  - Non-modifiable: Age, ♂, black race & +ve family history.
  - Modifiable:
    - Major: **HTN**, hypercholesterolemia, smoking & DM.
    - Minor: hypertriglyceridemia, obesity, sedentary life.
- Sites:
  - Adjacent to arterial bifurcations: *(most common site)* at the origin of major arterial branches & at sites where an artery passes beneath or through a fascial sling.
  - Atherosclerosis that causes lower limb ischemia affects most:
    - The aortic bifurcation (aortoiliac disease).

slowly progressive obstruction that gives enough time for collaterals to develop, therefore, gangrene does not occur rapidly.

- The superficial femoral artery (femoropopliteal disease).
- The leg arteries (tibial disease).

✓ The lesions have special distribution in descending order affecting coronaries > cerebrals & carotids > arteries of lower limbs > the renal > superior mesenteric > coeliac arteries & lastly arteries of upper limbs.

❖ Pathogenesis:

- Subintimal deposition of low-density lipoproteins (LDL) fatty streaks.
- **Endothelial dysfunction**:
  - ↓↓ nitric oxide counteracts free oxygen radials
  - Release of pro-coagulant
  - Release of inflammatory mediators
  - Release of angiotensin II
- Proliferation of smooth muscles of media due to release of growth factors as platelet derived growth factor and fibroblast growth factor.
- Eventually, formation of fibrous plaque leading to arterial narrowing.
- Complete arterial block may occur due to thrombosis on top of the atheroma or due to hemorrhage in the subintimal plaque.

❖ Clinical Picture:

- Symptoms:

a) Intermittent claudication: Cramping pain occurs on walking & relieved by rest. (*Early* there's *pain on walking but can continue walk*)

- The claudication distance: distance which the patient can walk until pain occur & patient stops.
- The claudication time: time which the patient stops to recover.
- As the disease progress: the claudication distance shortens and claudication time increases.
- Site of pain depends on site of obstruction:
  - Gluteal, thigh & Calf pain in aortic bifurcation block.
  - Calf pain in superficial femoral block.
  - Foot pain in distal arteries block.

- Mechanism: Accumulation of metabolites due to muscle ischemia during walking which are gradually washed away during rest.

b) Rest pain: with continuous progression of the disease, the blood supply of the limb cannot even satisfy the requirements at rest:

- ischemic neuritis: causes agonizing burning pain mainly in the foot.
- Pain is more severe at night when the patient sleeps probably due to further diminution of the blood supply caused by elevation of the foot. Another factor is that the warmth of the limb during sleep increases the metabolic demands while the blood supply cannot cope with it.
- Warmth also induces cutaneous vasodilatation which further deprives the nerves of their blood supply. Many of these patients spend the night sitting in a chair in an attempt to relieve the pain.

c) Ulceration and gangrene: In severe ischemia, superficial painful erosions may occur between the toes, on the dorsum of the foot, or around the malleoli. The end stage of ischemia is dry gangrene.

d) Impotence: occurs in aortic bifurcation block due to diminished blood flow in the internal iliac arteries (Leriche Syndrome).
e) Atherosclerosis symptoms: angina, cerebral stroke or transient ischemic attacks & postprandial abdominal pain (intestinal ischemia)

✓ **Leriche syndrome**
1. claudication in one or both lower limbs.
2. Reduced sexual potency in males.
3. ↓↓ or absent femoral pulses.

✓ **staging of chronic limb ischemia**
Stage I: Asymptomatic
Stage II: Intermittent claudication
Stage III: Rest pain
Stage IV: Ulceration or gangrene

➢ Signs:

a) General examination
- Pulse: All accessible arteries are palpated to assess the presence of the pulse, any arrhythmias or the condition of the arterial wall.
  A stethoscope over a stenosed artery may reveal a murmur.
- Blood pressure: should be measured.

b) Local examination
- Trophic changes:
  - **Loss of skin appendages:** (hairs and sebaceous & sweat glands): skin becomes dry, the nails become brittle, deformed & lose their luster.
  - **Loss of subcutaneous fat:** skin is thin & toes tapered.
  - **The muscles:** become wasted and the bones osteoporotic.
  - **ischemic ulcers:** may develop at the tips of the toes.

c) Temperature changes: Some degree of coldness will be detected in the ischemic limb up to a level below but near that of obstruction.
d) Color changes: Pallor, cyanosis or redness (rubor).
- Pallor is due to ↓↓↓ blood flow into the skin.
- Cyanosis & redness are due to the stagnation of blood in dilated capillaries under the effect of accumulated vasodilator metabolites.
- Rubor color of blood is red at first but then becomes blue due to extraction of oxygen by the tissues.

e) Absence of pulses:
- Pulses distal to the site of occlusion are absent or markedly weak.
- A murmur may be heard over site of the stenotic arterial segment.

✓ N.B: Dorsalis pedis artery is absent normally in 10%, Posterior tibial pulse is absent in 2%.

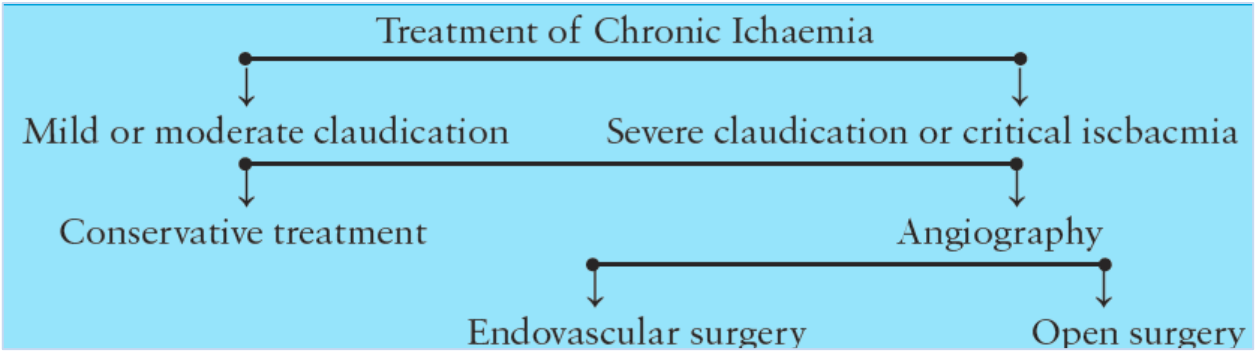
➲ **D.D of chronic lower limb pain**
1. Venous claudication incompetent deep or perforating veins.
2. Neurogenic claudication due to a herniated disc or spinal canal stenosis. The pain radiates from the gluteal region to the foot. The patient has to bend forwards or to lie flat on a hard matrix to relieve pain.
3. Osteoarthrosis of hip or knee joints.

- **Special tests**
  - Test for the capillary circulation: If one presses over the tip of the toe, it becomes pale. Once the pressure is released, the color returns within two seconds. In an ischemic limb the return of color is slow and is called sluggish capillary circulation.
  - Buerger's angle: Patient lies supine & limb is gradually elevated. Angle at which blanching of toes occurs is called Buerger's angle. Blanching does not occur even when the limb is raised up to 90°. The smaller the angle → the more severe the ischemia is.
  - Harvey's venous refilling time: With the patient supine, the limb is elevated to right angle until all veins empty. It is then brought down to the horizontal position. Normally the veins fill in 10-15 seconds, in chronic ischemia venous refilling is delayed to above 30 seconds.
- **Complication of Acute ischemia:**
  - Ulceration of atheromatous plaques invites platelet aggregation which later detach & cause distal embolism.
  - Acute thrombosis on top of atheroma → complete arterial block
  - hemorrhage may occur under intima → arterial block.
  - Aneurysm formation.
  - Critical limb ischemia
    I. Persistent recurrent rest pain needing opiates for at least 2 weeks.
    II. Ulceration or gangrene of the foot or toes.
    III. Ankle systolic pressure < 50 mm Hg or toe pressure <30 mmHg.
- **Investigation:**
  - Labs:
    - Blood sugar.
    - Blood picture to detect anemia or polycythemia.
    - Serum lipids & creatinine.
  - Imaging
    - Doppler flow study:
      - Detect stenosed or occluded segments.
      - Detecting collateral refilling of post-stenosed segments.
      - Measurement of the Ankle/Brachial (AB) index.
      - Measuring segmental pressures of the different arterial segments.
        Normal arterial flow → triphasic wave &collateral flow → biphasic wave.
    - Duplex scanning: visualize arteries & detect area of stenosis.
    - CT angiography (CTA).
    - Magnetic resonance angiography.
    - Direct arteriography: for endovascular surgery ONLY.

Don't forget ECG & fundus examination

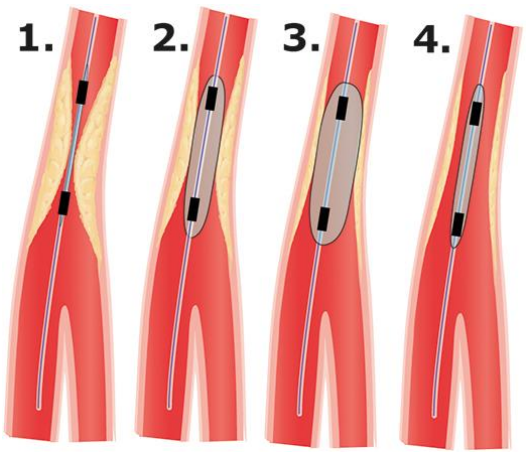
AB Index
1-1.2 Normal
< 0.9 Ischemia.
< 0.7 Severe Ischemia
< 0.3 Impending gangrene.

❖ Treatment:



- Conservative:
  1. Complete abstinence from smoking.
  2. Mild exercises help open the collateral circulation.
  3. Correction of anemia helps tissue oxygenation.
  4. Control of D.M, HTN & hyperlipidemia.
- Medical:
  1. Aspirin (150 mg/day) →↓↓ platelet adhesiveness.
  2. Statins to control hyperlipidemia.
  3. Calcium Channel Blockers.
  4. Cilostazol**:** ↓ platelet aggregation, ↑HDL, cholesterol & ↓triglycerides.
- Surgical:
  - **Indication for surgery:**
    - **Severe claudication** interferes with the patient's work & lifestyle.
    - **Critical ischemia** (ischemic ulcer, rest pain or minor gangrene) to save the limb (Limb salvage surgery).
  - **Types:**
    - Endovascular surgery:
      A. Percutaneous transluminal angioplasty (PTA): Special balloon catheter is introduced percutaneously till balloon reaches stenosed or occluded segment. It is then inflated & kept for about 1 min to dilate the stenosed segment.
        - **Indications:** short < 2 cm or significant stenotic segments in otherwise relatively normal arteries.
        - **Success rates:** about 95**%**.
        - **Complications**: Hematoma, AV fistula, Restenosis.

      B. Application of stent: After balloon angioplasty, stent can be inserted to prevent elastic recoil of the arterial wall & keep the lumen patent.



    - Open Surgery:
      A. Thrombo-endarterectomy:

- **Technique:** Removal of the thrombus, intima & inner media leaving a patent lumen that becomes rapidly endothelialized
- **Indication**: ONLY for limited number of patients applied for large artery with localized obstruction e.g. a localized narrowing in an iliac artery.

B. Bypass surgery (more frequently performed)

- **Idea:** bypass the obstruction by inserting a graft from the healthy artery above to another healthy artery below the obstruction.
- **Indication:** Big & medium sized artery with extensive or multiple lesion

- For Aorto- iliac disease → Aorto-bifemoral bypass using synthetic graft [Dacron or polytetrafluoroethylene (PTFE)].
- For Superficial femoral artery block→ Femoro-popliteal bypass.

✓ **For Femoro-Popliteal bypass**

The saphenous vein is the best graft for this operation BUT has valves that allow blood to flow from below upwards only, this problem is overcomed by:

- Reversed long Saphenous vein graft: to prevent obstruction
- In situ long saphenous vein graft: valves are destroyed by Valvotomy & the vein tributaries ligated to prevent A.V. fistula.

➢ What to do for patients who don't have good distal run-off?

- **Lumbar sympathectomy** doesn't improve bl. supply of the muscles. So, it's mainly indicated when there are cutaneous ulcers or gangrene
- **IV or Intra-arterial PGE.**
- **Amputation** for gangrene at a proper level or wait for separation.
  - Indications:
    1. Spreading or massive gangrene
    2. Spreading infection.
    3. Severe uncontrollable pain making the pt request.
  - Level of amputation: Depends on blood supply enough for healing.
    - If popliteal pulse is felt → below knee amputation.
    - Atherosclerosis (involve femoral artery) →above knee amputation. (The stump will be supplied by the profunda femoris).

Thrombo-Angiitis Obliterans (Buerger's Disease)

❖ Definition: chronic, non-atherosclerotic, segmental inflammatory, obliterative disease
❖ Sites: infrapopliteal & infrabrachial medium sized and small arteries.
❖ Etiology: young male, heavy smoker
❖ Pathology:

- The disease has patchy distribution & episodic course.
- It affects the vessels distal to the popliteal artery.
- Neurovascular bundle shows pan vasculitis & neuritis.
- The artery is obstructed by thrombus → organized.
- Affection of the nerves by inflammatory process & early block of arteries that explain the severe pain

- Clinical picture:
  - Chronic ischemia: young heavy smoker male
  - Migrating thrombophlebitis: precedes disease which appears as a small, tender cord along vein.
  - Raynaud's phenomenon: may be superimposed.
  - Interdigital fungus infection: is common association.
- Investigations:
  - Duplex: distal arterial occlusion with corkscrew collaterals.
  - Digital subtraction angiography: reveal involvement of small and medium sized arteries as the digital, planter and peroneal ones.
- Treatment:
  - Smoking cessation: to avoid disease progress.
  - Sympathectomy: short term pain relief & heal promotion.
  - Amputation: of one or more digits or toes is indicated for persisting pain or gangrene and can be performed adjacent to line of demarcation with satisfactory primary healing.

	Atherosclerosis	Buerger's disease
Age	Elderly	Young (20-40 y)
Sex	♀>♂	Only in ♂
Level	Aorta-iliac, femoro-popliteal	Distal vessels with patchy distribution
Etiology	•Hypertension • High cholesterol • DM	Excessive smoking
Pathology	Intimal (atheroma)	Inflamed neurovascular bundle & thrombi that block lumen
Migrating thrombophlebitis	Absent	Usually present
Rest pain	May be but LATE	Very EARLY

Vasospastic Disorder (Raynaud's Disease)

- Etiology: exact etiology is Unknown but certain factors may promote:
  a) Abnormal sensitivity of small arteries & arterioles of hand or feet to cold.
  b) Increased sympathetic tone.
- Clinical Picture:
  - Bilateral and Symmetrical.
  - Common in young females in both hands.
  - Attacks precipitated by coldness & relieved by warmth.
  - The attack consists of 3 consecutive phases:
    I. Pallor: due to spasm of the digital arterioles.
    II. Cyanosis: due to dilatation of the capillaries which are filled with slowly flowing deoxygenated blood.
    III. Redness: As the attack passes off, arterioles dilate & oxygenated blood passes into the dilated capillaries.
  - Radial & Ulnar pulses are preserved.
  - Only minute patches of ulceration or gangrene.

- Grades:
  - 1st → Only Raynaud's phenomenon.
  - 2nd → Mild trophic changes in the tips of fingers and nails.
  - 3rd → Gangrene of finger tips.

- Treatment:
  - In early stages>> conservative measures:
    1. Avoid cold weather and to wear woolen gloves in winter time.
    2. Vasodilator drugs & Calcium channel blockers.
  - In severe cases>> Surgical: **Cervico-dorsal sympathectomy:** Its immediate results are good but the symptoms usually recur after sometime. Recurrence, at least, is not severe.

Raynaud's phenomenon

- Color changes similar to Raynaud disease, may occur with:
  - Thoracic outlet syndrome
  - Certain occupations as typists, pianists and laborers who use vibrating tools
  - Vascular disorders as atherosclerosis and Buerger's disease
  - Drugs as chronic administration of ergot-containing drugs for migraine & ergot poisoning
  - Atrophic disorders of limbs, e.g., after poliomyelitis
- Treatment: treatment of the cause and Vasodilators and ß-blockers may be added

Diabetic Foot

- 25 times diabetics are more susceptible to foot problems
- 70% of amputation performed on diabetic patient
- Etiology:
  - Peripheral neuropathy: is present **in 25-40%** of patients.
    - **Sensory neuropathy** the patients are unaware of injurious insults.
    - **Motor neuropathy** of intrinsic muscles of the foot leading to clawing of the toes & subluxation of the metatarsophalangeal joints, which puts undue pressure on the metatarsal heads. A callosity forms over pressure areas & infection → Neuropathic ulcer.
    - **Autonomic neuropathy** → peripheral vasodilatation & A.V shunting. Sweating ↓ → cracking & fissuring of skin.
  - ischemia: premature atherosclerosis of major vessels & micro-angiopathy diminish the blood supply and nutrition of the tissues.
  - Hyperglycemia and compromise of the immune system: Both humeral and cellular immunity are inhibited in diabetic patients

- Clinical picture:
  - Neuropathic ulcers: **painless** with **punched-out** edges with no signs of healing usually on the planter surface of the metatarsal head. Examination reveals peripheral neuropathy.
  - ischemic ulcers: usually in the distal parts of the toes.

Symptoms and signs of chronic ischemia are present.

- Infection: usually follows a minor trauma e.g., a pin prick, careless cutting of a nail, scraping of a callosity or interdigital infection. Infection spreads rapidly leading to cellulitis, necrosis of tendons and muscles, lastly, osteomyelitis. Multiple pockets of pus are present.
  In severe cases→ localized or massive gangrene of the toes or foot

❖ Investigations:

- Blood sugar.
- X-ray may show osteomyelitis, pathological fracture or Charcot's joint.
- Swabs for culture and sensitivity should be taken from any discharge.
- Duplex ultrasound.
- CT angiography if there is clinical evidence of ischemia.
- MRI to assess the condition of the soft tissues.

❖ Treatment:

1. Hospitalization, Rest in bed & elevation of the foot.
2. Proper control of diabetes, better by crystalline insulin every 8 h.
3. Broad spectrum antibiotics started immediately until culture results
4. Drainage of infection and debridement under general anesthesia:
   - Drain all pockets of pus & all sloughs should be excised.
   - A gangrenous toe needs to be amputated leaving the wound open.
   - In cases with widespread infection, some form of a major amputation is necessary to save the patient's life.
5. Repeated dressings & debridement with drainage of pockets of pus.
6. Plastic skin coverage for large raw area when the wound is completely free of infection in order to shorten the recovery time.
7. Patients with clinical occlusion of a major artery → Angiography to assess the feasibility of vascular reconstruction.

✓ **Prevention**:

1. Control of diabetes.
2. Toe nails careful trimming
3. Avoid tight foot wear.
4. Early ttt of taenia pedis infection.
5. Careful inspection daily of the feet to look for wounds & interdigital infections.
6. Avoid walking bare-footed
7. Daily feet care by washing and drying or powdering.

Gangrene

❖ Definition: macroscopic death of tissues that is generally caused by loss of blood supply & usually associated with bacterial invasion & putrefaction.

❖ Site: Great toe is commonest site.

❖ Etiology:

- ischemic:
  - Thrombosis: e.g., on top of atherosclerosis (senile gangrene).
  - Embolism.
  - Vasospastic diseases e.g., Raynaud's disease & ergotism.
- Neuropathic: Diabetes mellitus, syringomyelia & leprosy.
- Traumatic:
  - Direct trauma: due to crushing or pressure (bed-sores).

- Indirect trauma: due to injury of the main vessels.

➢ Physicochemical: Burns, frost-bite and trench foot.
➢ Infective:
- Specific infection: Clostridial gas gangrene.
- Nonspecific infections: necrotizing fasciitis & carbuncle.

➢ Venous gangrene.

❖ Types:

Types	1.Dry Gangrene	2.Moist Aseptic gangrene
Etiology	Chronic ischemia e.g., atherosclerosis	• Acute ischemia e.g., ligature or embolus • Coincident venous occlusion e.g., traumatic gangrene
Mechanism	gradual slowing of blood flow permits evaporation	water logging of the tissues from sudden occlusion of the main artery
Gross	Shrunken, dry, wrinkled mummified & BLACK	Remains of same size & consistency, WHITE colored at first then purple or greenish-black
Odor	No odor or minimal	Very offensive
Demarcation	Well defined line	ill-defined line (no time for evaporation)
Fate	separation →ulcer	Spread →direct or by skipped lesions
C/P	Pain is marked & causes insomnia and exhaustion	

3.septic moist gangrene

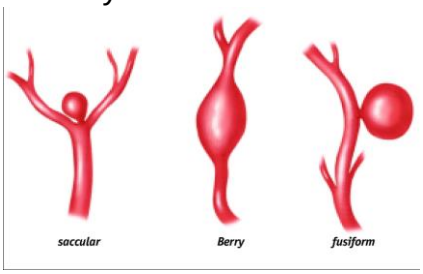
- Etiology:
  1. Infection of sterile gangrene (2ry infective gangrene)
  2. Infection of tissues with virulent organisms (1ry infective gangrene).
- Clinical picture:
  1. **Limb** is swollen, edematous & inflamed.
  2. **Skin** → moist and blotchy & raised from the surface by bullae filled with serum.
  3. **Odor** is very offensive, and may crepitate due to gas formation.
  4. **Constitutional signs** are present & death occurs from septicemia.
     - Treatment: amputation till level of pulsation (emergency situation)

✓ **The five cardinal signs of gangrene are remembered by:**
"Press and See How Color Fades"

1. Loss of **P**ulsation and sluggish capillary circulation
2. Loss of **S**ensation
3. Loss of **H**eat (coldness)
4. Change **C**olor of skin into blue & then black, the color doesn't change by local pressure "fixed color changes"
5. Loss of **F**unction

Aneurysm

- Definition:
  - SAC contains blood & communicates with the lumen of artery.
  - Permanent localized dilatation of an artery
  - At least 1.5 times the normal diameter of given segment.
- Types: classified according to:
  - Etiology:
    1. Pathological.
    2. Traumatic
    3. Congenital.
  - Structure: True or false.
    - True aneurysm: Wall is formed of the 3 layers of the dilated artery.
    - False aneurysm: a hematoma communicating with lumen of artery through a partial tear in its wall. So, wall of false aneurysm is formed by the fibrous wall of the hematoma.
  - Shape:
    1. Fusiform
    2. Saccular
    3. Dissecting



- Etiology:
  - Pathological: due to weakening of the arterial wall.
    - Atherosclerosis is the commonest cause e.g., the abdominal aorta.
    - cystic medial necrosis, septic emboli of subacute bacterial endocarditis & collagen diseases as Behcet's disease, Marfan & Ehler's Danlos syndromes, syphilis → thoracoabdominal aneurysms.
  - Traumatic:
    - Blunt trauma to artery → weaken its wall → true aneurysm.
    - Penetrating injury to artery → small hole in wall → hematoma → clot surrounded by false capsule of organized fibrous tissue →false aneurysm.
  - Congenital: circle of Willis (Berry aneurysms) → subarachnoid hemorrhage. Other sites include the splenic, renal or coeliac vessels.
- Clinical Picture:
  - Silent aneurysms e.g., the abdominal aorta is commonly silent. They are accidentally discovered at clinical or US examination
  - Swelling or symptoms of complications.
  - Symptoms due to compression on adjacent structures.
  - Local signs of an aneurysm:
    1) The swelling exhibits expansile pulsations (most important sign).
    2) Swelling can be moved **Across** the line of artery but **NOT** along it.
    3) Proximal pressure on artery → diminution of the pulsations.
    4) Distal pressure on artery → aneurysm ↑↑size & tenser.
    5) A systolic thrill felt or **bruit** heard.
- Complications:
  1. Rupture→ hemorrhage (most **fatal** complication)
  2. Distal ischemia due to thrombosis & occlusion of the aneurysm→ detachment of fragments of this thrombus → distal embolization e.g., gangrene in one of the digits due to subclavian aneurysm which develops in patients with thoracic outlet syndrome.

3. Infection →rupture → 2ry hemorrhage.
4. Compression on adjacent structures →obstruction or thrombosis e.g., compression of a nerve may cause motor or sensory affection.

- Investigations:
  - Duplex scanning is very useful.
  - CT angiography is very accurate
  - Magnetic resonance angiography.
- Treatment: The standard line of treatment is excision & graft
  1. Exclusion graft. insertion of a graft inside the sac of the aneurysm without removal of the sac, this is commonly done for aortic aneurysms.
  2. Excision with arterial ligation can be done for aneurysms of small arteries as the radial and ulnar arteries.
  3. Endovascular treatment with **stent** insertion.

✓ **Differential diagnosis**

1. A swelling overlying an artery. (Pressure on the proximal artery →NO change in size of the swelling)
2. A very vascular tumor as an osteosarcoma or metastases
3. An abscess.
4. Serpentine artery e.g., common carotid
5. An arterio-venous fistula

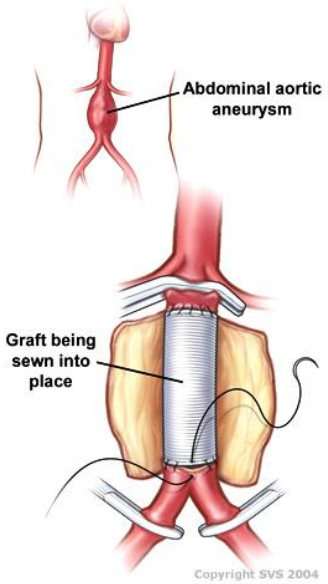
Abdominal Aortic Aneurysm (AAA)

- This is the most frequent type of aneurysms affecting Aorta below origin of the renal arteries in 95% of cases but may extend to iliacs.
  Rarely does it extend upwards to involve variable distance of the suprarenal abdominal aorta or the thoracic aorta in which case it is called a thoraco-abdominal aortic aneurysm.
- Etiology: Atherosclerosis in 95 % of cases.
- Clinical Picture:
  - Asymptomatic aneurysms: In **75%** of patients the AAA is discovered accidentally during a routine abdominal examination (As pulsatile epigastric mass) or during ultrasonography or CT scan.
  - Pain: is the commonest symptom. An A.A.A. gradually enlarges & impinges on surrounding structures causing vague abdominal pain. e.g., Back and flank pain from vertebral compression. These patients may be wrongly diagnosed as having lumbar disc prolapse.
  - Symptoms of rupture: The classic triad of AAA rupture is:
    1. **Acute upper abdominal pain:** abrupt onset in the back, flank or abdomen is characteristic of aneurysm rupture or acute expansion.
    2. **Pulsatile abdominal mass:** usually **tender**. It may be masked by obesity or abdominal distension.
    3. **Shock:** in most cases is the **1st** presentation. In other cases, rupture is contained within retroperitoneum that hypotension may be masked at time of initial examination. If patient is left untreated →shock eventually.

The 5-year rupture rates for untreated AAA of diameters **≥** 5cm is high, even if asymptomatic.

- Investigations:
  - Ultrasonography: If an AAA is *clinically suspected*, ultrasonography is the **screening test of choice** to document or to rule out the presence of an aneurysm. It is rapid, inexpensive & non-invasive.
  - C.T scan: If repair of an AAA is decided, provides data important for surgery especially if endovascular repair is considered**.**
  - Spiral CT: display information in multiple planes & allow three-dimensional reconstruction of the aneurysm sac.
  - MRI: detailed but *expensive*.
  - Transfemoral aortography:
    - Indication: suspected associated occlusive disease in the iliac, renal, or mesenteric arteries → plan repair of the occlusive disease.

- Treatment:
  - Immediate surgery
    for patients with the rupture. Should be taken to the operating room immediately for repair, no time should be lost in a surgical ward or in an ICU.
  - Urgent surgery: patients with symptoms of acute expansion (Severe pain of acute onset but with no leak on C.T. scan).
  - Elective surgery
    - Symptomatic aneurysms regardless of the size
    - Asymptomatic aneurysms **≥** 5cm in diameter.
  - Regular follow up for patients with asymptomatic aneurysms < 5cm Ultrasound is done every 6 months.



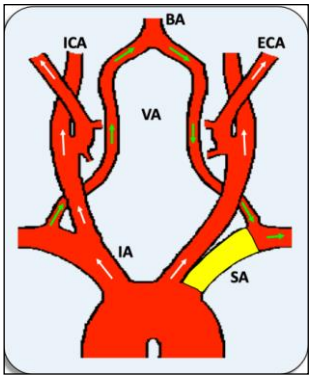
- ✓ Types of surgery:
  - **Open surgery** by opening & excluding the aneurysm then implanting a synthetic graft (**The standard surgery**)
  - **EVAR (Endovascular repair of AAA)** insertion of endoluminal stented graft through bilateral femoral arteriotomies **indicated for:**
    - High risk patients who cannot tolerate anesthesia & open surgery.
    - High-risk local abdominal factors, e.g., previous major surgery.

Acquired Arteriovenous Fistula (A-V fistula)

- Definition: Abnormal communication between an artery and a vein
- Other types: **Congenital** (associated with port wine hemangioma)
- Etiology:
  - Penetrating injury: by knife or bullet → inducing communication between an artery and a neighboring vein.
  - Iatrogenic during **angiographic** procedures the cannula may injure the vessels leading to A-V fistula.
    - Site femoral triangle *(most common)*
  - Intentionally: **chronic renal failure patients** needing regular dialysis.
    - Site usually between the cephalic vein and the radial artery.

- Clinical Picture:
  - Local signs:
    1. Small **swelling** along the vessel with **thrill** over it & continuous machinery **murmur** propagated along the vein by auscultation.
    2. **Branham's sign:** local pressure at fistula → marked slowing of pulse.
  - Distal signs: veins become dilated, tortuous, thick walled &pulsating.
  - Systemic signs: ONLY if fistula is moderate or large sized ↓ peripheral resistance &↑in the venous return →↑ cardiac output → tachycardia (water hammer pulse), ↑ systolic & ↓ diastolic pressure.
- Complication: A high output large fistula →cardiac failure.
- Investigations:
  - Doppler: can localize the site of the fistula.
  - Arteriography: show both artery & vein at the same time due to rapid venous filling.
    - Advantage: localize the site of the fistula.
- Treatment: Excision of the sac & restore continuity of both artery & vein.

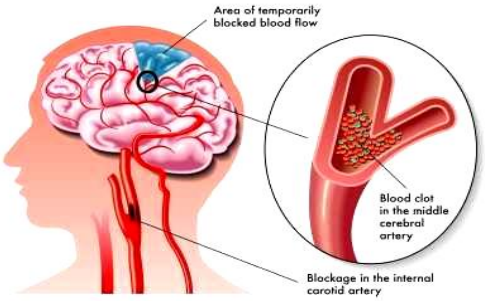
Subclavian Steal Syndrome (Cerebral Ischemia)



- Etiology: reversal of flow in ipsilateral vertebral artery distal to a proximal lesion in the **1st** part of the subclavian artery.
- Clinical picture:
  Vertebrobasilar insufficiency occurs on exercise of the upper limb due to ↑blood demand which the extremity steals from cerebral circulation through the ipsilateral vertebral artery.
- Investigations:
  - Duplex
  - CT angiography
- Treatment:
  - Carotid-subclavian artery bypass
  - PTA of subclavian stenosis.

Extracranial Cerebrovascular Disease

- Etiology: Embolization is the commonest cause of transient ischemic attacks (TIAs) & ischemic stroke (75 %)
- Pathogenesis:
  The embolus arises from heart, but more commonly arises from an ulcerating atherosclerotic plaque at the carotid bifurcation → ulceration & irregularity of the plaque → platelet aggregation →embolize to important vessel in the brain →either break up or persist.
  - If platelet aggregates **break up quickly → TIA**.
  - If embolic **persists** → focal infarction → **stroke**



- Clinical picture:
  - Asymptomatic patients discovered **accidentally** on screening carotid duplex prior to a major surgery e.g., coronary artery bypasses.
  - Transient ischemic attacks (TIAs) temporary focal neurologic or visual deficits lasting < 24 hours and end with complete recovery.
    - Onset: sudden & typically resolve within one hour.

- Site: Carotid artery TIAs are most common & caused by deficits in areas supplied by the **anterior** & **middle** cerebral arteries.

➢ Symptoms
1) Motor dysfunction as weakness or paralysis of one or both limbs ***contralateral*** to the affected hemisphere.
2) Sensory alterations numbness, loss of sensation or paresthesia in one or both contralateral limbs or in the opposite side of the face.
3) Receptive or motor aphasia if the dominant hemisphere is affected
4) Amaurosis fugax ipsilateral transient loss of vision described by the patients as a black curtain coming across the eye.

➢ Stroke residual neurologic deficits that could be minimal or profound depending on the extent of brain damage. **A stroke may be fatal**.

➢ Vertebrobasilar TIAs symptoms:
- Vertigo
- Ataxia
- Dizziness
- Visual hallucinations
- Bilateral paresthesia

❖ investigations:
➢ Duplex scan: **1st line investigation** in a symptomatic patient which can detect presence & the degree of stenosis.
➢ CT angiography.
➢ CT of the brain preoperative to detect pre-existing cerebral damage.

❖ Treatment: for symptomatic patients with 50-69% stenosis
➢ Surgical: **Carotid endarterectomy** Atherosclerotic plaque at the carotid bifurcation is removed →↓↓ risk of future stroke
- Indication Internal carotid artery stenosis of ≥ 70% in patient with:
  1) Carotid artery TIAs or Asymptomatic lesion
  2) Stroke leaving minimal neurologic deficit.

➢ Medical:
1) Antiplatelet drugs e.g., aspirin or clopidogrel.
2) Stop smoking & control of diabetes, HTN or hyperlipidemia.
3) Full anticoagulation in patients with cardiac embolic disease
   - Indication
     1. < 50% stenosis even if symptomatic.
     2. Patients during the acute phase of a stroke
     3. Patients suffering from stroke with poor recovery

✓ **Revascularization** contraindicated as it leads to hemorrhage in infarcted brain.

Endovascular Surgery

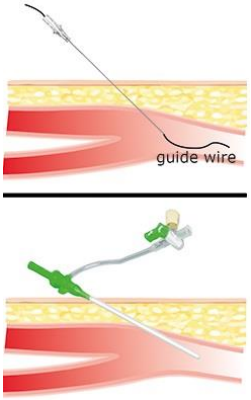
Balloon Angioplasty

Percutaneous transluminal angioplasty (PTA)

❖ Indication: adjunctive procedure with surgical revascularization.

1) Critical ischemia denoted by rest pain, ischemic ulcers or minor gangrene.
2) Severe claudication interferes with patient's work or life style.

❖ Ideal Lesion:
1) Best in iliac artery
2) Stenosis < 5cm length
3) At least 5mm beyond origin of an artery.

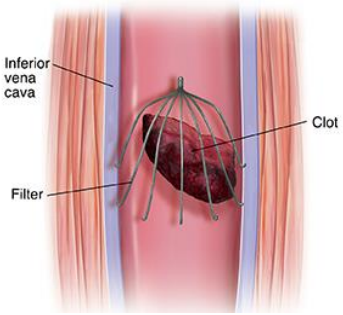


Arterial Stent

❖ Aim: A stent may be inserted in the same session after PTA to maintain patency of artery & prevent its drawbacks (dissection &elastic recoil)
❖ Principal:
- Metal vascular stent is guided over a guide-wire endoluminally & directed to the dilated arterial segment.
- The stent is carried over an expanding device that is kept closed until the stent reaches the desired site. Device is then opened to expand the stent to fit in the artery.

Inferior Vena Cava Filter

❖ Indications:
1) DVT or documented pulmonary embolism in a patient who has contraindication to anticoagulation, e.g., previous intracerebral hemorrhage or history of bleeding peptic ulcer.
2) Recurrent pulmonary embolism despite adequate anticoagulation.
3) Complications of anticoagulation that forced therapy to be discontinued.
4) Chronic pulmonary embolism in patients with pulmonary hypertension.



Endoluminal Grafts

❖ Principals: combination of a stent with prosthetic graft positioned with a delivery system.
❖ Uses:
1. Exclusion of A.A.A
2. Support of the intima of an artery after balloon dilatation.

Thrombolytic Therapy

❖ The main agents: streptokinase, urokinase & recombinant TPA.
❖ Aim: injects certain substances that activate the fibrinolytic system leading to dissolution of fresh thrombi in the arterial or venous system, given as a regional therapy directed into the thrombus via an endovascular catheter
❖ Indications:
1) Acute deep vein thrombosis
2) Acute thrombotic ischemia

3) Acute coronary occlusion
4) Acute embolic ischemia
5) Pulmonary embolism

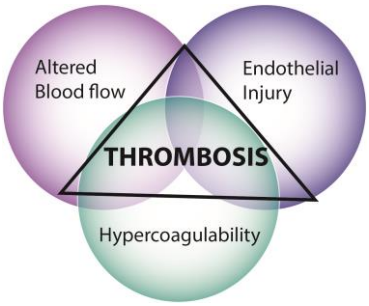
❖ Contraindications:

1) Active bleeding
2) Recent stroke
3) Infected bypass grafts
4) Infective endocarditis
5) Major operations within 2 weeks
6) Occlusion of vascular grafts
7) Recently occluded C.V.L

Venous Disorders

General Principles of venous thrombosis

- Types:
  I. Superficial venous thrombosis.
  II. Deep venous thrombosis mainly in the calf or iliofemoral veins.
- Pathogenesis: **Virchow's triad 3V (vessel wall, velocity, viscosity)**
  - Vessel Wall Damage to the endothelial lining of vein wall:
    1. Trauma to the vein wall, e.g., pelvic operations.
    2. Inflammation near vein, e.g., pelvic sepsis.
  - Venous stasis (Velocity)
    1. Prolonged confinement to bed, long trips or casts.
    2. Congestive heart failure
    3. Venous compression by tumor or pregnant uterus.
  - Hypercoagulability of blood due to (Viscosity)
    1. Polycythemia or Dysfibrinogenemia.
    2. ↓ Antithrombin III, proteins S & C, antitrypsin or macroglobulins.
    3. Factor V Leiden gene defect or activated protein C resistance.



- Factors that help venous return from the lower limbs:
  a) Muscle pump which requires strong muscles & intact deep fascia.
  b) Transmitted arterial pulsations to the venae commitants.
  c) The unidirectional valves.
  d) The -ve intrathoracic pressure.

Superficial Thrombophlebitis

- **Etiology:**
  - Idiopathic.
  - Varicose veins.
  - Veins cannulated for I.V. infusion.
  - After injection of irritant drugs e.g., diazepam.
  - Diseases: Buerger's, polycythemia, polyarteritis, Visceral cancers
- Clinical picture:
  - The vein becomes red, painful & cord like.
  - Mild pyrexia may be present.

- Complications:
  - Infection spread: rapid upward spread occurs with the danger of extension to the deep veins through the communicating veins.
    In this condition, proximal ligation and disconnection should be done.
  - Pulmonary Embolism: thrombus is adherent to vein wall due to inflammation.
- Treatment:
  - These are usually enough for the majority of cases:
    a) Compression by elastic stocking or compression bandage.
    b) Anti-inflammatory drugs e.g., aspirin.
  - The following are resorted to under special circumstances:
    - Antibiotics: ONLY if there is evidence of infection.

- Anticoagulant therapy (heparin and warfarin) in severe progressive cases (ascending thrombophlebitis & if associated DVT is suspected.
- Surgery: Prophylactic sapheno-femoral or sapheno-popliteal disconnection is done if thrombosis is ascending towards the junction with deep system.

Deep Venous Thrombosis (DVT)

❖ Etiology:
- **Virchow's triad**
- Obesity
- old age
- oral contraceptives intake
- previous DVT
- malignancy
- major trauma.

❖ Pathophysiology:
- Starts in the calf venous sinuses or in the iliac & femoral veins by adherence of platelets to the endothelial surface.
- More platelets then adhere. Fibrin and RBCs are deposited as layers in-between the platelets giving laminated appearance.
- When the vein is totally occluded, non-adherent jelly-like propagating thrombus spreads up the vessel as far as the next major tributary which is loosely attached & can be easily detached → pulmonary embolism.
- Later, the thrombus becomes adherent to the vein wall then organizes & contracts producing destruction of the valves & luminal narrowing, which are responsible for **post-phlebitic limb syndrome**.
- Later on, the process of fibrinolysis & phagocytosis starts and lead to recanalization of the vein but the valves are permanently destroyed.

❖ Clinical picture:
- **Classical group**
  - **Pain:** Aching discomfort & tightness in the involved calf or thigh.
  - **Swelling:** measuring difference in circumference between both limbs.
    - Calf thrombosis limited to the foot & ankle
    - Femoral thrombosis involves the calf & lower part of the thigh
    - ilio-femoral thrombosis massive swelling affecting whole limb.
  - **Tenderness:** present on grasping the affected calf or thigh or on compressing the muscles against bones
    Dorsi-flexion of foot → pain in calf (Homan's sign) "not reliable"

- **Complication Group**
  I. **Phlegmasia alba dolens:** Massive iliofemoral DVT associated with severe arterial spasm. The limb becomes pale, white, and massively swollen with absent peripheral pulses.
  II. **Phlegmasia cerulea dolens:** Massive iliofemoral DVT may cause severe congestion. The whole lower limb is massively

swollen & bluish. If not promptly treated it may lead to **venous gangrene**.

III. **Venous gangrene.**
IV. **Pulmonary embolism.**

➢ **Asymptomatic group:**
- **Silent DVT** is a frequent occurrence, there are NO local symptoms & patient may present later with either pulmonary embolism or with the manifestations of a post-phlebitic limb. However, it is suspected by the presence of unexplained rise of temperature or pulse rate.

✓ **D.D. of D.V.T.**
- **Sprain:** of calf muscles & hematoma.
- **Rupture of plantaris tendon** occur during exercise & can produce swollen painful calf which is usually difficult to differentiate from DVT. Duplex scan is needed for diagnosis
- **Ruptured Baker's cyst.**
- **Lymphatic obstruction:** chronic swelling & non-pitting.
- **Cellulitis:** systemic symptoms & inflammation. The leg is red, hot & tender.

❖ Investigations:
- ➢ Doppler: simple(bed-side), rapid & accurate in 80-85%.
- ➢ Duplex: Standard test for diagnosis of DVT Sensitivity & specificity reach 90-100%.
- ➢ Enhanced helical CT: can show the thrombus even in small veins.

Duplex & Doppler Findings	Normal veins	D.V.T.
Vein diameter	Normal	Dilated
Blood flow	Spontaneous	Poor
Vein compressibility	Normal	Poor
Echogenic material in lumen	None	Present
Distal compression	Augments blood flow	Poor augmentation
Blood flow with respiration	Phasic flow	Phasic flow lost

❖ Prevention of post-operative D.V.T:
- ➢ Physical measures
  1) Early ambulation after operations & active leg exercises while in bed.
  2) Adequate postoperative hydration.
  3) Elastic stocking support especially in the elderly.
  4) Intermittent pneumatic compression.
- ➢ Medical (Prophylactic anticoagulants)
  I. Low-dose heparin 5000 IU S.C. 2 h before operation & then every 12 h until the patient is ambulant (5-7 days) ↓ incidence of DVT by 50%.
  II. Low molecular weight heparin is more popular because it is given once daily & has lower risk of bleeding, more suitable for use at home.
    - **Indications:** High-risk cases for development of DVT
      - History of DVT or pulmonary embolism.
      - Elderly patients.
      - Major surgery e.g., cancer operations.
      - Obesity.
      - Females on contraceptive pills.

- **Contraindications:** large raw area is left after surgery.

❖ Treatment:

➢ Bed rest & elevation of the lower limb

a) Strict bed rest with the feet elevated 15-20 degrees above the level of the heart →↓ edema & pain, ↑ venous return →↓↓ thrombosis.
b) Application of elastic bandage or stocking help venous return.

✓ Thrombi usually take 7-10 days to become adherent to the vein wall, patient should be kept in bed for this period, usually swelling, pain and tenderness would have resolved by this time, gradual ambulation with elastic support is allowed but standing and sitting with legs dependent are forbidden because accompanying rise in venous pressure aggravates oedema and discomfort

✓ These measures are continued for 3 to 6 months until recanalization and collateralization develop.

➢ Anticoagulant therapy

▪ **Heparin:** given till all signs of active thrombosis subside which is known by disappearance of pain and tenderness. This usually takes 7-10 days

- Mode of action:
  - Enhances the activity of the naturally occurring antithrombin III.
  - The anti-thrombin III-heparin complex neutralizes the effect of thrombin.
  - Neutralizes factors IX, X, and XI.
  - Mild thrombolytic effect as it can cause mild activation of fibrinolysis.
- Types of heparin:
  - Fractionated heparin *(low molecular weight)*
    - Tinzaparin 175 IU/Kg/24 hours subcutaneously.
    - Enoxaparin 1 mg/Kg/12 hours subcutaneously.
  - Unfractionated heparin
    - Continuous IV infusion: ideal method, but requires hospitalization & pump which controls the dose per hour. A bolus dose of 5000 IU is given IV and then a Dextrose 5% drip containing heparin is infused at rate of 20-30 IU of heparin/kg/hour (1000-2000 IU/ hour for an adult) Dose is monitored by activated partial thromboplastin time (APTT) which should be kept between 2-2.5 times the control value. A more practical but less accurate method of monitoring is to do clotting time.
    - Bolus therapy 5000 IU are administered I.V. every 4-6 h, APTT is performed one hour before every injection until the dose is adjusted.

subcutaneous low molecular weight heparin is **less** likely to cause bleeding

TYPES	**Unfractionated heparin**	**low molecular weight**
Mode of action	Antithrombin III	Anti-factor Xa
Half life	1.5 - 2 hours	12 hours
Route	I.V.	Subcutaneous
Monitoring	**APTT**	Xa (usually not needed)
Thrombocytopenia	1-5%	<2%
Hospitalization	Is necessary	Can be taken at home

- Complications of heparin therapy:
  - **Bleeding:** due to over dosage.
    - Clinically: subcutaneous bruises, epistaxis, hematuria bleeding gums or gastro-intestinal bleeding
    - Prevention: by proper control of the dose by repeated APTT.
    - Treatment: **Protamine sulphate** 1 mg IV for every 100 IU heparin.
  - **Heparin resistance:** mild & requires ONLY increasing the dose.
  - **Heparin induced thrombocytopenia: 1-5%** in 2nd week of therapy. Platelet count drops <100,000/ul → heparin should be stopped.
- Contraindications to anticoagulant therapy:
  - **Absolute:**
    - Trauma or recent operation on brain or spinal cord
    - Hematological diathesis.
  - **Relative**
    - Major visceral injury.
    - Wide raw area
    - Major acute fractures.
    - Peptic ulcer.
    - History of cerebral hemorrhage.
    - Hypertension > 180/120 mm Hg.

- **Oral anticoagulants (Warfarin):**
  - Mode of action: block the synthesis of at least 4 vitamin K dependent clotting factors (prothrombin & VII, IX and X). So, its effect is **delayed**. ↓↓ synthesis of protein C & S which have an anticoagulant effect & so there is a period of relative hypercoagulability during first few days of warfarin therapy that should be covered by continuing heparin therapy together with the warfarin for the first 3 days.

  - Duration: Oral anticoagulants are given for **3-6** months which is the time needed for recanalization & collateralization.
    In some patients liable to re-thrombosis, warfarin is given indefinitely.
  - Administration:
    - Basal level of prothrombin time (PT) & PC prior to starting of warfarin.
    - An initial dose of 10 mg warfarin is followed by 5 mg daily dose.
    - Discontinue heparin after 3 days of overlap treatment.
    - Five days after the start of warfarin the prothrombin time (PT) & concentration (PC) are measured and the dose is adjusted to reach the therapeutic goal of PC 30-40%.
    - PT & PC should be repeated **every 2 weeks** during treatment course.
      These tests expressed more accurately as (**INR**) should be between **2.5-3.5** times control value.
  - Complications:
    - **Bleeding** → treated by ↓↓ dose & giving 10-20 mg vitamin K I.V. In cases of severe bleeding → **fresh frozen plasma** is given.
    - **Drug interaction** e.g., aspirin & non-steroidal anti-inflammatory agents.

- Thrombolytic therapy
  - Streptokinase, Urokinase and TPA (Fibrinolytic activators)
    - Indications:
      a) Severe DVT
      b) phlegmasia alba dolens.
      c) phlegmasia cerulea dolens.
    - Mechanism of action:
      1) Dissolve fresh thrombi.
      2) produce rapid clearance of the occluded veins.
      3) preserve the competence & function of venous valves.
    - Complications:
      - Severe bleeding
      - Streptokinase may lead to allergic reactions.
    - Contraindications:
      - old age
      - ulcer
      - history of hemorrhagic diathesis.

✓ Effect of these drugs is at its best in the first **3 days** of thrombosis after that they have NO advantage over heparin.
✓ Limit development of postphlebitic syndrome.

- Insertion of inferior vena cava filter
  - Under local anesthesia & radiographic control the filter is placed by open exposure or percutaneous through jugular vein in infrarenal position.
    - Indications:
      - contraindication to the use of anticoagulants.
      - Progressive thrombosis or recurrent pulmonary embolism.
    - Complications: **< 5**% of cases
      1) Hemorrhage
      2) Injury of IVC.
      3) Migration.
      4) perforation to the Aorta

✓ **Objectives of treatment**
- Prevention of formation of new thrombi.
- Prevention of pulmonary embolization.
- ↓↓ venous valves damage

Axillary Subclavian DVT

- Etiology:
  - Primary: (Spontaneous)→ **Cervical Rib** excessive muscle activity of upper limb (sports or occupational) compress vein in costoclavicular space.
  - Secondary:
    - Central venous catheters.
    - Metastatic tumors in axilla.
    - IV chemotherapy or TPN through upper limb veins.

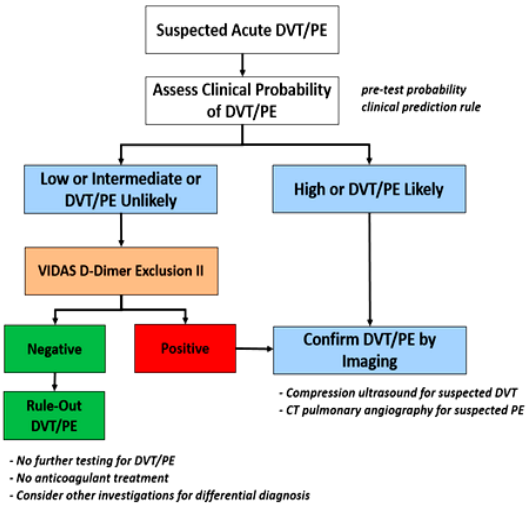
- Incidence: 2-3% of DVT
- Clinical picture: •
  - Pain, swelling
  - cyanosis of the entire upper extremity
  - Prominent venous collaterals over shoulder & anterior chest wall.
- Treatment:
  - Similar to that of lower limb DVT
  - Resection of cervical rib if it kinks the vein.

Pulmonary Embolism (PE)

- 2-3**%** of all hospital mortalities are due wholly or in part, to pulmonary embolism.
- Etiology: usually 2^{ry} to DVT or other thrombi in the venous circulation
- Pathophysiology:
  - Effects of PE occur when**>** 25% of pulmonary artery circulation is occluded.
  - Cardiac output ↓↓ when pulmonary artery occlusion > 50%.
  - Underlying cardiac or respiratory insufficiency contributes to premature failure of cardiac output. Decreased pulmonary blood flow results in:
    1) ↓ cardiac output (CO) → systemic hypotension & shock.
    2) High V/Q ratio due to reduced circulation in normal ventilation areas.
    3) Myocardial hypoxia.
    4) Pulmonary infarction in < 10% of PE cases depending on completeness of occlusion & effectiveness of collateral circulation.
- Clinical picture: according to the site where the embolus impacts.
  - Small emboli: usually **Silent**, Impacted in peripheral arterioles
    Recurrent small emboli → **pulmonary hypertension**.
  - Medium sized emboli: lodge in branches of the pulmonary arteries
    → **pulmonary infarction**.
  - Large emboli: Impacted in the main pulmonary artery or one of its major branches→ **massive pulmonary embolism**.
    - Symptoms: severe precordial pain, chest tightness, marked dyspnea, severe hypotension & marked tachycardia. Sudden death may occur.
    - **Unexplained dyspnea or heart failure appearing in a hospitalized patient is very suggestive of P.E.**
- Differential diagnosis:
  - Pneumonia
  - Congestive heart failure
  - M.I
  - ARDS
- Investigations:
  - Of the cause:
    - Duplex of L.L veins
  - To diagnose:
    - **D-dimer:** ↑↑ in PE &DVT (fibrin degradation product)
    - **Blood gases:** Low Pa02 & normal PaC02.
    - **ECG:** differentiate PE from M.I.
    - **Chest X-ray:** normal in 50% of cases:
      - Diminished vascular markings.

- Prominent pulmonary artery *(hilar shadow)*.
- Enlarged right ventricle.
- Small pleural effusion.

- **Ventilation perfusion pulmonary**
- **isotope scan V/Q scan:** show lung areas ventilated but not perfused.
- **CT pulmonary angiography Most popular imaging study, Non**-invasive & **No** vascular catheter.
- **Pulmonary arteriography:** abrupt vessel cut-off or intraluminal filling defect.
  - **Disadvantages:**
    1. dangerous in shocked patients.
    2. micro emboli might be overlooked



❖ Treatment:

- **Mild pulmonary embolism (low risk):** NO right ventricular strain → heparinization for 10 days then warfarin.
- **Submassive:** Rt ventricular strain detected by echocardiography & increased troponin level.
- **Massive:** Persistent hypotension < 90 mm Hg for 15 minutes.

✓ Treatment of submassive and massive cases in ICU:

1) Oxygen inhalation.
2) Cardiac support by dobutamine or epinephrine.
3) Pulmonary catheter thrombolytic therapy.
4) **Pulmonary embolectomy**: continuous deterioration after thrombolytic therapy.

✓ Prevention of recurrent P.E. **by Vena caval interruption** in:

1) Major bleeding complications?? anticoagulant therapy is contraindicated.
2) Recurrent embolism in spite of anticoagulant therapy.
3) First PE in high-risk patients.
4) Following pulmonary embolism.

Varicose Veins of Lower Limbs

❖ Superficial veins are **dilated, elongated & tortuous.**

❖ Types:

- Primary varicose veins (unknown cause)
  - Etiology:
    1. The weak wall theory: inherited weakness of vein wall →venous dilatation even with normal pressures, Secondary valvular incompetence occurs.
    2. The congenital valvular incompetence theory: **+ve** family history found in **50%** of cases of primary varicose veins.
    3. Aggravating factors:

- Female sex.
- High parity**.**
- Marked obesity.
- Estrogens intake.
- Occupations requiring prolonged standing.

➢ Secondary varicose veins These develop secondary to:

- Deep venous thrombosis: most common cause of 2ry V.V, Recanalization of the thrombosed deep veins leaves the valves of the perforating veins incompetent leading to reflux of blood. This puts unusually high strain on the superficial veins which have little external support → progressively dilate.
- Arterio-venous fistula: Varicose veins are prominent with traumatic or congenital (Klipple Trenaunay syndrome) A-V fistulae, Arterial pressure is transmitted to the deep system leading to valvular incompetence & reflux of blood to the superficial veins which dilate.
- Pelvic tumors and pregnancy: compression of the pelvic veins

❖ Clinical picture:

➢ Symptoms: 2ry > 1ry

- Cosmetic disfigurement.
- Dull, heavy, bursting sensation of the leg usually at end of the day or on prolonged standing, **relieved by elevating the limbs**.
- Mild swelling at the end of the day, particularly with secondary VV.
- **Pigmentation** due to ooze of blood in the subcutaneous tissue & deposition of hemosiderin.
- Night cramps.
- Itching.
- Ulceration.

➢ Signs: Aim of examination

1) The anatomical distribution of the veins.
2) Type of the varicosities? **1ry or 2ry**
3) Competency of saphenofemoral junction & communicating veins.
4) The condition of the deep system of veins.
5) The presence of complications.

✓ Patient examined while standing & exposed up to the umbilicus

- Varicose veins appear as elongated, dilated and tortuous veins.
- Veins may belong to the long or short saphenous system in **1ry v.v.** or may be arranged haphazardly in case of **2ry v.v.**
- Presence of veins crossing the suprapubic region denotes **2ry v.v.** caused by narrowing of the external iliac vein.
- Presence of complications as edema, eczema, liposclerosis or ulceration is in favor of **2ry v.v.**
- Palpation of a thrill over the veins denotes an arterio-venous fistula.
- Palpation of a thrill over the saphenofemoral junction on cough denotes an incompetent saphenofemoral junction.

✓ **Special Tests**

- **Multiple tourniquet test:** Rapid filling in any segment indicates the site of regurgitation.
- **Trendelenburg:**
  - Detection of saphenofemoral incompetence
  - Detection of incompetence of communicating
- **Perthe's & Modified Perthe's tests:** Detection patency of deep venous system.

❖ Complications:

- Oedema
- Venous ulceration
- Subcutaneous bruises due to rupture of small veins as result of venous hypertension.
- Itching, dermatitis &eczema are due to deposition of hemosiderin in the subcutaneous tissues.
- Liposclerosis: Extravasated fibrinogen → fibrous tissue Soft subcutaneous fat is replaced by tough fibrous tissue.
- Recurrent superficial thrombophlebitis.
- Chronic leg pain.
- Post-phlebitic syndrome: include all above symptoms as they follow previous DVT with incompetent perforating veins
- Hemorrhage may occur due to rupture of a varicose vein especially when the overlying skin is thin. It can be initially stopped by elevation & compression bandage. Later treatment is by injection sclerotherapy.

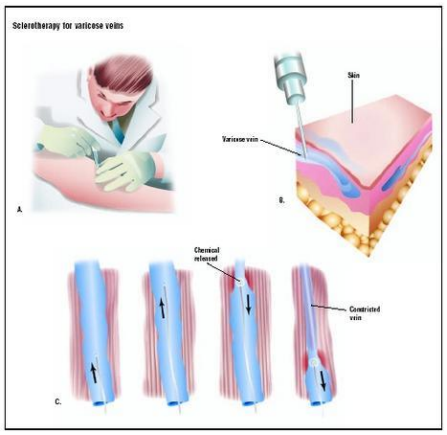
✓ **Post- phlebitic syndrome**

- History of DVT.
- Lower leg eczema and pigmentation.
- Leg pain on standing relieved by elevation.
- Lower leg chronic ulcer
- Leg edema on standing & by end of day.
- Small varicosities of irregular distribution.

❖ Types:

	1ry v.v.	2ry v.v.
Etiology	Idiopathic	previous DVT, A-V fistula pelvic tumors or pregnancy.
Pain	Slight or absent	Marked
Distribution	Along the long or short saphenous veins	Haphazard. (Veins may cross the groin)
Complications	Minimal or absent	Oedema, pigmentation, eczema & frequent features

- Investigations:
  - Aim:
    a) Detect sites of incompetent communicating veins
    b) Verify patency of the deep veins.
  - Duplex ultrasound: accurate detection of incompetent perforators & detection of accompanying deep vein thrombosis in suspected cases.
- Treatment:
  - Primary varicose veins
    - Conservative treatment:
      - Reassurance & elastic stocking
      - Indication: Minor cosmetic or spider varicosities.
    - Injection sclerotherapy:
      - Indications:
        - Small unsightly veins.
        - Localized dilated superficial veins.
        - Incompetent lower leg perforators.
        - Recurrent or persistent veins after operation.



      - Principle:
        - **Sclerosants** like polidocanol should be injected in an empty vein to induce injury of the endothelial layer of intima.
        - **Compression** applied by elastic bandage & left for 4-6 weeks (then Immediately, the patient is instructed to walk for a long distance in order to flush any amount of sclerosant that might have reached the deep veins)

    ✓ Walls of the vein will adhere together →permanent occlusion of the vein

      - Complications:
        - **Extravasation** of sclerosant material under the skin leads to sloughing of the skin & poor cosmetic results.
        - **Deep venous thrombosis** may occur if a large amount of sclerosant material reaches the deep system undiluted so no more than **1 ml** is injected at any point.

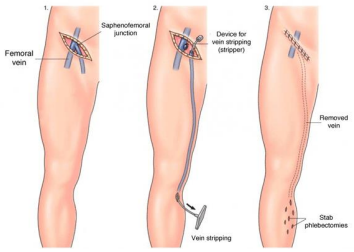
    ✓ **NO** injection is done for veins above the knee.

    - Surgery:
      - Indication: Patients with long or short saphenous incompetence or both are treated by saphenofemoral or saphenopopliteal disconnection combined with stripping.
      - Technique:
        - **Trendelenburg operation** (ligation of the saphenofemoral junction) Saphenous vein should be disconnected flush with the femoral or popliteal vein & all tributaries must be ligated & divided to prevent recurrence, The long saphenous vein is then stripped from above the knee to groin, The leg part of the vein is better not stripped to avoid saphenous nerve injury. The short saphenous is stripped from lateral malleolus to popliteal region.

- **Endovenous laser or radiofrequency current** used for obliteration of sapheno-femoral junction & medium sized incompetent perforators.

➢ Secondary varicose veins

- **Post phlebitic:** (i.e., following deep venous thrombosis)
  - Conservative: → **Elastic stockings**. Rarely the varicosities are large enough to require active treatment, **verify** that the deep system is recanalized, then treat as primary VV.



- **Varicose veins complicating AV fistula:** Surgical treatment of the fistula is usually followed by marked regression of varicosities, if residual veins remain, treat as primary varicose veins.
- **Varicose veins occurring during pregnancy:** A complete elastic stocking from the toes up to the groin all through the period of pregnancy. **After delivery** residual veins are treated as primary varicose veins.

Venous Ulcers

❖ Etiology:

A. Postphlebitic limb due to previous deep vein thrombosis.
B. Arterio-venous fistulas.
C. Complication of 1ry v.v. (transient & heals rapidly)

❖ Pathology:

➢ Recanalization of DVT is commonly followed by dysfunction of valves of both the deep veins & the perforating veins.
➢ Skin of the lower part of the leg is drained directly by the Perforators →drain into deep system →↑ venous pressure in medial side of lower 1/3 of leg **«ulcer bearing»** area.
➢ Combination of venous hypertension, liposclerosis & eczema will lead to ulceration after minor trauma.
➢ **Local hypoxia** due to venous stasis & impaired nutrition liberation of **free oxygen radicals** → toxic to the tissues.

❖ Clinical picture:

➢ Site: Ulcer bearing area or around medial malleolus.
- Skin around ulcer shows pigmentation, eczema with itching marks & induration ± 2ry varicose veins.
- Ulcer takes a long time to heal & liable to break down

❖ Treatment:

➢ Conservative: indicated for all cases.
1) Elevation of the foot in bed.
2) Ulcer care (Moist **saline** dressings twice weekly)
3) Elastic stocking or crepe bandage (Compression is the ***most important***)
- Medications to accelerate healing:
  1) Pentoxiphylline (Trental).
  2) Prostaglandin E1 analogue.

3) Diosmin.

- ✓ **Topical antibiotics** should not be used. may aggravate the condition by inducing allergy
- ✓ Conservative treatment is usually successful in allowing the ulcer to heal within a few weeks. The problem is that, once the patients return to normal activity, most ulcers will recur. If there is evidence of incompetent perforators in the leg, they can be either injected or excised.

➢ Surgical: open or endoscopic subfascial ligation of perforators, are obsolete now & NOT performed

Lymphatic System

Lymphadenopathy

- Definition: lymph node enlargement
- Etiology:
  - Inflammatory:
    - Acute: non specific and specific as infectious mononucleosis
    - Chronic: non specific and specific as T.B and syphilis
  - Metastases
  - Lymphomas:
    - Hodgkin's lymphoma
    - Non-Hodgkin's lymphoma
  - Blood diseases:
    - Acute leukemia
    - Chronic myeloid leukemia
    - Chronic lymphatic leukemia
  - Lipidosis:
    - Gaucher's disease.
    - Niemann-Pick's disease
    - Hand-Schuler-Christian's disease.

✓ **Functions of lymphatic system**

1.Uptake, transportation, & return of fluid, foreign substances &macromolecules from the interstitial space to the systemic circulation.

2. Protection of the host by a filtration system that resists infection & impede the spread of neoplasm.

3. Secretion of T cells, B cells, cytokines & vascular endothelial growth factor that stimulate Lymphangiogenic

Acute Lymphangitis

- Definition: Inflammation of lymphatic vessels
- Etiology: commonest organisms are streptococci
- Clinical picture:
  - General Fever, rigors & general constitutional disturbances.
  - Local pain, edema & red tender streaks.
  - Regional LNs are enlarged & tender.

- Complication: complete obliteration of the affected lymphatics & if the condition is repeated or involves major number of lymphatics draining an organ or a limb → permanent edema or elephantiasis.
- Treatment:
  a) Treatment of the cause.
  b) Antibiotics, especially penicillin & broad-spectrum antibiotics.

c) Local rest of the affected part & local heat to help resolution.
d) If suppuration occurs, it needs an incision.

Acute Septic Lymphangitis

- Definition: Inflammation of lymph Nodes
- Site: Along the lymphatics from inflamed focus
- Clinical picture:
  - The picture of the causative lesion.
  - General constitutional manifestations.
  - Locally the nodes are enlarged, red, hot, tender, firm or soft & if suppuration occurs→ fluctuation.
- Complications:
  1) Spread to proximal LNs
  2) Spread to nearby tissue
  3) Suppuration → abscess
- Treatment:
  a) TTT of the causative focus.
  b) General rest & antibiotics.
  c) If an abscess form → incision & drainage.

Chronic Non-Specific Lymphangitis

- Site: Submandibular & inguinal LNs.
- Clinical picture: Nodes are slightly enlarged, mobile, tender and firm in consistency.
- Treatment: treatment of the cause
  If persists for > 3 or 4 m, TB must be excluded.

T.B Lymphangitis

- Types:
  1. lymph borne (common in young).
  2. blood borne (in elderly).
- Site: Lymph-borne (fibrocaseous type) (the commonest)
  1) Cervical LN groups (commonest) the organisms reach from the tonsils
  2) Mediastinal & axillary groups (esp.in children): ± T.B lesions in the lung.
  3) Abdominal LNs (children & adolescents): Organisms from the ingested milk pass through the lacteals to reach LNs without affecting intestinal wall.
  4) Tabes mesentrica (>50y) common finding in x-ray is multiple mottled calcific shadows of mesenteric LNs (old T.B nodes healed by fibrosis & calcification).
- Pathophysiology:
  a) The organisms reach the LNs by afferent lymphatics thus, reaching the capsule 1st → T.B peri adenitis → matting of the nodes.
  b) Then the cortex will be affected & finally the medulla.
  c) Multiple tubercles will form, coalesce together, may caseate & break down → cold abscess that may rupture through the skin → T.B. sinus or ulcer.

❖ Clinical picture:

C/P	Lymph borne type	Blood borne type
Incidence	Common	rare
Age	Children	Usually elderly
Local signs	a. Localized lymphadenopathy affect upper deep cervical LNs. b. **Variable** consistency. • Early → Firm • Caseation → Cystic • Calcification → Hard c. LNs. are **matted** together. L.N.s. may be arranged in beaded cords due to thickening of connecting lymphatics.	a. Generalized lymphadenopathy b. **Firm** in consistency. c. LNs are **discrete**. As pt is usually an adult of fair resistance, there is no breaking down, caseation or cold abscess.
Caseation	cold abscess → T.B. sinus.	No cold abscess. No sinus.

❖ Complications:

- Caseation: may burrow through the deep fascia or an overlying muscle → bilocular (collar stud abscess).2ry infection may occur → difficult treatment
- Cold abscess: is actually neither cold (clinically warm) nor abscess (contents are not pus but caseating material).
- Sinus: with a thin cyanotic undermined edges & thin serous discharge.
- Spread to other groups of L.N.s: if the disease is left untreated.

❖ Investigation:

- Chest x-ray.
- Tuberculin test: good –ve indicator.
- Biopsy from the nodes → establish the diagnosis.
- Aspiration of cold abscess for microscopical pus exam
- Smears from a sinus & ex. for tuberculous organisms.

❖ Treatment:

- Tuberculous lymphadenitis before caseation:
  1. Good diet & vitamins.
  2. At least 2 antituberculous drugs (Rifampicin + INH) for at least 9 months.
  3. Surgical excision: for single LN group with no response to medical treatment after 6 M.
- Cold abscess:
  1. Antituberculous drugs.
  2. Aspiration & injection of streptomycin solution (to prevent sinus formation)
     - Needle is inserted in a healthy part of the skin away from the abscess.
     - The site of puncture should be in a non-dependent part.
     - The needle should pass in a Valvular manner (i.e., points of entry through skin & abscess cavity should not be opposite to each other).
     - Aspiration usually needs repetition every few days until abscess dries.
  3. Incision: Indicated in the following conditions:

- 2ry infection.
- If the abscess is imminent to rupture

4. Excision: if the nodes need excision.

➢ TTT of a tuberculous sinus:

1. Antituberculous treatment.
2. Dressing with streptomycin powder every 3 days until it closes.
3. If resistant to conservative measures → excise with underlying nodes

Chronic Lymphatic Obstruction (Lymphedema)

❖ Definition: Hypertrophic skin & SC tissues caused by chronic lymphatic obstruction.
❖ Sites: Limbs (L.L lymphedema /post-mastectomy U.L. lymphedema) **most common**
Others: scrotum, external genitalia & rarely breasts.
❖ Types:

Types	Lymphedema congenita	Lymphedema Praecox	Lymphedema Tarda
Incidence	**10%**	**80%**	**10%**
Age	At or within 1y of birth	Usually adolescence	After 35 years
Sex	M > F	F > M	M = F
Site	Commonly bilateral & involves whole leg	Commonly unilateral & below the knee	Usually unilateral

❖ Etiology:
➢ 1ry Lymphedema: congenital malformations of lymphatic vessels
➢ 2ry Lymphedema: Common
▪ Post-traumatic:
a) Injuries as circumferential skin loss.
b) Operations as Extensive block dissection of regional nodes.
c) Irradiation of the regional LNs.
▪ Parasitic: Filariasis is the commonest cause in Egypt.
▪ Post-Inflammatory:
a) Recurrent non-specific lymphangitis
b) Recurrent cellulitis e.g., interdigital infection &chronic leg ulcer.
c) post-erysipelas lymphoedema.
▪ Neoplastic:
a) Primary affection as lymphomas.
b) Secondary affection by metastases.

❖ Pathology: Pt with lymphatic obstruction will suffer from 2 problems:
➢ Lymph stagnation → accumulation of large amount of protein rich fluids (irritant) tissues → FB reaction → fibrosis in SC tissues → more obstruction.
➢ Recurrent attacks of lymphangitis → more obstruction → vicious circle.
➢ Pathological changes
1) Swelling: 1st due to accumulation of fluids & later due to fibrous tissue.
2) Edema: pitting then later, non-pitting (fibrosis hardens the skin).
3) The skin changes: negligible in 1ry lymphedema but in long standing cases, there is thickening & hyperkeratosis. Lymphatic vesicles may appear in the skin. In severe cases, the skin develops huge bulges (elephantiasis).

- Clinical picture:
  - Type of patient: according to history of the cause e.g., in filarialiasis adult pt from an endemic area (e.g., Giza, Imbaba) complaining of progressive leg swelling with exacerbations & partial remission.
  - During exacerbations: (Attacks of streptococcal lymphangitis) the limb shows red tender streaks traveling to draining LNs.
  - The swelling: is hard non-pitting with a crease in front of the ankle.
  - In late cases:
    a) Skin may show blebs, which may rupture → superficial ulcers oozing lymph (lymphorrhea)
    b) Skin is dark, thick with multiple deep furrows, thick callosities & warty over growths (Elephantiasis).

  - The inguinal LNs: may be enlarged, firm & tender (chronic septic lymphadenitis) or may form a soft lobulated mass (varicose LNs).
  - Other filarial manifestations: as lymphedema of scrotum, filarial funciulo-epididymitis, chylocele (rupture of lymphatics in tunica vaginalis), chylous ascites & chylothorax.

Filarial lymphoedema of the lower limbs

- Most common cause in Egypt is infection with **Wuchereria bancrofti.** Disease is endemic in Egypt in areas near the Mediterranean, e.g., Damietta & in areas at the desert edge e.g., Giza, Sharkiah & Assiut.
- Etiology: obstruction of the lymph nodes by adult worms leading to stagnation of lymph & dilatation of the lymph vessels.

- Investigation:
  - Mid-Night blood film: for microfilaria.
  - Intradermal skin tests.
  - Lymphoscintigraphy a nuclear medicine study of lymph vessels & nodes. Radio-labeled particles of protein are injected just under the skin.
  - Near infrared laser (fluorescence) imaging: uses indocyanine green imaged with infra-red fluorescence camera. Lymph vessels & nodes can be visualized. It is a dynamic study analysis of lymph flow.
  - Magnetic resonance lymphangiography by injection of contrast sub dermally to visualize lymphatic system (lymph vessels & nodes), to Evaluate lymphatic obstruction by size, function & dermal back flow
  - Ultrasonography to evaluate edema & fatty tissue in the limb
- ✓ Differential diagnosis of diffuse limb swelling:
  1. Postphlebitic limb
  2. Elephantiasis
  3. Neuro-fibromatosis
  4. Congenital A-V fistula (local gigantism)
  5. Lipedema
- Treatment:
  - Conservative treatment
    1) Limb hygiene Elevation, manual massage, exercise & weight loss.
    2) Compression garments ± intermittent pneumatic compression.
    3) Elastic stockings used permanently after reduction of size.

4) Diuretics(controversial).
5) Long-acting penicillin 1,200,000 units every 3 weeks
6) Diethyl carbamazine Active filarial infection**.**

- Surgical treatment → for severe cases
  1) Physiological operations to improve fault in Lymph drainage of limb.
     - **Indication:** early stages where imaging shows dilated lymphatic vessels.
     - **Types:**
       a) lymphovenous shunts
       b) lymphatico-lymphatic bypass to bridge the obstruction with lymphatic graft.
       c) Autologous lymph node transplantation to stimulate lymphangiogenesis.
  2) Excisional operations reduce bulk of the limb by eradication of swollen diseased tissues & skin with closure of the defect with skin flaps or grafts.
  3) Liposuction to treat residual swelling.

Lymphomas

- Definition: Malignant neoplasm that arises in LNs or extra nodal lymphoid tissue
- Types:
  1. Hodgkin's disease.
  2. Non-Hodgkin's lymphoma.
  3. Burkitt's lymphoma.

Hodgkin Disease

- Pathology:
  - Gross picture:
    - Site: lymph nodes & extranodal tissue in spleen, liver & bone marrow.
    - It usually starts in cervical lymph nodes.
    - Affected nodes are enlarged, discrete, rubbery, and have a pink color.
    - A large lymph node in the center with smaller lymph nodes around it.
  - Microscopically: there are variable densities of lymphocytes.
    - Diagnostic feature finding characteristic **Dorothy-Reed Sternberg cells**, Giant cells with even number of nuclei (2 or 4) arranged in a mirror image.

- Types:
  1) Lymphocyte predominance: Giant cells are rare, but there are abundant Lymphocytes & histiocytes. This type is the best prognosis.
  2) Nodular sclerosis: commonest type & characterized by broad fibrous bands that disrupt the lymph node architecture.
  3) Mixed cellularity: Heterogenous cellular component of eosinophils, lymphocytes, plasma cells & the giant Reed Sternberg cells
  4) Lymphocyte depletion: considerable fibrosis, variable amount of Sternberg cells & few lymphocytes Prognosis is the worst.
- Stages:
  - Stage I: Single involved lymph node group (I), or single extra lymphatic site (IE).
  - Stage II: Two or more involved lymph node groups limited to **one side** of the diaphragm or a solitary extra lymphatic site and one or more lymph node areas on the same side of the diaphragm (IIE).
  - Stage III: Involvement of both sides of the diaphragm.
  - Stage IV: Extra lymphatic spread including liver, lung, bone marrow, skin & gut.

✓ All stages are subdivided into either A →No systemic symptoms, Or B→One or more of 3 symptoms: fever, night sweats or weight loss > 10% in 6 M.

- Clinical Picture:
  - Incidence:
    1. Caucasians are affected by Hodgkin's disease more than the other races.
    2. Disease shows two age peaks, 1st between 15-35 y & other > 50 y.
  - Symptoms & Signs:
    1. Painless progressive enlargement of the **cervical** lymph nodes (**most common**). With progress of the disease other node groups in neck, axillae, groin, mediastinum & abdomen are affected.
       - ☆ These nodes are enlarged, discrete, non-tender & rubbery in consistency.
    2. Splenomegaly and hepatomegaly may also be present.

3. Some patients exhibit systemic manifestations in the form of fever, night sweats, weight loss, pruritus, anemia, & jaundice. Sometimes a characteristic intermittent fever which lasts for a few days followed by a remission for few weeks occurs (**Pel-Ebstein fever)**
4. Immediate pain in diseased areas after drinking alcoholic beverages.

❖ Investigations:
- ➢ Full blood picture:
  - Anemia
  - Eosinophilia or lymphopenia
  - High ESR.
- ➢ Alkaline phosphatase elevated in cases with bone or liver involvement
- ➢ Lactic dehydrogenase (LDH) & beta-2 microglobulin usually elevated
- ➢ Chest x-ray and CT scan detection of intrathoracic disease.
- ➢ Abdominal ultrasound and CT scan detecting intra-abdominal disease
- ➢ Lymph node biopsy under G.A. **(cornerstone of diagnosis)**
  - site of biopsy: **Neck** (Inguinal lymph nodes better avoided as they are commonly enlarged as a result of chronic nonspecific lymphadenitis)
- ➢ Bone marrow biopsy.
- ➢ Immunophenotyping technique of identifying molecules associated with lymphoma & leukemia cells and that help identify their subtypes.

❖ Treatment:
- ➢ Stage IA, IB, and IIA: **Radiotherapy** to affected &adjacent group of L.N.
- ➢ Stage IIB: **Radiotherapy** & **6** cycles of combination **chemotherapy**.
- ➢ Stage III and IV: **12** cycles of **chemotherapy**.

✓ **Radiotherapy** is used as a supplement to control bulky cervical nodes.
✓ **Chemo drugs**: **Doxorubicin, bleomycin, vinblastine and dacarbazine.**

❖ Prognosis:
- 80% 5-years survival with treatment.
- 100% for stage IA with lymphocytic predominance.

Non-Hodgkin Lymphoma (NHL)

❖ Classification: Each type the cells may be arranged in *nodular* or *diffuse*
- ➢ B-cell lymphoma (80-85%) is further classified into:
  - Small cell lymphoma
  - Mixed small & large cell lymphoma
  - Large cell lymphoma.
  - Immunoblastic lymphoma.
- ➢ T-cell lymphoma.
- ➢ Lymphoblastic lymphoma.
- ➢ Histiocytic lymphoma.

❖ Etiology: exact cause is **Unknown** but increased incidence with:
- ➢ Sjogren's disease & benign Lymphoepithelial lesions of salivary glands
- ➢ Immune deficiency AIDS & with prolonged immunosuppression.
- ➢ Human T –cell leukemia/lymphoma virus (HTLV-I).
- ➢ Systemic lupus erythematosus (SLE).

❖ Etiology:
- ➢ Lymph node enlargement is similar to that of Hodgkin's disease.
- ➢ Progression of Lymph node enlargement.

- NHL is more likely to affect **extranodal** sites than Hodgkin's lymphoma:
  - Gastric lymphoma produces manifestations that are similar to carcinoma.
  - Intestinal lymphomas → intestinal obstruction, bleeding or perforation.
- **Mycosis fungoides** is a variant of NHL affects skin & has eruptions.
- The disease is likely to be disseminated at the time of presentation.

❖ Treatment:

- Radiotherapy & combination chemotherapy according to the stage, Common drugs: cyclophosphamide, Adriamycin, Vincristine, bleomycin and prednisolone.
- Surgery Indication: Gastric & intestinal affection
  - Method: Gastrectomy & intestinal resection (Followed by radio & chemotherapy)

❖ Prognosis: Gastric lymphoma is better than Gastric adenocarcinoma.

Burkitt's Lymphoma

❖ Etiology: Unknown may be infection with EBV, Malaria may have a role to induce the disease.

❖ Pathology: malignant tumor of the **B**-lymphocytes.

❖ Type of patient: male **<**12 y in western Africa.

❖ Clinical picture:

- painless progressively enlarging jaw swelling.
- Distorts face, displaces eye & partially occludes the mouth.

❖ Treatment:

- **Chemotherapy:** Cyclophosphamide & cytosine arabinoside.

Leukemias

- Definition: Generalized enlargement of lymph nodes, spleen & liver with marked increase in total leucocytic count mostly made of **immature** forms.

Chronic Myeloid Leukemia

- Incidence: affects both sexes equally between the ages of 35 & 70 years.
- Clinical picture:
  - onset Insidious
  - Symptoms Anemia, weight loss, spontaneous hemorrhage
  - Signs:
    - Nodes are slightly enlarged & discrete.
    - Marked splenomegaly, hepatomegaly & generalized lymphadenopathy.
- Investigations
  - CBC progressive anemia with increased white cell counts up to 1000,000/uL, **80-90%** of them are immature with granular series.

Chronic Lymphocytic Leukemia

- Affects elderly people, especially males.
- Clinical picture: Moderate enlargement of all lymph nodes & lymphoid tissues, BUT SPLEEN is much smaller than myeloid leukemia.
- Investigation:
  - CBC shows moderate anemia with great increase in white count up to 500 ,000/uL, **80-90%** are immature lymphocytes.
- Treatment: Chemotherapy & biological therapy
- Prognosis: 5-year survival is about 80%.