# Atherosclerotic disease


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## Venous disease & Lymphedema - ILOs:

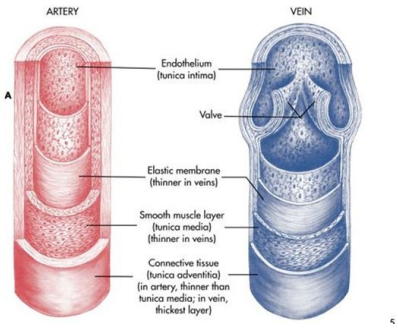
- State the normal anatomy of venous system of the lower limb
- Describe the pathogenesis, presentation, investigation, complications & management of varicose veins
- Describe chronic venous insufficiency of lower limb & its management
- State the etiology, diagnosis & management of DVT
- Describe prophylactic measures of DVT
- Describe etiology of primary & secondary LL lymphedema
- Describe the clinical features and management of lymphedema

**Color Index:**
**-Slides -Important -Doctor's Notes -Davidson's Notes**
**-Extra**


**Correction File** **Email: Surgeryteam434@gmail.com**

## Veins:

- Thin walled vessels & unidirectional valve.
- Transport deoxygenated blood from capillaries back to right side of heart
- Made of three layers
- Little connective tissue and smooth muscle makes veins more distensible
- Accumulate large volumes of blood - 70%




## Venous system of lower limbs:

| • Superficial veins: **It's important to know which system is affected for better management.** | • Deep veins | • Perforators |
|---|---|---|
| - Long saphenous venous system<br>- Short saphenous venous system | | |

## 1• Superficial veins:

### Long (Great) Saphenous System

- **From medial limb the dorsal venous arch to saphenous opening**

- sapheno femoral junction (SFJ) (**from the medial side extend to the groin to joins the common femoral vein and this is called SFJ**)

- **SFJ Tributaries: (Not important)**

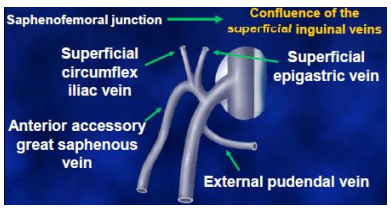
1. Superficial epigastric vien
2. Superficial external pudendal vein
3. Superficial lateral circumflex iliac vein

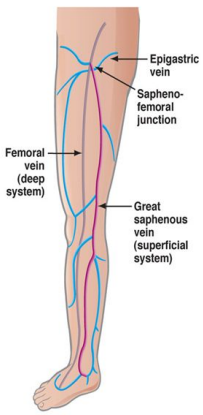
- **Thigh tributaries:**

1. Anterolateral Vein
2. Posteromedial Vein

- **Calf tributaries:**

1. Anterior arch vein
2. Posterior arch vein

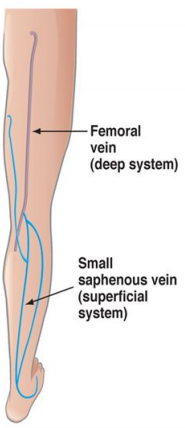



### Short (Lesser) Saphenous System

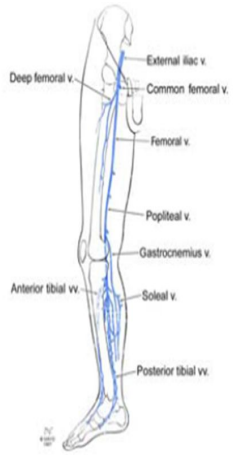
**(from the back of the leg starts from the lateral aspect of the frontal arch and joins the popliteal vein and this is called Sapheno-popliteal junction)**

- Branches: lateral & medial calf veins



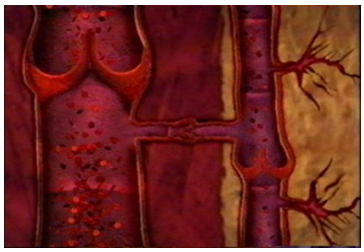
## 2• Deep veins

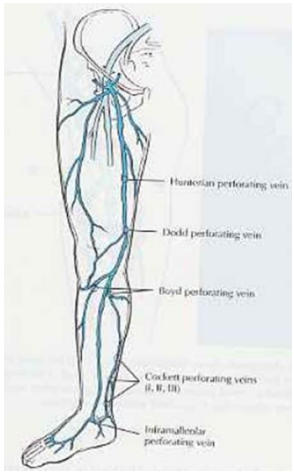
- Accompany arteries (And named as the arteries)
- Run with in muscles deep to the muscle fascia




## 3• Perforators (Communicating veins)

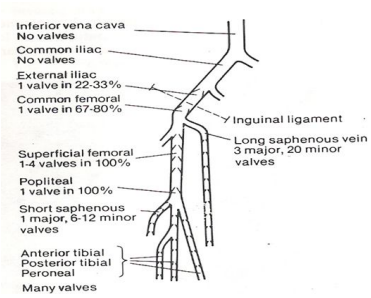
- Connect deep and superficial system (There is a lot of perforators around the ankle and knee
- Flow normally from superficial to deep
- Any abnormality in the valves will become incompetent, the blood will flow the opposite way for deep to superficial veins. So, because of the high density of perforators in the ankle then you have this clinical picture of chronic venous insufficiency, skin pigmentations and venous ulcer.
- Any pathology in the superficial veins or perforators causing chronic venous insufficiency this is known as primary chronic venous insufficiency.
- Any pathology in the deep veins such as DVT or tumor compressing the deep veins so this is known as secondary chronic venous insufficiency.

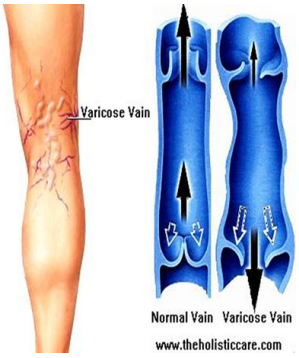




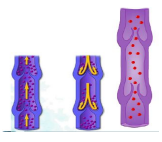
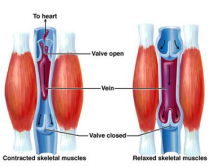

## • Valves

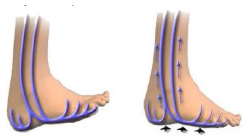
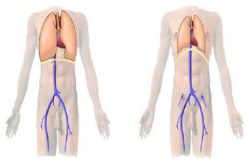
- These veins when coming from proximal to distal the valves will increase in number when going distally. The IVC has no valves and common iliac vein has valve and when you go down they will increase. Superficial veins have valves.



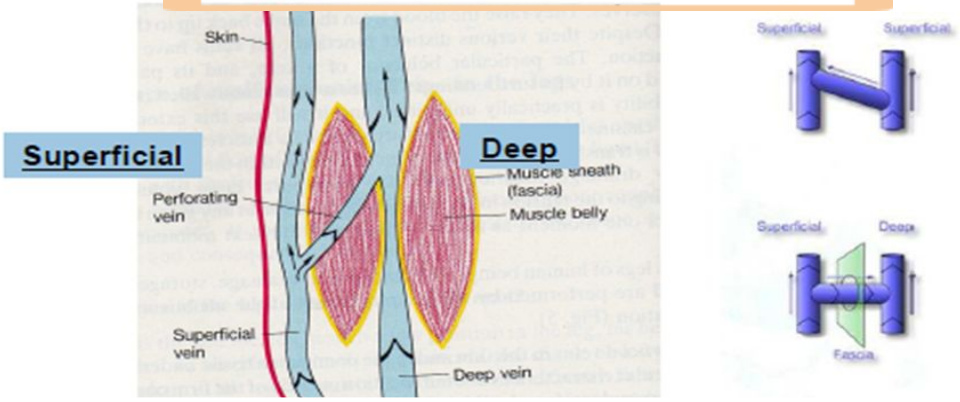

## Mechanism of Venous return

| | |
|---|---|
| **Unidirectional valves** | - Valves in veins<br>- One way valves prevent blood from flowing back wards & allow only movement toward the heart  |
| **Leg muscle pump** | - Calf-muscle pump<br>- 200-300 mm of Hg<br>- > 80 ml of blood<br>- Contractions propel blood towards heart<br>- Relaxation draws blood from superficial to deep veins  |

| | |
|---|---|
| **Foot sole & ankle pump** | - Veins in the sole of the foot are compressed<br>- The calf muscles can only function efficiently if mobility of the ankle joint is unimpeded<br>- Venous pressure > 100 mm of Hg<br>- Contributes > 50% blood leaving calf  |
| **Respiratory pump** | - Pressure changes induced in thoracic cavity by breathing sucks blood upward toward the heart<br>- In inspiration there is no venous return(+ve pressure) while in expiration there is venous return(-ve pressure which helps in venous return) because of the changes in the intra-abdominal and intrathoracic pressure  |

## Normal venous return flow

Blood flow upward toward heart & from the superficial veins to the deep veins




*** It's unilateral as mentioned in the picture and if this picture is reversed we then will have chronic venous insufficiency.**

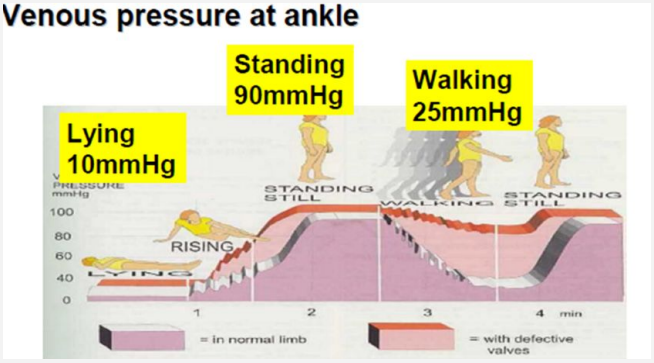
## Ambulatory Venous Pressure: (important)

• Minimal pressure in foot veins on **walking**

• Falls by 60-80% in few seconds

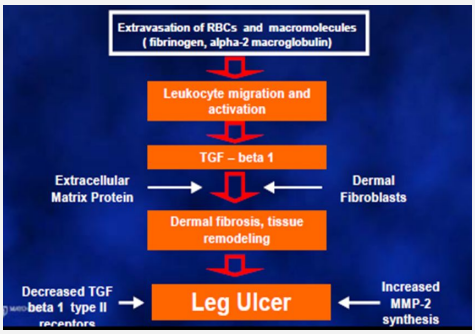
• 25 mm of Hg. And when you cross 30 mmHg and above this is when you can develop venous pressure.

- All the pathologies in the venous system of the lower limbs is due to abnormally high ambulatory venous pressure.




**Case: Young patient has venous ulcer or varicose vein and comes to you and you measure the pressure in the foot vein while ampulation. what do you expect the pressure:**

A. 0
B. 10
C. 100 because it's above 30 mmHg
D. 25




## Ambulatory Venous Hypertension

- Just to know that venous ulceration are caused by ambulatory venous hypertension by having an inflammatory response.

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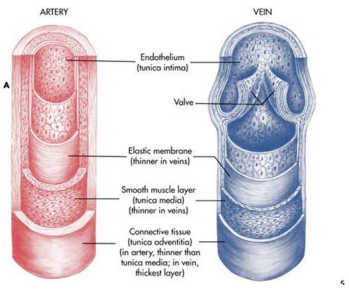
## Venous Disorders

### • Venous Thrombosis:

- **Superficial: Thrombophlebitis:** probably due to infections
- **Deep: Phlebothrombosis**

### • Chronic Venous Insufficiency: due to high venous pressure opposite to arterial disease where there is less blood flow (pressure).




## Chronic Venous Insufficiency (CVI)

### Chronic Venous Insufficiency (CVI):

- Valvular Incompetence
- Continued reflux
- Increased Ambulatory Venous Pressure
- Decreased Refilling Time < 10 sec
- More common in female

• CVI collectively describes the manifestations of impaired venous return due to abnormal venous system function
• Main defect may be in superficial, deep or perforating veins

- Primary :due superficial veins pathology related to structural weakness of valves or venous wall as in primary varicose veins Tx: Surgery or conservative.
- Secondary due deep venous pathology such as previous DVT as in post – phlebitis syndrome, a tumor blocking deep veins , AV fistula or Pregnancy . Tx: Treat the underlying cause.

**Case: Lady 30 years old teacher and mother of 6 children. She noticed varicose veins when she had her 3rd baby and with every pregnancy she had increase in varicose veins, limb swelling and pain. She denies any Hx. of DVT. Dx?** Dx. is she most probably has primary CVI. Because in the history she has no previous DVT and many other risk factors.

**Case: Young male in RTA that fractured his bones. He has been in a hospital for several weeks and immobilized during his stay he had DVT and Duplex scan was done to prove his lower limb DVT and has complications of PE. He was discharged the presented couple of months later with venous ulcer, and limb swelling. Dx?** Dx. is secondary CVI.

# Chronic Venous Disease: Varicose Veins

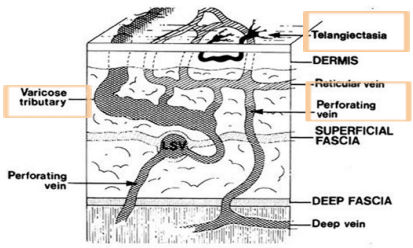
• Most common vascular disorder

• Incidence:

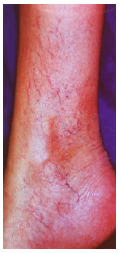
- Male : 10-15%
- Female : 20-25%

• When non saphenous varicosities (when the varicose veins are along the distribution of the long and short saphenous vein, and they are on the lateral and medial aspects and around the ankle) included:

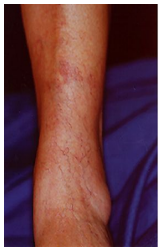
- Male : 45%
- Female : 50%



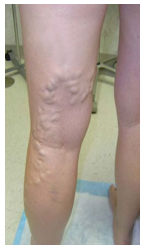

**- Telangiectasia:**
-In dermis (spider veins) and could be present in obese patients.
-Confluence of dilated intradermal venules less than 1 mm in diameter



**- Reticular veins:** (the abnormal ones)
-Dilatation of veins in subcutaneous tissue tissue that is not associated long or short saphenous veins
-Dilation bluish tortuous subdermal veins (subcutaneous) 1-3 mm in diameter



**- Varicose veins:** subcutaneous dilated , elongated , tortuous veins greater than 3 mm involving saphenous veins, saphenous tributaries or non saphenous tributaries



## Risk factors

- **Female gender**
- **Caucasian race**
- **Advanced age**
- **Family history**

## Accelerators

- **Pregnancy**
- **Obesity**
- **Professions that needs long standing, e.g teachers or nurses**
- **Oral contraceptives**

# Venous ulcers

**Theories of Etiology:** (will not be asked in the exam and the main cause is high ambulatory venous pressure)

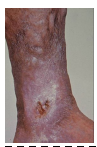
• Venous stasis and hypoxia (Homans)

• Arteriovenous fistula (Blalock, Haimovici, Pratt)

• Fibrin Cuff around capillary ( Burnand ): Barrier to oxygen diffusion and nutrient blood flow (

• Leukocyte trapping and activation (Coleridge-Smith) : Degranulation and endothelial damage, dermal hypoxia, venous ulceration.

- Painful ulcers near ankle
- Brownish pigmentation which precedes development of ulcer
- Lipodermatosclerosis (an inflammation of the layer of fat under the epidermis leading to loss of the soft feeling

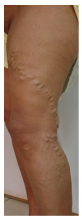
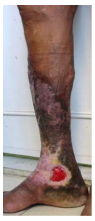


of the skin)

- Bleeding
- Superficial thrombophlebitis

* Location: we don't see venous ulcer in the foot, sole or toes.

* Ulcers are delayed manifestation while superficial veins like Telangiectasia is early.

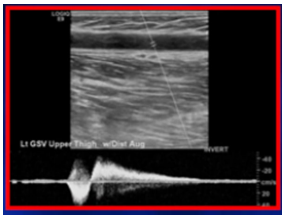
| | |
|---|---|
| **Clinical Evaluation** | 1-Asymptomatic:<br>Cosmetic<br>2-Symptomatic:<br>• Severity of symptoms and signs depend on the degree and duration of venous hypertension<br>• Pain:<br>- Throbbing, Aching. Stinging, Burning, Exercise – Variable effect on pain, Night pain – Cramping<br>• Itching<br>• Skin changes<br>• Complications<br>3-Effects of previous treatments |
| **Physical Examination *the patient should be both standing and supine position*** | - **Inspection:**<br>**1-Standing** Look for extent & distribution of Varicose veins and we mark it.<br>2-**Supine to** look for any ulcers(if +ve then describe it),skin changes ,limb swellings and venous emptying..<br>if you find the ulcer describe it<br>   |
| **Clinical tests** | • To know:<br>- Which system<br>- Which perforators<br>- Patency of deep veins<br>* All these tests are to assess the SFJ, no need to know them (will not be asked in the exam)<br>• Trendelenburg test<br>• Schwartz test (Cruvhellier's sign)<br>• Morissey's cough impulse<br>• Fegan's method (Phallen's test)<br>• Pratt's test<br>• Tourniquet test (Mahorne-ochsner}<br>• Perthe's test |

**Investigation**

**(Important)**

**Always start with Non-invasive**
Duplex or doppler

1- Doppler Examination:



- To assess flow and patency of veins
- Qualitative assessment of venous reflux ( if there reflux that means bidirectional)
- Evaluation of reflux in sapheno-femoral & popliteal junctions
- Does not give anatomic information ( you don't know which veins you asses)

2- Duplex Scan: (**near gold standard + very useful examination**)

- Direct detection of valvular efflux
- Physiologic reflux < 0.5 sec
- Pathologic reflux ≥ 0.5 sec
- Visualization of veins and valve leaflet motions and
- Quantify degree of incompetence
- Study of deep, superficial and perforator veins for patency and competency
- Most importantly is that the patient should be standing
- And the conclusion is finding the pathology if it's in the superficial or deep veins.

3- Plethysmography: (we don't do it anymore we just depend on the Duplex)

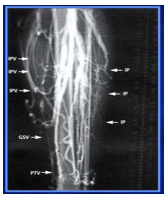
- Volume change of limb: secondary to changes in venous blood flow

4- Pressure Measurements: (we don't do it anymore we just depend on the Duplex)

- Transmural pressure
- Ambulatory venous pressure

AVP abnormal results if: Lack of sufficient drop: pressure doesn't decrease enough on walking and the difference between the standing and walking pressure is <50% Short venous refill time: It takes less than 20 seconds. (This means the blood is filling veins quickly and the valves aren't working efficiently to stop the blood from refluxing).

5- Invasive Procedures:



- Ascending venography(complications will be due to the contrast and locally like bleeding or injury to the surrounding structures or inflammation)
- Descending venography( Inject the contrast to IVC)
- CT Venography **the gold standard:** Contrast injected to visualize veins, not used much nowadays, due to its complications. But still has specific indication.
- MRV[1]

**Case: Patient has varicose veins he did venography then he developed painful swelling tender of the superficial veins. Dx?**

- Complication of the procedure.

[1] Magnetic Resonance Venography

## CEAP(سيب) Classification (will not be asked in the exam)

- Clinical
- Etiological
- Anatomical
- Pathophysiological

| | |
|---|---|
| **Clinical Classification** | - C0: no signs of venous disease<br>- C1: Telengiectasia & Spider veins<br>- C2: Varicose veins<br>- C3: Edema due to venous disease<br>- C4: Skin changes, Lipodermatosclerosis<br>- C5: Healed ulcer<br>- C6: Active ulcer |
| **Etiological** | • Congenital: EC **(the doctor didn't explain it)**<br>• Primary: EP **(the doctor didn't explain it)**<br>• Secondary : ES **(the doctor didn't explain it)**<br>- Post thrombotic<br>- Post traumatic<br>- Others |
| **Anatomic segments - 18** | • Superficial Veins As:<br>- 1: LSV **(Large Saphenous Vein)**<br>- 2: Above knee<br>- 3: Below knee<br>- 4: SSV **(Small Saphenous Vein)**<br>- 5: Non Saphenous<br>• Deep Veins Ad:<br>- 6: IVC **(Inferior Vena Cava)**<br>- 16: Muscular<br>• Perforator Veins As:<br>- 17: Thigh<br>- 18: Calf |
| **PATHOPHYSIOLOGIC** | - REFLUX: Pr **(the doctor didn't explain it)**<br>- OBSTRUCTION: Po **(the doctor didn't explain it)**<br>- REFLUX & OBSTR: Pro **(the doctor didn't explain it)** |

# Treatment Option

- **The treatment goal is reducing the Ambulatory venous pressure in primary venous insufficiency**
- **secondary venous insufficiency treat the underlying cause**

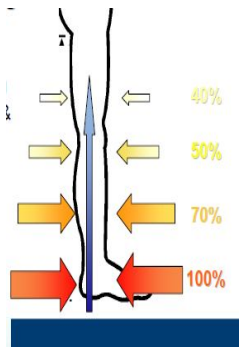
Principles of treatment:

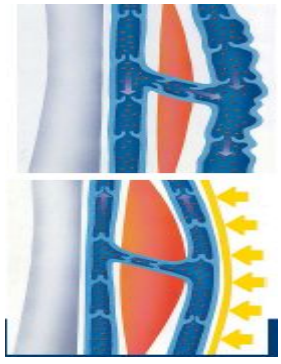
- ➢ Always exclude secondary causes by physical examination, history and investigations.
- ➢ Restoration of blood pumping towards the heart
- ➢ Remove the problematic vein (provided that there is another functioning vein draining the same area)

| | |
|---|---|
| • Conservative Treatment<br>• Compression garments<br>• Lifestyle and risk factors modification<br>• Skin care, ulcer treatment<br>• Phlebotropic drugs<br>• Compression Therapy | • Elastic compression:<br>- Bandage<br>- Stockings<br>• Paste gauze (UNNA) Boot<br>• Circaid orthosis<br>• Intermittent pneumatic compression |

## ★ Compression Therapy (indicated on both Primary and secondary CVI)

| Hemodynamic Effect: | Effects on tissues |
|---|---|
| • Compress the legs & superficial veins of leg<br>• Reduce vein caliber, helps the closing of valves<br>• Increase venous blood flow<br>• Decrease venous blood volume<br>• Reduce reflux in superficial & deep veins.<br>• Reduces pathologically elevated venous pressure | • Increase tissue pressure > edema<br>• Reduce inflammation<br>• Sustains reparative process<br>• Improves movements of tendon and joints and contraction of venous muscle pump |



| Class | Uses | Pressure At Ankle (mmHg) |
|---|---|---|
| I | Mild venous insufficiency veins | 14-17 |
| II | Treatment and prevention of venous ulcer recurrence | 18-24 |
| III | Treatment of severe venous hypertension and ulcer prevention in large diameter calves | 25-35 |

## ★ Pharmacologic Therapy

- Diuretics – limited use
- Zinc
- Fibrinolytic Agents

-Stanozolol - Androgeni steroid
-Oxypentiphylline - Cytokine Ant

- Phlebotrophic Agents:

1-Hydroxy - Rutosides 2-Calcium dobesilate 3-Troxerutin

- Hemorrheologic Agents

-Pentoxiphylline -Aspirin

- Free Radical Scavengers

-Topical Allopurinol -Dimethyl Sulfoxide

- Prostaglandins E & F
- Topical therapies
- Growth factors & Cytokines
- Skin substitutes Pharmacologic Therapy

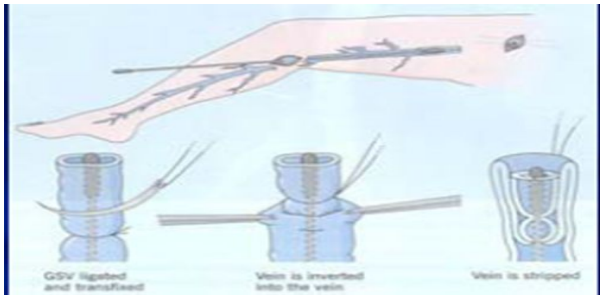
## ★ Sclerotherapy

Sclerotherapy is the injection of a sclerosing agent into a vein, causing an inflammatory reaction and destruction in the endothelium of the vein wall. The vein walls adhere together under compression and form a scar (fibrotic tissue) that is absorbed by the body. Remember this works only to the small veins not the big ones. Telangiectasias and reticular veins

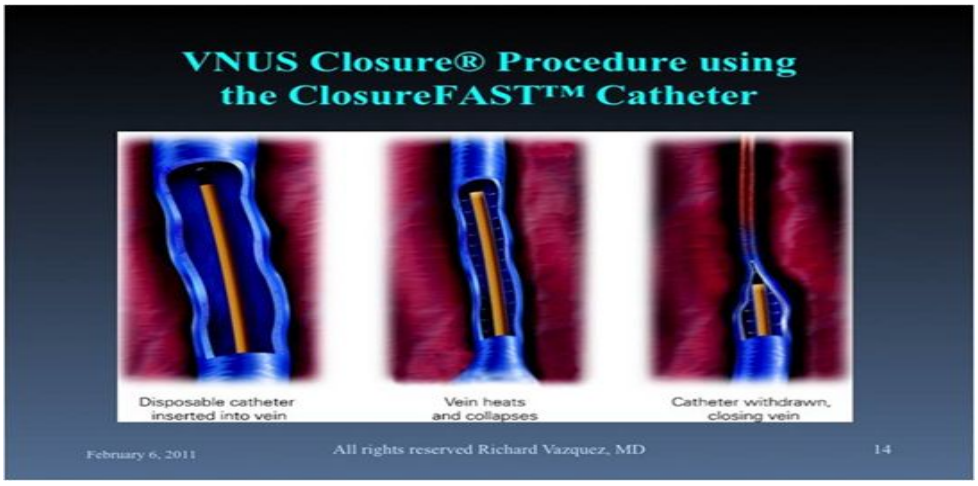
| Sclerosants | Detergents | Osmotic solutions | Chemical irritants |
|---|---|---|---|
| | ❖ Sodium tetradecyl sulfate (the most common use)<br>❖ Polidacanol<br>❖ Sodium morrhuate<br>❖ Ethnolamine oleate | ❖ Hypertonic Saline<br>❖ Hypertonic saline and Dextrose<br>❖ Sodium salicylate | ❖ Polyiodinated iodine<br>❖ Chromated glycerine |
| Microsclerotherapy | ❖ 30 G needle<br>❖ 0.1 – 0.25 Sodium tetradecyl sulphate<br>❖ Needs multiple sessions<br>❖ Needs compression therapy | | |
| Foam sclerotherapy | ❖ Tessari technique<br>❖ 1part of sodium tetradecyl sulphate and 4 parts of air agitated using two syringes | | |

## ★ Surgical treatment (gold standard)

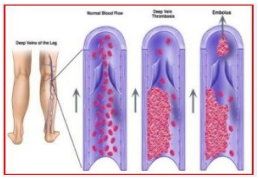
- ➔ Truncal Varicose veins (LSV and SSV) with incompetence
- ➔ Sapheno –femoral/sapheno-popliteal ligation & stripping of LSV/SSV
- ➔ Branch varicosities
- ➔ Avulsions via multiple stabs
- ➔ Incompetent perforators
- ➔ Individual ligation



| | |
|---|---|
| Endo venous laser surgery | ❖ Endoluninal obliteration by heat<br>❖ Induced collagen contraction and denudation of endothelium<br>❖ Fibrosis<br>❖ 810nm & 1470nm Diode laser |
| Radiofrequency ablation. | |




## Deep vein thrombosis (DVT) ( the doctor didn't go through all DVT )

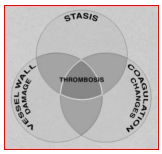


- ❑ In conjunction with Pulmonary Embolism ,DVT leading cause of in hospital mortality in USA
- ❑ 1 in 20 persons develop DVT in course of his or her lifetime
- ❑ Formation of semi solid coagulum with in flowing blood in any deep veins of body usually in lower limb and pelvic veins
- ❑ DVT originates in lower extremity venous system
- ❑ Starts at calf veins & progress proximally into popliteal, femoral, iliac veins and IVC
- ❑ 80-90% of PE originate here

## ★ Virchow Triad

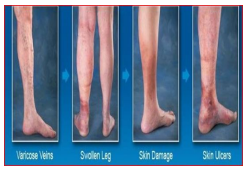
> 100 yrs ago Virchow described triad of factors for the development of venous thrombosis

- ➔ Alteration in normal blood flow
- ➔ Injury to vascular endothelium
- ➔ Alteration in constituents of blood




| Venous Stasis : | Hypercoagulable status : | Endothelial injury |
|---|---|---|
| • Advancing age<br>• Obesity<br>• Prolonged bed rest >4 days<br>• Immobilization<br>• Limb paralysis<br>• Extended travel | • Surgery, trauma, responsible for 40% TED<br>• Malignancy<br>• Increased estrogen levels – due fall in protein s<br>• Inherited disorders of coagulation<br>• Acquired disorders of coagulation<br>1. Nephrotic syndrome<br>2. Antiphospholipid Ab<br>3. inflammatory process<br>4. SLE | • Trauma<br>• Surgery<br>• invasive procedures iatrogenic |

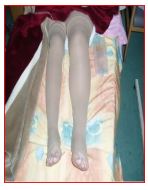
## Spectrum of Disease

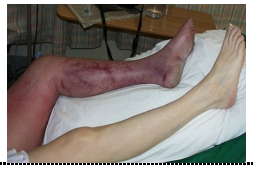
| Asymptomatic | Symptomatic | | |
|---|---|---|---|
| | Acute DVT limb | Pulmonary Embolism | Chronic Venous Insufficiency |
| | pain,edema,phlegmasia | Resp.distress, heart failure , Death |  |



## DVT Symptoms

- ➔ sudden Swelling
- ➔ Limb pain → pain on dorsiflexion of foot with knee flexed 90 degrees (Homan's sign)→ not sensitive or specific
- ➔ dilated veins, cyanosis,pallor
- ➔ Extensive thrombosis in thigh and pelvis may lead to Phlegmasia Alba Dolens → (inflammation white pain)
  1. Associated with arterial spasm
  2. Painful pale with poor or absent pedal pulses
- ➔ Massive iliofemoral DVT –complete impairment of venous outflow may lead to





cessation of arterial inflow

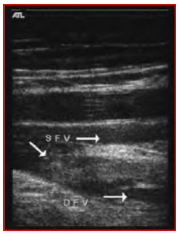
1. PHLEGMASIA CERULA DOLENS (inflammation blue pain)
2. Venous gangrene

| complications | |
|---|---|
| Immediate complications | Long term complications |
| ❖ PE 12-33%<br>❖ 200,000 deaths /year<br>❖ Leading cause of preventable in-hospital mortality | ❖ Post thrombotic syndrome<br>❖ Pain & swelling -67%<br>❖ Pigmentation – 23%<br>❖ Ulceration -5%<br>❖ 20-30% within 5yr valvular incompetence & luminal obstruction leads to more severe morbidity |

## Two-Level DVT Wells Score

| Clinical Feature | Point |
|---|---|
| Active Cancer (treatment ongoing, within 6 months or palliative) | 1 |
| Paralysis, paresis or recent plaster immobilisation of the lower extremity | 1 |
| Recently bedridden for 3 days or more or major surgery within 12 weeks requiring general/regional anaesthesia | 1 |
| Localised tenderness along the distribution of the deep venous system | 1 |
| Entire leg swollen | 1 |
| Calf swelling at least 3cm larger than asymptomatic side | 1 |
| Pitting oedema confined to the symptomatic leg | 1 |
| Collateral superficial veins (non-varicose) | 1 |
| Previously documented DVT | 1 |
| An alternative diagnosis is at least as likely as DVT | -2 |
| Clinical probability simplified score | |
| DVT likely | 2 points or more |
| DVT unlikely | <2 points |

| Diagnosis | |
|---|---|
| D-dimer Assay | ➢ Originally described in 1970s<br>➢ Fibrin fragment present in fresh fibrin clot & FDP of cross linked fibrin<br>➢ Marker for action of plasmin on fibrin<br>➢ Sensitivity 98.4%<br>➢ High - Does not confirm diagnosis due to low specificity<br>➢ Negative results rules out DVT<br>➢ Also elevated in trauma, recent surgery, haemorrhage, sepsis, cancer, pregnancy, liver disease |

| | |
|---|---|
| **Duplex** | ➢ Non invasive, Bedside exam<br>➢ 96% sensitive and 100% specific<br>➢ Normal veins –collapse with compression and lumen is free<br>➢ In DVT vein is dilated , lumen shows thrombus and noncompressible veins with poor or no venous flow  |
| **Venography** | ➢ Iodinated contrast injection into venous system<br>➢ DVT is visualized as filling defect<br>➢ Invasive<br>➢ Contrast complications<br>➢ Needs radiology suite |

## ACCP[2] Guidelines – Prevention Risk Stratification

| Risk | Patients |
|---|---|
| Low risk | Minor surgery in patients <40 years with no additional risk factors |
| Moderate risk | Minor surgery in patients with additional risk factors |
| | Surgery in patients aged 40-60 years with no additional risk factors |
| High risk | Surgery in patients >60 years |
| | Surgery in patients aged 40-60 years with additional risk factors (prior venous thromboembolism, cancer, hyper-coagulable state) |
| Highest risk | Surgery in patients with multiple risk factors (age >40 years, cancer, prior venous thromboembolism) |

## General Surgery Patients

| Patient group | Prophylaxis recommendation |
|---|---|
| Low risk, minor procedure | Early ambulation |
| Moderate risk, major procedure (benign disease) | LMWH |
| High risk, major procedure for cancer | LMWH |
| Multiple risk factors for VTE | LMWH plus mechanical* |
| High bleeding risk | Mechanical thromboprophylaxis |
| Laparoscopy, no risk factors | Early ambulation |
| Laparoscopy, with risk factors | LMWH, IPC or GCS |

[2] American College of Chest Physiciains

## Prevention

- 5000 units of heparin 2 hours preoperatively than every 12 hours postoperatively ( 5 days or until discharged)
- Twice or three times daily regimen :
  - ❏ twice has lower rates of bleeding complication
  - ❏ three times trend towards better efficacy in preventing VTE events
- LMWH Regimen Lovenox(Enoxaparin)

**Prophylaxis of DVT in abdominal surgery patients[6]**

| Patient type | Dosing | Duration of therapy |
|---|---|---|
| Abdominal surgery | 40 mg SC once daily (initiated 2 hours prior to surgery) | • Usual: 7 to 10 days<br>• Administered up to 12 days in clinical trials |
| Severe renal impairment (CrCl <30 mL/min) | 30 mg SC once daily | • Usual: 7 to 10 days<br>• Administered up to 12 days in clinical trials |

protects against DVT and PE. Treatment is continued until the patient is fully ambulant. In high risk patients, it can be continued following discharge and there is increasing evidence that this is of benefit in reducing venous thromboembolism and, probably therefore, the post thrombotic syndrome.

**Risk Assessment:**

Patients with limited mobility and at least one additional risk factor: consider VTE prophylaxis

| B. VENOUS THROMBOEMOLISM RISK ASSESSMENT | |
|---|---|
| • Acute medical illness<br>• Age >65<br>• Cancer (active or occult)<br>• Cancer therapy (hormonal, chemo- or radio-therapy, angiogenesis inhibitors)<br>• Estrogen-based OC or HRT<br>• Erythropoiesis-stimulating agents<br>• Immobility, lower-extremity paresis<br>• Indwelling central venous catheter<br>• Inflammatory bowel disease<br>• Inherited or acquired thrombophilia | • Myeloproliferative disorders<br>• Nephrotic syndrome<br>• Obesity<br>• Paroxysmal nocturnal hemoglobinuria<br>• Pregnancy and post-partum period<br>• Previous VTE<br>• Selective estrogen receptor modulators<br>• Smoking<br>• Surgery<br>• Trauma (major or lower extremity)<br>• Venous compression (tumor, hematoma, arterial abnormality |

| | Low Risk | Moderate Risk | High Risk |
|---|---|---|---|
| Population at Risk | • Mobile medical patients or minor surgery patient<br>• Perceived admit < 48 hrs | • Most general surgery patients<br>• Medical patients with limited mobility and one risk factor | • Hip or knee arthroplasty<br>• Hip fracture surgery<br>• Major trauma<br>• Spinal Cord Injury |
| Suggested Therapy | • No specific recommendation<br>• Order for ambulation<br>• +/- SCD's | • Heparin 5000 units SQ q8h<br>• Enoxaparin 40mg SQ daily<br>• +/- SCD's | • Enoxaparin 30mg SQ q12h<br>• Enoxaparin 40 mg SQ daily<br>• Heparin 5000 units SQ q8h<br>• +/- SCD's |

❖ Patients at moderate to high risk with bleeding risk should receive mechanical thromboprophylaxis

❖ Dosing adjustment should be considered for enoxaparin in patients with renal insufficiency (CrCl < 30ml/min) and should be considered in morbidly obese patients (BMI > 40 kg/m2)

## Treatment

### Anticoagulation

is the mainstay of initial treatment :

**TABLE 256-4 ANTICOAGULATION OF VTE**

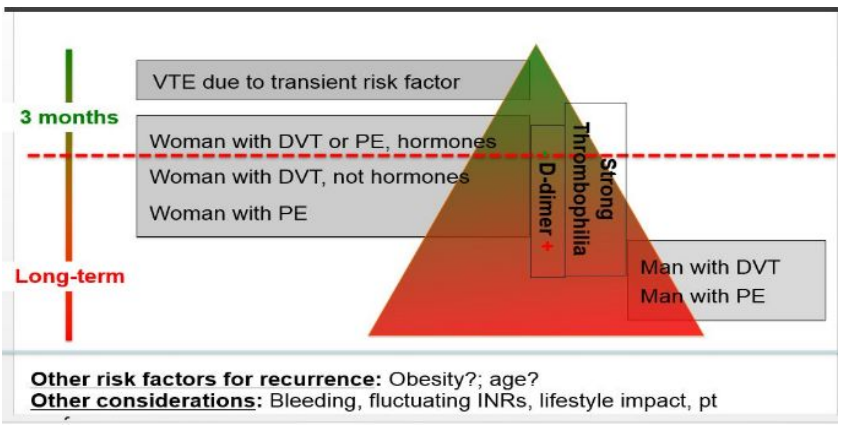
**Immediate Parenteral Anticoagulation**

Unfractionated heparin, bolus and continuous infusion, to achieve aPTT 2–3 times the upper limit of the laboratory normal, or
Enoxaparin 1 mg/kg twice daily with normal renal function, or
Tinzaparin 175 units/kg once daily with normal renal function, or
Fondaparinux weight based once daily; adjust for impaired renal function

**Warfarin Anticoagulation**

Usual start dose is 5–10 mg.
Titrate to INR, target 2.0–3.0.
Continue parenteral anticoagulation for a minimum of 5 days and until 2 sequential INR values, at least 1 day apart, return in the target range.

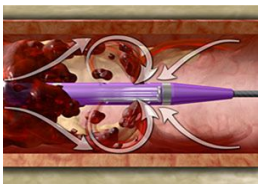
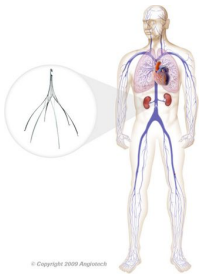
How long to treat with Anticoagulation




| Patient Group | Duration of anticoagulant |
|---|---|
| DVT/PE due to reversible factors | 3 months |
| 1st episode of unprovoked DVT/PE | 3-6 months vs.. long-term(2yrs) |
| 2nd episode of unprovoked DVT/PE | lifetime |
| cancer pts. with DVT/PE | 3-6 months minimum (until CA cleared) |

### Phlegmasia

★ Treat with thrombolysis or surgery,
★ anticoagulation may not prevent progression in these patients.
★ Limbs loss possible

| | |
|---|---|
| Catheter Directed Thrombolysis: | ★ iliofemoral DVT<br>★ symptoms <14 days<br>★ low risk of bleeding<br>★ have good functional status<br>★ reduce acute symptoms<br>★ reduce post thrombotic syndrome<br>➢ Post thrombolysis, balloon /stent for obstructing lesion<br>➢ Pharmaco-mechanical thrombolysis may shorten treatment time<br>➢ After thrombolysis, anticoagulate  |
| Vena cava filters indications: | ★ Patient cannot tolerate even a small pulmonary emboli<br>★ Anticoagulation contraindicated (intracranial bleed)<br>★ Anticoagulation fails to prevent embolization or extension of thrombus<br>★ Major bleed while on anticoagulation<br>★ After risk of bleeding resolves pt. should receive full course of anticoagulation  |
| | ★ Operative venous thrombectomy<br>1. Phlegmasia when thrombolysis contraindicated<br>2. Symptoms < 7days<br>3. Good functional status<br>★ Early ambulation<br><br>Leg elevation >> Compression stockings (30-40 mm of Hg) continue for 2yrs, longer if PTS symptoms |

## Lymphedema

Lymphedema (1-2 MCQ questions on this part)

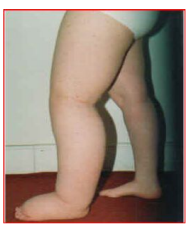
➔ Abnormal collection of interstitial lymph fluid due to either congenital maldevelopment of lymphatics or secondary to lymphatic obstruction

➔ It affects 2% of population causing limb swelling

➔ **It could be cause both unilateral or bilateral limb swilling**

➔ How to differentiate between the causes of unilateral limb swelling ??

1. lymphedema >>> swelling of the foot ( look at the picture: child with congenital lymphedema due to absence of lymphatic drainage or hyperplasia `` dilated lymph node `` )
2. infection ( such as cellulitis)
3. chronic venous insufficiencies (DVT) >>> no foot swelling



➔ **Patterns of lymphatic abnormalities:**

1-Aplasia -15% 2-Hypoplasia – 55%

3-Hyperplasia -35% 4-Dermal Backflow – Chylous Reflux

➔ **Predisposing factors**

1-Any inflammatory process 2-Trauma
3-Pregnancy 4-Puerperal sepsis (Postpartum infections)
5-Most of primary lymphedema appear spontaneously

## Etiology

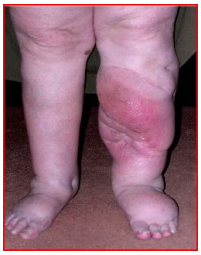
### Primary lymphedema

- Developmental error in lymphatic vessels
- Depending on the severity it appears ( MCQ )

1. At birth < year – lymphedema congenital – familial - Millroy's Disease
2. Between 1-35yr – lymphedema precox
3. Later >35yr - lymphedema tarda

### Secondary lymphedema

Trauma
infections
filariasis
post phlebitic limb
irradiation
malignancy
allergy.



**Cases :**

Child with normal delivery , mother notice limbs swelling with foot swelling , Arterial and venous examination were normal >> you suspect lymphedema

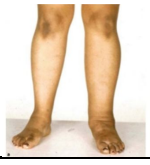
## Clinical Classification of lymphedema ( no body ask about it )

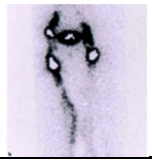
| | |
|---|---|
| **Subclinical (latent)** | **There is excess interstitial fluid and histological abnormalities in lymphatics and lymph nodes, but no clinically apparent lymphedema** |
| **I** | **Edema pits on pressure and swelling largely, or completely disappears on elevation and bed rest.** |
| **II** | **Edema does not pit and does not significantly reduce upon elevation** |
| **III** | **Edema is associated with irreversible skin changes, i.e. fibrosis, popillae** |

**Diagnosis**

➔ Lymphangiography
➔ Isotope lymphoscintigraphy (lymphoscintigram) the best

and first choice ,but can cause some complications.





| | | |
|---|---|---|
| | ➔ CT<br>➔ MRI<br>➔ Duplex Ultrasound | |
| **Complications important** | ➔ Local infection<br>➔ Systemic infection<br>➔ lymphangiosarcoma | |
| **Management:** | ➔ The patient should elevate the foot above the level of the hip when sitting, elevate the foot of the bed when sleeping, and avoid prolonged standing. Various forms of massage are effective at reducing oedema. Intermittent pneumatic compression devices are also useful.<br>➔ Diuretics are of no value and are associated with side effects, including electrolyte disturbance. No other drugs are of proven benefit.<br>➔ Early treatment is highly effective<br>➔ Late disease is difficult to treat<br>➔ Care to avoid any injury to limb<br>➔ Skin care to avoid infection<br>➔ Reduce swelling by decongestive lymph therapy<br>➔ Manual lymph therapy – massage<br>➔ Multilayer compression bandage & physiotherapy<br>➔ Intermittent pneumatic compression devices | |
| **Surgical treatment** | **Reducing Surgery** | **Bypass surgeries (best procedure)** |
| | ➔ Sistrunk - wedge removal & skin closure.<br>➔ Homans - Subcutaneous tissue removal & skin closure.<br>➔ Thompson - denuded skin sutured to deep fascia.<br>➔ Charles - remove every thing & cover raw area with skin graft | lymph venous shunts |

# MCQs.

Which of the following is the best initial test in Venous ulcer ?
A-Duplex ultrasound B-CT venography C-Ascending venography D-MRI

Which of the following is the first choice of diagnosis of lymphoedema ?
A-MRI B-Duplex scan C- lymphoscintigraphy D-Lymphangiography

What is the pathophysiology of CVI?
A-High ambulatory venous pressure B- Less blood flow C-Heart failure D-Venous obstruction

ANSWER :A, C, A