L15-Venous Diseases

Surgery Team
MED 433

MED 433

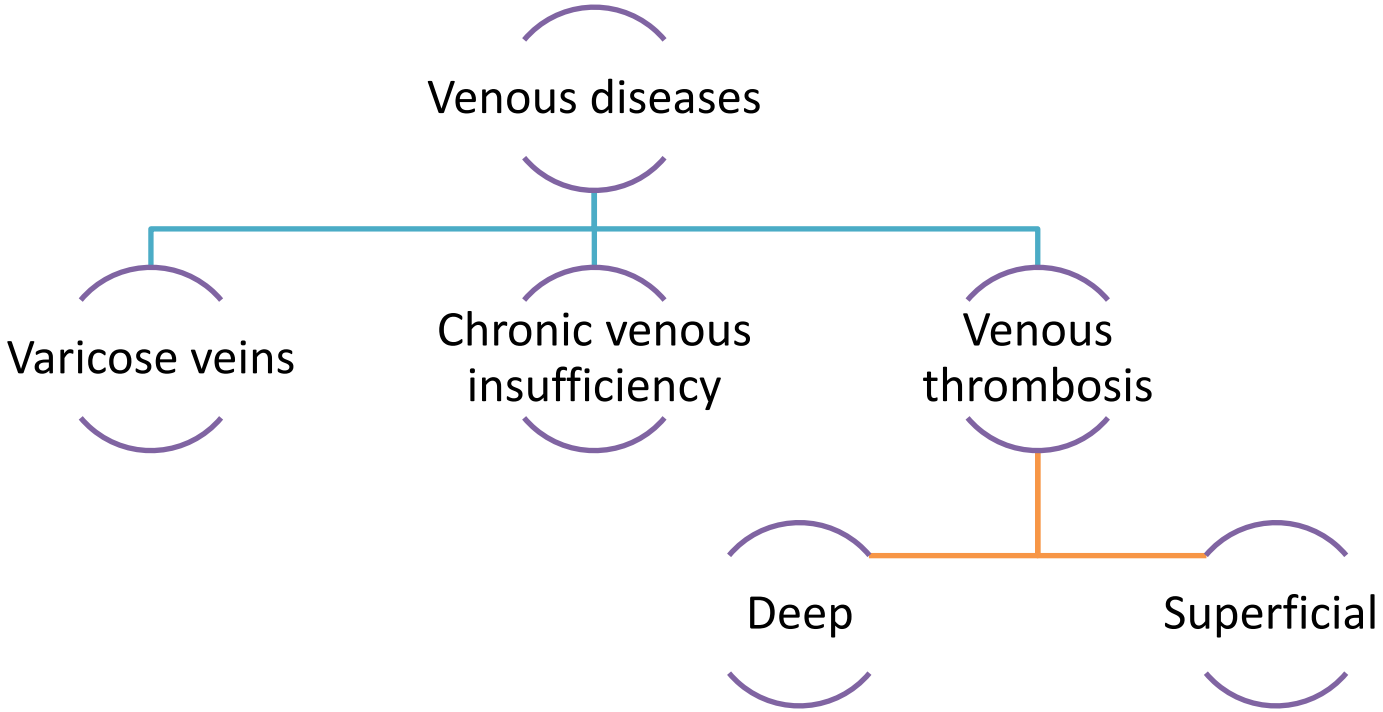
Objectives :

- 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 & Raslan's () | Doctor's Notes | Extra Explanation | Additional

This work is based on doctor's Slides +Notes and Raslan's only (Does not include the book)

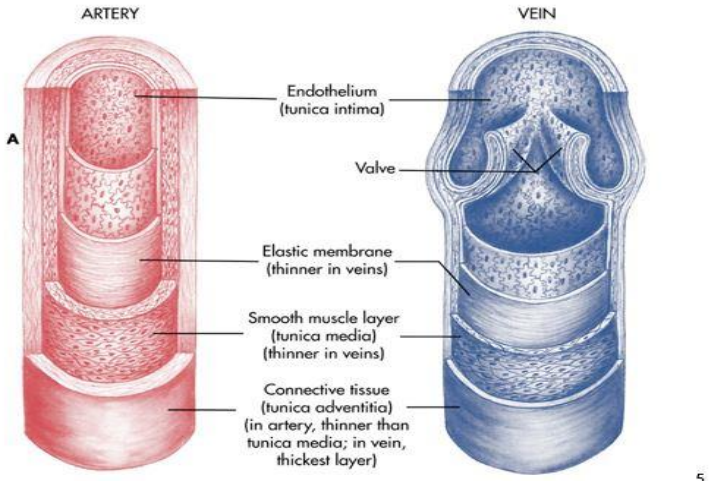
Mind Map



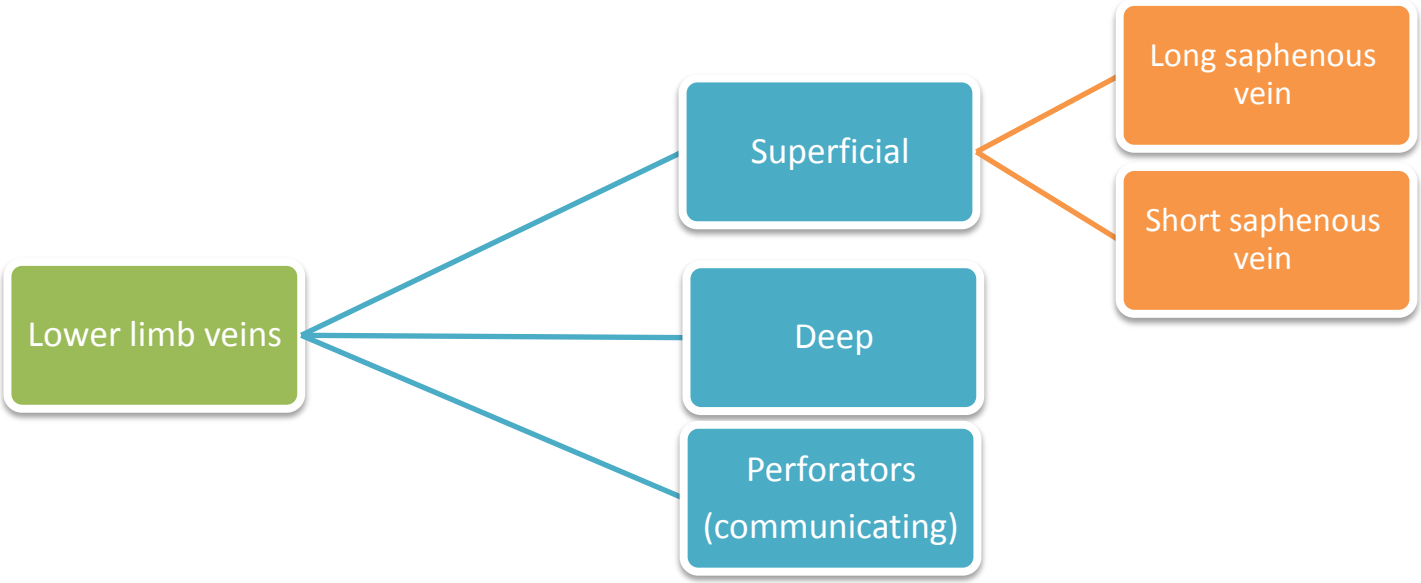
Please understand the next three slides to understand the rest of the lecture, although it's less likely to be asked about

1)Anatomy and Physiology of veins

- Veins are thin walled vessels, they transport deoxygenated blood from capillaries back to right side of heart.
- They are made of three layers (Just like arteries: intima, media and adventitia)
- They contain little connective tissue & smooth muscles making them more elastic and distensible, so they contain 70% of total blood without any increase in pressure.



Venous system of lower limbs



Cont. Venous system of lower limbs:

1/Long (Great) saphenous vein

- It runs medially in lower limb from the dorsal venous arch of foot till the saphenous opening in the Sapheno-femoral junction lies 2.5cm below and lateral to the pubic tubercle. (SFJ) to drain into the femoral vein.
- Tributaries of Great saphenous vein:

- SFJ Tributaries

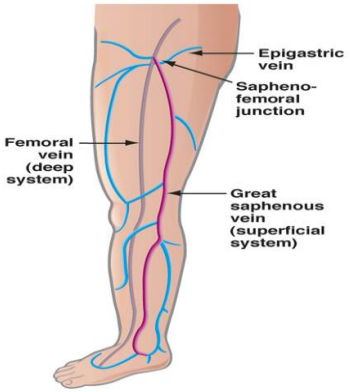
Superficial epigastric vein
Superficial external pudendal vein
Superficial lateral circumflex iliac vein

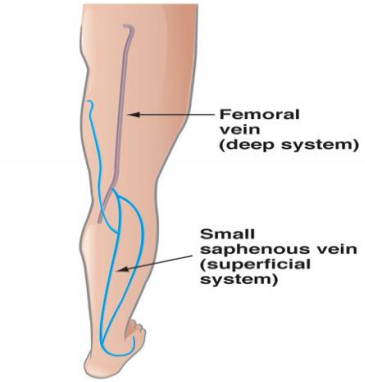
- Thigh tributaries

Anterolateral Vein
Posteromedial Vein

- Calf tributaries

Anterior arch vein
Posterior arch vein





2/Short (Lesser) saphenous vein

- It joins the Sapheno-popliteal junction
- Tributaries of Great saphenous vein:

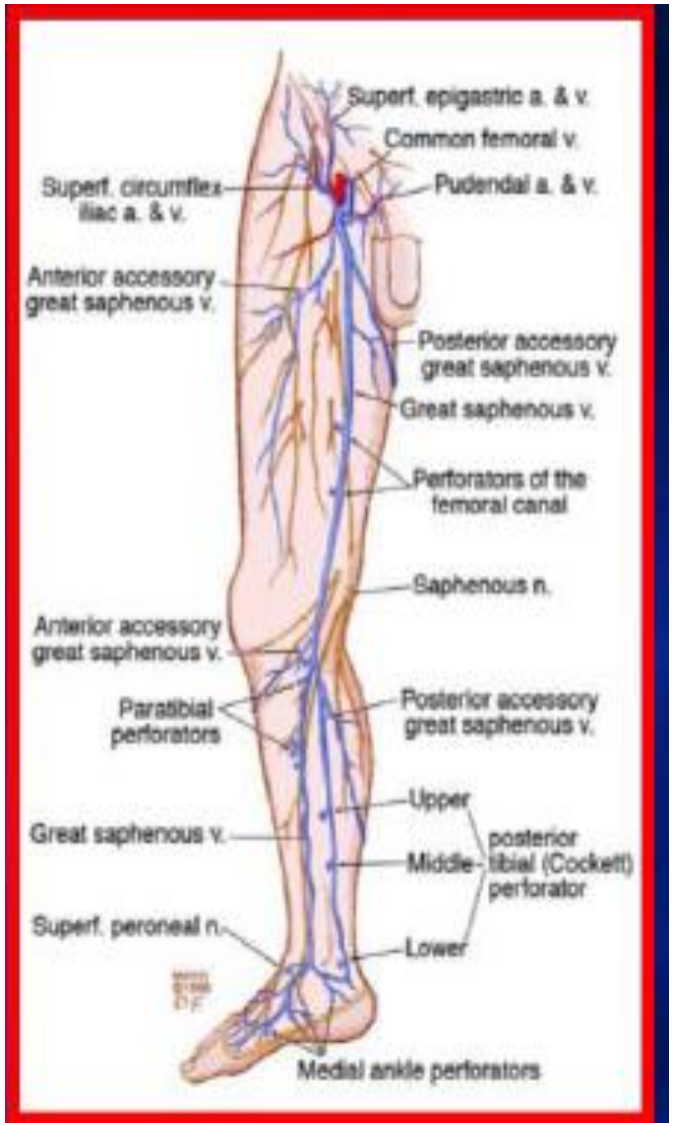
Lateral & Medial calf veins

3/Deep veins

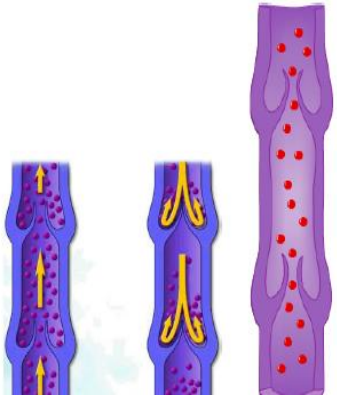
They accompany arteries and Run within muscles deep to the muscle fascia

4/Perforators (Communicating veins)

Flow the blood normally from superficial vein to deep vein



Cont. Venous system of lower limbs



Valves of veins:

Both superficial and deep veins contain valves that permit only one-way direction of venous blood flow (Toward the heart) and prevent backward reflux. (Note: There are **no valves** in SVC, IVC and iliac veins)

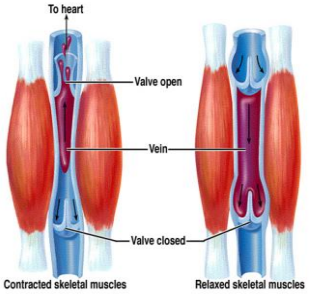
Venous cycle and venous return

Venous blood inside deep veins is pumped from lower limbs toward the heart by the aid of sole and calf muscles contractions, after which valves of deep veins close to prevent that blood from being refluxed back. When this occurs, blood from superficial veins passes to deep veins through perforators when muscles are relaxed, then cycle can be repeated again.

*Alternating pressures of the chest and abdomen during breathing:
o Chest is always negative in pressure. o But abdomen:
- In expiration diaphragm will go up creating a (–) ve pressure, which will lead to blood sucking to heart, and subsequent blood sucking from the legs.
- On inspiration diaphragm will go down, creating a (+)ve pressure which will lead to closure of veins.
- That cycle will continue and form a valve like function.

Mechanisms of venous return:

Unidirectional valves	Already discussed.
Leg muscle pump	They produce up to 200-300 mm of Hg on vein and able to propel >80ml of blood. Their contractions propel blood towards heart. While their relaxation draws blood from superficial to deep veins
Foot sole & ankle pump	The calf muscles can only function efficiently if mobility of the ankle joint is unimpeded, when ankle moves venous pressure can reach >100mm of Hg .Contributes >50% blood leaving calf.
Respiratory pump*	Pressure changes induced in thoracic cavity during inspiration sucks blood upward toward the heart.



2)Venous diseases

- Normal ambulatory venous pressure (AVP) shouldn't exceed **25mmHg** at the level of the ankle.
- Any disturbance in the normal venous cycle whether at the level of superficial veins (Primary cause), deep veins or perforators (Secondary causes) may lead to blood accumulation and venous hypertension.
- Any kind of lesion that is able to intervene with the venous cycle can lead to an elevated venous pressure.

Long standing hypertension can then result in:

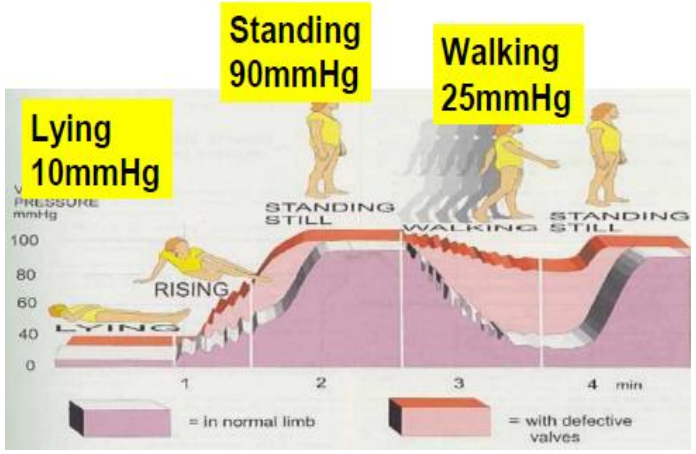
1/Varicose veins (Congested tortuous veins due to long standing high pressure)

2/Chronic venous insufficiency

(collection of signs, symptoms and complications due to long standing high pressure)

*AVP is the minimal pressure in foot veins on walking (Normally ≤ 25mmHg)

On motionless standing, venous pressure can reach 90mmHg, this pressure Falls by 60-80% in few seconds after walking.



Normal change in venous pressure in different situations

Ambulatory Venous Pressure:

NORMALLY:

o When lying supine: pressure in lower limbs is low (10 mmHg)

o When rising: deep veins start pulling blood from superficial veins slowly→ so pressure starts to increase gradually.

o When standing: deep veins continue to pull blood and pressure increases reaching (90 mmHg)

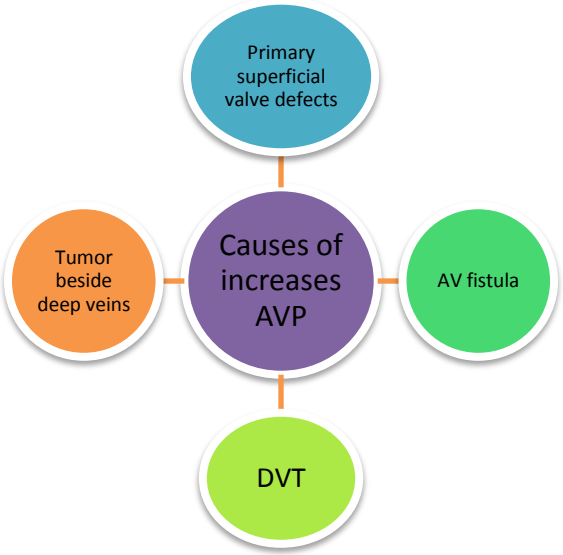
o When walking: calf muscle pump starts working and pushes blood up the vein through the valves→ so pressure drops to (25 mmHg). Valves then close, to prevent the pressure from increasing again by preventing the blood from refluxing.

o If you stop and stand still, calf pump stops and the deep veins start to pull blood from the superficial veins again, so pressure builds up again.

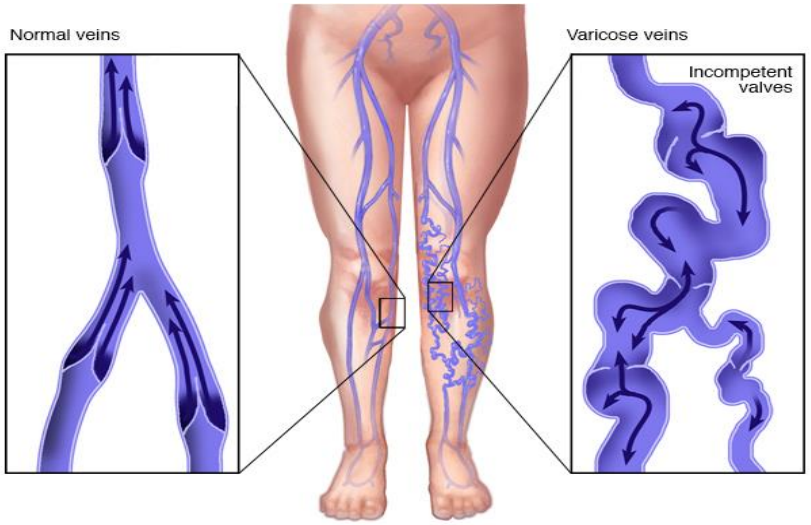
If Valves Defected;

o When rising and standing: blood will reflux from the valve, so the pressure will increase rapidly.

o When walking: blood will reflux through the valve and the pressure remains high.



1-Varicose veins



- Most common vascular disorder, can be saphenous or non-saphenous
- Incidence of saphenous varicosities:

Male : 10-15%

Female : 20-25%

- When non-saphenous varicosities are included:

Male :45%

Female : 50%

Risk factors Increase the risk to develop varicose veins	**Accelerators** Doesn't increase the risk, but when the risk is there they accelerate their occurrence
Female gender	Pregnancy
Advanced age	Obesity
Caucasian race	Professions need long standing e.g: surgeon, nurse, teachers < gravity cause high venous pressure
Family history	Oral contraceptives (vasodilators)

Types of varicose veins

Telangiectasia

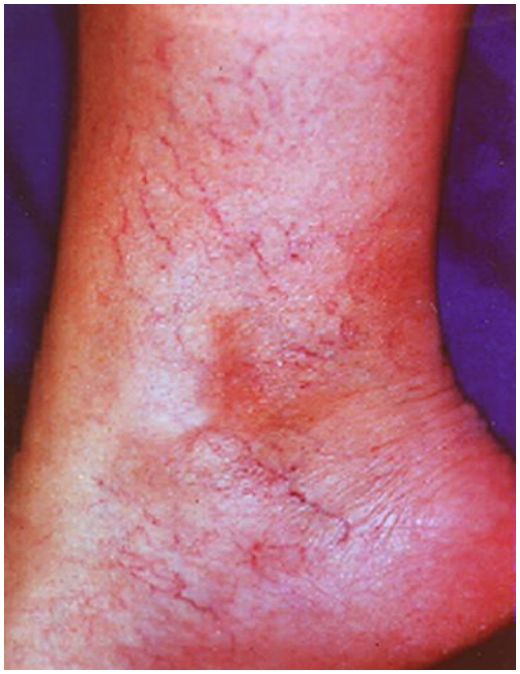
- confluence of dilated intradermal venues less than 1mm in diameter

Reticular veins

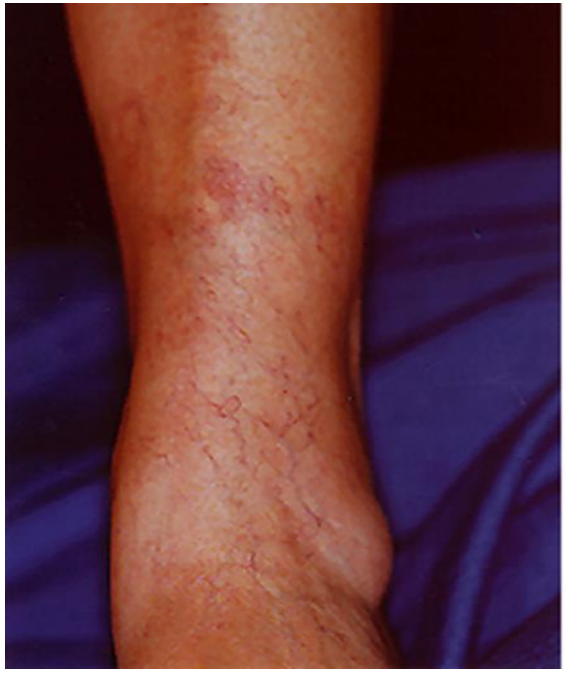
- dilate bluish tortuous sub dermal veins
  1-3mm in diameter

Trunk varices(varicose vein)

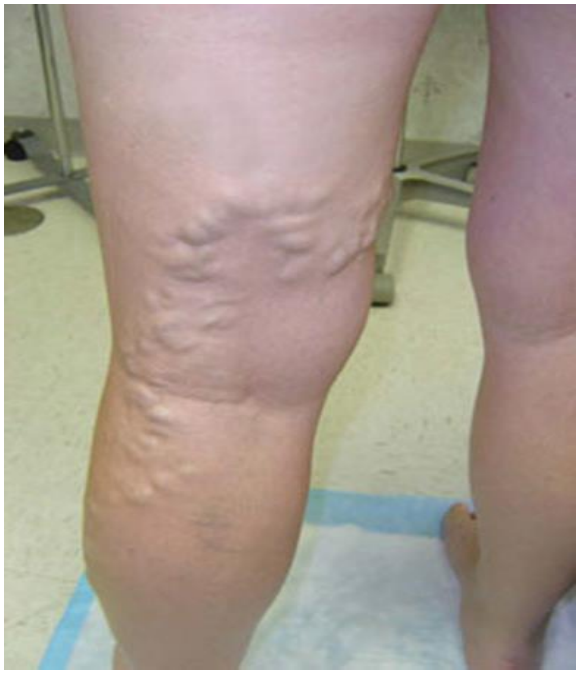
- subcutaneous dilated, elongated, tortuous veins grater than 3mm involving saphenous veins, saphenous tributaries or non saphenous tributaries



Telangiectasia



Reticular veins



Trunk varices

2-Chronic venous insufficiency:

Arterial insufficiency = decrease blood supply
Venous insufficiency = venous hypertension

Pathophysiology

- 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 causes (superficial veins)**: related to structural weakness of valves (Floppy valves) or venous wall as in primary varicose veins.**

Tx. Surgery or conservative.

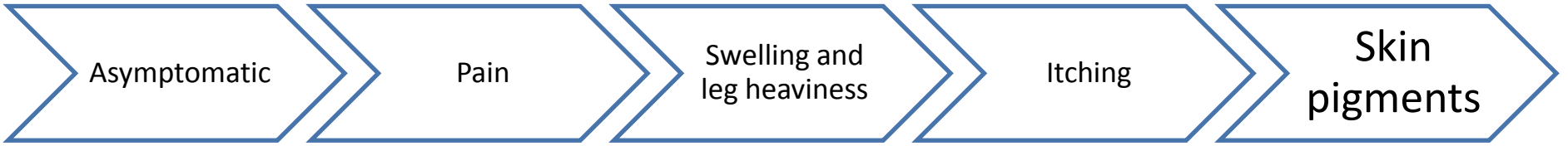
- Secondary causes (deep veins) **include those due to previous DVT as in post-phlebitis syndrome, a tumor blocking deep veins , AV fistula or Pregnancy .**

Tx. Treat the underlying cause.

Cont. Chronic venous insufficiency

Clinical picture

- Clinical presentation ranges from an asymptomatic to a cosmically and symptomatically annoying condition



- Pain can be Throbbing, Aching. Stinging, Burning, Night pain, Crampiness.
- Severity of symptoms and signs depend on the degree and duration of venous hypertension.
- Patients can also manifest with complications such as Bleeding, Leg ulcers, Lipodermatosclerosis and Superficial thrombophlebitis.
- Leg ulcers are painful and primarily located near the ankle, their development can be preceded by brownish pigmentations.(venous ulcer located around the ankle on the medial aspect)

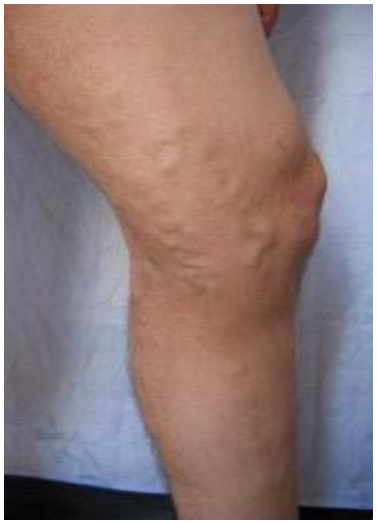
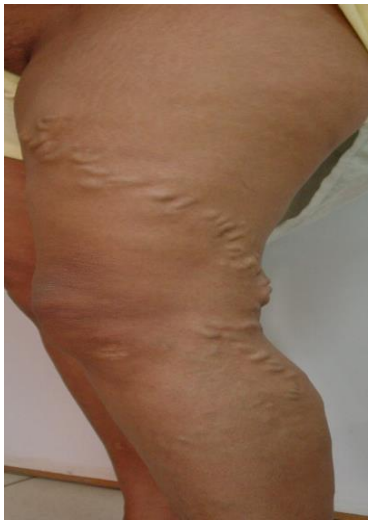
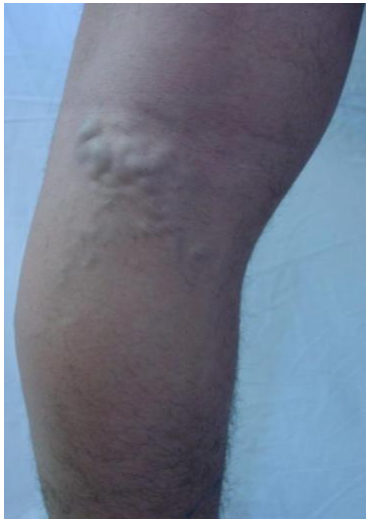
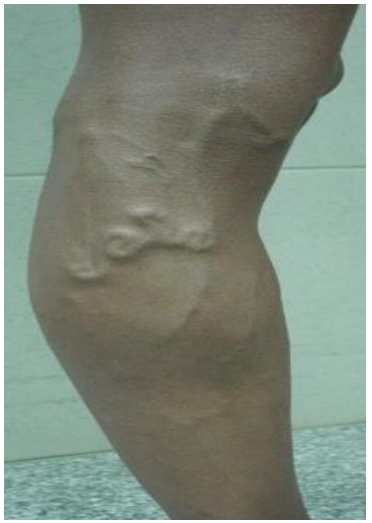
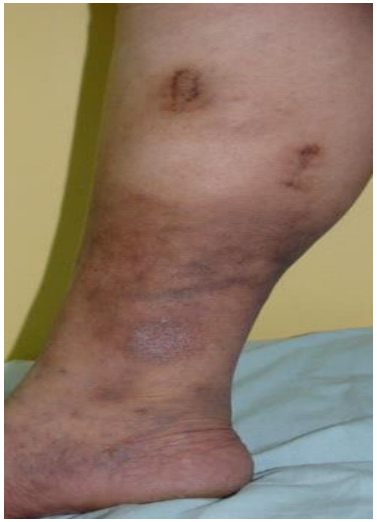
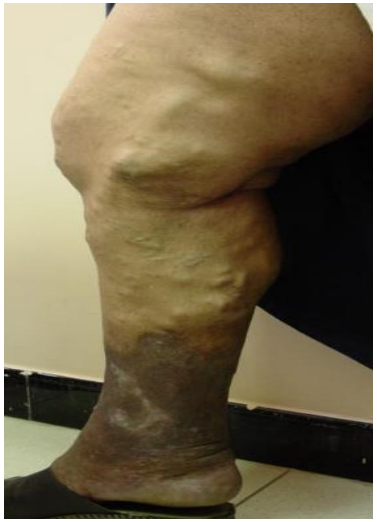
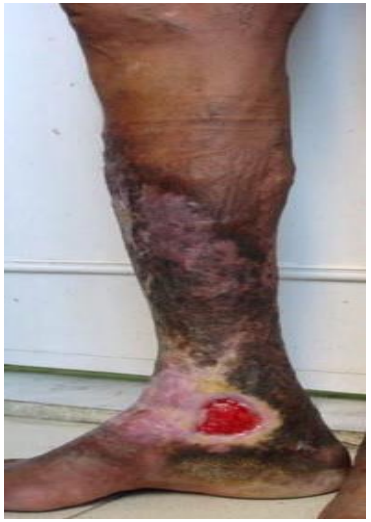
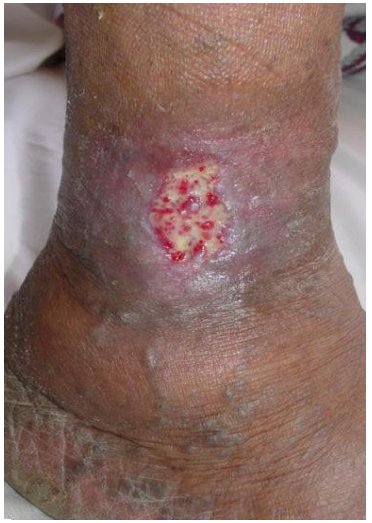
3)Clinical Examination

- Patient should be in standing position.
- First, Inspect for extent & distribution of Varicose veins.
- There are many tests can be done in order to know Which system? Which perforators? Are involved and to assess the patency of deep veins.

- Trendelenburg test
- Schwartz test (Cruvhellier's sign)
- Morissey's cough impulse
- Fegan's method (Phallen's test)
- Pratt's test
- Tourniquet test (Mahorne-ochsner}
- Perthe's test

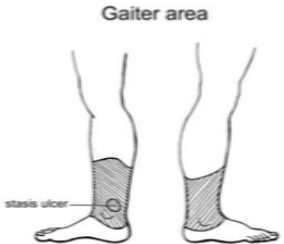
According to doctor, You won't be asked about these tests. However, if you are interested you can Google them

Cont. Clinical Examination

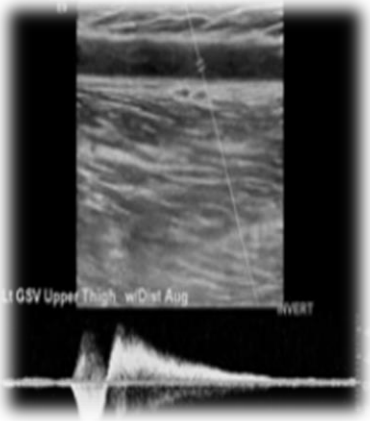
Gaiter's Area:

Area around medial and lateral malleolus is common site for venous ulceration because this area is skin on bone (no tissue and fat between them) so the blood and inflammatory stuff will go directly from the veins to skin leading to certain manifestation.



4)Investigations

1-Doppler ultrasound



To assess flow & patency of veins

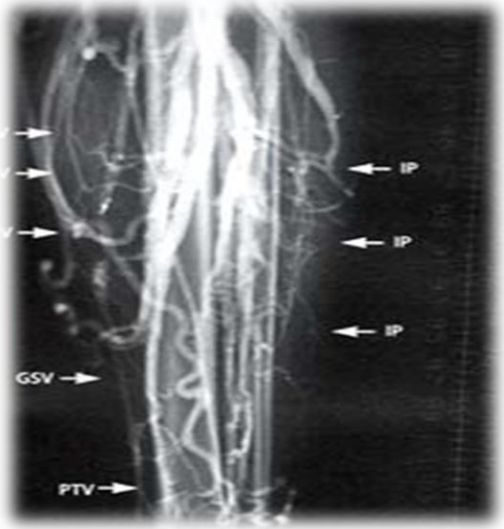
- It gives a qualitative assessment of venous reflux as well as an evaluation of reflux in Sapheno-femoral & popliteal junctions.
- It does not give anatomic information

○ **Duplex Scan:** Direct detection of valvular efflux →**best initial test , save in pregnancy**

1/Physiologic reflux < 0.5sec

2/Pathologic reflux > 0.5sec

- Visualization of valve leaflet motions
- Quantify degree of incompetence
- Study of deep, superficial and perforator veins for patency and competency.



2-Plethysmography

- Detects volume change of limb secondary to changes in venous blood flow

3-Pressure measurements

A/Transmural pressure B/Ambulatory venous pressure

4-Invasive Procedures

- Ascending venography
- Descending venography
- CT Venography
- MRV

Venography: **the gold standard**
o Contrast injected to visualize veins.
o Not used much nowadays, due to its complications. But still has specific indications

Investigations Cont. From Raslan's

* AVP (ambulatory venous pressure)

It is a test to measure the venous pressure in supine, standing, andwalking positions. To compare it with normal changes.

Method:

- It is performed by placing a small needle into one of the veins on the back of the foot and connecting the needle to a blood pressure measurement machine.
- 20-21gauge Butterfly Needle
- Superficial Dorsal Vein (Foot) or Ankle Vein

Normally:

when walking

- A decrease in pressure from Pressure 80 - 90mm Hg to

20-30 mm Hg

- Or a: > 50% drop

Then after standing still Venous Refilling Time: ?20 seconds

Abnormal results if:

Lack of sufficient drop in pressure with ambulation

- Pressure doesn't decrease enough on walking and the difference between the standing and walking pressure is <50%
- This means that the pressure remains high in the veins although it is supposed to drop because of walking

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

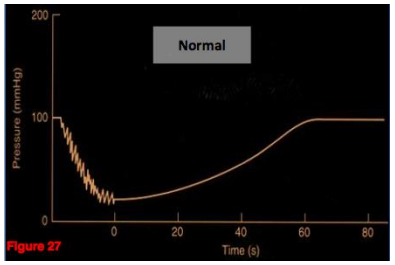


Figure 27

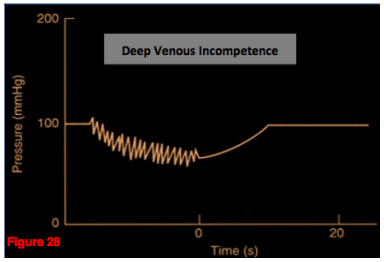


Figure 28

5)CEAP Classification (Just go through it)

In order to standardize the reporting and treatment of the diverse manifestations of chronic venous disorders, a comprehensive classification system (CEAP) has been developed to allow uniform diagnosis.

Clinical*

- C_0 - No clinical signs
- C_1 - Small varicose veins
- C_2 - Large varicose veins
- C_3 - Edema
- C_4 - Skin changes without ulceration
- C_5 - Skin changes with healed ulceration
- C_6 - Skin changes with active ulceration

Etiology*

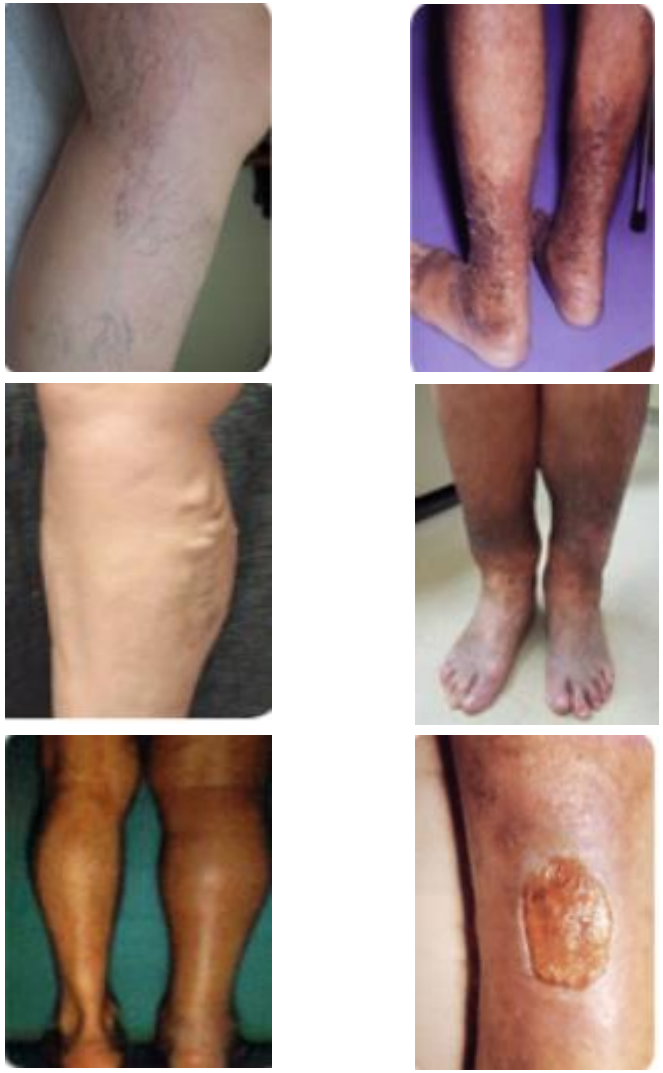
- E_C - Congenital
- E_P - Primary
- E_S - Secondary
  (usually due to prior DVT)

Anatomy*

- A_S - Superficial veins
- A_D - Deep veins
- A_P - Perforating veins

Pathophysiology*

- P_R - Reflux
- P_O - Obstruction



6) Treatment Options

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)

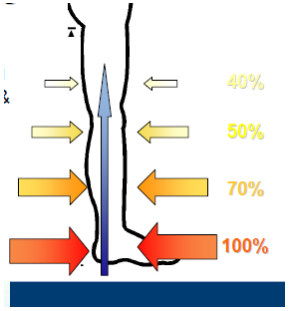
Treatment options:

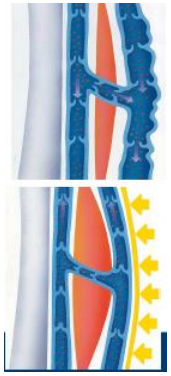
- Conservative Treatment
- Compression garments
- Life style and risk factors modification
- Skin care, ulcer treatment
- Phlebotrophic drugs
- Compression Therapy
- Elastic compression
  - Bandage
  - Stockings
- Paste gauze (UNNA) Boot
- Circa diorthosis
- Intermittent pneumatic compression
  - **Effects on tissues**
  - **Increase tissue pressure > edema**
  - **Reduce inflammation**
  - **Sustains reparative process**
  - **Improves movements of tendon and joints and contraction of venous muscle pump**

Compression therapy

Hemodynamic Effect:

- Compress the legs & superficial veins of leg
- Reduce vein caliber, helps the closing of valves
- Increase venous blood flow
- Decrease venous blood volume
- Reduce reflux in superficial & deep veins.
- Reduces pathologically elevated venous pressure





Class	Uses	Pressure at Ankle (mmHg)
I	Mild venous insufficiency or varicose 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**
  - Hydroxy - Rutosides
  - Calcium dobesilate
  - Troxerutin
- **Haemorrheologic Agents**
  - Pentoxiphylline
  - Aspirin
- **Free Radical Scavengers**
  - Topical Allopurinol
  - Dimethyl Sulfoxide
- **Prostaglandins E & F**
- **Topical therapies**
- **Growth factors & Cytokines**
- **Skin substitutes**

Sclerotherapy

- Sclerotherapy is the injection of a sclerosing agent into a vein, causing an inflammatory reaction 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

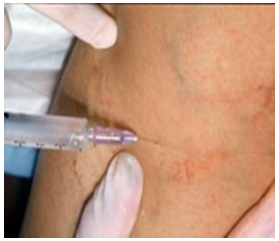
- Sodium tetradecyl sulfate
- Polidacanol
- Sodium morrhuate
- Ethnolamine oleate

Osmotic solutions

- Hypertonic Saline
- Hypertonic saline & Dextrose
- Sodium salicylate

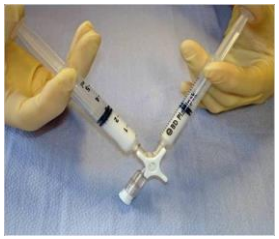
Chemical irritants

- Polyiodinated iodine
- Chromated glycerine



Microsclerotherapy

- 30 G needle
- 0.1 – 0.25 Sodium tetradecyl sulphate
- Needs multiple sessions
- Needs compression therapy



Foam sclerotherapy

- Tessari technique
- 1part of sodium tetradecyl sulphate and 4 parts of air agitated using two syringes

Surgical treatment (gold standard)

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

Endo venous laser surgery

- Endoluninal obliteration by heat
- Induced collagen contraction and denudation of endothelium
- Fibrosis
- 810nm & 1470nm Diode laser.

Radiofrequency ablation.

7) Deep vein thrombosis (DVT)

In conjuction with Pulmonary Embolism DVT leading cause of in hospital mortality in USA
1 in 20 persons develop DVT in course of his or her life time
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 & IVC
80-90% of PE originate here

Virchow's 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 :

- Advancing age
- Obesity
- Prolong bed rest >4days
- Immobilization
- Limb paralysis
- Extended travel

Endothelial injury

- Trauma
- Surgery
- invasive procedures
- iatrogenic

Hypercoagulable status :

- Surgery, trauma, responsible for 40% TED
- Malignancy
- Increased estrogen levels – due fall in protein s
- Inherited disorders of coagulation
- Acquired disorders of coagulation
  - Nephrotic syndrome
  - Antiphospholipid Ab
  - inflammatory process
  - SLE

Cont. DVT

Spectrum of Disease

- **Asymptomatic**
- **Symptomatic**

Acute DVT
limb pain,edema,phlegmasia

Pulmonary Embolism
Resp.distress, heart failure , Death

Chronic Venous Insufficiency

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)**

- Associated with arterial spasm
- Painful pale with poor or absent pedal pulses

Diagnosis

D-dimer Assay

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

Duplex

- Non invasive, Bedside exam
- 96% sensitive & 100% specific
- Normal veins –collapse with compression and lumen is free
- In DVT vein is dilated , lumen shows thrombus and non compressible veins with poor or no venous flow

Venography

- Iodinated contrast injection into venous system
- DVT is visualized as filling defect
- Invasive
- Contrast complications
- Needs radiology suite

ACCP 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

Prevention

- 5000 units of heparin 2 hours preoperatively than every 12 hours post operatively (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

Cont. DVT

(Not mentioned in the lecture *from TEXTBOOK*)

- **Pathophysiology:**

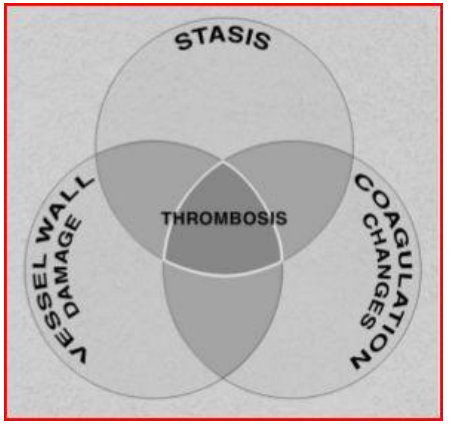
DVT probably begins in the calf in most cases. At first, the clot is free-floating within a column of flowing blood. The risk of PE is highest at this point. Later, when thrombus has completely occluded the vein and incited an inflammatory reaction in the vein wall, the clot becomes densely adherent and is unlikely to embolize. The classic features of DVT are due to this occlusion (**leg swelling, dilated superficial veins**), thrombophlebitis (**redness, pain and tenderness, heat**) and Homan's sign*. The important point is that most surgical patients developing a clinically significant postoperative pulmonary emblosim (PE) do so on about the 7th to 10th day and nearly always have *clinically normal legs*. By the time a clinically apparent DVT has developed, the danger period for pulmonary embolism has largely passed; for this reason DVT prophylaxis must be considered in all thrombo-embolic prophylaxis must be considered in all patients undergoing open vascular or endovascular surgery.

- 1 in 20 persons develop DVT in course of his or her life time.
- Starts at calf veins & progress proximally into popliteal, femoral, iliac veins & IVC
- 80-90% of PE originate here.

*Pain in the calf of the leg upon dorsiflexion of the foot with the leg extended. It's not sensitive nor specific.

- **Virchow's triad:**

Three factors are traditionally associated with thrombogenesis (Virchow's triad): **Venous stasis, Intimal damage and Hypercoagulability of the blood**. Many of the recognized clinical risk factors for DVT relate to venous stasis. In most instances of DVT no evidence of direct intimal damage can be detected. However, external trauma to a vein, for example, during a hip replacement operation, can provide a starting point for thrombogenesis.



Cont. DVT

(Not mentioned in the lecture *from TEXTBOOK*)

- **Venous gangrene:**

In certain circumstances, DVT may propagate the venous collaterals or microcirculation (arterioles and venules). The former leads to an intensely swollen, cyanosed limb (**phlegmasia caerula dolens**), whereas the latter can lead to obstruction of the arterial inflow and the development of a swollen white leg (**phlegmasia alba dolens**) which is associated with arterial spasm and poor or absent pedal pulses. The patient may then go on to develop venous gangrene.

- **Complications:**

- PE 12-33%
- PTS*
- Pain & swelling - 67%
- Pigmentation - 23%
- Ulceration - 5%

*Magnetic Resonance Venogram.
*Low-dose subcutaneous low molecular weight heparin.
*Post Thrombotic Syndrome.

- **Diagnosis:**

Clinical examination alone is unreliable at confirming or excluding the presence of DVT. This means that the diagnosis of DVT cannot be made on clinical grounds alone, and that some form of investigation is required.

1-Color flow duplex ultrasound:

Color duplex ultrasound imaging has largely replaced conventional venography in the diagnosis of DVT. It has a sensitivity of 96% and a specificity of 100%. It is noninvasive, avoids ionizing radiation and contrast, and is as accurate as venography in most cases. In DVT vein is dilated , lumen shows thrombus and non compressible veins with poor or no venous flow. At times of doubt, MRV* or CT venography may be useful.

- **D-dimer:**

- It is sensitive but not specific
- Fibrin fragment present in fresh fibrin clot & FDP of cross linked fibrin
- Marker for action of plasmin on fibrin

- **Prevention:**

LMWH* (Lovenox(Enoxaparin) 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.

Cont. DVT

(Not mentioned in the lecture *from TEXTBOOK*)

- **Treatment:**

The aims of treatment are to relieve the acute symptoms, protect against PE, and minimize the risk of recurrent thrombosis and post-thrombotic sequelae to the limb.

Uncomplicated DVT:

If thrombus is confined to the calf, the patient is fully mobile and other risk factors are reversible, then an **elastic stocking and physical exercise may be all that is required**. For most uncomplicated DVT, it is now clear that:

1- Bed rest is unnecessary and the patient can be mobilized immediately, wearing an appropriately fitted compression stocking.

2- LMWH given by intermittent subcutaneous injection is more effective than unfractionated heparin given by infusion.

Complicated DVT:

In a proportion of patients, treatment is more complicated because of one or more of the following:

1- The DVT is more extensive (iliofemoral, vena cava, phlegmasia)

2- The DVT is recurrent

3- The patient has had a PE

4- The patient has one or more major irreversible congenital and/or acquired thrombophilia

5- Heparinization is contraindicated.

In these circumstances, treatment must be tailored to the individual patient, and in selected cases it may be appropriate to use thrombolysis, insert a caval filter or consider thrombectomy.

Cont. DVT

(Not mentioned in the lecture *from TEXTBOOK*)

- **Phlegmasia**
- Treat with thrombolysis or surgery, and anticoagulation may not prevent progression in these patients.

- **Catheter Directed Thrombolysis:**

- iliofemoral DVT
- symptoms <14 days
- low risk of bleeding
- have good functional status
- reduce acute symptoms
- reduce post thrombotic syndrome

- **Post thrombolysis, ballon /stent for obstructing lesion**
- **Pharmaco-mechanical thrombolysismay shorten treatment time**
- **After thrombolysis, anticoagulate**
- **Vena cava filters indications:**
- Patients cannot tolerate even a small pulmonary emboli.
- Anticoagulation contraindicated (intracranial bleed)
- Anticoagulation fails to prevent embolization or extension of thrombus
- PE are still occurring despite adequate anticoagulation.

The rationale behind inserting an inferior vena cava filter (IVC) is that it will trap embolus that would otherwise have been destined for the lungs causing a PE. The use of IVC filters varies enormously around the world.

8) Lymphedema (Not mentioned in the lecture *from TEXTBOOK*)

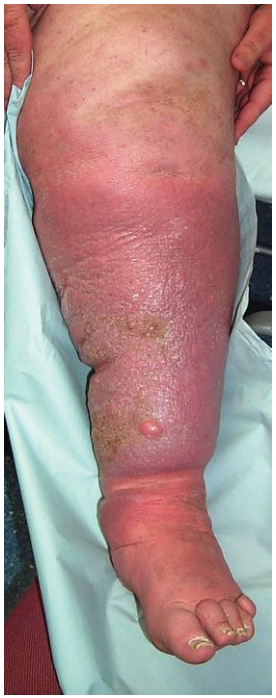
- **Abnormal collection of interstitial lymph fluid** due to either congenital maldevelopment of lymphatics or secondary to lymphatic obstruction
- It affects 2% of population causing limb swelling

Patterns of lymphatic abnormalities:

- Aplasia -15%
- Hypoplasia – 55%
- Hyperplasia -35%
- Dermal Back flow – Chylous Reflux

Predisposing factors

- Any inflammatory process
- Trauma
- Pregnancy
- Puerperal sepsis
- Most of primary lymphedema appear spontaneously



Etiology

Primary lymphedema:

- Developmental error in lyphatic vessels
- Depending on the severity it appears

  At birth < year – lymphedema

  congenital – familial - Milroy;s Disease
- Between 1-35yr – lymphedema precox
- Later >35yr - lymphedema tarda

Secondary lymphedema:

- Trauma, infections, filariasis*, post plebitic limb, irradiation, malignancy, allergy.

Clinical Classification of lymphedema

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

*is a parasitic disease caused by an infection with roundworms of the Filarioidea type.These are spread by blood-feeding black flies and mosquitoes.

Cont. Lymphedema (Not mentioned in the lecture *from TEXTBOOK*)

Clinical features:

Lymphedema is like other forms of edema, in that it is present only upon dependency; that is, **worse at the end of the day and absent in the morning**. However, as the edema fluid becomes more protein-rich, it is less and less affected by position. Lymphedema nearly always commences distally on the foot and extends proximally, usually only to the knee. Some patients first present to medical attention because of acute cellulitis. Such patients are prone to recurrent episodes, each one of which damages the lymphatic system still further, leading to cycle of deterioration.

Diagnosis:

- **Lymphoedema is essentially a clinical diagnosis and most patients require no further investigation.**
- Lymphangiography (invasive, inject contrast)
- Isotope lymphosintigraphy (best initial test)
- CT
- MRI
- Duplex Ultrasound

Complications:

- Local infection
- Systemic infection
- 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.
- Diuretics are of no value and are associated with sideeffects, including electrolyte disturbance. No other drugs are of proven benefit.
- Early treatment is highly effective
- Late disease is difficult to treat
- Care to avoid any injury to limb
- Skin care to avoid infection
- Reduce swelling by decongestive lymph therapy
- Manual lymph therapy – massage
- Multilayer compression bandage & physiotherapy
- Intermittent pneumatic compression devices

Surgical treatment

Reducing Surgery

- Sistrunk - wedge removal & skin closure.
- Homans - Subcutaneous tissue removal & skin closure.
- Thompson - denuded skin sutured to deep fascia.
- Charles - remove every thing & cover raw area with skin graft.

Bypass surgeries (best procedure)

- lymph venous shunts

Table 21.6 Differential diagnosis of the swollen limb

Non-Vascular or Lymphatic

General disease states
- Cardiac, renal and liver failure
- Hyperthyroidism (myxoedema)
- Allergic disorders
- Immobility and lower limb dependency

Local disease processes
- Ruptured Baker's cyst
- Myositis ossificans
- Bony or soft tissue tumours
- Arthritis
- Haemarthrosis
- Calf muscle haematoma
- Achilles tendon rupture
- Other trauma
- Reflex sympathetic dystrophy

Gigantism
- Rare; all tissues are uniformly enlarged

Drugs
- Steroids

Obesity
- Lipodystrophy, lipoidosis

Venous

Deep venous thrombosis
- The classic signs of pain and redness may be absent

Post-thrombotic syndrome
- Venous skin changes, secondary varicose veins on the leg and collateral veins on the lower abdominal wall
- Venous claudication may be present

Varicose veins
- Do not usually cause significant swelling

Venous malformations
- Most common is Klippel–Trenaunay syndrome
- Abnormal lateral venous complex, capillary naevus, hypo(a)plasia of deep veins and limb lengthening
- Lymphatic abnormalities often co-exist

External venous compression
- Pelvic or abdominal tumour including the gravid uterus
- Retroperitoneal fibrosis

Arterial ischaemia-reperfusion
- Following lower limb revascularization for chronic and particularly acute ischaemia

Arteriovenous malformation
- May be associated with local or generalized swelling

Aneurysm
- Popliteal
- Femoral
- False aneurysm following (iatrogenic) trauma

e.g: 50 years old male or female present in your clinic with unilateral leg swelling, painless , gradually increasing . No Hx. Of trauma. On examination there is diffuse swelling ?

Dx: best initial tests

Duplex Scan for venous disease , Isotope lymphosintigraphy for lyphoedema

Thank You..

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