DISORDERS OF THE VENOUS SYSTEM
The supine foot venous pressure measures 15 mmHg, and represents the residual kinetic energy of the heart reduced by the capillary and arteriolar resistance.

The vein wall is composed of collagen, elastic tissue and smooth muscle fibers, the smooth muscle of the venous wall is important in regulating the venous volume. And is important in keeping the central venous pressure constant despite of variations in blood volume.

The venous return is affected by a number of factors; namely: Calf muscle pump, Arterial pulsations, Arterial pressure, Negative intrapleural pressure and the body position.

Bicuspid valves are present in the peripheral veins and decreases in number proximally to direct the blood flow towards the heart.
DISORDERS OF THE SUPERFICIAL VEINS

- Primary varicose veins arise spontaneously in the absence of deep system pathology, while secondary varicose veins occur due to chronic venous insufficiency.
- Women exhibit a higher incidence of varicose veins than men especially between 40-49 years.
- Predisposing factors include physical inactivity, obesity, smoking, hypertension and menopause.

**Treatment:**

- **Conservative treatment:** for both primary and secondary varicose veins include, elastic stockings, periodic elevation and exercise wearing elastic stockings.
- **Surgical treatment:** is indicated in, pain over the varicosities, superficial thrombophlebitis, disabling edema, dermatoliposclerosis, bleeding and manifestations of CVI especially ulceration.
- **Contraindication** to varicose vein stripping and ligation include; cosmetic reason only, secondary V.V. due to insufficiency (thrombosis) of the deep system, V.V associated with other diseases, causing the symptoms like, arthritis, arterial occlusive disease, lymphedema, CHF, & obesity. Or in association with arteriovenous fistula or congenital anomalies like Klipple-Trainaunay disease.
- **Sclerotherapy:** is frequently done for spider varicosities and telangiectatic vessels.
- **Indicated in:** presence of pain, presence of CVI symptoms especially during menstruation, bleeding, ulceration, history of thrombophlebitis.
- **Contraindicated in:** active thrombophlebitis, pregnancy and lactation, bed ridden patients, arterial occlusive disease, inability to apply bandages after injection and pure cosmostis.
**DISORDERS OF THE DEEP VEINS**

- Traditionally CVI has been considered to occur mainly as a consequence of DVT, recent reports suggest that CVI occurs almost equally in primary valvular incompetence.

- Other causes include hemangioma, congenital A-V fistula and pelvic tumors.

- **Pathogenesis:** Incompetent perforators produce elevated venous pressure especially at the ankle region due to the effect of gravity and due to the presence of direct perforators due to lack of soft tissue support at the gaiter area, causing edema, capillary rupture and extravasation of RBCs causing eczema, pigmentation, and inflammatory reaction followed by subcutaneous fibrosis that result in local tissue ischemia and ulceration.

- Venous ulcers are large, irregular, painful, granulation tissue forms the floor, occurs in the gaiter area and the surrounding skin shows eczema, edema and pigmentation.

- **Symptoms include:** Aches, fatigue, night cramps, itching, and venous claudication due to compartmental syndrome caused by edema during exercise.

- **Epidemiology:** 25% of the general population suffer from CVI.

  75% of cases of DVT develop complications if untreated varicose ulcers start to appear 2-3 years from the onset of DVT.

  Valvular incompetence increases from 17% to 66% during the 1st year.
Treatment:

- Once venous insufficiency develops it is an incurable but manageable problem.

- **Conservative management;** consists of * elastic compression stockings to be worn as soon as the patient gets up from bed,  
  * leg elevation above the level of the heart during sleep and frequently during the day,  
  * exercise with elastic stocking support

- **Surgical treatment;** most patients are treated conservatively only 1-2% of cases benefit from surgical treatment.

**Preoperative evaluation;** Should be done preoperatively to confirm the diagnosis and to select the proper surgical procedure * non-invasive vascular laboratory testing photo-plethysmography PPG, air plethysmography OPG, doppler studies.

* duplex scanning, is of prime importance in identifying and localizing the pathology

* venography ascending or descending is also as useful but invasive in nature

**Operative procedures;**
DEEP VENOUS THROMBOSIS

- **Epidemiology:** 1% of the population develop DVT every year, the most serious complication of which is pulmonary embolism which occurs in 2.5-10% of cases of DVT. It is attended with a high mortality rate which is difficult to determine as it can only be proved by autopsy. Deep vein thrombosis is frequently followed by post-phlebitic syndrome which is debilitating and costly.

- **Etiology & Pathogenesis:** Wirchow triad includes intimal injury, stasis and hypercoagulability. Other predisposing factors include 1) >40 years, 2) males, 3) malignancy, 4) previous DVT, 5) surgical intervention, 6) pregnancy, 7) contraceptive use, 8) nephrotic syndrome, 9) SLE, 10) dysfibrinogenemia, 11) deficiency or disorder of protein C & S, antithrombin III, plasminogen, and 12) drug abuse.

  The thrombus starts by platelet accumulation in the vein, stasis retains the pro-coagulation factors leading to activation of the coagulation cascade and further fibrin and platelet accumulation, the thrombus then enlarges attaches to the other wall of the vein occluding the lumen and edema and pain develops. A floating tail thrombus then forms due to stagnation of blood proximal to the occluding thrombus, this can detach and cause pulmonary embolism.

  Antithrombin III inhibits activated factor X stopping the coagulation cascade. Deficiency of this factor as well as protein C & S and heparin cofactor II predisposes to DVT.
**DIAGNOSIS**

- **CLINICALLY:**
  - Insidious development of lower limb pain,
    - Extensive pitting edema, blanching due to edema called *{phlegmasia alba dolens}*.
    - The condition is common in the left lower limb {3:1}, due to compression of the left iliac vein by crossing beneath the right iliac artery at the level of the pelvic brim, if a gravid uterus causes the compression the condition has been called *{milk leg}*.
    - If venous thrombosis is extensive arterial circulation becomes impaired producing the characteristic picture of reddish blue color change *{Phlegmasia cerulea dolens}*, changes are severe causing motor and sensory loss and venous gangrene if left untreated.
    - Physical signs occur below the level of venous obstruction.
    - Homans sign which describes the inability of passive dorsiflexion is unreliable.
  - Unfortunately the clinical findings are non-specific and diagnosis of DVT based on clinical data is unreliable.

- **VENOGRAPHY:**
  - Was historically considered the gold standard for providing an accurate anatomic diagnosis of DVT, by visualizing filling defects in the deep system,
    - has the disadvantages of -being invasive -causing patient discomfort & -the injected dye being thrombogenic.
    - the procedure can also be done using radioactive isotope and gamma camera, which may also be useful in detecting silent pulmonary embolism.
**DUPLEx STUDY:** of comparable accuracy to venography, possible bedside use, noninvasive and non thrombogenic, easy and can be repeated for the follow-up of the disease process, the problem is that it is an operator dependant study. Doppler study is based on impairment of flow signals in the presence of venous thrombi.

**IMPEDANCE PLETHYSMOGRAPHY:** Measures the volume response of the lower limb to temporary occlusion of the venous system, and the rate of venous emptying after release of occlusion.

**RADIOACTIVE AGENTS:** Radioactive iodine labeled fibrinogen carries the risk of contracting hepatitis, so other radioactive materials are now used like technetium labeled anti-fibrin monoclonal antibody. Detects DVT & pulmonary embolism.

**ASSAY OF FIBRIN AND FDP:** Sensitive but not specific.

**PROPHYLAXIS:** Consists of altering coagulation & prevention of stasis

Early ambulation is controversial in the prevention of stasis

Anticoagulation using low dose heparin prevents the complication of intra and post operative hemorrhage and depresses factor Xa activity producing sufficient anticoagulation to decrease the incidence of DVT and pulmonary embolism in high risk patients

The dose of 5000IU S.C. heparin 2h, before the operation and a similar dose every 12h, is used in high risk orthopedic and urologic patients low molecular weight heparin offers more significant protection.

The use of intermittent pneumatic compression pumps on the lower limbs during major operative procedures decreases the incidence of intraoperative thrombi formation.
Three main objectives should be achieved: 1) minimize the risk of pulmonary embolism. 2) to limit further thrombosis. 3) to facilitate resolution of the existing thrombus to prevent the occurrence of the post-phlebitic syndrome.

The patient should rest in bed in the Trendelenburg position for 4-5 days under anticoagulant until edema and pain subside, then the patient could ambulate using elastic stockings.

**ANTICOAGULATION:** Therapy in DVT is based on adequate systemic anticoagulation, initially with heparin then with warfarin. **Heparin** is given in the dose of 100-150 IU/kg/dose IV., controlled by partial thromboplastin time [PTT] which should be 2-3 times the normal. **Side effects include:** bleeding, -thrombocytopenia, -thromboembolism, -hypersensitivity and – osteoporosis. **Oral anticoagulants** are given shortly after the initiation of heparin therapy as several days are needed to bring the international normalized ratio INR within the therapeutic range of 2.0-3.0, i.e. 1.3-1.5 times control. **Side effects include:** bleeding, -skin necrosis, -subcutaneous painful erythema in fatty areas. [NB: fresh frozen plasma rather than vit K restores the prothrombin time to normal in case of bleeding]. Anticoagulation should be maintained for at least 3-6 months oral anticoagulants are teratogenic, interact with barbiturates and the dose should be monitored during its use.

**FIBRINOLYSIS:** **TPA, Streptokinase & Urokinase** activate the plasmin system better done by direct catheter lyses to reduce the dose and hence the side effect, they have no advantage over heparin after 72h. **Side effects include:** bleeding, hence contraindicated in post-op. & post-traumatic patients. -streptokinase may cause allergic reactions in 10% of patients. They have no advantage over heparin in the prevention of the post-phlebitic syndrome.
**SURGICAL APPROACH**

- **Operative thrombectomy;** consists of operative removal of the thrombus by the use of fogarty catheter although post-operative results may be impressive re-thrombosis evidenced by venography is noticed in all patients before discharge from hospital, only reserved for limb-salvage in patients with phlegmasia cerula dolens and impending venous gangrene.

- **Vena cava interruption;** A means of mechanical protection from pulmonary embolism.

  **Indications:**
  1. recurrent embolism in spite of adequate anticoagulation
  2. contraindication to anticoagulation
  3. complication of anticoagulation that mandates cessation
  4. pulmonary embolism in association with pulmonary hypertension
  5. after pulmonary embolectomy
  6. failed previous filter
  7. **relative indications;** patients with pulmonary vascular disease involving $>50\%$ of the pulmonary vascular bed - large free floating iliofemoral thrombus.

  **Complications:** hematoma, migration, misplacement, recurrent embolism 2-4%, venacaval occlusion.
PULMONARY THROMBOEMBOLISM

**EPIDEMIOLOGY:** It is the most serious complication of DVT.
- Approximately 50,000 patients die from pulmonary embolism every year in the USA
- 5 patients every 1000 major surgery die from pulmonary embolism
- The full spectrum of the disorder ranges from asymptomatic minor embolism to sudden death from massive disease.

**DIAGNOSIS:**

**Clinical presentation:** symptoms and signs vary depending on the amount of embolus. Sudden chest pain, cough, dyspnea, tachypnea, anxiety and hemoptysis. Signs include: cyanosis, engorged neck veins, tachycardia, increased second sound and collapse, less commonly wheeze, pleural rub, low grade fever, ventricular gallop and wide splitting of the second sound.

*Differential diagnosis include:* myocardial infarction, esophageal perforation, pneumonia & septic shock.

**Electrocardiography:** Is of value in the exclusion of myocardial infarction, non-specific ST-segment and T wave changes is present in 66% of cases.

**Chest X-ray:** Is important in exclusion of esophageal perforation, pneumonia, pneumothorax. Some changes may be observed like asymmetry of the vascular markings with segmental lobar ischemia {Westermark sign}. 
Arterial blood gases: Hypoxemia with PO2 less than 60 mmHg, [hypoxemia may be present in various disorders that could be misdiagnosed as massive embolism eg; septic shock].

Central venous pressure: Elevated central venous pressure with systemic hypotension is suggestive of pulmonary embolism the catheter is valuable in fluid and drug administration. Low CVP excludes pulmonary embolism, but it can be normal in chronic and subacute cases.

Lung scan: Ventilation perfusion scanning is more accurate than perfusion scans alone, presence of a normal scan excludes pulmonary embolism. Wide variation in scan interpretation among observers, led to the much more reliability of the diagnosis when based on arteriography.

Pulmonary vascular imaging: Selective pulmonary arteriography is the most accurate method confirming the presence, size and distribution of pulmonary emboli, but carries a mortality rate of 0.5%. Dynamic contrast enhanced magnetic resonance arteriography, has proved to be more accurate, But cannot detect peripheral embolism. Trans-esophageal ultrasound can also be useful as a bedside investigative modality to confirm or exclude the diagnosis.
**PATHOPHYSIOLOGY**

- 85-90% of pulmonary emboli originate from veins of the lower extremity, less than 33% of patients with documented pulmonary embolism show signs of DVT.

- Once the embolus lodges in the pulmonary arterial tree the ratio of the regional perfusion to ventilation increases, the lung responds by bronchoconstriction in response to low CO2 in the expired air, resulting in reduced lung volume and pulmonary compliance.

- The cardiac and pulmonary vascular effects depends in severity upon the amount of occlusion of the pulmonary vascular bed, over 30% occlusion elevates the pulmonary artery pressure and occlusion of greater than 50% reduces the systemic blood pressure.

- In suspected massive embolism, the patient is given intravenous heparin 150-200IU/kg urgent pulmonary angiography is done and an arterial line is inserted to monitor the blood gases, standby endotracheal intubation for ventilation if required, and proceed to the operating room to perform pulmonary embolectomy.

### CLASSIFICATION AND MANAGEMENT

<table>
<thead>
<tr>
<th>Category</th>
<th>signs &amp; sym.</th>
<th>Gases</th>
<th>PO2%</th>
<th>Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td>MINOR</td>
<td>Anxiety &amp; hyperventilation</td>
<td>PO2 &lt;80mmHg, PCO2 &lt;35mmHg</td>
<td>20-30</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>MAJOR</td>
<td>Dyspnea collapse</td>
<td>PO2 &lt;65mmHg, PCO2 &lt;30mmHg</td>
<td>30-50</td>
<td>CVP elevated, PA press. &gt;20mmHg responds to resuscitation</td>
</tr>
<tr>
<td>MASSIVE</td>
<td>Dyspnea shock</td>
<td>PO2 &lt;50mmHg, PCO2 &lt;30mmHg</td>
<td>&gt;50</td>
<td>CVP elevated, PA press. &gt;25mmHg requires inotropes</td>
</tr>
<tr>
<td>CHRONIC</td>
<td>Dyspnea syncope</td>
<td>PO2 &lt;70mmHg, PCO2 30-40mmHg</td>
<td>&gt;50</td>
<td>CVP elevated, PA press. &gt;40mmHg fixed low cardiac output</td>
</tr>
</tbody>
</table>
Anticoagulation:
Minor embolism can usually be managed by anticoagulants alone, heparin provides adequate protection against further thrombosis and embolism, recurrent embolism under anticoagulant therapy occurs in 10-16% of cases, oral anticoagulation is continued for such patients.

Thrombolytic therapy:
Streptokinase, urokinase & tissue plasminogen activator [TPA] are being assessed in the treatment of pulmonary embolism without hypotension systemic administration is more commonly used with a loading dose and booster doses thereafter allergic reactions are observed more with streptokinase treatment. Bleeding complications occur with all drugs and is dose dependant. Results of therapy is proving to be superior to anticoagulant therapy.

Pulmonary hypertension:
results from accumulation of pulmonary emboli not undergoing lysis in the vascular bed clinically is the picture of chronic cor-pulmonale. When the diagnosis is made the life expectancy is 2 years, but the patient may benefit from a venacaval filter preventing further pulmonary embolism which is lethal in such cases. Some cases may be considered for heart-lung transplantation.

Pulmonary embolectomy:
Is reserved for those patients with massive pulmonary embolism causing systemic hypotension. The operation should be done within minutes after heparinization, inotropic therapy and angiographic confirmation of the diagnosis. Mortality rate is in excess of 40%
Transvenous catheter embolectomy
Thank you