

VENOUS DISORDERS

VENOUS PATHOPHYSIOLOGY

INVESTIGATION

DEEP VENOUS THROMBOSIS

SUPERFICIAL VEIN THROMBOSIS

VARICOSE VEINS

Venous disorders are associated with a high morbidity and a significant mortality. Twenty per cent of the population suffers with varicose veins. 2% of the population have skin changes associated with venous.

Venous Pathophysiology

Blood returns to the heart through the deep veins. Blood from the skin and superficial tissues, (external to the deep fascia) drains via communicating veins into the deep vein. Valves prevent the flow of blood from the deep to the superficial system and on muscle contraction, blood is forced towards the heart. The venous pressure in the foot vein on standing is equivalent to the height of a column of blood, extending from the heart to the foot. On exercise the calf muscle pump contracts and the venous pressure in the dorsal vein of the foot drops. With both superficial and deep venous insufficiency this drop may not be apparent. In patients with superficial venous insufficiency alone, a simple tourniquet applied at the knee or the groin may correct this abnormality, resulting in a fall in foot vein pressure on exercise.

In patients with venous obstruction (iliofemoral occlusive disease), the foot vein pressure may increase on exercise. It is the inability to reduce pressure on exercise

that leads to skin changes around the ankle. These changes, lipodermatosclerosis, are secondary to maintained venous pressure. With persistently raised venous pressure, increased capillary leakage results in blood and fibrin getting into the surrounding tissue. The blood is broken down to produce haemosiderin resulting in pigmentation. The fibrin is deposited around the capillaries, leading to a barrier (Fig. 12.1).

Fig. 12.1 Patient with extensive lipodermatosclerotic change and ulceration.

Hippocrates recognised an association between varicose veins and leg ulcers. He was the first person to advocate the use of compression bandages to treat leg ulcers. Homans attributed leg ulceration to tissue hypoxia. The presence of a fibrin cuff was thought to impede oxygen transfer, resulting in skin breakdown.

Professor Michel was the first to recognise that white cells became trapped in the dependent limb. Dormandy observed an increased white-cell trapping in the dependent limb of patients with chronic venous insufficiency. Coleridge Smith, Thomas, Dormandy and Scurr proposed the hypothesis that white-cell trapping was important in the aetiology of venous ulceration. In patients with venous hypertension, the arteriovenous pressure gradient decreases, blood slows in the capillaries, white cells marginate and become trapped. This may result in a decreased flow through the capillary bed, leading to hypoxia. White cells adherent to the endothelium become activated, releasing proteolytic enzymes and oxygen free radicals. This process may also produce microthrombosis, further compromising the capillary circulation.

Attempts to prevent white cell trapping, to prevent white cell activation and to improve the capillary circulation, will lead to better therapeutic measures for the treatment of venous ulceration.

Investigations

A full history and clinical examination should be undertaken. Patients presenting with arterial disease may have coexisting varicose veins and are inappropriately referred to a venous clinic. Venous symptoms include tiredness, aching, tingling and cramps which get progressively worse towards the end of the day and are relieved by elevating the leg. Ankle swelling and skin changes may also occur. The bursting sensation in the calf on exercise is usually associated with venous obstruction.

A clinical examination carried out with the patient standing will reveal ankle

swelling, skin discoloration, eczematous change, and the presence of varicosities including the presence of a saphena varix. An examination of the peripheral pulses should be carried out. By the use of a tourniquet (Brodie—Trendelenburg test) some information can be obtained about the position of incompetent valves, demonstrating, for example, incompetence of the saphenofemoral junction, a midhigh perforator or a saphenopopliteal valve incompetence.

Doppler ultrasonography is now the minimum level of investigation required before treating venous disease. A Doppler flow probe can be used to exclude arterial disease, to determine the patency of a vein and a bidirectional flow probe used to detect venous reflux. The major disadvantage of this technique is that it is not apparent which vein is being insonated and this may cause confusion, particularly when using the probe in the region of the popliteal fossa.

Photoplethysmography is the technique, which measures the amount of blood in the skin. On dorsiflexion of the ankle the blood is pumped from the skin. When the ankle movements cease the blood returns. In the presence of venous insufficiency the blood flows rapidly back into *the* skin via either the deep or superficial veins (Fig. 12.2). The application of tourniquets above and below the knee will separate patients with superficial, from those with deep, venous insufficiency.

Strain gauge plethysmography measures volume changes in the leg in response to a tourniquet applied around the thigh. It provides useful information on venous outflow and can be used to diagnose iliofemoral venous thrombosis.

Duplex imaging involves the use of B-mode ultrasound and a coupled Doppler probe. It allows direct visualisation of the veins and provides functional as well as anatomical information.

By colour coding, the direction of blood flow can be seen easily. This allows the detection of reflux to be determined. The position of communicating veins can be determined with precise localisation of the saphenopopliteal junction. This entirely noninvasive technique is now replacing venography in the management of patients with venous disorders and for the diagnosis of deep vein thrombosis (Figs 12.3—12.6).

Venography. A fine tourniquet is applied just above the malleoli and an injection of nonionic contrast material given to outline the veins. The technique provides anatomical information and is now only indicated in the absence of good duplex ultrasonography facilities.

Deep Vein Thrombosis

Definition: is thrombosis of part or all the deep venous system of an extremity.

Pathogenesis: *Virchow triad leading to deep vein thrombosis.*

- Trauma to the vein wall: caused by surgery or physical injury.
- Decreased venous blood flow: due to postoperative immobility or drugs decreasing venous tone (estrogen).
- Increased viscosity (coagulability) or change of cellular components of blood (due to surgery or polycythemia vera).

N.B.: Some patients have a congenital thrombophilia with the abnormalities of protein C, protein S and antithrombin III deficiencies.

Clinical features

- Classical signs & symptoms of DVT → Swelling, calf or thigh pain, redness, tenderness, dilated superficial veins, low grade pyrexia, +ve *Homan's sign* (calf pain on dorsiflexion of foot).
- Pulmonary embolism is the presenting symptom in some patients.

Risk factors

- *Low risk:* Young patients, minor illnesses, operation lasting < 30 minutes with no additional risk factors.
- *Moderate risk:* Patients > age of 40 with debilitating illnesses, undergoing a major surgery but no additional risk factors.
- *High risk:* Patients > age of 40 with serious medical conditions such as stroke, myocardial infarction and undergoing major surgery with additional risk factors (past history of venous thromboembolism, extensive malignant disease, obesity).

Precipitating factors for venous thromboembolism

- Obesity
- Varicose veins
- Immobility (bed rest over 4 days)
- Pregnancy, Puerperium
- High-dose oestrogen therapy
- Previous DVT or pulmonary embolism
- Disease: Thrombophilia, Deficiency of antithrombin III, protein C or protein S
Antiphospholipid antibody or Lupus anticoagulant

Differential diagnosis:

1. Superficial thrombophlebitis

2. Calf muscle haematomas & cellulitis
3. Ruptured plantaris tendon
4. Ruptured Baker's cyst

Diagnosis

- Duplex ultrasound is accurate and noninvasive method. Duplex refers to two modes of ultrasound:
 1. Doppler ultrasound flow examination: accuracy rate 80%—90%. It determines: Phasic (variation)flow, Augmentation (increased)flow, Venous velocity, Differentiate between diseased and normal extremity
 2. B-mode evaluation displays the ultrasound beam as an actual image of the vein being evaluated.
- Venography of the ascending venous system: Radiopaque dye is injected into the pedal veins, and a tourniquet is loosely applied at the ankle to direct the flow of dye into the deep venous system.
- Impedance plethysmography: (accurate in 90% of cases) measures the variations in the volume of calf blood upon releasing a blood pressure cuff placed so as to cause temporary thigh venous occlusion.

N.B.: Duplex ultrasonography & venography are diagnostic. Doppler ultrasonography & impedance plethysmography are confirmatory.

Treatment

- (1) Continuous heparin infusion is given for 5-10 days followed by administration of warfarin or subcutaneous of heparin for 3-6 months, control till prothrombin time is prolonged 2-3 times normal.
- (2) Thrombolytic therapy with streptokinase & urokinase is used if extensive DVT results in impaired perfusion of the extremity.
- (3) Inferior vena cava filter is used if heparin is contraindicated or if pulmonary embolus occurs in spite of adequate anticoagulation therapy. However, this treatment prevents only pulmonary embolism and does not treat the DVT.
- (4) Venous thrombectomy is rarely performed for massive venous obstructions leading to arterial insufficiency and hence venous gangrene.

Prevention

- (1) Simple preventive measures include leg elevation, early mobilization after surgery, the use of support hose (which compresses superficial veins in legs and increases flow in deep veins), and the correction of preoperative risk factors, such as polycythemia vera.
- (2) Intermittent calf compression by means of a pneumatic cuff increases

leg blood flow velocity and helps to prevent stasis.

(3) Pre- & postoperative administration of prophylactic heparin may be effective in preventing DVT. An intermittent subcutaneous dose of 3000—6000 units is given every 8—12 hours. At this dose, heparin activates antithrombin III, inhibits platelet aggregation, and decreases thrombin availability.

(4) Low-dose heparin, low-molecular-weight heparin, dextran and adjusted-dose warfarin are effective in reducing the incidence of DVT, pulmonary embolism & postoperative phlebitis.

N.B.: Advantages of low-molecular-weight heparin include once /day administration (suitable for use outside hospital), low risk of bleeding complications.

Complications

1. Postphlebitic syndrome
2. Phlegmasia alba dolens
3. Phlegmasia cerulea dolens
4. Pulmonary embolism
5. Venous gangrene

Postphlebitic syndrome is a common late complication of DVT, often occurring several years after the acute event.

Clinical presentation:

- Swelling and ulceration
- Chronic valvular incompetence.
- Leg edema (It usually resolves with leg elevation and rest).
- Local superficial venous hypertension leads to edema and interstitial exudation of plasma, cells, and protein → brawny induration from hemoglobin metabolism.

Treatment

- Support hose worn continually to prevent superficial venous hypertension and swelling → prevent ulcers.
- Ligation of local perforating veins, or Unna boot (medicated pressure bandage) is used to lower the venous pressure at the ulcer if it will not heal.

Phlegmasia alba dolens is caused by acute occlusion of the iliac and femoral veins due to DVT.

Clinical presentation: This phlebitis results in a pale cool leg with a diminished arterial pulse due to spasm.

Treatment is thrombolytic therapy followed by heparin administration to prevent progression to phlegmasia cerulea dolens.

Phlegmasia cerulea dolens is secondary to acute and nearly total venous occlusion of the extremity outflow, including the iliac and femoral veins. It is more common in the left leg. Association with another disease is common: For example, 30% of cases occur postoperative, postpartum and in pelvic malignancy.

Clinical presentation

- Physical findings include cyanosis of the extremity with massive edema, severe pain, and absent pulses, followed by venous gangrene.
- Shock may occur as a result of sequestration of a significant amount of blood in the leg.

Treatment

- Thrombolytic therapy followed by heparin administration
- Thrombectomy occasionally if nonoperative therapy is unsuccessful
- Bed rest with leg elevation

Superficial Vein Thrombosis

Superficial venous thrombosis (thrombophlebitis) commonly occurs in superficial veins, particularly when cannulated for an infusion. Spontaneous occurrence can occur in the presence of varicose veins, polycythaemia, polyarthritis and Buerger's disease and may also herald the presence of a visceral cancer (thrombophlebitis migrans). Treatment involves application of compression stockings or bandages and use of anti-inflammatory drugs.

Axillary vein thrombosis

Thrombosis of the axillary vein may occur following excessive exercise. It is occasionally associated with a cervical rib. The arm becomes swollen and the superficial veins distended (Fig. 12.10). Early treatment with anticoagulants may result in rapid resolution. In severe cases the use of fibrinolytic therapy, streptokinase or tissue plasminogen activator may be considered.

Fig. 12.9 Venous gangrene due to polycythaemia Vera.

Fig. 12.8 Intermittent pneumatic compression device commonly used for prevention of deep vein thrombosis.

Fig. 12.10 Collateral venous circulation following thrombosis of the axillary vein, Infrared photograph (*Max Pemberton*).

Varicose Veins

Definition: it is a dilated, tortuous, elongated and superficial vein, which permits flow in the reverse direction due to incompetence of its valves. It is a condition mainly seen in the legs, but can occur in the arms, scrotum and the lower end of oesophagus.

Types of Varicose veins:

- Primary varicose veins due to (1) changes in the vein wall (2) progressive venous dilatation (3) valvular failure. Primary varicose veins is due to congenital predisposition combined with occupational influences.
- Secondary varicose veins occur following a DVT with valvular damage & ↑ venous pressure in the superficial veins, leading to varicosities. Arteriovenous malformations such as congenital A-V fistulas (Klippel Trenaunay syndrome) or extensive cavernous haemangiomas may lead to varicose veins.

Clinical features

- Local pain ache, tiredness and limb swelling (edema) are common complaints, especially after long periods of standing.
- Local hemorrhage into the surrounding area (due to Rupture of a varix).
- Dilated superficial veins → discomfort & cosmetic complaint
- Skin changes → pigmentation or eczema or ulceration.

Examination

The aim is to identify the sites of incompetent connections between the deep and superficial system.

Percussion over a varix while palpating with the other hand at a higher or lower level will help trace the pattern.

The level at which deep-to-superficial reflux is occurring can be checked by the *Trendelenberg test* (Fig. 21.23). The leg is elevated and a rubber tourniquet applied just at or below the saphenofemoral junction. The patient is then asked to stand. Veins fill slowly from arterial inflow but quickly from venous influx. If venous distension below the tourniquet is controlled, the site of reflux must be above it. By moving the tourniquet to different levels in the limb the pattern of incompetence can be mapped out.

A more effective way to demonstrate reflux is to insonate over the site of incompetence and reflux with a portable directional *floppier ultrasound flowmeter* (Fig. 21.24). This is particularly valuable in obese patients or those with recurrent

varicose veins in whom the anatomy may be obscure.

If it is suspected that the varicose veins are secondary to previous deep vein thrombosis (on account of the history or the presence of stigmata of chronic venous insufficiency), *ascending phlebography* of the deep veins may be indicated. This is performed by injecting contrast medium into a foot vein while occluding the superficial system with a tourniquet around the ankle, and then taking serial X-rays as the dye ascends the limb. Before operating on difficult cases of varicose veins, particularly recurrent ones, *varicography* is used, i.e. the direct injection of contrast material into varices to demonstrate deep connections.

Severe varicose veins, especially if in children, of atypical distribution, or associated with cutaneous haemangioma, should raise the suspicion of congenital arteriovenous fistulas (see Fig. 21.17).

Management

- ❖ ***Conservative treatment:*** for mild disease → elastic stocking, weight reduction, regular exercise, elevation of the limb
 - ❖ ***Injection sclerotherapy:*** It is used for moderate cases & cosmetic disfigurement. Idea is to make permanent venous occlusion of varicose veins by injections of empty veins so that the wall adheres to the wall.
Technique → Needle is inserted into the vein with the patient sitting down and his leg in a horizontal position. A latex foam pad is put over the site of the injection and along the whole length of the vein and compression bandaging applied. Injections should be given at the site of perforating veins and following this the patient is encouraged to exercise to the maximum.
 - ❖ ***Surgical treatment:*** The basic principle of operative treatment is to separate the superficial veins from the deep veins by ligation of the communicating vein. The main sites of communicating veins are the groin, the saphenofemoral junction and, behind the knee, the saphenopopliteal junction. To prevent small communicating veins refilling the long saphenous vein or gastrocnemius veins, from the short saphenous vein, removal of both the long and short saphenous vein is recommended. To avoid nerve injury, the long saphenous vein should not be removed below midcalf and great care should be exercised in removing the short saphenous vein.
- (One) ***Saphenofemoral junction ligation:*** An oblique incision is made in the groin starting over the femoral artery and extending 4 cm medially. The long saphenous vein is exposed and the common femoral and superficial femoral veins identified before dividing the long saphenous vein. Having divided the long saphenous vein, all branches should then be isolated and divided. The saphenofemoral junction should be tied flush with the femoral vein. A flexible stripper can then be passed down the long saphenous vein. The end can

be identified in the midcalf region. A 2-mm incision is made and the end of the stripper recovered. Inverse stripping is associated with a lower incidence of bleeding. A 1-Vicryl suture is attached to the end of the stripper; the stripper passed into the long saphenous vein without a stripping head and the suture attached to the vein. Pulling gently on the stripper, the long saphenous vein will invert and can be delivered through a 2-mm incision in the midcalf region.

(Two) *Saphenopopliteal junction ligation*: A skin incision is made over the junction. The deep fascia is incised. Large gastrocnemius veins if present may require division at the same time. The short saphenous vein (under deep fascia) can be identified. The saphenopopliteal junction is where the short saphenous vein enters the side of the popliteal vein. Divide the short saphenous vein, then a thin metal rod is passed down the short saphenous vein 10—15 cm above the ankle. The short saphenous vein is again stripped by inverting it, having tied a 1-Vicryl suture to the stripper and then surrounding the vein.

Residual varices are removed by making 1.5 mm incisions, inserting an Oesch hook, bringing the vein onto the surface and then avulsing lengths of vein.

Venous reconstructive surgery may be carried out for venous occlusion and for deep venous insufficiency. In patients with venous obstruction, venous bypass procedures can be performed.

Simple bypass using vein or prosthetic material, particularly with the larger veins, such as the iliac and vena cava, works well. The indications for venous bypass surgery with established venous obstruction are based on a proper venous assessment. Those patients whose venous pressure rises on exercise are candidates for venous bypass surgery.

For occluded iliac veins, a Palma operation can be carried out. This involves mobilising the long saphenous vein in the opposite leg, tunnelling the distal end of the long saphenous vein across suprapubically and inserting it into the femoral vein below the obstruction. Blood then drains from the affected leg via the long saphenous vein into the femoral vein in the opposite leg.

Treatment of deep venous insufficiency is far from satisfactory. Attempts at valve repair in nonthrombotic disease or valve transplantation have been carried out. In those patients where valvular insufficiency is related to thrombotic disease, the incidence of rethrombosis and valve failure is so high as to make these operations unsatisfactory. Valve repair procedures for primary valve failure have achieved a moderate amount of success. Venous ligation and the diversion of blood through the superficial and competent veins can sometimes be effective. A patient who has deep venous incompetence, yet competent valves in the superficial system, may benefit from femoral vein ligation. These operations are difficult to predict and

should only be carried out after a proper and formal venous assessment.

Venous ulcers. Any patient presenting with ulceration of the lower leg should be properly assessed. The cause of ulceration may be arterial, or associated with other underlying medical conditions.

In those patients with venous ulceration, between 40 and 50 per cent have ulceration due to superficial venous insufficiency alone (Fig. 12.16). Simple, non-invasive tests can exclude those patients with arterial insufficiency and identify those patients with deep venous insufficiency and superficial venous insufficiency. Those patients with pure superficial venous insufficiency, i.e. varicose ulcers, ulcers due entirely to varicose veins, respond well to surgical treatment of their varicose veins.

All patients with venous ulcers will achieve healing, given intensive medical treatment. The difficulty is in preventing them recurring. If the cause of the venous ulceration is superficial venous insufficiency, not only does the ulcer heal, but also the patient will remain ulcer-free.

In those patients with deep venous insufficiency, standard treatment involves debriding the ulcer, keeping it clean and applying adequate compression. Compression can be applied in the form of elastic stocking or using a multilayer bandage technique.

Pulmonary embolism

Definition: Pulmonary embolism is a mechanical obstruction of blood flow in the pulmonary arterial system due to lodgment of a thromboembolus → ↓ cardiac output, pulmonary vasospasm, hypertension, impaired blood oxygenation, and bronchospasm.

Pulmonary embolism is one of the most common causes of sudden death in hospitalized patients.

1. Normal individuals can tolerate a 60%—70% occlusion of the pulmonary vasculature, but patients with preexisting cardiac or pulmonary disease tolerate much smaller occlusions poorly.
2. Pulmonary embolism is frequently sudden and seemingly unheralded, although it is often preceded by the development of small and clinically unrecognized emboli. Only 10% of all autopsy-proven cases of pulmonary embolism are diagnosed pre-mortem.
3. Ninety percent of deaths occur within 2 hours after the onset of the initial symptoms. Therefore, if the patient lives longer than 2 hours, the chance of

survival is very high.

4. Pulmonary embolism develops in 10%—40% of patients with **deep venous thrombosis** (see I B 3). However, approximately 33% of patients with pulmonary embolism have no antecedent symptoms of deep venous thrombosis.
 - a. Thrombosis in the venous system is caused by situations described in **Virchow's triad** (see 1 A 2 b).
 - b. Pulmonary embolus formation can be prevented through the early diagnosis and prevention of deep venous thrombosis.

Risk factors

1. Pregnancy & postpartum period have an incidence 5 times higher.
2. Estrogen therapy is associated with an increased risk of pulmonary embolism 4—7 times that of controls. The risk is dose-dependent and is eliminated within several weeks after cessation of therapy.
3. Heart disease is associated with a 3—4 times higher risk of pulmonary embolus formation. This risk is directly related to the severity of the heart disease.
4. Obesity is associated with a 1 1/2—2 times greater risk of pulmonary embolism.
5. Carcinoma is associated with a 2—3 times greater risk of pulmonary embolism.
6. Major trauma, especially spinal cord injury and pelvic or femoral shaft fractures, carries an increased risk of pulmonary embolus formation.
7. A history of pulmonary embolism increases the risk of later pulmonary embolus formation, especially after surgery.
8. Varicose veins are associated with a 2 times greater risk of pulmonary embolism.
9. Older age-groups are associated with an increased risk of developing pulmonary emboli.

Symptoms of pulmonary embolism range from none to severe cardiopulmonary dysfunction. In general, the more complicated the symptoms, the more unreliable the clinical diagnosis.

1. **Classic signs**—hemoptysis, pleural friction rub, cardiac gallop, cyanosis, and chest splinting—are present in only 24% of patients.
2. **Nonspecific findings**, including tachycardia (in 60% of patients), tachypnea (in 85% of patients), and dyspnea (in 85% of patients), are common. Bronchospasm and pleuritic chest pain also occur frequently.
3. **Electrocardiographic changes**, including arrhythmias and evidence of right ventricular strain, may appear.
4. **Chest x-ray** may be abnormal or totally normal.

- a. Occasionally, a marked diminution of the pulmonary vasculature produces increased radiolucency in the area of the embolus (**Westermarck's sign**).
 - b. Pleural effusion, which is usually hemorrhagic, or pulmonary infiltration may be present, especially in cases of pulmonary infarction, which occurs in 10%—25% of cases of pulmonary embolism.
- 5. Arterial blood gases** frequently show hypoxemia with a low carbon dioxide partial pressure (P_{CO_2}) associated with hyperventilation.
- a. Although serial measurements may document a sudden drop in oxygen partial pressure (P_{O_2}), a single measurement is unlikely to confirm the diagnosis of pulmonary embolism.
 - b. A normal P_{O_2} does not eliminate the possibility of pulmonary embolism.

Diagnosis of pulmonary embolism is based on the results of several tests.

- 1. Pulmonary arteriogram** is the best technique for diagnosing pulmonary embolism. This test is virtually 100% accurate, but it is invasive, as a radiopaque dye is injected directly into the pulmonary artery. The attendant risk of cardiac arrest is small in stable patients, but it can be unacceptably high in hypotensive, unstable patients.
- 2. Pulmonary radioisotope scanning** is less invasive than arteriography.
 - a. Perfusion lung scan.** A radioactive particle, small enough to block a small number of pulmonary capillaries temporarily, is injected. A camera records different views of the uptake in the vasculature.
 - (1) A major difficulty with this test is that many acute and chronic pulmonary diseases can result in similar perfusion defects. It is critical to compare the chest x-ray with the scan to determine the presence of other abnormalities.
 - (2) A normal scan is very reliable in determining the absence of a pulmonary embolus.
 - (a) The presence of segmental or large defects predicts pulmonary embolism in 71% of patients.
 - (b) A subsegmental or small perfusion defect is associated with pulmonary embolism in only 27% of patients.
 - b. Ventilation scan** performed simultaneously with the perfusion lung scan improves the accuracy of the latter. An inert radioactive gas, such as xenon, is inhaled, and the patency and ventilation of the bronchial tree are assessed.
 - (1) **Ventilation—perfusion mismatch** occurs when a perfusion defect is present but the ventilation scan is normal. **Matched ventilation—perfusion defect** occurs when a defect in the same location is revealed by both tests.

- (a) A segmental or large perfusion defect mismatched with a normal ventilation scan is associated with pulmonary embolism in 91% of patients.
 - (b) A subsegmental or small perfusion defect mismatched with a normal ventilation scan is associated with pulmonary embolism in only 27% of patients.
 - (c) A perfusion defect matched with a ventilation defect is associated with pulmonary embolism in 23% of patients.
- (2) When pulmonary embolus is clinically suspected but the lung scan is equivocal, an additional test should be performed to increase the reliability of those results. A **pulmonary arteriogram** is most reliable. A **leg venogram (or venous duplex)** is also useful in documenting the presence of deep venous thrombosis in this situation and, therefore, the likelihood of pulmonary embolism.

Treatment of pulmonary embolism includes both supportive measures to maintain circulatory function and administration of heparin for systemic anticoagulation.

1. **Cardiovascular support** is frequently necessary in patients with significant pulmonary embolism and should be instituted immediately. The supportive measures include oxygen administration, assisted ventilation, correction of cardiac arrhythmias, and treatment of shock by means of adequate hydration and vasopressors.
2. **Heparin as an anticoagulant** should be administered in an initial bolus of 10,000—20,000 units to halt the thrombotic process and to stabilize platelets in the embolus to prevent the release of vasoactive and bronchoactive substances. Administration begins with a continuous intravenous drip of heparin at approximately 1000 units an hour; the dosage is then adjusted to maintain the partial thromboplastin time at 1 1/2—2 times the control time. Heparin is continued for a minimum of 7 days and is followed by long-term anticoagulation therapy for 3—6 months.
3. **Thrombolytic therapy** with streptokinase, urokinase, or tissue plasminogen activator may be used in cases of acute life-threatening pulmonary embolism when cardiopulmonary function is severely compromised as evidenced by shock, profound hypoxemia, or elevated pulmonary arterial pressure. Because thrombolytic therapy results in the lysis of preexisting thrombi, this therapy may be even more dangerous than heparin therapy, and it is contraindicated in patients with recent intracranial hemorrhage, recent surgery, or conditions associated with bleeding, such as peptic ulcer disease, large tumors, or urinary tract

diseases associated with bleeding.

4. **Pulmonary embolectomy** is reserved for very ill patients. If patients remain significantly unstable despite rapid resuscitation and, in the opinion of the attending physician, are too unstable to endure the several-hour treatment with thrombolytic therapy, then embolectomy should be considered. A closed embolectomy using a suction catheter or open embolectomy on cardiopulmonary bypass are the two most acceptable methods.
5. **Long-term anticoagulation** may be maintained by either oral administration of warfarin or subcutaneous intermittent administration of heparin. The warfarin dosage should be carefully regulated to maintain the international normalized ratio (INR) at 2—3 times normal. However, warfarin interacts with many other drugs, and its effectiveness can be severely altered by these drugs and by hepatic disease.

Complications of anticoagulation therapy

1. **Major hemorrhage** requiring transfusions occurs in 1 %—2% of patients on anticoagulants; **minor bleeding episodes** are common in more than 16%; and fatal hemorrhage occurs in 0.1%—1%. The risk of hemorrhage is greater if heparin is administered intermittently or is given to elderly or severely hypertensive patients.
2. **Pulmonary embolism** recurs despite anticoagulation therapy in 1 %—8% of patients.
3. **Heparin-induced thrombocytopenia** occurs in up to 5% of patients on heparin and may be related to the development of heparin-induced antibodies directed toward platelets. All heparin infusions must be discontinued if thrombocytopenia occurs, because of the risk of either hemorrhage or heparin-induced necrosis, a form of tissue necrosis that is related to localized intravascular thrombosis.

Pulmonary embolism is found in approximately 50% of all autopsies. It is the commonest acute lung disorder in hospital patients and an important cause of postoperative death.

Like DVT the majority of pulmonary emboli are silent and many of those which give rise to symptoms are not diagnosed. Their features are protean and non-specific.

Approximately 90% of pulmonary emboli arise from the veins of the lower limbs or pelvis. A few come from the right heart. The recent increase in the use of central venous lines for monitoring and parenteral nutrition has caused an increase in emboli from the subclavian veins.

Major embolism

Massive embolism with occlusion of two-thirds or more of the pulmonary arterial flow causes acute central chest pain, followed by severe dyspnoea, cyanosis, hypotension and collapse.

Early resuscitation is essential and includes cardiac massage, administration of oxygen (at a rate of 6 litres/min) and immediate injection of heparin 15 000 units intravenously to prevent extension of thrombus. Intravenous fluid is given to support right ventricular filling and a pressor agent (i.e. 1 in 1000 noradrenaline made up as 2 mg in 500 ml of isotonic saline) given via a paediatric burette at a rate titrated to maintain blood pressure at a minimum of 80 mmHg systolic.

An urgent pulmonary angiogram is obtained and, if the diagnosis is confirmed and the patient's condition is still serious, thrombolytic therapy is commenced with a loading dose of 250 000 units of streptokinase intravenously, followed by 100 000 units per hour by infusion pump for 24 hours. Hydrocortisone 100 mg intravenously is given before the streptokinase. New thrombolytic agents (tissue plasminogen activator; plasminogen-streptokinase complex; pro-urokinase) may prove to be equally effective but with fewer bleeding complications.

The patient's vital signs are carefully monitored. Thrombolytic therapy is followed by heparin and later by oral antieoagulants.

Rarely pulmonary embolectomy may have to be considered, but few pulmonary emboli occur in circumstances which permit immediate cardiopulmonary bypass. A new technique under trial allows extraction of the embolus from the pulmonary artery under radiological control by means of a catheter introduced through the femoral vein.

Minor embolism

This includes any embolism which does not immediately threaten life. Most emboli are multiple and many do not cause infarction. The symptoms may therefore be insidious and a high level of suspicion is essential.

Dyspnoea may be sudden or gradual. Infarction results in pleuritic chest pain, tachycardia and pyrexia. Haemoptysis is relatively uncommon. Auscultation reveals diminished air entry, moist rales and a friction rub. Some pulmonary emboli cause bronchospasm and are mistaken for asthmatic attacks. Others, by reducing cerebral oxygenation, may present as confusion, impaired consciousness or syncopal attacks. Recurrent pulmonary emboli may over a period of months or years lead to the development of pulmonary hypertension>

Blood gas analysis shows a low P_{O_2} . ECG may show signs of right heart strain with right axis deviation, a prolonged PR interval, depressed ST segments in leads I and II and an inverted T wave in leads II and III. The chest X-ray may be un-

remarkable or may show diminished lung markings, a prominent pulmonary artery and enlarged cardiac shadow. Linear atelectasis may be noted. Later there may be pleural effusion, elevation of the diaphragm and wedge-shaped areas of consolidation.

Lung scanning and phlebography are the principal investigations - A perfusion scan is performed with a gamma-camera after intravenous injection of macroaggregates of albumin which have been labelled with technetium-99m. To distinguish perfusion defects from those caused by emphysematous bullae a ventilation scan may be added. This will reveal areas of increased uptake following the inhalation of xenon-133. If there is doubt about the diagnosis or if thrombolytic therapy is being considered, a pulmonary angiogram is obtained.

At least one-third of the patients who die of pulmonary embolism have had previous episodes of 'herald' embolism. In patients suspected of minor pulmonary embolism, attention must be focused on the lower limbs and residual life-threatening DVT excluded by bilateral phlebography.

Management

It is important, as the term 'thromboembolism' implies, to consider pulmonary embolism and DVT as one disease. [The main objective of management is to prevent further embolism. Systemic heparin therapy is started at once, followed by an oral anticoagulant. Antibiotics and analgesics are indicated for pulmonary infarction. If phlebography reveals loose thrombus in femoral or iliac veins, embolism can be prevented by insertion of a filter into the inferior vena cava (Fig. 21.32). The filter is introduced percutaneously under radiological control through a femoral or a jugular vein. A further indication for insertion of a caval filter is the recurrence of embolism despite anticoagulation. Surgical removal of the thrombus is associated with a high thrombosis rate and is seldom indicated. Oral anticoagulants are continued for at least 6 months.