CHAPTER 10

Arterial Disease

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Assessing the patient with arterial disease

Arterial disease may result in impaired blood supply to the limbs or other end organs, aneurysm formation, or haemorrhage due to trauma or disease. The commonest causes of arterial disease are:

- atherosclerosis;
- embolus;
- thrombosis;
- spasm — Raynaud's phenomenon;
- diabetes;
- Buerger's disease;
- ergot poisoning — usually iatrogenic from migraine therapies;
- vessel injury due to trauma, cold or chemicals.

In addition, there is a genetic component predisposing to both occlusive disease and aneurysm formation. Although there are a large number of diseases that can cause impaired arterial blood flow to the limbs, these conditions are relatively common in the UK. First, and most important, atherosclerotic disease including the premature atherosclerosis seen in diabetics; second, embolism; and third, arteriolar spasm in Raynaud's phenomenon.

Clinical features

Accurate pathological and anatomical diagnosis can be made by careful history taking and clinical examination.

History

The time course of the symptoms is important, ranging from the insidious progression of intermittent claudication of the calves over a period of months or years to the acute onset of ischaemia following an embolus. Sudden onset of pain in the leg suggestive of an embolus should prompt the student to seek a likely source such as atrial fibrillation, recent myocardial infarction or aortic aneurysm. Acute deterioration in a patient with claudication is suggestive of thrombosis on the background atherosclerotic occlusive disease. A history of cold, painful hands since childhood, especially in the female, will be suggestive of Raynaud's disease, and coexistence of connective tissue disorders, such as systemic lupus erythematosus (SLE) or systemic sclerosis (scleroderma) favours Raynaud's phenomenon. The change in colour (pale and deathly white, then blue and finally a dusky red) precipitated by cold immersion is typical.

The symptoms of atherosclerosis occurring
• Pain (in the limb supplied, starting distally and progressing proximally);
• Pallor;
• Paresthesiae;
• Paralysis; and
• Coldness.

Haemorrhage may be overt (bright red blood), or concealed (e.g. closed limb fractures). Symptoms are those of rapidly developing hypovolaemic shock (cold, clamminess, tachycardia, hypotension, loss of consciousness, oliguria progressing to anuria).

Treatment
Closed injuries
• Treat causative factors. If the cause of ischaemia is a tight plaster cast, remove or split the cast. If it is due to a supracondylar humeral fracture, the peripheral pulses should return when the fracture is reduced; if the radial pulse does not return rapidly surgical exploration is indicated.
• Angiography. An angiogram will reveal whether ischaemia is due to spasm, intimal tear or arterial disruption. It may be performed in a radiology suite, or intra-operatively. Partial tears in large vessels may be amenable to intravascular stenting.
• Operative exploration. If a limb fails to reperfus after a fracture or dislocation is reduced, and angiography is unhelpful or shows a tear or block, exploration is mandatory. Either the affected vessel is repaired directly or a segment of saphenous vein interposed to replace the injured area.
• Fasciotomy. Muscle ischaemia leads to swelling and compartment syndrome. The fascial compartments should be opened by splitting the deep fascia widely to relieve compartment pressure.

Open injuries
• Direct compression. Primary measures to staunch haemorrhage should include direct pressure. The use of a proximal tourniquet usually exacerbates blood loss, as it seldom generates sufficient pressure to occlude arterial flow, but does block venous return, which results in increased blood loss.
• Resuscitation. Replace blood loss.
• Exploration. Small vessels that are part of a large collateral supply may be sacrificed and ligated above the site of injury. Partial tears may be directly sutured or closed with a vein patch; complete division often requires interposition of reversed saphenous vein. The use of prosthetic material after trauma is avoided if possible due to the risk of contamination and graft infection.

Aneurysm
An aneurysm is an abnormal permanent dilatation of an artery or part of an artery or the heart. Morphologically it may be fusiform or saccular. The term aneurysm is also used to describe any condition in which there is a sac communicating with an arterial lumen, in which case the aneurysms are false or pseudo-aneurysms. These false aneurysms may also involve arteriovenous fistulae (arteriovenous aneurysms) and dissecting aneurysms, although the latter is now more usually termed aortic dissection rather than a dissecting aneurysm.

Aneurysm types (Fig. 11.1)
Saccular aneurysms
A dilated portion of the artery joins the main lumen by a narrow neck. Mycotic aneurysms are often of this sort, where infection causes a local weakness of the wall which gives way to aneurysmal dilatation.

Fusiform aneurysm
A generalized dilatation of the artery exists, and this is the commonest type of aneurysm to affect the abdominal aorta.

False (pseudo-) aneurysm
Blood leaks out of an artery and is contained by the surrounding connective tissue lined with thrombus. The resultant blood collection communicates with the artery so it is pulsatile and expansile. It will either thrombose spontaneously or enlarge and rupture.
**ANEURYSMS**

(a) True
- Saccular
- Fusiform

(b) False

(c) Arterio-venous

(d) Dissecting

**Fig. 11.1** The types of aneurysm.

**Arteriovenous aneurysm**
A communication between adjacent artery and vein, this is a false aneurysm intervening between artery and vein.

**Dissecting aneurysm**
Blood forces a passage through a break in the intima of a vessel, creating a separate 'false' channel between the external layers of the arterial wall. This false channel may then either rupture back into the lumen, or rupture out of the adventitia externally. Over the arterial segment where flow is extraluminal, vessels taking their origin from the true lumen will be deprived of blood (see aortic dissection, p. 68).

**Aetiology**
**Congenital**
The small berry aneurysms that occur intracranially on the circle of Willis* and also the less common arteriovenous aneurysms and fistulae in the limbs are congenital.

**Degenerative**
Atheromatous degeneration of the vessel wall is the commonest cause of a true aneurysm.

* Thomas Willis (1612–75), Physician and Anatomist, first in Oxford then in London.

**Traumatic**
Penetration or weakening of the arterial wall by a penetrating wound such as a bullet or knife, or iatrogenic injury during catheterization for angiography and angioplasty, may result in a true aneurysm or false aneurysm, possibly with an associated arteriovenous fistula.

**Infection**
Mycotic aneurysms, previously seen in the thoracic aorta of patients with tertiary syphilis, are now more commonly seen in the abdominal aorta or femoral artery as a consequence of salmonellosis, or resulting from mycotic emboli in patients with infective endocarditis. Patients with immunodeficiency, whether as a result of immunosuppression for organ transplantation or as a result of human immunodeficiency virus (HIV) infection are prone to mycotic aneurysms from unusual bacteria or fungi.

**Inflammatory**
This is a subtype of atherosclerotic and mycotic aneurysms in which there is an immune response to components in the aneurysm wall resulting in a dense inflammatory response with a ring of inflammatory tissue surrounding the lumen. In non-infective cases this may sub-
side with corticosteroid treatment. It is also associated with retroperitoneal fibrosis and ureteric obstruction. The erythrocyte sedimentation rate is raised.

Clinical features of true aneurysms
The clinical features of an aneurysm depend on its location, and it may present with symptoms far distant from the aneurysm itself. Abdominal aortic aneurysms may present with back pain but they are frequently asymptomatic and picked up incidentally during the course of investigation for some other condition. The patient may feel a sensation of abdominal bloating or may have noticed the pulsatile swelling, or may present with distal emboli from the sac contents. When the peripheral arteries are involved it is more common to find a complaint of a pulsatile mass or distal ischaemia. On examination there is a dilatation along the course of the artery. The aneurysm itself is both pulsatile and expansile. In smaller peripheral aneurysms, direct compression may empty the sac or diminish its size, and pressure on the artery proximal to the aneurysm may reduce its pulsation. If the feeding vessel has a narrow orifice there may be a thrill and bruit, and if there is an arteriovenous communication a machinery murmur is audible.

Differential diagnosis
- A dilated, tortuous, atheromatous artery; common in elderly subjects.
- A mass overlying or displacing the artery superficially. In the abdomen a carcinoma of the pancreas may have a transmitted pulsation from the underlying aorta but will not be expansile, distinguishing it from an aneurysm.

Complications
- Rupture. The likelihood of rupture increases as the diameter of the artery increases relative to its normal size.
- Thrombosis. Thrombus lines the wall of the aneurysm, and may dislodge or extend to completely occlude the artery. This results in acute impairment of the distal circulation.
- Embolism. Lining thrombus may detach and embolize to distal circulation, either as small emboli resulting in digital ischaemia or as a large mass of thrombus threatening the entire limb.
- Pressure. Adjacent structures may be eroded or displaced. Hence backache and sciatica are common in patients with large abdominal aortic aneurysms, and occlusion of the femoral vein is common with large femoral aneurysms.
- Infection. An aneurysm may become infected or arise secondary to infection and consequent weakening of the arterial wall.

Special investigations
- Abdominal X-ray. This may show calcification in the wall of the aneurysm. A lateral film is particularly helpful in demonstrating aortic aneurysm calcification.
- CT, MR and ultrasound scanning may delineate the size and extent of an aneurysm, its relationship to other structures and evidence of leakage.
- Angiography underestimates the size and extent of a true aneurysm, as it images the lumen, which is usually narrowed by thrombus. In addition, it may be dangerous, as the guide wire or cannula may perforate the aneurysm wall. It is useful in false aneurysms to identify the connection between the artery and the sac.

Treatment
The treatment of an arterial aneurysm depends on its nature (true or false), location, size and symptoms. Abdominal aortic aneurysms should be resected when they become symptomatic or reach a size at which the risk of rupture outweighs the likely operative mortality for the individual. Aneurysms of other large vessels, such as the femoral and popliteal arteries, may be replaced with a prosthetic graft or saphenous vein, while a small peripheral aneurysm can usually be excised without endangering the distal circulation, assuming an adequate collateral circulation. False aneurysms and mycotic aneurysms are more prone to rupture and require urgent attention.

Abdominal aortic aneurysm
Dilatation of the abdominal aorta is a common finding in older males, and in those with a posi-
tive family history. Around 10% will have a coincidental popliteal aneurysm. Small aneurysms (less than 4 cm), are generally benign and grow slowly (1–2 mm per annum). As they enlarge, the growth rate increases, and the risk of symptoms increases. The most feared complication is rupture. This has an incidence of around 5% per annum once the aneurysm reaches 6 cm in anteroposterior diameter. With operative mortality at or below 5%, resection of the aneurysm is advised at 5.5–6 cm as prophylaxis against rupture.

Management
Patients with small asymptomatic aortic aneurysms are followed up by regular ultrasound scans to monitor the rate of growth. Once the threshold diameter is reached, or the aneurysm becomes symptomatic, elective surgical resection is advised. Preoperative assessment includes evaluating the patient’s operative risk by screening for coincident cardiac disease (by resting and exercise, ECG or echocardiography) and for carotid arterial disease. This information may affect the decision to operate.

Operative management
Surgery involves replacement of the aneurysmal aorta with an artificial graft, usually made of Dacron. Endoluminal repair of some aneurysms may be possible in some patients by passing a graft via the femoral artery up to the aorta, and anchoring it proximally and distally with self-expanding stents. This avoids a major abdominal procedure.

Complications of surgery
- Renal failure. The renal arterial ostia are often compressed when the aorta is clamped and are thus rendered ischaemic for the duration of cross-clamping. The left renal vein may be ligated and divided as part of the operative procedure. Hypotension pre- or postoperatively may exacerbate the renal injury.
- Distal embolization. Thrombus from the sac may disperse distally and block the small vessels in the foot and lower leg causing acute ischaemia, in this context called ‘trash foot’.
- Myocardial infarction. Coronary artery disease is common in the population who develop aortic aneurysms. Cross-clamping the aorta during surgery dramatically increases the peripheral resistance against which the heart must work, and this extra stress, coupled with the metabolic stress that occurs when the legs are reperfused, may precipitate a myocardial infarct.
- Graft infection. This occurs in about 1% of cases and may lead to an aorto-enteric fistula.

Ruptured abdominal aortic aneurysm
A patient with a ruptured aneurysm usually presents with severe back pain, frequently with radiation to the groin, and the diagnosis is often confused with renal colic, although renal colic is less likely in the elderly population (60 years and over) than a ruptured aneurysm. Occasionally, only groin or iliac fossa pain may be the presenting symptom. Sometimes, the pain is confined to the epigastrium, leading to the mistaken diagnosis of myocardial infarction.

Fifty per cent of patients die from the initial rupture and never reach hospital. Those that do reach hospital are usually profoundly shocked (cold, clammy, tachycardic, hypotensive) with generalized abdominal tenderness. A pulsatile mass is an indication for immediate laparotomy. In most patients reaching hospital, the rupture is contained by the retroperitoneum, helped by the hypotension following rupture. Injudicious fluid replacement to restore normal blood pressure prior to surgery may lead to further bleeding and breaches of the retroperitoneum, resulting in haemoperitoneum and exsanguination. Occasionally, the aortic aneurysm may rupture into the inferior vena cava (aorto-caval fistula, diagnosed by a machinery murmur and pulsatile veins) or into the duodenum (aorto-duodenal fistula, diagnosed by coexistence of an aneurysm and brisk haematemesis).

Acute aortic expansion
The aneurysm may expand acutely and result in the typical pain of rupture but without the
haemodynamic consequences of a bleed. Indeed, some patients are paradoxically hypertensive during this phase. At laparotomy the aneurysm sac is found to be oedematous or a local rupture is found.

Special investigations
Investigation of a patient with a suspected rupture should only be performed if there is reasonable doubt about the diagnosis, as delay may be fatal. Investigation should answer two questions:

1. **Does the patient have an aneurysm?** Often an aneurysm is difficult to feel because of hypotension and a large retroperitoneal haematoma masking the sac. A plain X-ray will frequently show calcification in the wall of an aneurysm, particularly in an aortic aneurysm. A dorsal decubitus film is particularly valuable, showing the calcified sac displacing the bowel anteriorly.

2. **Is the aneurysm bleeding?** A patient known to have an aneurysm presents with abdominal pain and is normotensive. In this context a CT is useful to identify a leak, but no modality will distinguish an uncomplicated aneurysm from one that has acutely expanded and that may imminently rupture.

**Treatment**
Urgent surgery is indicated in anyone with a high suspicion of a ruptured abdominal aortic aneurysm. Prior investigations are only indicated where doubt exists. Even with prompt surgery there is a significant mortality, together with morbidity, including acute renal failure, myocardial infarction and distal embolization.

**Popliteal aneurysm**
Popliteal aneurysms are the most common peripheral aneurysms, and historically were the first to be diagnosed and treated surgically. They are usually associated with other aneurysms, and are frequently bilateral.

**Clinical features**
Popliteal aneurysms are generally asymptomatic. When they do present it is either in association with distal embolization of sac contents leading to claudication or digital infarction, acute occlusion or rupture (uncommon). Examination confirms a prominent pulsation in the popliteal fossa, often extending proximally. Distal pulses should be sought for evidence of embolization.

**Special investigations**
- **Duplex ultrasonography.** Delineates the extent and size of the aneurysm.
- **Angiography.** To examine the arterial tree distal to the aneurysm.

**Treatment**
Symptomatic aneurysms should be treated by femoral to distal popliteal bypass, with ligation of the feeding vessels. Aneurysms containing clot should be repaired electively. Distal emboli may be treated by thrombolysis at the time of surgery.

**Assessing the patient with arterial disease**

Diseases of the arteries may result in impaired blood supply to the limbs. It is important to remember when assessing a particular patient that arterial disease is rarely localized to the peripheries; involvement of other organs, particularly the heart, central nervous system and abdominal viscera must be kept in mind.

The vascular diseases to be considered are:
- atherosclerosis;
- diabetic micro-angiopathy;
- thromboembolism;
- Raynaud’s phenomenon;
- Buerger’s disease;
- ergot poisoning—usually iatrogenic from migraine therapies;
- arterial injury due to trauma (see p. 73); and
- cold or chemical injury.

By far the commonest of these is atherosclerosis, which may often be complicated by coexisting diabetes.

**Clinical features**
Accurate pathological and anatomical diagnosis can often be made by careful history-taking and clinical examination.
Assessing the patient with arterial disease

History
The time course of the symptoms is important, ranging from the insidious progression of intermittent claudication of the calves over a period of months or years to the acute onset of ischaemia following an embolus. Sudden onset of pain in the leg suggestive of an embolus should prompt the student to seek a likely source such as atrial fibrillation, recent myocardial infarction or aortic aneurysm. Acute deterioration in a patient with claudication is suggestive of thrombosis on the background of atherosclerotic occlusive disease. A history of cold, painful hands since childhood, especially in the female, will be suggestive of Raynaud’s disease, and coexistence of connective tissue disorders, such as systemic lupus erythematosus (SLE) or systemic sclerosis (scleroderma) favours Raynaud’s phenomenon. The change in colour (pale and deathly white, then blue and finally a dusky red) precipitated by cold immersion is typical.

Symptoms of atherosclerosis occurring in a young person, especially a heavy smoking male, is typical of Buerger’s disease.

Ergot poisoning is occasionally seen in patients with migraine who are consuming large doses of ergotamine.

It is important to determine the degree of handicap produced by the symptoms, for on this will depend the selection of patients for reconstructive surgery. Similarly, if a patient suffers from angina pectoris as well as intermittent claudication, there may be more handicap from the angina than from the claudication, and more benefit from coronary revascularization (see p. 65).

Atherosclerosis is a generalized disease, and the cerebral circulation is often affected in addition to the circulation in the legs. Thus, a history of intermittent loss of consciousness, blindness and hemiparesis is of importance and may indicate coexisting carotid artery disease.

Examination
Careful clinical examination will usually provide a very clear indication of the severity and nature of the ischaemic disease. It is important that attention should be directed to other systems of the body, especially the heart and blood pressure (is the poor circulation due to a poor cardiac output?).

Heart rhythm. The presence of atrial fibrillation or other cardiac arrhythmias should be noted, particularly if there is a history of acute limb ischaemia or stroke. The heart should be examined with particular attention to the apex beat (ventricular aneurysm) and auscultated for evidence of valvular disease (e.g. mitral stenosis).

Inspection of limbs. Attention is then directed to the legs. Inspection may reveal marked skin pallor, an absence of hairs, ulcers (usually lateral malleolus and often in the interdigital clefts) and gangrene, all being evidence of impaired circulation. Fixed staining (purpuric areas not blanching on pressure) in the context of an acutely ischaemic limb is a sign of irreversible tissue injury. A tense, tender calf with impaired dorsiflexion in acute ischaemia signifies compartment compression and requires urgent fasciotomy in addition to revascularization.

Venous guttering. The veins of the foot and leg in a patient with diminished arterial supply are often very inconspicuous compared with normal veins. Indeed, the veins may be so empty that they appear as shallow grooves or gutters, especially in the elevated limb.

Buerger’s test. Buerger’s test involves raising the legs to 45° above the horizontal and keeping them there for a couple of minutes. A poor arterial supply is shown by rapid pallor. The legs are then allowed to hang dependent over the examination couch. The feet repurpose with a dusky crimson colour in contrast to a normally perfused foot, which has no colour change. In severe cases, the foot may remain pale and some time may pass before the reactive hyperaemia appears.

* Leo Buerger (1879–1943), Born in Vienna; Urologist, Mount Sinai Hospital, New York, USA.
Capillary return. The speed of return of capillary circulation after the blanching produced by pressure is a very useful gauge of the peripheral circulation.

Skin temperature. In addition to the pulses, skin temperature can be readily assessed by palpation, which is especially sensitive when the dorsum of the hand is used. A difference between the temperatures of one part of the leg and another or between the two legs can be ascertained when it is as small as 1°C. A clearly marked change of temperature may reveal the site of blockage of a main artery.

Peripheral pulses. The peripheral pulses throughout the body should be examined. Whereas normal pulsation can be appreciated easily, palpation of weak pulsation requires practice, care and, above all, time. The presence of a weak pulse that is definitely palpated is of considerable significance diagnostically and can be important prognostically, as even a weak pulse means the vessel is patent.

Careful recording of the peripheral pulses will often clearly delineate a blockage in the arterial system. For instance, the presence of a good femoral pulse and absence of pulses distal to the femoral suggests a superficial femoral arterial block. Ischaemia of the digits in the presence of all pulses including the radial and ulnar pulses is a typical finding in Raynaud’s phenomenon.

Aortic pulsation. The abdomen should be examined for any evidence of abnormal aortic pulsation; the popliteal and femoral arteries are also often aneurysmal and should be examined with this in mind. If distal pulses are absent then it is possible that no aortic pulsation will be felt due to thrombosis of the terminal aorta.

Auscultation of vessels. In all areas where pulses are felt, auscultation should be performed. Partial blockage of arteries very often causes bruits, which are usually systolic in timing. They may even be felt as thrills. Arteriovenous communications will produce continuous bruits with systolic accentuation (machinery murmur) and pulsating dilated veins.

Ankle brachial pressure index (ABPI). The ABPI should be measured in each leg as part of the routine examination. A Doppler probe is held over the brachial artery and a blood-pressure cuff inflated to occlude the blood flow. As the blood-pressure cuff is deflated, a Doppler signal reappears and a systolic pressure can be recorded. Similar pressure readings are taken from the dorsalis pedis and posterior tibial arteries with a cuff just above the ankle. The ABPI is the ratio of pressure at the foot pulse to that at the brachial artery. Values less than 0.5 indicate significant ischaemia. Heavily calcified vessels may be incompressible and give false high readings.

Exercise test. If it is difficult to obtain a clear history of the exact severity of intermittent claudication, the patient should be taken for a walk with the doctor who observes the time and nature of the onset of symptoms. Measurement of the ABPI pre- and post-exercise may show a significant fall from normal after exercise, indicating a critical stenosis in the proximal vessels.

Special investigations
- Urine for sugar and blood glucose to exclude diabetes, a common accompaniment of peripheral artery disease. If necessary, a fasting blood glucose estimation or glycosylated haemoglobin (HbA1c) may be necessary.
- Haemoglobin estimation to exclude anaemia or polycythaemia. Anaemia may sometimes precipitate angina or claudication.
- Erythrocyte sedimentation rate and CRP are raised in inflammatory and mycotic aneurysms.
- Serum cholesterol is often raised in atherosclerosis, and is treatable.
- Electrocardiogram (ECG) to exclude associated coronary disease.
- Echocardiogram to confirm valvular lesions, mural thrombus on an akinetic ventricular wall, ventricular aneurysm and atrial myxoma.

* C. J. Doppler (1803–53), Professor of Physics, University of Vienna, Austria.
- **Chest X-ray.** Bronchial carcinoma is a common finding in end-stage vascular disease, both caused by smoking. Chest X-ray also allows assessment of the cardiac silhouette.
- **Doppler ultrasound.** The Doppler ultrasonic probe can be used to generate a waveform of the arterial pulse in the peripheral vessels in addition to allowing the measurement of pressure and derivation of the ABPI. The waveform is biphasic in normal elastic arteries, but becomes monophasic in hardened arteries.
- **Duplex ultrasonography.** Combining Doppler ultrasound with real time produces duplex scanning, which is a sensitive method of imaging blood vessels. By measuring flow patterns it can quantify the degree of stenosis of a vessel because the blood velocity increases as it crosses a stenosis in order to maintain the same flow rate. Summation of scans produces a result similar to angiography, but non-invasively. It is particularly useful in assessing carotid artery disease.
- **Arteriography** is used to determine the site and extent of a blockage, and is performed if reconstructive surgery or angioplasty is contemplated to identify the severity and distribution of disease, whether atheromatous plaques, stenoses, or complete blocks as well as demonstrating run-off (Fig. 11.2).
- **Angioplasty.** At the time of arteriography, a stenosed segment of artery may be dilated using a specially designed balloon catheter. This percutaneous transluminal angioplasty (PTA) is now commonly undertaken for coronary as well as peripheral arteries. It may be combined with endoluminal stenting to maintain the patency of the dilated segment.
- **Computed tomography (CT) and ultrasound scanning.** These are useful in determining the presence and extent of aneurysmal disease, and their relation to other structures.

**Principles of treatment**
There are two treatment principles underlying the management of patients with vascular disease; both come under the adage of *primum non nocere* (first do no harm).

1. **Treat handicap not disability.** Treatment must be tailored to the patient. If a patient claudicates at 500 m (the disability), but seldom needs to walk that distance, there is no handicap with this disability and therefore the patient needs no treatment. However, if the patient is young and work requires him or her to walk 500 m (e.g. on a milk round) then the patient is handicapped by the disability and merits treatment.

   There are usually two treatment options: conservative management and surgery. Reconstructive surgery can produce dramatic results but at a risk.

2. **Prophylactic surgery is only appropriate when the risk of the event outweighs the risk of the procedure.** For example, surgical repair of an aortic aneurysm is advised when the risk of rupture (which is usually fatal) outweighs the operative mortality. If the patient is a poor operative risk then the threshold for surgery increases.

**Atherosclerotic arterial disease**
Arterial disease may be divided into occlusive disease and aneurysmal disease (see p. 74), the commonest cause of both being atherosclerosis. Both manifestations may coexist; hence patients with an abdominal aortic aneurysm frequently also have coronary artery disease.

**Aetiology**
Many factors have been shown to contribute to the genesis of atherosclerosis. While there is a

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**RISK FACTORS FOR Atherosclerotic Disease**

- Smoking.
- Hyperlipidaemia.
- Hypertension.
- Diabetes mellitus.
- Male sex.
- Increasing age.
- Family history.
familial tendency to the disease, the most common aetiological factors are smoking, hyperlipidaemia and hypercholesterolaemia, hyper-tension and diabetes. It is a disease that affects predominantly males, although with increasing age females become more susceptible.
Smoking
There are three components of the serious effects of smoking in atherosclerotic disease.
1 Nicotine, which induces vasospasm.
2 Carbon monoxide, present in inhaled smoke, which is taken up by haemoglobin to form carboxyhaemoglobin, which dissociates slowly and is, therefore, unavailable for oxygen carrying, resulting in relative tissue hypoxia.
3 Increased platelet stickiness, with increased risk of thrombosis formation.

Hyperlipidaemia
Raised cholesterol and raised triglycerides are both implicated in vascular disease and cholesterol-lowering agents have been shown to reduce the risk of death from coronary artery disease in patients with hypercholesterolaemia.

Diabetes
Diabetic patients are prone to higher incidence of atherosclerosis, and also are at risk of diabetic micro-angiopathy resulting in poor tissue perfusion, ulceration and gangrene, due to both tissue perfusion and the neuropathy that accompanies diabetes. Two clinical manifestations of diabetic arterial disease should be distinguished.
1 The young diabetic patient with peripheral gangrene but with good pulses in the limb. Control of infection and improved diabetic control, together with local debridement of the gangrenous tissue, usually results in limb salvage.
2 The elderly patient with atherosclerosis (as shown by absent peripheral pulses) who is also diabetic. Here the diabetes makes the prognosis of the disease much worse.

Atherosclerotic occlusive arterial disease
Occlusive disease results in ischaemia of the end organ or tissue that is supplied. In the peripheral arteries the three cardinal features are exercise-induced pain (intermittent claudication), which may progress as the disease progresses to pain at rest and gangrene. The progression is not necessarily a smooth one in the early stage, with deterioration in claudication distance, followed by some improvement as collateral circulation develops, before further deterioration due to thrombosis.

Parallels to peripheral artery occlusive disease are present in the other circulatory systems.

Coronary occlusive disease
Angina pectoris is the coronary circulation’s equivalent of intermittent claudication, with pain on exertion as oxygen demand exceeds supply, and rest pain being analogous to unstable angina with resultant infarction if the coronary circulation is not revascularized either by thrombolysis or bypass surgery.

Mesenteric occlusive disease
Mesenteric angina occurs when the blood supply to the gut is impaired and follows the ingestion of food. Patients present with pain after meals, a history of marked weight loss and fear of eating because of pain. Acute occlusion results in bowel infarction.

Cerebral occlusive disease
In the cerebral circulation, progressive occlusive disease manifests as dementia, while small emboli causing occlusion of small vessels may appear as transient ischaemic attacks, complete occlusion resulting in cerebral infarction in the absence of a collateral circulation.

Intermittent claudication
Intermittent claudication manifests as a gripping, tight, cramp-like pain in the calf on exercise, and usually affects one leg in advance of the other. The pain disappears on resting. Pain that is present on standing and that requires the patient to sit down before it is relieved is more typical of cauda equina compression (spinal claudication) (p. 128).

The pathology lies in one of the main arteries supplying the leg. Calf claudication is usually due to a lesion in the thigh, while buttock claudication is due to a reduced blood flow down the internal iliac arteries, due to a lesion either there or higher up in the common iliac artery or the aorta. Bilateral buttock claudication is associated with impotence, as both in-
ternal iliac arteries are compromised (Leriche's syndrome: absent femoral pulses, intermittent claudication of the buttock muscles, pale cold legs and impotence).

Management
Conservative treatment
If patients stop smoking and continue exercise, or better still are enrolled into a programme of supervised exercise, over one-third of patients will extend their claudication distance due to the development of collateral vessels that bypass the blockage. Only one-third will deteriorate. In addition to cessation of smoking, the other risk factors for the development of arterial disease should be treated, so diabetes should be sought and treated aggressively and hyperlipidaemia if present should be treated.

The work performed by the legs is greater if the patient is overweight so strict dieting may well improve exercise tolerance. If the claudication is limited to the calf, raising the heels of the shoes 2 cm will relieve the work performed by the calf muscles, and therefore allow the patient to walk a greater distance. Careful chiropody is important. Gangrene can commence from a minor trauma such as faulty nail or corn cutting and may result in limb loss.

Interventional treatment
If claudication is a significant handicap to the patient, the possibility of reconstructive surgery or angiographic intervention should be considered.

Special investigations
Special investigations detailed above should be arranged, including the following in particular:

- Arteriography. This should include the aorta and iliac, femoral, popliteal and distal arteries on the affected side. In particular, this should look for short (less than 10 cm) occlusions or significant (greater than 70%) stenoses, which would be amenable to angioplasty.
- Duplex sonography. Duplex scanning is now replacing angiography in many centres. It takes longer to perform and is more subjective but can give better information as to the significance of stenoses and has the benefit of being non-invasive.

Treatment choices
- Angioplasty. Angioplasty involves blowing up a balloon within the vessel to stretch and fracture the stenosis or blockage, and allow more blood to pass through. This is most successful with concentric stenoses or blocks in the iliac system and is less successful with long blocks over 10 cm, particularly in the distal femoral and popliteal arteries. An endovascular stent may be used to maintain patency. Angioplasty carries the risk of distal embolization and vessel perforation.
- Thrombolysis. Where there has been an acute deterioration in claudication distance due to thrombosis occurring on a background of pre-existing disease, thrombolysis may be appropriate. A fibrinolytic enzyme such as streptokinase or tissue plasminogen activator (TPA) is infused into the clot, which it dissolves. Complete dissolution of thrombus takes time, so the technique is not appropriate where limb viability is acutely threatened.
- Bypass surgery. Bypass surgery should not be undertaken for minimal symptoms and is now generally reserved for limiting claudication or rest pain. Complications include intimal dissection, distal embolization and graft thrombosis, which worsen the initial situation.

Critical ischaemia

Critical ischaemia may be defined as rest pain, ulceration or gangrene associated with absent pedal pulses.

Rest pain
Rest pain occurs when the blood supply to the leg is insufficient. Initially the pain occurs at night
after the foot has been horizontal for a few hours in bed. The patient gains relief by sleeping with the leg hanging out of bed. As the disease progresses the pain becomes continuous.

Gangrene
The presence of gangrene indicates a severe degree of vascular impairment. Typically, it occurs in the toes or at pressure areas on the foot, particularly the heel, over the malleoli or on the plantar aspect of the ball of the hallux. Gangrene results from infection of ischaemic tissues. Minimal trauma, such as a nick of the skin while cutting the toenails or an abrasion from a tight shoe, enables ingress of bacteria into the infarcted tissues; the combination of these two factors results in clinical gangrene.

Investigation
Critical ischaemia needs investigating with great urgency to relieve the patient’s pain and to prevent irreversible damage leading to limb loss. The investigations are the same as those used to evaluate claudication.

Treatment
Non-operative treatment
Arteriography and angioplasty. Arteriography should be performed with a view to angioplasty or stenting where possible, and to identify surgically reconstructable disease.

Lumbar sympathectomy. Sometimes, palliation may be achieved by lumbar sympathectomy, which increases the blood supply to the skin, and which may be performed percutaneously. The small increase in blood supply may be sufficient to allow an ulcer to heal but will not generally improve rest pain.

Operative treatment
Reconstructive surgery. Successful surgical reconstruction demands four things.
1 Inflow. A good arterial supply up to the area of blockage is necessary to ensure that enough blood can be carried distally via the conduit to the ischaemic area.
2 Outflow (run-off). There should be good vessels below the area of disease on to which a conduit can be anastomosed. If there is nowhere for the blood to go the conduit will occlude.
3 The conduit. A graft of saphenous vein, reversed or used in situ with valve destruction, or an inert prosthetic material such as polytetrafluoroethylene (PTFE), may be used for the conduit to take blood from the proximal to the distal segment of the artery beyond the blockage. In grafts that start and finish above the knee there is little to choose between PTFE and vein in terms of long-term patency, but a graft that crosses the knee is much more likely to remain patent if it is saphenous vein rather than PTFE. Infection is less likely with autologous vein.
4 The patient. Critical ischaemia is often the first sign of the end-stage vascular disease which inevitably results in death. Surgery for critical ischaemia has a high mortality reflecting this general deterioration.

Amputation. Pain that is not controlled by sympathectomy or reconstructive surgery, and gangrene that is associated with life-threatening infection are indications for amputation of the limb or part of the limb. The general principle is to achieve a viable stump that heals primarily, and a secondary goal is to make the stump as distal as possible.

Carotid artery disease (Fig. 11.3)
Atheroma usually affects the bifurcation of the carotid artery into the internal and external carotid arteries. Atheromatous plaques may ulcerate and thrombus form on their surface. If this thrombus breaks off, it forms an embolus comprising platelet clumps or atheromatous debris. This may impact in the ipsilateral retinal artery producing ipsilateral blindness, or the cerebral arteries of the ipsilateral hemisphere producing contralateral paralysis. Alternatively,
INTERNAL CAROTID ARTERY STENOSIS

Fig. 11.3 Symptoms and treatment of carotid artery stenosis.

the atheroma may so narrow the artery that blood flow is critically limited or totally occluded, producing similar symptoms.

Clinical features
- **Amaurosis fugax.** The patient commonly complains of a loss of vision like a curtain coming down across his or her visual field. The blindness is unilateral and usually lasts a few minutes.
- **Cerebrovascular accidents (stroke).** Emboli in the carotid territory of the cerebral circulation of the ipsilateral hemisphere will result in symptoms affecting the contralateral side of the body, commonly loss of use of the arm. If the dominant hemisphere is involved, speech may be affected.
- **Transient ischaemic attack (TIA).** By definition, these mimic strokes but last less than 24 hours.
- **Cerebral hypoperfusion.** Bilateral severe stenoses may result in critical ischaemia in the brain such that cerebral or physical exertion may result in relative hypoperfusion and confusion or TIA.

Examination may reveal a bruit over the affected side (although very tight stenoses are often silent) and evidence of vascular disease elsewhere. During an attack, unilateral weakness affecting arm or leg, dysphasia, and retinal emboli and infarction may be noted.

Differential diagnosis
Other causes of focal neurological deficits include hypoglycaemia, focal epilepsy, migraine, intracerebral neoplasm and emboli secondary to cardiac arrhythmias and valve disease.
Special investigations
- **Duplex ultrasonography.** This gives a non-invasive assessment of degree of stenosis and is useful to screen for the disease.
- **Angiography** allows accurate assessment of the degree of stenosis, but carries the risk of dislodging thrombus and precipitating an embolic stroke.
- **Magnetic resonance (MR) angiography** can also give good images of the carotid vessels and also allows good visualization of the vertebral system to assess the complete cerebral perfusion. It is less accurate in the measurement of the degree of stenosis.
- **MR/CT of the brain** is indicated if any doubt over symptoms exists, as intracranial tumours may mimic carotid artery disease, and may coexist.
- **Cerebral reactivity.** If cerebral perfusion is marginal, with bilateral stenoses or an occlusion on one side and stenosis on the other, the haemodynamic response to stress can be gauged by measuring the change in cerebral blood flow using intracranial duplex scanning while the patient breathes CO₂, which causes vasodilatation, so intracranial blood flow should increase. If there is a critical stenosis affecting the carotid artery, and the collateral cerebral circulation provided by the circle of Willis is not intact or sufficient, there will be no reactive increase in perfusion.
- **ECG/echocardiography.** This may be necessary to exclude a cardiac cause of cerebral symptoms.

**Treatment**
Patients who have had recent TIA, amaurosis fugax or stroke with full recovery in the presence of an internal carotid stenosis of 70% or more are at high risk of a subsequent stroke in the months following. These patients benefit from endarterectomy to remove the diseased intima and re-establish normal carotid flow. All patients should be started on aspirin upon diagnosis, and this should be continued indefinitely as prophylaxis against further events. Patients with asymptomatic stenoses may also benefit from surgery, but here the risk/benefit ratio is not as favourable.

**Carotid endarterectomy** is performed as prophylaxis against future stroke. The diseased intima is removed, and peroperatively a shunt is used to keep blood flowing to the brain.

**Complications of carotid endarterectomy**
- **Death and disabling stroke.** Up to 5% of patients will suffer a stroke, some of whom will die as a consequence.
- **Haemorrhage.** Bleeding is common, as the patients are on aspirin therapy. Occasionally, postoperative haemorrhage requires re-exploration.
- **Hypoglossal neuropraxia.** The hypoglossal nerve crosses the upper part of the incision and may be damaged during surgery resulting in a hypoglossal palsy, manifested by protrusion of the tongue to the ipsilateral side.
- **Reperfusion syndrome.** The sudden increase in blood flow to the brain may result in cerebral oedema and fitting or haemorrhage. Good postoperative blood-pressure control is therefore vital.
- **Restenosis.** The vessel may restenose at the site of the arteriotomy. To overcome this, a patch is usually used, made from saphenous vein or prosthetic material such as PTFE or Dacron.

**Raynaud's phenomenon**

**Clinical features**
The syndrome occurs due to intermittent spasm of the small arteries and arterioles of the hands (and feet). Spasm is usually precipitated by cold exposure. During the spasm the hands go white. As the vasoconstriction resolves, the pallor changes to cyanosis and then crimson red as reperfusion and hyperaemia occurs, the process commonly taking 30–45 minutes.

* Maurice Raynaud (1834–81), Physician, Paris, France.
The underlying cause
The history and physical signs may reveal a cause for the embolus.

- **Atrial fibrillation** is by far the most common cause of arterial emboli. The atrial fibrillation may be due to rheumatic heart disease or myocardial ischaemia.
- A **mural thrombus**, typically following a myocardial infarction, may also dislodge and embolize. This typically occurs around 10 days post infarct.
- **Aortic dissection** is an uncommon differential diagnosis, when ischaemia may progress down the body, often with spontaneous recovery corresponding to the intimal flap dissecting away from the true lumen (see p. 68).
- **Paradoxical emboli** are also uncommon. In patients with a patent foramen ovale, or other septal defect, a clot originating in the veins may pass up towards the chest. In addition to impacting in the pulmonary arterial tree, the clot may pass across the septal defect and lodge in the arterial system. This is particularly likely after a pulmonary embolus, as the resultant raised pulmonary artery pressure results in increased shunting across a septal defect if present.

Treatment
1. **Assessment.** The limb is exposed to room temperature and observed for signs of impairment to the circulation. If the block seems to be resolving, with the appearance of pulses that had previously been absent, the collateral circulation may produce adequate distal
POTENTIAL SOURCES OF EMBOLI

- Left atrium: atrial fibrillation and mitral stenosis, atrial myxoma.
- Heart valves: infective endocarditis.
- Left ventricular wall: mural thrombus after myocardial infarction or from ventricular aneurysm.
- Aorta: from aneurysm or atheroma.
- Interventricular septum: rare paradoxical embolus via a septal defect, originating in the systemic veins.

ACUTE LIMB ISCHAEMIA

- Pain.
- Pallor.
- Pulseless.
- Paraesthesiae.
- Paralysis.
- Perishingly cold.

arterial blood supply and surgery may not be required; thrombolysis may be an appropriate alternative. If the distal limb has apparently no blood supply and there are neurological changes, urgent surgery is indicated. Absent femoral, popliteal or aortic pulsations are indications that operation will probably prove necessary.

The likelihood of surgical removal of an embolus successfully restoring viability to a limb is inversely proportional to the time since the onset of the arterial occlusion; after 24 hours have elapsed successful revascularization of the limb becomes unlikely. Fixed staining of the skin is a sign that it is too late.

2 Heparinization. As soon as the diagnosis is made, the patient should be systemically heparinized, so as to prevent propagation of clot from the site of blockage.

3 Surgical embolectomy. The approach to the involved vessel will depend on physical findings indicating the level of the block. The operative treatment is relatively simple: the vessel is exposed, opened and the clot removed. A special balloon catheter (designed by Thomas Fogarty* when he was a medical student) is passed into the vessel with the balloon collapsed. The balloon is then inflated and pulled back, the clot being expelled by the balloon via the arteriotomy. Poor results will be due to propagation of clot beyond the embolus, particularly down the branches of the popliteal artery, and local thrombolysis may be required. Emboli in the upper limb vessels usually produce less disability than those in the lower limb, as a collateral circulation in the upper limb is better. Surgery is therefore indicated less often.

4 Thrombolysis. Where there is no obvious cause for an embolus, a spontaneous thrombosis in situ must be considered. This is more likely if the patient has a previous history of occlusive symptoms such as claudication. In this case, collaterals have already developed and the limb remains viable. Thrombolysis may restore patency, followed by angioplasty to treat the underlying disease. Occasionally, in situ thrombosis may be a manifestation of malignancy.

It is most important that after the successful outcome of an embolectomy the cause of the embolism be treated if this is possible.

Cold injury

Frostbite may result from prolonged exposure to cold and results from a combination of ice crystal formation in the tissues, capillary sludging and thrombosis within small vessels of the exposed extremities. Treatment comprises gentle warming, anticoagulation with heparin.
to prevent further thrombosis, and antibiotics to inhibit infection of necrotic tissues. Local amputation to remove necrotic digits is performed once clear demarcation develops. Raynaud's phenomenon may be experienced as a late complication.