CHAPTER 11

ARTERIAL DISORDERS

ARTERIAL STENOSIS OR OCCLUSION
ACUTE ARTERIAL OCCLUSION
GANGRENE
AMPUTATION
ARTERIAL DILATATION (ANEURYSM)
ABDOMINAL AORTIC ANEURYSM
ARTERITIS
VASOSPASTIC CONDITIONS
Chronic Arterial Occlusion

**Definition:** Chronic Arterial occlusion is the decrease in arterial blood supply to the tissues due to partial occlusion of arteries ➔ Chronic Ischemia

**Cause:** common causes are
1. Atherosclerosis
2. Burger’s disease
3. Diabetic vascular disease
4. Raynaud’s disease

**Symptoms & signs of lower limb arterial occlusion:**

**Claudication pain:**
- It is a intermittent cramp-like pain felt in the muscles that:
  * Brought on by walking
  * Not present on taking the first step (contrast osteoarthrosis)
  * Relieved by standing still (contrast lumbar intervertebral disc nerve compression).
- Claudication pain is most commonly felt in calf, but can affect thigh or buttock.

  *N.B.: Distance walked is called claudication distance*

**Rest pain:** It is severe pain felt in the foot at rest, made worse by lying down, or elevation of the foot, the pain is worse at night, and relieved by hanging the foot out of bed, or by sleeping in a chair.

**Coldness, numbness & paraesthesia:** are common in moderate & severe ischaemia but, in the absence of colour changes, it is essential to exclude neurological cause.

**Colour changes:** Severely ischaemic limbs develop purple discoloration on dependency. Bright red colour is due to extravasation of RBCs through capillary walls.

**Ulceration:** occurs with severe arterial insufficiency and often presents as painful, superficial erosion between toes.

**Gangrene:** has blackened mummified skin and tissues.

**Sensation & movement:** Severe chronic ischaemia produce hyperaesthesia, especially on the borderline skin of gangrene.

**Arterial pulsations:** below occlusion are usually absent or, diminished in the presence of good collaterals. Expansile arterial pulsation with a mass may indicate an aneurysm.

**Other physical findings:** include loss of hair, nail changes, pallor on elevation, and dependent rubor.
Symptoms related to the organ supplied by the artery:

* Lower limb ➔ claudication, rest pain and gangrene
* Brain ➔ transient ischaemic attacks and hemiplegia
* Myocardium ➔ angina and myocardial infarction
* Kidney ➔ hypertension or infarction
* Intestine ➔ abdominal pain and infarction.

**Symptoms & signs of Aorto-Iliac occlusive disease:**
- Asymptomatic or may present with buttock or thigh claudication.
- LeRiche syndrome.
- Severe ischemia ➔ gangrene or limb loss, is rare.

**Symptoms & signs of Mesenteric vascular disease:**
- Abdominal pain following a meal; leading to “food fear”
- Weight loss secondary to avoiding food due to pain
- An epigastric bruit

**Symptoms & signs of Renal artery stenosis disease:**
- Sudden onset of severe hypertension in patients of 35-55 years of age
- Sudden worsening of hypertension in a well controlled patient
- Inability to control blood pressure despite multiple-drug therapy
- Presence of flank bruits associated with any of the preceding characteristics

**Symptoms & signs of Cerebrovascular disease:** (Carotid stenosis)
- Transient ischemic attacks (TIAs) are recurrent and short-lived mini-strokes.
  Resolution within 2-15 or occurs within 24 hours, but they are a warning of impending major stroke.
- Amaurosis fugax is transient monocular blindness caused by an embolus to a retinal vessel. Fundus examination show a gray fibrin plaque or a bright-yellow (Hollenhorst) cholesterol plaque in a retinal artery.

**Physical signs in case of arterial occlusion with normal distal pulses & highly developed collateral circulation:**

1. **Disappearing pulse:** on exercising till claudication ➔ palpable pulse disappear. Then after 1-2 minutes of rest the disappearing pulse reappears.
2. **Arterial bruits:** auscultation of subclavian arteries ➔ Systolic bruits. Bruit in neck at the level of mandibular angle without supraclavicular bruit means carotid artery stenosis. The patient with renal artery stenosis has a bruit over the renal artery. Continuous ‘machinery’ murmur over an artery ➔ A-V fistula.
3. Increased venous return & varicosities of veins are associated with A-V fistulas.
Diagnosis & Investigations:

**Routine:** Hb, full blood count, ESR, plasma fibrinogen, protein electrophoresis, blood & urine glucose, blood lipid profile and Exercise ECG.

**Noninvasive tests:**
- *Doppler ultrasound:* use a stethoscope with sphygmomanometer to assess the systolic blood pressure in relatively small vessels
  - *Pulse volume recording (PVR)* measures pulse volume changes in the extremity during the cardiac cycle. Using waveform analysis, the presence and the location of occlusive lesions can be ascertained.
  - *Ankle : brachial index (ABI)* is the ratio of systolic pressure at ankle artery to that in brachial artery. Numerator ➔ systolic pressure in dorsalis pedis & posterior tibial arteries, denominator ➔ systolic pressure of brachial arteries.
    - Normal ABI ➔ 1 or slightly greater ➔ ABI < 0.3 ➔ imminent necrosis.
    - ABI < 0.8 ➔ arterial obstruction.
    - ABI < 0.5 ➔ rest pain.
- *Duplex imaging:* provide an image of vessels, can give detailed knowledge of vessel blood flow & turbulence. Colour-Duplex ➔ visualisation of blood flow, indicates change in direction & velocity of blood flow (↑flow ➔ stenosis).
- *Treadmill:* is a useful in the assessment of walking distance in claudicants.

**Invasive tests**
- *Intravenous digital subtraction angiography (IVDSA)* performed by injecting contrast material into a large vein. The arterial system can be visualized by proper timing of the X-ray exposures.
- *Arterial digital subtraction angiography (DSA)* uses less contrast material, it is computerised angiography ➔ provides image before contrast injection to be subtracted from the contrast image, yielding great clarity.
- *Conventional arteriography:* Contrast material is injected into the artery of interest, usually by way of a femoral artery puncture, and sequential X-ray exposures are made. This procedure is associated with several serious complications (acute renal failure, acute arterial occlusion, Pseudo-aneurysms).
- *Brachial artery or Axillary artery catheterization:* One study is performed frequently for cardiac catheterization or for angiography when the lower extremity vessels (femoral vessels) are not adequate for angiography.
- *Abdominal plain X-ray:* show the presence of arterial calcification and flecks of calcium may outline an aneurysm.
Plethysmography assesses changes in limb or digit volume over cardiac cycle.

**Management of arterial occlusion**

*Conservative treatment:*
- Stop smoking, particularly patients with Buerger’s disease.
- Taking regular exercise, within the limits of the pain.
- Suitable Diet to reduce weight in the obese & hyperlipidaemics.
- Heel raise: Claudication distance may be increased by raising the heels of shoes by 1 cm. The work of the calf muscles is reduced thereby.
- Drugs: Rest pain can be relieved by analgesics and elevation of the head of bed (Buerger’s position), Pentoxifylline for treatment of intermittent claudication, Aspirin in small dose of 150 mg/day to prevent thrombosis.
- Sympathectomy can occasionally relieve ischaemic rest pain and ulceration.

*Nonoperative procedures to improve arterial flow*
- Transluminal angioplasty: inserting a balloon catheter into an artery and inflating it within a narrowed area.
- Percutaneous transluminal angioplasty (PTA) inflatable balloon may be used for stenosis or short occlusions.
- Intraluminal stents: after balloon dilatation, the vessel may fail to stay dilated then it is possible to keep open lumen by a metal stent.
- Lasers & Atherectomy catheters Lasers can be used to open occluded arteries so that a balloon angioplasty catheter can be inserted. Atherectomy catheters actually remove atherosclerotic plaque from arterial wall, either by cutting or extracting.

*Operations for arterial occlusion*
- Aortoiliac occlusion: good calibre vessels below the site of disease responds well to Aorto-femoral bypass. If not, an iliac endarterectomy might be considered, but PTA with or without a stent is probably a better alternative.
- Superficial femoral & profunda femoris artery occlusion (with unilateral symptoms): For severe disease angioplasty or bypass may be used. A femoropopliteal bypass graft is the most usual operation (to overcome a blocked superficial femoral artery). Patient’s own saphenous vein is the best graft.
- Occlusive disease below the popliteal artery (usually unreconstructable) Bypass to tibial vessels (down ankle level) can be successful. Long saphenous vein used in the in Situ fashion after disrupting the valves with a valvulotome. If the saphenous vein is not available a polytetrafluoroethylene (PTFE) graft is used.
Patient unable to withstand major abdominal surgery + ischaemia due to aortoiliac occlusion, following is done: (extra-anatomic bypass)
- Femoro-femoral crossover graft is useful for relieving an iliac artery occlusion if only one iliac system is involved with disease.
- Axillo-femoral graft is useful for a pre gangrenous limb in a poor-risk patient with bilateral iliac obstruction (i.e. both iliac segments are diseased). Axillary artery carries sufficient volume of blood to maintain the circulation in the arm and revascularise the lower limb.

Treatment of Other sites of atherosclerotic obstruction

- Carotid stenosis: carotid endarterectomy: an arteriotomy in the common carotid artery continued up into the internal carotid artery through the diseased segment, removal of the occlusive disease (endarterectomy) and closure of the arteriotomy usually with a patch (vein or PIPE).
- Subclavian artery stenosis: May cause claudication and rarely ischaemia of arm. Artery to artery embolisation occurs loss of digits. Treatment by endarterectomy or bypass but PTA balloon is the treatment of choice.
- Mesenteric artery occlusion: Treatment: surgical endarterectomy or bypass.
- Renal artery stenosis Treatment: control hypertension by drugs. PTA and endarterectomy, aortorenal bypass, renal artery revascularisation using another vessel as splenic artery, to renal auto-transplantation.

Prosthetic materials: (Types of Dacron woven or knitted)
- For aortoiliac bypass segment the favoured material is Dacron.
- For femoropopliteal bypass region, if long or short saphenous or arm vein is not available, PTFE or glutaraldehyde-tartned, Dacron-supported or human umbilical vein may be employed.
- For profundaplasty, a small piece of vein may be used or PTFE or Dacron.

N.B.: Suture materials for vascular surgery are usually mono-filament in nature; polypropylene has been particularly popular.

Atherosclerosis

Definition: is a disease process that involves both large and small arteries. Arterial lesions tend to occur at proximal internal carotid artery, the infrarenal aorta, and superficial femoral artery. The supraceliac aorta and the distal deep femoral artery are rarely involved. The reason for this pattern is not known.

Pathology: arterial lesions can be divided into three general types.
1. Fatty streaks are discrete, subintimal lesions, which are composed of
cholesterol-laden macrophages and smooth muscle cells. These streaks may occur early in life and are not hemodynamically significant.

2. **Fibrous plaques** are more advanced lesions, which also contain an extracellular matrix. These plaques may progress to cause an obstruction to flow.

3. **Complex plaques** are characterized by intimal ulceration or intraplaque hemorrhage. These plaques may cause local occlusion of the vessel or may result in embolization of clot or cholesterol, causing distal arterial occlusion.

### Thromboangitis obliterans (Buerger’s disease)

**Definition:** N.B. 1 or 2 are present
- It is occlusive disease of small & medium size arteries (plantars, tibials, radial,...)
- Thrombophlebitis of superficial or deep veins
- Raynaud’s phenomenon occurring in male patients

**Etiology:** It occurs in young men, aged 20-40 years. The cause is unknown, but it may be heavy smoking or fungal infection of the feet.

**Pathology:** Localised inflammatory changes occur in walls of arteries and veins leading to thrombosis. It usually affects distal arteries below the mid-calf level.

**Clinical picture:** The usual symptoms and signs of arterial occlusive disease will be present. Claudication & Gangrene of the toes and fingers are common.

**Investigation:** Arteriography: shows a characteristic corrugation of femoral arteries as well as distal arterial occlusions and helps to distinguish the condition from presenile atherosclerosis. ESR and autoantibodies

**Treatment:** as usual + the following:
- Total prevention of smoking will arrest the disease but don’t reverse established arterial occlusions.
- Direct arterial surgery is not usually applicable and sympathectomy is the most useful procedure. It results in healing of ischaemic ulcers and improvement in skin nutrition, with relief of pain. Amputations may be required.

### Diabetic Vascular disease

**Infection and gangrene** are common problems among diabetic patients. Diabetics tend to have more diffuse, more distal (infrapopliteal) disease.

**Clinical picture:**
- Peripheral neuropathy ↓ sensation & proprioception  ➔ No recognition of minor trauma. Painless ulcers can form over the weight-bearing areas of foot.
- Infection due to impaired immunity (↓ leukocyte chemotaxis, ↓ adherence to vascular endothelium, ↓ phagocytosis, ↓ intracellular killing activity).
(1) Cellulitis of the dorsum of the foot
(2) A deep plantar space infection
(3) Osteomyelitis of the metatarsal or phalangeal bones

**Clinical types:**
1. Micro-angiopathy: patient presents with swollen inflammed foot, lymphangitis, early gangrene, all pulses are felt.

**Diagnosis:**
- ABI is artificially elevated due to calcified vessels in diabetic. Calcification frequently spares digital vessels.
- Toe waveforms and pressures are frequently useful in evaluating wound healing potential.

**Treatment:**
- Broad-spectrum antibiotics for treatment.
- If necrotic tissue is present or a plantar abscess is suspected, urgent operative debridement is necessary.
- An adequate arterial blood supply to the affected foot and toes must be assured for healing to occur.

**Raynaud’s Syndrome**

- **Primary Raynaud’s disease**
  It is idiopathic, episodic vasoconstriction, affects commonly the fingers but occasionally the feet. It is usually initiated by cold exposure or emotional stimuli and occurs mainly in young women.

**Clinical picture:**
- Color changes:
  - Pallor due to severe vasospasm in the dermal vessels
  - Cyanosis due to sluggish blood flow and resultant marked blood desaturation
  - Rubor due to the reactive hyperemia
    - numb discomfort localized in the fingers, ulceration or gangrene.

_N.B.: Associated local or systemic disease. Although scleroderma is most commonly associated with Raynaud’s phenomenon, it may develop in anyone with a collagen vascular disease._

**Management:**
- Cold should be avoided by gloves or hand warmers in extremely cold weather
- Tobacco should be avoided because it stimulates vasoconstriction.
- Calcium channel blockers, such as nifedipine, are the drugs of choice. Use of phenoxybenzamine for alpha-blockade may be therapeutic.
- Sympathectomy is rarely recommended in these patients because they have vascular occlusion of the digital vessels.

**Secondary Raynaud’s phenomenon**

**Secondary causes:** Peripheral vasospasm occurs secondarily to:
- Atherosclerosis
- Scleroderma
- Systemic sclerosis
- SLE
- Cervical rib
  - Follows use of industrial tools, e.g. pneumatic road drills and chain saws, which vibrate at certain frequencies.

**Tests:** Full blood count, ESR, urea and electrolytes, antinuclear factor, rheumatoid factor, cryoproteins, immunoglobulins; cold agglutinins, chest X-ray; skin biopsy.

**Treatment:** It is directed to the cause. Sympathectomy may occasionally be required but the results are not impressive. *Drugs.* a-Blockers, ergotamine-based preparations and other vasospastic antagonists can also be tried.

*N.B.: Cold hypersensitivity may occur following frostbite. The affected area is bluish with a burning pain. Medical management and occasionally sympathectomy control the symptoms.*
N.B.

* **Subclavian steal syndrome:** If 1st part of the subclavian artery is obstructed, the vertebral artery may provide a collateral circulation into the arm by reversing its direction of flow → periods of cerebral ischaemia. Treatment: PTA, endarterectomy or bypass from ipsilateral common carotid to the 3rd part of subclavian.

* **Pregangrene:** refers to the combination of rest pain, colour changes, oedema, and hyperaesthesia, with or without ischaemic ulceration.

* **Ischaemic sites**
  1. Behind angle of the mandible → supraclavicular fossa & carotid arteries
  2. Groin & over adductor canal → abdominal aorta & femoral arteries.

* A systolic bruit over an artery is due to turbulence, and indicates a stenosis of the artery.

* ‘Leriche syndrome’
  - Claudication of the thighs and buttocks
  - Diminished femoral pulses
  - Impotence
  - Atrophy of the leg muscles

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**Acute Arterial Occlusion**

**Definition:** Sudden occlusion of an artery is commonly due to either emboli or trauma.

**Clinical picture:** six *P’s:*

1. **Pain**
2. **Pallor**
3. **Pulselessness**
4. **Paresthesia**
5. **Paralysis**
6. **Progressive coldness**

In thrombosis the limb has developed adequate collateral pathways around the occlusion to interrupt the sequence leading to tissue death, and the patient is seen when symptoms of claudication from chronic ischemia lead to the evaluation.

**Factors Suggesting Embolism versus Thrombosis**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Embolism</th>
<th>Thrombosis</th>
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</thead>
<tbody>
<tr>
<td>- Identifiable source or risk factor</td>
<td>- Usual, particularly atrial fibrillation or prior</td>
<td>- Unusual</td>
</tr>
</tbody>
</table>
- History of claudication
- Physical findings suggestive of occlusive disease
- Arteriography

<table>
<thead>
<tr>
<th>history of embolism</th>
<th>- Rare</th>
<th>- Common</th>
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<td>- Few; proximal and contralateral limb pulses normal</td>
<td>- Minimal atherosclerosis; sharp cutoff; few collaterals</td>
<td></td>
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</tbody>
</table>

- History of embolism
- Rare
- Few; proximal and contralateral limb pulses normal
- Minimal atherosclerosis; sharp cutoff; few collaterals
- Common
- Often present; proximal or contralateral limb pulses diminished or absent
- Diffuse atherosclerosis; tapered, irregular cutoff; well-developed collaterals

**Embolism**

Embolus is a foreign body to the bloodstream and may become lodged in a vessel and causes obstruction, producing the classic symptom of acute ischemia, because it frequently lodges in a vessel previously carrying a relatively normal flow; thus no collateral pathways had been established previously.

*Aetiology:* Simple emboli are due to blood clot. The sources of blood clot are most commonly mural thrombus following a myocardial infarct (1/3 cases), mitral stenosis, cardiac arrhythmias, particularly atrial fibrillation, and aneurysms.

*Types of emboli:*
1. Generated within the vascular system, e.g. fragments of thrombus, material from atheromatous plaque.
2. Matter entering the vessels:
   (One) Solid ➔ tumour cells, bacterial clumps, parasites, foreign bodies
   (Two) Gaseous ➔ air
   (Three) Liquid ➔ amniotic fluid, fat

*Sites of Embolic lodgement & symptoms:*
- **Brain** ➔ the middle cerebral artery is most commonly affected, resulting in permanent or temporary hemiplegia (transient ischaemic attacks).
- **Retina** ➔ Complete obstruction of central retinal artery causes total and permanent blindness.
- **Lungs** ➔ pulmonary embolism is a fatally interrupt after operation.
- **Mesenteric vessels** ➔ causing engorgement and may be gangrene of the corresponding loop of intestine.
- **Spleen** ➔ commonly affected with local pain and enlargement.
- **Kidneys** ➔ resulting in loin pain and haematuria.
- **Lower limb** ➔ pain, pallor, paresis, pulselessness and paraesthesia.

The limb is cold and almost immediately the toes cannot be moved (contrast with
venous occlusion when muscle function is not affected). The patient, who has no previous symptoms of claudication or limb pain, and has a source of emboli, suddenly develops severe pain or numbness of the limb, which becomes cold with mottled blue and white discoloration. Movement of the toes becomes progressively more difficult and sensation to touch is lost. Pulses are absent distally, but the femoral pulse may be palpable if the clot is lodged in a low bifurcation of the femoral artery. This is because distal occlusion results in forceful expansion of the artery with each pressure wave, despite the lack of flow.

**Difference between Acute Arterial Occlusion due to Embolus & Atherosclerosis**

<table>
<thead>
<tr>
<th>Embolic arterial occlusion</th>
<th>Atherosclerotic narrowing + thrombosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>- No preceding history of claudication</td>
<td>- There is a preceding history of claudication</td>
</tr>
<tr>
<td>- A source for emboli can usually be found, e.g. mitral stenosis, cardiac arrhythmias, aortic aneurysm, recent myocardial infarct, artificial valve</td>
<td>- A source for emboli cannot be found</td>
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<tr>
<td>- Loss of function occurs within 4-6 hours after the onset of pain, e.g. patient unable to move toes</td>
<td>- Loss of function not present (because collaterals have had time to be established)</td>
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</table>

* In acute ischaemia, a dead, white limb which becomes mottled means impending gangrene and not an improvement in the circulation

* Acutely ischaemic limbs are frequently paralytic and without sensation.

**Treatment:**

*Administration of heparin* IV 5000 units, then continuous infusion can reduce proximal and distal extension of thrombus until the embolus can be treated.  

*Embolectomy (thrombectomy)*: Through a longitudinal or transverse incision the clot is removed, together with the embolus.  

*Intra-arterial thrombolysis*: to treat either embolus or thrombosis if ischaemia is not so severe. Thrombolytic agents are streptokinase, urokinase & tissue plasminogen activator.  

*Fogarty catheterisation*: effective in removing proximal & distal extensions of thrombus. The Fogarty catheter is like a ureteric catheter, with a balloon tip, and is introduced until it passes the limit of the thrombus. The balloon is inflated and the catheter withdrawn slowly, together with the clot. The procedure is repeated until bleeding occurs.  

*N.B.: Prevention of further emboli is achieved by treatment of the cause, and using*
long-term anticoagulation (warfarin) to reduce chance of thrombus re-formation.

Mesenteric artery occlusion
Acute mesenteric occlusion can be either thrombotic (atherosclerotic) or embolic. Thrombotic occlusion follows progressive narrowing, and so the symptoms are progressive with weight loss, abdominal pain and leukocytosis. Severe abdominal pain → diarrhoea, systemic hypovolaemia and haemoconcentration. Treatment is arteriography followed by PTA or surgical bypass if the bowel has not already infarcted.

Embolic occlusion results in sudden, severe abdominal pain, with bowel emptying (vomiting and diarrhoea), and a source of emboli present (usually cardiac). Treatment is Arteriography and embolectomy or bypass surgery.

Other Types of embolism
Air embolism:
Etiology:
1. Accidentally injected air e.g. sucked into open vein.
2. Complicates operations on the neck or axilla, open heart surgery (artificial pneumothorax), fallopian tube insufflation, and following illegal abortion.
3. Following cut throat.
Pathogenesis: When air enters Rt atrium it is churned up, and the foam enters Rt ventricle and causes an air-lock in the pulmonary artery → Rt-sided heart failure. Treatment: Trendelenburgs position encourages air to pass into the veins of the lower body half, and the patient is placed on the left side so that air will float into the apex of the ventricle, away from the pulmonary artery. Oxygen is administered to counteract hypoxaemia, and to assist in the excretion of nitrogen. In serious cases, the right ventricle should be aspirated by a needle passed upwards and backwards from below the left costal margin. If this fails, the heart is rapidly exposed for aspiration under direct vision.

Fat embolism:
Etiology:
1. Follows severe injuries with multiple or major fractures.
2. Follows convulsive therapy.
The fat may be derived from bone marrow or adipose tissue, but recent work suggests that it is metabolic in origin, perhaps by aggregation of chylomicrons.
Symptoms according to type:
1. Cerebral type: patient is drowsy, restless, and disorientated. Subsequently, the patient is comatose, the pupils become small and pyrexia ensues.
2. Pulmonary type: is cyanosed + signs of Rt heart failure. White froth at mouth and nostrils (Mistaken for bronchopneumonia or Lt ventricular
failure).

**Signs:**
- Emboli in the retinal arteries ➔ striate haemorrhages & ‘fluffy’ patches of exudate. Fat droplets in sputum
- Fat may be excreted in the urine.
- Fall in Hb value of blood.
- Petechial haemorrhages often occur.

*Treatment:* oxygen, early heparin, and IV low-molecular-weight dextran.

Other forms of emboli include *infective emboli* of bacteria or infected clot, which may cause mycotic aneurysms (see below), pyaemia, or infected infarcts, *parasitic emboli* due to the ova of *Taenta echinococcus* and *Filaria sanguinis hominis*, and emboli of *malignant cells* (e.g. hypemphroma).

**Therapeutic embolisation:**

*Uses:*
1. Arrest haemorrhage from GIT, urinary and respiratory tracts
2. Treat A-V fistula by blocking their arterial supply
3. Control the growth of unresectable tumours

*N.B.: Arterial embolisation requires accurate selective catheterisation using the Seldinger technique.*

*Examples:*
- Occlusion of left gastric or gastroduodenal to treat a bleeding ulcer.
- Occlusion of the hepatic artery relieves the pain of 1ry & 2ry liver tumours and Control endocrine effects of hormone-secreting tumours e.g. metastatic carcinoid.
- Renal artery embolisation to devascularise renal tumour prior to surgery and to arrest persistent haemorrhage from an unresectable tumour.
- Oesophageal varices: by entering portal system of liver, and embolising the veins supplying the varices.

A wide range of materials have been used and they include blood clot, gel foam sponge, human dura, plastic microspheres, balloons, ethyl alcohol, quick-setting plastics and mechanical devices made of stainless steel coils and wool.

**Caisson and decompression disease:**

These similar conditions may affect divers, those who work in compressed air chambers, or who ascend in open aeroplanes to above 7620 m. If decompression is too rapid, bubbles of nitrogen are set free in the tissues & bloodstream, and occlude small vessels.

*Symptoms:*
- Pain in muscles or joints
Neurological disturbances
- Weakness of legs and sphincters if the spinal cord is affected.
- Chest tightness and dry cough (the ‘chokes’) if lungs affected.

_Treatment:_ Caisson disease requires recompression and thereafter gradual decompression. The high-altitude flyer is relieved by gradual descent, Inhalation of oxygen assists the excretion of nitrogen. If the spinal cord is not permanently damaged the prognosis is good, but hypertrophic changes may persist in the ends of long bones.

- **Acute arterial occlusion due to trauma**

  Arteries (like all tubes) can be occluded as a result of changes:

  1. In the lumen, e.g. thrombosis;
  2. In the wall, e.g. subintimal haematoma;
  3. In the surrounding tissues, e.g. anterior tibial compartment syndrome.

  Absent pulses with rest pain or skin colour and temperature change suggest arterial occlusion. In this group, the commonest event is femoral or brachial artery damage at cardiac catheterisation; the latter vessel is much more prone to iatrogenic complications and should be avoided if possible by those using catheter techniques.

  Preoperative assessment including arteriography is valuable. It is also useful to recognise pre-existing arteriosclerotic disease.

  Operative procedure. On exposing a damaged artery, an obvious laceration may be found — remember to look for a puncture wound in the back of an artery in a stabbing. If this’ is very small, e.g. from a needle puncture, a single Suture may suffice to repair a leak. If damage is more widespread and if thrombosis forms part of the picture, it may be necessary to resect a damaged segment. If the resection is very limited, it may be possible to reconstitute the vessel by direct anastomosis of the cut ends. In general, however, a short interposition graft (vein) is to be preferred. The vein should not be taken from the damaged limb because concomitant deep venous trauma may be present and superficial veins may be required to return blood centrally.

  It should first be appreciated that occlusion due to trauma should never be casually ascribed to arterial spasm: ischaemia after trauma demands urgent action. Second, the results of operating on traumatiaed arteries are, in general, good. The outflow is not compromised by atheromatous disease in most cases. It the return of blood supply to the limb after arterial declamping is not very obvious, then peroperative angiograph~’ is mandatory. Not only may there be a problem with the local arterial reconstruction but also thrombosis (from Stasis) or embolism at a distal site may be present and may require separate attention.
Fractures of bone occur frequently alongside arterial injuries and require stabilisation, both in their own right and to protect the vascular repair. Stabilisation may be carried out before or after the vascular reconstruction (depending on how acute is the ischaemia) and fixation (often external) is greatly to be preferred to traction, for obvious reasons.

Compartment syndrome. It must also be remembered that in the lower limb the additional problem of compression of the main artery due to haematoma or oedema in the fixed fascial compartments of the calf, especially the anterior tibial compartment, can cause distal ischaemia (and crush syndrome renal effects). The treatment is urgent fasciotomy releasing the external compression on the artery.

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**Gangrene**

**Definition:** Gangrene is death with putrefaction of macroscopic portions of tissue.

**Sites:** common in distal part of a limb, appendix, a loop of small intestine, and sometimes organs such as gallbladder, pancreas, or testis.

*N.B.: Necrosis means death of groups of cells. A slough is a piece of dead soft tissue, e.g. skin, fascia or tendon.*

**Etiology:**
1. *Secondary to arterial obstruction:* Thrombosis, embolism (from heart in AF), arteritis, Buerger’s disease, Raynaud’s disease, ergotism, intra-arterial injections (thiopentone & cytotoxic substances).
2. *Infective:* boils & carbuncles, gas gangrene, scrotum (Fournier’s) gangrene.
5. *Neuropathic:* syringomyelia, leprosy.
6. *Venous gangrene* due to extensive thrombosis in peripheral veins.
Clinical features:
Gangrenous part lacks arterial pulsation, venous return, capillary response to pressure (colour return), sensation, warmth and function.

<table>
<thead>
<tr>
<th>Types</th>
<th>Dry gangrene</th>
<th>Moist gangrene</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site</td>
<td>Occurs in parts poor in tissue fluid and exposed to dryness by evaporation.</td>
<td>Occurs in parts rich in tissue fluid and not exposed to dryness by evaporation.</td>
</tr>
<tr>
<td>Cause</td>
<td>Gradual slowing of blood-stream, as in atherosclerosis. Minimal infection (aseptic ulcerative separation).</td>
<td>Sudden venous, arterial occlusion by ligature or embolus, and DM. Infection and putrefaction (septic ulcerative separation) are present.</td>
</tr>
<tr>
<td>Infection</td>
<td>- Affected part is dry, wrinkled, dark in colour (Hb disintegration), and greasy to the touch.</td>
<td>- Affected part is swollen, dark in colour, edematous, offensive odor</td>
</tr>
<tr>
<td>Pathology</td>
<td>- There is line of demarcation separate viable &amp; dead tissue.</td>
<td>- Skin: tense, ulcerated, may show raised bleb (filled with gas bubbles) - No line of demarcation.</td>
</tr>
</tbody>
</table>

Separation of gangrene
Separation by demarcation: A zone of demarcation, between viable & dead tissue, appears first. It is indicated on the surface by a band of hyperaemia & hyperaesthesia. Separation is achieved by development of a layer of granulation tissue forms between dead & living parts. These granulations extend into the dead tissue, until those which have penetrated farthest are unable to derive adequate nourishment. Ulceration follows, and thus a final line of demarcation (separation) forms which separates the gangrenous mass from healthy tissue.

Treatment of gangrene
General principles. A limb-saving attitude is needed in most cases of symptomatic gangrene affecting hands and feet. The surgeon is concerned with how much can be preserved or salvaged. With arterial disease all depends upon there being a good blood supply to the limb above the gangrene, or whether a poor blood supply can be improved by such measures as percutaneous transluminal angioplasty or direct arterial surgery. A good or improved blood supply indicates that a
conservative excision is likely to be successful and a major amputation may be avoided. A *life-saving amputation* is required for a badly crushed limb, rapidly spreading symptomatic gangrene, and gas gangrene (see section on amputation).

General treatment includes that of cardiac failure, atrial fibrillation and anaemia, to improve the tissue oxygenation. A nutritious diet, essential in all forms of gangrene, and the control of diabetes when present, are additional items of care. Pain, especially night pain, may be difficult to relieve. Nonaddictive drugs should be used whenever possible.

Local treatment. Care of the affected part includes keeping it absolutely dry. Exposure, and the use of a fan may assist in the desiccation and may relieve pain. *The limb must not be heated.*

Protection of local pressure areas, e.g. the skin of the heel or the malleoli, otherwise fresh patches of gangrene are likely to occur in these places. A bed-cradle, padded rings, foam PVC blocks, a ‘sheepskin’ and air beds are useful preventative aids.

*Minor surgical toilet.* Careful observation of a gangrenous part will show whether the lifting of a crust, or the removal of hard or desiccated skin, will assist in demarcation, the release of pus and the relief of pain.

**Varieties of gangrene**

*Diabetic gangrene* is due to three factors:

1. Trophic changes resulting from peripheral neuritis
2. Atheroma of the arteries resulting in ischaemia
3. Excess of sugar in the tissues which lowers their resistance to infection.

The neuropathic factor impairs sensation, and thus favours the neglect of minor injuries and infections, so that inflammation and damage to tissues are ignored. Muscular involvement is frequently accompanied by loss of reflexes, and deformities. In some cases, the feet are splayed and deformed (neuropathic joints). Thick callosities develop on the sole, and are the means whereby infection gains entry, often following amateur chiropody. Infection involving fascia, tendon, and bone can spread rapidly upwards via subfascial planes.

*Clinical examinations and investigations* include those on the urine and blood for diabetes. Palpable dorsalis pedis and posterior tibial pulses, and the absence of rest pain and intermittent claudication, imply that there is no associated major arterial disease (atherosclerosis). A bacteriological examination is made of any pus. A radiograph will reveal the extent of any osteomyelitis.

*Treatment.* The diabetes must be brought under control by diet and appropriate drugs. The gangrene is treated on the lines already described, the accent being on conservatism if there is no major arterial obstruction. A rapid spread of infection
requires drainage of the area by incision and the removal of any obviously dead tissue. This may often involve free and extensive laying open of infected tissue planes. Adequate surgical drainage of pus and the control of infection due to bacteria and fungi may then be followed by rapid healing. After healing, protection of the affected parts is essential to promote further healing.

Direct traumatic gangrene is due to local injury, and may arise as a result of crushes, pressure (as in the case of splints or plasters), or bedsores. Gangrene following severe injury, e.g. a street accident in which a heavy vehicle passes over a limb, is of the moist variety and excision without delay is usually indicated. Amputation may be performed as close to the damaged part as will leave the most useful limb (see Amputation below).

Bedsores (decubitus ulcers) are predisposed to by five factors — pressure, injury, anaemia, malnutrition and moisture. They can appear and extend with alarming rapidity in patients with disease or injury of the spinal cord and other patients with debilitating illness. It is important to recognise patients at risk and take adequate prophylactic measures. These measures include the avoidance of pressure over the bony prominences, regular turning of patients and nursing on specially designed beds, which reduces the pressure to the skin. These beds include the high air loss Clintron bed, low air loss Mediscus bed, and the very low air loss OSA 1000. There are advantages in not blowing large quantities of air around the ward, and also advantages in being able to articulate the patient, yet removing the increased pressure and sheer forces produced by such articulation (Chapter 26). Preventive measures are of the utmost importance. Thus pressure over bony prominences is counteracted by a 2-hourly change of posture and protection by foam PVC blocks, or a ‘sheepskin’ (see footnote, earlier). A water bed or a ripple bed is sometimes desirable. Injury due to wrinkled draw-sheets and maceration of the skin by sweat, urine, or pus is combated by skilled nursing and the use of an adhesive film such as Opsite (Fig. 11.34).

A bedsore is to be expected if erythema appears which does not change colour on pressure. The part must be kept dry. An aerosol silicone spray may be used. Actual bedsores may either be treated by lotions or by exposure to keep them as dry as possible. Once pressure sores develop, they are extremely difficult to heal. They should be kept clean, débrided and the use of rotation flaps should also be considered. The haemoglobin of the patient should be maintained at a normal level by transfusions of packed cells if need be. If the patient is young and otherwise healthy, excision of the dead tissue and flap pedicle skin grafting is often successful.

Indirect traumatic gangrene is due to interference with blood vessels:
1. From pressure by a fractured bone in a limb, or by strangulation
2. Thrombosis of a large artery, following injury
3. Ligation of the main artery of a limb, as after division by injury
4. Poor technique for local anaesthesia e.g. tourniquet + adrenaline-containing local anaesthetic solution ➔ permanent occlusion of all the arteries.

_Treatment_ directed to the cause:
Fracture ➔ closed or open reduction + direct arterial surgery for damaged vessel + limb cooling ➔ will usually prevent the onset of gangrene.
If moist gangrene occurs and spreads rapidly, amputation may be needed to save the patient’s life.

Ergot, a cause of gangrene among dwellers on the shores of the Mediterranean Sea and the Russian steppes who eat rye bread infected with _C. ziceps purpurea_, also occurs in migraine sufferers, who, for prophylactic reasons, unwittingly take ergot preparations over a long period. The fingers, the nose arid ears may be affected.

**Physical and chemical causes of gangrene**
Frostbite is due to exposure to cold, especially if accompanied by wind or high altitudes (e.g. climbers and explorers). It is also encountered in the elderly or the vagrant during cold spells (Fig. 11.37). Pathologically, there is damage to the vessel walls, which is folky.sed by transtidation and oederna. The sufferer notices severe burning pain in the affected part, after which it assumes a waxy appearance and is painless. Blistering, and then gangrene, follows.

Fig. 11.36 Gangrene due to ergot. The patient had taken repeated doses of ergotamine tartrate for ‘migraine’ while on a transatlantic flight.
Fig. 11.37 Frostbite In Fulham, London, England. An elderly lady, living with insufficient food or heating. Conservative amputations were successful.

_Treatment_. Frostbitten parts must be warmed very gradually. Any temperature higher than that of the body will be detrimental. Many frostbitten limbs have been either stewed or wasted in ignorance, and gangrene thereby encouraged. _The_ part should be wrapped in cottonwool and kept at rest. Friction, e.g. rubbing with snow, may damage the already devitalised tissues. Warm drinks and clothing are provided and powerful analgesics are required to relieve the pain which heralds the return of circulation. Paravertebral injection of the sympathetic chain may be helpful in relieving associated vasospasmt. Amputations should be conservative. Hyperbaric oxygen (Chapter 4) is effective.

Trench foot is due to cold, damp, and muscular inactivity, and is predisposed to by tight clothing, such as garters, puttees, or ill-fitting boots. Prophylaxis is therefore of paramount importance. Numbness is followed by pain, which is
excruciating when boots are removed. The skin is mottled like marble, and in severe cases blisters containing blood-stained serum develop, and moist gangrene follows. The pathology is similar to that of frostbite, and the treatment is the same. Inadvertent intra-arterial injection of thiopentone can happen when a high division of the brachial artery results in one of its two terminal branches, usually the ulnar, passing superficially downwards in the antecubital fossa. The appreciation by palpation of pulsation of the vessel, and of the withdrawal of bright red blood prior to injection should prevent this calamity.

Injection causes immediate and severe burning pain and blanching of the hand. The needle should be left in position, and 5 ml of 1 per cent procaine and/or 2 per cent papaverine sulphate injected to obviate vascular spasm. Dilute heparin solution may also be given if the needle is in position. Intra-arterial thrombolysis intravenous low-molecular-weight dextran may be employed. Brachial block must also be performed, and repeated if necessary. Even so, gangrene of one or more fingers may occur.

Drug abuse, inadvertent arterial injection of drugs is becoming common in many countries with significant numbers of drug addicts. Usually the femoral artery in the groin is involved and presentation is with pain and mottling distally in the leg. Often all pulses down to ankle level are retained. If pulses have been lost, angiography and inka-arterial thrombolysis may be considered (possibly with dextran and heparin in addition). If pulses are retained, dextran and heparin may be given but there is no firm evidence of their efficacy in this condition. Many cases are self-limiting and resolve spontaneously. It should be remembered that many of these patients carry the human immunodeficiency virus (HIV) or have frank acquired immunodeficiency syndrome (AIDS). Chemical gangrene. Carbolic acid (phenol) is the most dangerous, as anaesthesia masks the pain which occurs before the onset of gangrene. Carbolic compresses should never be used, for fingers have been lost by application of compresses even as dilute as 1:80. The gangrene is due to local arterial spasm. In addition, there is danger of severe systemic effects from absorption of phenol. Local bicarbonate soaks should be applied. Later, excision of the slough and skin grafting are necessary.

Ainhum (Fig. 11.38), a disease of unknown aetiology, usually affects black males (but some females) who have run barefoot in childhood. Besides Central Africa, there are reports from Central America and the East. A fissure appears at the level of the interphalangeal joint of a toe, usually the 5th, the fissure becomes a fibrous band, which encircles the digit and causes necrosis. The treatment is either early Z-plasty or, later, amputation.

Venous gangrene is discussed in Chapter 12 (Fig. 12.9).

Fig. 11.38 Ainhum (see text)
Amputation should be considered when part of a limb is Dead, Deadly or a Dead loss.

Dead. Arterial occlusion or stenosis, if sufficiently severe, will lead to tissue infarction with putrefaction of macroscopic portions of tissue (gangrene). The occlusion may be in major vessels (atherosclerotic or embolic occlusions) or in small peripheral vessels (diabetes, Buerger’s disease, Raynaud’s disease, inadvertent intra-arterial injection, ergotism (see preceding pages)). If the obstruction cannot be reversed and the symptoms are severe, amputation is indicated.

Deadly. Moist gangrene with its accompanying putrefaction and infection is dangerous, for the infection spreads to surrounding viable tissues, and cellulitis with severe toxemia and overwhelming systemic infection can occur. Amputation is indicated as a life-saving operation. (Compare with dry gangrene — see above.) Antibiotic cover should be broad and massive.

Other life-threatening situations for which amputation may be required include gas gangrene (as opposed to simple gas infection, Chapter 7), neoplasms such as osteogenic sarcoma and for arteriovenous fistulas.

Dead loss.

- Severe laceration and fracture with partial amputation due to the trauma of road accident or bomb-blast injury (mines) (Chapter 3).
- Severe contracture or paralysis, e.g. poliomyelitis, may make the limb impossible to use, and may hinder walking or any movement. Amputation can improve mobility.
- Severe rest pain without gangrene in a patient with an ischaemic foot may be an indication for amputation because of the relentless severity of the pain. Amputation under those circumstances can improve the quality of life.

**Distal amputation**

In patients with small-vessel disease (diabetes and Buerger’s disease), gangrene of the toes occurs with relatively good blood supply to the surrounding tissues. Therefore local amputation of the toe can result in healing.

- in diabetic patients:
  - infection tends to track up the tendon sheath;
  - infection tends to recur if the wound is closed;
  - neuropathy often makes early mobility possible because of lack of pain.

For these reasons, when the metatarsophalangeal joint region is involved in
diabetes, ‘ray’ excision is recommended, taking part of the metatarsal and cutting tendons back (Fig. 11.39). The wound should not be sutured but loosely packed with gauze soaked in an antiseptic solution such as proflavine (Chapter 9). Early mobility aids drainage provided cellulitis is not present. For less extensive gangrene, if amputation is taken through a joint, healing is improved by removing the cartilage from the joint surface.

Transmetatarsal amputation can be used in similar circumstances, where several toes are affected and irreversible ischaemia has extended to the forefoot, as in Buerger’s disease; but a viable long plantar flap is essential for this operation to heal successfully (Fig. 11.40).

Fig. 11.39 Conservative amputation for diabetic gangrene (‘ray’ excision).
Fig. 11.40 Transmetatarsal amputation for diabetic gangrene of the toes.

**Major amputation**
Preoperative preparation/informed consent. The patient should, whenever possible, be given time to come to terms with the inevitability of amputation, and ideally, once the alternatives between a painful useless limb or a painless useful (artificial) one are explained, the patient will make the final decision. This approach to the matter prevents the feeling by the patient that the loss of the limb is being imposed, which otherwise tends to make him or her less positive in attitude to retraining. In gangrene of the foot, especially with ‘skip’ areas, this is the time for explanation of, and consent for, above knee amputation should an attempt at below knee section prove inadvisable on account of inadequate blood supply to the flaps. The general condition of the patient needs to be maintained and/or improved, e.g. anaemia corrected and pain controlled.

**Physiotherapy** before the operation enables the patient to get used to the exercises that will prevent muscle wasting and flexion deformity of the hip.

**Antibiotics** should be given with the premedication to prevent clostridial infection (Chapter 7), particularly in above knee amputations.

**Analgesia.** The appropriate level of analgesia should be maintained up to the time of operation (and see postoperative analgesia in Chapter 4).

**Assessment of joints.** Flexion contracture, or severe arthritis may influence the level of amputation or the final degree of mobility.

Choice of operation (Fig. 11.41). Where good limb-fitting facilities exist, above or below knee amputations are preferable because the best cosmetic and functional results can be obtained by the cone-bearing amputation stumps. (Note the words ‘cone-bearing’. The term conical stump is reserved for an entirely different pathological entity: that which occurs when the growing humerus (or tibia), following amputation in a child, stretches the stump tissues and skin into an
unsightly cone. (Main growth occurs in the epiphyses located ‘toward the knee and away from the elbow’.) If Limb-fitting facilities are limited (e.g. where distances are prohibitive) end-bearing amputation may be preferable (Syme, through-knee, Gritti—Stokes) so that simple prostheses (peg leg or simple boot) can be used. Syme amputations are not suitable for severely ischaemic atherosclerotic limbs because of the poor healing of the heel flap.

Fig. 11.41 Choice of site: (a) cone-bearing and (b) end-bearing amputations.

**Cone-bearing amputations. General.** In above or below knee amputations, with good stump shape and limb-fitting facilities, it is possible to have a prosthesis held in place simply by suction, and no cumbersome and unsightly straps are necessary.

- The stump must be of sufficient length to give the required leverage (below the knee — not less than 8 cm (preferably 10—12 cm); above the knee — not less than 20 cm).
- There must be room for the artificial joint (the stump must not be too long): above the knee ideally 12 cm above the knee joint and below the knee 8 cm above the ankle joint are needed for the mechanism.

A below knee amputation is much better than an above knee (or Gritti—Stokes) amputation in terms of eventual mobility. Every attempt should be made to preserve the knee joint if the extent of ischaemia or trauma allows this.

**Below-knee amputations**

*Three* types of skin flap are commonly used: equal anterior and posterior flaps, long posterior flap, and the skew flap. Skew flaps were described by K. P. Robinson. Whatever method is chosen it is wise to remember the old rule that the total length of flap or flaps will need to be at least one and a half times the diameter of the leg at the point of bone section. One can always trim (subtract) but not add!

Long posterior flap below-knee amputation (Fig. 11.42). In cases of trauma, a tourniquet is applied at the thigh, but not in cases of ischaemia due to atherosclerosis or embolus. Anteriorly, the incision is deepened to bone, and the lateral and posterior incisions are fashioned to leave the bulk of the gastrocnemius muscle attached to the flap, muscle and flap being transected together at the same level. If bleeding is inadequate, the amputation is refashioned at a higher level.

**Fig. 11.42 Inflatable artificial limb,**

Blood vessels are identified and ligated. Nerves are not clamped but pulled down gently and transected as high as possible. Vessels in nerves are ligated. The fibula
is divided an proximal to the level of tibial division using bone cutters, the skin and muscle being retracted to avoid damage. The tibia is cleared and transected at the desired level, the anterior end of the bone at section being sawn obliquely before the cross-cut is made. This, with filing, gives an anterior smooth bevel which prevents pressure necrosis of the flap. The long muscle/skin flap is tapered after removing the bulk of soleus muscle (most of gastrocnemius may be left), the area is washed with saline to remove bone fragments and the muscle and fascia sutured with catgut or Dexon to bring the flap over the bone ends. Suction drains are placed deep to the muscle and brought out through a stab incision in the skin. The skin flap should lie in place with all tension taken by the deep sutures. Interrupted skin sutures are inserted. Drains can be attached to the skin by adhesive tape instead of sutures, thus allowing removal of the drain without taking down the stump dressing. Gauze, wool and crepe bandages make up the stump dressing.

Skew flaps. This form of below-knee amputation seeks to make use of anatomical knowledge of the skin blood supply. Equally long flaps are developed; they join anteriorly 2.5cm from the tibial crest, overlying the anterior tibia! compartment, and posteriorly at the exact opposite point on the circumference of the leg. After division of bone and muscle in a fashion similar to that above, the gastrocnemius flap is sutured over the cut bone end to the anterior tibial periosteum with catgut or Dexon. Finally, drainage and skin sutures are inserted and the limb dressed as in the long posterior flap operation.

Above-knee amputation. The site is chosen as indicated above, but may need to be higher if bleeding is poor on incision of the skin. Equal anterior and posterior skin flaps are made curved and of sufficient total length (one and a half times the anterior/posterior diameter of the thigh). Skin, deep fascia, and muscle are transected in the same line. Vessels are ligated. The sciatic nerve is pulled down and transected cleanly as high as possible and the accompanying artery ligated. Muscle and skin are retracted, the bone cleared and sawn at the point chosen. Haemostasis is achieved. The muscle ends are grouped together over the bone by means of catgut or Dexon sutures incorporating the fasoa. Two auction drains deep to the muscle are brought out through the skin clear of the wound and affixed with tape so that removal can take place without disturbing the stump dressing. The fascia and subcutaneous tissues are further brought together so that the skin can be apposed by interrupted sutures without tension. Gauze, wool and crepe bandages form the stump dressing.

Gritti—Stokes and through-knee amputations are rarely done nowadays. In the Gritti-Stokes type. the section is transcondylar.

Sytne’s amputation. It is essential to preserve the blood supply to the heel flap by meticulous clean dissection of the calcaneum. The tibia and fibula are sectioned as low as possible to the top of the mortice joint. This type of procedure is rarely
applicable in patients with occlusive vascular disease.

**Postoperative care of an amputation**

Pain relief. Diamorphirte or other opiates should be given regularly, and the reader should refer to the special section concerning pain relief in Chapters 4 and 5.

Care of the good limb. Attention is focused on the amputation, but a pressure ulcer on the good foot will delay mobilisation, despite satisfactory healing of the stump. The use of a cradle to keep the weight of bed clothes off the foot, and a ‘sheepskin’ (see above) are adjuncts to good nursing care.

Exercises and mobilisation. Immediately, the prevention of flexion deformity can be achieved by the use of a cloth placed over the stump with sand bags on each side to weight it down. Once the drains have been removed, exercises are started to build up muscle power and coordination. A stump bandage is applied each day to mould the shape of the stump. Mobility is progressively increased with walking between bars and the use of an inflatable artificial limb which allows weight-bearing to be started before a pylon or temporary artificial limb is ready (Fig. 11.43). It is emphasised that the whole episode in the patient’s life should be conducted in an attitude of promotion through the stages towards full independence. Early assessment of the home (part of the whole programme) allows time for minor alterations, such as the addition of stair rails, movement of furniture to give support near doors, and clearance in confined passages.

Complications. *Early*, include the following: reactionary haemorrhage, which requires return to the theatre for operative haemostasis; a haematoma, which requires evacuation; and infection, usually from a haematoma. Any abscess must be drained. Depending upon the sensitivity reactions of the organisms cultured, the appropriate antibiotics are given. Gas gangrene can occur in a midthigh stump — the organisms coming from contamination by the patient’s faeces. Wound dehiscence and gangrene of the flaps are due to ischaemia. and a higher amputation may well be necessary. Amputees are at risk of deep vein thrombosis and pulmonary embolism in the early postoperative period. Prophylaxis with subcutaneous heparin 5000 units twice daily is advised for at least 2 weeks after operation.

*Late*. Pain is usually the presenting symptom. due to: unresols’ed infection, e.g. a sinus, osteitis, sequestrum (even a complete ‘ring’ sequestrum); a bone spur; a scar adherent to bone; an amputation neuroma from the outgrowth of nerve fibrils which become attached to skin, muscle or fibrous tissue; a phantom limb.

*Phantom pain*. Patients frequently remark that they can feel the amputated limb and sometimes that it is painful. The surgeon’s attitude should be one of reassurance that these feelings will disappear. He or she should on no account foster the subject and talk about phantom-limb pain in front of the patient, as it is
very refractory to treatment once it is established (see also Chapter 5). Other late complications include ulceration of the stump (pressure effects of the prosthesis or increased ischaemia). Rarely, an ulcer is artefacta (Fig. 9.16). Some patients are troubled by cold and discoloured stumps, especially during the winter.

Fig. 11.43 Classic long posterior flap type of below-knee amputation.

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**Arterial Dilatation**

**Aneurysm**

**Definition:** Aneurysm is abnormal dilated localised segment of an artery.

**Types:**

- **Types according to aetiology:**
  - **True aneurysms:** containing the three layers of the arterial wall
    1. Congenital
    2. Atherosclerosis
    3. Mycotic
    4. Syphilitic
    5. Dissecting
  - **False aneurysms:** having a single layer of fibrous tissue at the wall of sac
    1. Pulsating hematoma (aneurysm following trauma)
    2. Arteriovenous (A-V) fistula

_N.B.: Mycotic is a misnomer, as the cause is not due to a fungus but to_
**bacterial infection (SBE).**

- **Types according to shape:**
  1. Fusiform
  2. Saccular
  3. Dissecting

**Site of Aneurysm:** Aneurysms can occur all over the body in major vessels such as aorta, femoral, popliteal, subclavian and carotid arteries, or in smaller vessels, such as the cerebral, mesentric, splenic and renal arteries. The majority are true fusiform atherosclerotic aneurysms.

**Clinical picture:** symptoms due to expansion, thrombosis, rupture, or emboli. Symptomatic aneurysms cause either minor symptoms, such as back pain and abdominal pain, or sudden, severe symptoms when they expand and rupture. **Extrinsic.** Neighbouring or distal structures are affected. Thus pressure on veins or nerves causes distal oedema or altered sensation. Bones, joints, or tubes, such as the trachea or oesophagus, are sometimes affected, but structures which are resilient, such as the intervertebral discs, often withstand prolonged pressure. **Intrinsic.** A swelling exhibiting expansile pulsation is present in the course of an artery. The pulsation diminishes if proximal pressure can be applied, and the sac itself is compressible, filling again if proximal pressure is released. A thrill may be palpable, and auscultation sometimes reveals a bruit.

**Differential diagnosis**

- **Swelling under an artery:** An artery may be pushed forwards, e.g. the subclavian by a cervical rib. Careful palpation distinguishes this condition.
- **Swelling over an artery:** Transmitted pulsation is mistaken for expansion pulsation. However, posture may diminish pulsation, e.g. pancreatic cyst over aorta.
- **Pulsating tumours:** such as bone sarcoma, osteodastoma, and a metastasis, especially from a hypernephroma.
- **An abscess**
- **A serpentine artery,** e.g. innominate, carotid.

**Abdominal Aortic Aneurysm**

Abdominal aortic aneurysm is the commonest type of aortic aneurysm. Generally, an aneurysm is considered significant if its diameter is twice that of the normal artery. The cause of abdominal aortic aneurysms is multifactorial. Age, smoking, hypertension, and family history are all predisposing factors toward aneurysm formation. Aneurysms are more common in males (4:1).
Clinical picture:
- Patients most commonly present without symptoms although they may have back pain in lumbar region, and upper abdomen.
- Pain can occur in the thigh and groin due to nerve compression.
- Gastrointestinal, urinary and venous symptoms can also be caused by abdominal aneurysm.
- As rule, the presence of pulsatile mass must be assumed to be due to aneurysm (until proved otherwise).

Investigations
Abdominal aortic aneurysms most often occur below the level of the renal arteries.
- X-ray: Calcification of part of the abdominal aorta seen on posterior—anterior or lateral abdominal x-ray is present in 60% of patients.
- Abdominal U/S & CT-scan \(\Rightarrow\) dilated aorta, aneurysm size, aneurysm diameter.
- Angiography & Aortogram useful in delineating proximal and distal extent of aneurysm \(\text{(it does not delineate diameter because the sac is usually filled with circumferential clot leading to a falsely narrow angiographic appearance)}\).
- Routine investigations: Urine analysis to exclude D.M, Hb, full blood count, ESR, blood group & cross-match, ECG, liver function, blood lipids, electrolytes & urea.

Indication for surgery
- Symptomatic aneurysm
- Asymptomatic aneurysm: Aneurysm found incidentally on radiography or U/S needs repair if >5 cm in diameter.

Surgical treatment:
Aneurysms are repaired by replacing the diseased segments of the aorta with prosthetic grafts. Intraoperatively, patients should be very carefully controlled.
1. Swan-Ganz catheter to monitor cardiac output during aortic clamping.
2. Mannitol is given to stimulate diuresis.
3. Heparin is given
4. Aortic clamp time is kept to a minimum.
5. Acidosis and hyperkalemia may occur after clamps are removed and must be treated promptly.

Postoperative complications
- Respiratory complications are commonest
- Haemorrhage.
- Ischaemia of the colon due to lack of collateral supply occurs in 10 % of cases.
- Renal failure, infection of the graft and wound dehiscence are rarely seen.
- Sexual dysfunction, fistula formation and spinal cord ischaemia.
- Aortoduodenal fistula is uncommon but treatable. It should be suspected whenever haematemesis or melaena occurs after operation.

**Atypical aneurysms of the abdominal aorta**

1. **Inflammatory aneurysms** are characterized by dense fibrotic reaction, involving anterior and lateral walls of the aneurysm and the surrounding tissues. The aneurysm is repaired by dissecting the neck above the area of inflammation for proximal control, frequently above the duodenum near the renal vein.

2. **Mycotic abdominal aortic aneurysms** are caused by bacterial inflammation of the arterial wall. In the infrarenal aorta, commonest organism is *Salmonella*. Mycotic aneurysms usually are saccular, and lack calcification of the wall.

**Clinical picture:** Patients present with fever, elevated white blood cell counts, and positive blood cultures. Evidence of septic embolization may also be present. Treatment begins with culture & sensitivity. Patients are then surgically explored.
   1. If there is no peri-aortic purulence and Gram stain of the proximal and distal artery is negative, the aneurysm is repaired with an interposition graft.
   2. If gross purulence is present, the aneurysm and surrounding tissue are resected, the aorta is closed, and axillofemoral bypass bypass is constructed.
   3. Long-term antibiotic therapy is indicated in these patients.

**Ruptured abdominal aneurysm**

**Site of rupture:** Abdominal aortic aneurysms can rupture: anteriorly into peritoneal cavity (20%) or posteriorly into retroperitoneal space (80%).
- Anterior rupture results in free bleeding into the pentoneal cavity. Patients have prolonged period of hypotension and shock.
- Posterior rupture produces retroperitoneal haematoma. Patients have moderate hypotension and the resistance of the retroperitoneal tissues stops the haemorrhage. The patient remains conscious, but in severe pain.

**Risk of rupture:**
   a. It is related to the size of the aneurysm, rates of rupture are:
      1. If less than 4.5 cm in diameter, 9%
      2. If 4.5—7 cm in diameter, 35%
      3. If more than 7 cm in diameter, 75%
   b. The expansion rate of an aneurysm is 0.4 cm in diameter per year. More rapid expansion suggests an unstable aneurysm and is an indication for repair.

**Clinical picture:**
- Severe central abdominal or back pain, localized to lower abdomen, groin, or
testes. It may be accompanied by a brief loss of consciousness.
- Pulsatile, tender abdominal mass.
- Femoral pulses in one or both groins may be diminished or absent.
- Shock due to rupture.

**Diagnosis:**
- Endoscopy of the esophagus, stomach, and duodenum.
- CT-scan → demonstrate air or fluid around an infected graft.
- Indium-tagged WBC scan → localize the area where the graft is infected.
- Aortogram → demonstrate a false aneurysm.
- Sinogram → outlines the graft.

**Surgical repair:**
- Definitive treatment of burst aneurysm is operation, not resuscitation!
- Two good infusion lines and a central venous pressure line must be inserted as soon as the patient arrives in hospital. Infusion are given to raise systolic BP to approx 100 mmHg. A urinary catheter is passed.
- Operation of ruptured aortic aneurysm into retroperitoneal space: The patient is anaesthetised, full length midline transverse incision to the patient’s right side to expose the aorta and the haematoma lying behind the posterior peritoneum. Aortic pulsation is palpated through the haematoma and fingers are insinuated each side of aorta.

**Surgical complications:**
1. **Aortic graft infection**, commonest infecting organism is *Staphylococcus aureus*. First-generation cephalosporins (cefazolin) are the drugs of choice.
2. **Postoperative renal failure**, treatment is hemodialysis.
3. **Ischaemia of colon** due to lack of collateral supply, treatment (Hartmann’s procedure) → dead colon resection + proximal colostomy + distal colon closure.
4. **Acute leg ischaemia**, treatment is repair of the injury or embolectomy.
5. **Sexual dysfunction**: The sympathetic nerves controlling ejaculation cross the left common iliac artery near the aortic bifurcation. If injured, retrograde ejaculation will occur.
6. **Spinal cord ischemia**, The artery of Adamkiewicz supplies the spinal cord and arises from the left side of the abdominal aorta, usually at T₈ to L₁ but occasionally as low as L₄. Spinal cord ischemia produces a classic anterior spinal artery syndrome, which is characterized by:
   1. Paraplegia
   2. Rectal and urinary, incontinence
   3. Loss of pain and temperature sensation but preservation of vibratory and proprioceptive sensation
Other arterial aneurysms:

1. **Iliac artery aneurysms:** usually are extensions of aortic aneurysms, diagnosed as pulsatile masses that are palpable on rectal examination; occasionally, they rupture into the sigmoid colon or any part of GIT and present as GIT bleeding.

2. **Splanic artery aneurysms:** are common after aortic aneurysms. Causes—fibrous dysplasia, portal hypertension, multiparity, and inflammation secondary to pancreatitis. Diagnosis by plain film of the abdomen shows a left upper quadrant ring-shaped calcification. Indications for repair include rupture, symptoms (pain in left upper quadrant), and the presence of an aneurysm in a woman of childbearing age.

3. **Peripheral arterial aneurysms:** The popliteal artery is the most common location for peripheral aneurysms. The usual cause is atherosclerosis.
   - **Diagnosis:** Physical examination detects prominent popliteal pulses, and the aneurysm can be seen on ultrasound. An arteriogram guides reconstruction.
   - **Rupture** is rare, but embolization and thrombosis are common.
   - **Treatment:** by ligation and bypass. Attempts to completely remove the aneurysm are dangerous because injury to adjacent nerves and veins can occur.

**Arteriovenous (A-V) fistula**

**Definition:** Abnormal communication between artery and vein

**Types:**
- **Congenital fistula:** represent development anomalies. They range from single (birthmarks) to complex (Klippel-Trenaunay syndrome)
- **Acquired fistula:** by the trauma of a penetrating wound or a sharp blow.

*N.B.: Arteriovenous fistulas are also created surgically in the forearms or legs of patients undergoing renal dialysis.*

**Effect:**
- **Structural effect:** of the arterial blood flow on the veins is characteristic, as they become dilated, tortuous, and thick walled (arterialised).
- **Physiological effect:** high-pressure in arterial system + ↑ venous return & pressure ➔ ↑ pulse rate & cardiac output ➔ Left ventricular enlargement ➔ cardiac failure.

A congenital fistula in the young may cause overgrowth of a limb. In the leg, indolent ulcers may result from relative ischaemia below the short circuit.

**Clinical picture & Diagnosis:**
- Pulsatile swelling may be present if the lesion is relatively superficial.
- Dilated veins may be seen, in which there is a rapid blood flow.
- On palpation: a thrill is detected
- Auscultation reveals a buzzing continuous bruit
- Pressure on the artery proximal to the fistula causes the swelling to diminish in size, the thrill and machinery bruit to cease, the pulse rate to fall (Branham’s sign) and pulse pressure to return to normal.
- Arteriography confirms the lesion.

Treatment:
- Congenital lesions (usually stationary): excision is advocated only for severe deformity or recurrent haemorrhage.
- Acquired lesions (progressive): quadruple ligation operation is indicated: ligation of the involved artery and vein is required both above and below the lesion. Vein or Dacron grafts may be required.

Arteritis

Temporal, occipital and ophthalmic arteritis
Localised infiltration with inflammatory and giant cells leads to arterial occlusion and features ischaemic headache and tender, palpable, pulsetess (throm. bose) arteries in the scalp, and the major catastrophe of irreversible blindness when the ophthalmic artery is occluded.
A raised ESR and a positive scalp artery biopsy calls for immediate prednisolone therapy (20 mg three times daily) to arrest and reverse the process before the ophthalmic artery is involved. The dose must be reduced as soon as possible, in line with clinical improvement and a fall of the ESR, to a maintenance dose which is controlled under long-term surveillance.
Takayasu’s arteriopathy (obliterative arteritis of females, pulseless disease) causes narrowing and obstruction of major arteries and usually pursues a relentless course (Fig. 11.57).
Cystic myxomatous degeneration. An accumulation of clear jelly (like a synovial ganglion) in the outer layers of a main artery may occasionally be encountered, especially in the popliteal artery. The lesion so stiffens the artery that pulsation disappears, and claudication occurs when the limb is flexed (as on waUdng up stairs). Arteriography shows a smooth narrowing of an otherwise normal artery,
and a sharp kink, or buckling, when the knee is flexed.
Decompression, by removal of the myxomatous material, is all that is required, but
the ‘ganglion’ may recur and require excision of part of the artery with
interposition vein graft repair.
Other types of arteritis are encountered in rheumatoid arthritis, diffuse lupus
erythematosus and polyarteritis. Treatment is similar. Diabetes is discussed earlier.
The subclavian and other arteries were also occluded by Takayasu’s arteriopathy
(see text).

Vasopastic Conditions
(Small-vessel abnormalities)

Vasopastic diseases affect the upper extremities and involve usually small palmar
and digital arteries and arterioles. Common symptoms include pain, numbness,
coldness, and, occasionally, skin ulcers. Vasospasm may be associated with
collagen vascular disease, atherosclerosis, trauma, and embolism from peripheral
arterial lesions, or may be without an identifiable associated disease.

Associated disorders
1. Cryoglobulinemia or cold hemagglutinins
2. Myxedema
3. Ergotism
4. Thrombocytosis
5. Macroglobulinemia
6. Occupations in which the hands suffer repeated trauma, such as a
   jackhammer operator
7. Nerve compression syndromes, such as the carpal tunnel syndrome
8. Arterial compression syndromes, such as the thoracic outlet
   syndrome

Diagnosis. Tests may be performed to document vasospasm.
1. Doppler examination to measure arm and wrist blood pressure
2. Digital plethysmography to study the digital arteries. If they are
diminishing and unresponsive to temperature change, the arteries are
occluded. If warming reverses the changes, vasospasm is likely.
3. The presence of a cervical or an axillary bruit suggests arterial
   occlusive disease. Arteriography may be necessary to eliminate this
   possibility totally. During arteriography, a vasodilator may be injected
intra-arterially and the dye injection repeated. Demonstration of increased blood flow and vasodilation is strong support for the presence of vasospastic disease.

Acrocyanosis, *crurum puellarsm frigidum*’ may be confused with Raynaud’s disease, but it is painless and is not paroxysmal. Affecting young females, the cyanosis of the fingers, and especially the legs, may be accompanied with paraesthesia and chilblains. In severe cases, sympathectomy may be tried. If merely affecting the calves, a differential diagnosis is Bazin’s disease.

Pregangliomic cervicodorsal sympathectomy. **Supraclavicular method.** Through a supraclavicular incision, the clavicular part of the sternomastoid, the posterior belly of the omohyoid, and the scalenus anterior muscles are divided, the phrenic nerve being displaced inwards. The subclavian artery is exposed, and the thyrocervical trunk is divided. The subclavian artery is depressed and the suprapleural faunas is divided, so that the dome of the pleura can be displaced downwards. The atelate ganglion is identified as it lies on the neck of the first rib (Fig. 11.58). The sympathetic trunk is traced downwards and divided below the third thoracic ganglion. All rami communicantes associated with the second and third ganglia and the nerve of Kuntz, a grey ramus running upwards from the second thoracic ganglion to the first thoracic nerve, are meticulously divided. Occasionally, the approach is under a high arching subclavian artery.

Fig. 11.58 Cervical sympathectomy: exposure of the superior ganglion of the sympathetic chain through a supraclavicular approach.

**Transthoracic method.** This gives a greater exposure and facilitates the removal of the sympathetic chain from the fifth ganglion up to the lower fringe of the stellate ganglion. It tends to give better results than the supraclavicular method and can be employed when that has failed. In women where cosmetic effects are a consideration, the approach can be made via an axillary incision through the third space (Hedley Atkins). The sympathetic chain is easily seen and after dividing the pleura, it is dissected out, care being taken to avoid damage to the intercostal vessels, which may cause tedious haemorrhage. Care should also be taken, when making and suturing the approach wound, to avoid damage to the nerve to serratus anterior, giving rise to ‘winging’ of the scapula. This procedure can now be performed using the laparoscope and diathermy to cauterise the sympathetic chain.

**Endoscopic method.** This seeks to achieve sympathectomy via the transthoracic route using a suitable endoscope, e.g. a cystoscope or laparoscope. A Verres needle is passed via the axilla to induce a CO₂ pneumothorax. A trochar and cannula are then employed to introduce the endoscope. The sympathetic chain is visualised and
a coagulating electrode used to disrupt the ganglia. The endoscopic method is now the procedure of choice for cervicodorsal sympathectomy.

Lumbar sympathectomy. **Operative method.** Using a transverse loin incision, an extraperitoneal approach is used in which the colon and peritoneum, to which the ureter clings, are stripped inwards so as to expose the inner border of the psoas muscle (Fig. 11.59).

Fig. 11.59 Left lumbar sympathectomy. The central retractor is holding back the peritoneum and ureter to expose the aorta and the psoas muscle, between which ties the sympathetic chain. Note that the second lumbar ganglion, adjacent to the tower pole of the kidney, has connecting rami to the spinal cord. The genitofemoral nerve is seen emerging through the fibres of the psoas.

The sympathetic trunk lies on the sides of the bodies of the lumbar vertebrae, and on the right side is overlapped by the vena nays. Lumbar veins are apt to cross the trunk superficially. The sympathetic trunk is divided on the side of the body of the fourth lumbar vertebra, and is traced upwards to be divided above the large second lumbar ganglion, which is easily recognised by the number of white rami which join it. Care should be taken not to mistake small lymph nodes, lymphatics, the genitofemoral nerve or the occasional tendinous strip of the psoas minor for the sympathetic chain. Along with a decline in the recognised indications for sympathectomy there has been a move away from the operative approach in favour of the less hazardous chemical (phenol) sympathectomy.

**Fig.** 11.80 Lumbar paravertebral injection.

**Chemical method.** This is contraindicated in patients taking anticoagulants. Under radiographic fluoroscopic control, with the patient in the lateral position, local anaesthetic is injected. A long spinal needle is then inserted (Fig. 11.60) to seek the side of the vertebral body and to pass alongside it to reach the lumbar sympathetic chain. After confirming the needle position by injection of contrast agent, approximately 5 ml of phenol in water (1:16) is injected. This is usually done at two sites: beside the body of the second and fourth lumbar vertebrae. Great care is needed to avoid penetrating the aorta, cava or ureter; the plunger of the syringe must always be drawn back before injection to exclude the presence of blood.