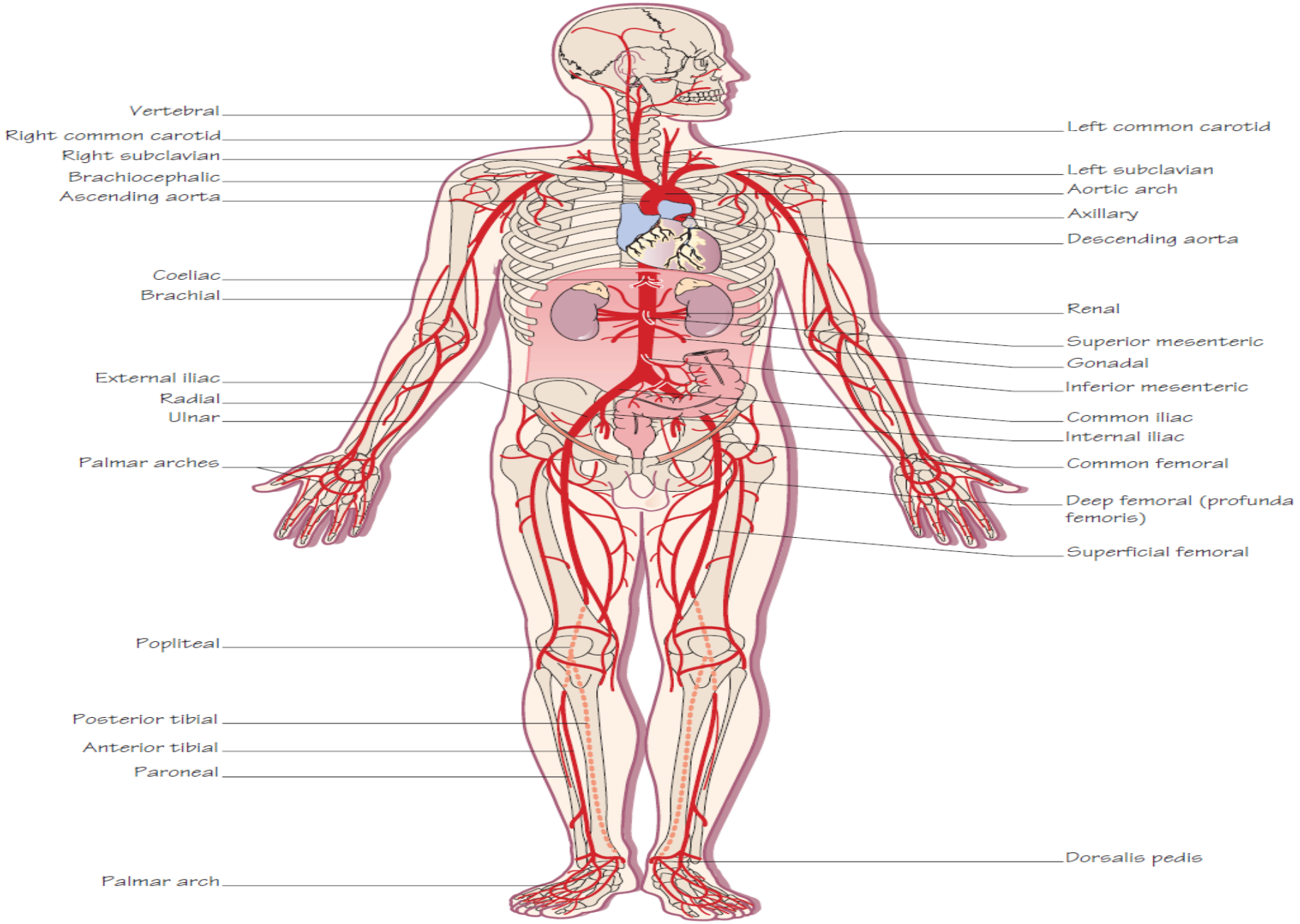

Vertebral
Right common carotid
Right subclavian
Brachiocephalic
Ascending aorta
Coeliac
Brachial
External iliac
Radial
Ulnar
Palmar arches
Popliteal
Posterior tibial
Anterior tibial
Paroneal
Palmar arch
Left common carotid
Left subclavian
Aortic arch
Axillary
Descending aorta
Renal
Superior mesenteric
Gonadal
Inferior mesenteric
Common iliac
Internal iliac
Common femoral
Deep femoral (profunda femoris)
Superficial femoral
Dorsalis pedis

Histology

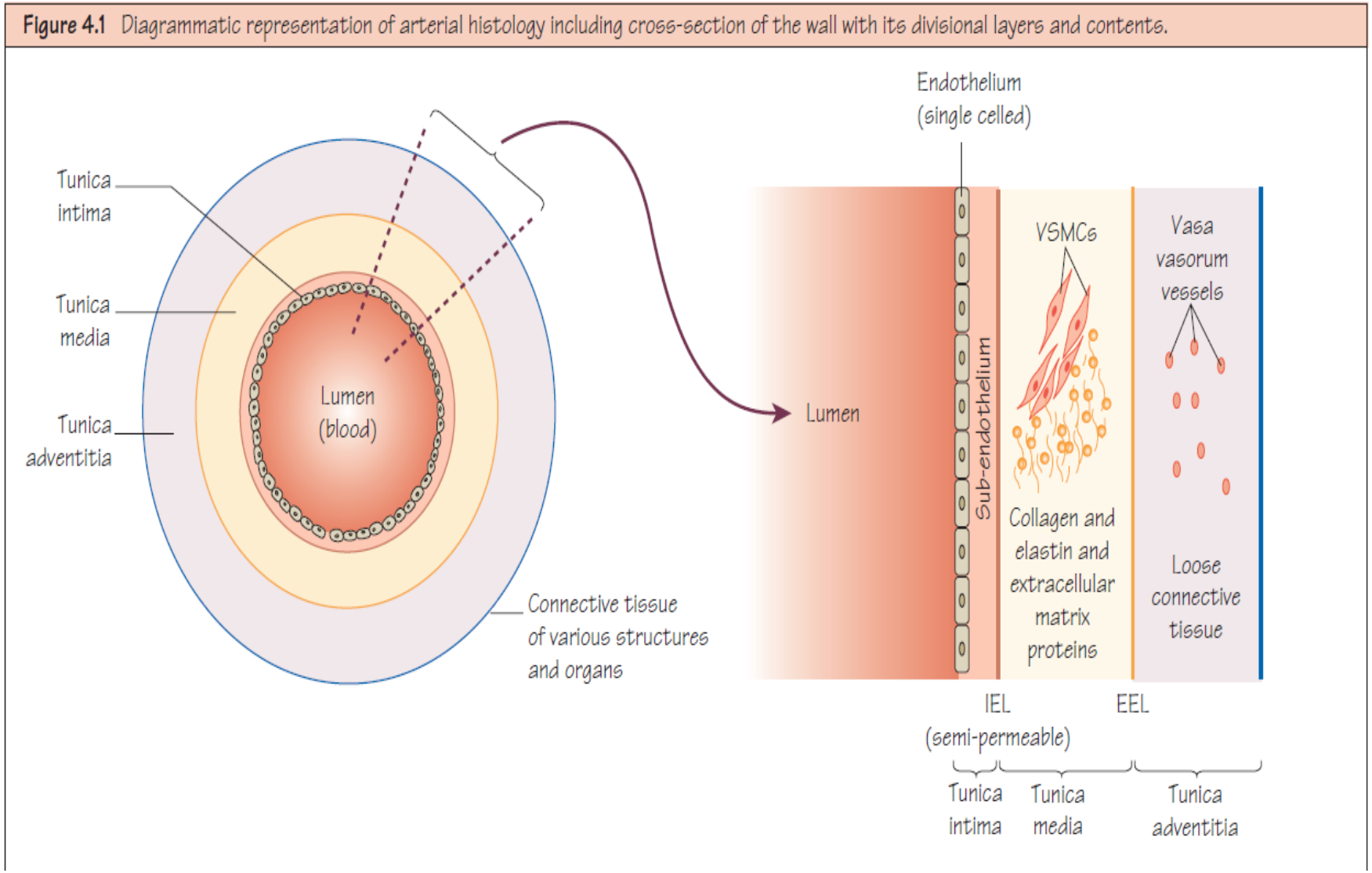
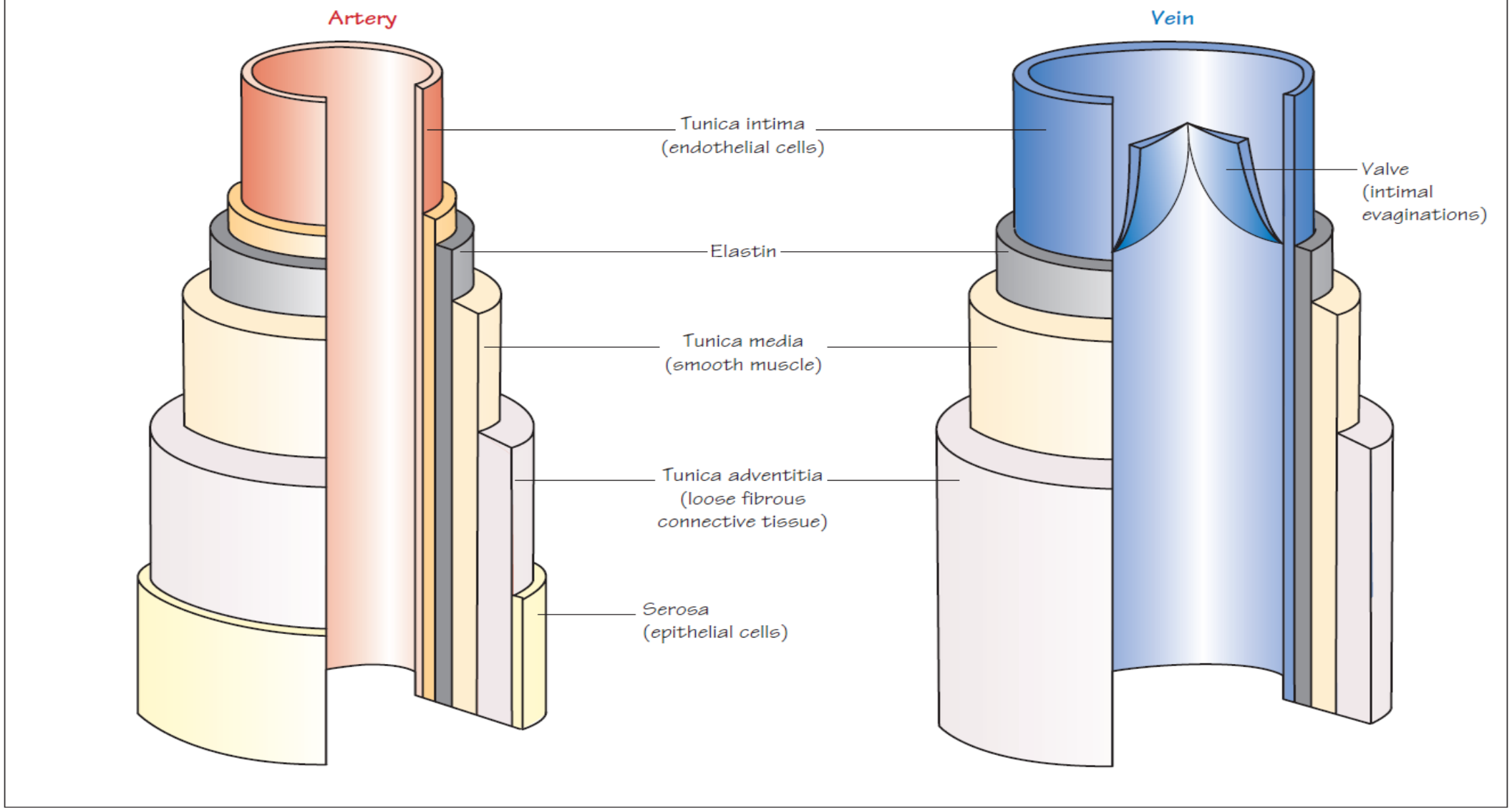


Figure 4.1 Diagrammatic representation of arterial histology including cross-section of the wall with its divisional layers and contents.
Tunica intima
Tunica media
Tunica adventitia
Lumen (blood)
Connective tissue of various structures and organs
Endothelium (single celled)
Lumen
Sub-endothelium
VSMCs
Collagen and elastin and extracellular matrix proteins
Vasa vasorum vessels
Loose connective tissue
IEL (semi-permeable)
EEL
Tunica intima
Tunica media
Tunica adventitia

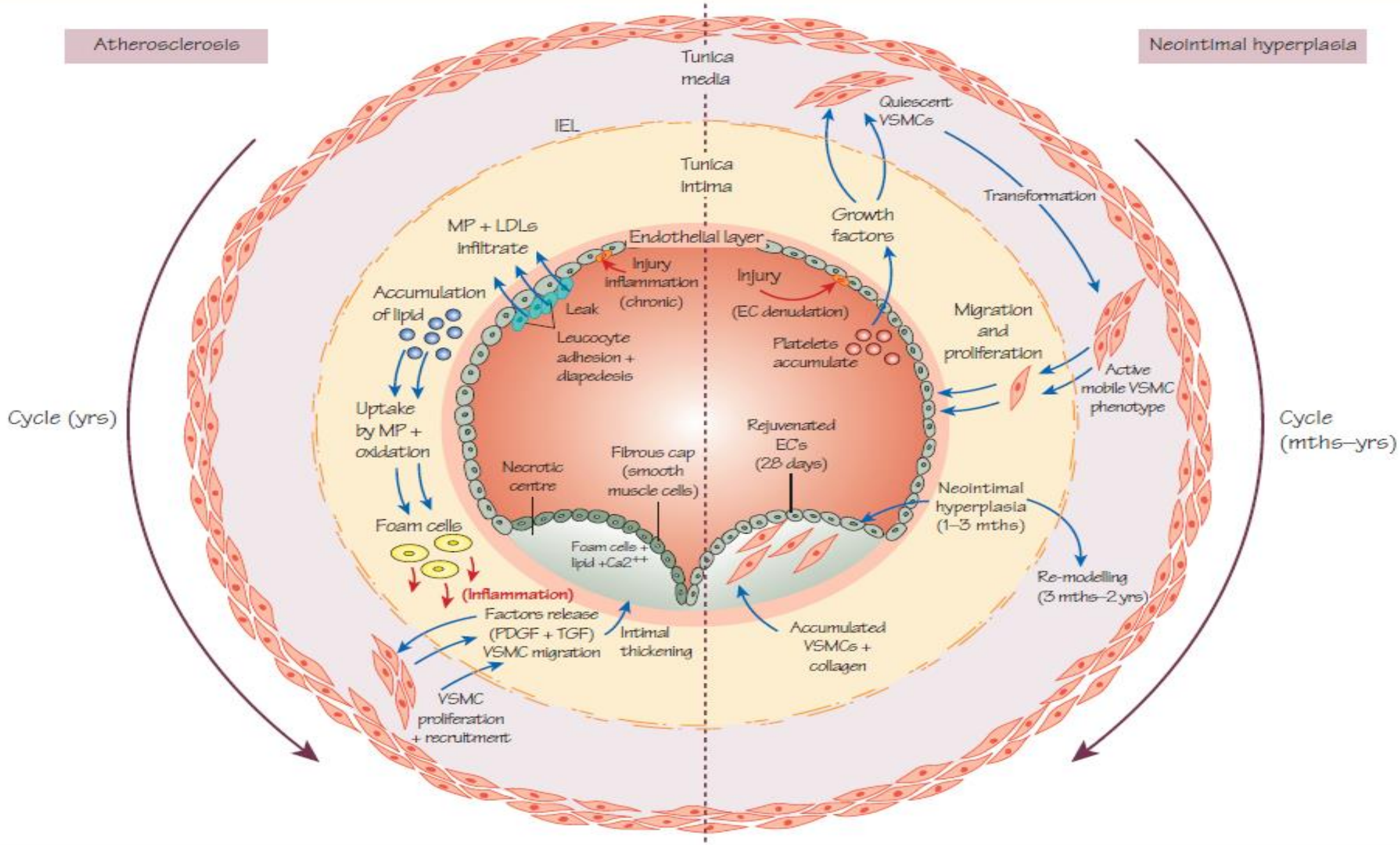
Figure 4.2 Diagram illustrating the histological divisional layers that make up arteries and veins.



Abbreviations: EEL, external elastic lamina; IEL, internal elastic lamina; VSMCs, vascular smooth muscle cells

Vascular pathobiology

Figure 5.1 Illustration of the histopathological changes that occur with the two most prevalent and troublesome pathological conditions in vascular surgery: Atherosclerosis (left) and neointima hyperplasia (right).



Atherosclerosis

This principally affects large and medium-sized arteries (the aorta and its branches including coronaries, carotid, mesenteric and lower limb), but has a preponderance for occurring at branching sites (e.g. carotid bifurcation). Known risk factors for its development include male gender, advancing age, smoking, dyslipidaemia, diabetes mellitus and hypertension. Atherosclerotic lesions may occur in isolation, but, as a rule, atherosclerosis is a systemic disease affecting numerous arterial locations. Furthermore, an atherosclerotic lesion in one location (e.g. lower limbs) serves as a surrogate marker for disease elsewhere (e.g. coronary arteries).

Pathophysiology

Atherosclerosis is a degenerative disease of large and medium-sized arteries characterized by lipid deposition and fibrosis.

- There are *three stages* of atheromatous lesion; *fatty streaks* are linear lesions on the artery lumen, composed of lipid-filled macrophages, and which progress to *fibrolipid plaques* (unstable plaques), and finally *complex lesions* (stable plaques).

Pathophysiology

Atherosclerosis is a degenerative disease of large and medium-sized arteries characterized by lipid deposition and fibrosis.

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Neointimal (myointimal) hyperplasia

- This is the vascular histological response to acute injury (e.g. surgery, angioplasty, stent insertion), initiated by endothelial injury or denudation (response is proportional to the severity [depth] of injury [i.e. if the media is also involved]).

Arteriosclerosis

- This is a general term for sclerosis or ‘hardening’ of the arteries and
- is broadly subdivided into two types:
- **1 *Arteriosclerosis obliterans.*** This is characterised by gradual fibrosis and calcification of the intima and media leading to stenosis and eventual obliteration, and it mostly affects the medium and large arteries of the lower extremities.
- **2 *Medial calcific sclerosis.*** Also called *Monkeberg’s arteriosclerosis*, this is characterised by dystrophic calcification of the media without intimal involvement or luminal narrowing, commonly affecting the extremities with advancing age.

Ischaemia-reperfusion injury

- This phenomenon occurs after restoration of blood flow following a (variable) period of ischaemia resulting in further tissue damage (due to the reperfusion) with both systemic and local effects. It is caused by the uncontrolled release of oxygen-free radicals and superoxide moieties (especially the oxidation of hypoxanthine) that are generated in response to tissue ischaemia.
- **Local effects**
- Tissue *oedema* and *necrosis* leading to *compartment syndrome* (further potentiating the *ischaemia*).
- **Systemic effects**
- *Acidosis* and *hyperkalaemia* (release of accumulated acid moieties and intracellular potassium, respectively), *coagulopathy* (prothrombotic necrotic tissue) and *myoglobinuria* (*rhabdomyolysis* [↑ creatine kinase (CK)]) leading to *acute kidney injury*.

Aneurysmal degeneration

- This is a degenerative condition of the vessel wall perhaps due to abnormal metalloproteinase (MMP) production and regulation. MMPs (especially MMP-2 and MMP-9) are thought to have enzymatic properties that degrade elastin, which in combination with years of increased wall stress leads to progressive vessel dilatation.

Peripheral vascular resistance (PVR)

- This is the effect the pressure (energy) drop has on flow rates (akin to Poiseuille's law) and is dependent on *radius* of the vessel (r4), *length* of vessel (L) and *viscosity* of fluid.

9 Cardiovascular risk factors

Figure 9.1 Known significant vascular risk factors.

Modifiable

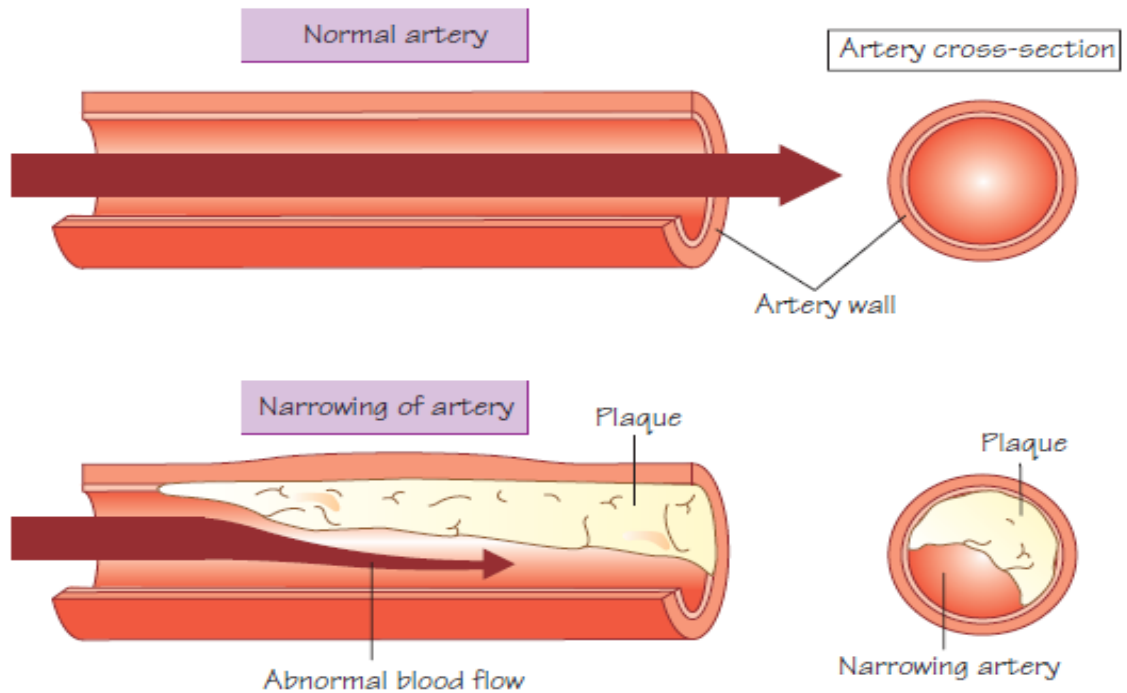
Smoking

Diabetes mellitus

Hypercholesterolaemia

Hypertension

Obesity & lack of exercise



Non-modifiable

Increasing age

Ethnicity

Family history

Ankle brachial pressure index

Figure 13.1 The patient is supine and the probe is angled at 45–60 degrees over the brachial artery.

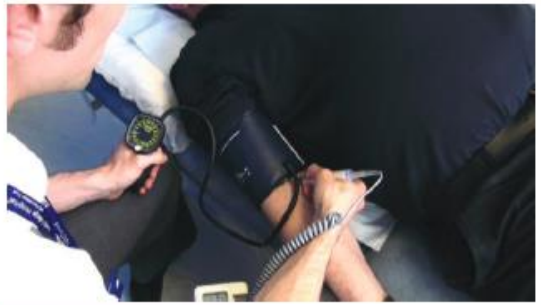
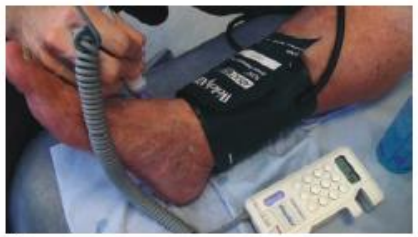
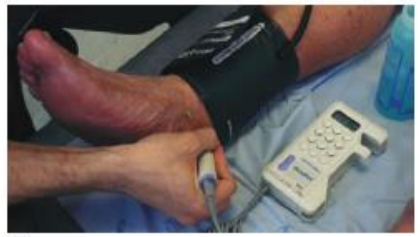


Figure 13.2 (a) The probe is angled over the DP artery and (b) over the PT artery.



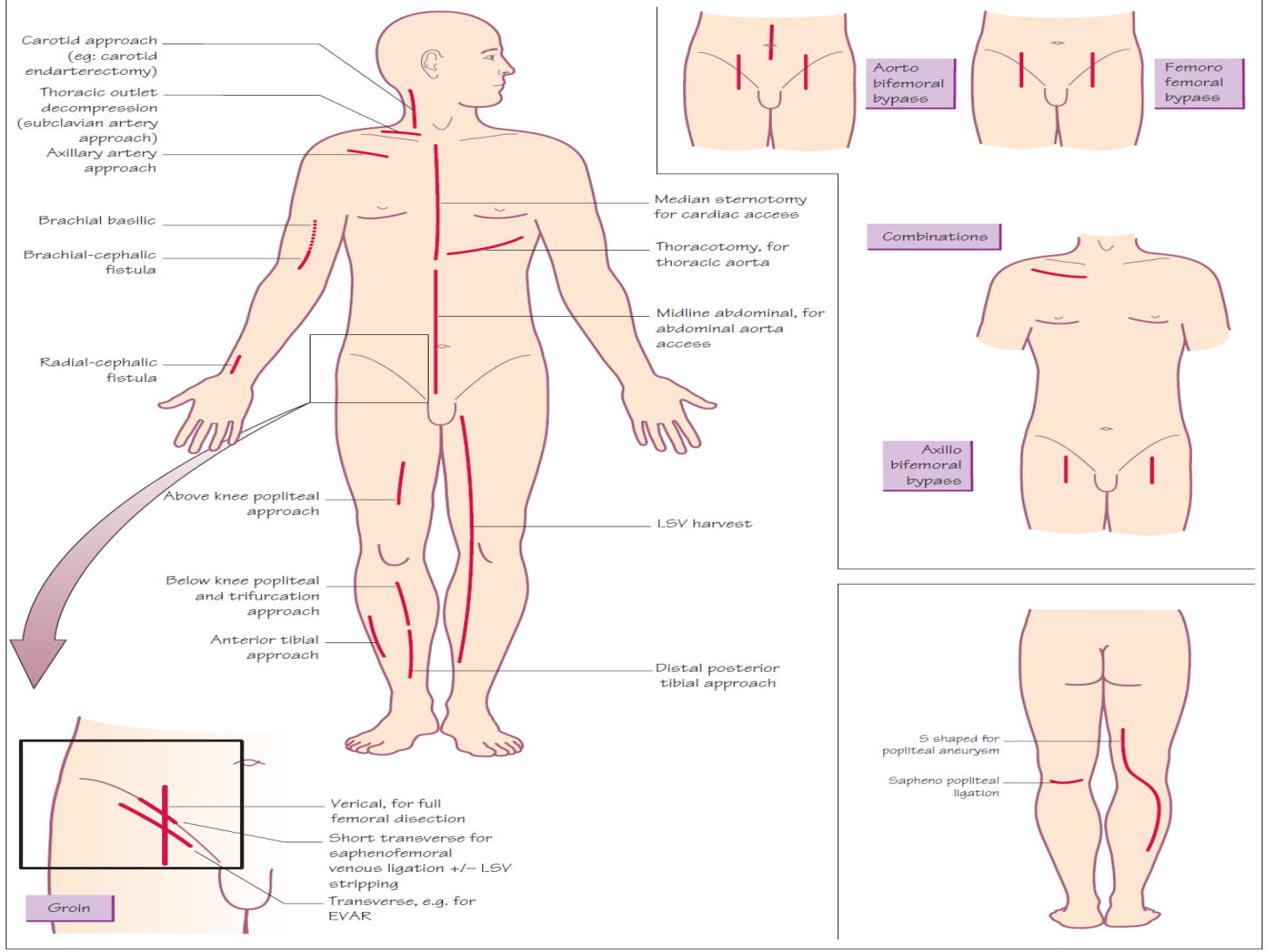
(a)



(b)

Resting	Disease severity
1.4 or greater	Calcification likely
0.9–1.3	Not suggestive of arterial disease
0.5–0.89	Suggests arterial minor disease (likely causing claudication)
0.40–0.5	Severe occlusive disease
Less than 0.3	Critical ischaemia with likely rest pain and tissue loss

Figure 15.1 Interpreting vascular scars.



Abbreviations: EVAR, endovascular aneurysm repair; LSV, long saphenous vein

16 Vascular investigations: Overview

Figure 16.1 Treadmill machine for measuring ABPIs before and after exercise.

Figure 16.2 Equipment for measuring ABPI including hand-help Doppler device, BP cuff and manometer and ultrasound gel.

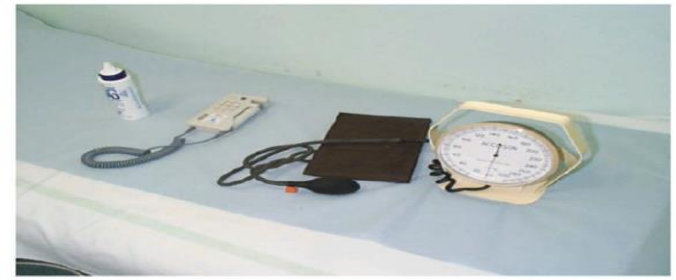


Figure 16.3 Measuring ABPI's at dorsalis pedis artery. Note BP cuff around calf.

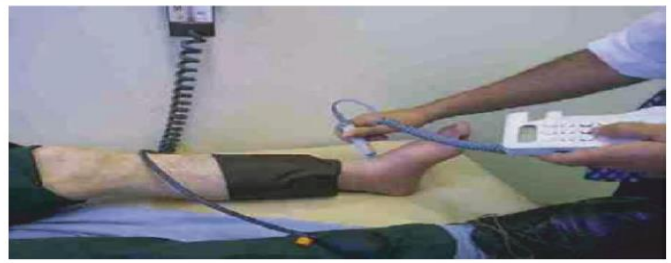


Figure 16.4 Typical state-of-the-art vascular ultrasound machine.

Figure 16.5 Outline of normal artery on colour flow Doppler (red).

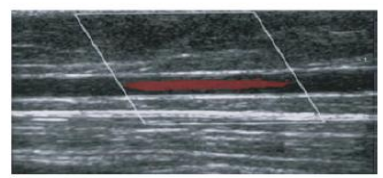


Figure 16.6 CT angiogram outlining left lower limb vessels including profunda femoris and SFA. Occlusion at level of adductor hiatus (AK popliteal artery).

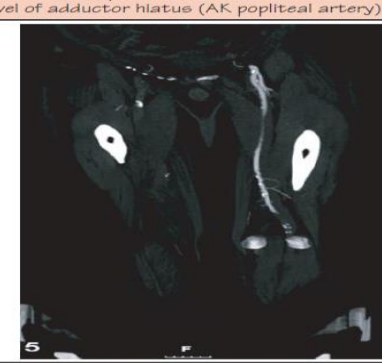
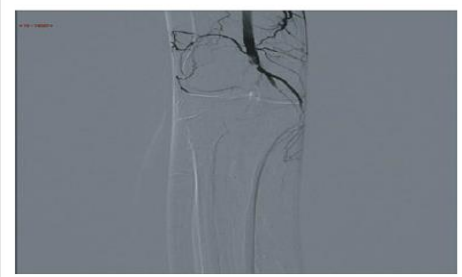


Figure 16.7 Catheter-directed angiogram showing occlusion at the AK popliteal artery with preserved collateral branches (geniculate vessel).



Abbreviations: AK, above knee; ABPI, ankle-brachial pressure index; BP, blood preaaure; CT, computed tomography; SFA, superficial femoral artery

20 Angiography I: Overview

Figure 20.1 Layout of a typical hybrid angiography suite.

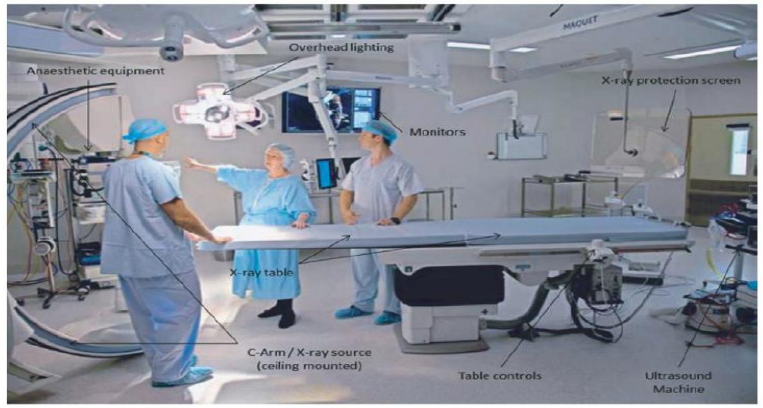
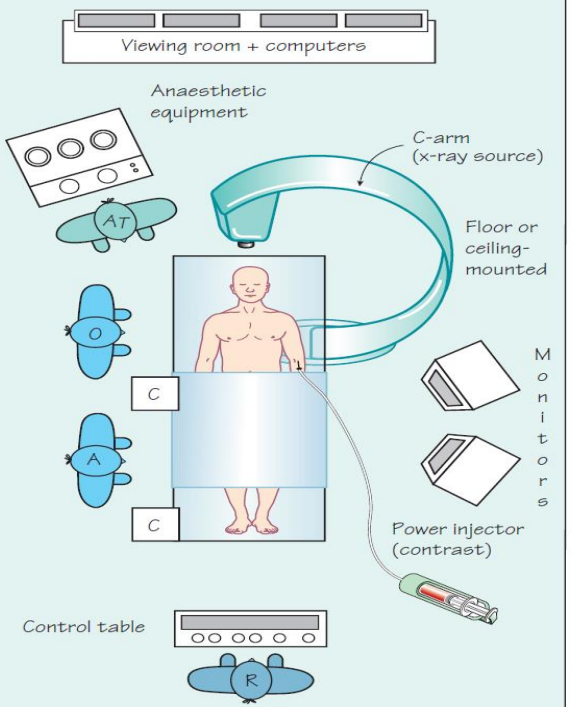


Figure 20.2 Layout of angiography suite.



Abbreviations: A, assistant; AT, anaesthetist (if required); C, controls (for table); O, operator; R, radiographer

Figure 20.3 Potential side effects of intravenous iodinated contrast agents.

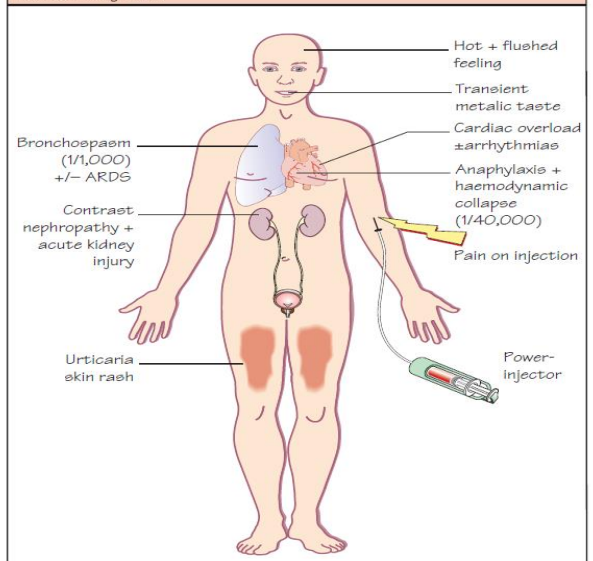


Table 20.1. Examples of commercially available contrast agents and their corresponding iodine concentration and osmolality. Ionic hyperosmolar agents give superior images (higher concentrations of iodine give 'denser' images) but have worse side effect profiles (more iodine atoms per molecule). Nonionic agents covalently bind iodine with less dissociation into component molecules, hence less side effects. In addition, nonionic compounds are more expensive.

Ionic			Nonionic		
Name	Iodine content (mg/ml)	Osmolality (mOsm/kg H_2O)	Name	Iodine content (mg/ml)	Osmolality (mOsm/kg H_2O)
Hypaque 25% (sodium diatrizoate)	150	696	Omnipaque 140 (iohexol)	140	322
Hypaque 50% (sodium diatrizoate)	300	1550	Omnipaque 240 (iohexol)	240	520
Renografin-60 (meglumine diatrizoate)	292	1549	Omnipaque 350 (iohexol)	350	884
Isopaque 370 (metrizoate)	370	2100	Visipaque 320 (iodixanol)	320	290
Hexabrix (meglumine ioxglate)	320	600	Optitray 320 (ioversol)	320	702
Conray-30 (meglamine iothalamate)	141	681	Isovue #28 (iopamidol)	128	290
			Isovue 370 (iopamidol)	370	796
			Oxilan 350 (ioxilan)	350	695
			Ultraviet (iopromide)	370	774

21 Angiography II: Access and imaging

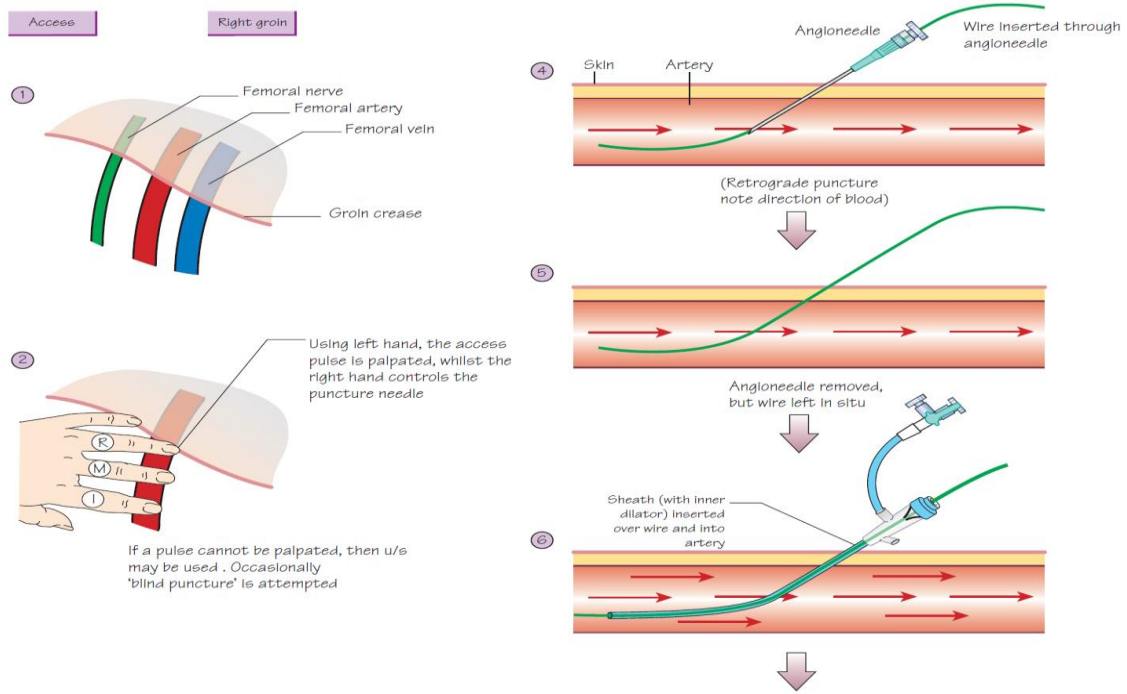
Figure 21.1 Basic steps and fundamental principles of angiointervention.

1. Safe access
2. Safe travel (to the site of intervention)
3. Safe haemostasis

Basic steps in angiography:
1. Access (vessel for intervention)
2. Travel (to vessel of interest)
3. Imaging
4. Intervention
5. Re-imaging
6. Removal of access and haemostasis

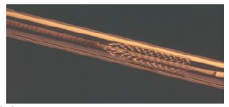
General Principles:
- All devices with a central lumen must be pre-flushed with heparinised saline to remove any air or fragments and to ensure patency
- Access vessel
  - Puncture and sheath insertion
  - US may be necessary
- Fluoroscopic imaging is used at all times when passing or withdrawing wires and catheters
- Travel from access vessel to target lesion
  - Variety of guidewires and catheters
  - Fluoroscopic guidance
- Diagnostic angiography
  - Images of the vessel and the lesion are taken using contrast
  - Digital subtraction of the images (eg: smart masking)
- Traversing the target lesion safely
  - Variety of guidewires and catheters
  - Digital subtraction used
- Treatment at the target lesion
  - Angioplasty
  - Subintimal angioplasty
  - Stent insertion
- Post-therapy angiography (check angiography)
- Instrument withdrawl (under flouroscopy)
- Haemostasis
  - Manual pressure
  - Closure devices

Figure 21.2 Basic steps to gain vascular access for angiography.

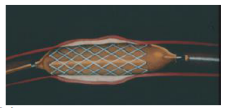


22 Angiography III: Angiointervention

Figure 22.1 Various stents and their deployment: (a) stent deployed within vessel (b) balloon expandable stent (c) self-expanding stent partially unhoused for deployment.



(a) Diagrammatic representation of stent partially deployed from its housing catheter within vessel



(b) Diagrammatic representation of balloon-expandable stent being deployed within vessel

(c) Stent partially deployed from its housing catheter

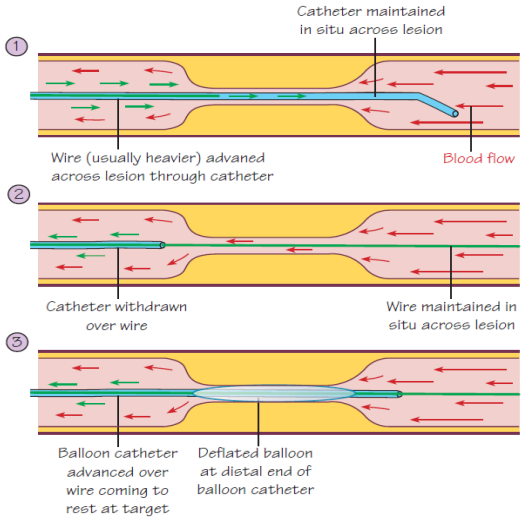
Vascular balloon characteristics

- ☐ Size
- ☐ Profile, pushability, tackability
- ☐ Compliance
- ☐ Cutting vs. non-cutting
- ☐ OTW vs. monorail techniques

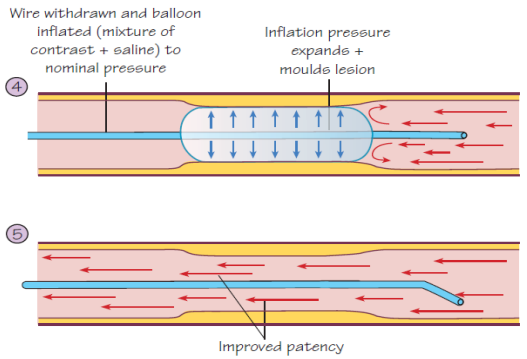
Vascular stent characteristics

- ☐ Size
- ☐ Balloon expandable vs. self-expanding
- ☐ Metal framework (stainless steel, nitinol)
- ☐ Covered vs. uncovered
- ☐ BMS vs. DES

Figure 22.2 Steps in performing balloon angioplasty.

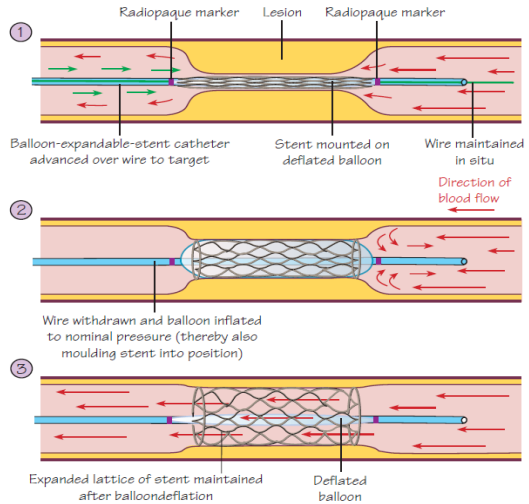


(Flouroscopy + road mapping used to identify where lesion is and thus where balloon target is)



Wire is advanced again through balloon catheter and balloon catheter withdrawn. A flush catheter is again advanced across lesion, wire withdrawn and a post-procedure image taken. If satisfactory result, the wire is advanced again and the catheter removed 'over-the-wire' to minimise intimal injury.

Figure 22.3 Steps in deployment of balloon expandable stents.



Catheter + devices removed over the wire as per angioplasty and post-procedure image taken

Abbreviations: BMS, bare metal stents; DES, drug eluting stents; OTW, over the wire

34 Peripheral vascular disease I: Overview

Figure 34.1 Illustration of the pattern of intermittent claudication on history taking: ischaemic muscle pain coming on with exercise and relieved with rest and reproducible at the same claudication distance.

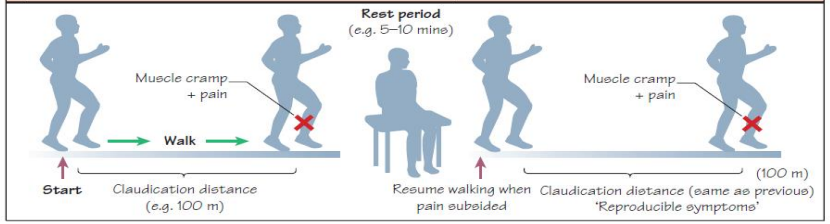


Figure 34.2 Scope of clinical presentations of critical and subcritical limb ischaemia.

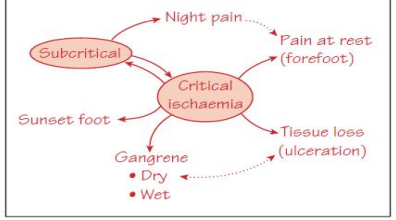


Figure 34.3 Lower limb arterial anatomy.

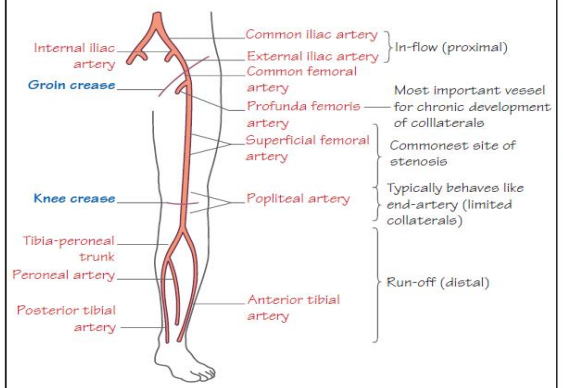


Table 34.1 Fontaine classification of severity of PAD.

Fontaine stage	Clinical description	Definition	Typical ABPI
I	Asymptomatic	Incidental finding with no symptoms	0.9–1.2
II	Intermittent claudication	Cramp-like pain in muscle brought on by exercise and relieved with rest (IIa=mild, IIb=moderate to severe)	0.5–0.7
III	Rest pain	Constant pain in forefoot (worse at night)	<0.4
IV	Tissue loss	Arterial ulceration/gangrene (wet/dry)	<0.3

Table 34.2 Rutherford classification of PAD.

Category	Clinical	Objective findings	Fontaine equivalent
0	Asymptomatic	Normal treadmill test	I
1	Mild claudication	Symptoms on treadmill, but AP>50 mmHg at end (but <20 mmHg compared to resting AP)	IIa
2	Moderate claudication	(between 1 and 3)	IIa–IIb
3	Severe claudication	Cannot complete treadmill test/ AP<50 mmHg at end	IIb
4	Rest pain	Resting AP<40 mmHg/TP<30 mmHG	III
5	Minor tissue loss	Resting AP<60 mmHg/TP<40 mmHG	IV
6	Major tissue loss/ gangrene	Resting AP<60 mmHg/TP<40 mmHg	IV

Figure 34.4 'Sunset foot' with characteristic rubor of cold, ischaemic forefoot. Note small patch of dry gangrene at tip of great toe.

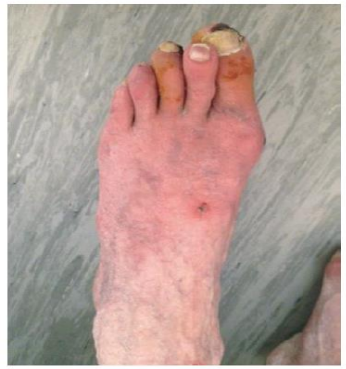


Figure 34.5 Trash foot. Foot is viable, but note skin discolouration (ischaemic skin).

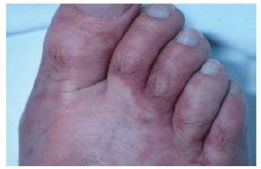


Figure 34.6 Dry gangrene of left foot requiring forefoot amputation and restoration of perfusion.

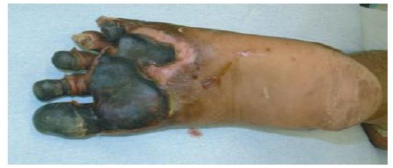


Figure 34.7 Pressure ulceration over medial aspect of first metatarsal head in a neuropathic foot (diabetic).

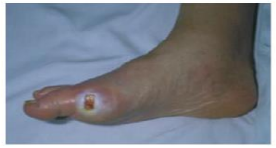


Figure 34.8 Severe ischaemic ulceration of heels (pressure areas) with superimposed infection.