جامعة الملك سعود
King Saud University

Management of atherosclerotic disease (PAD, carotid stenosis) & Acute Limb Ischemia

SURG 351

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Outline

- **Pathophysiology of atherosclerosis**
- Describe the etiology, clinical features, investigations & management of **chronic LL ischemia**
- Explain the differentiating features & significance of **critical limb ischemia**
- Describe etiology, presenting features & management of **acute limb ischemia**
- **Reperfusion injury** & its management
- Pathogenesis & management of **Diabetic foot**
- Describe the etiology, clinical features, investigations & management of **Carotid artery atherosclerotic disease**

The Arterial system

- Contains 30% of blood volume
- Normal systolic pressure <130 mmHg
- Arterial capillary pressure 25 mmHg
- High pressure /low volume system

The Arterial system

- **Elastic arteries**: Aorta & Beginning of its large branches have preponderance of elastic fibers in media

- **Muscular arteries**: medium sized arteries, distributing arteries exhibit smooth muscles in their walls

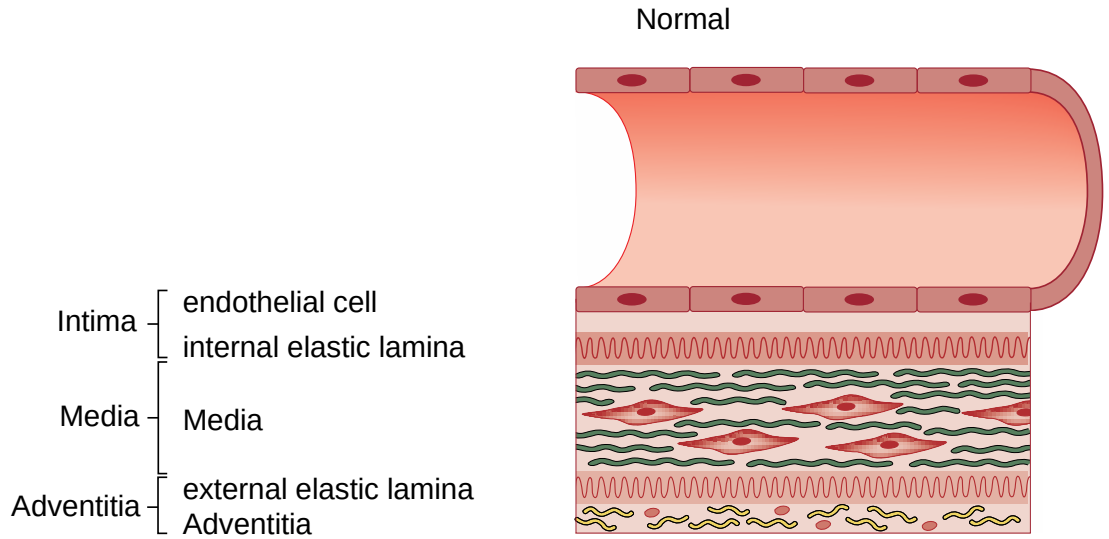
- **Small arteries**: major site of autonomic regulation of blood flow

Risk Factors

- **Non Modifiable**
  - Age
  - Male gender
  - Menopause
  - Familial predisposition
  - Genetic
- **Modifiable – Major Factors**
  - Smoking
  - HTN
  - DM
  - Dyslipidemia
- **Other risk factors**
  - Sedentary life style
  - Obesity
  - Elevated homocysteine
  - Stressfull & competitive life style
  - Type A personality
  - High carbohydrate intake

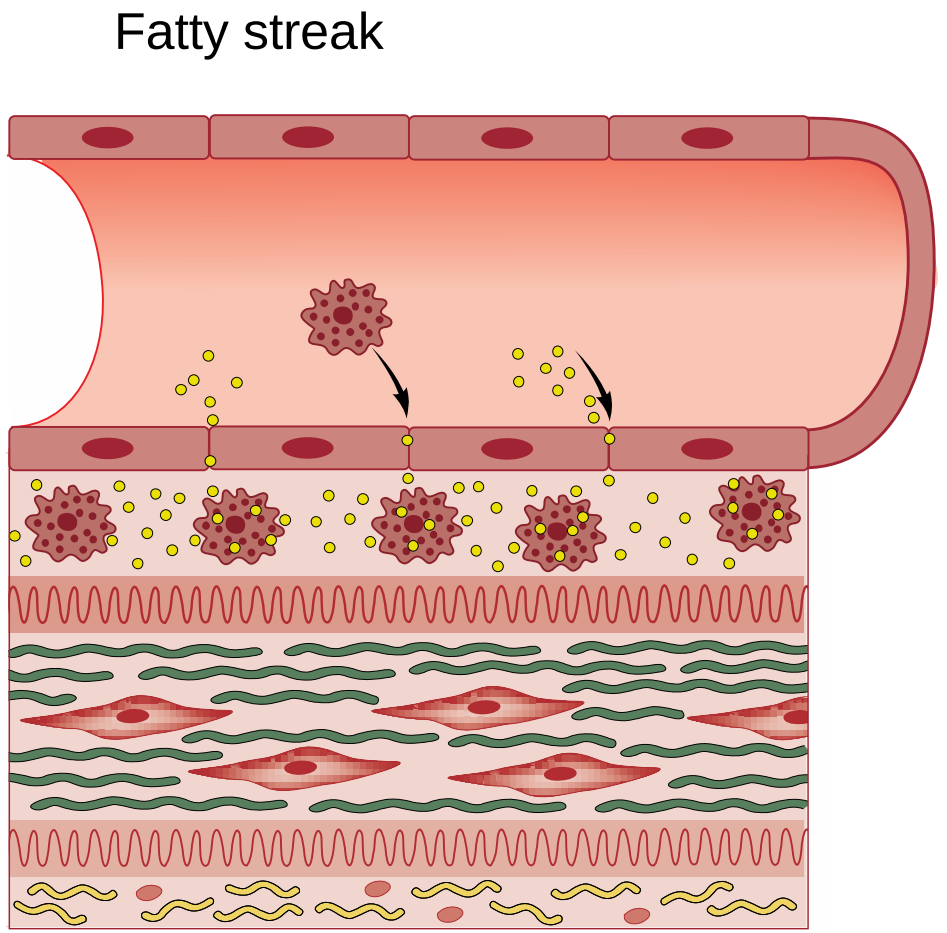
Pathophysiology of atherosclerosis

- Endothelial injury
  - Chemical
  - Physical
  - Atheroma
- Hypertension, increases this stress



Pathophysiology of atherosclerosis

- Increased permeability to lipids and inflammatory cells
- Leucocytes adhere into the subendothelial space and digest lipids to become foam cells
- Protease and free radicals liberated
- Cytokines attract leucocytes and smooth muscle cells



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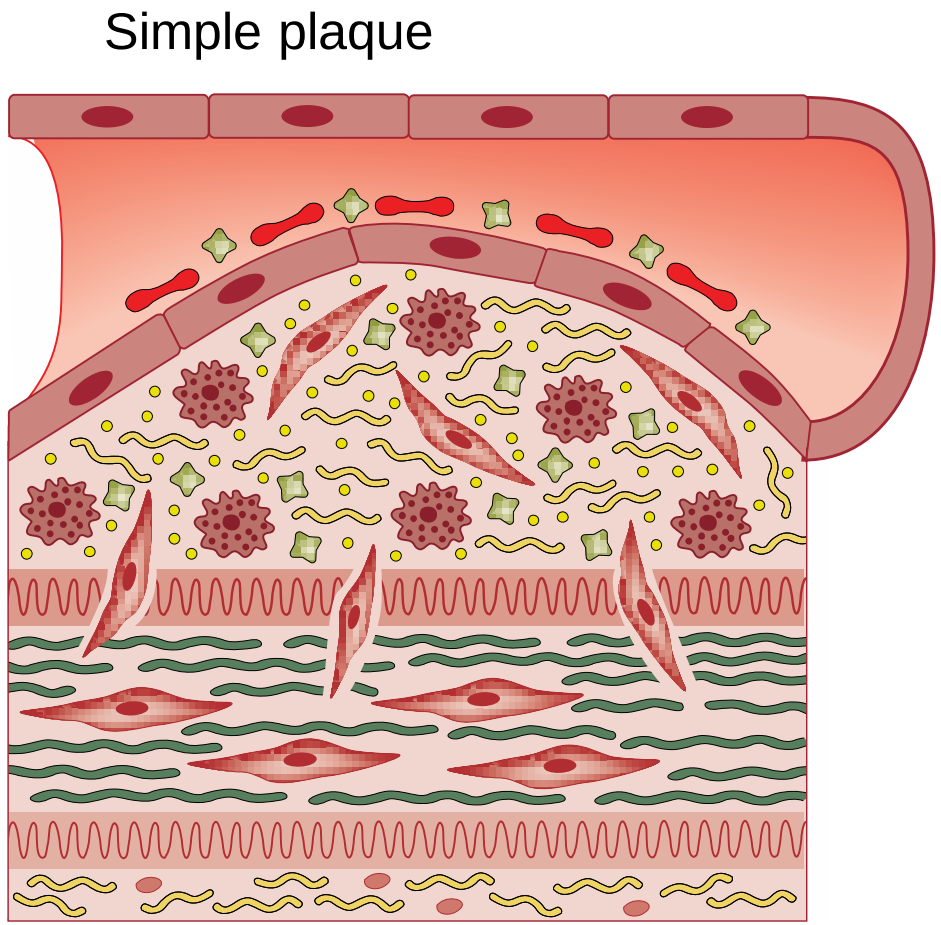
Pathophysiology of atherosclerosis

- Smooth muscle cells exit the media

- **Proliferate**, take on the characteristics of fibroblasts and produce collagen, raising the atheroma



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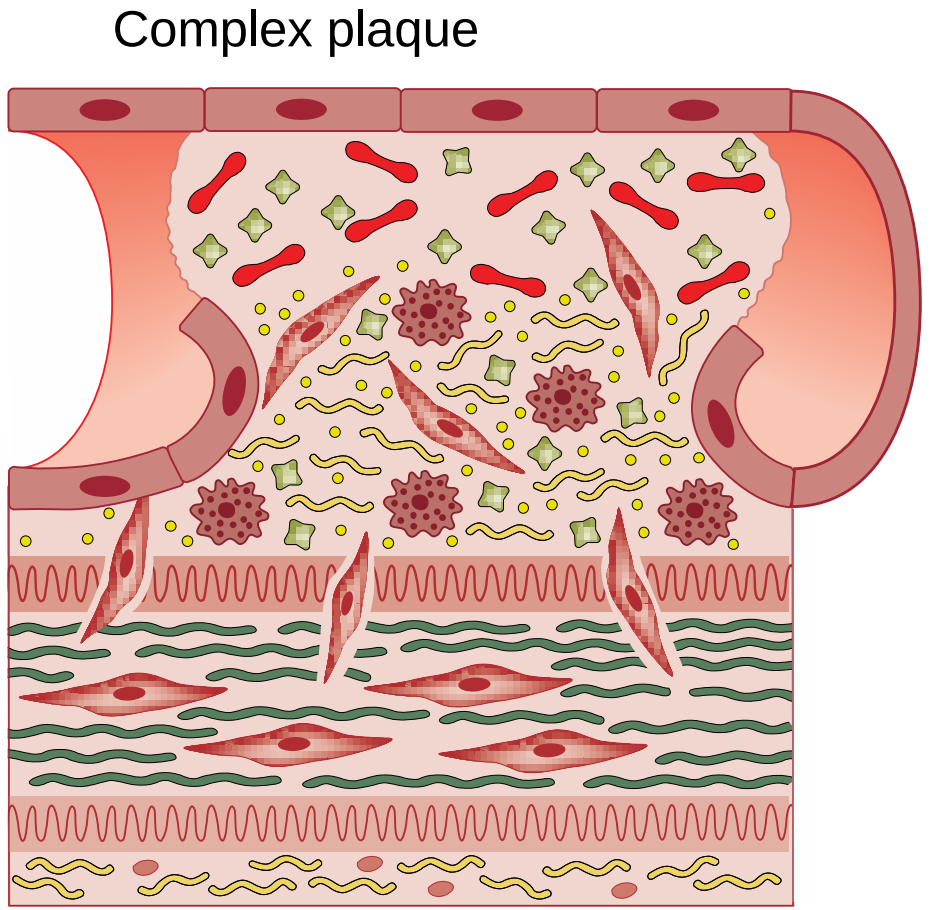
Pathophysiology of atherosclerosis

- Proliferation forms an endothelial cap, which may rupture, ensuing further endothelial injury

- This results in thrombosis and distal embolization



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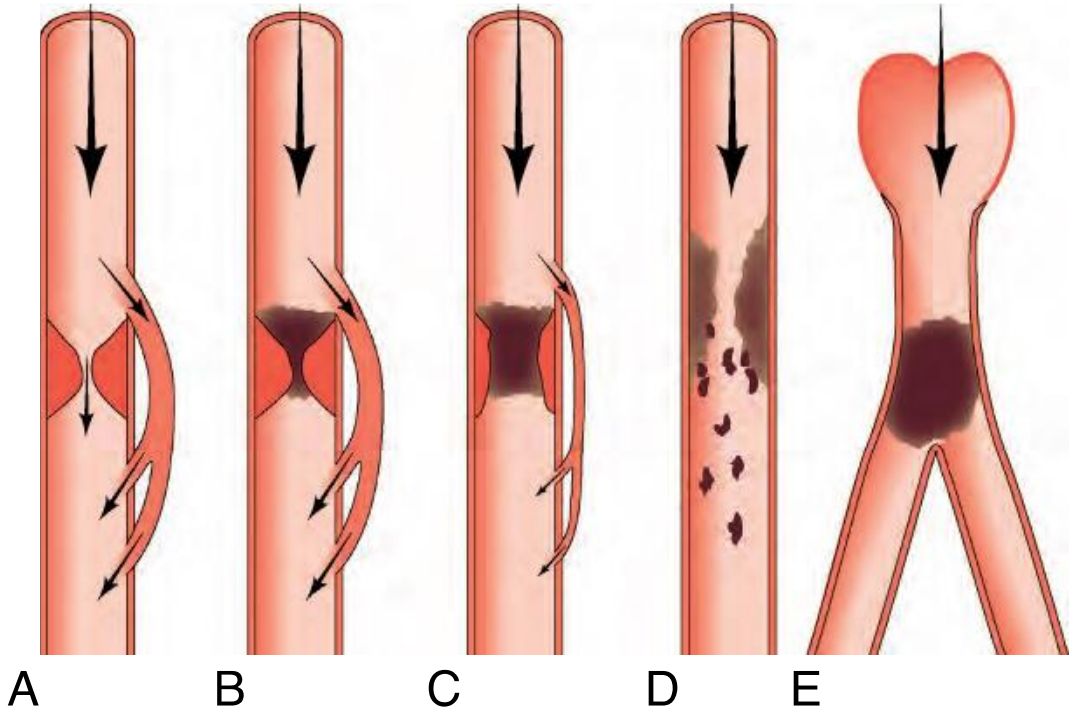
Mechanisms of injury in atherosclerotic disease

A. Critical stenosis compensated by collateral vessels
   - symptomatic on exercise

B. Acute thrombosis of a critical stenosis
   - little change in symptoms due to collateral development

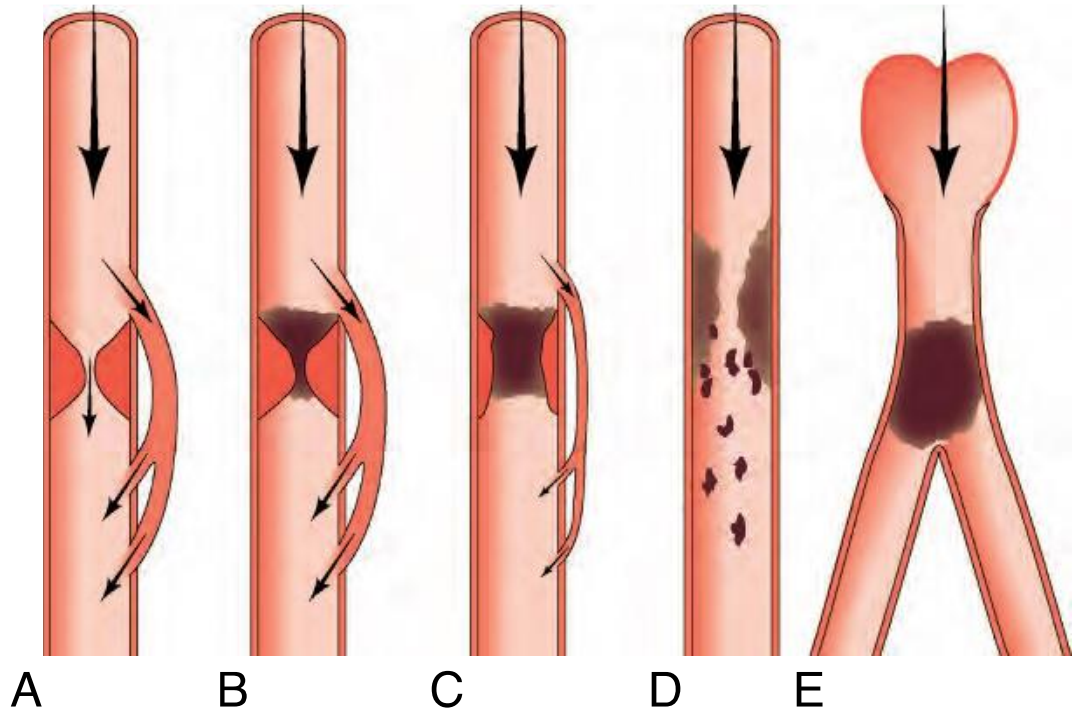


Mechanisms of injury in atherosclerotic disease

C. Acute thrombosis of noncritical stenosis
   - Severe symptoms due to poorly developed collaterals

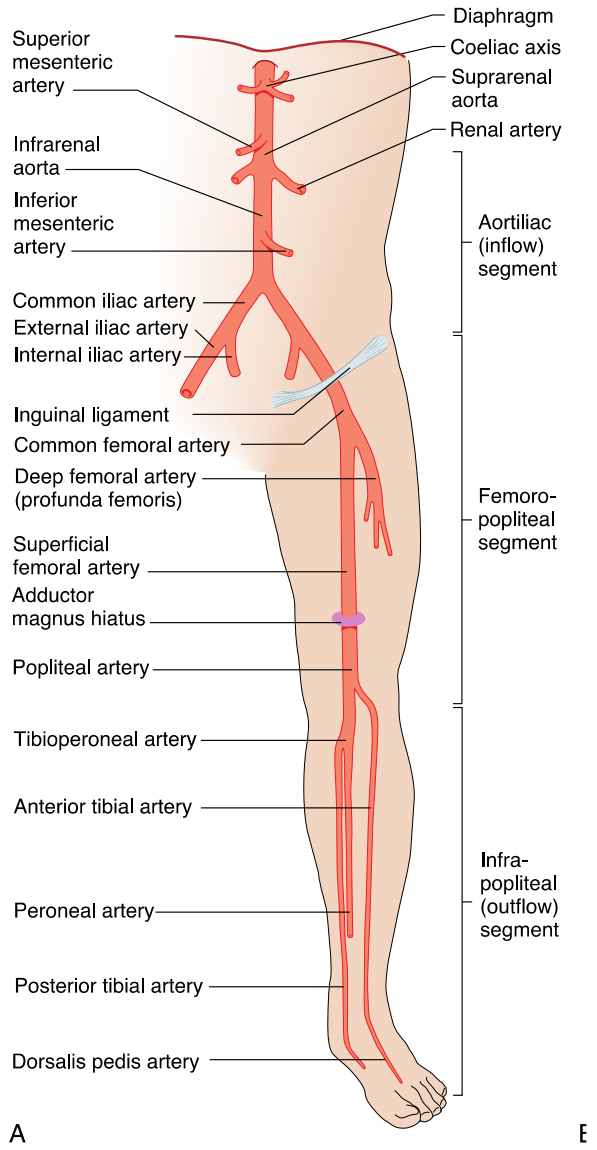
D. Atheroembolism from ruptured plaque

E. Thromboembolism
   - severe ischemia because of lack of collateral supply



- The aortoiliac segment above the inguinal ligament **(inflow)**

- The femoropopliteal segment and the infrapopliteal segment **(outflow)**



Peripheral Arterial Disease (PAD)

- **Chronic**
  - Slow gradual luminal stenosis secondary to plaque
  - Collateral development compensate
  - Symptoms proportional to disease burden
  - Exertional symptoms appear first
- **Intermittent Claudication (IC)**
- **Critical limb ischemia (CLI)**
- **Diabetic foot (DF)**

- **Acute**
  - Sudden occlusion in the absence of adequate collaterals
  - **Embolism**
  - **Thrombosis**
  - **Injury**
- **Acute Limb Ischemia (ALI)**

IC Epidemiology

- Up to 5% of people >60 years
- 1–2% of patients will deteriorate if they comply with best medical treatment (BMT)
- The annual mortality rate is 5–10% per year,
  - 2–3-times higher than non-claudicant
  - Marker of atherosclerosis, and most of these patients succumb to myocardial infarction (MI), stroke and limb loss
- The emphasis is on the preservation of life.

IC Clinical features

- Claudication pain is a muscular pain
  - **Not present at rest**
  - Comes on walking a particular distance, which is known as the **claudication distance**
  - It is quickly **relieved by resting**
  - It is **repetitive**; the patient will develop the pain after walking the claudication distance

IC Clinical features

- The site of claudication gives a clue to site of arterial disease:
  - **Bilateral thigh and/or buttock: aortoiliac arteries**
  - **Unilateral thigh and/or buttock: iliac arteries**
  - **Calf: femoropopliteal arteries**
  - **Foot (instep): tibial arteries**

IC Clinical features

- Impotence
- Weakness / decreased mobility
- Skin changes
- Toe nail changes
- Muscle wasting

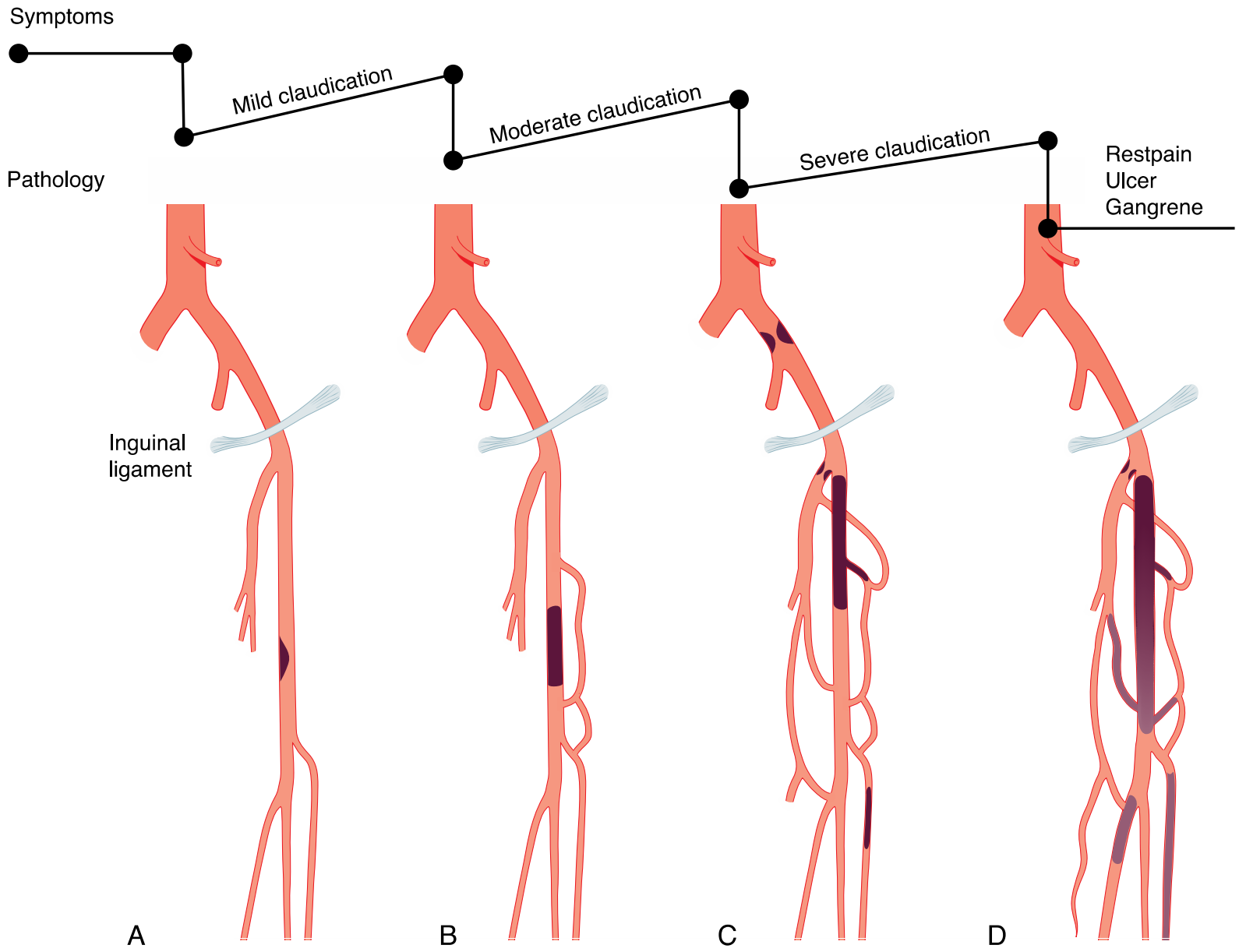


Fig. 21.5 Symptoms and pathology in intermittent claudication. **(A)** Superficial femoral artery (SFA) stenosis at adductor canal. **(B)** Occlusion of the SFA and development of a collateral circulation between the deep femoral (profunda femoris) artery (PFA) and the popliteal artery. **(C)** Iliac artery and PFA stenosis leading to worsening symptoms of intermittent claudication and further collateralisation. **(D)** Eventually critical limb ischaemia characterised by ischaemic rest pain and tissue loss develops due to multilevel disease affecting tibial arteries and collateral supply.

Critical Limb Ischemia (CLI)

- Caused by multiple lesions affecting different arterial segments in the affected limb
  - **Rest pain**
  - **Tissue loss** in the form of an ulcer or gangrene

Critical Limb Ischemia (CLI)

- **Rest pain**
- Exacerbated by lying down or elevation of the foot
- Classically felt at night and is relieved by sleeping with feet hanging over the bed or sleeping on a chair
- The patient may present with foot swelling

CLI Examination findings

- Skin is thin and dry
- Pallor, particularly on elevation
- Brittle Nails
- Muscle wasting
- Reduced temperature

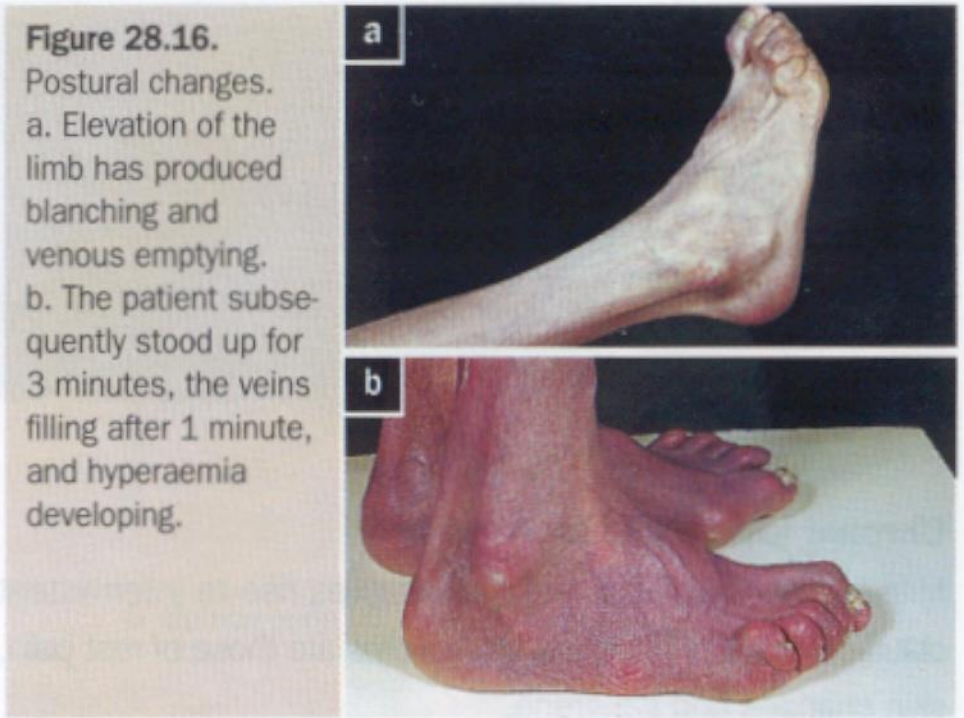


Figure 28.16. Postural changes. a. Elevation of the limb has produced blanching and venous emptying. b. The patient subsequently stood up for 3 minutes, the veins filling after 1 minute, and hyperaemia developing.

CLI Examination findings

- Pallor, particularly on elevation

- Upon dependency, the foot becomes bright red; this is known as dependent rubor or ‘sunset foot’, and is due to **reactive hyperaemia (Buerger’s test)**

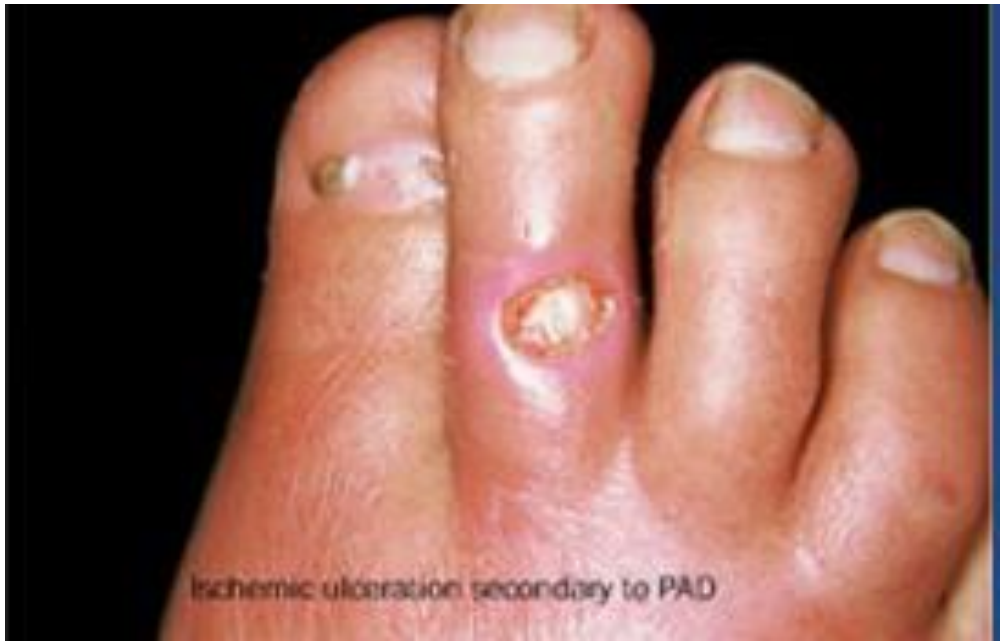
- Superficial veins that fill sluggishly in the horizontal position and empty upon minimal elevation **(venous guttering)**

CLI Examination findings

- All patients must have their pulse status recorded
  - This includes, carotid, subclavian, brachial, radial, ulnar, femoral, popliteal, posterior tibial and dorsalis pedis
- The pulses are recorded as normal, weak or absent
- The presence of a thrill and/or bruit denotes turbulent flow
- Ankle/brachial pressure index should be recorded

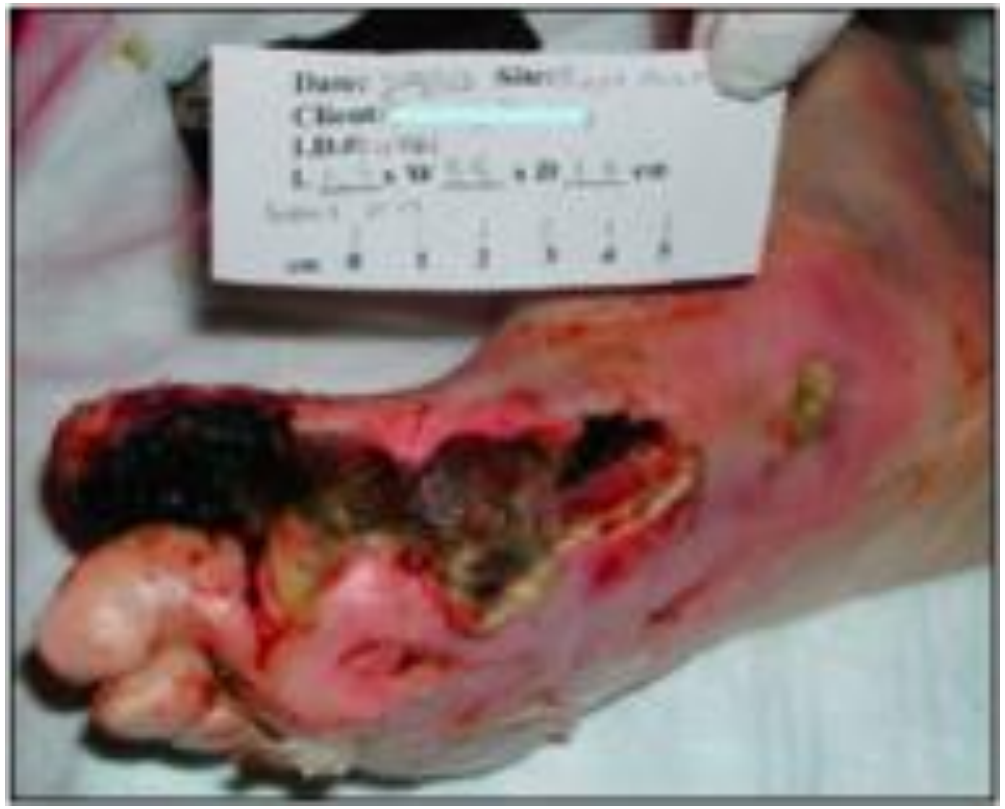
Tissue Loss

- Arterial Ulcers
  - Often located on toes or foot
  - Pale and with necrotic floor,
  - Irregular margins
  - Painful
  - Surrounding ischemic features



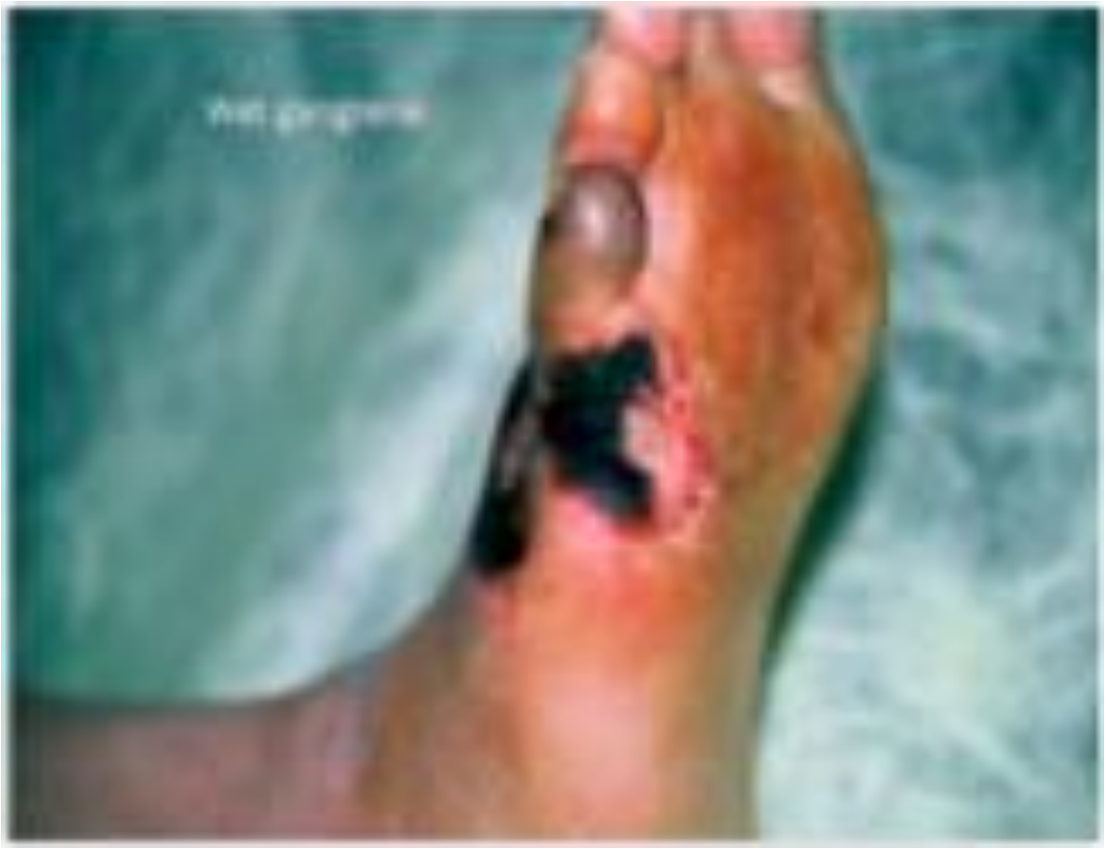
Ulcer Examination

- **Inspection**
- Site (location)
- Number
- Size
- Shape
- Floor - The exposed part of an ulcer (Inspection)
- Edges – Part between the margin and the floor of an ulcer (Undermined, Punched out, Sloping, Rolled, Raised)
- Depth
- Exudate (Discharge)
- Surrounding area
- Margin- Line of demarcation between normal and abnormal



Ulcer Examination

- **Palpation**
- Tenderness
- Base- the structure on which the ulcer rests (Felt on palpation)
- Relation with Deeper structures



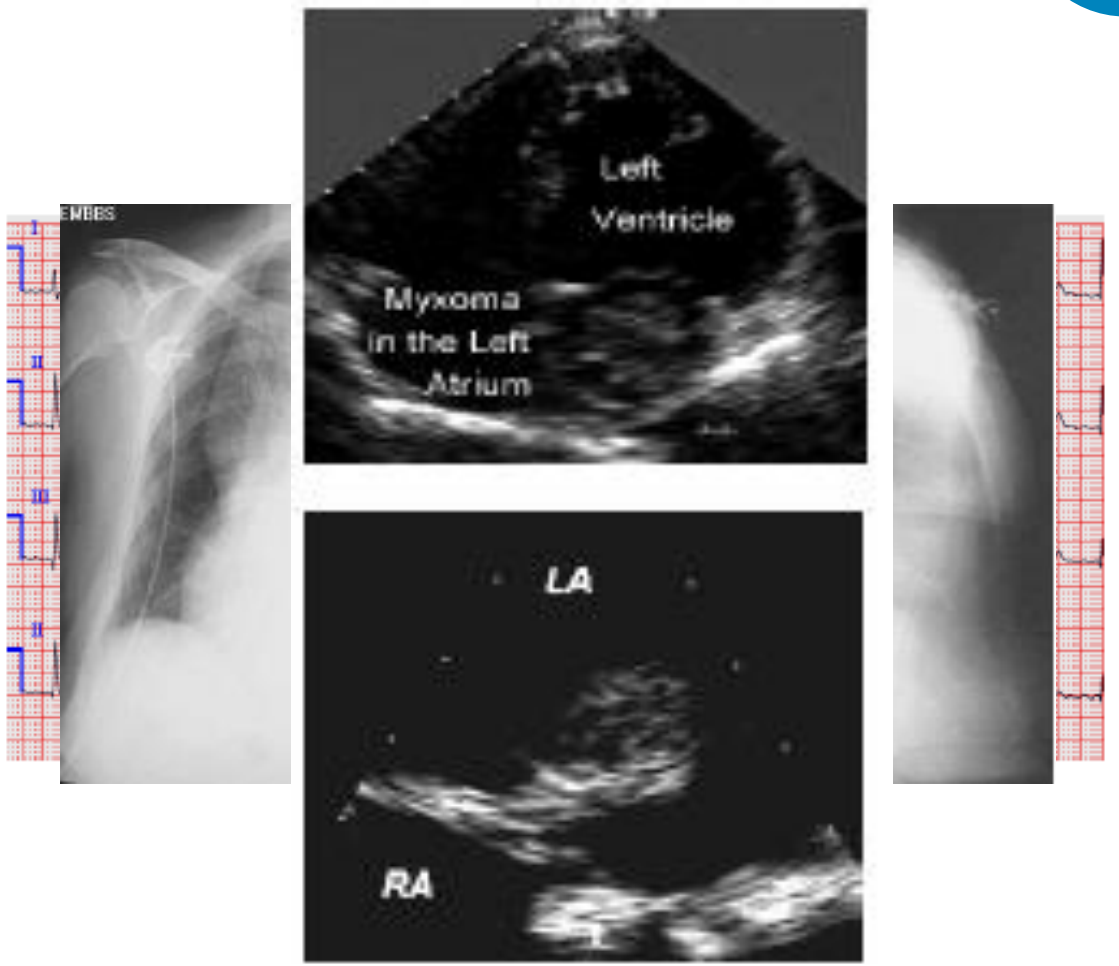
- **Examination of Surrounding Area**
- **Examination of Lymph Nodes**
- **Examination of the pulse**

Investigations

- CBC, Electrolytes, creatinine, coagulation profile (aPTT, INR)
- Type and screen

- Lipid profile
- **Hemoglobin A1c**

- ECG
- Chest Xray
- Echocardiogram

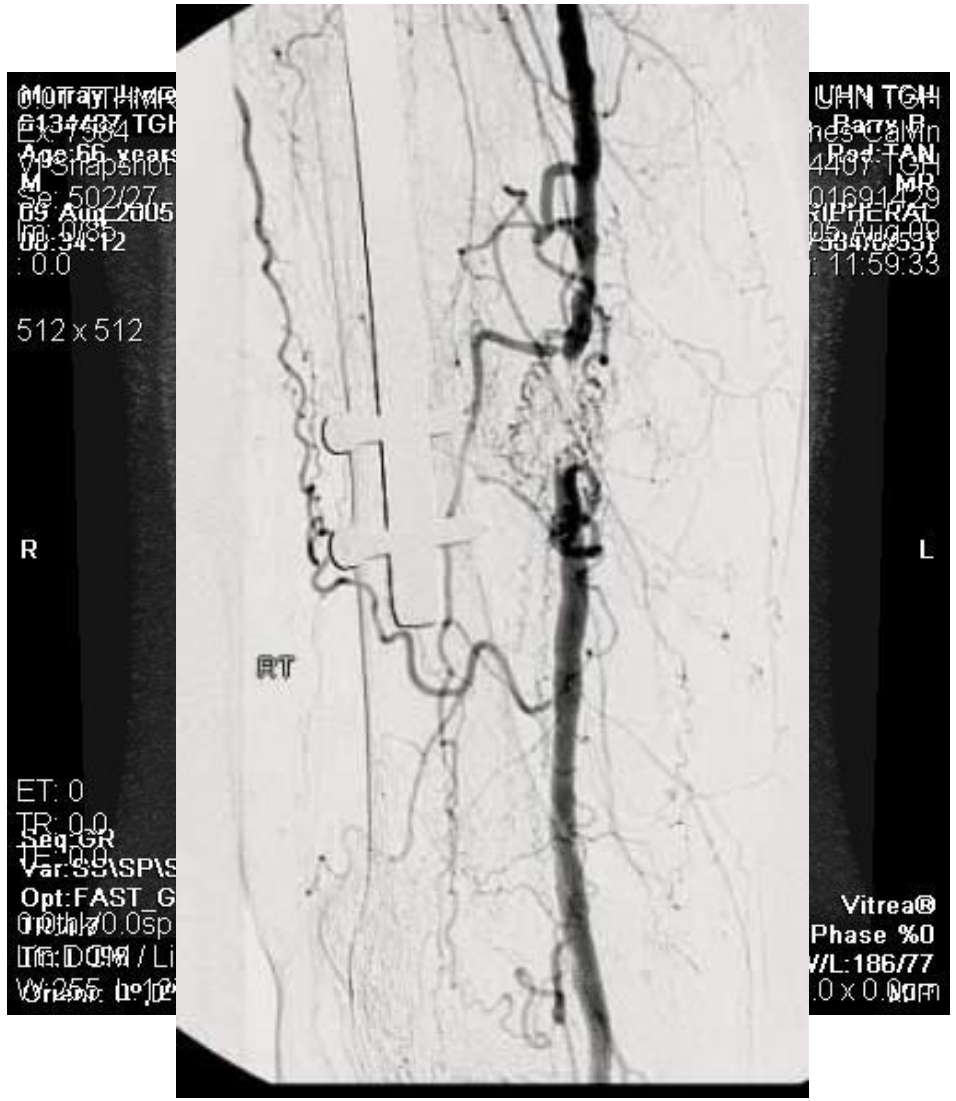


IC Investigations

- **Ankle Brachial Index (ABI)**
  - 0.8-0.4
- **Toe pressures**
  - <50 mmHg
- **Segmental pressure**
  - 20 mmHg reduction
- **Volume Plethysmography**
  - Measures arterial volume changes

- **Duplex Ultrasound**
  - Stenosis or single occlusion
- **CT Angiogram**
- **MRA**
- **Invasive Vascular Investigations**

CLI Investigations



- **Duplex Ultrasound**
  - Multiple Stenoses or occlusions
- **CT Angiogram**
- **MRA**
- **Invasive Vascular Investigations**

PAD Prevention

- Primary Prevention
  - Modifiable risk factors – lifestyle changes
- Secondary prevention
  - Best medical treatment
- Treatment
  - Endarterectomy
  - Percutaneous Angioplasty
  - Bypass procedures

PAD Medical Management

- All patients should be strongly urged to comply with Best Medical Therapy (BMT):

  - Cessation from **smoking (most important)**
  - Control of **hypertension**, (ACE Inhibitors)
  - Prescription of a **statin** despite the absence of dyslipidemia
  - Prescription of **antiplatelet agent**: aspirin (81 mg daily), or clopidogrel (75 mg daily)
  - Regular **exercise**
  - Control of **obesity**
  - The identification and treatment of patients with **diabetes (Hb A1c <7%)**

PAD Medical Management

- Compliance with BMT increases
  - Walking distance
  - Affords protection against cardiovascular events
  - Improves the quality of life and life expectancy
  - BMT reduces the overall intervention risks and increases the likely success
- Many patients fail to comply

Endovascular and Surgical Interventions

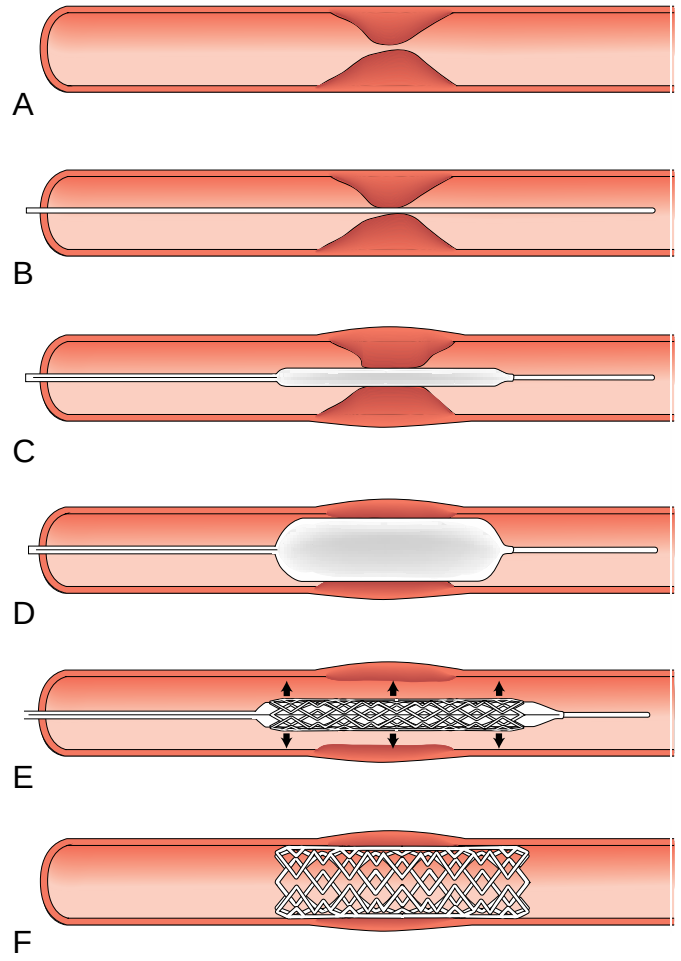
- Indications for intervention are:
  - Disabling claudication pain
  - CLI

- Intervention includes
  - Balloon angioplasty, with or without stenting
  - Surgery

Endovascular Intervention

- The lesion is identified
- Crossed with a wire
- A balloon is inserted and inflated
- This enlarges the lumen by disrupting the plaque

- In patients with occlusions and complex disease, stents may be deployed

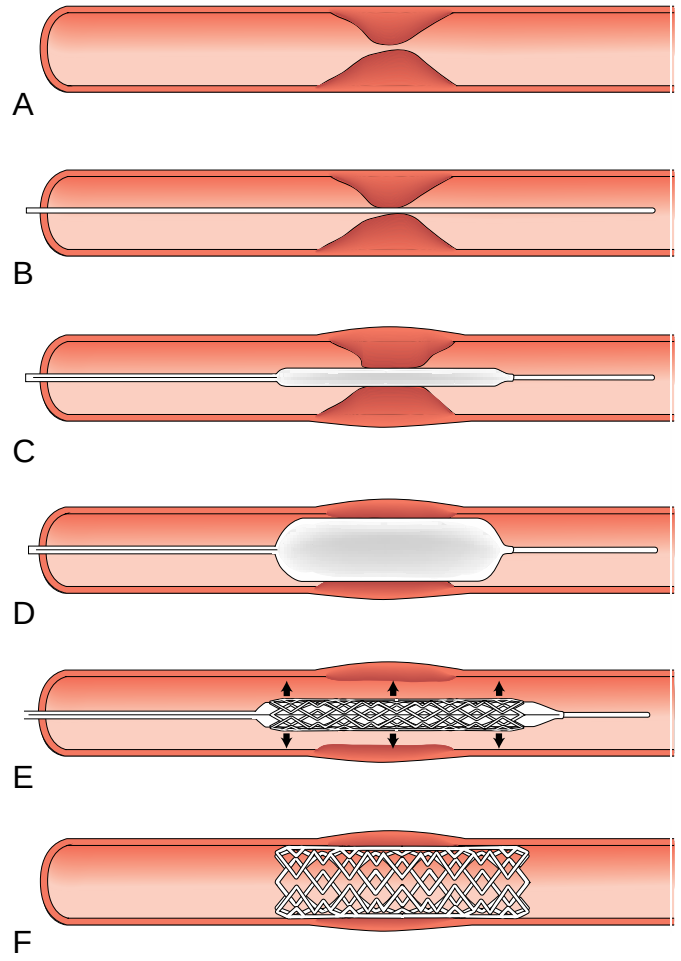


Endovascular Intervention

- Drug-eluting balloons and stents reduce the neointimal hyperplasia that can lead to restenosis and occlusion

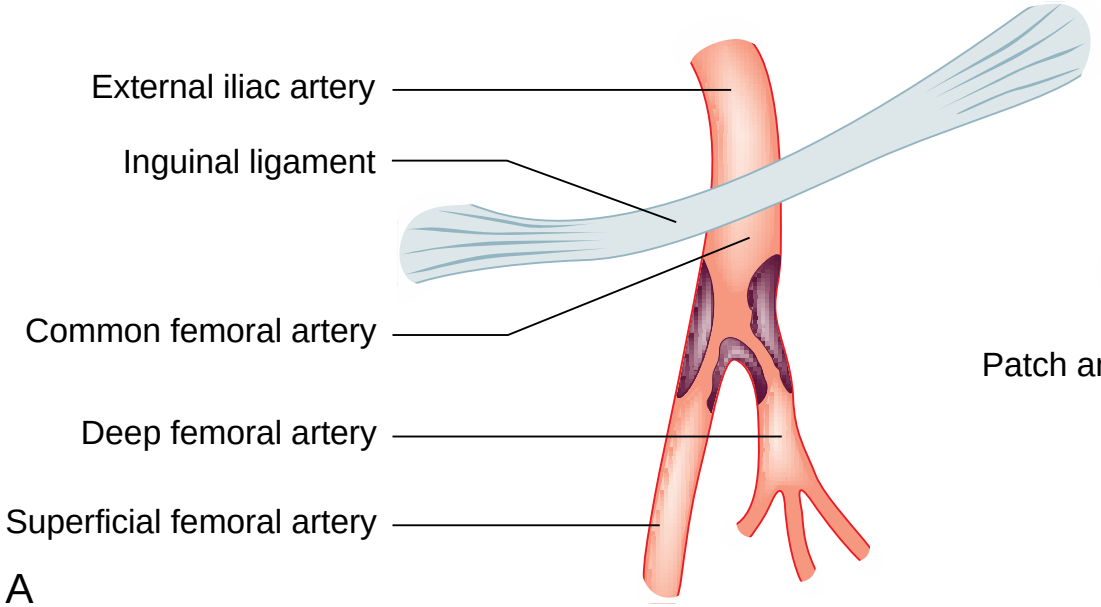
- Favorable lesions - short concentric stenosis

- Unfavorable lesions- long eccentric stenosis or occlusion



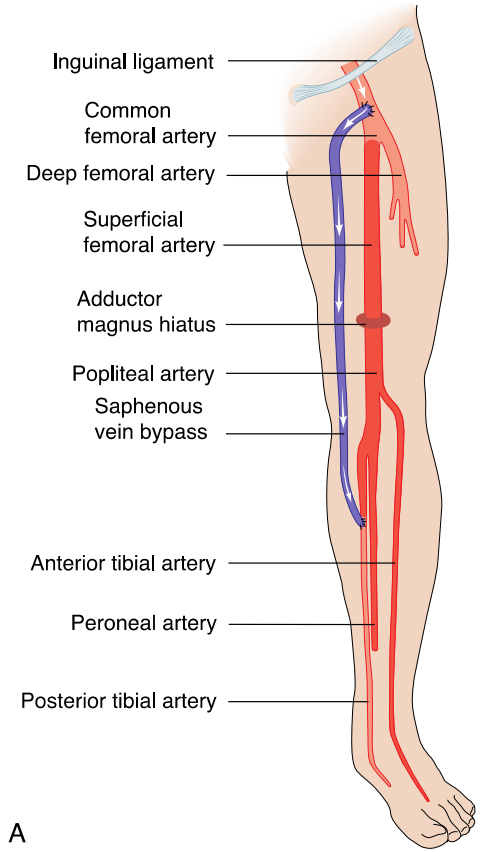
Surgery

- **Endarterectomy**
- Direct removal of atherosclerotic plaque and thrombus, usually done at the **carotid and femoral** bifurcations



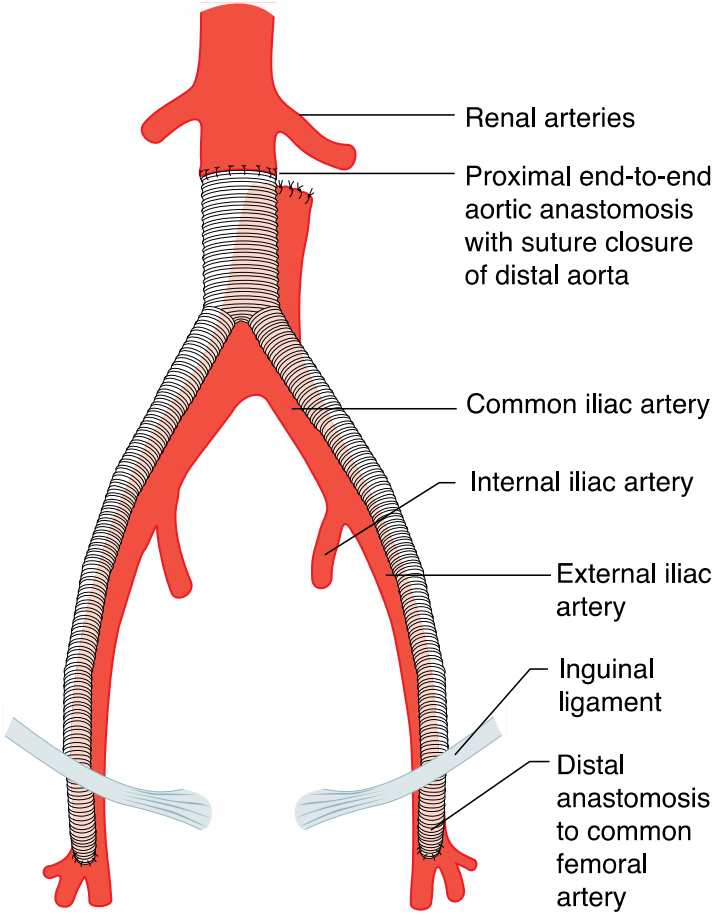
Bypass Grafting

- For a bypass operation to be successful in the long term, three conditions must be fulfilled:
- There must be high-flow, high-pressure blood entering the graft **(inflow)**
- The **conduit** must be suitable
- The blood must have somewhere to go when it leaves the graft **(outflow or run-off)**



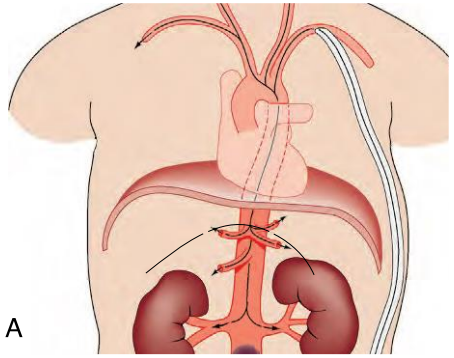
Bypass Grafting

- Two main types of conduit are available:
- **Autogenous material**, most commonly a vein
- **Prosthetic material**, expanded polytetrafluoroethylene (ePTFE) or Dacron



Bypass Grafting

- **Extraanatomic bypass**
- Lesser procedures, and preferred in high-risk patients or those that have a limited life expectancy
  - Fem-Fem crossover for patients with an occluded iliac
  - Axillobifemoral if both iliac arteries are occluded
- Do not have as good long-term patency as anatomic



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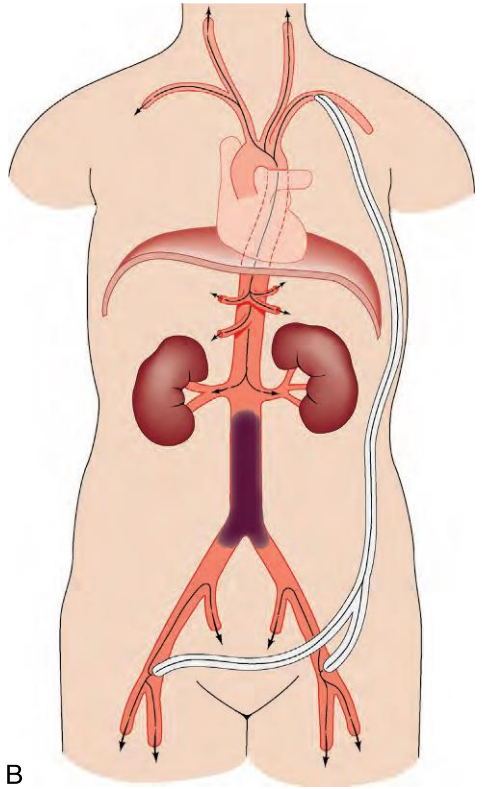

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Choice of treatment

- Patients symptoms
- Comorbidities
- Life expectancy
- Risk and benefits
- Anatomy of the disease
- Prior interventions

Diabetic Foot

- Approximately 40% of patients with CLI have diabetes
- Combination of **ischemia, neuropathy and immunocompromise pt**
- **Arteries are often calcified**
- Severe multisystem arterial disease (CAD, CVD and PAD)

Diabetic Foot

- The feet of diabetic patients are very susceptible to sepsis, ulceration and gangrene

- Diabetic vascular disease has a tendency for the infrapopliteal vessels

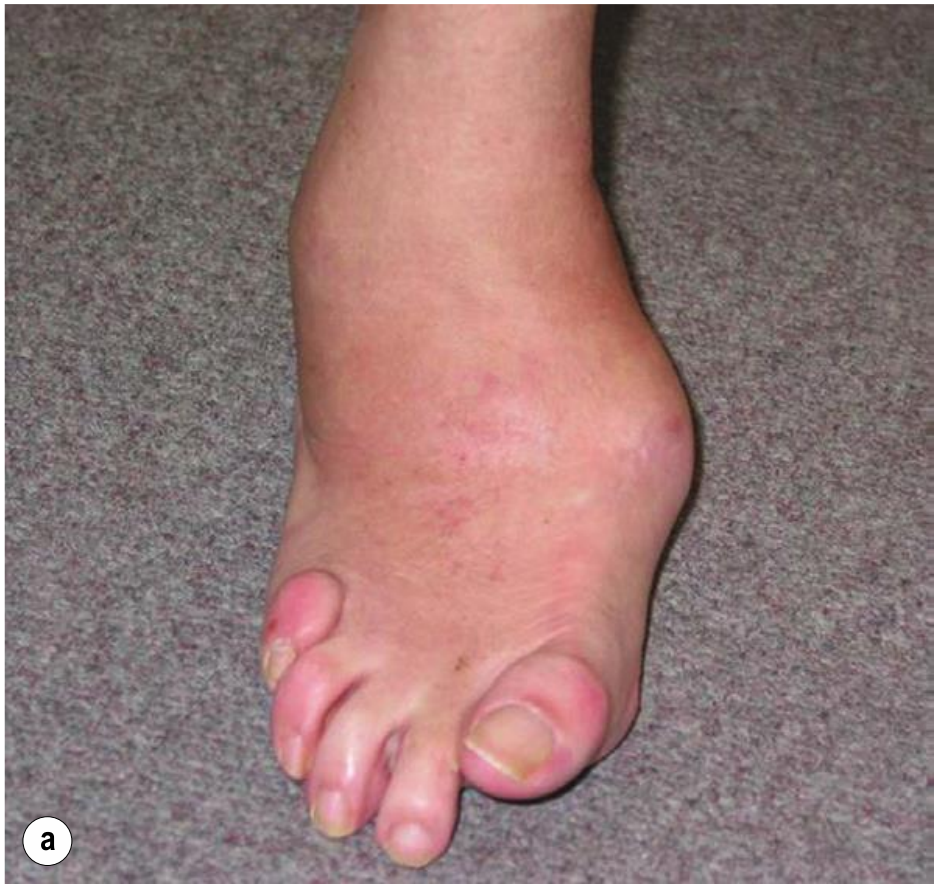
Diabetic Foot

- **Diabetic neuropathy affects the motor, sensory and autonomic nerves**

- Diabetic neuropathy may lead to foot ulceration in its own, and also complicates peripheral ischemia

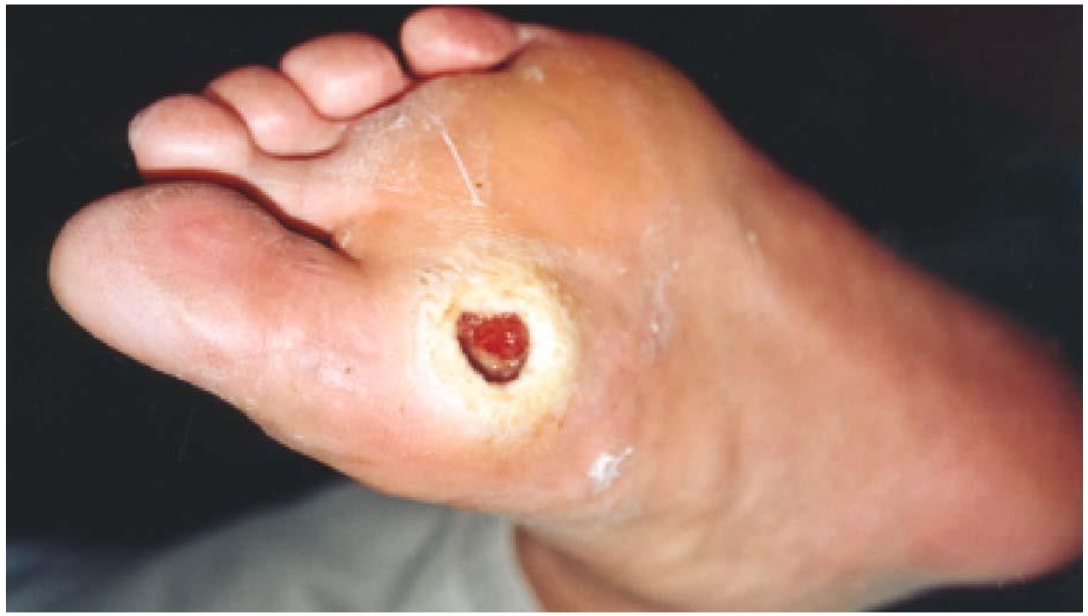
Sensory Neuropathy

- Patient incapable of feeling pain
- It affects proprioception such that, when walking, pressure is applied at unusual sites
- This leads to ulcer formation and joint destruction (Charcot's Foot)



Motor Neuropathy

- The flexors are affected more than the extensors
- The extensors are unopposed and the toes become dorsiflexed
- This exposes the metatarsal heads to abnormal pressure, and they are a frequent site of callus formation and ulceration



Autonomic Neuropathy

- Dry foot deficient in the sweat that normally lubricates the skin and contains antibacterial substances
- Causing scaling and fissuring
- Abnormal flow in the bones due to loss of autonomic control may also contribute to osteopenia and bony collapse

DF

- History
- Physical exam
- Investigations
- Similar to PAD

DF Prevention

- **Diabetic control (Hb A1c <7%)**

- **Comprehensive behavioral foot care education**

  - **Washing** the feet with soap daily and dry it thoroughly
  - **Use a file** to shape the nails (not a clipper)
  - Keep the **skin moisturized**
  - **Don’t walk barefoot**
  - Change daily into **clean soft socks**
  - **Daily foot inspection** for injuries
  - Therapeutic **foot wear**

DF Management

- **If the blood supply to the foot is adequate**
  - Excise dead tissue
  - Control the Infection
  - Protected the foot from pressure (off-loading)

- **If there is ischemia, the priority is to revascularize the foot, if possible**

- Many patients present late, with extensive tissue loss and un-reconstructable disease accounting for the very **high amputation rate**

Acute Limb Ischemia (ALI)

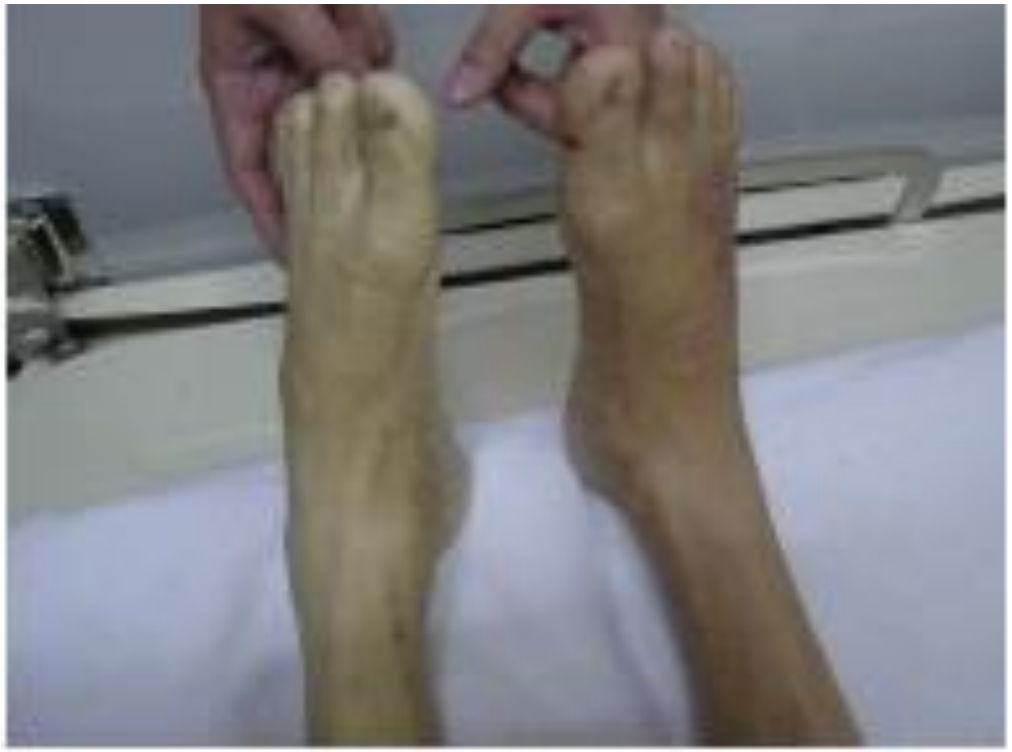
- **Etiology**
- Acute limb ischemia is caused most frequently
  - Acute thrombotic occlusion of a preexisting stenotic arterial segment (60%)
  - Embolism (30%)
  - Trauma

- Distinguishing between thrombosis and embolism is important because investigation, treatment and prognosis are different

ALI Etiology

- More than 70% of peripheral emboli are due to AF

- Thrombosis in situ may arise from
  - Acute plaque rupture
  - Hypovolaemia
  - Increased blood coagulability (e.g., in association with sepsis, malignancy)
  - Pump failure (e.g., cardiac event)



ALI Classification

- On the basis of onset and severity

- **Incomplete acute ischemia** (usually due to thrombosis in situ) can often be treated medically, at least in the first instance

- **Complete ischemia** (usually due to embolus) will normally result in extensive irreversible tissue injury within **6 hours** unless the limb is revascularized

- **Irreversible ischemia** mandates early amputation or, if the patient is elderly and unfit, end-of-life care

ALI Clinical features

6 Ps :

- Pain
- Pallor
- Pulselesness
- Paresthesia
- Paralysis
- Poikilothermia

ALI Clinical features

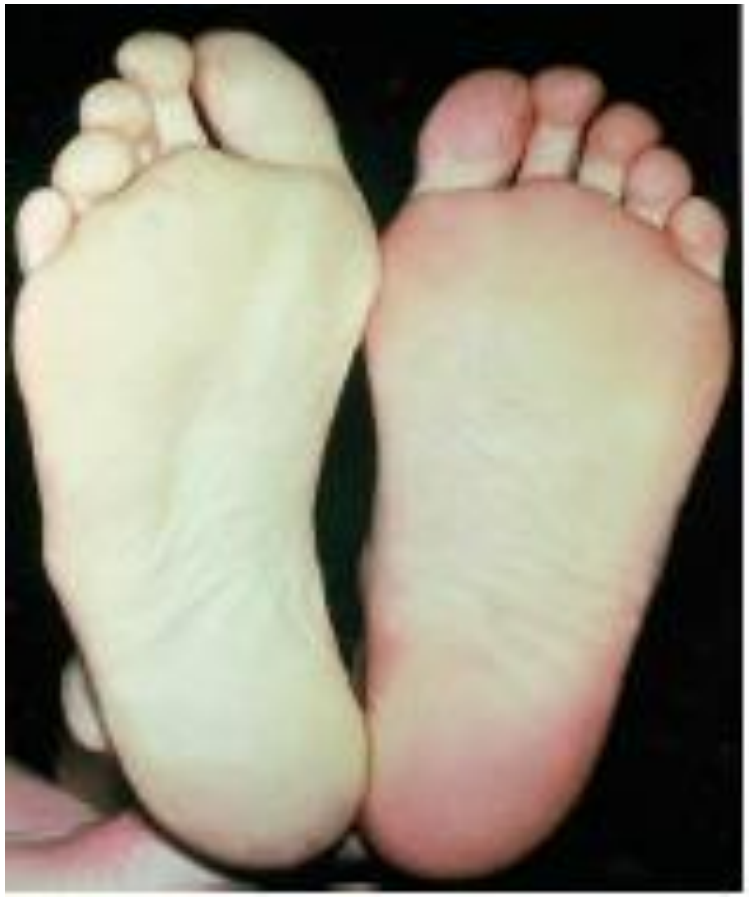
- **Paralysis** (inability to wiggle toes/fingers)
- **Paraesthesia** (loss of light touch over the dorsum of the foot/hand
- **The other Ps assist the diagnosis**

Table 21.6 Symptoms and signs of acute limb ischaemia

Symptoms/ signs	Comment
Pain	May be absent in complete acute ischaemia; severe pain is also a feature of chronic ischaemia
Pallor	Also a feature of chronic ischaemia
Pulseless	Also a feature of chronic ischaemia
Perishing cold	Unreliable, as the ischaemic limb takes on the ambient temperature
Paraesthesia and paralysis	Loss of function is the most important feature of acute limb ischaemia and denotes a threatened limb that is likely to be lost unless it is revascularised within a few hours

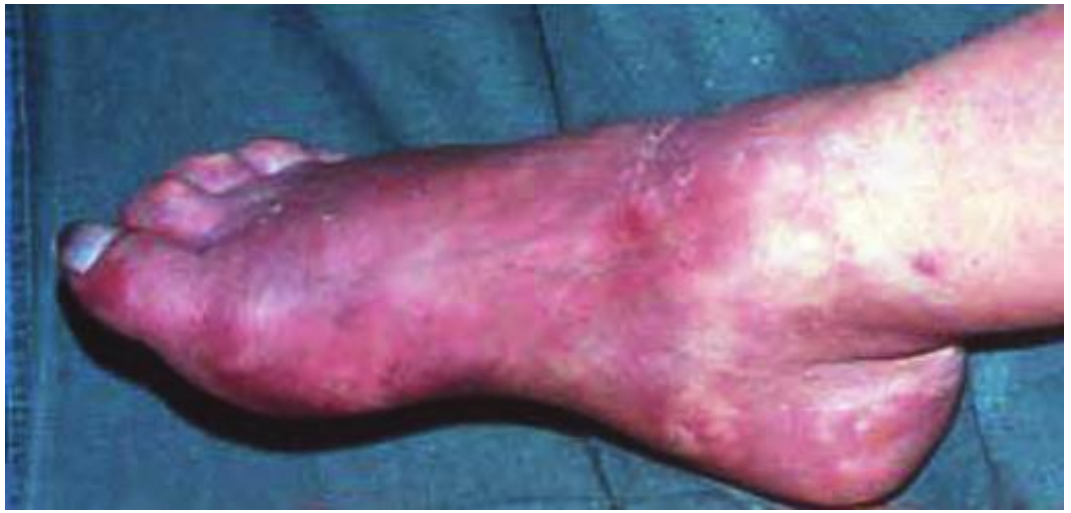
ALI Clinical features

- Acute complete ischemia is associated with intense distal arterial spasm and the limb is ‘marble’ white
- As the spasm relaxes over the next few hours and then fills with deoxygenated blood, mottling appears
- This appears light blue or purple, has a fine reticular pattern, and on pressure, so-called nonfixed mottling
- At this stage, the limb is salvageable



ALI Clinical features

- As ischemia progresses, blood coagulates in the skin, leading to mottling that is darker in colour and does not blanch
- Blistering and liquefaction
- Attempts at revascularisation are futile and will lead to life-threatening reperfusion injury



Management

- Must be discussed immediately with a vascular surgeon
- Blood work, ECG, and cross match
- If there are no contraindications, IV heparin (5000-8000 IU) is administered
  - to limit propagation of thrombus and protect the collaterals

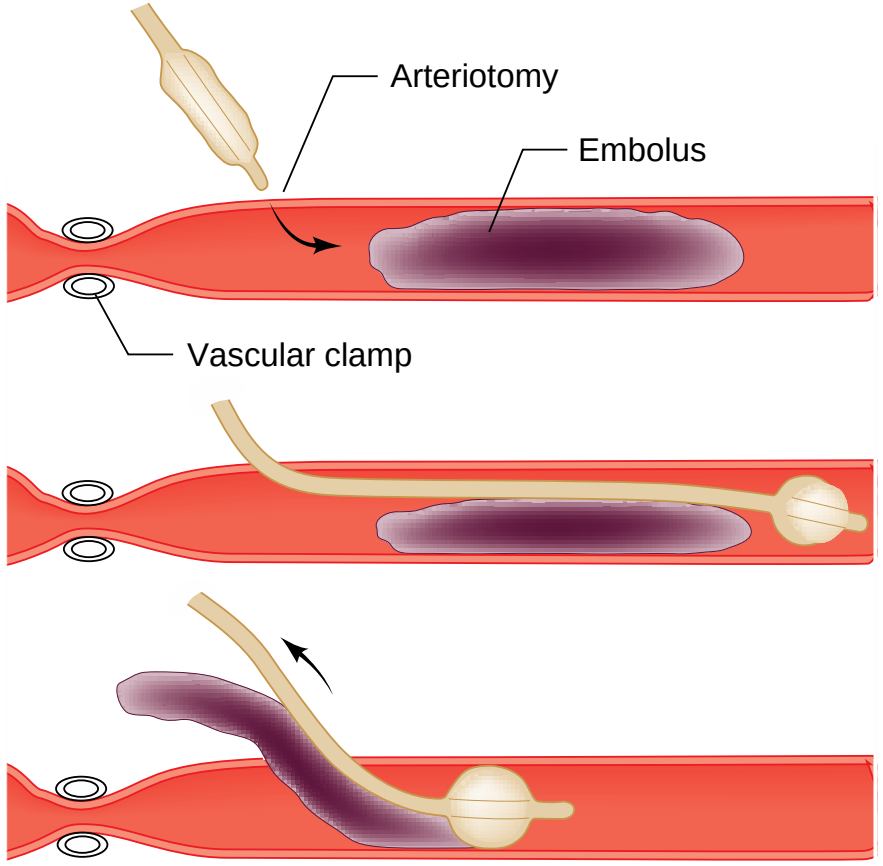
Management

- If ischemia is complete
  - The patient proceeds for embolectomy

- If ischemia is incomplete
  - Preoperative imaging is obtained (simple embolectomy or thrombectomy is unlikely to be successful)
  - Preoperative optimization

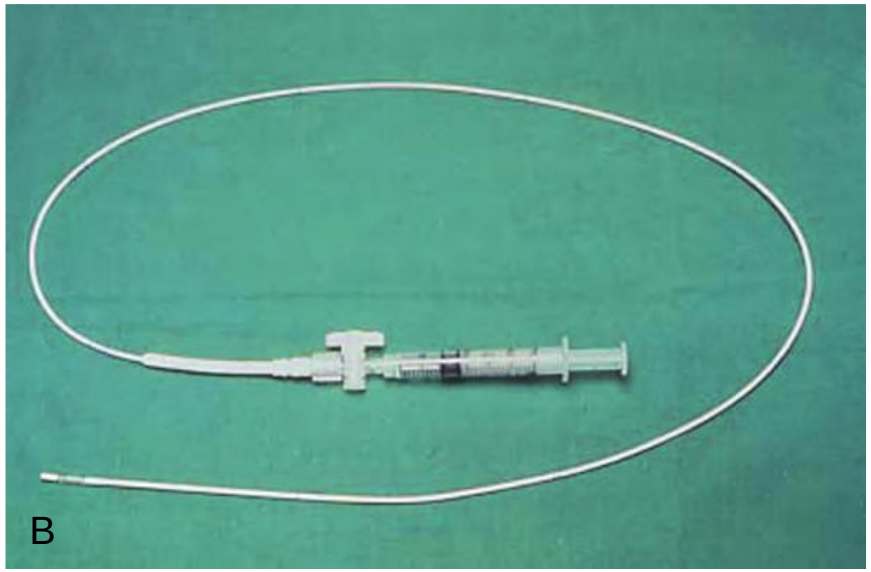
Acute Embolus

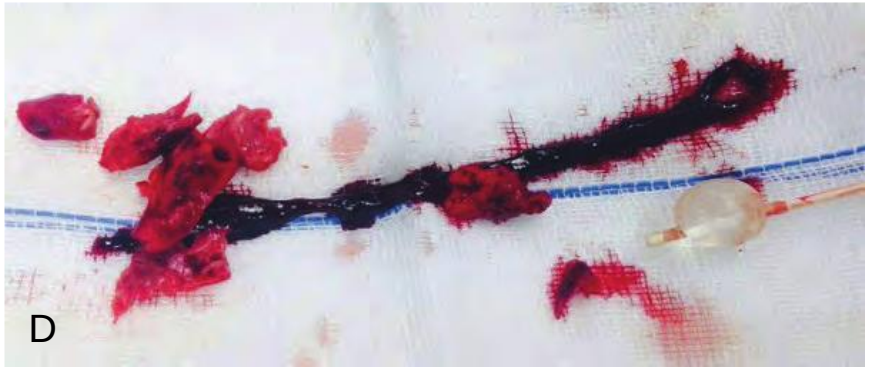
- Femoral embolus is associated with ischemia to the upper thigh
- Acute embolic occlusion of the aortic bifurcation (saddle embolus) leads to absent femoral pulses and having white or mottled waist & legs
  - May also present with paraplegia due to ischemia of the cauda equina



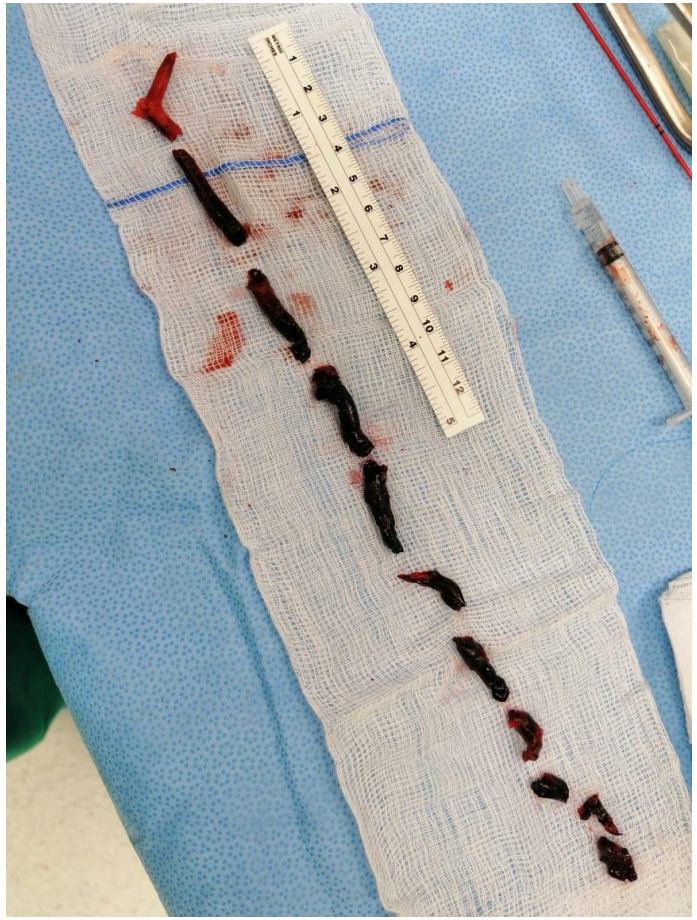
Acute embolus

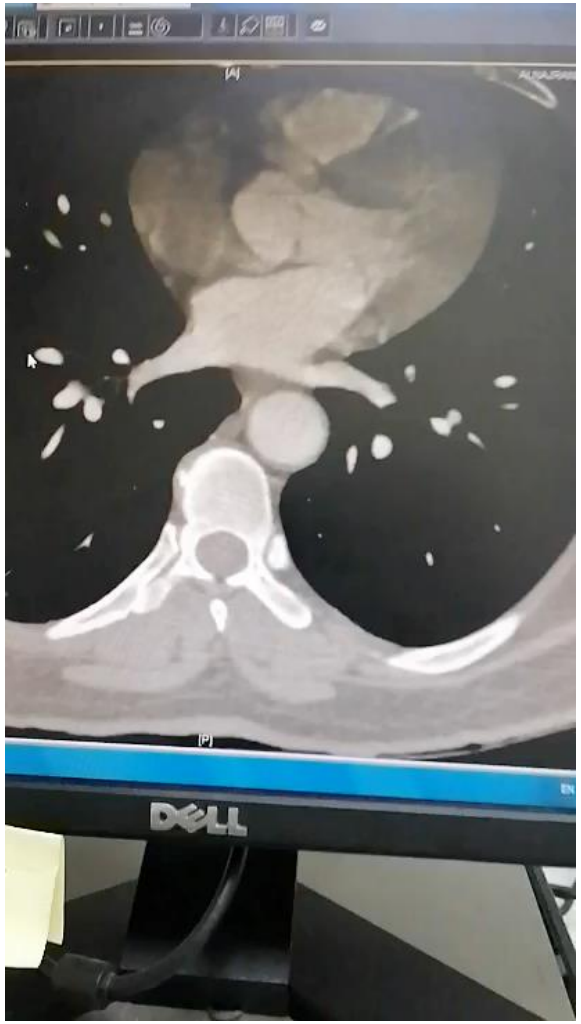
- Embolectomy can be performed under LA or GA
- Postoperatively, the patient should continue on IV heparin
- Warfarin reduces the risk of recurrent embolism but is associated with an annual risk of significant bleeding of 1–2%
- in-hospital mortality from cardiac death or recurrent embolism, e.g. stroke, is 10-20%





Acute embolus





Thrombosis in situ

- Generally occurs in vessels affected by pre existent atherosclerosis
- Ischemia is often less severe than with acute embolism
- Location of occlusion may play a role in the severity of limb ischemia

- Causes include
  - Pump failure (e.g., silent or overt MI)
  - Hypovolaemia, which may be associated with widespread thrombosis
  - Increased blood coagulability (e.g. sepsis, malignancy)

Thrombosis in situ

- Many patients can be managed medically

- If the limb remains threatened then it may be possible to clear thrombus by

  - Thrombectomy
  - Endoluminal techniques
  - Thrombolysis
  - Bypass

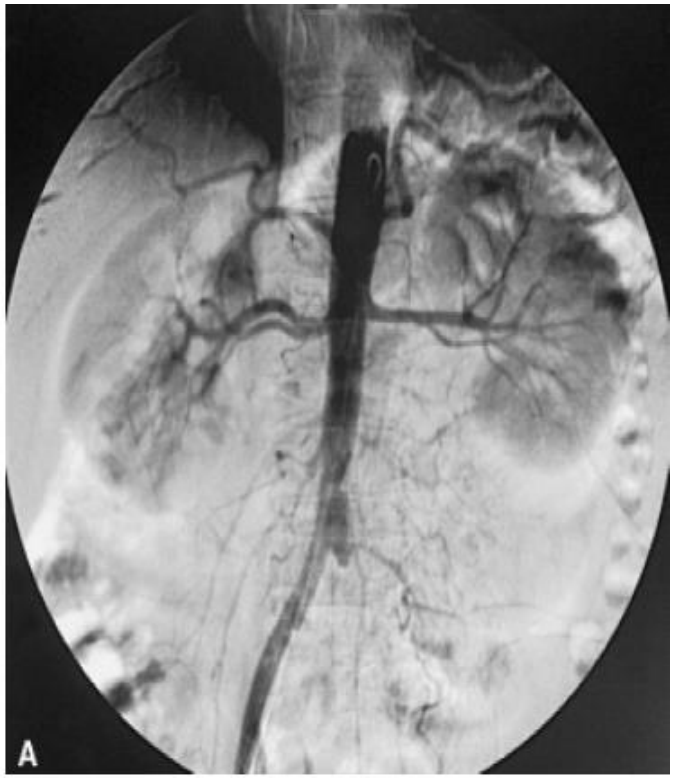
Thrombosis in situ

- If an urgent intervention is required, the in-hospital limb loss rate may approach 30%, with an in-hospital mortality rate of 10–20%.

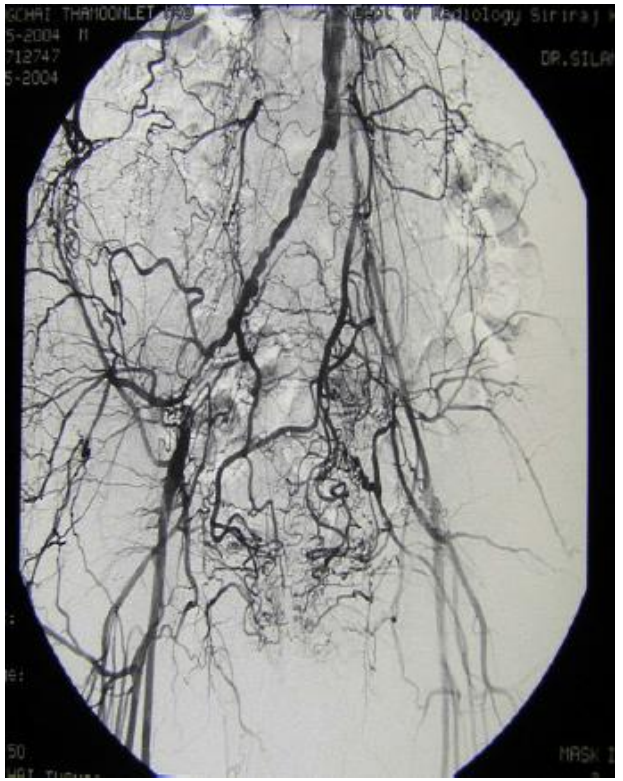
Table 10.2. History and clinical findings differentiating the etiology of acute ischemia

Thrombosis	Embolism
Previous claudication	No previous symptoms of arterial insufficiency
No source of emboli	Obvious source of emboli (arterial fibrillation, myocardial infarction)
Long history (days to weeks)	Sudden onset (hours to days)
Less severe ischemia	Severe ischemia
Lack of pulses in the contralateral leg	Normal pulses in the contralateral leg
Positive signs of chronic ischemia	No signs of chronic ischemia

Acute Embolus



Acute Thrombosis



Post Ischemic Syndrome

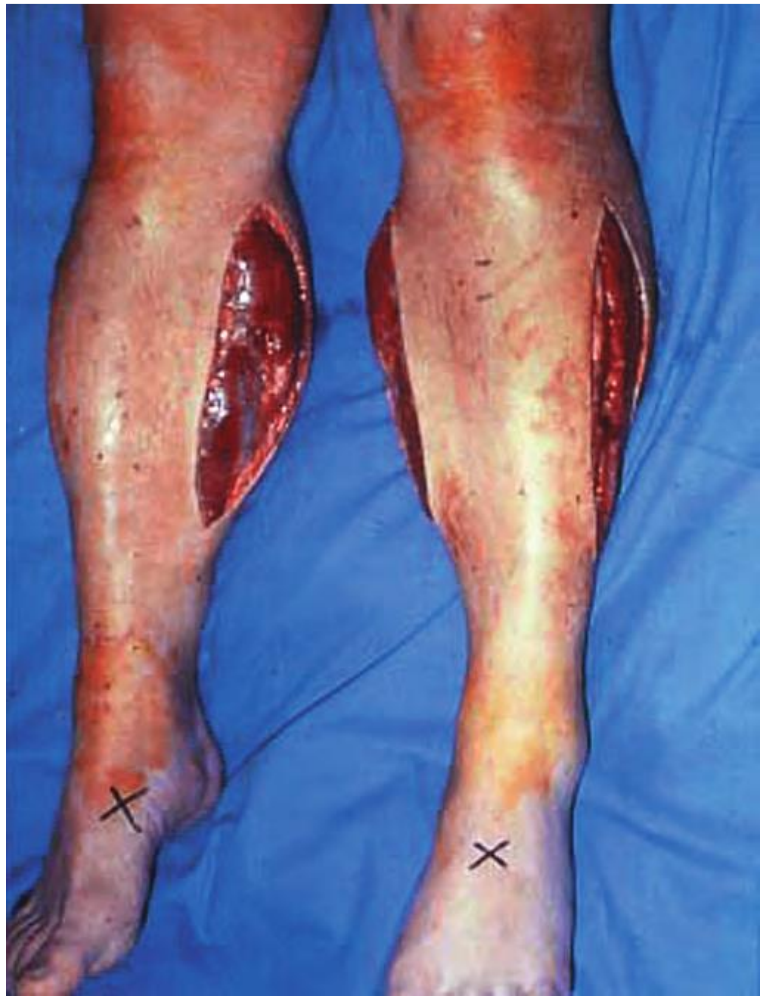
- **Compartment syndrome (local)**
  - Endothelial cell injury leads to increased permeability
  - The calf muscles are confined within tight fascial compartments
  - The increase in interstitial tissue pressure leads to muscle necrosis despite adequate arterial inflow: **compartment syndrome (>25mmHg)**
  - There is swelling and pain on squeezing the calf muscle or moving the ankle
  - Palpable pedal pulses do not exclude compartment syndrome

Post Ischemic Syndrome

- **Management**

- **Prevention** through expeditious revascularisation

- Low threshold for **fasciotomy** to relieve the pressure



Post Ischemic Syndrome

- **Reperfusion injury (systemic)**
  - Caused by activated neutrophils, free radicals, enzymes, hydrogen ions, carbon dioxide, potassium and myoglobin released from reperfused tissue

- Leads to

  - Acute respiratory distress syndrome (ARDS)
  - Myocardial stunning
  - Endotoxaemia
  - Acute Tubular Necrosis
  - Multiple organ failure and death

Post Ischemic Syndrome

- **Reperfusion injury (systemic)**

- Treatment

  - **Hydrate** the patient
  - **Communication** with the anesthesiologist and intensivist
  - Protect the heart with **calcium**
  - Prevent and treat hyperkalemia before reperfusion
  - **Correct acidosis and produce alkalosis** in anticipation to reperfusion
  - Use **inotropic** support liberally

Cerebrovascular disease CVD

- **Stroke**
  - An episode of **focal** neurological dysfunction lasting **>24 hours**, of vascular etiology

- **Transient ischaemic attack**
  - Symptoms last for **less than 24 hours**

- **Amaurosis fugax**
  - **Transient incomplete unilateral loss of vision**, never synchronously bilateral
  - A veil or curtain coming across the eye

CVD Pathophysiology

- Approximately **80% of strokes are ischemic**
- **About half** of these are thought to be due to atheroembolism from the carotid bifurcation
- The origin of the internal carotid artery is most common site of atheroma formation
- The tighter the degree of stenosis, the more likely it is to cause symptoms

CVD Pathophysiology

- Emboli entering the ophthalmic artery leads to **amaurosis fugax** or permanent monocular blindness on the **same side (ipsilateral)**

- If they enter the middle cerebral artery they may cause **hemiparesis and hemisensory loss on the opposite side (contralateral)**

- If the dominant hemisphere is affected there may also be dysphasia

CVD Clinical Assessment

- Complete History
- Risk factor assessment

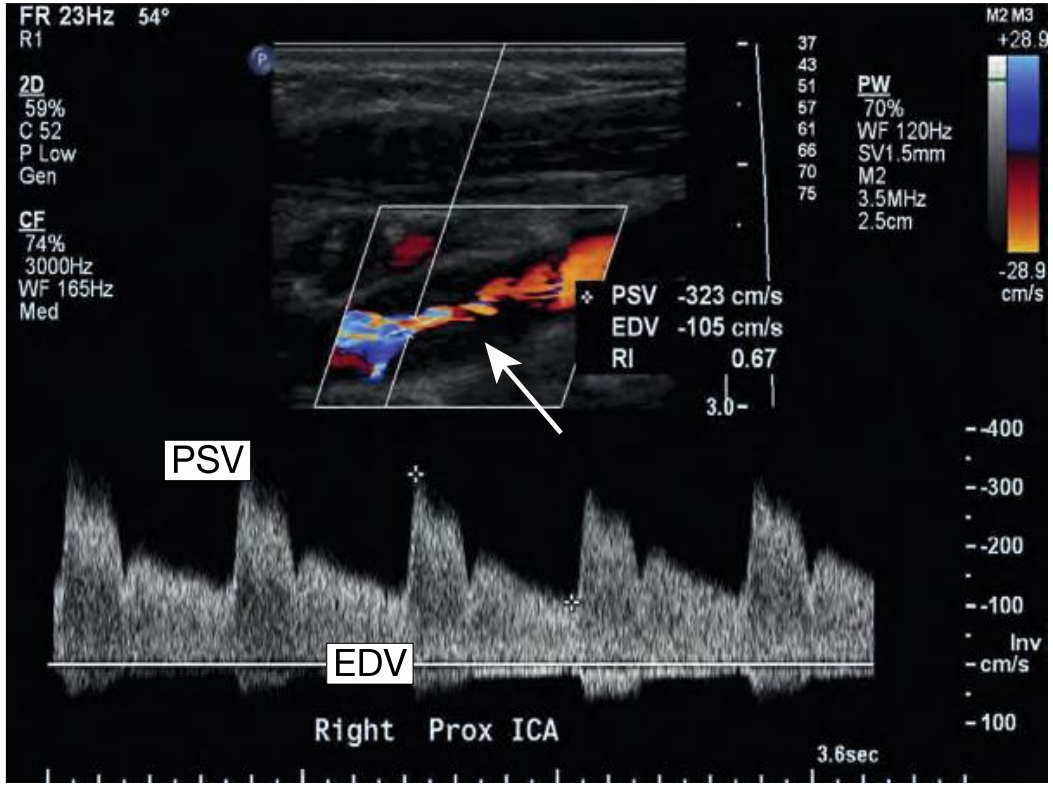
- It is important to exclude other causes of cerebral ischemia and haemorrhage

- Complete Neurological exam

- ‘Carotid’ bruit has no reliable relationship to the severity of the internal carotid artery disease and the risk of stroke

CVD Investigation

- **Doppler (duplex) ultrasound**
- **Magnetic resonance angiography (MRA)**
- **Computed tomographic angiography (CTA)**
- Intraarterial digital subtraction **angiography** is associated with risk of TIA/stroke as it is an invasive procedure



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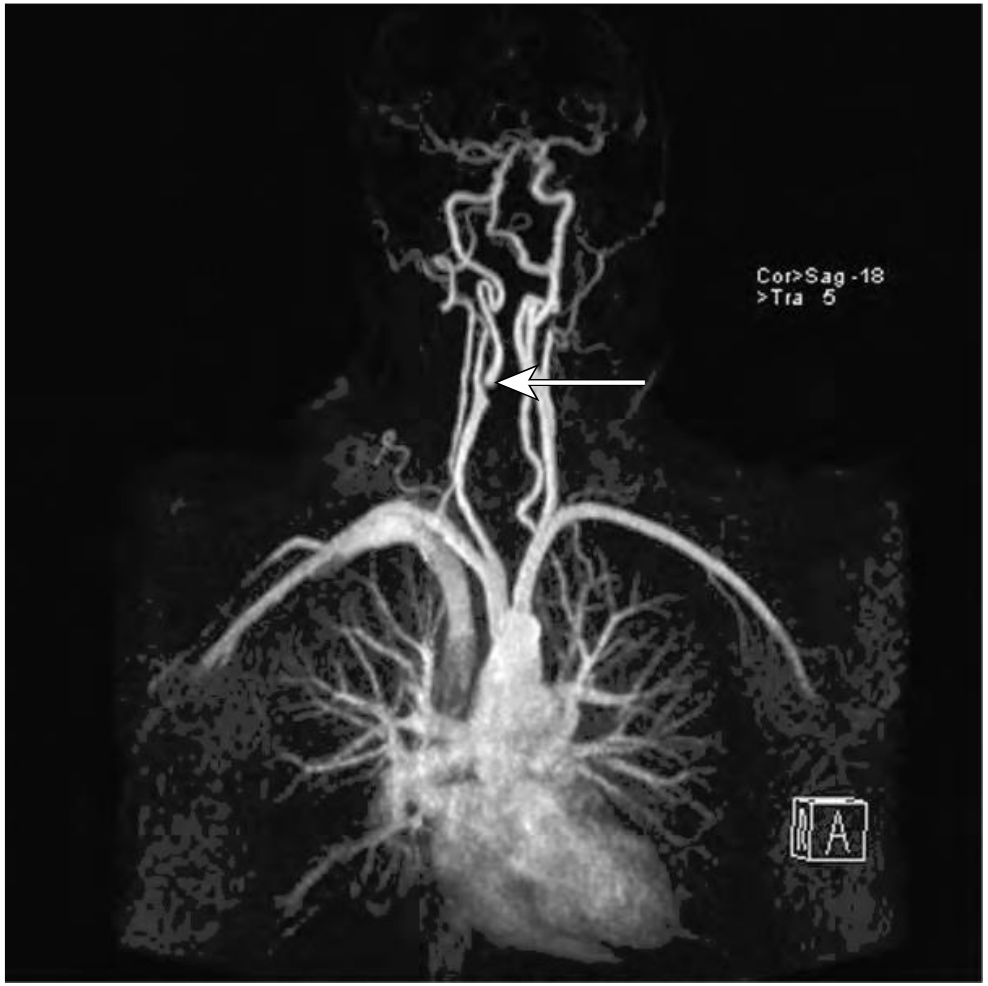
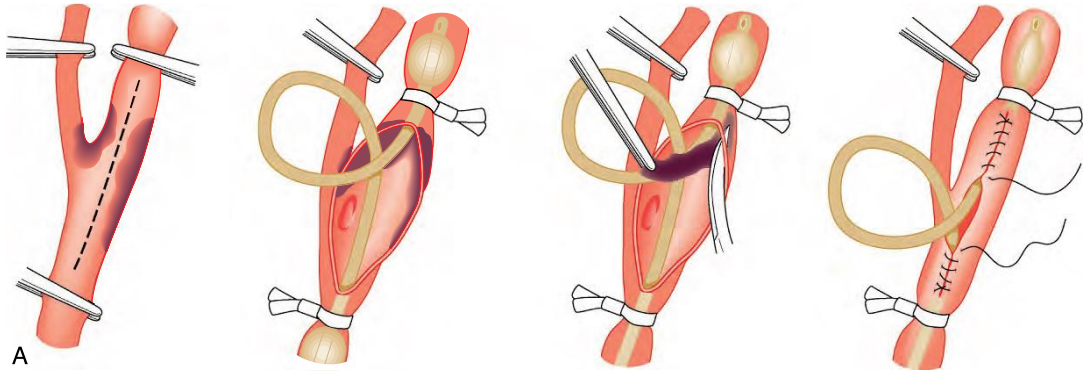


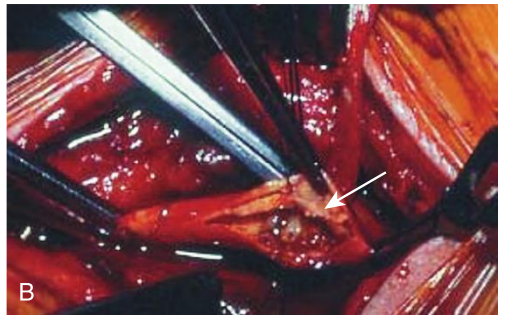
Asymptomatic CVD Management

- **Are treated with BMT**
- Risk of developing TIA/stroke are low (<10% at 5 years)
- The RRR is 50%, the ARR would be only 1% per year
- The number needed to prevent one TIA or stroke is at least 20–30
- The number needed to treat for symptomatic disease is less than 10

Carotid endarterectomy (CEA)

- CEA with BMT is associated with a significant reduction in recurrent stroke, compared with BMT alone



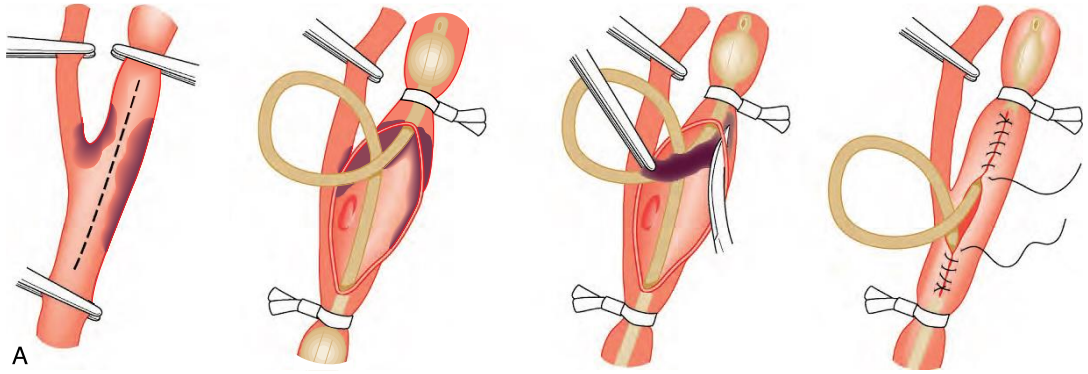


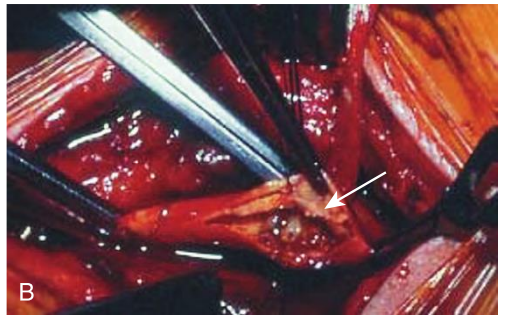
  - ICA stenosis (> 50%)
  - Life expectancy of at least 2y
  - Can be undertaken with a stroke and/or death rate of <5%
  - The intervention can be performed soon

Carotid endarterectomy (CEA)

- The sooner the better
- Performed under GA or LA

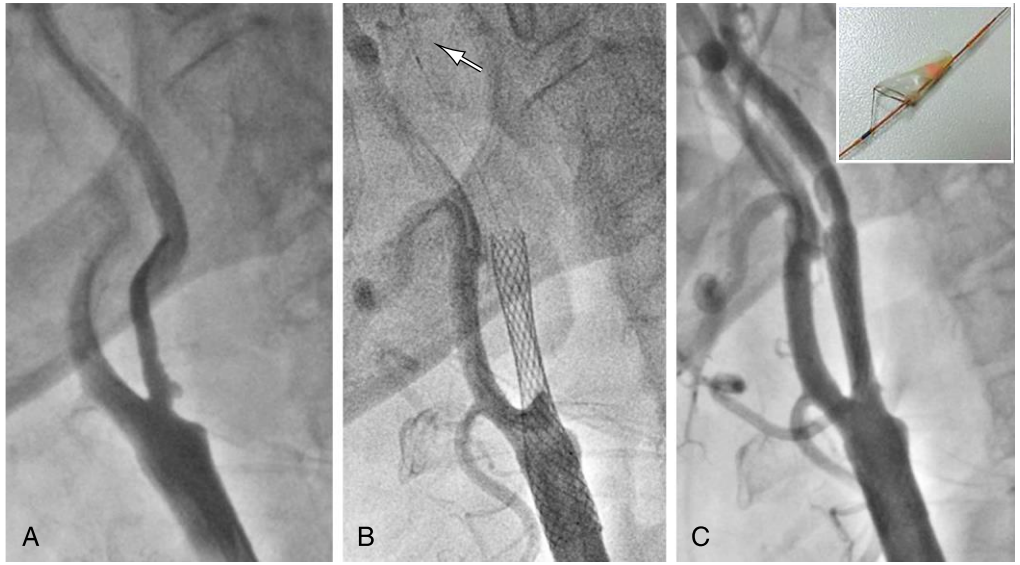
- Patients with major stroke and little in the way of recovery are not candidates for carotid intervention
- Patients with an occluded ICA are not candidates for carotid intervention





Carotid Artery Stenting (CAS)

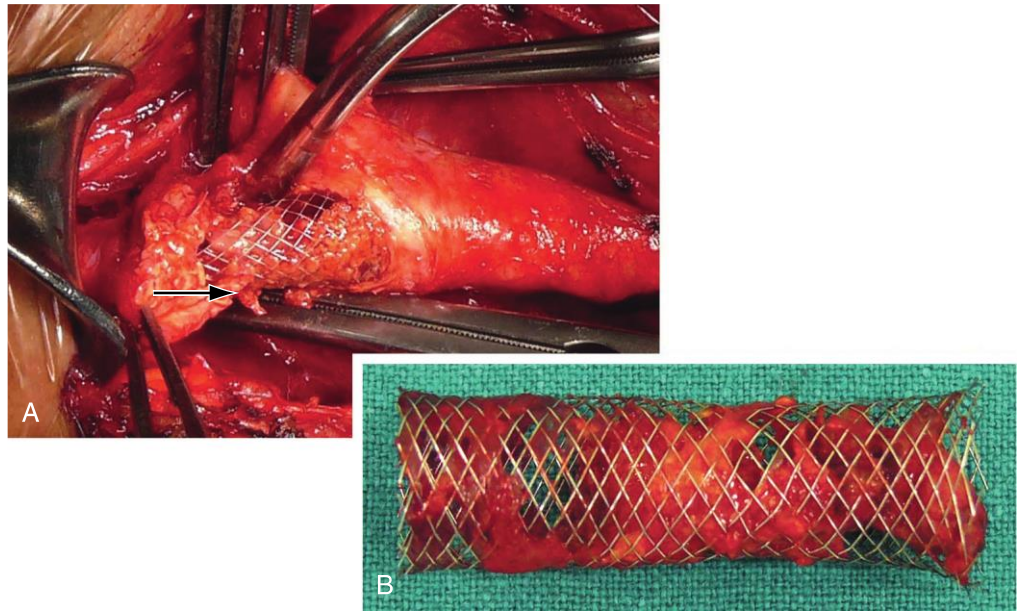
- The role of (CAS) remains controversial
- Avoids a neck wound and the risks of cranial nerve injury
- Reduces the risk of MI
- Short-term risks of clinical and subclinical strokes are greater than CEA



Carotid stenting

- CAS should be reserved for patients where CEA is not possible or desirable because of anatomic and clinical factors

  (e.g., recurrent stenosis after previous surgery or radiation arteritis)



THANK YOU

Principles and Practice of Surgery

Garden and Parks

Chapter 21. Vascular and endovascular surgery