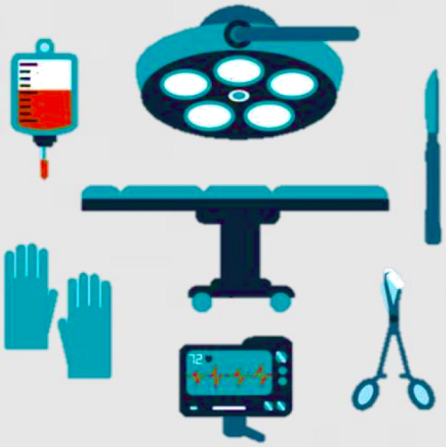


Atherosclerosis (PAD, Carotid stenosis & Acute limb ischemia)

Objectives:

1- To know the pathophysiology of atherosclerosis.

2- Describe the etiology, clinical features, investigations & management of chronic lower limb ischemia.

3- Explain the differentiating features & significance of critical limb ischemia.

4- Describe etiology, presenting features & management of acute limb ischemia.

5- Reperfusion injury & its management.

6- Pathogenesis & management of Diabetic foot.

7- Describe the etiology, clinical features, investigations & management of Carotid artery atherosclerotic disease.

Resources:

- Davidson's.
- 436 doctors slides.
- Surgical recall.
- 435' team work

Done by: Jawaher alkhayyal, Ghada almazrou, Noura alhugail, Shahad alanzan
Leaders: Heba Alnasser, Jawaher Abanumy, Mohammed Habib, Mohammad Al-Mutlaq
Revised by: Maha AlGhamdi

Color Index:

Notes , Important , Extra , Davidson's

Editing file

Feedback

Atherosclerosis Helpful video

The Arterial system:

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

Types of arteries:

Elastic arteries	Muscular arteries	Small arteries
Aorta & Beginning of its large branches have preponderance of elastic fibers in media	medium sized arteries, distributing arteries exhibit smooth muscles in their walls	major site of autonomic regulation of blood flow

---

Risk factors :

Modifiable		Non-modifiable
Major	**Minor**	Male
Smoking	Physical inactivity / sedentary lifestyle	Age
Hypertension	Obesity and high carb intake	After menopause in female
Diabetes	Homocysteinemia (Hyperhomocysteinemia)[1]	Family predisposition
	type A personality[2]	Genetic abnormality things you are born with you can't change.
Hyperlipidemia	Stressful, Competitive life style &	

Major factor: We act on these factors, so if someone asked what is the best medical therapy it mostly act on these factors.

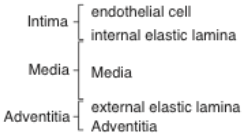
[1] Elevated homocysteine

[2] Type A behavior characterized by excessive ambition, aggression, competitiveness, drive, impatience, need for control. It is commonly associated with risk of coronary disease and other stress-related ailments.

Pathophysiology:

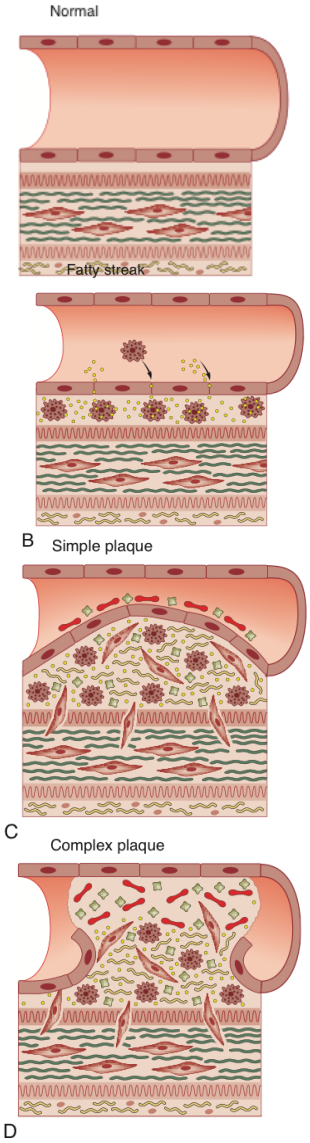
Atherosclerosis is a disease process which is triggered by sometimes subtle physical or chemical insults to the endothelial cell layer of arteries. At bifurcations, lots of Atherosclerosis comes at bifurcations like carotid bifurcation, or femoral bifurcation. Hypertension may increase that stress.

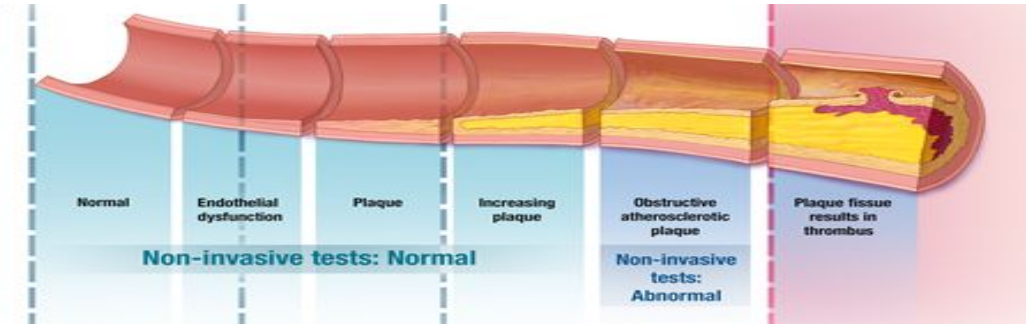
- Endothelial injury
  - Chemical
  - Physical
  - Atheroma



- Hypertension increases the stress

- Increased permeability to lipids and inflammatory cells > Leukocytes adhere into the subendothelial space and digest lipids to become foam cells
- Protease and free radicals liberated
- Cytokines attract leukocytes and smooth muscle cells
- Smooth muscle cells exit the media
- Proliferate, take on the characteristics of fibroblasts and produce collagen, raising the atheroma
- Proliferation forms an endothelial cap, which may rupture, ensuing further endothelial injury
- This results in thrombosis and distal embolization



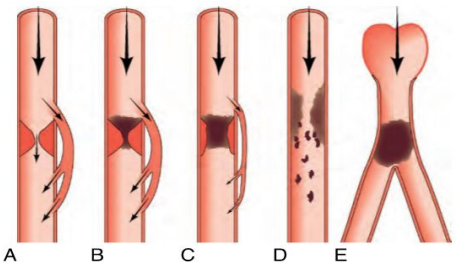


Mechanisms of injury in atherosclerotic disease:

A. Critical stenosis compensated by collateral vessels

- symptomatic on exercise

in figure A there is stenosis but the stenosis have happen through time, so, the body will develop collaterals and the collaterals will compensate, this what happens with claudication. Instead of walking for 5 km he walks for 500m, 300m, there is enough blood to keep it alive, there is enough blood to prevent neural injury, there is enough blood to heal, small wound but there is not enough blood to go forward and go along with daily activities,



B. Acute thrombosis of a critical stenosis

- little change in symptoms due to collateral development

Picture B: the stenosis is there because of so tight thrombosis might happen but you also have some collaterals because this happened overtime, pathology of the occlusion.
Not each patient has occlusion must be accompanied with ischemia per say because this occlusion happened overtime

C. Acute thrombosis of noncritical stenosis

- Severe symptoms due to poorly developed collaterals

D. Atheroembolism from ruptured plaque

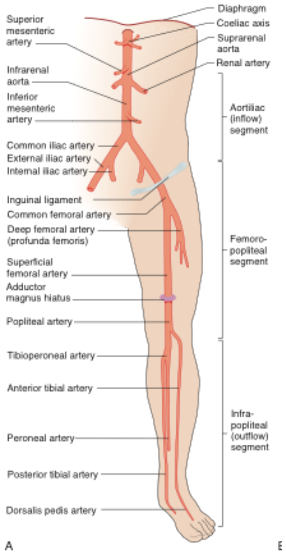
E. Thromboembolism

- severe ischemia because of lack of collateral supply

Didn't understand? Check this video!

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

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



Peripheral Arterial Disease (PAD)

Chronic	Acute
Slow gradual luminal stenosis secondary to plaque	Sudden occlusion in the absence of adequate collaterals
Collateral development compensate	Embolism
Symptoms proportional to disease burden	Thrombosis
Exertional symptoms appear first	Injury
Can lead to : • **Intermittent Claudication (IC)** • **Critical limb ischemia (CLI)** • **Diabetic foot (DF)**	Can lead to : • **Acute Limb Ischemia (ALI)**

Generally speaking, it is either chronic or acute: chronic: slow, gradual, usually there is a plaque, collaterals. symptoms are provoke propotentianal to disease burden, as much as you get stenosis as much as you get occlusions the symptoms will get worse. If you get one occlusion or one tight stenosis you might have marked indication you might not feel. If you have multiple occlusions it might become critical limb ischemia, you might have tissue loss.

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 THEN limb THEN function.

IC (intermittent claudication)

- Claudication pain is a muscular pain
IC is usually characterized by pain on walking
in the muscles of one or both calves.
- Not present at rest
-The pain comes on after 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:

- **Common site** (IMPORTANT)

(the site of claudication gives a clue to site of arterial disease):
In the lower extremities in

- Bilateral thigh and/or buttock: aortoiliac arteries
- Unilateral thigh and/or buttock: iliac arteries
- Calf: femoropopliteal arteries
- Foot (instep) : tibial arteries usually rare

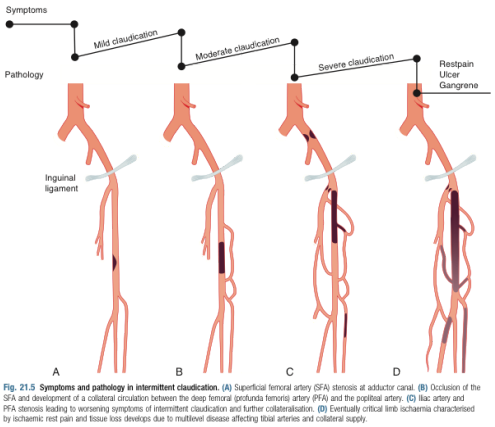


Fig. 21.5 Symptoms and pathology in intermittent claudication. (A) Superficial femoral artery (SFA) stenosis at adductor canal. (B) Occlusion of the SFA and development of a collateral circulation between the deep femoral (profunda femoris) artery (PFA) and the popliteal artery. (C) Iliac artery and 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.

Signs and symptoms:

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

- IC Investigations:

We Will have other lectures explaining them in details

- 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 (chronic limb ischemia)

Whereas IC is usually due to single-level disease, CLI is caused by **multiple lesions** affecting different arterial segments in the affected limb. These patients usually have

- Rest pain.
  - Exacerbated by lying down or elevation of the foot,relieved by sleeping with feet hanging over the bed or sleeping on a chair.
  - 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
- Tissue loss in form of (ulceration or gangrene)
- May present with foot swelling
- Low ABPI[3]

Without revascularization, such patients will often lose their limb, and sometimes their life, in a matter of months.

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

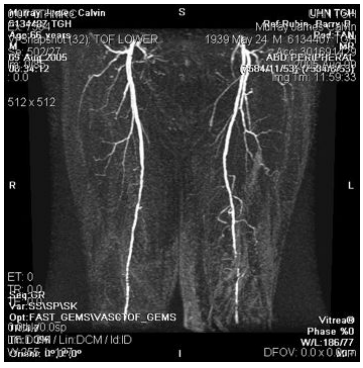
Buerger test: it is basically, when you have ischemia, what happen to your body will signal the capillaries which are going to dilate because they wants more blood, for example when the patient is sitting his feet is little bit red once you rise it his feet is back to normal color so you explain it is 25 degree burger test or 10 degree burger test.

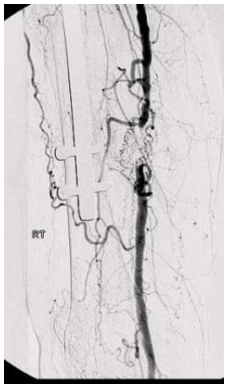
Pallor, particularly on elevation

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

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





Investigation:

➔ Duplex Ultrasound
  ◆ Multiple Stenoses or occlusions
➔ CT Angiogram
➔ MRA
➔ Invasive Vascular Investigations

Common site:
dorsalis pedis artery (on the dorsum of the foot above the metatarsal) classically awaken the patient .

[3] The severity of ischaemia in the leg can be simply estimated by determining the ratio between the ankle and brachial blood pressures.

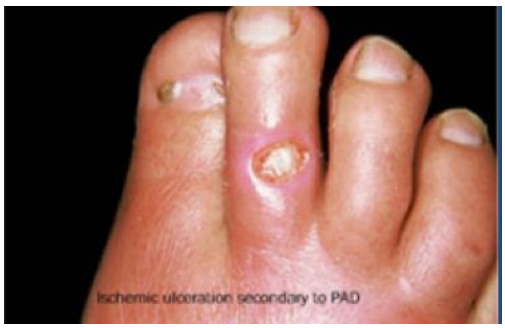
Intervention: angioplasty, or surgical.
Surgical: Endarterectomy or bypass.

Indication of surgery: (STIR)

- **S**evere claudication - disabling claudication pain (depends on the patient, for example: If the patient occupation needs a lot of walking i will perform surgery, but if the patient is a 70 years old will start medical treatment and wait.)
- **T**issue necrosis
- **I**nfection
- **R**est pain.

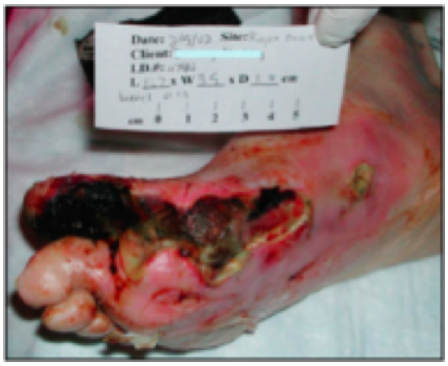
Tissue loss:

- Arterial ulcers
  - Type and screen
  - Often located on toes or foot
  - Pale and with necrotic floor
  - Irregular margins
  - Painful
  - Surrounding ischemic features

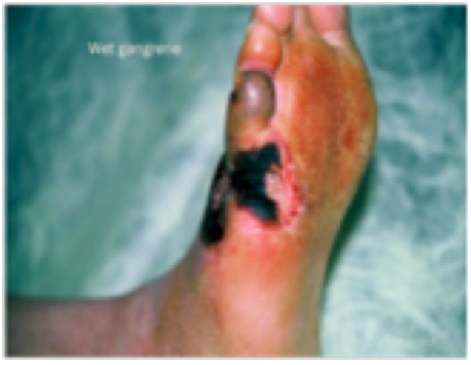


Ulcer Examination:

If you have an ulcer in your osce, make sure to cover all these point



- 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



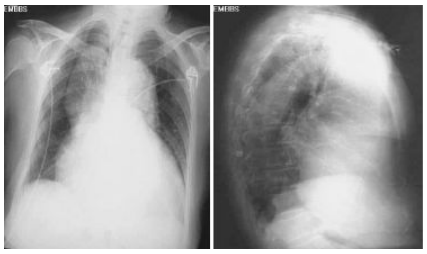
Floor is what you see (inspect) , base is what you feel (palpate)

- 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

- 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 X-ray
- Echocardiogram



PAD Prevention:

A) Primary Prevention
- Modifiable risk factors – lifestyle changes

B) Secondary prevention
- Best medical treatment (treat the risk factors: DM,HTN ec..)

The best treatment is to prevent the patient from having peripheral arterial disease.
Primary prevention: to modify your lifestyle, become active, reduce weight, ... secondary prevention is medical treatment for the risk factors, if the patient has diabetes, or hypertension, we will start treating that, or smoker > stop smoking.
Treatment: it can be by surgical, or angioplasty.
You have to ask him to stop smoking, you have to control their hypertension preferably with ACE inhibitors, you start him with statins regardless if they have dyslipidemia or not because statin have anti-inflammatory process it is smooth in atherosclerosis. And you start him with antiplatelet agents either aspirin or plavix or the new antiplatelet agents like that they give for coronary artery disease patients.

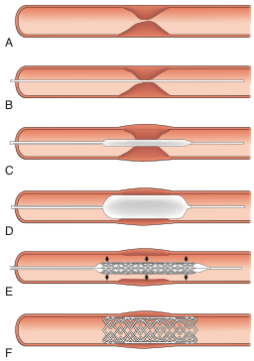
- Treatment
  - Endarterectomy
  - Percutaneous Angioplasty
  - Bypass procedures

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
  - why? They have anti-inflammatory characteristics which will help with the atherosclerosis
  - 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 (HbA1c < 7%)

- 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 Intervention:



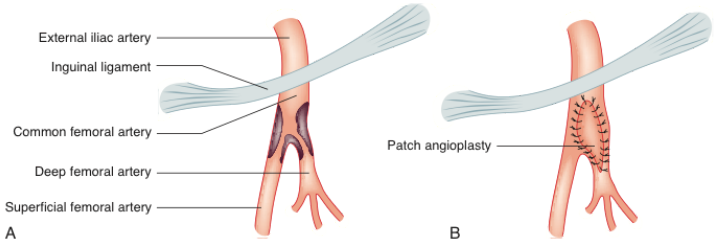
- Indications for intervention are:
  - Disabling claudication pain
  - CLI
- Intervention includes
  - Balloon angioplasty, with or without stenting
  - Surgery
- Endovascular interventions:
  - The lesion is identified
  - Crossed with a wire
  - A balloon is inserted and inflated
  - This enlarges the lumen by disrupting the plaque
  - In patients with occlusions and complex disease, stents may be deployed
  - 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

Endovascular intervention: you try to do everything within the vessels.
Balloons is multiple: there is regular balloons, and there is balloons with (chemotherapy agent) why because you want to deliver chemotherapy to the area of atherosclerosis to prevent the prefiltration of smooth muscles, sometimes with atherosclerosis with the occlusion we put stent, as much as the lesion was shorter, and the lesion is not occlusion, the outcome is favorable.

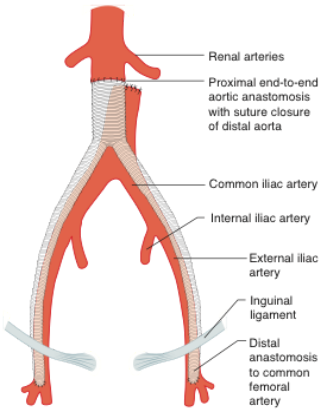
Surgery:

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

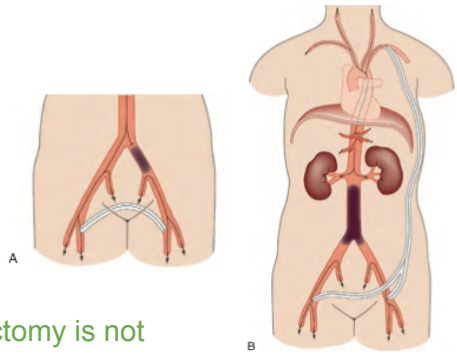
the doctor dissect, heparinize the patient, get proximal and distal control, open the vessel then try to see if there is a layer within atherosclerotic area or between the atherosclerosis and adventitia you shed it out, clean it and put a patch or close it primarily.



- **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 blood must have somewhere to go when it leaves the graft **(outflow or run-off)**
  - The conduit must be suitable, two main types of conduit are available :
    - **Autogenous material**, most commonly a vein.
    - **Prosthetic material**, expanded polytetrafluoroethylene (ePTFE) or Dacron.



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



Bypass: While having long occlusion or multiple area of disease, endarterectomy is not feasible, then doctors try to bypass that occlusion, for bypass to work they have to have sufficient blood flow because if blood flow was sluggish “low” thrombosis develops.

Choice of treatment decided depending on :
Patient symptoms
Comorbidities
Life expectancy
Risk and benefits
Anatomy of the disease
Prior interventions

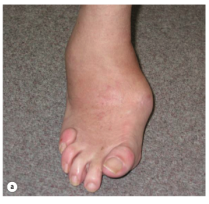
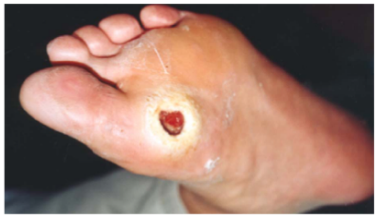
Diabetic foot cont. chronic PAD

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

- 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 is a combination of three things, patients who have ischemia, because they have small vessels disease, and neuropathy, they don't feel their feet, so the injuries are elevated and because they have diabetes they are immunocompromised. These are the three things that make diabetic foot clinically different than coronary arterial disease.

Diabetic neuropathy:

SENSORY	MOTOR	AUTONOMIC
- Patient incapable of feeling pain - Affects proprioception such that, when walking, pressure is applied at unusual sites -This leads to ulcer formation and joint destruction (Charcot's Foot) In this case distal nerves get inflamed and irritated, and in the end you start to feel pins and needles and you lose your sensation, starting with the feet, patient cant feel while he is walking. While the patient has lost the proprioception. 	- 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. 	- 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 Prevention:

- Diabetic control (HbA1c <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 footwear

DF investigations:

➔ Similar to PAD.
➔ Depends on the history and the physical exam.

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 unreconstructable disease accounting for the very **high amputation rate**

Acute Limb Ischemia (ALI):

Etiology

- Acute limb ischaemia is caused most frequently by acute thrombotic sudden **occlusion** of a pre-existing stenotic arterial segment (60%), thromboembolism (30%) and trauma.
- Distinguishing between thrombosis and embolism is important because investigation, treatment and prognosis are different
- 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

1. **Incomplete acute ischemia** (usually due to thrombosis in situ) can often be treated medically, at least in the first instance
2. **Complete ischemia** (usually due to embolus) will normally result in extensive irreversible tissue injury within **6 hours** unless the limb is revascularized
3. **Irreversible ischemia** mandates early amputation or, if the patient is elderly and unfit, end-of-life care

Signs and symptoms ALI : the 6P's very important

- **Pain**
- **Paresthesia**
- **Paralysis**
- **Poikilothermia**[4]
- **Pallor**
- **Pulselessness**

Paralysis (inability to wiggle toes / fingers)

Paraesthesia (loss of light touch over the dorsum of the foot / hand)

The other Ps assist the diagnosis

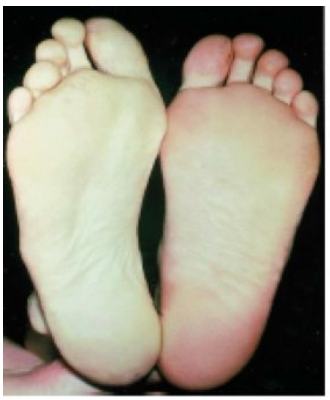
Table 21.6 Symptoms and signs of acute limb ischaemia

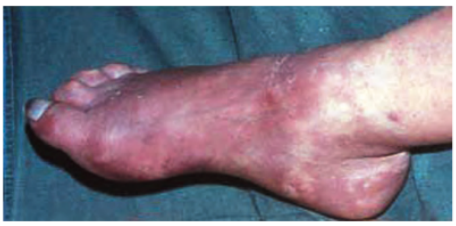
Symptoms/ signs	Comment
Pain	May be absent in complete acute ischaemia; severe pain is also a feature of chronic ischaemia
Pallor	Also a feature of chronic ischaemia
Pulseless	Also a feature of chronic ischaemia
Perishing cold	Unreliable, as the ischaemic limb takes on the ambient temperature
Paraesthesia 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

[4] Poikilothermia: impaired regulation of body temperature of the limb usually cool, reflecting the ambient temperature.

Signs and symptoms ALI:

- ★ 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 non-fixed mottling
- ★ At this stage, the limb is salvageable
- ★ 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
- 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

ECG and some other modalities are important to use because of the risk of having silent MI with no apparent signs or symptoms

Complete: plaqutomy within 6 hours

Incomplete: IV heparin, control the pain, imaging to reach best treatment plan.

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

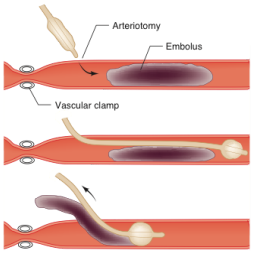
- May also present with paraplegia due to ischemia of the cauda equina

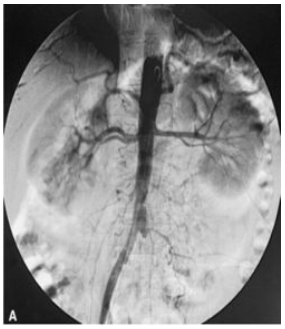
Embolectomy can be performed under LA or GA

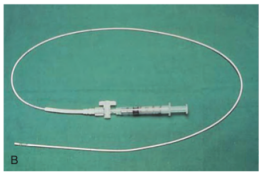
Postoperatively, the patient should continue on IV heparin

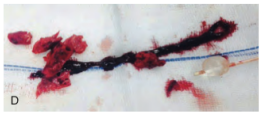
Warfarin reduces the risk of recurrent embolism but is associated with an annual risk of significant bleeding of 1–2%

In-hospital mortality from cardiac death or recurrent embolism, e.g. stroke, is 10-20%









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)

Many patients can be managed medically

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

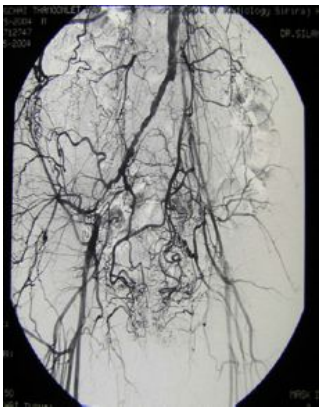
- Thrombectomy
- Endoluminal techniques
- Thrombolysis
- Bypass

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

In thrombosis in situ, there is the disease and atherosclerosis that have 5 elements, there will be stenosis, plaque causing thrombosis, later patient will have symptoms, but not as bad as acute complete lower limb ischemia .



Surgical recall:

What is atherosclerosis?
Diffuse disease process in arteries; atheromas containing cholesterol and lipid form within the intima and inner media, often accompanied by ulcerations and smooth muscle hyperplasia
What is the common theory of how atherosclerosis is initiated?
Endothelial injury → platelets adhere → growth factors released → smooth muscle hyperplasia/plaque deposition
What are the risk factors for atherosclerosis?
Hypertension, smoking, diabetes mellitus, family history, hypercholesterolemia, high LDL, obesity, and sedentary lifestyle
What are the common sites of plaque formation in arteries?
Branch points (carotid bifurcation), tethered sites (superficial femoral artery [SFA] in Hunter's canal in the leg)
What must be present for a successful arterial bypass operation?

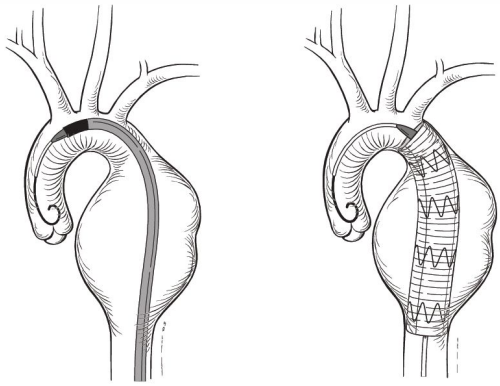
1. Inflow (e.g., patent aorta)
2. Outflow (e.g., open distal popliteal artery)
3. Run off (e.g., patent trifurcation vessels down to the foot)

What is the major principle of safe vascular surgery?
Get proximal and distal control of the vessel to be worked on!
What does it mean to "POTTS" a vessel?
Place a vessel loop twice around a vessel so that if you put tension on the vessel loop, it will occlude the vessel
What is the suture needle orientation through graft versus diseased artery in a graft to artery anastomosis?
Needle "in-to-out" of the lumen in diseased artery to help tack down the plaque and the needle "out-to-in" on the graft

What are the three layers of an artery?

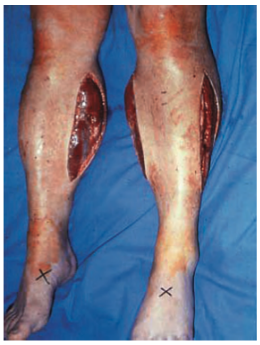
1. Intima
2. Media
3. Adventitia

Which arteries supply the blood vessel itself?
Vaso vasorum
What is a true aneurysm?
Dilation (>2× nL diameter) of all three layers of a vessel
What is a false aneurysm (a.k.a pseudoaneurysm)?
Dilation of artery not involving all three layers (e.g., hematoma with brous covering) Often connects with vessel lumen and blood swirls inside the false aneurysm
What is "ENDOVASCULAR" repair?
Placement of a catheter in artery and then deployment of a graft intraluminally



Post Ischemic Syndrome

patient who completed 6 hours without blood flow to his leg. First of all, he'll have anaerobic metabolism > exudate, lactate, acid, then with time you will have necrosis you will have potassium leaking out from the cells. What will happen initially you will have endothelial injury which will increase the permeability, then you will have edema, increased permeability blood flow along with acidosis, will result in increased interstitial pressure, over than 25 mmHg it will be higher than the pressure in the capillaries, at this point necrosis starts. the patient has pulse because pressure in the arteries is 120 but when the blood goes to the capillaries it don't prefuse because the pressure in the compartment is higher

Compartment injury (local)	Reperfusion injury (systemic)
• 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	• 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 - Endotoxemia - Acute Tubular Necrosis - Multiple organ failure and death Ischemia activates neutrophils, free radicals follows muscle will reach necrosis level, enzymes and hydrogen ions, carbon dioxide anaerobic metabolism all of them will be released to the systemic flow and it may cause acute respiratory distress syndrome ...
Management: •**Prevention** through expeditious revascularisation •Low threshold for **fasciotomy** to relieve the pressure 	**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

Up to 50% of all ischaemic strokes may be caused by atheroembolism from the carotid bifurcation. Patients with carotid territory transient ischaemic attacks (TIA) and amaurosis fugax (painless temporary loss of vision) **should be assessed by a vascular surgeon with a view to carotid endarterectomy (CEA)**

CVDs:

- **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
- 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:

1. Complete History
2. Risk factor assessment
3. It is important to exclude other causes of cerebral ischemia and haemorrhage
4. Complete Neurological exam
5. ‘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)**
- Intra-arterial digital subtraction **angiography** is associated with risk of TIA/stroke as it is an invasive procedure

Asymptomatic CVD management:

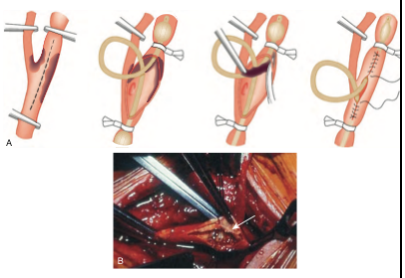
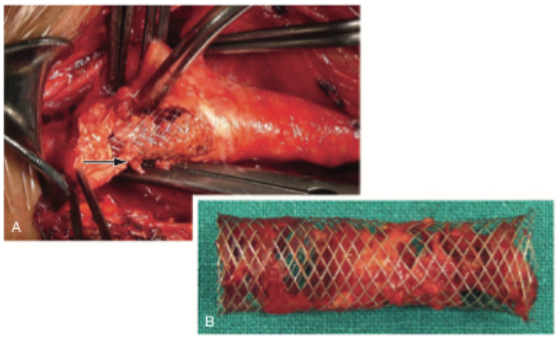
Are treated with BMT

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)	Carotid Artery Stenting (CAS)
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 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 	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 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) 

Summary

Atherosclerosis :

- an **inflammatory process** that causes clogging, narrowing and hardening of the **large** and **medium** sized arteries, the atheromatous plaque must reduce the artery cross sectional area about **70%** In order to cause a significant drop of the arterial flow at rest .
- Pathophysiology :**Toxic insult (DM,HTN,..)-->Endothelial Dysfunction --->Inflammatory Response-->Foam cell Formation (engulfed LDL by the macrophages)-->Fatty streaks-->Intermediated lesion -->Fibrous Plaque -->Thrombus formation .**
- The risk factors for atherosclerosis can be divided into :
  1-Modifiable Major(Smoking , Hypertension,Diabetes,Hyperlipidemia)
  2- Modifiable minor (Physical inactivity,Obesity and high carb intake,Homocysteinemia , type A personality ,Stressful, Competitive lifestyle)
  3- Non-Modifiable (M,Age ,after menopause in female,family predisposition ,genatic abnormality
- **Prevention and Treatment:**
  Primary prevention for modifiable risk factors like lifestyle
  Secondary prevention
  - **Statins ,Low dose Aspirin** ,Beta blockers, ACE-I, and Angiotensin receptor antagonists

  Treatment
  - Endarterectomy
  - Percutaneous Angioplasty
  - Bypass procedures

Peripheral Arterial Disease (PAD):

is the sequence of atherosclerosis of peripheral vessels excluding the carotids and coronaries arteries.

- it presents as **acute arterial diseases** : acute thrombotic sudden occlusion of a pre-existing stenotic arterial segment (60%), thromboembolism (30%) and trauma. it can be:

1. 1- Embolic an Embolus can be from Cardiac source, Non-Cardiac, Iatrogenic. The most Common sites of embolus: Femoral artery
2. 2-Thrombotic The local formation or presence of a blood clot in a blood vessel.
3. 3-,Traumatic The most common causes of injury are limb fractures, dislocations, blunt injuries, road traffic accidents, and stab wounds.

- The classic signs/ symptoms include Pain ,paralysis,pallor,paresthesia,poikilothermia,pulselessness.

Pain location and site of occlusion:

Pain location	Site of occlusion
Buttock and hip 30%	Aortoiliac disease "**Leriche's syndrome triad":** Bilateral claudication + absent femoral pulses +/- erectile dysfunction (impotence)
Thigh	Aortoiliac or common femoral artery[5]
Upper ⅔ of the calf 60% (most common)	Superficial femoral artery
Lower ⅓ of the calf	Popliteal artery
Foot claudication	Tibial arteries (especially in **DM**)

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 neuropathy :

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

Diabetic control ,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 footwear.

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,amputation rate

5

Questions

1. Which of the following Peripheral Arterial Disease is acute?

A. Intermittent Claudication
B. Acute Limb Ischemia
C. Critical limb ischemia
D. Diabetic foot

2. The site of arterial disease if the pain is in Unilateral thigh is?

A. Aortoiliac arteries
B. Tibial arteries
C. Iliac arteries
D. Femoropopliteal arteries

3. What is the Management for complete ischemia?

A. Embolectomy
B. IV heparin
C. Both A & b
D. None of the above

4. Arterial Ulcers is a feature of:

A. Intermittent Claudication
B. Acute Limb Ischemia
C. Critical limb ischemia
D. All of the above

7. Which of the following differentiate btw CLI & ALI ?

A. Paralysis
B. Paresthesia
C. Pain
D. A&B

5. How much increase in interstitial tissue pressure needs to cause compartment syndrome?

A. > 30 mmHg
B. > 20 mmHg
C. > 25 mmHg
D. > 18 mmHg

Q6: Which of the following causes transient incomplete unilateral loss of vision

A. Stroke
B. Transient ischaemic attack
C. Sensory Neuropathy
D. Amaurosis fugax

Answers:
1: B 2: C 3: A 4: C 5:C 6:D 7:D