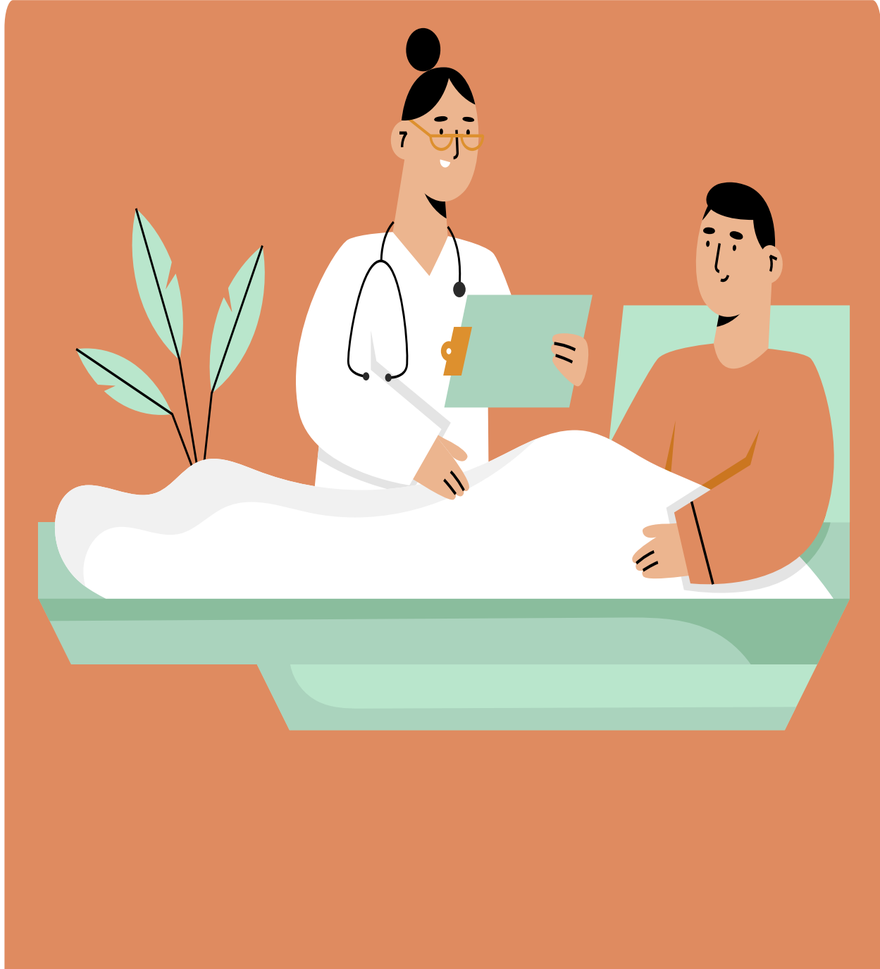
MED437
KING SAUD UNIVERSITY

SURGERY TEAM 437



Atherosclerosis

Objectives:

1. Pathophysiology of atherosclerosis
2. Describe the etiology, clinical features, investigations & management of chronic LL 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

Team leaders: Alanoud Almansour, Ghaida Al Musma , Muath Alhamoud and Mohammed Alqwaifli

Done by: Ebtsam Almutairi, Laila Alsabbagh & Abdulrahman Alkholaif

Revised by: Yazeed Al-Dossare

Color Index:

- Important
- Doctor's Notes
- Extra
- Davidson's

Editing File / Feedback

Atherosclerosis

13:27

The Arterial system:

- Contains 30% of blood volume
- Normal systolic pressure <130 mmHg
- Arterial capillary pressure 25 mmHg
- High pressure / low volume system , But in Venous system → low pressure, high volume.

Types of arteries:

Elastic arteries	Muscular arteries	Small arteries
Aorta and 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 blood flow

Risk Factors:

We act on major factors, so if someone asked what is the best medical therapy it mostly act on these factors

Modifiable		Non-Modifiable
Major	Minor	
Smoking	Physical inactivity/ sedentary lifestyle	Gender (male)
Hypertension	Obesity and high carb intake	Age
Dyslipidemia	Elevated homocysteine (Homocysteinemia) (Hyperhomocysteinemia)[1]	Family predisposition
Diabetes	Type A personality [2]	Genetic abnormalities
	Stressful, competitive lifestyle	After menopause in female , We can delay the menopause by pharmacological way, but physically no.

Box 21.1 Factors determining clinical manifestations of arterial disease

Anatomical site

- Coronary arteries: myocardial infarction
- Cerebral circulation: stroke, transient ischaemic attack, amaurosis fugax, vertebrobasilar insufficiency
- Renal arteries: hypertension, renal failure
- Mesenteric arteries: mesenteric angina, acute intestinal ischemia
- Limbs: intermittent claudication, chronic limb ischaemia, acute limb ischemia

Type of arterial supply

- End artery (only supply to tissue)
- Well collateralised (one of several arteries)
- For example, in a patient with a complete circle of Willis, occlusion of one internal carotid artery may be asymptomatic, whereas with an incomplete circle occlusion is more likely to cause a stroke

Speed of onset

- Slow development of atheroma may give chance for collateral development (e.g., profunda [deep] femoral artery collateralises around a diseased superficial femoral artery in patients with intermittent claudication).
- Sudden occlusion of a previously normal artery may cause severe ischemia (as there has been no time for collateral vessels to develop)

Mechanism of injury

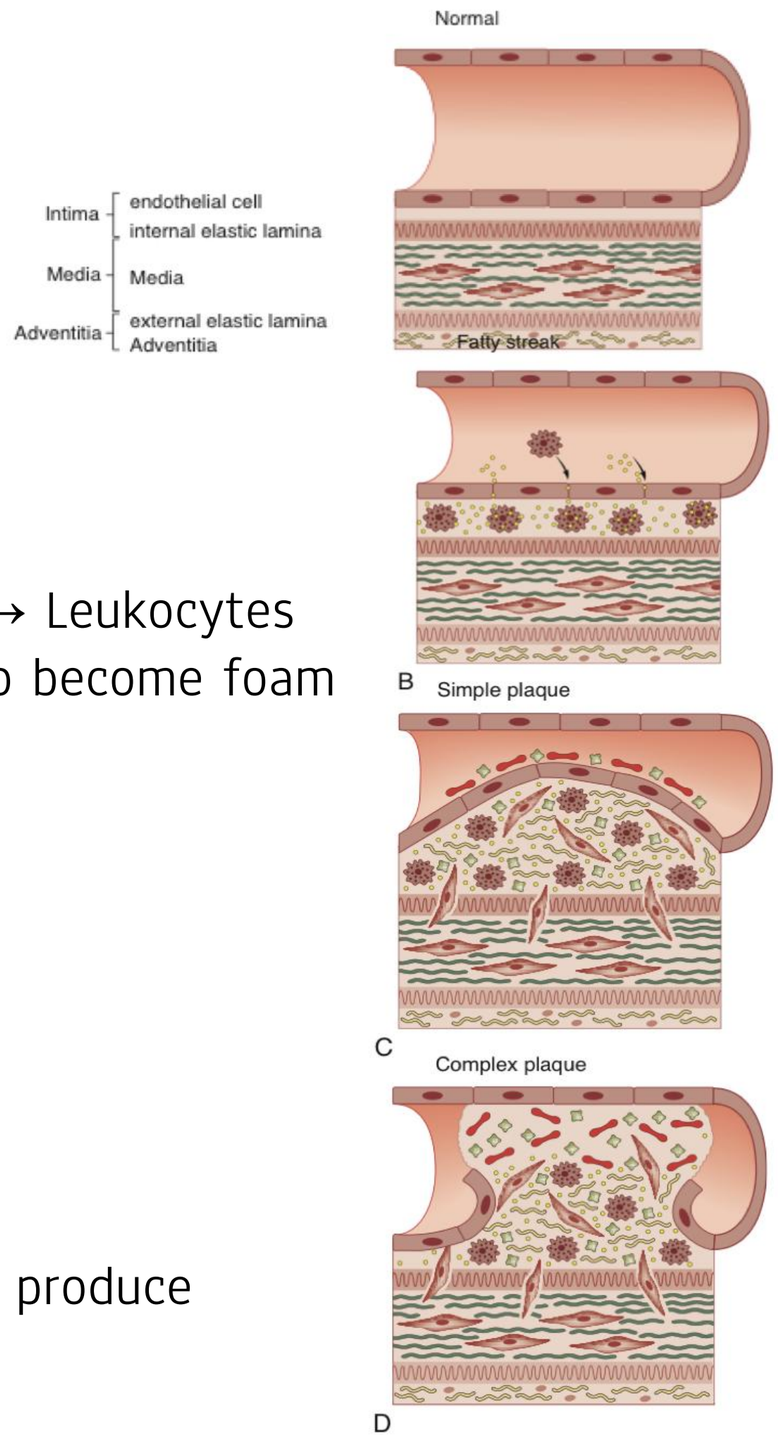
- Haemodynamic mechanism: pressure drop across a stenosis is proportional to blood velocity. During walking, pressure drop is increased and is called critical stenosis (e.g., intermittent claudication)
- Thrombosis: rupture of plaque (myocardial infarction or stroke)
- Atheroembolism: embolic load. Small load = temporary (e.g., transient ischaemic attack), high load = irreversible (e.g., retinal infarct)
- Thromboembolism: most common source left atrium, thrombus load is high and often causes sudden occlusion (e.g., stroke or limb ischemia).

[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 bifurcation, lots of atherosclerosis comes at bifurcation, like carotid bifurcation or femoral bifurcation.



★ Endothelial injury:
- Chemical
- Physical
- Atheroma

★ Hypertension increases that stress

★ Increase permeability to lipids and inflammatory cells → Leukocytes adhere into the subendothelial space and digest lipids to become foam cells

★ Protease and free radicals liberated

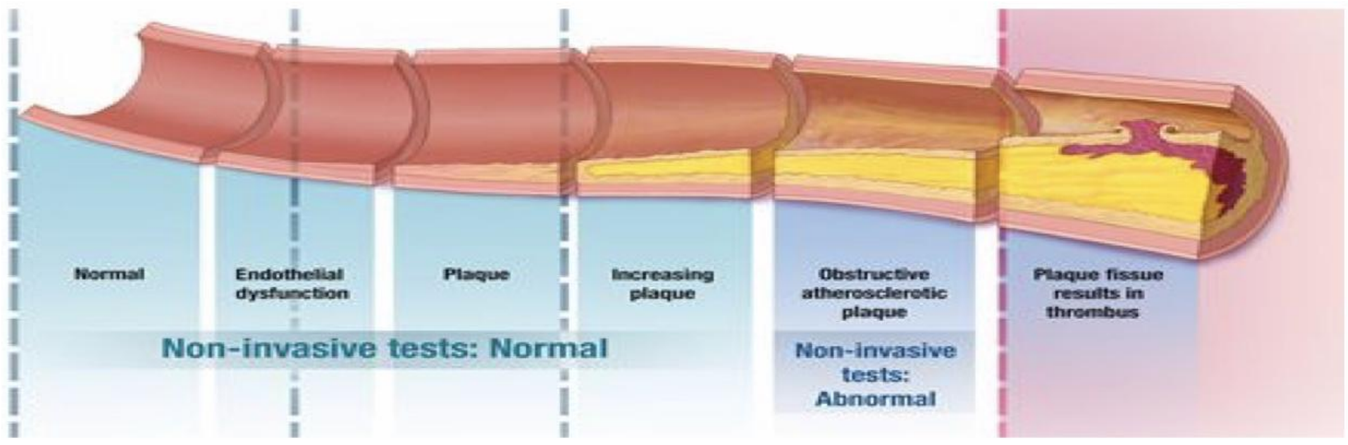
★ Cytokines attract leukocytes and smooth muscle cells

★ Smooth muscle cells exit the media

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

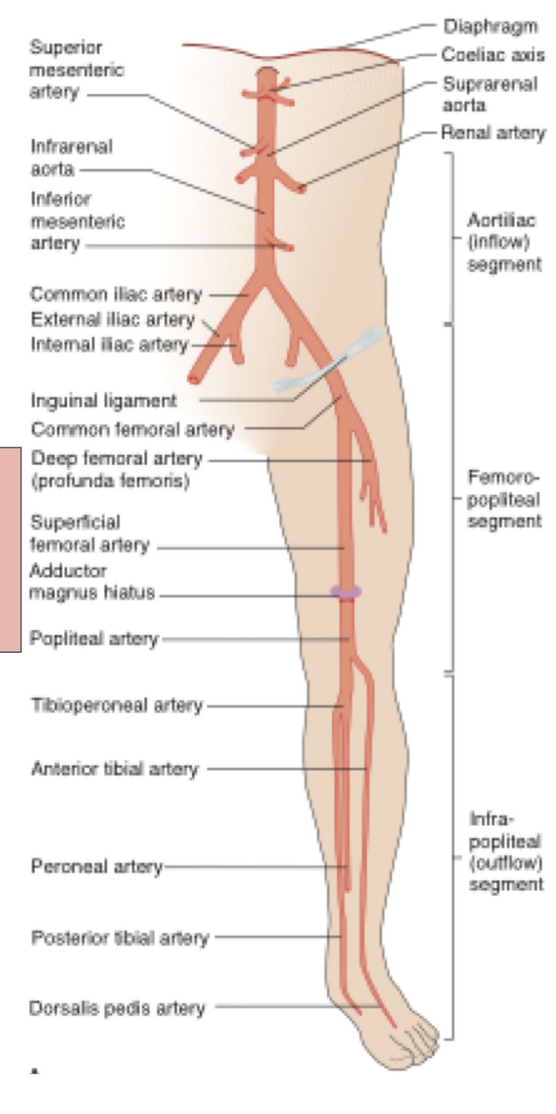
★ Proliferation forms an endothelial cap, which may rupture, ensuing further endothelial injury

★ This results in thrombosis and distal embolization injury



The aortoiliac segment above the inguinal ligament (inflow)

The femoropopliteal segment & the infrapopliteal segment (outflow)



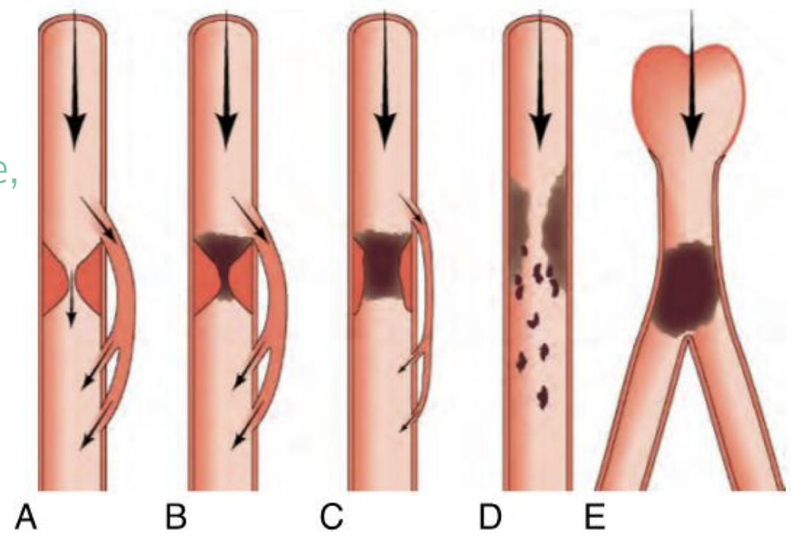
Mechanisms of Injury in Atherosclerotic Disease

13:45

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 or 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 no enough blood to go forward along the daily activities

Important pic



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

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

Peripheral Arterial Disease (PAD)

1. Intermittent Claudication (IC)

- ★ 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.**
- ★ **Repetitive**, the patient will develop the pain after walking the claudication distance

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 Clinical Features

a. Common site: (the site of claudication gives a clue to site of arterial disease):
   i. 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

b. Signs and symptoms:
   - Impotence if the problem in aortoiliac segment.
   - Weakness / decreased mobility
   - Skin changes dry and scaly.
   - Toe nail changes
   - Muscle wasting

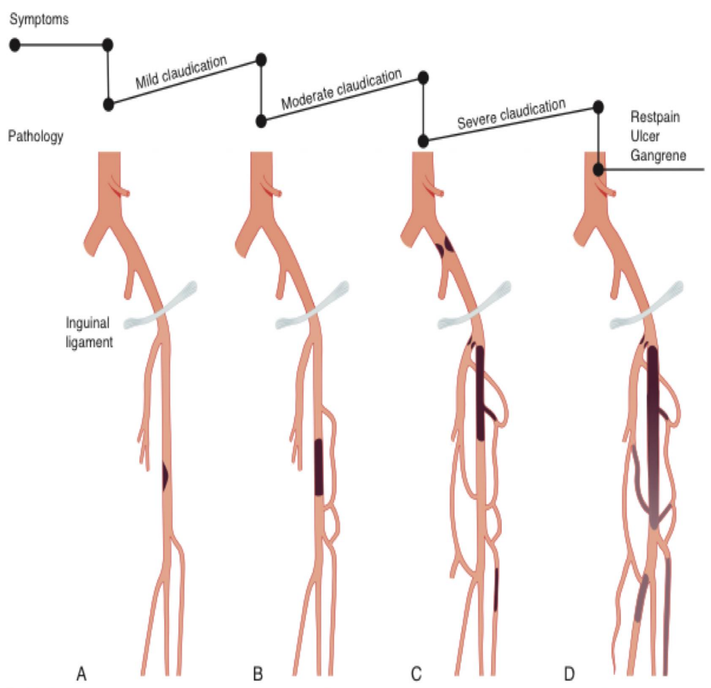


Fig. 21.5 **Symptoms and pathology in intermittent claudication.** (A) Superficial femoral artery (SFA) stenosis at adductor canal. (B) Occlusion of the SFA and development of a collateral circulation between the deep femoral (profunda femoris) artery (PFA) and the popliteal artery. (C) Iliac artery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

IC investigations

Ankle Brachial Index (ABI): 0.8-0.4 normal is 0.9-1.30 (low)

Toe pressures: <50 mmHg normal is 70-110 (low)

Segmental pressure: 20 mmHg reduction (low)

Volume Plethysmography: measure arterial volume changes

Duplex Ultrasound: stenosis or single occlusion

Invasive Vascular Investigations

CT Angiogram

MRA

2. Critical Limb Ischemia (CLI)

★ 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. (gravity)
- Classically felt at night, awakening the patient 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

★ More than 50% of patients will have amputation of the limb

★ Low ABPI ⟶ Severity of ischaemia in the leg can be estimated by determining the ratio b/w the ankle & brachial BP

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

**the difference between CLI and intermittent claudication ⟶ rest pain and tissue loss (in CLI).

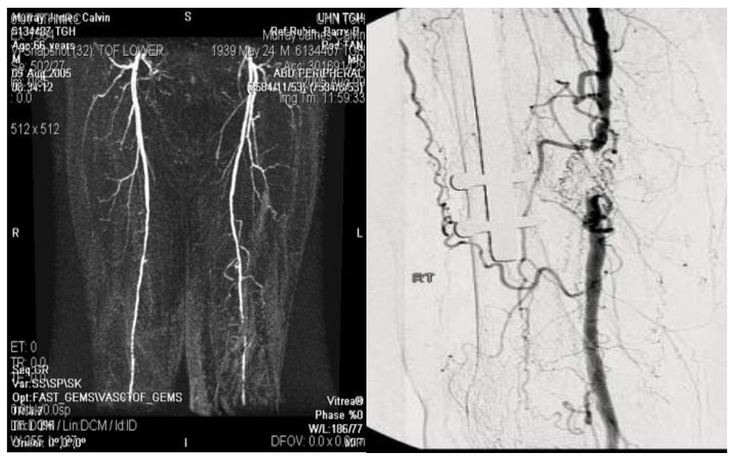
CLI Examination Findings

1. Skin is thin & dry	2. **Pallor**, particularly on elevation	3. Brittle Nails	4. Muscle wasting	5. Reduced temperature

2. 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 & 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

- Ankle Brachial Index (ABI) <0.5 (lower than IC)
- Toe pressures < 30 mmHg (lower than IC)
- Segmental pressure
- Volume Plethysmography
- Duplex Ultrasound (Multiple Stenoses or occlusions) CT Angiogram & MRA
- Invasive Vascular Investigations

Intervention:

1. Angioplasty
2. Surgical: endarterectomy or bypass.

Indication of Surgery: (STIR)

- **Severe** claudication - refractory to conservative treatment that affects quality of life (depends on the patient, ex: if the patient occupation needs a lot of walking i will perform surgery, but if the patient is a 70 yo start medical treatment and wait.)
- **Tissue** necrosis
- **Infection**
- **R**est pain.

2. Critical Limb Ischemia (CLI)

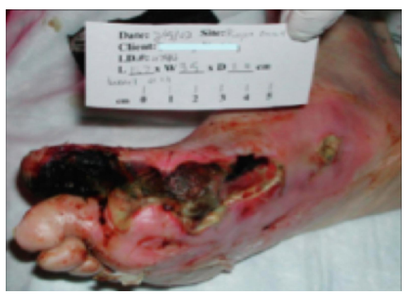
Tissue Loss

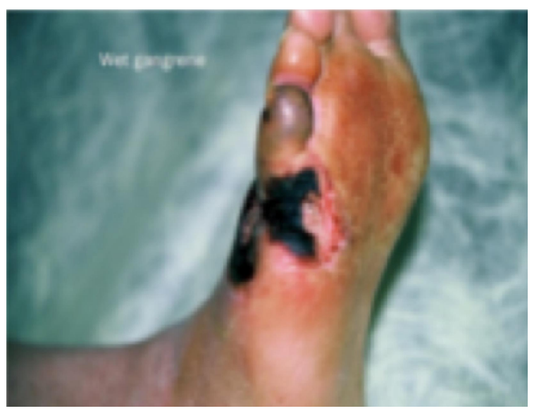
Arterial Insufficiency Ulcers

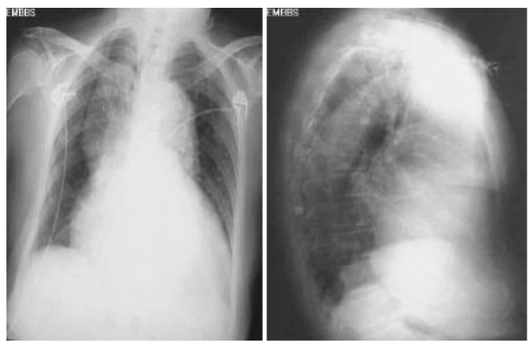
- ★ Type and screen
- ★ Often located on toes or foot
- ★ Pale and with necrotic floor
- ★ Irregular margins
- ★ Painful
- ★ Surrounding ischemic features

Ulcer Examination:

Inspection	- Site (location) - Number - Size - Shape - Floor (The exposed part of an ulcer (inspection) it is what you see - 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
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
Investigation	- CBC, Electrolytes, creatinine, coagulation profile (aPTT, INR) - Type and screen - Lipid profile - Hemoglobin A1c - ECG - Chest X-ray - Echocardiogram In the clinic ankle-brachial index (ABI) test is the first step







Peripheral Arterial Disease (PAD)

PAD Prevention: The most important treatment is prevention!

1. Primary Prevention
   a. Modifiable risk factors – lifestyle changes
   b. to modify your lifestyle, become active, reduce weight.
2. Secondary prevention
   a. Best medical treatment (treat the risk factors: DM,HTN ec..)
   b. medical treatment for the risk factors. If the patient has diabetes or HTN, we will start treating that, or smoker → stop smoking

Treatment

Endarterectomy	Percutaneous Angioplasty	Bypass procedures
Remove diseased intima & media		

PAD Medical Management: PACE

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

- **C**essation from smoking
- 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: **A**spirin (81 mg daily) with or without **P**ento**X**ifylline*, or clopidogrel (75 mg daily)
- Regular **E**xercise & diet
- Control of obesity
- The identification and treatment of patients with diabetes (HbA1c < 7%)

* Increase RBC deformity and fle**X**ibility

★ *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*

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
- Walking and exercise program
- Many patients fail to comply

To improve the collaterals and anaerobic metabolism of muscles.

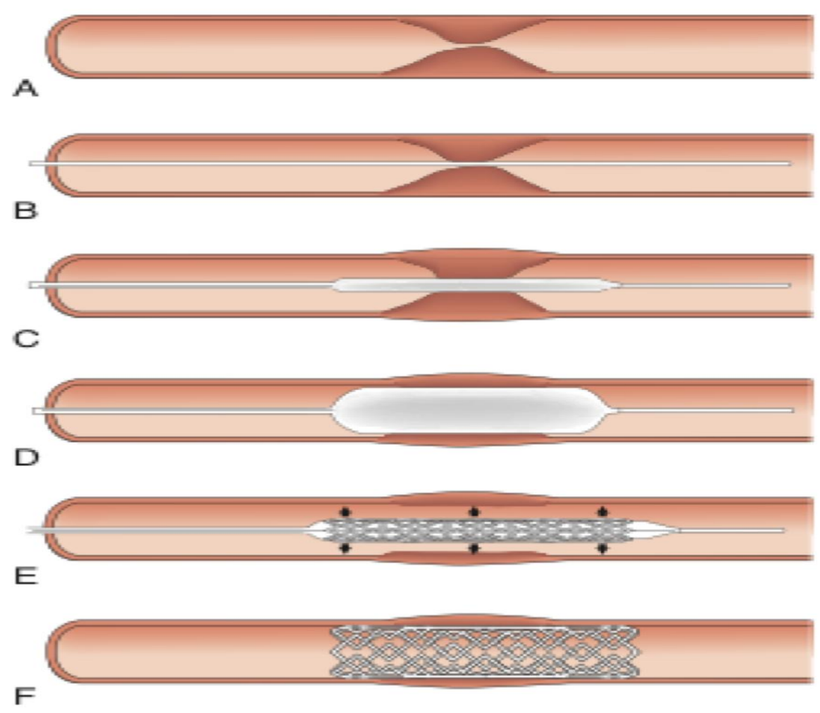
Indications for intervention are:

- Disabling claudication pain
  لما يكون المريض يحتاج يمشي مسافات كبيره مثلاً موظف في مصنع كبير وهذا يمنعه، لكن لو شخص ما يحتاج مشي كثير ما نسوي العمليه
- CLI

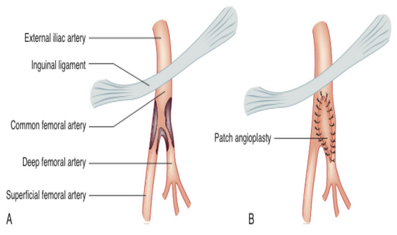
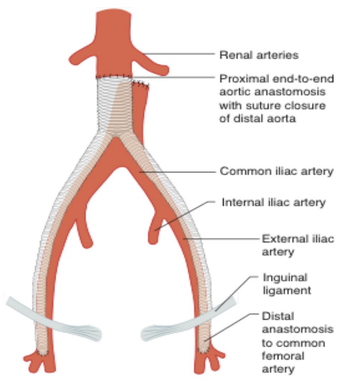
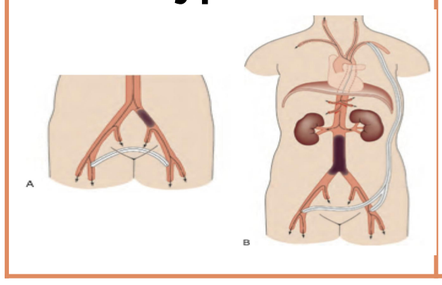
Endovascular & Surgical Intervention:

1. **Endovascular Interventions:** (balloon angioplasty with or without stenting)

- You try to do everything within the vessels.
  - The lesion is identified ⟶ crossed with a wire ⟶ balloon is inserted & inflated
    ⟶ this enlarges the lumen by disrupting the plaque
  - In patients with occlusions and complex disease, stents may be deployed
  - Drug-eluting balloons & stents reduce the neointimal hyperplasia; can lead to restenosis & occlusion
  - Favorable lesions: short concentric stenosis
  - Unfavorable lesions: long eccentric stenosis or occlusion
- There are 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.



2. Surgery Intervention

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, <u>three conditions</u> must be fulfilled: 1. There must be high-flow, high-pressure blood entering the graft (inflow) 2. The blood must have somewhere to go when it leaves the graft (outflow or run-off) 3. 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.
Extra Anatomical Bypass 	Lesser procedures, and preferred in high-risk patients or those that have a limited life expectancy. (Don't 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 Do it if we can't open the abdomen, so that is why we should check all pulses including the upper limb.

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.

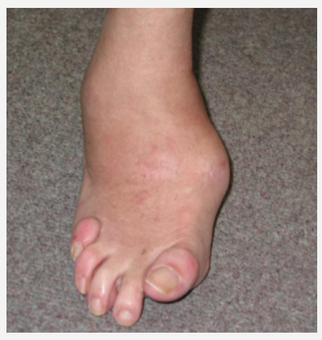
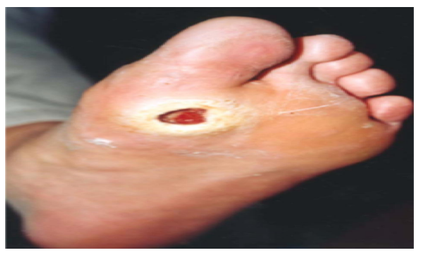
Choice of Treatment

Decided depending on:
Patient's symptoms, comorbidities, life expectancy, risk & benefits, anatomy of the disease & prior interventions

Diabetic Foot

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

Diabetic Nephropathy

Sensory 	• Patient is incapable of feeling pain • Affects proprioception such that when walking pressure is applied at **<u>unusual sites</u>** ○ This leads to ulcer formation and joint destruction (Charcot's Foot) loss of the arch • 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.
Motor	• 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	• 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 main goal of treatment

- 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 :

➔ **Depends on the history and the physical exam and it is similar to PAD**

DF Management :

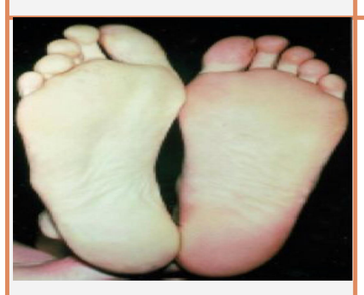
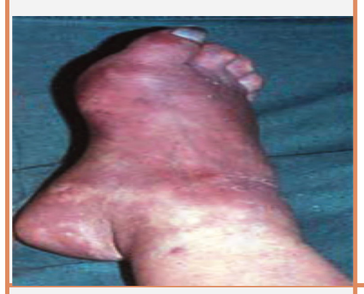
- **If the blood supply to the foot is adequate**
  - Excise dead tissue
  - Control the Infection
  - Protect the foot from pressure (off-loading) doesn't heal if there is persistent pressure
- **If there is ischemia**
  - The priority is to revascularize the foot, if possible
  - Many patients present late, with extensive tissue loss and unreconstructable disease accounting for the very high amputation rate

Acute Limb Ischemia

Etiology	• Acute limb ischaemia is caused most frequently by:
	  ○ Acute thrombotic sudden occlusion Of a pre-existing stenotic arterial segment (60%) and Embolism (needs immediate treatment) (30%)
	  ○ Trauma.
	• Distinguishing between thrombosis and embolism is important because investigation, treatment and prognosis are different (ALI is acute patient can exactly tell when and where)
	• More than 70% of peripheral emboli are due to **AF**
	• Thrombosis in situ may arise from
	  ○ Acute plaque rupture & Hypovolemia
	  ○ Increased blood coagulability (e.g, in association with sepsis, malignancy)
	  ○ Pump failure (e.g, cardiac event)

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

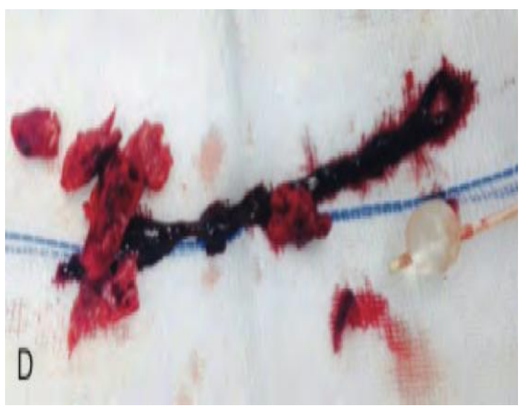
Signs & Symptoms **the 6P's**	• **P**ain (absent in complete acute ischemia & severe pain in chronic ischemia) • **P**aralysis* (inability to wiggle toes or fingers) • **P**allor (feature of chronic ischemia) • **P**aresthesia* (loss of light touch over the dorsum of the foot or hand) • Poikilothermia (impaired regulation of body temperature of the limb usually cool, reflecting the ambient temperature) • **P**ulselessness (feature of chronic ischemia) *Loss of function is the are the most important feature of ALI and denotes a threatened limb that is likely to be lost unless it is revascularized within a few hours

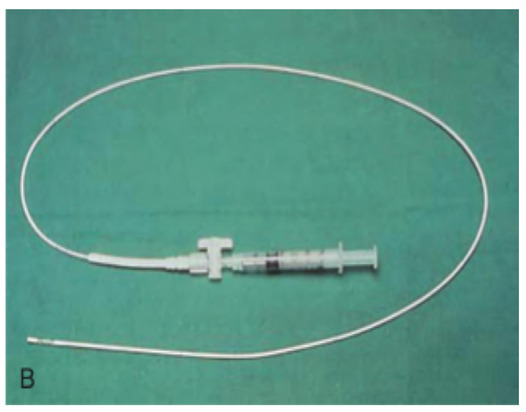

Clinical Features
 
	• Acute complete ischemia is associated with intense distal arterial spasm and the limb is 'marble' white
	  ○ As the spasm relaxes over the next few hours and then fills with deoxygenated blood, mottling appears
	  ○ This appears light blue or purple has a fine reticular pattern, and on pressure, so-called non-fixed mottling (limb is salvageable)
	• As ischemia progresses, blood coagulates in the skin, leading to fixed mottling that is darker in colour and does not blanch
	  ○ Blistering and liquefaction
	  ○ Attempts at revascularization are futile and will lead to life-threatening reperfusion injury

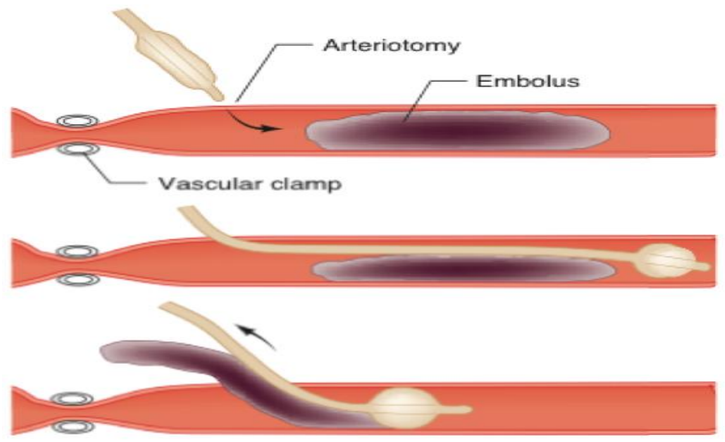
Management	• Must be discussed immediately with a vascular surgeon
	• Blood work, ECG, and cross match
	• If there are no contraindications, IV heparin (5000-8000 IU) is administered
	  ○ To limit propagation of thrombus and protect the collaterals
	• If ischemia is complete: The patient proceeds for **embolectomy** (surgery)
	• If ischemia is incomplete:
	  ○ Preoperative imaging is obtained (simple embolectomy or thrombectomy is unlikely to be successful)
	  ○ Preoperative optimization (control their BP and glucose level before surgery)
	- 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 Embolism

- ★ 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
- ★ <u>Clinical findings:</u> No previous symptoms of arterial insufficiency, obvious source of emboli, sudden onset, severe ischemia, normal pulses in the collateral leg & no signs of chronic ischemia
- ★ 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%

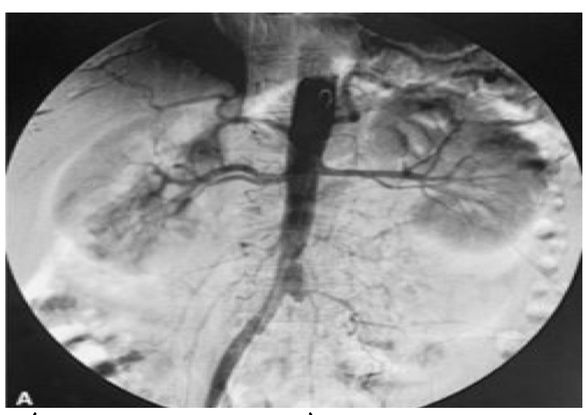




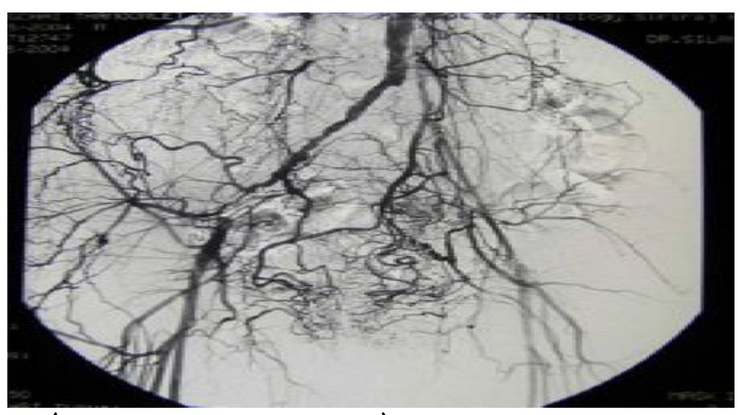


Thrombosis In Situ

- ★ Generally occurs in vessels affected by pre-existent atherosclerosi
- ★ Ischemia is often less severe than with acute embolism
- ★ Location of occlusion may play a role in the severity of limb ischemia
- ★ <u>Causes include:</u>
  - ○ Pump failure (e.g., silent or overt MI)
  - ○ Hypovolaemia, which may be associated with widespread thrombosis
  - ○ Increased blood coagulability (e.g. sepsis, malignancy)
- ★ <u>Clinical findings:</u>
  - ○ Previous claudication, no source of emboli, long history, less severe ischema, lack of pulses in the contralateral leg, & positive signs of chronic ischemia.
- ★ 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%
- ★ 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



(Acute embolus) no collaterals



(Acute Thrombosis) lots of collaterals

Post Ischemic Syndrome

Compartment Injury (local)

- Endothelial cell injury leads to increased permeability
- The calf muscles are confined within tight fascial compartments
- The increase in interstitial tissue pressure → muscle necrosis despite adequate arterial inflow: Compartment syndrome (>25mmHg) pressure in compartment is higher than capillary pressure
- There is swelling and pain on squeezing the calf muscle or moving the ankle
- Palpable pedal pulses do not exclude compartment syndrome
- **Management:**
  - Prevention through expeditious revascularization
  - Low threshold for fasciotomy to relieve the pressure (open the fascia to let the muscles breath)

Patient who has acute limb ischemia for up to 6 hours.

- First of all, his muscles will be having an 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

Reperfusion Injury (systemic)

- Caused by activated neutrophils, free radicals, enzymes, hydrogen ions, carbon dioxide, potassium and myoglobin released from reperfused tissue
- 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.
- Leads to:
  - Acute Respiratory Distress Syndrome (ARDS)
  - Myocardial stunning (hyperkalemia)
  - Endotoxemia
  - Acute Tubular Necrosis
  - Multiple organ failure and death
- **Treatment (Prevention!!)**
  - **Hydrate** the patient
  - **Communication** with the anesthesiologist and intensivist
  - Protect the heart with **calcium**
  - Prevent and treat hyperkalemia before reperfusion (induce hypokalemia)
  - Correct acidosis and **produce alkalosis** in anticipation to reperfusion
  - Use **inotropic** support liberally

Cerebrovascular Disease (CVD)

- **Stroke** (contralateral side)
  - An episode of focal neurological dysfunction lasting > 24 hours, of vascular etiology
- **Transient ischaemic attack** (contralateral side)
  - Symptoms last for less than 24 hours
- **Amaurosis fugax** (embolus ⟶ ophthalmic artery ⟶ patient describes it as curtain coming down
  - Transient incomplete unilateral loss of vision, never synchronously bilateral (ipsilateral side)
  - A veil or curtain coming across the eye

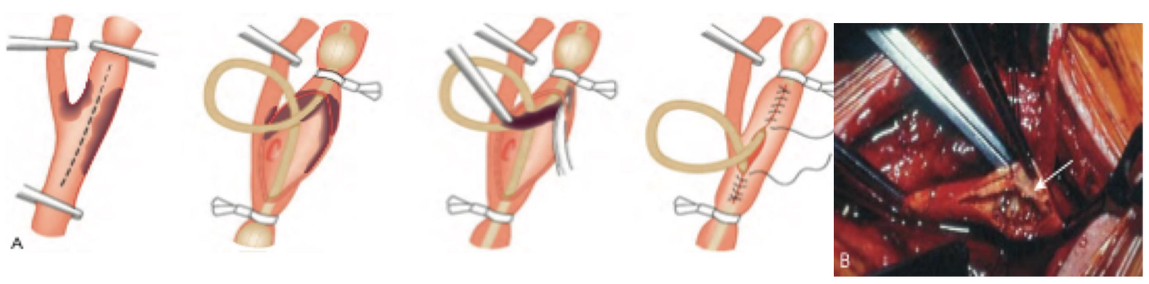
Pathophysiology	- Approximately 80% of strokes are ischemic - About half of these are thought to be due to atheroembolism from the carotid bifurcation - The origin of the internal carotid artery is most common site of atheroma formation - The tighter the degree of stenosis, the more likely it is to cause 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
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
Investigations	- Doppler (duplex) ultrasound (noninvasive) - 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** (goal is prevention!) e.g. patient underwent cardiac bypass, or fainted and we did full investigation and found 50% stenosis, how to treat? BMT to prevent stroke - Risk of developing TIA/stroke are low (< 10% at 5 years) - The Relative Risk Reduction is 50%, the Absolute Risk Reduction 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

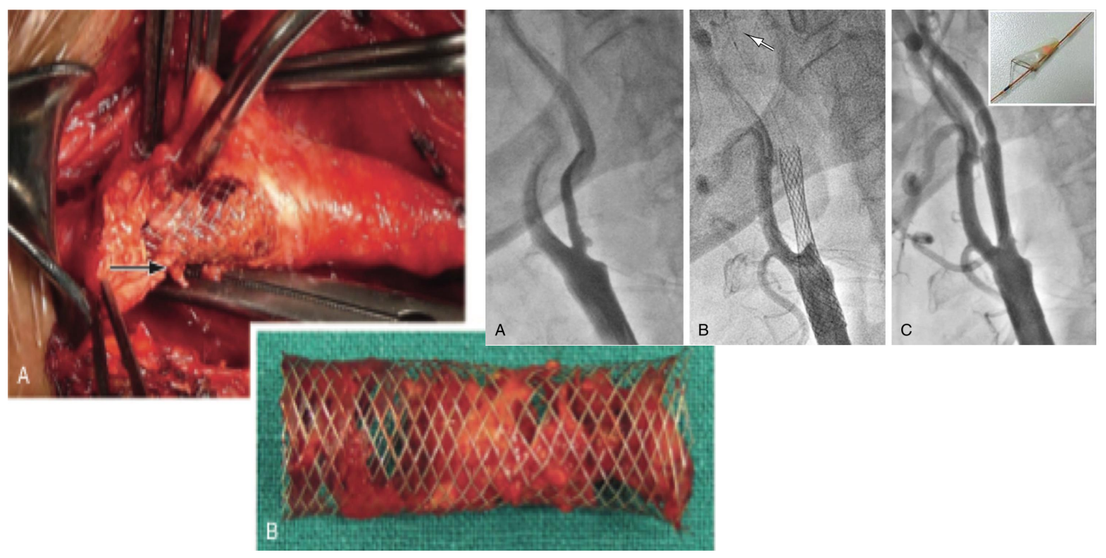
We first heparinize the vessel to prevent the blood from coagulating ⟶ put a shunt in the vessel ⟶ clean the area of atherosclerosis ⟶ close it primarily or put a patch

- ICA stenosis (> 50%)
- Life expectancy of at least 2y
- Undertaken with a stroke and/or death rate of <5%
- The intervention can be performed soon
- The sooner the better (golden time is first 3-6 months)
- 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)

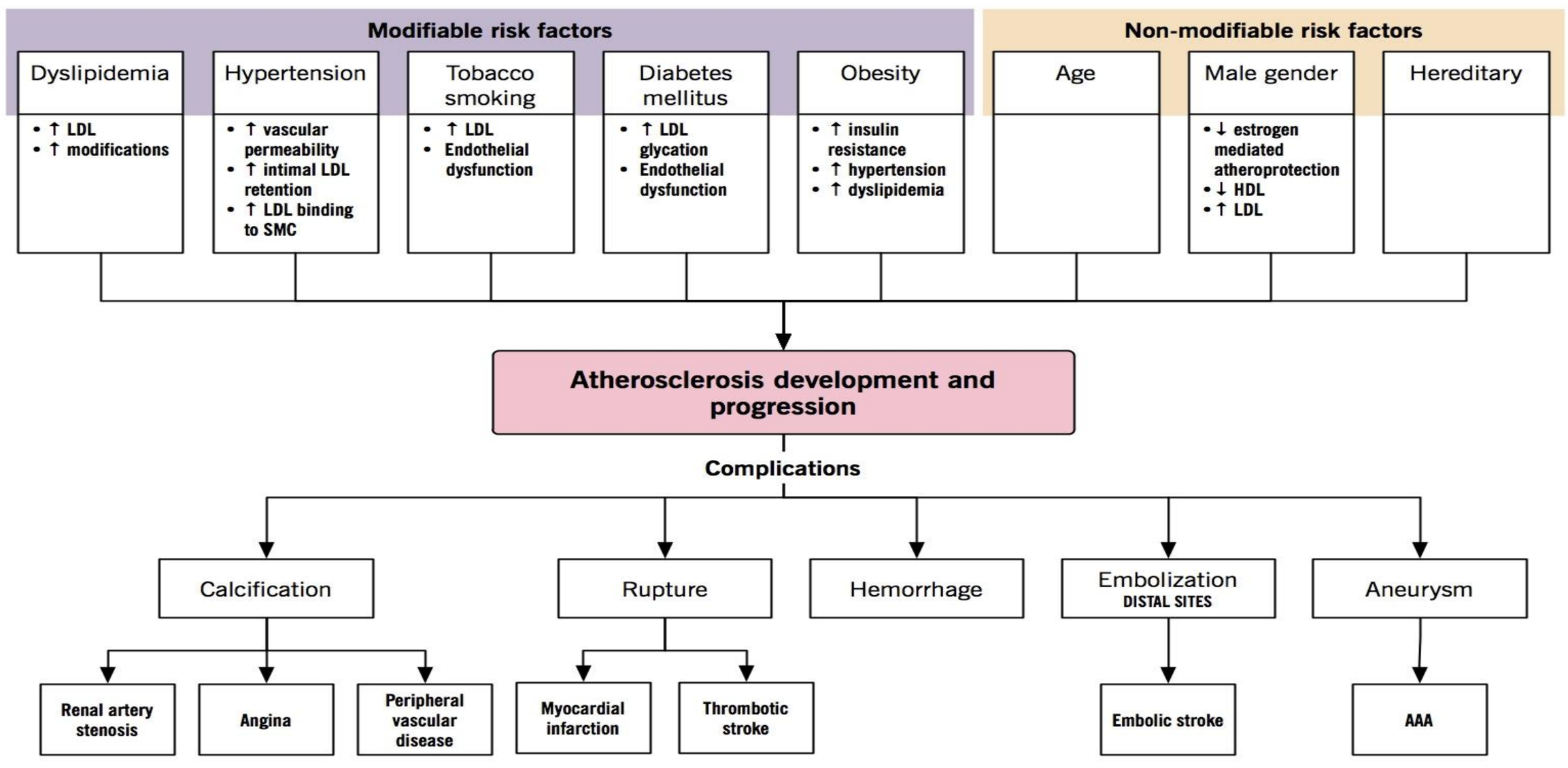
- The role of (CAS) remains controversial
- Avoids a neck wound and the risks of cranial nerve injury
- Reduces the risk of MI
- Short-term risks of clinical and subclinical strokes are greater than CEA
- CAS should be reserved for patients where CEA is not possible or desirable because of anatomic and clinical factors (e.g., recurrent stenosis after previous surgery or radiation arteritis)

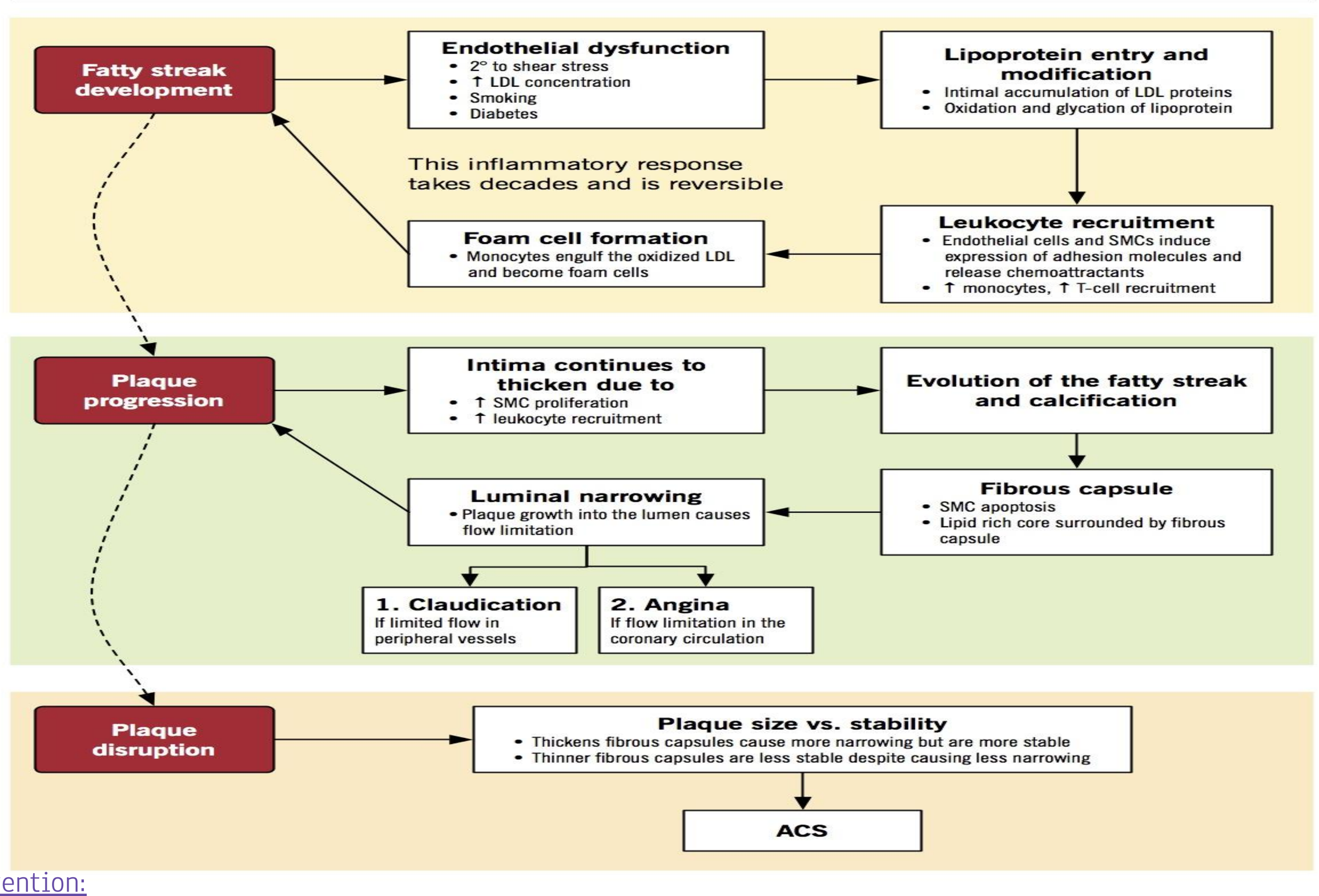


Summary

1. Atherosclerosis

Endothelial injury ⟶ platelets adhere ⟶ growth factors released ⟶ smooth muscle hyperplasia/plaque deposition





Prevention:

- 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

Summary

2. Peripheral Arterial Disease

- Chronic ⟶ intermittent claudication, critical limb ischemia or diabetic foot
- Acute ⟶ acute limb ischemia

Intermittent Claudication	Critical Limb Ischemia
- Pain comes on walking a particular distance (claudication distance) & relieved by rest - Site: aortic arteries, iliac arteries, femoropopliteal arteries, and tibial arteries - Clinical features: impotence, weakness, skin changes, toe nail changes & muscle wasting - Investigation: ABI 0.8-0.4, toe pressure <50 mmHg and segmental pressure 20 mmHg reduction	i. Rest pain, exacerbated by lying down and felt at night, patient may present with foot swelling ii. Tissue loss ⟶ ulcer or gangrene iii. Clinical findings: dry & thin skin, pallor on elevation, brittle nails, muscle wasting, reduced temperature, venous guttering, & bright red foor (dependant rubor or sunset foot) bc of reactive hyperaemia iv. Arterial ulcer: painful & pale w/ necrotic floor v. Investigation: ABI <0.5, & toe pressure <30 mmHg

- **Investigations**:
  - CBC, electrolyte, creatinine coagulation profile, type & screen, lipid profile, HbA1c, ECG, CXR, Echo, Duplex US, CT Angiogram, MRA, invasive vascular investigation, ankle brachial index, toe pressure, segmental pressure, and volume plethysmography
- **Prevention:**
  - Primary: modifiable risk factors and lifestyle changes
  - Secondary: BMT ⟶ cessation from smoking, control HTN, prescribe statin, and antiplatelet, exercise , & treat patient with DM ⟶ HbA1c <7%
- **Treatment:**
  - Indication for endovascular and surgical intervention: disabling claudication pain & CLI
  - Endarterectomy, percutaneous angioplasty and bypass procedure

3. Diabetic Foot

- The patient is immunocompromised, has neuropathy (can lead to foot ulceration) and ischemic

Sensory	Patient is incapable of feeling pain ⟶ Affects proprioception such that when walking pressure is applied at unusual sites ⟶ ulcer formation & joint destruction (Charcot's Foot)
Motor	The flexors are affected more than the extensors ⟶ toes become dorsiflexed ⟶ exposes the metatarsal heads to abnormal pressure ⟶ frequent site of callus formation and ulceration.
Autonomic	- Deficient in the sweat that normally lubricates the skin & contains antibacterial substances ⟶ scaling & fissuring - Abnormal flow in the bones due to loss of autonomic control may also contribute to osteopenia and bony collapse.

- **Prevention!:** diabetic control HbA1c <7% & comprehensive behavioral foot care education
- **Management:**
  - Good blood supply ⟶ excise dead tissue, control infection & protect foot from pressure
  - Ischemia ⟶ revascularize

4. Acute Limb Ischemia

- 60% due to thrombotic occlusion, 30% due to embolism or due to trauma
  a. Incomplete acute ischemia ⟶ thrombosis in situ (pump failure, hypovolemia & increased blood coagulability)
  b. Complete ischemia ⟶ embolus & must be treated within 6 hours
  c. Irreversible ischemia ⟶ amputation
- Clinical features (6 Ps): pain, pallor, pulselessness, paresthesia, paralysis & poikilothermia
- **Management:**
  - If no contraindications, IV heparin is administered to limit propagation of thrombus & protect the collaterals
  - If ischemia is complete: The patient proceeds for embolectomy (surgery)
  - If ischemia is incomplete: Preoperative imaging is obtained & Preoperative optimization

5. Post Ischemic Syndrome

Compartment Injury (local)	Reprefusion Injury (systemic)

- Endothelial cell injury leads to increased permeability ⟶ increase in interstitial tissue pressure ⟶ muscle necrosis despite adequate arterial inflow: Compartment syndrome (>25mmHg)
- There is swelling and pain on squeezing the calf muscle or moving the ankle
- **Management:**
  - Prevention through expeditious revascularization
  - Low threshold for fasciotomy to relieve the pressure
	- 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 & death
	- **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

Quiz

Special thanks to team 436❤️

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

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

6. Which of the following causes transient incomplete unilateral loss of vision

A. Stroke

B. Transient ischaemic attack

. Sensory Neuropathy

D. Amaurosis fugax

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

A. Paralysis

B. Paresthesia

C. Pain

D. A&B

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