Atherosclerosis:
A Surgical Look

Mohammed Al-Omran, MD, MSc, FRCSC

Associate Professor & Consultant
Vascular Surgery
King Saud University

Surgery team:
Shoog Alaqeel, Fatima AlKhashram

What is Atherosclerosis?

- It as an inflammatory process that causes clogging, narrowing, and hardening of <u>large</u> and <u>medium-</u>sized arteries

It's one of the important diseases and you have to know it very well from surgical prospective

What are the risk factors for Atherosclerosis?

Non-Modifiable Risk Factors:
Male gender
Advanced age
Family history (genetic predisposition)

Modifiable Risk Factors:

Major
Smoking
Hypertension
Diabetes 1st in KSA
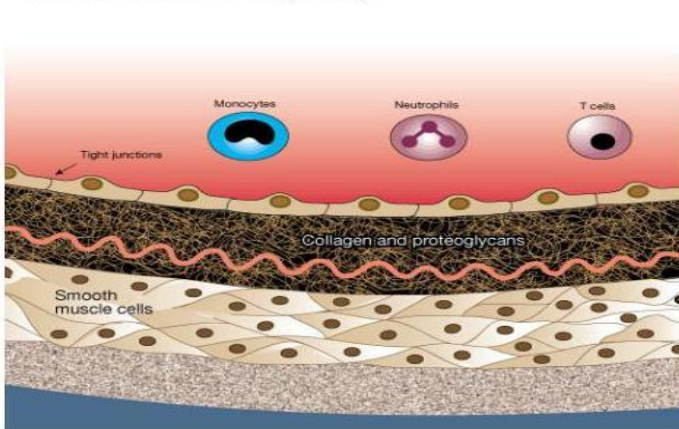Hyperlipidemia

Minor
Homocystenemia
Obesity
Hypercoaguable state
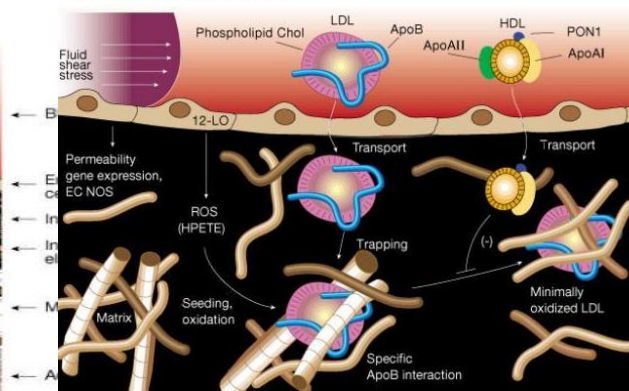Physical inactivity
(Sedentary life style)
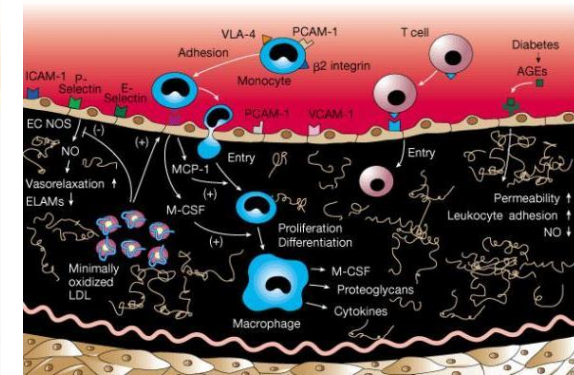
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Pathogenesis

Structure of a normal large artery
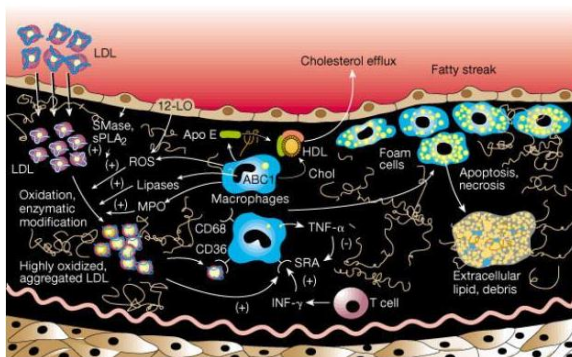

Atherosclerosis-Lesion initiation

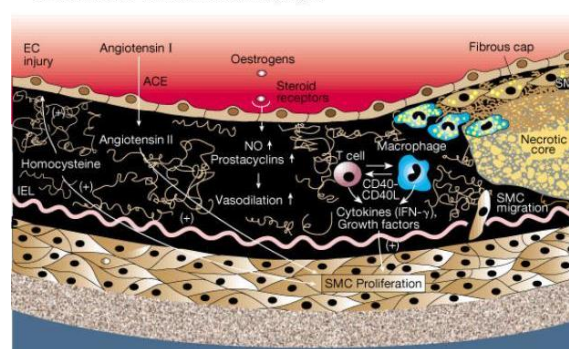Atherosclerosis- Inflammation

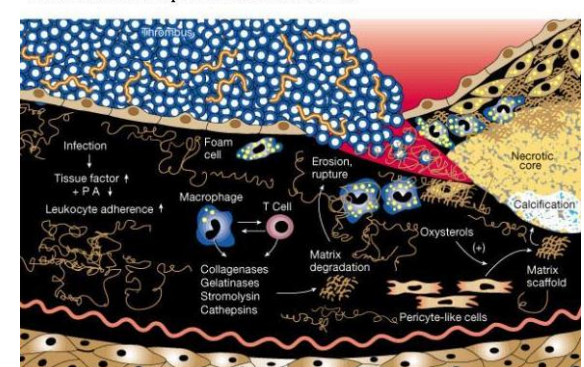The key word in the arthrosclerosis is inflammation.

Atherosclerosis-Foam-cell formation

Atherosclerosis- Formation of fibrous plaques

Atherosclerosis- Complex lesions and thrombosis

Notes

- It is an inflammation:
- There is an injury usually it related to fat droplet that injure the endothelium then get to subendothelium cell, that will initiate the inflammatory process.
- WBC will migrate from blood vessels to tissue and macrophage engulf the fat droplet and change into foam cell >>then usually we will have necrosis, that the end of the inflammatory process and the body will try to isolate the inflammation alone and here where we have the fibrous cap then it cover with Ca
- It is dangerous when its rapture

Notes

- The blood vessel consist from three layers, which are from outside : adventitia, media and intimae which covered by the endothelium .

- Blood is fluid serving three type of cell RBC, WBC and platelets, these cell have some kind of homeostasis function where they do not intervene with each other and they support each other.

- Problem with the atherosclerosis that we will have injury, and we do not know when the injury started

- The fibrous cap came from fibrin which produce by platelets , when it become older body will cover this older material with calcium, so it isolated and that what give the Hardening of the artery, the key here is that atherosclerosis is an inflammatory process.

- The problem is not in the present of the plaque or in the stenosis of the artery but when the plaque rupture, this will have rough surface in the inner layer of the artery, platelet will adhere and thrombus will form thrombosis

Pathogenesis
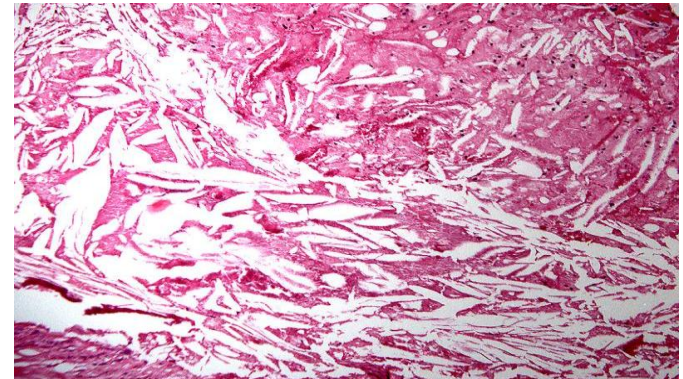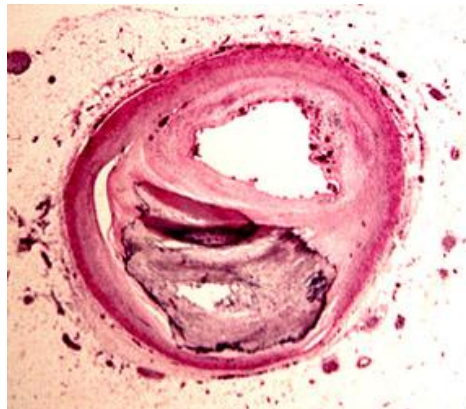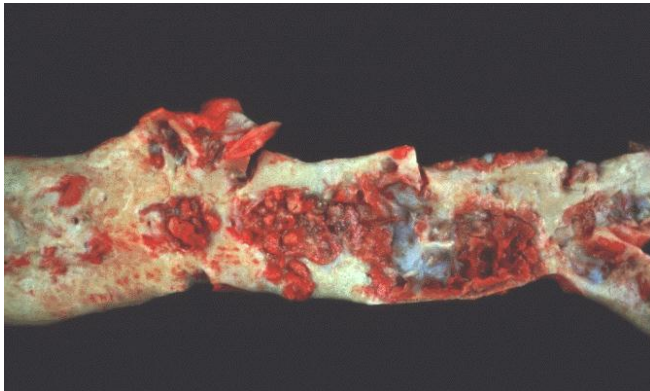
Pathogenesis

- Fat deposits accumulate and will cause endothelial injury that will initiate the inflammatory process
- Formation of fibrous plaque by platelets
- Calcification of the arterial wall (this is the cause of atherosclerosis)
- Fat by itself is NOT harmful
- Rupture of the wall will cause clotting(atherothrombosis) >> this what we are worrying about

What is the Clinical Spectrum of Atherosclerosis?

- The other message from this lecture is to put in mind that atherosclerosis is a systematic disease when its affect one area by default it affect the others.

– **Cerebrovascular disease**

– **Coronary artery disease**

– **Renal artery Diseases**

– **Visceral (mesenteric) arterial disease**

– **Peripheral arterial disease (Aorto-iliac & upper and lower limb) is a marker for atherosclerosis**

  - **Intermittent claudication**
  - **Critical limb ischemia**
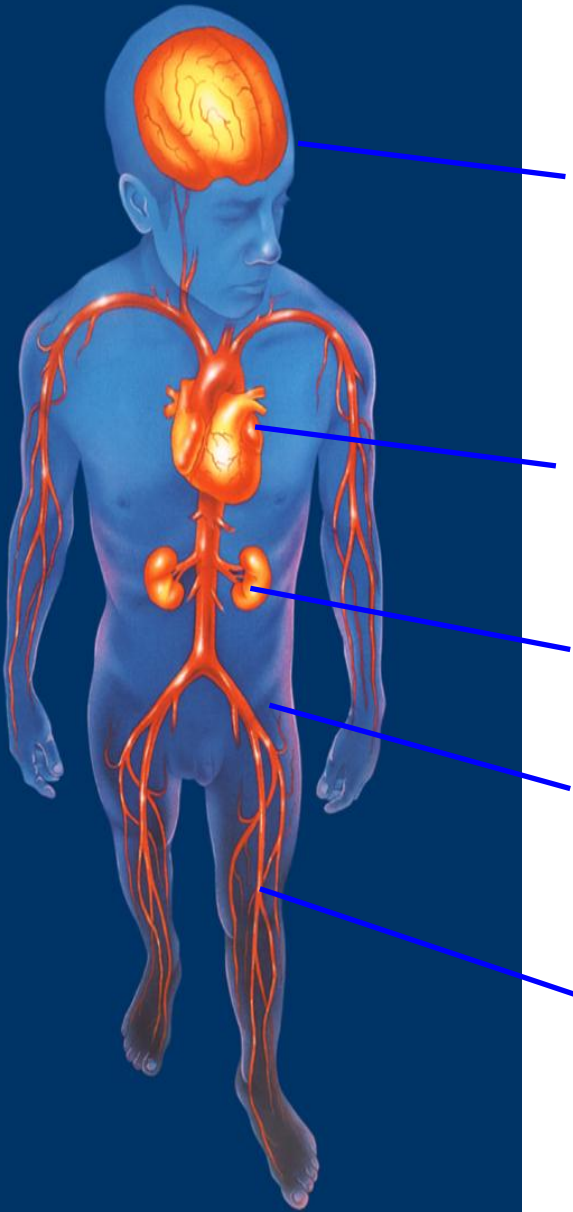
Notes

- When it affect one part of the arterial tree by default it affect all parts of the arterial tree which start in the aortic and then stat to give branches to all around the body ,each organ supplied by group of arteries , we give the circulation the name of the organ it supply (cerebrovascular circulation , coronary circulation,……..etc), so if it affect any one of this arteries the rest of the arterial tree will be affected .

- The patient usually presents with symptoms related to the organs been affected by the blood supply but it Haden the atherosclerosis is present in the rest of the tree and we give the disease name according to the circulation been affected .

What is the burden of Atherosclerosis?

Why it important to study atherosclerosis ? Because it burden of the disease, it the number one killer world wide and here in Saudi Arabia and it predicted to increase
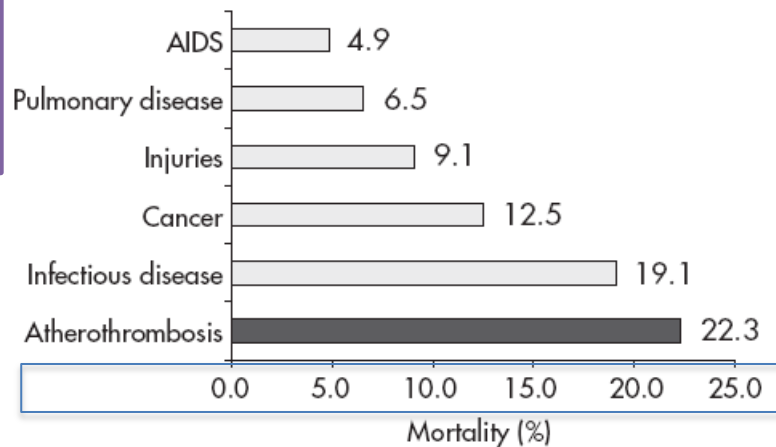

Fig. 1. Atherothrombosis is the leading cause of death worldwide. Data from the World Health Organization Report, Geneva.[3]

Let's Talk about Peripheral Arterial Disease

Now we will take system by system

The Message : any things apply to any system it apply to the others systems

Why it is important to recognize patients with PAD?

PAD is a marker of systemic atherosclerosis

Patients with either symptomatic or asymptomatic PAD generally have widespread arterial disease

it is the largest arterial tree of the body , it is a superficial for us, we can diagnoses and we can screen, it not make different if it symptomatic or not .

Why it is important to recognize patients with PAD?

- Coexisting vascular Disease:
  CAD*-- 35 % to 92%
 CVD*-- 25 % to 50%

Coronary Arterial disease
Cerebrovascular disease

Why it is important to recognize patients with PAD?

- **Cause of death:**
  CAD– 40%-60% main cause of death
  CVD– 10%-20%
  Non-cardiovascular causes--Only 20% to 30 %

- Patients with PAD have a **6 fold** (imp!) increased risk of cardiovascular disease mortality compared to patients without PAD even the patient with or without symptoms

Pts with PAD are called the walking bomb

Natural History
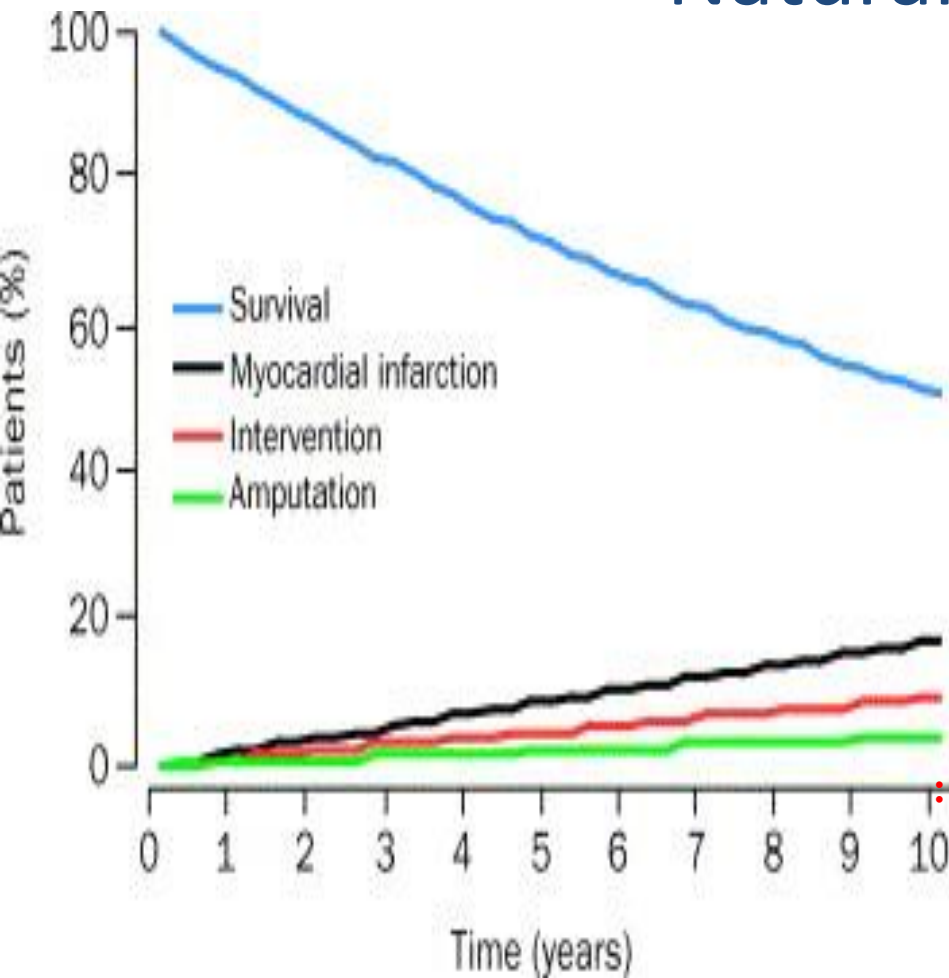
- Annual risk :
  - Mortality 6.8%
  - MI 2.0%
  - Intervention 1.0%
  - Amputation 0.4%

We always concentrate on the symptoms related to the organ it self and forget to think about the bigger picture, when we thinks about the peripheral artery disease we thinks only about the peripheral complication like claudication, amputation
In the reality we have to think about the mortality about 7% per year

Ouriel K, Lancet 2001; 358: 1257-64.

How do patients with PAD present?

Symptomatic

- Intermittent **claudication** =pain of the lower limb's ms at excretion relive by rest it is the same with angina but different location

- Critical Limb Ischemia (limb-threating condition)

  Pain at rest =unstable angina

  Tissue loss (Ulcers)

  Gangrene

Asymptomatic

Notes

- intermittent claudication is same as Angina but at different location

- So the concept I want you to keep in your mind that atherosclerosis is one disease, one symptoms and one treatment , one every things but different arterial tree so different symptomatology pts will present with, but at the end of the day they are exactly the same disease.

- Pts may have the disease sclerotic(stenosis artery) but still the blood go to the organ and even if it excreted there is no problem because the demand is meet , but if dose not meet only in at excise that what we called intermittent claudication and relief by rest and if it reach to the next stage which is pain at rest it called critical limb ischemia and if it happen in the heart it called (unstable angina) ,in this stage the organ still function but we enter to the stage of the critical ischemia, when move to the next stage where the functions and the integrity of the cell will go(cell die) , in the leg gangrenes or ulcer will result and in the heart MI will occur and that how the symptoms will appear.

How do patients with PAD present?

Lower Limb Ischemia Resulting from Peripheral Arterial Occlusive Disease

Peripheral arterial occlusive disease

Atrophy of the calf muscle

Gangrene of the digits

Loss of hair over the dorsum of the toes

Arterial ulcers on the heel and tip of the toe

How do we diagnose PAD?

Symptomatic

History: **The better the history we take the better physician that we are, it like the detective we gather the information**

Physical Examination/ Hx and PE represent 90 %of the diagnosis

ABI measurement

Non-invasive tests (arterial duplex, CTA, MRA)

Invasive test (Conventional angiogram) (the gold standard)

Asymptomatic screening

Note: The same things we apply to the heart except the ABI measurement which is specific for the limb

ABI measurement

Investigation usually to :
1-confirm the diagnosis (NOT for diagnosis)> To exclude or include certain ddx
2-to inform us about the severity> category the disease that I have

How do we diagnose PAD?
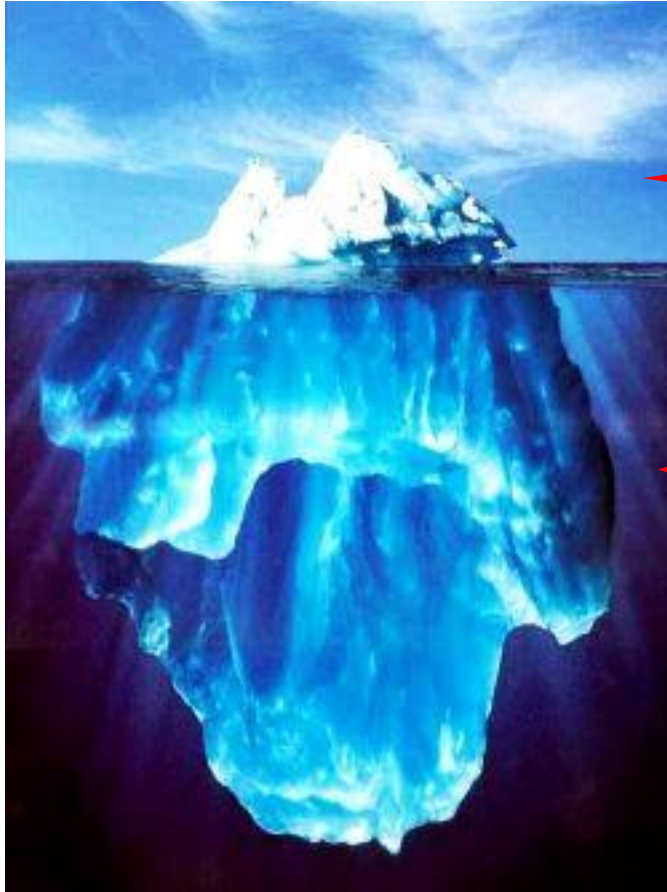

Symptomatic 10%

Asymptomatic 90%

Even Asymptomatic patients Carrie the same risks of symptomatics

Notes

- THE PROBLEM WE HAVE IS WITH ASYMPTOMATIC patient , what we do with them ?

- We screen the high risk people even when they do not have symptoms, by default we have to screen people with high risk factor(age more than 50, Fx, male),

- we do the screen by the ABI measurement

- Remember the slide of the percentage, why that we have 7% mortality compared to only 1% for the peripheral intervention ? That because 90% of the PAD pts are asymptomatic and that decrease the intervention

- So What we seen is only the tip of the ice Berge , so we really have to go and search for those 90% to decrease the mortality and the cardiovascular mortality and morbidity associated with those pts.

Ankle Brachial Index
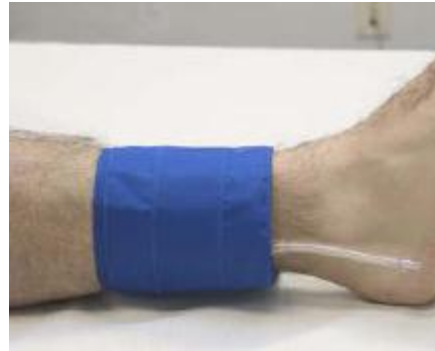
ABI= <u>Highest</u> Ankle Systolic BP (PT or DP) / <u>Highest</u> Arm Systolic BP

- It is the index between the ankle and the brachial pressure, only the systolic
- In the ankle we have PT and DP we measure both and take the highest one of them

PT : posterior tibial artery
DP : dorsalis pedis artery

Ankle Brachial Index normally > ,9

- It is very sample to do the ABI
- any thing below 0.9 this qualify for PAD you start to do every thing related to management that will reduce the mortality and save the money for the care health system.

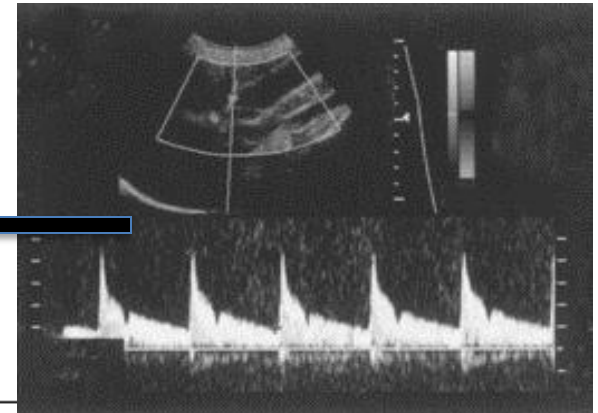| ABI value	Indicates
<0.9	Abnormal [MCQ]
0.8- 0.9	Mild PAD
0.5- 0.8	Moderate PAD
<0.5	Severe PAD
<0.25	Very Severe PAD

The ABI has limited use in evaluating calcified vessels that are not compressible as in Diabetics
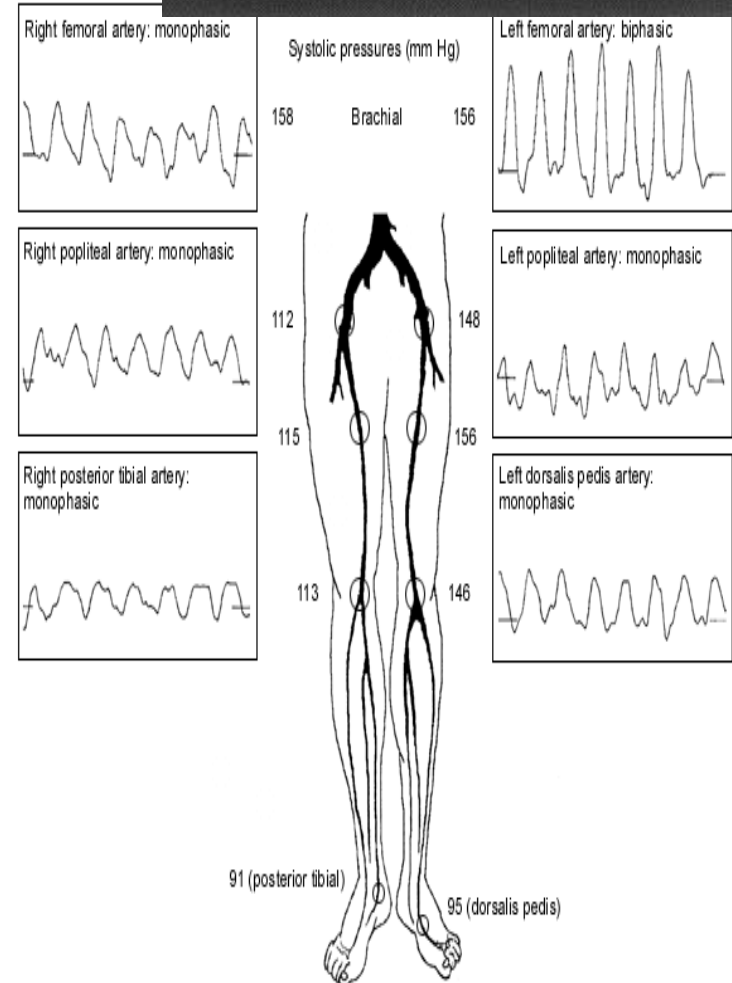
Arterial duplex
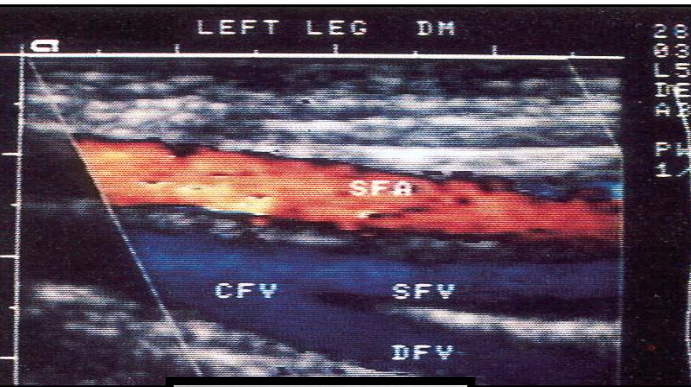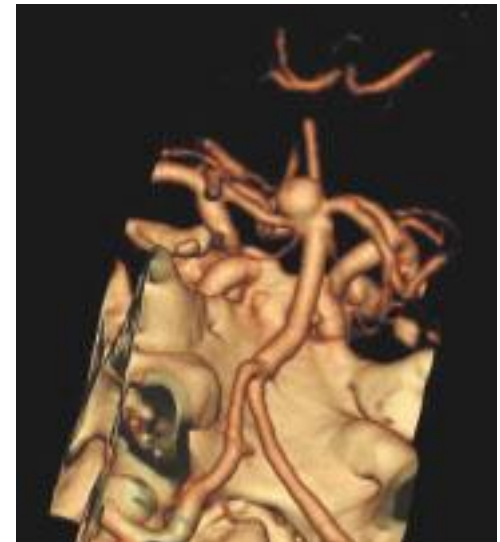

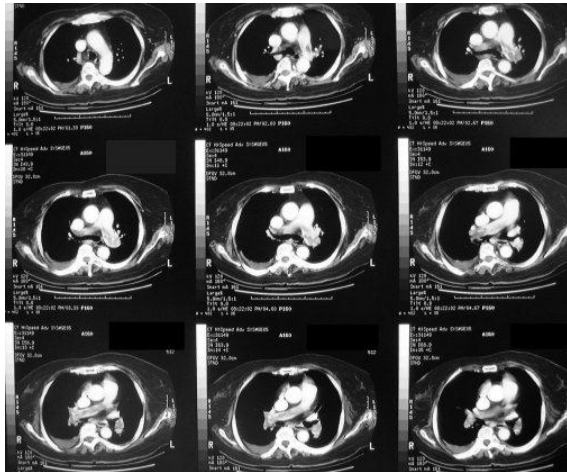**Doppler**
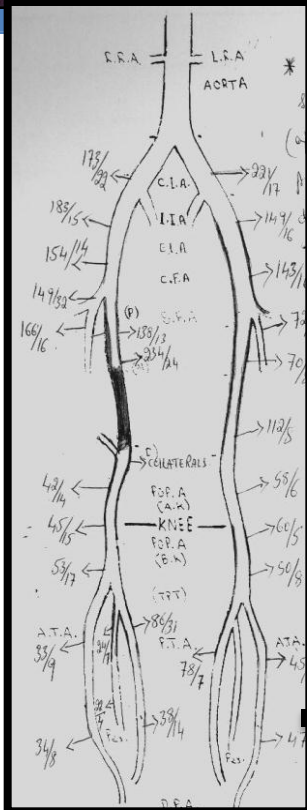
Anatomical function

Notes

Duplex :It a US and Doppler

- the US Give us the anatomy ,we draw the anatomical map and go over the arteries to see where is the occlusion

- Doppler see the waveform of the arteries , each waveform have a certain indication (monophasic ,biphasic and triphase) that will indicate something about the fluid in the arteries> u do not have to know that !

CTA

The usual CT but we give contrast

Angiogram

very accurate in mapping artery but Doppler better in dynamic

It is invasive, we go with the needle in to the artery so it will give good anatomy but not that much of the dynamic issues duplex will give more dynamic

What are the Goals of treating patients with PAD?

This four goals apply to all the disease, but The strategy to how achieve this goal it will be different

- Relief symptoms

- Improve quality of life (or keep the quality of life)

- Limb salvage (Preserve the organ function)

- Prolong survival

Strategies in treating patients with PAD

We have tow major strategies :

Risk Factors Modification

The modifiable factor have to be control

- Strategies mean that we have to pack one or two

- The less strategies we have the better we achieve

Improve Lower Limb Circulation

# Risk Factors Modification

- Diet and weight control
- Exercise

Antiplatlets (to prevent the thrombus when the artery is ruptured) prevent the adherent of the platlets in case rupture happen

- Hypertension control <140/90 (130/80 for diabetic)
- Diabetes control H1c < 6
- Lipid control < 2.5
- Smoking Cessation

Strategies in treating patients with PAD

Improve Lower Limb Circulation

- Conservative (Exercise Program) promote angiogenesis
- Intervention (Revascularization)
        - Angioplasty +/- Stenting (inside the artery)
        - Surgical Bypass (outside the artery)
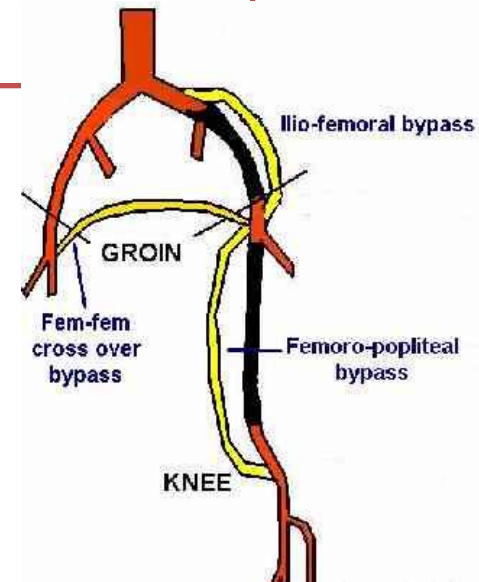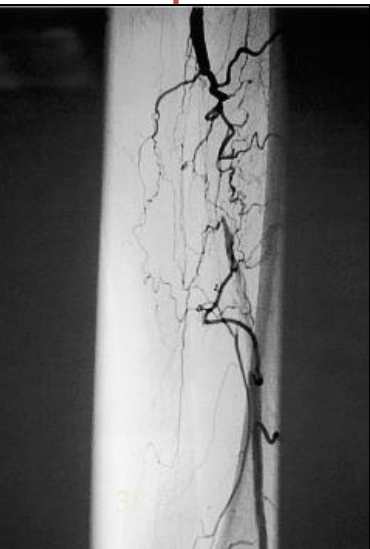
Notes

- Why the excises is important ? to promote angiogenesis
- So we will have more collateralization around the artery that's the main idea and as well with walking (we feel the pain because of the lactate irritate the nerve ending) so it will increase pain threshold so they walk more
- Same strategies we do it for the heart:
- Excises for angina
- Unstable angina we start to do cath
- We check if we can manage the pts vascular by angioplasty and stent if does not we move to bypass

Percutanous Transluminal Angioplpasty PTA

Surgical Bypass

Last Strategy in treating patients with PAD

- **Major amputation :** **(it affects the functionality e.g. whole leg amputation)**

1-Primary (we start with amputation)

2-Secondary (we start with angioplasty or Bypass but the patient does NOT response)

- Minor vs BKA vs AKA (doesn't affect the functionality e.g. toe amputation)

BKA: Below the knee amputation
AKA: Above the knee amputation

- Functions of LL : walk and stand.
- Below knee or above it major amputation.
- Primary: my first chose is the amputation .
- Secondary: we start with vascularization if it fail for what ever reason we move to amputation.
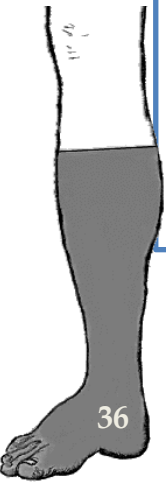
NOW

Let's Talk about Carotid Artery Disease

Why it is important to recognize patients with CAS?

- Stroke is the **third** leading cause of death and a principal **cause of long-term disability** in much of the western countries
- Prevention of stroke is more IMP than treatment

Healthy

Plaque build up

Carotid artery

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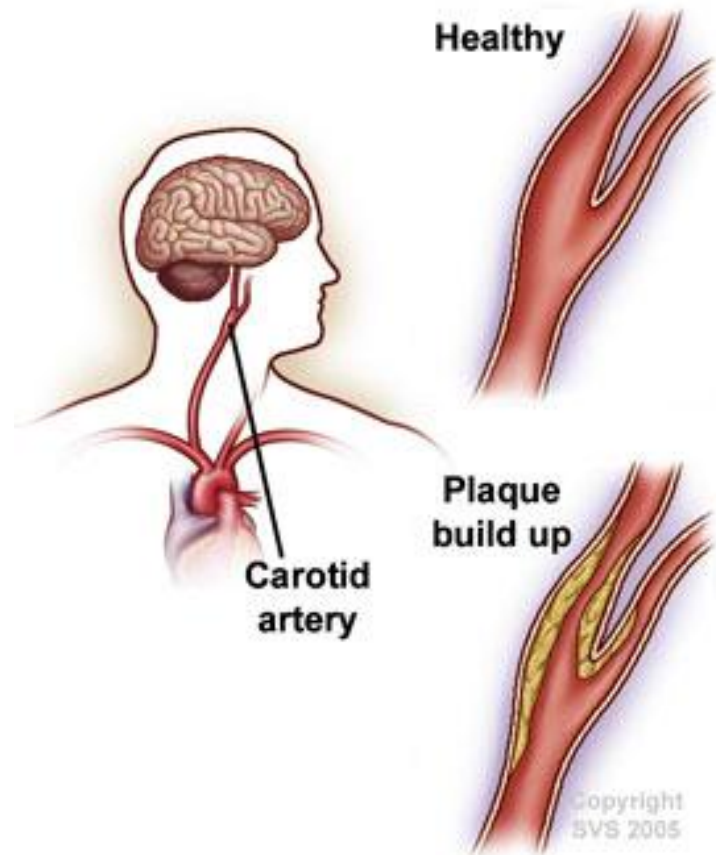
How do patients with CAS present?

Symptomatic

- Transient Ischemic Attacks (TIA) loss of the motor or sensory function for <u>less than </u>24 hours
- Amurosis Fugax (Transient Visual Loss)less than 24 hours
- Stroke <u>more than </u>24 hours

Asymptomatic

المريض يقولك فجأة وانا أتقهوى طاحت أيدي أو ممكن وهو يتكلم مال لسانه لخمس دقايق وبعدها رجع ، او انا قايم لصلاة خانتني رجلي وطحت
بالنسبة للعين: اسأله ما حسيت بغباشة على عينك فجأة أو فقدت النور عن عينك

How do we diagnose CAS?

Symptomatic
  History
  Physical Examination
  Non-invasive tests (arterial duplex, CTA, MRA)
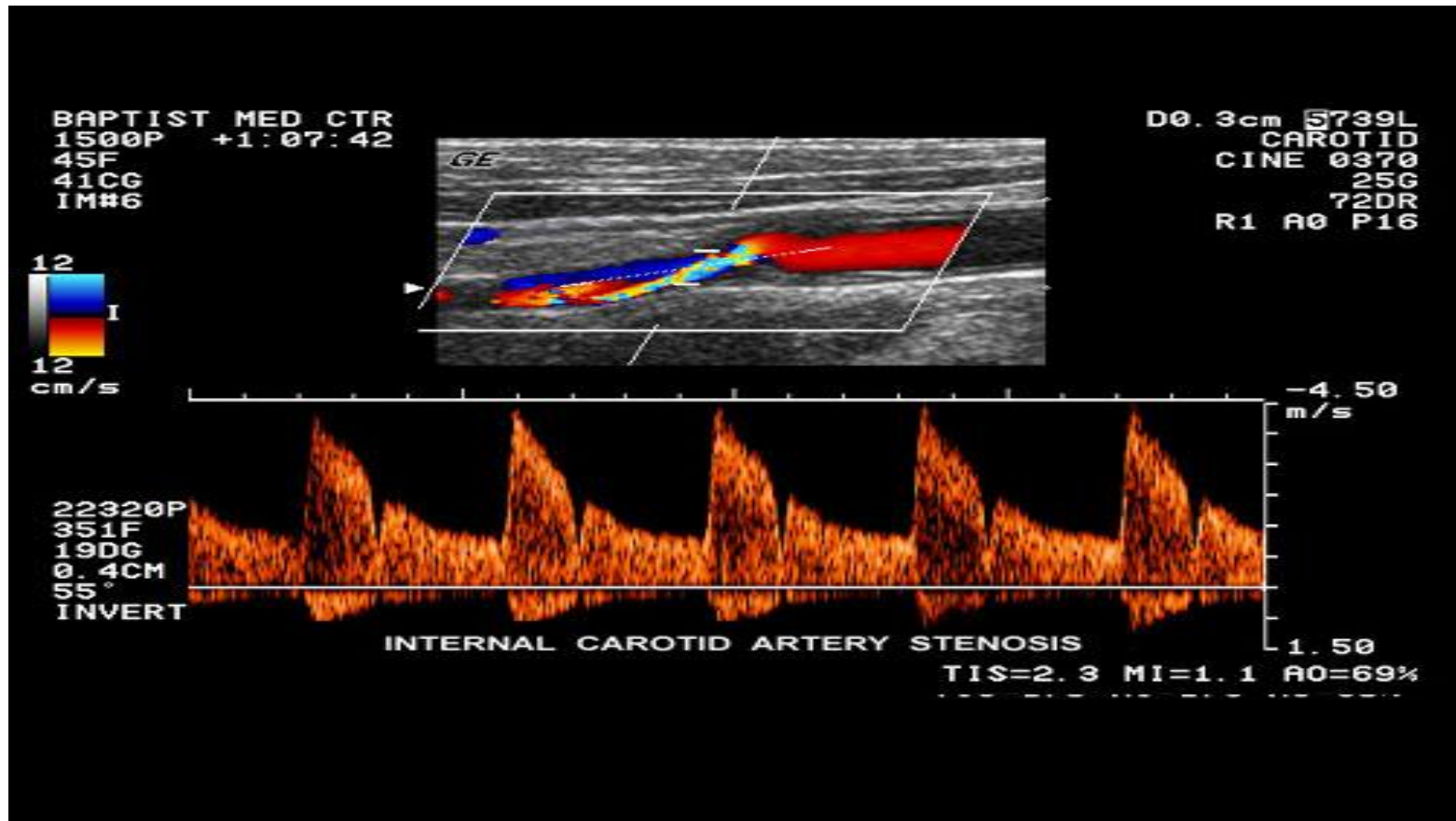 Invasive test (Conventional angiogram)

Asymptomatic IMP

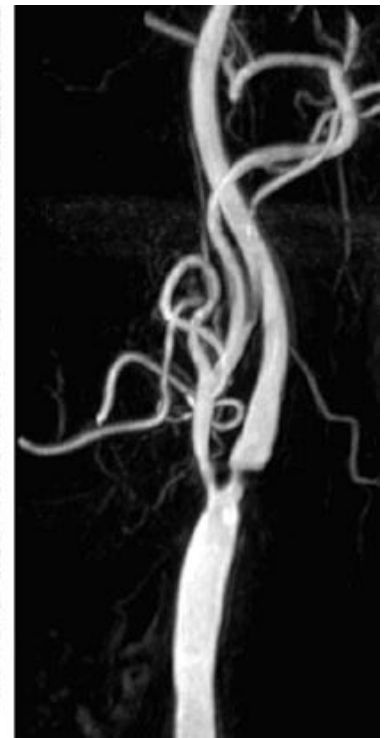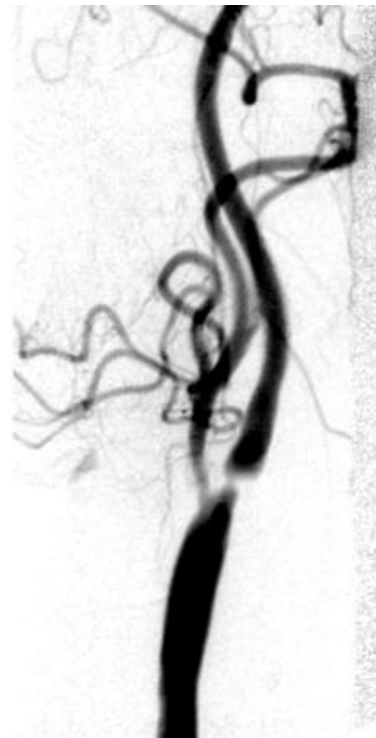Carotid Bruit plz use ur stethoscope for pt above 50 y/o
  Arterial duplex

Asymptomatic we screen them by using the stethoscope , plz plz plz do it

Arterial duplex

- **Stenosis is determined by measuring Velocities NOT anatomical diameter**

Angiogram

What are the Goals of treating patients with CAD?

- Prevent Stroke

- Prolong survival

Strategies in treating patients with CAD

Risk Factors Modification

Improve Brain Circulation

Risk Factors Modification

- Diet and weight control
- Antiplatlets double dose sometime
- Exercise
- Hypertension control
- Diabetes control
- Lipid control
- Smoking Cessation

Strategies in treating patients with CAS

Improve Brain Circulation
- Intervention (Revascularization)
  - <u>Carotid Endarterectomy</u> (the best method to increase the brain circulation)
  - Angioplasty +/- Stenting

- There is no exercise for the brain so we do the intervention directly.
- Stenting (from inside)still need evidences (we reserve it for certain group of pts) .
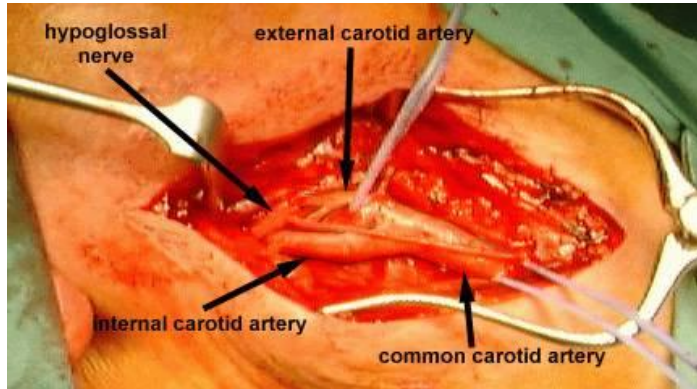
What are the indications to intervene?

Symptomatic

- \> 70% stenosis- NACET

    Decrease Stroke at 2 years from 26% to 9%
- 50-69% stenosis- marginal benefit, greater for male
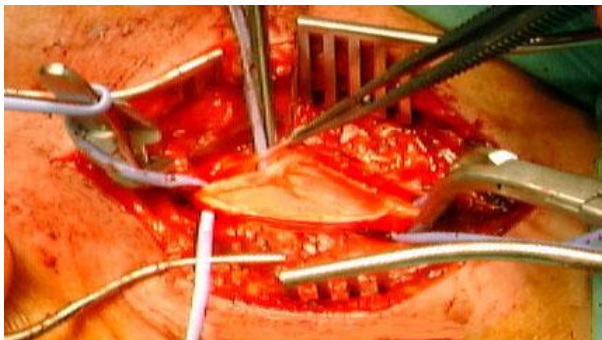- Recovered Ischemic Stroke Patients

Asymptomatic

- \> 60% stenosis- ACAS

    Decrease Stroke at 4 years from 11% to 5%
    (should be done in high volume centers only)

Carotid Endarterectomy: The Standard of Care

Open carotid and take out plaque and then close the carotid

Carotid Angioplasty and Stenting

**This interventional procedure is currently under investigation**

Relative Indications
- Hostile Neck
- Hostile Carotid Disease
- As part of a Randomized Clinical Trial

Carotid Angioplasty and Stenting

We reserve it for the redoes-hostile neck /hostile carotide- (some one who already do carotid Endarterectomy and the stenosis get back so it's difficult to do it again , or patient with radiation or have high lesion in the carotide here the surgery is so difficult to do) this indication for the angioplasty

Carotid Angioplasty and Stenting

Arm insertion point

Groin insertion point

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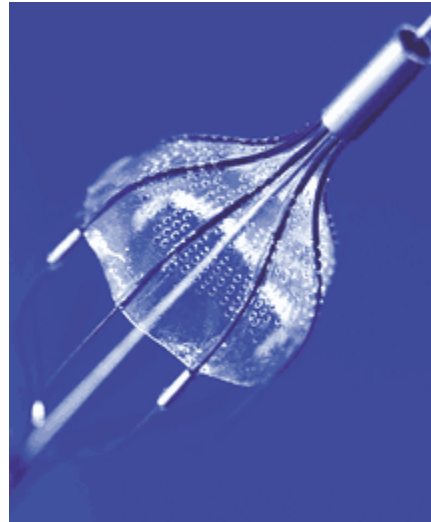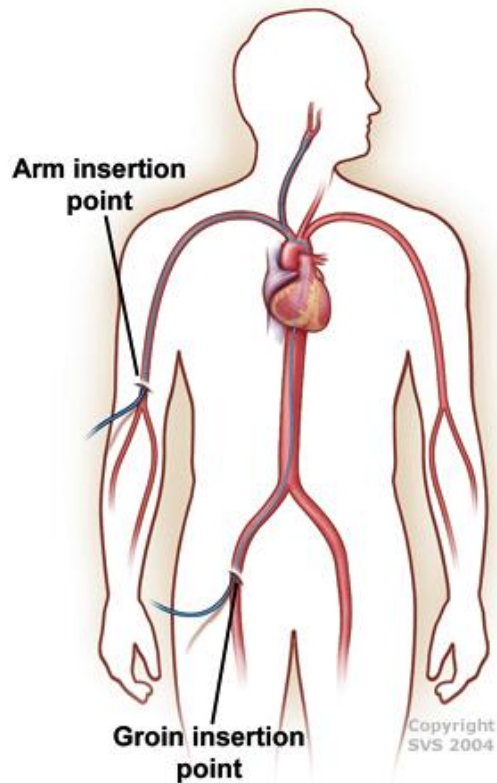
Carotid Angioplasty and Stenting

Acute Limb Ischemia

What is an Acute Limb Ischemia? IMP

- Sudden(there is no time) decrease or worsening in the limb perfusion causing a potential threat to the limb viability resulting from a sudden obstruction of the arterial system

What are the causes of acute arterial occlusion ?

- Embolus the commonest cause MCQ
- Thrombosis
- Others

- Trauma > واحد جاي منطعن وانقطع الشريان

- Iatrogenic (the orthopedic works in hip and by mistake he removes the femoral artery)

  - Arterial dissection

What is the possible source for an embolus?

Spontaneous (80%)

Cardiac source commonest cause [MCQ]

arrhythmias, MI, prosthetic valve, endocarditis

Non-Cardiac source

Proximal AS plaque, Proximal Aneurysm, Paradoxical emboli

Iatrogenic (20%)

Angiographic manipulation
Surgical manipulation

Notes

- Classic example : patient has MI>arterial fibrillation >form clot inside the heart the start to showering

- After this clot get out from the heart it goes to circulation and close one of the arteries.

- What is the commonest arties ? Common femoral artery

What are the common sites for embolus lodgment in the arterial tree?

Upper extremity 14%

Visceral 7%

Aortoiliac 22%

Femoral 36%

Popliteal 15%
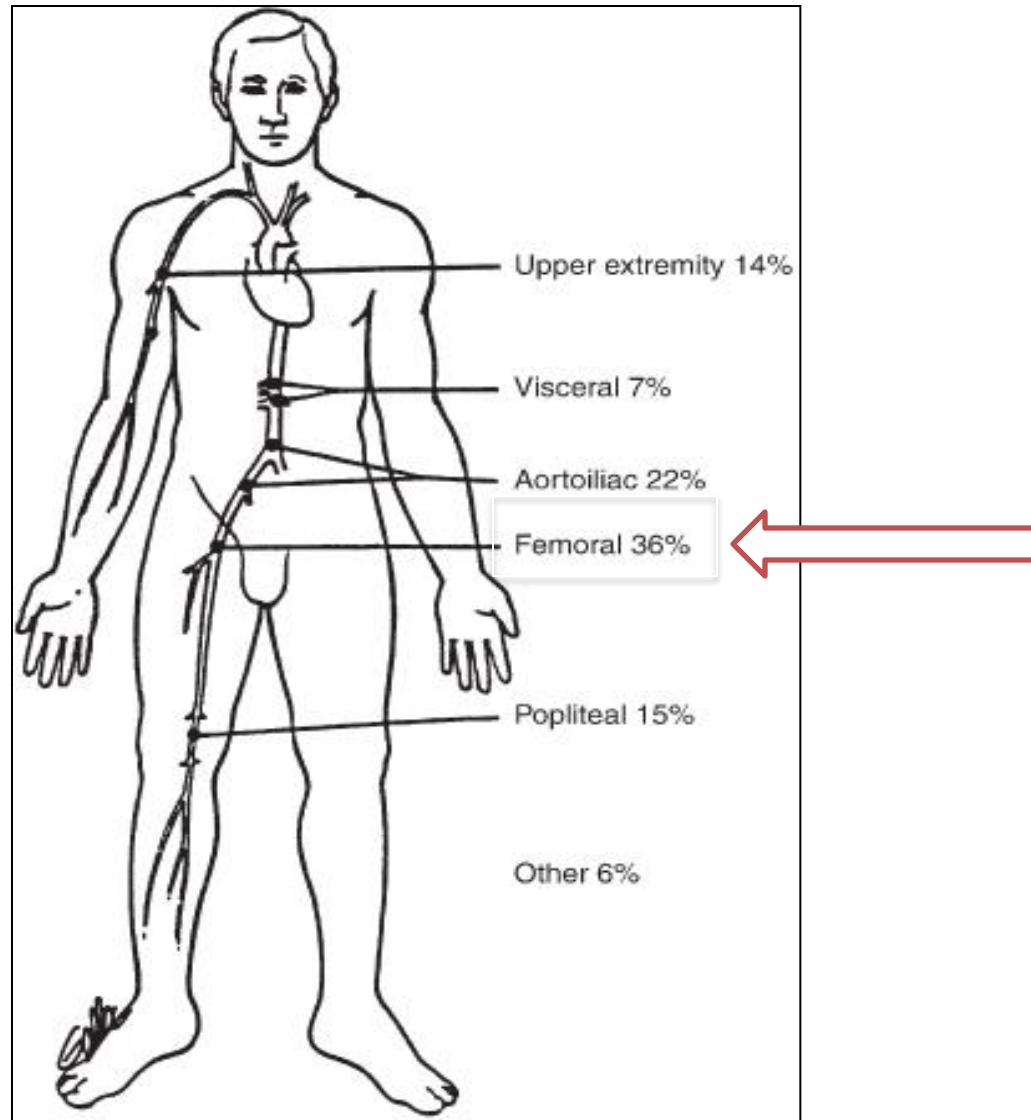
Other 6%

How do patients with acute limb ischemia present?

- Sudden onset of diffuse and poorly localized leg pain
- 6 Ps imp!
  Paresthesias (no blood to the nerve)
  Pain(coz there is no blood)
  Poikilothermia (coolness)
  Pallor (no blood to the skin)
  Pulselessness(no blood go to the arteries)
  Paralysis(no blood go to the muscle)

Investigations

- Acute Limb Ischemia is a **CLINICAL DIAGNOSIS** *(coz there is no time to do investigation)*

- If time allows, especially if atherosclerotic thrombosis is suggested, preoperative angiography is often wise

Goal of treating patients with Acute Limb Ischemia

- Rapid restoration of adequate arterial perfusion without the development of morbid local or systemic complications

Preserve the limb but not in the expense of life

Treatment

- *EMEGENCY (Golden time is 6 hours from the appearance of symptoms)*
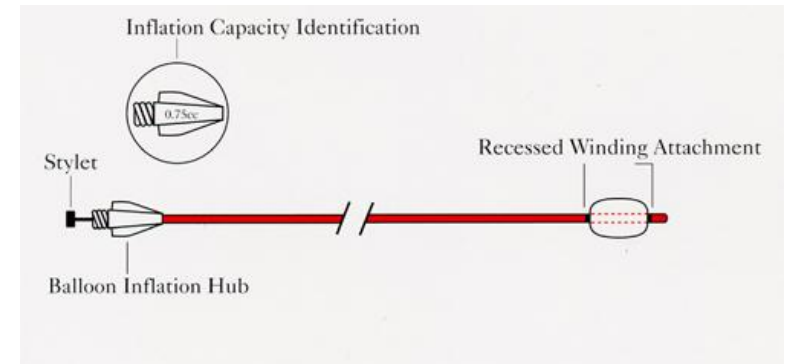 - ABC> this is the most important
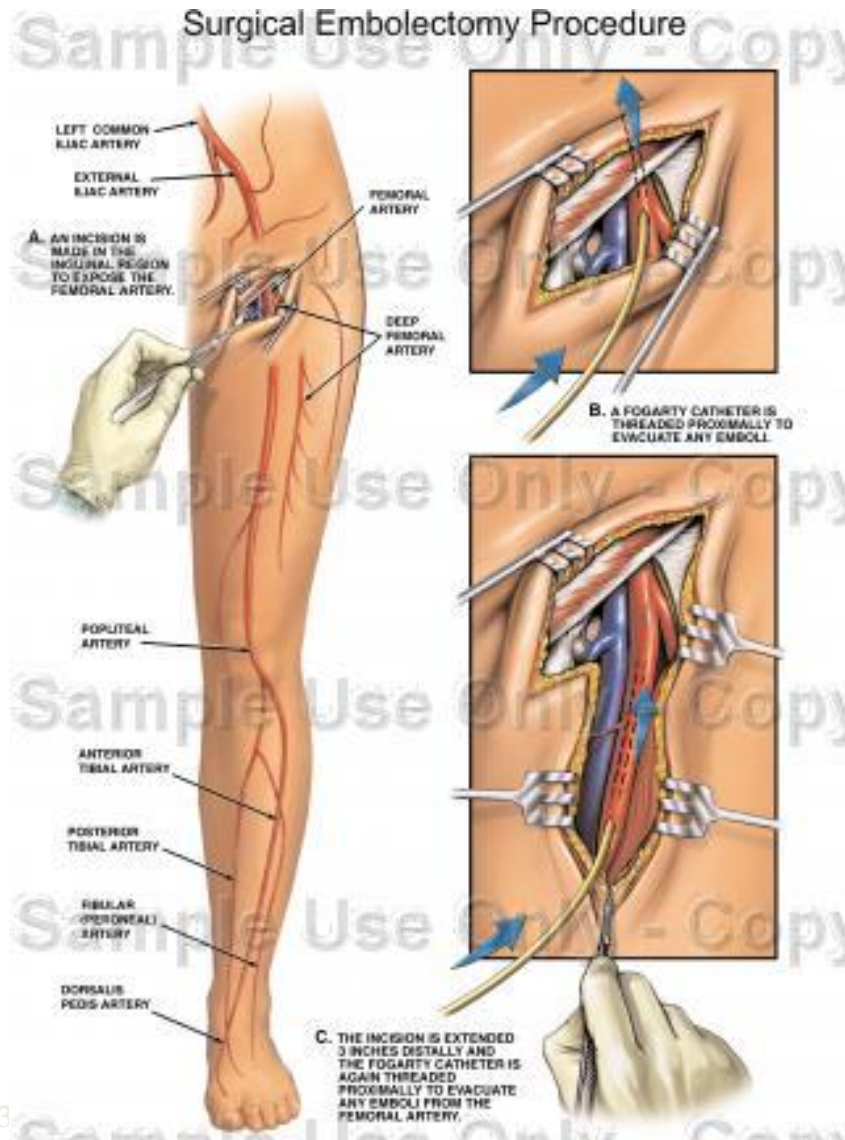 - IV Heparin (anticoagulation)
  - Rapid surgical thromboembolectomy
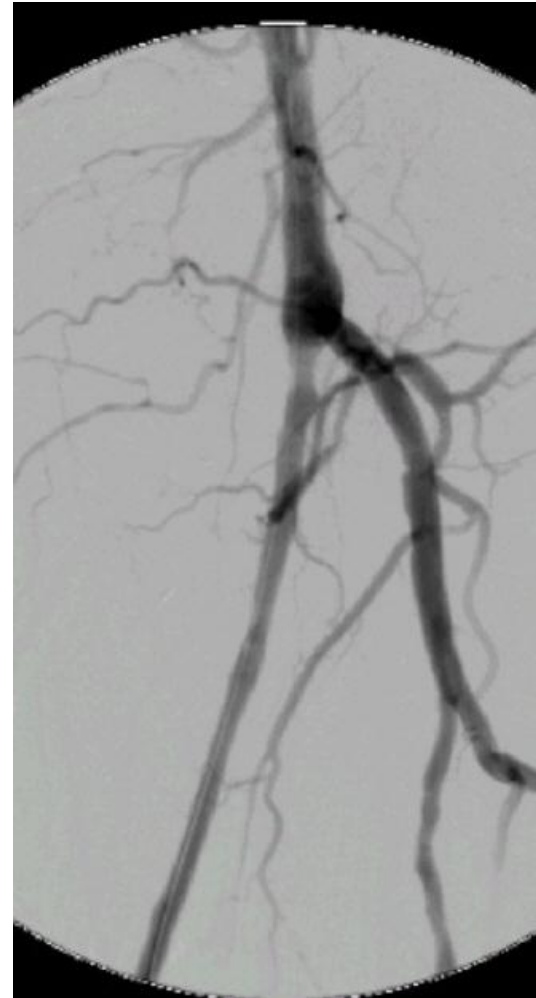 - +/ - surgical bypass
  - +/- thrombolytic therapy
  - +/- primary amputation

Surgical Thrmboemblectomy Procedure

Thrombolysis

What do we worry about after revascularization?

## • Reperfusion Injury

-Local

         - Compartment Syndrome we should do fasciotomy

-Systemic

         - Hyperkalemia leads to cardiac arrest we should give Calcium Gluconate

         - Acidosis we should give bicarbonate

         - Myoglobulinuria leads to acute renal injure we should give a lot of fluids

One of the problem with revascularization that after we restore the blood we can have compartment syndrome (local reperfusion) or the systemic reperfusion problem such as hyperkalemia, acidosis and Myoglobulinuria

Compartment Syndrome
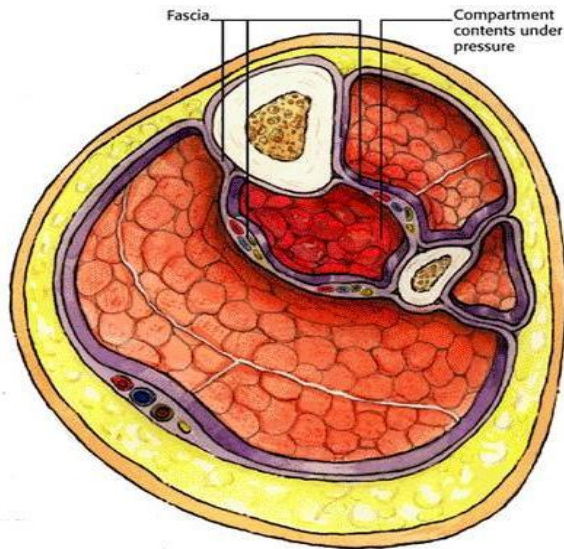
Why compartment ?
Because we have the fascia
and you can not expand

Compartment Syndrome of Left Leg with Surgical Fasciotomies