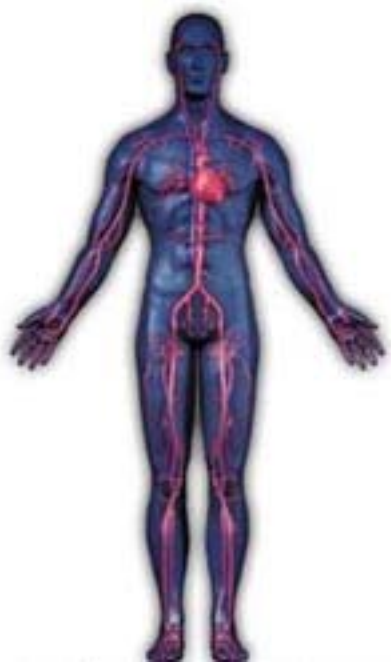
Peripheral Vascular Disease



Judith Barbaro-Brown

judith brown
on-line cpd

What is PVD ?

Any condition or pathology that detrimentally affects the function of the peripheral vascular system is termed ;

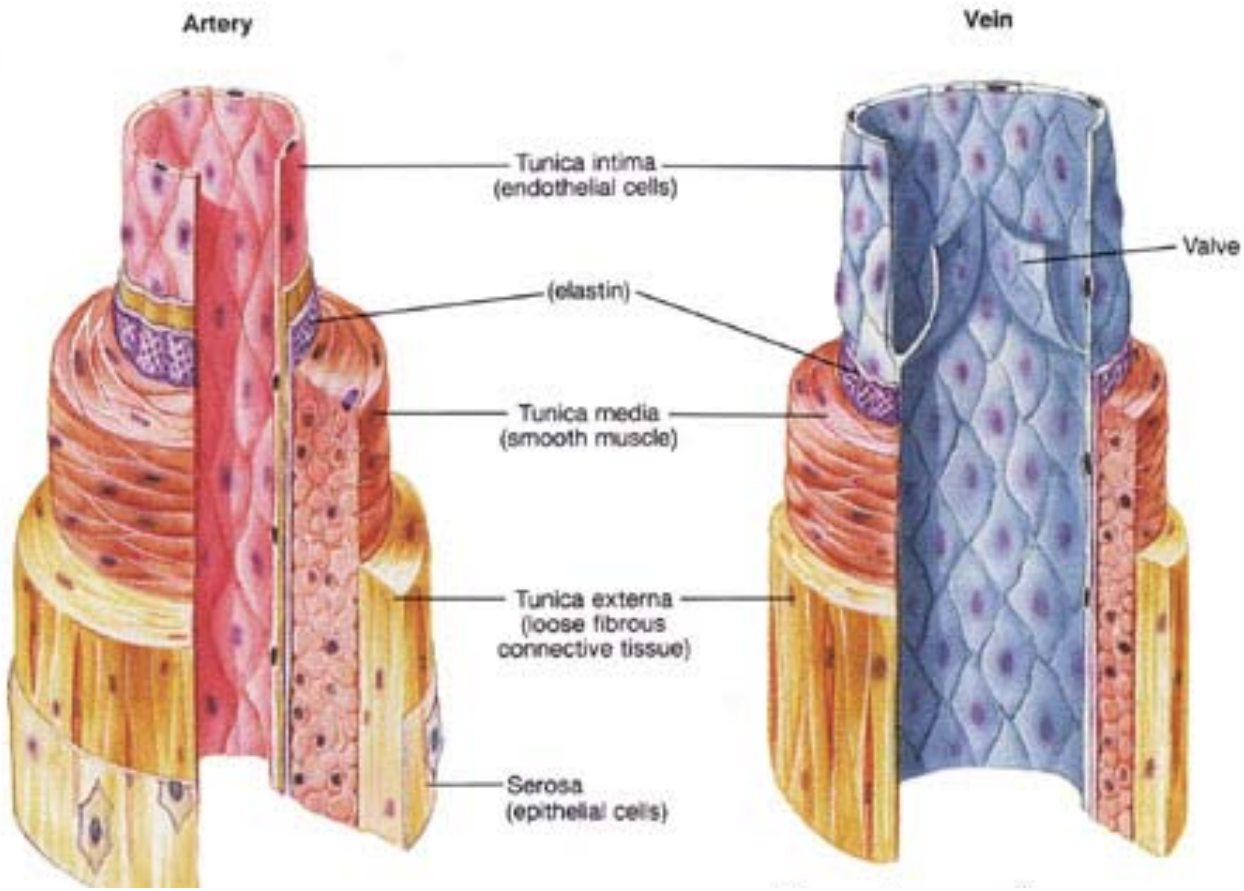
“peripheral vascular disease”

The peripheral vascular system relates to structures involved in the carriage of blood that are not within the cardiac tissue.

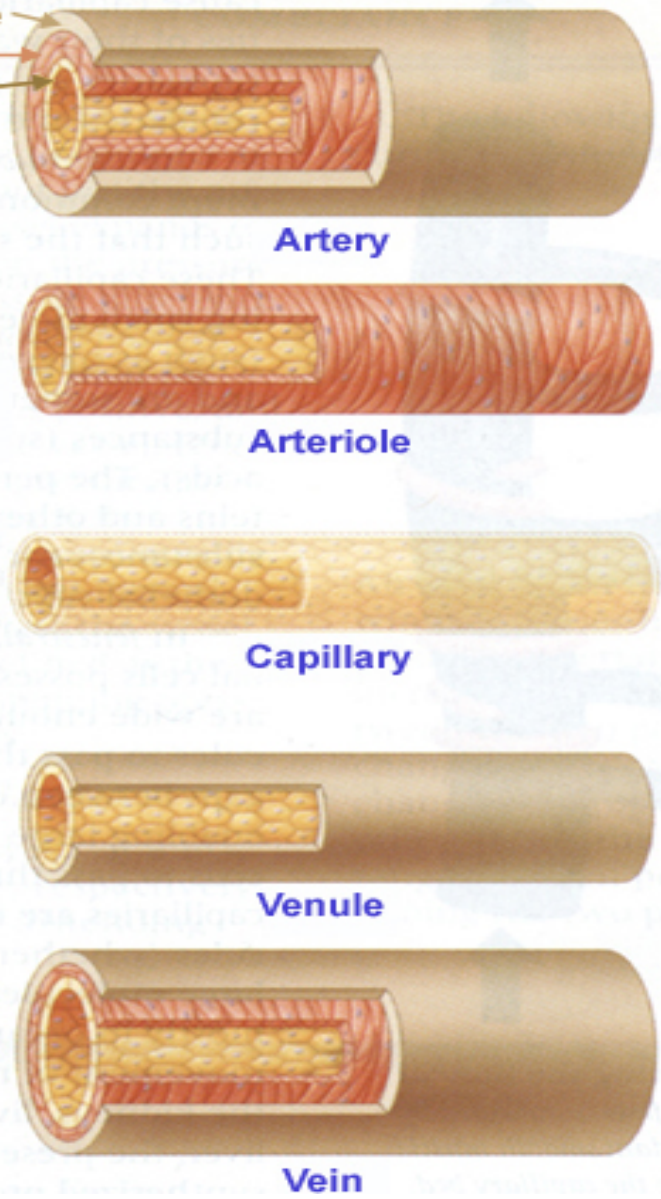
So this involves…….

- Arteries
- Veins
- Capillaries
- Arterioles, venules, lymphatic system

Arteries and Veins



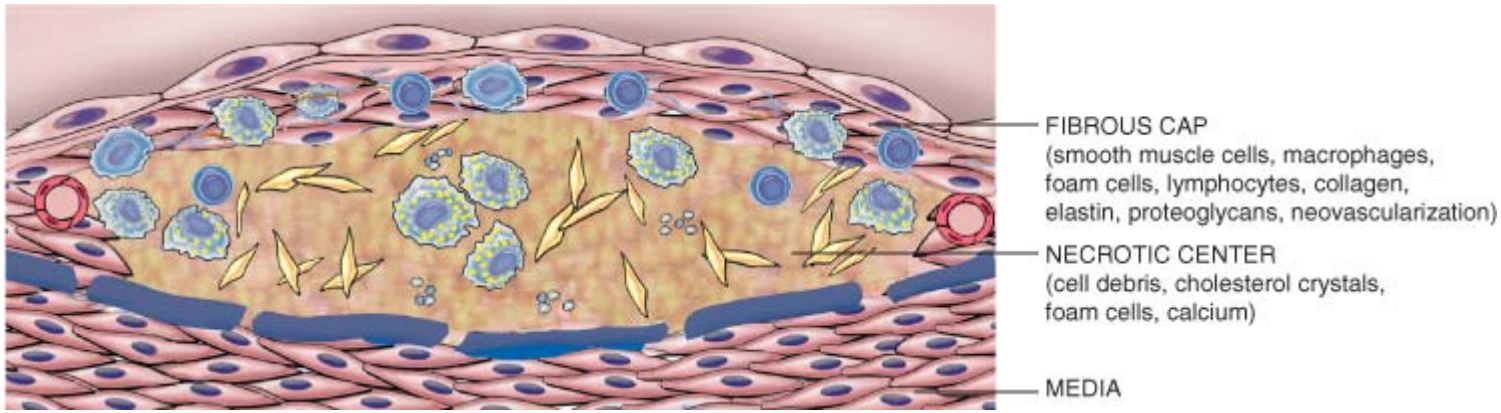
Vessel Characteristics



Lets start with the biggest problem.

ATHEROMA

What is atheroma ?



atheroma (plural: atheromata) is an abnormal accumulation of inflammatory cells (macrophages, WBCs), lipids, and a variable amount of connective tissue within the walls of arteries.

Can begin as young as 1 year of age, and appears in ALL children over 10 years of age.

Why is it a problem?

It leads to the narrowing of the vessel, and therefore interferes with the flow, and dynamics of flow.

Consequences:

MI, CVA, ischaemia, angina, occlusion, thrombosis, embolism, and many more…….

Who does it affect ?

Most patients have underlying atherosclerosis.

Major risk factors include hypertension, elevated levels of low-density lipoprotein, reduced levels of high-density lipoprotein, cigarette smoking, diabetes mellitus, obesity, male sex, elevated homocysteine levels, and family history of

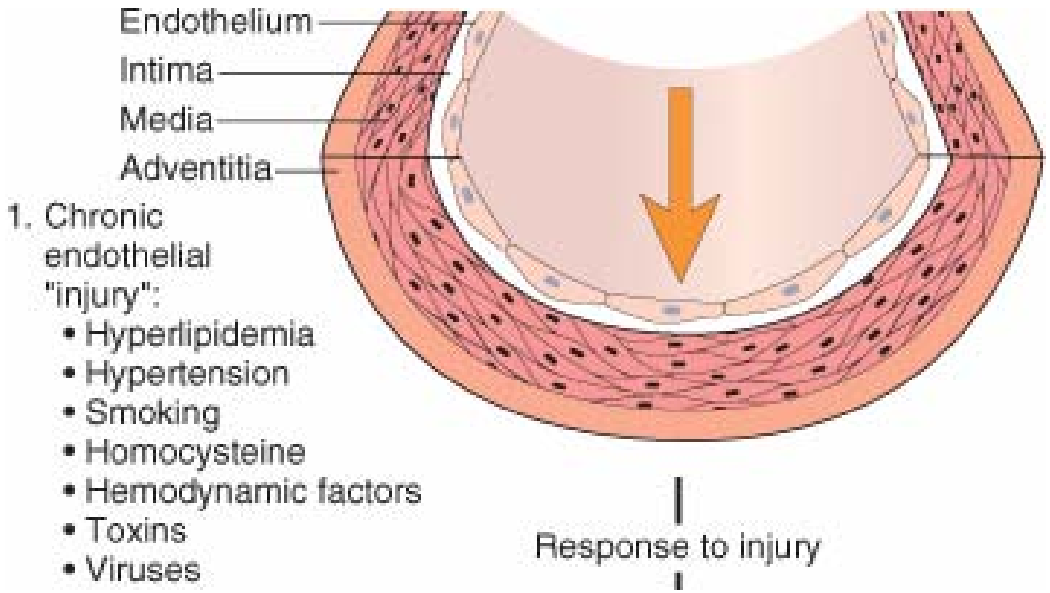
premature atherosclerosis.

"Would you like a bypass with that?"

How does it develop ?

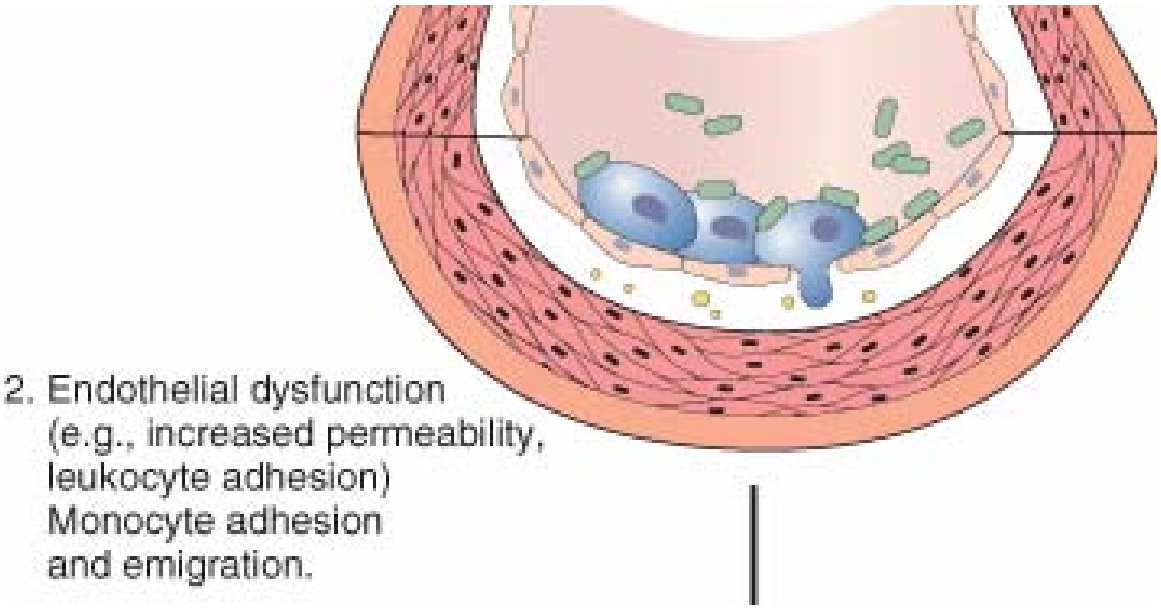
Stage 1-

chronic endothelial injury



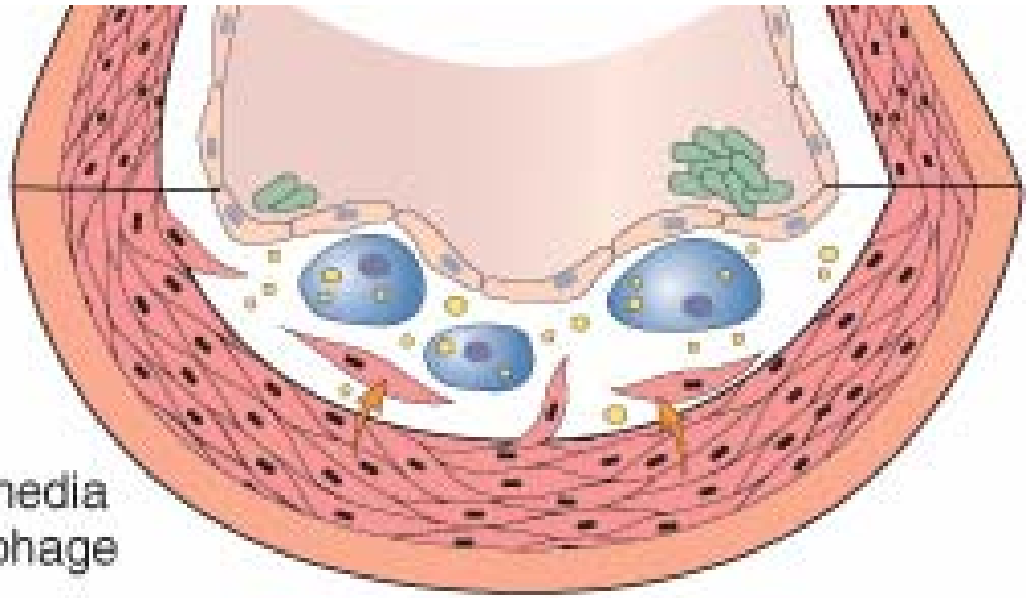
Development, Stage 2.

Endothelial dysfunction, allowing adhesion and invasion



Development, Stage 3

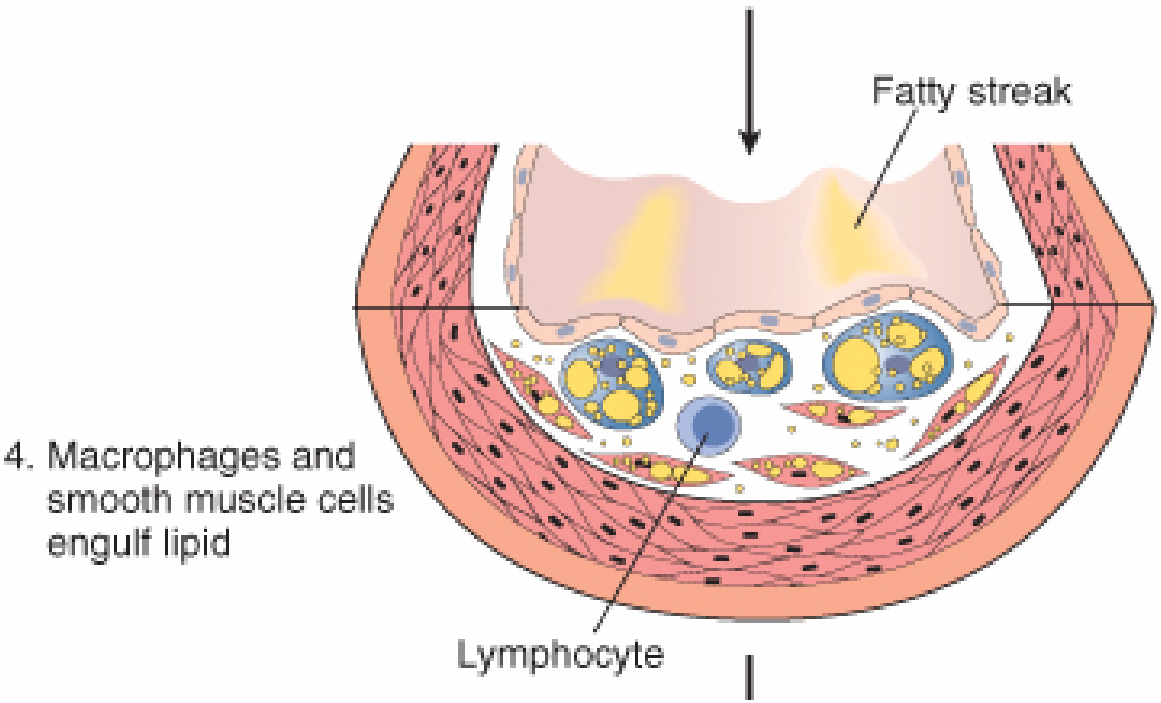
Smooth muscle begins to emmigrate, macrophage activity.



3. Smooth muscle emigration from media to intima. Macrophage activation.

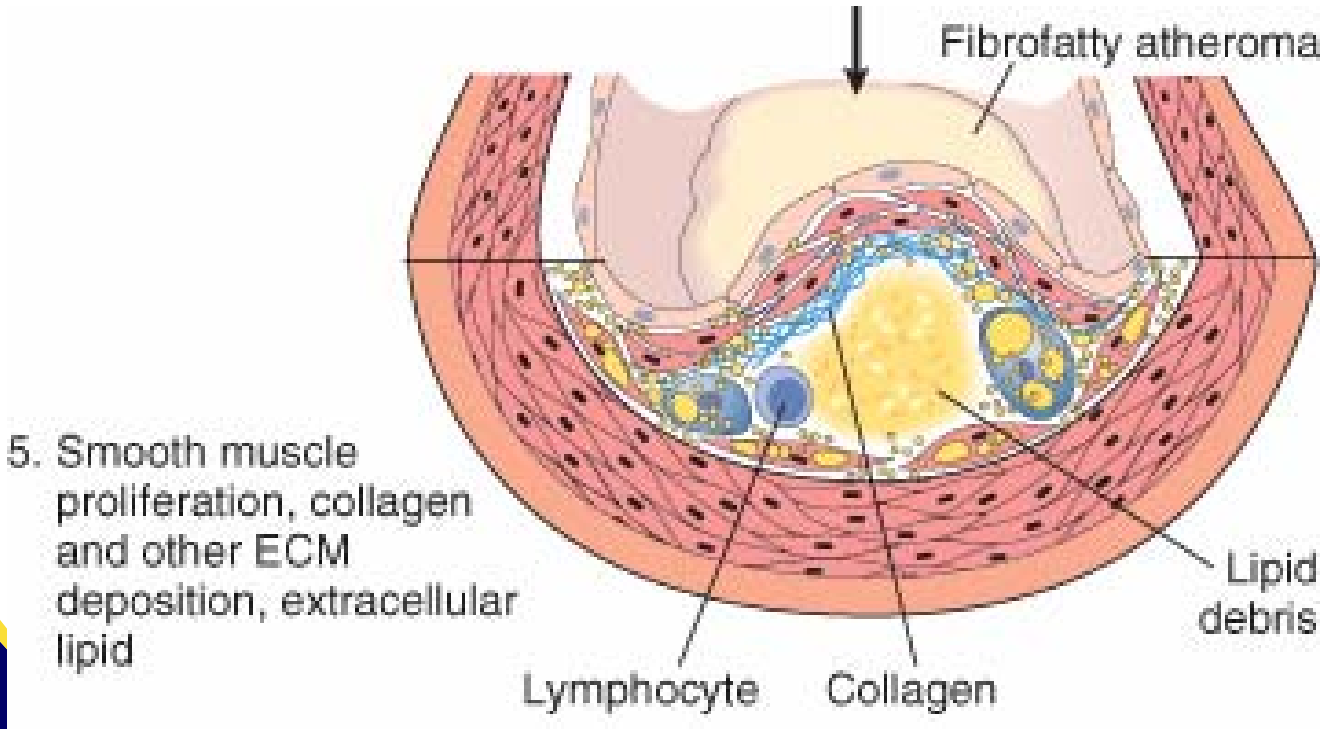
Development, Stage 4.

Engulfment of lipid material into artery wall, development of ‘fatty streak’.



Development, Stage 5.

Smooth muscle proliferates, extra collagen laid down, lipid debris, narrowing of lumen.



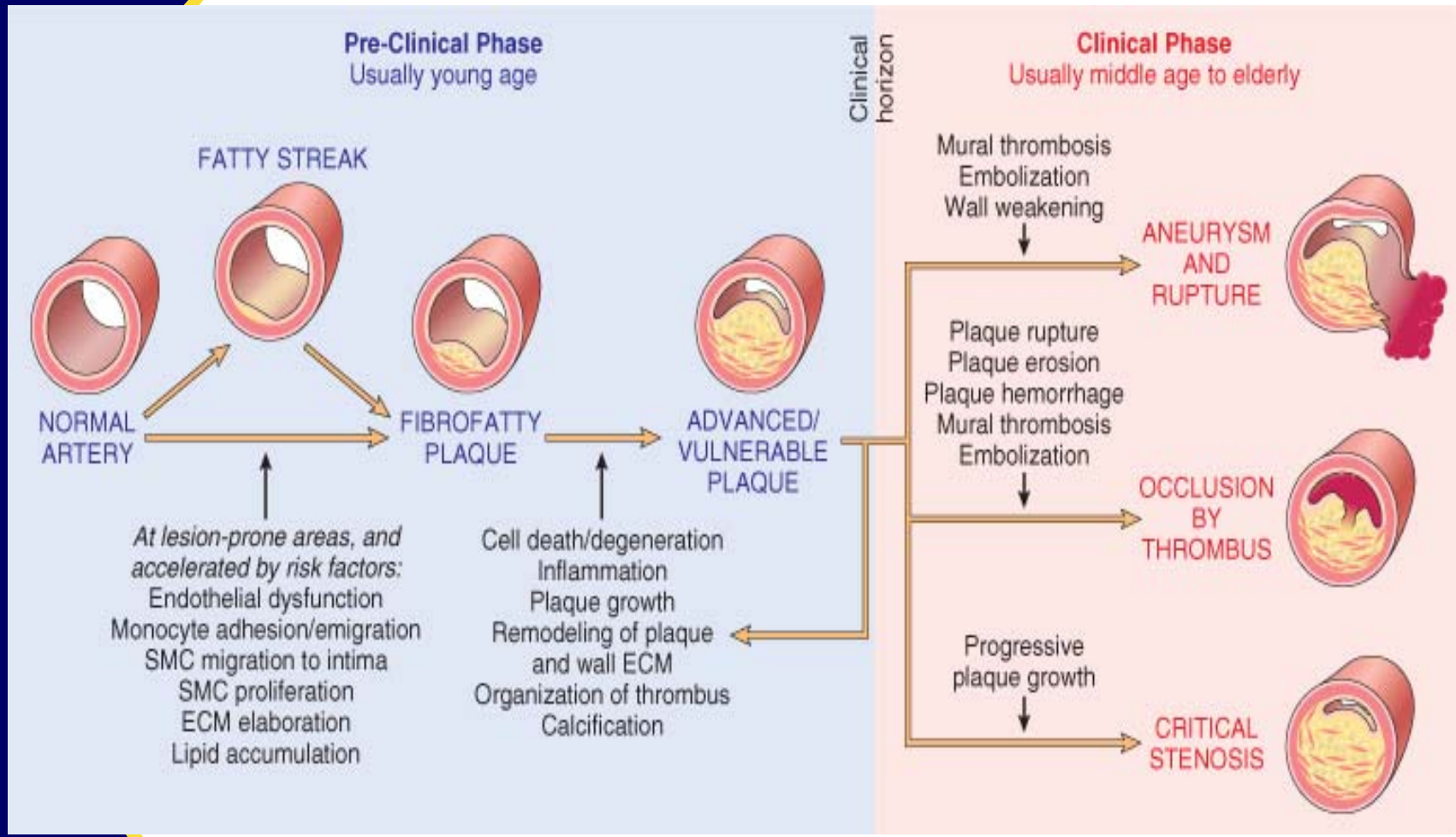
Signs & Symptoms

Acute Occlusion

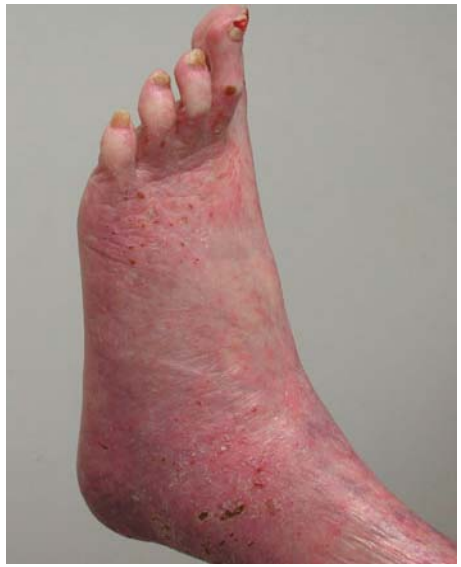
- Sudden onset of pain
- Coldness
- Numbness
- Pallor
- Absent pulses

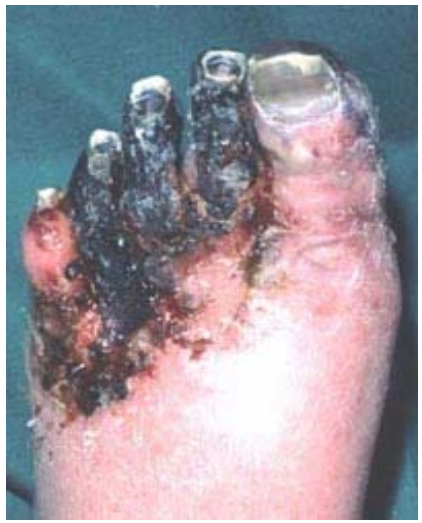
Chronic Occlusion

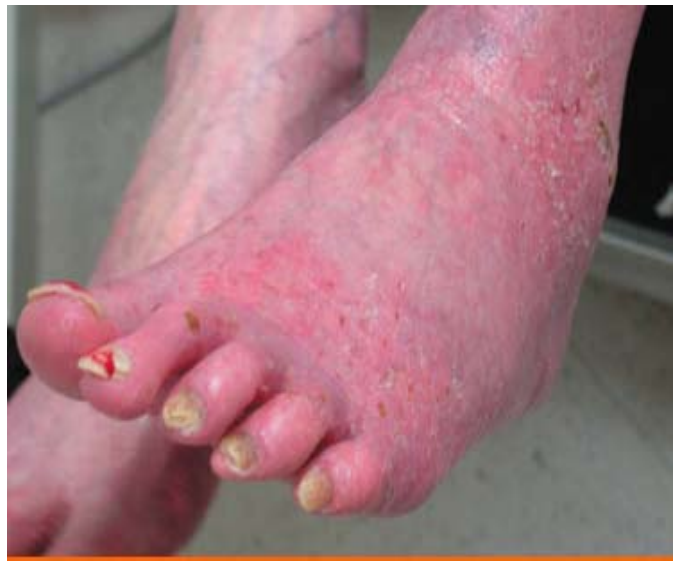
- Insidious onset of ischaemia
- Intermittent claudication
- Night cramps


Pre-Clinical Phase
Usually young age
Clinical horizon
Clinical Phase
Usually middle age to elderly
FATTY STREAK
NORMAL ARTERY
FIBROFATTY PLAQUE
ADVANCED/ VULNERABLE PLAQUE
At lesion-prone areas, and accelerated by risk factors:
Endothelial dysfunction
Monocyte adhesion/emigration
SMC migration to intima
SMC proliferation
ECM elaboration
Lipid accumulation
Cell death/degeneration
Inflammation
Plaque growth
Remodeling of plaque and wall ECM
Organization of thrombus
Calcification
Mural thrombosis
Embolization
Wall weakening
ANEURYSM AND RUPTURE
Plaque rupture
Plaque erosion
Plaque hemorrhage
Mural thrombosis
Embolization
OCCLUSION BY THROMBUS
Progressive plaque growth
CRITICAL STENOSIS

Potential consequences in the limb







Management of atheroma

- Stop smoking
- Increase exercise (gently!)
- ? Vasodilators
- Percutaneous transluminal angioplasty
- Grafting, bypass
- Thrombolytic therapy
- Growth factor therapy

Raynaud's Disease & Phenomenon

Spasm of the digital arterioles, with intermittant pallor and/or cynaosis.

Disease – idiopathic

Phenomenon – secondary to other pathology

What happens ?

- Reversible constriction of the vessels, leading to a reduction of oxygenation in the related tissues.
- May be linked with prostaglandin metabolism.
- Associated with migraine, variant angina, pulmonary hypertension.

A vasospastic response is normal where the digital arteries will constrict when the temperature lowers.

In Raynaud’s, the threshold for this response is lowered, meaning that the temperature does not need to reduce dramatically for an attack to occur.

Signs & Symptoms

Exposure to cold or emotional upset precipitates colour change.

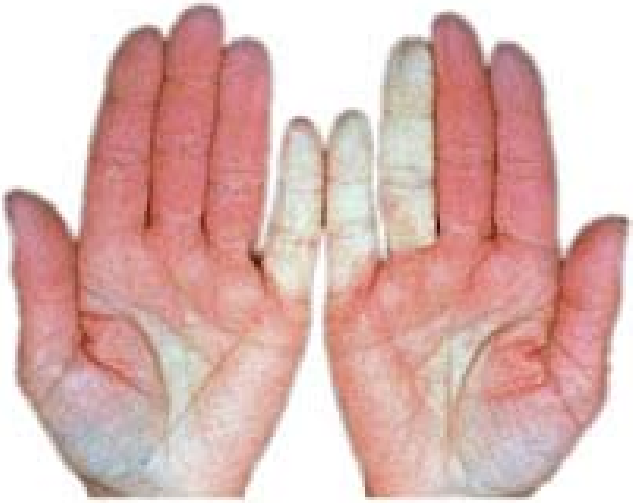
Triphasic – pallor, cyanosis, hyperaemia

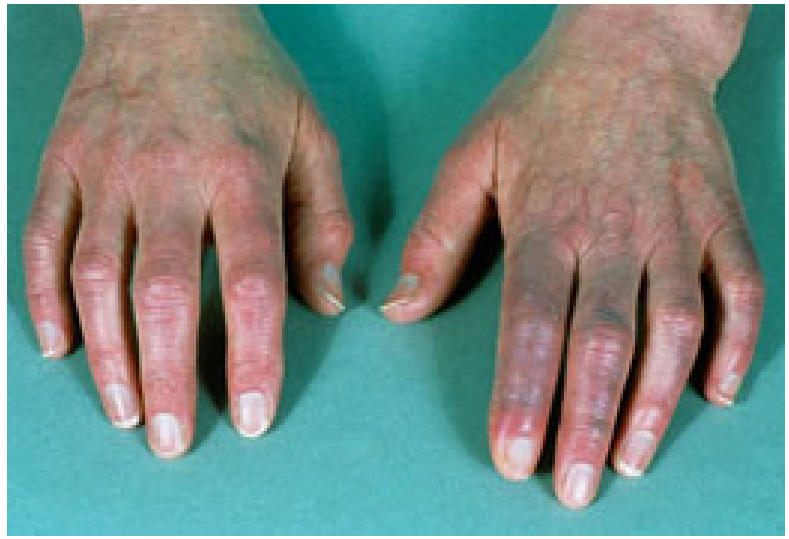
Biphasic – cyanosis, hyperaemia

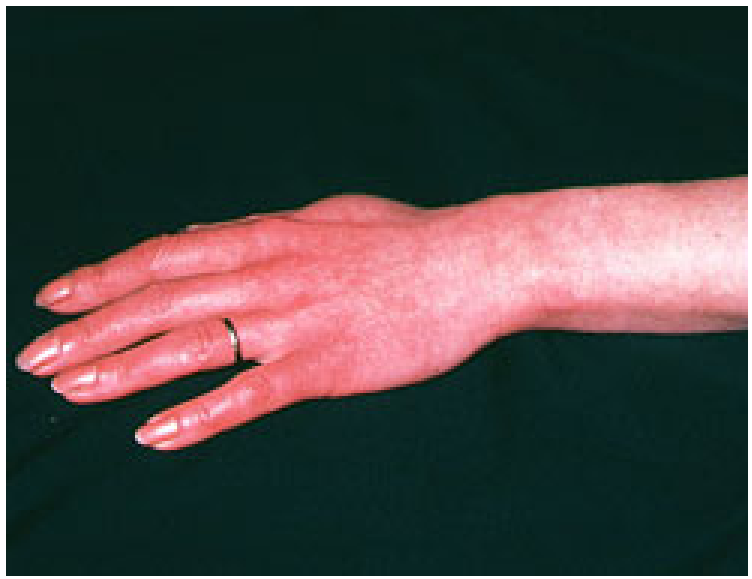
Sometimes painful

Paraesthesia

Spontaneous return to normality on warming area







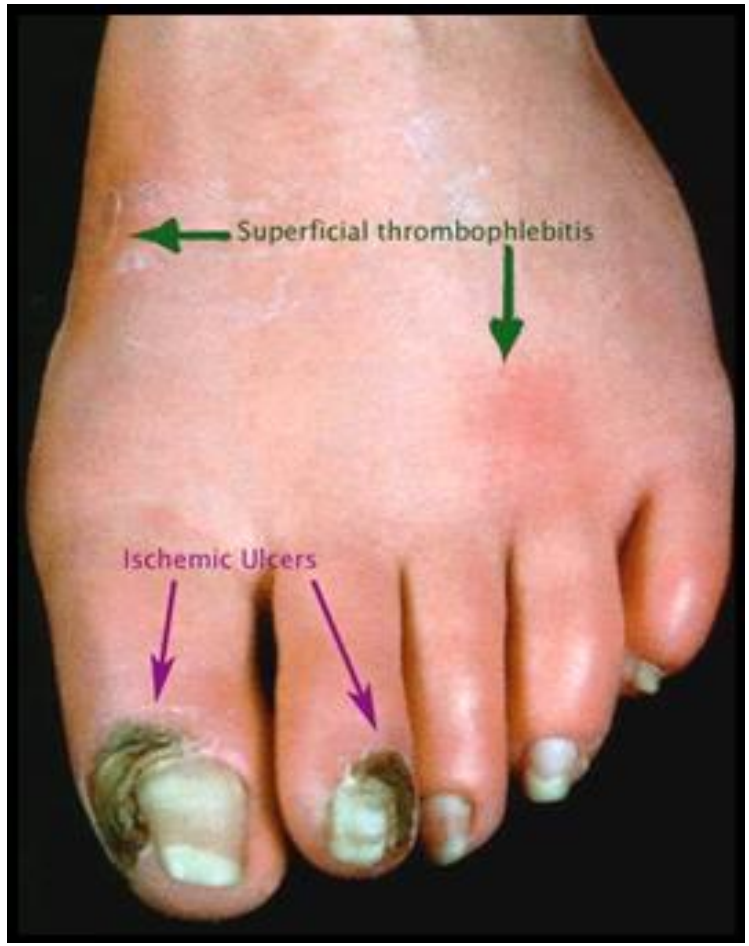
Management

- Protection from cold
- Stop smoking
- Calcium channel blockers (nifedipine)
- Pentoxifylline

Thromboangiitis Obliterans

AKA Buerger's Disease

An obliterative disease, characterised by inflammatory changes in small and medium arteries and veins.



Pathology

- Involves small & medium sized arteries
- Proliferation of endothelial cells
- Infiltration of intimal layer by lymphocytes
- Thrombus development
- Fibroblast infiltration

Signs & Symptoms

Arterial ischaemia

Superficial thrombophlebitis

Gradual onset, from distal to proximal

Digital gangrene

Coldness, numbness, tingling, burning

Intermittant claudication

Impaired or absent pulses

Management

Stop smoking

Avoid exposure to cold

Increase exercise

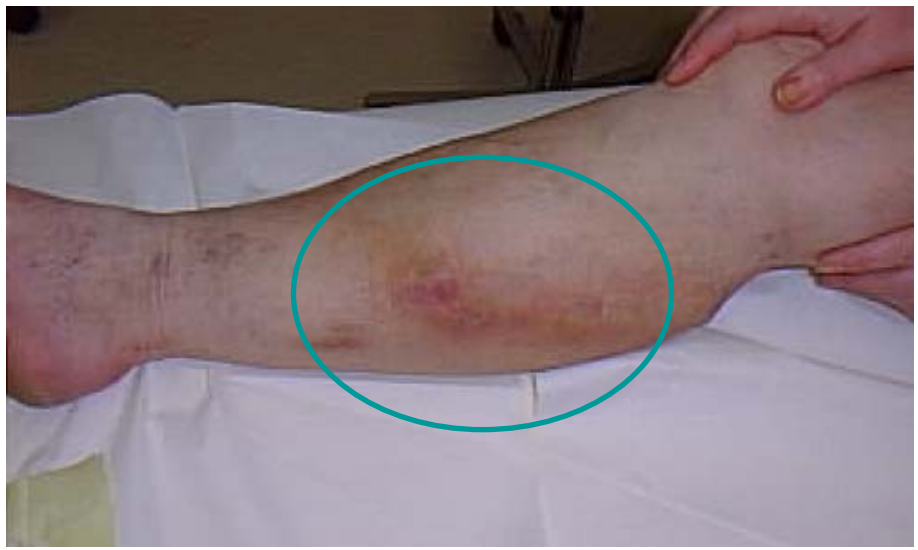
Drug therapy not useful

Venous Thrombosis

Can affect superficial or deep veins.

Prolonged thrombus may lead to chronic venous insufficiency

Always accompanied by phlebitis

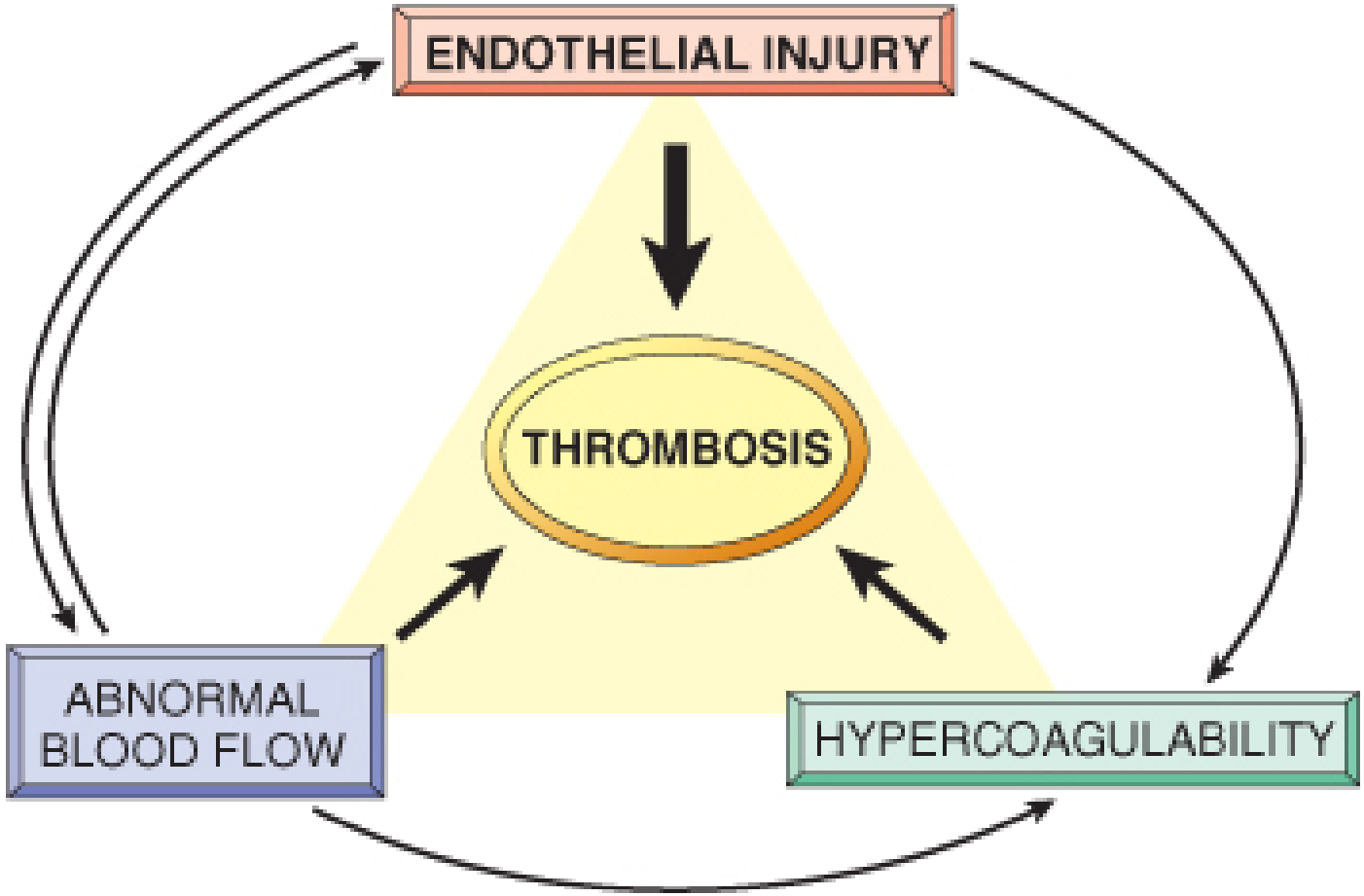


Pathology

- Endothelial injury exposes collagen, leading to platelet aggregation
- Release of thromboplastin
- In combination with stasis and/or hypercoagulability

VIRCHOW'S TRIAD

Virchow's Triad



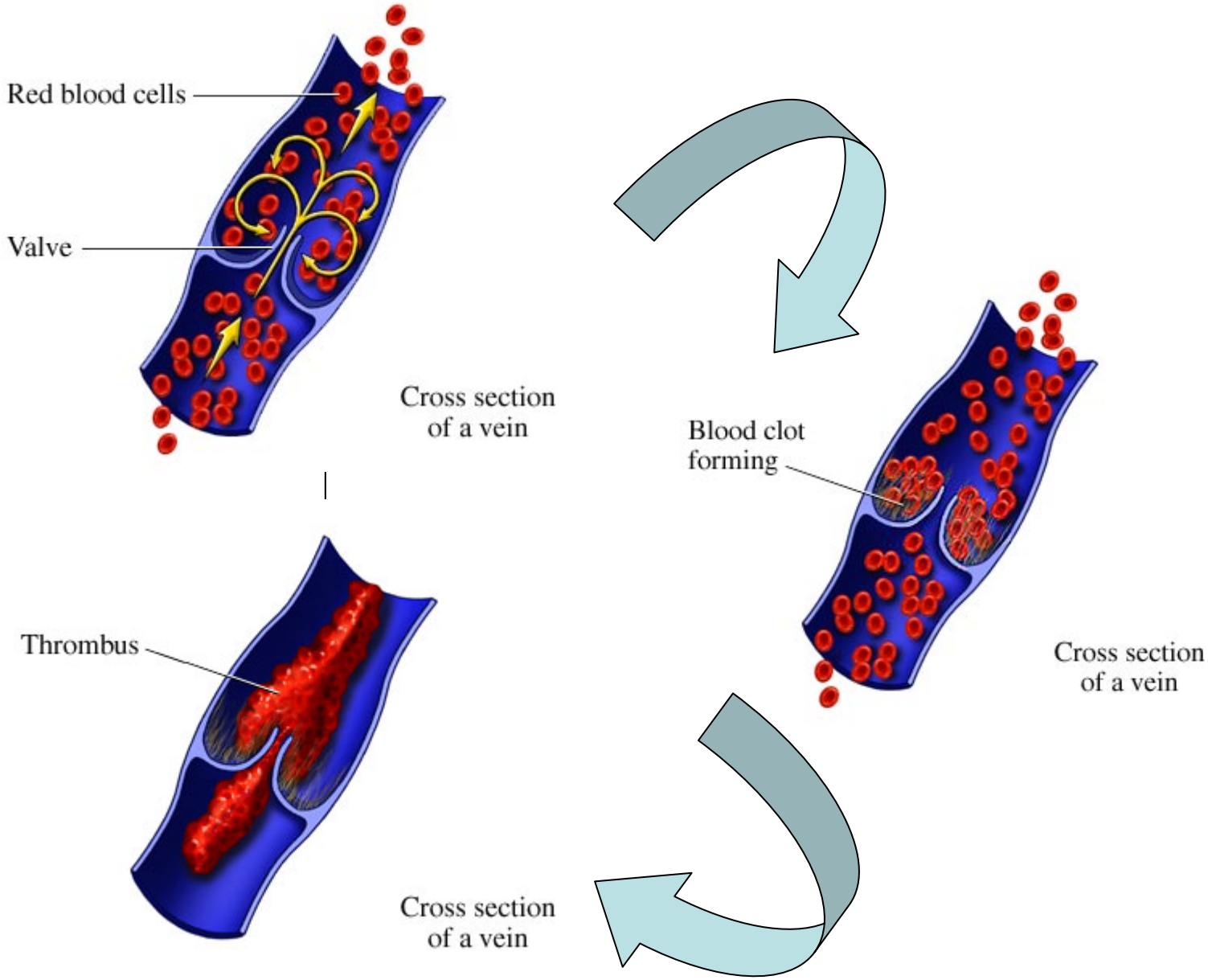
Most begin around the valves within the deep veins

Thromboplastic released

Trapping of RBC’s

Propagation of red thrombus

(white thrombi occur in arteries as the flow in these vessels is faster)


Red blood cells
Valve
Cross section of a vein
Blood clot forming
Cross section of a vein
Thrombus
Cross section of a vein

Acute thromboplebitis

- Symptoms usually arise over 1-2 days
- Usually self-limiting, lasting up to two weeks, after which acute process subsides.
- Can be painful, swollen.
- Can be superficial or deep

Superficial thrombophlebitis

- Thrombosed superficial vein
- Can often be palpated
- Pain, tenderness, erythema, warmth

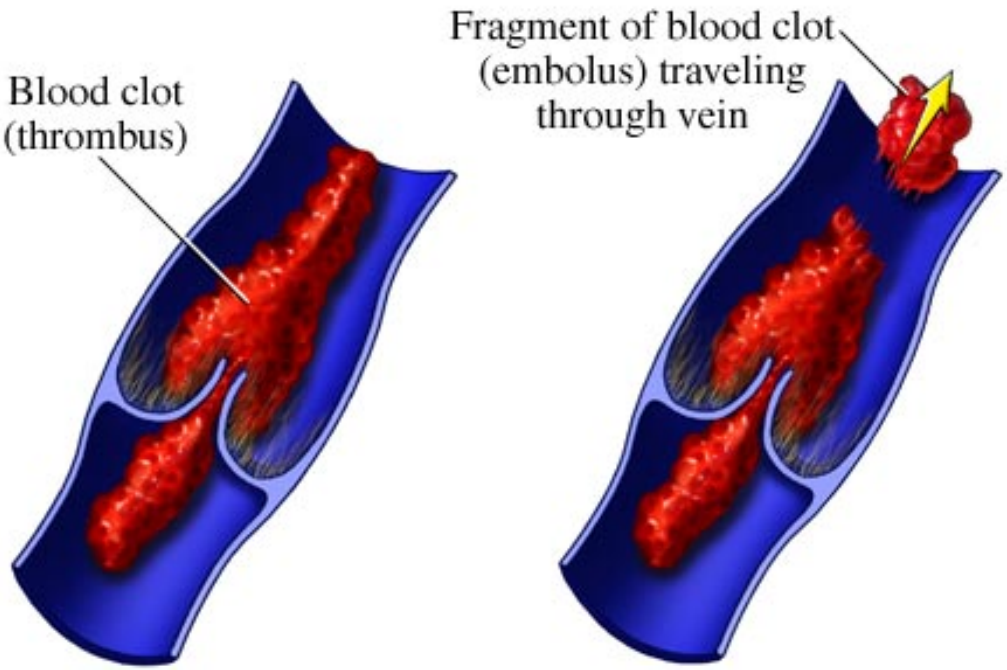
Deep vein thrombosis

- Can be silent
- Pain, tenderness, swelling, warmth, redness, prominent superficial veins
- Soreness when standing and/or walking
- Deep calf pain on palpation
- Sometimes difficult to differentiate from muscle pain
- Differential diagnosis – Popliteal cyst

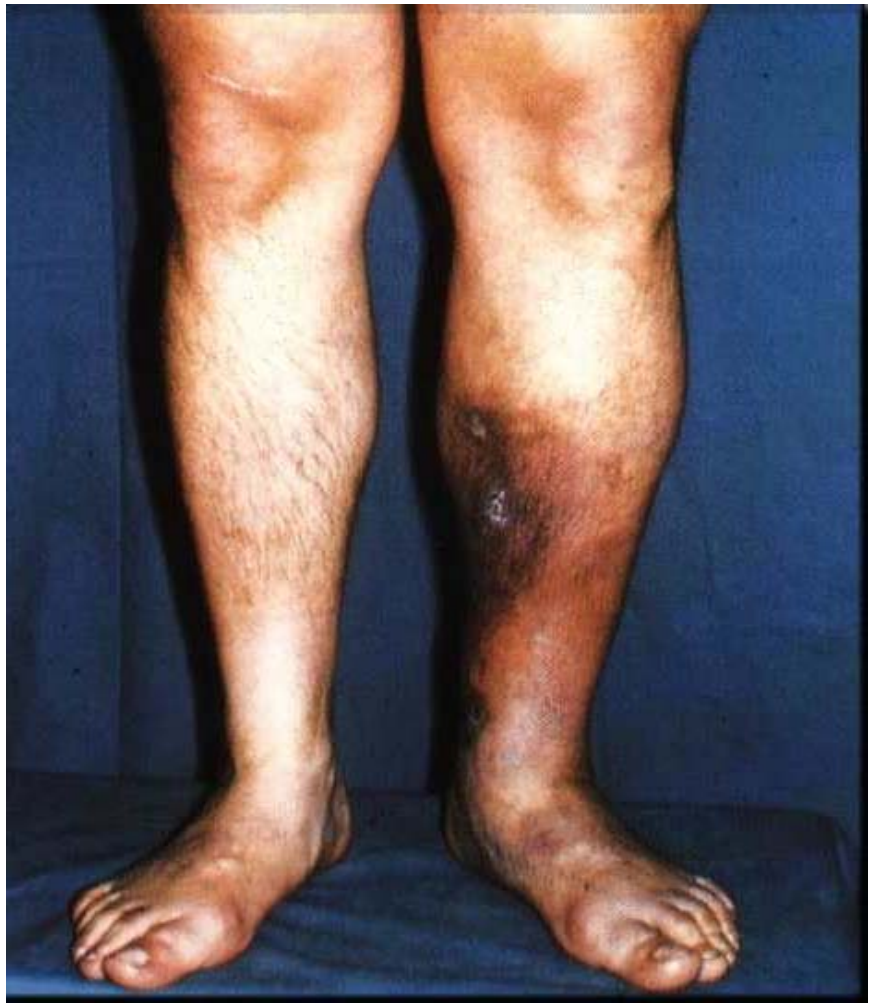
Consequences of DVT

Embolism

- Pulmonary embolism
- MI, CA
- CVA



Chronic venous insufficiency (CVI)



Management

For DVT, the objective is to prevent PE and CVI.

Initially, hospitalisation

Low molecular weight heparin via infusion

Elevation of limb

Oral warfarin, to achieve INR 2-3

Use of compression hosiery

Key Points……..

1. PVD is extremely common
2. It can have major consequences for morbidity and mortality
3. It is one of the main causes of lower limb gangrene and tissue loss
4. It's progress is accelerated by underlying pathologies such as diabetes
5. One of the major contributing factors to it's development is SMOKING ……


WE UNDERSTAND FROM THE NEIGHBOURS THAT YOU'VE BEEN ALLOWING YOUR YOUNG BOY TO SMOKE...?

Thank you for your attention.