ARTERIOSCLEROSIS

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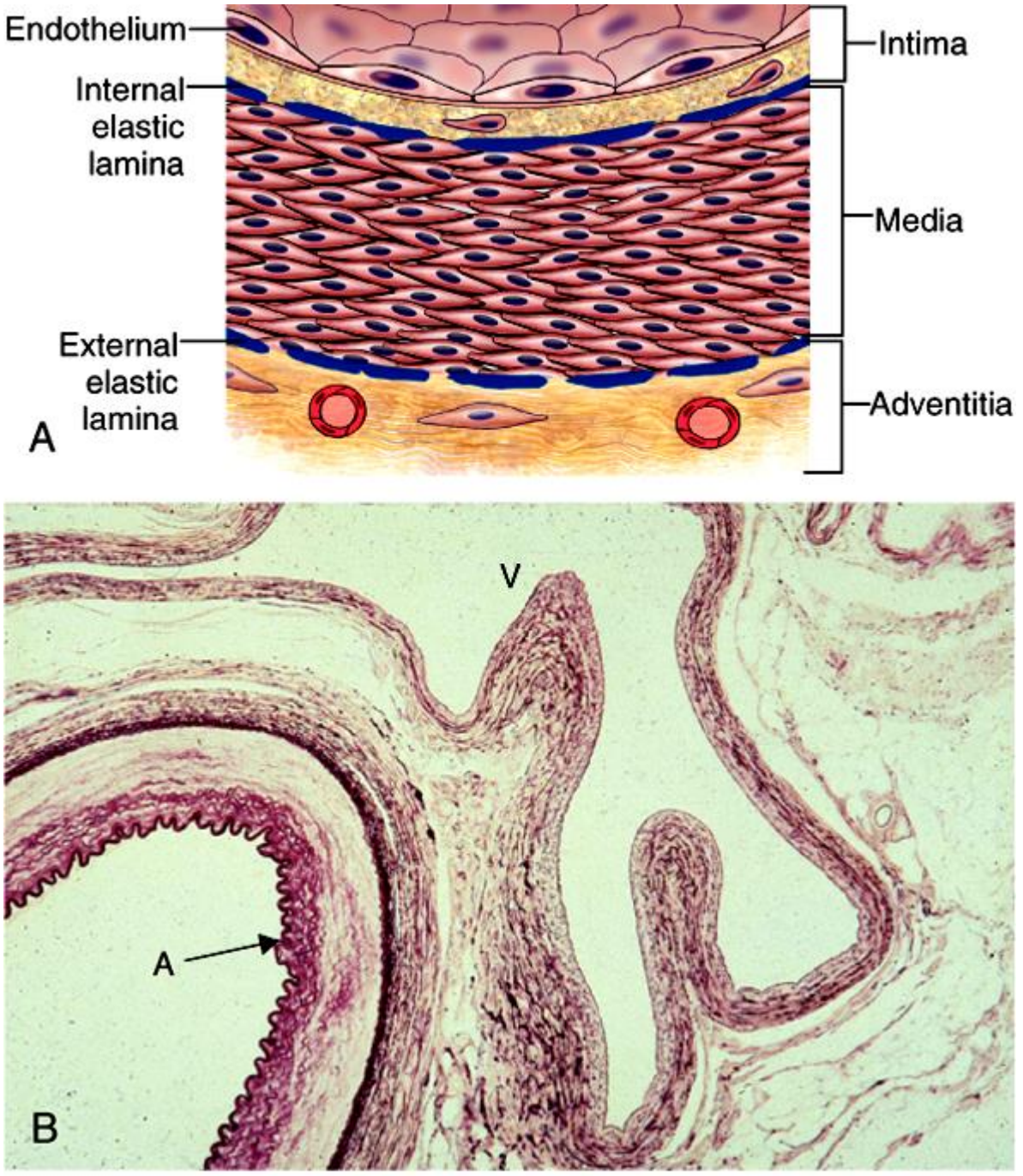
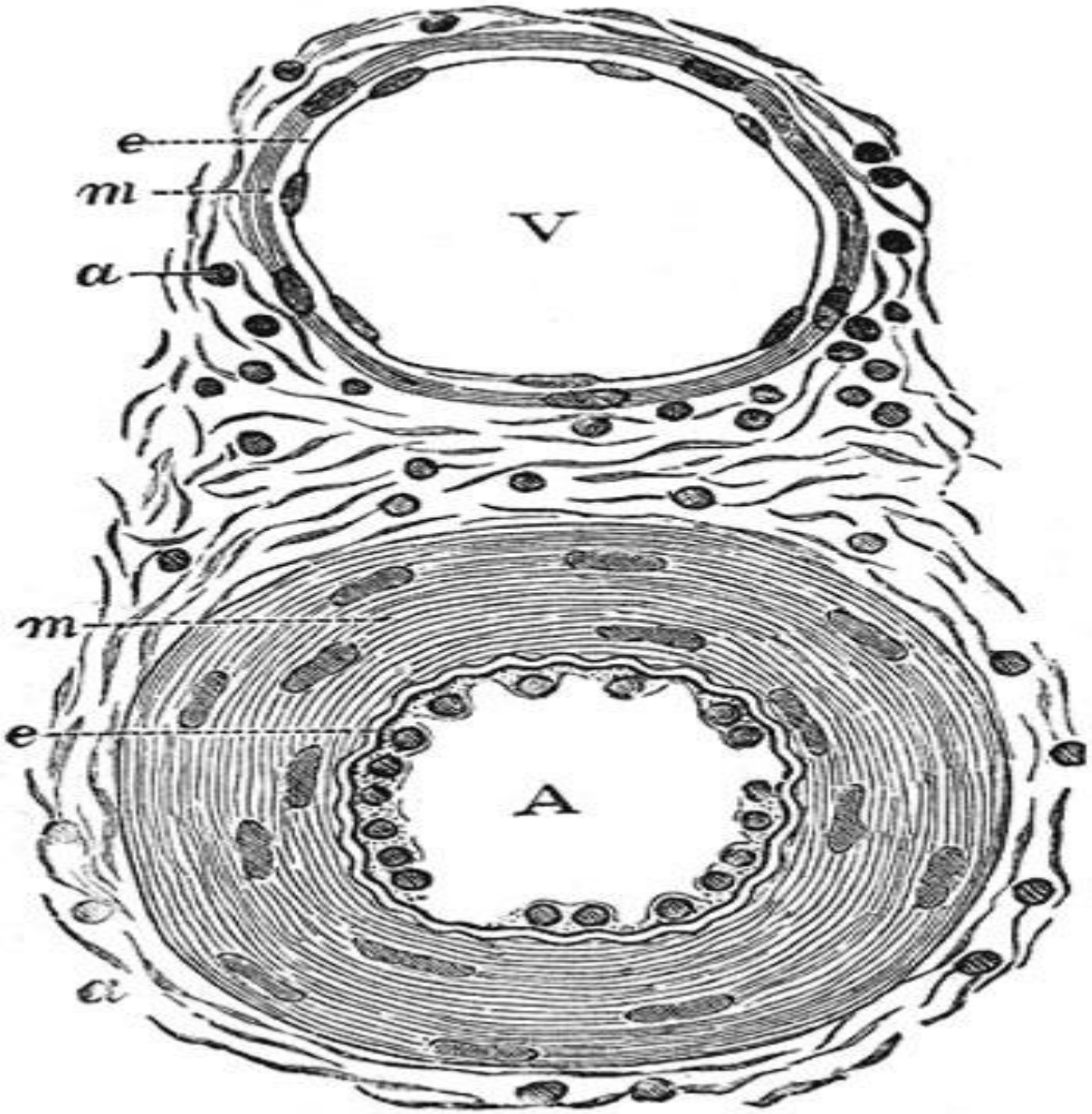
Normal
blood vessels
A= artery
V= vein



Artery (A) versus vein (V)

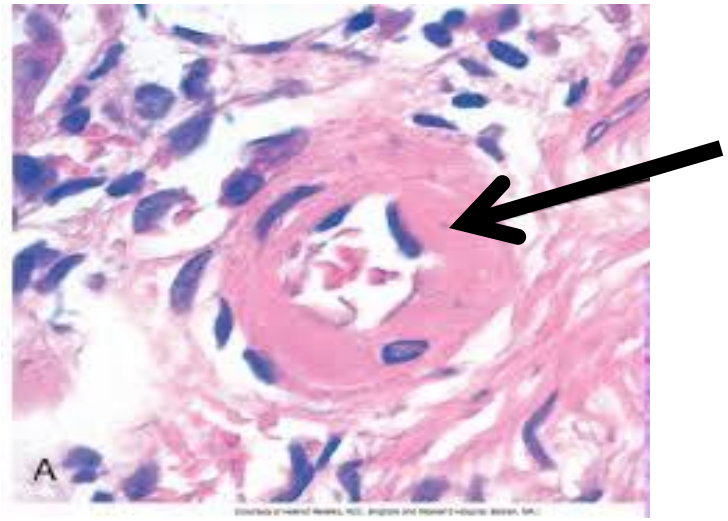


ARTERIOSCLEROSIS

- ***Arteriosclerosis*** **="hardening of the arteries"**
- **arterial wall thickening and loss of elasticity.**
- **Three patterns are recognized, with different clinical and pathologic consequences:**

1-Arteriolosclerosis

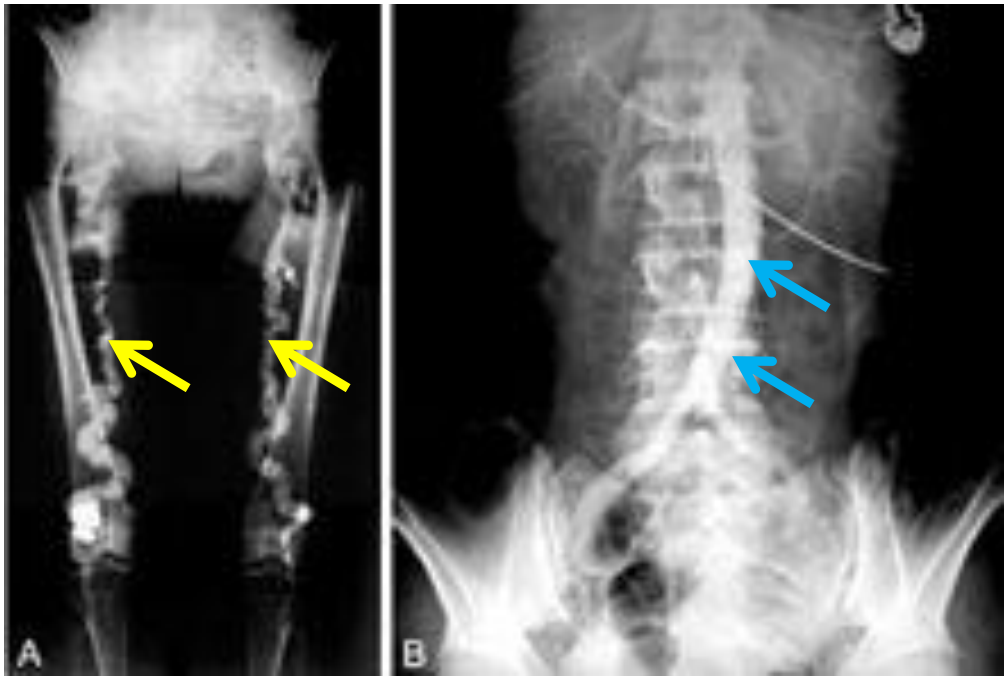
- affects small arteries and arterioles
- associated with hypertension and/or diabetes mellitus

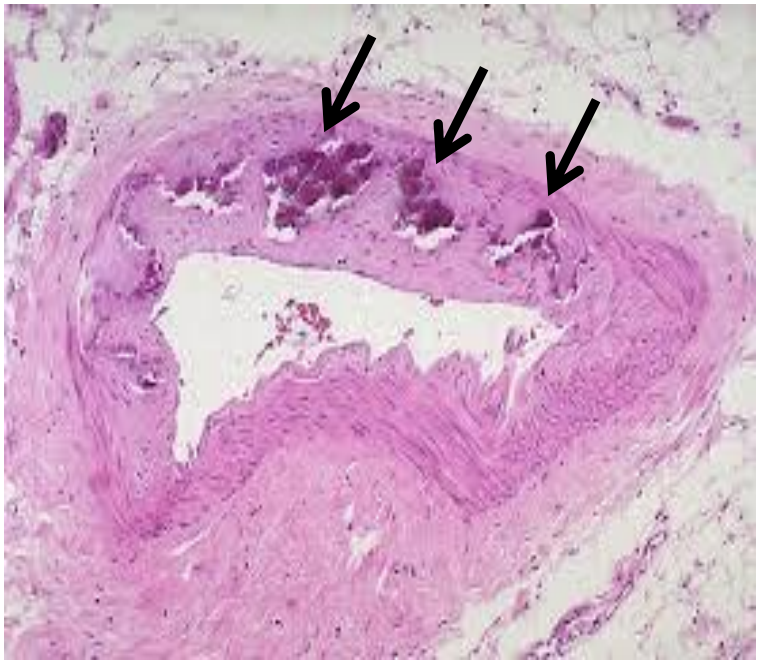


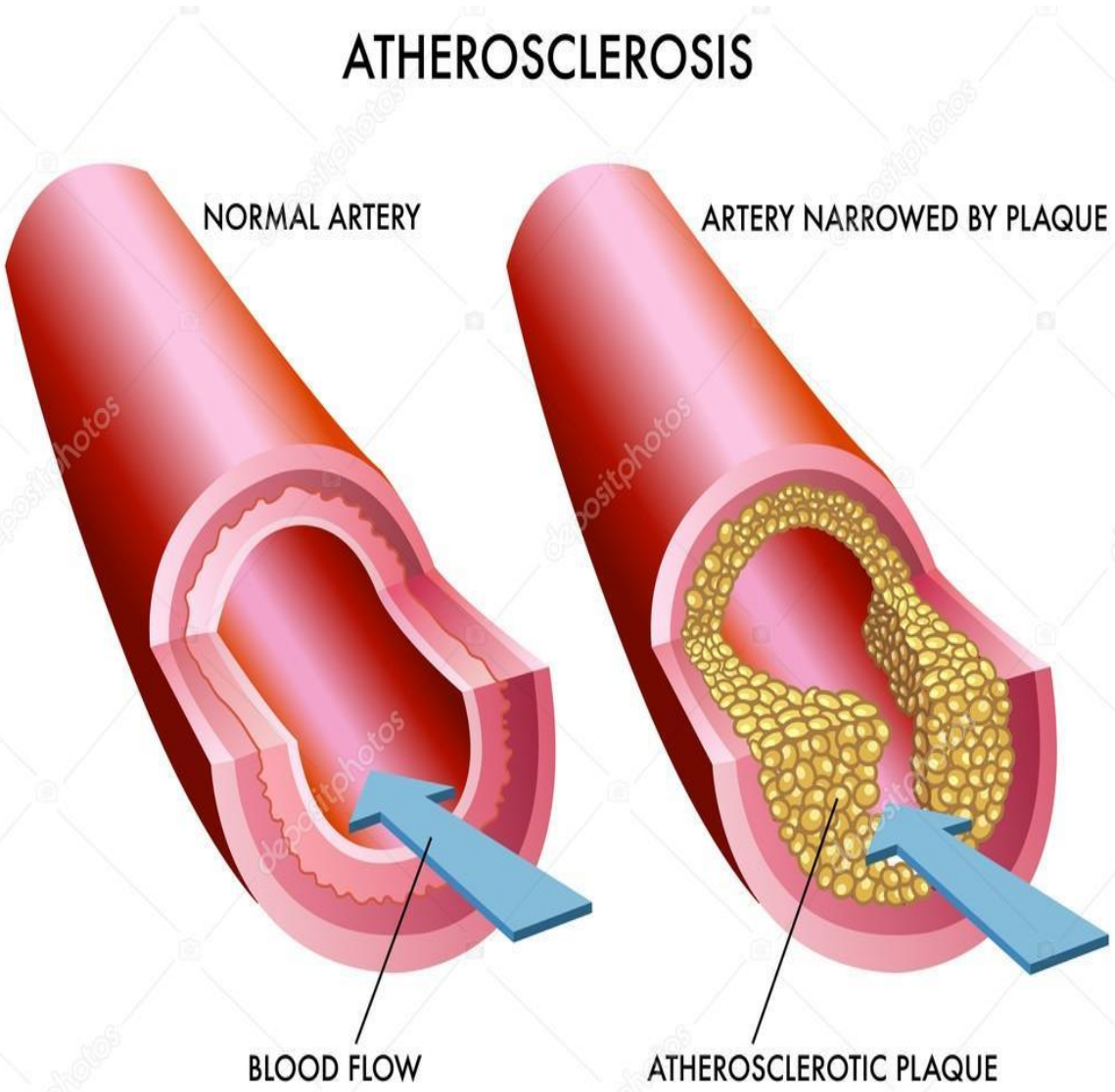
2- Mönckeberg medial calcific sclerosis

- **calcific deposits in muscular arteries**
- **typically in persons > age 50**
- **radiographically visible (x-rays, etc…)**
- **palpable vessels**
- **do not encroach on vessel lumen and are usually not clinically significant**

2-Mönckeberg medial calcific sclerosis





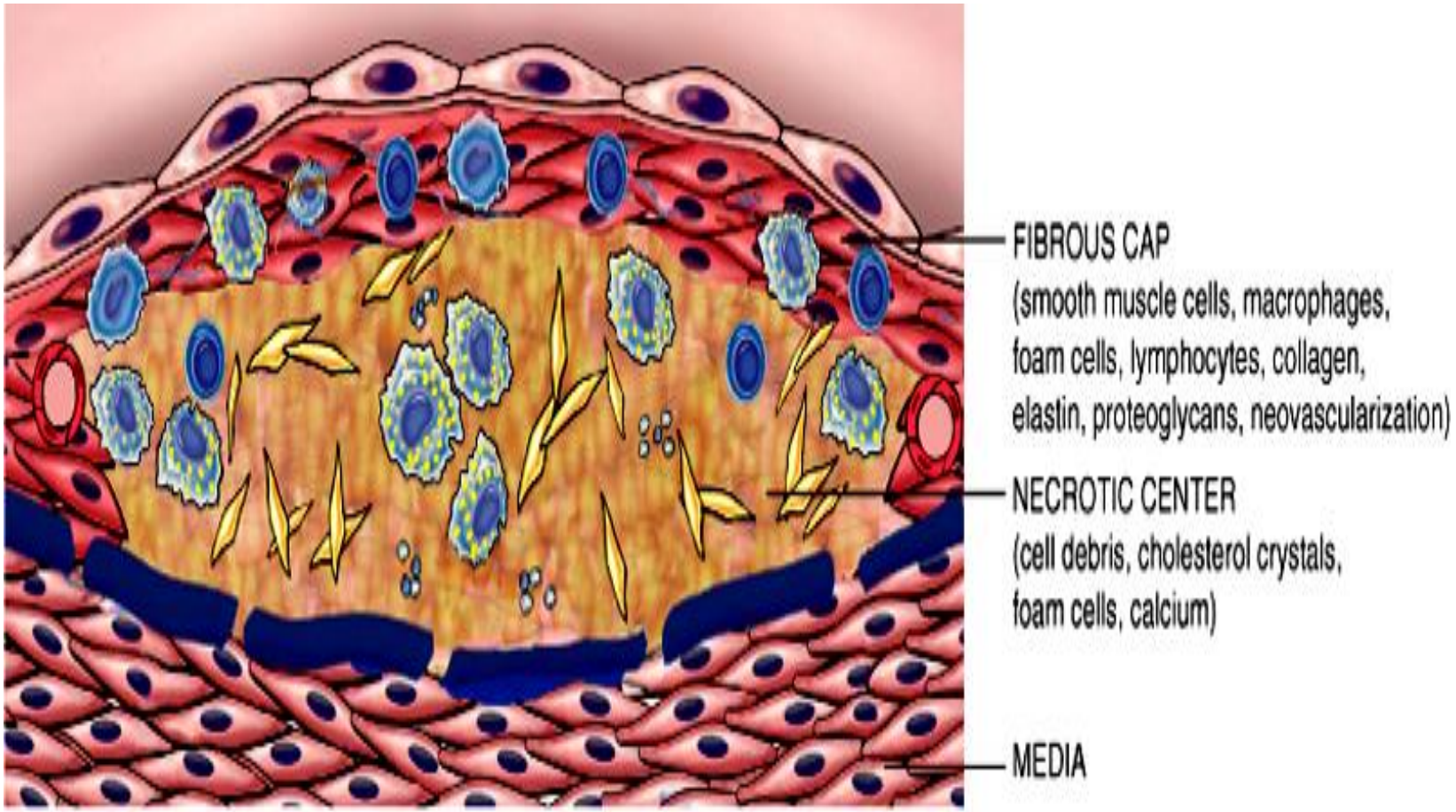


- Greek word "gruel" ,"hardening,"
- most frequent and clinically important pattern of arteriosclerosis
- characterized by intimal lesions =*atheromas* (a.k.a. *atherosclerotic plaques*)
- atheromatous plaque = raised lesion with a core of lipid (cholesterol and cholesterol esters) covered by a firm, white fibrous cap

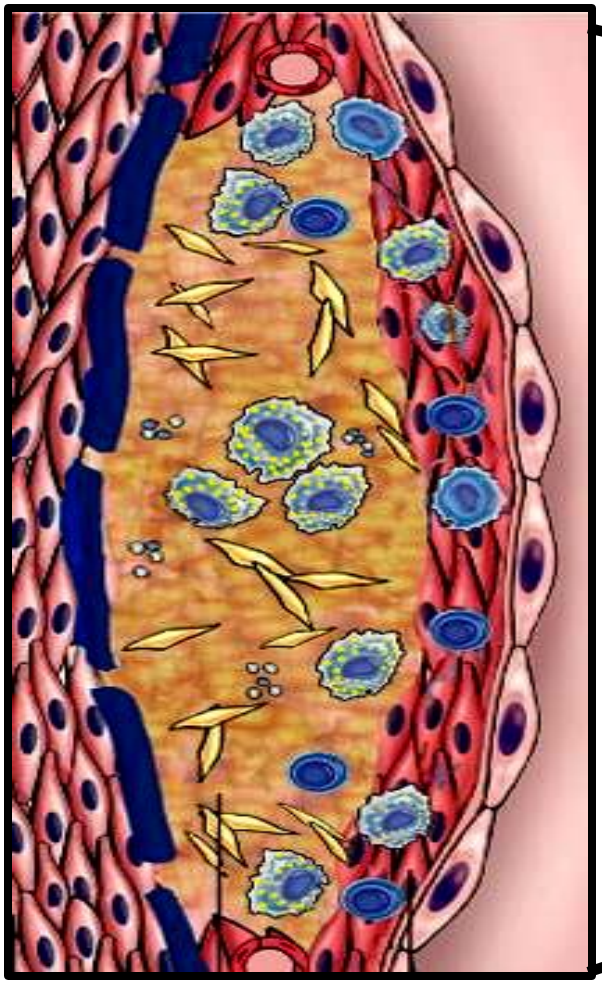
Atherosclerosis- Pathogenesis

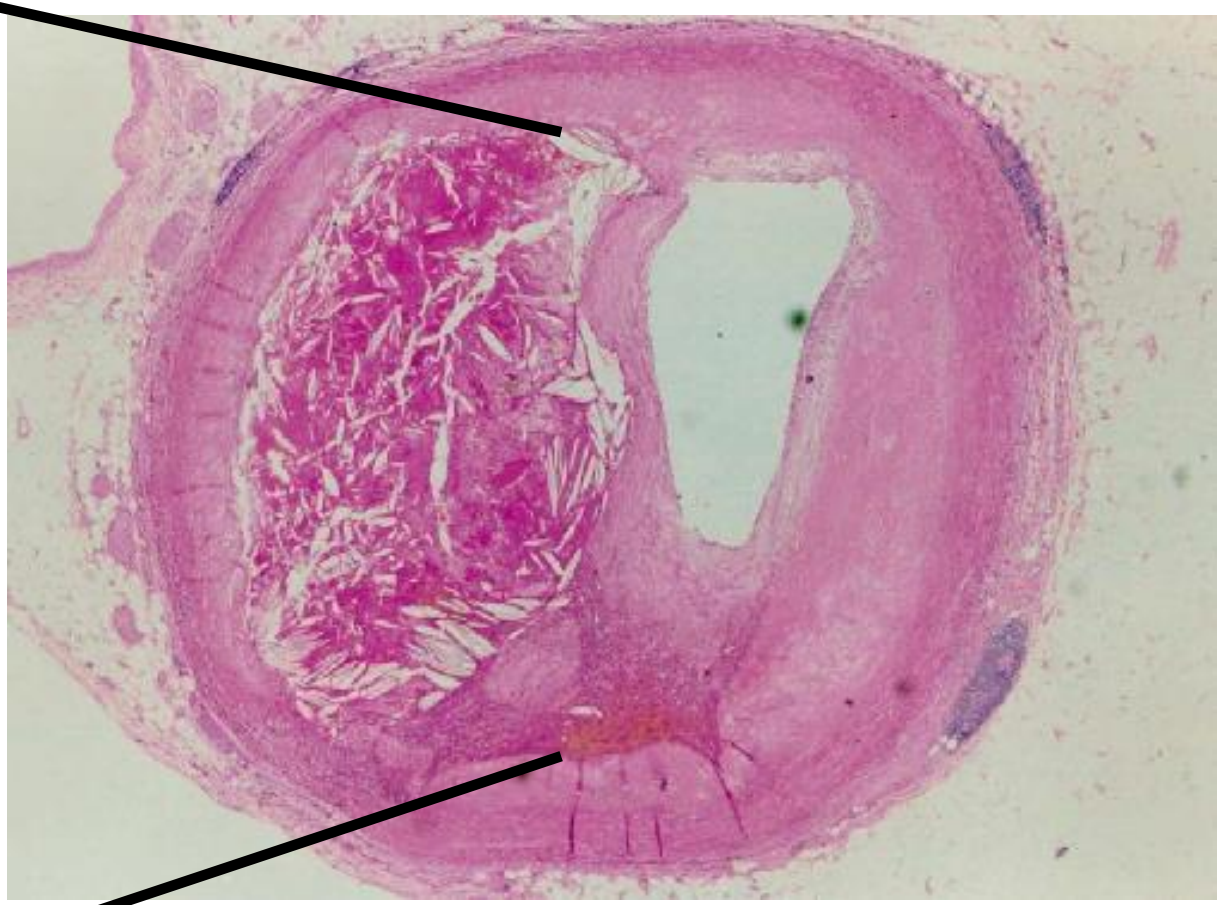
- **not fully understood**
- **? inflammatory process in endothelial cells of vessel wall associated with retained low-density lipoprotein (LDL) particles→ ? a cause, an effect, or both, of underlying inflammatory process**

The major components of a well-developed intimal atheromatous plaque

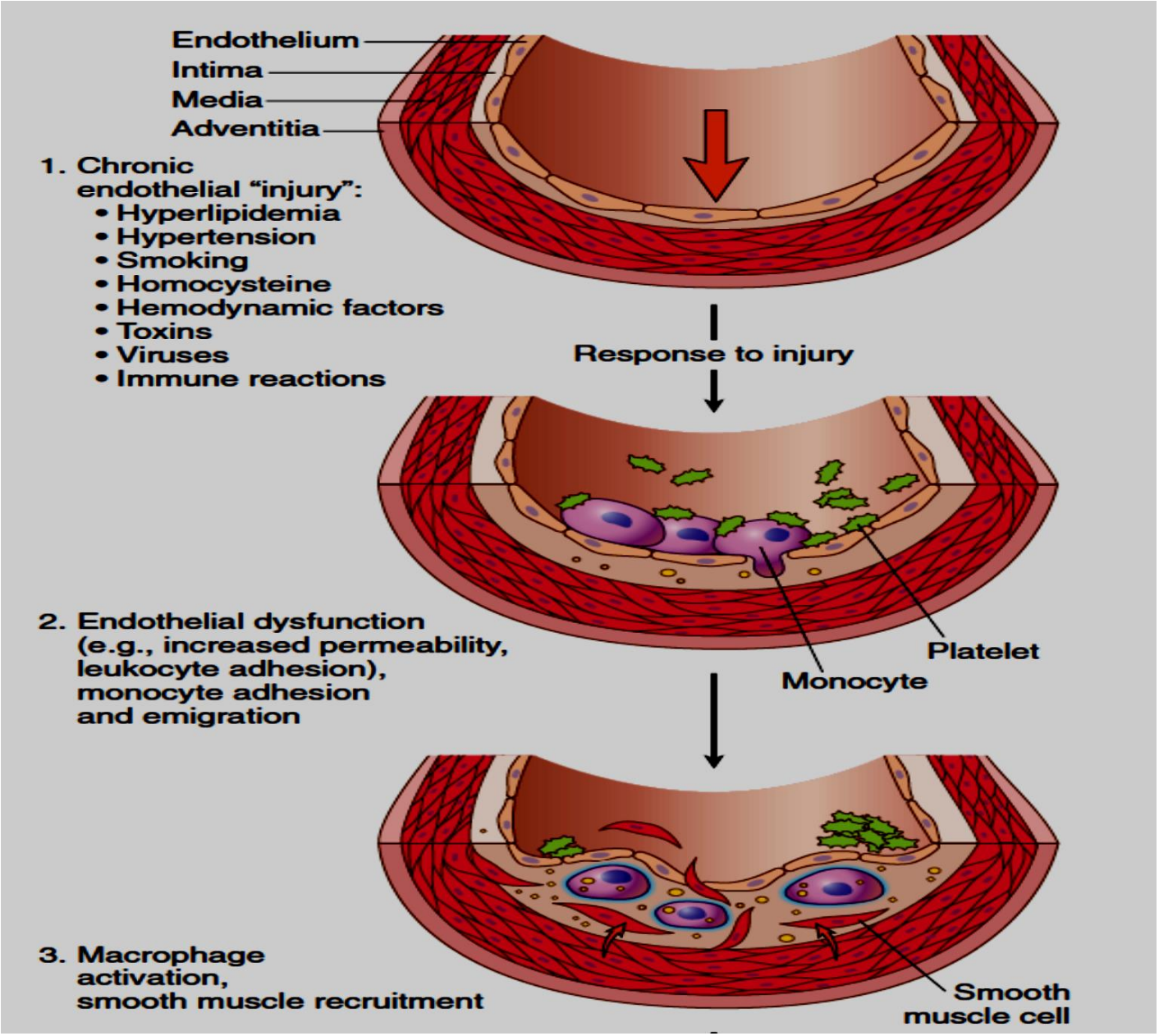


Atheromatous plaque

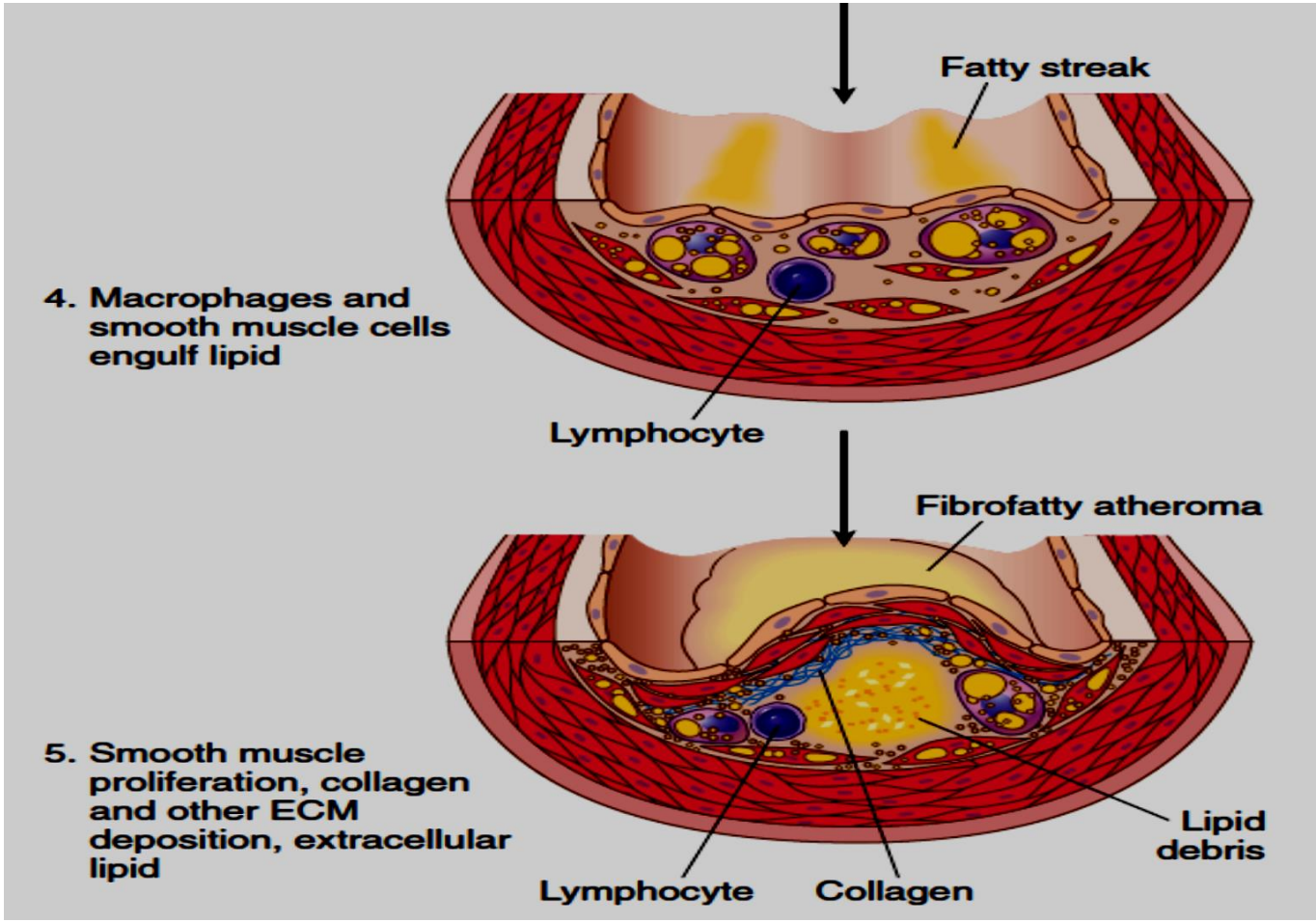


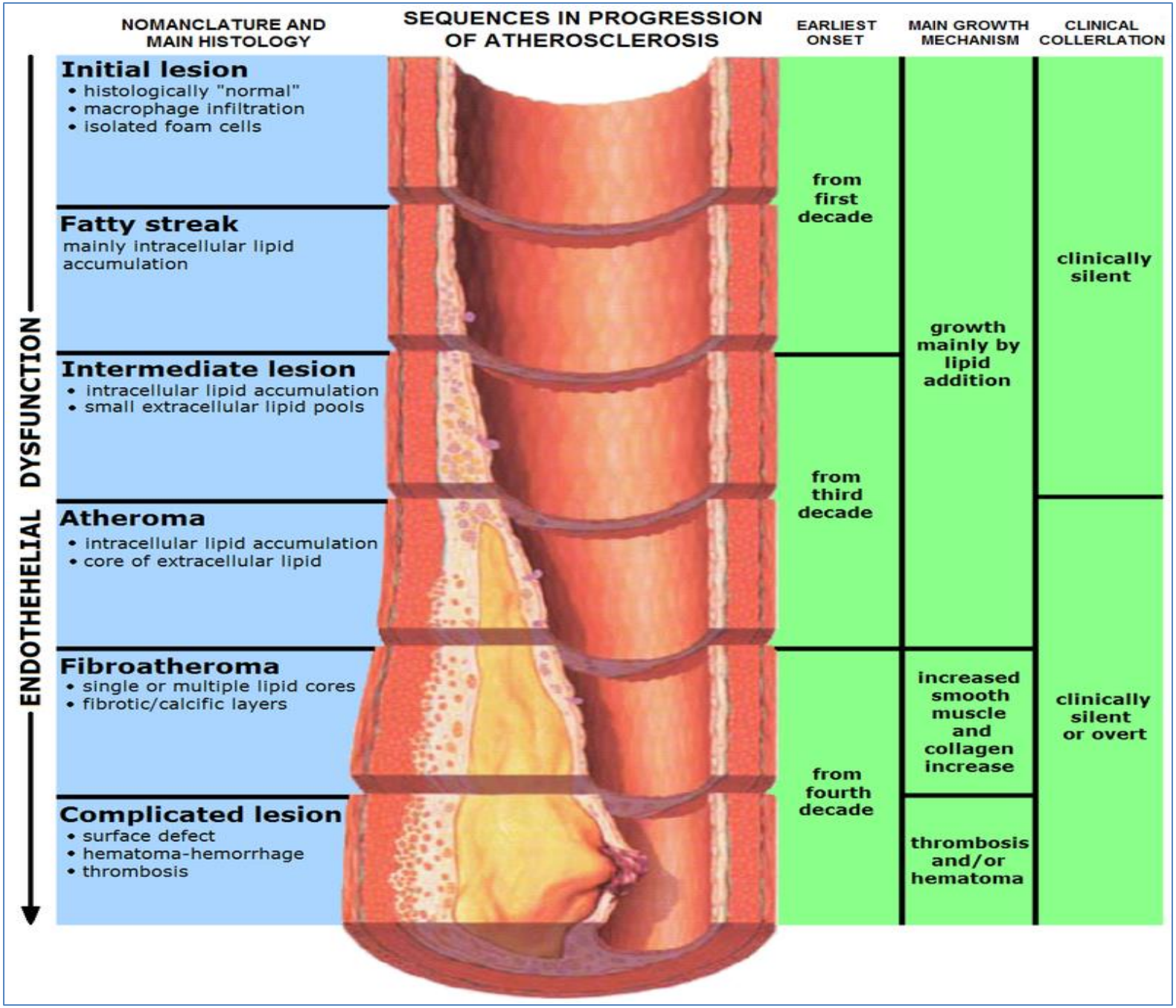


Formation of atheromatous plaque

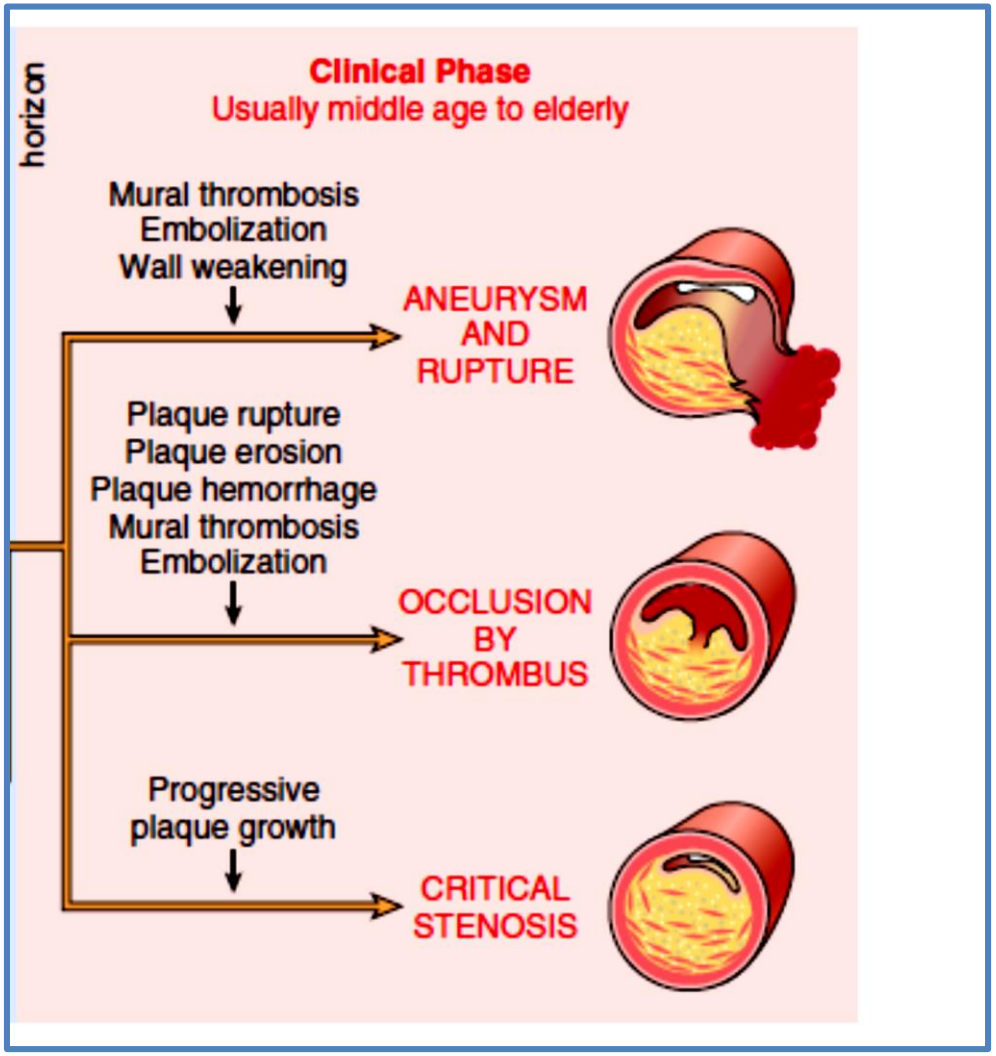


Formation of atheromatous plaque

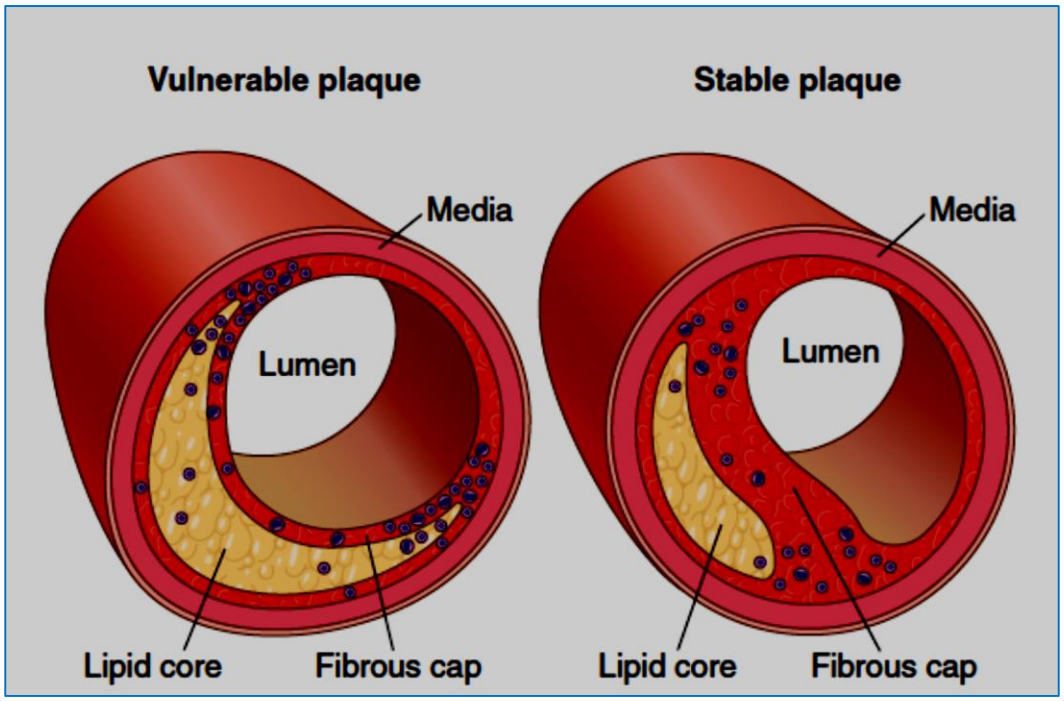



NOMANCLATURE AND MAIN HISTOLOGY
SEQUENCES IN PROGRESSION OF ATHEROSCLEROSIS
EARLIEST ONSET
MAIN GROWTH MECHANISM
CLINICAL COLLERLATION
ENDOTHEHELIAL DYSFUNCTION
Initial lesion
• histologically "normal"
• macrophage infiltration
• isolated foam cells
Fatty streak
mainly intracellular lipid accumulation
Intermediate lesion
• intracellular lipid accumulation
• small extracellular lipid pools
Atheroma
• intracellular lipid accumulation
• core of extracellular lipid
Fibroatheroma
• single or multiple lipid cores
• fibrotic/calcific layers
Complicated lesion
• surface defect
• hematoma-hemorrhage
• thrombosis
from first decade
from third decade
from fourth decade
growth mainly by lipid addition
increased smooth muscle and collagen increase
thrombosis and/or hematoma
clinically silent
clinically silent or overt

Atherosclerosis: progression



Vulnerable vs stable plaque



Thick fat core
Thin fibrous cap
More inflammation

Thin fat core
Thick fibrous cap
less inflammation

Risk Factors for Atherosclerosis

Major Risks	Lesser, Uncertain, or Non-quantitated Risks
Non-modifiable (non-controllable)	**Obsesity**
Increasing age	**Physical inactivity**
Male gender	**Stress ("type A personality)**
Family history	**Postmenopausal estrogen deficiency**
Genetic abnormalities	**High carbohydrate intake**
	Lipoprotein(a)
Potentially modifiable (Controllable)	**Hardened (trans)unsaturated fat intake**
Hyperlipidemia	
Hypertension	**Chlamydia pneumoniae infection**
Cigarette smoking	
Diabetes	
C-reactive protein (inflammation)	

1-age

- **ages 40 to 60, incidence of MI in men increases 5 x**
- **Death rates from IHD rise with each decade**

2-Gender

- **Premenopausal* → protected against atherosclerosis compared with age-matched men.**
- **After menopause→ incidence of atherosclerosis-related diseases increases**

- ***** **unless they are otherwise predisposed by diabetes, hyperlipidemia, or severe hypertension.**

3-Genetics

- **familial predisposition is multifactorial.**
- **Either :**

1- familial clustering of other risk factors

- e.g. HTN or DM

or :

2- well-defined genetic derangements in lipoprotein metabolism

- e.g. familial hypercholesterolemia

Additional Risk Factors for atherosclerosis

- 20% of cardiovascular events occur in the *absence of identifiable risk factors:*

- **Hyperhomocystinemia**
- ***Metabolic syndrome***
- **Lipoprotein a levels**
- **Factors Affecting Hemostasis (***Elevated levels of procoagulants; Clonal hematopoiesis)*
- **Others:**

-lack of exercise

-competitive, stressful lifestyle ("type A" personality)

-obesity

-High carbohydrate intake