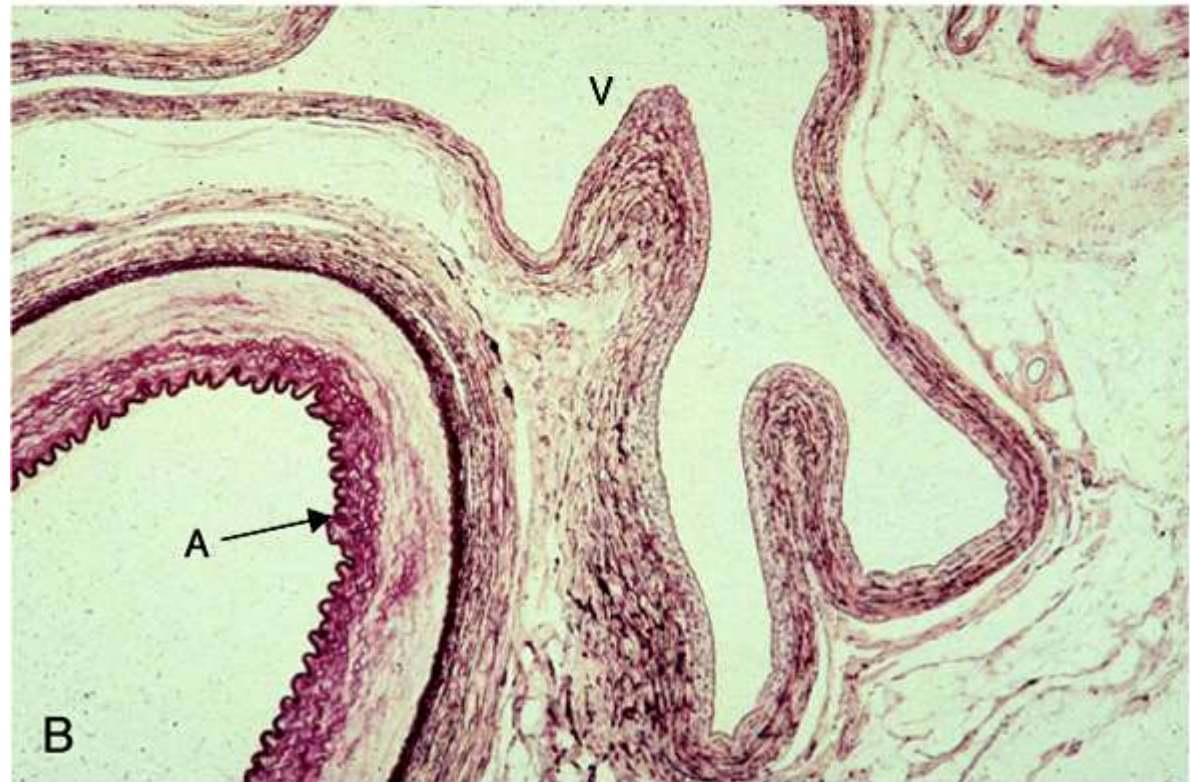
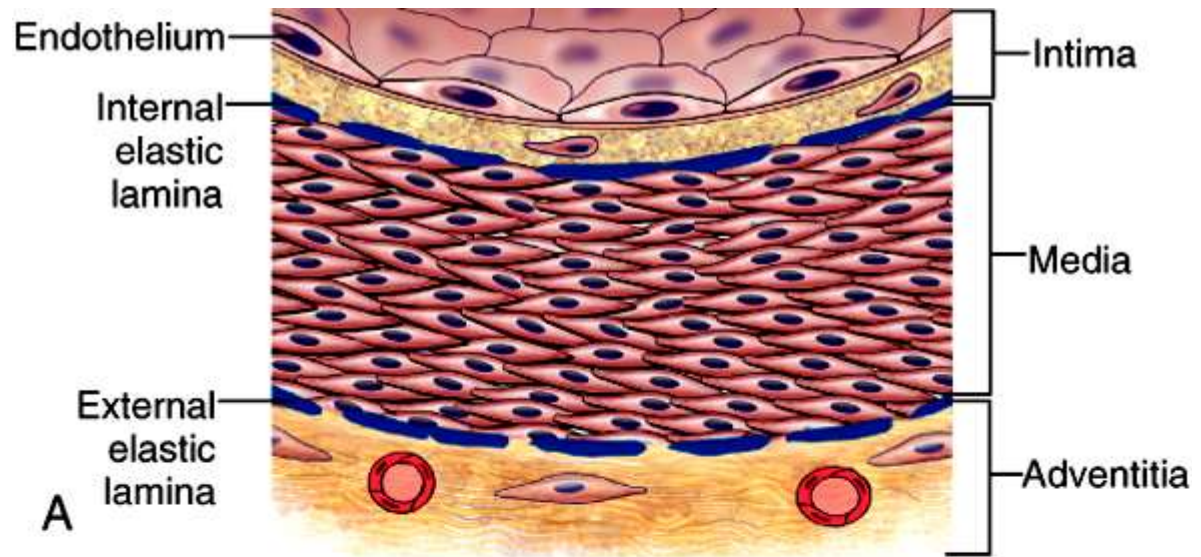




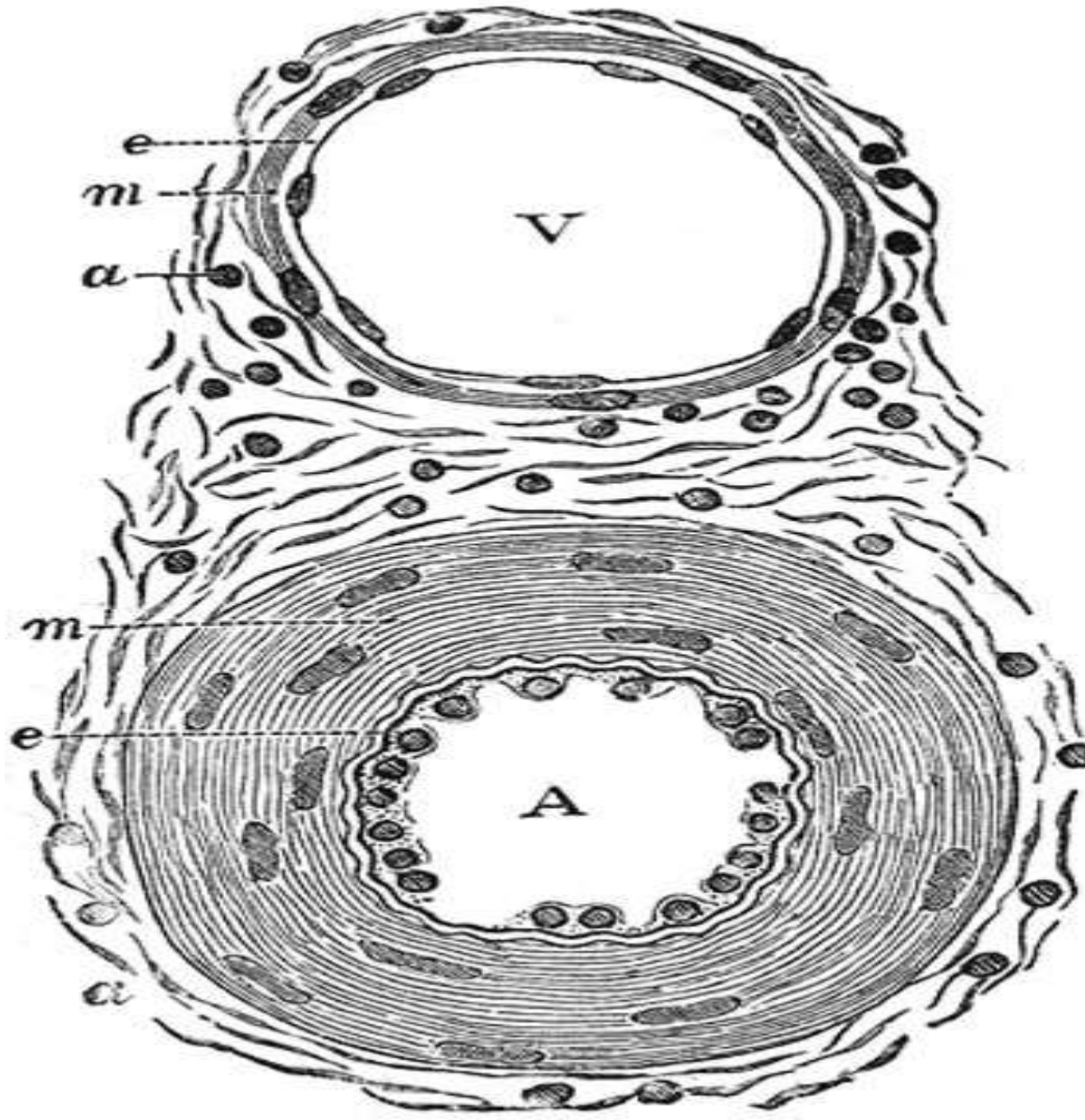
ARTERIOSCLEROSIS

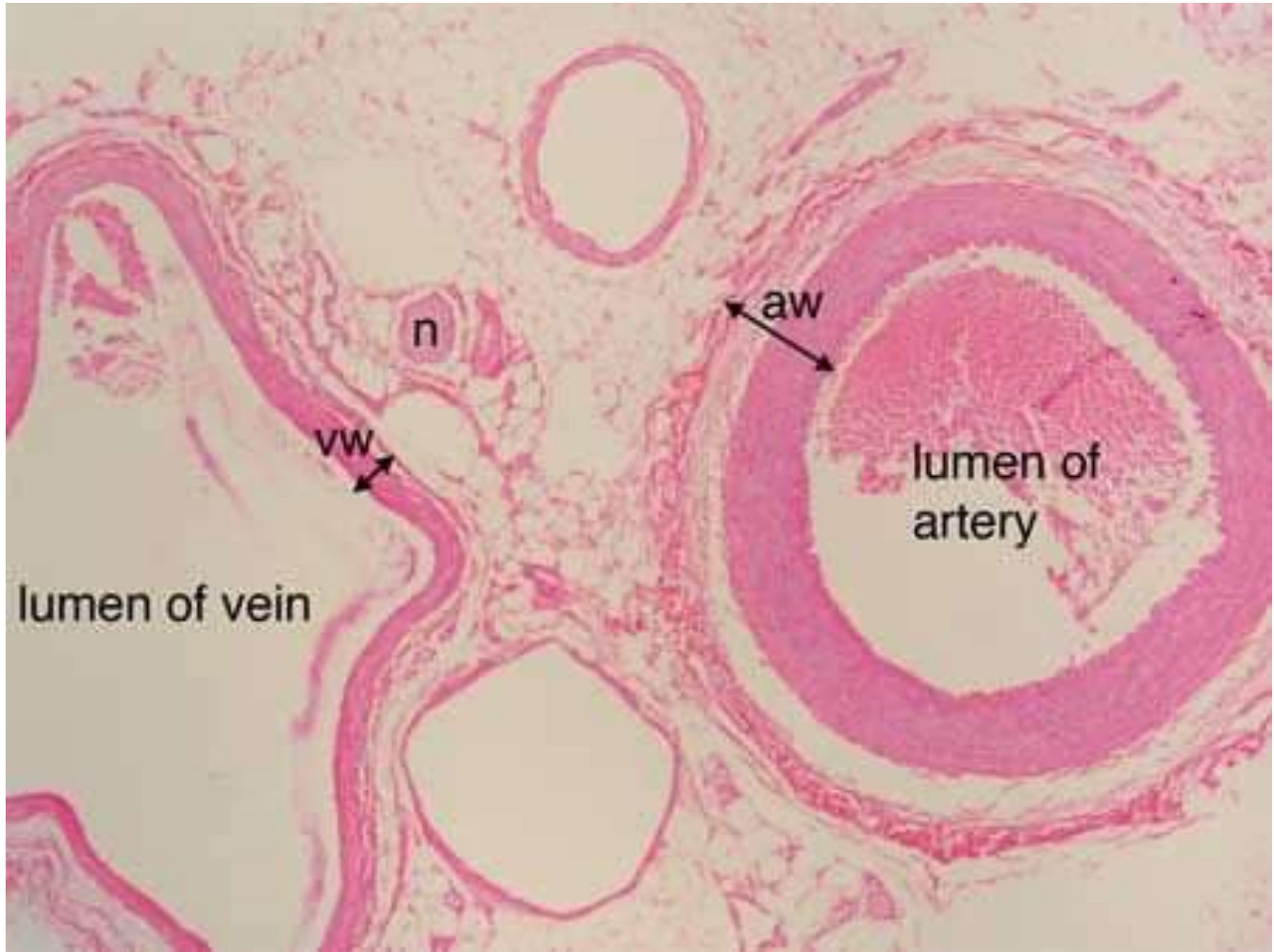
Dr. Nisreen Abu Shahin
Professor of Pathology
Pathology Department
University of Jordan

Normal
blood vessels
A= artery
V= vein



Artery (A) versus vein (V)



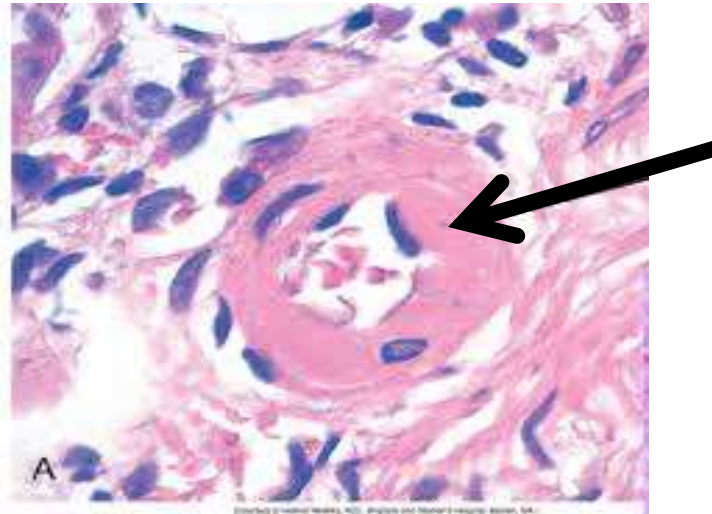


ARTERIOSCLEROSIS

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

1-Arteriolo sclerosis

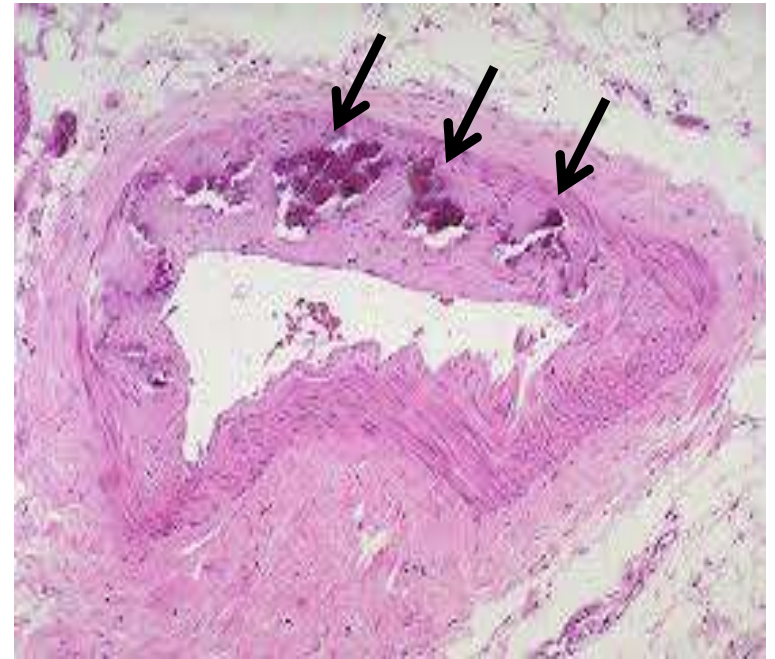
- affects small arteries and arterioles
- associated with hypertension and/or diabetes mellitus



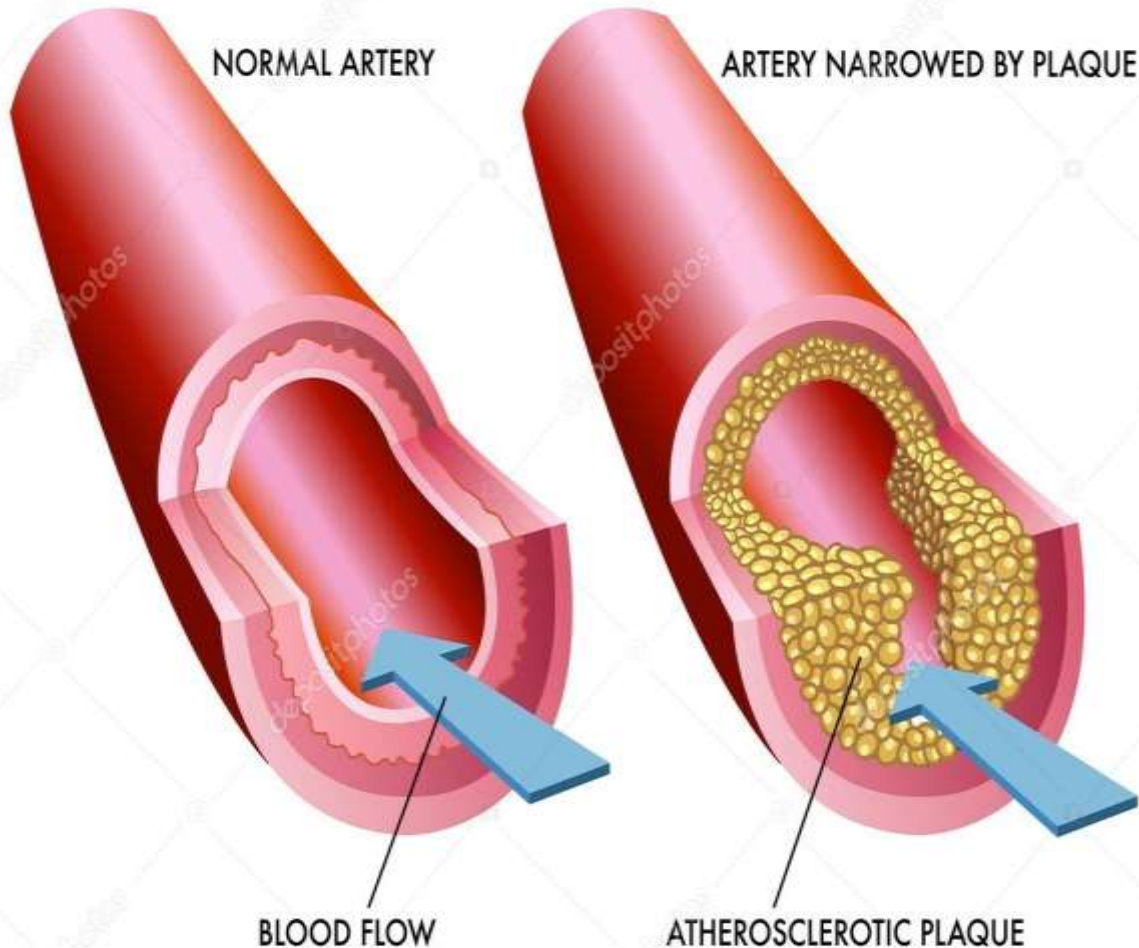
2- Mönckeberg medial calcific sclerosis

- **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



ATHEROSCLEROSIS

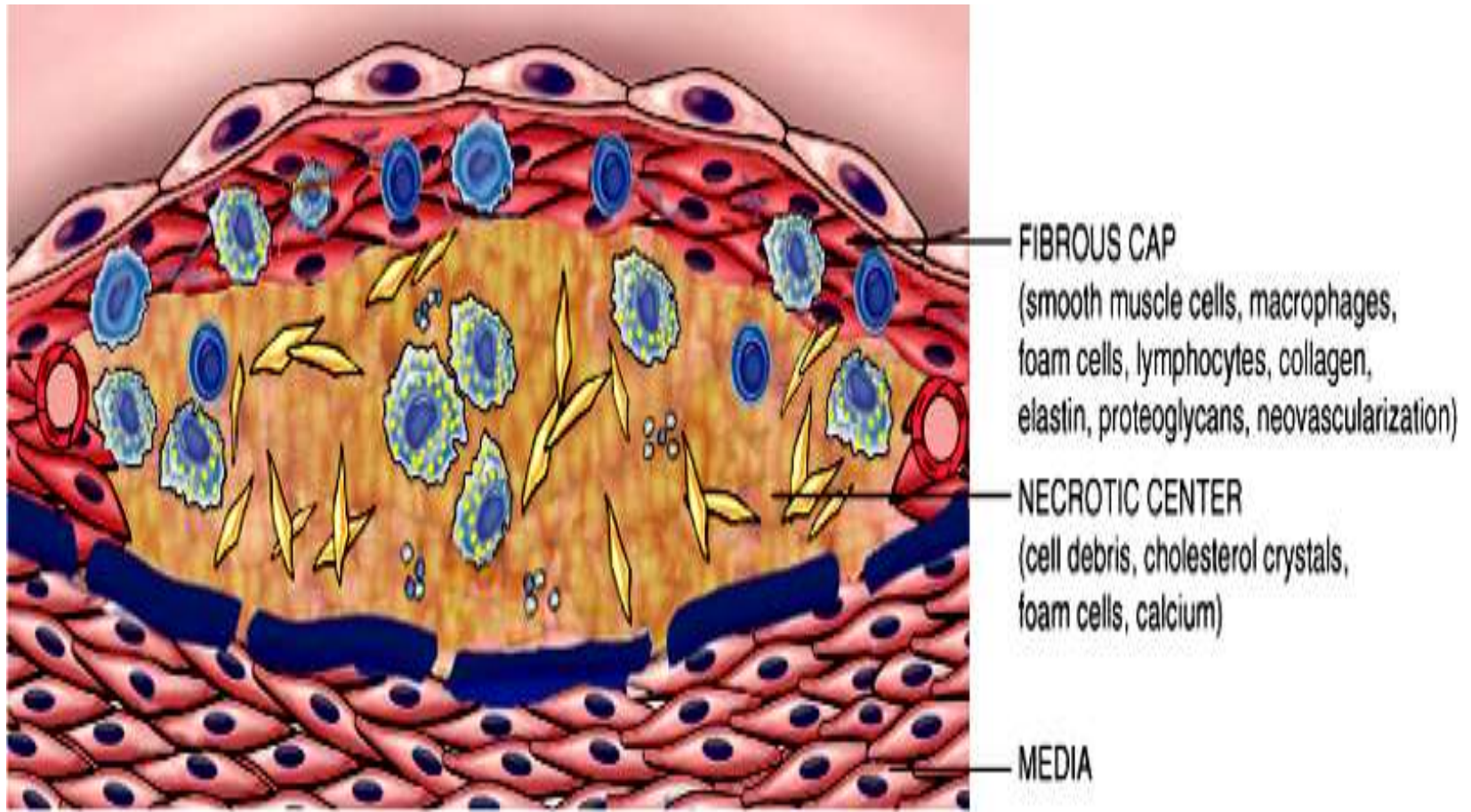


- Greek word "gruel" , "hardening,"
- most frequent and clinically important pattern of arteriosclerosis
- characterized by intimal lesions = *atheromas* (a.k.a. *atherosclerotic plaques*)
- atheromatous plaque = raised lesion with a core of lipid (cholesterol and cholesterol esters) covered by a firm,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

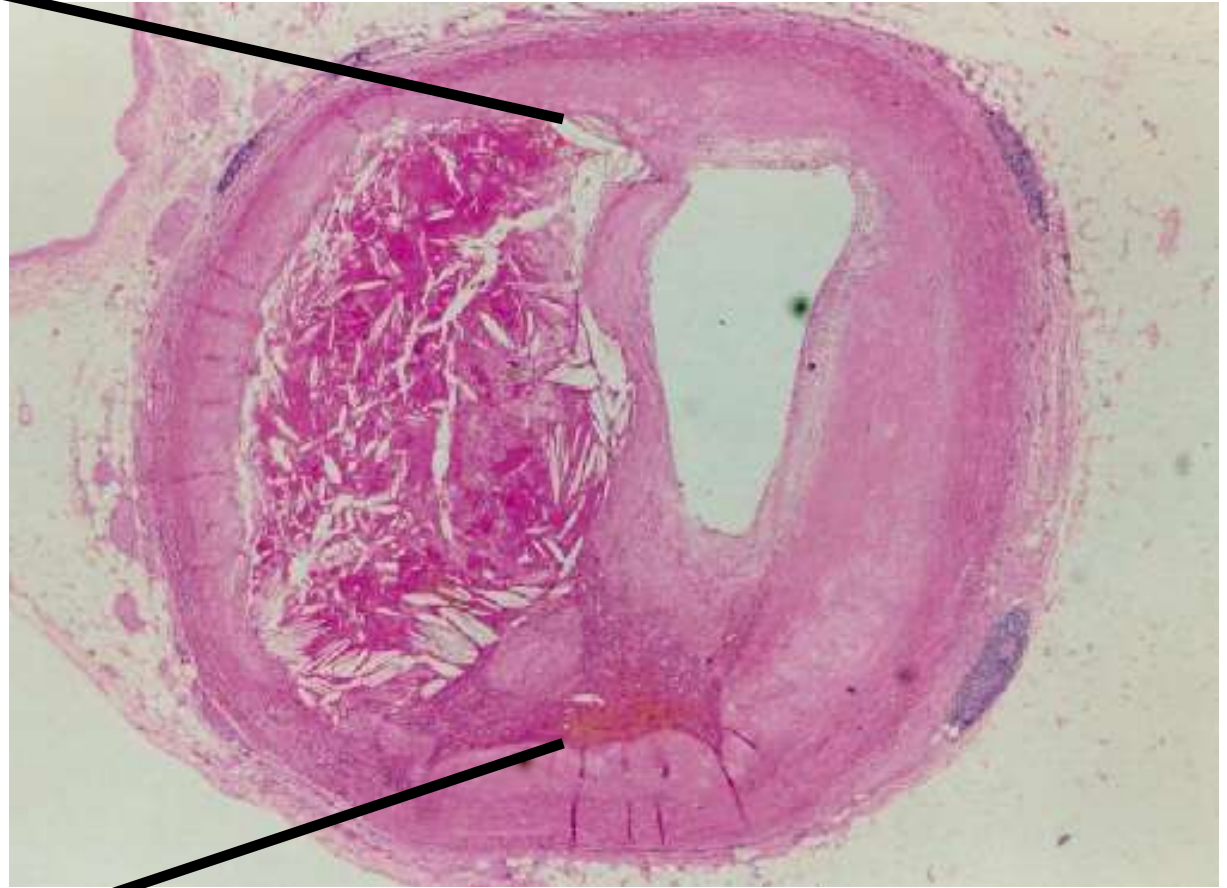
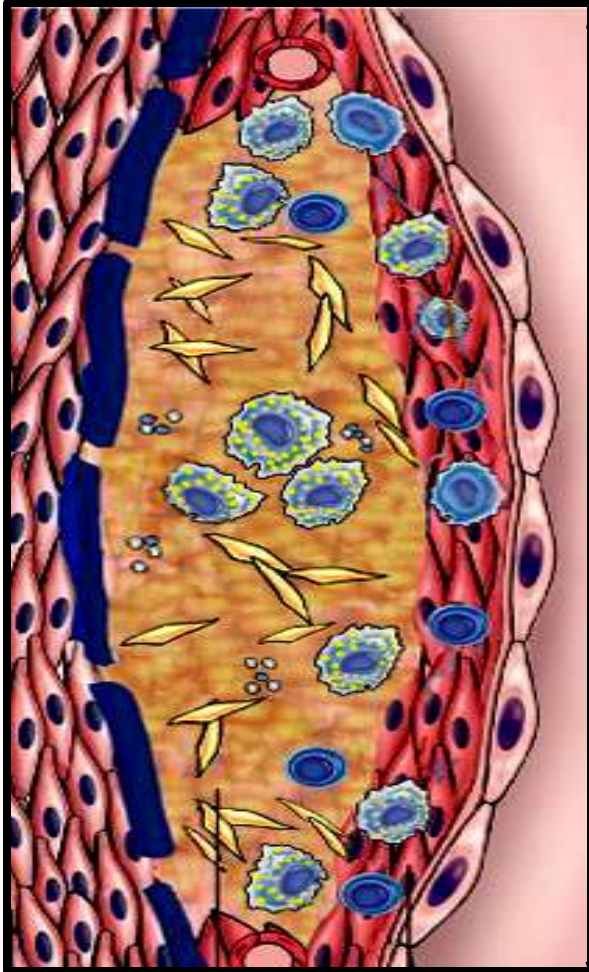
Atherosclerosis- Pathogenesis

- **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**

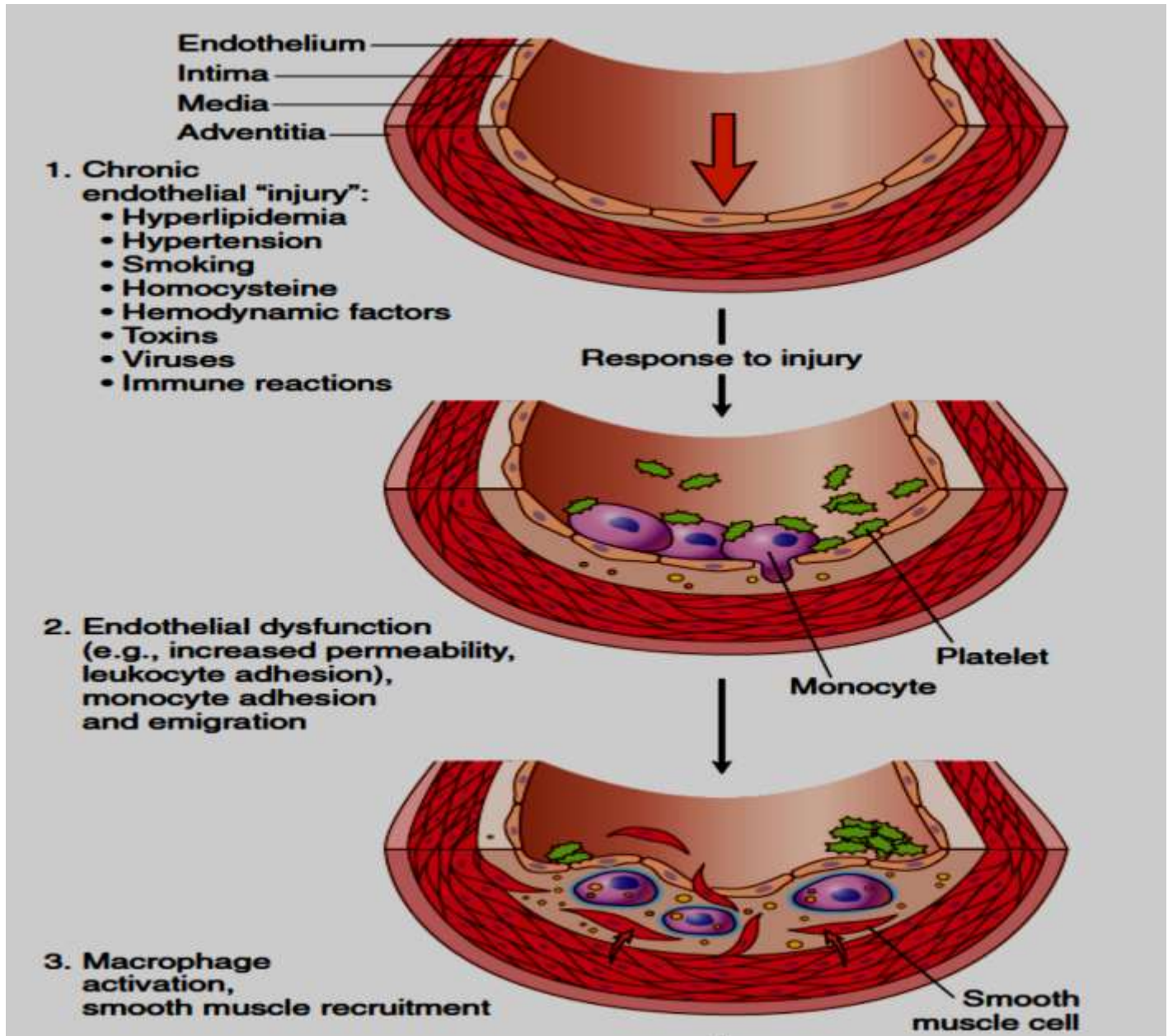
The major components of a well-developed intimal atheromatous plaque



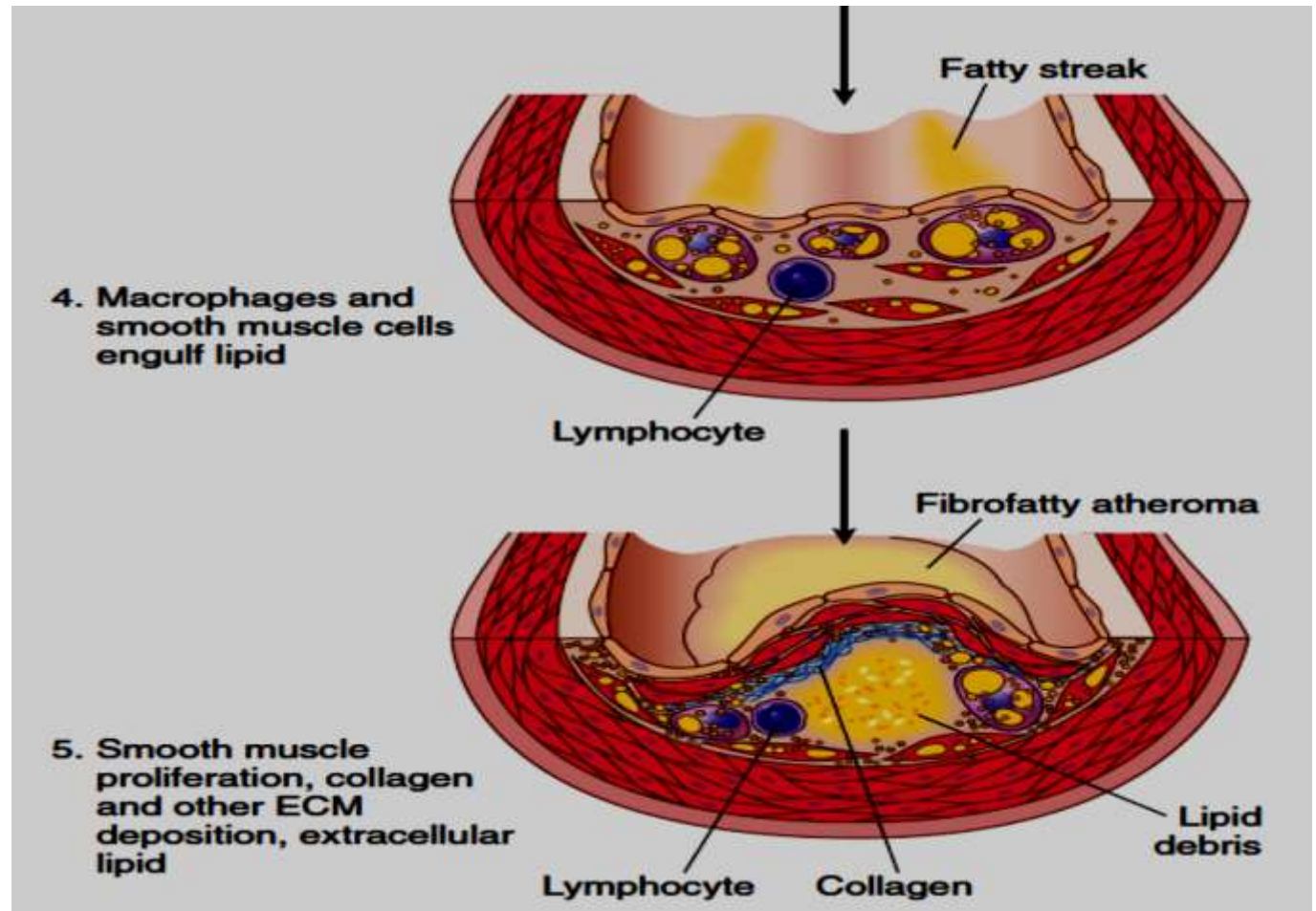
Atheromatous plaque

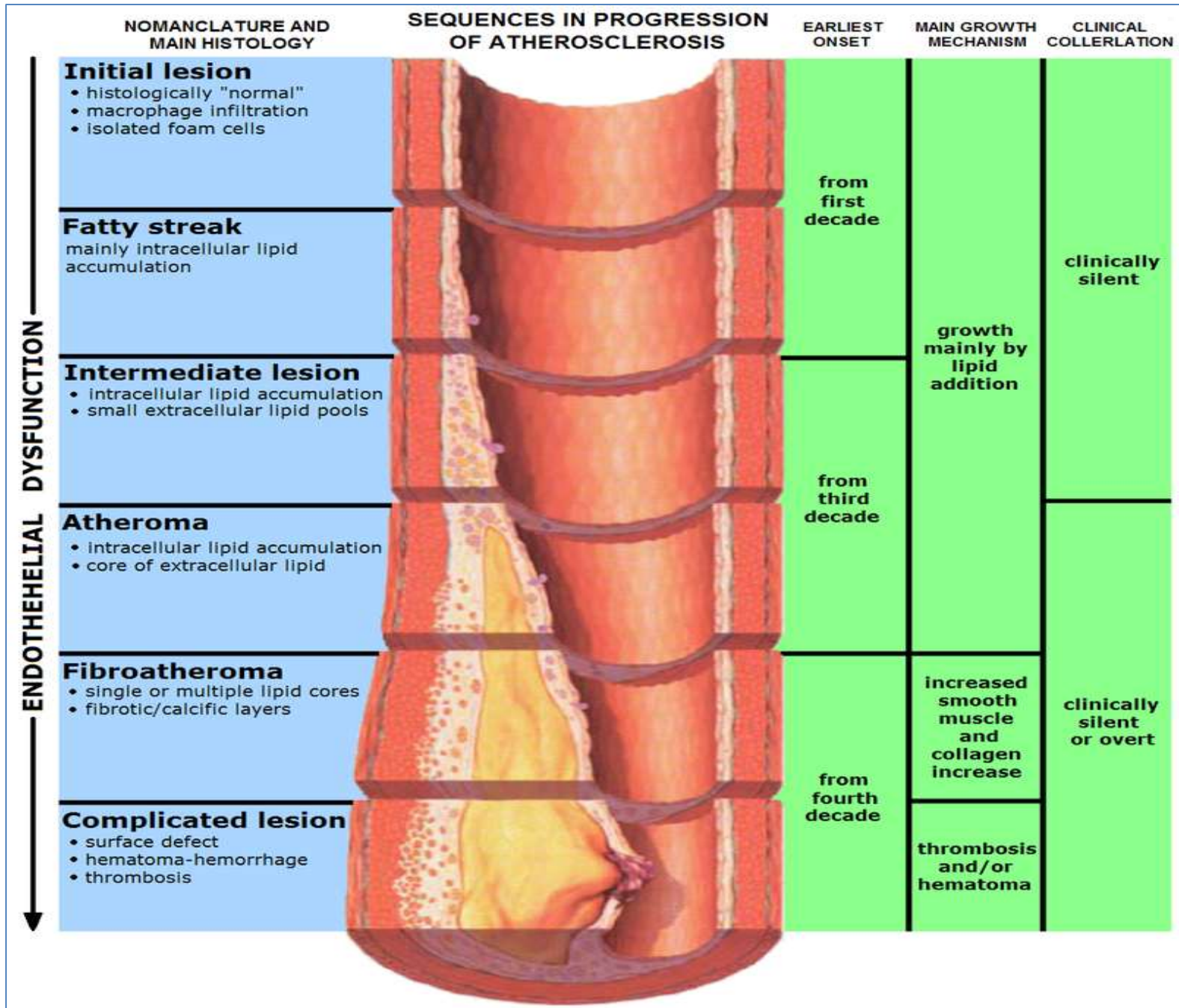


Formation of atheromatous plaque

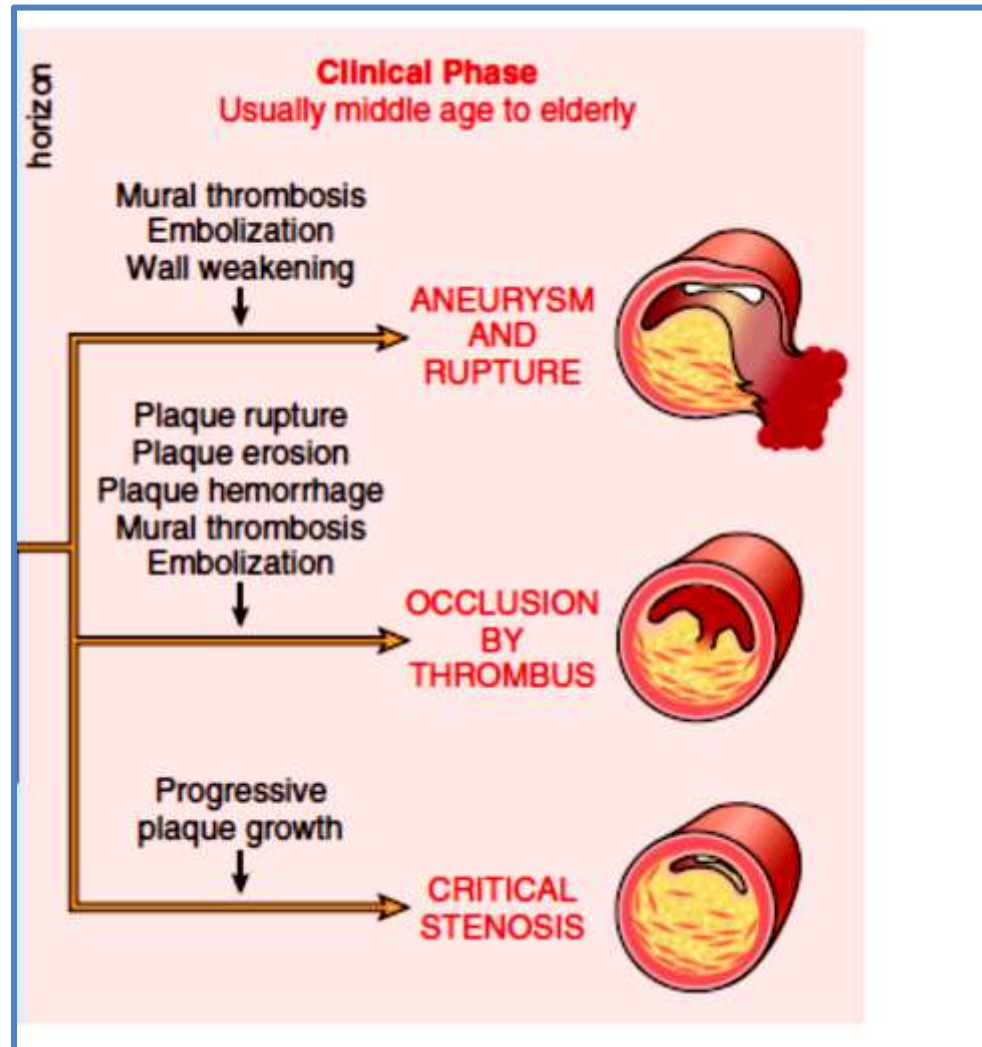


Formation of atheromatous plaque

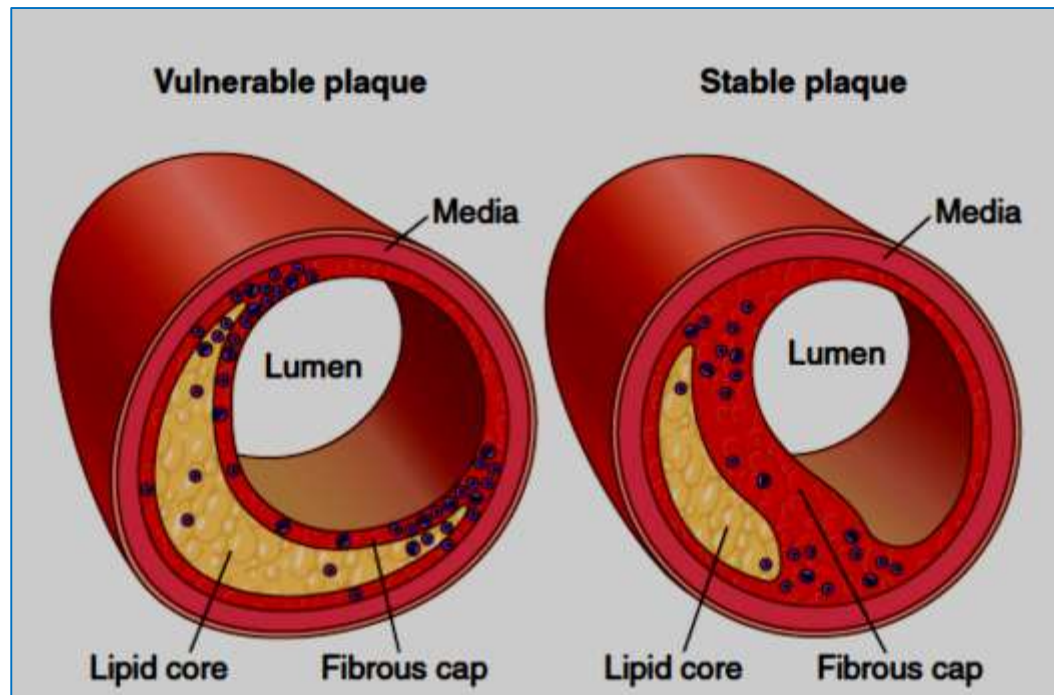




Atherosclerosis: progression



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)	Obesity	
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....*)
 - **Others:**
 - lack of exercise
 - competitive, stressful lifestyle ("type A" personality)
 - obesity
 - High carbohydrate intake