

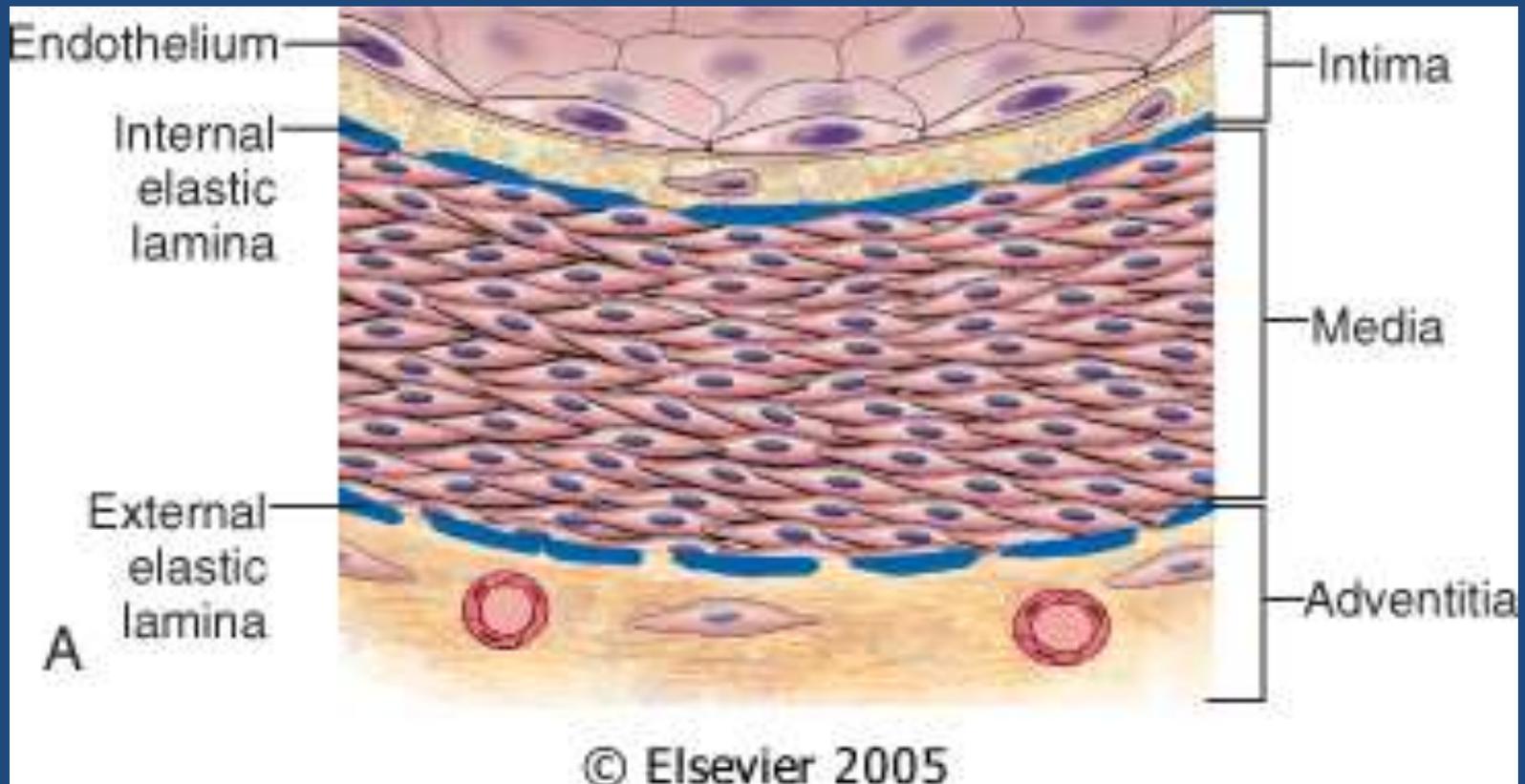
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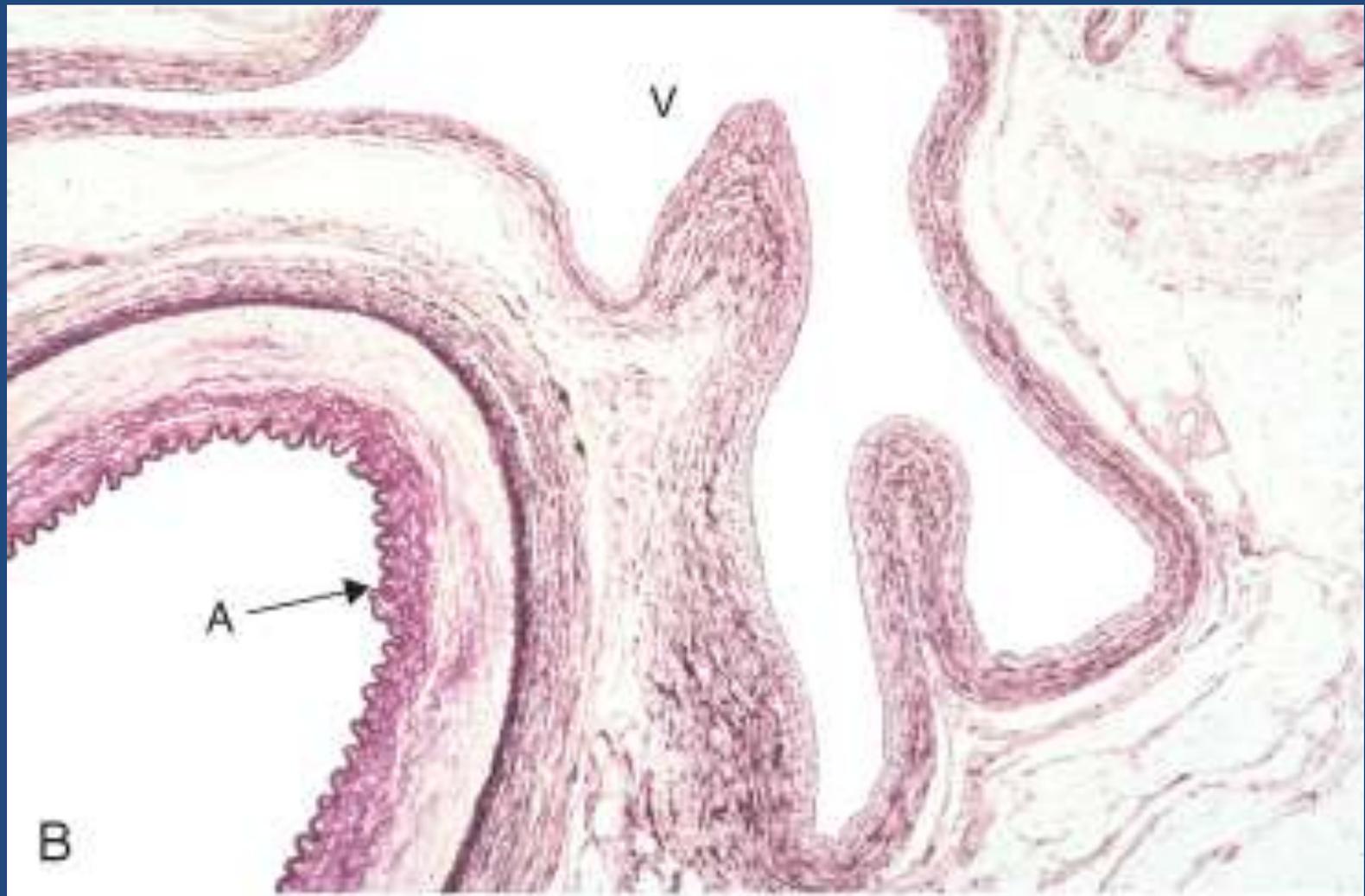
Atherosclerosis

CVS 1

Hisham Al Khalidi

Vessel wall structure





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cm

A70-130

1

2

3

4

5

6





Atherosclerosis

- A type of arteriosclerosis
- Chronic inflammatory response in the walls of arteries
- Slowly progressive
- A build-up of fat (cholesterol) within the artery wall
- Characterized by intimal lesions called: atheromas, atheromatous or fibrofatty plaques

Atherosclerosis

Common sites

- Abdominal aorta
- Coronaries
- Popliteal artery
- The internal carotid arteries
- The vessels of the circle of Willis

Atherosclerosis

Risk factors

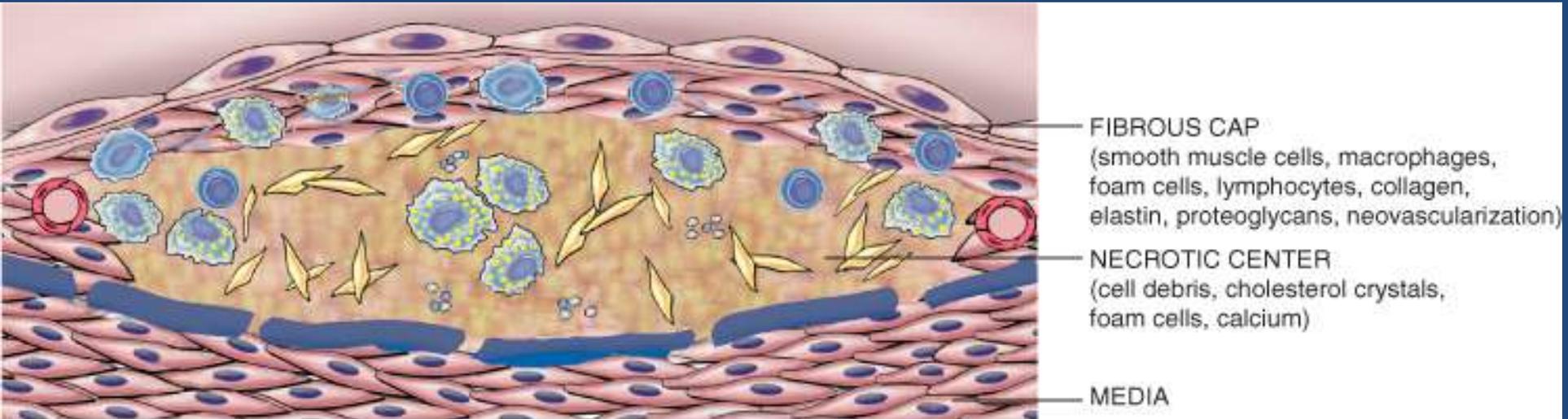
Major	Lesser, Uncertain, or Nonquantitated
<i>Nonmodifiable</i>	
Increasing age	Obesity
Male gender	Physical inactivity
Family history	Stress ("type A" personality)
Genetic abnormalities	Postmenopausal estrogen deficiency
	High carbohydrate intake
<i>Potentially Controllable</i>	
Hyperlipidemia	Alcohol
Hypertension	Lipoprotein Lp(a)
Cigarette smoking	Hardened (trans)unsaturated fat intake
Diabetes	<i>Chlamydia pneumoniae</i>

LDL Vs. HDL

- LDL cholesterol : deliver cholesterol to peripheral tissues
- HDL, "good cholesterol": mobilizes cholesterol from developing and existing atheromas and transports it to the liver for excretion in the bile

Atherosclerosis

Fibrous cap



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Atherosclerosis Pathogenesis

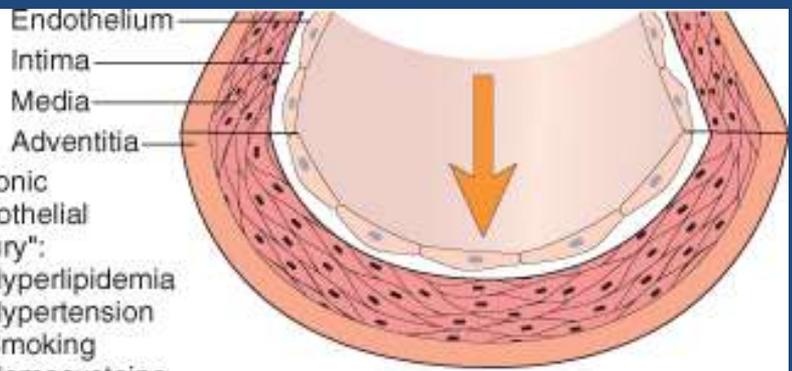
- The main components of a fibrofatty plaques:
 - Lipids
 - Extracellular matrix
 - Cells, Proliferating smooth muscle cells

Atherosclerosis

Pathogenesis

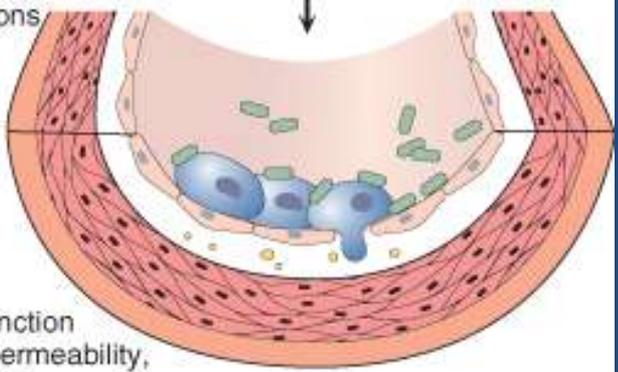
response-to-injury hypothesis

- Endothelial injury
 - Not completely understood
 - Nevertheless, the two most important causes of endothelial dysfunction are:
 - Hemodynamic disturbances
 - Hypercholesterolemia
 - Inflammation is also an important contributor.
- Smooth muscle cell proliferation

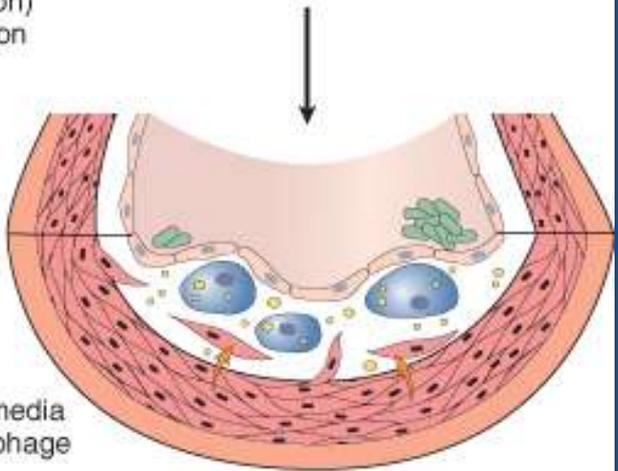


1. Chronic endothelial "injury":
- Hyperlipidemia
 - Hypertension
 - Smoking
 - Homocysteine
 - Hemodynamic factors
 - Toxins
 - Viruses
 - Immune reactions

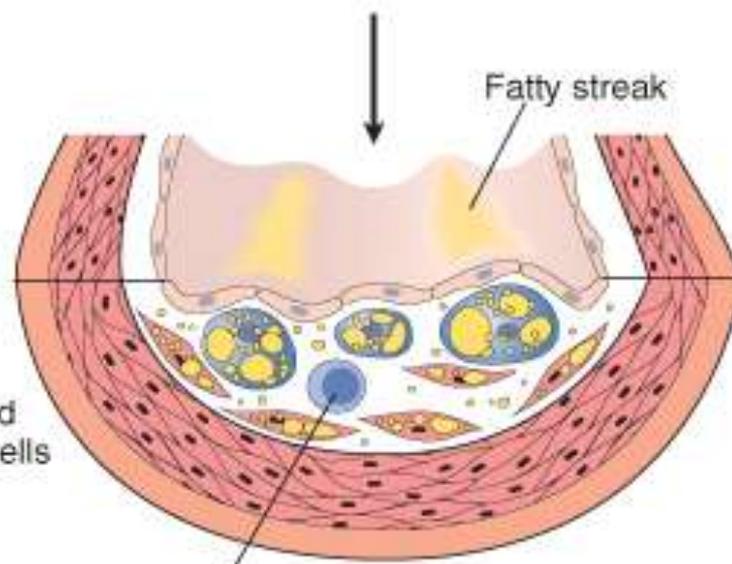
Response to injury



2. Endothelial dysfunction (e.g., increased permeability, leukocyte adhesion) Monocyte adhesion and emigration.

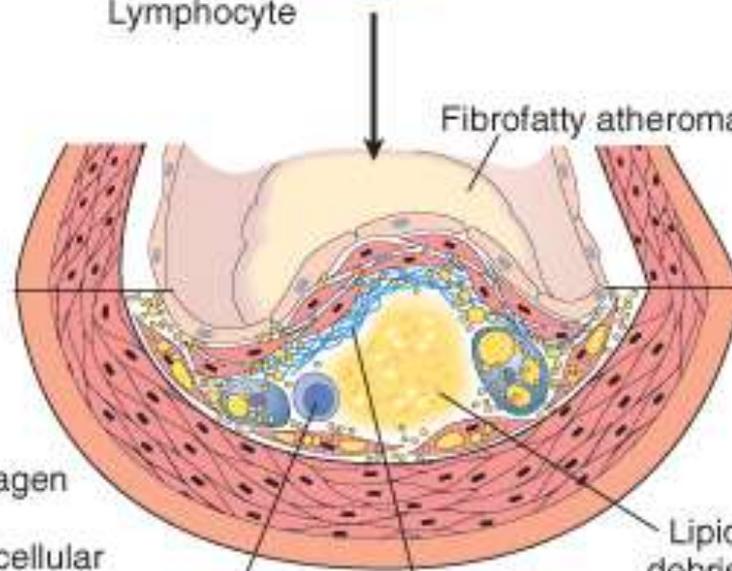


3. Smooth muscle emigration from media to intima. Macrophage activation.



4. Macrophages and smooth muscle cells engulf lipid

Lymphocyte



5. Smooth muscle proliferation, collagen and other ECM deposition, extracellular lipid

Fibrofatty atheroma

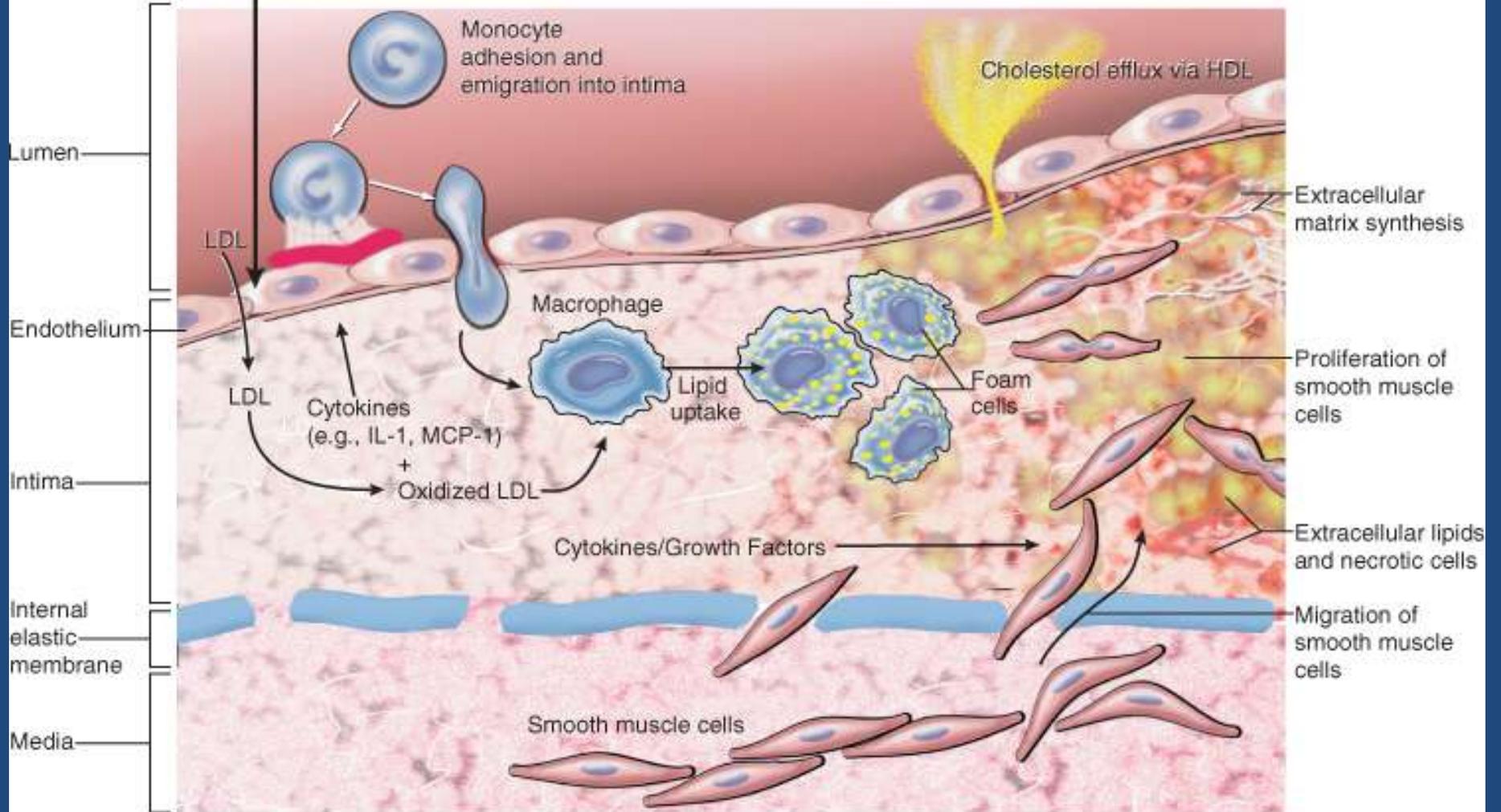
Lymphocyte

Collagen

Lipid debris

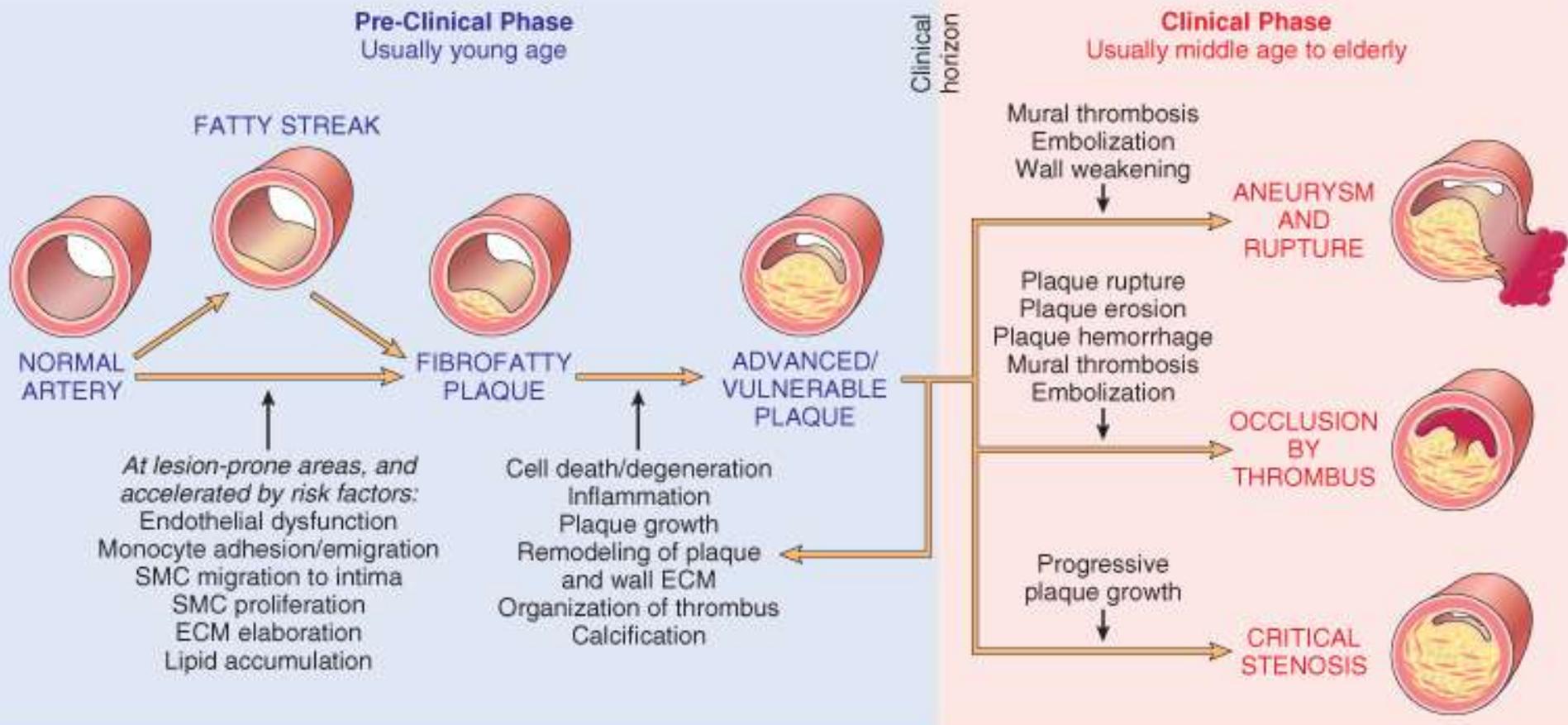
Hyperlipidemia, Hypertension,
Smoking, Toxins, Hemodynamic
factors, Immune reactions, Viruses

Endothelial Injury/Dysfunction



Atherosclerosis

Consequences



Atherosclerosis

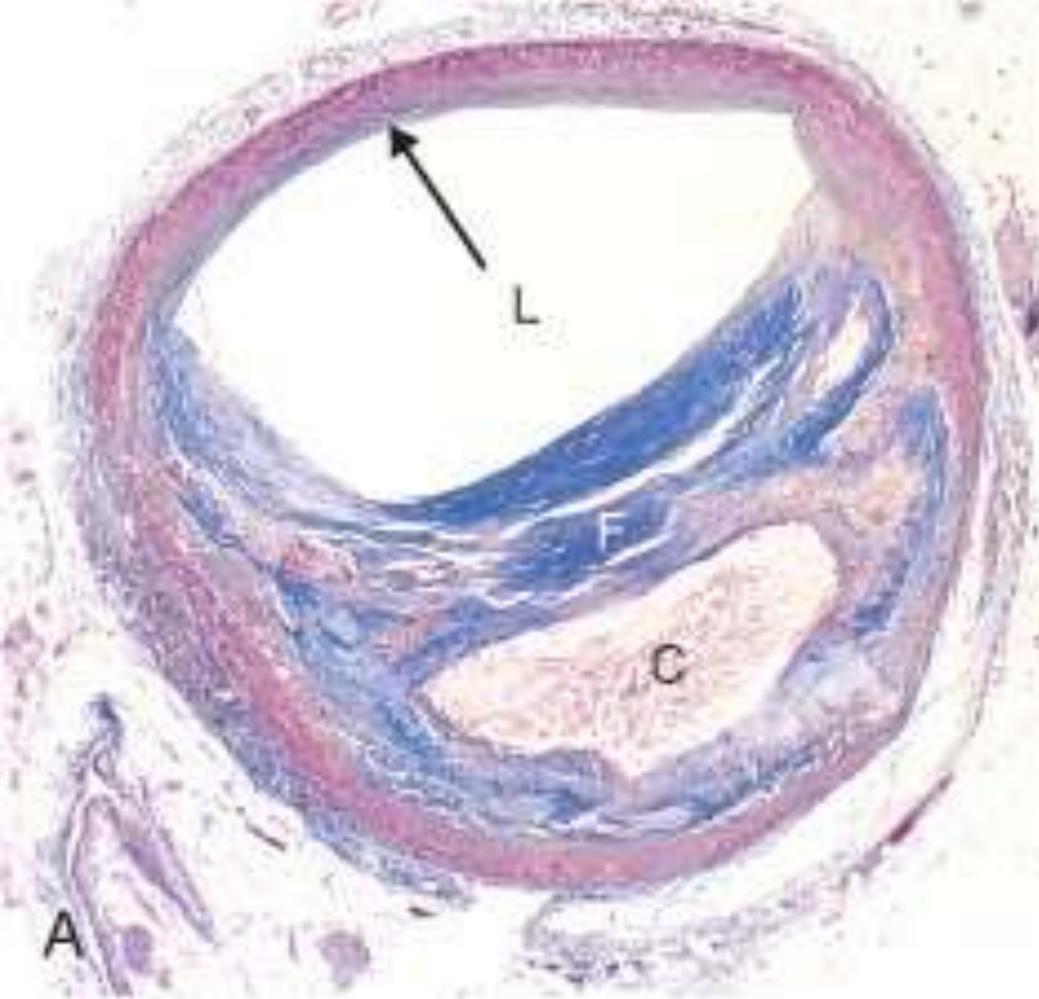
Clinical Complications

- Myocardial infarction (heart attack)
- Cerebral infarction (stroke)
- Aortic aneurysms
- Mesenteric occlusion
- Peripheral vascular disease (gangrene of the legs)

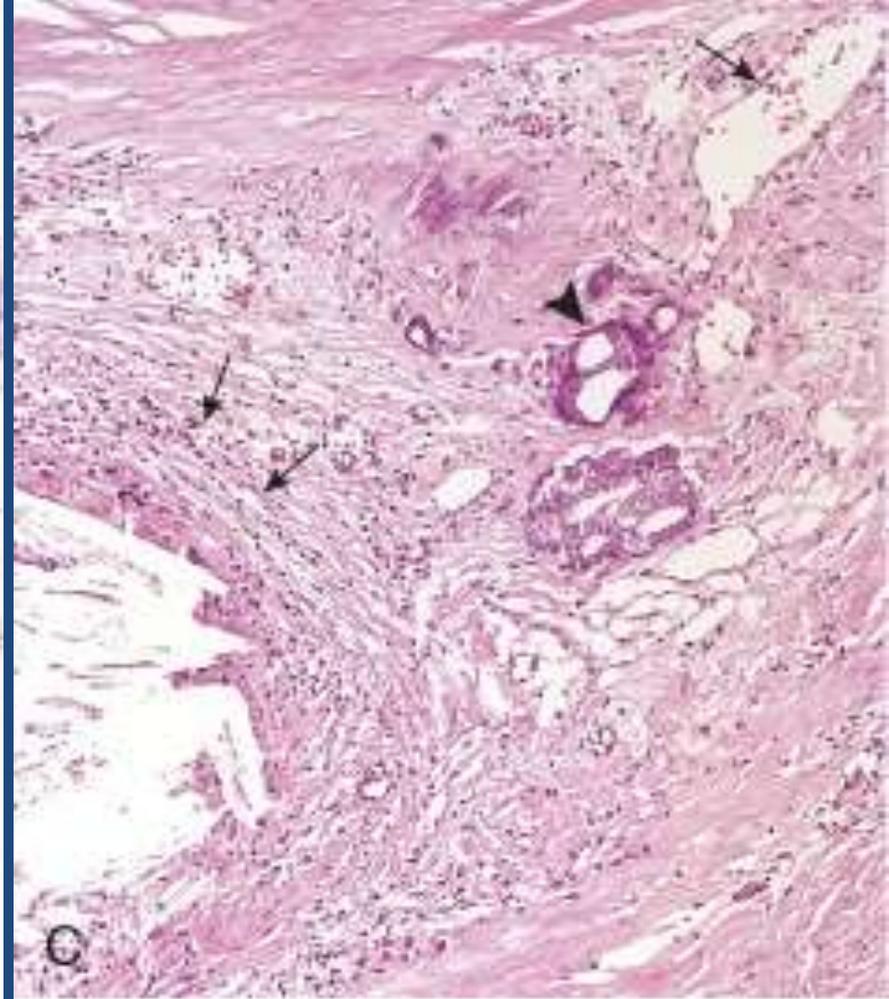
Morphological terms that are seen on macro and microscopic levels

- Calcification
- Hemorrhage
- Fissure
- Ulcer
- Thrombosis
- Neovascularization
- Medial thinning
- Cholesterol microemboli
- Aneurysmal dilatation





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