Ischemic Heart disease

IHD

- Acute or chronic form of cardiac disability arising from myocardial ischemia- imbalance between myocardial supply and demand for oxygenated blood
- Ischemia comprises insufficiency of O2 & nutrients; inadequate removal of metabolites
- In 90% cases, cause is ↓ blood flow due to obstructive atherosclerotic lesion, so alternatively called Coronary artery dis (CAD)
- M> F

Clinical syndromes

1. Myocardial Infarction
2. Angina pectoris: 3 variants:
   - *stable angina*
   - *Prinzmetal angina*
   - *unstable angina*
3. Sudden Cardiac Death
4. Chronic IHD with heart failure

Acute coronary syndrome

Etiopathogenesis

1. Coronary atherosclerosis
2. Superadded changes in coronary atherosclerosis
3. Non- atherosclerotic causes

Coronary atherosclerosis

- Fixed lumen obstruction major cause in 90% of cases
- Distribution- highest incidence in LAD > RCA>LCX
- >75% occlusion causes symptomatic ischemia induced by exercise
- 90% occlusion can lead to symptoms even at rest
- Location- area of severe involvement is 3-4cms from coronary ostia; most often at/ near the bifurcation
- Slowly developing atheromas over long periods lead to collateral circulation

Super-added changes in Plaques

Acute coronary syndromes are precipitated by changes superimposed on pre-existing fixed coronary atheroma:

- haemorrage: causes volume expansion
- fissuring, ulceration→ exposure of highly thrombogenic subendothelial tissues to blood
- local platelet aggregation plug → which release TxA2 (vasospasmic mediator), responsible for coronary vasospasm
- thrombosis, emboli & microinfarcts

Classification of atherosclerosis

- Type I- initial lesions
- Type II- fatty streaks
- Type III- intermediate lesions
- Type IV- atheromatous lesions
- Type V- fibrofatty lesions
- Type VI- complicated lesions

Cause of plaque change

- Extrinsic influences

- adrenergic stimulation →rise in B.P, sudden coronary A spasm, tachycardia, platelet hyper- reactivity

- acute coronary synd occur most commonly b/w 6.00 A.M to 12 noon concurrent with adrenaline surge

- intense emotional stress

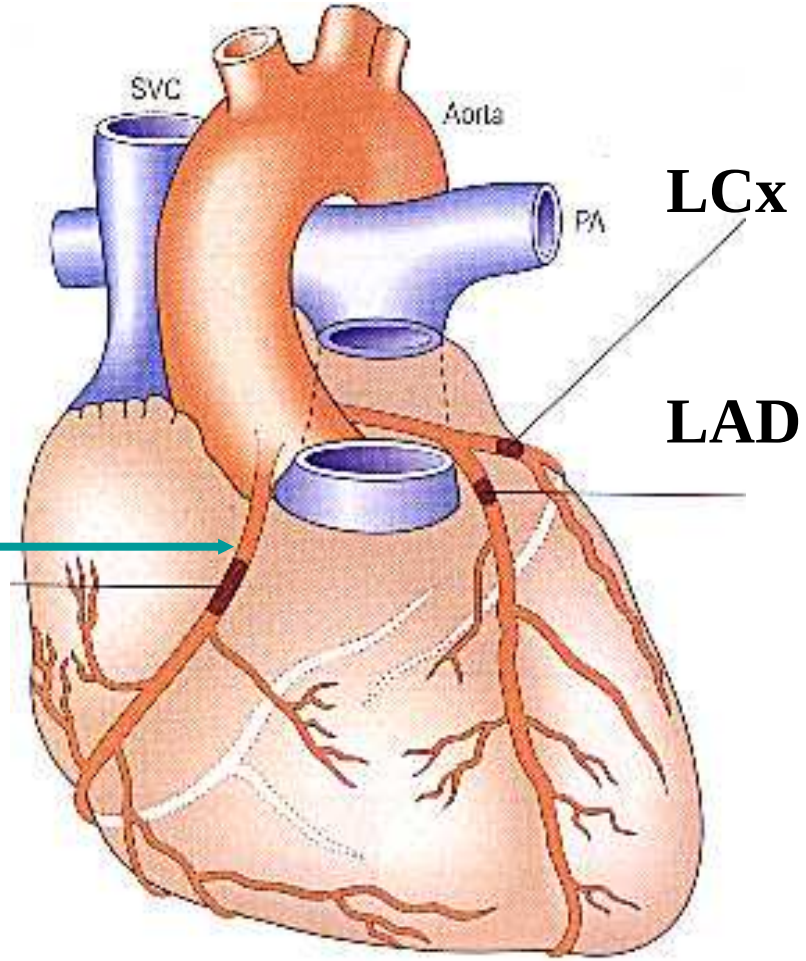
- Intrinsic influences

- plaque structure & composition

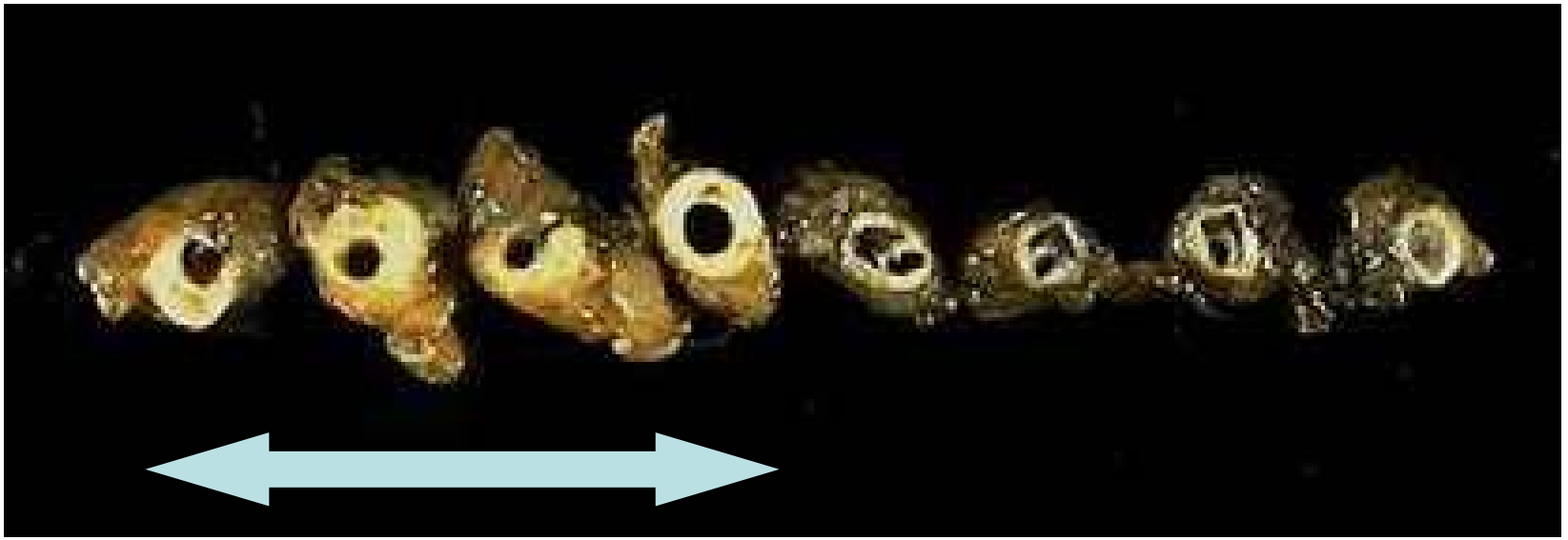
  plaques with more foam cells and extra cellular lipids & thin fibrous caps with deficient smooth muscle are vulnerable plaques & predisposed to rupture

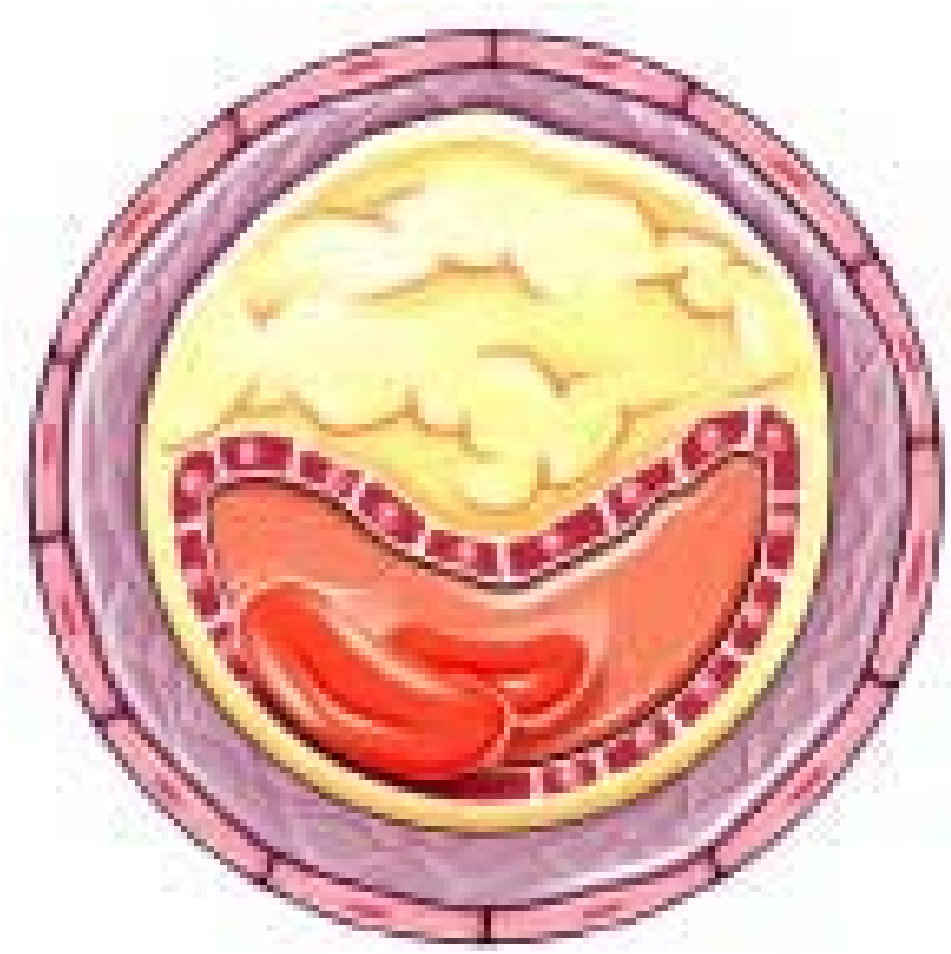
Coronary Atheorsclerosis

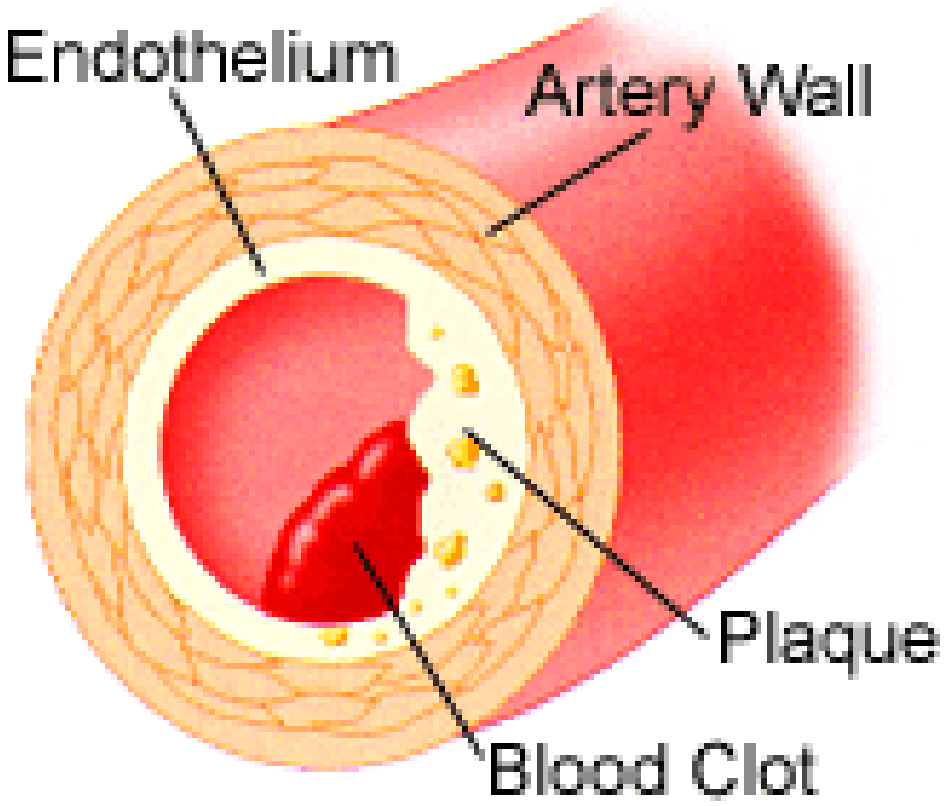
- **Left Coronary Artery.**
  - Anterior Descending (**LAD**)
  - Left Circumflex (**LCx**)
- **Right Coronary Artery.**



Coronary Narrowing in Atherosclerosis:

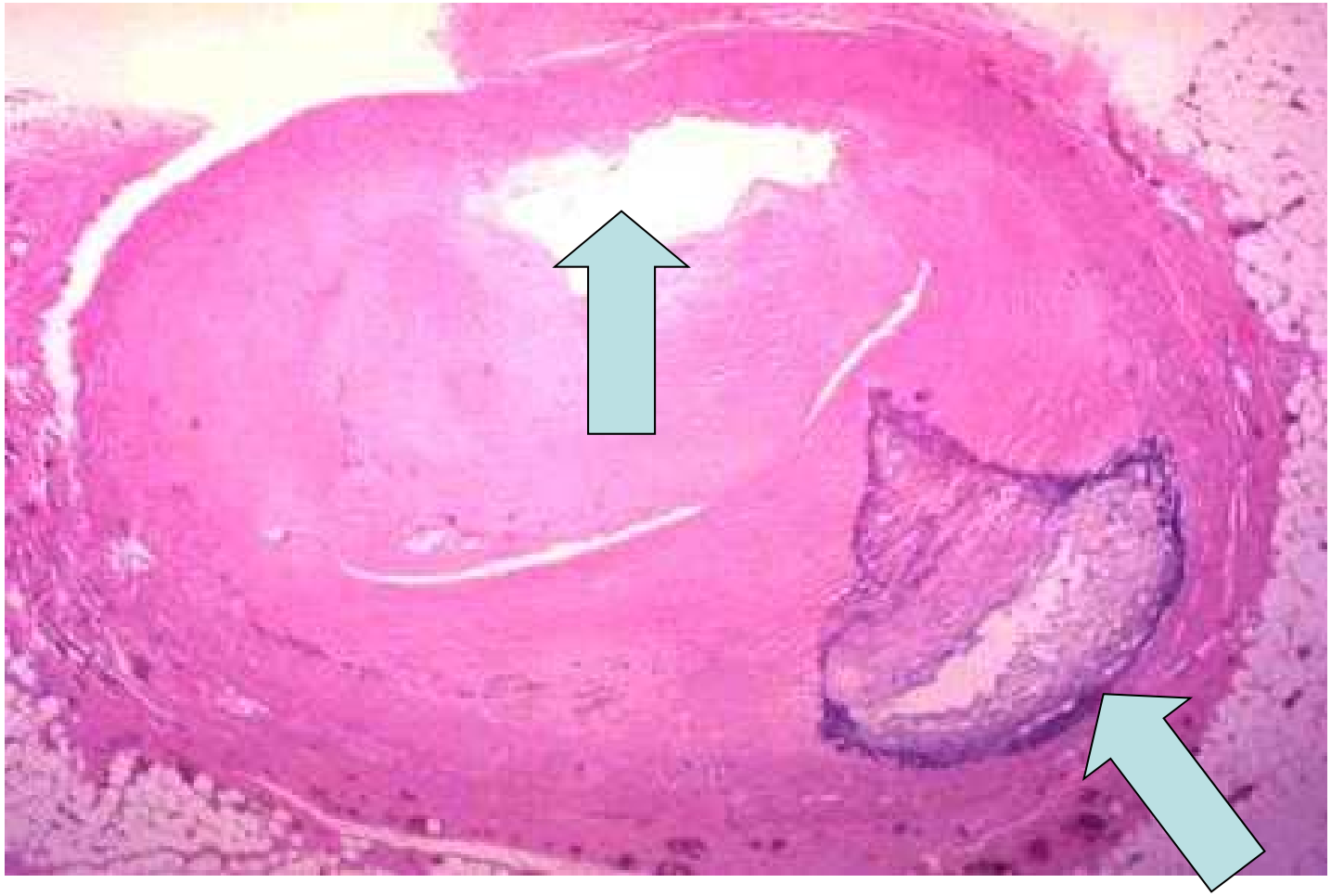






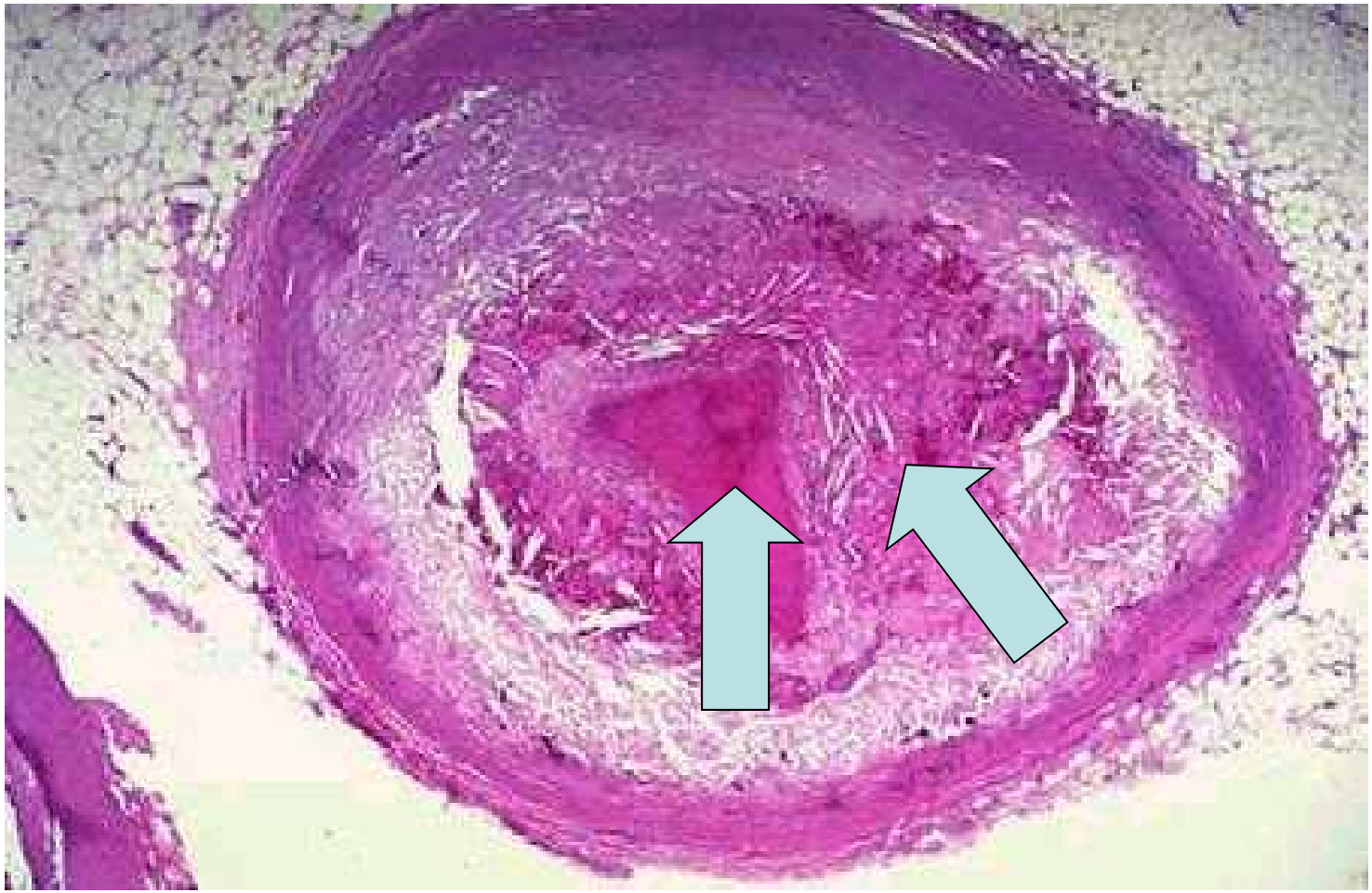
Diseased Artery

Atheroma Coronary Artery:

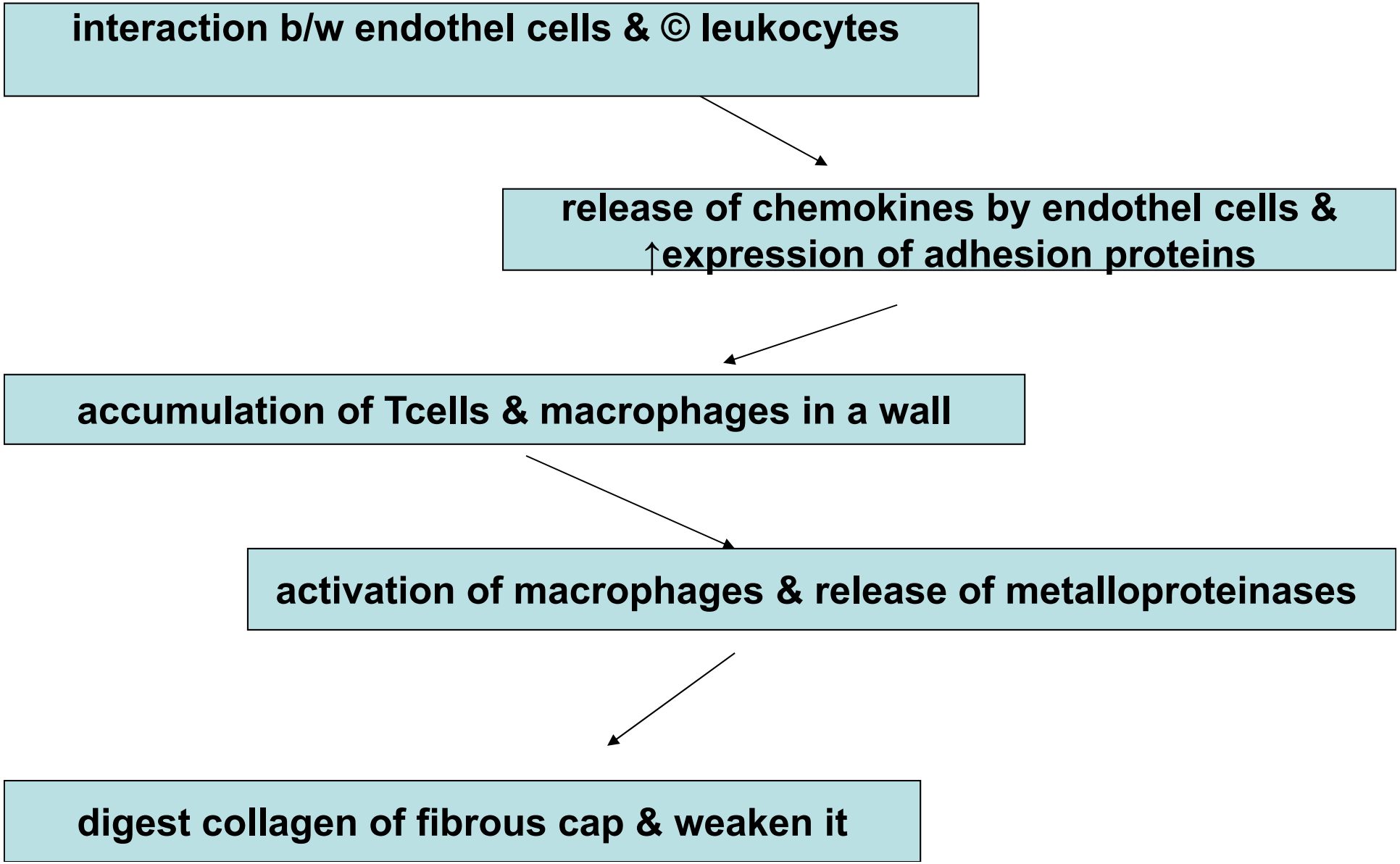


Calcification

Atheroma with Thrombosis:



Role of inflammation



Role of inflammation

- C-reactive protein- CRP, an acute phase reactant secreted by liver, is a predictor of coronary a dis.
- Used to estimate risk of M.I in patients with angina & risk of reinfarct in M.I patients

Non- atherosclerotic causes

1. Vasospasm: despite no significant atherosclerotic coronary narrowing may cause angina/ M.I.

- circulating adrenergic agonists
- locally released contents of platelets
- ↓ secretion of relaxing factors

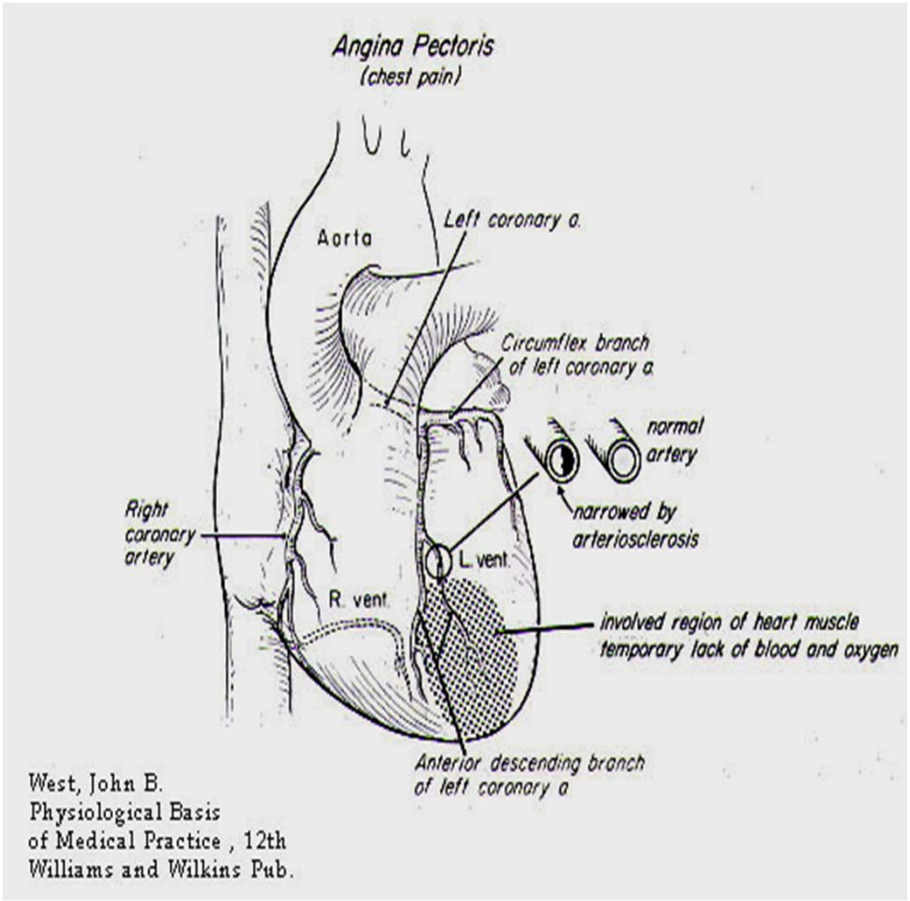
2. Stenosis of coronary ostia: from syphilitic aortitis
3. Arteritis: PAN, Kawasaki dis, Takayasu's dis
4. Embolism: fat, air, vegetations from endocarditis
5. Thrombotic dis: TTP, sickle cell dis: hypercoagulability of the blood→coronary occlusion
6. Trauma
7. Aneurysms/ Dissections occluding the lumen of the ostia
8. Compression from adjacent structures: tumors, tension pneumothorax

Angina pectoris

- Clinical synd arising from transient myocardial ischemia (15 sec to 15 min) that falls short of inducing cellular necrosis that defines infarction
- Characterized by paroxysmal and recurrent attacks of substernal/ precordial chest discomfort (knife-like, choking, squeezing)

Stable Angina

- Typical angina pectoris
- Most common form
- Caused by chronic fixed stenosing obstruction
- Heart vulnerable to ischemia on increase in demand i.e physical activity or emotional excitement
- Attacks of pain following physical exertion
- Usually relieved on rest/ nitroglycerin (a strong vasodilator)
- ECG- depression of ST segment
- No elevation in enzymes



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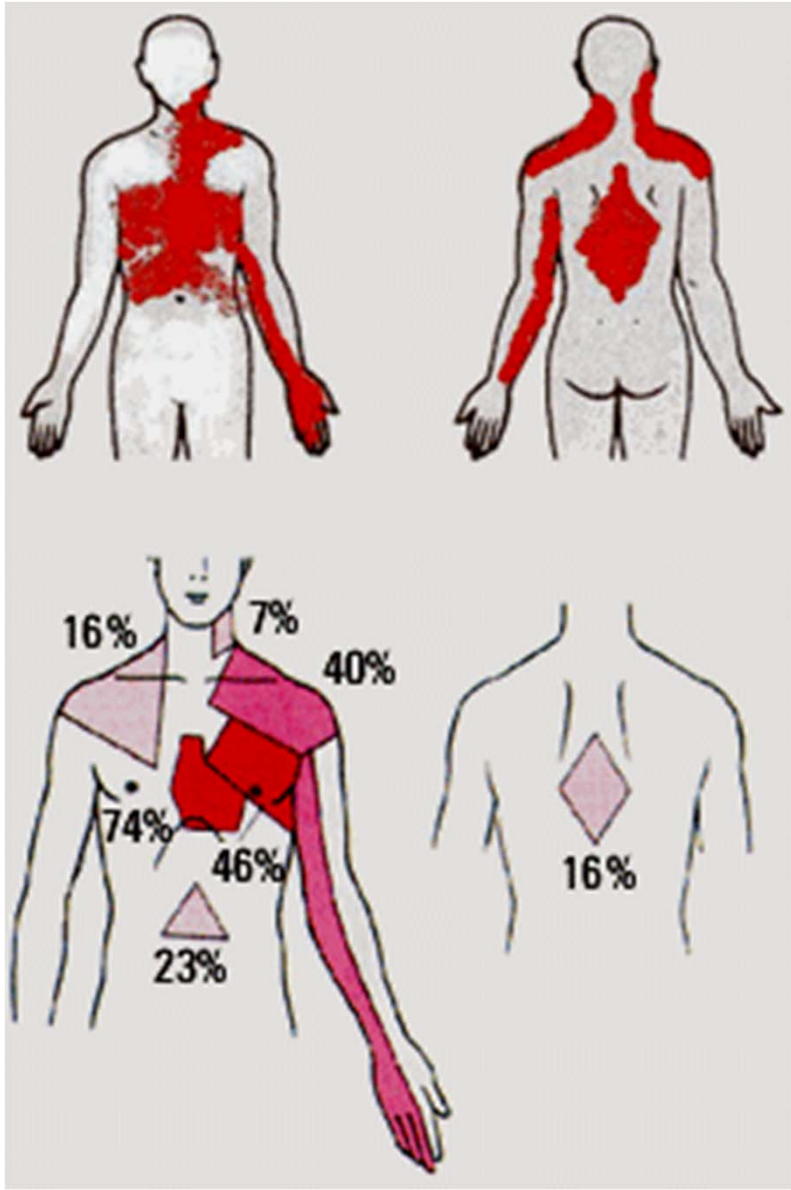
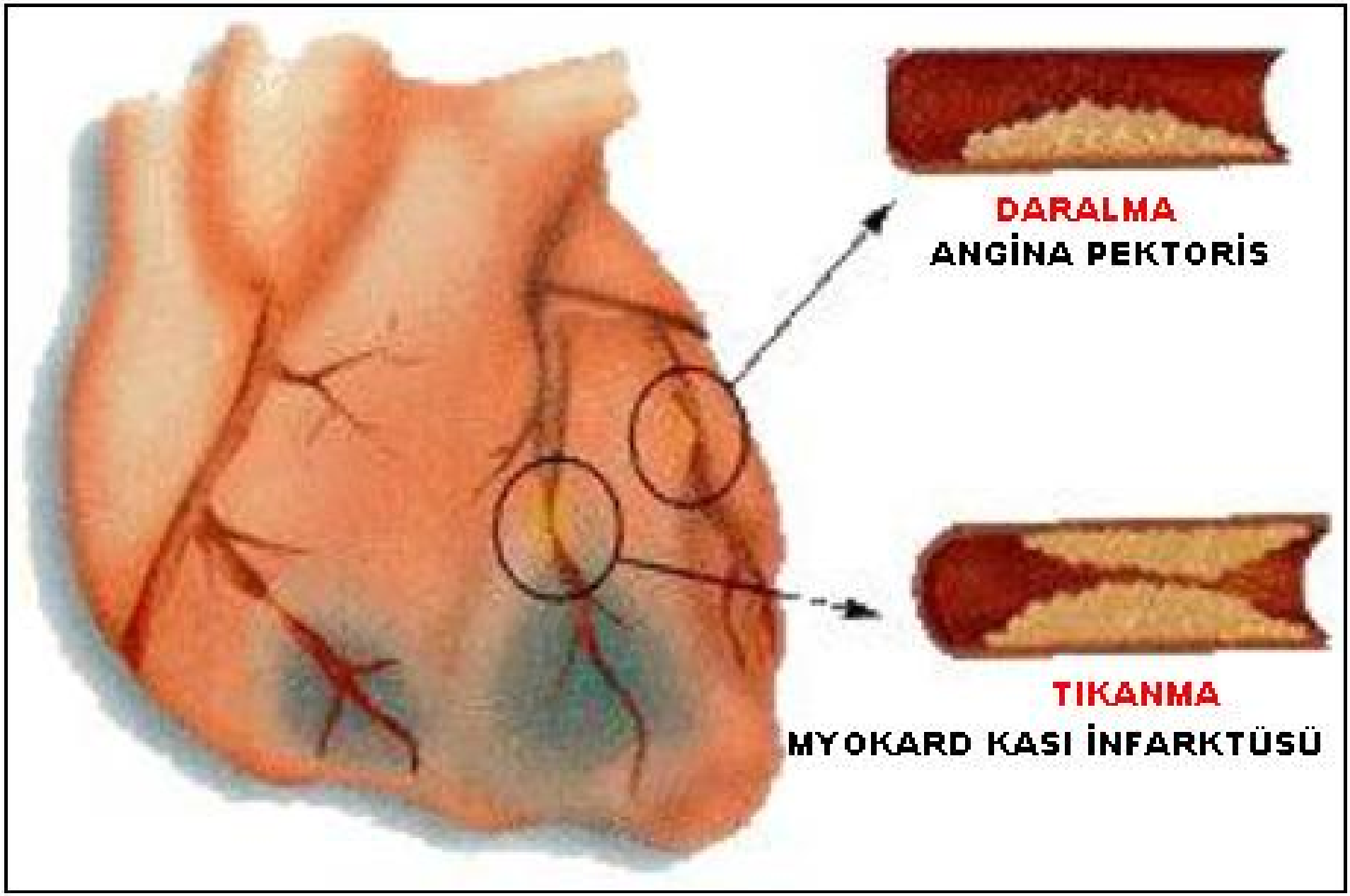
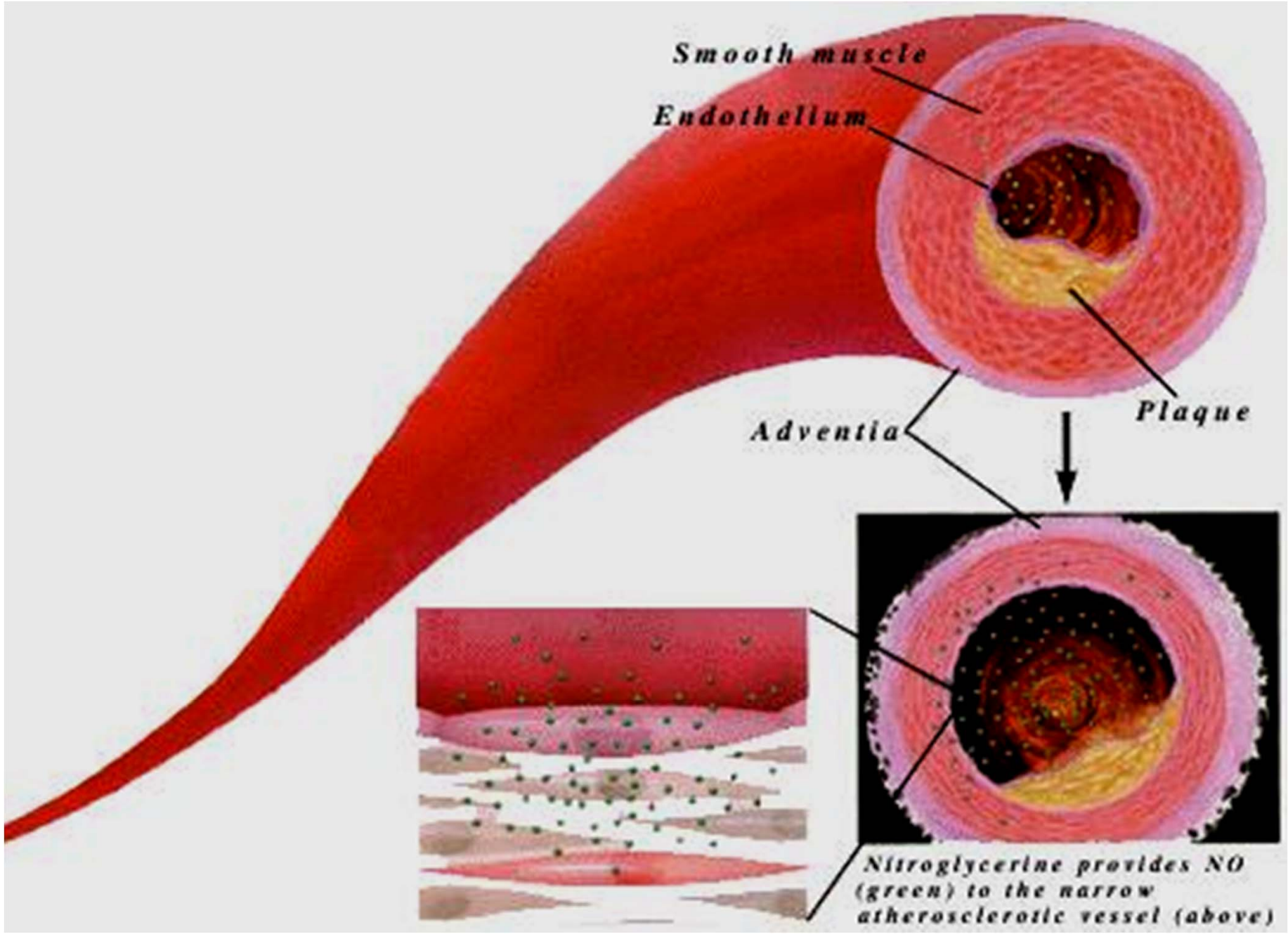



DARALMA
ANGİNA PEKTORİS
TIKANMA
MYOKARD KASI İNFARKTÜSÜ


Smooth muscle
Endothelium
Plaque
Adventia
Nitroglycerine provides NO (green) to the narrow atherosclerotic vessel (above)

Prinzmetal Variant Angina

- Occurs at rest
- Due to sudden vasospasm of coronary A
- Uncommon pattern
- Unrelated to physical activity, heart rate or B.P
- ST elevation on ECG indicative of transmural ischemia
- Responds to vasodilators and CCB

Unstable / Crescendo Angina

- Pain occurs with increasing frequency, is precipitated with lesser effort, often occurs at rest and is of prolonged duration
- Induced by plaque disruption with superadded changes i.e vasospasm, thrombosis, embolism
- Prodrome for subsequent M.I, thus called Preinfarction angina