

Ischemic heart disease

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Definition

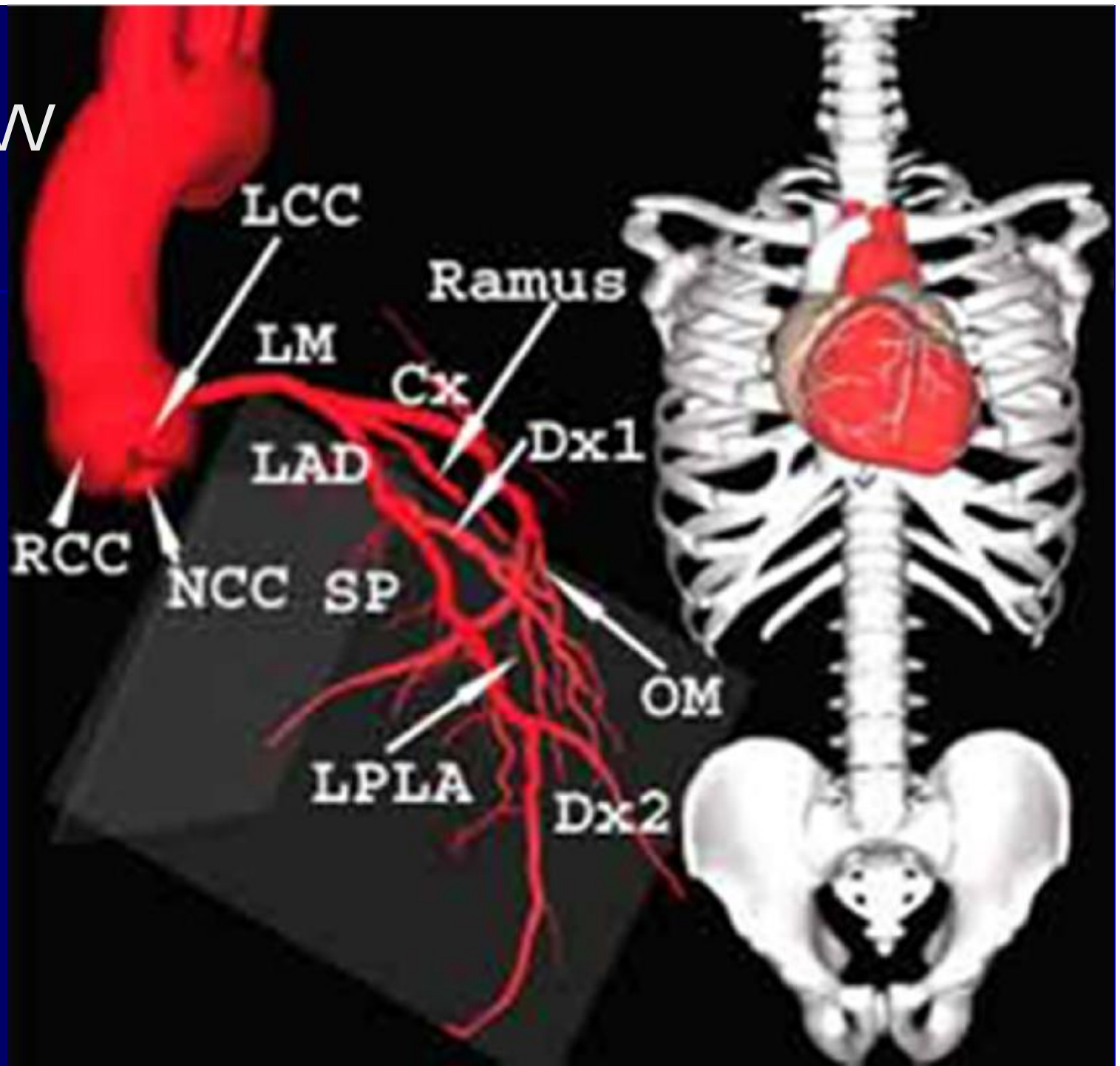
- n Ischemia : inadequate tissue perfusion
- n This may result from imbalance between oxygen demand and oxygen supply to tissue

Coronary blood flow

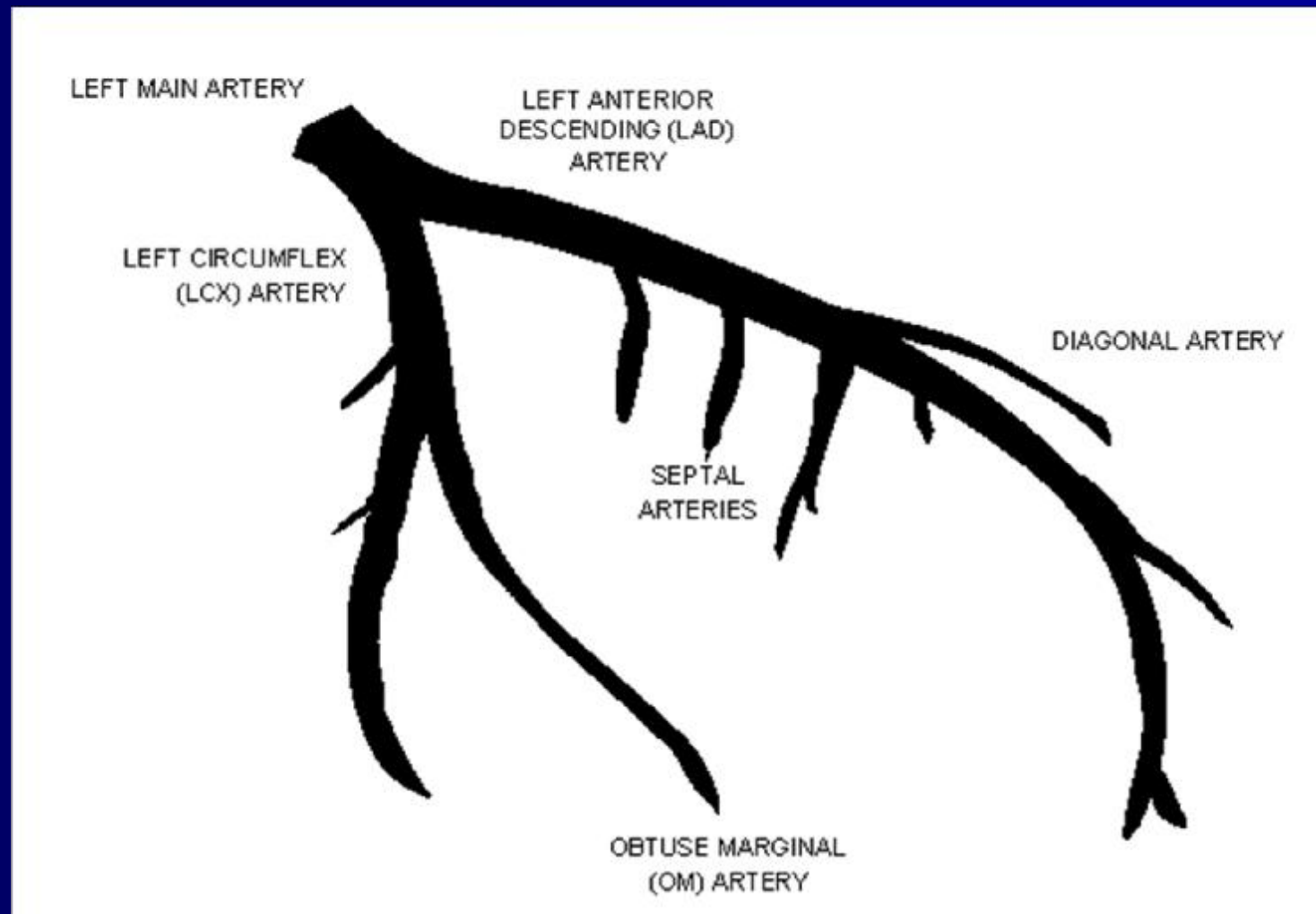
- n 0.8ml/g/min at rest this increase to 5-6time in exercise
- n Narrowing of CA lead to diminshed blood flow
- n In absence of collateral narrowing >75% oclusions of cross sectional area(>50% of lumen) lead to stable angina

Anatomy of coronary arteries

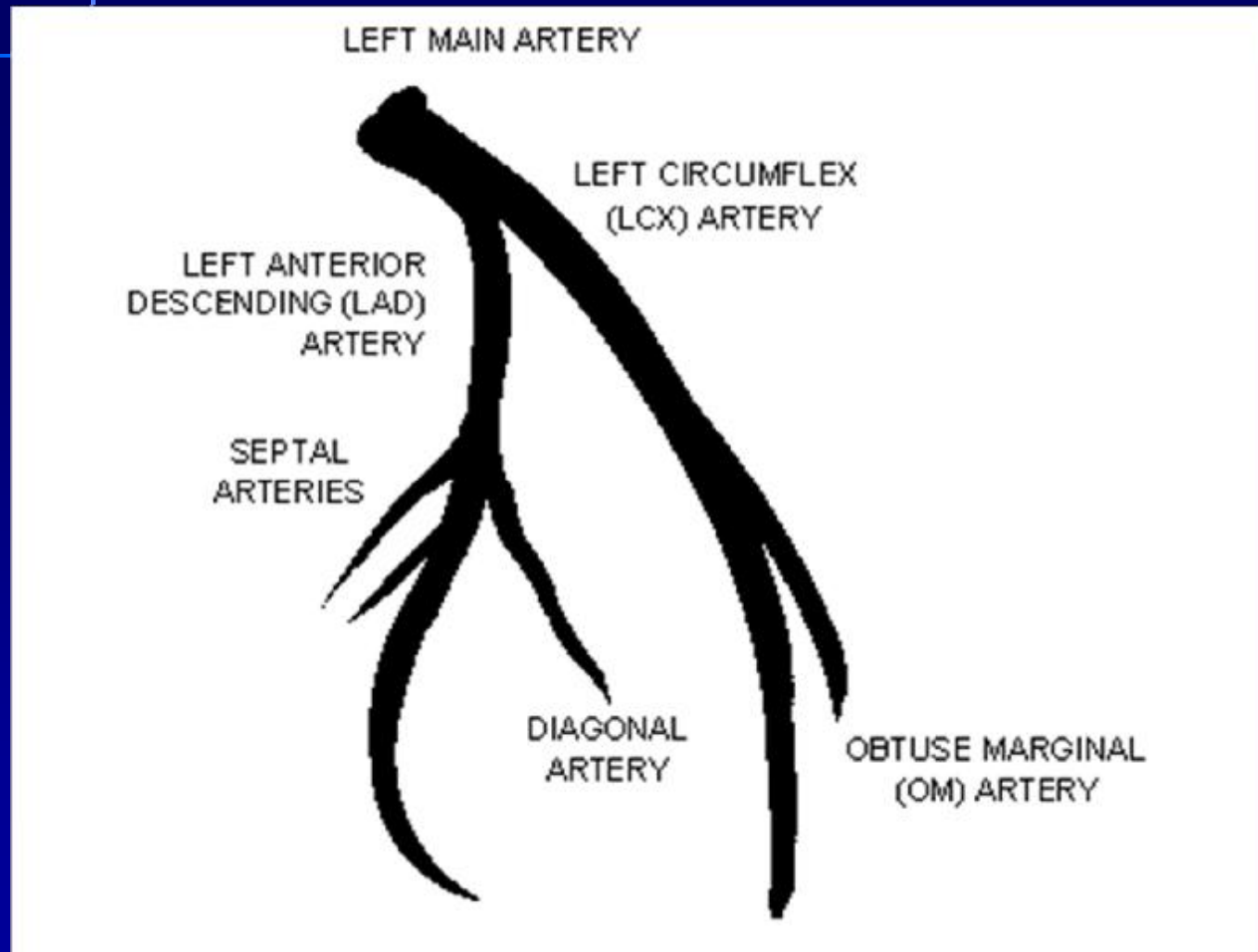
AP view



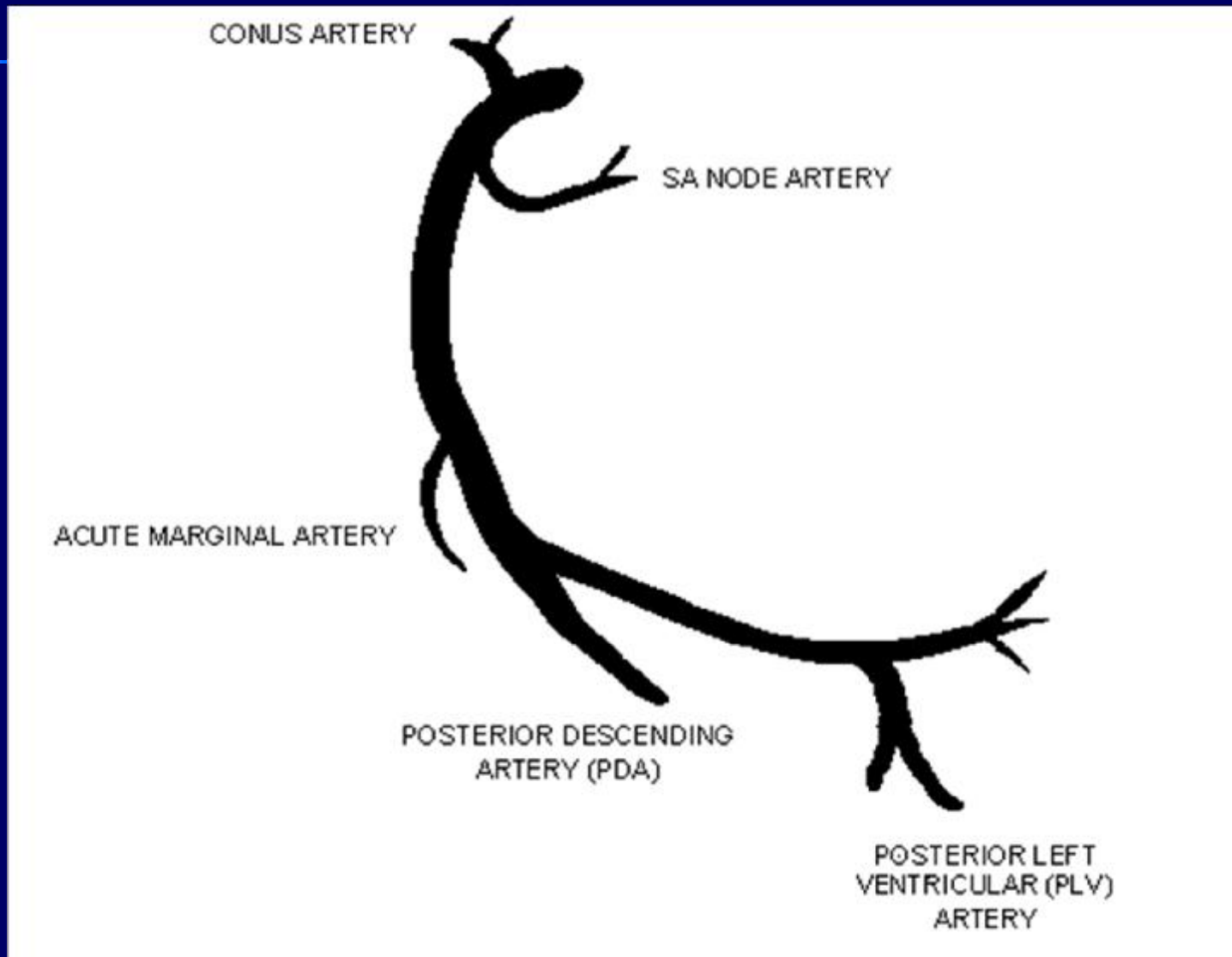
LCA RAO



LCA cranial LAO



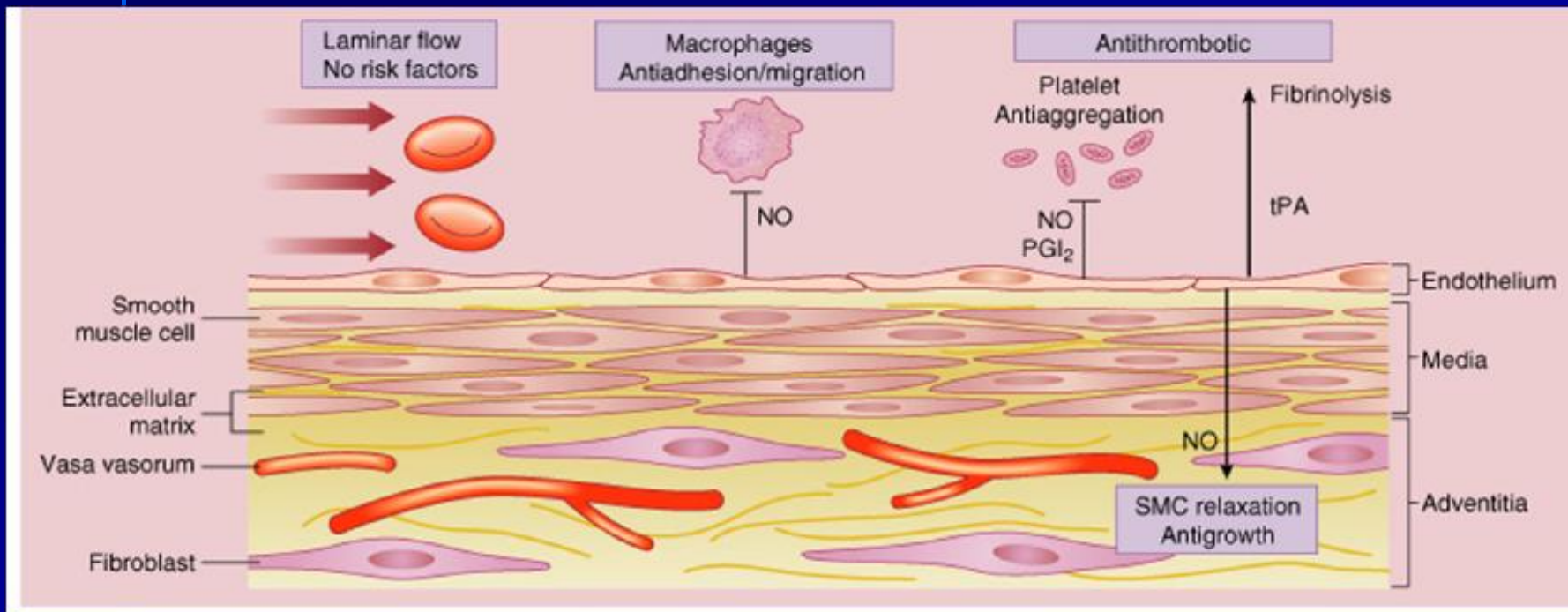
RCA

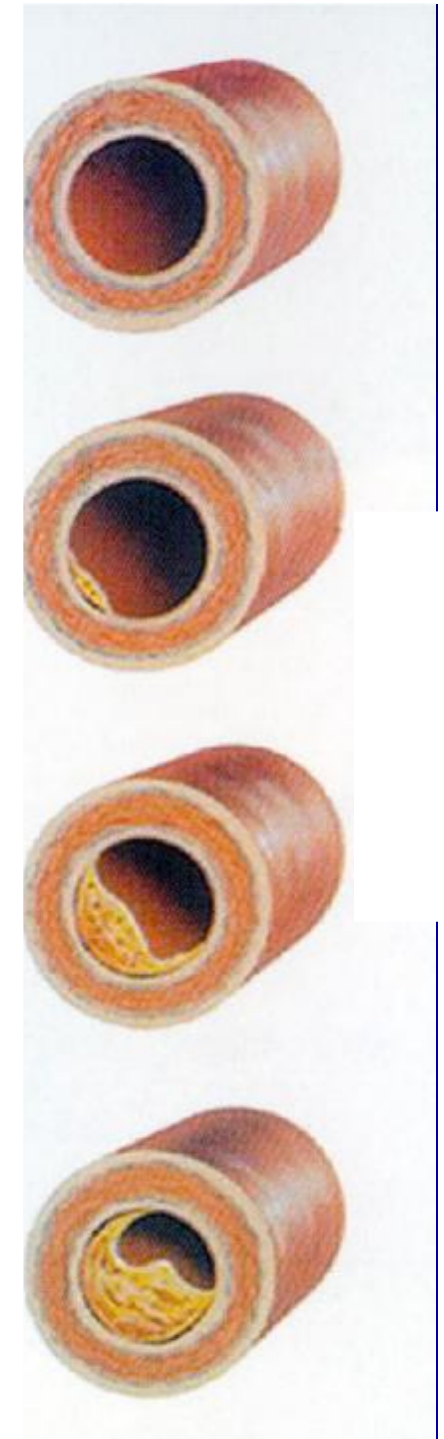
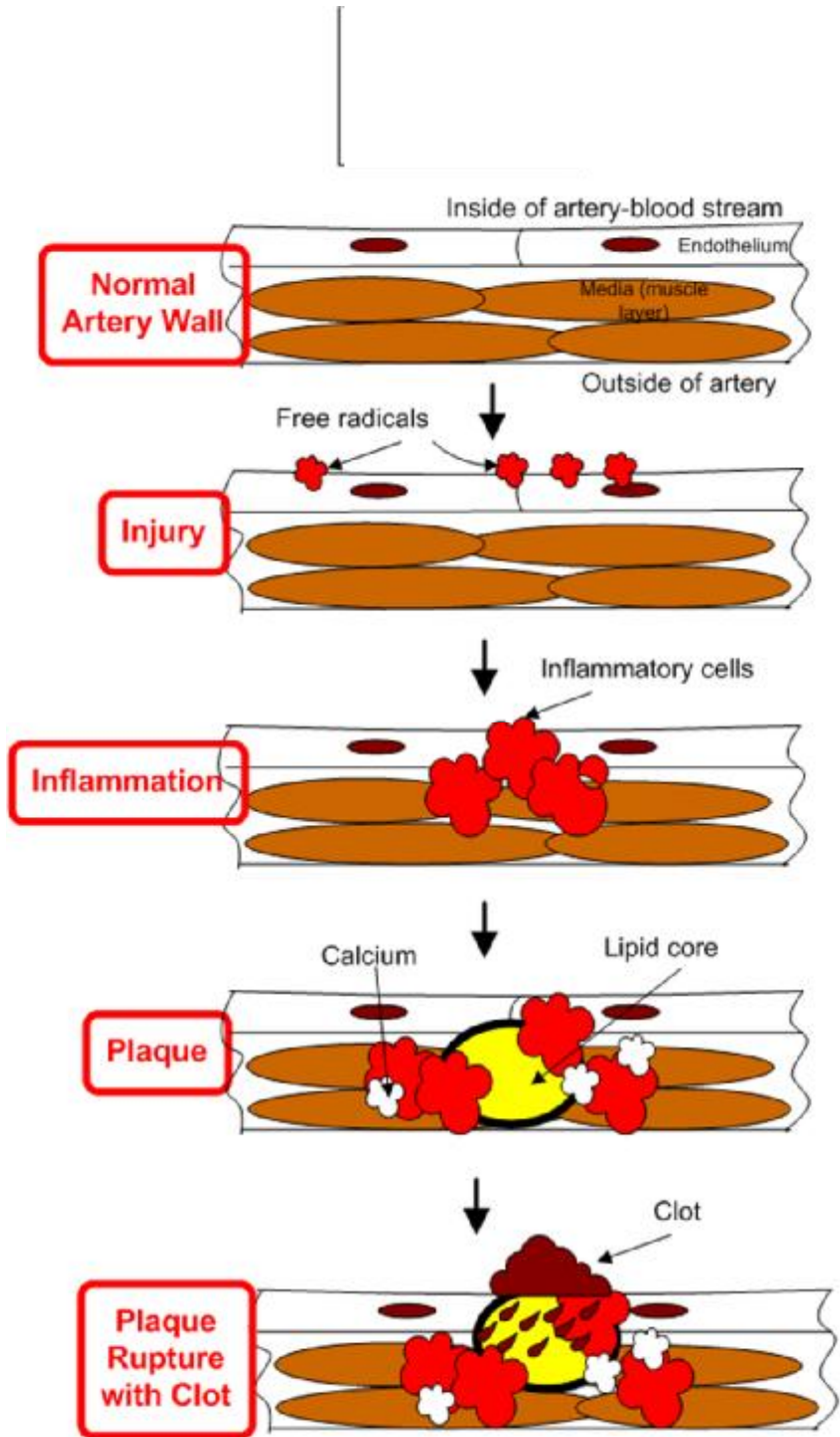


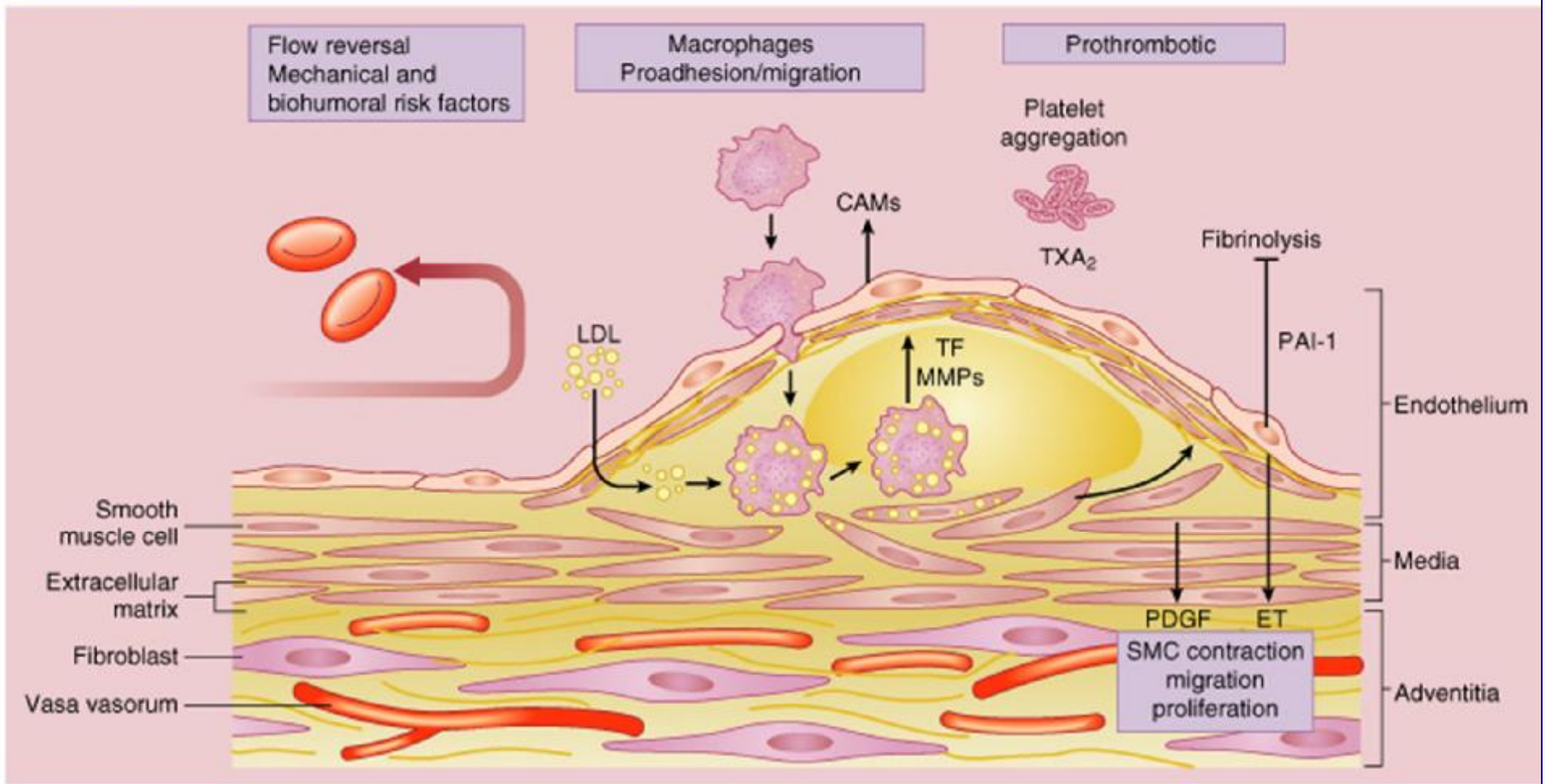
Etiology

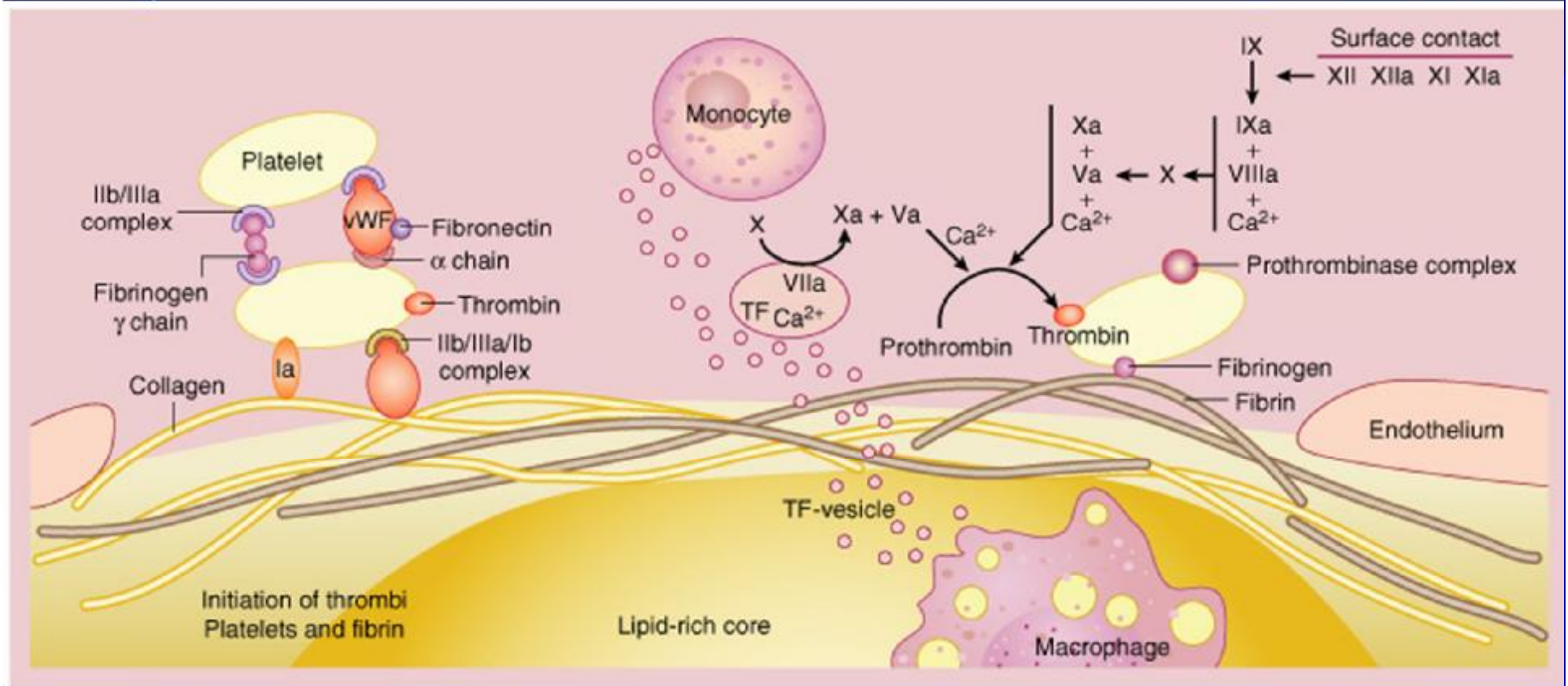
- n Atherosclerosis
- n Congenital anomalies of coronary artery
- n Vasculitis (Kawasaki syndrome, Takayasu arteritis)
- n Aortic valve disease, HOCM

Atherosclerosis









Risk factors

- n Smoking
- n Hypertension
- n Diabetes mellitus
- n Hyperlipidemia
- n Obesity
- n Physical inactivity
- n Family history

Presentation of pt with cardiac ischemia

- n Chest pain : Angina or MI
- n Arrhythmia
- n Heart failure
- n Sudden death
- n Silent
- n Rarely, confusional state, hypotension
disturbance consciousness ...etc

Angina

n Chest pain due to cardiac ischemia

n Type:

Stable

unstable

Angina variant

decubitus angina

Stable angina

- n Chest pain that occurs on exercise and is relieved by rest and NTG, lasting a few minutes
- n Usually retrosternal and may radiate



Unstable angina

- n Chest pain at rest
- n Chest with marked ECG changes
- n Crescendo Angina(Increase in severity and duration of stable angina)
- n Post MI angina
- n Any recent onst Angina with 2 weeks of onset

Myocardial infarction

n Infarction : Ischemic necrosis of tissue

Chest pain at rest ,

last 1-2 hours ,

not relive by NTG

Usually associated with homodynamic changes

(Pale, sweaty ,hypotension ...etc

Myocardial infarction

Types:

- n Transmural ,subendocardial
- n Q wave type ,non Q wave type
- n ST segment elevation ,Non ST segment elevation

Acute coronary syndrome (ACS)

Acute coronary syndrome (ACS) describes a spectrum of clinical conditions ranging from ST segment elevation myocardial infarction (MI) to non-ST segment elevation MI and unstable angina
(ACS without enzyme or marker release)

The syndrome is the consequence of disruption of a vulnerable coronary artery plaque, complicated by intraluminal thrombosis, embolisation, and varying degrees of obstruction to perfusion

The severity of coronary arterial obstruction and the volume of affected myocardium determine the characteristics of clinical presentation.

Patients with complete occlusion may manifest ST segment elevation infarction if the lesion occludes an artery supplying a substantial volume of myocardium,

but the same occlusion in the presence of extensive collateralisation may manifest as infarction without ST segment elevation (non-ST elevation ACS).

Similarly, incomplete occlusion at the site of a disrupted arterial plaque may produce ischemia or microinfarction, depending on the volume of myocardium affected and the extent of distal embolisation.

Sensitive and specific markers of myocyte injury (troponins)

allow the detection of more subtle volumes of infarction than possible using conventional cardiac enzymes.

Investigations of patient with cardiac ischemia

Next lecture

