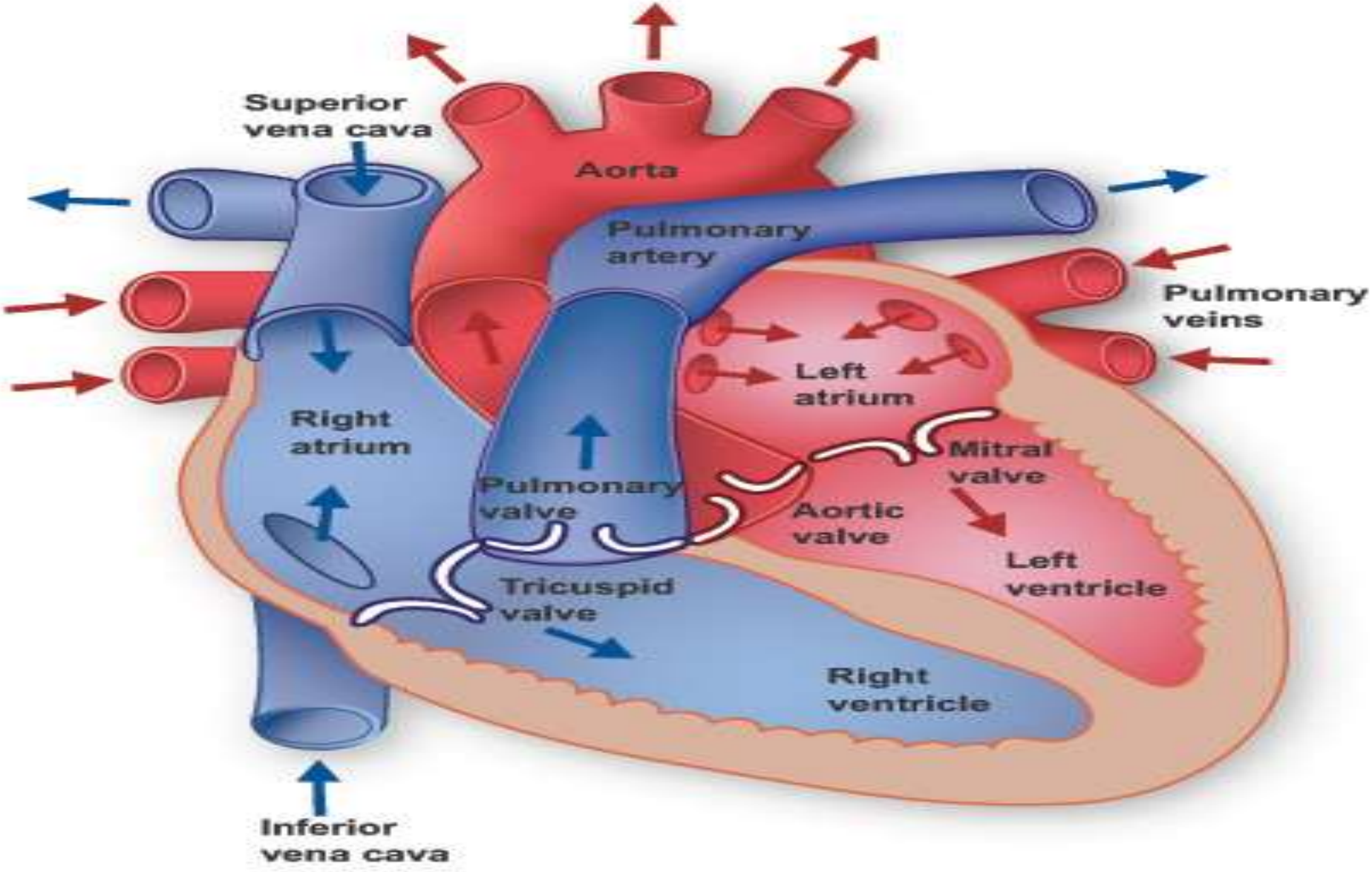
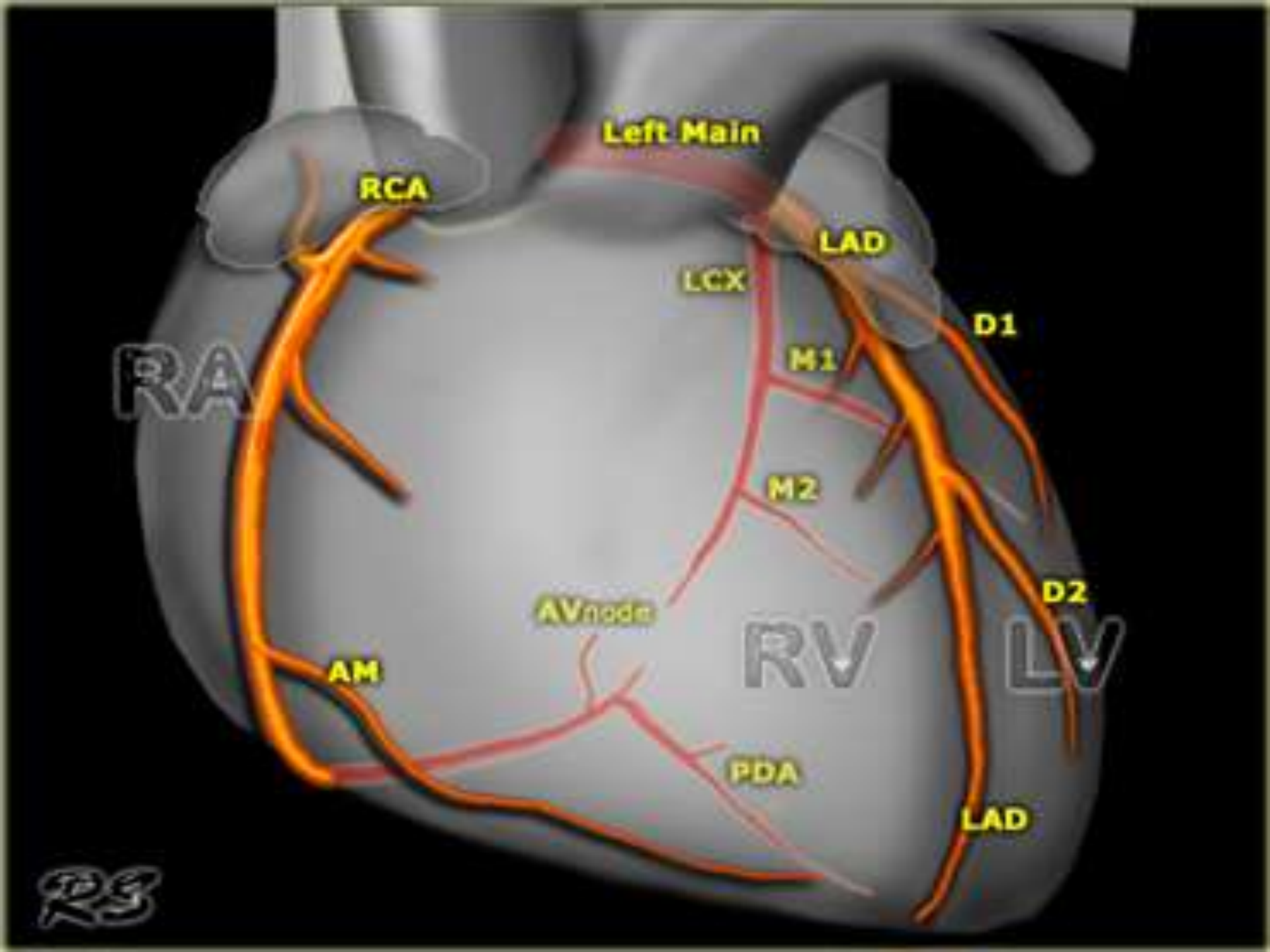
Coronary Vascular Disorder

Anatomy of the Heart



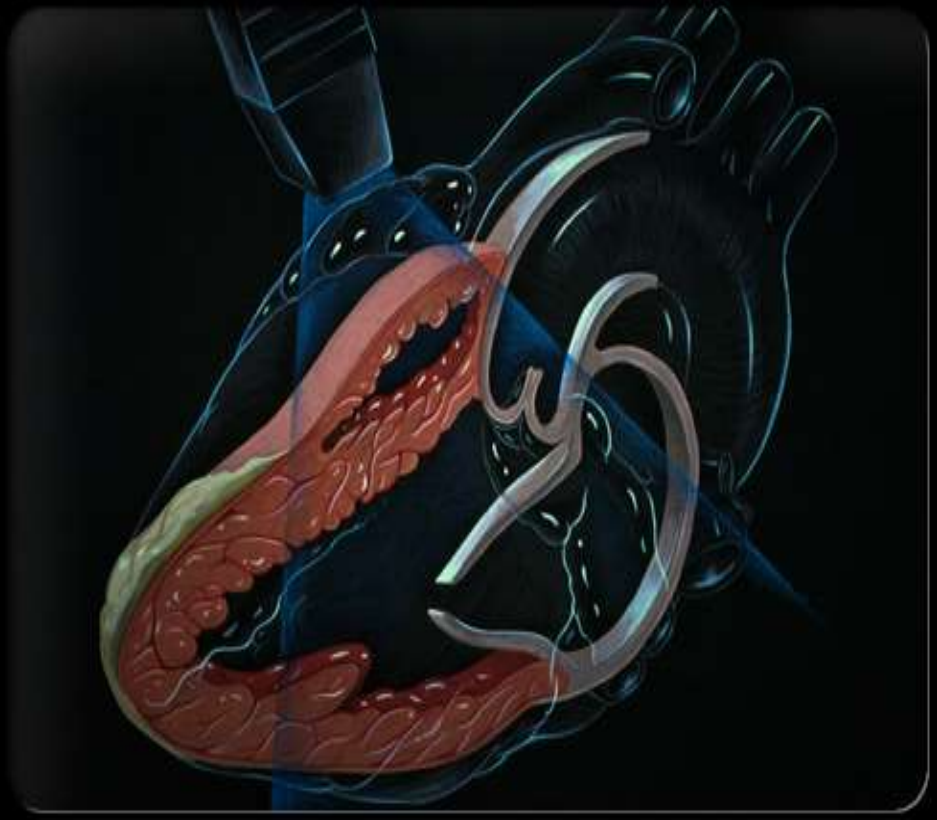

Left Main
RCA
LAD
LCX
D1
M1
RA
M2
D2
AVnode
RV
LV
AM
PDA
LAD

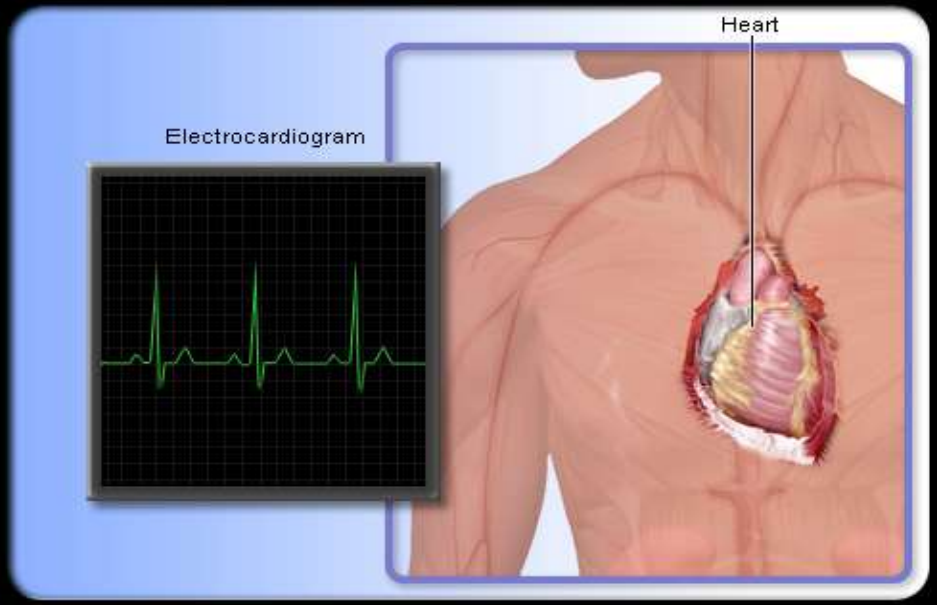
- Conduction system of heart:
  - SA node (60- 100)
  - Av node (40-60)
  - Bundle of his (30-40)
  - Purkinje fiber
- Heart Muscle
  - Endocardium
  - Myocardium
  - Pericardium

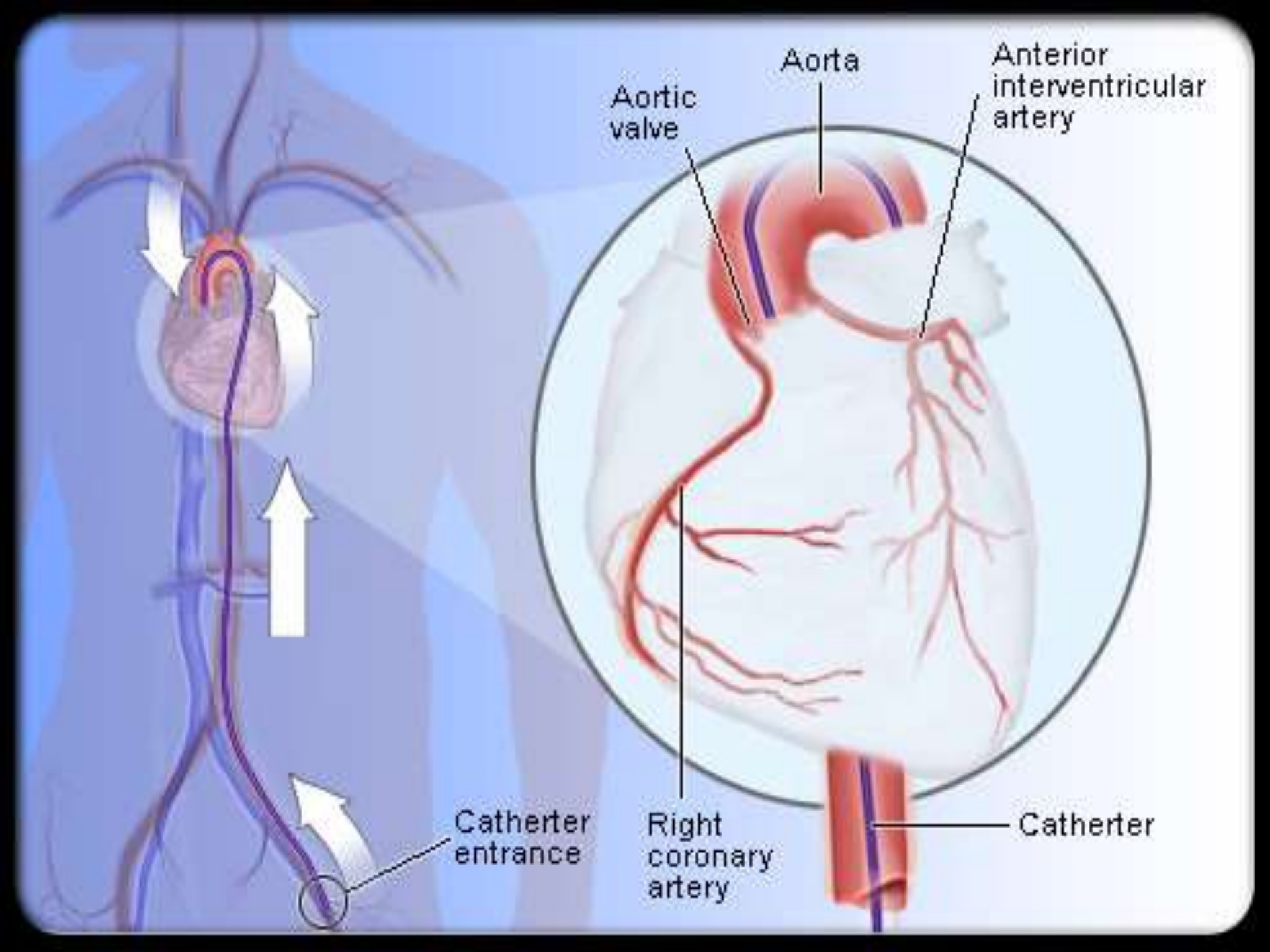
30 ml of fluid present in the pericardial space to decrease friction during systole

Assessment of the CV

- Health history
- Clinical manifestations
- Physical examination
- Review of diagnostic examination and laboratory test (Cardiac biomarkers, lipid profile , ECG, CXR, Treadmill exercise, CT scan, MRI, Echo, catheterization, Angiography)

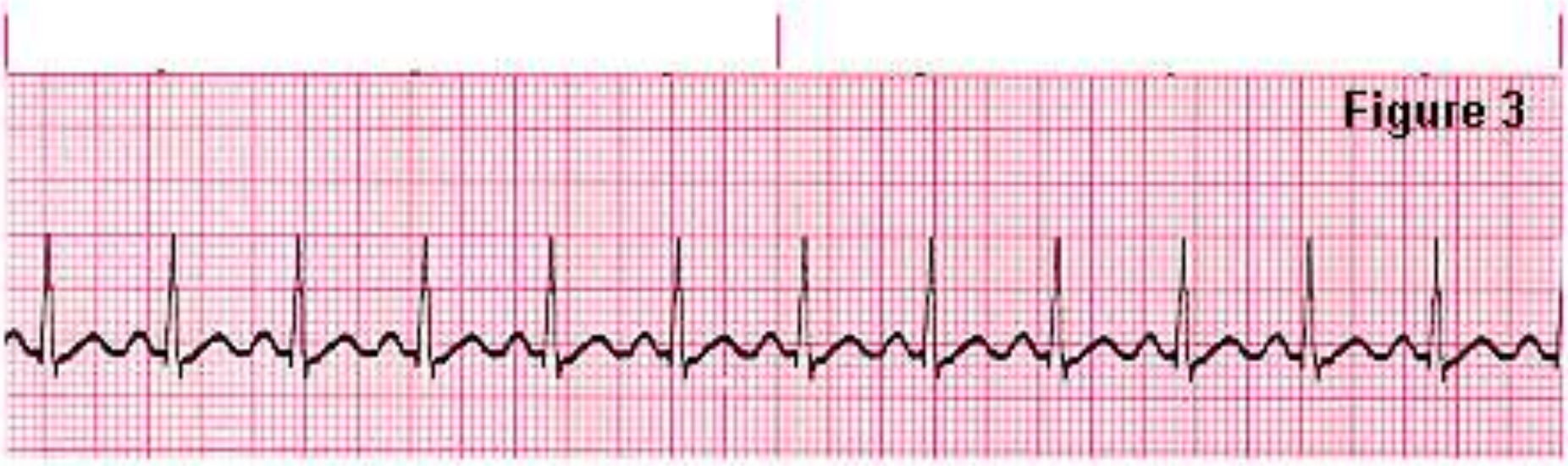


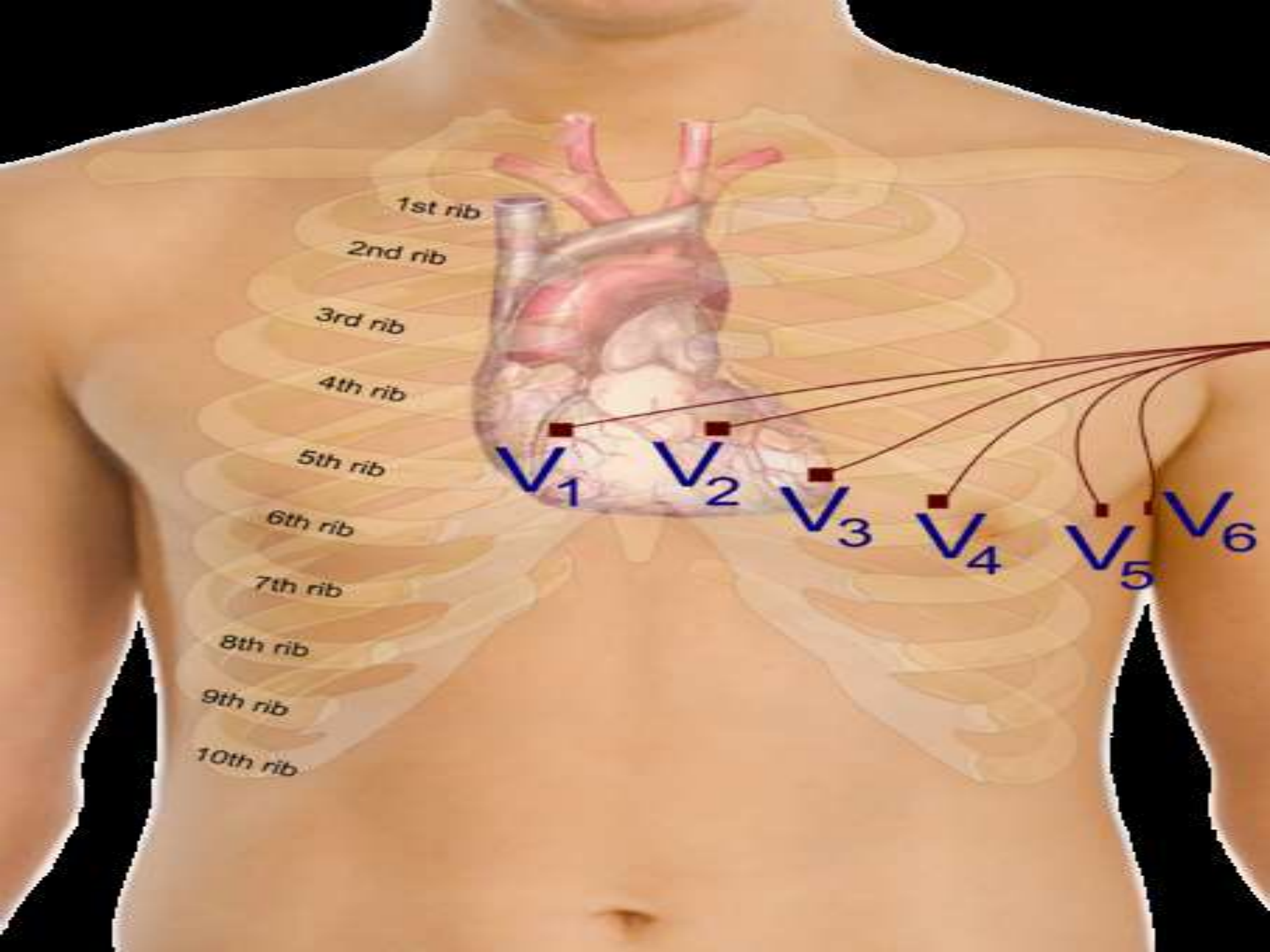

Heart
Electrocardiogram


Aorta
Aortic valve
Anterior interventricular artery
Catherter entrance
Right coronary artery
Catherter

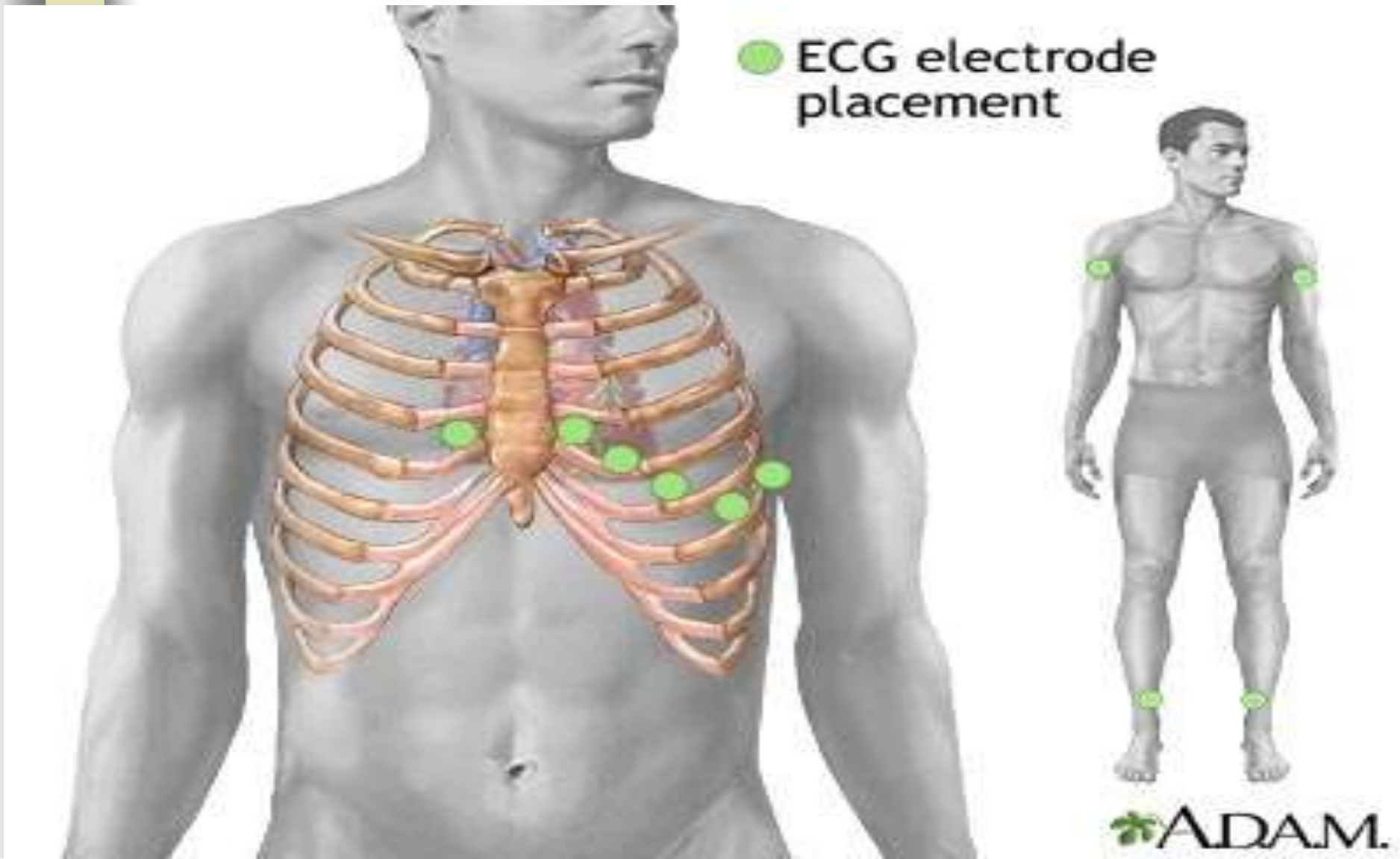
Electrocardiogram (ECG)

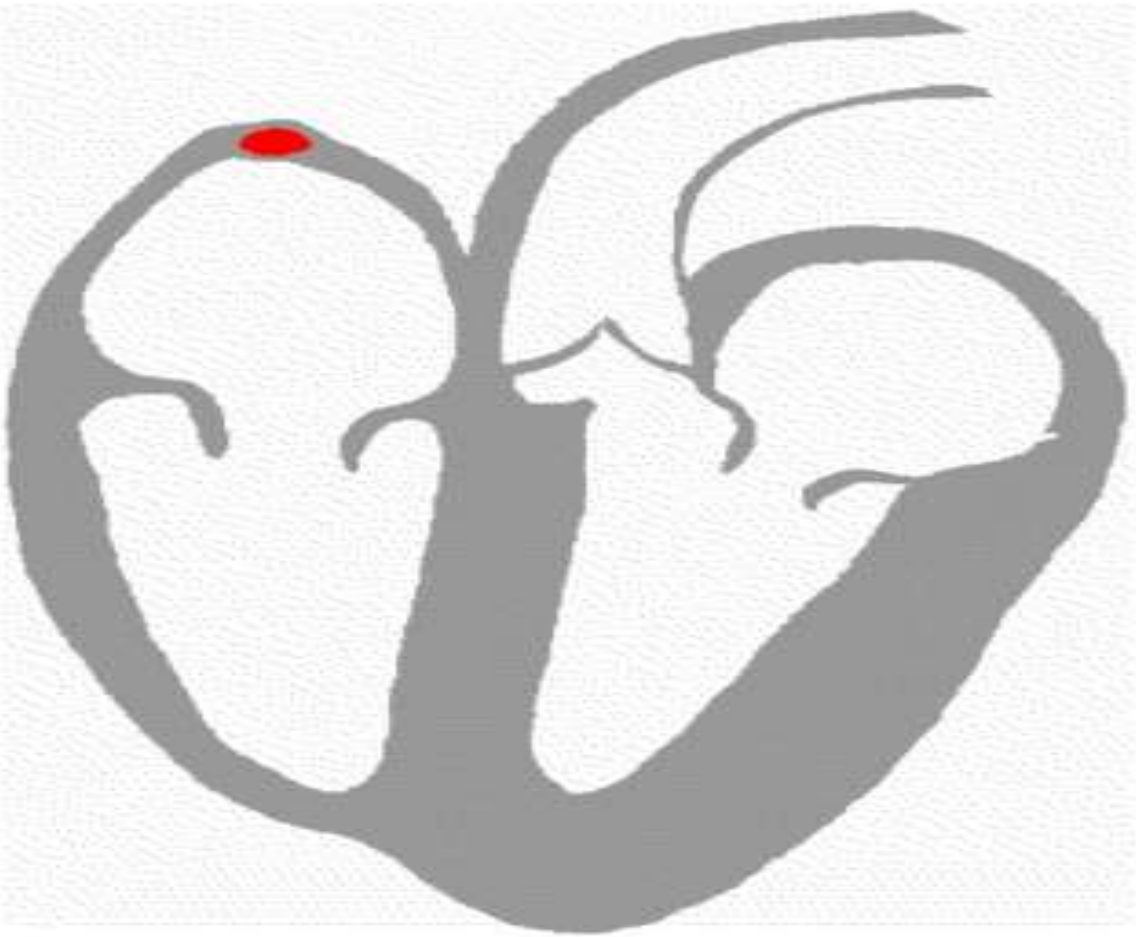
- Is a graphic (wave form) with horizontal and vertical lines for recording of electrical activity of the heart: 12 lead ECG: Lead I, II, III, AVR, AVL, AVF, V1-V6

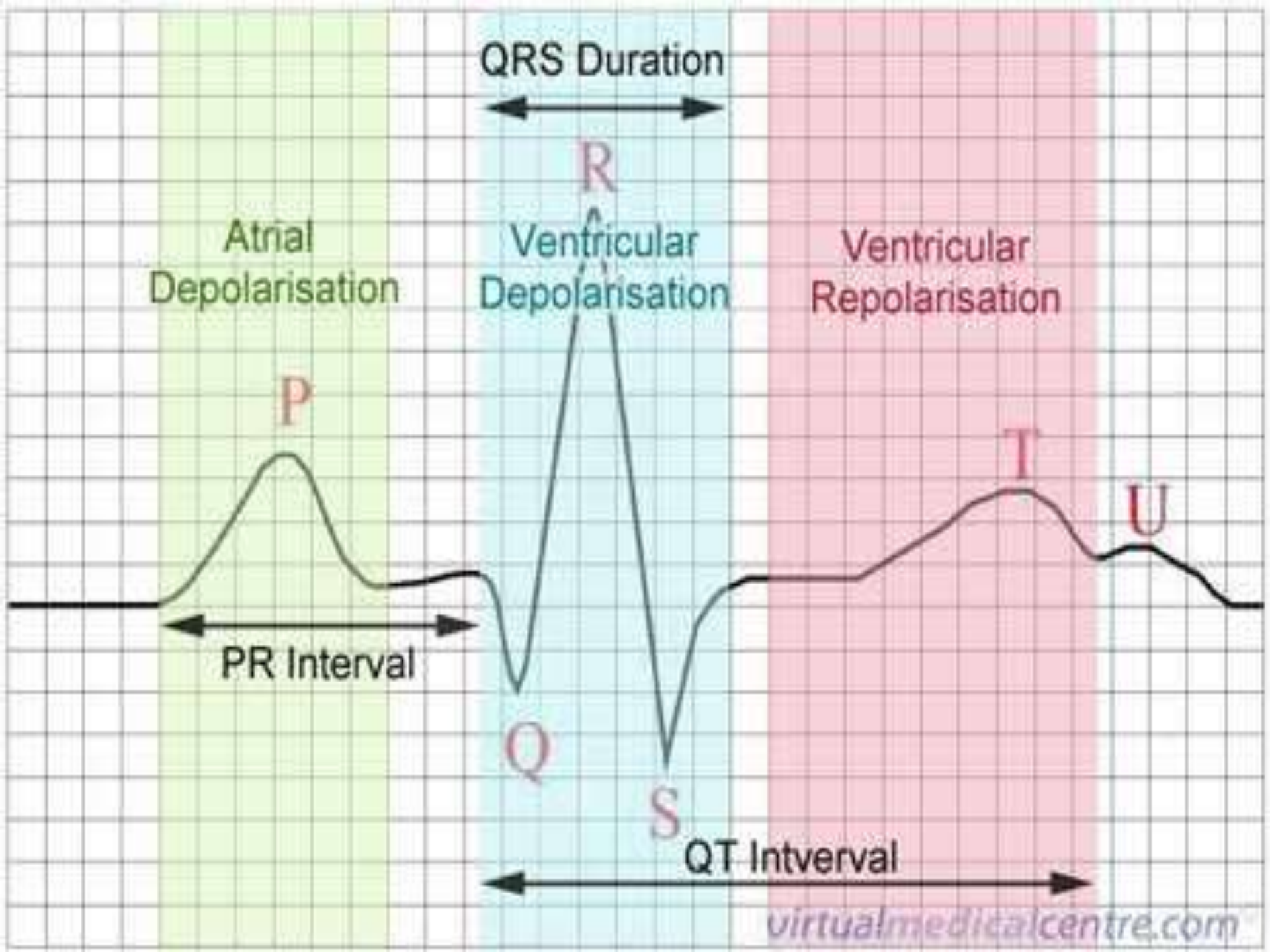



1st rib
2nd rib
3rd rib
4th rib
5th rib
6th rib
7th rib
8th rib
9th rib
10th rib
V1
V2
V3
V4
V5
V6

Places of ECG electrodes






QRS Duration
R
Atrial
Depolarisation
Ventricular
Depolarisation
Ventricular
Repolarisation
P
T
U
PR Interval
Q
S
QT Intverval
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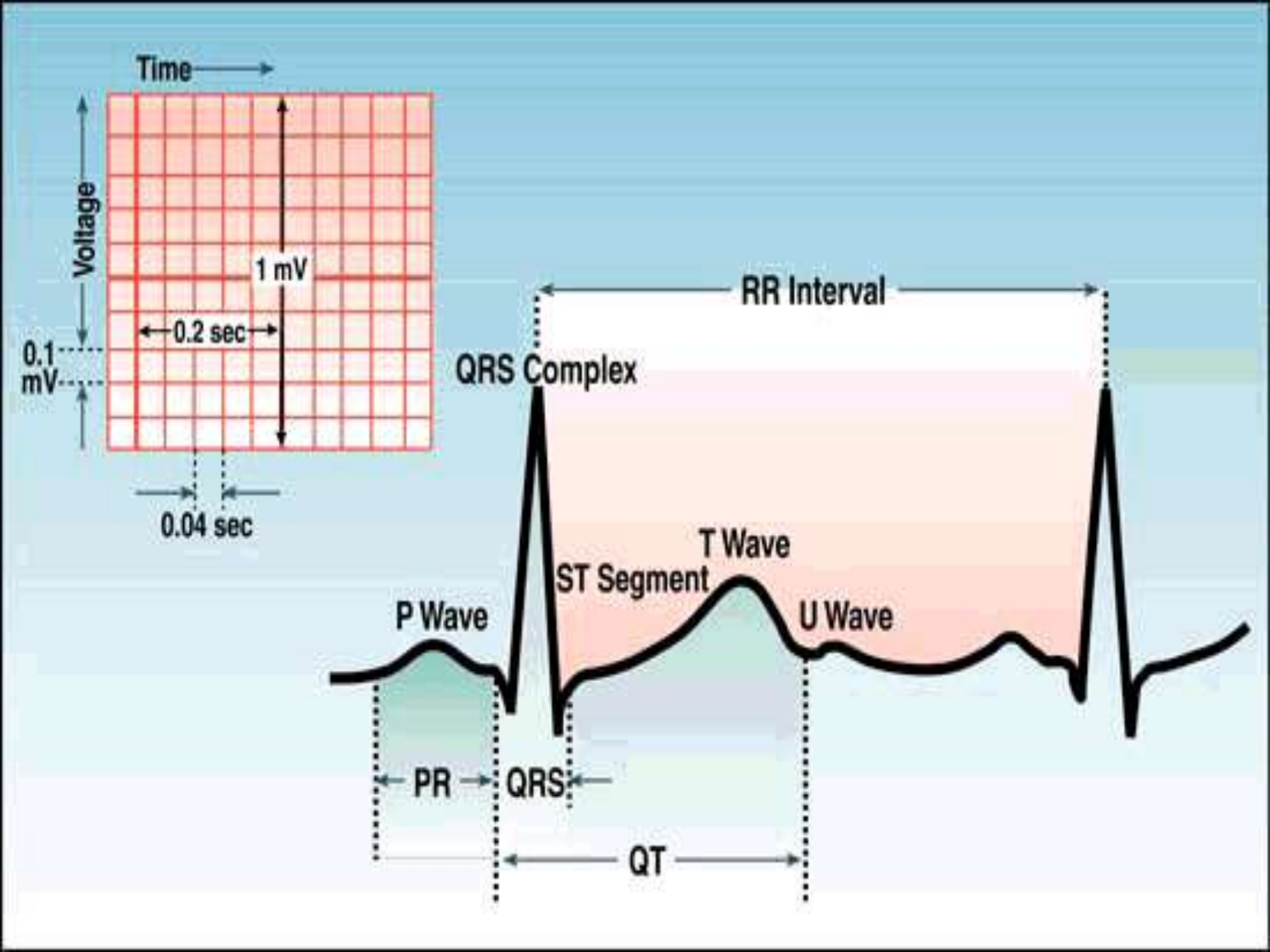
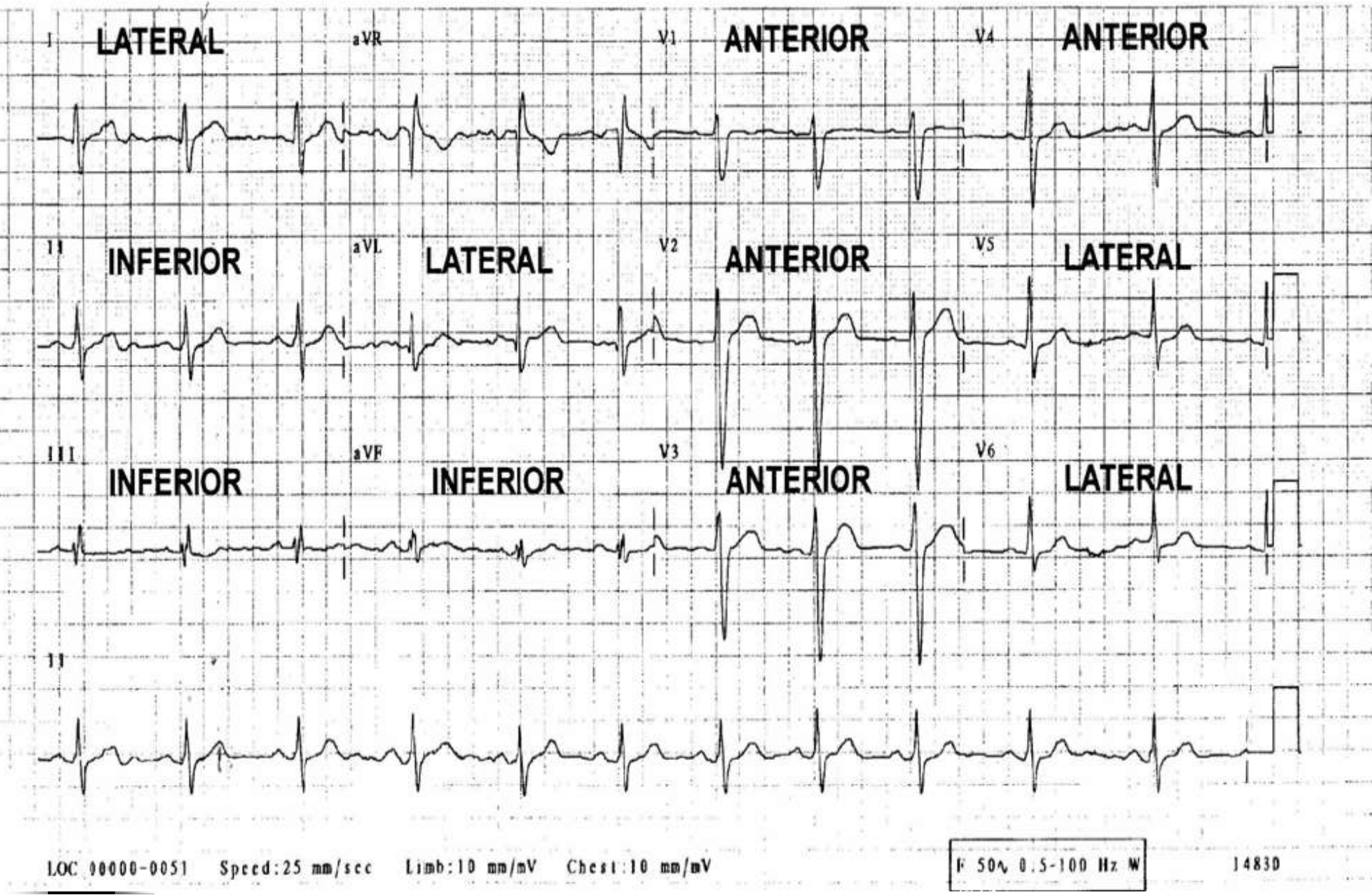

Time
Voltage
1 mV
0.2 sec
0.1 mV
0.04 sec
RR Interval
QRS Complex
T Wave
ST Segment
P Wave
U Wave
PR
QRS
QT


I
LATERAL
aVR
V1
ANTERIOR
V4
ANTERIOR
II
INFERIOR
aVL
LATERAL
V2
ANTERIOR
V5
LATERAL
III
INFERIOR
aVF
INFERIOR
V3
ANTERIOR
V6
LATERAL
II
LOC 00000-0051 Speed:25 mm/sec Limb:10 mm/mV Chest:10 mm/mV
F 50∿ 0.5-100 Hz W
14830

Waves

- P wave: represent electrical impulses starting in SA node spread through atria, represent atrial depolarization, 0.11 sec in duration
- QRS complex: ventricular depolarization, less than 0.12 sec in duration
- T wave: ventricular repolarization, same direction with QRS complex
- U wave: represent repolarization of purkinje fiber, seen in pt with ↓K, HTN and heart disease, smaller than the P wave

Intervals

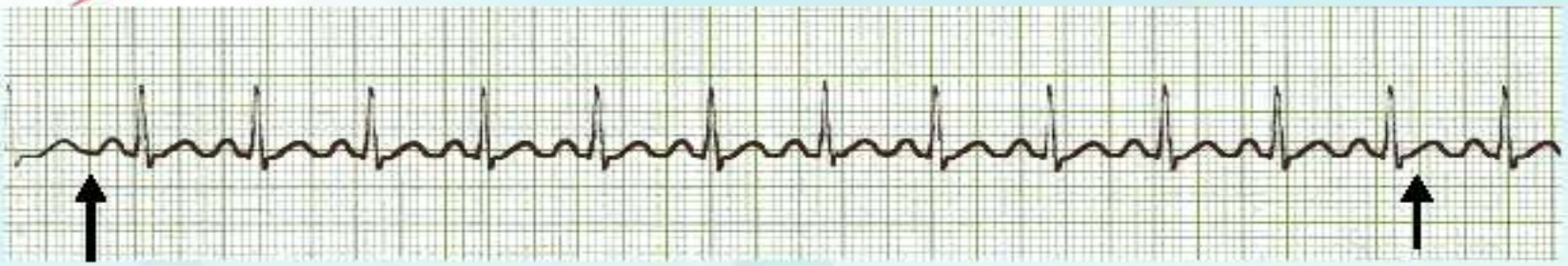
- PR interval: time interval from onset of atrial depolarization (P wave) to onset of ventricular depolarization (QRS complex). The normal range of PR interval is 0.12 to 0.20 seconds duration
- ST-segment: last from the end of the QRS to beginning of the T wave, important in determine cardiac ischemia
- QT: total time of ventricular depolarization and repolarization, measured from beginning of QRS complex to the end of T wave, 0.32- 0.4 sec, varies with age, gender and HR

- TP interval: from the end of T wave to the beginning of the next P wave (isoelectric period)
- RR interval: duration of ventricular cardiac cycle (an indicator of ventricular rate and rhythm), from QRS to the next QRS
- PP interval: duration of atrial cycle (an indicator of atrial rate and rhythm), From the P wave to the next P wave

Calculation of HR from ECG strip

Calculating the Heart Rate

NHS
Essex Cardiac Network



1) Count the number large squares between two consecutive 'R' waves
then divide the result by 300
i.e. 300/4 = 75 bpm

2) Count the number of 'R' waves in a 6 second strip (30 large squares)
then multiply the result by 10
i.e. 10 X 12 = 120 bpm
(this method is especially good for irregular heart rates)

3) With a rate ruler

Analyzing ECG Strip

- Normal Sinus Rhythm (NSR):

Rhythm: regular

Rate: 60 – 100

- Sinus Node Dysrhythmias:
  - Sinus Bradycardia:

Rhythm: regular

Rate: < 60

Seen in hypovolemia, hypoxia, acidosis, hypoglycemia, pulmonary embolism, T's

- Sinus Tachycardia:

Rhythm: regular

Rate:> 100 and <120

Occur in HF, fever, exercise, anxiety, pain

- Sinus Arrhythmia:

Rhythm: irregular

Rate: 60 – 100

Coronary Artery Disease (CAD)

- *Atherosclerosis:*

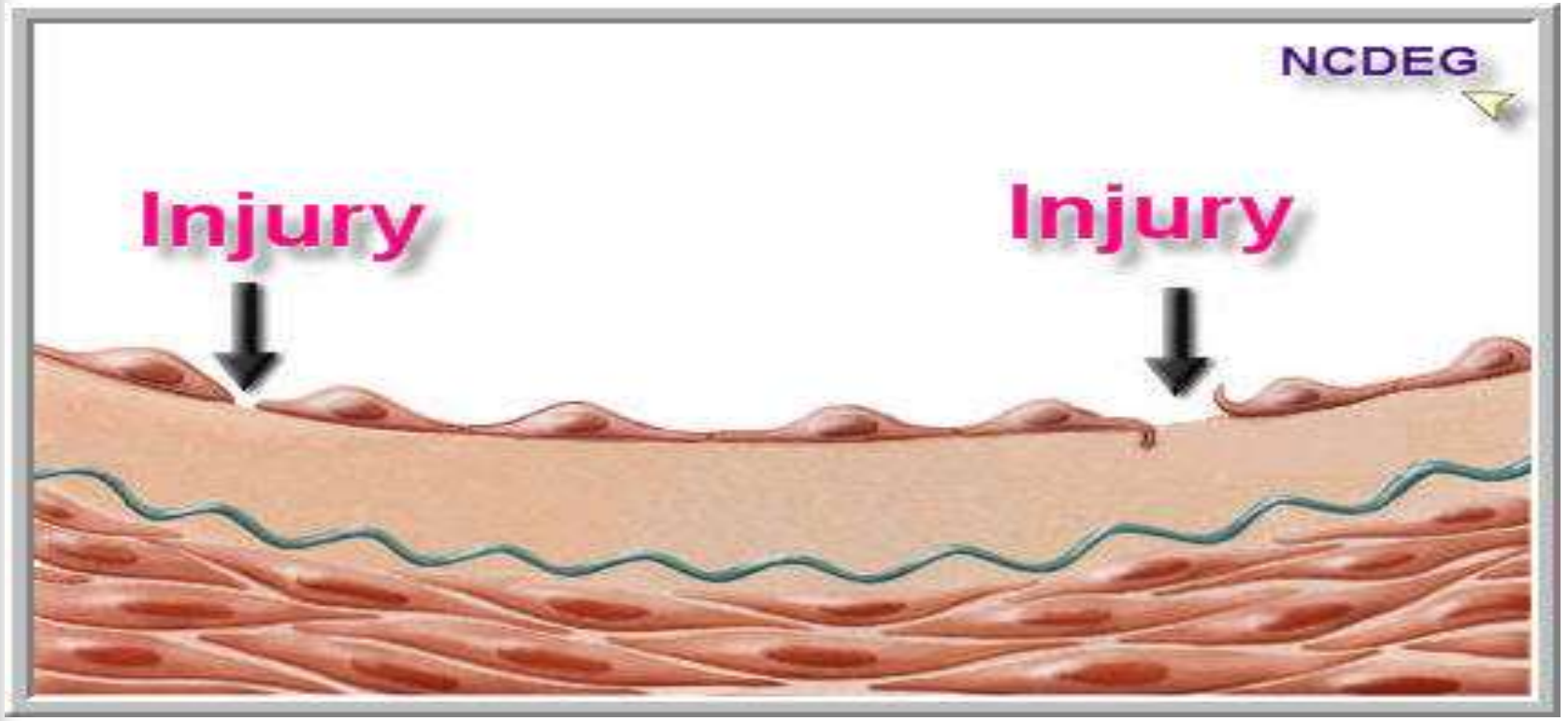
- Atherosclerosis is the abnormal accumulation of lipid deposits and fibrous tissue within arterial walls and lumen.
- blockages and narrowing of the coronary vessels reduce blood flow to the myocardium.
- Most common cause of CVD

Pathophysiology of atherosclerosis

- The formation of atherosclerosis start with

1- Injury to endothelium wall: which lead to alteration in structure and function of endothelial lining of arterial wall, this injury initiated by HTN, hyperglycemia, smoking and hyperlipidemia and other factors

Pathophysiology

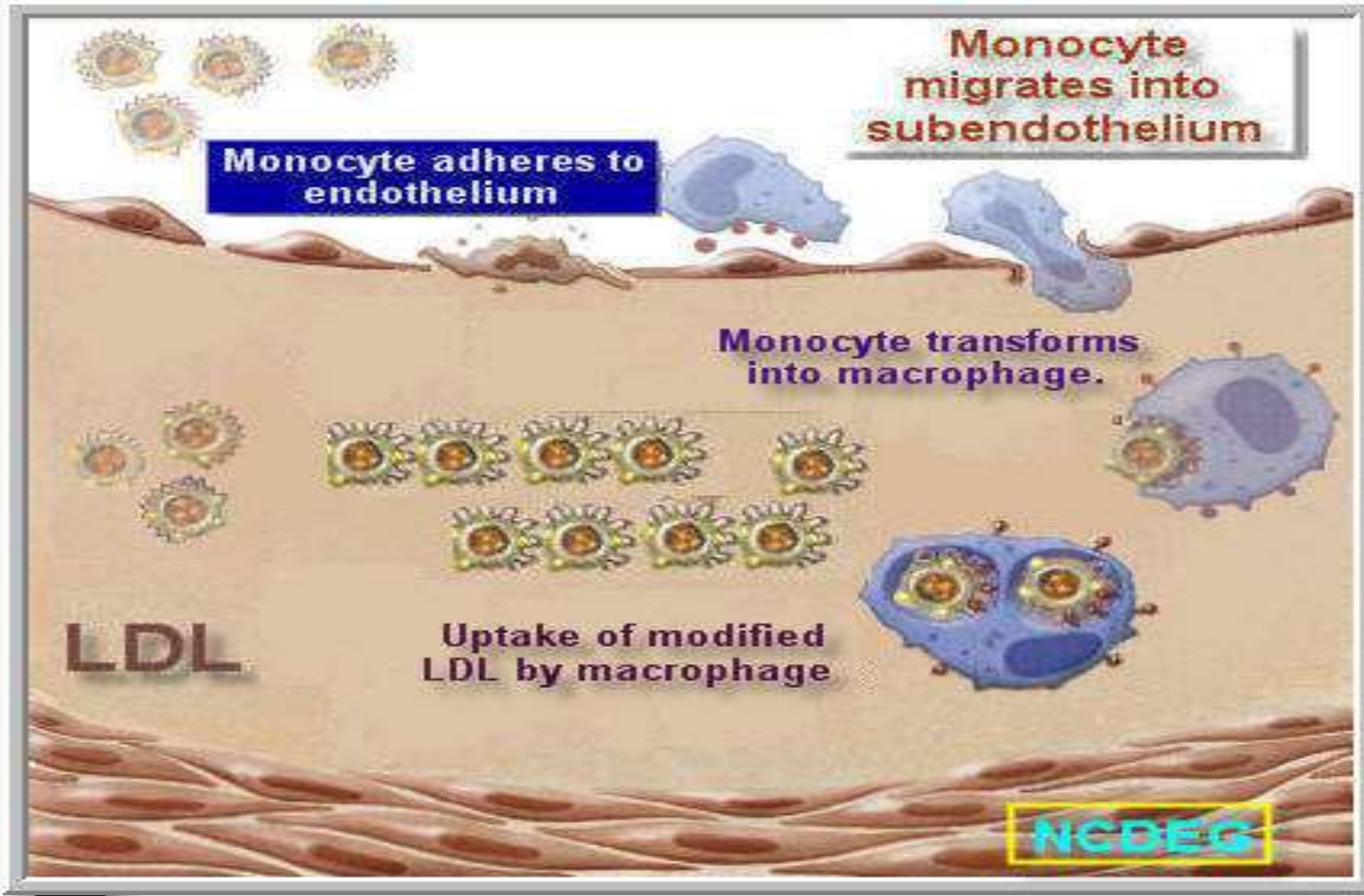


The initial injury alters the structure and function of the vascular endothelial.

- The second step is:

2- Formation of fatty streak: where circulating LDL filter through endothelial layers to intimal space, where modification occur as (denaturation, aggregation and oxidization), oxidized LDL is cytotoxic lead to additional endothelial injury, it attract circulating monocytes, also injured endothelial secreat attractant substances to monocytes which make it adhere to endothelium, then migrate subendothelial where it called macrophage, this engulf LDL and form foam cell

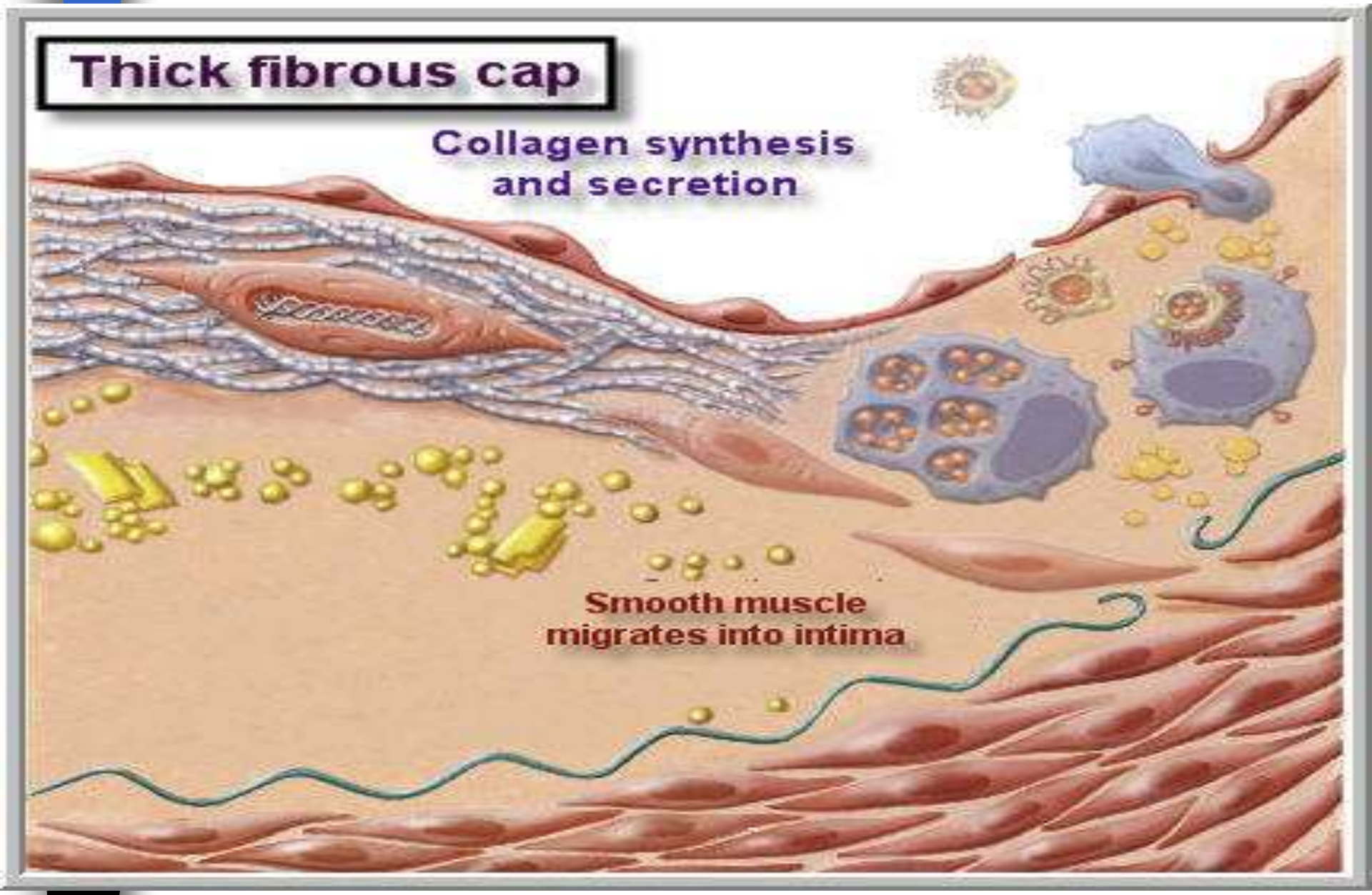
fatty streak:



- Third step (fibrous plaque formation):

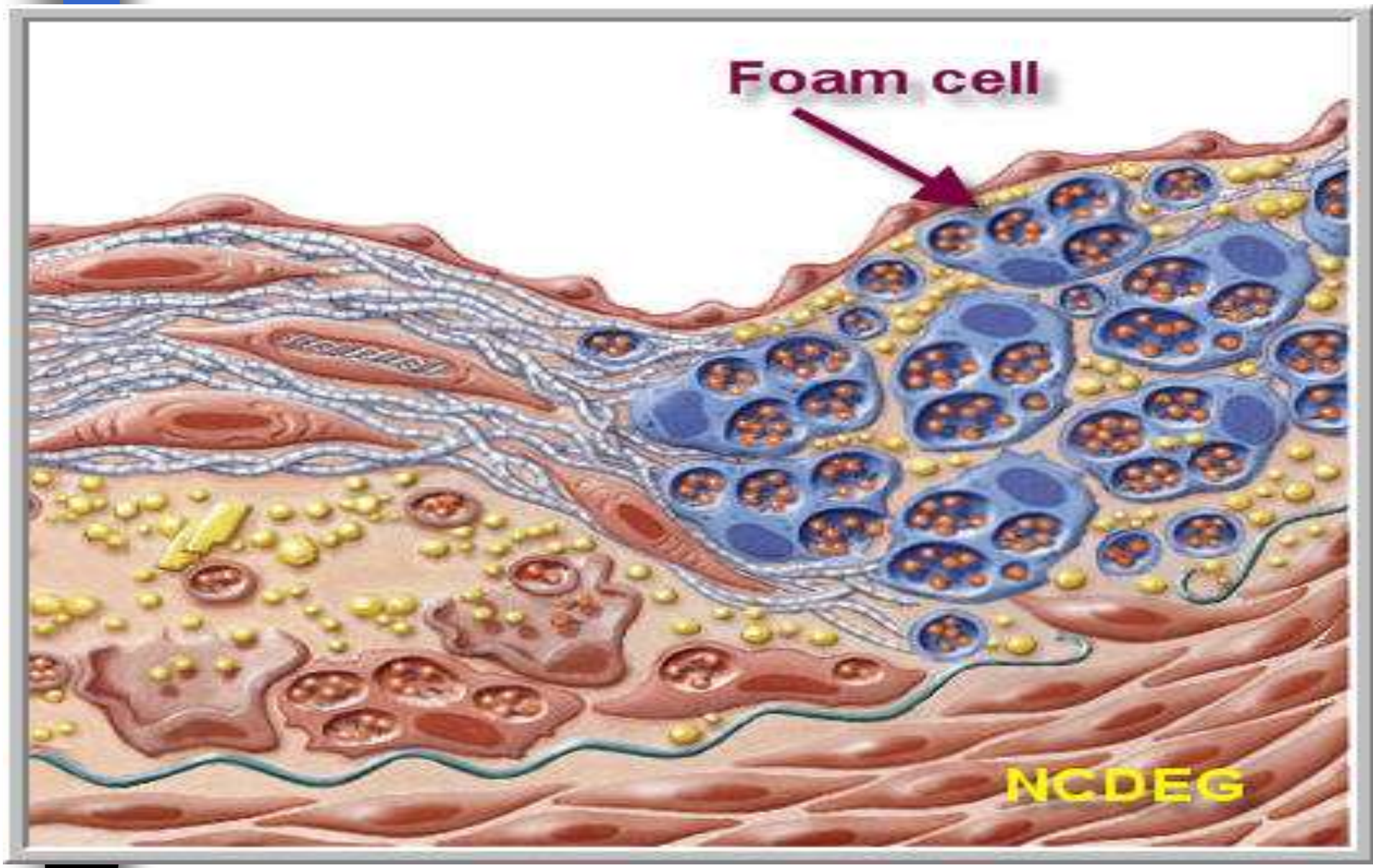
Macrophages continue to take up lipid, then the internal elastic membrane rupture after that the smooth muscle migrates into intima where it begins to synthesize and secret collagen fiber, collagen fiber + smooth muscle+ macrophage form thick fibrous cap which continue to grow lead to narrowing of the arterial lumen

Fibrous Plaque Formation:



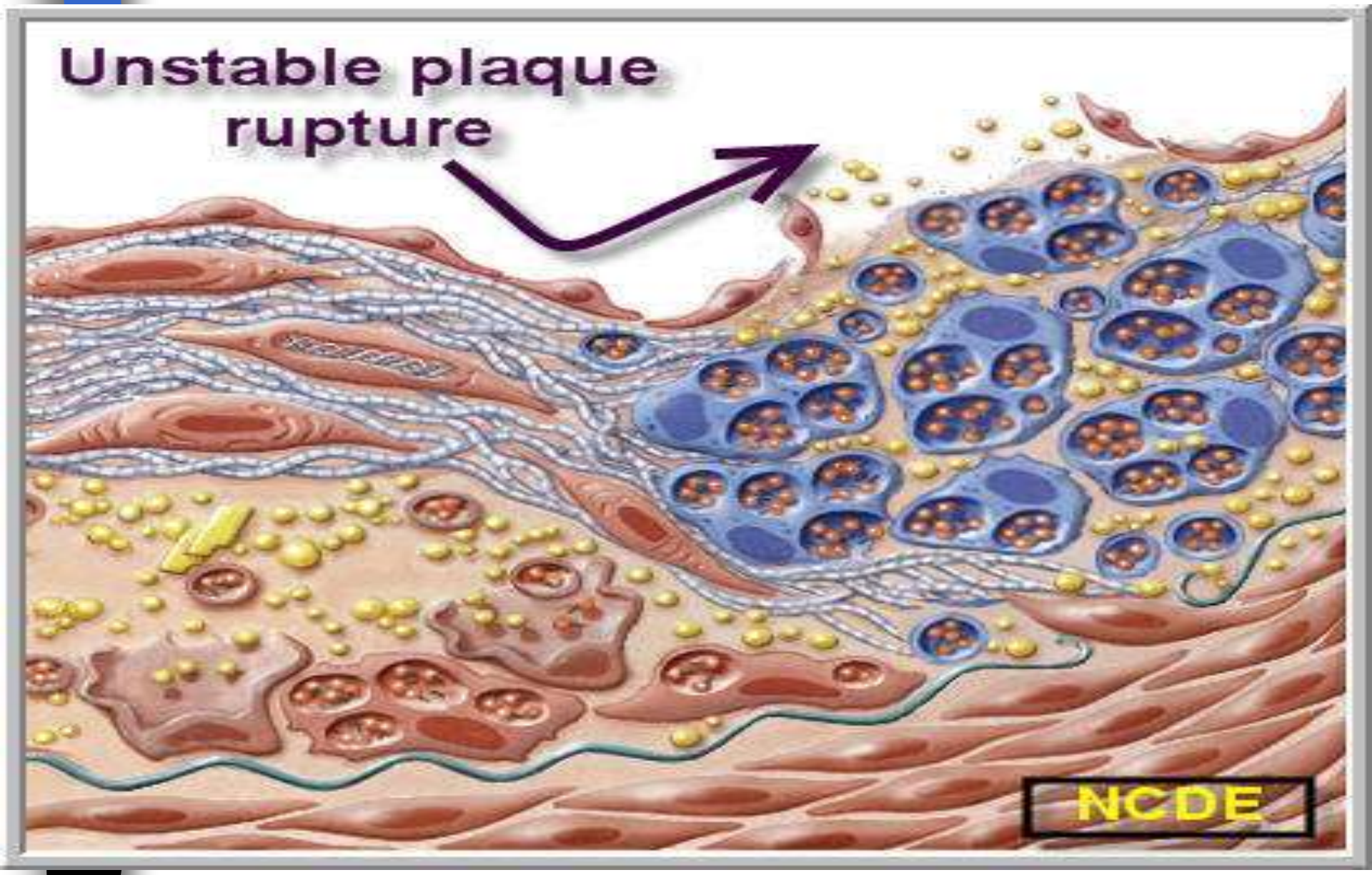
- Degree of stability depends upon degree of inflammation and consequent thickness of the fibrous cap, also in this stage macrophages release enzymes that eat thin wall of foam cell, the plaque become more likely to rupture leading to erosion on endothelium

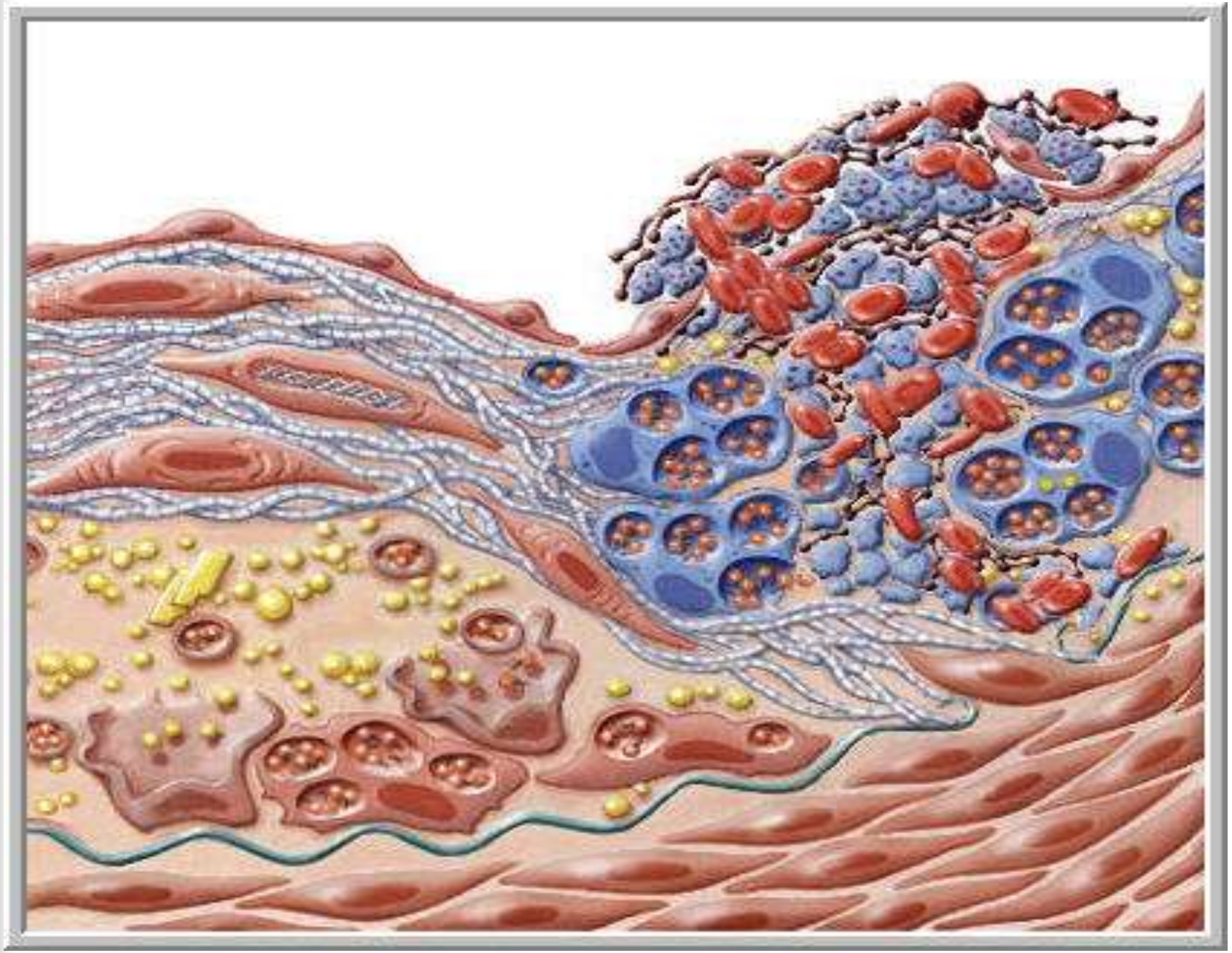
Unstable plaque

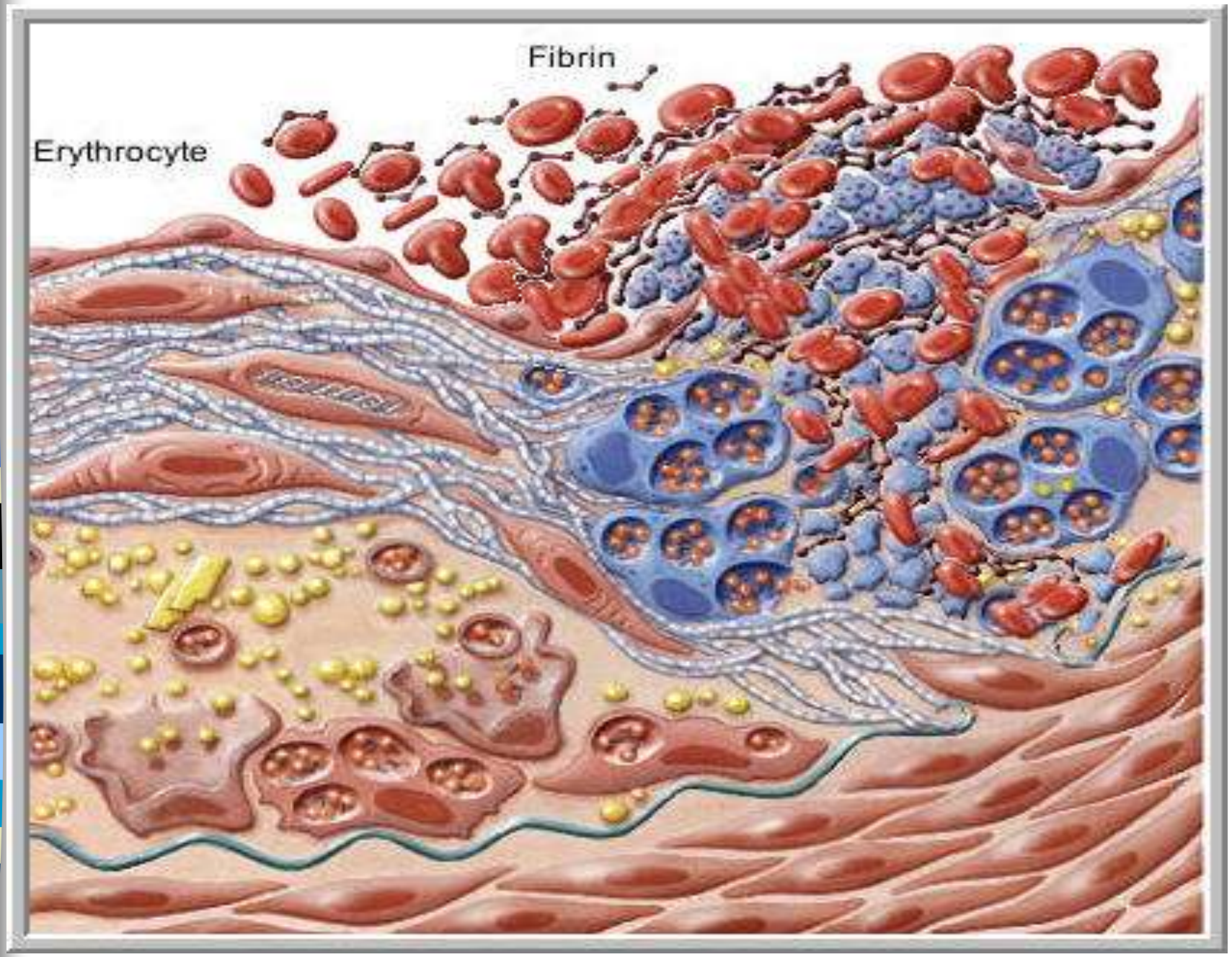


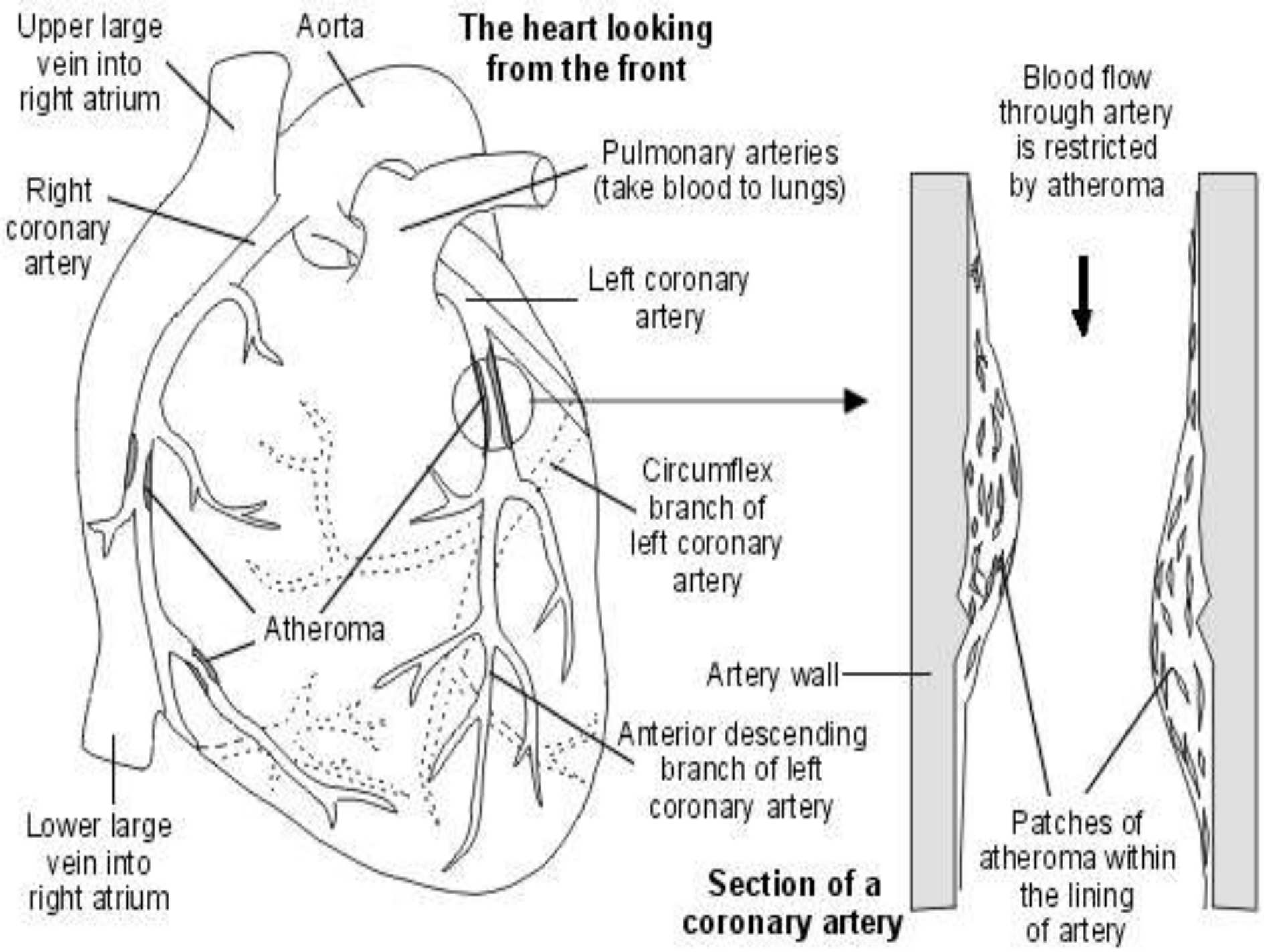
- When the plaque rupture erythrocyte adhere to the site and accumulate to form thrombus

Plaque rupture






Fibrin
Erythrocyte


Upper large vein into right atrium
Aorta
The heart looking from the front
Pulmonary arteries (take blood to lungs)
Right coronary artery
Left coronary artery
Circumflex branch of left coronary artery
Atheroma
Lower large vein into right atrium
Anterior descending branch of left coronary artery
Blood flow through artery is restricted by atheroma
Artery wall
Patches of atheroma within the lining of artery
Section of a coronary artery

- There are other phenomena causes decrease blood flow to the heart as:

- ✓ Vasospasm
- ✓ Decrease O2 supply as in anemia and hypotension
- ✓ Increase O2 demeaned (increase HR or in cocaine use)

Clinical Manifestation

- Symptoms and complications are related to the location and degree of vessel obstruction
- Symptoms are due to myocardial ischemia.
- Angina pectoris
- Myocardial infarction
- Heart failure
- Sudden cardiac death
- **chest pain**; however, some individuals may be asymptomatic or have atypical symptoms such as weakness, dyspnea, and nausea.

- Atypical symptoms are more common in women and in persons who are older or who have a history of heart failure or diabetes.
- Prodromal symptoms may occur as first indication of coronary atherosclerosis

Risk Factors

- Non modifiable:
  - Age (>45y for men, >55y for women)
  - Gender (men > women)
  - Hereditary (1st degree relative with CVD at 55y or younger for men, at 65y and younger for women)

Risk Factors

- Modifiable:
  - Hypercholesterolemia – dyslipidemia
  - Hypertension
  - Smoking
  - Diabetes
  - Physical inactivity
  - Obesity
  - Metabolic syndrome
  - Elevated C-reactive protein (CRP)

Prevention of CAD

- Controlling Cholesterol:

Therapeutic Lifestyle changes: -

- Diet: total fat < 35%, 50-60% CHO, 15% protein, cholesterol < 200 mg, 20-30 g\d fiber
- Exercise (walking for 30m\day)
- Smoking cessation
- Wt loss
- Control other risk factors (BS, BP)

Medications:

- Statins (atorvastatin, simvastatin):

Block cholesterol synthesis, decrease LDL &TG, increase HDL, LFT should monitor

- Nicotinic Acid (niacin):

Decrease lipoprotein synthesis, decrease LDL & TG, increase HDL

- Fibric Acid or fibrat (Clofibrate, fenofibrate):

Decreases cholesterol synthesis, decrease TG, increase HDL

LDL target

- less than 160 mg/dL for patients with one or no risk factors
- Less than 130 mg/dl for patients with 2 or more risk factors
- Less than 100 mg/dl for patients with CAD or at high
- Less than 70 for patient with diabetes and CAD

Case 1

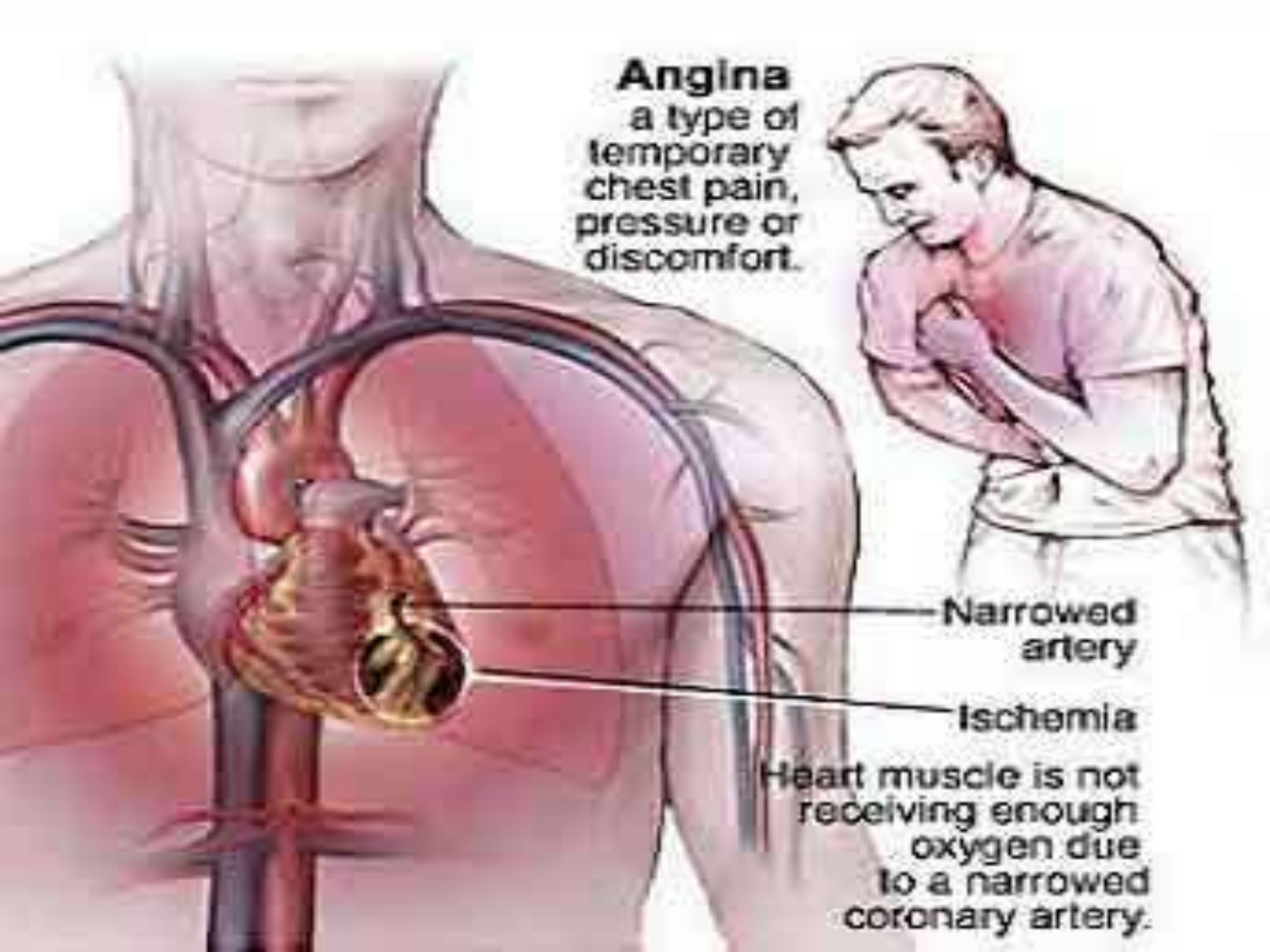
- Mrs. S 64y old female pt, obese, has a HX of HTN, type 2 DM, presented to ER C/O chest discomfort that attack here >3t/wk, induced by climbing stairs and stress relived by rest, radiated to jaw, shoulder and neck, associated with dyspnea and vomiting, the pain like heavy rock over the chest.

Case2

- Mr. Y 50 y old male pt, smoker 2pk/d, hypertensive, wc=110cm, works as a farmer, presented to a clinic c/o chest pain 10/10, as tightness last >20m came suddenly while he is watching TV, radiated to shoulder, inner aspect of hand, associated with sweating, vomiting and dyspnea, its not relived by rest

Angina Pectoris

- Clinical syndrome characterized by episodes of discomfort (pain) or pressure in the anterior chest caused by insufficient coronary blood flow
- Physical exertion or emotional stress increases myocardial oxygen demand, and the coronary vessels are unable to supply sufficient blood flow to meet the oxygen demand.
- Most common cause atherosclerosis

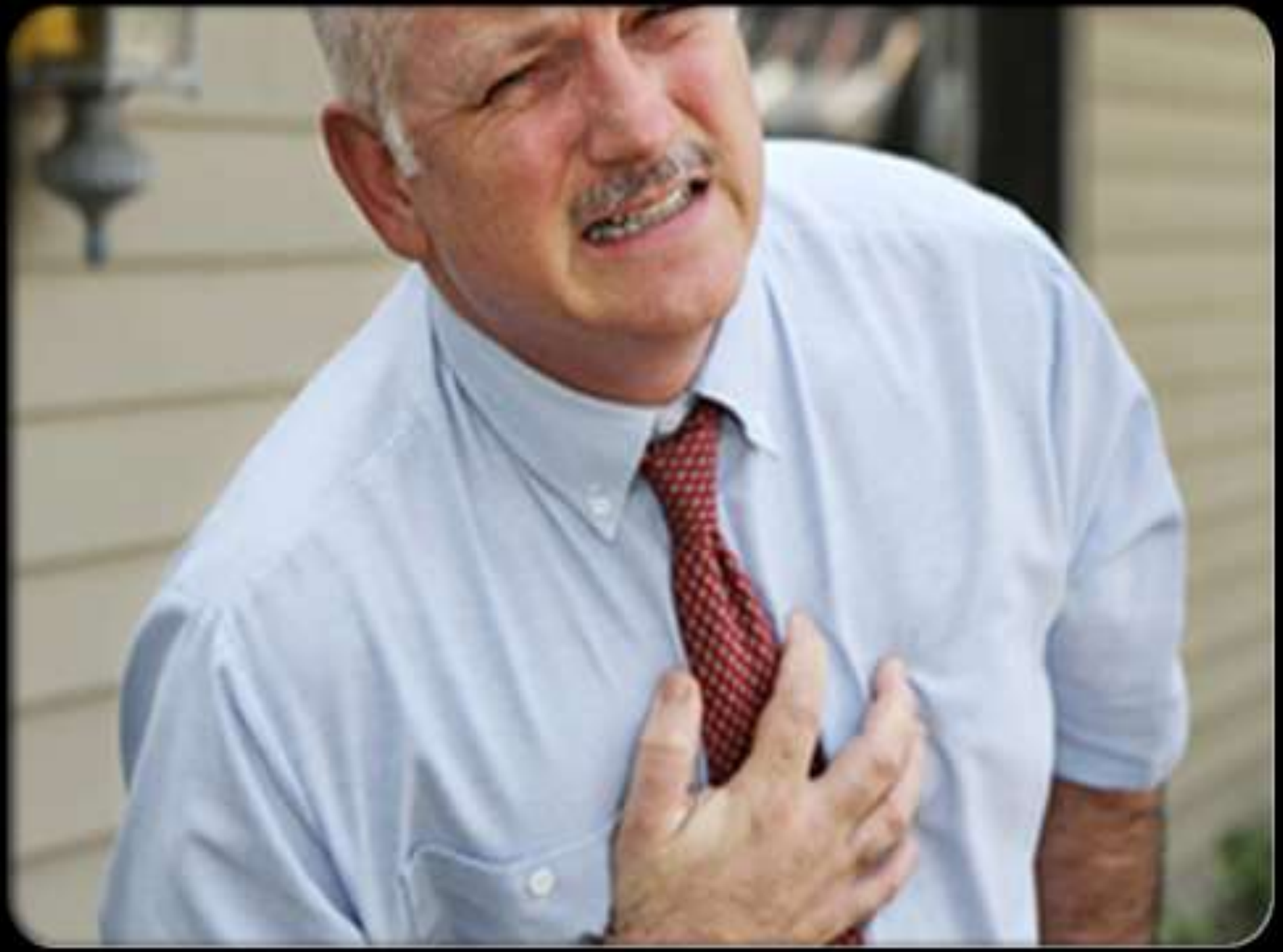

Angina
a type of temporary chest pain, pressure or discomfort.
Narrowed artery
Ischemia
Heart muscle is not receiving enough oxygen due to a narrowed coronary artery.

- Several types:
  - Stable
  - Unstable (preinfarction, crescendo)
  - Intractable or refractory
  - Variant (prinzemetal's)
  - Silent ischemia

Factors Known To Precipitate Typical Angina

- Physical exertion
- Exposure to cold
- Eating a heavy meal
- Stress or emotional situation

Unstable angina not associated with these factors it may occur with rest



Clinical Manifestation

- May be described as tightness, choking, or a heavy sensation in the upper chest
- Pain felt deep in the chest (retrosternal), poorly localized
- It is frequently retrosternal and may radiate to neck, jaw, shoulders, or inner aspect of arms (usually left).
- Anxiety frequently accompanies the pain.
- Other symptoms may occur: dyspnea/shortness of breath, dizziness, nausea, and vomiting.
- The pain of typical angina subsides with rest or NTG.
- Unstable angina is characterized by increased frequency and severity and is not relieved by rest and NTG. Requires medical intervention

Diagnostic Findings With Angina

- Diagnosis often made by evaluating clinical manifestations and history
- 12 lead ECG (T wave depression)
- Stress test
- Cardiac catheterization or coronary angiography
- Laboratory values (lipid profile, CRP, cardiac biomarkers), to rule out ACS

Medical Management

- Objective: decrease O2 demand and increase O2 supply By:
- Pharmacologic therapy
- Control risk factors
- Revascularization
  - PCI procedures (PTCA)
  - Coronary artery bypass grafting (CABG)

Medications

- ***Nitroglycerin:*** vasoactive agent mainstay of treatment
- Dilate veins – decreases preload
- Dilate arteries – decreases afterload as well as dilates coronary arteries
- Administer- spray, sublingually, PO, IV, topically
- Side effects – hypotension, HA, flushing, tachycardia
- Ex: Nitrostat, Nitrol, isordile
- DO NOT administer with Viagra
- Chart 27-3 (important)

- Client teaching related to sublingual (SL) nitroglycerine (NTG)
  - Carry NTG all times
  - Heat, light, and moisture cause NTG to lose its potency. Store in original container (dark capped glass container).
  - Renew every 6 months
  - Sit or lie down when taking
  - Take one tablet under tongue every 5 min until angina relieved. If no relief after 3 tabs, call emergency
  - May take immediately before activity causing angina
  - Instruct pt about side effect

- ***Beta blockers***
  - Reduce myocardial oxygen consumption by decreasing heart rate, contractility and blood pressure
  - Caution client not to stop med abruptly; may cause worsening of angina, MI may develop
  - Monitor heart failure clients for worsening failure
  - Side effects – hypotension, bradycardia, bronchial spasm, masks hypoglycemia
  - Ex: metoprolol (Lopressor or Toprol), propranolol (Inderal), Atenolol (Tenormin)

Calcium channel blockers

- Dilate arteries – decreases SVR which decreases workload and O2 consumption
- Decrease heart rate and myocardial contractility – decreases O2 consumption
- Avoid in clients with severe heart failure (decreased myocardial contractility)
- Side effects - hypotension, bradycardia, constipation, edema, AV blocks
- Ex: nifedipine, diltiazem, amlodepine

- **Antiplatelet medications**
  - Prevent platelet aggregation on atheroma or thrombus
    - ASA – side effects: GI irritation, bleeding, increased bruising, used with H2 blockers or proton pump inhibitor (omeprazole)
    - Ticlid (ticlopidine) – antiplatlet, side effects: neutropenia, GI upset, N/V/D, rash. Must monitor CBC
    - Plavix (clopidogrel) – side effects: increased bleeding tendencies, N/V/D, rash

- **Anticoagulants**
  - ***Heparin***
    - Given IV in acute situations or subcutaneous in non-acute situations
    - Monitor partial thromboplastin time (PTT)
    - Antidote – Protamine Sulfate
    - perform bleeding precautions
    - Therapeutic effect when PTT 2 – 2.5 times the normal
    - Monitor for signs and symptoms of bleeding
    - Monitor for Heparin induced thrombocytopenia (HIT)

– **Coumadin (warfarin)**

- Used long term; given PO
- Effects do not occur for 3-5 days
- Monitor Prothrombin time (PT) or International Normalized Ratio (INR)
- Antidote – Vitamin K
- Affected by certain foods
- Contraindicated in pregnancy, clients with liver dysfunction or those at risk for bleeding

Glycoprotein IIb\IIIa agent (aggrastat) :

- Prevent platelet aggregation by blocking the GPIIb\IIIa receptors on the platelet
- Major S\E Bleeding, bleeding precusion should be initiated

- **Oxygen therapy**
  - Administered usually at 2 L/min per nasal cannula
  - Initiated at the onset of chest pain
  - To Increases amount of O2 delivered to myocardium
  - Monitor O2 sat > 90%

Nursing Process: The Care of the Patient with Angina Pectoris: Assessment

- Symptoms and activities, especially those that precede and precipitate attacks
- See **Chart 27-4 p 740**
- Risk factors, lifestyle, and health promotion activities
- Patient and family knowledge
- Adherence to the plan of care

Collaborative Problems

- ACS/Myocardial infarction
- Dysrhythmias and cardiac arrest
- Heart failure
- Cardiogenic shock

Diagnosis

- Ineffective cardiac tissue perfusion R/T atherosclerosis AMB chest pain, dyspnea, cardic cath result other symptoms
- Chronic pain R/T increase O2 demand and Decrease O2supply.
- Death Anxiety R\T cardiac symptoms

- Activity intolerance R/T myocardial ischemia
- Deficient knowledge regarding medication
- Noncompliance, ineffective management of therapeutic regimen R/T failure to accept life style changes.

Planning

- Goals include the immediate and appropriate treatment of angina, prevention of angina, reduction of anxiety, awareness of the disease process, understanding of prescribed care, adherence to the self-care program, and absence of complications.

Treatment of Anginal Pain

- Treatment of anginal pain is a priority nursing concern
- Patient is to stop all activity and sit or rest in bed in Semi fowler position.
- Assess the patient while performing other necessary interventions. Assessment includes VS, observation for respiratory distress, and assessment of pain. In the hospital setting, the ECG is assessed or obtained.
- Administer oxygen .
- Administer medications as ordered or by protocol, usually NTG.

- Anxiety:
  - Use a calm manner
  - Stress-reduction techniques
  - Patient teaching
  - Addressing patient spiritual needs may assist in allaying anxieties
  - Address both patient and family needs

- Patient Teaching:

- Lifestyle changes and reduction of risk factors
- Explore, recognize, and adapt behaviors to avoid to reduce the incidence of episodes of ischemia.
- Teaching regarding disease process
- Medications (how to use NG)
- Stress reduction
- When to seek emergency care