ISCHEMIC HEART DISEASE

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Ischemic Heart Disease (IHD) Outline

When to suspect patient with IHD

Basic: coronary circulation

Myocardial oxygen supply and demands

Causes of IHD

Management

Case presentation

A 65 Year old male, presented to outpatient clinic complaining of chest pain of 5 months duration.

What are the possible anatomical causes of chest pain?

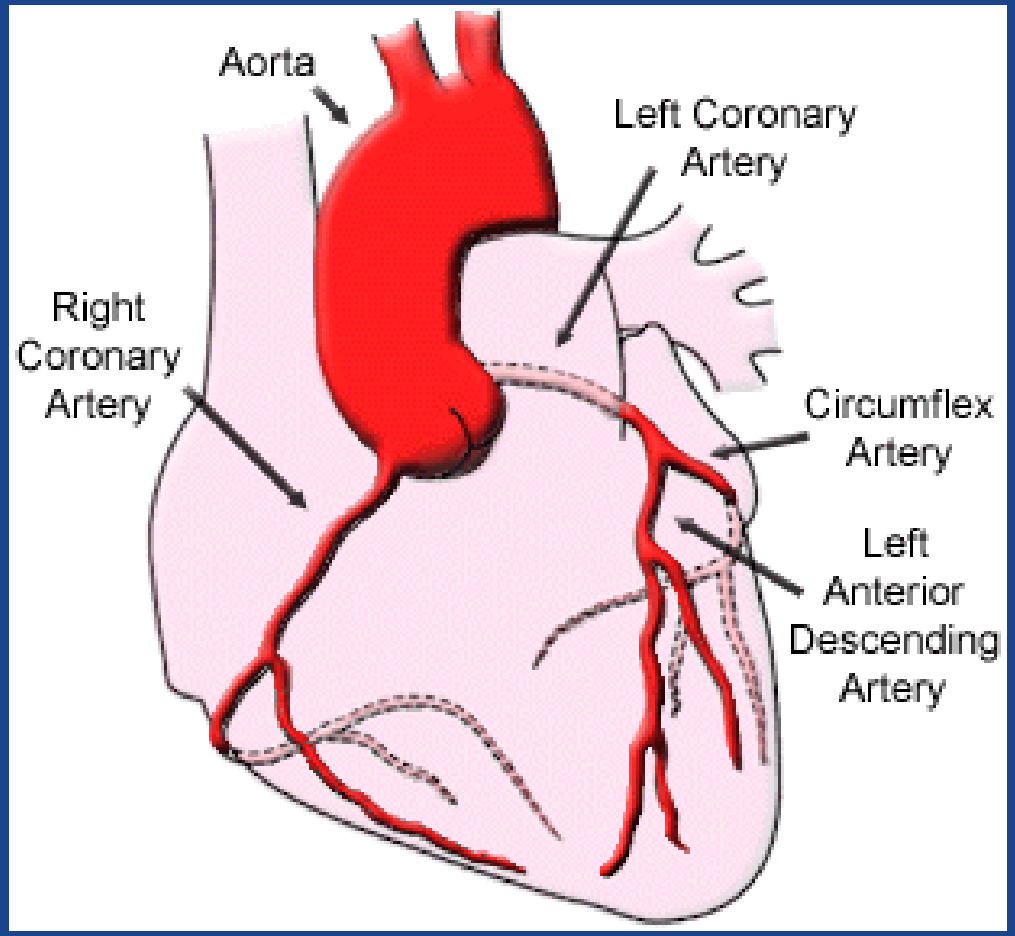
The pain is retrosternal, compressive in nature, precipitated by ~~wakening of~~ *walking 400 meter , relieved by rest, radiated to left shoulder, associated with sweating.

Patient is diabetic => A major risk factor for atherosclerosis
And smoker

On examination:
Blood pressure:160/100. pulse rate: 88 bpm
Heart auscultation : normal
WHAT IS THE PROBLEB? => Possibly IHD
What is abnormal physical findings? => Hypertension
What to do? Investigations

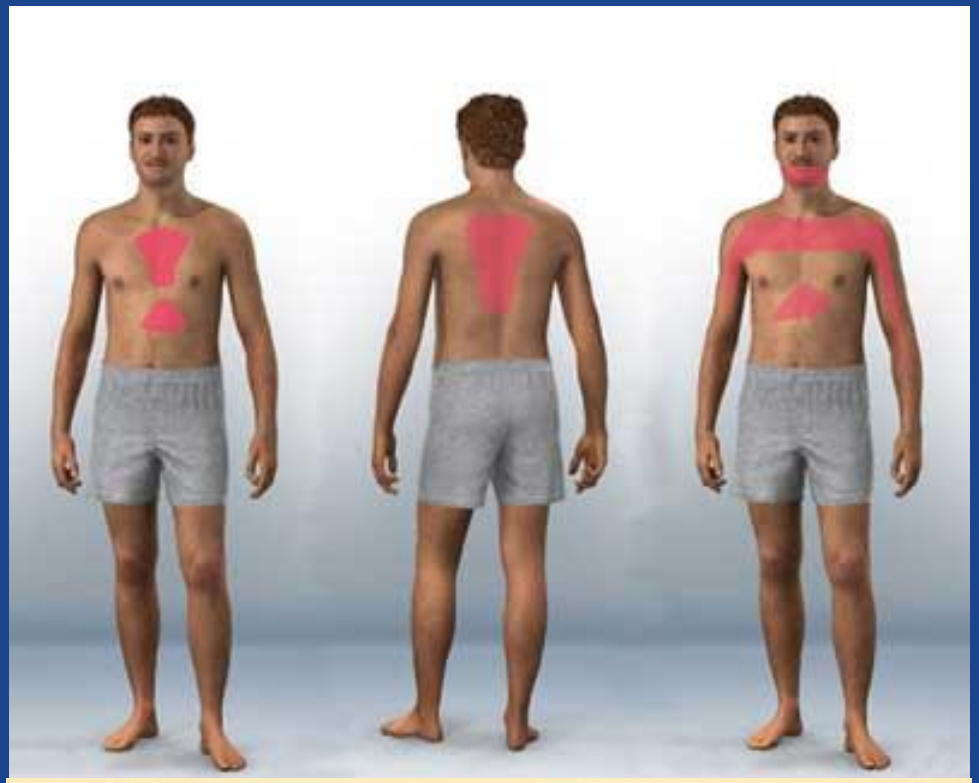
Main complaint in a patient with IHD:
- Chest pain
- Shortness of breath (dyspnea)
- Palpitations
- Fatigue

Coronary Anatomy



Symptoms of Angina

Angina can spread anywhere between the belly button and the jaw, including to the shoulder, arm, elbow or hand- usually on the left side.



Focus on: the causes of increased blood demand and decreased blood supply.

Ischemic Heart Disease

: occurs when there is an imbalance between the myocardial blood supply and blood demand. Factors affecting each are:

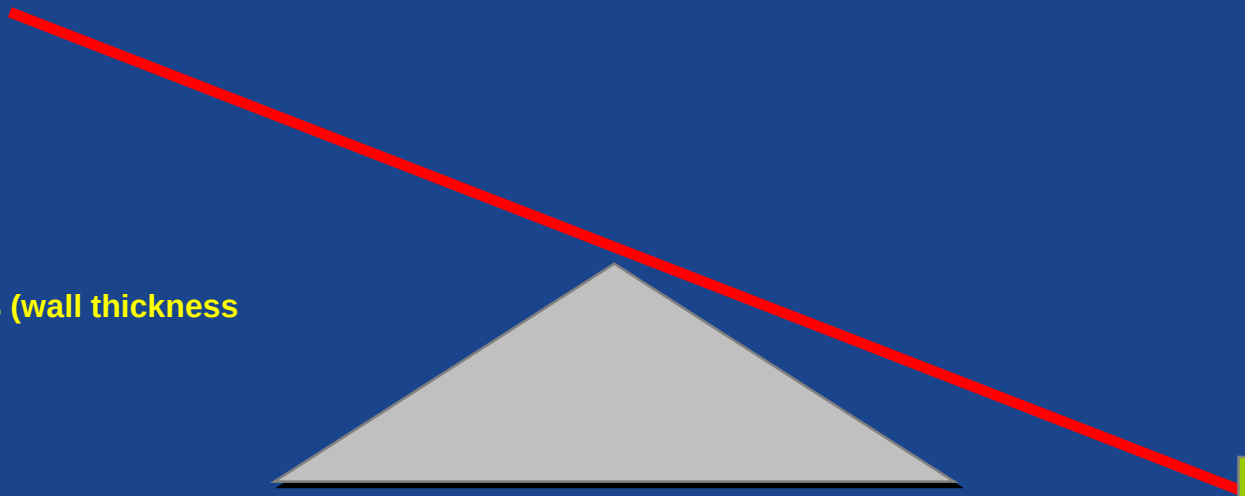
demand

1- Heart rate

2- Contractility

3- Wall tension

4- Muscle mass (wall thickness



supply

1- Coronary flow (patency of coronary artery)

2- Hemoglobuline level

3- Myocardial oxygen extraction

4- Arterial oxygen saturation

- The faster the heart rate > more blood / oxygen demand
  => Patients with IHD should avoid tachycardia (beta-blockers)

- Heart's blood supply is mainly during diastole; during systole, the majority of the coronary vessels are compressed.

Causes of coronary artery disease

95% Atherosclerosis

It is a diffuse disease capable of affecting any artery in our body in which plaque builds up inside them; coronary arteries can be affected.

The following slide shows the risk factors of atherosclerosis:

5% Nonatherosclerosis

Risk Factors for Cardiovascular Disease (atherosclerosis risk factors)

(Reversible)

Modifiable

- Hypertension
- Smoking
- Hyperlipidaemia
  - Raised LDL-C
  - Low HDL-C
  - Raised triglycerides
- Diabetes mellitus => 4th major risk factor
- Dietary factors
- Lack of exercise
- Obesity
- Homocysteinemia
- Lipoprotein a
- Gout
- Thrombogenic factors: fibrinogen, factors V,VII
- Excess alcohol consumption

(Irreversible)

Non-modifiable

- Personal history of CVD
- Family history of CVD
- Age: M>45, F>55
- Gender M>F (Premenopausal)
- Personality type A
- Genetic factors: ACE gene

Focus on: the pathophysiology of atherosclerosis.

Endothelial Dysfunction in Atherosclerosis

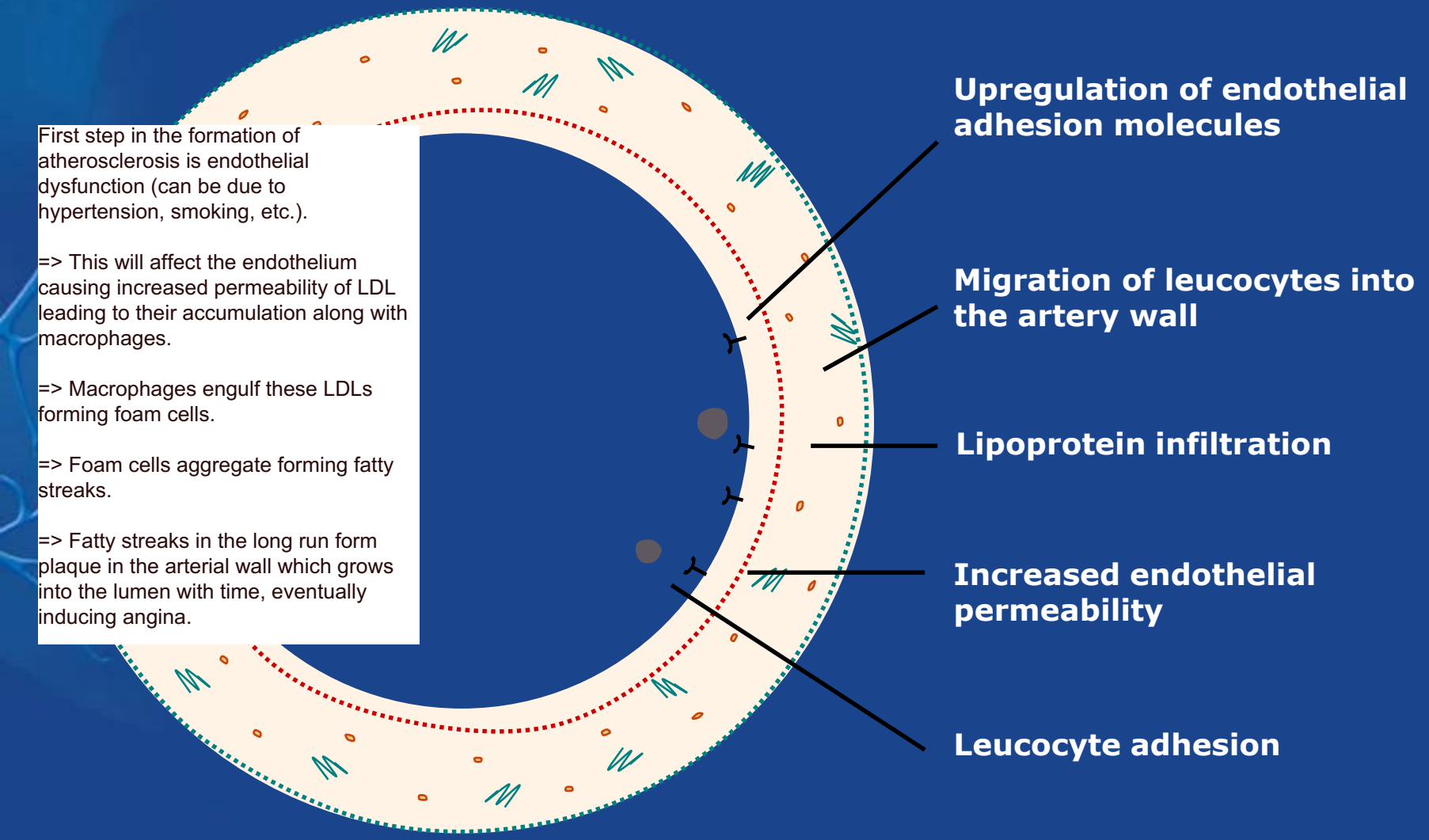
First step in the formation of atherosclerosis is endothelial dysfunction (can be due to hypertension, smoking, etc.).

=> This will affect the endothelium causing increased permeability of LDL leading to their accumulation along with macrophages.

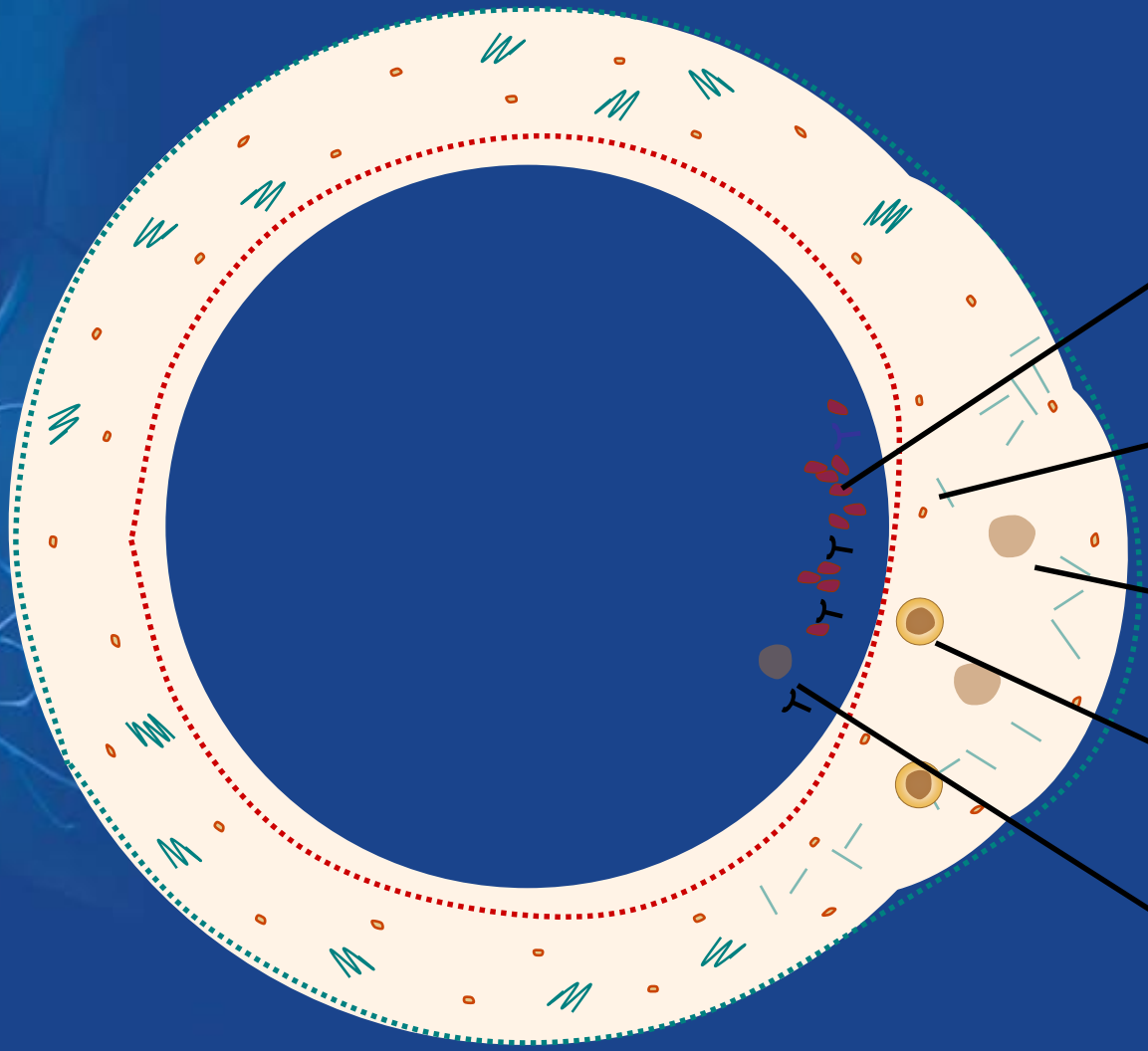
=> Macrophages engulf these LDLs forming foam cells.

=> Foam cells aggregate forming fatty streaks.

=> Fatty streaks in the long run form plaque in the arterial wall which grows into the lumen with time, eventually inducing angina.



Fatty Streak Formation in Atherosclerosis



Adherence and aggregation of platelets

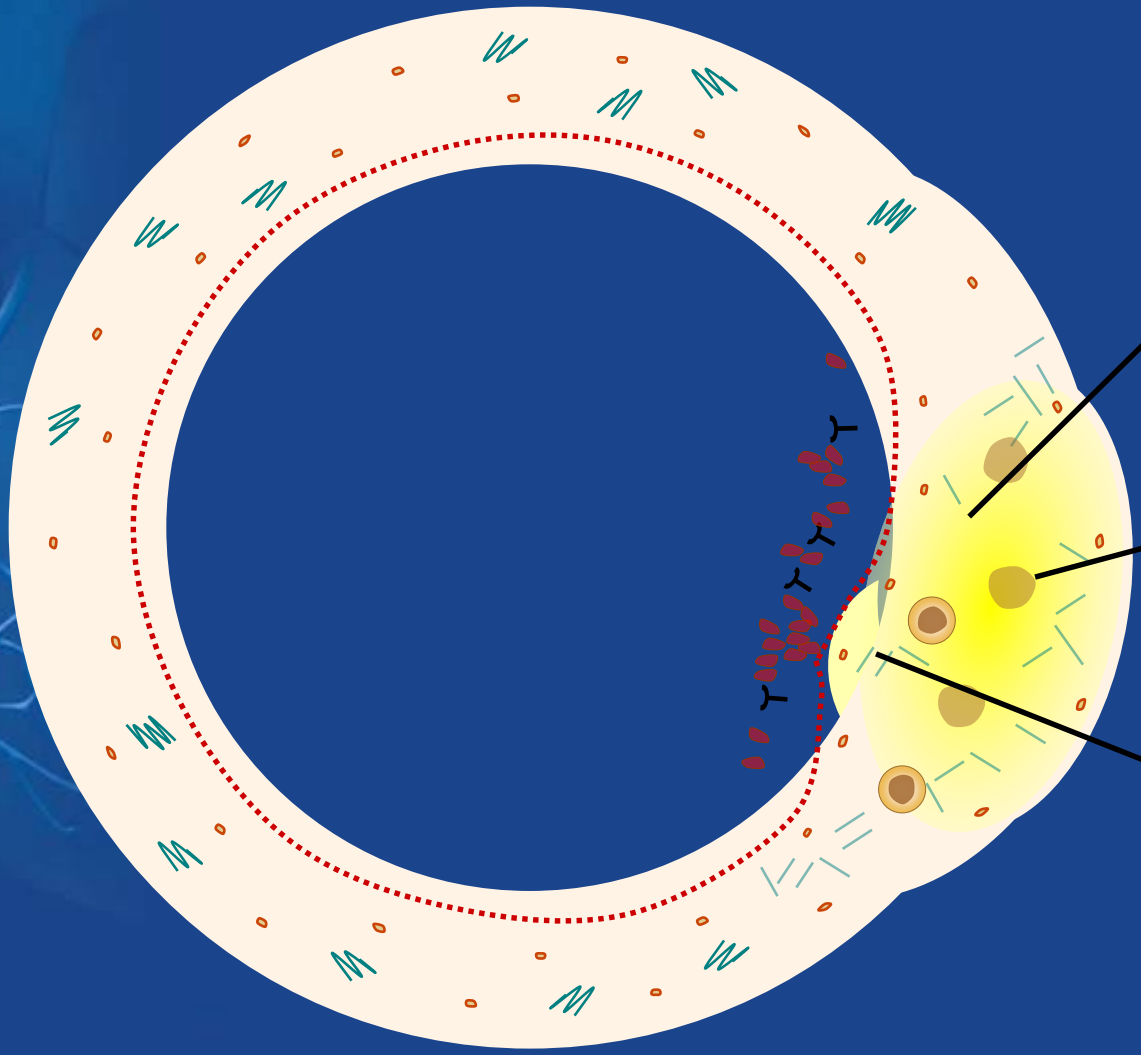
Migration of smooth muscle cells

Formation of foam cells

Activation of T cells

Adherence and entry of leucocytes

Formation of the Complicated Atherosclerotic Plaque



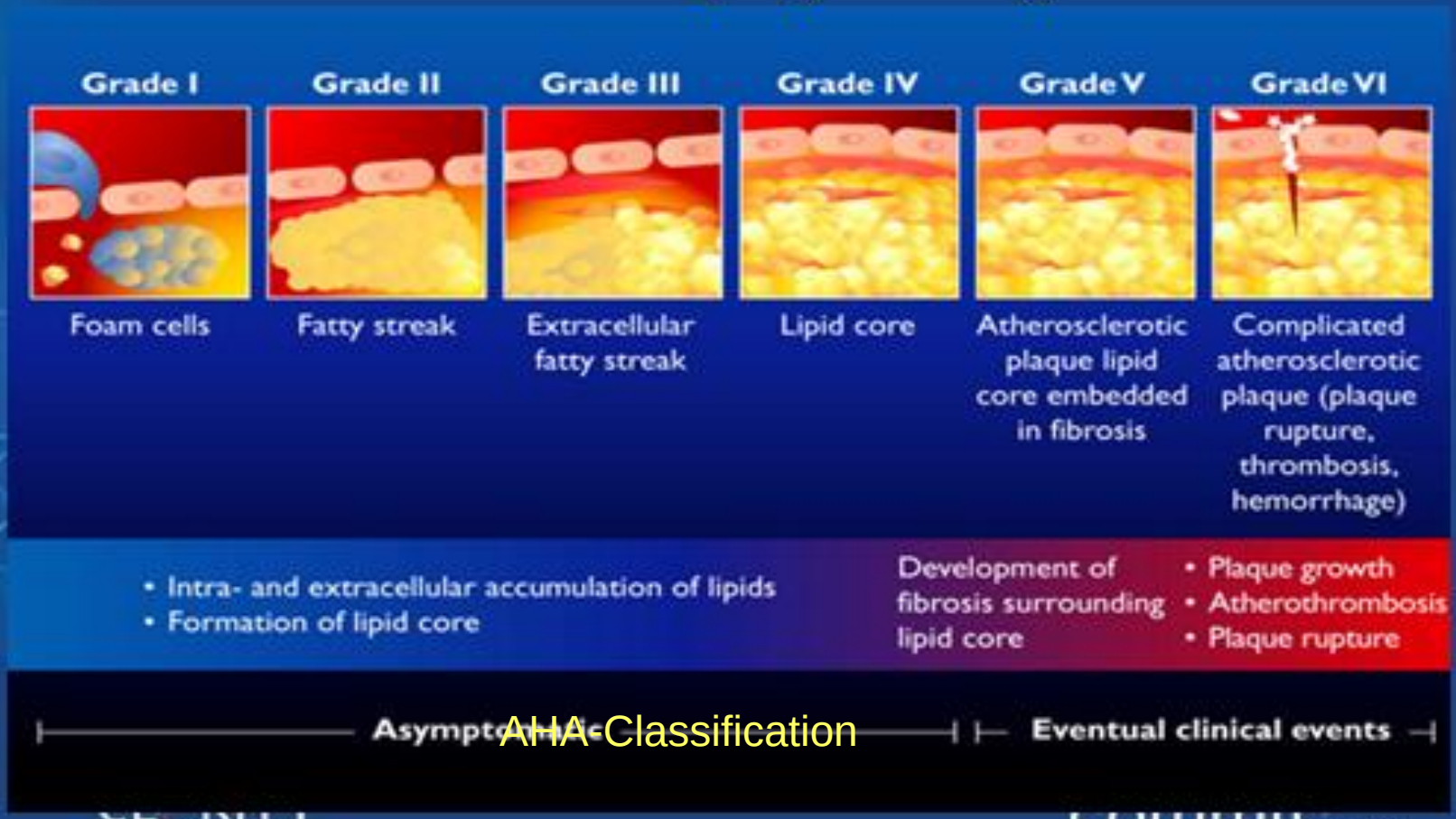
Formation of necrotic core

Note: when this core is exposed due to plaque rupture, it serves as a surface for platelet adhesion inducing thrombosis.

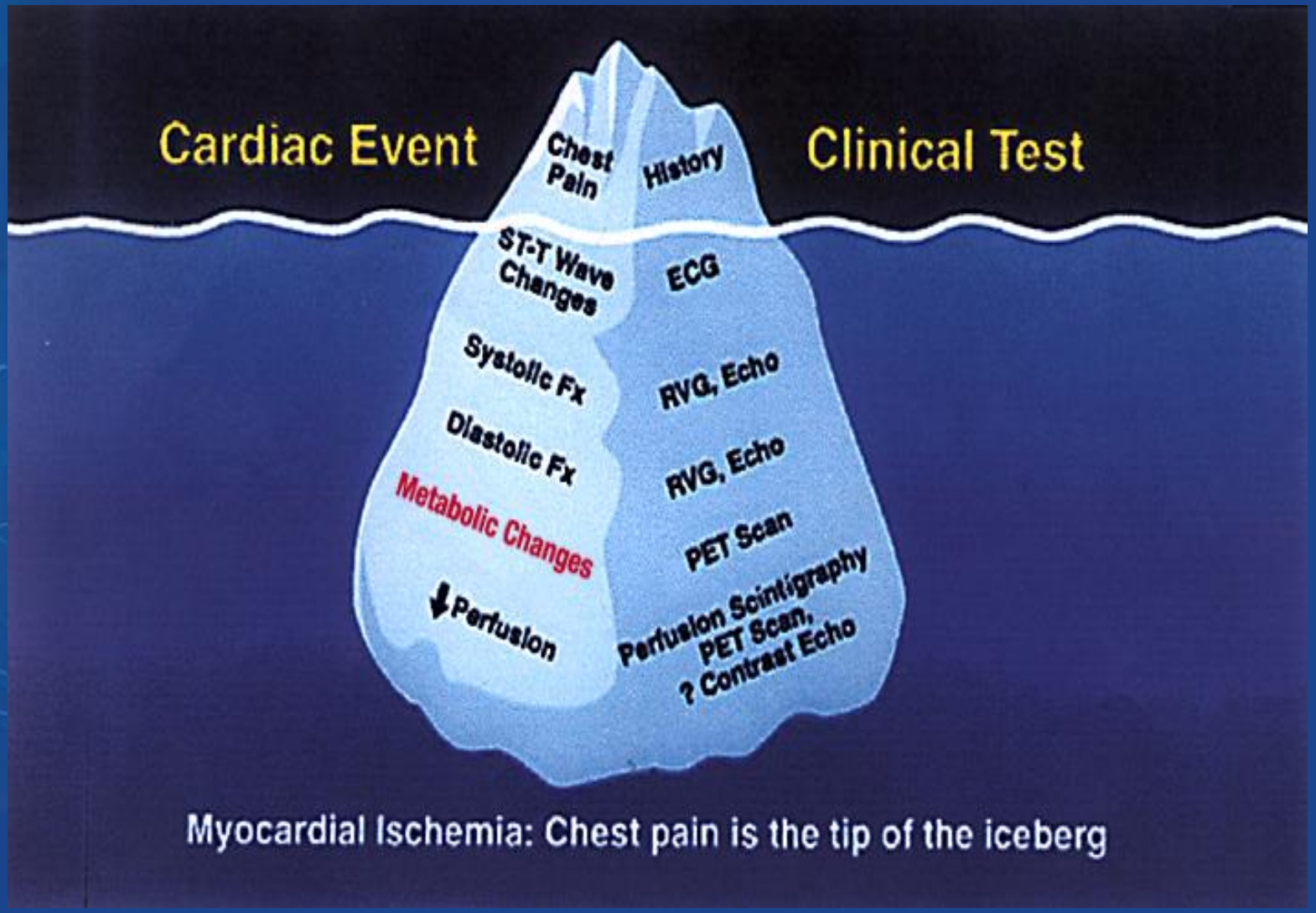
Accumulation of macrophages

Formation of the fibrous cap

Cardiovascular risk factors and the stages of atherosclerotic plaque development



AHA-Classification



Gottdiener JS. In: ACCSAP 1997-98 by the ACC and AHA

Clinical Manifestations of Atherosclerosis

Coronary heart disease

- Asymptomatic
- **Angina pectoris, variant angina**
- **Myocardial infarction, Unstable angina**
- Heart failure (HF)
- Arrhythmias
- Sudden cardiac death.

IHD-clinicopathological correlation

1- stable angina: stenosis > 70% luminal narrowing

2-variant angina: increase coronay tone
30% normal coronaries

3-unstable angina: rupture plaque
subocclusive thrombus
progress to myocardial infarction 15-30%

4-myocardial infarction: rupture plaque
occlusive thrombus

Focus on: stable vs unstable angina.

Stable angina: it is triggered by physical activity due to stenosis (>70% luminal narrowing). It resolves with rest or nitrates.
Unstable angina: it is unpredictable and cause ACS which may lead to MI. it does not resolve with rest nor with drugs.

Acute Coronary Syndrome (ACS) will be discussed more later on.

Angina Chest Pain:

Clinical Diagnosis

The most important step in diagnosis is taking history.

CAUSES OF ANGINA

Reduced Myocardial O2 Supply

1-Coronary artery disease

2-Sever Anemia

Increased Myocardial O2 Demand

1-Left Ventricular Hypertrophy:

- hypertension
- aortic stenosis
- hypertrophic cardiomyopathy

2- Rapid Tachyarrhythmias

Differential diagnosis of angina

History is important to diagnose and understand what's the main problem causing angina.

1- Neuromuscular disorder

2- Respiratory disorders

3-Upper GI disorder

4- Psychological

5- Syndrome X:

Typical angina with normal coronary angio

? Increase tone or decrease coronary vasodilatation

excellent prognosis

antianginal therapy is rarely effective

Case presentation

A50 year old male presented to emergency room complaining of sudden sever chest pain of 1 hour duration. It is retrosternal, compressive, and radited to left shoulder and arm.

Associated with sweating, nausea and vomiting

This is a classical scenario indicating myocardial infarction most of the time:
=> a sudden persistent severe chest pain lasting for more than 30mins.

On examination: patient is anxious, in pain, sweaty.

BP: 100/60. PULSE: 120 BPM, RR: 26/min

Chest: basal crepitations

=> Hypotension, tachycardia.
=> RR: respiratory rate is high (normal: 18-20).
=> Basal crepitation: pulmonary edema.

What is the most likely diagnosis

=> Myocardial infarction

pathophysiology

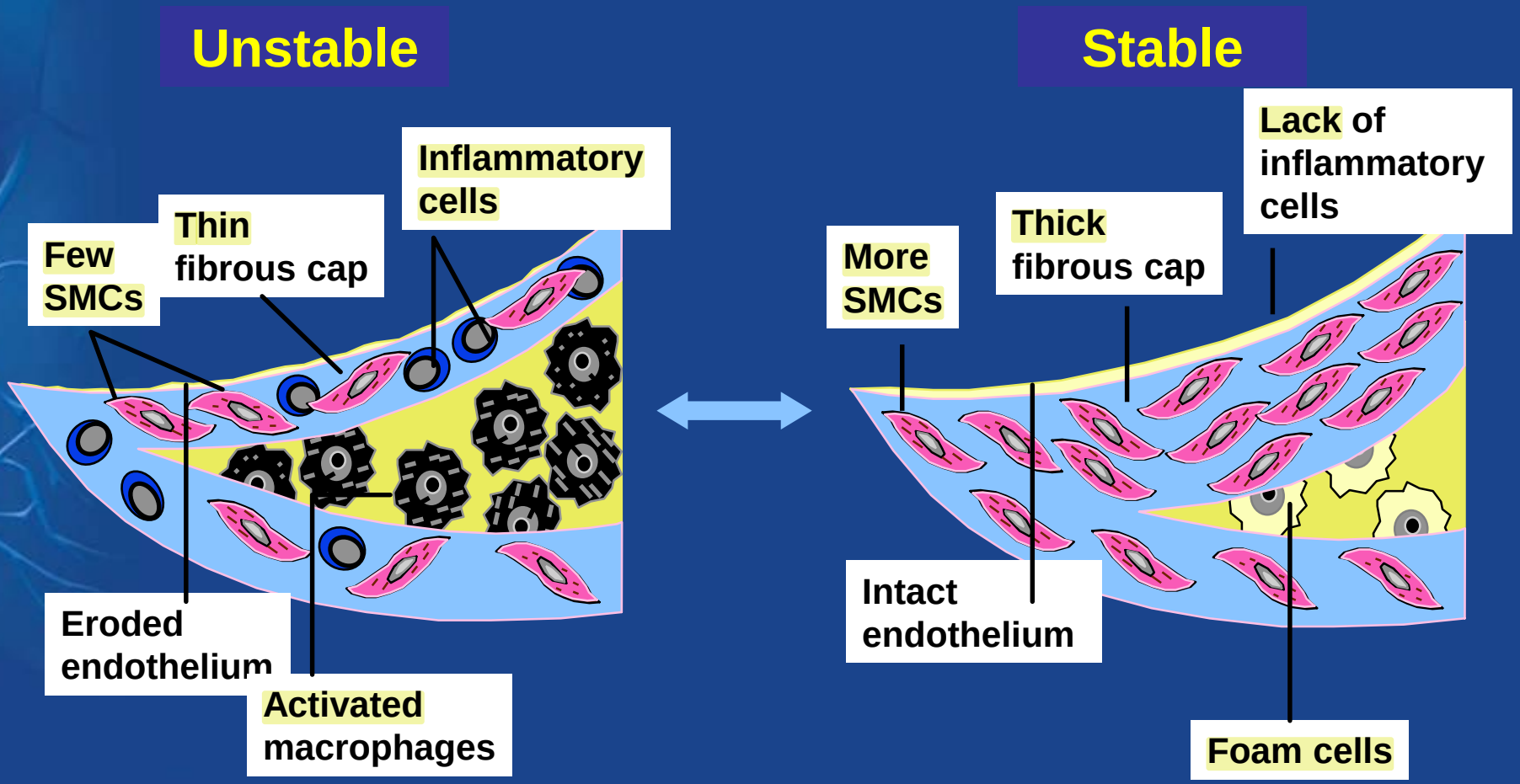
=> Most likely the patient had a total occlusion of one of the coronary arteries.
=> Most of the time what causes the occlusion is a weak unstable plaque 'discussed further on'.

Symptoms of Angina

Angina can spread anywhere between the belly button and the jaw, including to the shoulder, arm, elbow or hand- usually on the left side.

Focus on: the characteristics of stable and unstable plaques.

Characteristics of Unstable(RUPTURE-PRONE PLAQUE) and Stable Plaque



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PATHOGENESIS OF ACS

Acute coronary syndrome (ACS) is a syndrome (set of signs and symptoms) due to decreased blood flow in the coronary arteries causing dysfunction to that part of the heart muscle.

=> It is mainly caused by an unstable plaque rupture.

* **Plaque rupture-----Platelet adhesion---activation---aggregation THROMBOSIS**

1- Primary hemostasis: Initiated by platelet

platelets adhesion, activation, and aggregation---platelet plug

As mentioned before, when the unstable plaque ruptures, it exposes the thrombogenic necrotic core which becomes in contact with platelets inducing their activation.

2- Secondary hemostasis:

activation of the coagulation system---fibrin clot.

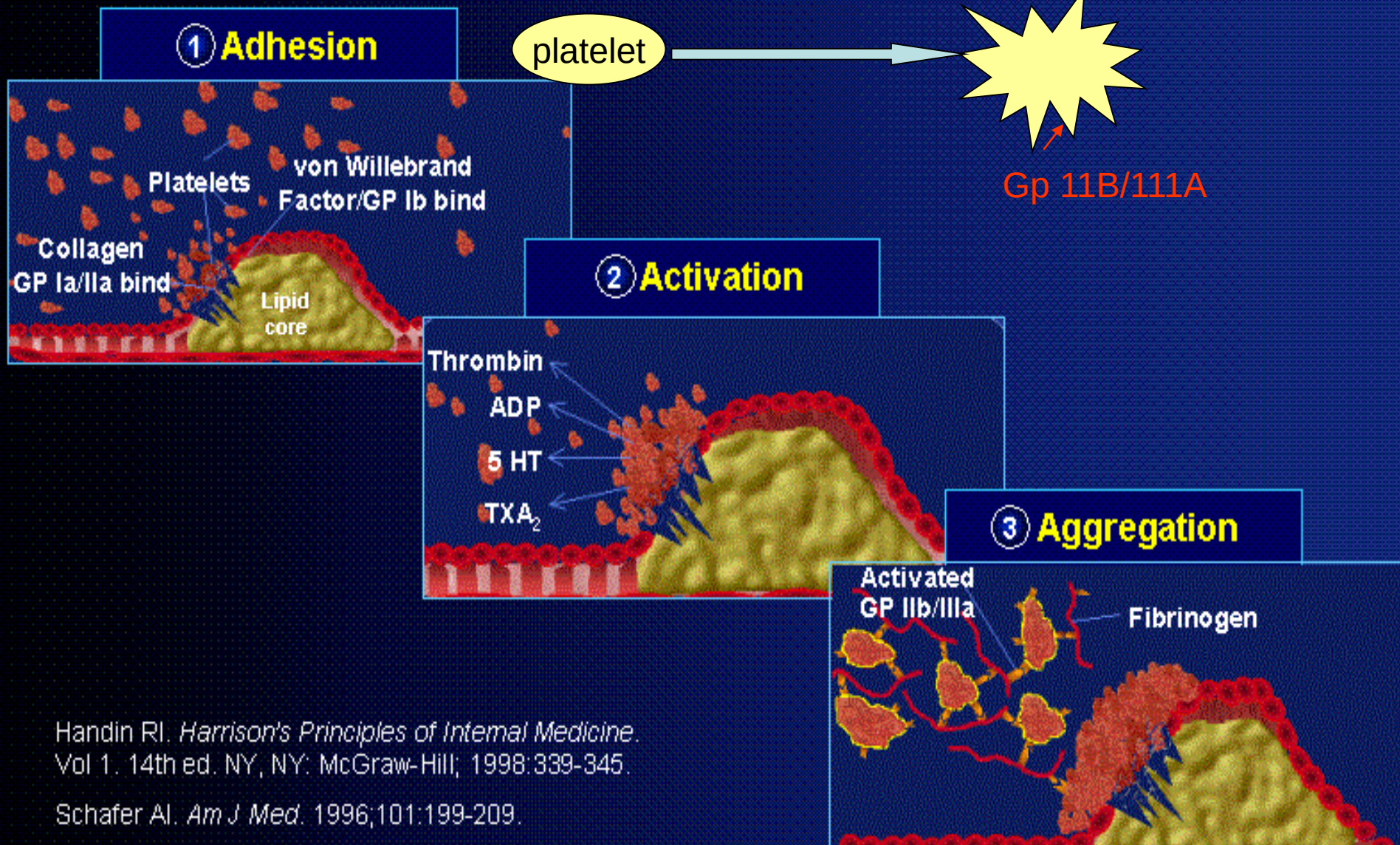
These two phases are dynamically interactive:

Platelet can provide a surface for coagulation enzymes

Thrombin is a potent platelet activator

=> In the end a thrombus will be formed and if it is big enough, it will cause artery occlusion => MI (ST segment elevation)

Platelet Cascade in Thrombus Formation



Handin RI. *Harrison's Principles of Internal Medicine*. Vol 1. 14th ed. NY, NY: McGraw-Hill; 1998:339-345.

Schafer AI. *Am J Med*. 1996;101:199-209.

Diagnosis of Myocardial Infarction

1-History

2-ECG (Electrocardiogram): STMI and NSTMI

- Hyperacute T wave
- ST-segment elevation
- Q- wave
- T- inversion
- ST-segment depresion

normal ECG will not exclude MI

3-Cardiac Marker: Troponin,CPK, myoglobulin,..

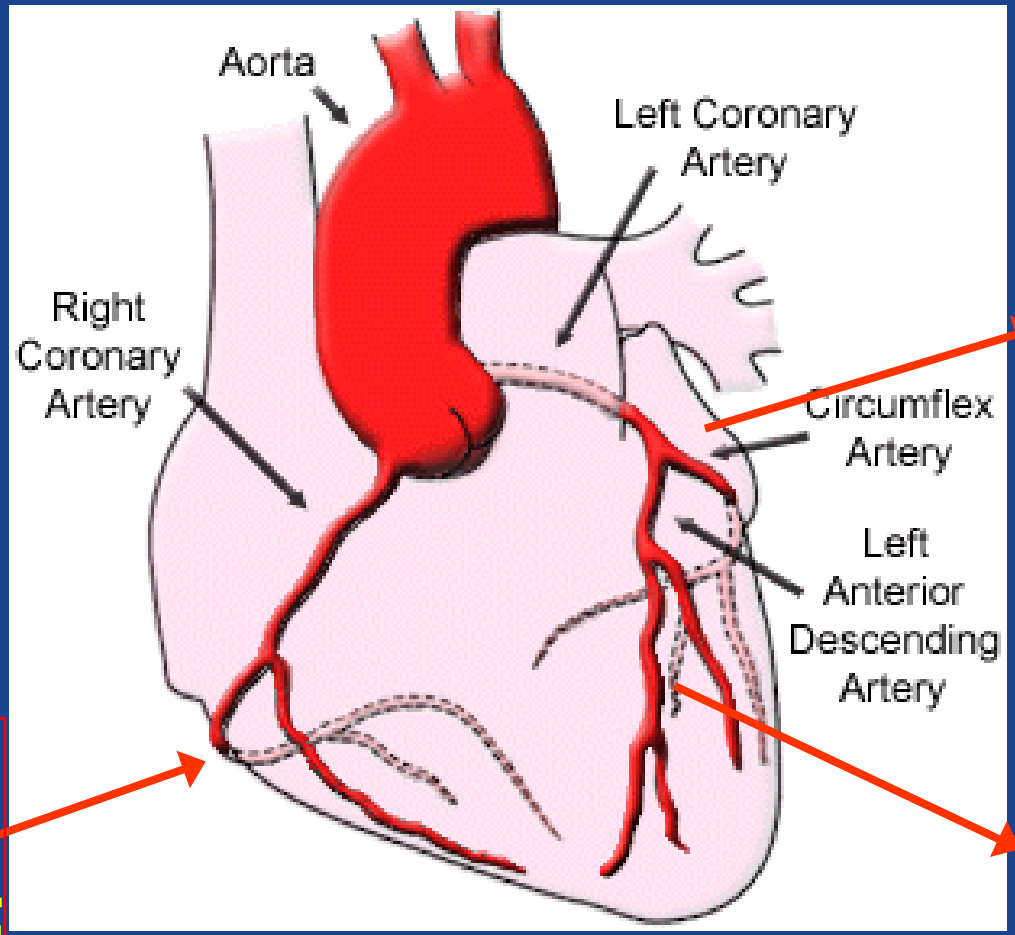
- Troponin T,I: 4-6 Hr
  - last 10-14 days
- CPK:4-6 Hr, peak 17-24hr, normal 72 hr
  - MB(MM,BB)
  - MB2/MB1 >1.5

=> Cardiac markers are detected after a few hours.
=> High-sensitivity cardiac troponin assays are mainly used as results show in 2-3hrs after MI.

=> Troponin lasts for 10-14 days, while CPK lasts for only 72hrs.
=> In MI, positive troponin is a must. If it continues to be negative then there is no MI

Regions of the Myocardium

We can predict which coronary artery is occluded from ECG:

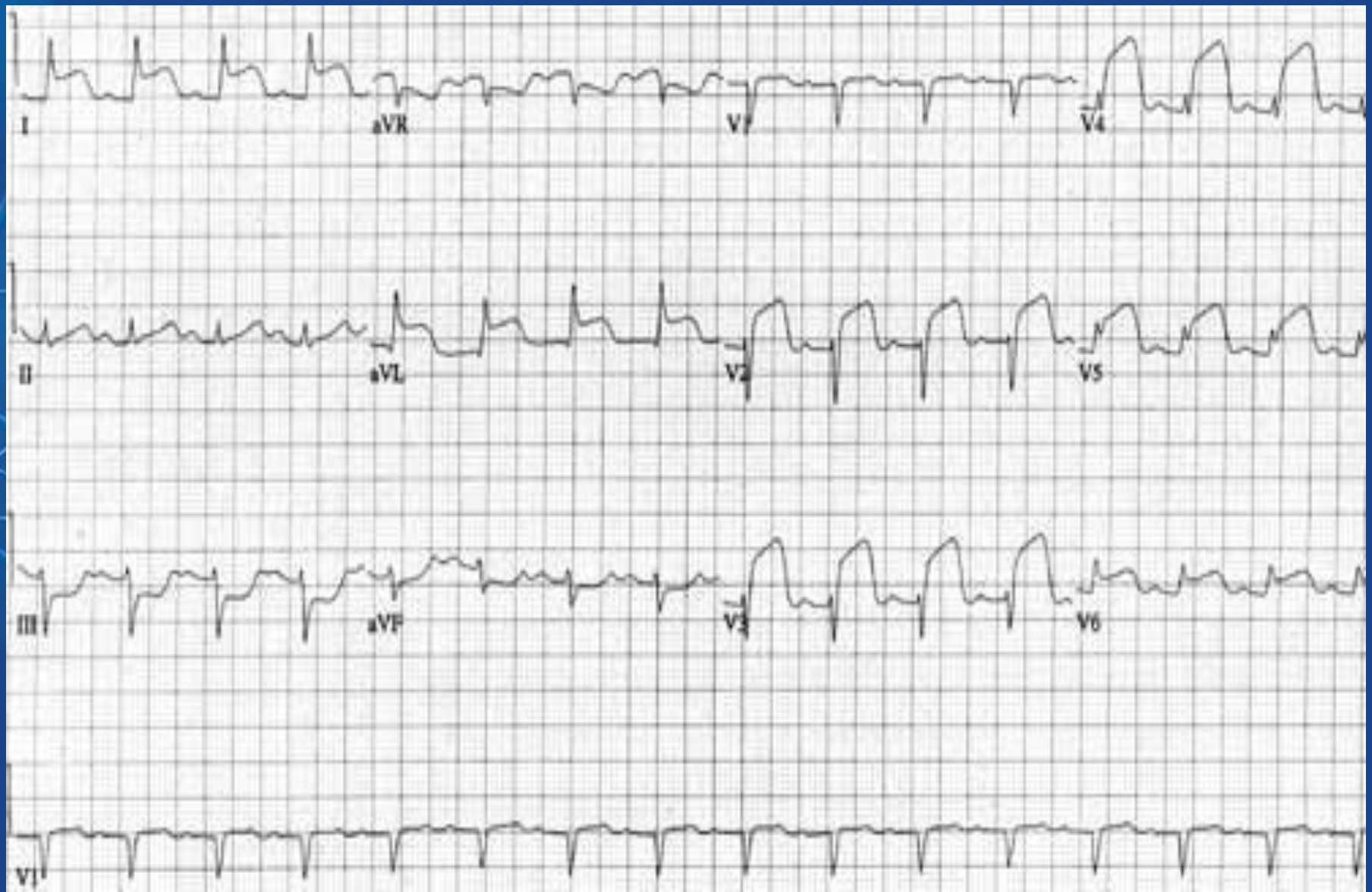


Lateral
I, AVL, V5-V6

Inferior
II, III, aVF

Anterior / Septal
V1-V4

ST Elevation



I couldn't find the slides corresponding for this information (which was mentioned by the doctor), I did my best to try and write it down:

Treatment of IHD (start immediately):

1- Sub-lingual Nitrates.
2- Aspirin.
3- Angioplasty: PTCA, CABG.
4- Reperfusion (most important): using PCI or thrombolytics.

- ⇨ To improve prognosis, by regressing atherosclerosis: any patient with IHD should be on anti-platelets, ACEI/ARBs, or statins.
- ⇨ To improve symptoms: we decrease O2 demand *(decrease contractility by CCBs or wall tension by Nitrates)*, increase O2 supply *(beta-blockers)*, and by revascularization.
- ⇨ Percutaneous Coronary Intervention (PCI) is a non-surgical procedure that uses a catheter to place a stent to open up blood vessels in the heart that have been narrowed.
- ⇨ PCI has a higher success rate than thrombolytics.
- ⇨ If there is no chance to have PCI within 90mins, thrombolytics are given.
- ⇨ The main point to affect MI prognosis is the time until reperfusion is established; the sooner the better.

Complications of MI:

1- Death (20%) without immediate treatment.
2- Heart failure (left-sided): when >25% of the myocardium is lost to infarction.

Heart Failure Treatment:

1- Improve prognosis
   a- ACEI / ARBs: reducing ejection fraction.
   b- Beta-blockers (β-1 selective and mixed α-β blockers): used to be contraindicated
   c- Spironolactone
   d- Digoxin

2- Improve symptoms
   a- Diuretics: does not affect prognosis nor does it decrease the mortality rate.