

# ISCHEMIC HEART DISEASE

## ETIOLOGY AND PATHOPHYSIOLOGY

- *Ischemia* refers to a lack of oxygen due to inadequate perfusion, which results from an imbalance between oxygen supply and demand.
- The most common cause of myocardial ischemia is **atherosclerotic disease** of epicardial coronary arteries.

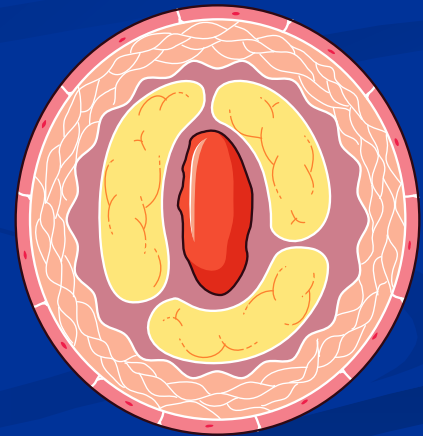
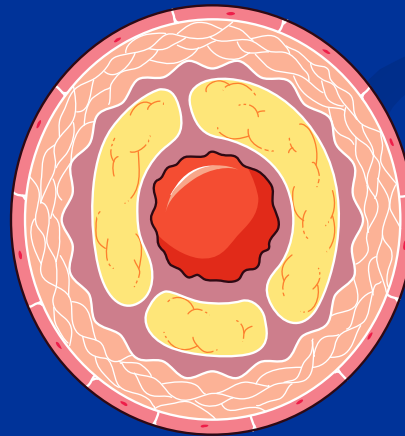
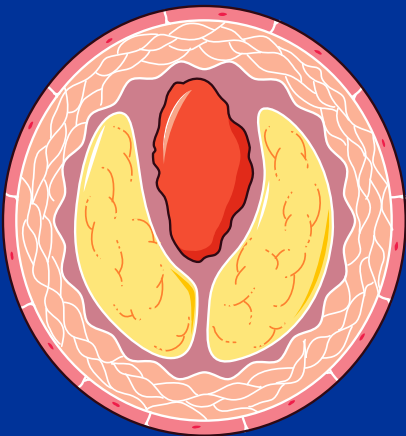
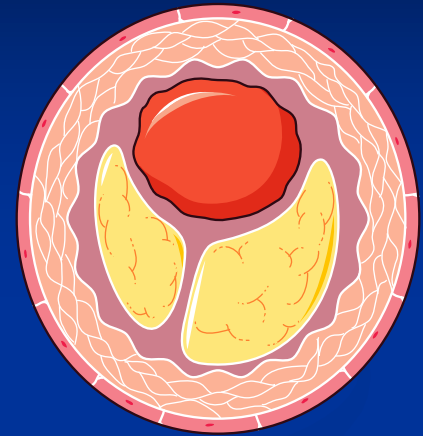
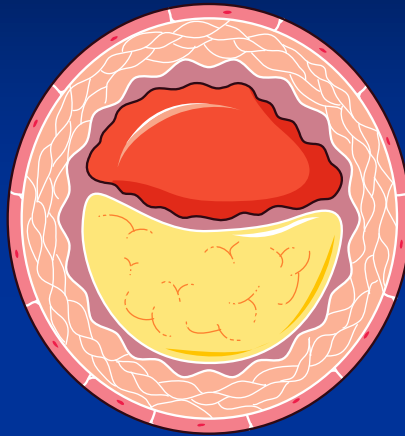
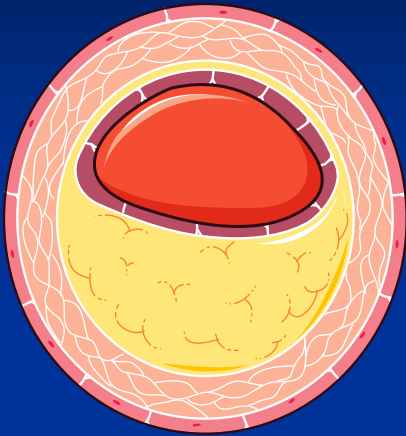
# Ischemic heart disease (IHD)

- is the most common, serious, chronic, life-threatening illness in the Moldova, Europe, United States, where more than 40 million persons have IHD.
- This condition causes more deaths and disability and incurs greater economic costs than any other illness in the developed world.

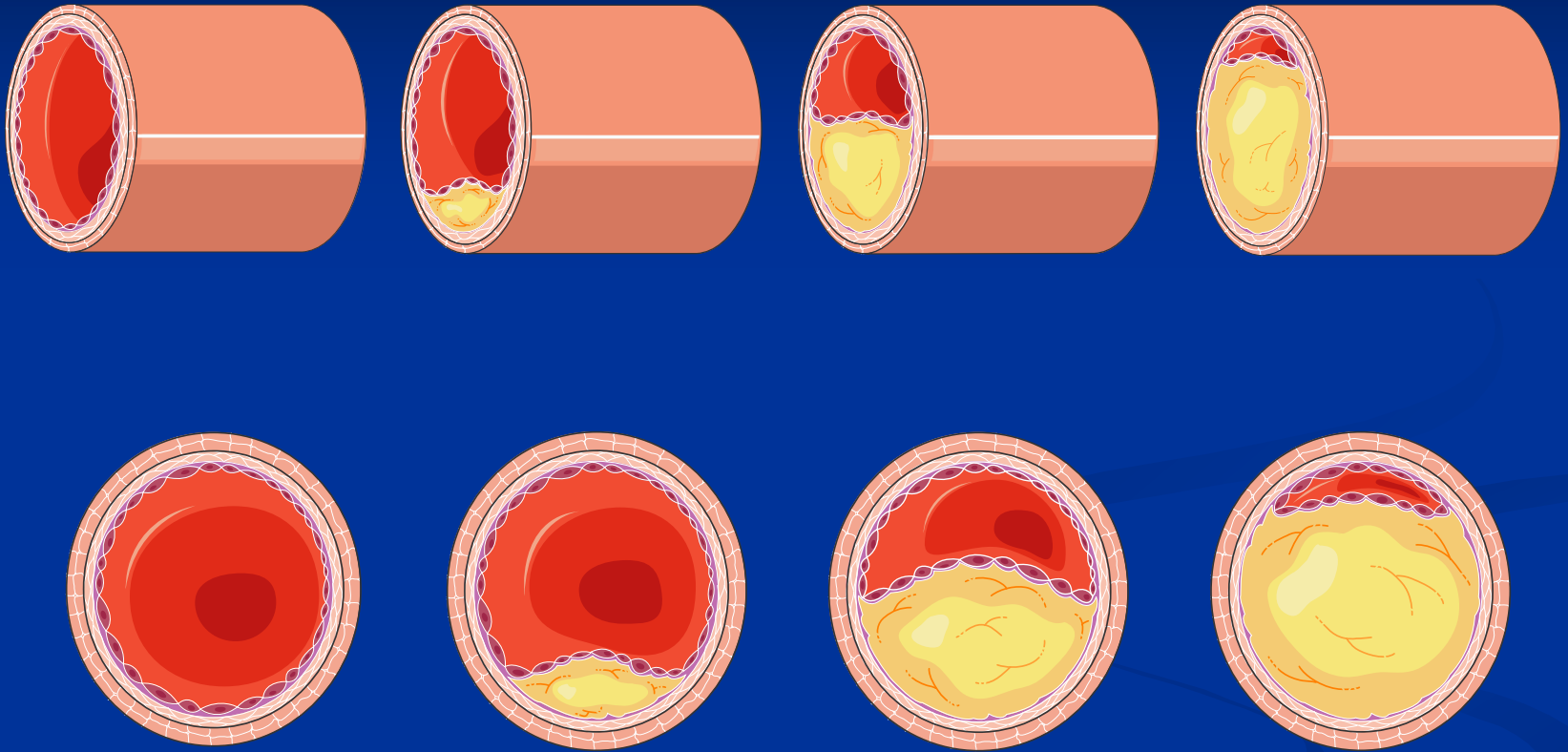
# Causes of IHD

- atherosclerosis - By reducing the lumen of the coronary arteries it reduces myocardial perfusion when the demand for flow is augmented, as occurs during exertion or excitement.
- spasm,
- arterial thrombi,
- rarely, coronary emboli as well as by ostial narrowing due to luetic aortitis.
- Congenital abnormalities,

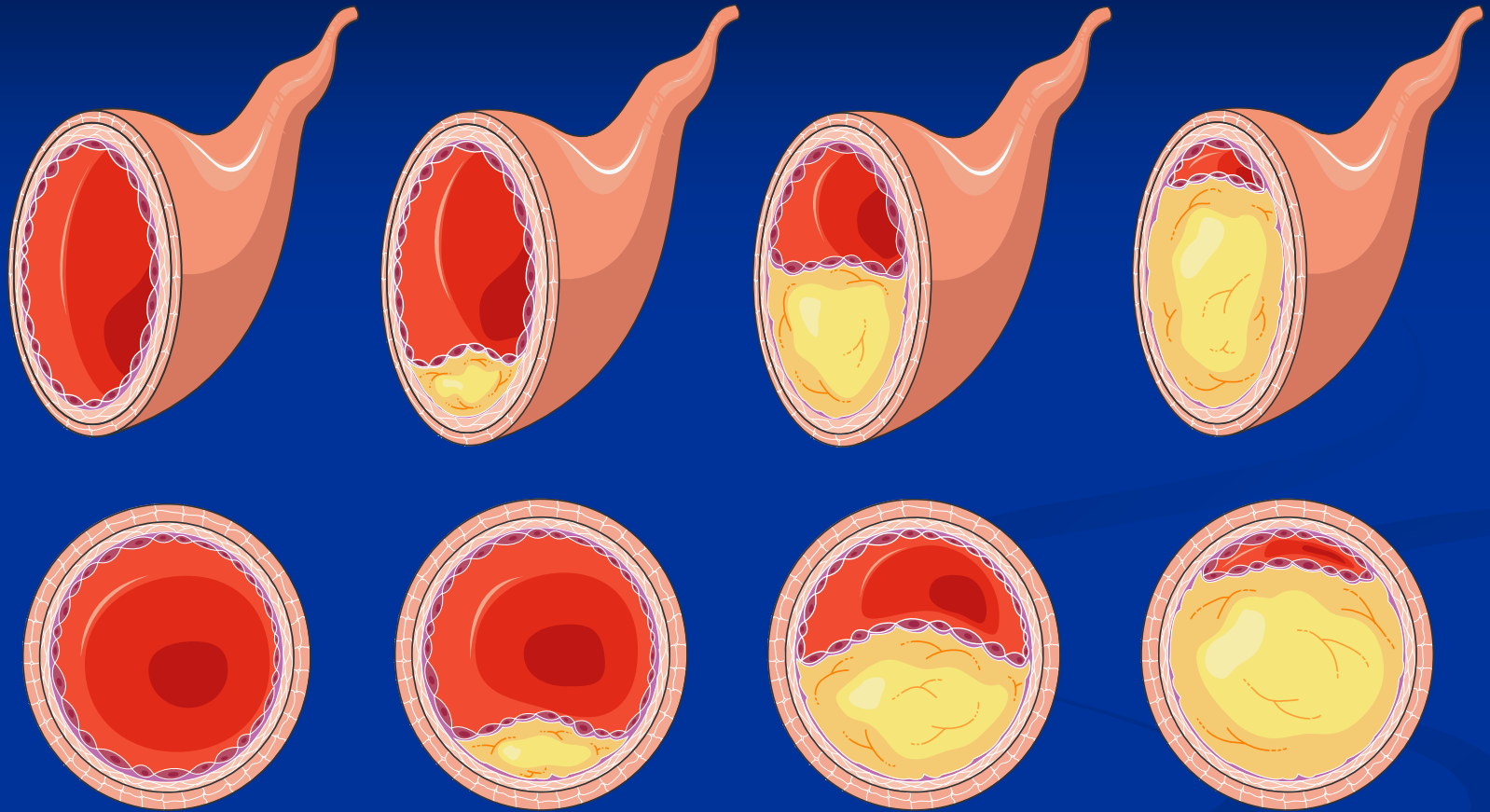
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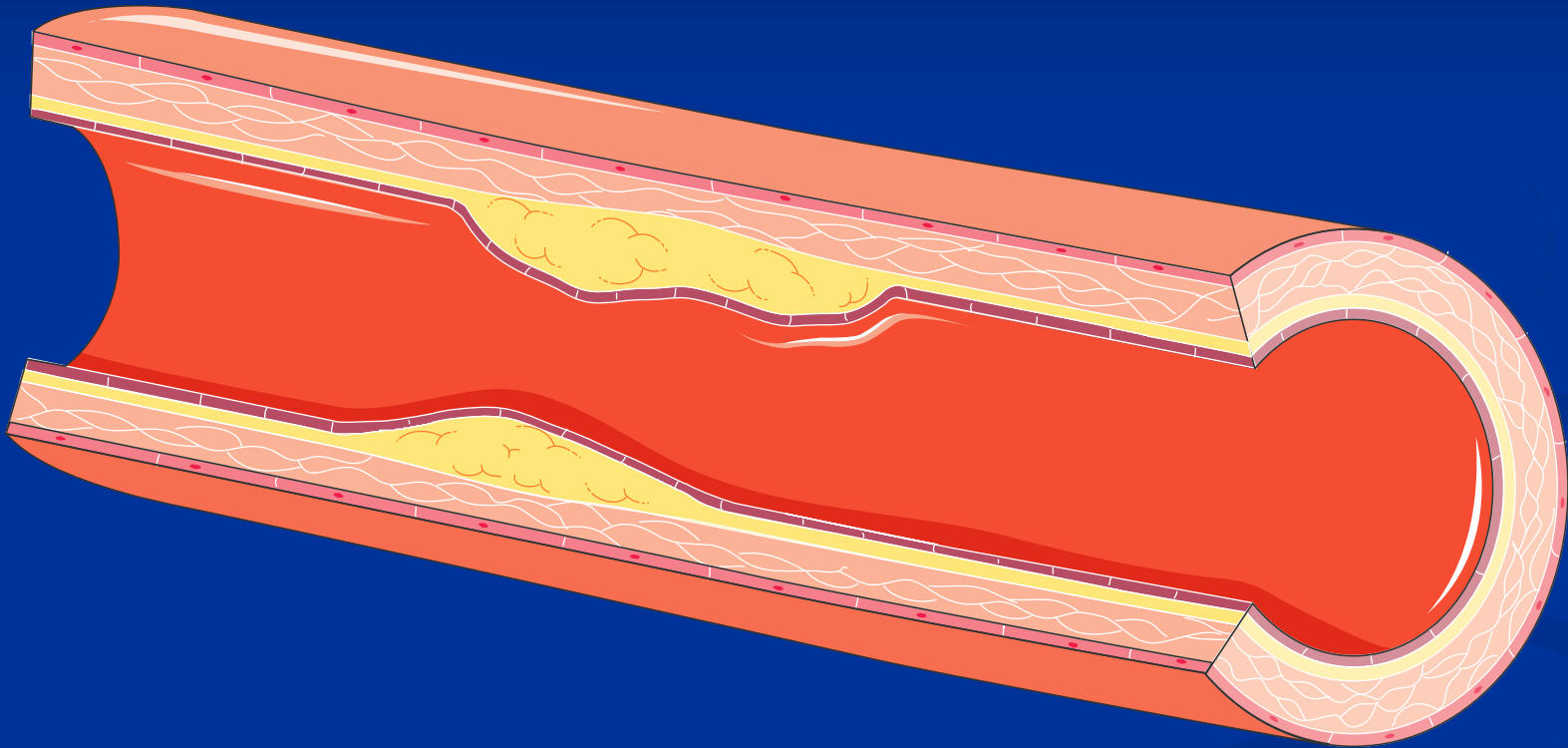
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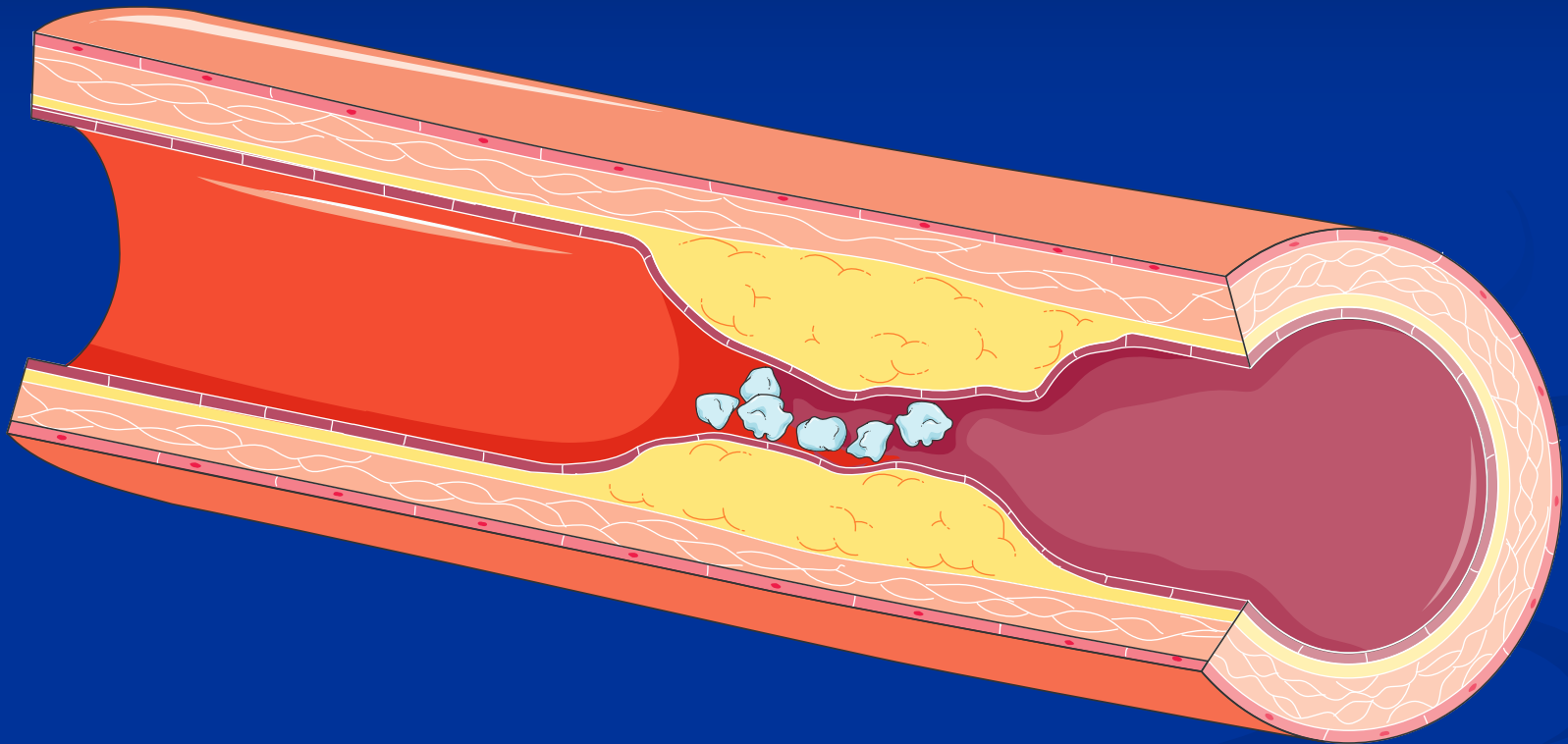
# Atheroma (4)



# From Atheroma To Thrombus (1)

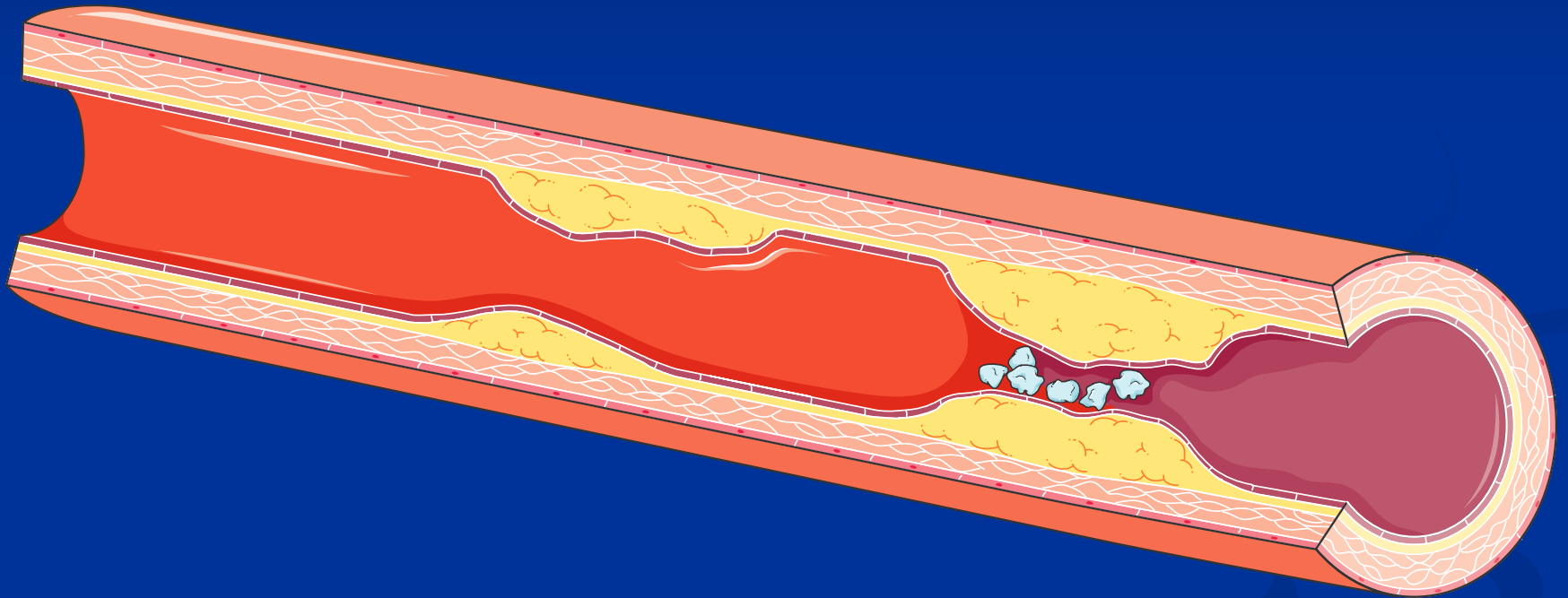


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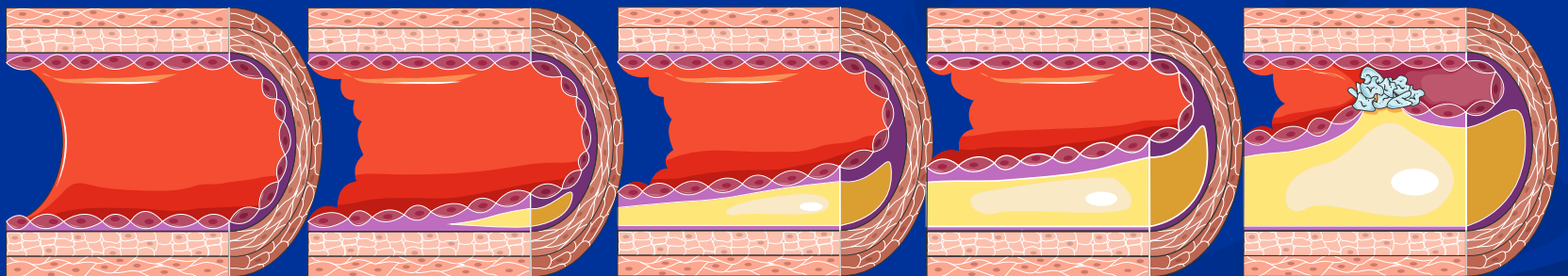
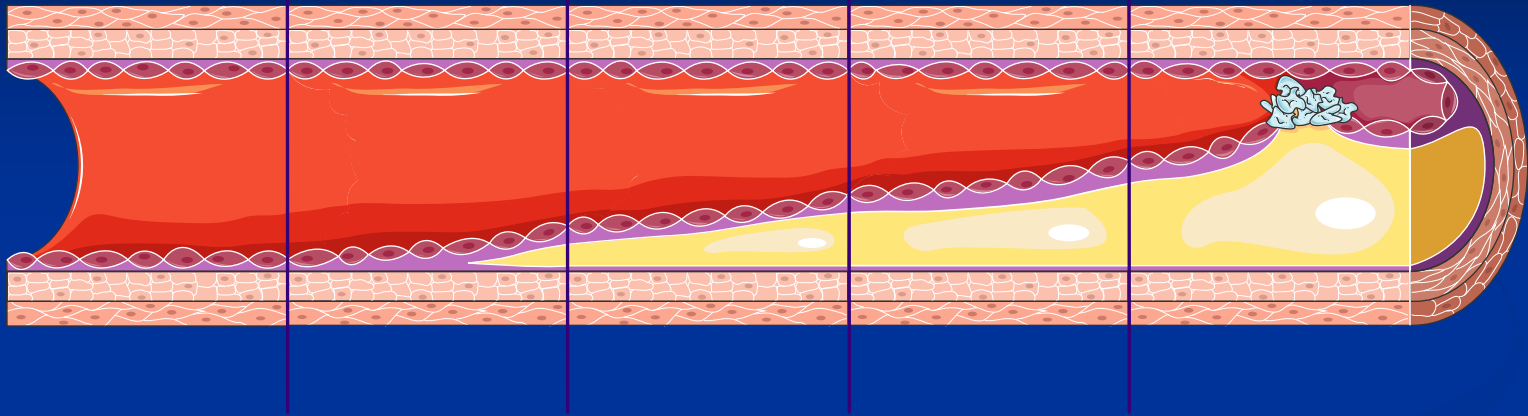




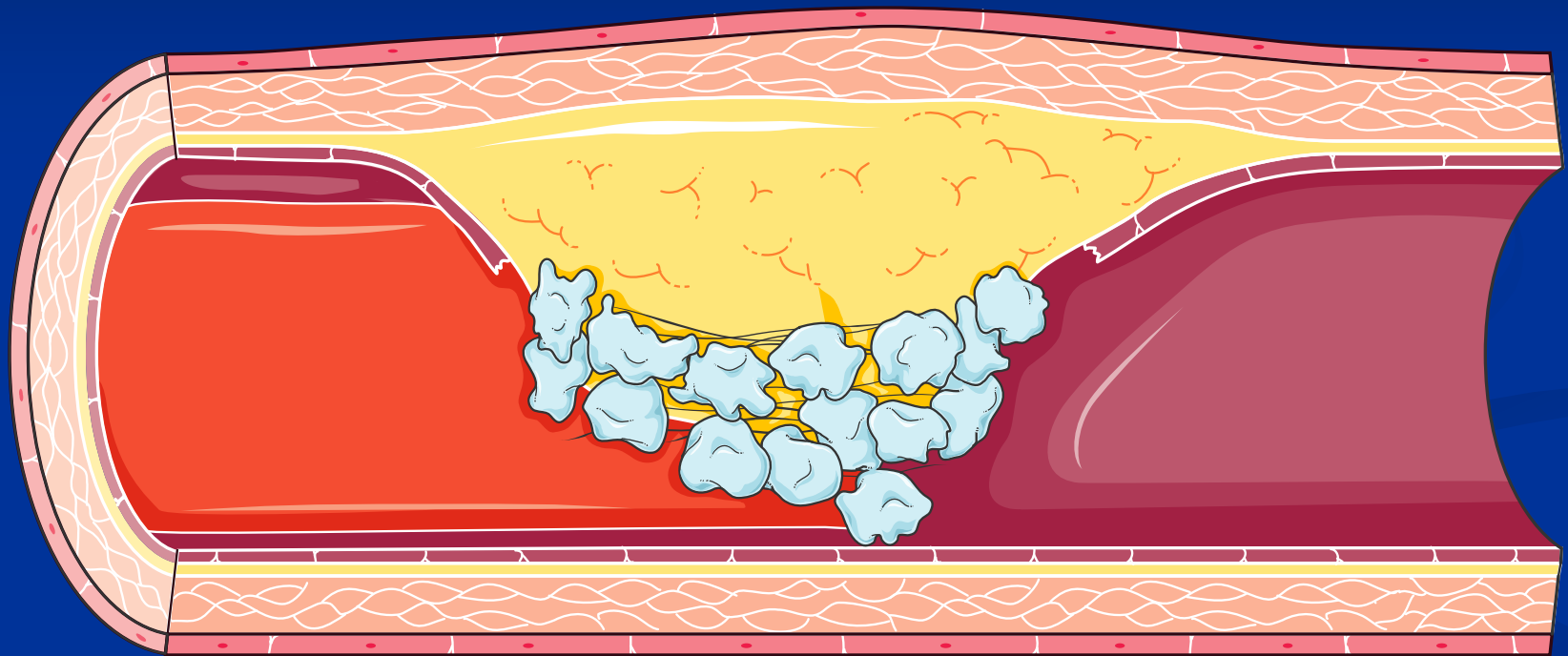
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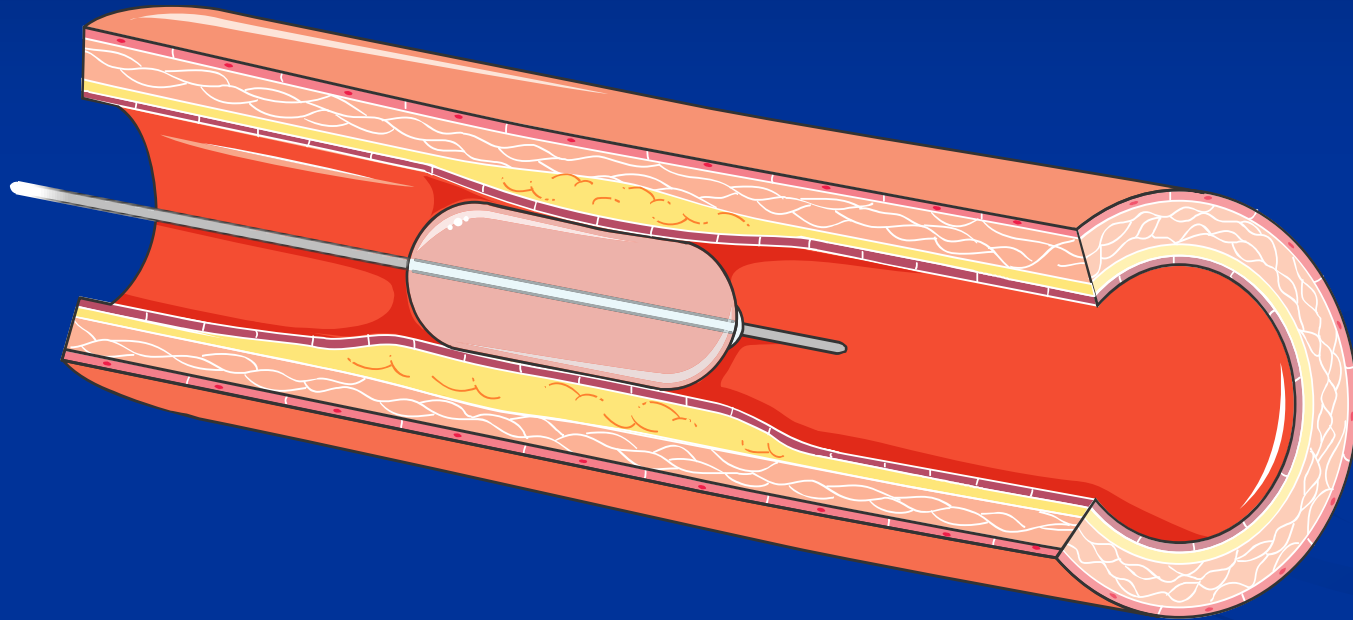
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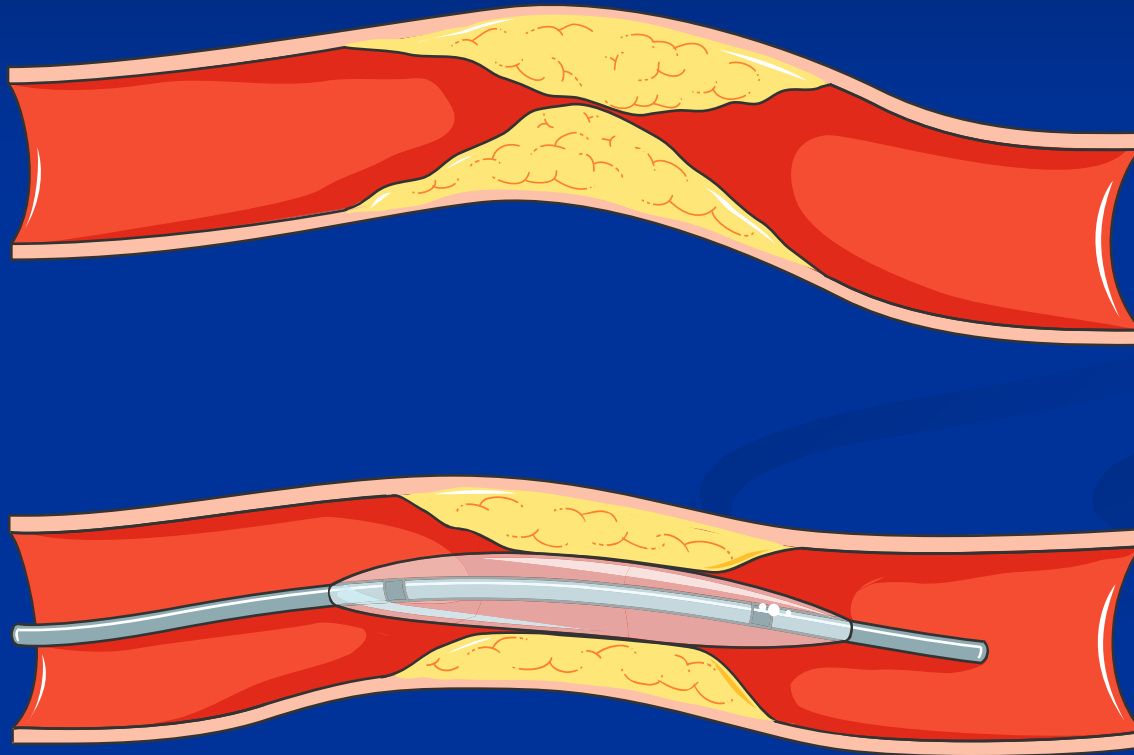
# Atherothrombosis



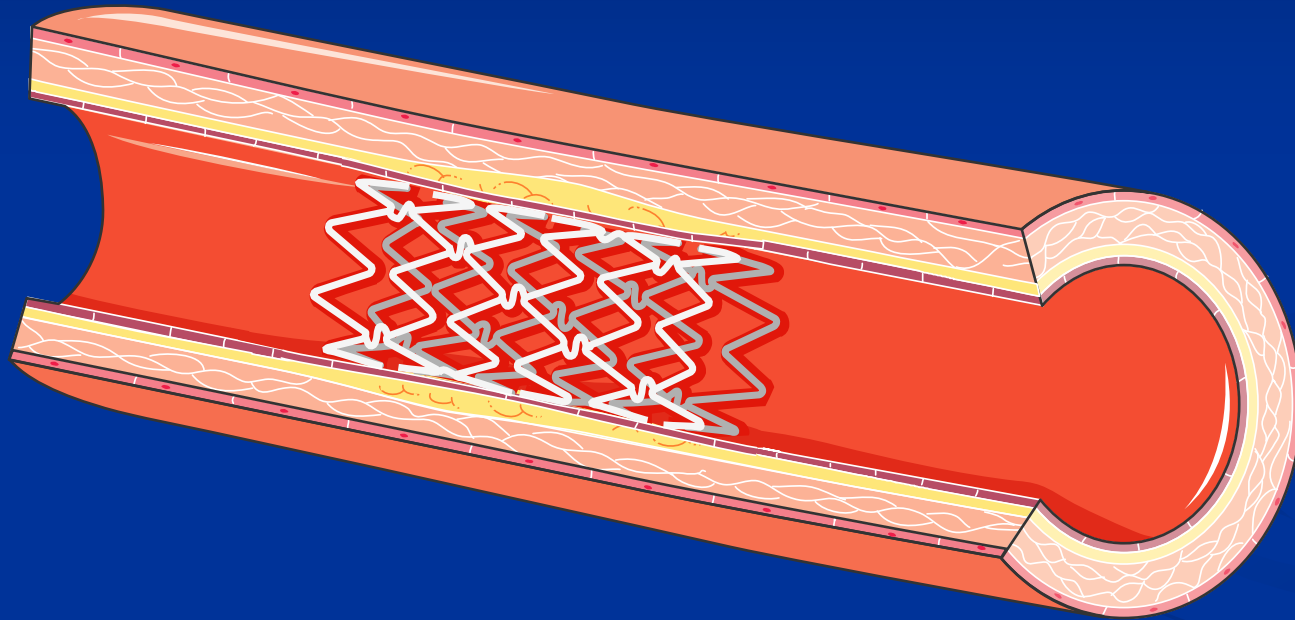
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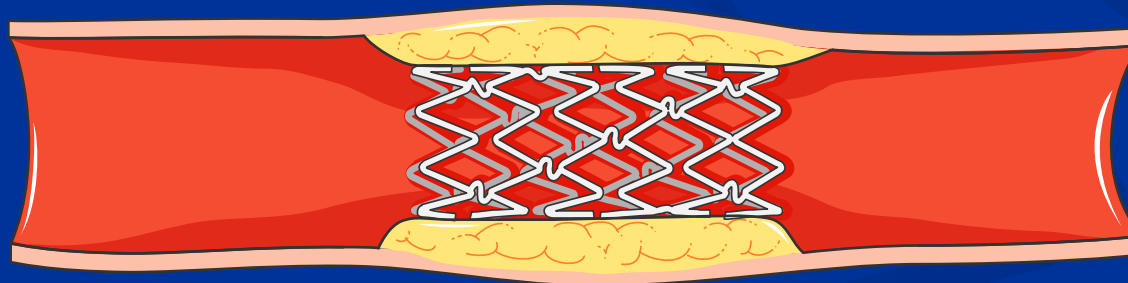
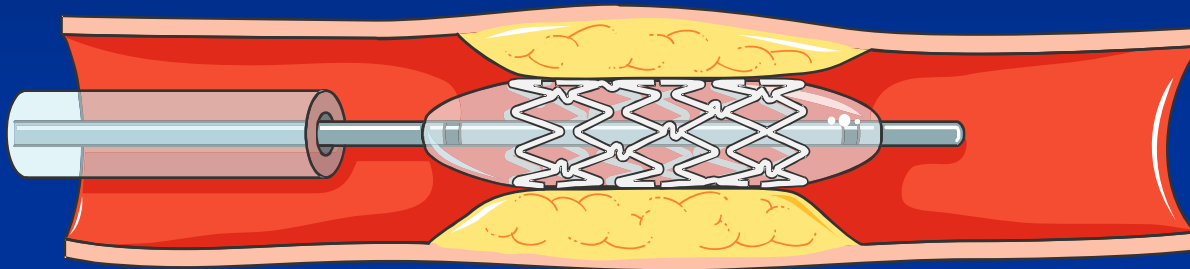
# Angioplasty (2)



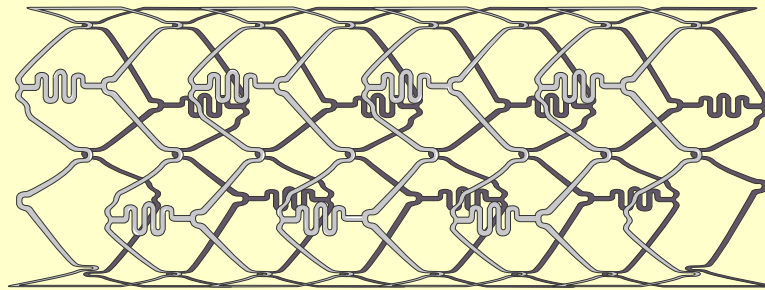
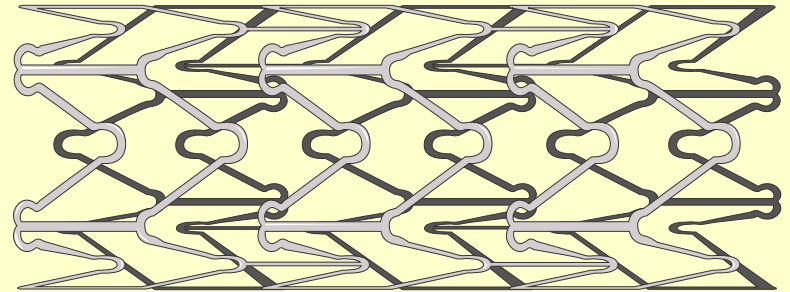
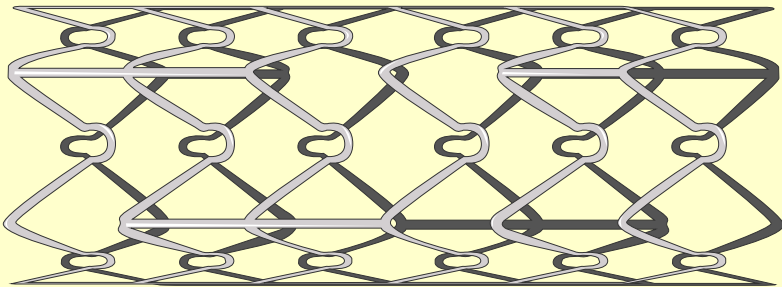
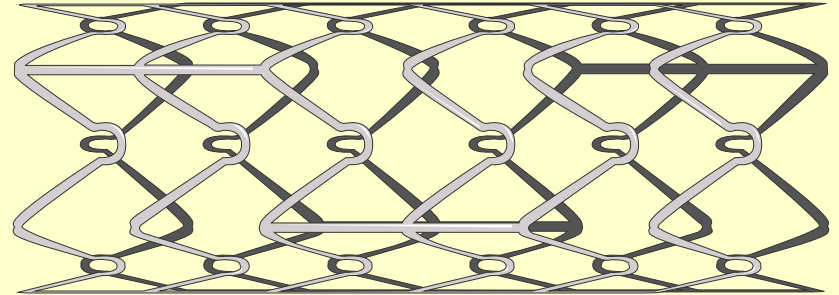
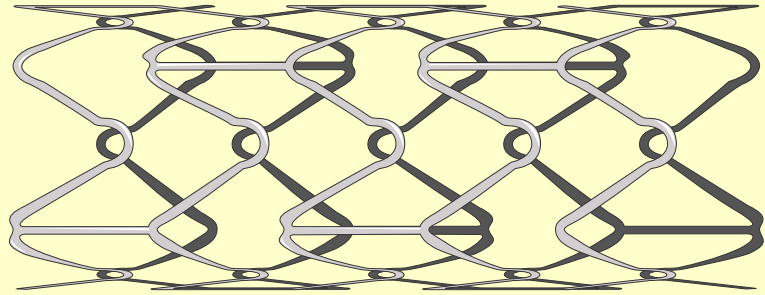
# Stent (1)



# Stent (2)

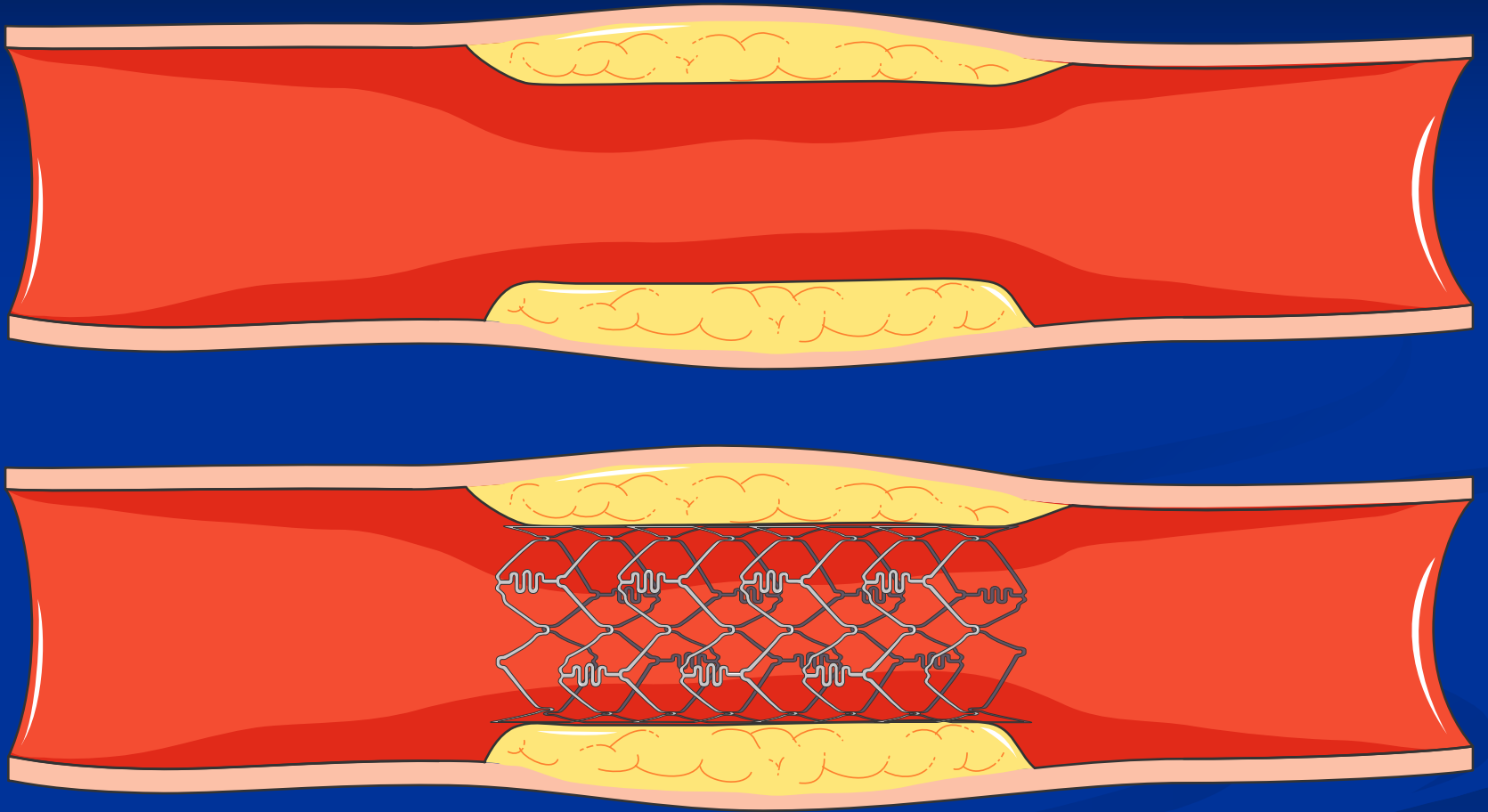


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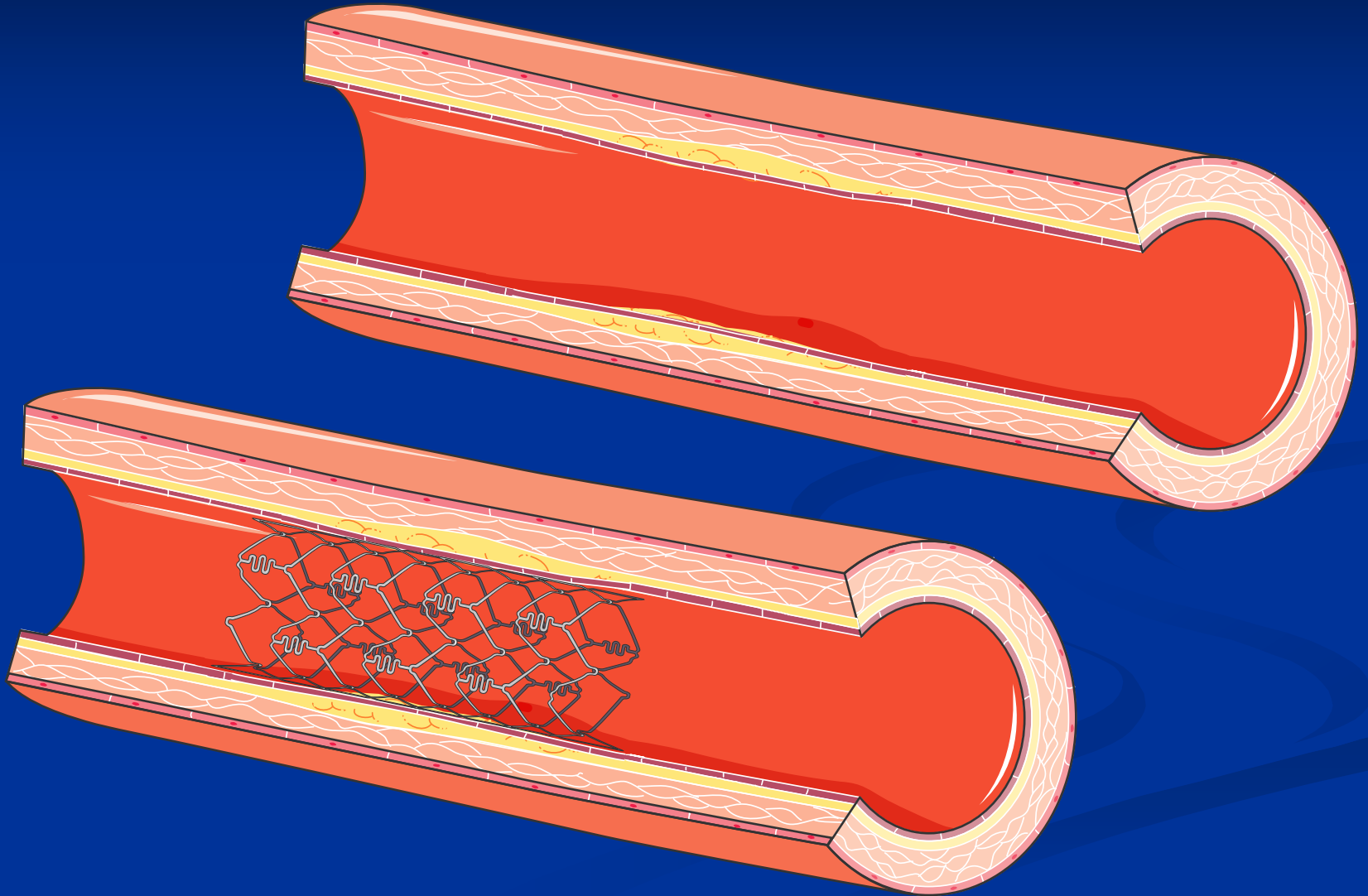




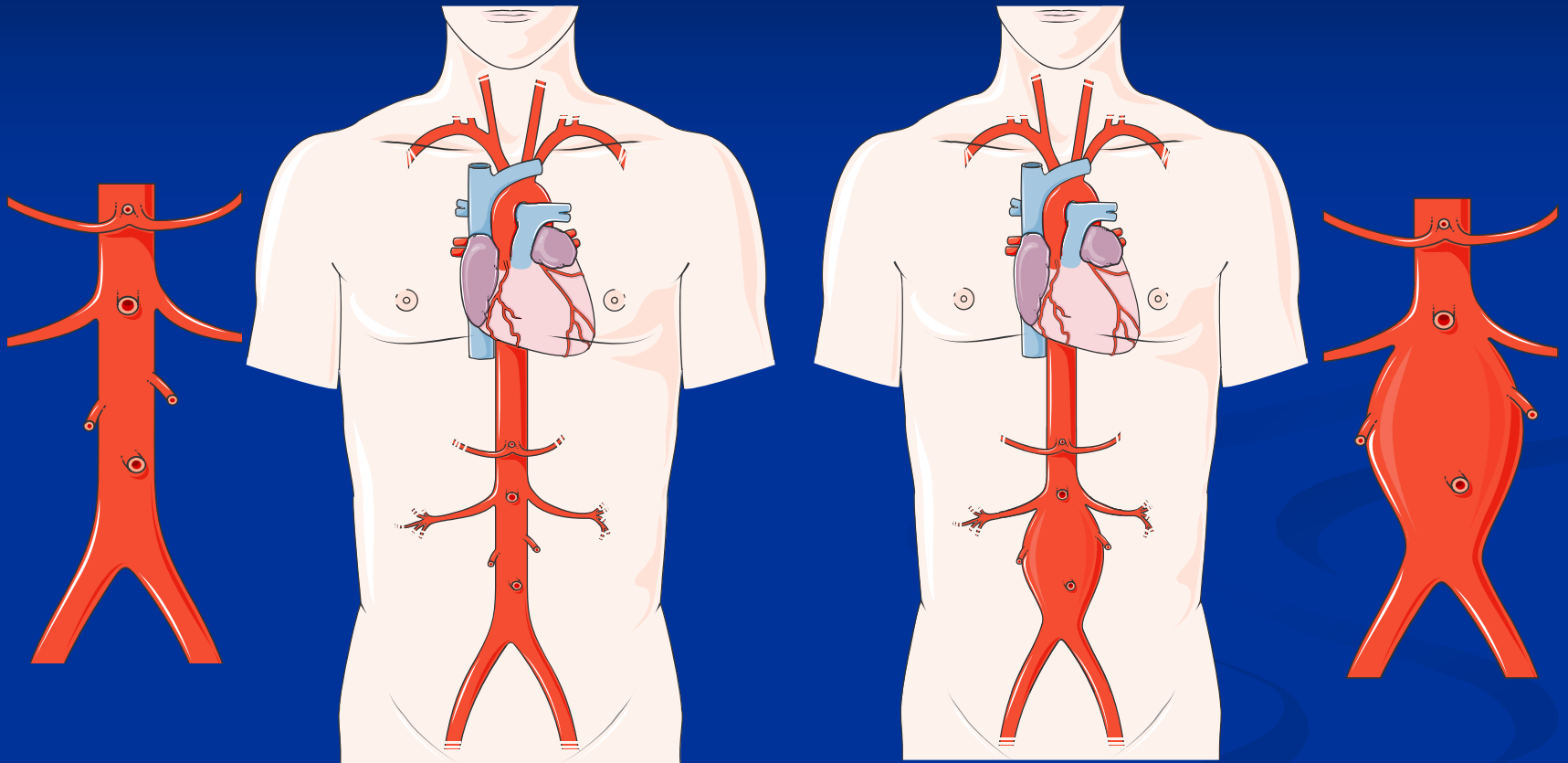
# Stent (4)



# Stent (5)



# Abdominal Aortic Aneurysm



- Myocardial ischemia can also occur if myocardial oxygen demands are markedly increased, as in severe ventricular hypertrophy due to aortic stenosis.

- the large epicardial coronary arteries are capable of constriction and relaxation, in healthy persons they are referred to *conductance vessels*,
- the intramyocardial arterioles normally exhibit striking changes in tone and are therefore referred to as *resistance vessels*.
- Abnormal constriction or failure of normal dilation of the coronary resistance vessels can also cause ischemia. When it causes angina this condition is referred to as *microvascular angina- X syndrome*.

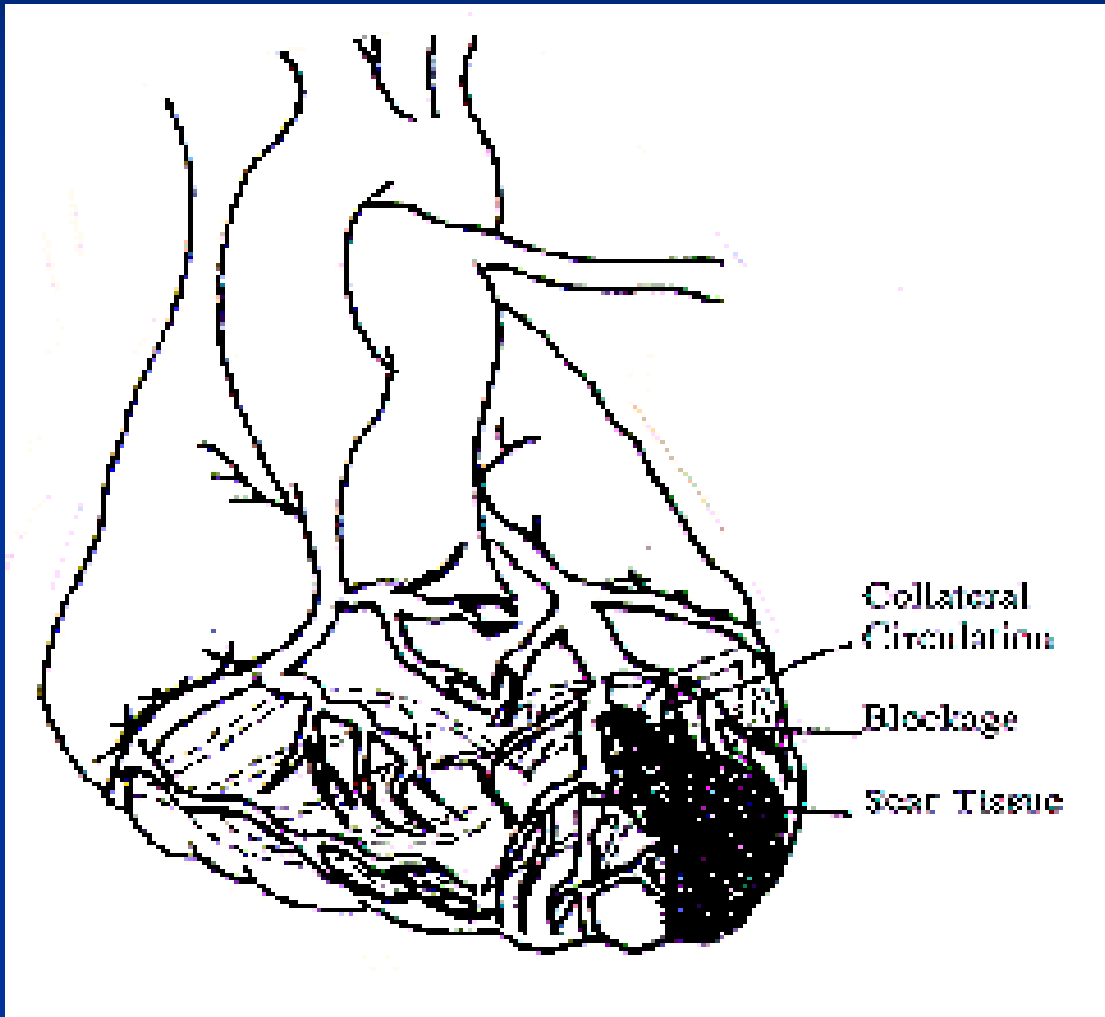
*Risk factor* is something that increases your chance of getting a disease or condition. IHD:

- Sex: Male
- Age: 45 and older for men; 55 - for women
- Heredity
- Diabetes
- Obesity and overweight
- Smoking

- High blood pressure
- High blood cholesterol  
(specifically, high LDL cholesterol, and low HDL cholesterol)
- Sedentary lifestyle
- Stress
- Excessive alcohol use

- Atherosclerosis is characterized by an abnormal thickening and hardening of the walls of arteries, with a resulting loss of elasticity.





# Classification of the IHD (WHO)

1. Sudden coronary death

2. Angina pectoris

- Stable angina pectoris

- Unstable angina pectoris

- Angina pectoris *de novo*

- Prinzmetall angina pectoris

- Progressive angina pectoris

# Classification of the IHD (WHO)

## 3. Myocardial infarction

- Acute

■ Transmural MI

■ Intramural MI (subendocardial, subepicardial)

- Chronic (old MI)

## 4. Cardiac failure in IHD

## 5. Arrhythmias

# 1. Sudden death

- - is a coronary death in 2 hours that happens suddenly, without serious previous cardiac diseases.
- *Etiology of Sudden death* - usually the course of it is ventricular tachy-arrhythmias.

## 2. Angina pectoris

- - its main clinical symptoms are attacks of retrosternal pain due to acute but transient disorder in the coronary circulation - Ischemia

### *Etiology of Angina pectoris*

- the atherosclerosis of the coronary arteries of the heart
- coronary spasms.

# Angiography

- is the most commonly used diagnostic tool used to view blood vessels.
- contrast material is injected into the coronary arteries, to diagnose blockages in the blood vessels



# *Clinical picture*

## Symptoms

Angina - intermittent chest pain that often has a squeezing or pressure-like feeling, which may radiate into the shoulder(s), arm(s), etc.

Angina usually lasts for about 2 to 10 minutes, not more than 20 min, and is often relieved with rest.

# Angina can be triggered by:

- Exercise or exertion
- Emotional stress
- Cold weather
- A large meal



- If angina is unrelieved by rest or nitroglycerin, is severe, begins at rest (with no activity), or lasts more than 20 minutes, these are warning signs of unstable angina or a heart attack.

# Accompanying symptoms may include:

- Shortness of breath
- Sweating
- Nausea
- Weakness

# Objective examination

- During the attack, the pulse is usually slow, rhythmical, may be tachycardia, slightly increasing of the arterial pressure.
- Palpation, percussion and auscultation cannot reveal any abnormalities.

# Instrumental and laboratory examinations

Tests may include:

1. Blood Tests - to look for certain substances in the blood; helps the doctor determine if you are having angina or an acute myocardial infarction .
  - Blood tests during a myocardial infarction will show elevated Creatinine kinase (CK), Troponin level, or LDL level, AsT level.

## 2. Electrocardiogram (ECG, EKG)

- EKG shows characteristic abnormalities of ST segment or T waves:
  - ST elevations  $> 1$  mm or depression of the ST segment  $> 1$  mm (below isoelectrical line)
  - T wave inversions (negative T wave) or T wave may be - "high, positive, sharp"
  - It may also show evidence of an old heart attack, i.e. Q waves or poor R wave progression.
  - When the attack is abated the ECG soon normalizes.

3. Echocardiogram - uses high-frequency sound waves to examine the size, shape, and motion of the heart; gives information about the structure and function of the heart
4. Tests with physical effort:
  - cycloergometria - physical effort on bicycle with permanently taking the ECG
  - treadmill - running road with permanently taking the ECG

5. Exercise Stress Test - records the heart's electrical activity during increased physical activity
6. **Holter monitorization** - is a method of taking ECG during 3-6-24 hours, when electrodes are placed on the patient's body and he is acting as usual.

- 7. Stress Echocardiogram -- this is where stress treadmill is combined with Echocardiogram before and after exercise. It is also about 90% sensitive for detecting heart disease.
- 8. Thallium Stress Test - thallium is used to scan the myocardium, the muscle layer of the heart
- 9. Nuclear Scanning - radioactive material is injected into a vein and observed as it is absorbed by the heart muscle



- 10. Electron-beam CT Scan - a type of x-ray that uses a computer to make pictures of the inside of the heart
- 11. Coronary Angiography -
- 12. **Cardiac catheterization** -- this is the gold standard, allows for exact determination of blockages. Heart function and valve function may also be assessed at the same time.

# Preventive Blood Testing

- Lipid profile (this includes total cholesterol, LDL, HDL, and triglyceride levels)
  - Homocystine level -- this blood test seems to correlate with increased reactivity of the lining of the artery walls to atherosclerosis formation
  - C-reactive protein -- this is an inflammatory marker. This is useful for predicting which patients will respond to aspirin as a preventative in the case of an ischemic event, such as a heart attack.

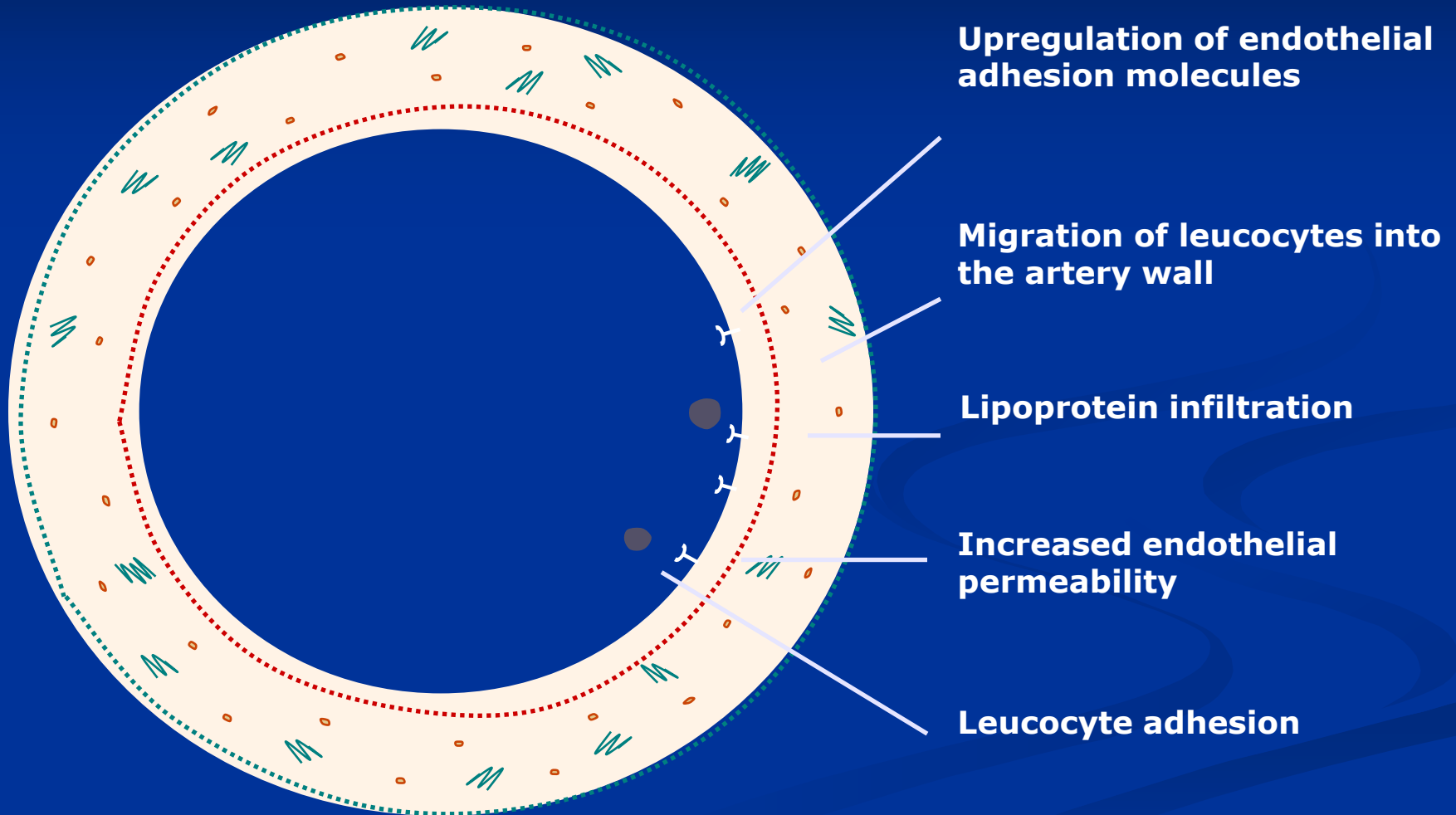
### 3. Acute myocardial infarction (AMI).

- is formation of a necrotic focus in the heart muscle due to upset coronary circulation.
- In survived patients necrosis is substituted in several weeks by a scar.

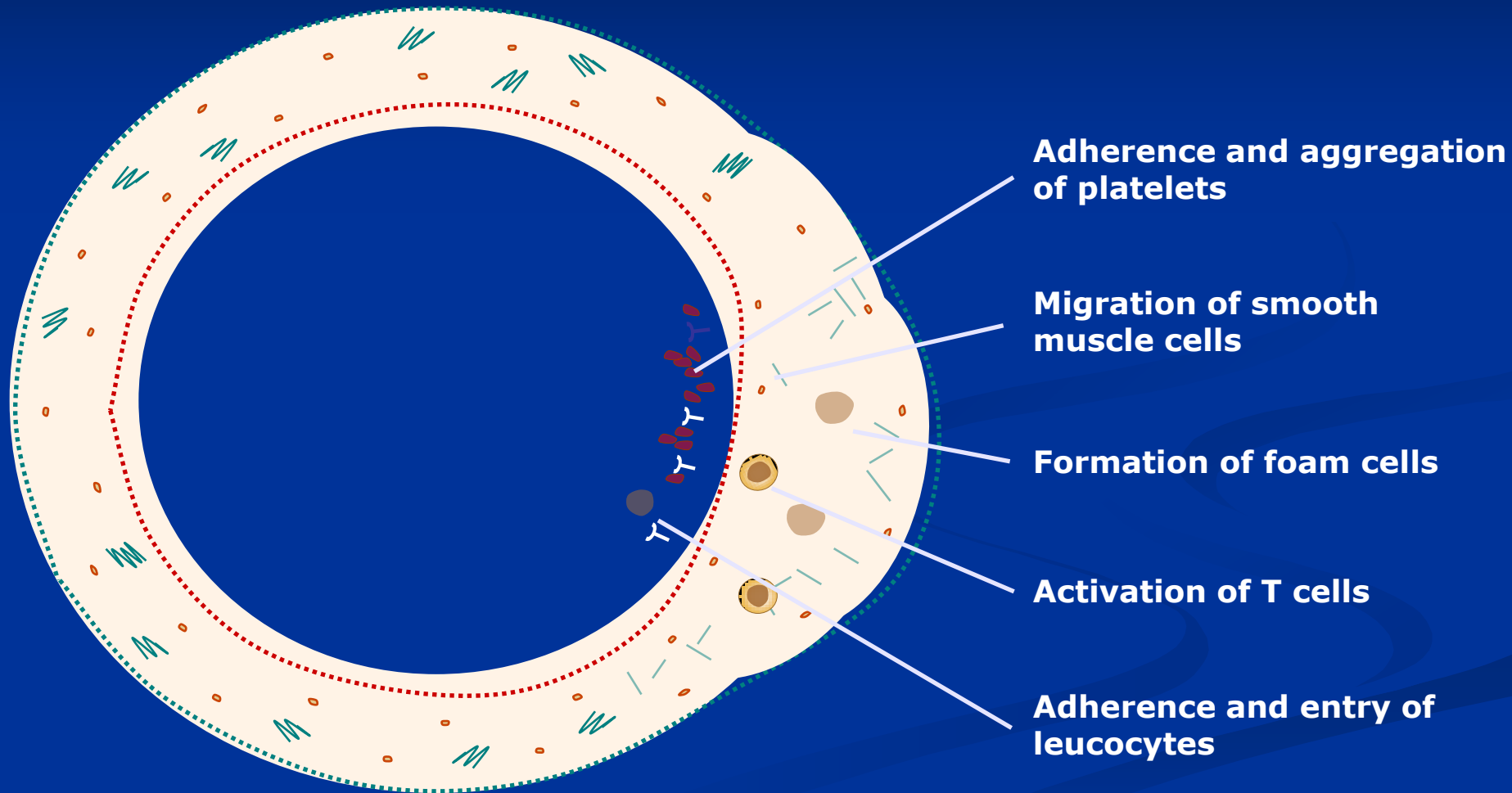
## Etiology

- One of the main causes of the AMI (90-95%) is the rupture of the atherosclerotic plaque with the following thrombosis of the vessel, sometimes and spasm

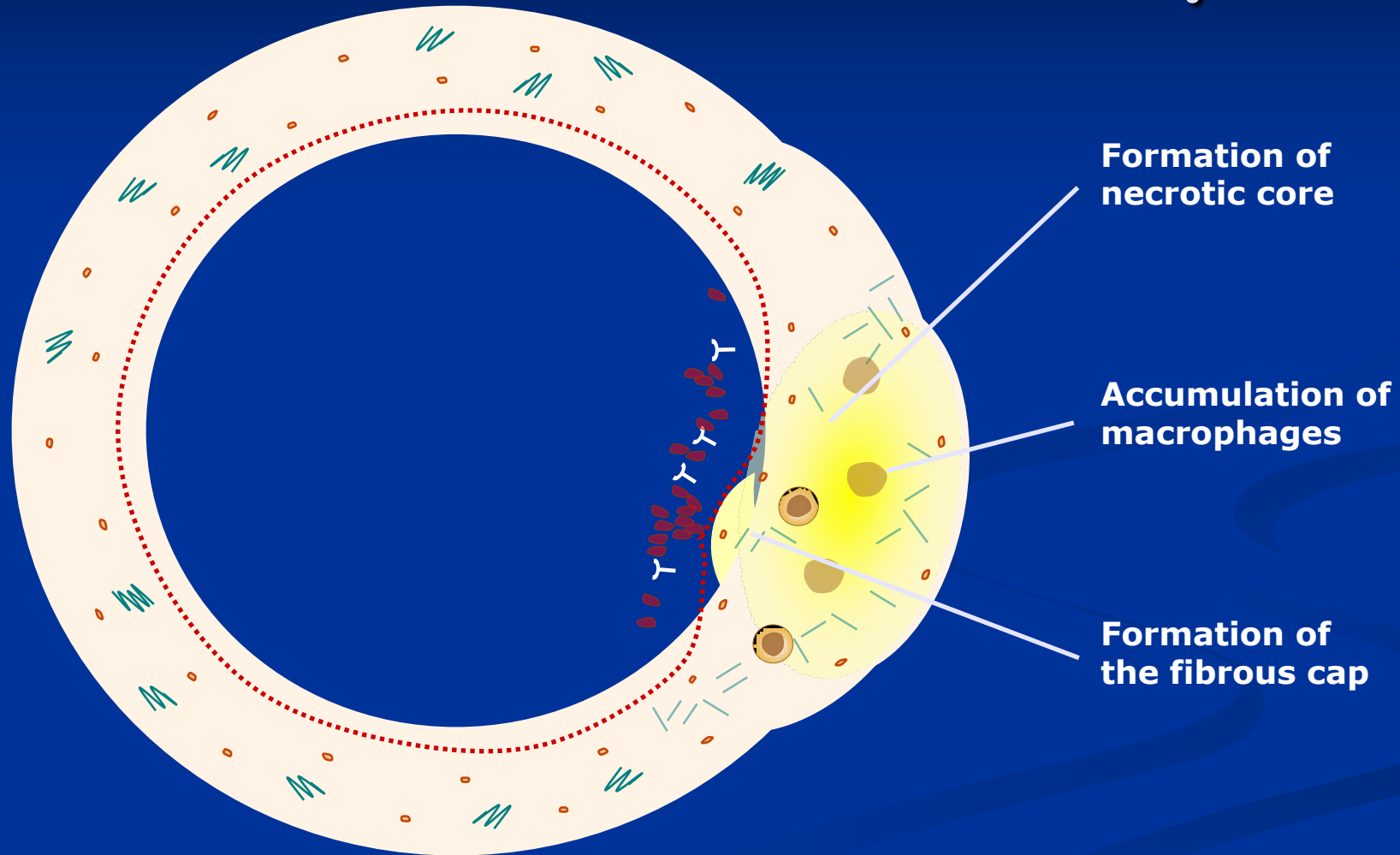
# Endothelial Dysfunction in Atherosclerosis



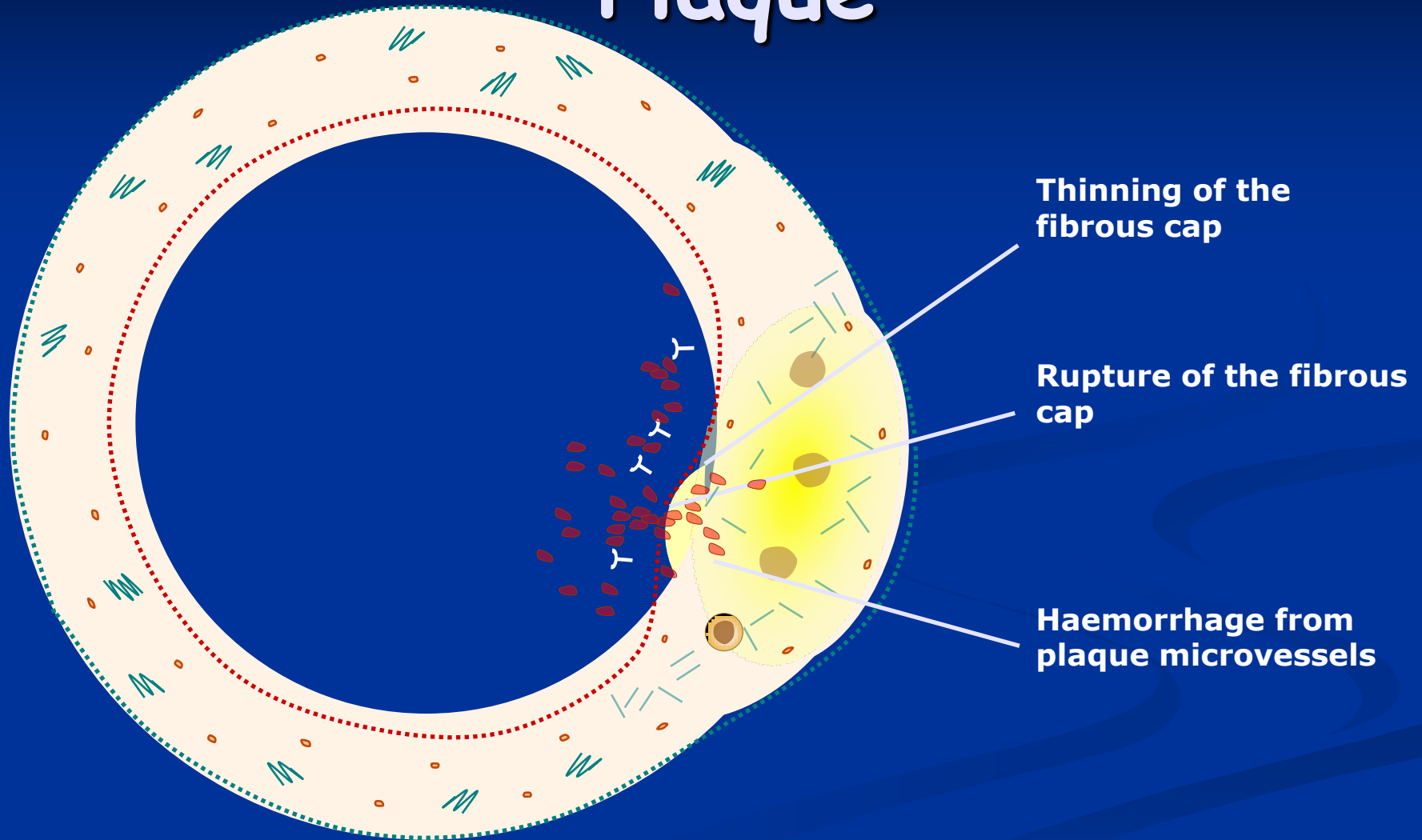
# Fatty Streak Formation in Atherosclerosis



# Formation of the Complicated Atherosclerotic Plaque

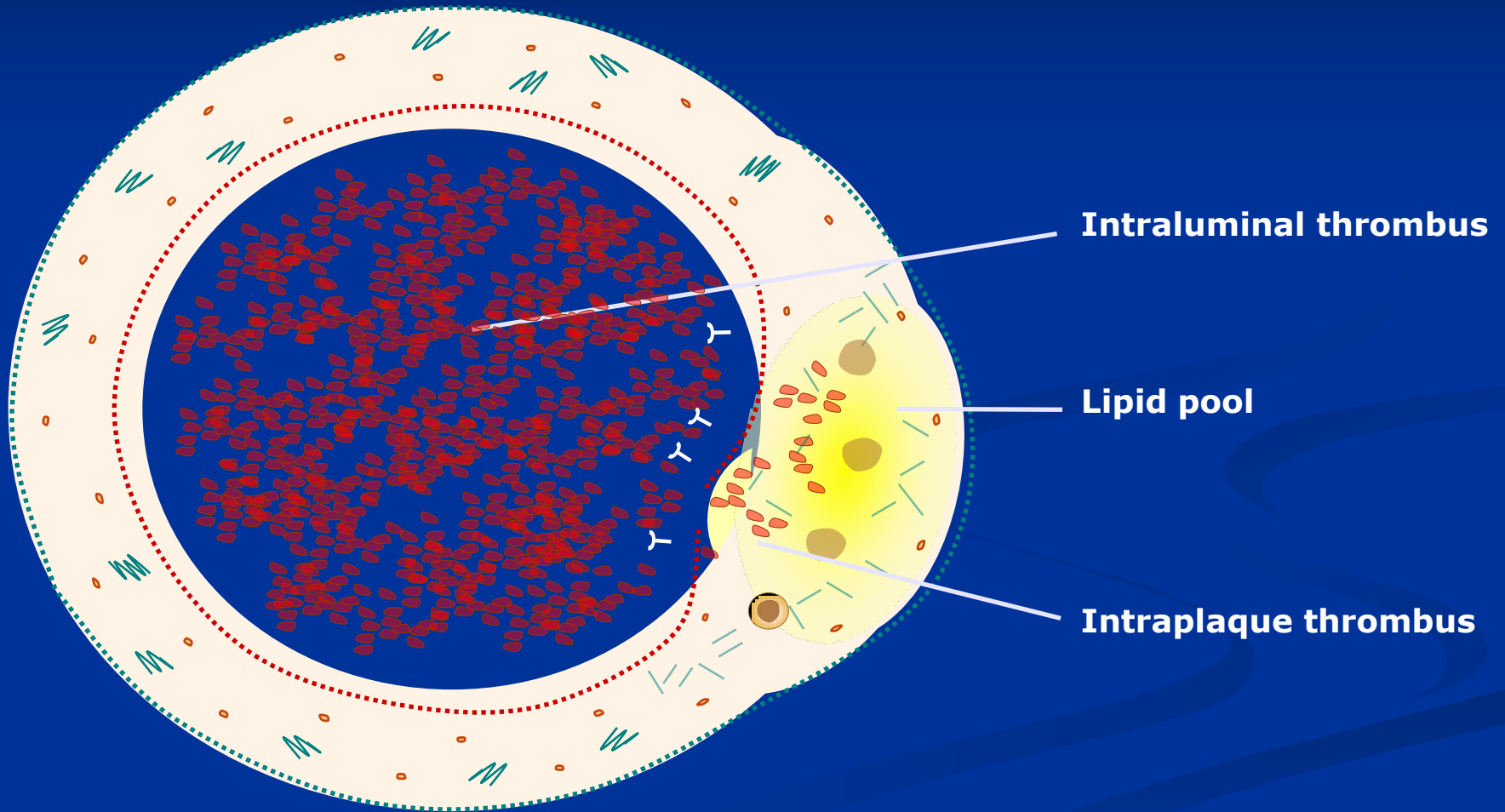


# The Unstable Atherosclerotic Plaque





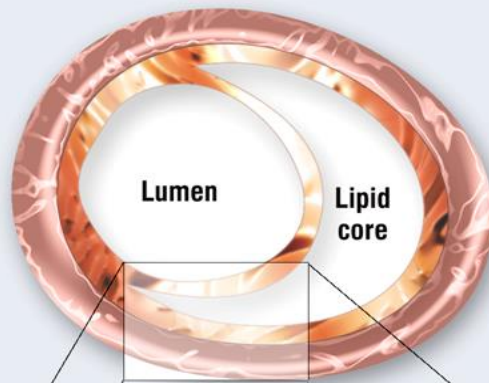
# Atherosclerotic Plaque Rupture and Thrombus Formation



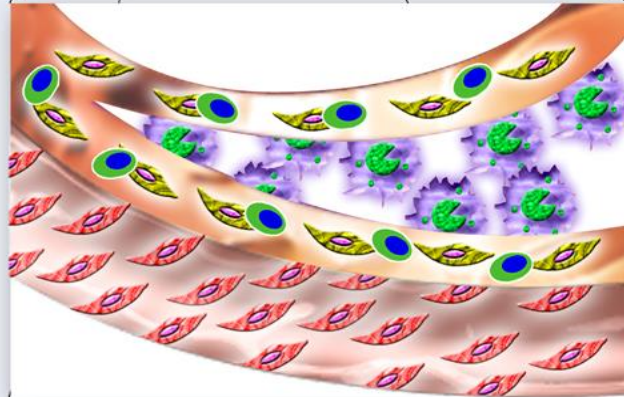
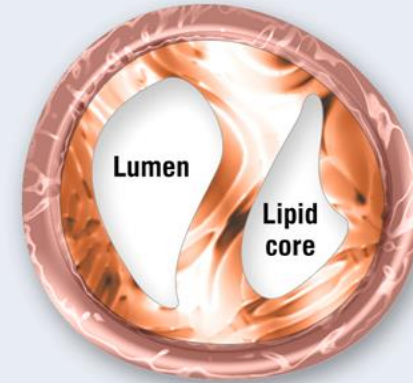
# The Vulnerable Atherosclerotic Plaque



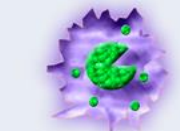

## Characteristics of vulnerable plaques

'Vulnerable' plaque



'Stable' plaque



-  - 'Activated' intimal SMC (HDL-DR<sup>+</sup>)
-  - Normal medial SMC
-  - Macrophage foam cell (tissue factor<sup>+</sup>)
-  - T Lymphocyte

SMC – smooth muscle cell

HDL-DR – transplantation antigen indicating 'activation' of SMCs

The diagnosis of AMI is based on 2 from 3 signs:

- characteristic clinical picture
- characteristic ECG changes
- Characteristic blood serum analysis.

# Clinical picture.

## Typical form of AMI:

- anginous form ( pain lasts more than 30 min)

## Atypical forms of AMI:

- Asthmatic form - like an hit of bronchial asthma
- Abdominal form - like an acute abdomen, with a dyspeptic syndrome (often in old people)
- Cerebral form - like the symptoms in stroke
- Arrhythmic form - it begins with arrhythmias.

- As distinct from angina pectoris, pain in myocardial infarction is not removed by nitroglycerin and persists for a longer time (from 30 - 60 min to several hours).
- Sometimes AMI may begin with a sudden heart failure or collapse, various disorders in the cardiac rhythm or heart block, while the pain syndrome is absent or weak.

# *Cardiovascular system.*

- Examination reveals enlargement of cardiac dullness and low auscultation sounds.
- Sometimes can be heard gallop rhythm.
- Pericardial friction is audible over restricted area on the 2nd or 3rd day after debut of AMI.
- Pulse in AMI is often small, accelerated, or arrhythmic.
- Arterial pressure increases during attack but then it falls.

- Depending on the localization of the AMI, circulation may be disordered by the left-ventricular or right-ventricular type.
- In the former case, congestive moist rales can be heard in the lungs.

General blood analysis - leucocytosis, elevation of ESR.

Blood serum analysis - elevated level of :

- lactic dehydrogenase (after 48 hours, 1-3 weeks)
- creatine phosphokinase - MB (is elevated after 6 hours from beginning of the MI) - 3-4 days
- myoglobine
- troponine fractions (is elevated after 3-4 hours from beginning of the MI).



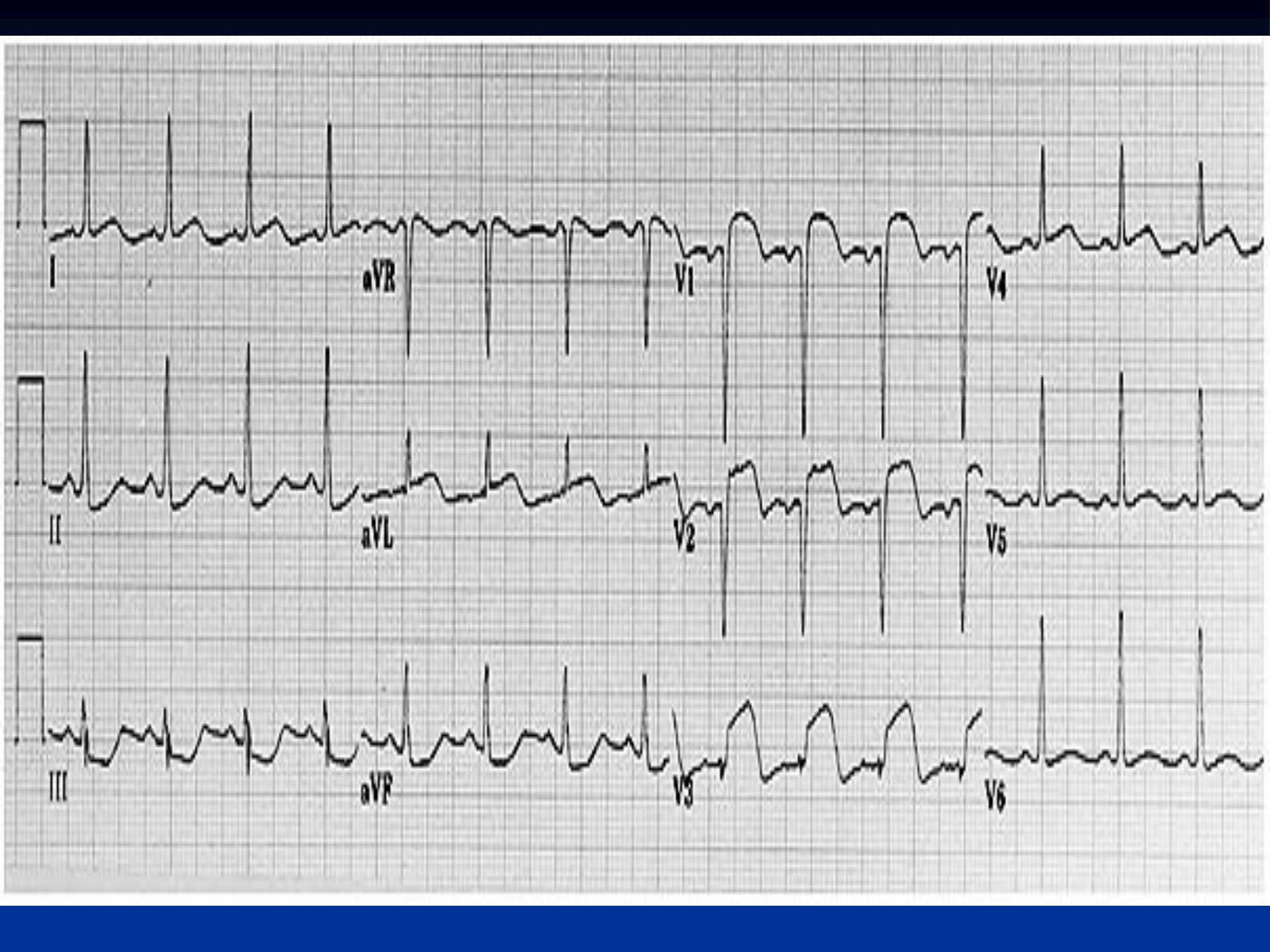
# ECG investigation -

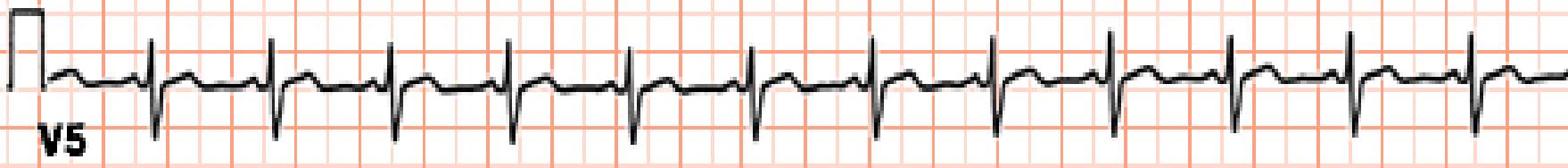
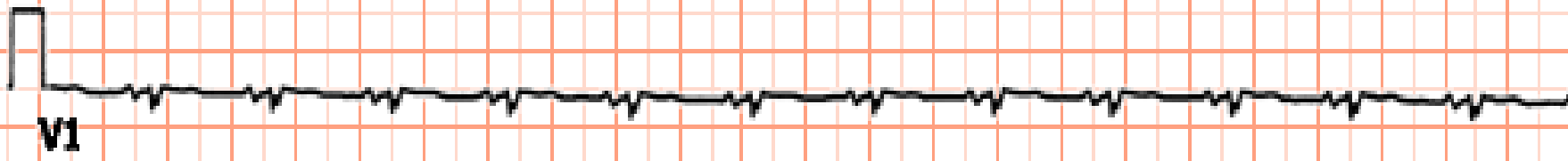
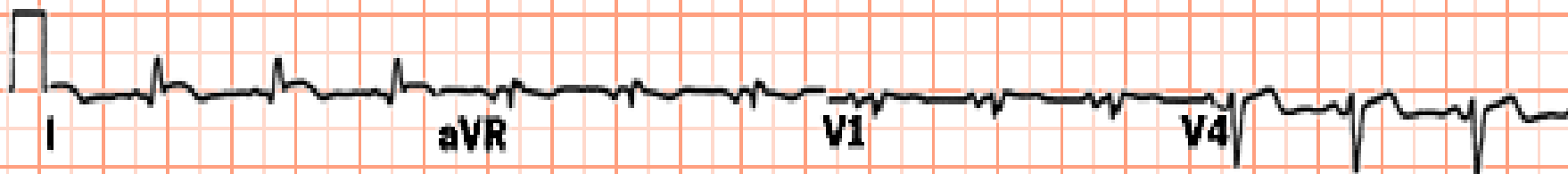
## 1. Acute phase:

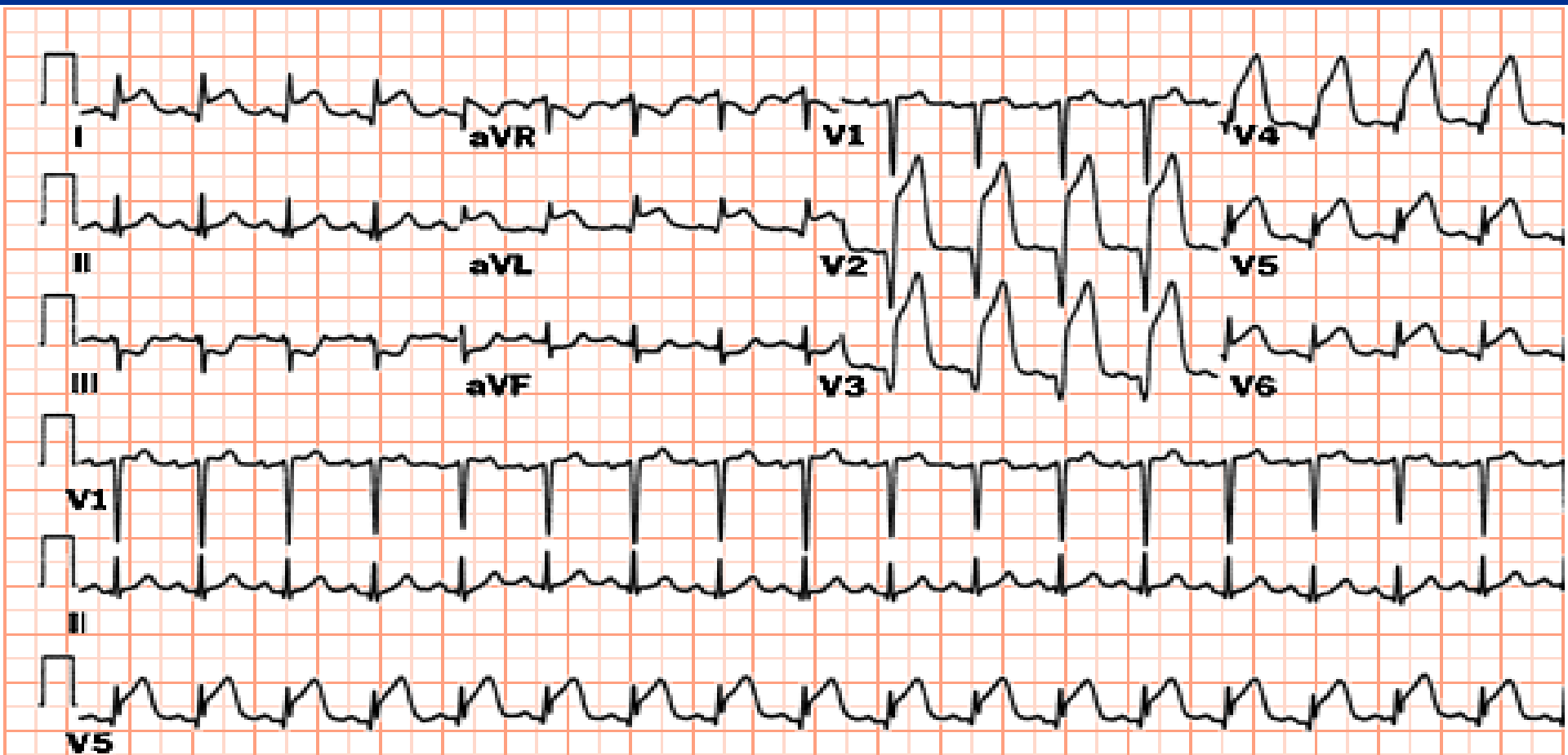
- Initially acute phase - (3-6 hours) - progressive elevating of ST segment and T wave. Together they formed monophasic curve.
- Acute defined phase - appearance of the pathological Q wave with concomitant slowly diminishing of the R wave. ST segment becomes to the isoelectric line to the 10-15 day

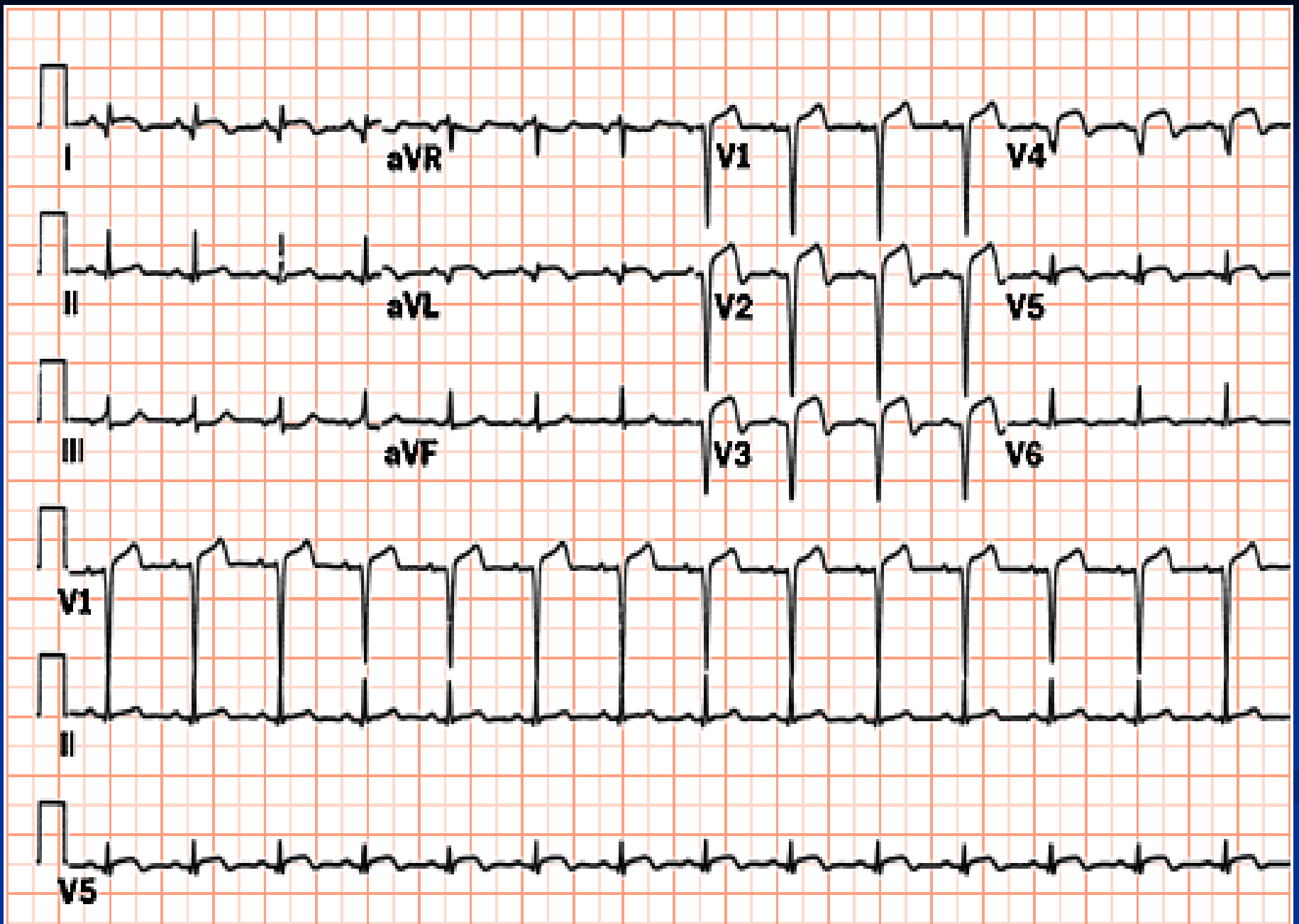
# ECG investigation

2. *Subacute phase* - (few weeks or months) - ST segment is on the isoelectric line, T wave becomes positive.
3. *Chronic phase* - presence of the QS complex, T wave is positive, absence of the R wave (or R wave is very small).









- **Ecocardiography** - is revealing the presence of the hypokinetic or akynetic zones, aneurism of the heart muscle.
- **Coronarography** - presence of the occlusions of the arteries, zones of necrosis.
- **Radionuclide method** with Tl 99 can be helpful in diagnosing of the AMI.