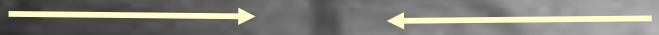
Chair of propaedeutics of internal medicine with care of patients

The CHIEF SYMPTOMS and DIAGNOSTICS on CORONARY HEART DISEASE and Arterial Hypertension

Dr. Chekalina N.

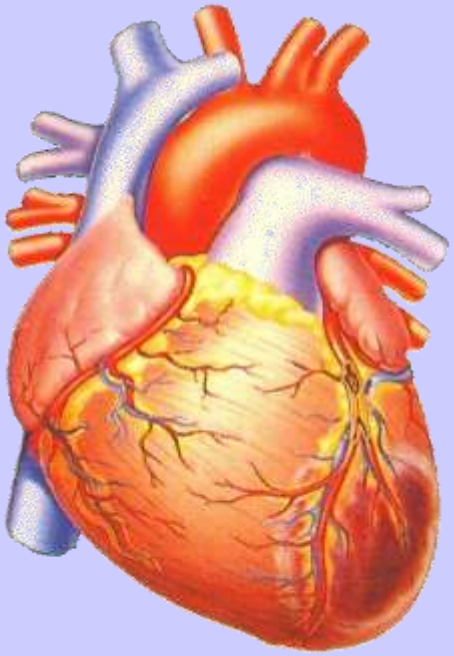
What is coronary artery disease?



A narrowing of the coronary arteries that prevents adequate blood supply to the heart muscle. Usually caused by atherosclerosis, it may progress to the point where the heart muscle is damaged due to lack of blood supply. Such damage may result in infarction, arrhythmias, and heart failure.

Myocardial Ischemia

- Results when there is an imbalance between myocardial oxygen supply and demand
- Most occurs because of atherosclerotic plaque with in one or more coronary arteries
- Limits normal rise in coronary blood flow in response to increase in myocardial oxygen demand



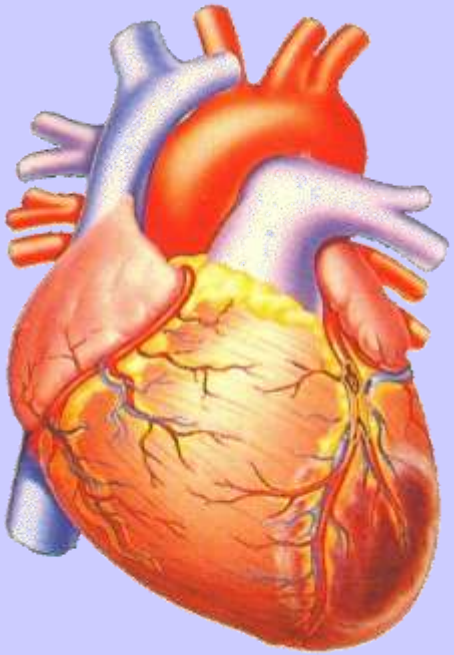
Pathology of atherosclerosis

- Lipid laden macrophages form fatty streak
- This allows lipids to deposit in the arterial wall
- Smooth muscle cells, cholesterol and lymphocytes also join the plaque

- Eventually a fibrous cap forms around the whole structure causing a narrowing

- Causes ischemia by three mechanisms
  - Platelet aggregation can form clots – emboli
  - Plaque rupture
  - Progressive stenosis

Stable Angina

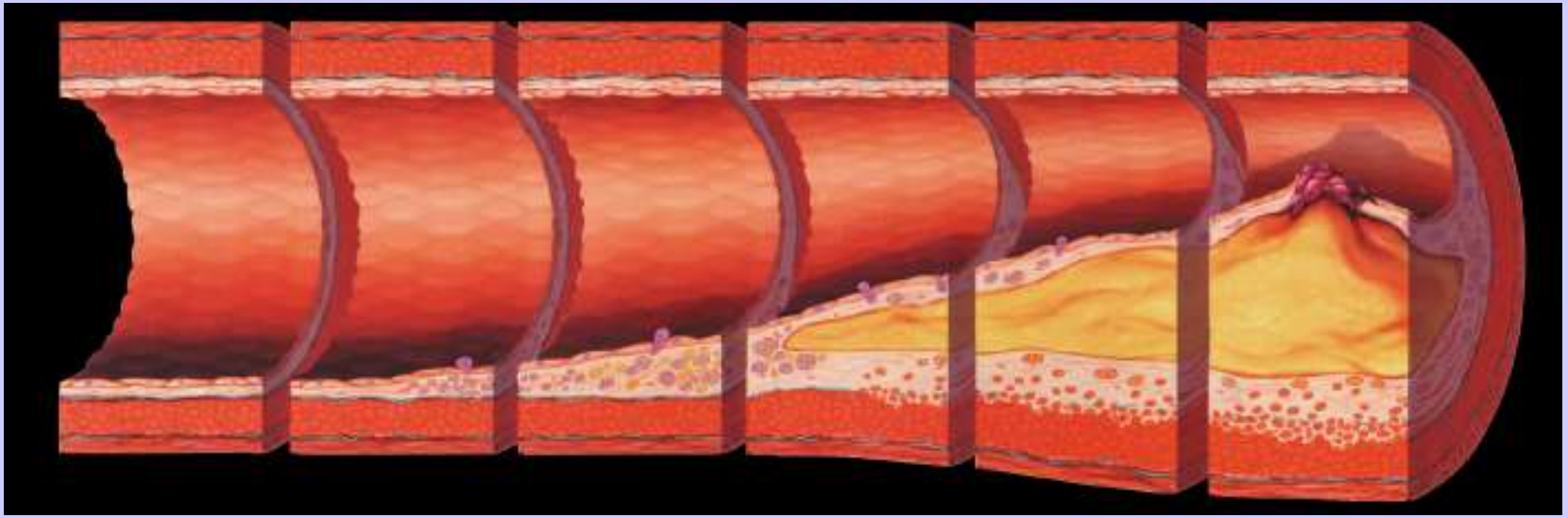


- myocardial cells have to do only 2 things: contract and relax; both are aerobic, O2 requiring processes
- oxygen extraction in the coronary bed is maximal in the baseline state; therefore to increase O2 delivery, flow must increase
- large visible epicardial arteries are conduit vessels not responsible for resistance to flow (when normal)

- small, distal arterioles make up the major resistance to flow in the normal state
- atherosclerosis (an abnormal state) affects the proximal, large epicardial arteries
- once arteries are stenotic (narrowed) resistance to flow increases unless distal, small arterioles are able to dilate to compensate

Atherosclerosis Timeline

Foam Cells | Fatty Streak | Intermediate Lesion | Atheroma | Fibrous Plaque | Complicated Lesion / Rupture



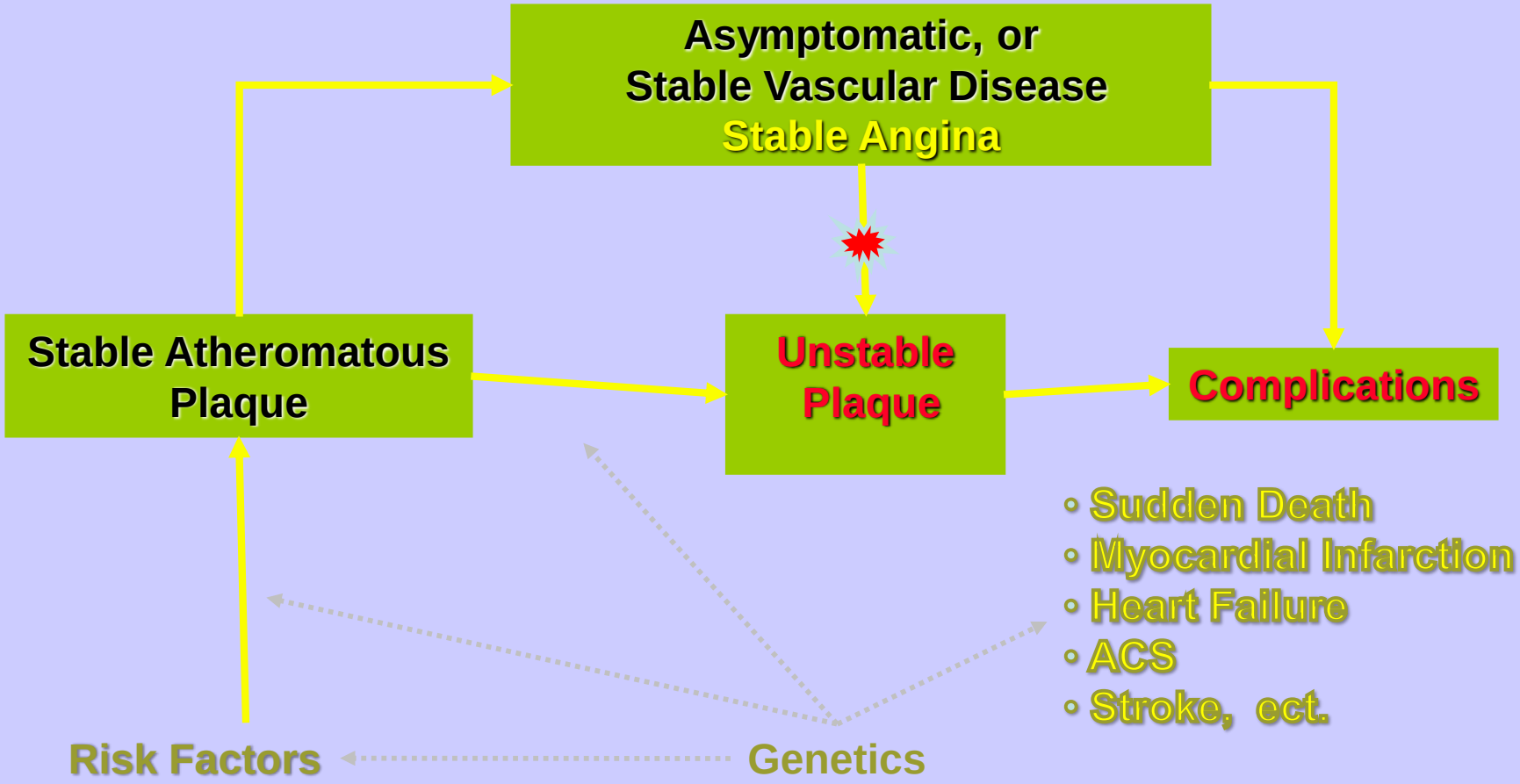
Endothelial Dysfunction →

From First Decade

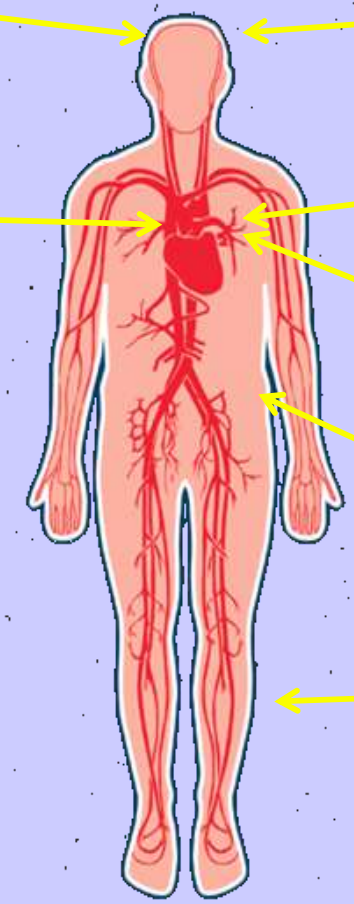
From Third Decade

From Fourth Decade

Progression of Vascular Disease



Clinical Manifestations of Atherothrombosis



Transient ischemic attack (TIA)

Angina

Ischaemic stroke (CVA)

Acute Coronary Syndrome (ACS)

Myocardial infarction (MI)

Renovascular Disease

Peripheral Vascular Disease (PVD)

Ischemic Heart Disease

- 80% of deaths in Ukraine by heart disease and 30% of total mortality
- Mortality from IHD declined in the Ukraine by 40% in the past 20 years
- Major cause of cardiac arrest
- Survival to discharge of all rhythm cardiac arrests is 10.7%

Causes of Ischemic Heart Disease

- **IHD has many risk factors, including:**
  - **Smoking,**
  - **Hypertension,**
  - **Diabetes,**
  - **Hyperlipidemia,**
  - **Hypodynamia,**

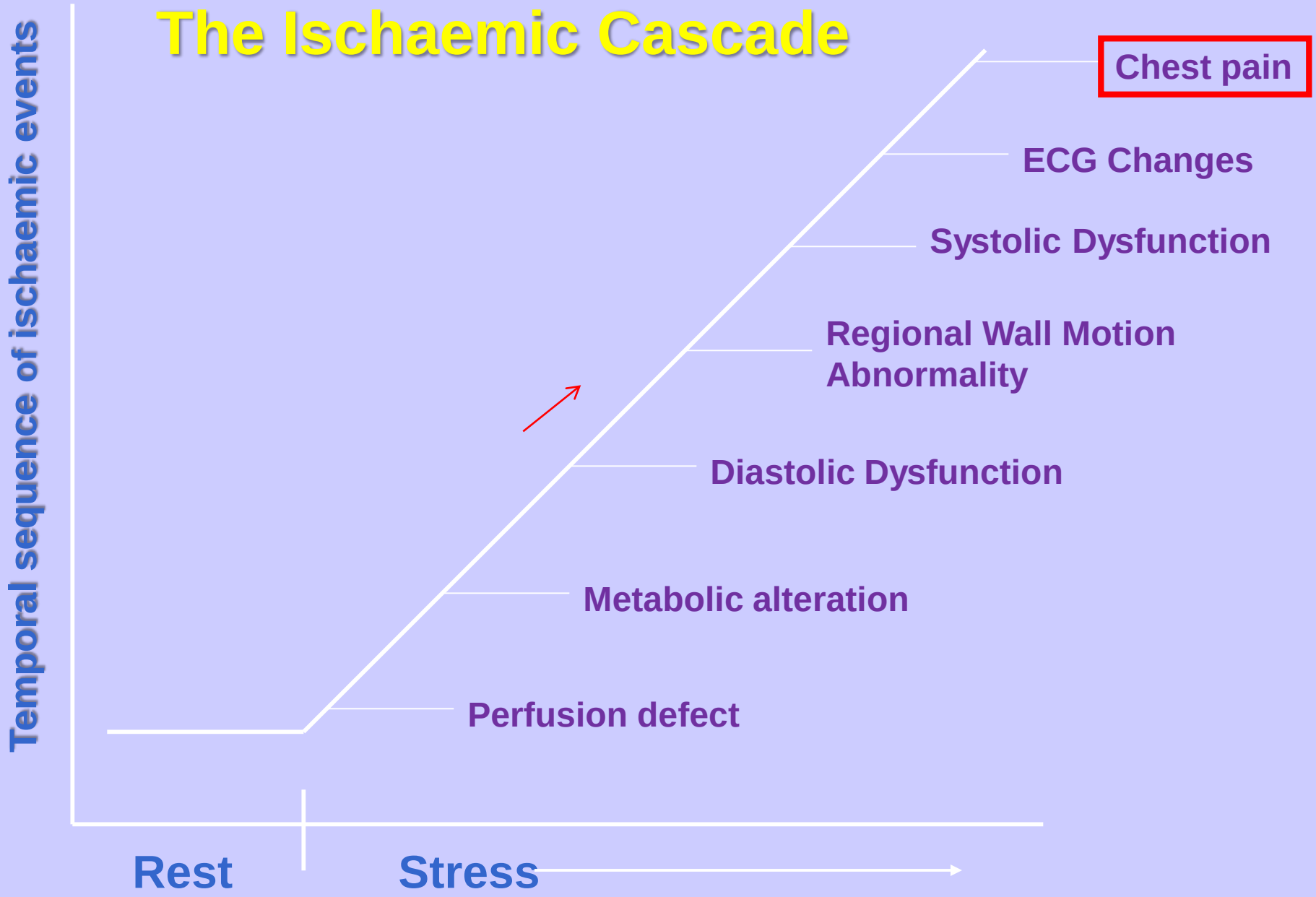
Signs & Symptoms

- **Coronary heart disease may be asymptomatic.**
- **If not, symptoms can include:**
  - **Chest heaviness**
  - **Dyspnea**
  - **Fatigue**
  - **Chest pain**
  - **Angina**
  - **Myocardial infarction**

Quality

- **Tightness, squeezing, heaviness, pressure, burning, indigestion or aching sensation**
- **It is rarely "PAIN"**
- **Never: sharp, stabbing, prickly, spasmodic, or pleuritic**
- **Lasts a few seconds < 10 minutes**
- **Fist to sternum**

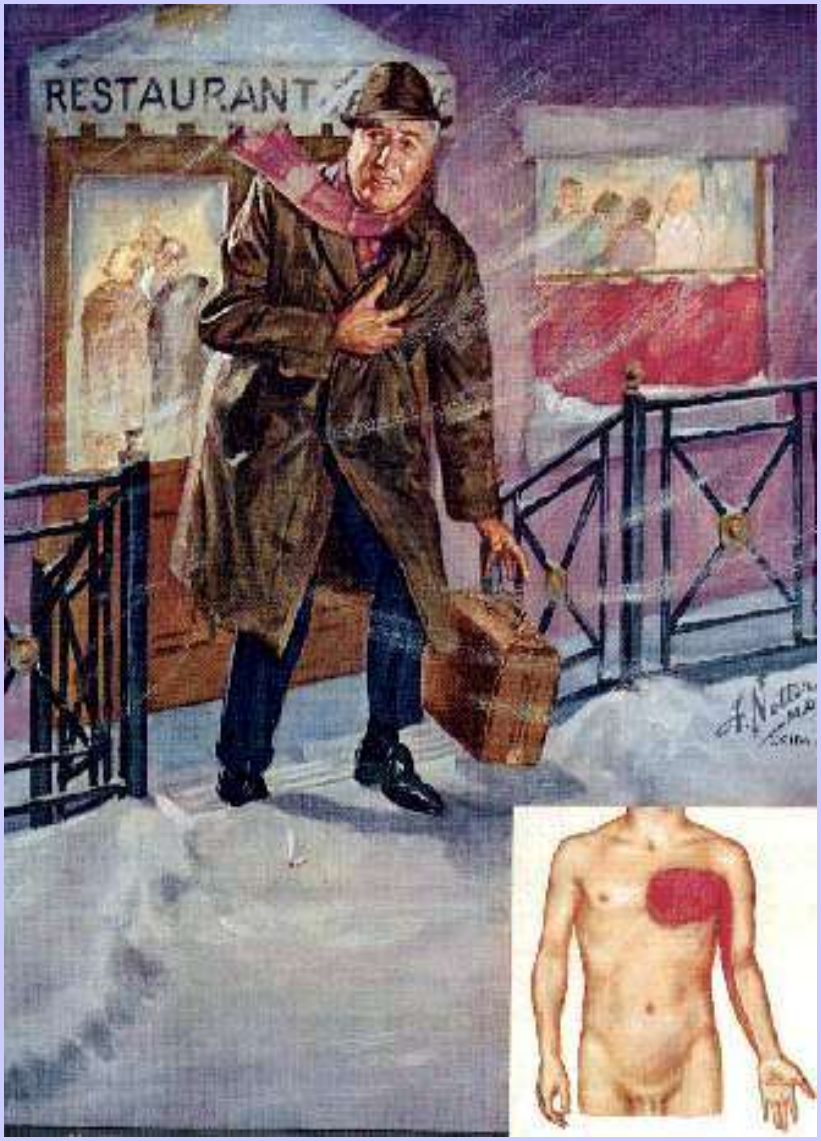
The Ischaemic Cascade



Stable Angina - Symptoms

- mid-substernal chest pain
- squeezing, pressure-like in quality (closed fist = Levine's sign)
- builds to a peak and lasts 2-20 minutes
- radiation to left arm, neck, jaw or back
- associated with shortness of breath, sweating, or nausea
- exacerbated by exertion, cold, meals or stress
- relieved by rest, NTG

Symptoms and Signs: Coronary Ischemia



- **Stable Angina:** chronic pattern of transient angina pectoris precipitated by physical activity or emotional upset, relieved by rest with in few minutes
- Temporary depression of ST segment with no permanent myocardial damage

- Angina Pectoris: uncomfortable sensation in the chest or neighboring anatomic structures produced by myocardial ischemia

- **Variant Angina:** Typical anginal discomfort usually at rest
- Develops due to coronary artery spasm rather than increase myocardial oxygen demand
- Transient shifts of ST segment – ST elevation

- **Unstable Angina:** **Increased frequency and duration of Angina episodes, produced by less exertion or at rest = high frequency of myocardial infarction if not treated**

- **Silent Ischemia:** **Asymptomatic episodes of myocardial ischemia**
- **Detected by electrocardiogram and laboratory studies**

- **Myocardial Infarction:**
- **Region of myocardial necrosis due to prolonged cessation of blood supply**
- **Results from acute thrombus at side of coronary atherosclerotic stenosis**
- **May be first clinical manifestation of ischemic heart disease or history of Angina Pectoris**

Precipitants

- **Exertion:** walking, climbing stairs, vigorous work using arms, sexual activity
- **Vasoconstriction:** increased systemic vascular resistance, increased in myocardial wall tension and oxygen requirements
- **Myocardial Ischemia displays a circadian rhythm threshold for Angina it is lower in morning hours.**

- **Signs and symptoms:** hyperthyroidism with increased myocardial oxygen demand, hypertension, palpitations, xanthomas, exudates
- **Auscultate carotid and peripheral arteries and abdomen:** aortic aneurysm
- **Cardiac:** increased heart rate, increased blood pressure

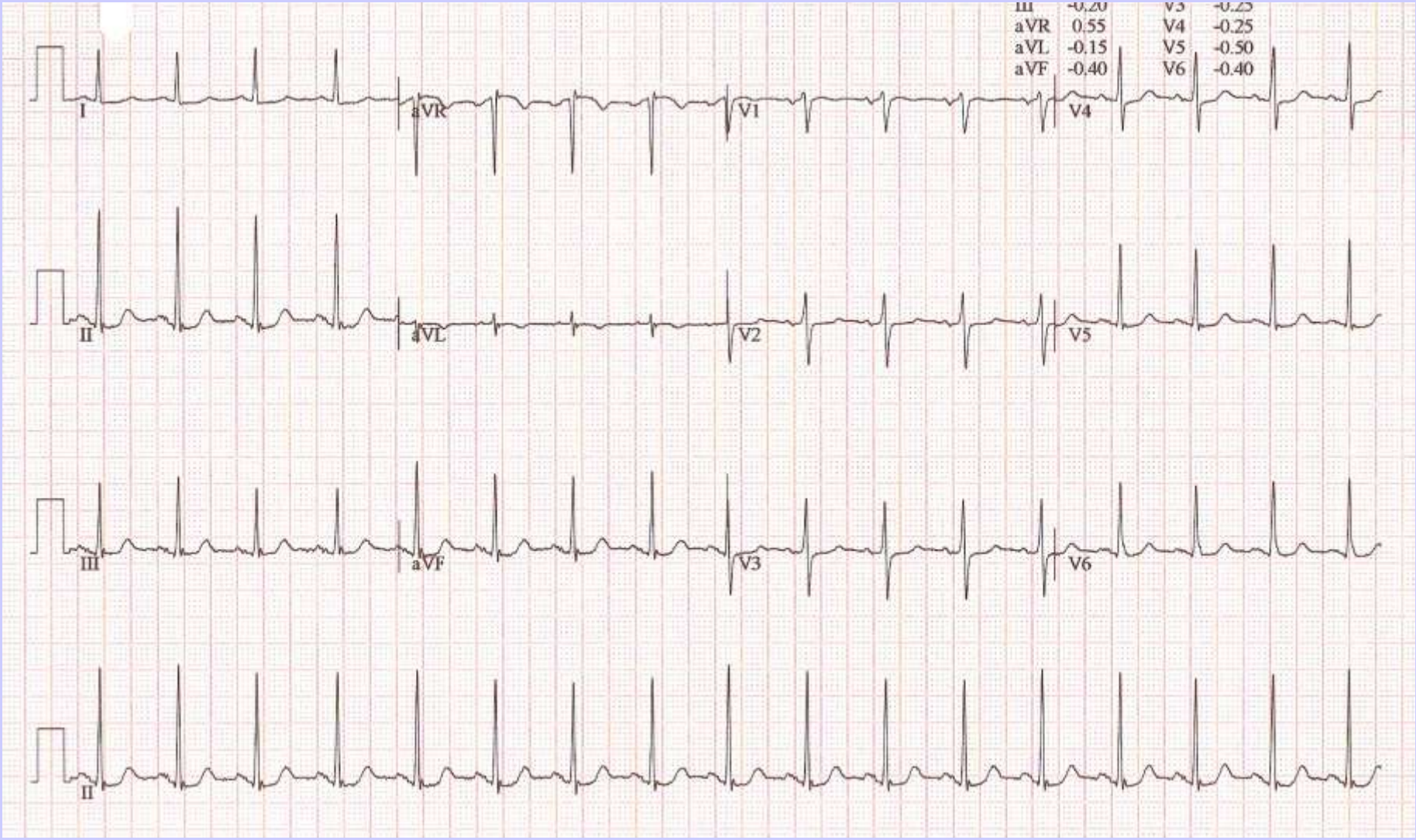
Diagnostic Tests

- **Blood tests include serum lipids, fasting blood sugar, Hematocrit, thyroid**
  (anemias and hyperthyroidism can exacerbate myocardial ischemia)
- **Resting Electrocardiogram: CAD patients have normal baseline ECGs**
  - **pathologic Q waves = previous infarction**
  - **minor ST and T waves abnormalities not specific for CAD**

Electrocardiogram

- Electrocardiogram: is useful in diagnosis during chest pain
- When ischemia results in transient horizontal or downsloping ST segments or T wave inversions which normalize after pain resolution
- ST elevation suggest severe transmural ischemia or coronary artery spasm which is less often

46 male with chest tightness on effort. Diabetic with raised cholesterol.



Exercise Stress Test

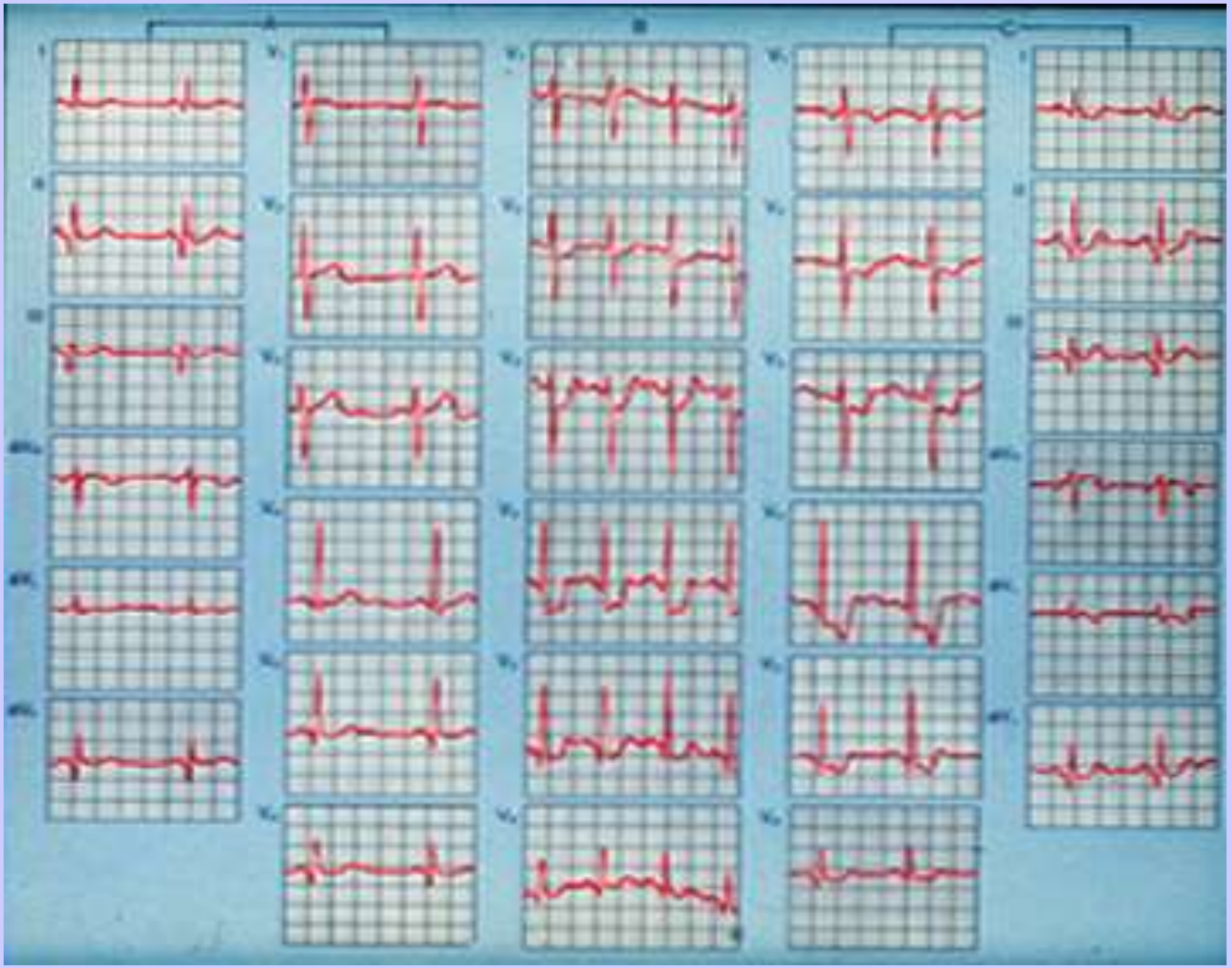
- Used to confirm diagnosis of angina
- Terminate if hypotension, high grade ventricular disrrhythmias, 3 mm ST segment depression develop
- (+): reproduction of chest pain, ST depression
- Severe: chest pain, ST changes in 1st 3 minutes, >3 mm ST depression, persistent > 5 minutes after exercise stopped
- Low systolic BP, multifocal ventricular ectopy or V- tach, ST changes, poor duration of exercise (<2 minutes) due to cardiopulmonary limitations

Cardiac Stress Test

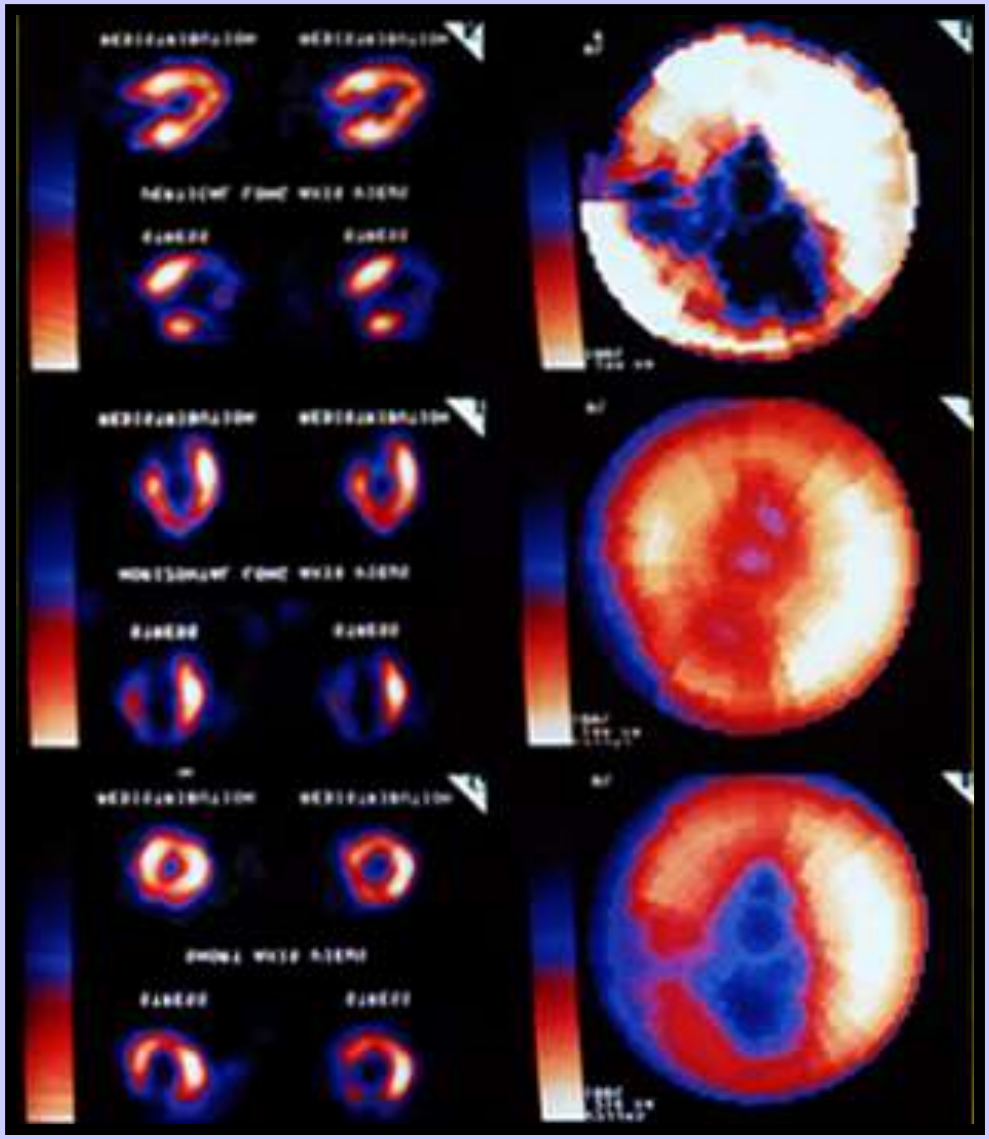
Other Diagnostic Tests

- Radionuclide studies
- Myocardial perfusion scintigraphy
- Exercise radionuclide ventriculography
- Echocardiography
- Ambulatory ECG monitoring
- Coronary arteriography

Stable Angina - Diagnosis
Exercise Treadmill Test



Stable Angina - Diagnosis
Thallium Stress Test



Unstable Angina

- Ischemic episodes occur more frequently more intense, last longer.
- Precipitated by less activity or even at rest
- May progress to acute MI due to presence of complicated coronary lesions with ulceration, hemorrhage or thrombosis at side of atherosclerotic plaque
- Lesions may heal: s/s return to more stable pattern

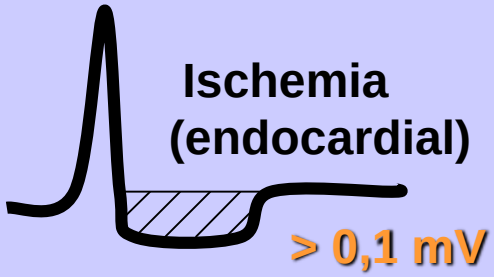
Unstable Angina

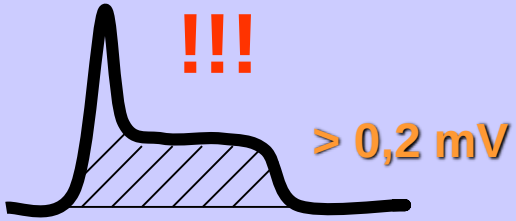
- It is a medical emergency
- During episodes of angina, transient ST segment shifts or T wave flattening or inversion is likely
- Signs of LV dysfunction (pulmonary rales, mitral regurgitation) may accompany ischemic episodes.

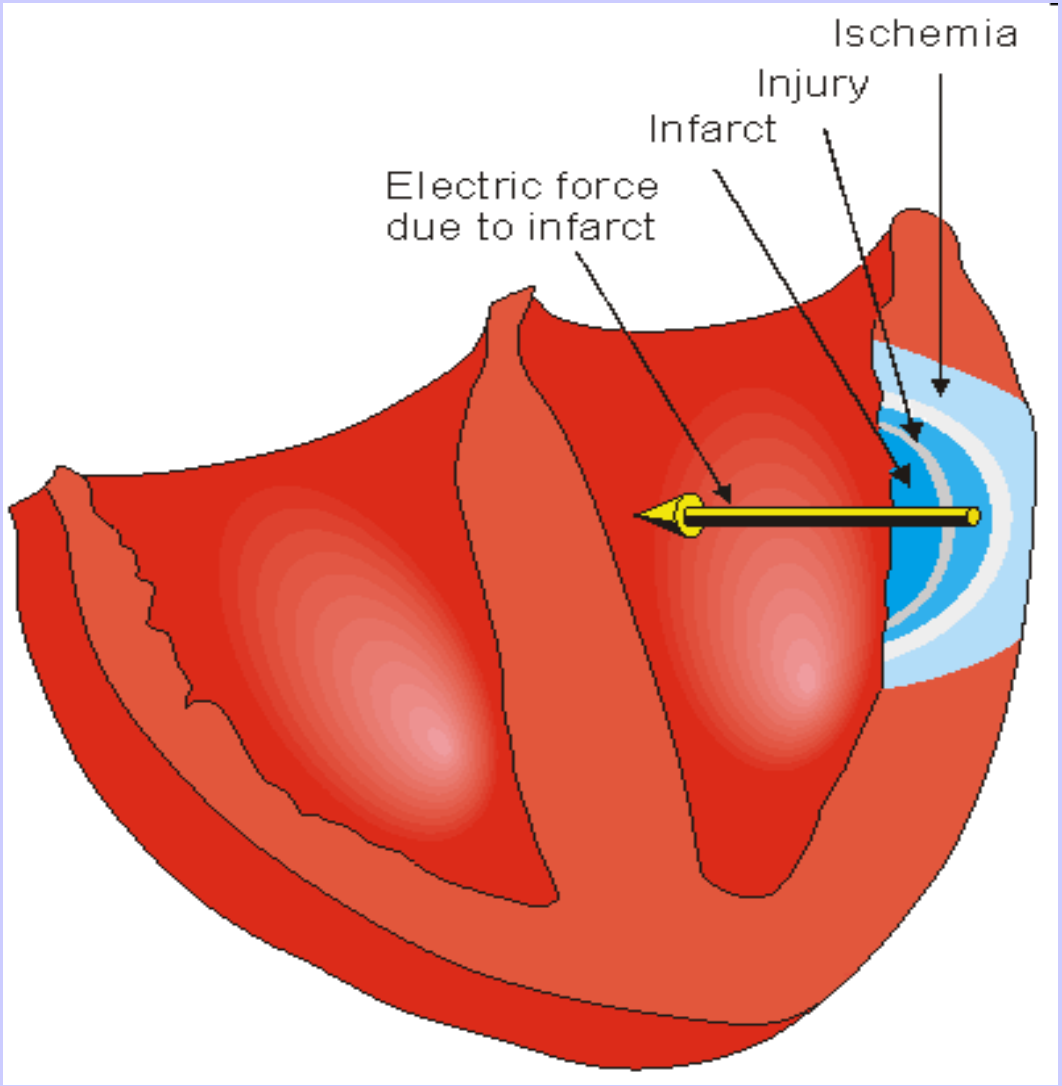
Acute Myocardial Infarction

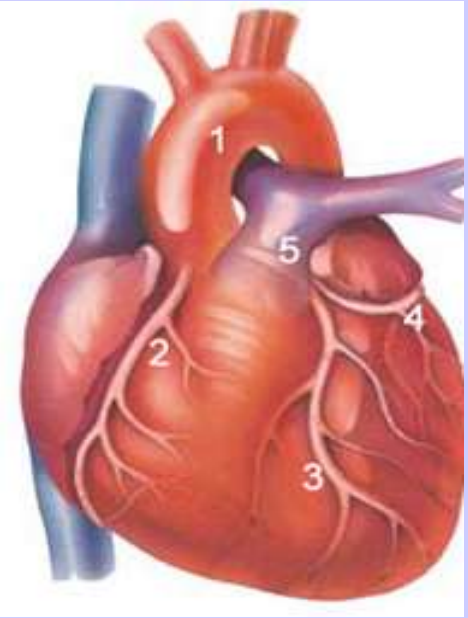
- Is dreaded outcome in patients with ischemic heart disease
- Mortality rate of 25%
- 60% of MI related deaths occur before medical facilities are reached

Myocardial infarction









Coronary Artery (CA) Distribution

1. Aorta
2. Right Coronary Artery
3. Left Anterior Descending Artery
4. Circumflex Coronary Artery
5. Left main stem

Left Anterior Descending Artery (40-50%) - anterior wall LV, apex, anterior IV septum

Right CA (30-40%) - posterior wall LV, posterior IV septum

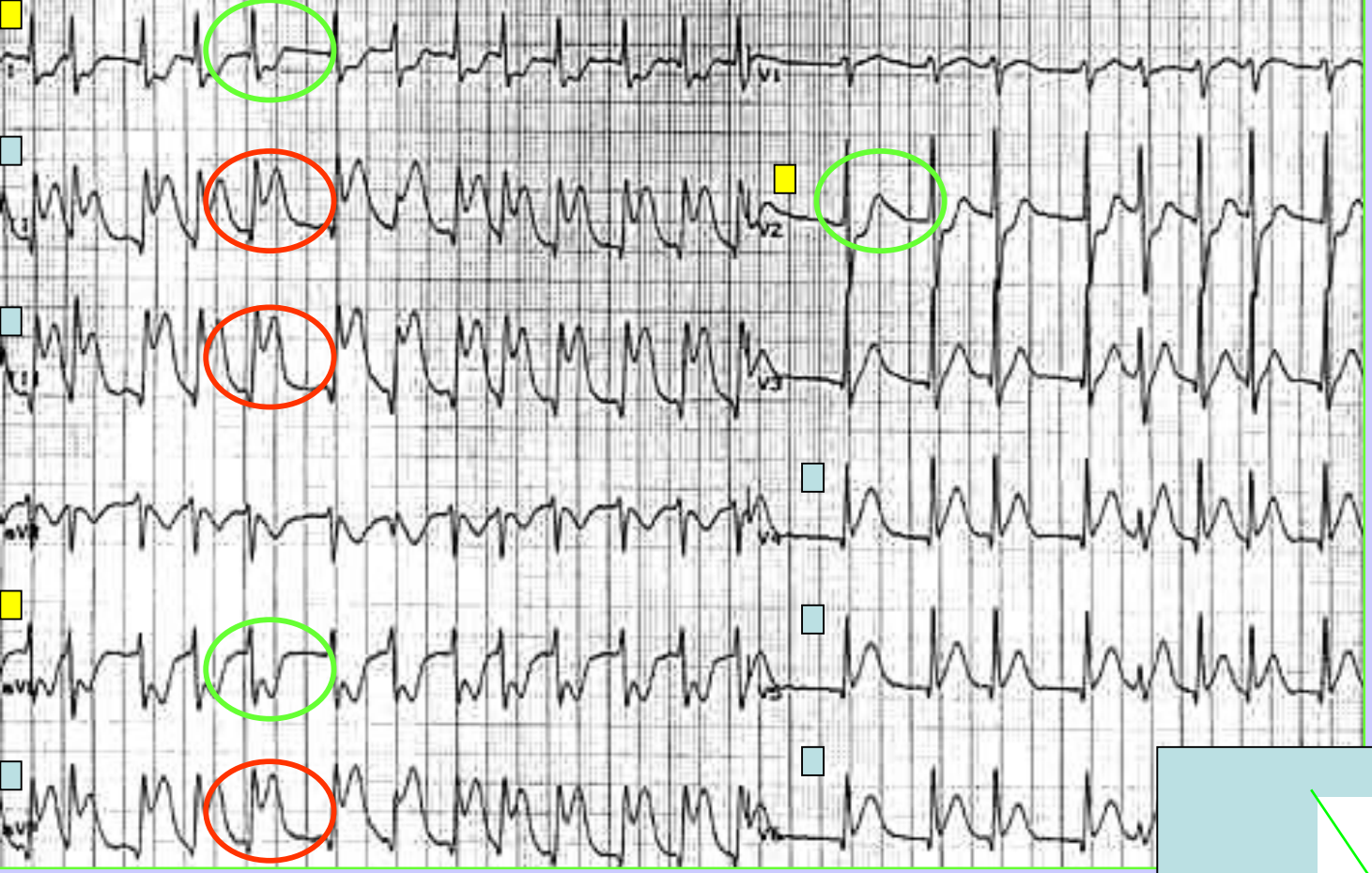
Left circumflex CA (15-20%) - lateral wall LV

- **Circumflex artery obstruction**
  - **Lateral infarction**
  - **ECG changes leads I, AVL and V4-V6**
  - **20% cases**

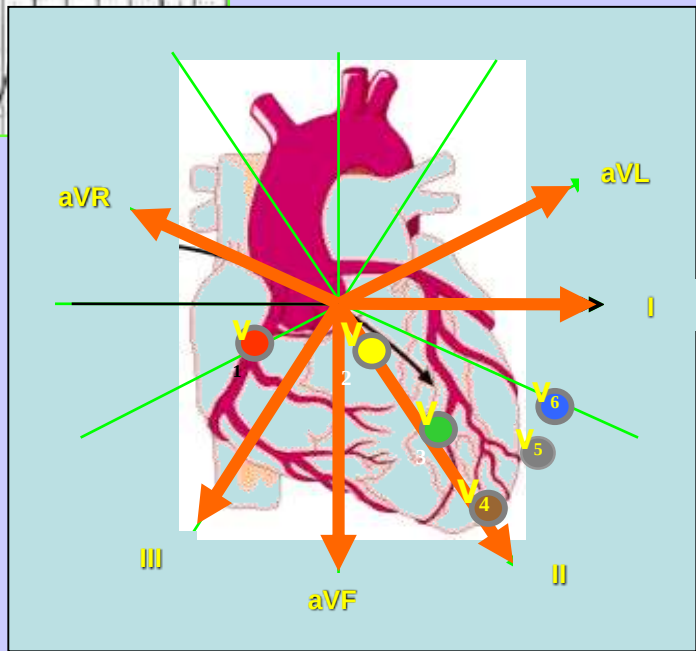
- **Left anterior descending obstruction**
  - **Anterior infarction**
  - **ECG changes V1-V4**
  - **50% cases – most likely to cause left ventricular dysfunction**

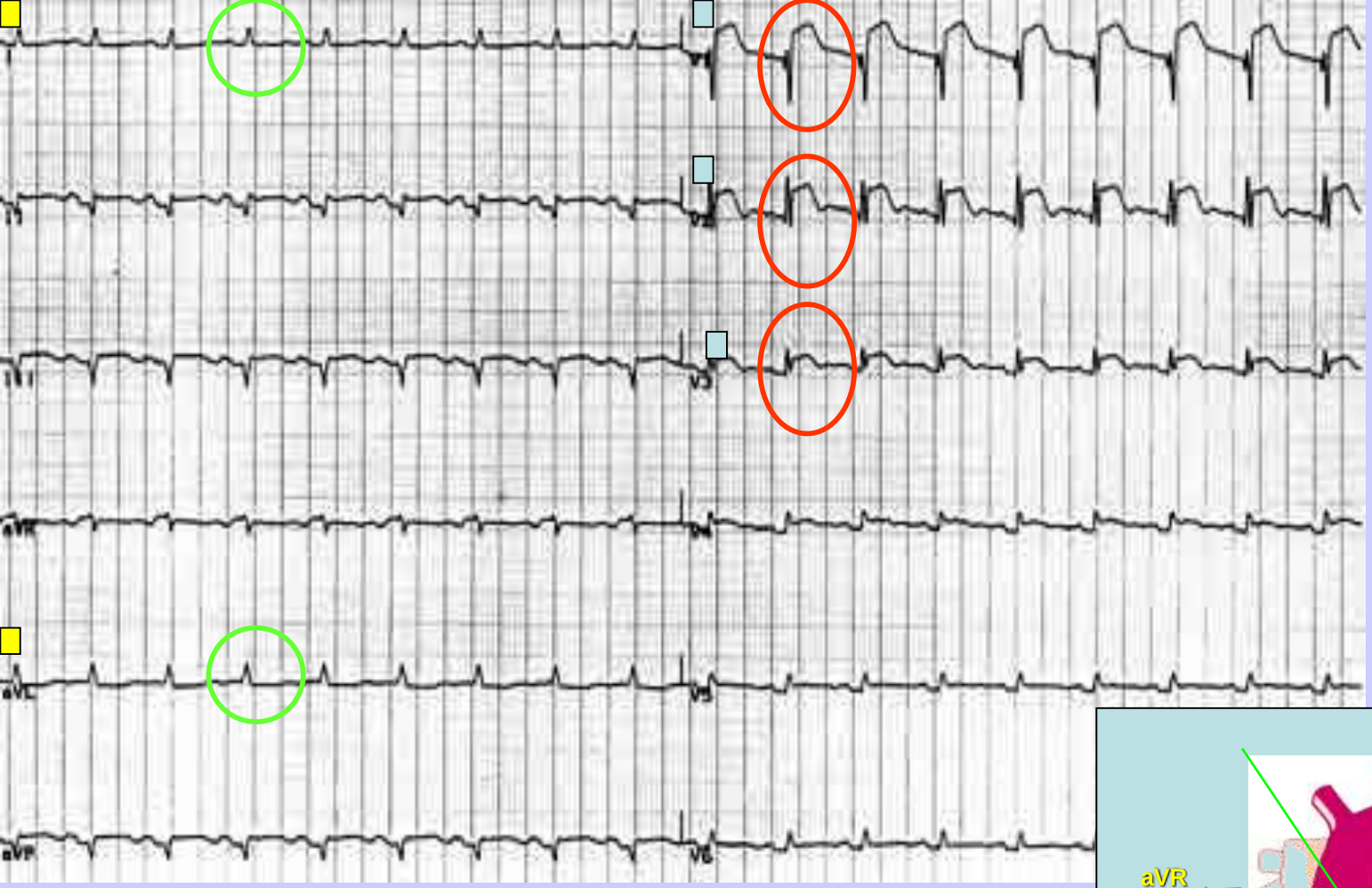
- **Right coronary artery obstruction**
  - **Inferior infarction**
  - **ECG changes in II, III & AVF**
  - **30% cases - often causes bradyarrythmias**

- **Right coronary artery but may be circumflex**
  - **Posterior myocardial infarction**
  - **ST depression in anterior leads**
  - **Dominant R wave is actually a Q wave**

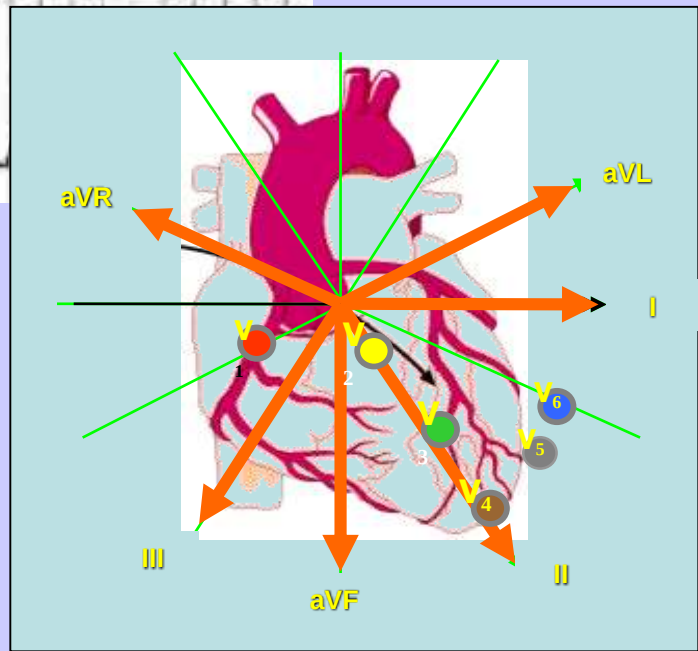


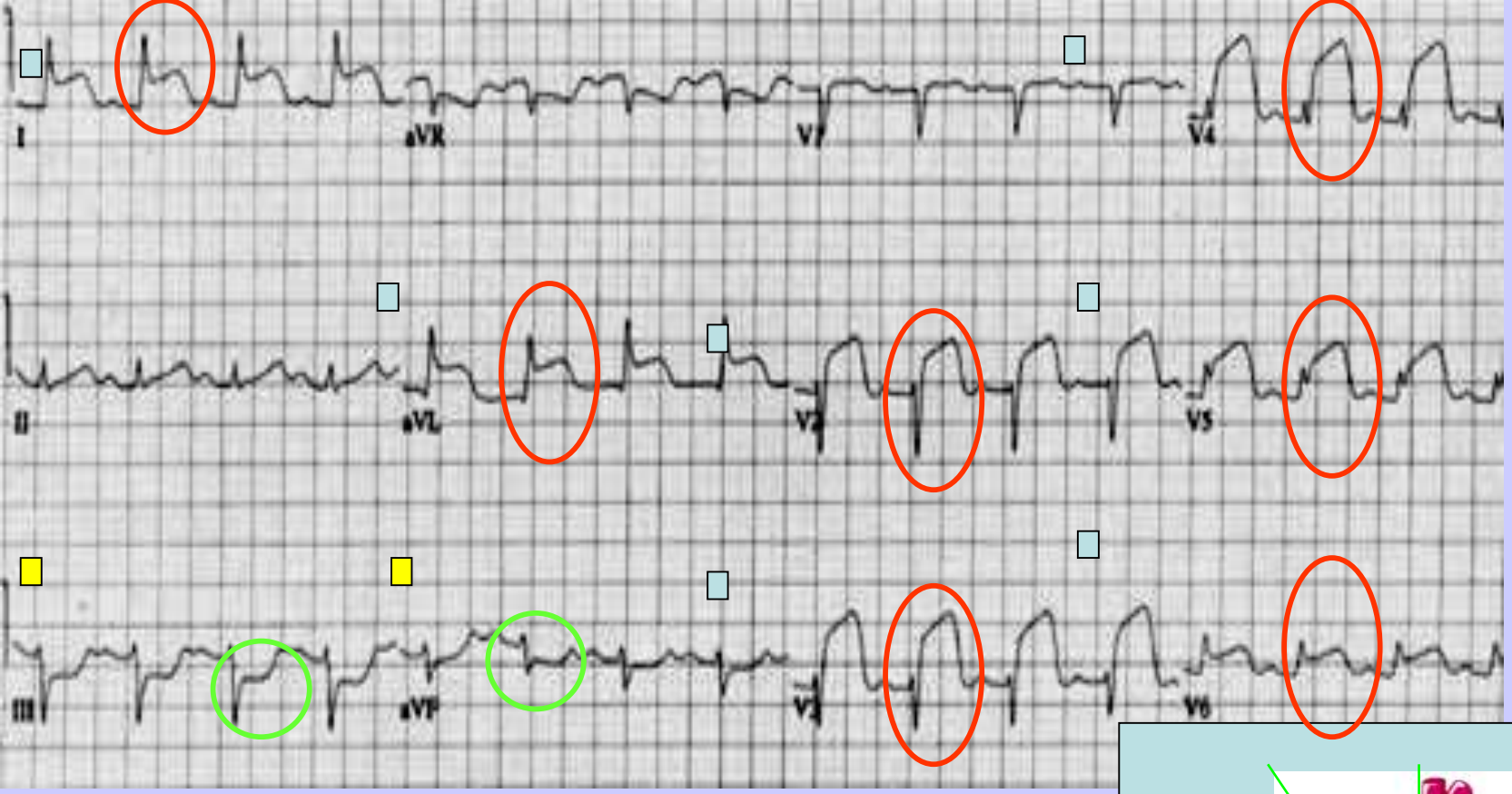
Inferior myocardial
infarction of a left ventricle





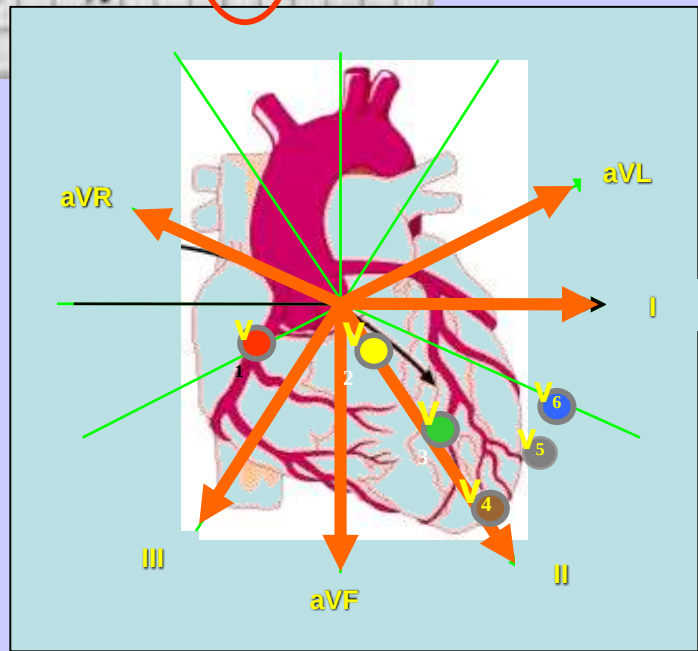
Myocardial infarction of a
anterior and septum
of a left ventricle

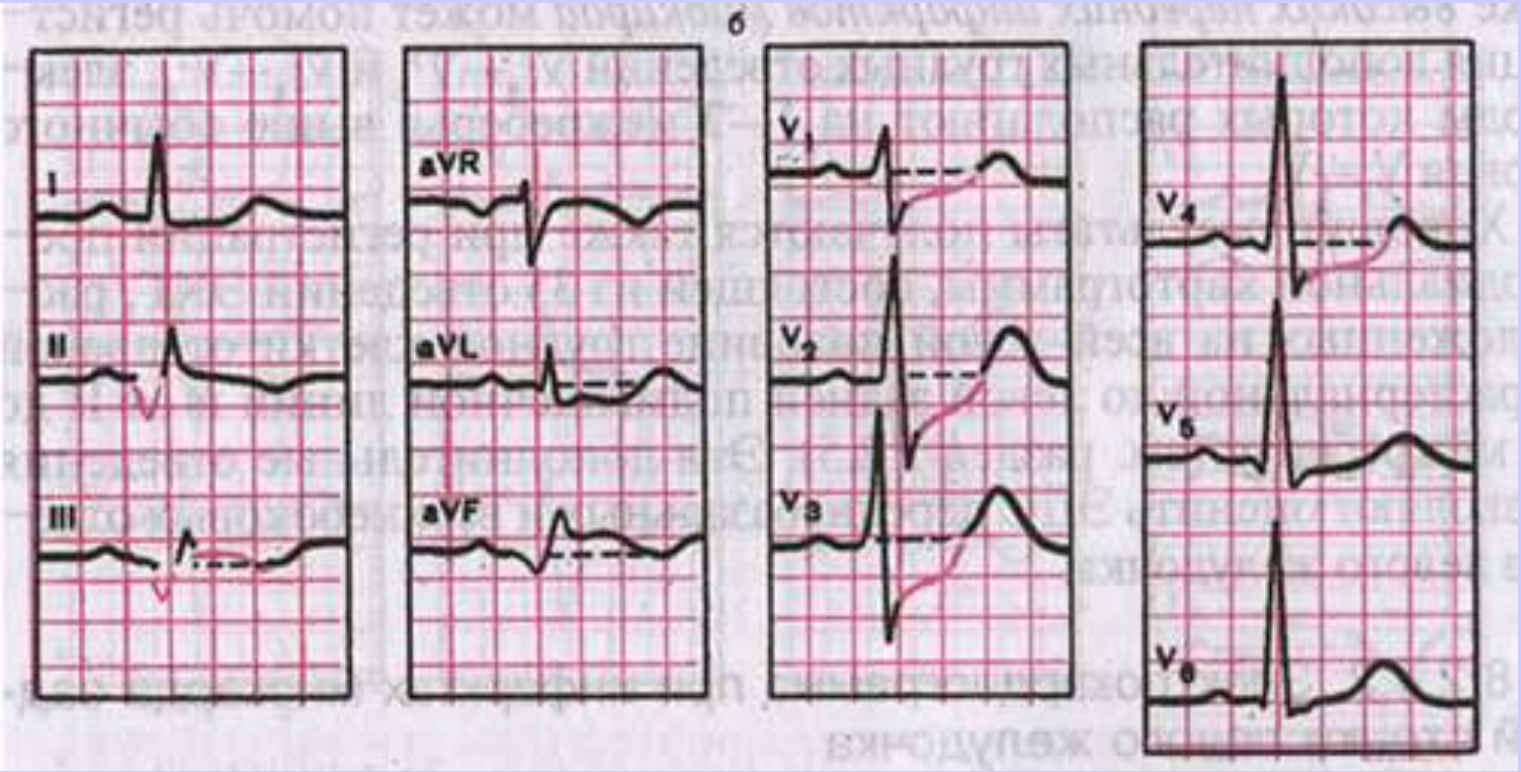




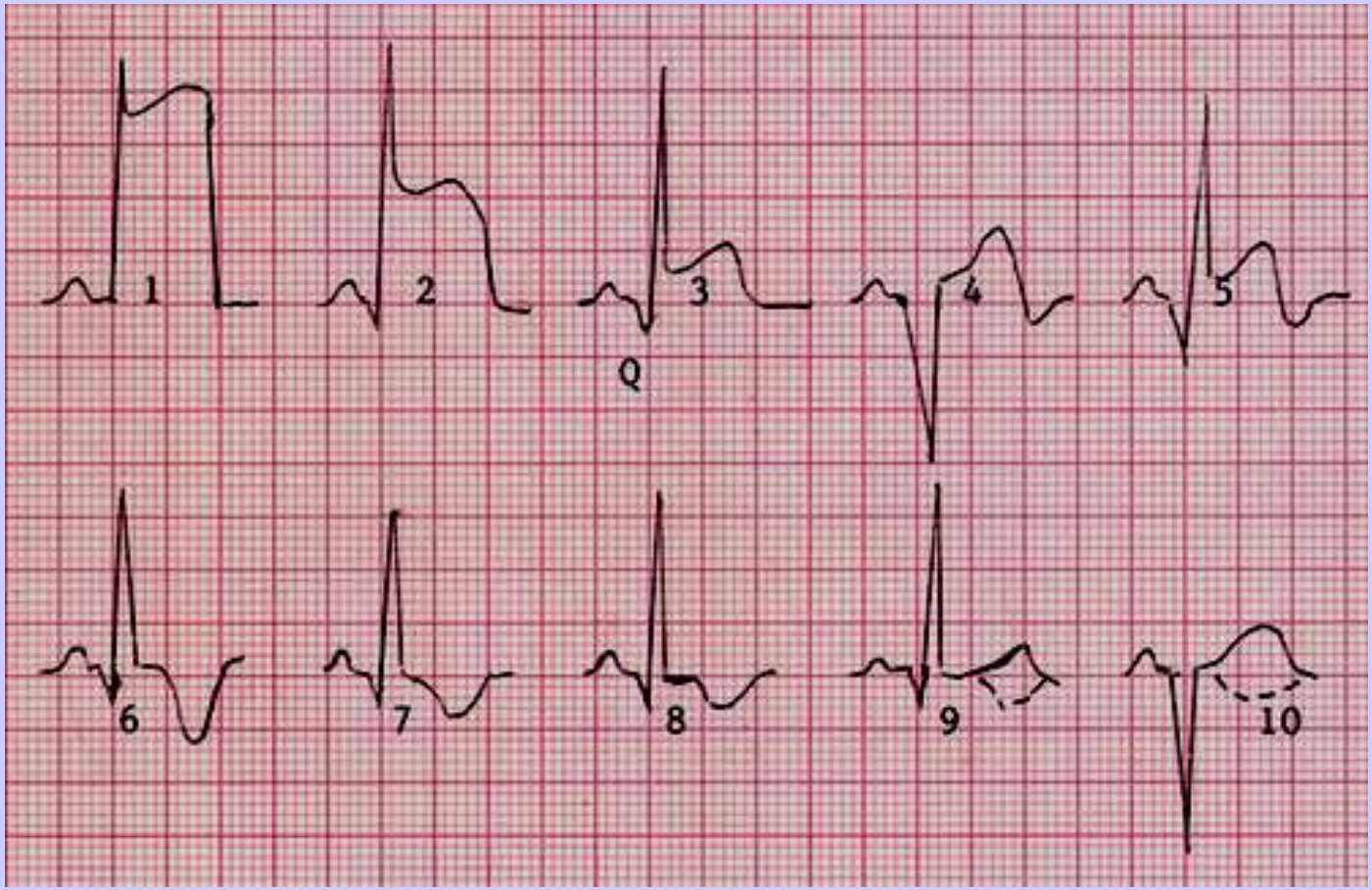
Myocardial infarction

of anterior, apex, lateral side of a left ventricle




б
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

?


1
2
3
Q
4
5
6
7
8
9
10

MVO_2 = Myocardial Oxygen Demand

MVO_2 determined by:

Heart Rate
Contractility
Wall Tension

Increased Myocardial Demands

Tachycardia
Hypertrophy
Hypermetabolism
Hyperthyroidism
Drugs

Availability of Oxygen in Blood

Anemia

Carboxyhemoglobin

Pulmonary disease

Right to left shunts

Sudden Cardiac Death

Unexpected death within one hour of cardiac event

300,000-400,000 persons per year

Usually high grade coronary stenosis

Ventricular electrical instability

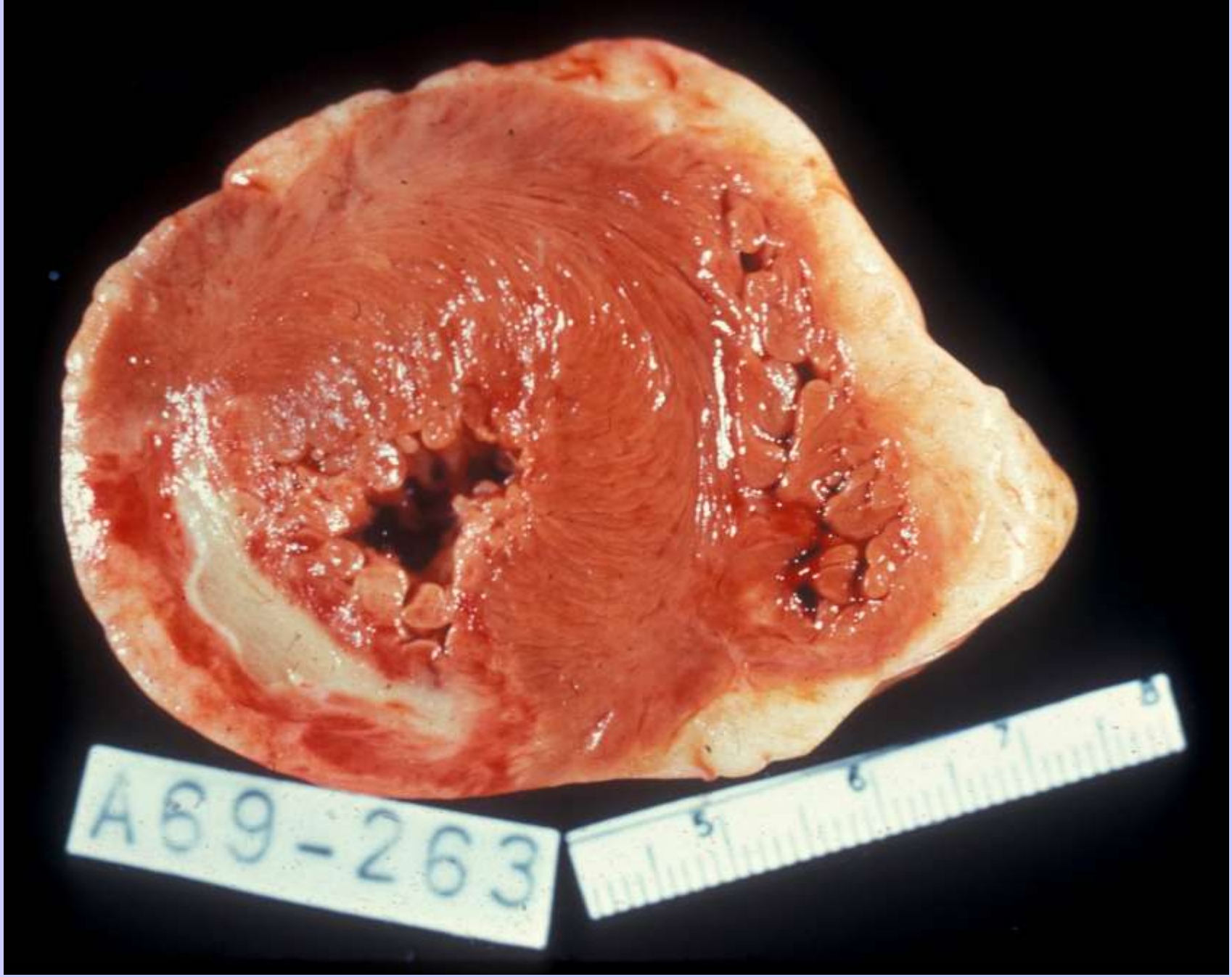
Subendocardial Infarct

- Multifocal areas of necrosis confined to inner 1/3-1/2 of LV wall
- Infarct evolution different than transmural infarct

Transmural Infarct

Endocardium to epicardium

Usually involving LV anterior and posterior free wall or septum with extension into RV wall in 15-30% of cases



Recent transmural infarct

MI - Microscopic Appearance

1-3 hrs - Wavy fibers

2-3 hrs - staining defect

4-12 hrs - Coagulation necrosis

18-24 hrs - Pyknosis, contraction bands

24-72 hrs - Neutrophils, loss of striations

3-7 days - Macrophages and fibrosis

7 weeks and beyond - Fibrosis

MI - Complications

None 10-20%

Arrhythmias 75-95%

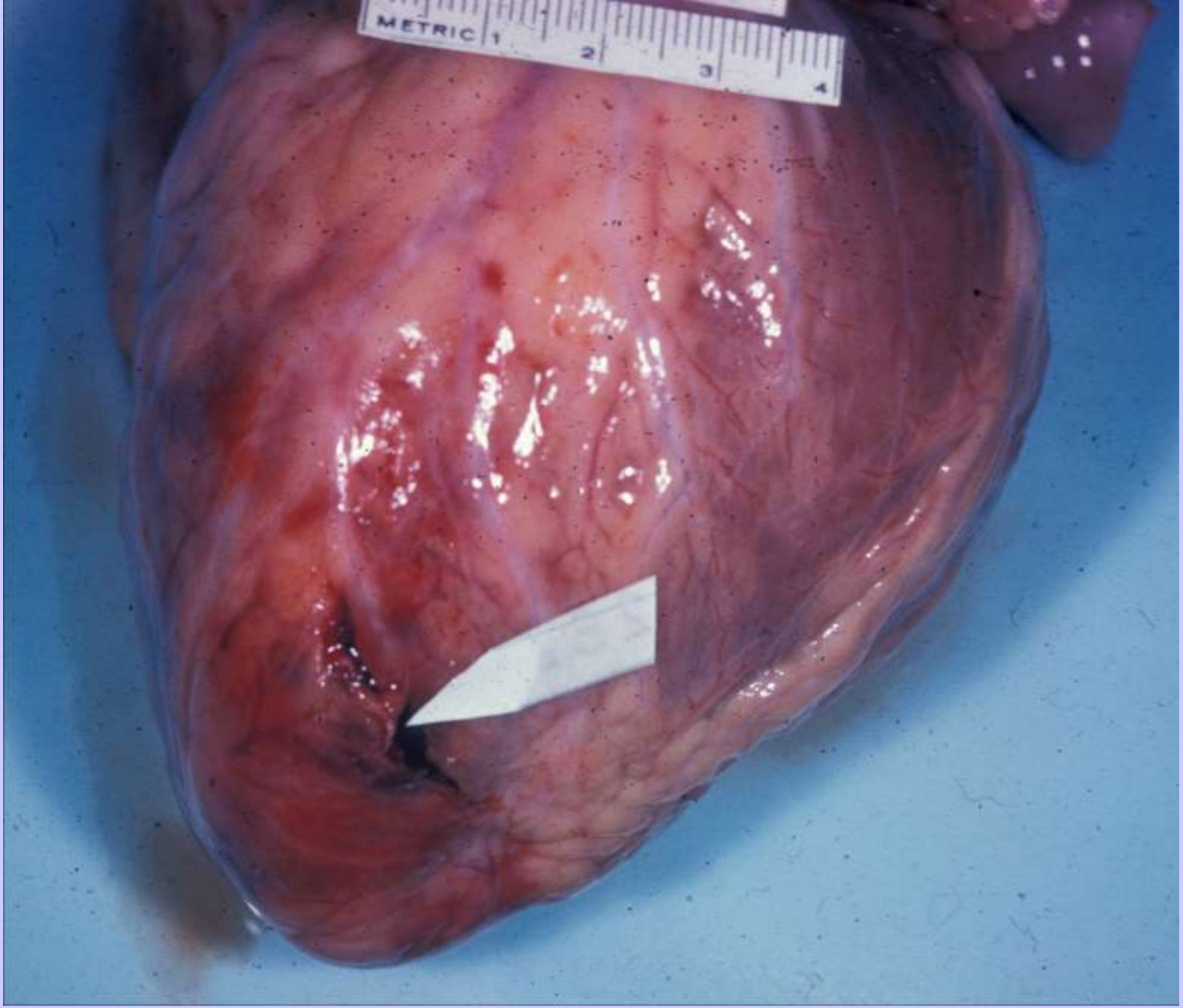
Pulmonary edema 60%

Cardiogenic shock 10-15%

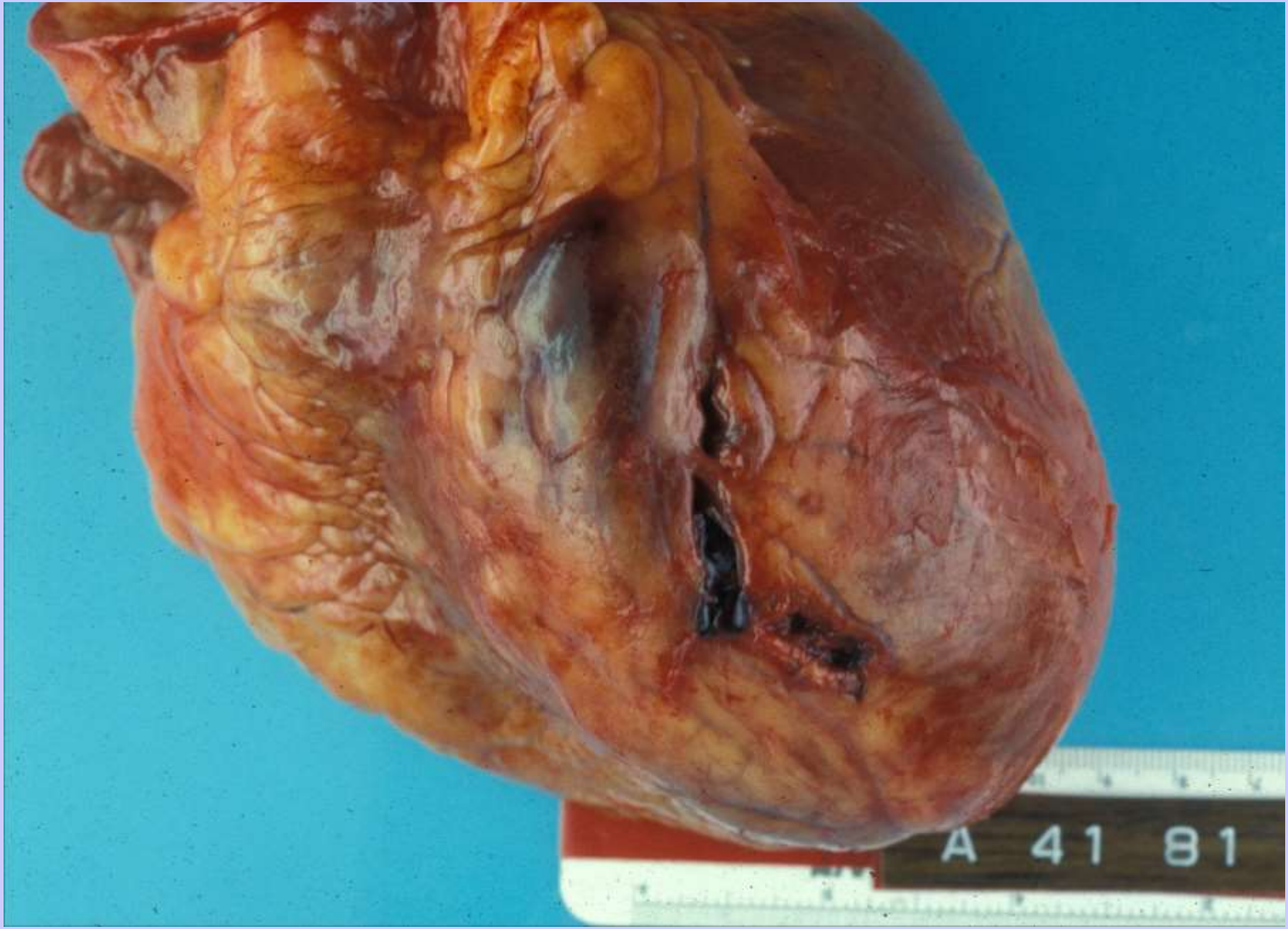
Pericarditis 50%

Mural thrombosis 40%

Rupture ventricle or papillary muscle 4-8%



Recent infarct with perforation



Another Rupture in an acute MI. This typically happens 3-5 days after the infarct.

Creatine Kinase - CK Isoenzyme

Tissue	BB	MB	MM
Muscle	0%	2%	98%
Heart	0%	15-40%	60-85%
Brain	90%	0%	10%
Bladder	95%	0%	5%
Bowel	100%	0%	0%

Lactate Dehydrogenase - LD

LD-1	19-9%	**Heart,** Kidney
LD-2	25-30%	Kidney
LD-3	16-31%	Lung
LD-4	2-9%	Muscle
LD-5	2-17%	Liver, Muscle

Temporal Sequence of Enzymes

Enzyme	Appear	Peak	Duration
CK-MB	2-8 hr	6-8 hr	1-3 day
Total CK	4-8 hr	18-24 hr	1-3 day
LD-1	4-8 hr	12-24 hr	5-14 day
Total LD	12-24 hr	48-96 hr	5-14 day
AST	6-8 hr	24-48 hr	3-5 day

Troponin for Acute MI

Troponin - regulatory protein release when cardiac cell necrosis occurs

Serum levels within 4 hours of AMI

Troponin I - inhibits myosin ATPase

Troponin T - binds to tropomyosin

Troponin C - binds to calcium

Hyperhomocysteinemia

Homocysteine levels are often elevated by 15-40% in patients with coronary artery disease

Normal levels are less than 16 micromols/liter

Treated with folic acid, pyridoxine or vitamin B12

BNP - Brain Natriuretic Peptide

- Neurohormone produced in the LV in response to pressure and volume
- Up-regulated in patients with heart failure
- Resulting in vasodilation and diuresis/natriuresis Detect asymptomatic CHF
- Elevated BNP - hypertension, tachycardia, cardiomyopathy, MI, mitral and aortic stenosis
- Predict 30-day and 10-month mortality after AMI

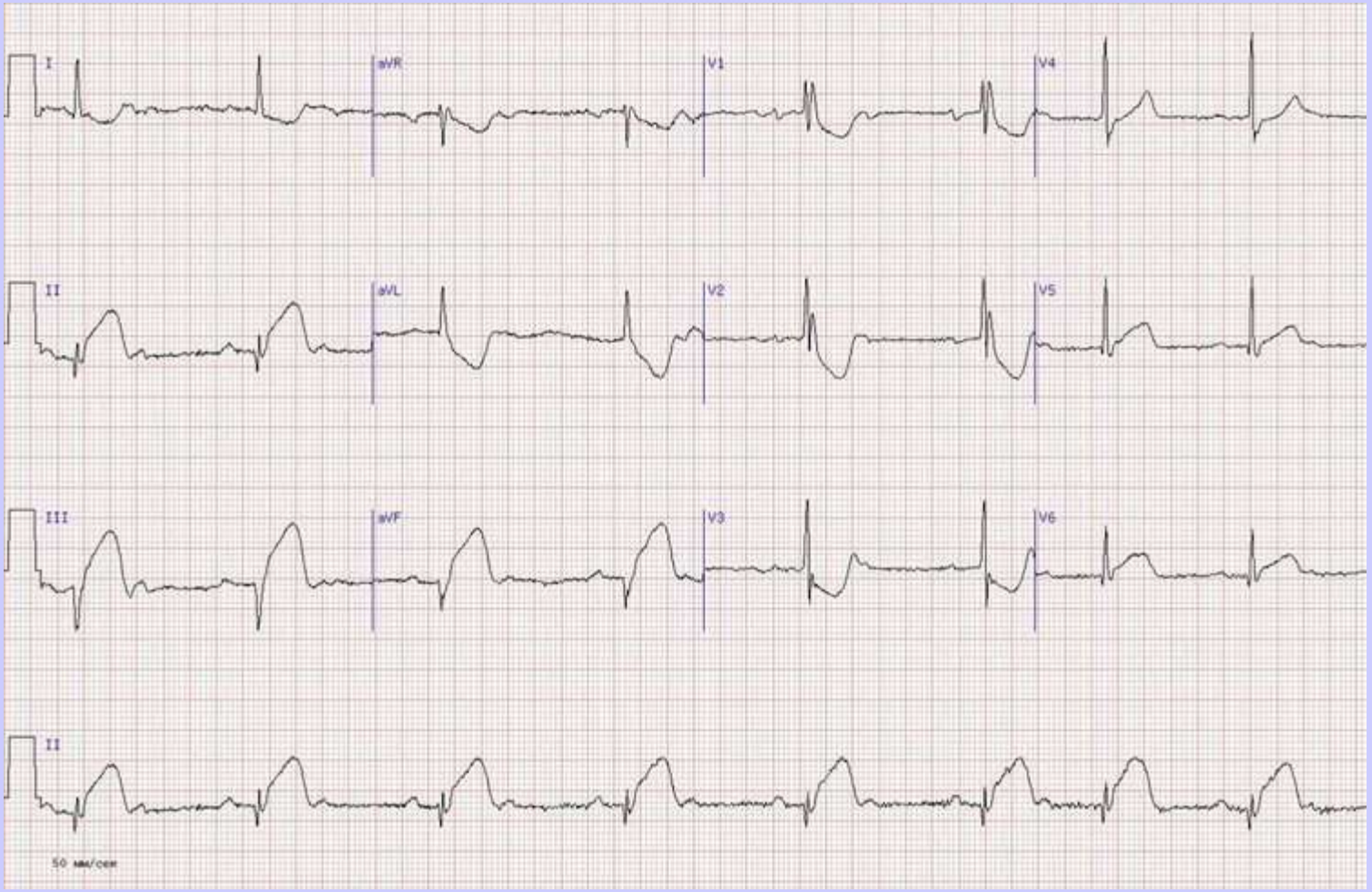
hs-C Reactive Protein

(hs = high sensitivity)

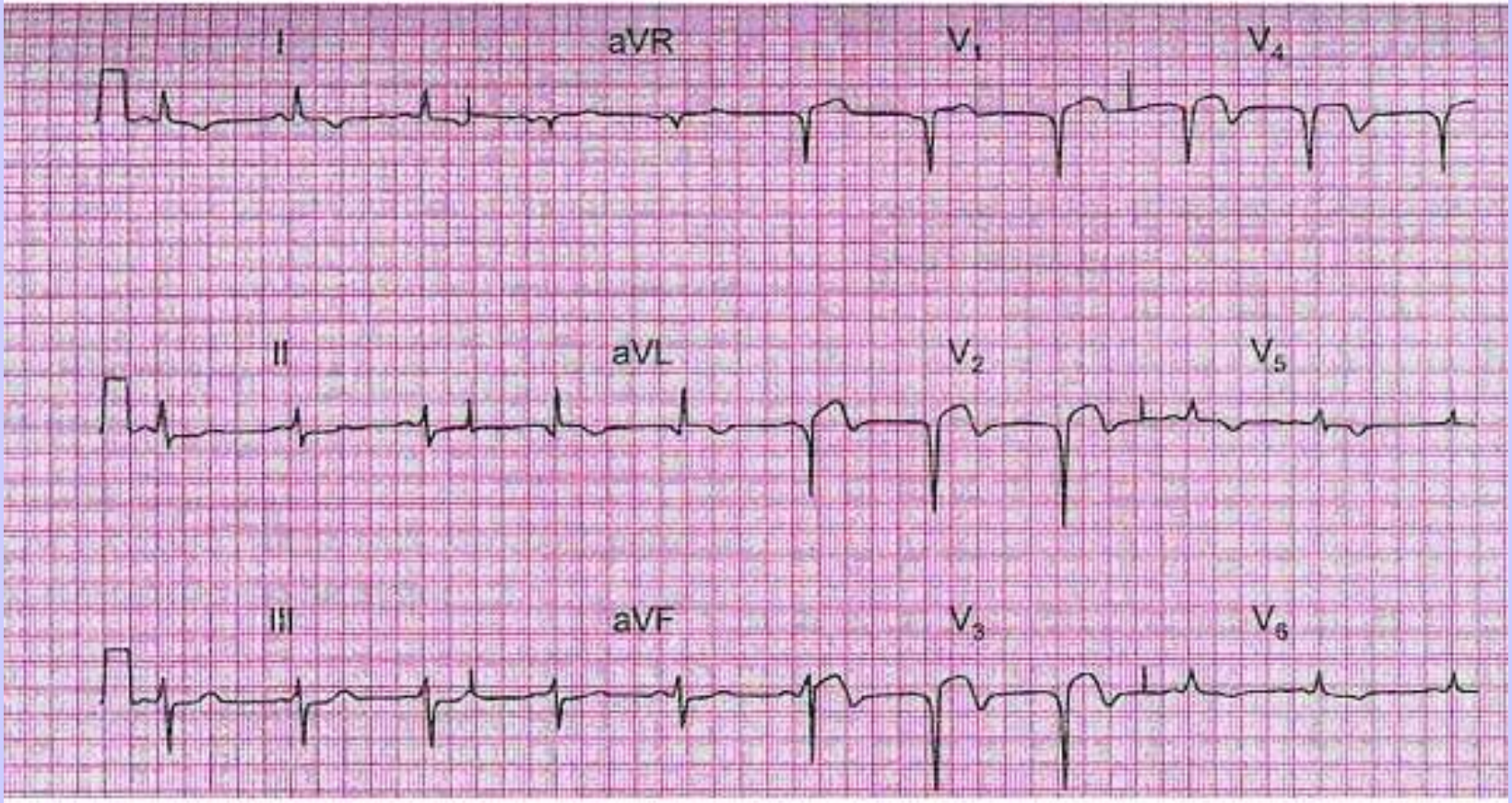
- Independent risk factor for first MI and ischemic stroke in healthy people
- Not associated with risk of venous thrombosis
- Short term risk factor in patients with unstable angina and long term risk factor in patients for MI and ischemic stroke occurring six or more years later

hs-C Reactive Protein

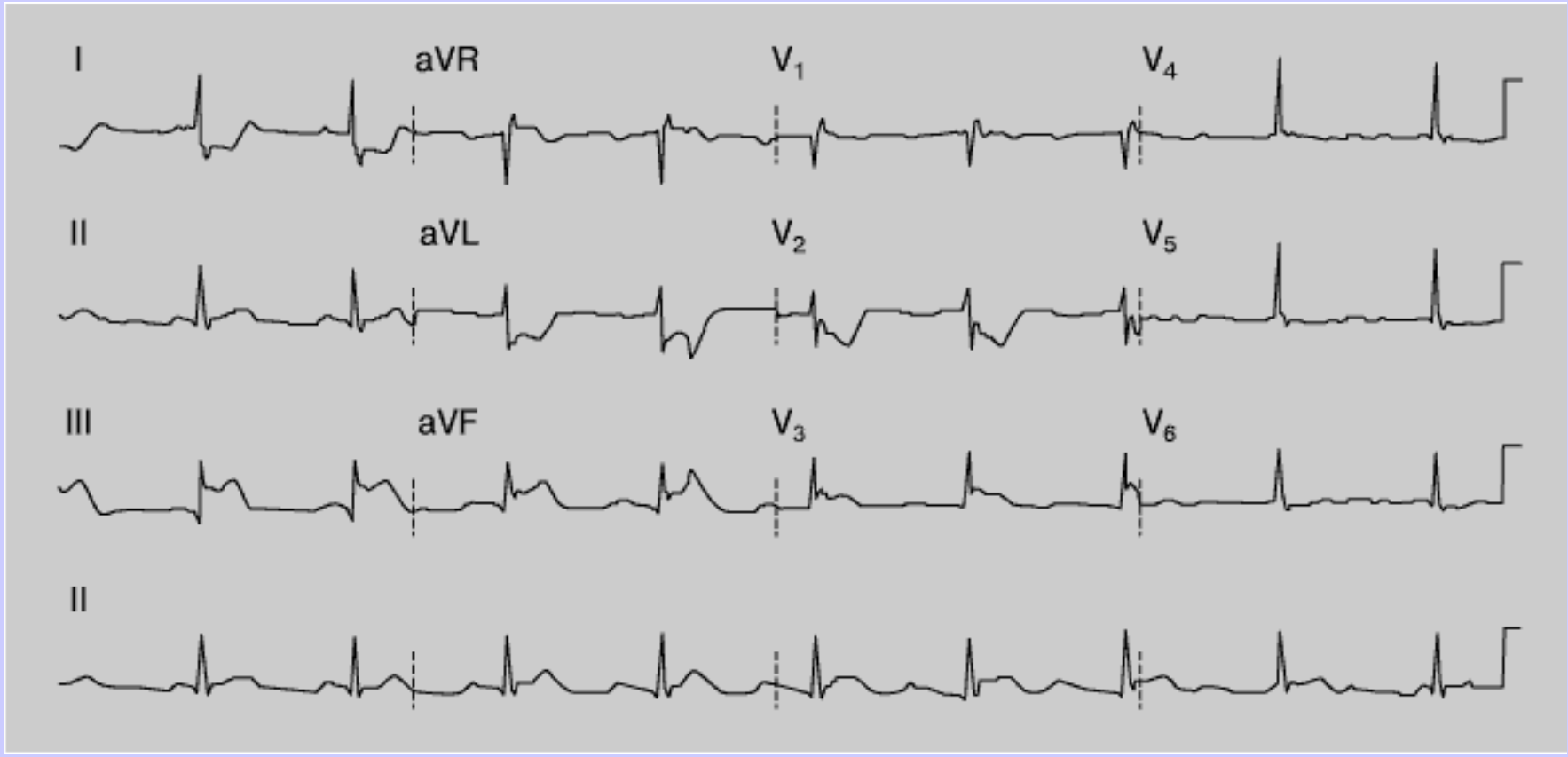
- Inflammation mediated by a chronic process and excludes undetected acute illness
- Aspirin and other antiinflammatory agents may have a role in preventing cardiovascular disease
- May predict patients who will benefit from aspirin or other antiinflammatory therapy


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
50 mm/sec

?


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

?


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

?


Thank you for attention!