ISCHEMIA HEART DISEASES

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Angina pectoris

- Is a clinical syndrome characterized by paroxysmal chest pain due to transient myocardial ischemia . It may occur whenever there is imbalance between myocardial oxygen supply and demand the most common cause is atherosclerosis .however angina may also develop in aortic stenosis and hypertrophic cardiomyopathy even there is no coronary atheroma

- precipitated by the " E's"
- Emotional stress
- Exertion
- Exposure to very hot or cold temperatures
- Eating (Heavy meals)

Higher-risk signs in MI or unstable Angina (USA) . Signs include the following:

- Systolic blood pressure less than 100 mm Hg or overt hypotension
- Elevated jugular venous pressure
- Presence of a third or fourth heart sound
- New or worsening apical systolic murmur due to papillary muscle dysfunction
- Rales or crackles

Summary :

Acute coronary syndrome (ACS) refers to acute **myocardial ischemia** and/or infarction due to partial or complete occlusion of a **coronary artery**. There are three clinical entities grouped under ACS: **unstable angina pectoris**, non-ST-segment elevation **myocardial infarction** (**NSTEMI**), and ST-segment elevation **myocardial infarction** (**STEMI**). These conditions are often difficult to distinguish from one another based on clinical symptoms alone and require **ECG** and **cardiac biomarker** measurement to diagnose.

Typical cardiac **chest pain** is substernal in nature, often described as a feeling of pressure, and is relieved with rest and/or **nitrate** use. The **pain** may radiate to the left **jaw**, neck, epigastrium, upper back, and/or left arm. Additionally, autonomic symptoms such as diaphoresis, nausea, and vomiting are common. **ECG** and laboratory tests are important diagnostic tools in the initial evaluation.

In contrast to **angina pectoris**, **NSTEMI** and **STEMI** are characterized by the destruction of **cardiac muscle** tissue, which results in elevated **cardiac enzymes** in the blood (i.e., the elevation of **troponin** after 3–4 hours). Unlike **unstable angina** and **NSTEMI**, **STEMI** results in specific **ECG** changes (e.g., ST-segment elevation), which can help to determine the location and stage of the infarct.

The need for revascularization with either **fibrinolysis** or **cardiac catheterization** should be evaluated immediately, as revascularization significantly affects the prognosis of patients with **myocardial infarction**. **Cardiac catheterization** should be performed as soon as possible in **STEMI** and electively within 2–72 hours in high-risk **NSTEMI** and/or **unstable angina**.

Medical **management of ACS** includes anticoagulation, analgesics, and **antiplatelet agents**. Complications of ACS include **congestive heart failure**, **papillary muscle rupture**, **arrhythmias**, and sudden cardiac death. Subsequent management and **secondary prevention** of ACS depends on the presence of comorbidities, but most patients should be started on indefinite **aspirin** and **statin** therapy.

Summary :

Definition

Myocardial infarction: **myocardial cell** death caused by prolonged **ischemia**

Acute coronary syndrome: suspicion or confirmed presence of
acute **myocardial ischemia** and/or **myocardial infarction**

Further classified as **unstable angina**, **NSTEMI**, and **STEMI**

Sudden cardiac death (SCD): sudden, unexpected death caused by loss of cardiac function (most commonly due to lethal **arrhythmia**, e.g., **ventricular fibrillation**)

Acute Coronary Syndrome

- It consist of

–**Myocardial Infarction: ST-elevation myocardial infarction (STEMI) or Non-ST-elevation myocardial infarction (NSTEMI)**

- symptoms occur at rest
- evidence of myocardial necrosis - increased **cardiac troponin** or **Creatine kinase-MB** isoenzyme

–**Unstable angina (UA).**

- new-onset or rapidly worsening angina (crescendo angina),
- angina on minimal exertion
- or angina at rest in the absence of myocardial damage

Incidence

♂ > ♀ (3:1)

Risk factors: See atherosclerosis.

- Increasing age
- Male gender
- Personal history of angina and/or known coronary artery disease
- Family history of CAD
- Diabetes mellitus
- Systolic hypertension
- Tobacco use
- Hyperlipidemia

Table 5. Risk Factors for Atherosclerotic Heart Disease

Non-modifiable Risk Factors	Modifiable Risk Factors
Age	Hyperlipidemia*
Male, postmenopausal female	Hypertension (HTN)*
Family history (FHx) of MI*	Diabetes mellitus (DM)*
First degree male relative <55	Cigarette smoking*
First degree female relative <65	Metabolic syndrome
	Obesity
	Sedentary lifestyle
	Heavy alcohol intake

* Major risk factor

Etiology

Most common cause: coronary artery **atherosclerosis**

Less common

- Coronary artery dissection
- **Coronary artery vasospasm** (e.g., Prinzmetal angina, cocaine use)
- Vasculitis (e.g., polyarteritis nodosa, Kawasaki syndrome)
- Myocardial oxygen supply-demand mismatch
  - Hypotension
  - Severe anemia
  - Hypertrophic cardiomyopathy
  - Severe aortic stenosis

	Unstable angina	Non-ST-segment elevation myocardial infarction (NSTEMI)	ST-segment elevation myocardial infarction (STEMI)
Description	•Acute myocardial ischemia that is not severe enough to cause detectable quantities of myocardial injury biomarkers or ST-segment elevations on ECG	•Acute myocardial ischemia that is severe enough to cause detectable quantities of myocardial injury biomarkers but without ST-segment elevations on ECG	•Acute myocardial ischemia that is severe enough to cause ST-segment elevations on ECG

	Unstable angina	Non-ST-segment elevation myocardial infarction (NSTEMI)	ST-segment elevation myocardial infarction (STEMI)
Clinical presentation	•**Angina** at rest or with minimal exertion •New-onset angina •Severe, persistent, and/or worsening angina (crescendo angina) •**Autonomic symptoms** may be present: diaphoresis, syncope, palpitations, nausea, and/or vomiting		

	Unstable angina	Non-ST-segment elevation myocardial infarction (NSTEMI)	ST-segment elevation myocardial infarction (STEMI)
Pathophysiology	•**Partial occlusion** of coronary vessel → decreased blood supply → ischemic symptoms (also during rest)	•Classically due to partial occlusion of a coronary artery •Affects the inner layer of the heart (**subendocardial infarction**)	•Classically due to complete occlusion of a coronary artery •Affects full thickness of the myocardium (**transmural infarction**)
Cardiac biomarkers	•**No elevated cardiac biomarkers**	•**Elevated cardiac biomarkers** (e.g., troponin)	•Cardiac biomarkers usually elevated (e.g., troponin)

	Unstable angina	Non-ST-segment elevation myocardial infarction (NSTEMI)	ST-segment elevation myocardial infarction (STEMI)
ECG findings	•**Normal**	•Normal or nonspecific (e.g., ST depression, loss of R wave, or T-wave inversion) •**No ST elevations**	•**ST elevations** (in two contiguous leads) or new **left bundle branch block**
Treatment	•Anticoagulation, aspirin, ADP receptor inhibitor •Invasive management depends on risk stratification (TIMI score).		•**Immediate revascularization** •Anticoagulation, aspirin, ADP receptor inhibitor

Pathophysiology

ACS is most commonly due to unstable plaque formation and subsequent rupture.

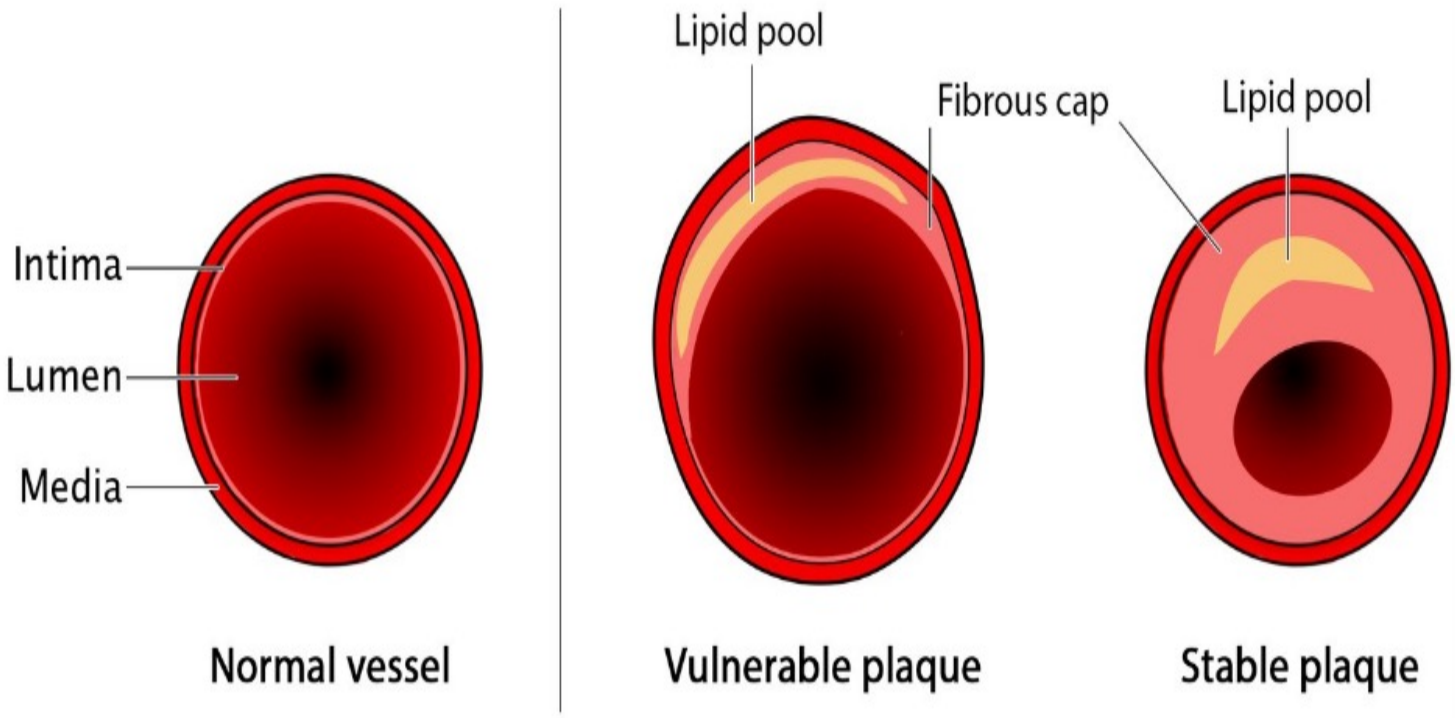
Plaque formation and rupture

For plaque formation, see principles of coronary heart disease and atherosclerosis.

Stable atherosclerotic plaque: manifests as stable angina (symptomatic during exertion)

Unstable plaques are **lipid-rich** and covered by **thin fibrous caps** → high risk of rupture

Inflammatory cells in the plaque (e.g., macrophages) secrete matrix metalloproteinases → breakdown of extracellular matrix → weakening of the fibrous cap → minor stress → rupture of the fibrous cap → exposure of highly thrombogenic lipid core → thrombus formation → coronary artery occlusion



Types of plaque in coronary heart disease

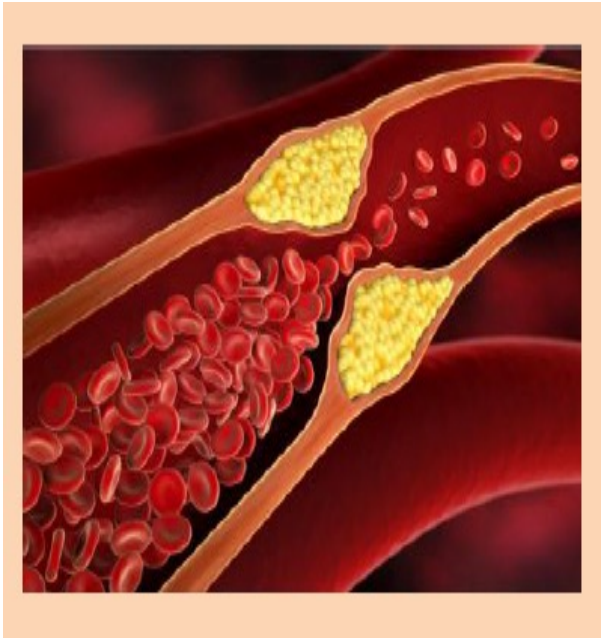
In a stable plaque, the lipid pool is contained by a stronger fibrous cap than in a vulnerable plaque. Despite the extensive narrowing of the lumen, the stable plaque is less likely to rupture and lead to myocardial infarction. In contrast, a vulnerable plaque hardly narrows the lumen, which makes it more difficult to distinguish from a physiological finding on angiogram.

Pathophysiology of myocardial ischemia

- Myocardial **oxigen supply is decreased**
  - Narrowed coronary arteries (sclerosis, thrombus, spasmus, coronary embolism, vasculitis)
  - Hypotension
  - Severe anemia
  - Methemoglobinemia, increased carboxyhemoglobin
- Myocardial **oxigen demand is increased**
  - Left ventricle hypertrophy
  - Fever
  - Hyperthyroidism
  - Tachycardy

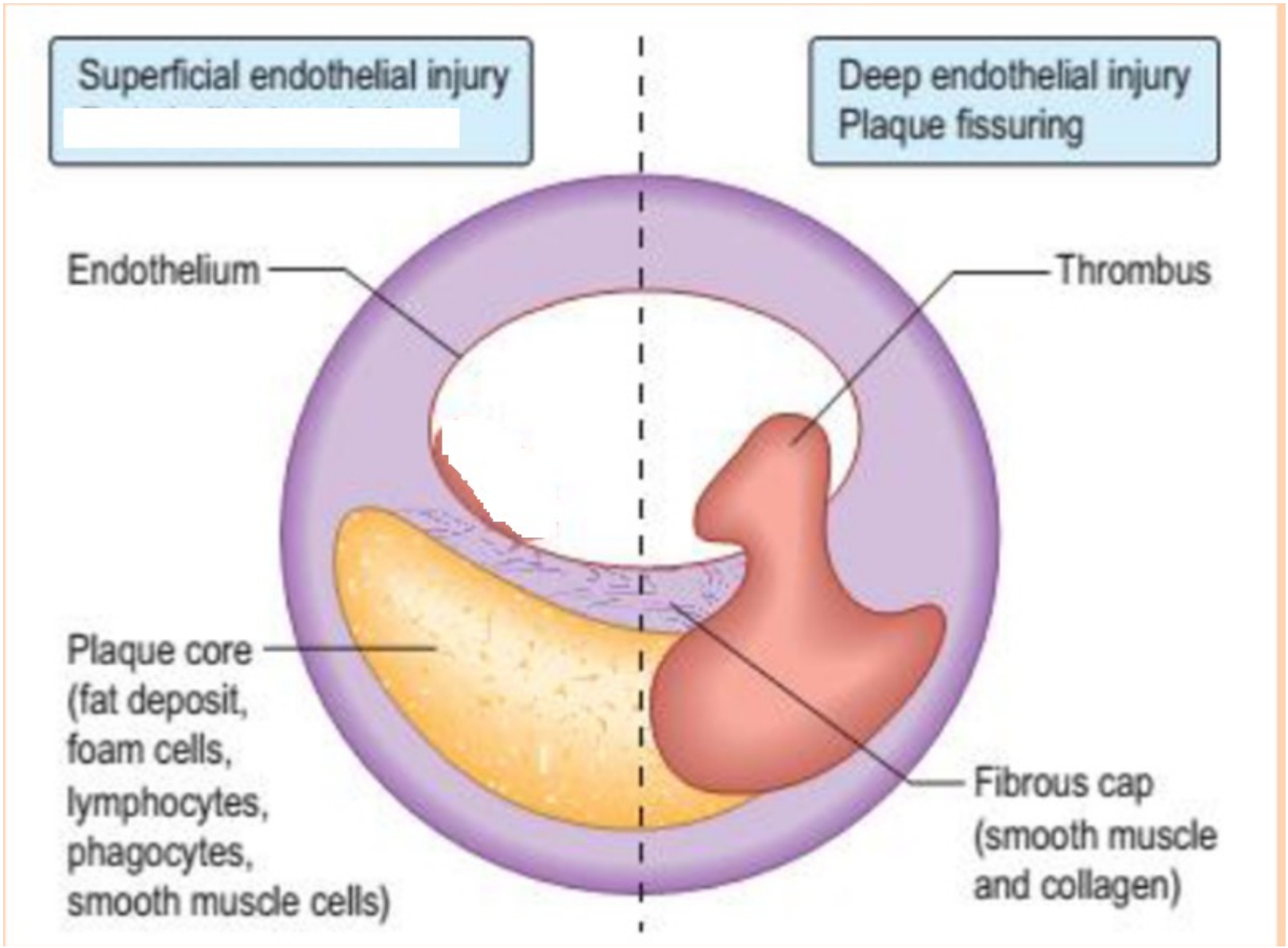
Atherosclerosis
The leading cause of ischemia

- Can affect any artery in the body
- **Heart**: angina, MI and sudden death;
- **Brain**: stroke and transient ischaemic attack;
- **Limbs**: claudication and critical limb ischaemia.

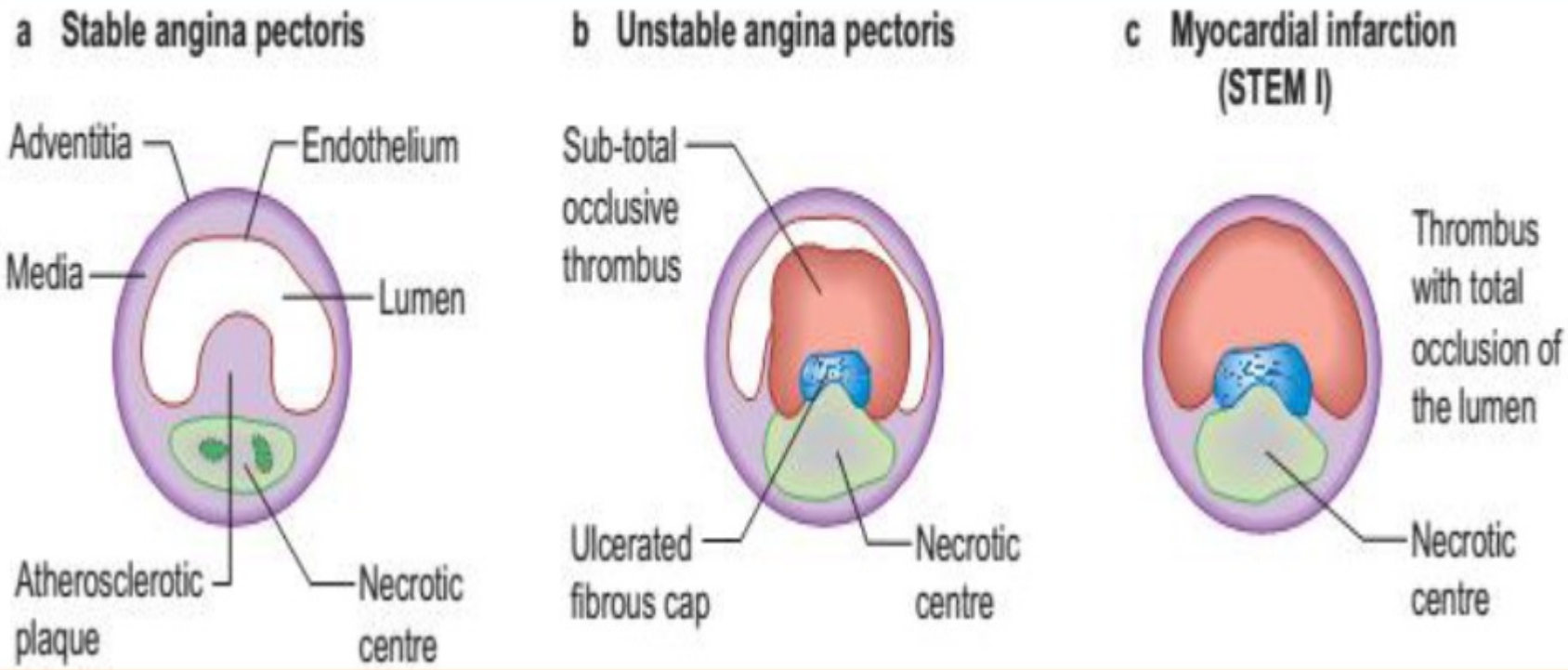


Atherosclerosis

- Progressive **inflammatory disorder** of the arterial wall characterised by focal lipid rich deposits of **atheroma**
- Remain clinically asymptomatic until
  - large enough to impair tissue **perfusion**,
  - Ulceration and disruption of the lesion result in **thrombotic occlusion**
- Clinical manifestations depend upon the **site of the lesion.**


Superficial endothelial injury
Deep endothelial injury
Plaque fissuring
Endothelium
Thrombus
Plaque core
(fat deposit,
foam cells,
lymphocytes,
phagocytes,
smooth muscle cells)
Fibrous cap
(smooth muscle
and collagen)

Advanced Atherosclerosis



Coronary artery occlusion

Partial coronary artery occlusion

Decreased myocardial blood flow → supply-demand mismatch → myocardial ischemia

Usually affects the inner layer of the myocardium (subendocardial infarction)

Typically manifests clinically as unstable angina and/or NSTEMI

Complete coronary artery occlusion

Impaired myocardial blood flow → sudden death of myocardial cells (if no reperfusion occurs)

Usually affects the full thickness of the myocardium (transmural infarction)

Typically manifests clinically as STEMI

Clinical features

Classic presentation

- Acute **retrosternal chest pain**
  - Typically described as **dull, squeezing** pressure and/or tightness
  - Commonly radiates to left chest, arm, shoulder, neck, jaw, and/or epigastrium
  - Precipitated by exertion or stress
  - See also angina.
  - The peak time of occurrence is usually in the morning (8–11 a.m.). [10]
- Dyspnea (especially with exertion)
- Pallor
- Nausea, vomiting
- Diaphoresis, anxiety
- Dizziness, lightheadedness, syncope

Other findings

Tachycardia, arrhythmias

Symptoms of CHF (e.g., orthopnea, pulmonary edema) or cardiogenic shock (e.g., hypotension, tachycardia, cold extremities)

New heart murmur on auscultation (e.g., new S_4)

Specific to inferior wall infarction

Epigastric pain

Bradycardia

Clear lung fields

Atypical presentation: minimal to no chest pain More likely in **elderly, diabetic individuals, and women**
Autonomic symptoms (e.g., nausea, diaphoresis) are often the chief complaint.
In patients with **diabetes**, chest pain may be completely absent (e.g., **silent MI**) due to polyneuropathy.

STEMI classically manifests acutely with more severe symptoms, while unstable angina/NSTEMI has a continuous course with milder symptoms.

The clinical manifestations of ischemic heart disease

- Ischemic heart disease **without clinical symptoms**. **Sudden death** can be the presenting manifestation.
- Cardiomegaly and heart failure that may have caused no symptoms prior the development of heart failure – **ischemic cardiomyopathy**.
- **Angina pectoris.** Stable angina pectoris.
- Unstable angina/Non ST-elevation myocardial infarction (NSTEMI)/STEMI = **acut coronary syndromes**

Angina pectoris

- Angina pectoris is the most common manifestation of the ischemic heart disease.
- The prevalence of angina pectoris is 16% in male and 11% in female population between 65 and 74 year of age.

The context of the symptom development can give clues to diagnosis and management

- Effort angina
  - Angina, which occurs predictably at a certain level of activity – **stable exertional pectoris**
  - Angina only after minor exertion (a short walk or shaving) in the morning: **first effort or warm-up angina** The patient by midday may capable of much greater effort without symptoms.
  - Emotional stress situation, haevy meal, exposure to cold, or smoking induced angina
- Angina (1) that occurs at rest or with minimal exertion, usually lasts more than 10 min, (2) is severe and new of onset, and/or (3) that occurs with a crescendo pattern – more severe, prolonged, or frequent than previously - **unstable angina, acute coronary syndrome**
- Focal spasm of an epicardial coronary artery (usually close to a noncritical obstruction of right coronary artery) leading to severe myocardial ischemia. It occurs at rest, and associated with transient ST-segment elevation. **Prinzmetal's variant angina**

The typical clinical features of angina pectoris

- The typical location of pain is **retrosternal.**
- When the patient is asked to localize the sensation, he or she will typically place their hand over the sternum, somtetimes with a clenched fist, to indicate the squezzing. **The pain can not be localized with one finger**.
- Usually described as **heaviness, pressure, squezzing**, or choking.
- Usually associates with **gradual intensification** of symptoms over a period of minutes.
- It **lasts** typically **2-5 min**.
- It can **radiate to** either **shoulder** and to **both arms** (especially the ulnar surfaces of the forearm and hand.
- It can also arise in or radiate to the back, interscapular region, root of neck, jaw, teeth, and epigastrium. Rarely localized below the umbilicus or above the mandible.
- Exertional angina is typically **relieved by rest and nitroglycerin.**

Associated symptoms and physical signs of angina pectoris

- **Associated symptoms**
  - Dyspnoe
  - Fatique, faintness
  - Nausea, vomiting
  - Sweating
  - Sense of impending doom (mostly in case of myocardial infarction)
- **Physical signs**
  - Third and fourth heart sounds
  - Apical systolic murmur due to mitral regurgitation (impaired papillary muscle function)
  - Pulmonary congestion

Summary of the characteristics of angina pectoris

- **Typical angina pectoris**:
  - Retrosternal chest pain (discomfort)
  - Complaints occur after exertion or emotional stress
  - The pain is relieved by rest and nitroglycerin

- **Atypical angina pectoris**: only two from three characteristics (especially in women and diabetics, angina may be atypical in location and not strictly related to provocing factors)

Cardial and extracardial causes of chest discomfort

- **CARDIOVASCULAR DISEASES**
  - Ischemic heart disease
  - Pericarditis
  - Aortic dissection
  - Aortic stenosis and regurgitation
  - Hypertrophic cardiomyopathy
  - Pulmonary hypertension
- **LUNG DISEASES**
  - Pulmonary embolism
  - Pneumothorax
  - Pleuritis
- **GASTROESOPHAGEAL DISEASES**
  - Gastroesophageal reflux
  - Esophageal motility disorders
  - Paptic ulcer
  - Gallstones
- **NEUROMUSCULOSKELETAL DISEASES**
  - Fracture of a rib
  - Spondylarthrosis
  - Intercostal muscle cramp
  - Tietze’ s syndrome
- **MISCELLANEOUS**
  - Subphrenic abscess
  - Herpes zoster
  - Splenic infraction

Differencial diagnosis of chest discomfort

- **Acute myocardial infarction**
  - The duration of the pain often more than 30 min
  - Often more severe than angina
  - Unrielived by nitroglicerin
  - May be associated with evidence of heart failure or arrhythmia
- **Aortic dissection**
  - Tearing, ripping pain with abrupt onset
  - Associated with hypertension, and/or connective tissue disorder
  - Depending on the location of dissection:
    - Loss of peripheral pulse
    - Pericardial tamponad
    - Murmur of aortic insufficiency

Differencial diagnosis of chest discomfort

- **Pericarditis**
  - The duration of the pain is hours to days
  - Sharp, retrosternal pain that is aggravated by coughing, deep breath, or changes in body position (relieved by sitting and leaning forward)
- **Pulmonary embolism**
  - Abrupt onset of the pain. Location is often lateral
  - Associated symptoms are dyspnea, tachycardy,and occasionally hemoptysis
- **Pneumothorax**
  - Sudden onset of pleuritic chest pain. Location:lateral to side of pneumothorax
  - Dyspnea, decreased breath sounds, tympanic percussion sound.
- **Pneumonia or pleuritis**
  - Localized sharp, knifelike pain
  - Pain is aggravated by inspiration and coughing
  - Dyspnea, fever, rales, occasionally pleural rub

Differencial diagnosis of chest discomfort

- **Esophageal reflux**
  - Deep, burning discomfort that may be exacerbated by alcohol, aspirin, or some foods
  - Worsened by postprandial recumbency, relieved by antacids
- **Ulcer disease**
  - Symptoms do not associated with exertion
  - Prolonged burning pain
  - Typically occurs 60 to 90 min after meals.

ECG

12-lead ECG is the best initial test if ACS is suspected.

Dynamic changes require serial ECG evaluation.

Compare to prior ECGs (if available).

ECG changes in STEMI

Acute stage: myocardial damage ongoing

- Hyperacute T waves ("**peaked T wave**")
- **ST elevations** in two contiguous leads with reciprocal ST depressions

Intermediate stage: myocardial necrosis present

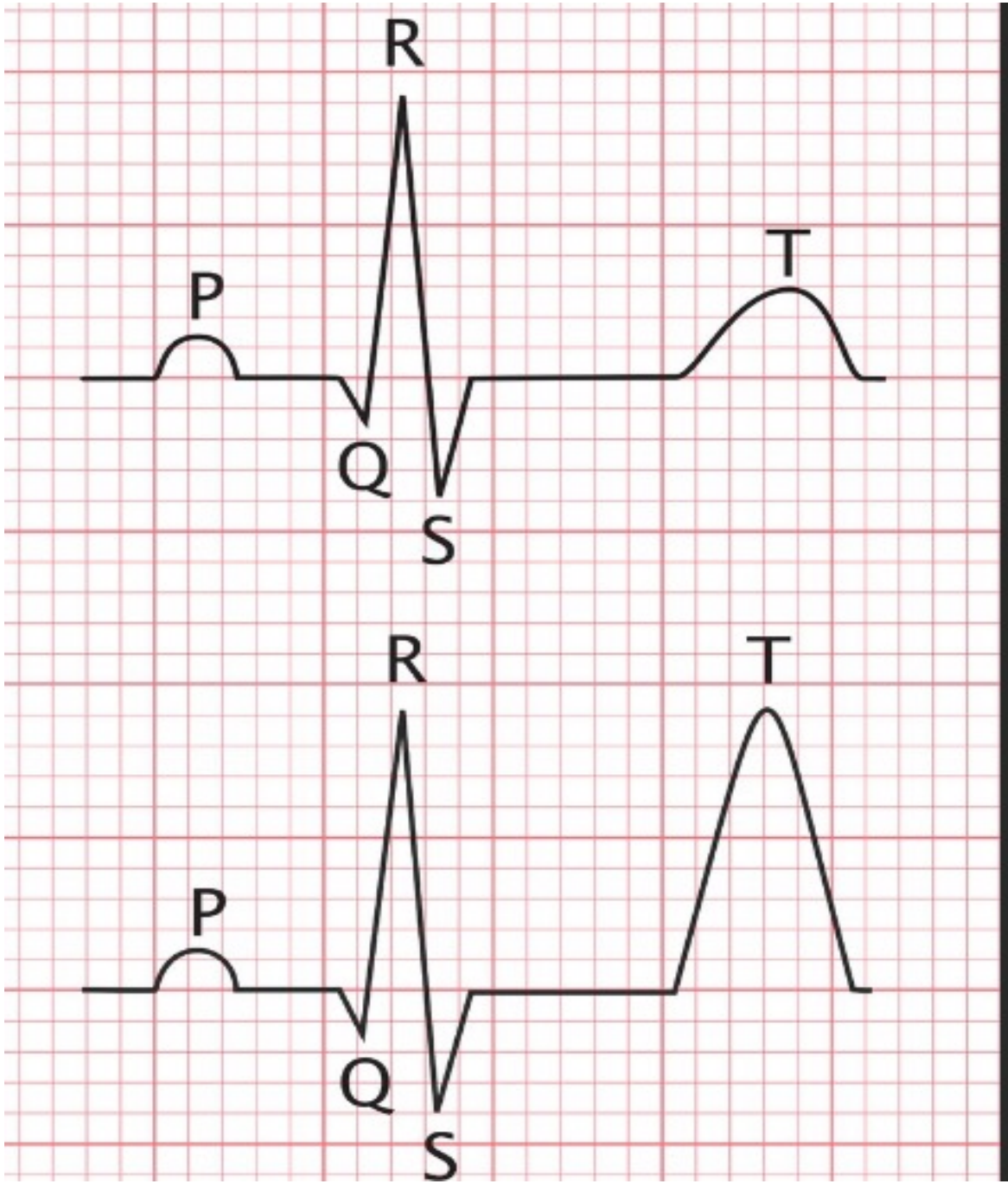
- Absence of R wave
- T-wave inversions
- Pathological Q waves

Chronic stage: permanent scarring

- Persistent, broad, and deep **Q waves**
- Often incomplete recovery of R waves
- Permanent T wave inversion is possible.

The sequence of ECG changes over several hours to days: hyperacute T wave → ST elevation → pathological Q wave → T-wave inversion → ST normalization → T-wave normalization

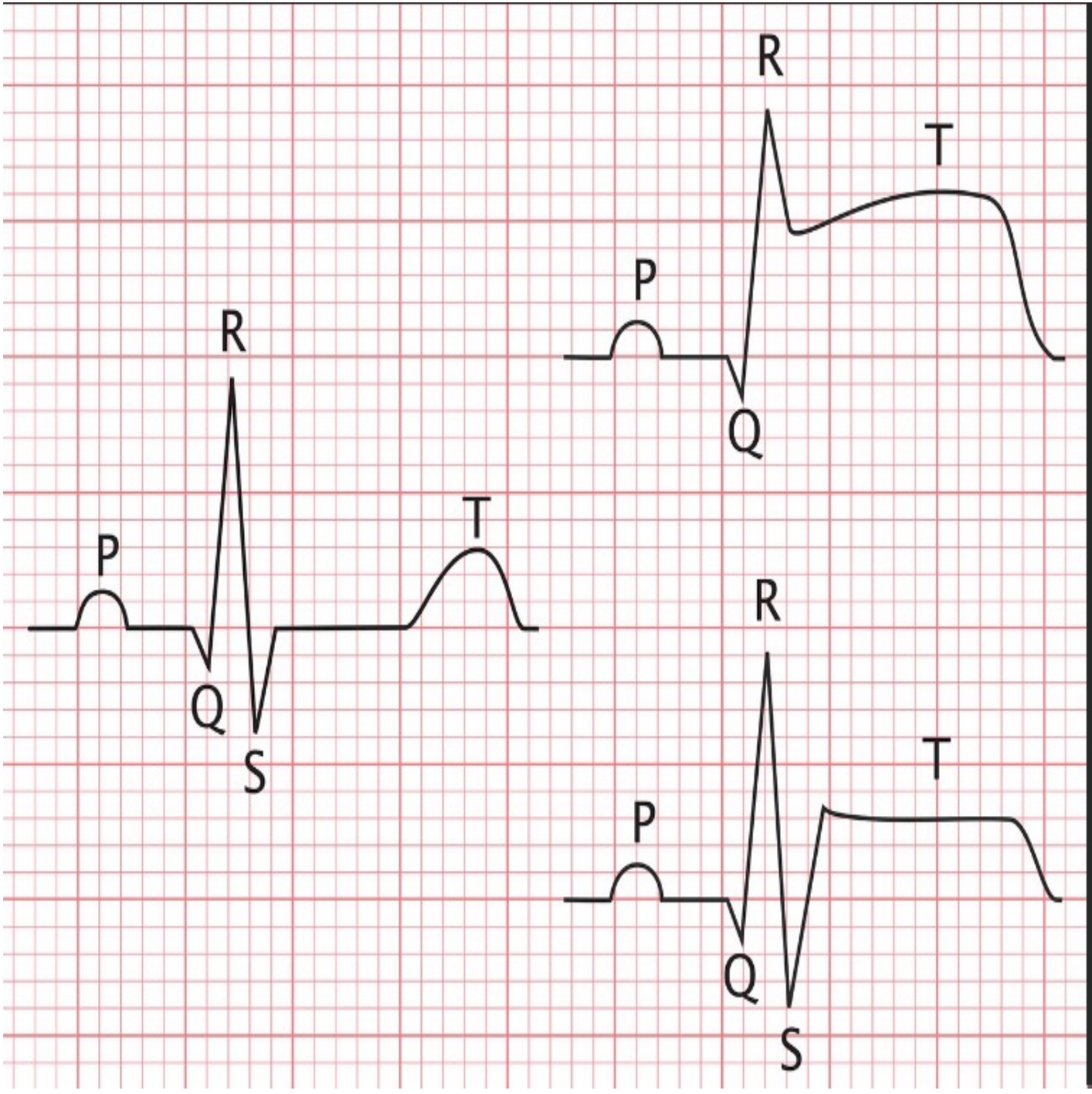
An acute left bundle branch block accompanied by symptoms of acute coronary syndrome is also considered an ST-elevation myocardial infarction (STEMI) because ST elevations cannot be adequately assessed in the setting of an LBBB.



Peaked T wave

Top: normal ECG

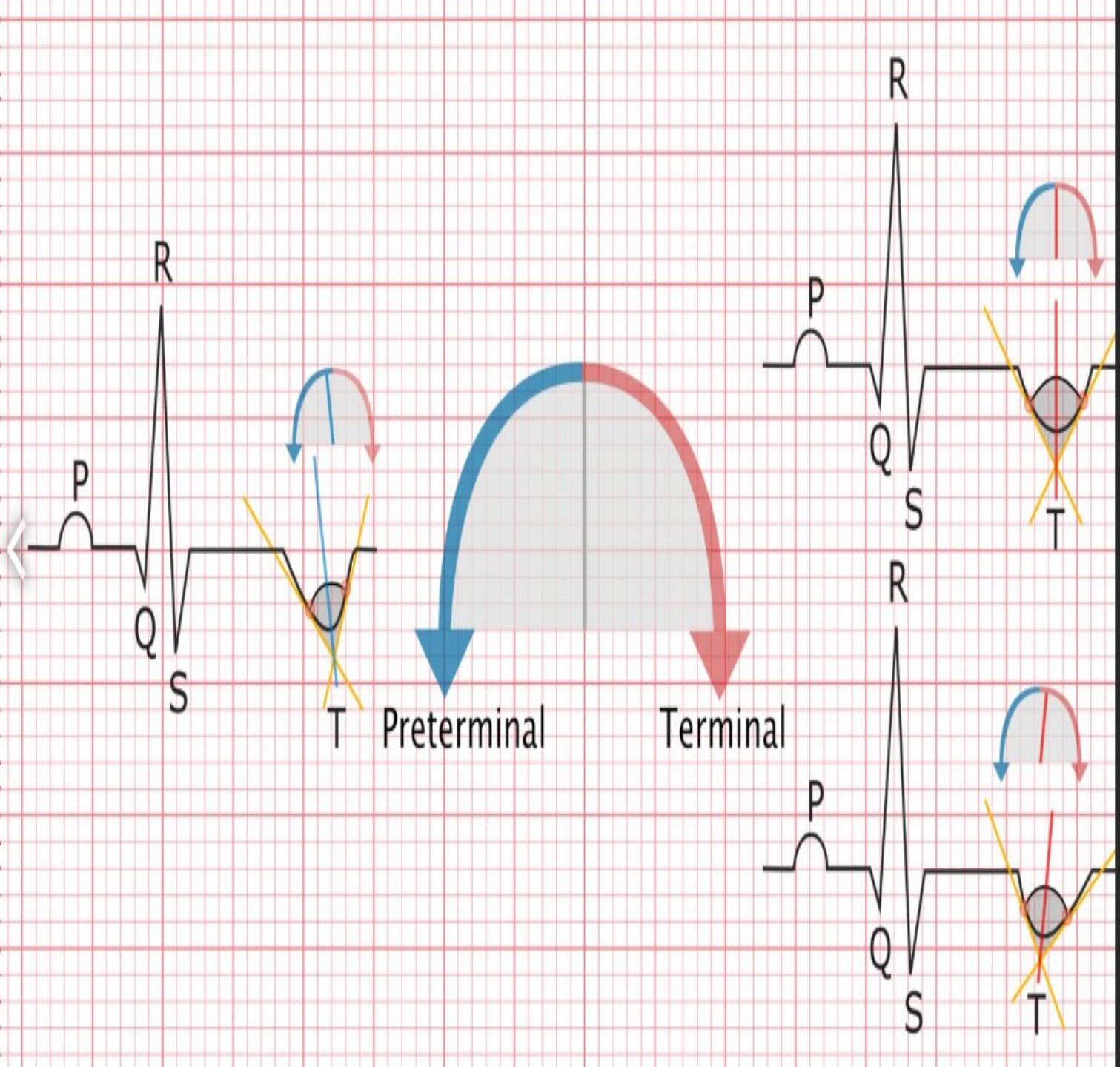
Bottom: significantly elevated, "tent-shaped" T wave. Physiological in patients with a high vagal tone; pathological in, e.g., hyperkalemia or may be an early sign of myocardial infarction.



Types of ST–segment elevation

Left: normal ECG
Top right: convex upward ST elevation from descending R
Bottom right: concave ST elevation from deep S

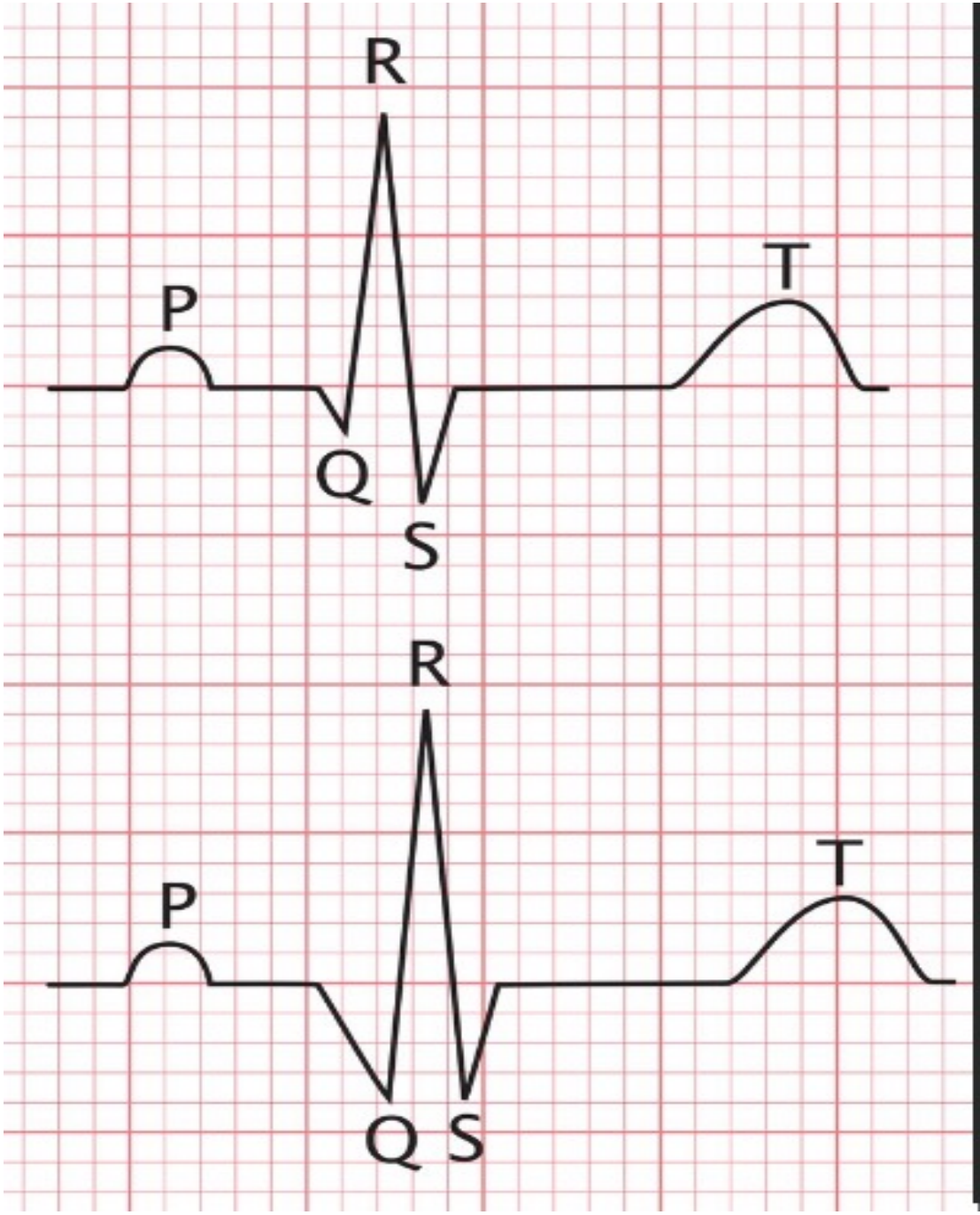
An ST elevation may be visible in various conditions including myocardial infarction, myocarditis, or pericarditis.



Types of T-wave inversion

To distinguish between terminal and preterminal T-wave inversion, first apply the tangents (yellow lines) to the legs of the T wave. Next, draw a line bisecting the inner angle (blue/red lines).

The upper end of the bisecting line, which points towards the R wave, indicates a preterminal T-wave inversion. If the bisecting line is perpendicular to the isoelectric line or if its upper end points away from the R wave, this indicates a terminal T-wave inversion.



Pathologic Q wave

Upper image: normal ECG
Lower image: pathological Q wave (duration ≥ 0.04 s; ≥¼ of preceding R wave)

A pathological Q wave is frequently seen after myocardial infarction or in hypertrophic cardiomyopathy.

ECG changes in NSTEMI/unstable angina

No ST elevations present

Nonspecific changes may be present.

- ST depression
- Inverted T wave
- Loss of R wave

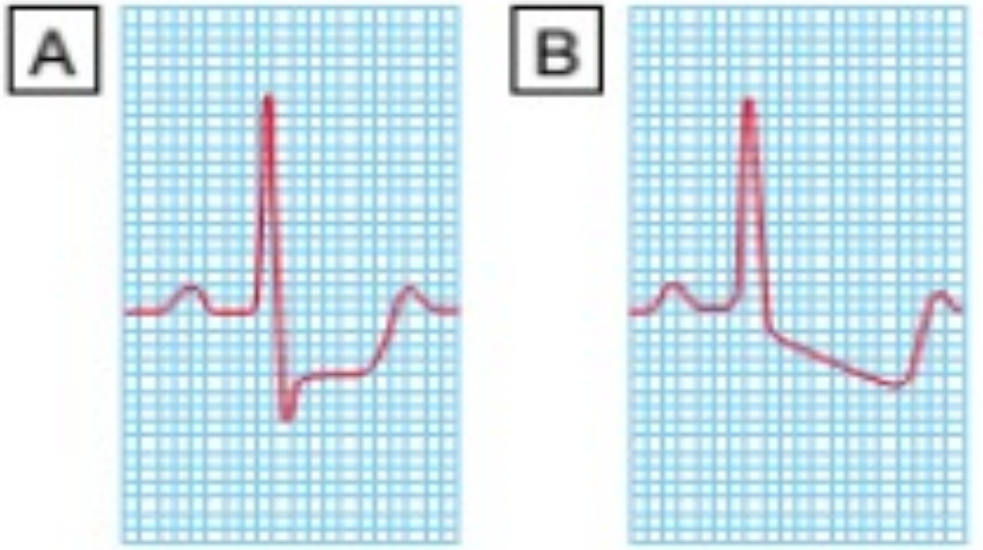
Localization of the myocardial infarct on ECG

ECG leads affected	Infarct location	Vessel involved
V_1–V_6	•Extensive anterior (Leads aVL and I can also be affected.)	•Proximal left anterior descending artery (LAD)
V_1–V_2	•**(Antero)septal**	•**LAD**
V_3–V_4	•**(Antero)apical**	•**Distal LAD**
V_5–V_6	•**(Antero)lateral**	•Diagonal branch of LAD •Distal LAD •**Left circumflex artery (LCX)** •In rare cases, can also be caused by right coronary artery (RCA) infarct
I, aVL	•Lateral	•Proximal LCX
II, III, aVF	•Inferior	•RCA (more common) •Distal LCX (less common)
V_3R–V_6R		
V_7-V_9	•Posterior/posterolateral	•Posterior descending artery (from RCA or LCX) •Reciprocal ST depressions in $V_{1–3}$ may also be seen

Infarction of the **anterior wall** is caused by obstruction of the **LAD** or its branches. Depending on the extent of anterior wall infarction, it results in ECG changes in the anterior wall leads ($\mathbf{V_{1–6}}$) and/or **I and aVL**. Infarction of the **inferior wall** is caused by obstruction of the **LCX** or **RCA** or their branches, and ECG changes are seen in leads **II, III,** and **aVF**.

To remember the ECG leads with maximal ST elevation in **anterior MI**, think **“SAL”**: “**S**eptal (V1–2), **A**pical (V3–4), **L**ateral (V5–6).

In severe transmural posterior wall infarction, there may not be any ST elevation on a standard 12-lead ECG.



Investigation

- **Resting ECG**

–may show evidence of previous MI but is often normal, even in patients with severe CAD.

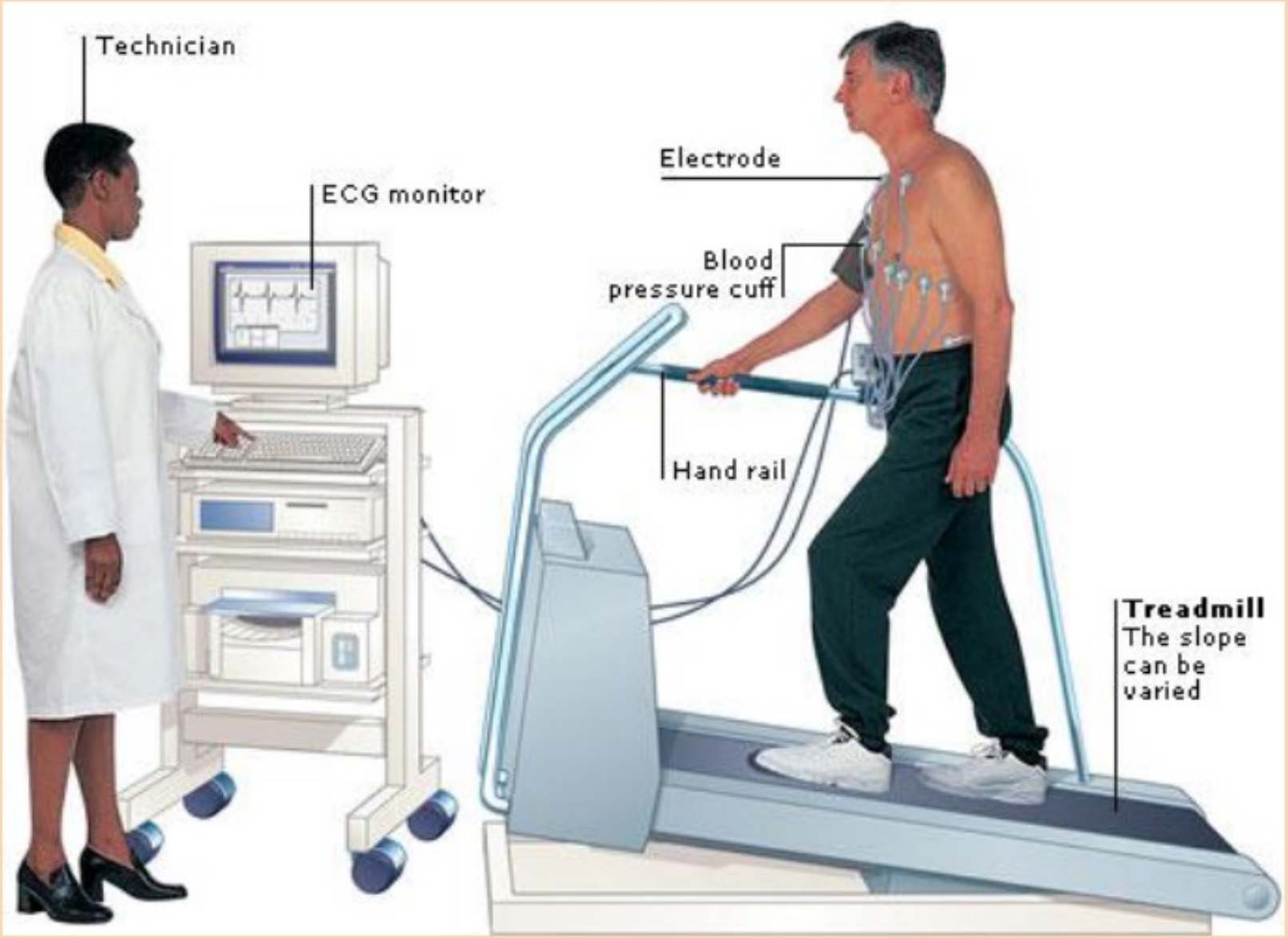
–The most convincing evidence of myocardial ischaemia - reversible ST segment depression or elevation, with or without T-wave inversion, at the time the patient is experiencing symptoms (during Angina or MI)

- **Exercise ECG**

–Exercise tolerance test (ETT) - standard treadmill or bicycle while monitoring the patient's ECG, BP and general condition.

–Planar or down-sloping ST segment depression of ≥ 1mm is indicative of ischaemia (**A, B**)

Investigation



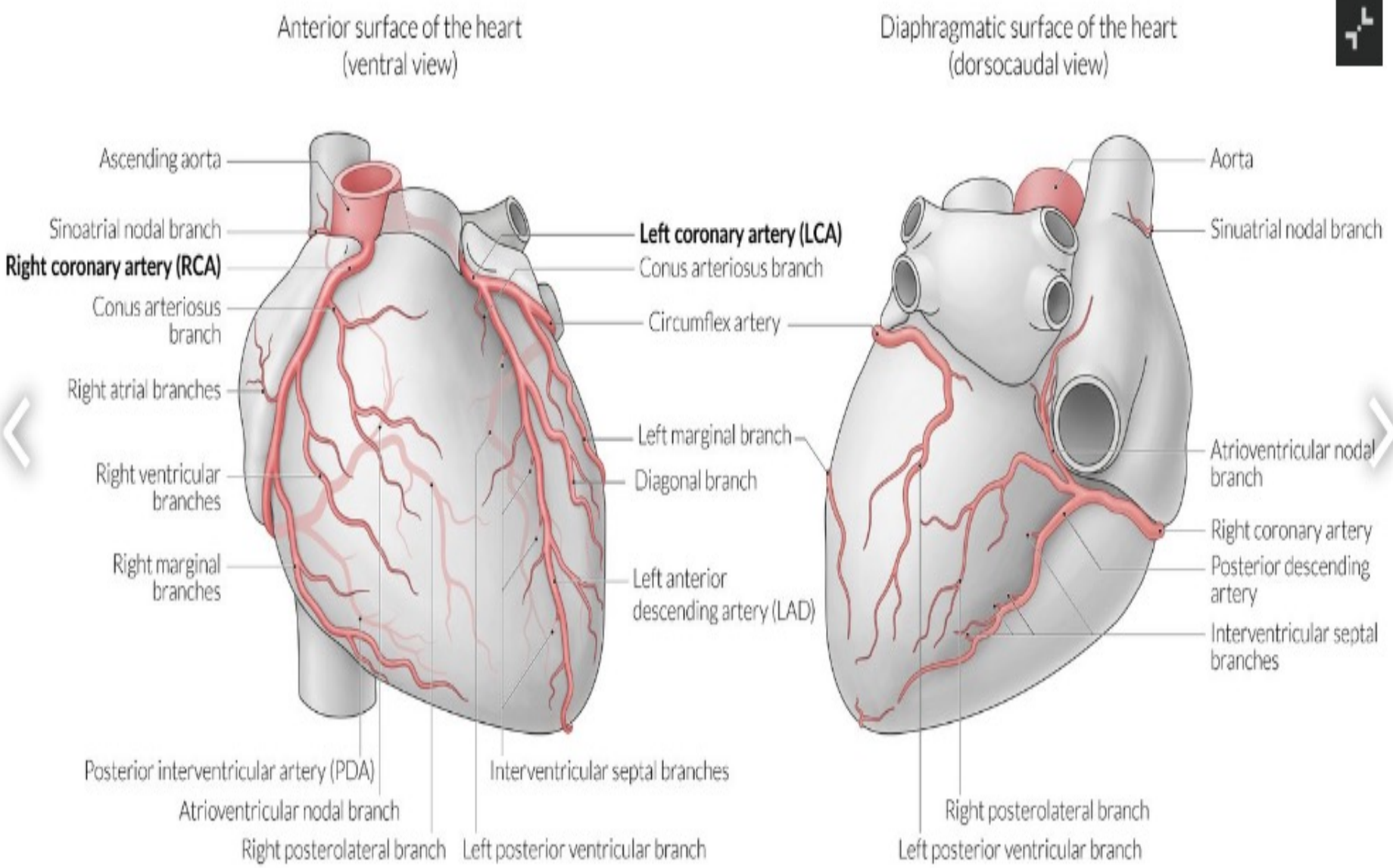
ECG in MI :

ST segment elevation / or depression , and/ or T inversion.

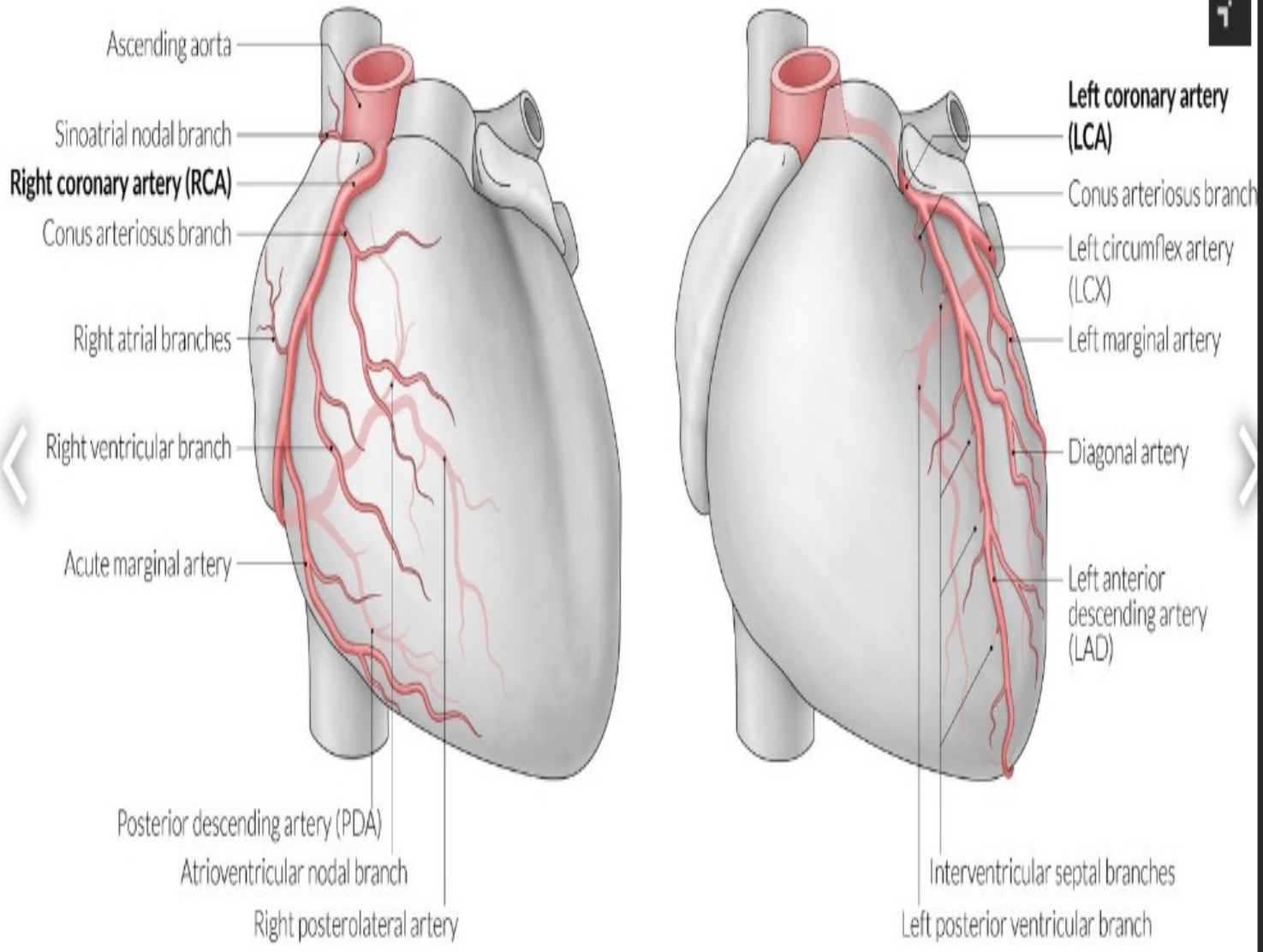
- Differential diagnosis of ST segment depression
- Myocardial Ischemia
- LVH
- Severe hypertension
- Cardiomyopathy
- Anemia
- Hypokalemia
- Digitalis effect

Differential diagnosis of ST segment elevation

- Myocardial infarction
- Prinzmetal's angina
- Ventricular aneurysm (post MI)
- Acute pericarditis


Anterior surface of the heart
(ventral view)
Ascending aorta
Sinoatrial nodal branch
Right coronary artery (RCA)
Conus arteriosus branch
Right atrial branches
Right ventricular branches
Right marginal branches
Left coronary artery (LCA)
Conus arteriosus branch
Circumflex artery
Left marginal branch
Diagonal branch
Left anterior descending artery (LAD)
Posterior interventricular artery (PDA)
Atrioventricular nodal branch
Right posterolateral branch
Left posterior ventricular branch
Interventricular septal branches
Diaphragmatic surface of the heart
(dorsocaudal view)
Aorta
Sinuatrial nodal branch
Atrioventricular nodal branch
Right coronary artery
Posterior descending artery
Interventricular septal branches
Right posterolateral branch
Left posterior ventricular branch

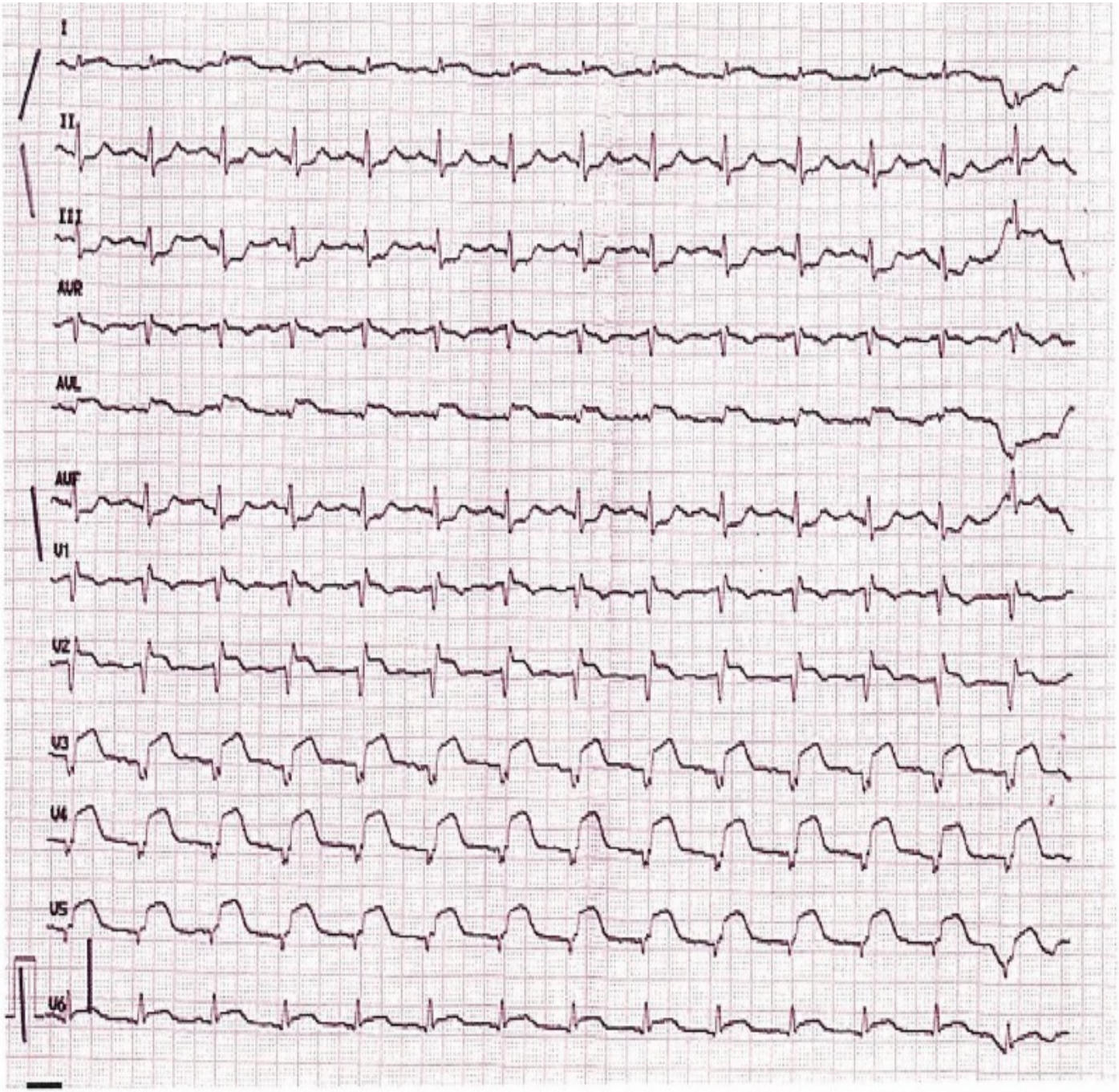
Coronary arteries



Coronary artery anatomy in the majority of the population:

Branches of the right coronary artery supply the sinoatrial node, the right atrium and ventricle, the atrioventricular node, approx. ⅓ of the interventricular septum, and a small part of the inferior surface of the left ventricle.

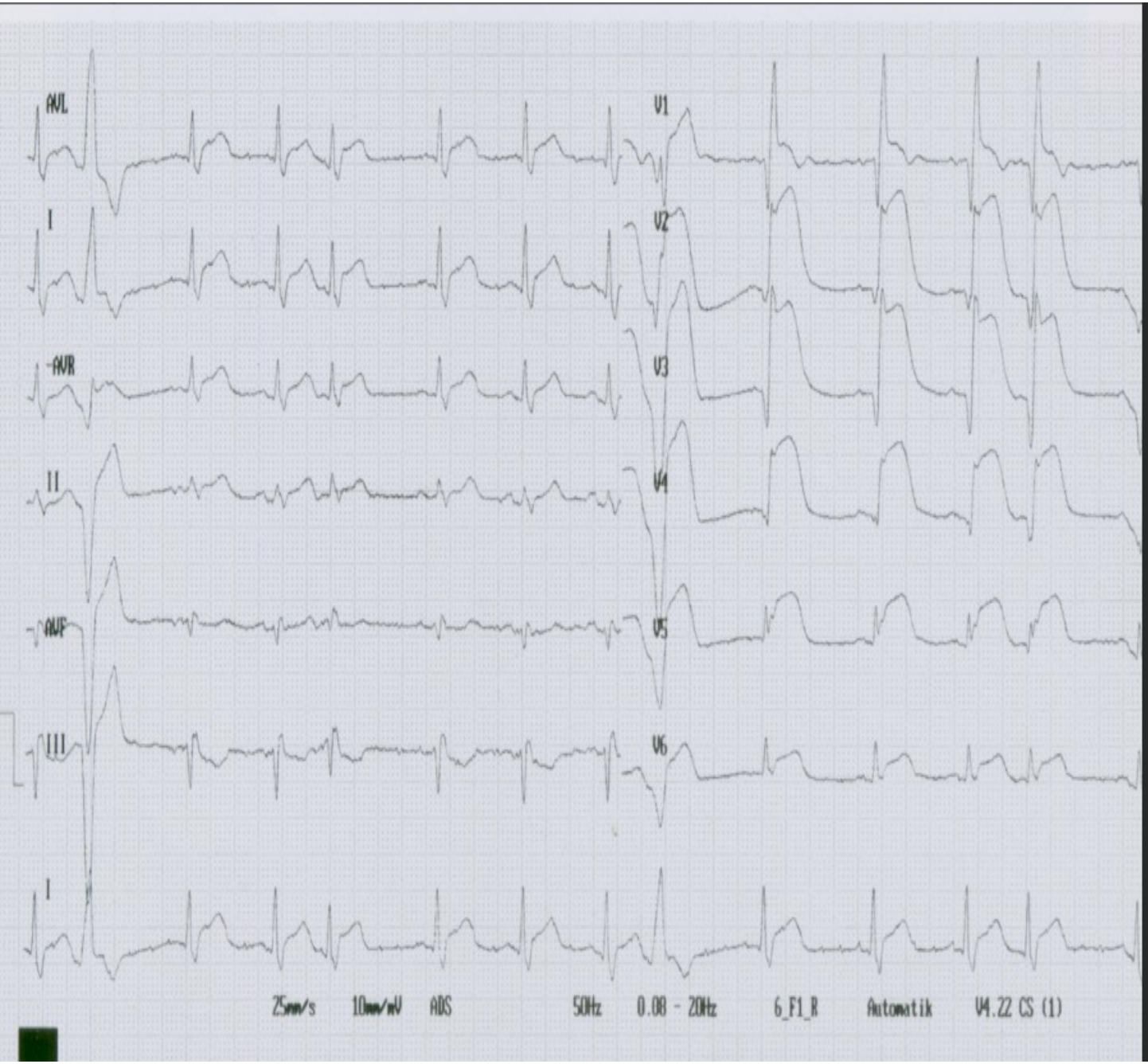
Branches of the left coronary artery supply the left atrium and ventricle, the anterior aspects of both ventricles, the cardiac apex, and ⅔ of the interventricular septum.



Acute anterior ST-elevation myocardial infarction (STEMI)

12-lead ECG (paper speed of 25 mm/s)

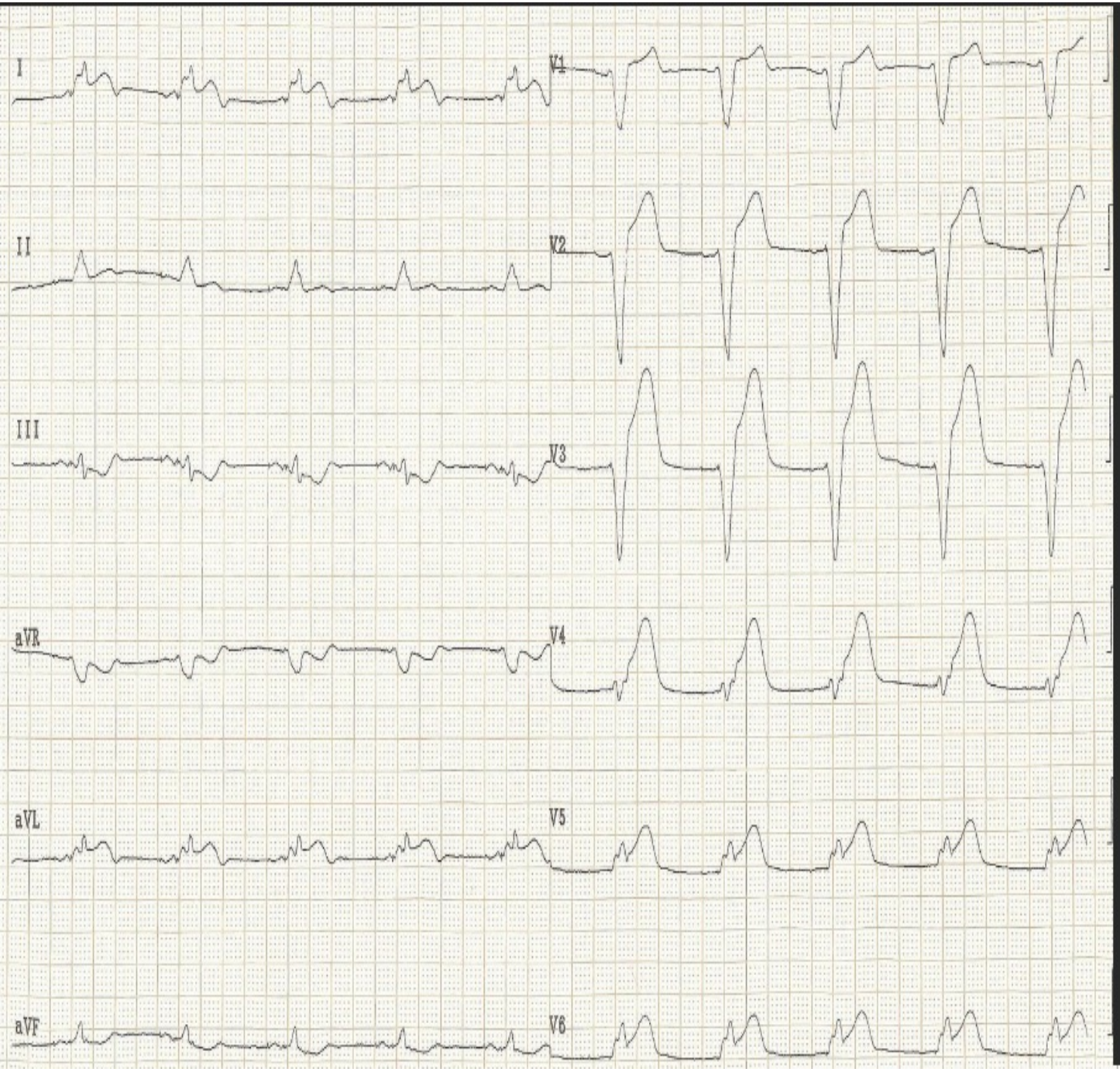
- Sinus rhythm with a heart rate of ~85/min
- Normal cardiac axis (leads I, II, and III positive, but difficult to interpret because of the ST elevation)
- ST elevation in V2-V6, AVL and slightly in I
- Reciprocal ST depression in leads II, III, and AVF



Acute anterior ST-elevation myocardial infarction (STEMI)

12-lead ECG (paper speed 25 mm/s)

- Sinus rhythm with a heart rate of ~90/min
- Left axis deviation (R > S in I, S > R in aVF)
- There are both ventricular (A) and supraventricular (B) extrasystoles.
- ST elevation in I, aVL and V1-V6. Note the pathological Q in V3.

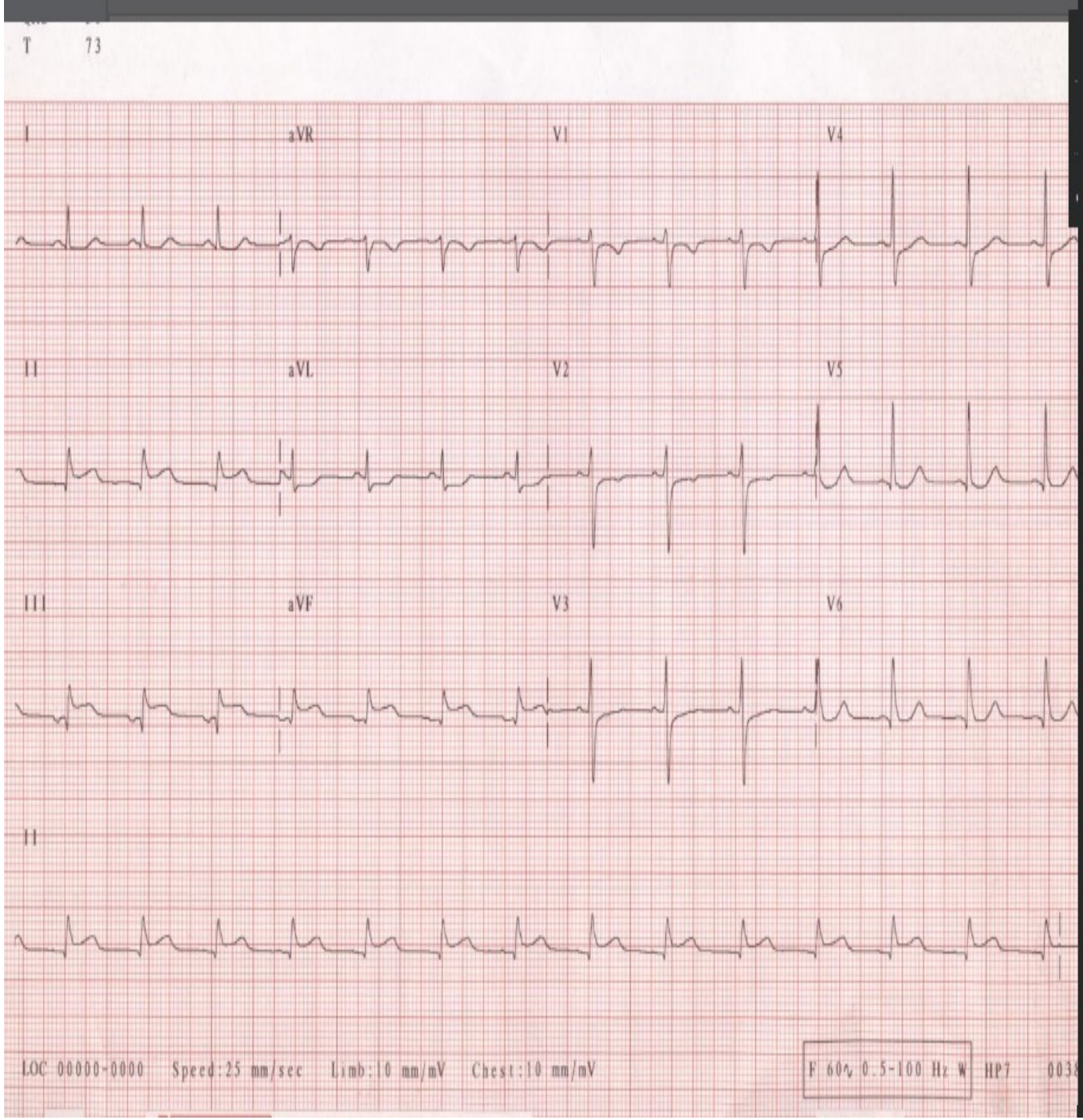


Anterior ST elevation myocardial infarction ECG

12-lead ECG (paper speed 25 mm/s)

- Sinus rhythm with a rate of ~75/min
- Normal heart axis (R > S in I and aVF)
- PR interval normal (< 200 ms)
- Significant ST segment elevation in V2-V6 (and mild ST elevation in I, aVL and V1)
- New left bundle branch block demonstrated by prominent W-shaped S waves in V1 and M-shaped R waves in V6.

These findings indicate an acute anterior ST-elevation myocardial infarction (STEMI).

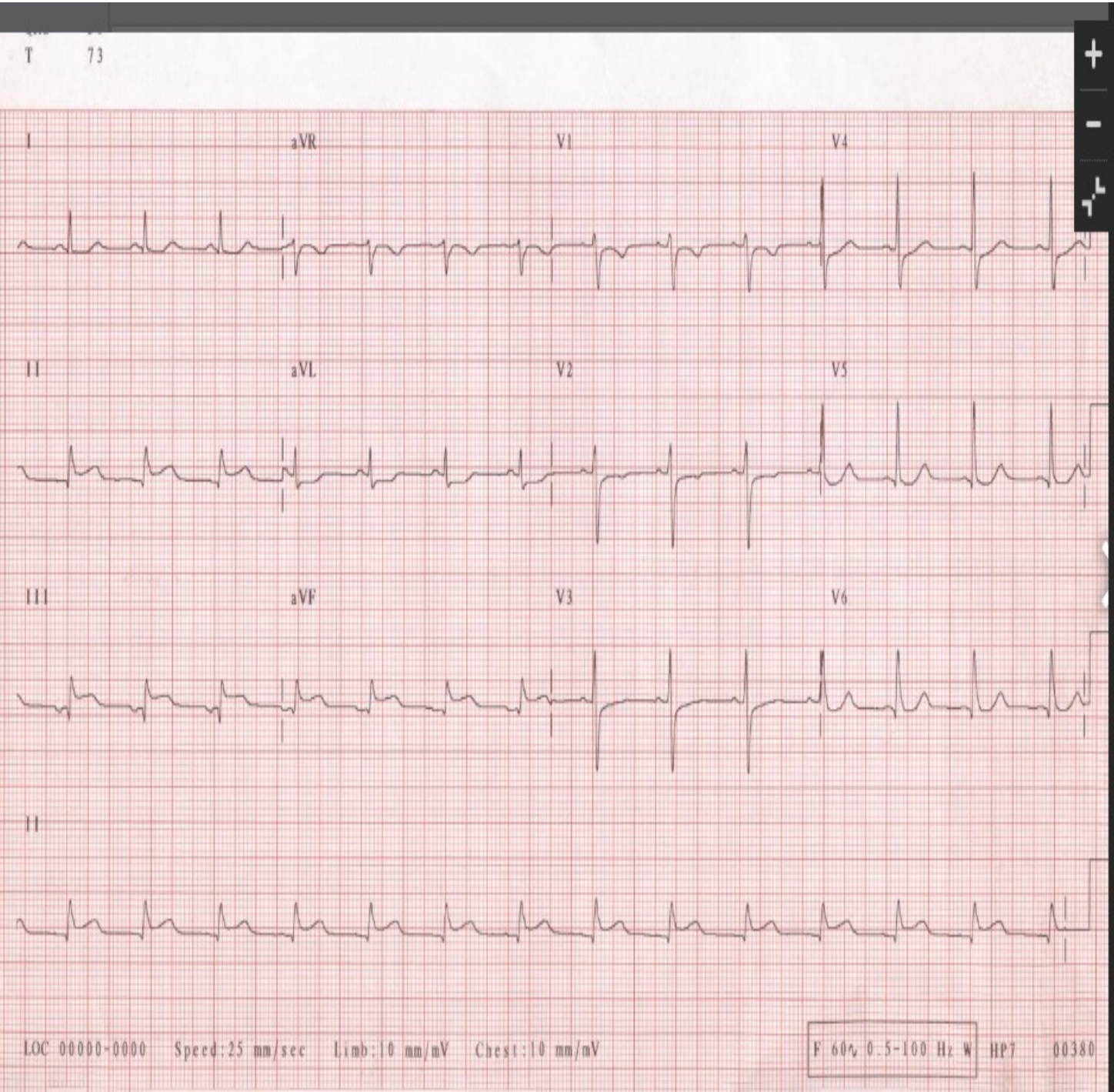


ST-elevation myocardial infarction (STEMI)

12-lead ECG (paper speed 25 mm/s)

- Sinus rhythm with a heart rate of ~83/min
- Normal axis (R > S in I and aVF)
- Normal PR, normal QRS morphology, normal QT interval
- ST elevations in the inferior leads (II, aVF, III), ST depressions in aVL, V2, V5-6 and borderline Q wave in III

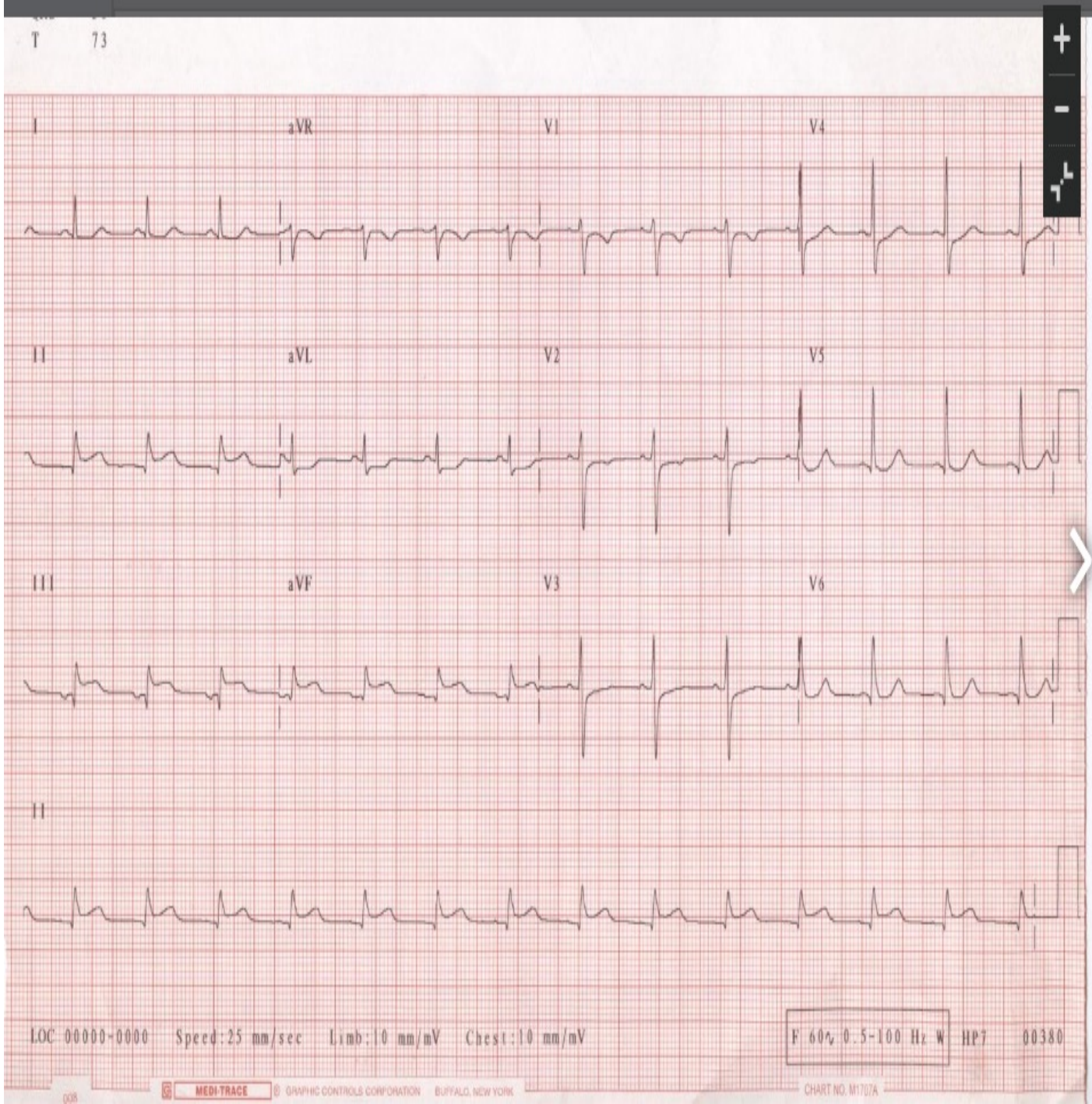
These findings indicate an acute inferior ST-elevation myocardial infarction (STEMI).



Acute inferior ST-elevation myocardial infarction (STEMI)

12-lead ECG (paper speed 25 mm/s)

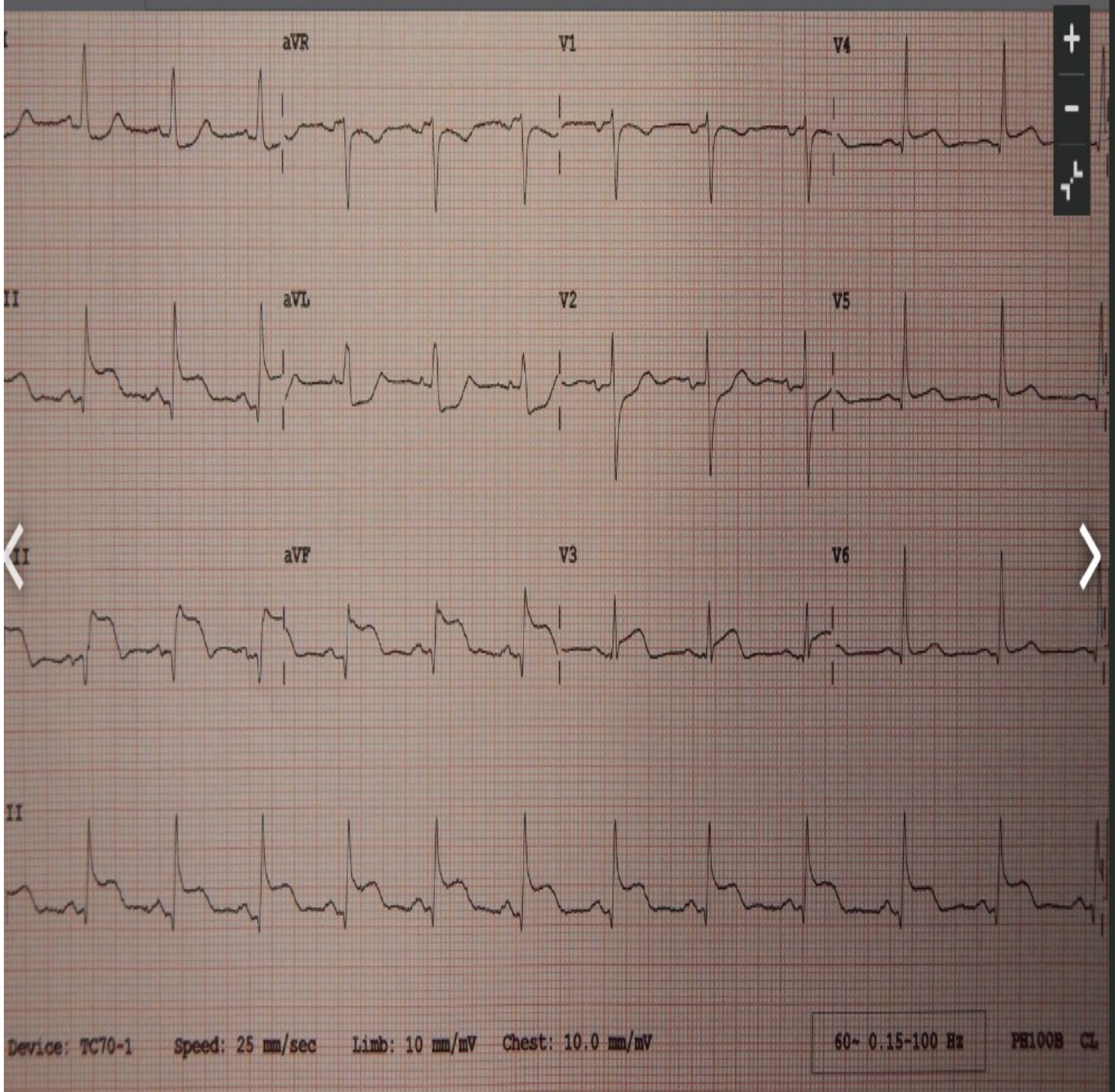
- Sinus rhythm with a heart rate of ~75/min
- Normal cardiac axis
- Normal QRS complex
- ST elevation in II, III, and aVF



Acute inferior ST-elevation myocardial infarction (STEMI)

12-lead ECG (paper speed 25 mm/s)

- Sinus rhythm with a heart rate of ~66/min
- Normal cardiac axis
- Normal QRS complex
- ST elevation in II, III, and aVF



Acute inferior ST-elevation myocardial infarction (STEMI)

12-lead ECG (paper speed 25 mm/s)

- Sinus rhythm with a heart rate of ~90/min
- Normal heart axis (R > S in I and aVF)
- Normal PR interval (~120 ms), narrow QRS complex (~80 ms), normal QT interval (~ 300 ms)
- ST elevation in II, III, and aVF with reciprocal ST-depressions in lateral leads (I and aVL)

Laboratory findings
Cardiac biomarkers

Biomarker/enzyme	Rise*	Maximum*	Normalization*	Characteristics
Troponin T/I	6-8 h	12–24 h	7–10 days	•Cardiac-specific with **high sensitivity for myocardial ischemia** •The degree of elevation often correlates with the size of the infarct. •High sensitivity troponin assays (HscTn) may detect an increase in serum troponin level as early as 90 to 180 minutes after myocardial ischemia has occurred [20] •Can also be elevated in other cardiac and noncardiac conditions: See differential diagnosis of increased troponin below.
Myoglobin	~ 1 h	4–12 h	24 h	•Nonspecific marker that is no longer commonly used
CK-MB	~ 4–9 h	12–24 h	2–3 days	•CK-MB is more specific to cardiac tissue than total CK. •Can be helpful for **evaluating reinfarction** because of its short half-life but is no longer commonly used •The degree of elevation often correlates with the size of the infarct.

* The values rise, reach a certain maximum, and normalize in the span of hours or days following the onset of myocardial infarction or its symptoms. Values and time references may vary based on the precise laboratory methods employed.

Serum **troponin T** is the most important cardiac-specific marker and may be measured **3–4 hours after the onset** of myocardial infarction. CK-MB values correlate with the size of the infarct, reach a maximum after approximately 12–24 hours, and normalize after only 2–3 days, making **CK-MB** a good marker f**or evaluating reinfarction**.

Additional findings

Elevated inflammatory markers: **↑ WBC, CRP**

Elevated BNP: especially in heart failure

Elevated LDH

Elevated AST (SGOT)

Coronary angiography

Best test for **definitive diagnosis** of acute coronary occlusion

Can be used for **concurrent intervention** (e.g., PCI with stent placement)

Can identify site and degree of vessel occlusion

Indications include

- **Acute STEMI**
- Other high-risk ACS (TIMI score below)
- See also cardiac catheterization.

The most commonly occluded coronary arteries (descending order): left anterior descending artery, right coronary artery, circumflex artery.

Coronary angiograph

- Coronary angiography visualizes the location and severity of coronary after stenosis. Narrowing greater than 50% of luminal diameter is considered clinically significant.

Indication of cath :

- Unstable angina
- Post myocardial infarction
- Stable angina

Additional studies

Transthoracic echocardiogram

Identification of any wall motion abnormalities and to assess LV function

Important for risk assessment: In STEMI, the best predictor of survival is LVEF.

Evaluation for complications: aneurysms, mitral valve regurgitation, pericardial effusion, free wall rupture

Cardiac CT

May be considered as an alternative to invasive coronary angiography in patients with an intermediate risk of ACS (based on TIMI score)

Allows for noninvasive visualization of the coronary arteries

Contraindication: arrhythmias, tachycardia

Differential diagnosis of increased troponin

Cardiac causes

- Myocarditis
- Decompensated congestive heart failure
- Pulmonary embolism
- Cardiac arrhythmia, tachycardia
- Cardiac trauma
- Takotsubo cardiomyopathy

- **Noncardiac causes**
  - Renal failure
  - Stroke
  - Critical illness (e.g., sepsis)

Differential diagnosis of ST-elevations on ECG

- Early repolarization
- LBBB
- Brugada syndrome
- Myocarditis
- Pericarditis
- Pulmonary embolism
- Hyperkalemia
- Tricyclic antidepressant use
- Poor ECG lead placement

Treatment

Any patient with ST elevations on ECG requires immediate evaluation for urgent revascularization. The administration of other therapies should not delay care.

All patients

- **Monitoring**
  - Serial 12-lead ECG
  - Continuous cardiac monitoring
  - Serial serum troponin measurement
- **Pharmacologic therapy**
  - Sublingual or intravenous **nitrate** (nitroglycerin or ISDN)
    - For symptomatic relief of chest pain
    - Does not improve prognosis
    - Contraindications: inferior wall infarct (due to risk for hypotension), hypotension, and/or PDE 5 inhibitor (e.g., sildenafil) taken within last 24 hours

- **Morphine** IV or SC (3–5 mg)
  - Only if the patient has severe, persistent **chest pain** or severe anxiety related to the **myocardial** event
  - Administer with caution due to increased risk of complications (e.g., hypotension, respiratory depression) and **adverse events**
- **Beta blocker**
  - Recommended within the first 24 hours of admission
  - Avoid in patients with hypotension, features of **heart failure**, and/or risk of **cardiogenic shock** (e.g., large LV infarct, low **ejection fraction**).
- **Statins**: early initiation of high-intensity **statin** (such as **atorvastatin** 80 mg) regardless of baseline **cholesterol**, LDL, and HDL levels
- **Loop diuretic** (e.g., **furosemide**) if the patient has flash pulmonary edema or features of **heart failure**

- **Supportive care**
  - **Intravenous fluids** (e.g., normal saline): in patients with an inferior **MI** that causes RV dysfunction
  - Oxygen: only in case of cyanosis, severe **dyspnea**, or SpO_2 < 90% (< 95% in **STEMI**)

Primary interventions of MI treatment include "**MONA**": **M**orphine, **O**xygen, **N**itroglycerin, and **A**spirin.
But remember: Morphine, oxygen,
and nitroglycerine are not necessarily indicated for every patient .

STEMI

- **Immediate revascularization**
- Revascularization is the most important step in the management of acute STEMI and initiation of further therapies (e.g., DAPT, anticoagulation) should not delay this step in management.
- **Emergent coronary angiography**: with percutaneous coronary intervention (**PCI**)
  - Preferred method of revascularization
  - Balloon dilatation with stent implantation (cardiac catheterization)
  - Ideally, door-to-PCI time should be < 90 minutes. It should not exceed 120 minutes.

- **Thrombolytic therapy**: tPA, reteplase, or streptokinaseIndications:
  - **If PCI cannot be performed** < 120 minutes after onset of STEMI
  - If PCI was unsuccessful
  - No contraindications to thrombolysis
- Contraindications
  - Any prior intracranial bleeding
  - Recent large GI bleeding
  - Recent major trauma, head injury, and/or surgery
  - Ischemic stroke within the past 3 months
  - Hypertension (> 180/110 mm Hg)
  - Known coagulopathy

- Timing
  - Symptom onset was within the past 3–12 hours
  - Should be administered within < 30 minutes of patient arrival to the hospital
  - Contraindicated if > 24 hours after symptom onset
- PCI should be performed even if lysis is successful.

- **Coronary artery bypass grafting**
- Not routinely recommended for acute STEMI
- Indications
  - If PCI is unsuccessful
  - If coronary anatomy is not amenable to PCI
  - If STEMI occurs at the time of surgical repair of a mechanical defect

Medical therapy

- **Dual antiplatelet therapy**: start as soon as possible
  - Aspirin loading dose 162 mg–325 mg
  - PLUS ADP receptor inhibitor: prasugrel, ticagrelor, or clopidogrel
  - Dual antiplatelet therapy should be continued for at least 12 months after PCI with DES.
- **GP IIb/IIIa receptor antagonist** (e.g., eptifibatide or tirofiban): should be considered in precatheterization setting
- **Anticoagulation**
  - Heparin or bivalirudin recommended
  - Continue until PCI is performed or for 48 hours after a fibrinolytic is given.

"**Time is muscle**": Revascularization should occur as soon as possible in patients with STEMI!

Unstable angina/NSTEMI

- **Dual antiplatelet therapy**: start as soon as possible
  - Aspirin loading dose
  - Plus ADP receptor inhibitor: clopidogrel or ticagrelor
  - Dual antiplatelet therapy should be continued for at least 12 months if PCI with DES was performed.
- **Anticoagulation**
  - Heparin or enoxaparin
  - Continue for the duration of hospitalization or until PCI is performed.
- **Immediate vs. delayed revascularization**
  - The indication for and timing of revascularization depends on the mortality risk (e.g., TIMI score).
  - In patients with therapy-resistant chest pain, a TIMI score ≥ 3, ↑ troponin, and/or ST changes > 1 mm
    - Consider the addition of a GPIIb/ IIIa inhibitor (e.g., tirofiban or eptifibatide)
    - Plan for revascularization within 72 hours (e.g., angiography with PCI or CABG)

Fibrinolytic treatment is not recommended in patients with unstable angina or NSTEMI.

Management: Medical

- **Antiplatelet therapy**

–Low-dose (75-100 mg) aspirin

- reduces the risk of adverse events such as MI
- prescribed for all patients with CAD indefinitely

–Clopidogrel (75 mg daily)

- equally effective ALTERNATIVE and additive to Aspirin in cases of MI or USA.
- if aspirin causes dyspepsia or other side-effects
- **Anti-anginal drug treatment**: Five groups of drug

–Nitrates

–β-blockers

–calcium antagonists

Preparation	Peak action	Duration of action
Sublingual GTN	4–8 mins	10–30 mins
Transdermal GTN	1–3 hrs	Up to 24 hrs
Oral isosorbide dinitrate	45–120 mins	2–6 hrs

Management: Medical

- Nitrates

–act directly on **vascular smooth muscle** to produce **venous** and **arteriolar dilatation**

–Increase in myocardial oxygen supply

–Continuous nitrate therapy can cause pharmacological tolerance

- avoided by a 6–8-hour nitrate-free period
- **Nocturnal angina:** longacting nitrates can be given at the end of the day

Management: Medical

- **β-blockers**: lower myocardial oxygen demand by reducing heart rate, BP and myocardial contractility
- First line treatment
- provoke bronchospasm in patients with asthma.
- **Calcium antagonists (Non-dihydropyridine drugs)**
- Second line treatment (because of their rate-limiting action, verapamil and diltiazem are the calcium channel antagonists of choice if a β-blocker is contraindicated).
- Don't use combination of β-blockers and Non-dihydropyridine drugs) , **Dihydropyridine calcium channel antagonist like** nifedipine or amlodipine can be added to the β-blocker if there is persistent chest discomfort , but may cause an unwanted tachycardia if used alone.
- **A statin**

Other drugs that occasionally used in Refractory Angina

- **Potassium channel activators**

–Do not exhibit the tolerance seen with nitrates.

–**Nicorandil (10–30mg 12-hourly orally)** - only drug in this class currently available for clinical use

- **If channel antagonist**

–**Ivabradine** is the first of this class of drug

–Induces **bradycardia** by modulating ion channels in the **sinus node**

–Comparatively, does not have other cardiovascular effects

–Safe to use in patients with heart failure

Coronary artery bypass grafting (CABG)

- Alternative treatment to Percutaneous intervention cath.

•Stenosed artery is by-passed with

–**internal mammary arteries**

–**radial arteries**

–reversed segments of the patient's own **saphenous vein**

•Major surgery under **cardiopulmonary bypass**,

•But in some cases, grafts can be applied to the beating heart: '**off-pump' surgery**

•Operative mortality is approximately 1.5% but risks are higher

–elderly patients,

–with poor left ventricular function

–those with significant comorbidity, such as renal failure .

Subsequent measures

Secondary prophylaxis: See prevention of coronary heart disease, therapy of atherosclerotic diseases, and PTCA. See coronary artery surgery.

Complications

- **0–24 hours post-infarction**
- **Sudden cardiac death**
  - The most common underlying cause in elderly individuals is acute coronary syndrome (~ 70% of cases).
  - The most common cause is ventricular arrhythmia.
- **Arrhythmias**
  - Ventricular tachyarrhythmias
  - AV block
  - Asystole
  - Atrial fibrillation
- **Acute left heart failure**: death of affected myocardium → absence of myocardial contraction→ **pulmonary edema**
- **Cardiogenic shock**

1–3 days post-infarction

- **Early infarct-associated pericarditis**
  - Typically occurs within the first week of a large infarct close to the pericardium
  - Clinical features of acute pericarditis: pleuritic chest pain , dry cough , **friction rub**, diffuse ST elevations on ECG
  - Complications (rare): hemopericardium, pericardial tamponade

3–14 days post-infarction

- **Papillary muscle rupture**
  - Usually occurs 2–7 days after myocardial infarction
  - Can lead to acute **mitral regurgitation**
  - Rupture of the posteromedial papillary muscle due to occlusion of the posterior descending artery is most common.
  - Clinical features
    - New holosystolic, blowing murmur over the 5th ICS on the midclavicular line
    - Signs of acute mitral regurgitation: dyspnea, cough, bilateral crackles, hypotension

- **Ventricular septal rupture**Usually occurs 3–5 days after myocardial infarction
- Due to structural degradation by macrophages
- Most commonly due to LAD infarction (septal arteries arise from LAD)
- Clinical features
  - **New holosystolic murmur** over the left sternal border
  - Acute-onset **right heart failure** (jugular venous distention, peripheral edema)
  - Can progress to cardiogenic shock: tachycardia, hypotension, cool extremities, altered mental status
- Treatment: emergency surgery and revascularization (often via CABG)

- **Left ventricular free wall rupture** Usually occurs 5–14 days after myocardial infarction
- Greatest risk during macrophage-mediated removal of necrotic tissue
- Clinical features: chest pain, dyspnea, signs of cardiac tamponade (e.g., Beck triad)
- Complications
  - **Cardiac tamponade**
  - LV **pseudoaneurysm formation**: rupture of LV wall that is contained by the pericardium

2 weeks to months post-infarction

- **Atrial and ventricular aneurysms**
  - Clinical features
    - Persistent (> 3 weeks post-MI) ST elevation and T-wave inversions
    - Systolic murmur, S_3 and/or S_4
  - Diagnosis: echocardiography
  - Complications
    - Cardiac arrhythmias (risk of ventricular fibrillation)
    - **Rupture** → cardiac tamponade
    - **Mural thrombus formation** → thromboembolism (stroke, mesenteric ischemia, renal infarction , acute obstruction of peripheral arteries)
  - Treatment: anticoagulation, possibly surgery

- **Postmyocardial infarction syndrome** (**Dressler syndrome**): pericarditis occurring 2–10 weeks post-MI without an infective cause Thought to be due to circulating antibodies against cardiac muscle cells (autoimmune etiology)
- Clinical features
  - Signs of acute pericarditis: pleuritic chest pain , dry cough , friction rub
  - Fever
  - Laboratory findings: leukocytosis, ↑ serum troponin levels
  - ECG: diffuse ST elevations
- Treatment: aspirin, acetaminophen
- Complications (rare): hemopericardium, pericardial tamponade

- **Arrhythmias** (e.g., AV block)
- **Congestive heart failure** (e.g., ischemic cardiomyopathy)
  - Can occur at any time after an ischemic event
  - Treatment: for patients with LVEF < 40% or signs of heart failure, ACE inhibitor/ARB and aldosterone antagonists have been shown to confer a mortality benefit.
- **Reinfarction**

Prevention

Primary prevention

- Treatment/avoidance of modifiable risk factors for atherosclerosis (e.g., smoking cessation, treatment of hypertension, etc.)
- Healthy, plant-based diet
- Regular physical activity and exercise
- Low-dose aspirin is beneficial for certain high-risk groups. The choice to prescribe it should be made on an individual basis.

Secondary prevention

- Lifestyle modification and treatment of modifiable risk factors (see "Primary prevention" above and treatment of diseases caused by atherosclerosis)
- Platelet-aggregation inhibitors
  - **Lifelong low-dose aspirin** 75–100 mg/day
  - **DAPT** with the addition of an ADP receptor inhibitor (e.g., prasugrel, ticagrelor, or clopidogrel) is recommended **for 12 months** for all patients who have undergone PCI.
  - Glycoprotein IIb/IIIa antagonists (e.g., abciximab) may be considered but are not used routinely.
- *Beta blockers*: Unless contraindicated, all patients should be started on a beta blocker, which has been shown to confer a mortality benefit.
- *Statin*: All patients should be started on a high-intensity statin (e.g., atorvastatin).
- An aldosterone antagonist and ACE inhibitor/ARB are recommended for all patients with ischemic cardiomyopathy and an LV ejection fraction < 40% or symptoms of heart failure.

THANKS……