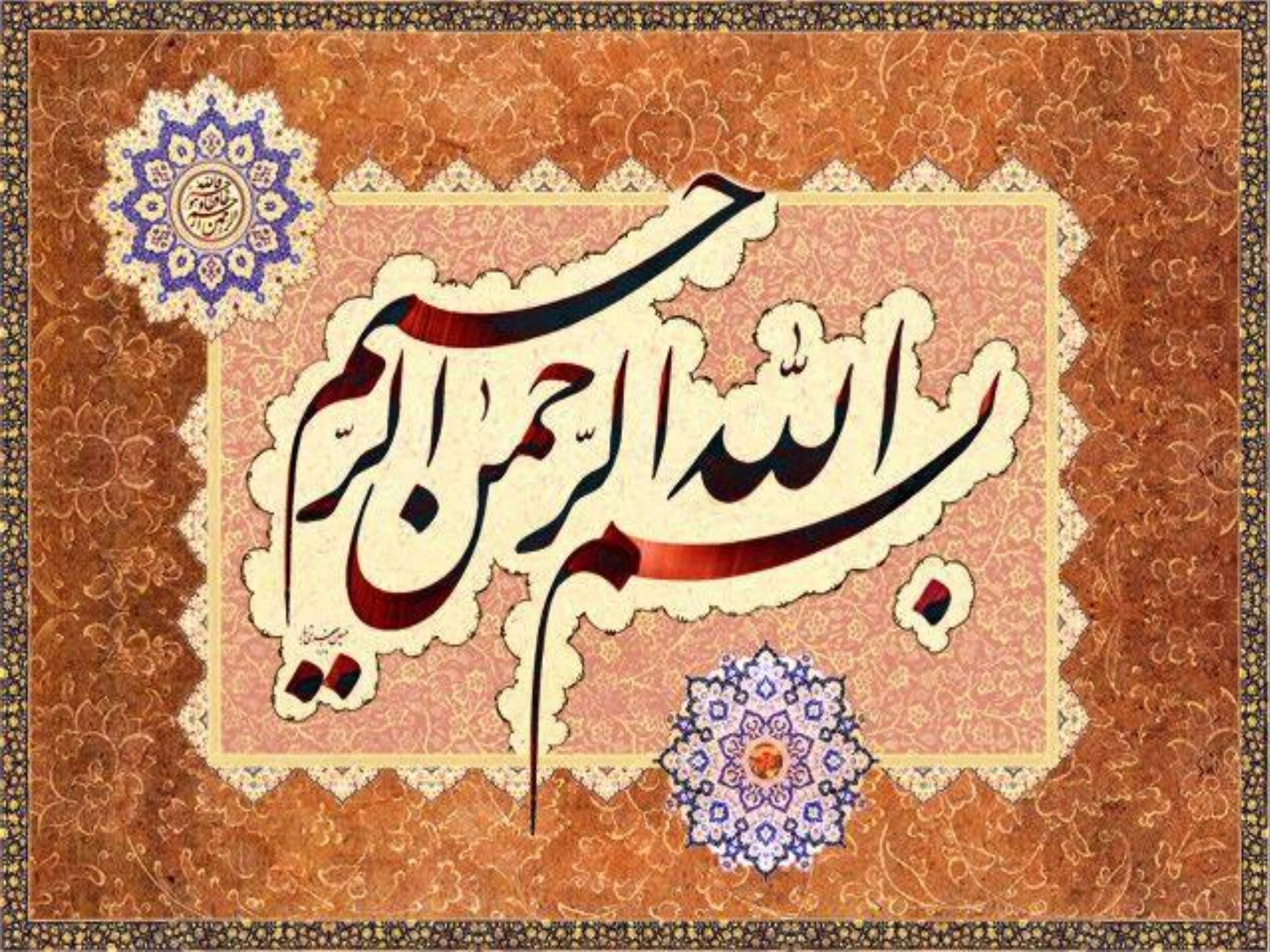
بسم الله الرحمن الرحیم

ST-Elevation Myocardial Infarction

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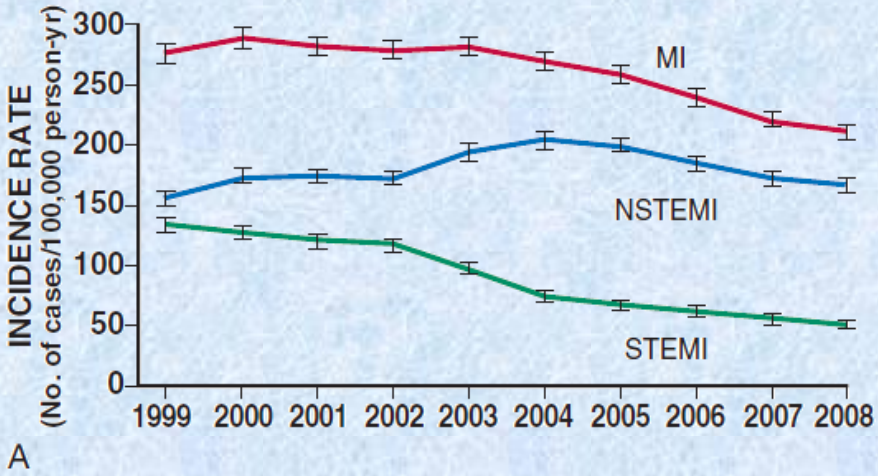
CHANGING PATTERNS IN INCIDENCE AND CARE

- ✓ STEMI remains:
- A major public health problem in the <u>industrialized</u> world and
- Rise in <u>developing</u> countries

- ✓ Between 1999 and 2008, the ACS and STEMI declined by almost 50%.



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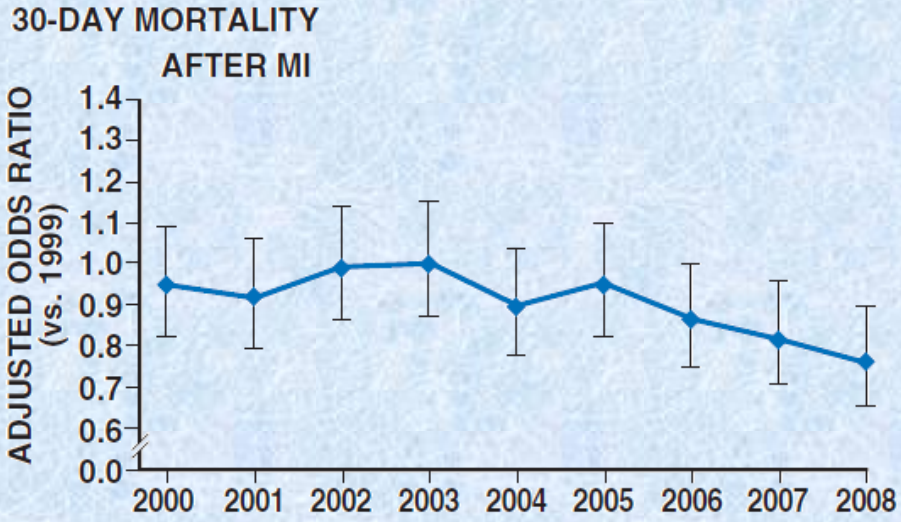


IMPROVEMENTS IN OUTCOME

- Improvements in the management of STEMI:

1. The “clinical observation phase” (focused on detailed recording of physical and laboratory findings)

2. The “coronary care unit phase” (emphasized early detection and management of cardiac arrhythmias)

3. The “high-technology phase,” (introduction of the pulmonary artery balloon catheter, set the stage for bedside hemodynamic monitoring)

4. The modern “reperfusion era” (intracoronary and then intravenous fibrinolysis, and development of primary PCI)

Universal Definition of Myocardial Infarction

Criteria for Acute Myocardial Infarction

- ✓ Rise and/or fall in cardiac biomarker values (preferably cTn) and one of the following:

A. Symptoms of ischemia

B. New or presumed new significant ST-T changes or new LBBB

C. Development of pathologic Q waves on the ECG

D. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

E. Identification of an intracoronary thrombus by angiography or autopsy

- ✓ Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic changes on the ECG or new LBBB but death occurred before cardiac biomarkers were determined or before cardiac biomarker values would be increased.

Criteria for Acute Myocardial Infarction

✓ PCI-related MI :

- elevation of cTn values (to >5 × the 99th percentile) in patients with normal baseline values or
- a rise in cTn values >20% if the baseline values are elevated and are stable or falling.
- In addition, either
- (1) symptoms suggestive of MI,
- (2) new ischemic changes on the ECG,
- (3) angiographic findings consistent with a procedural complication, or
- (4) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality is required.

✓ CABG-related MI:

- elevation of cardiac biomarker values (to >10 × the 99th percentile) in patients with normal baseline cTn.
- In addition, either
- (1) new pathologic Q waves or new LBBB,
- (2) angiographically documented new graft or new native coronary artery occlusion, or
- (3) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality is required.

Criteria for Previous Myocardial Infarction

- Any one of the following criteria:

1. Pathologic Q waves with or without symptoms (in the absence of nonischemic causes)

2. Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract (in the absence of a nonischemic cause)

3. Pathologic findings of previous MI

Universal Myocardial Infarction Classification of Type

- **Type 1: Spontaneous MI**
  (Atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection)

- **Type 2: MI Secondary to Ischemic Imbalance**
  (Imbalance between myocardial oxygen supply and/or demand)

- **Type 3: MI Resulting in Death When Biomarker Values Are Unavailable**
  (Cardiac death + symptoms + new ischemic changes on the ECG or new LBBB)

- **Type 4a: MI Related to PCI**

- **Type 4b: MI Related to Stent Thrombosis**

- **Type 5: MI Related to CABG**

PATHOLOGIC FINDINGS

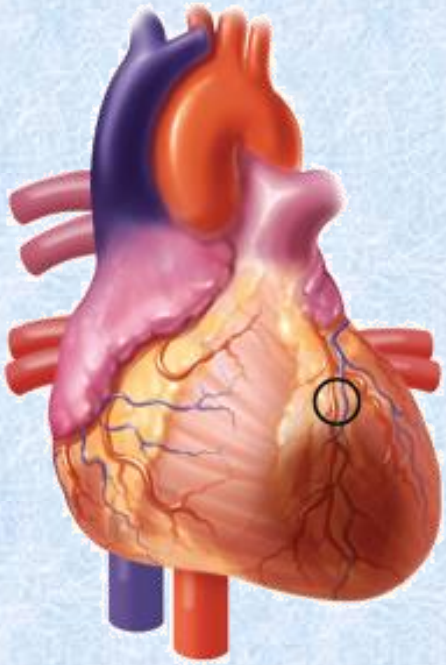
- Almost all ACS events result from coronary **atherosclerosis**, generally with superimposed coronary **thrombosis** caused by **rupture or erosion** of an atherosclerotic lesion

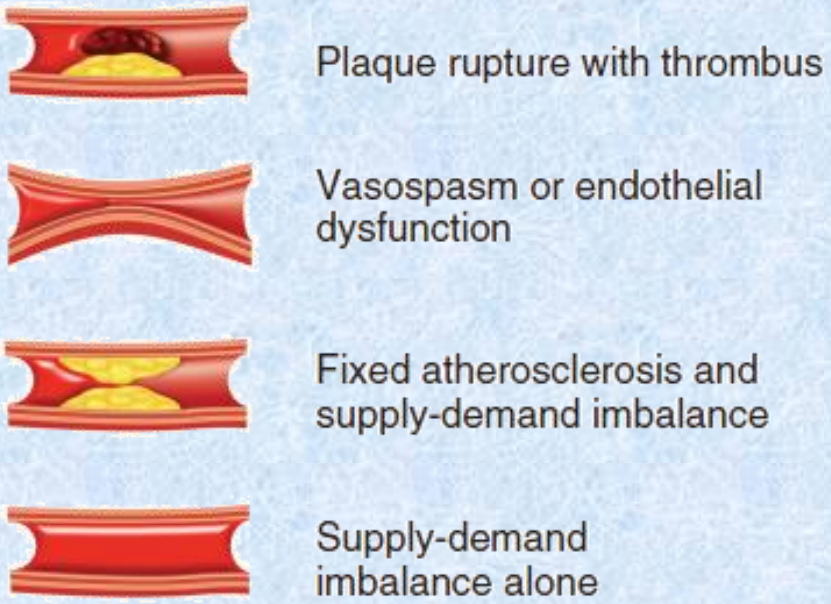
- Non-atherogenic forms of coronary artery disease are uncommom.

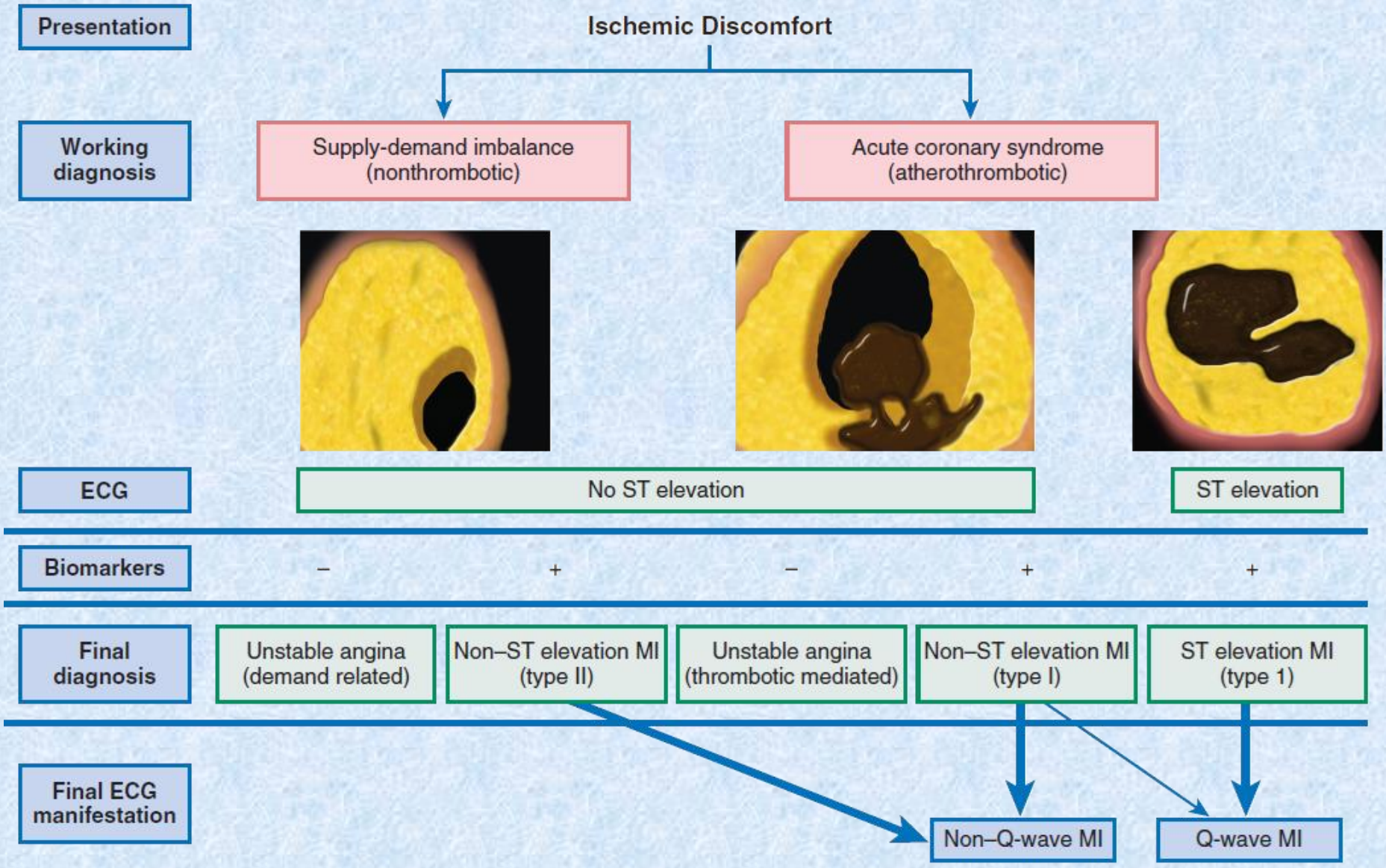
TABLE 51-3 Causes of Myocardial Infarction Without Coronary Atherosclerosis

Coronary Artery Disease Other than Atherosclerosis
Arteritis
Luetic
Granulomatous (Takayasu disease)
Polyarteritis nodosa
Mucocutaneous lymph node (Kawasaki) syndrome
Disseminated lupus erythematosus
Rheumatoid spondylitis
Ankylosing spondylitis
Trauma to coronary arteries
Laceration
Thrombosis
Iatrogenic
Radiation (radiation therapy for neoplasia)
Coronary mural thickening with metabolic disease or intimal proliferative disease
Mucopolysaccharidoses (Hurler disease)
Homocystinuria
Fabry disease
Amyloidosis
Juvenile intimal sclerosis (idiopathic arterial calcification of infancy)
Intimal hyperplasia associated with contraceptive steroids or with the postpartum period
Pseudoxanthoma elasticum
Coronary fibrosis caused by radiation therapy
Luminal narrowing by other mechanisms
Spasm of coronary arteries (Prinzmetal angina with normal coronary arteries)
Spasm after nitroglycerin withdrawal
Dissection of the aorta
Dissection of the coronary artery
Emboli to Coronary Arteries
Infective endocarditis
Nonbacterial thrombotic endocarditis
Prolapse of the mitral valve
Mural thrombus from the left atrium, left ventricle, or pulmonary veins
Prosthetic valve emboli
Cardiac myxoma
Associated with cardiopulmonary bypass surgery and coronary arteriography
Paradoxical emboli
Papillary fibroelastoma of the aortic valve ("fixed embolus")
Thrombi from intracardiac catheters or guidewires
Congenital Coronary Artery Anomalies
Anomalous origin of the left coronary from the pulmonary artery
Left coronary artery from the anterior sinus of Valsalva
Coronary arteriovenous and arteriocameral fistulas
Coronary artery aneurysms
Myocardial Oxygen Demand-Supply Disproportion
Aortic stenosis, all forms
Incomplete differentiation of the aortic valve
Aortic insufficiency
Carbon monoxide poisoning
Thyrotoxicosis
Prolonged hypotension
Takotsubo cardiomyopathy
Hematologic (In Situ Thrombosis)
Polycythemia vera
Thrombocytosis
Disseminated intravascular coagulation
Hypercoagulability, thrombosis, thrombocytopenic purpura
Miscellaneous
Cocaine abuse
Myocardial contusion
Myocardial infarction with normal coronary arteries
Complication of cardiac catheterization

Causes of myocardial oxygen supply-demand imbalance






Presentation
Ischemic Discomfort
Working diagnosis
Supply-demand imbalance (nonthrombotic)
Acute coronary syndrome (atherothrombotic)
ECG
No ST elevation
ST elevation
Biomarkers
–
+
–
+
+
Final diagnosis
Unstable angina (demand related)
Non–ST elevation MI (type II)
Unstable angina (thrombotic mediated)
Non–ST elevation MI (type I)
ST elevation MI (type 1)
Final ECG manifestation
Non–Q-wave MI
Q-wave MI

- Before the fibrinolytic era, clinicians typically divided patients with MI into those in whom

1. a Q wave developed on the ECG (*transmural infarction*s)
2. those with non–Q-wave MI (*subendocardial infarctions)*

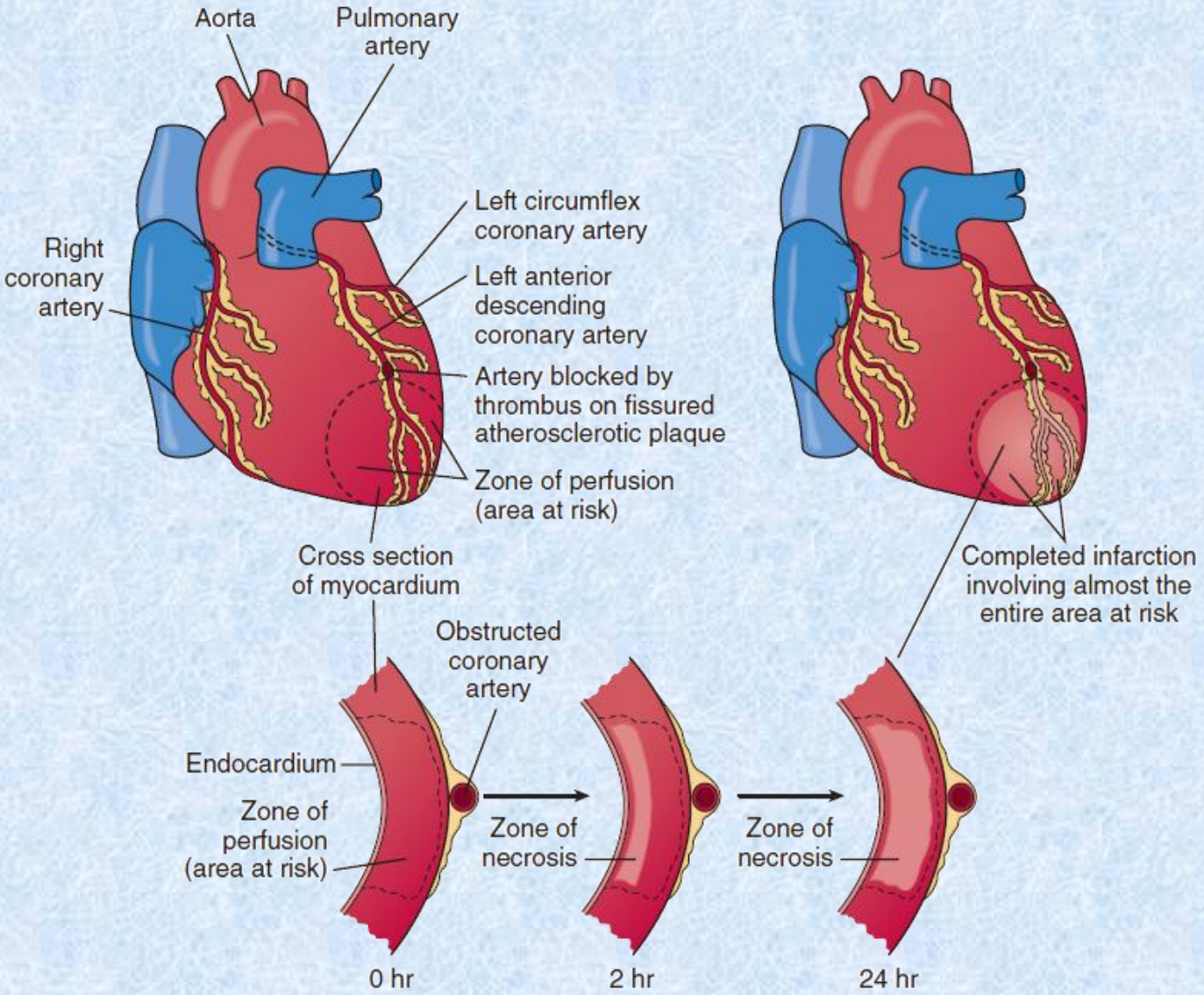
- New studies using CMR indicate that the development of a Q wave is determined more by the size of the infarct than by the depth of mural involvement.

Plaque

- **Plaque disruption** exposes substances that promote platelet activation and aggregation, thrombin generation, and ultimately thrombus formation.

- The resultant thrombus interrupts blood flow and leads to an **imbalance between oxygen supply and demand** and, if this imbalance is severe and persistent, to myocardial necrosis.

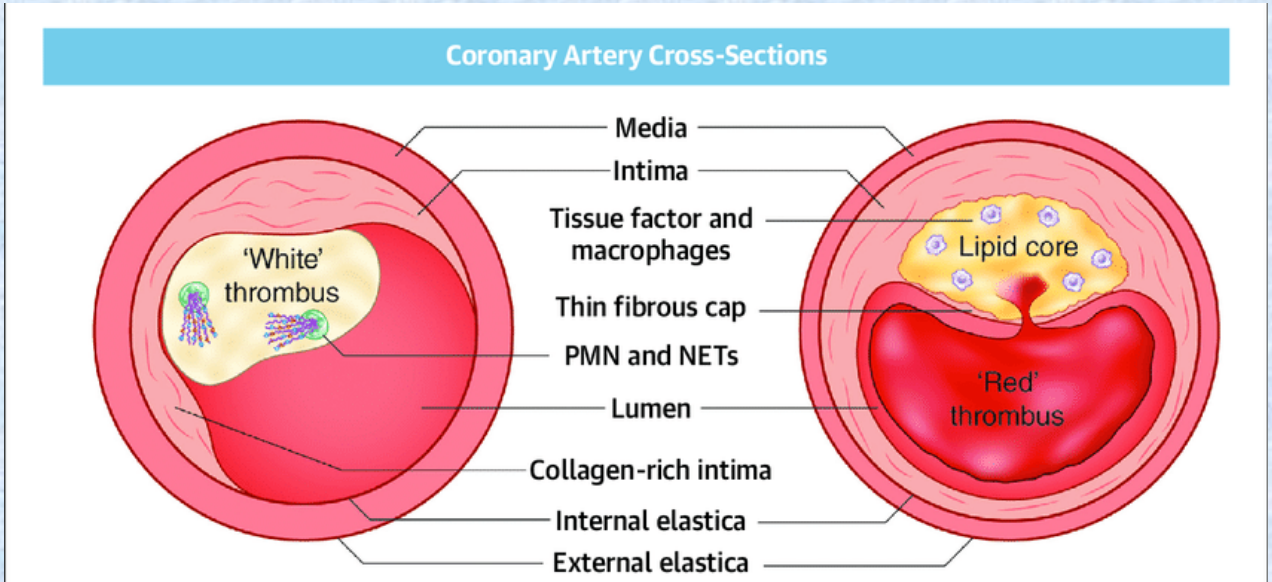
Schematic representation of the progression of myocardial necrosis after coronary artery occlusion



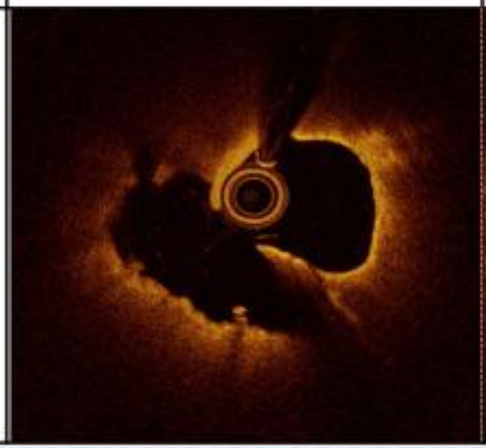
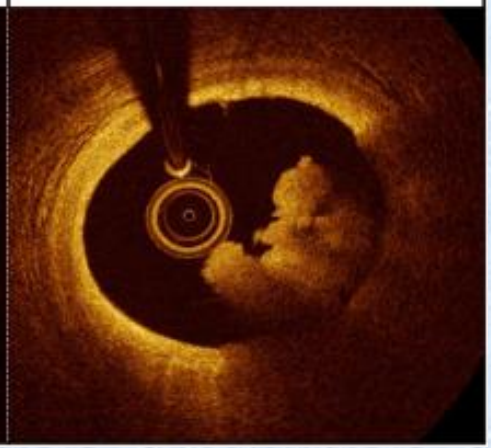
Plaque Fissuring and Disruption

- In autopsy studies, **plaque rupture** and **plaque erosion** are the most common underlying causes of MI and sudden cardiac death.
- **Plaque rupture** is present in almost three quarters of cases and is more prevalent in men.
- **Plaque erosion** is more frequent in women younger than 50 years, although the **prevalence of rupture increases as women age.**
- Atherosclerotic plaque considered prone to disruption or erosion is most likely plaque that has evolved to a morphology that includes a **necrotic core filled** with **lipids** and **inflammatory cells** and covered by a **thin and inflamed fibrous cap**.
- A prospective study of 697 patients with ACS who underwent three-vessel coronary angiog- raphy and gray-scale radiofrequency intravascular ultrasonographic imaging after PCI found that three lesion characteristics—**lipid burden greater than 70%, thin-cap broatheroma morphology**, and a **minimal luminal area of 4.0 mm2 or smaller**—were independent correlates of future atherosclerotic events

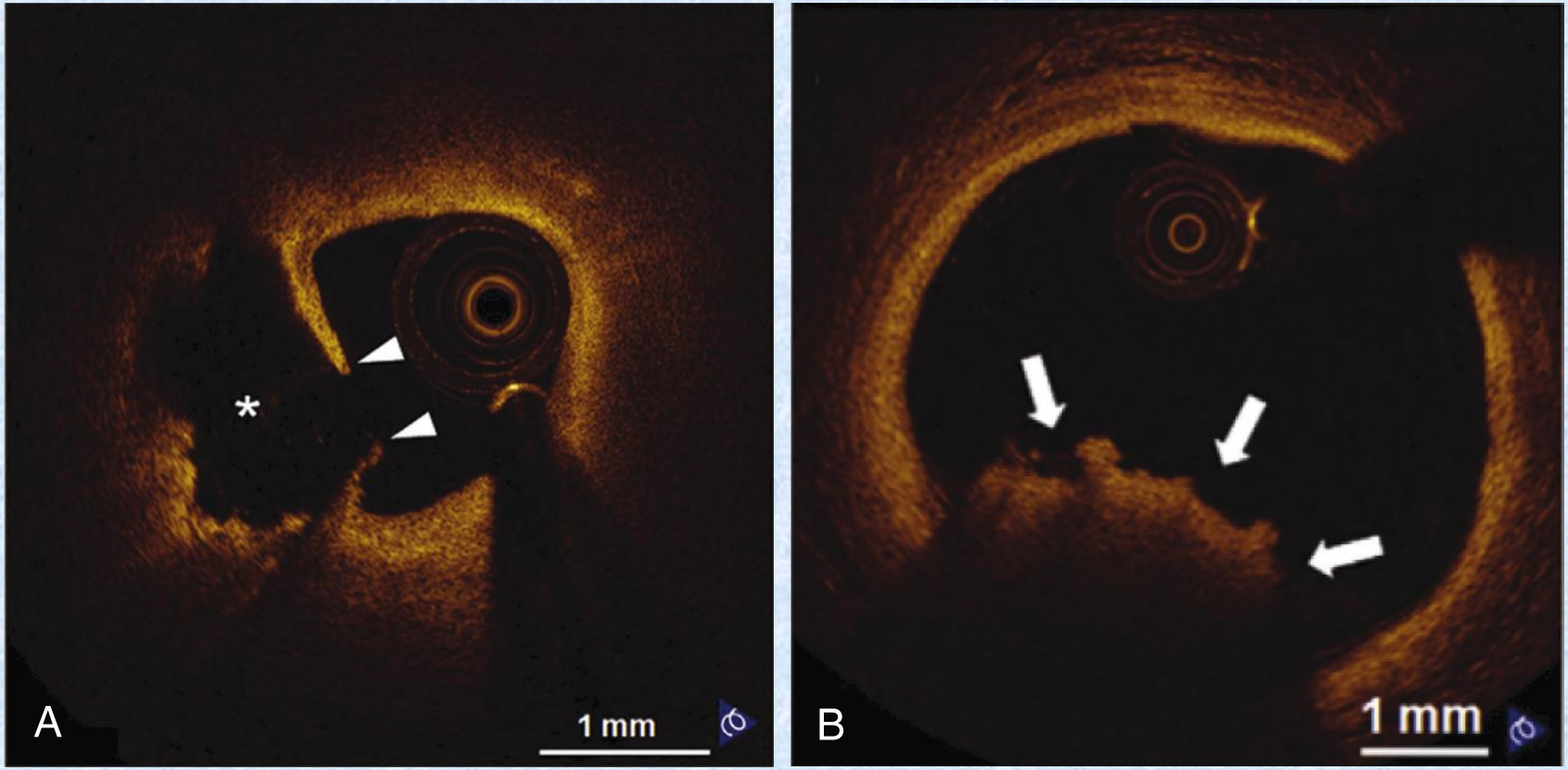
plaque rupture vs. plaque erosion



The coronary artery cross section showing thrombosis due to erosion **(left)** and rupture **(right)**. Reproduced with permission from Libby et al. (27). ACS = acute coronary syndrome; NET = neutrophil extracellular trap; PMN = polymorphonuclear leukocytes; STEMI = ST-segment elevation myocardial infarction.

	Plaque Rupture	Plaque Erosion
Estimated incidence ACS	67% (Predominant in STEMI)	25% (NSTEMI>STEMI)
Clinical Profile	Older Traditional risk factors (dyslipidemia, hypertension, diabetes)	Younger Women Smoker Lower LDL
Angiographic characteristics	Higher anatomical complexity Smaller lumen Occlusive thrombus	Predominant LAD Lower anatomical complexity Larger lumen Non-occlusive thrombus
Underlying Plaque	Lipid plaque TCFA Red thrombus	Fibrous plaque White thrombus
Optical Coherence Tomography (OCT)		

Plaque Rupture and Thrombosis

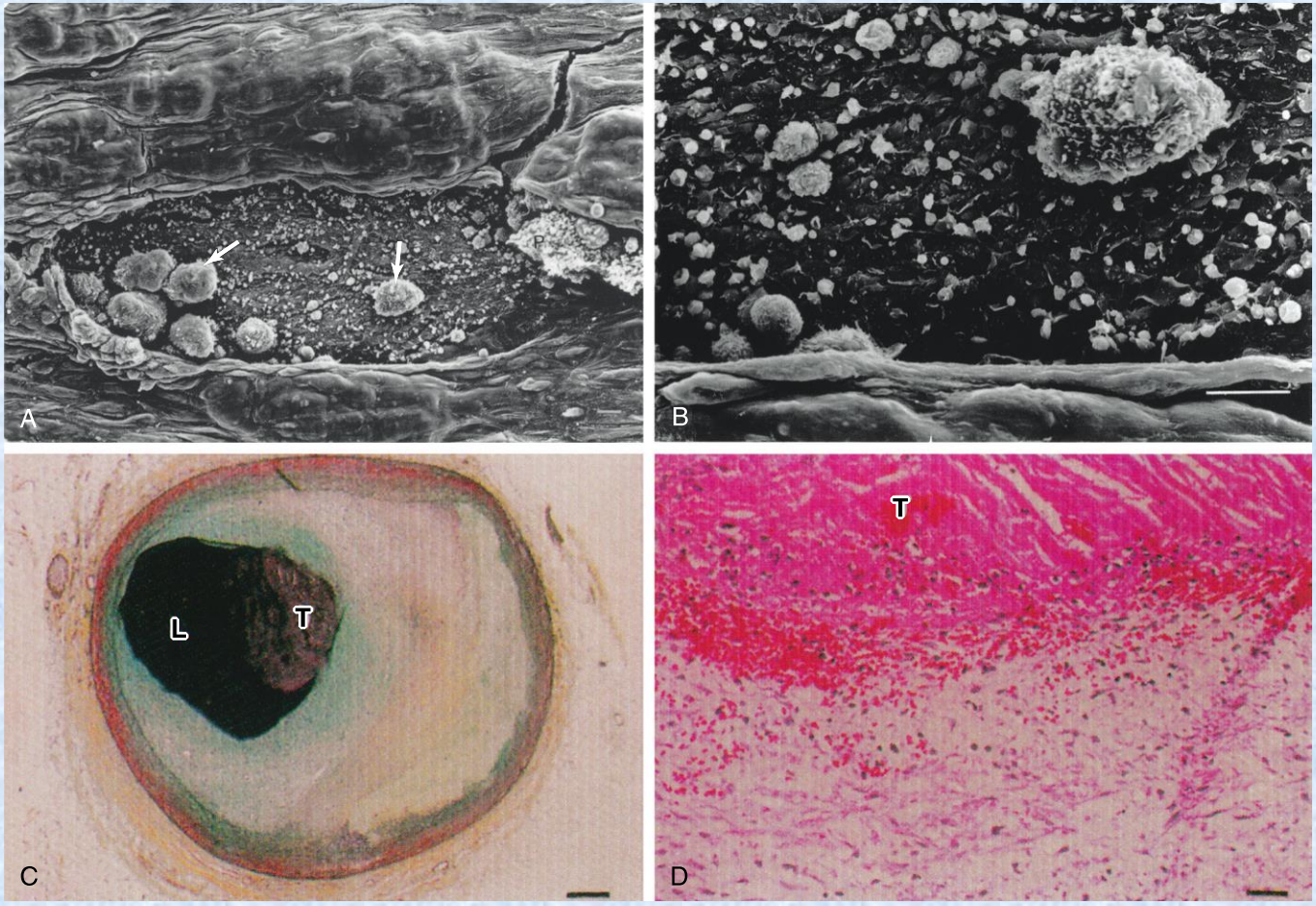


Examples of disrupted plaques in coronary arteries visualized by optical coherence tomography.

A, Rupture of a fibrous cap. *Arrowheads* point to the intimal discontinuity; the lucent cavity below (*asterisk*) probably represents an ulcer containing a lipid-rich core. Some or all of the thrombogenic contents of this core may have herniated into the artery and embolized.

B, An apparent thrombus (*arrows*) in a region without evident fibrous cap rupture probably represents superficial erosion.

Thrombosis Due to Superfacial Erosion of Plaques

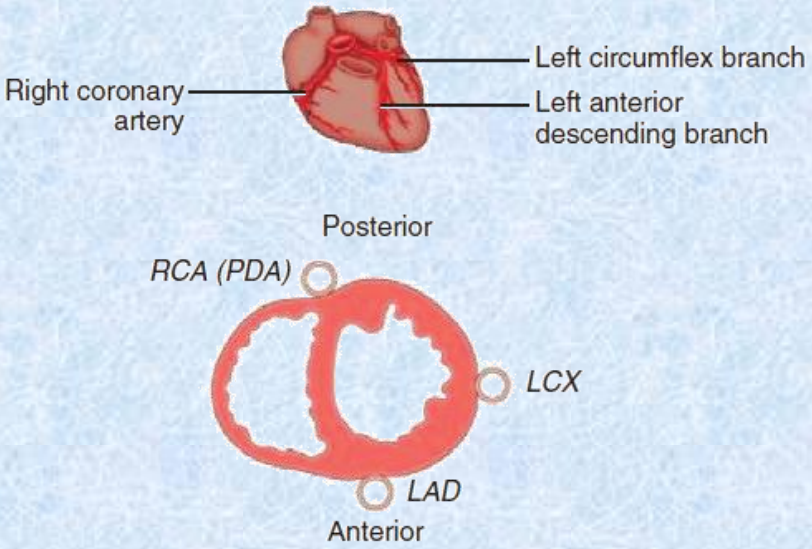


Acute Coronary Syndromes

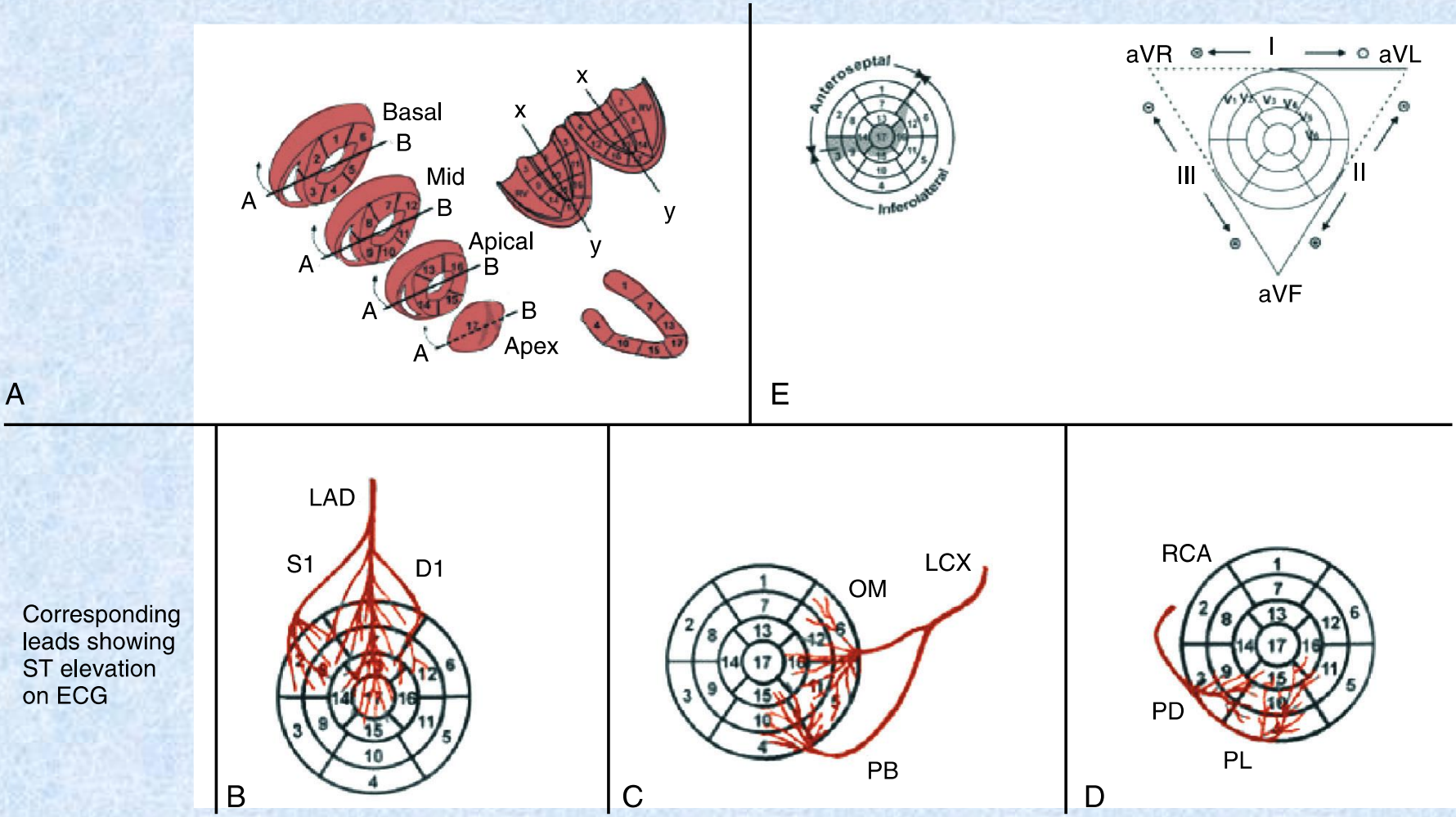
- Plaque disruption exposes thrombogenic substances that may produce an extensive thrombus in the infarct-related artery.
- An **adequate collateral network** that prevents necrosis from occurring can result in clinically silent episodes of coronary occlusion;
- in addition, **many plaque ruptures are asymptomatic** if the thrombosis is not occlusive.

- Characteristically, completely occlusive thrombi lead to transmural injury to the ventricular wall in the myocardial bed subtended by the affected coronary artery.

- The most characteristic change in the QRS that develops in most patients with STEMI is the evolution of Q waves in leads overlying the infarct zone.
- In a minority of patients with ST elevation, **no Q waves develop** but other abnormalities in the QRS complex occur frequently, such **as diminution in R wave height** and **notching or splintering of the QRS**.



Location of zones of necrosis following occlusion of major epicardial coronary arteries	Permanent occlusion of left anterior descending branch	Permanent occlusion of left circumflex branch	Permanent occlusion of right coronary artery (or its posterior descending branch)



For example, ST elevation seen most prominently in the leads overlying segments 1, 2, 7, 8, 13, 14, and 17 indicates that the LAD is the infarct artery.

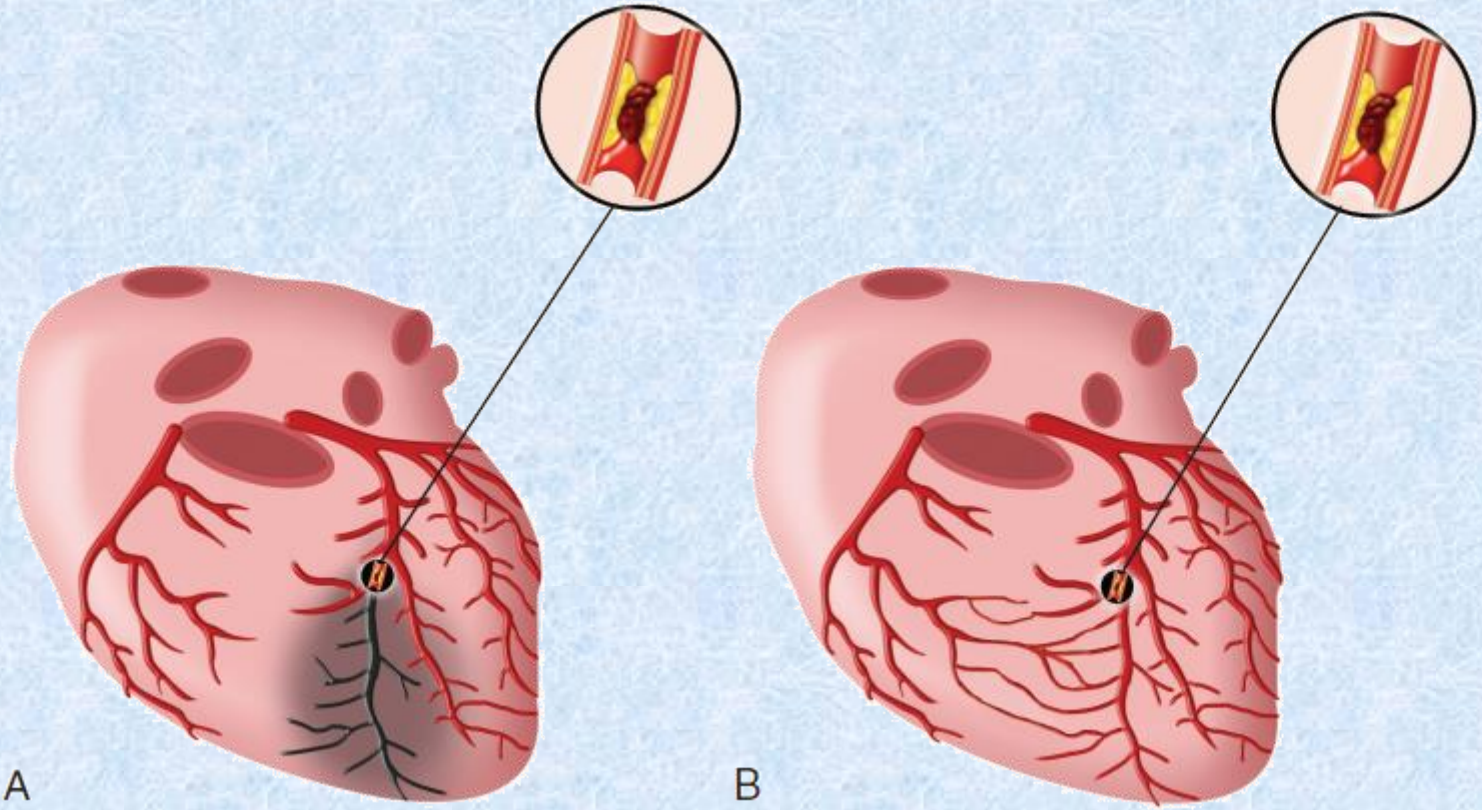
Heart Muscle

- The cellular effects of ischemia commence within seconds of the onset of hypoxia with the loss of adenosine triphosphate (ATP) production.

- Myocardial relaxation-contraction is compromised, and **irreversible** cell injury begins within as early as **20 minutes**.

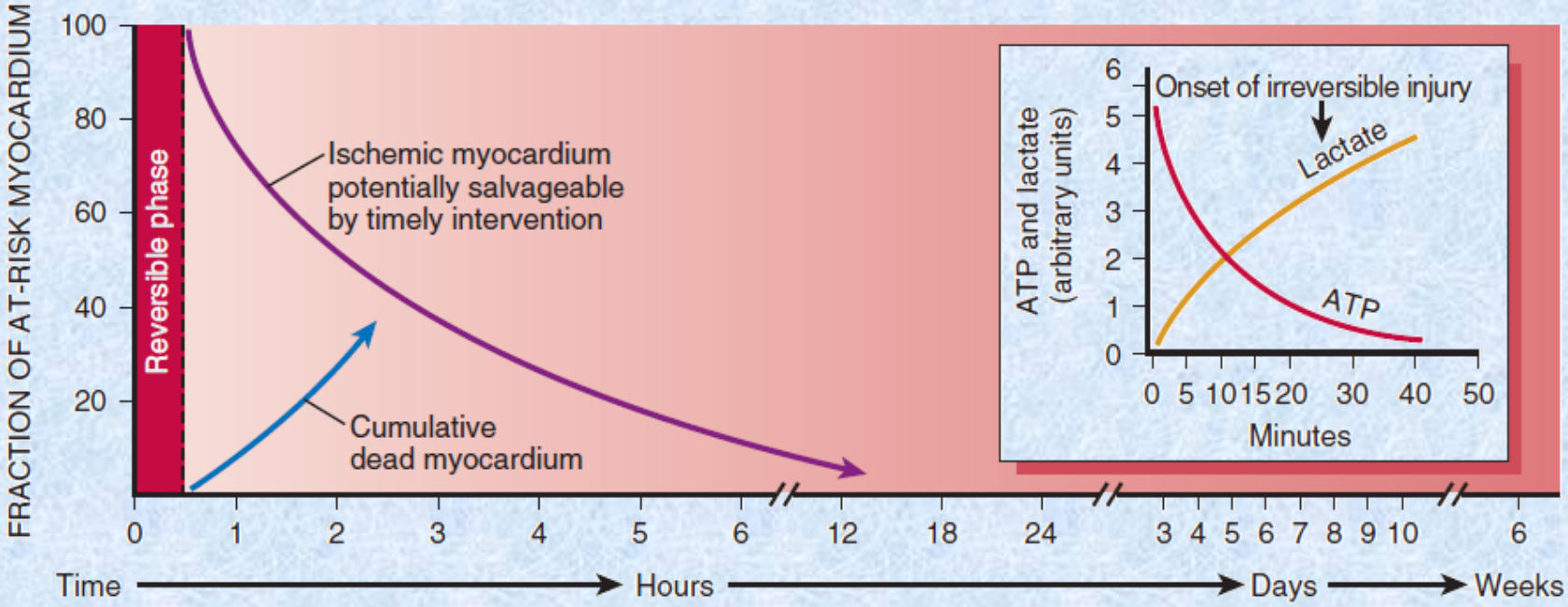
- **Necrosis** is usually **complete in 6 hours** unless reperfusion occurs or an extensive collateral circulation is present.

Schematic drawing of the coronary artery circulation of LAD



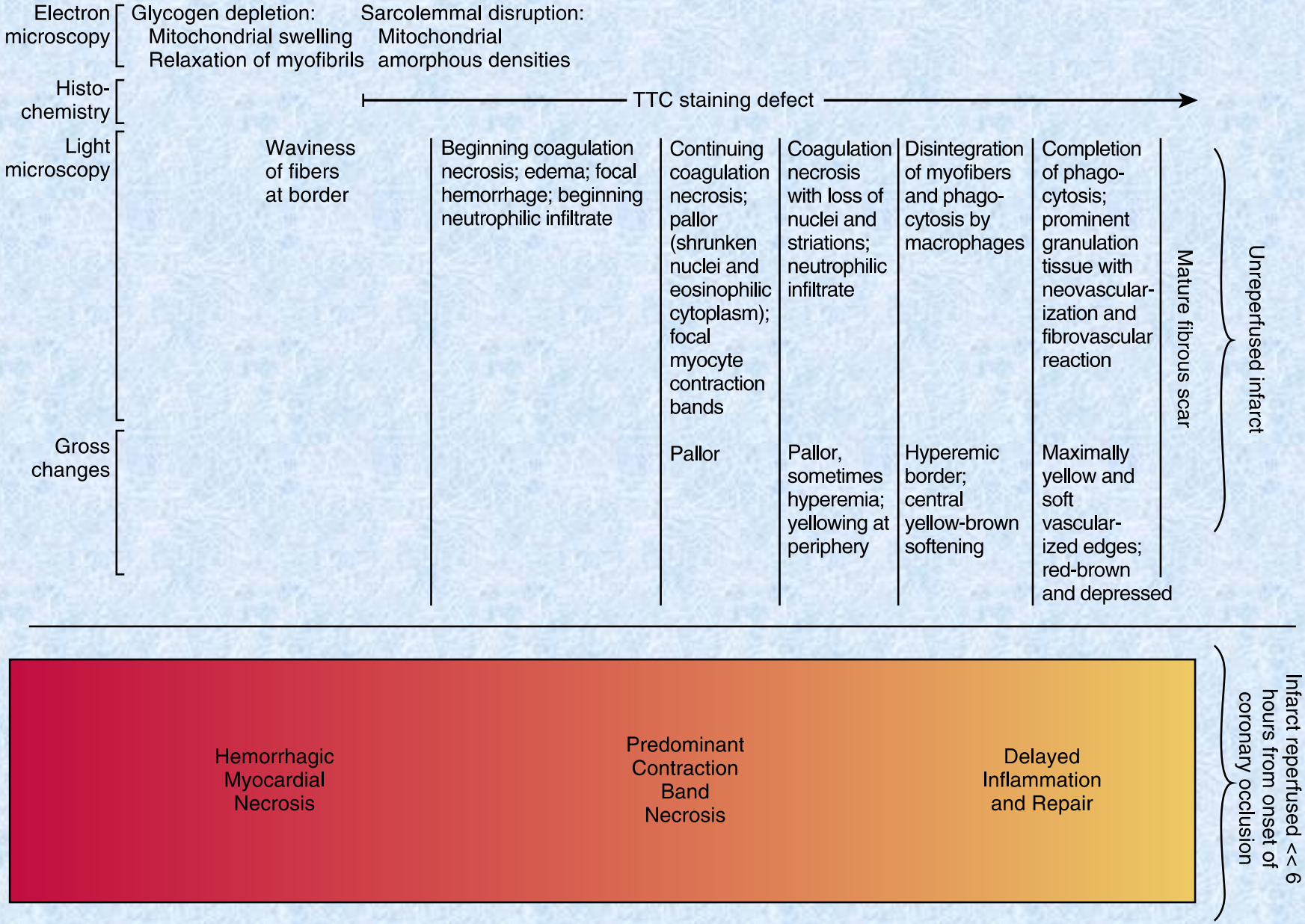
Without interarterial anastomoses

With interarterial anastomoses



✓ 30 minutes after the onset of severe ischemia, myocardial injury is potentially reversible;

✓ after this point, progressive loss of viability occurs and is complete by 6 to 12 hours.



Contraction band necrosis is a type of uncontrolled cell death (necrosis) unique to cardiac myocytes and thought to arise in reperfusion from hypercontraction, which results in sarcolemmal rupture

Right Ventricular Infarction

- Approximately 50% of patients with inferior infarction have some involvement of the right ventricle.
- Among these patients, right ventricular (RV) infarction occurs exclusively in those with transmural infarction of the inferoposterior wall and the posterior portion of the septum.
- RV infarction almost invariably develops in association with infarction of the adjacent septum and inferior left ventricular (LV) walls, **but isolated infarction of the right ventricle is seen in just 3% o 5% of autopsy-proven cases of M**I.
- RV infarction occurs less commonly than would be anticipated from the frequency of atherosclerotic lesions involving the right coronary artery.
- The right ventricle can sustain long periods of ischemia but still demonstrate excellent recovery of contractile function after reperfusion.

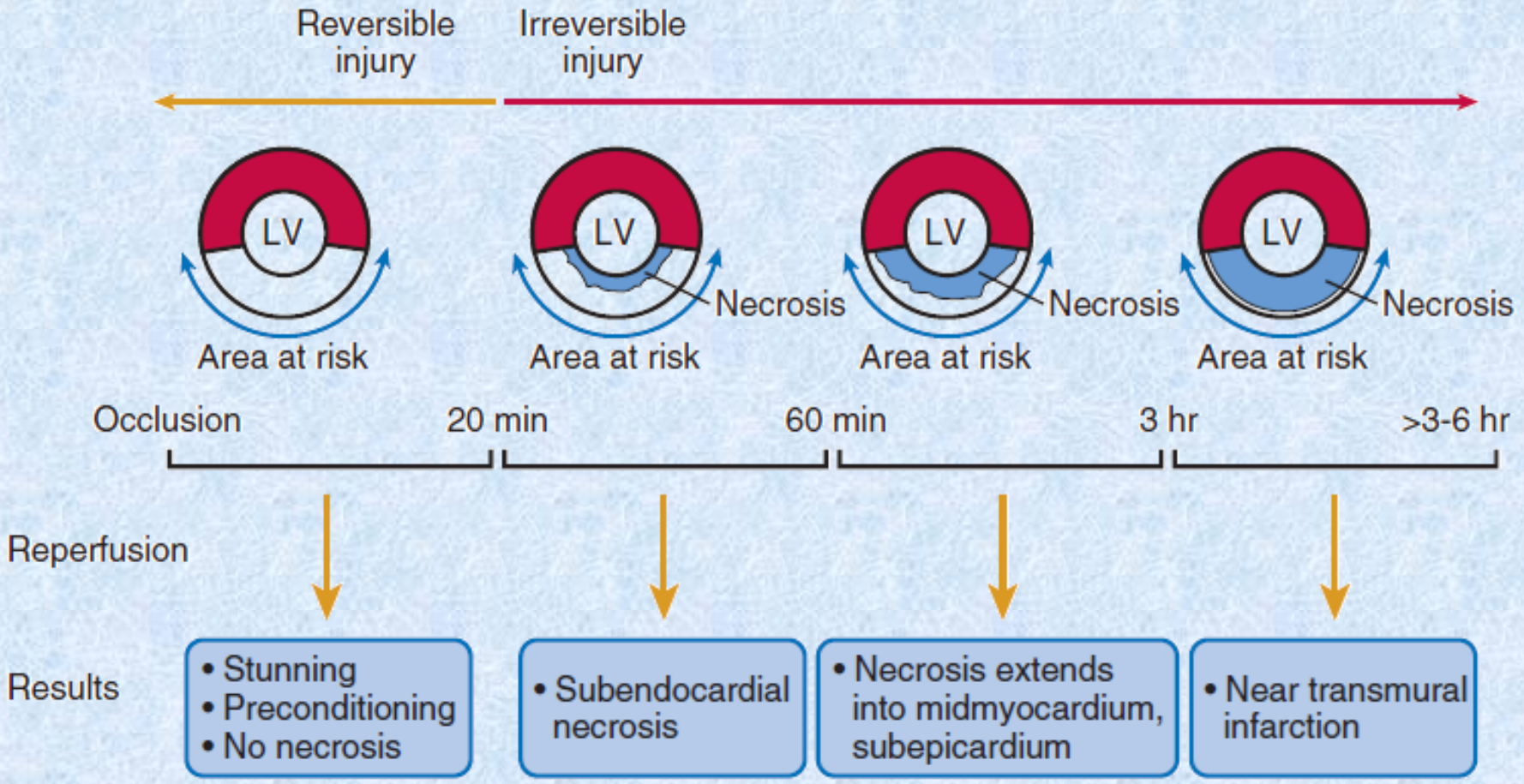
Atrial Infarction

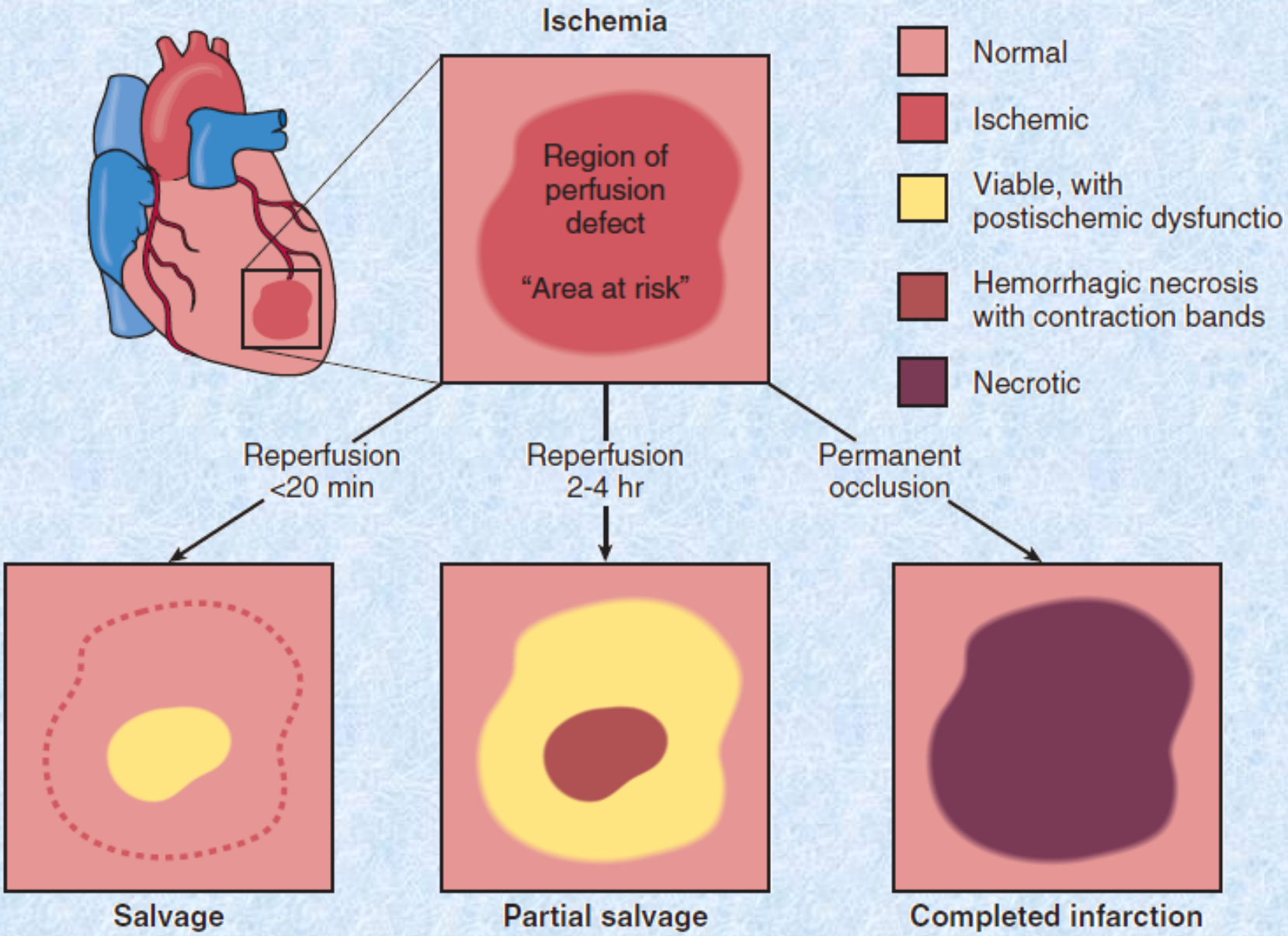
- This type of infarction occurs in **up to 10%** of patients with STEMI if PR-segment displacement is used as the criterion.

- Although **isolated atrial infarction is observed in only 3.5% of patients** with STEMI at autopsy, it often occurs in conjunction with ventricular infarction and can cause rupture of the atrial wall.

- This type of infarction is **more common on the right side than on the left side**,
- occurs **more frequently in the atrial appendages** than in the lateral or posterior walls of the atrium,
- and can **result in thrombus formation**.

- Atrial infarction is frequently accompanied by atrial arrhythmias and has been linked to **reduced secretion of atrial natriuretic peptide** and to a **low–cardiac output syndrome when RV infarction coexists**.


Reversible injury
Irreversible injury
LV
LV
LV
LV
Necrosis
Necrosis
Necrosis
Area at risk
Area at risk
Area at risk
Area at risk
Occlusion
20 min
60 min
3 hr
>3-6 hr
Reperfusion
Results
• Stunning
• Preconditioning
• No necrosis
• Subendocardial necrosis
• Necrosis extends into midmyocardium, subepicardium
• Near transmural infarction


Ischemia
Region of perfusion defect
"Area at risk"
Normal
Ischemic
Viable, with postischemic dysfunctio
Hemorrhagic necrosis with contraction bands
Necrotic
Reperfusion <20 min
Reperfusion 2-4 hr
Permanent occlusion
Salvage
Partial salvage
Completed infarction

Collateral Circulation in Acute Myocardial Infarction

- The coronary collateral circulation is particularly well developed in patients with coronary occlusive disease, **especially with reduction of the luminal cross-sectional area by more than 75% in one or more major vessels**; in patients with chronic hypoxia, as occurs in cases of severe anemia, chronic obstructive pulmonary disease, and cyanotic congenital heart disease; and in patients with LV hypertrophy.

- The magnitude of coronary collateral flow is a principal determinant of infarct size.

- **Even if the collateral perfusion existing at the time of coronary occlusion does not prevent infarction**, it may still exert a **beneficial effect by preventing the formation of LV aneurysms**.

- Patients with angiographic evidence of collateral formation have **improved angiographic and clinical out-comes after MI**.

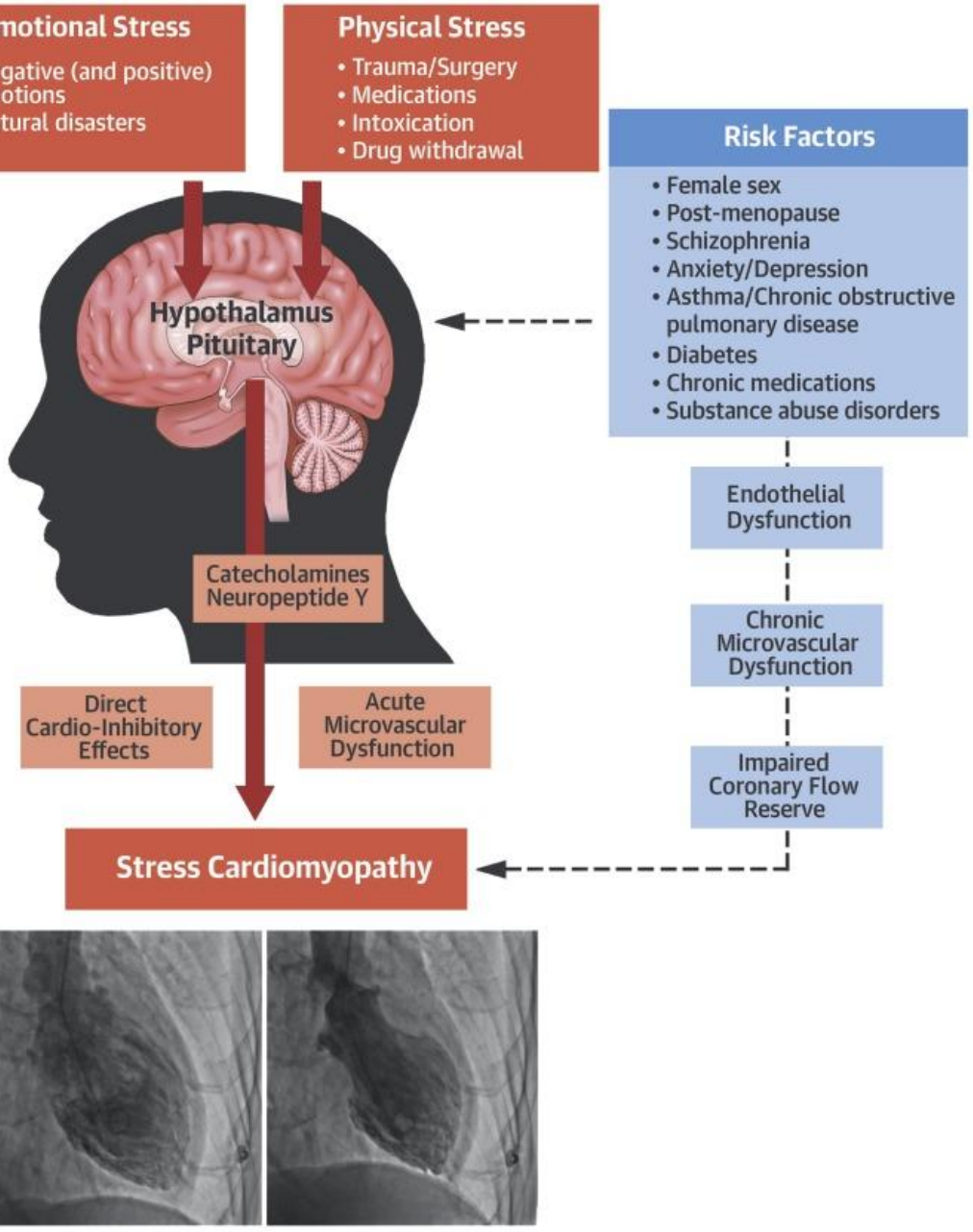
Myocardial Infarction with Angiographically Normal Coronary Vessels

- Patients with STEMI and normal coronary arteries tend to be **young** with **relatively few coronary risk factors** except that they often have a history of **cigarette** smoking.
- Usually, they have **no history of angina pectoris before the infarction**.
- These patients **do not generally have a prodrome before infarction**, but the clinical, laboratory, and electrocardiographic features of STEMI otherwise resemble those present in the overwhelming majority of patients with STEMI who have classic obstructive atherosclerotic coronary artery disease.
- Patients who recover frequently have areas of localized **dyskinesis and hypokinesis identified at LV angiography.**
- Many of these cases result **from coronary artery spasm and/or thrombosis**, perhaps with underlying endothelial dysfunction or plaque not apparent on coronary angiography.

Myocardial Infarction with Angiographically Normal Coronary Vessels

- The transient LV apical ballooning syndrome (**takotsubo cardiomyopathy**) is characterized by transient wall motion abnormalities involving the LV apex and mid-ventricle.
- This syndrome occurs in the absence of obstructive epicardial coronary disease and can mimic STEMI.
- Typically, an episode of psychological stress precedes the development of takotsubo cardiomyopathy.
- Initial ECGs demonstrate **significant and often diffuse ST-segment elevations** that when coupled with the typical (frequently severe) chest discomfort, prompts the appropriate immediate referral for coronary angiography.
- The cause is not clear, but **catecholamine- mediated myocardial stunning** and **microvascular dysfunction** play important roles.

CENTRAL ILLUSTRATION: Pathophysiology of Stress Cardiomyopathy



Medina de Chazal, H. et al. J Am Coll Cardiol. 2018;72(16):1955–71.

Myocardial Infarction with Angiographically Normal Coronary Vessels

- Additional suggested causes include
- (1) **coronary emboli** (perhaps from a small mural thrombus, a prolapsed mitral valve, or a myxoma);
- (2) coronary artery disease in **vessels too small to be visualized** on coronary arteriography or coronary arterial thrombosis with subsequent recanalization;
- (3) a **hematologic disorder** (polycythemia vera, cyanotic heart disease with polycythemia, sickle cell anemia, disseminated intravascular coagulation, thrombocytosis, and thrombotic thrombocytopenic purpura) causing in situ thrombsis in the presence of normal coronary arteries;
- (4) **augmented oxygen demand** (e.g., thyrotoxicosis, amphetamine use);
- (5) **hypotension secondary** to sepsis, blood loss, or pharmacologic agents; and
- (6) **anatomic variations** such as anomalous origin of a coronary artery, coronary arteriovenous fistula, or a myocardial bridge.

Myocardial Infarction with Angiographically Normal Coronary Vessels

- ***Prognosis***

- The long-term outlook for patients who have survived STEMI with angiographically normal coronary vessels appears to be **brighter** than for those with STEMI and obstructive coronary artery disease.

- After recovery from the initial infarct, recurrent infarction, heart failure, and death are **unusual** in patients with normal coronary arteries.

- Indeed, most of these patients have **normal finding on the exercise ECG**, and **very few develop angina** pectoris.

CLINICAL FEATURES

Predisposing Factors

- 1/2 of patients with STEMI have an **identifiable** precipitating factor:
- Accelerating angina and rest angina (UA) may culminate in STEMI.
- Noncardiac surgical procedures may also precede STEMI.

Predisposing Factors

- ✓ Reduced myocardial perfusion secondary to:

1. hypotension (e.g., hemorrhagic or septic shock) and
2. increased myocardial oxygen demands (aortic stenosis, fever, tachycardia, and agitation)

- ✓ Other factors:

- Respiratory infections,
- hypoxemia from any cause,
- pulmonary embolism,
- hypoglycemia,
- cocaine use,
- sympathomimetics,
- serum sickness,
- allergy,
- Prinzmetal angina (coronary artery spasm).

Circadian Periodicity

- The peak incidence of events occurring in the **morning.**

- Because:

1. increases in plasma catecholamines
2. Increases in cortisol and
3. increases in platelet aggregability.

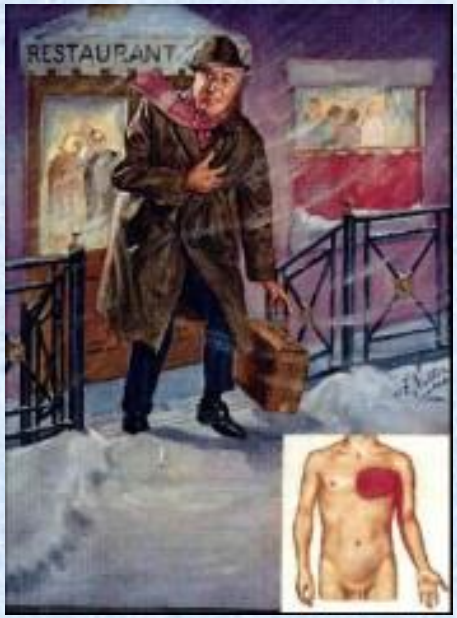
- This peak is *absent in patients receiving a* beta-blocking agent or aspirin

History

Nature of the Pain

- ❖ The pain in most patients is:
- ✓ severe
- ✓ intolerable.
- ✓ prolonged (> 30 minutes)
- ✓ discomfort (constricting, crushing, or compressing)
- ✓ complains of a sensation of a heavy weight or a squeezing in the chest.
- ✓ choking, viselike, or heavy pain,
- ✓ stabbing, knifelike, boring, or burning discomfort.

- ❖ Location of pain:
- ✓ usually localizes retrosternally
- ✓ frequently spreads to both sides of the anterior part of the chest, with a predilection for the left side.
- ✓ Often the pain radiates down the ulnar aspect of the left arm
- ✓ may begin in the epigastrium



Other Symptoms

- ✓ Nausea and vomiting

1. because of activation of the vagal reflex, Bezold-Jarisch reflex or side effects of opiates).
2. more commonly in patients with inferior STEMI

- If epigastric pain + nausea and vomiting, can be confused with:

1. acute cholecystitis,
2. gastritis, or
3. peptic ulcer.

- Other:

- ✓ feelings of profound weakness,
- ✓ dizziness,
- ✓ palpitations,
- ✓ cold perspiration, and
- ✓ a sense of impending doom.

Differential Diagnosis

1. Acute pericarditis (pleuritic features, coughing and often involves the shoulder, ridge of the trapezius, and neck).

2. Pulmonary embolism (laterally pain in the chest, pleuritic, and may be associated with hemoptysis).

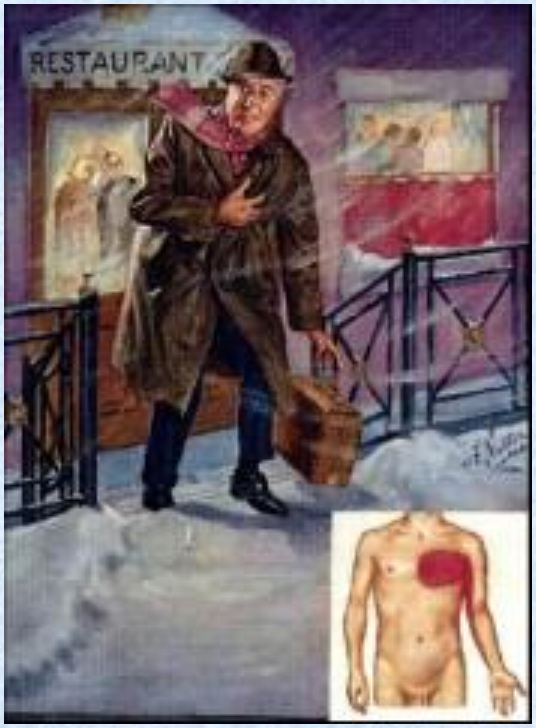
3. Acute aortic dissection (localized to the center of the chest, severe, described a “ripping” or “tearing” sensation, maximal intensity shortly after onset)

4. Tension pneumothorax

Physical Examination

General Appearance

- Appear:



- ✓ anxious
- ✓ distress.
- ✓ restless and
- ✓ move about in an effort to find a comfortable position.

- ✓ often massage or clutch their chests
- ✓ Levine sign

- ✓ If LV failure and sympathetic stimulation: cold perspiration and skin pallor

- **Heart Rate**

✓ The heart rate can vary from marked bradycardia to a rapid regular or irregular tachycardia

✓ Most commonly: sinus tachycardia at 100 to 110 beats/minute

- **Blood Pressure**

✓ Most patients with uncomplicated STEMI are normotensive.

✓ In *previously normotensive* patients, a hypertensive response is seen (secondary to pain, anxiety, and agitation).

✓ *Previously hypertensive* patients often become normotensive, although many of them eventually regain their elevated levels of blood pressure, generally 3 to 6 months after MI.

- **Temperature**
- Fever:

✓ a nonspecific response to tissue necrosis, develops in most patients
✓ within 24 to 48 hours of the onset of infarction.
✓ rectal temperature may reach 38.3°C to 38.9°C
✓ The fever usually resolves by the 4^{th} to 5^{th} day after infarction.

- **Respiration**

✓ The respiratory rate may rise slightly after STEMI;

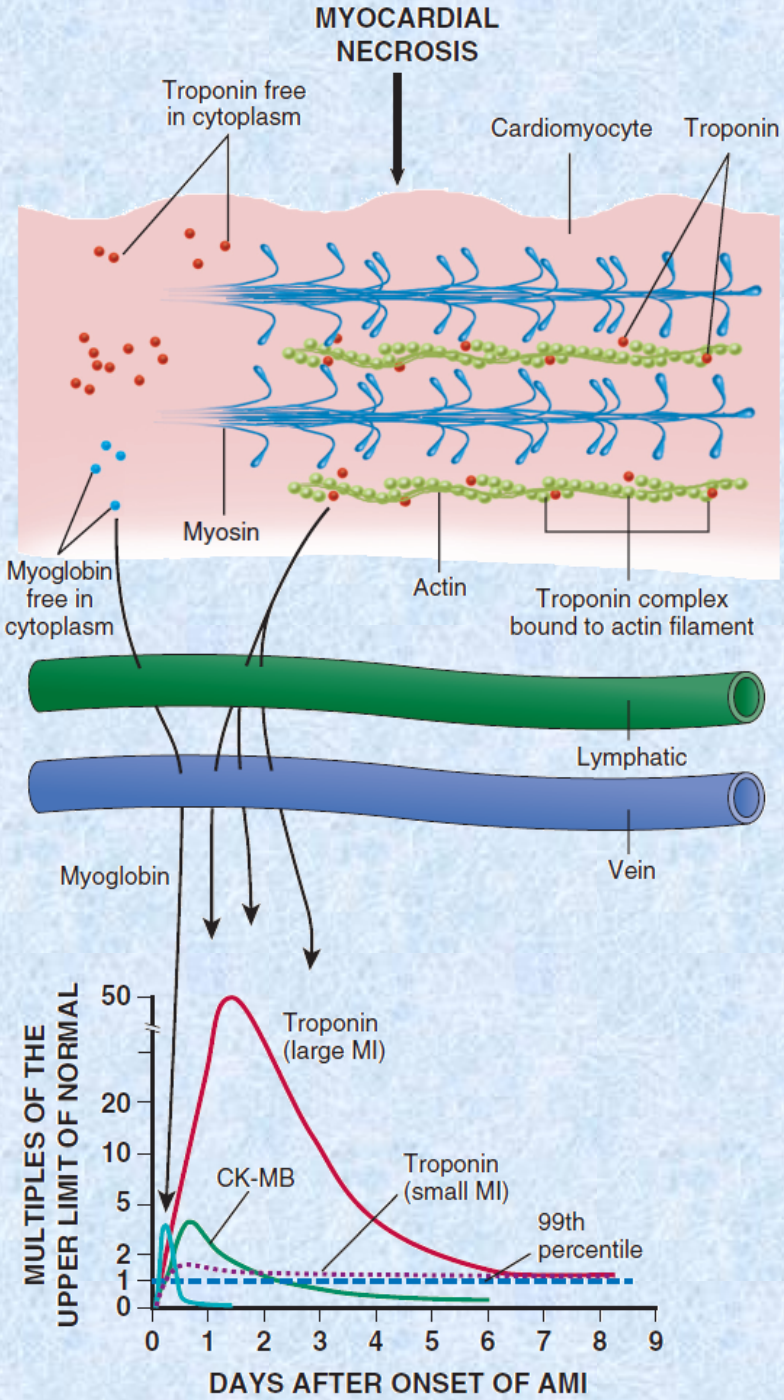
✓ In patients with LV failure, the respiratory rate *correlates with the severity of the failure*;
✓ patients with pulmonary edema may have respiratory rates > 40 per minute.

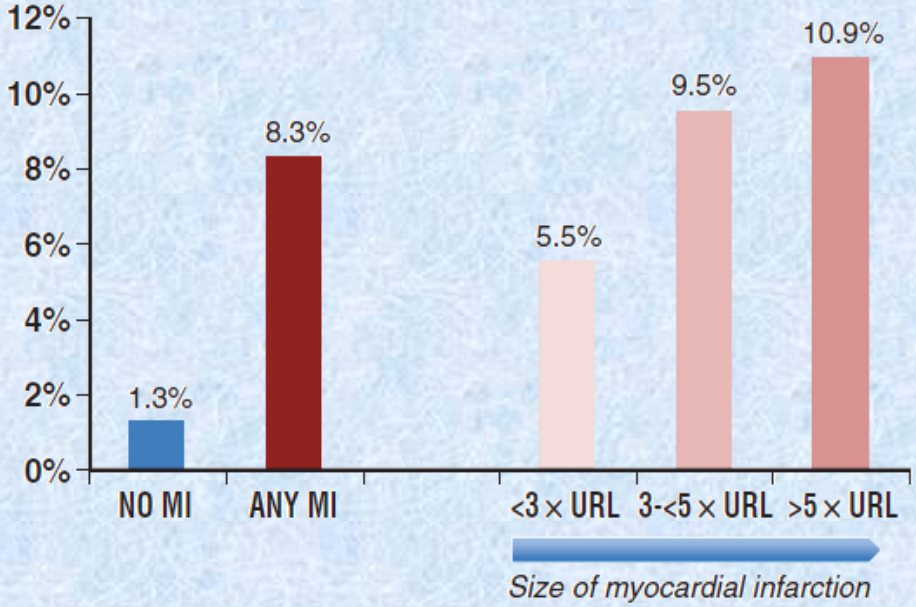
- **The Chest**
- Rales are audible in patients in whom LV failure and/or a reduction in LV compliance with STEMI develops.

- Cough with hemoptysis, suggesting pulmonary embolism with infarction, can also occur.

- *Killip* classes:
- Class I (free of rales and a S3).
- Class II (Rales <50% of lung fields ± S3).
- Class III (Rales >50% of lung fields and pulmonary edema).
- Class IV (Cardiogenic shock).

Laboratory Findings





Risk for cardiovascular death associated with new or recurrent MI stratified according to MI size

Electrocardiography

ECG OF AMI (in the absence of LBBB)

ST Elevation

New ST elevation at the J point in two contiguous leads with the following cut points:

- ≥0.1 mV in all leads (except V_2-V_3)
- In leads V_2-V_3 the following cut points apply:
  - ≥0.2 mV in men ≥40 years
  - ≥0.25 mV in men <40 years
  - ≥0.15 mV in women

ST Depression and T Wave Changes

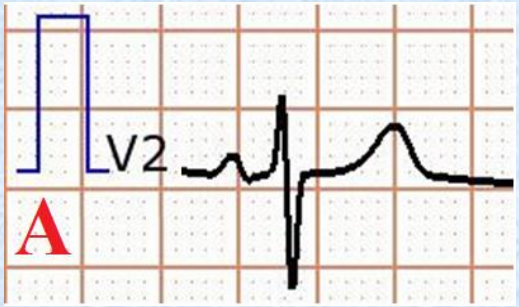
- New horizontal or downsloping ST depression ≥0.05 mV in two contiguous leads
- T-wave inversion ≥0.1 mV in two contiguous leads with a prominent R wave or R/S ratio >1

ECG OF AMI (in the setting of LBBB)

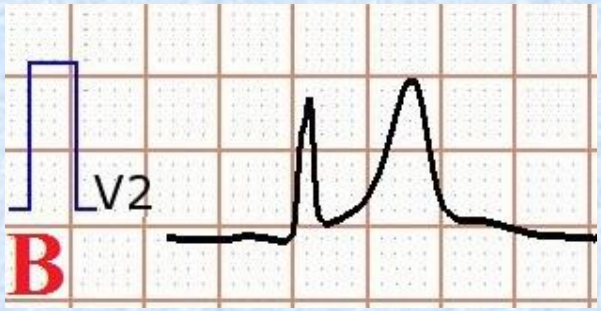
Electrocardiographic Criterion	Points
ST-segment elevation ≥1 mm and concordant with the QRS complex	5
ST-segment depression ≥1 mm in lead V_1, V_2, or V_3	3
ST-segment elevation ≥5 mm and discordant with the QRS complex	2

A score of ≥3 had a specificity of 98% for acute MI

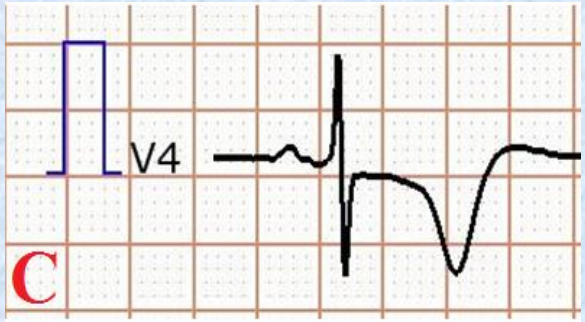
Table 6. Upper limits of normal J point elevation based on various conditions.	
Leads V2 and V3	
men ≥ 40 years	0.2 mV
men < 40 years	0.25 mV
Women	0.15 mV
Leads (except V2 and V3)	0.1 mV



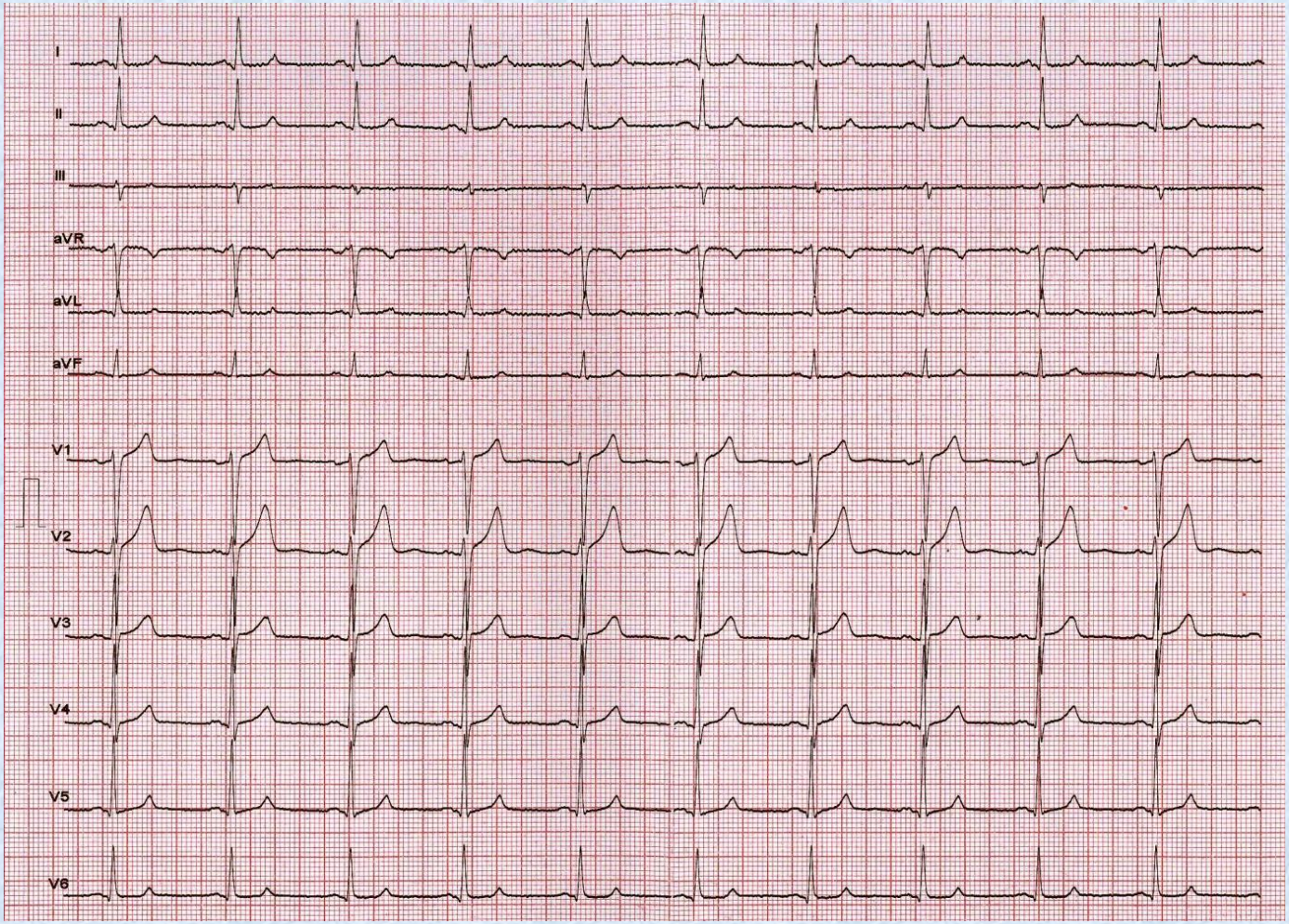
Normal T wave



Hyperacute T wave, suggesting subendocardial ischemia



T wave, suggesting transmural ischemia



Subendocardial Ischemia. ECG from a 50 years old man with significant stenosis in mid portion of left anterior descending coronary artery. The amplitude of upright T wave in lead V1 is larger than lead V6.

Location of STEMI

Inferoposterior STEMI

RCA occlusion

- ST depression in leads I and aVL > 1 mm
- ST elevation lead III greater than in lead II
- ST depression in lead aVL > lead I

Proximal

- ST elevation more than 1 mm with positive T wave in lead V4R
- ST elevation in lead V1

Distal

- ST isoelectric with a positive T wave in lead V4R
- ST depression in leads V1-V2

CX coronary artery occlusion

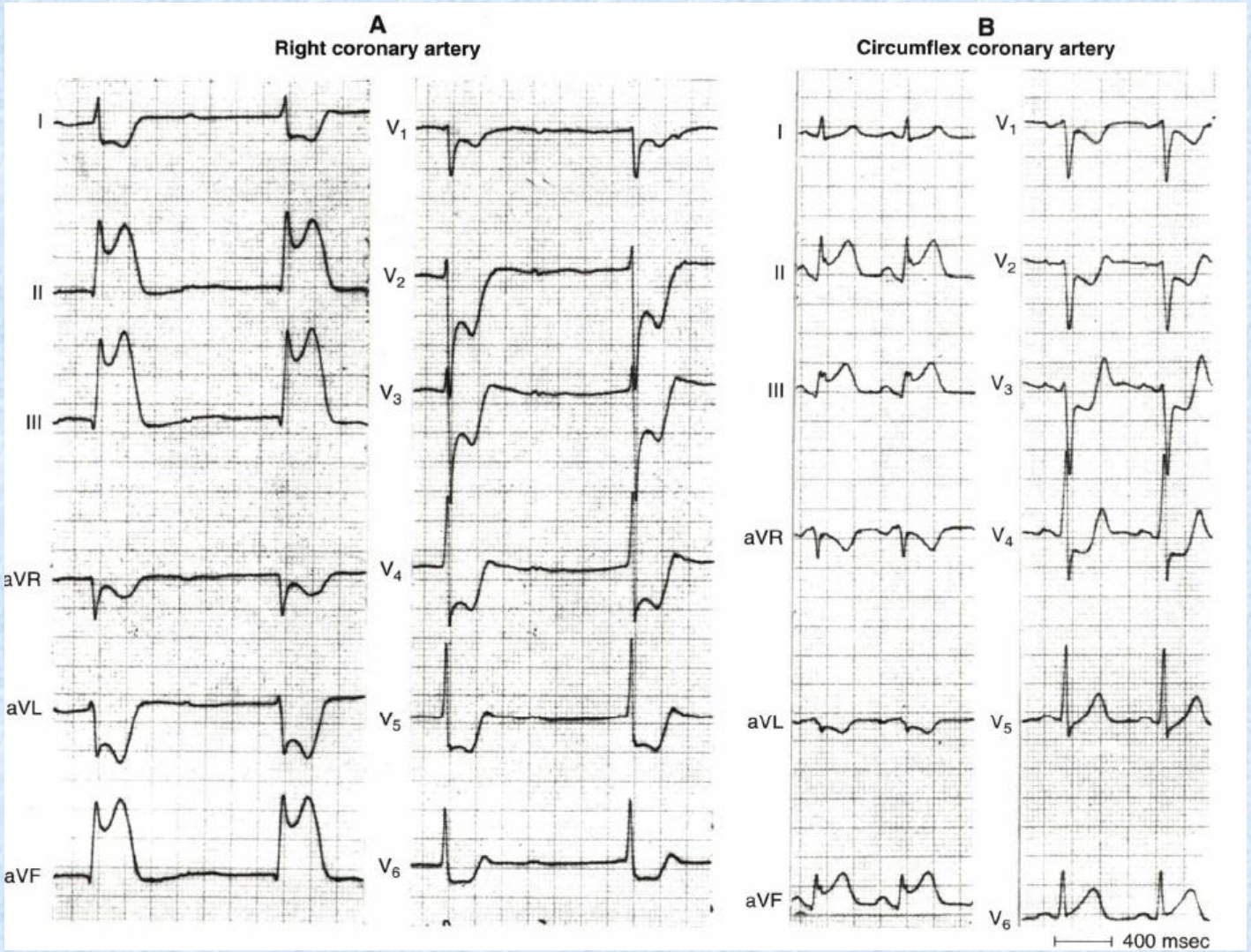
- ST elevation in lead II greater than lead III
- ST isoelectric or elevated in lead I
- ST isoelectric or depressed with negative T wave in lead V4R
- ST depression in leads V1- V3

Extension to Posterior Wall

- ST depression in precordial leads
- Tall R-wave in V1-V2

Extension to Lateral Wall

- ST elevation in leads I, aVL, V5 and V6


A
Right coronary artery
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
B
Circumflex coronary artery
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
400 msec

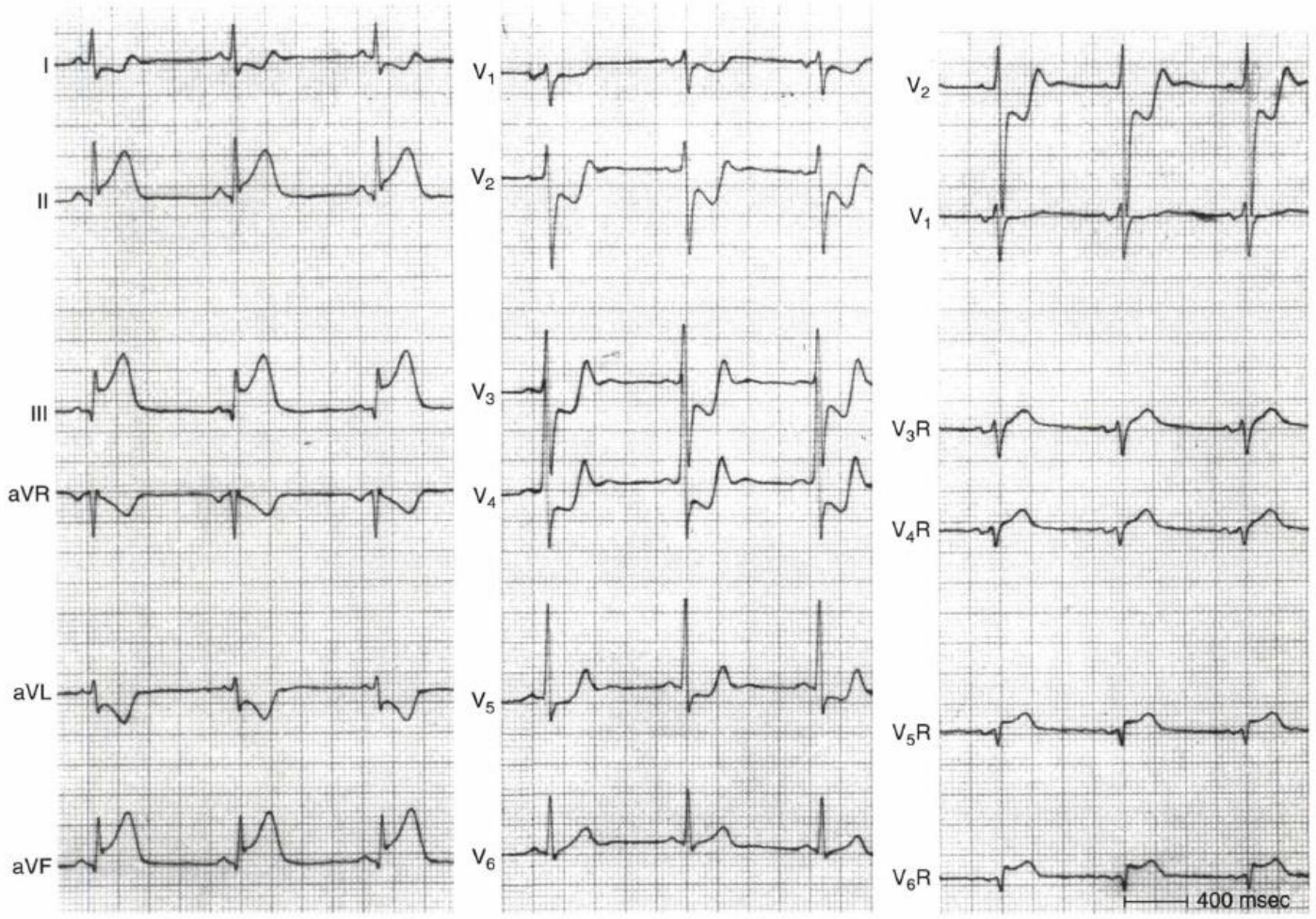


Figure 1-4 Inferoposterior ST segment elevation in MI with RV involvement caused by occlusion of the proximal RCA. RCA occlusion is diagnosed by ST depression in lead I and ST elevation in the inferior leads (more ST elevation in lead III than in lead II). The diagnosis that occlusion is proximal in the RCA is based on ST elevation with a positive T wave in lead V_4R.

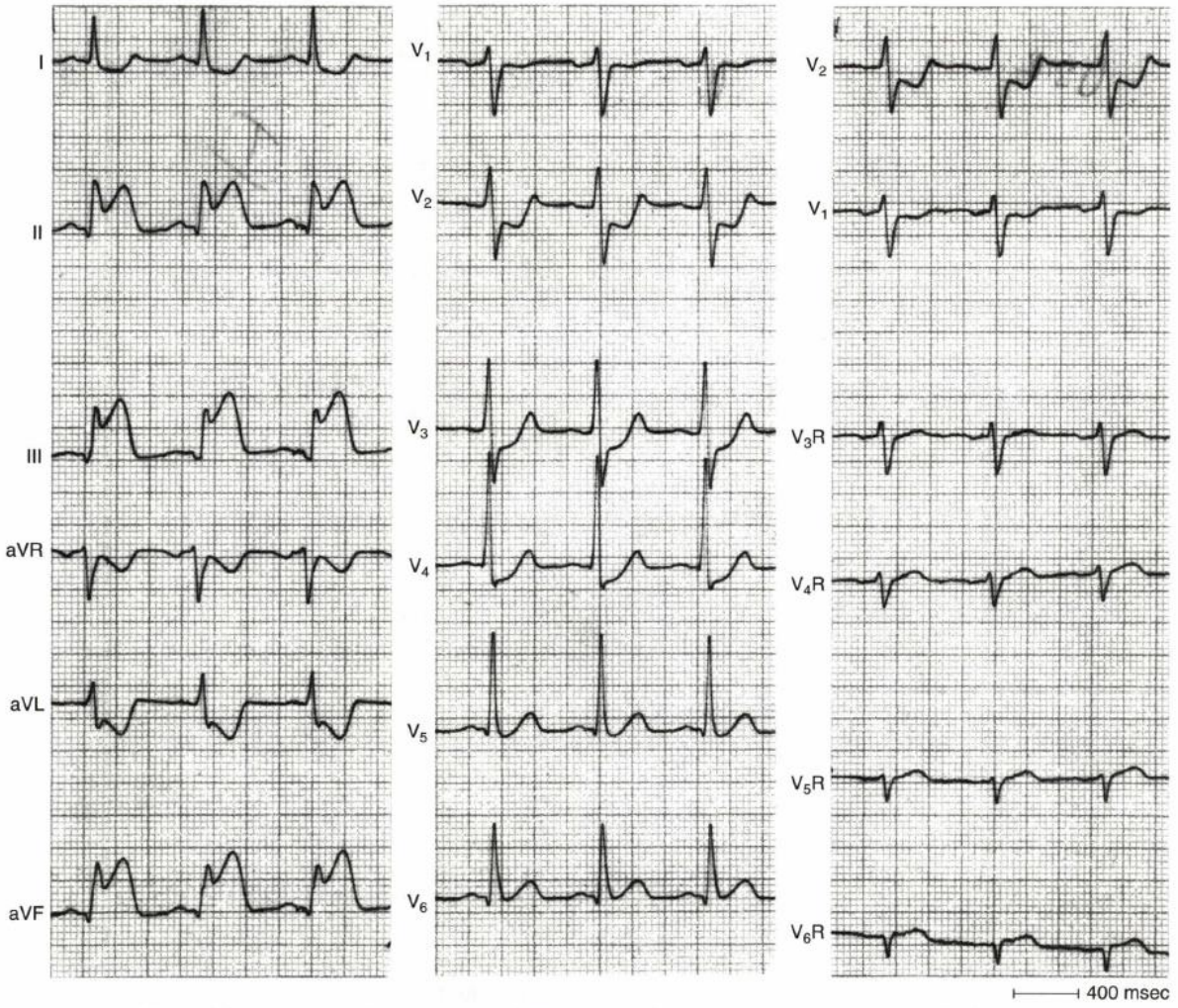


Figure 1-5 Inferoposterior ST segment elevation MI with no RV involvement. Note that leads I, II, and III indicate occlusion of the RCA (ST depression in lead I and elevation in II, III, and aVF, with more ST elevation in III than in II). Lead V_4R shows no ST elevation because the occlusion is distal to the RV branch.

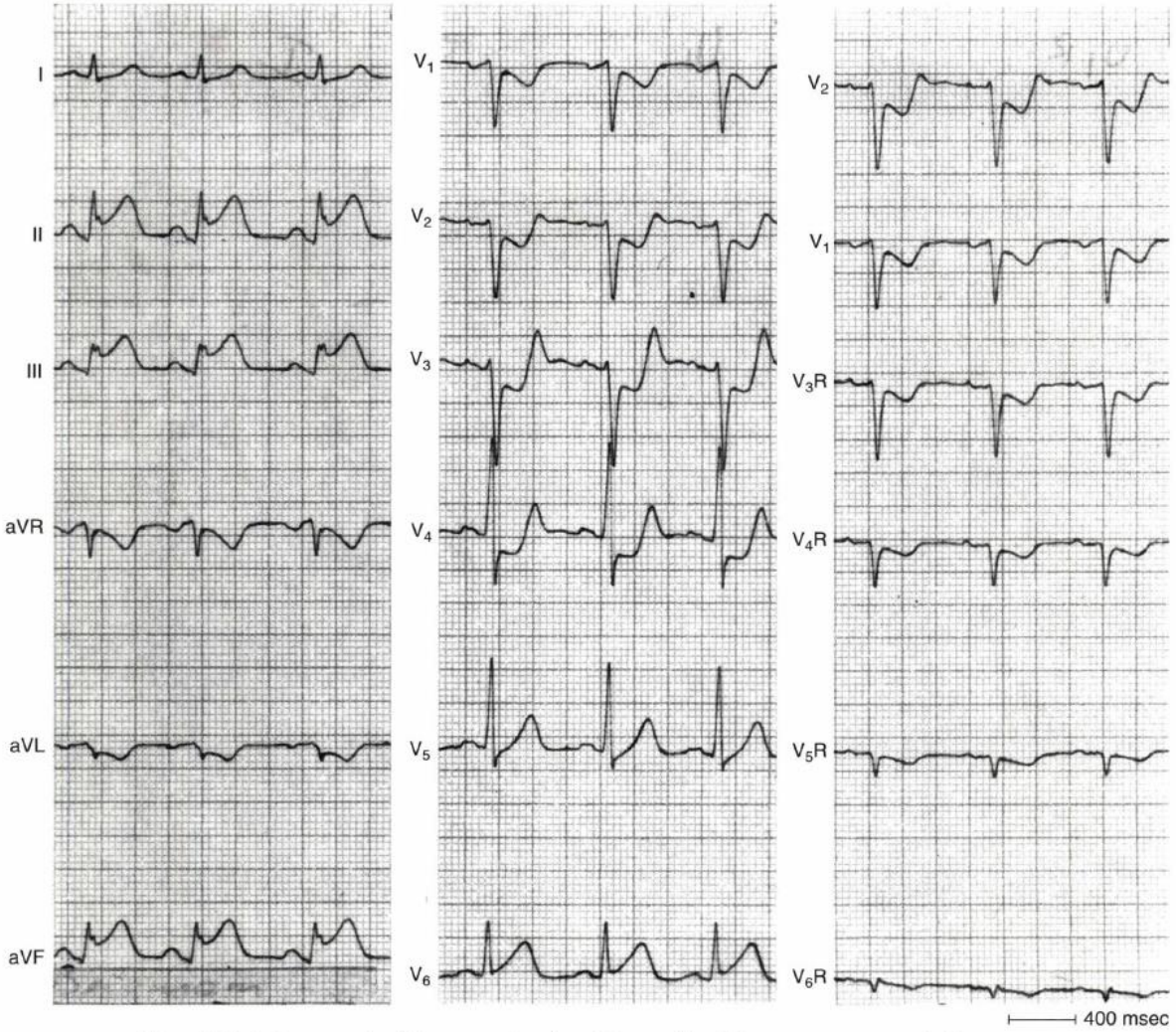


Figure 1-6 Inferoposterior ST segment elevation MI caused by CX coronary artery occlusion, reflected in the facts that ST elevation is more marked in lead II than in lead III, and ST segment depression and a negative T wave are shown in lead V_4R. Lead I also shows a positive T wave. Of interest is the notch at the end of the QRS in leads II, III, and aVF, indicating delayed activation of the basolateral area, a typical finding in CX coronary artery occlusion.

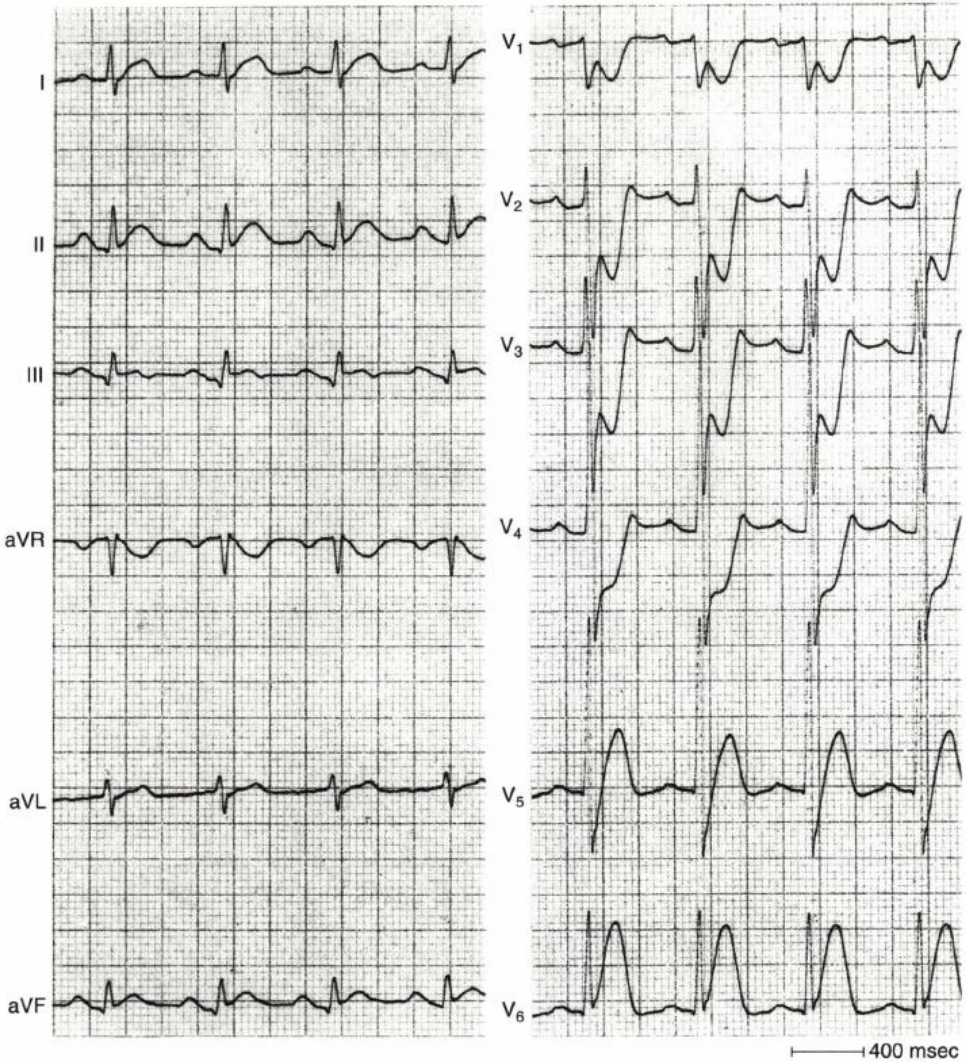


Figure 1-7 Inferoposterior ST segment elevation MI with lateral wall involvement. Note the ST elevation in leads reflecting the lateral wall (I, aVL, V_5, and V_6). As indicated by the ST depression in V_1 to V_4, this is an occlusion of a dominant CX coronary artery.

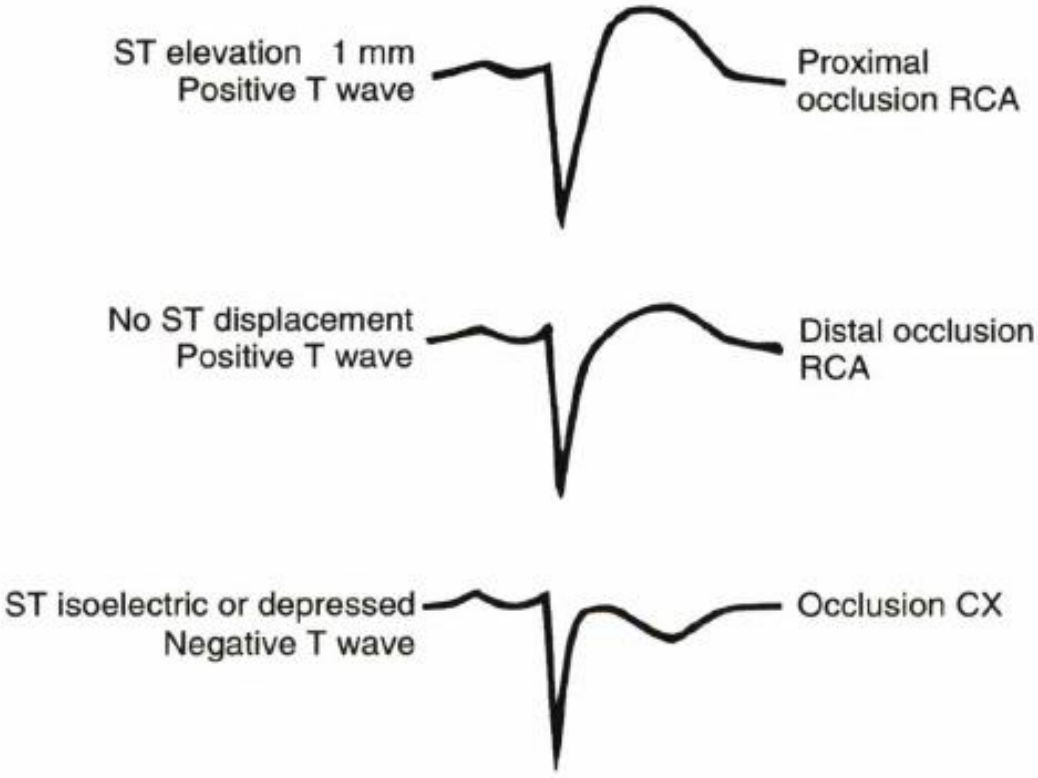
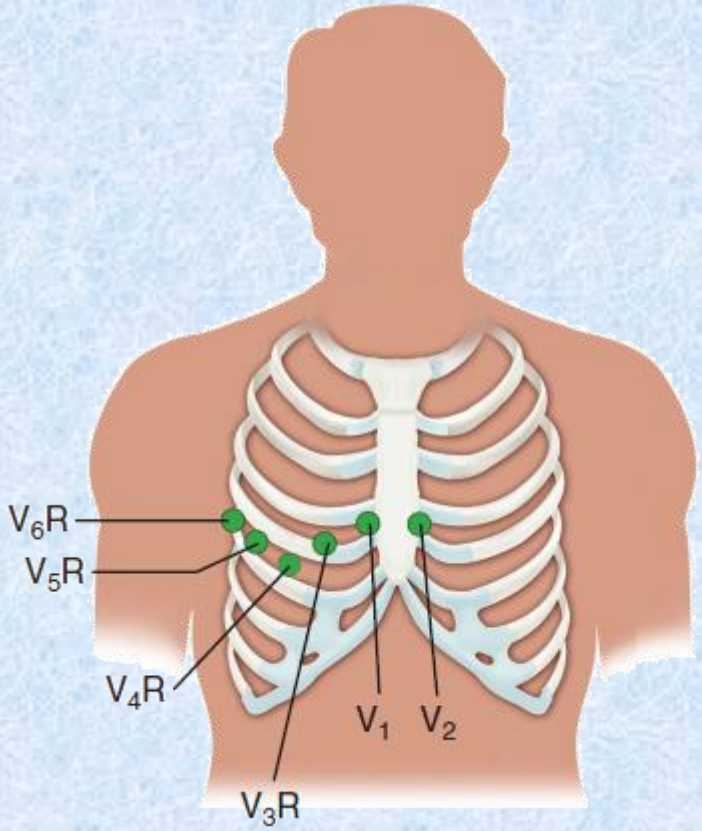


Figure 1-8 Value of ST-T segment changes in lead V_4R in acute inferoposterior MI.

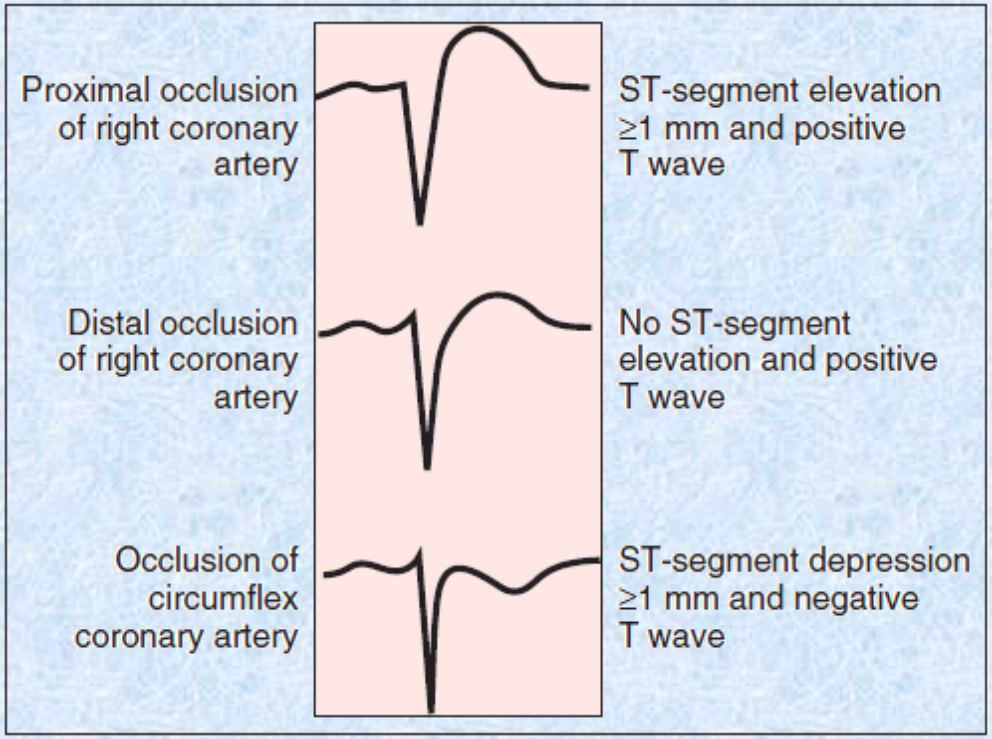
RV MI



Clinical findings:
- Shock with clear lungs, elevated JVP
- Kussmaul sign

Hemodynamics:
- Increased RA pressure
- Square root sign in RV tracing

Management:
- Maintain RV preload
- Lower RV afterload
- Restore AV synchrony
- Inotropic support
- Reperfusion



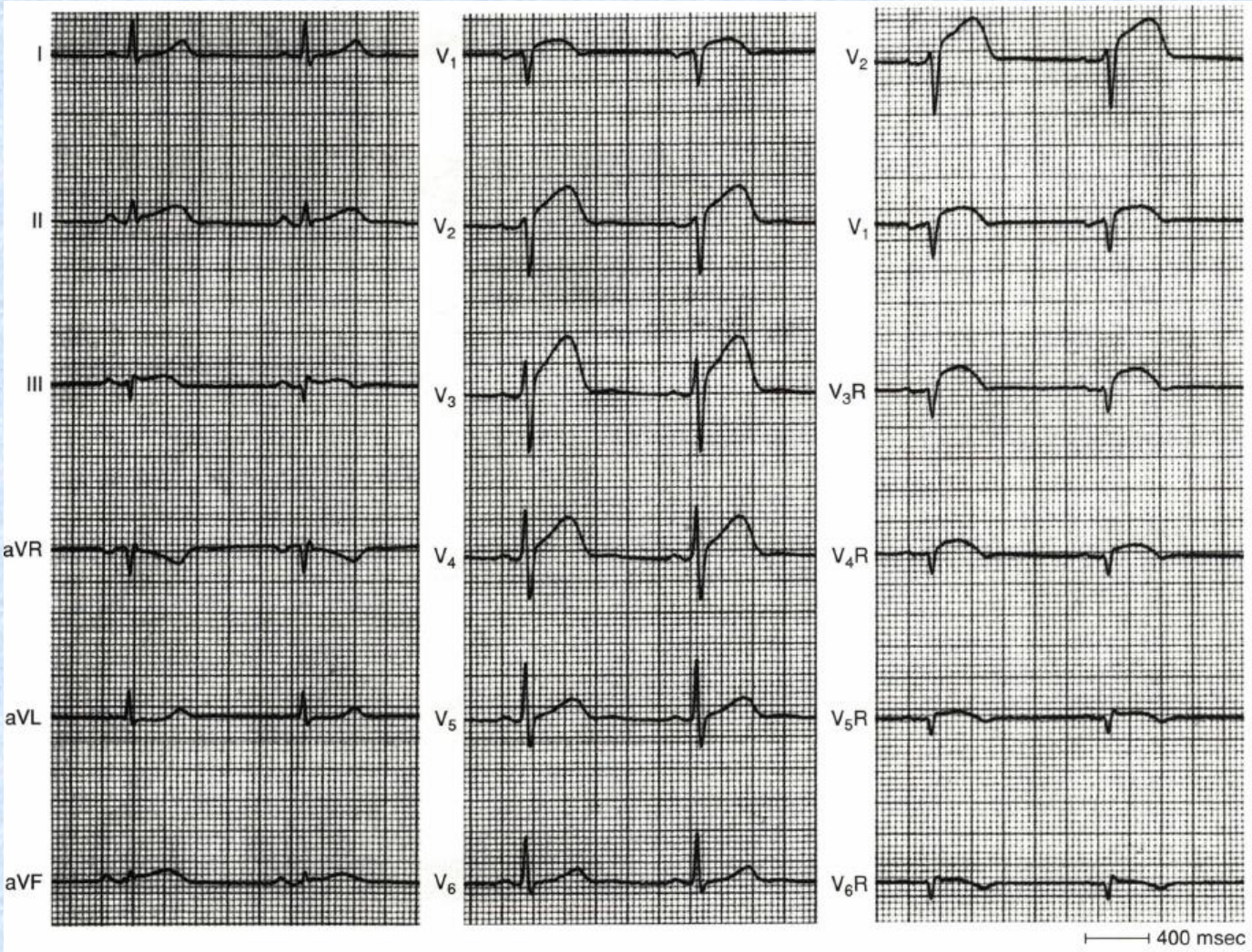


Figure 1-11 Acute isolated RV MI. Note that ST elevation is present in right and left precordial leads. This pattern should not be confused with that of anterior wall STEMI.

Anterior wall STEMI (LAD Occlusion)

Proximal to first septal branch and first diagonal branch

ST deviation vector points to the base of the heart, causing:

- ST elevation in leads aVR and aVL
- ST depression in leads II and III (> 1mm), and aVF (> 2mm)
- ST elevation in lead V1 (>2 mm) and leads V2 to V4
- ST isoelectric or depressed in leads V5 and V6

NOTE: Acquired intra-Hissal block or RBBB may occur.

Distal to first septal branch, proximal to first diagonal branch

ST deviation vector points to lead aVL,causing:

- ST elevation in leads I and aVL
- ST depression in lead III (lead II is isoelectric), but not ST depression in leads II, aVF
- ST elevation in leads V2 to V6 but not in lead V1

Distal to first diagonal branch, proximal to first septal branch

ST deviation vector points away from lead aVL and toward lead III, causing:

- ST depression in lead aVL and aVR
- ST elevation in inferior leads, highest in lead III
- ST elevation in leads V1 to V4

Distal LAD

ST deviation vector points apically toward lead III, causing:

- ST depression aVL and aVR
- ST elevation in inferior leads, highest in II
- ST elevation in leads V3 to V6

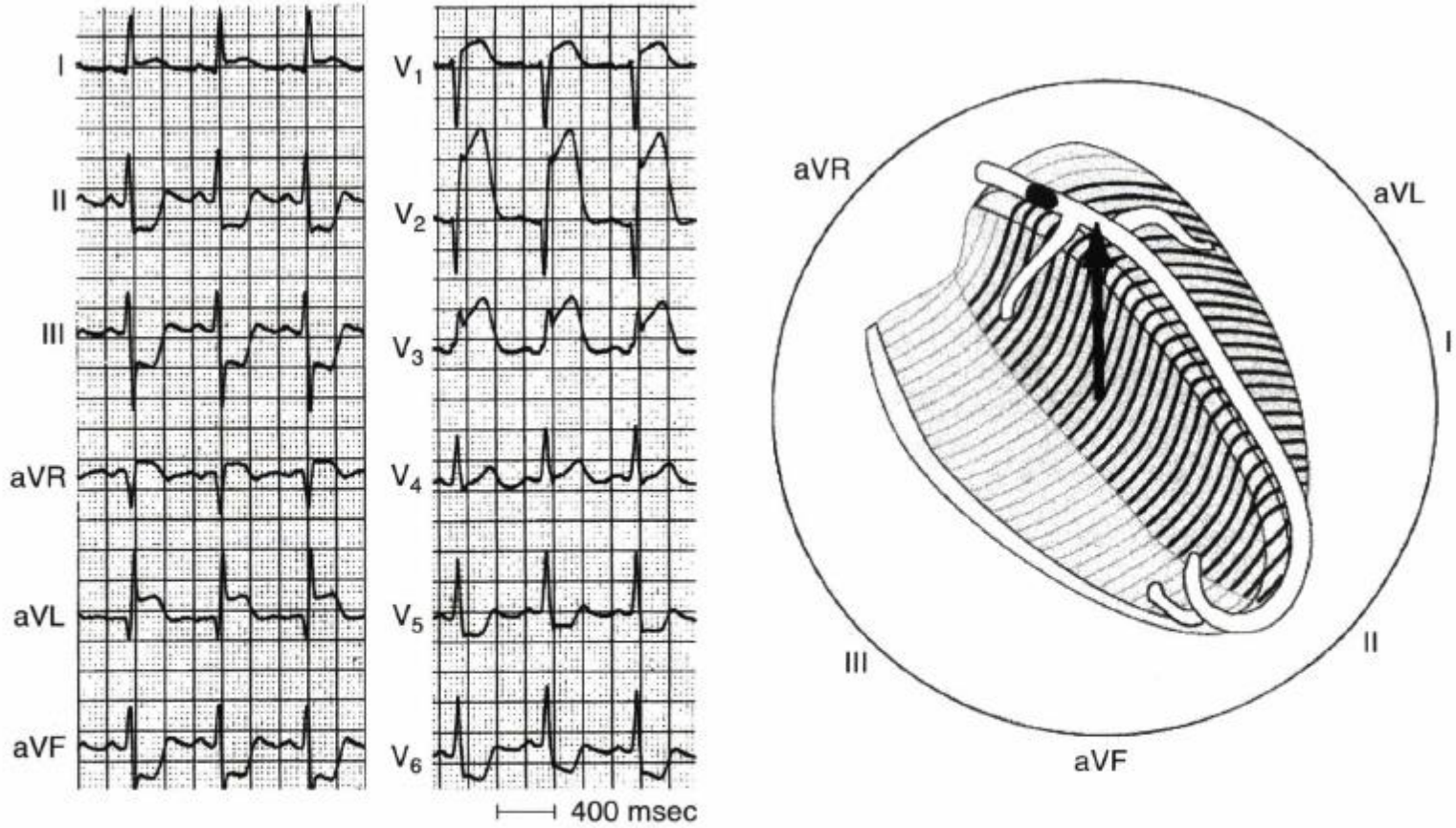


Figure 1-12 Acute anterior MI caused by an LAD occlusion proximal to the first septal and the first diagonal branch. Global ischemia of the whole anterior and septal aspects of the LV leads to a superiorly directed ST deviation vector because the anterobasal segment is the dominant ischemic area. This vector leads to ST elevation in leads aVR, aVL, and V_1, with reciprocal ST depression in the inferior leads and leads V_5 and V_6.

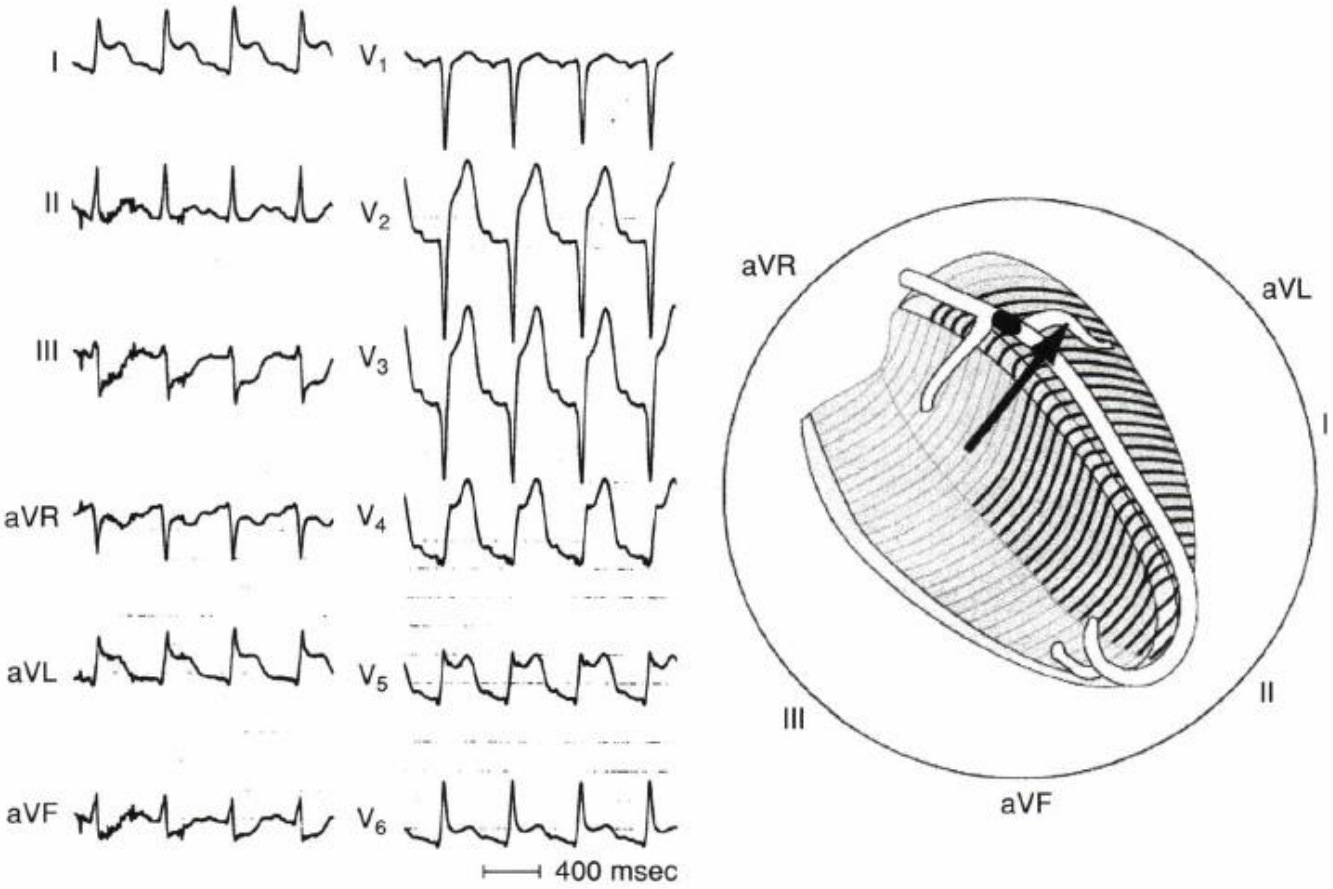


Figure 1-13 Acute anterior MI caused by an occlusion site in the LAD distal to the first septal branch but proximal to the first diagonal branch. The dominant ischemic area is located anterolaterally, leading to an ST deviation vector pointing in that direction. This results in ST elevation in leads I and aVL and ST depression in lead III. Lead II is isoelectric because of the perpendicular orientation of the ST vector in that lead.

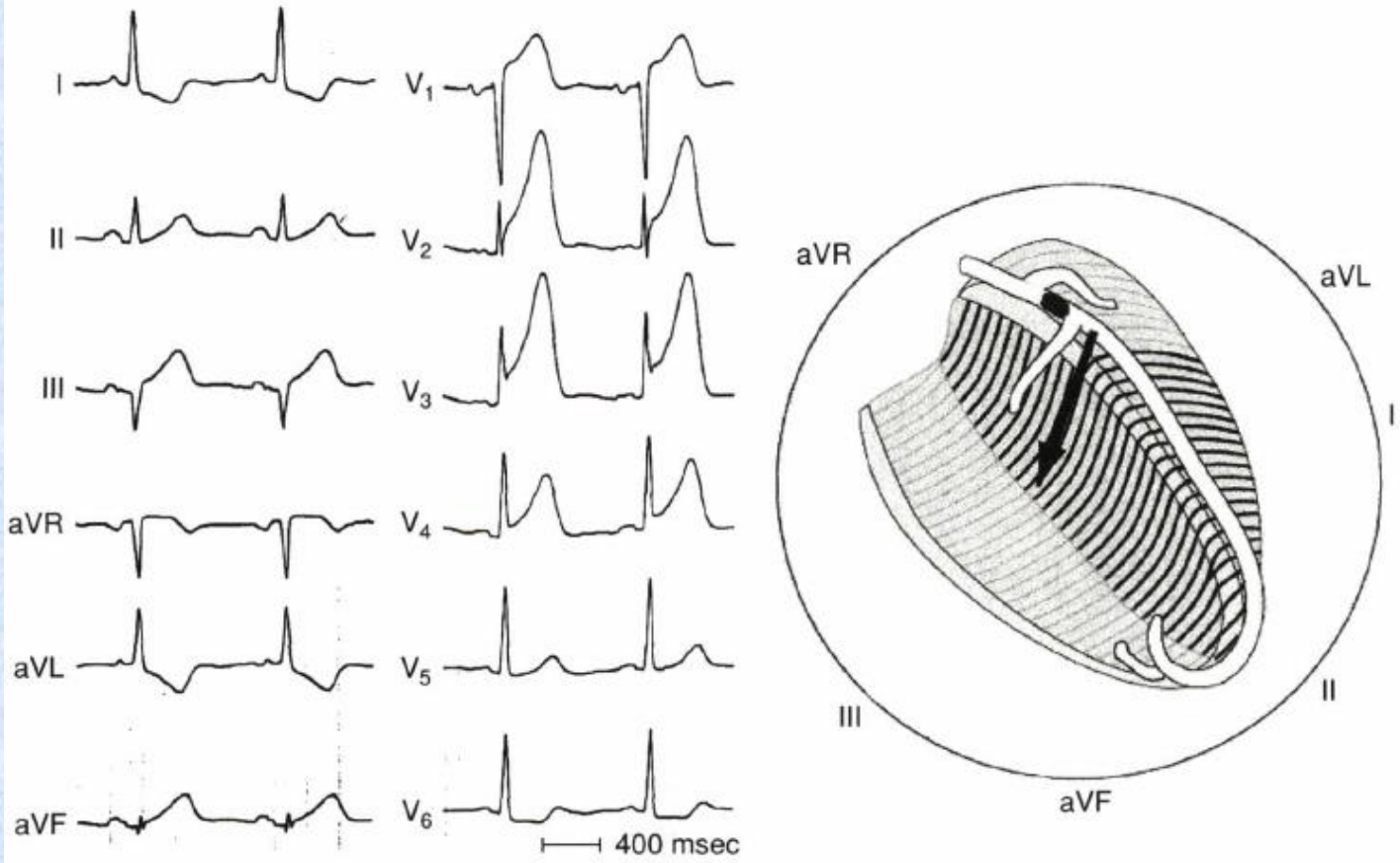


Figure 1-14 Acute anterior MI caused by an occlusion in the LAD distal to the first diagonal or intermediate branch but proximal to the first septal branch. This occlusion results in dominant ischemia in the anteroseptal area leading to a rightward and inferiorly directed ST deviation vector, which in turn results in ST elevation in leads V_1, aVR, and III and ST depression in leads I and aVL.

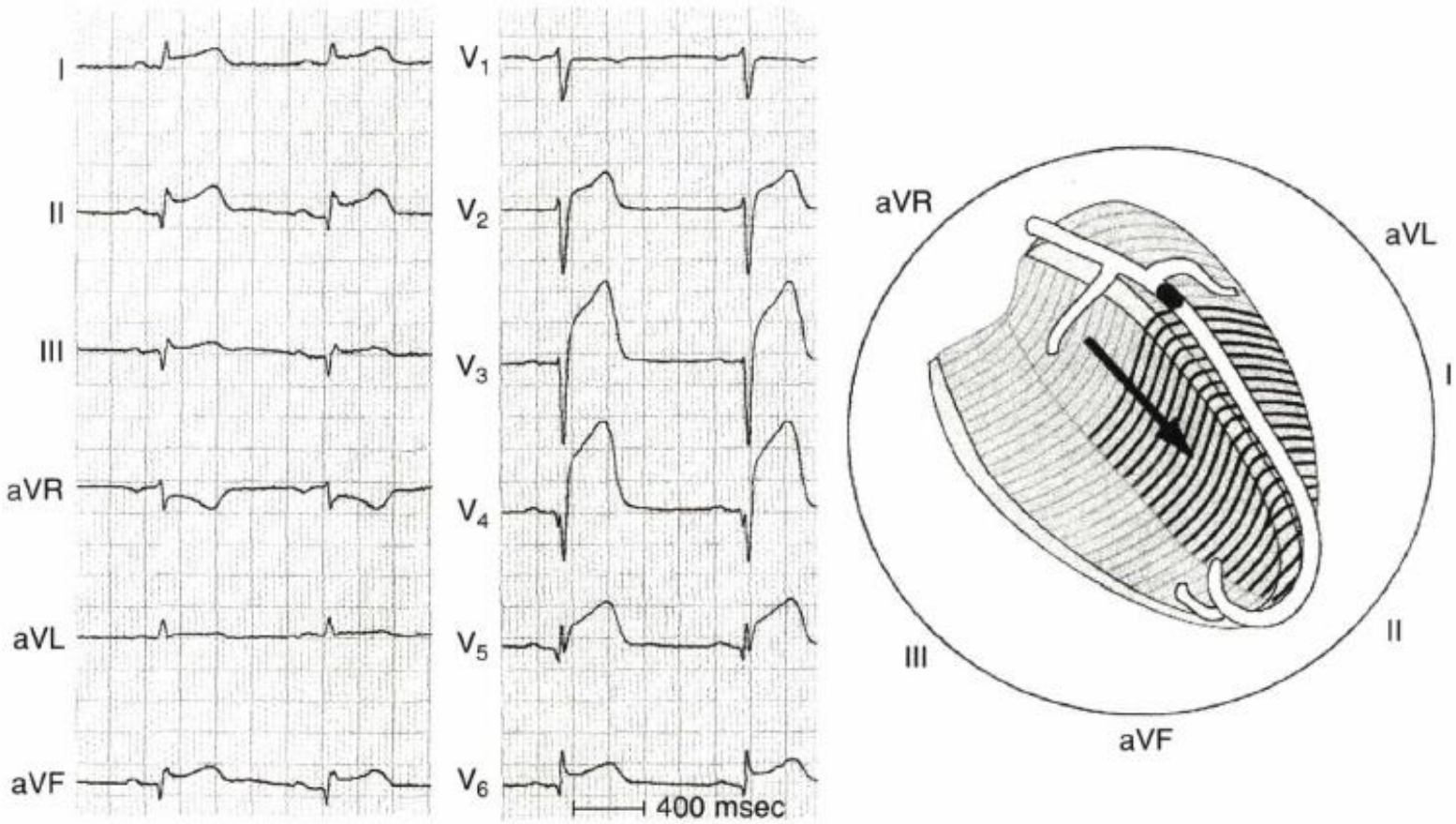


Figure 1-15 Acute anterior MI caused by a distal LAD occlusion. Because the ischemic area is located inferoapically, the ST deviation vector in the frontal plane points in an apical direction, resulting in ST elevation in the inferior leads (lead II greater than lead III), V_5, and V_6.

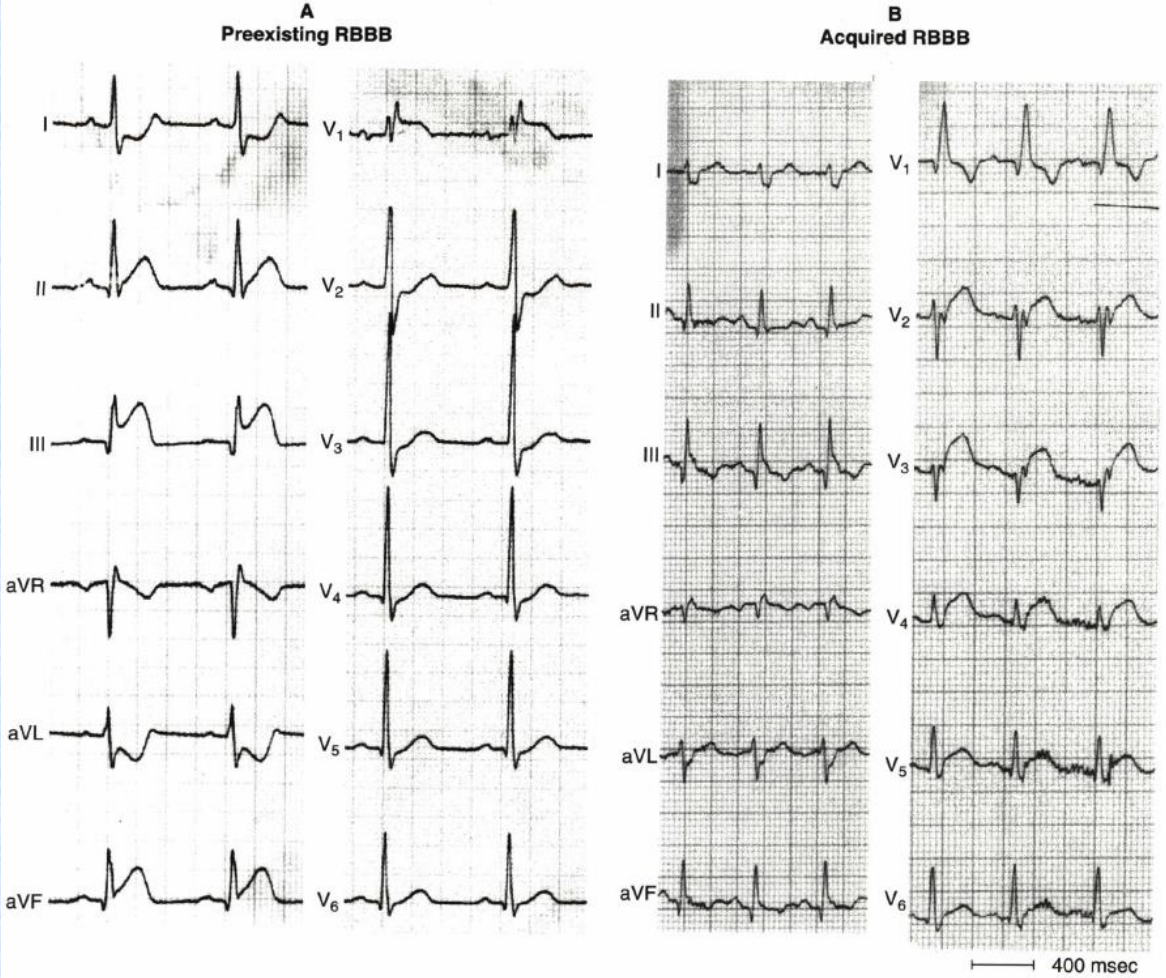
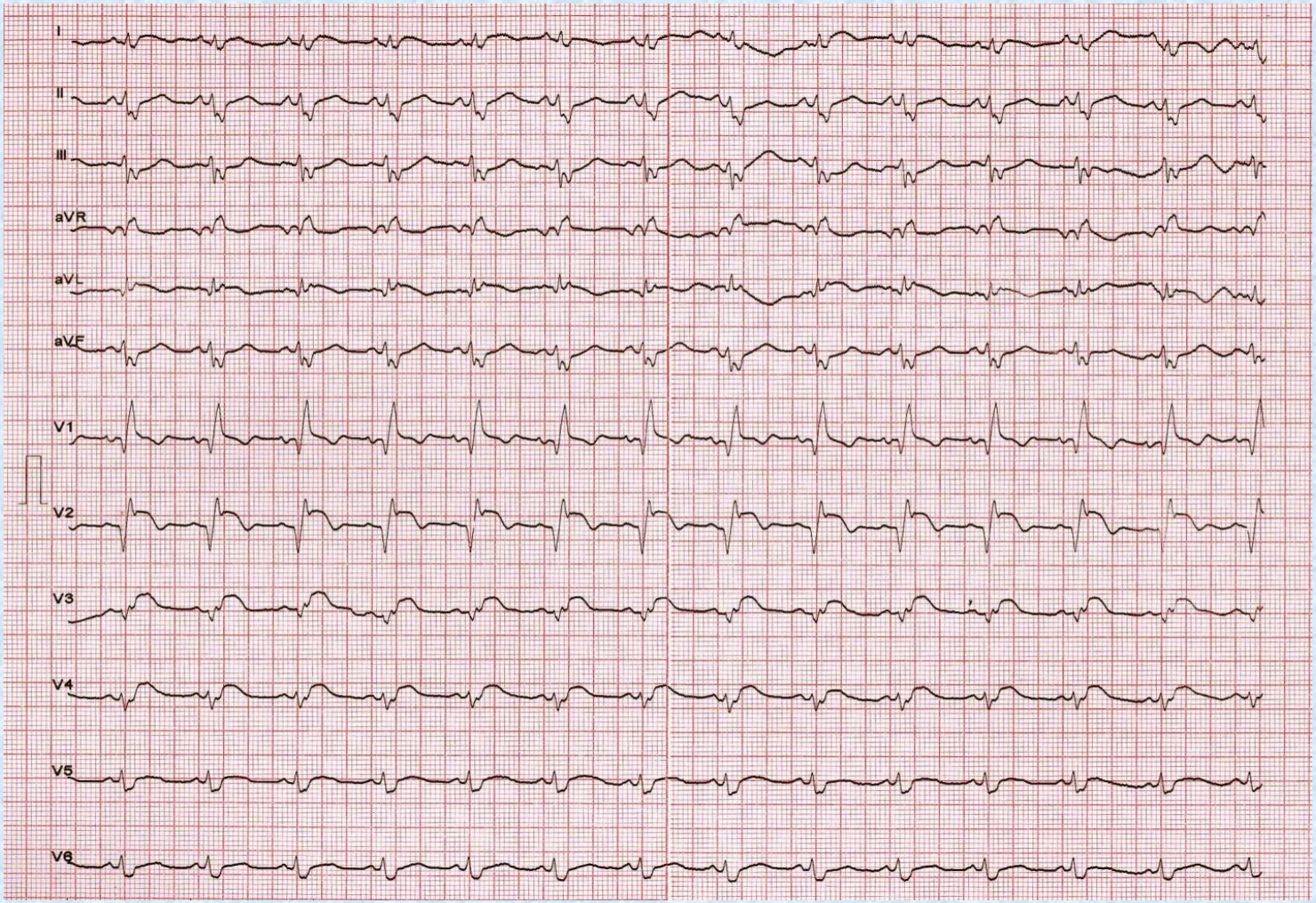


Figure 1-33 Preexisting RBBB versus acquired RBBB after MI. **A,** The ECG of a patient with preexisting RBBB admitted with an inferior MI from an RCA occlusion. The QRS during RBBB shows an RsR′ configuration in lead V_1. The ST elevation in lead V_1 indicates RV involvement. **B,** The ECG of a patient with an acute anterior wall MI with acquired RBBB and left posterior hemiblock. The QRS during RBBB shows a qR configuration in lead V_1.



Acquired RBBB in patient with extensive anterior STEMI. In contrast to preexciting RBBB (rs R' or RsR' configuration), the QR complex in lead V1 is seen.

- STEMI equivalents:

1. Isolated posterior MI.
2. Left main coronary artery occlusion pattern.
3. De Winter pattern.
4. Wellens syndrome.
5. LBBB with Sgarbossa criteria.

Diagnostic criteria for specific ECG patterns in patients with angina.
Isolated posterior MI
▪ Horizontal ST depression in lead V1-4 ▪ Dominant R wave (R/S ratio > 1) in V1 or V2 ▪ Upright T waves ▪ ST elevation in posterior leads (leads V7-9)
Left main coronary artery occlusion pattern.
▪ ST elevation in lead aVR and V1 (aVR > V1) ▪ ST depression in leads II and aVF (basal ischemia) ▪ ST depression in the precordial lead to the left of V2 (posterior wall ischemia) ▪ RBBB (may be present, because of ischemia in sub-AV nodal conduction system)
De Winter pattern
▪ Tall, prominent, symmetric T waves in the precordial leads ▪ Upsloping ST segment depression (1-3 mm) at the J-point in the precordial leads ▪ Absence of ST elevation in the precordial leads ▪ ST segment elevation (1-2 mm) in lead aVR
Wellens syndrome
▪ Symmetric and deeply inverted T waves in leads V2 and V3 (occasionally in leads V-6) or ▪ Biphasic T wave in leads V2 and V3; plus ▪ Isoelectric or minimally elevated (<1 mm) ST segment ▪ No precordial Q waves
LBBB with Sgarbossa criteria
▪ ST-segment elevation ≥1 mm and concordant with the QRS complex (5 points) ▪ ST-segment depression ≥1 mm in lead V1, V2, or V3 (3 points) ▪ ST-segment elevation ≥5 mm and discordant with the QRS complex (2 points) A score of ≥3 had a specificity of 98% for acute MI

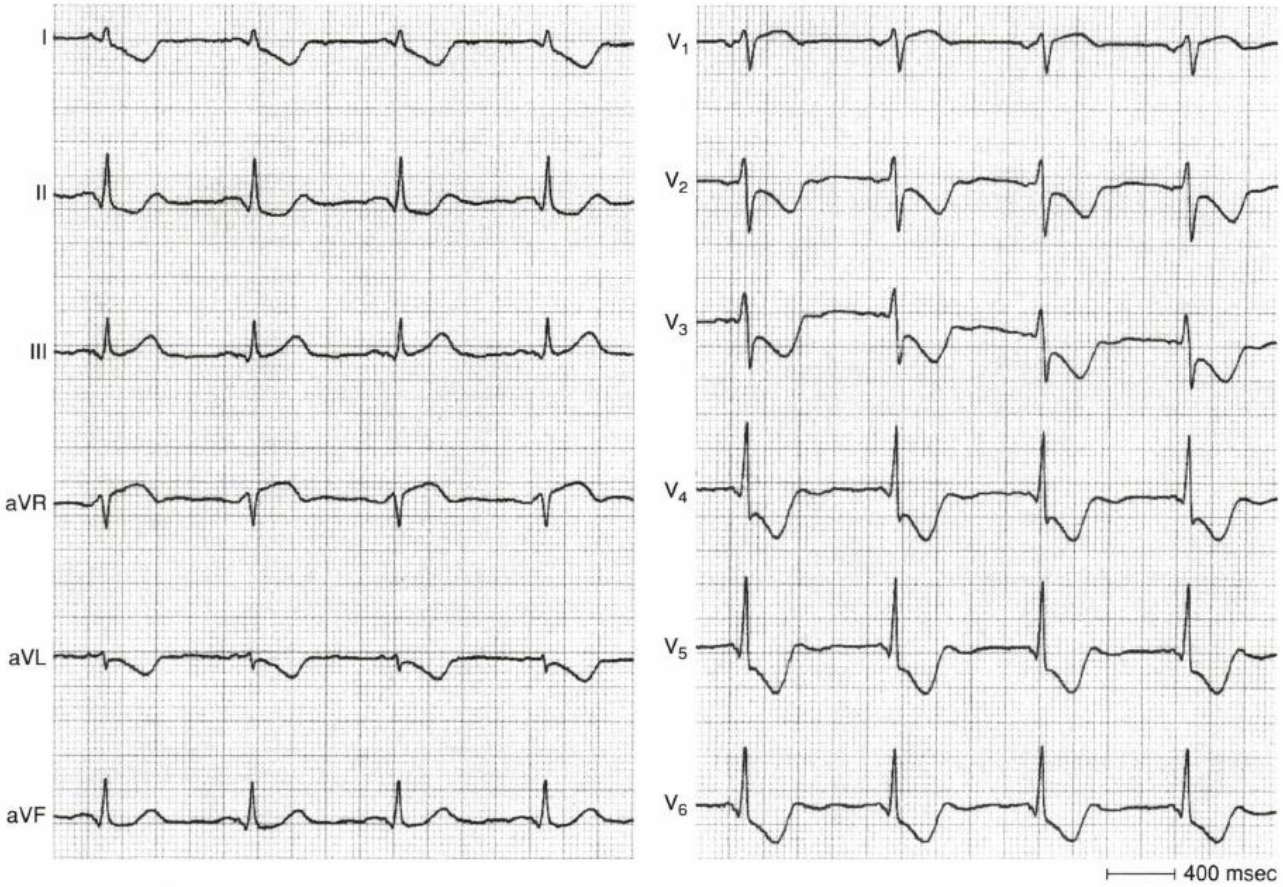
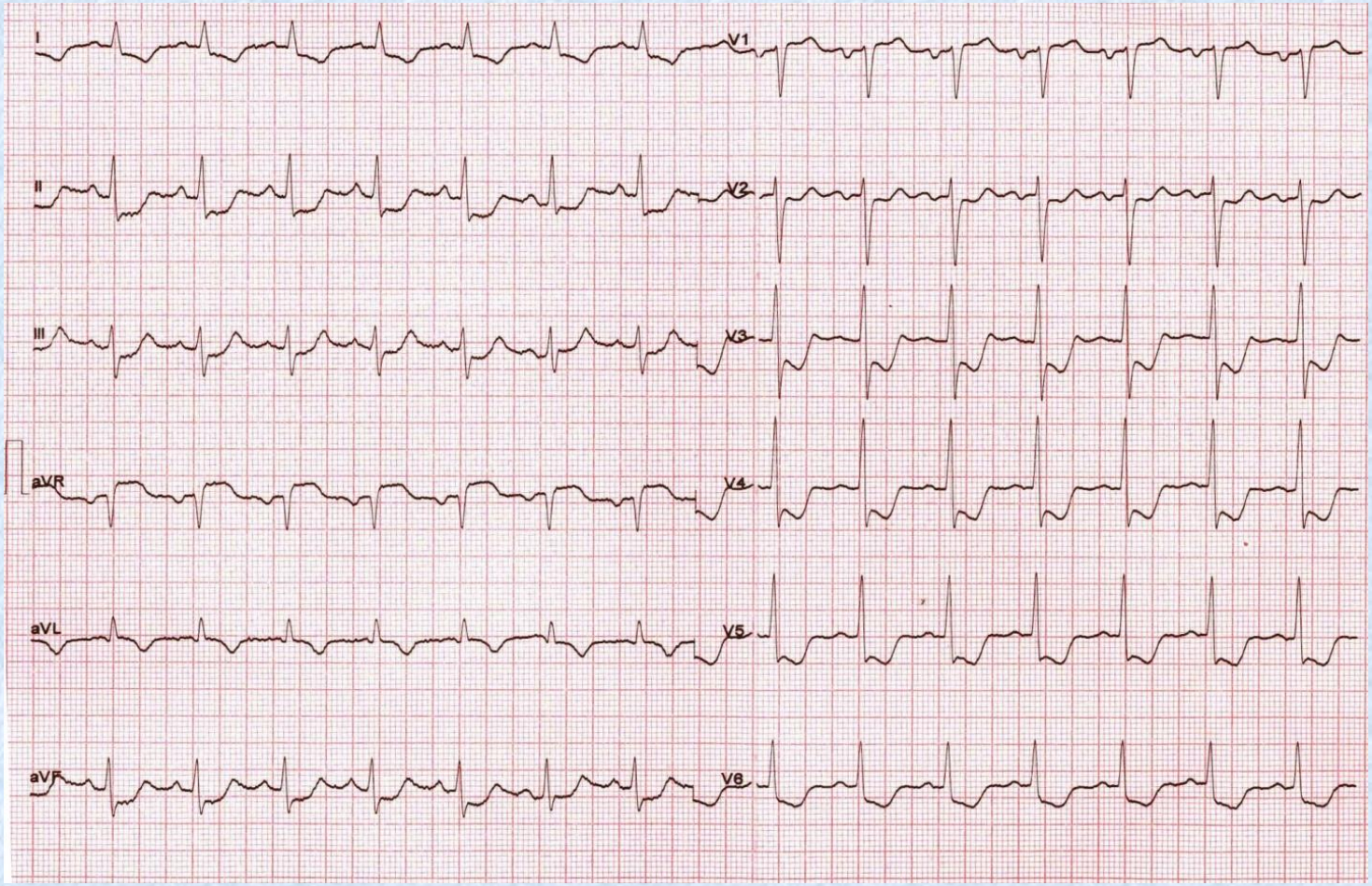
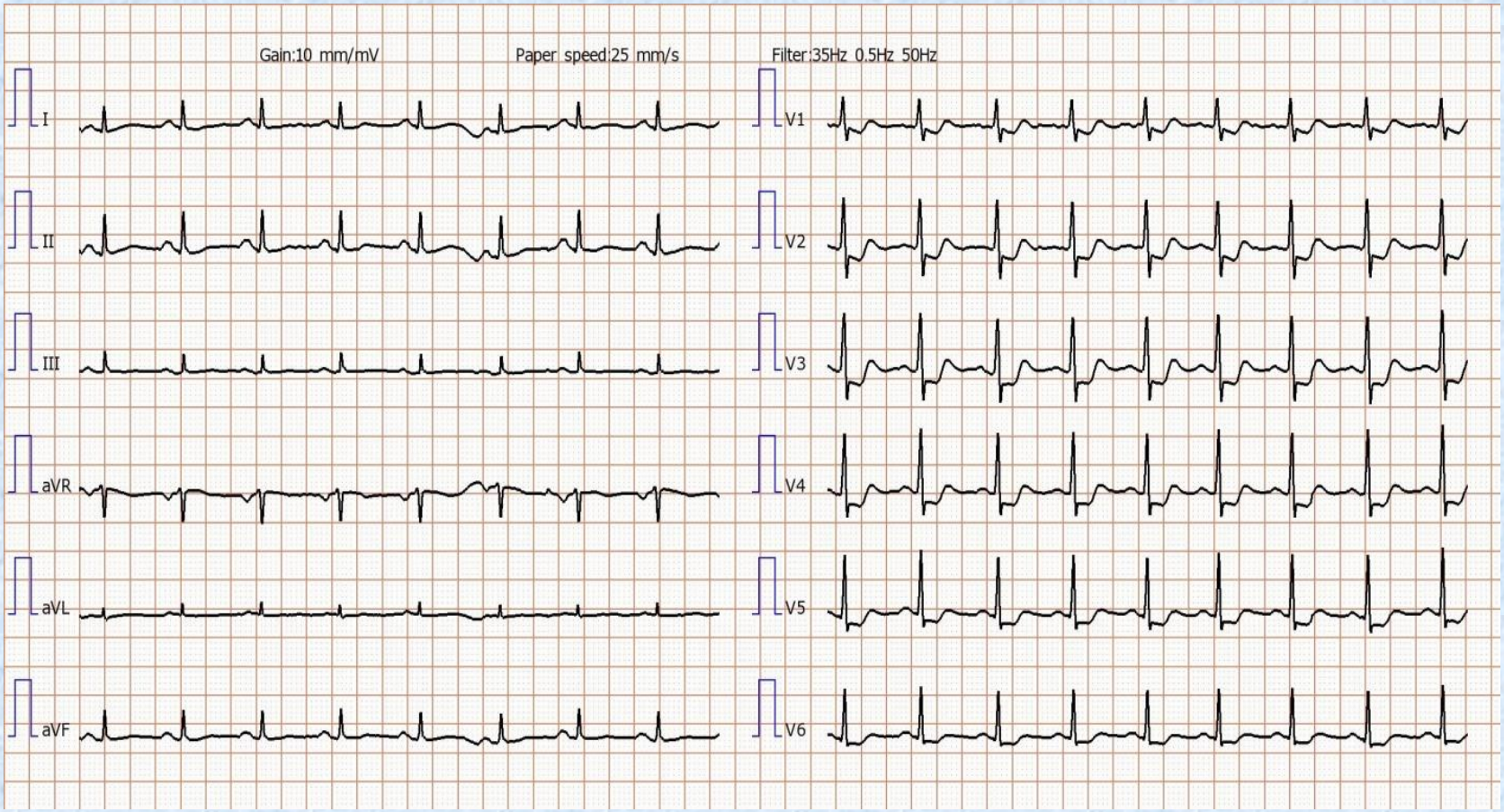


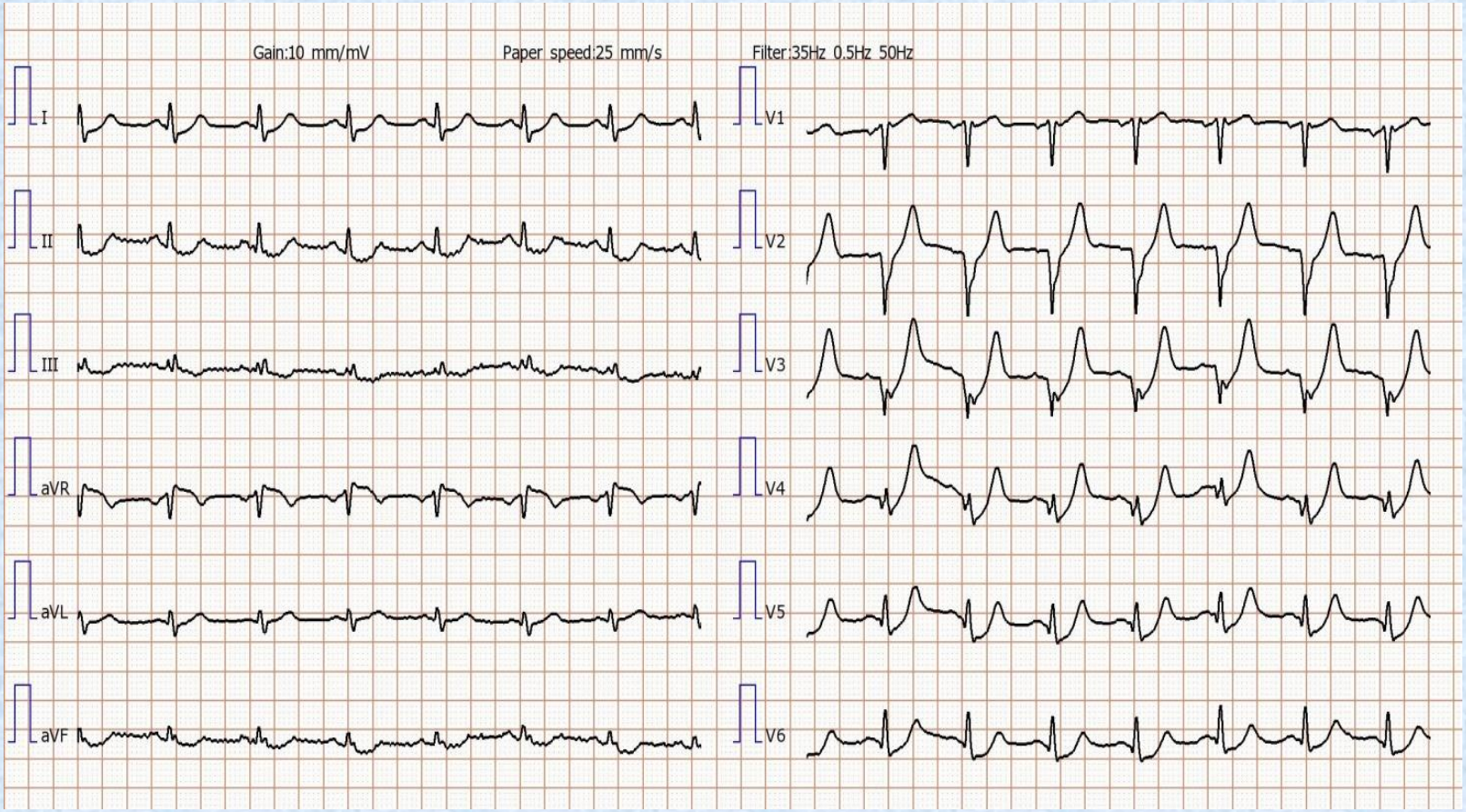
Figure 1-16 Left main occlusion. Apart from signs of an occlusion proximal to the first septal branch (ST elevation in leads aVR, aVL, and V_1), the ECG also shows evidence of posterobasal ischemia (ST depression in leads II, aVF, V_4, and V_5). Note that ST elevation in lead aVR is greater than that in V_1 and that the amount of ST depression in lead V_6 is more than the ST elevation in lead V_1.



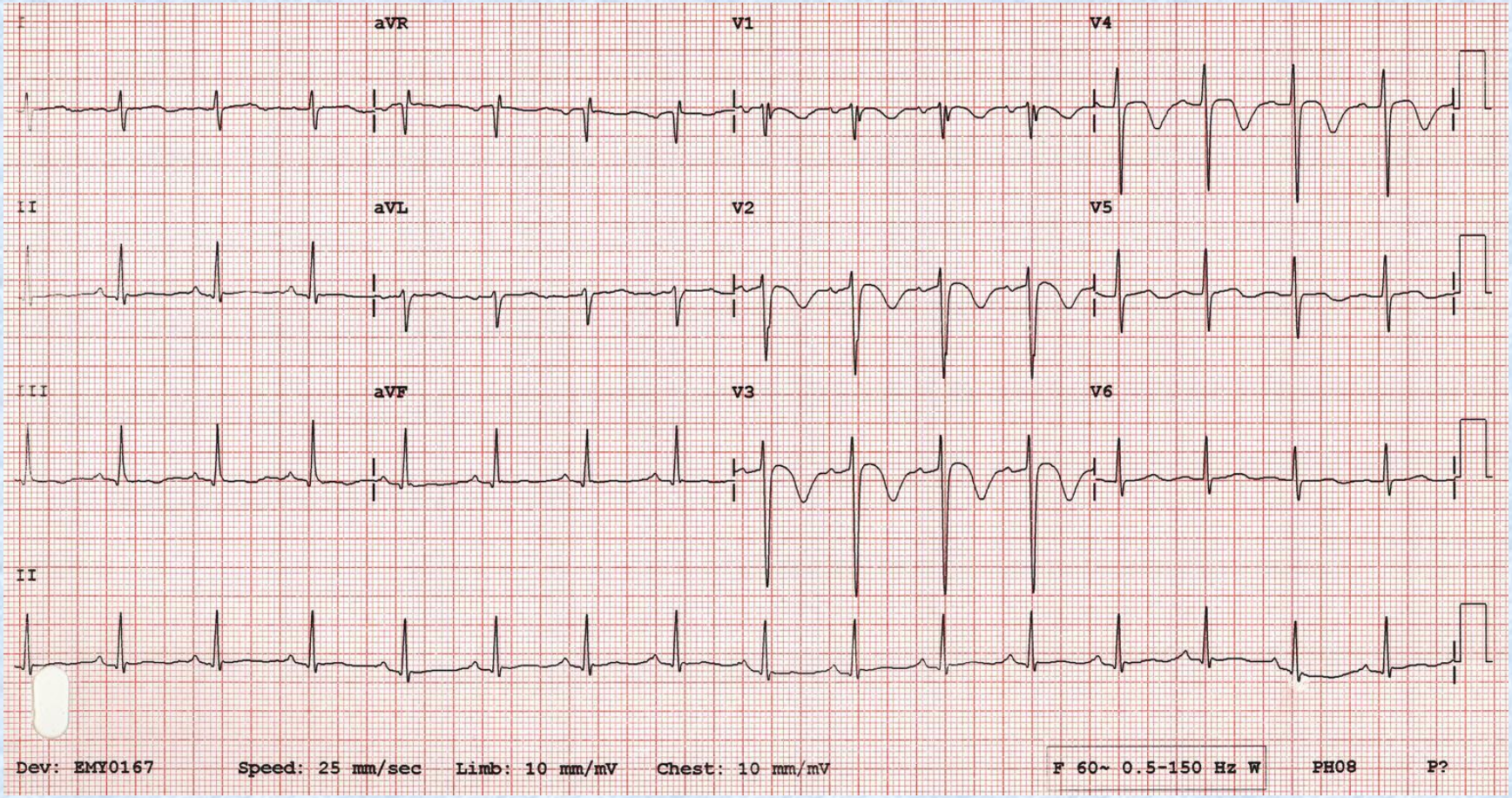
Left main occlusion pattern. Note to ST segment elevation in aVR and V1 and ST segment depression in other leads.



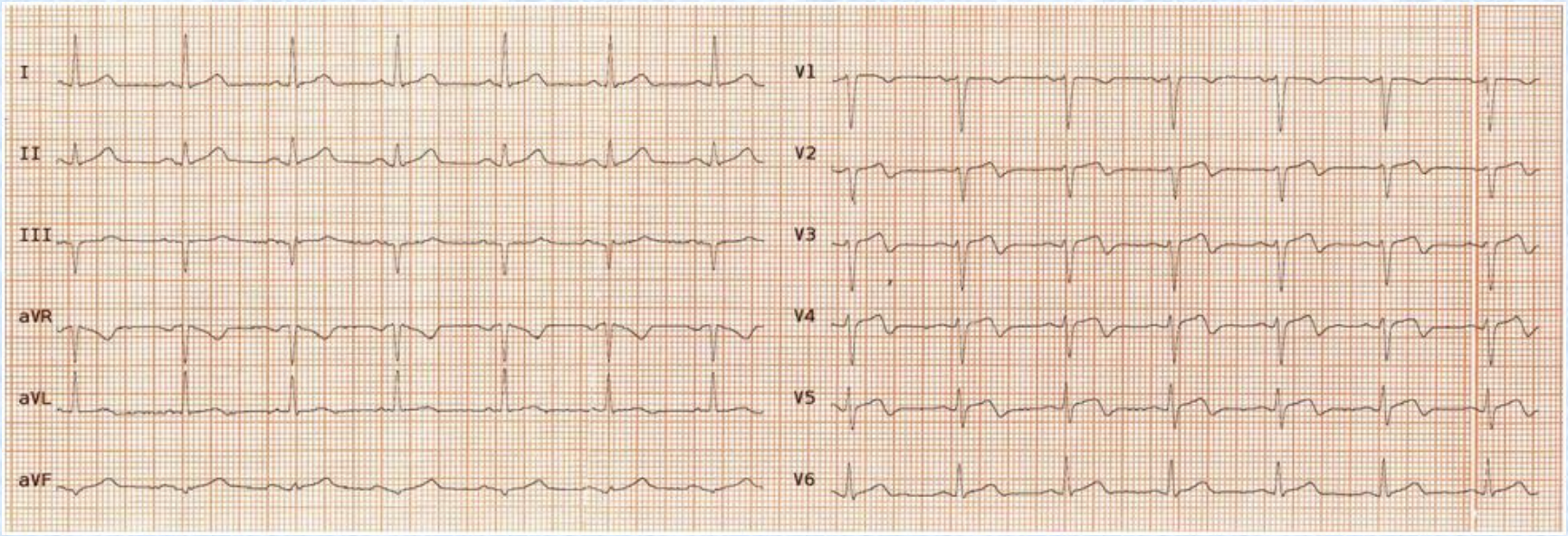
Isolated posterior STEMI. Note to horizontal ST depression in pericordial leads especially V1-4, dominant R wave (R/S ratio > 1) in V1 or V2, and upright T waves.



De Winter ECG pattern. ECG from a 50 years old male patient presented with at rest retrosternal angina. Note to broad, symmetrically peaked (hyperacute) T-waves in pericorial leads, ST segment elevation in lead aVR, and ST segment depression in other leads. The left anterior descending artery was cut at proximal portion in coronary angiography.



Wellens' Syndrome



Wellens syndrome. Note to biphasic T wave inversion in pericardial lead. This form is a more common type of wellens syndrome.

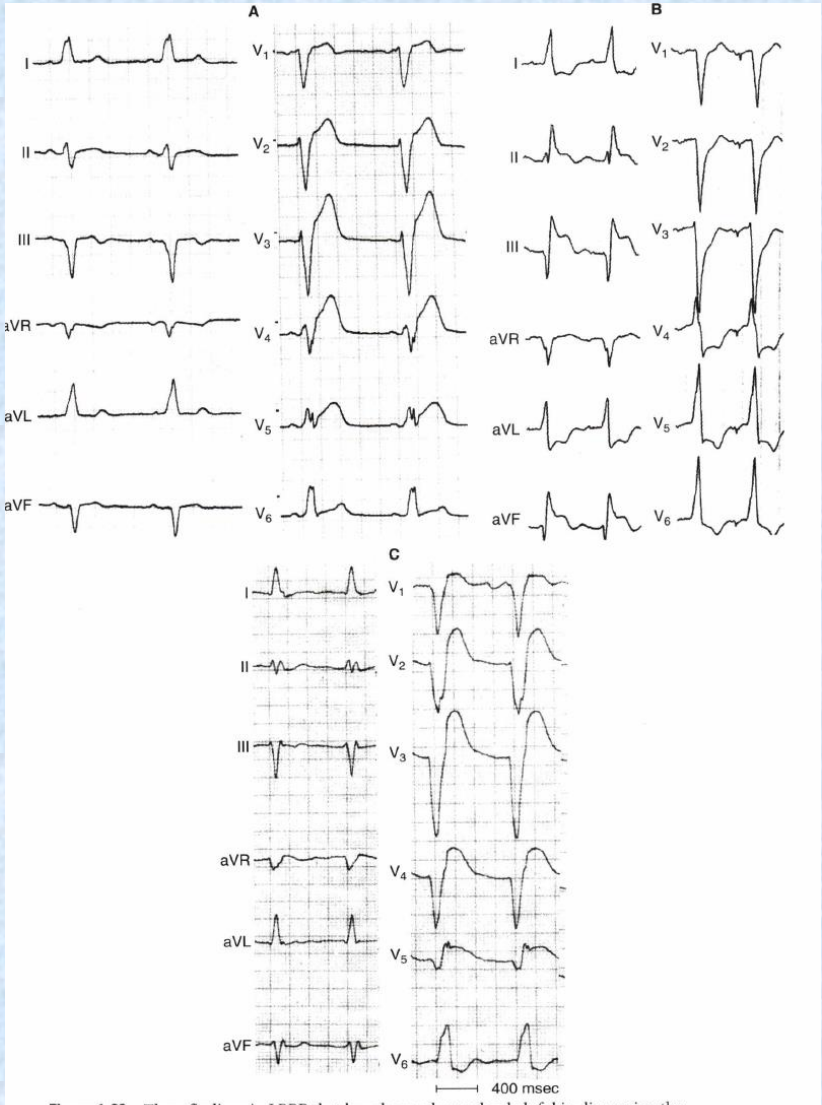
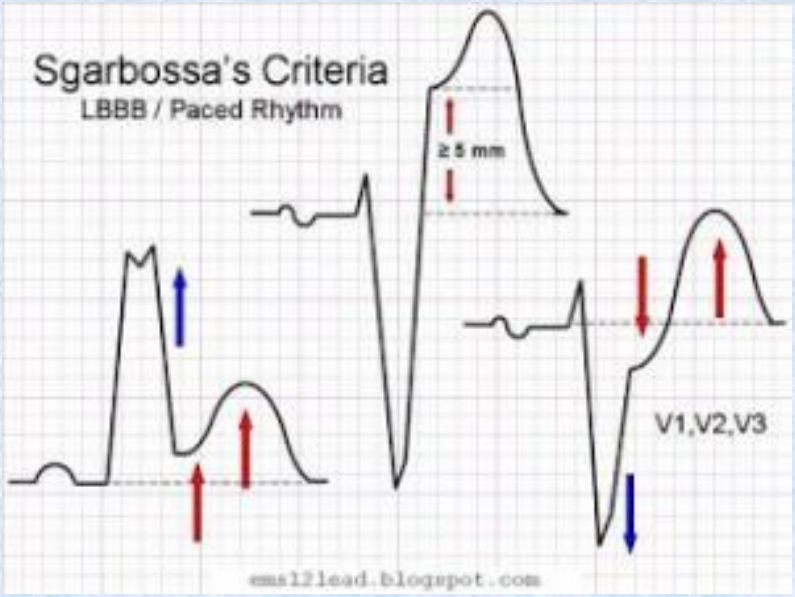
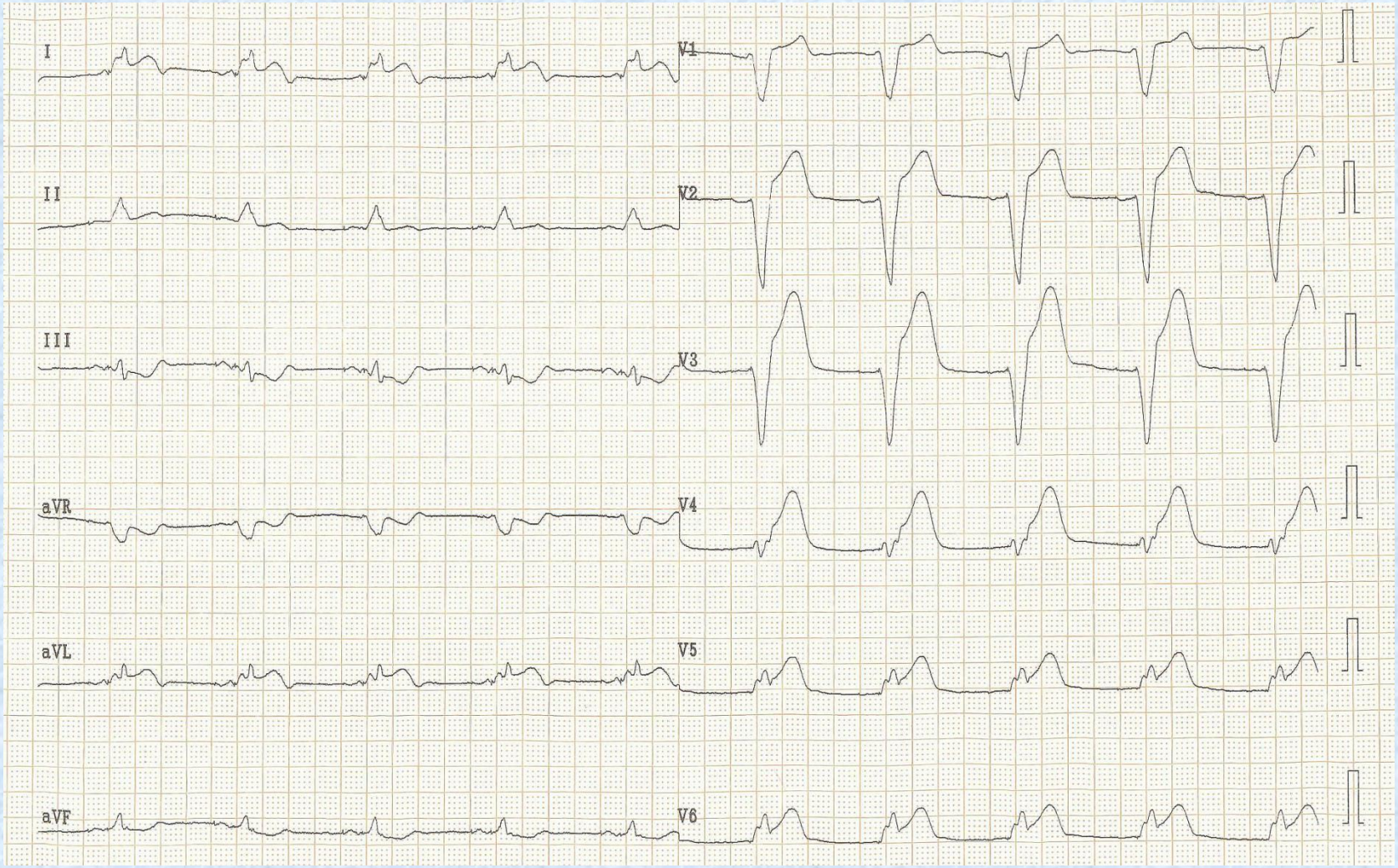


Figure 1-36 Three findings in LBBB that have been advanced as helpful in diagnosing the presence of MI. **A,** ST segment elevation more than 1 mm, concordant with the QRS complex in leads I, V_5, and V_6. **B,** ST segment depression more than 1 mm in leads V_1 to V_3. **C,** ST segment elevation more than 5 mm discordant with QRS in leads V_2 to V_4. Also see Table 1-3.




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

BOX 1-4

Reperfusion Arrhythmias

Accelerated idioventricular rhythm: Run of more than three ventricular complexes with a rate between 60 and 120 beats/min starting late in diastole

Nonsustained VT: Ventricular rhythm more than 120 beats/min lasting less than 30 seconds

Increase in VPBs: Twofold increase in the number per 5-minute interval

Ventricular fibrillation

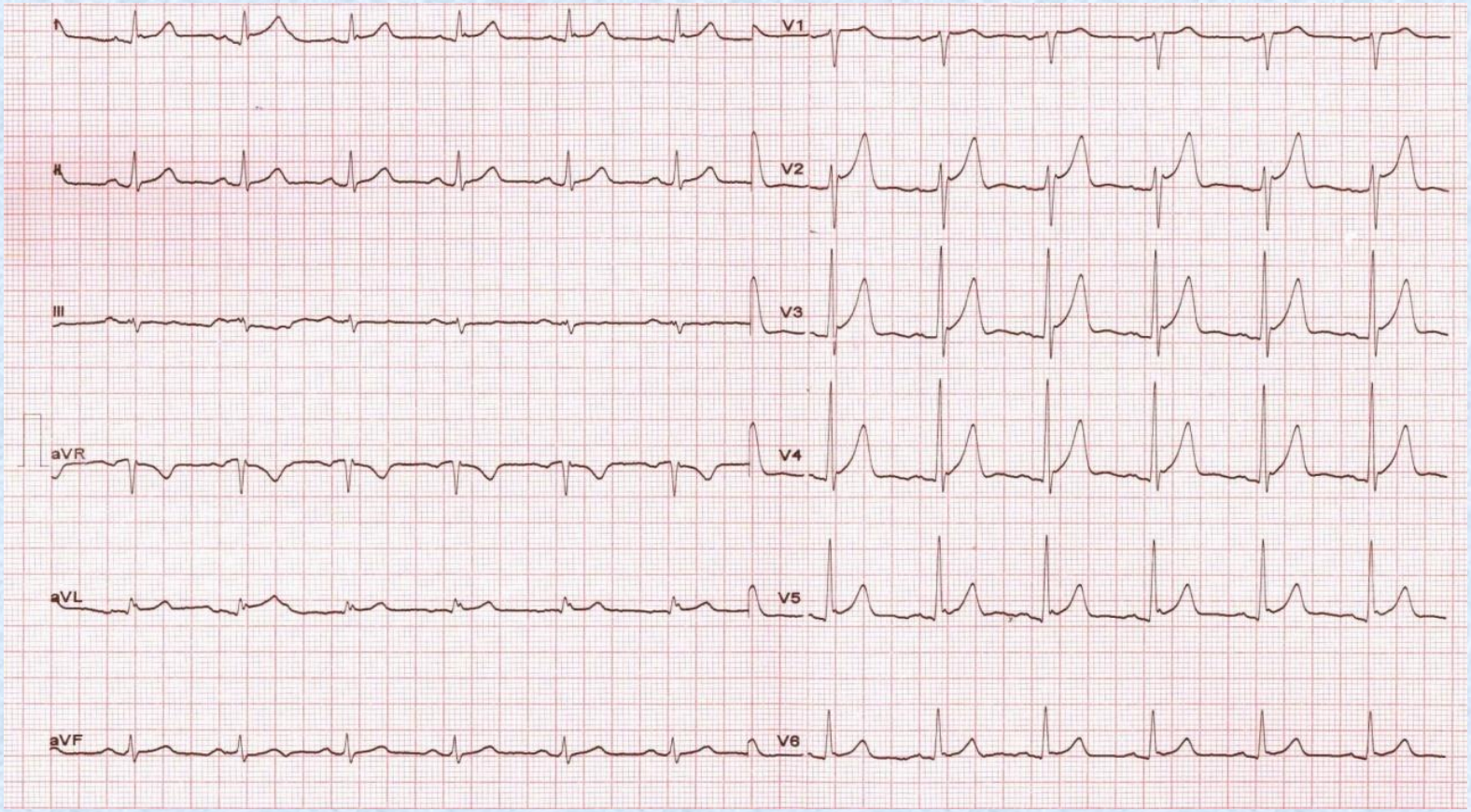
Atrial tachycardia/atrial fibrillation: Run of more than three regular or irregular rapid atrial complexes

Benign early repolarisation

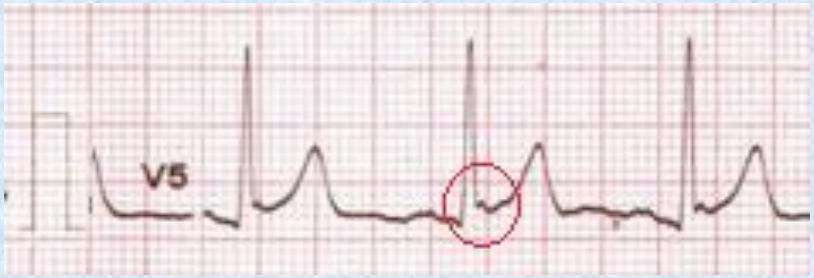
- **Benign early repolarisation**
- The ECG pattern of benign early repolarisation (BER) is most commonly seen in young, healthy patients with age of less 50 years of age.

- It produces widespread ST segment elevation that may mimic pericarditis or acute MI.

Table 7. criteria for benign early repolarisation
Widespread concave ST elevation, most prominent in the mid- to left precordial leads (V2-5).
Concavity of initial upsloping portion of ST segment
Notching or slurring at the J-point.
Prominent, slightly asymmetrical T-waves which are concordant with the QRS complexes (pointing in the same direction).
The degree of ST elevation is modest in comparison to the T-wave amplitude (less than 25% of the T wave height in V6; against pericarditis)
ST elevation compatible with criteria in table 6.
No reciprocal ST depression to suggest STEMI (except in aVR).
ST changes are relatively stable over time (no progression on serial ECG tracings).
Reduction in ST segment elevation with sympathomimetic factors



Benign early repolarization pattern in a 47 years old man with normal coronary arteries. Note to “Notching” and “slurring” of J point and concave upward ST-segment elevation in precordial, especially leadV5.



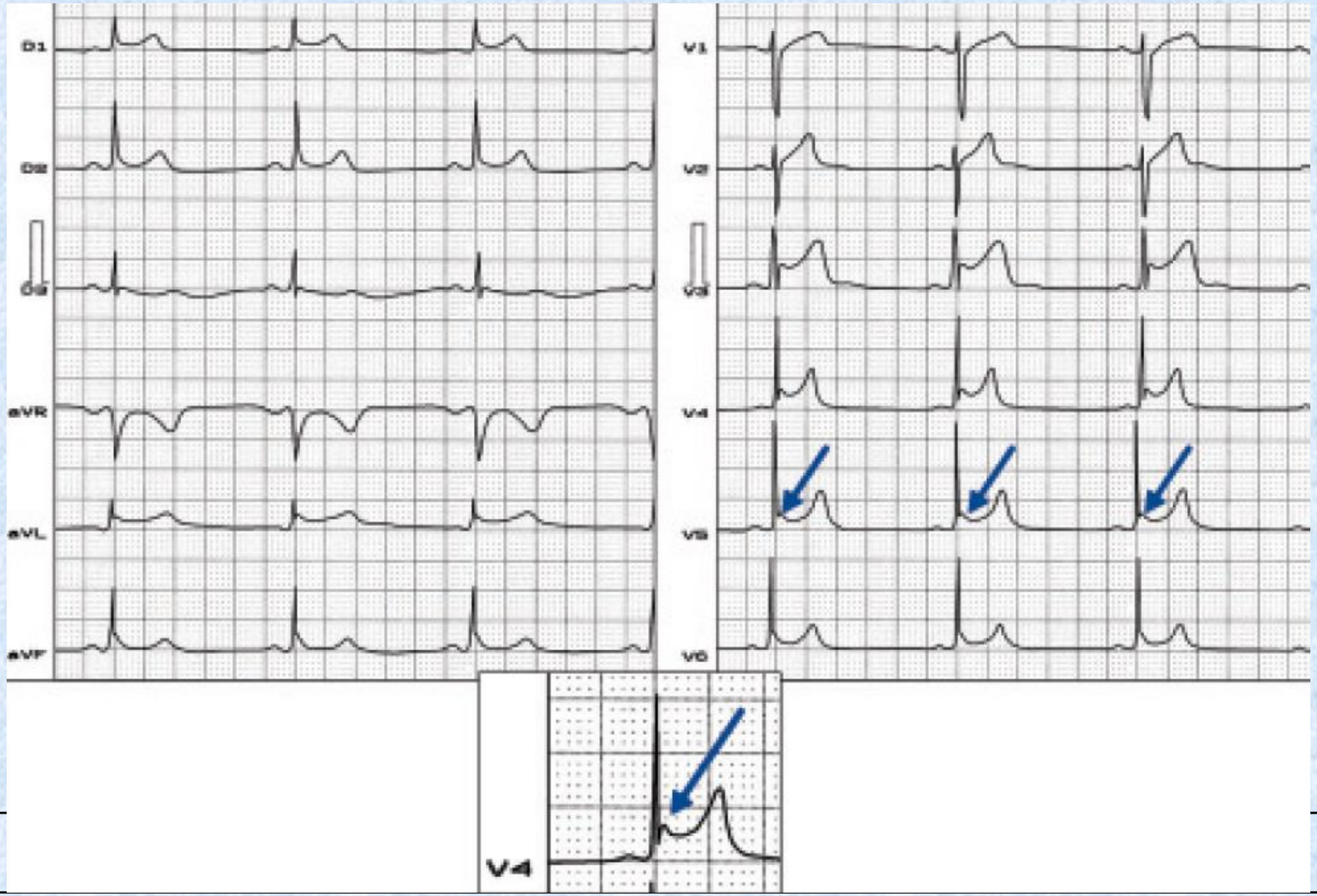
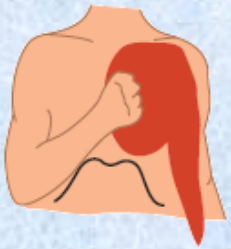


Figure 4: Benign early repolarization pattern in a male athletic with 24 years old. Note to “Notching” and “slurring” of J point and ST-segment elevation in precordial leads from V3–V5 of concave upward. (from: Pérez-Riera AR, de Abreu LC, Yanowitz F, Barros RB, Femenía F, McIntyre WF, et al. “Benign” early repolarization versus malignant early abnormalities: Clinical-electrocardiographic distinction and genetic basis. Cardiology journal. 2012;19(4):337-46.)

PREHOSPITAL MANAGEMENT



Activate EMS

Patient symptom onset of STEMI

EMS dispatch

EMS on scene
Encourage 12-lead ECGs
Consider prehospital fibrinolytic if capable and EMS-to-needle time ≤30 min

EMS triage plan

Hospital arrival

STEMI confirmed 12-lead ECG (≤10 min)

Hospital fibrinolysis: Door-to-needle time ≤30 min

Not PCI capable

Interhospital transfer

PCI capable

Goals

Patient — 5 min after symptom onset

Dispatch — 1 min

EMS on scene — ≤8 min

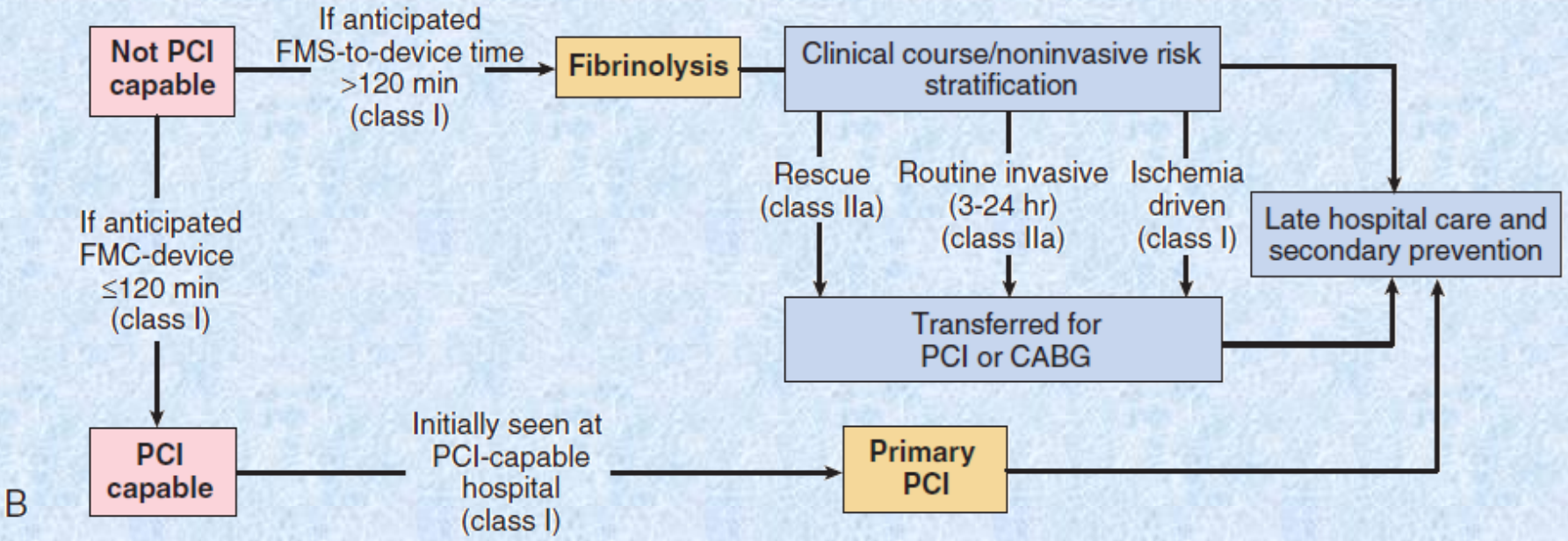
EMS transport

EMS transport: EMS-device ≤90 min

Patient self-transport: hospital door–to-device ≤90 min

Total ischemic time: <120 min

A



first medical contact (FMC)

MANAGEMENT IN THE EMERGENCY DEPARTMENT

General Treatment Measures

- ***Aspirin*** (162 to 325 mg should chew)

- ***<u>Control of Cardiac Pain</u>:***

1. **ANALGESICS** (choice: morphine 4 to 8 mg IV & 2 to 8 mg repeated at intervals of 5 to 15 minutes)

2. **NITRATES** (Exclude**:** RVMI, SBP <90 mm Hg especially if accompanied by bradycardia, or recent use of PDE-5 inhibitors)

3. **BETA-ADRENERGIC BLOCKING AGENTS** (Exclude: heart failure, SBP <90 mm Hg, HR <60 beats/min, or significant AV block).

4. **OXYGEN** (if SO_2<90%)

Contraindications to and Cautions in the Use of Fibrinolytics

Absolute Contraindications

1. Any previous intracranial hemorrhage
2. Known structural cerebral vascular lesion (e.g., AVM)
3. Known malignant intracranial neoplasm (primary or metastatic)
4. Ischemic stroke within 3 months *except acute ischemic stroke within* 4.5 hours
5. Suspected aortic dissection
6. Active bleeding or bleeding diathesis (excluding menses)
7. Significant closed-head or facial trauma within 3 months
8. Intracranial or intraspinal surgery within 2 months
9. Severe uncontrolled hypertension (unresponsive to emergency therapy)
10. For streptokinase, previous treatment within the previous 6 months

Relative Contraindications

1. History of chronic, severe, poorly controlled hypertension
2. Significant hypertension at initial evaluation (SBP > 180 mm Hg or DBP > 110 mm Hg)† (Could be an absolute contraindication in low-risk patients with MI)
3. History of previous ischemic stroke >3 months
4. Dementia
5. Known intracranial pathology not covered in Absolute Contraindications
6. Traumatic or prolonged (>10 minutes) cardiopulmonary resuscitation
7. Major surgery (<3 weeks)
8. Recent (within 2 to 4 weeks) internal bleeding
9. Noncompressible vascular punctures
10. Pregnancy
11. Active peptic ulcer
12. Oral anticoagulant therapy

REPERFUSION THERAPY

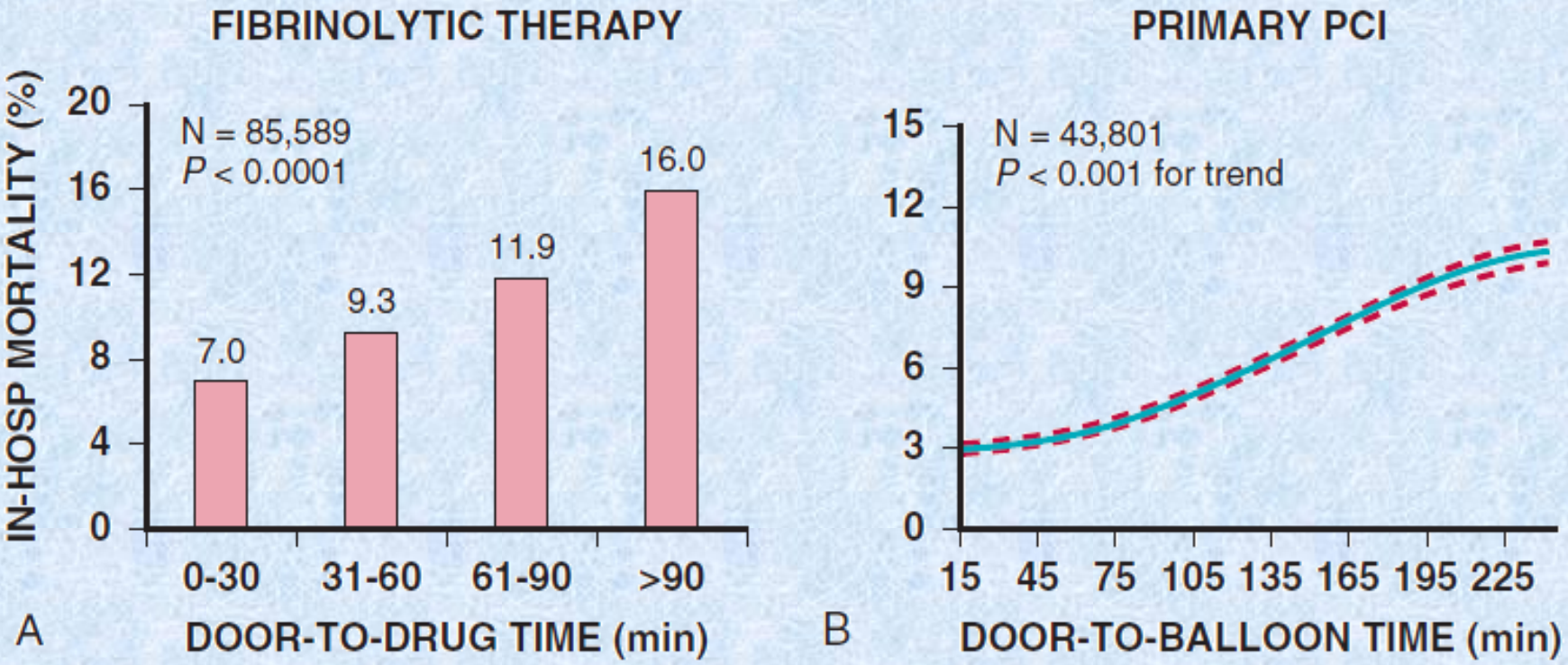

FIBRINOLYTIC THERAPY
IN-HOSP MORTALITY (%)
20
16
12
8
4
0
N = 85,589
P < 0.0001
7.0
9.3
11.9
16.0
0-30
31-60
61-90
>90
DOOR-TO-DRUG TIME (min)
A
PRIMARY PCI
15
12
9
6
3
0
N = 43,801
P < 0.001 for trend
15 45 75 105 135 165 195 225
DOOR-TO-BALLOON TIME (min)
B

TABLE 52-5 Comparison of Approved Fibrinolytic Agents

FIBRINOLYTIC AGENT	DOSE	FIBRIN SPECIFICITY	FIBRINOGEN DEPLETION	ANTIGENIC	PATENCY RATE (90-MIN TIMI 2 OR 3 FLOW)
Fibrin Specific					
Tenecteplase (TNK)	Single IV weight-based bolus[†]	++++	Minimal	No	85%
Reteplase (r-PA)	10 units + 10-unit IV boluses given 30 min apart	++	Moderate	No	84%
Alteplase (t-PA)	90-min weight-based infusion[‡]	++	Mild	No	73-84%
Non–Fibrin Specific					
Streptokinase[§]	1.5 million units IV given over 30-60 min	No	Marked	Yes[¶]	60-68%

TNK: Bolus of 30 mg for weight less than 60 kg, 35 mg for 60 to 69 kg, 40 mg for 70 to 79 kg, 45 mg for 80 to 89 kg, and 50 mg for 90 kg or greater.

T-PA: Bolus of 15 mg, infusion of 0.75 mg/kg for 30 minutes (maximum, 50 mg), then 0.5 mg/kg (maximum, 35 mg) over the next 60 minutes; the total dose not to exceed 100 mg.

TABLE 52-6 Indications for Coronary Angiography in Patients Who Were Managed with Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

RECOMMENDATION	COR	LOE
Cardiogenic shock or acute severe HF that develops after initial evaluation	I	B
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	B
Spontaneous or easily provoked myocardial ischemia	I	C
Failed reperfusion or reocclusion after fibrinolytic therapy	IIa	B
Stable* patients after successful fibrinolysis—before discharge and ideally between 3 and 24 hr	IIa	B

ACCF/AHA Guidelines

Time: Primary PCI

TABLE 52G-1 Primary Percutaneous Coronary Intervention for ST-Elevation Myocardial Infarction

	COR	LEVEL OF EVIDENCE
Ischemic symptoms <12 hr	I	A
Ischemic symptoms <12 hr and contraindications to fibrinolytic therapy irrespective of delay in time after FMC	I	B
Cardiogenic shock or acute severe HF irrespective of delay in time after the onset of MI	I	B
Evidence of ongoing ischemia 12-24 hr after the onset of symptoms	IIa	B
PCI on a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise	III: Harm	B

Drugs: Primary PCI

TABLE 52G-2 Adjunctive Antithrombotic Therapy to Support Reperfusion with Primary Percutaneous Coronary Intervention

	COR	LEVEL OF EVIDENCE
Antiplatelet Therapy		
Aspirin		
• 162- to 325-mg loading dose before the procedure	I	B
• 81- to 325-mg daily maintenance dose (indefinite)*	I	A
• 81 mg daily is the preferred maintenance dose*	IIa	B
$P2Y_{12}$ Inhibitors		
Loading Doses		
• Clopidogrel: 600 mg as early as possible or at the time of PCI	I	B
• Prasugrel: 60 mg as early as possible or at the time of PCI	I	B
• Ticagrelor: 180 mg as early as possible or at the time of PCI	I	B
Maintenance Doses and Duration of Therapy		
DES placed: Continue therapy for 1 year with		
• Clopidogrel: 75 mg daily	I	B
• Prasugrel: 10 mg daily	I	B
• Ticagrelor: 90 mg twice a day*	I	B
BMS† placed: Continue therapy for 1 year with		
• Clopidogrel: 75 mg daily	I	B
• Prasugrel: 10 mg daily	I	B
• Ticagrelor: 90 mg twice a day*	I	B
DES placed:		
• Clopidogrel, prasugrel, or ticagrelor* continued beyond 1 year	IIb	C
• Patients with STEMI and previous stroke or TIA: prasugrel	III: Harm	B
Intravenous Glycoprotein IIb/IIIa Receptor Antagonists in Conjunction with Unfractionated Heparin or Bivalirudin in Selected Patients		
• Abciximab: 0.25-mg/kg IV bolus, then 0.125 µg/kg/min (maximum, 10 µg/min)	IIa	A
• Tirofiban (high bolus dose): 25-µg/kg IV bolus, then 0.15 µg/kg/min	IIa	B
• In patients with CrCl <30 mL/min, reduce the infusion by 50%		
• Eptifibatide (double bolus): 180-µg/kg IV bolus, then 2 µg/kg/min; a second 180-µg/kg bolus is administered 10 min after the first bolus	IIa	B
• In patients with CrCl < 50 mL/min, reduce the infusion by 50%		
• Avoid in patients on hemodialysis		
• Pre–catheterization laboratory administration of IV GP IIb/IIIa receptor antagonist	IIb	B
• Intracoronary abciximab: 0.25-mg/kg bolus	IIb	B

Drugs: Primary PCI

TABLE 52G-2 Adjunctive Antithrombotic Therapy to Support Reperfusion with Primary Percutaneous Coronary Intervention—cont'd

	COR	LEVEL OF EVIDENCE
Anticoagulant Therapy		
• UFH		
• With a GP IIb/IIIa receptor antagonist planned: 50- to 70-unit/kg IV bolus to achieve therapeutic ACT‡	I	C
• With no GP IIb/IIIa receptor antagonist planned: 70- to 100-unit/kg bolus to achieve a therapeutic ACT§	I	C
• Bivalirudin: 0.75-mg/kg IV bolus, then 1.75-mg/kg/hr infusion with or without previous treatment with UFH. An additional bolus of 0.3 mg/kg may be given if needed	I	B
• Reduce the infusion to 1 mg/kg/hr with estimated an CrCl <30 mL/min		
• Preferred over UFH with a GP IIb/IIIa receptor antagonist in patients at high risk for bleeding	IIa	B
• Fondaparinux: not recommended as the sole anticoagulant for primary PCI	III: Harm	B

Drugs: Fibrinolytic therapy

TABLE 52G-5 Adjunctive Antithrombotic Therapy to Support Reperfusion with Fibrinolytic Therapy

	COR	LEVEL OF EVIDENCE
Antiplatelet Therapy		
Aspirin		
• 162- to 325-mg loading dose	I	A
• 81- to 325-mg daily maintenance dose (indefinite)	I	A
• 81 mg daily is the preferred maintenance dose	IIa	B
$P2Y_{12}$ Receptor Inhibitors		
• Clopidogrel:	I	A
• Age ≤ 75 yr: 300-mg loading dose		
• Followed by 75 mg daily for at least 14 days and up to 1 yr in the absence of bleeding	I	A (14 days) C (up to 1 yr)
• Age > 75 yr: no loading dose, give 75 mg	I	A
• Followed by 75 mg daily for at least 14 days and up to 1 yr in the absence of bleeding	I	A (14 days) C (up to 1 yr)

Drugs: Fibrinolytic therapy

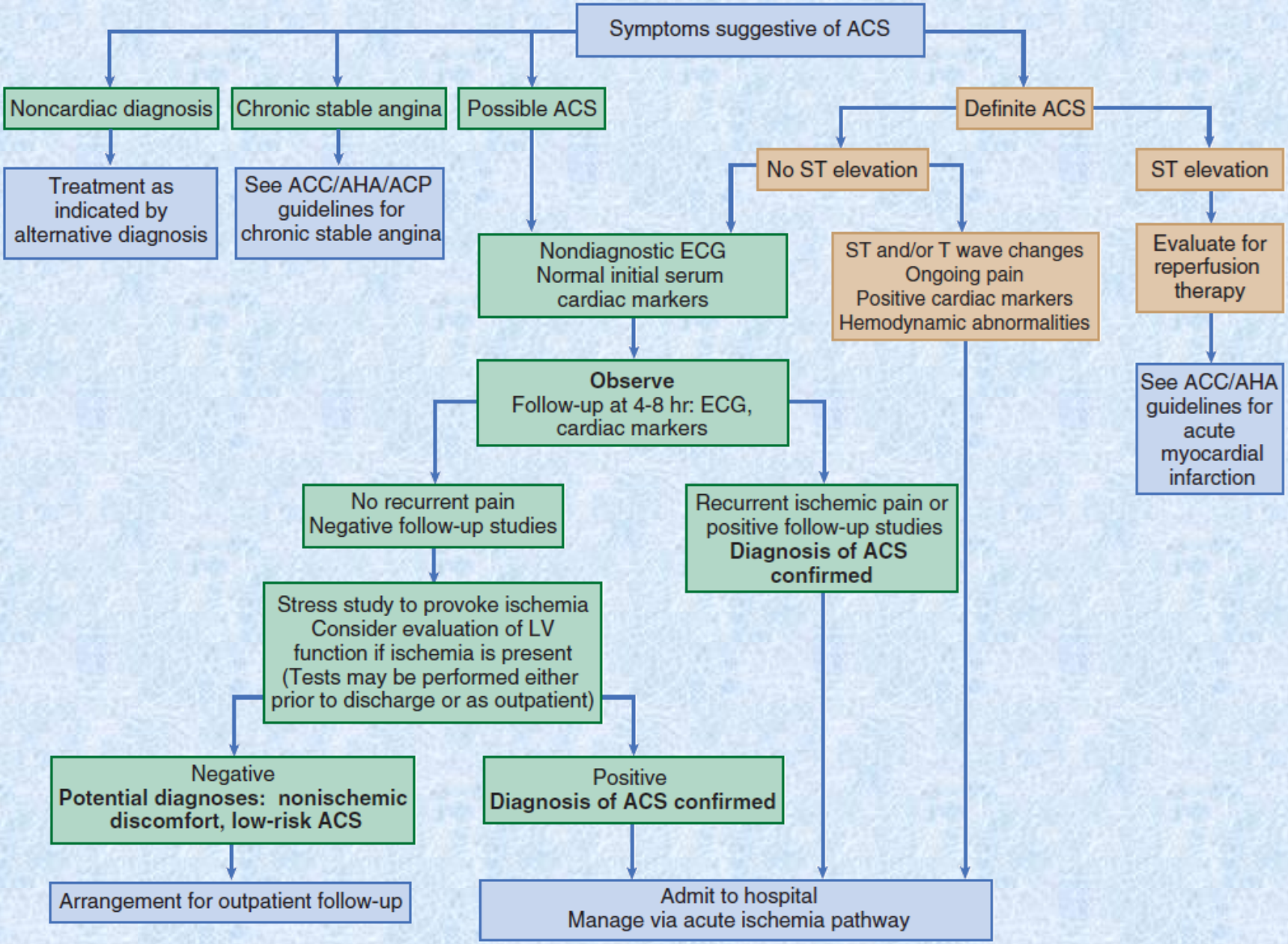
TABLE 52G-5 Adjunctive Antithrombotic Therapy to Support Reperfusion with Fibrinolytic Therapy—cont'd

	COR	LEVEL OF EVIDENCE
Anticoagulant Therapy		
• UFH:	I	C
• Weight-based IV bolus and infusion adjusted to obtain an APTT of 1.5-2.0 times control for 48 hr or until revascularization. IV bolus of 60 units/kg (maximum, 4000 units) followed by an infusion of 12 units/kg/hr (maximum, 1000 units) initially, adjusted to maintain the APTT at 1.5-2.0 times control (≈50-70 sec) for 48 hr or until revascularization		
• Enoxaparin:	I	A
• If age < 75 yr: 30-mg IV bolus, followed in 15 min by 1 mg/kg subcutaneously every 12 hr (maximum, 100 mg for the first 2 doses)		
• If age ≥ 75 yr: no bolus, 0.75 mg/kg subcutaneously every 12 hr (maximum, 75 mg for the first 2 doses)		
• Regardless of age, if CrCl < 30 mL/min, 1 mg/kg subcutaneously every 24 hr		
• Duration: For the index hospitalization, up to 8 days or until revascularization		
• Fondaparinux:	I	B
• Initial dose of 2.5 mg IV, then 2.5 mg subcutaneously daily starting the following day, for the index hospitalization up to 8 days or until revascularization		
• Contraindicated if CrCl < 30 mL/min		

Drugs: Medical therapy

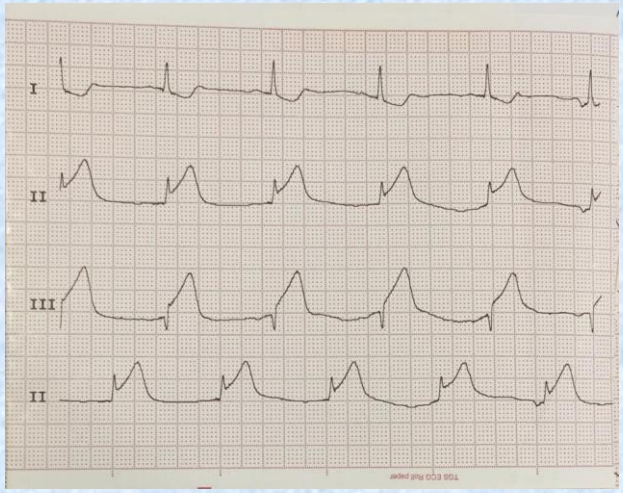
TABLE 52G-9 Indications and Cautions for Adjunctive Medical Therapies for Patients with ST-Elevation Myocardial Infarction

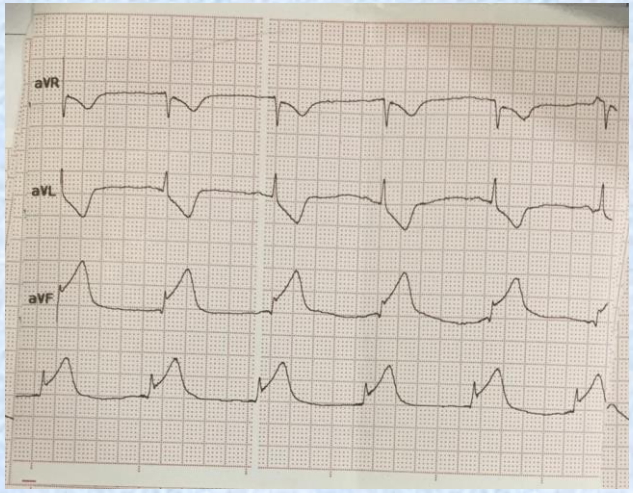
Beta-adrenergic receptor–blocking agents	Oral: All patients without contraindication IV: Patients with refractory hypertension or ongoing ischemia without contraindication	Signs of CHF Low-output state Increased risk for cardiogenic shock Prolonged first-degree or high-grade atrioventricular block Reactive airways disease
Angiotensin-converting enzyme (ACE) inhibitors	Anterior MI and EF ≤0.40 or CHF All patients without contraindication	Hypotension Renal failure Hyperkalemia
Angiotensin receptor–blocking agents (ARBs)	Intolerant of ACE inhibitors	Hypotension Renal failure Hyperkalemia
Statins	All patients without contraindications	With drugs metabolized via CYP3A4, fibrates Monitor for myopathy, hepatotoxicity Adjust dose for lipid targets
Nitroglycerin	Ongoing chest pain Hypertension and CHF	Suspected right ventricular infarction SBP <90 (or 30 mm Hg below baseline) Recent use of a type 5 PDE inhibitor
Oxygen	Clinically significant hypoxemia (Spo_2 < 90) CHF Dyspnea	Chronic obstructive pulmonary disease and CO_2 retention
Morphine	Pain Anxiety Pulmonary edema	Lethargic or moribund patient Hypotension Bradycardia Known hypersensitivity


Symptoms suggestive of ACS
Noncardiac diagnosis
Chronic stable angina
Possible ACS
Definite ACS
Treatment as indicated by alternative diagnosis
See ACC/AHA/ACP guidelines for chronic stable angina
No ST elevation
ST elevation
Nondiagnostic ECG
Normal initial serum cardiac markers
ST and/or T wave changes
Ongoing pain
Positive cardiac markers
Hemodynamic abnormalities
Evaluate for reperfusion therapy
Observe
Follow-up at 4-8 hr: ECG, cardiac markers
See ACC/AHA guidelines for acute myocardial infarction
No recurrent pain
Negative follow-up studies
Recurrent ischemic pain or positive follow-up studies
Diagnosis of ACS confirmed
Stress study to provoke ischemia
Consider evaluation of LV function if ischemia is present
(Tests may be performed either prior to discharge or as outpatient)
Negative
Potential diagnoses: nonischemic discomfort, low-risk ACS
Positive
Diagnosis of ACS confirmed
Arrangement for outpatient follow-up
Admit to hospital
Manage via acute ischemia pathway

Case Presentation

- A 50 years old man presented with Inf STEMI
- CAD RF: Smoking, DLP
- Echo: LVEF: 35%, inf HK


I
II
III
II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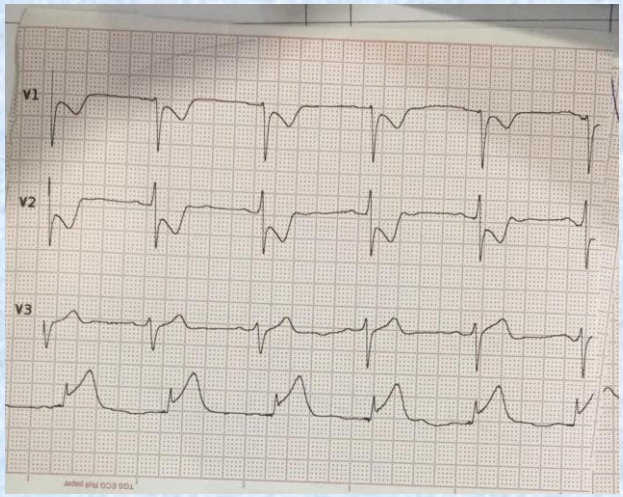
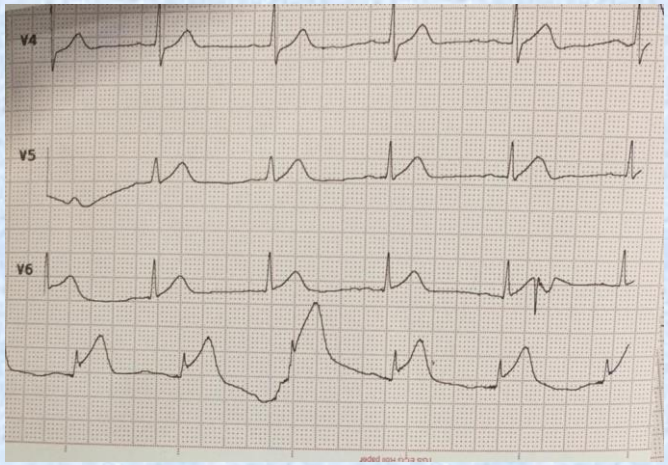
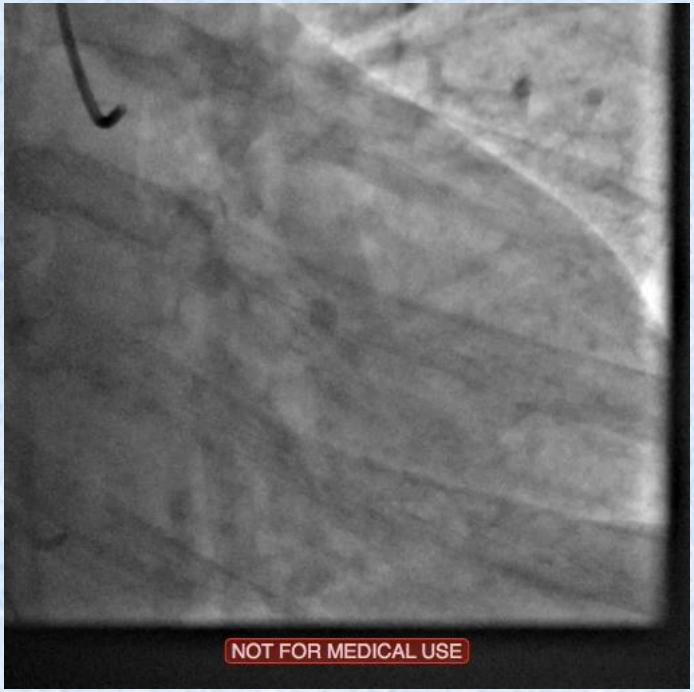
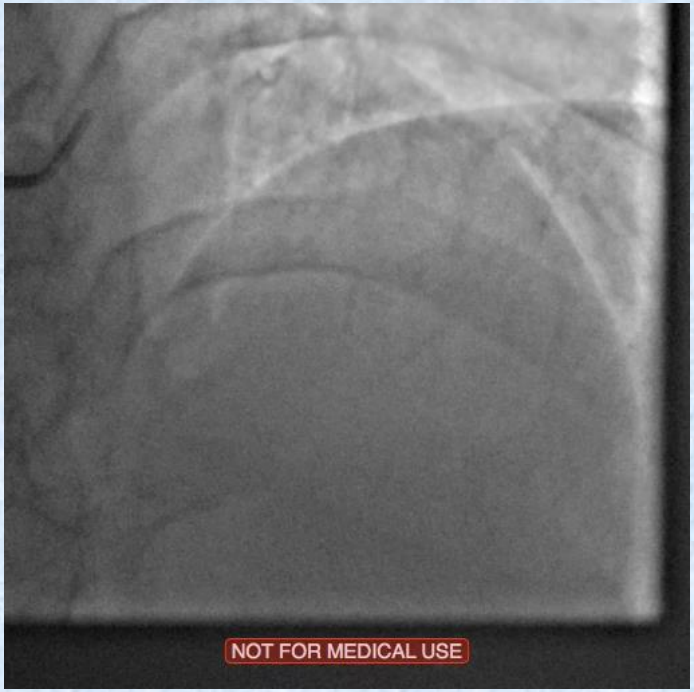
aVL
aVF


V1
V2
V3


V4
V5
V6


NOT FOR MEDICAL USE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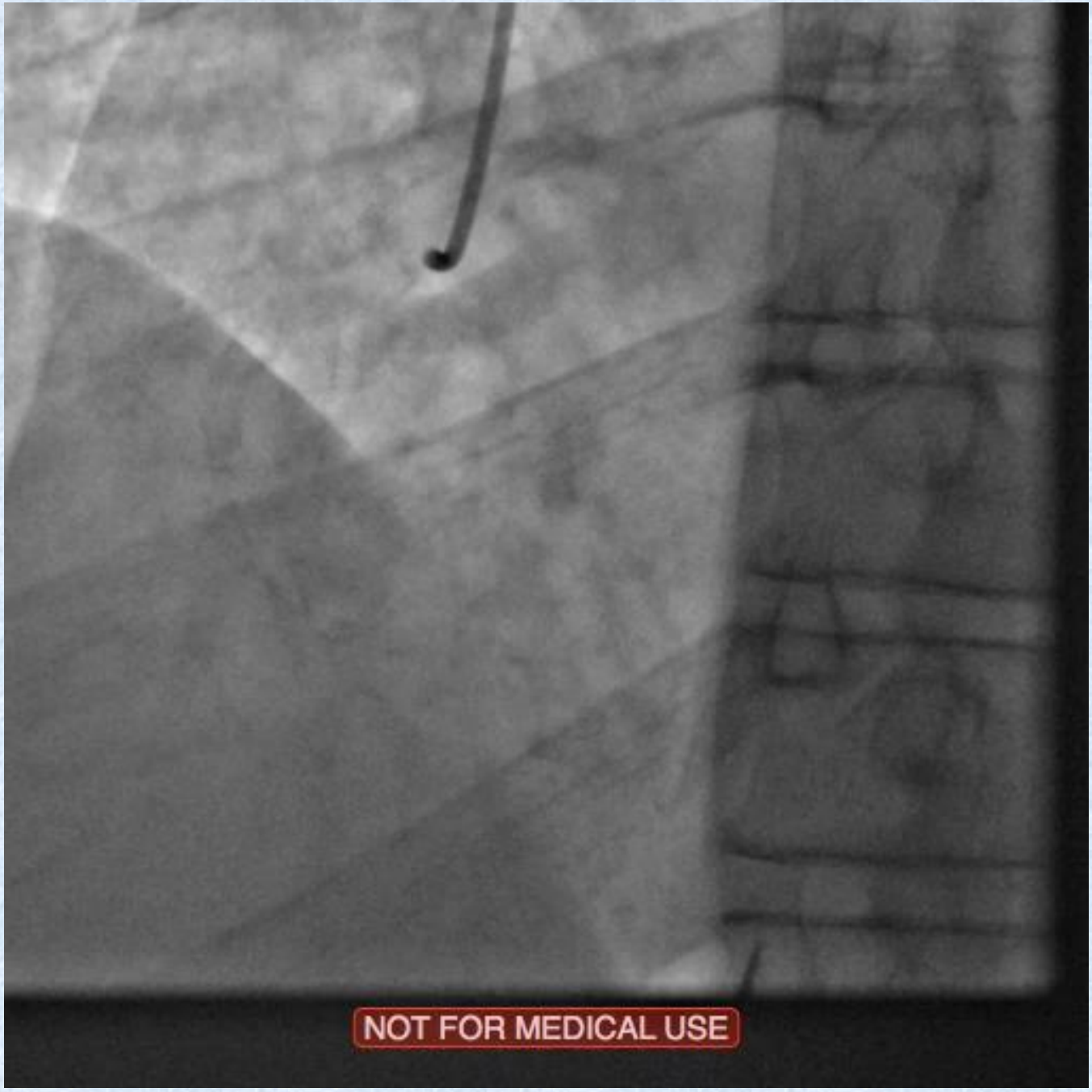
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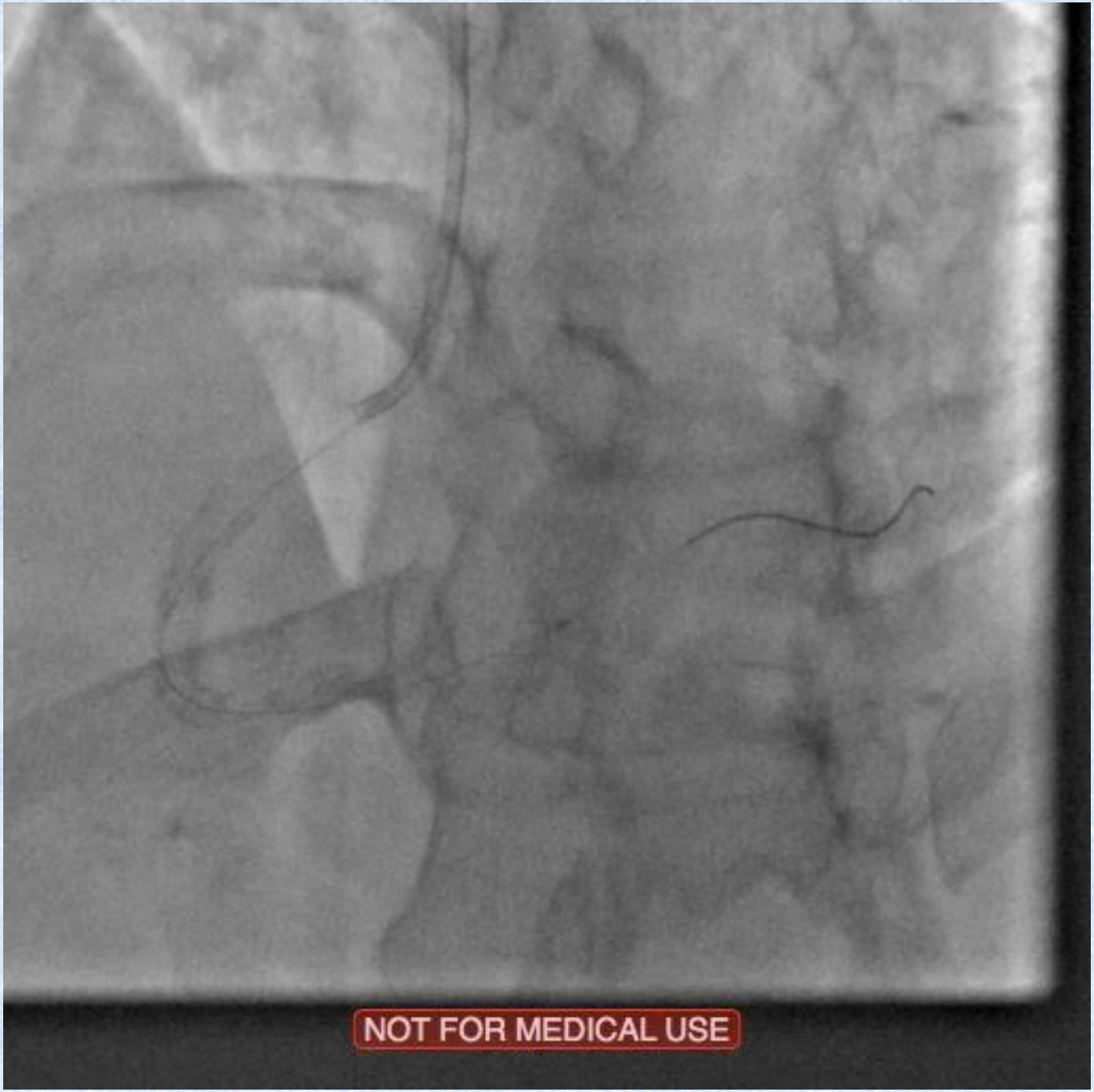
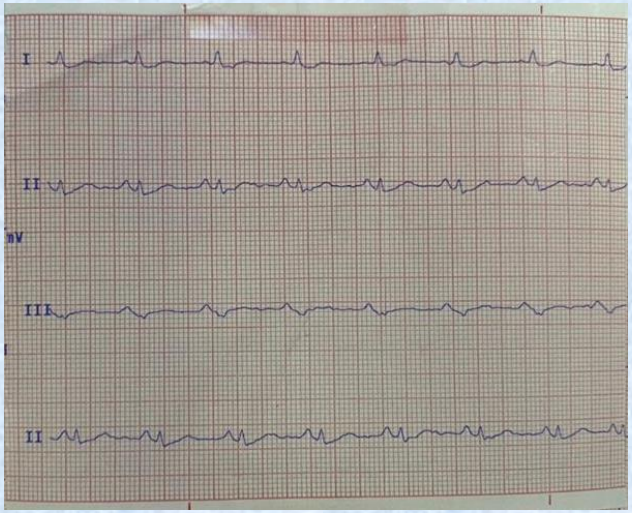
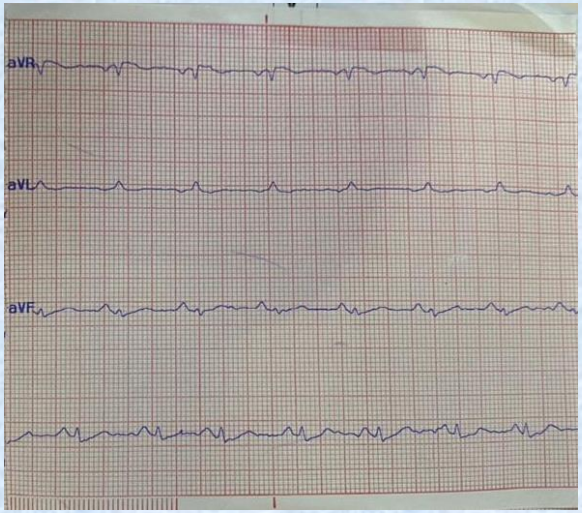

NOT FOR MEDICAL USE

Case Presentation

- A 66 years old man presented with ACS
- CAD RF: Smoking, DLP
- Echo: LVEF: 30%, inf HK


I
II
III
II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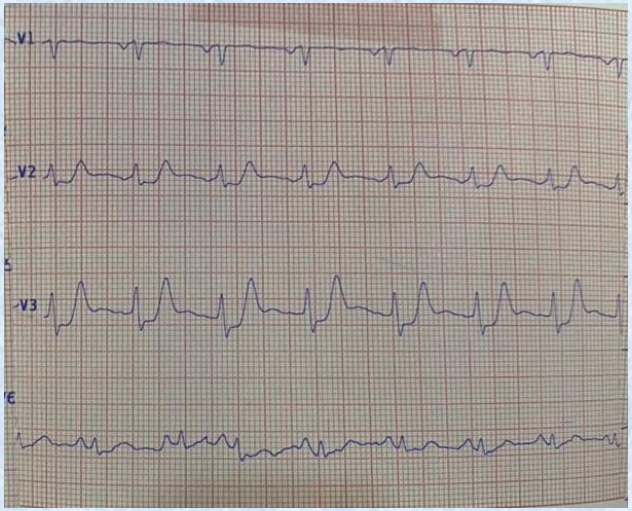
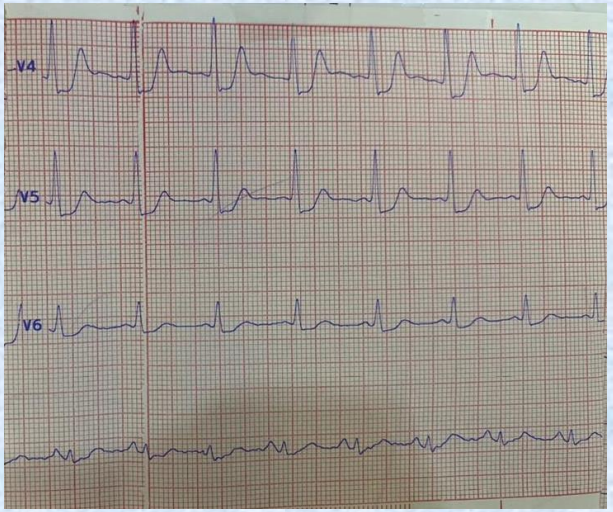
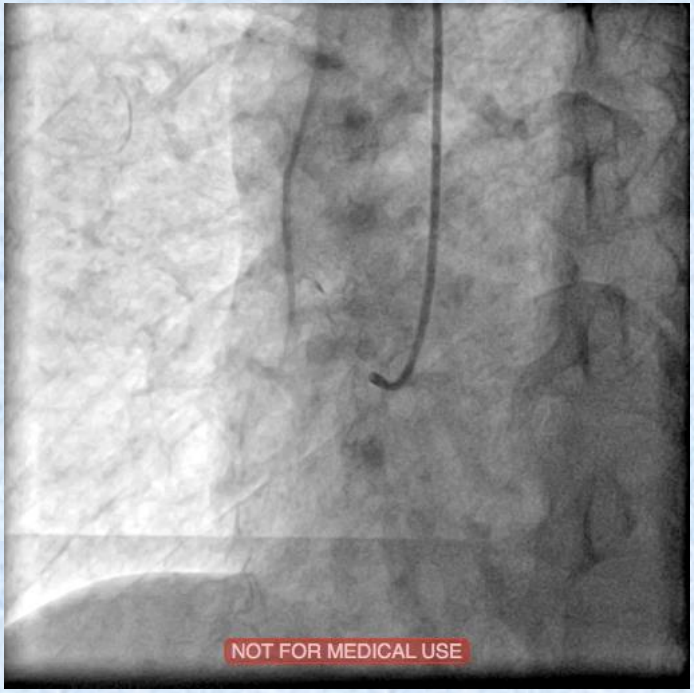
aVL
aVF


V1
V2
V3


V4
V5
V6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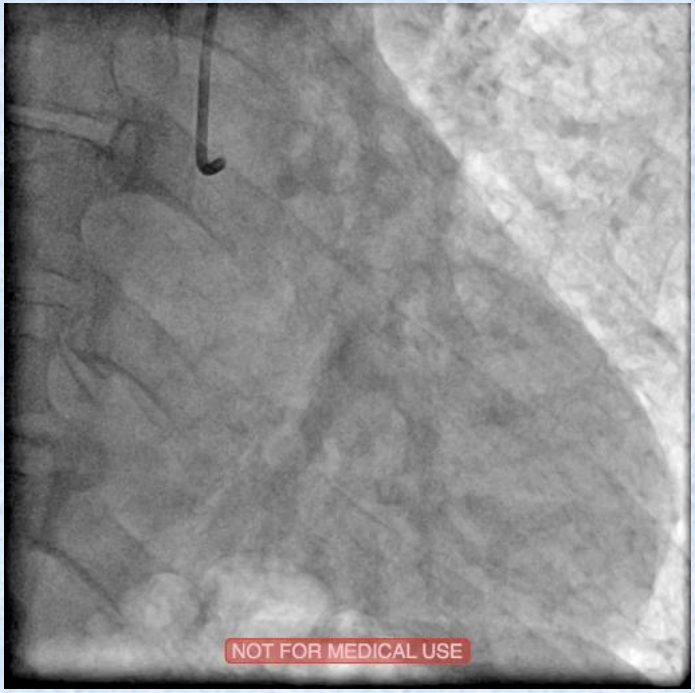
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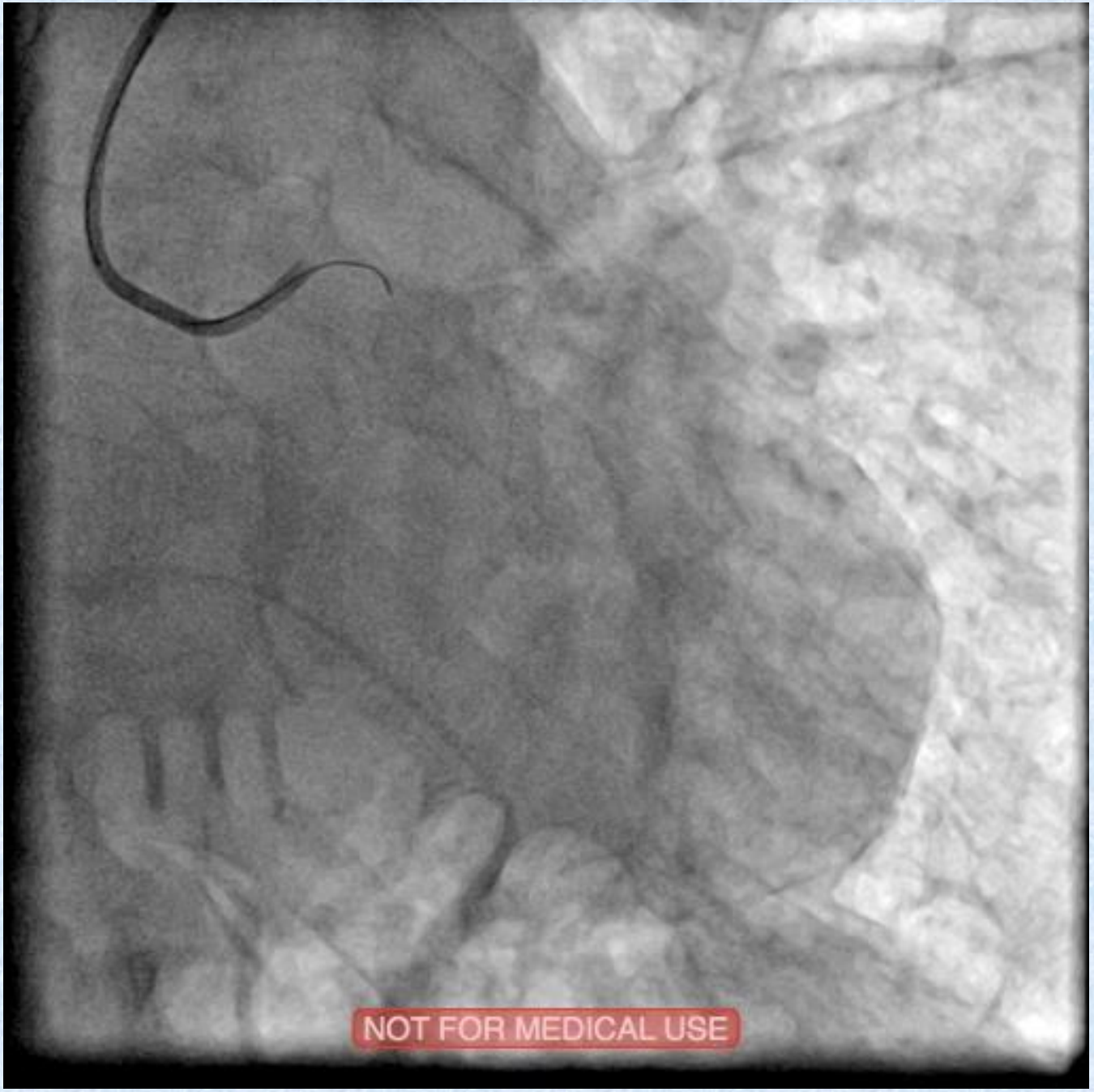
NOT FOR MEDICAL USE


Thanks for Your Attention