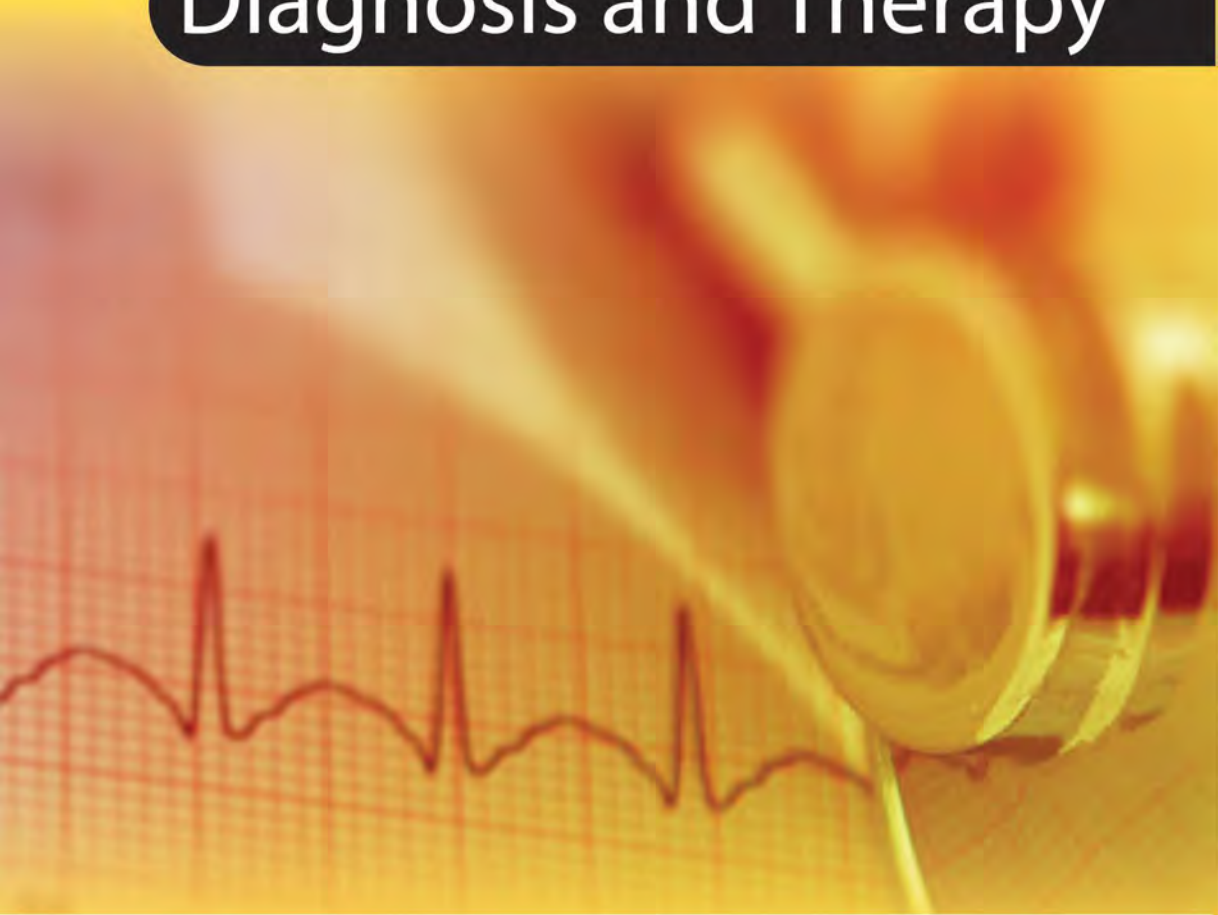
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INTERNAL MEDICINE

Diagnosis and Therapy



M Gabriel Khan

2nd Edition

JAYPEE

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CHAPTER

3 Acute Myocardial Infarction

PATHOPHYSIOLOGIC IMPLICATIONS

- An acute myocardial infarction (MI) is nearly always caused by occlusion of a coronary artery by thrombus overlying a fissured or ruptured atheromatous plaque. The ruptured plaque, by direct release of tissue factor and exposure of the subintima, is highly thrombogenic. Exposed collagen provokes platelet aggregation. Coronary angiography performed during the early hours of infarction has confirmed the presence of total occlusion of the infarct-related artery in over 90% of patients. It is not surprising that aspirin, through inhibition of platelet aggregation, reduces the incidence of coronary thrombosis and is especially useful in prevention of the progression of unstable angina to thrombosis and MI.
- Soft chewable aspirin 240 mg is particularly useful when given at the onset of chest pain produced by infarction.
- Aspirin, however, does not block all pathways that relate to platelet aggregation. In addition, aspirin does not ↓ the incidence of sudden death in patients with acute MI but an appropriate β-blocker does.
- Beta-blockers are the only cardioactive agents currently available that are proven to prevent sudden cardiac death (Table 3.1).
- The increased morning incidence of acute MI documented in several studies of the diurnal variation of infarction is related to the early morning catecholamine surges, which induce platelet aggregation, and an ↑ in blood pressure (BP) and hydraulic stress, which may lead to plaque rupture (Flowchart 3.1).
- Beta-adrenergic blockers have been shown to ↓ the early morning peak incidence of acute infarction and sudden death.
- Unfortunately, when an atheromatous plaque ruptures, the thrombogenic effect of plaque contents cannot be completely nullified by the inhibition of all aspects of platelet aggregation, and chemical agents that can arrest the effects of these thrombogenic substances deserve intensive study.
- Use of a β-blocking agent may inhibit plaque rupture perhaps by its ability to ↓ cardiac ejection velocity. This action reduces hydraulic stress on the arterial wall that might be critical at the arterial site where the atheromatous plaque is predisposed to rupture (Flowchart 3.2).

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Table 3.1: Total deaths and sudden deaths in beta-blocker randomized control trials of postmyocardial infarction and heart failure patients.

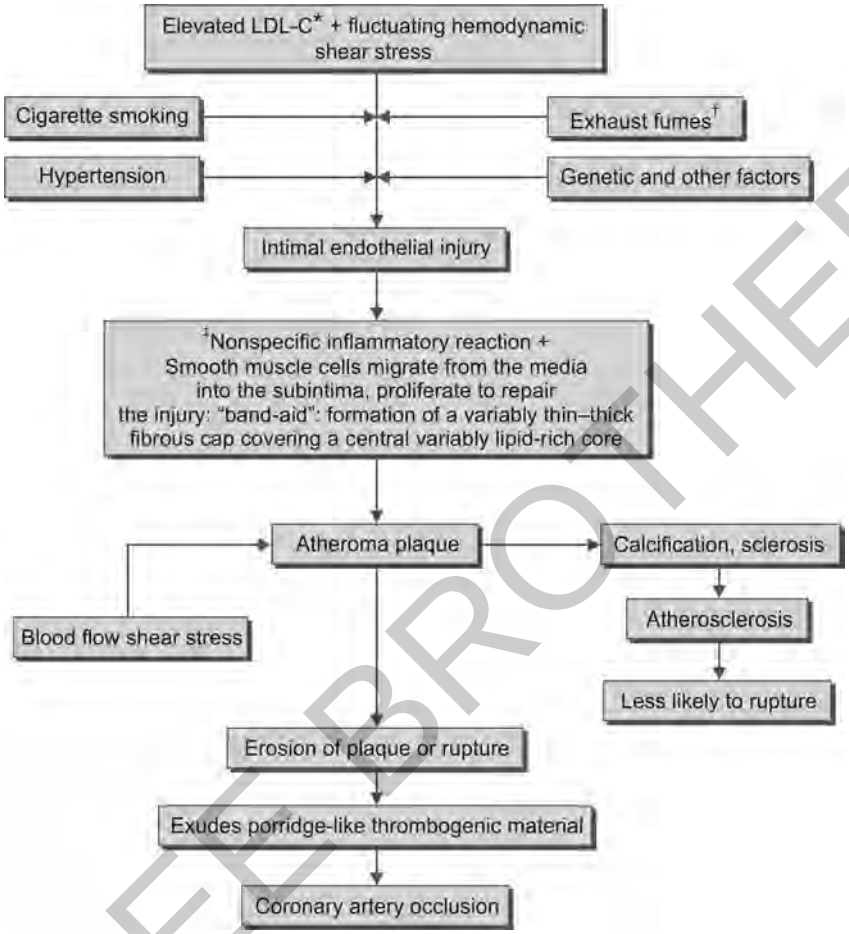
	Control	*Drug*	*% Decrease*
Propranolol*			
Total sudden deaths	78	60	64
5 am–11 am	31	11	64
Total deaths	188/1,460	138/1,456	26
Timolol†			
Total sudden deaths^	95	47	50
Instant, within seconds	38	11	71
Total deaths	117/718	67/670	42
Metoprolol#			
Total sudden deaths	21	9	57
Total deaths	31/147	25/154	19##
MERIT-HF^			
Total sudden deaths	132	79	40.2
Total deaths	217/2,001	145/1,990	33.2
Bisoprolol [CIBIS]‡			
Total sudden deaths	83	48	42.2
Total deaths	228/1,320	156/1,327	31.6

*BHAT study, AM sudden death (Peters et al. 1989) follow-up 25 months, 120–240 mg daily.
†Timolol Norwegian MI study; mean duration 17 months.
Follow-up: 10 mg twice daily.
#Metoprolol: A very small study of only approximately 150 patients in each group followed for 3 years.
##"This reduction in cardiac mortality did not reach statistical significance, as was to be expected from our sample size" (Olsson et al. 1985).
^In MERIT-HF, there were fewer sudden deaths in the metoprolol CR/XL group than in the placebo group [79 vs 132, 0.59 (0.45–0.78); p = 0.0002] and deaths from worsening heart failure [30 vs 58, 0.51 (0.33–0.79); p = 0.0023].
‡Bisoprolol in CIBIS; significant decrease in deaths and sudden deaths.
Carvedilol did not prevent sudden death in two large RCTs; total mortality in RCT was not significantly reduced in post-MI patients.

- Occlusion of a coronary artery leads, in about 20 minutes, to death of cells in areas of severely ischemic tissue, which will usually become necrotic over 4–6 hours. Since early and late mortality are directly related to the size of the infarct, limitation of infarct size (or even prevention of necrosis) by means of thrombolytic therapy initiated at the earliest possible moment, is of the utmost importance.
- The ischemic zone surrounding the necrotic tissue provides electrophysiologic (EP) inhomogeneity that predisposes the occurrence of

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Flowchart 3.1: Pathophysiology of atheroma formation, erosion, rupture, causing sudden cardiac death and myocardial infarction. Why the coronary arteries?



**Coronary arteries prone to atheroma:* As empty in systole and refilled during diastole, hemodynamically fluctuating shear stress causes injury to the intima; finally erosion or rupture of atheroma plaque occurs particularly in genetically susceptible individuals. Coronary arteries are hydraulically vulnerable unstable channels. During an average lifespan, they empty and fill rapidly at fluctuating shear stress more than 2 billion times with altered arterial hemodynamics at bends, bifurcations and at ostia. Over time the altered shear stress causes erosion and fissuring of plaques. High levels of low-density lipoprotein cholesterol (LDL-C) and in diabetics elevated sitosterol (Khan 2017) cause atheroma and arterial obstruction.

The algorithm suggests that fatal events may be best prevented by:

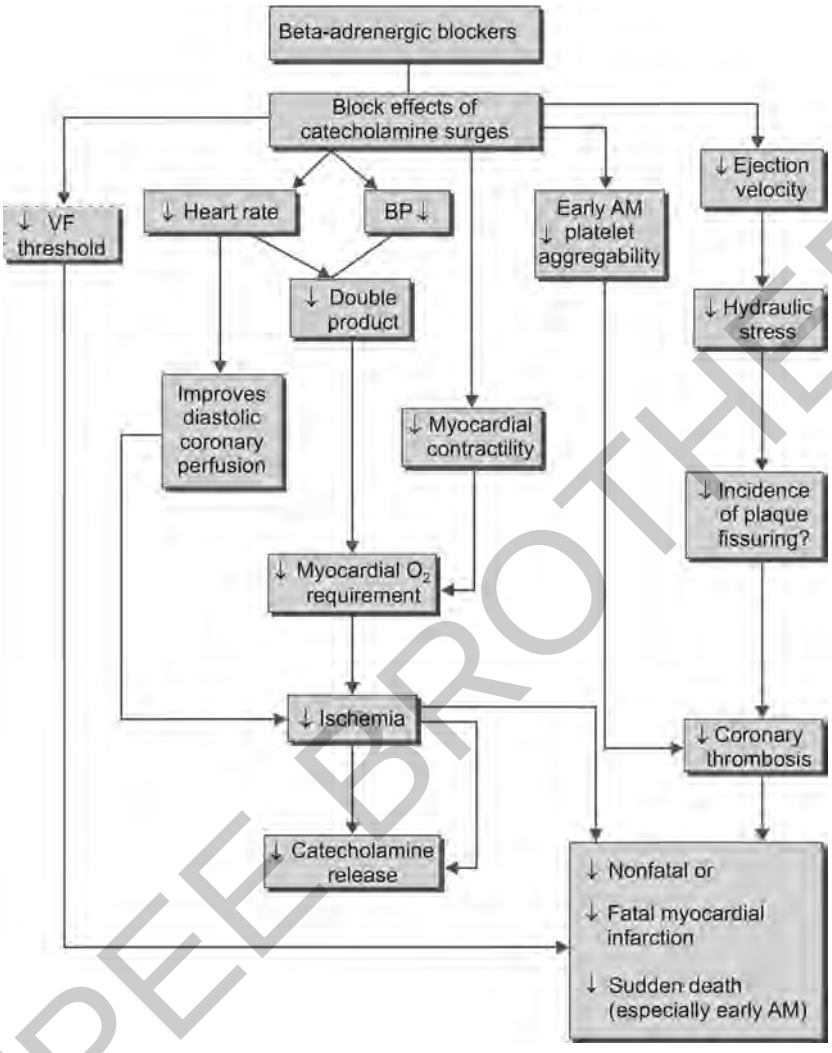
- Greatly reducing culprit LDL-C levels and sitosterol in diabetics, and administration of an appropriate β-blocker to alter hemodynamic shear stress and prevent erosions. For individuals with diabetes mellitus, blood sitosterol levels must be maintained less than 4.4 mg/L (Khan 2017).
- Fatal or nonfatal MI and sudden deaths were significantly prevented by timolol and propranolol, but by no other cardioactive agents; perhaps β1 and β2 agents are more cardioprotective than β1 agents.

†From motor vehicles, as dangerous as smoking.

‡Aspirin: A double agent; inhibits platelet aggregation and perhaps the inflammatory process.

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Flowchart 3.2: Salutary effects of beta-adrenergic blockade. (↑, increase; ↓, decrease).



Source: Khan MG. On Call Cardiology, 3rd edition. Philadelphia: WB Saunders, Elsevier Science; 2006.

lethal arrhythmias. These arrhythmias are most common during the early hours after onset and contribute to one of the major mechanisms of sudden death.

Extensive myocardial necrosis is the major determinant of congestive heart failure (CHF); papillary, septal, and free wall rupture; and cardiogenic shock, in which greater than 35% of the myocardium is usually infarcted.

- The most effective means of reducing the extent of myocardial necrosis is timely percutaneous coronary intervention (PCI) (within 60 minutes) or administration of thrombolytic therapy, aspirin, and a β-blocking agent as soon as possible after the onset of symptoms of coronary thrombosis.

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Diabetic sitosterolemia: High blood sitosterol causes heart attacks and stroke in diabetics. This important novel discovery is discussed at the end of this chapter.

DIAGNOSIS

Chest Pain

- Usually lasts more than 20 minutes and often persists for several hours. However, the pain of infarction can last for only 15 minutes and, occasionally, fatal infarction is ushered in by only a few minutes of severe pain or even unheralded cardiac arrest. Infarction may be silent, particularly in diabetic patients and in the elderly.
- Typically retrosternal and across the chest.
- Variations of a crushing, vise-like, heavy weight on the chest, pressure, tightness, strangling, aching.
- At times, only a discomfort with an oppression and burning or indigestion-like feeling.
- May radiate to the throat, jaws, neck, shoulders, arms, scapulae, or the epigastrium. At times, pain is centered at any one of these areas, e.g. the left wrist or shoulder, without radiation.
- Usually builds up over minutes or hours, as opposed to aortic dissection, in which pain has an abrupt onset like a gunshot.

  Associated symptoms and factors include:
- Diaphoresis, cold clammy skin, apprehension.
- Shortness of breath, nausea, vomiting, dizziness.
- Presyncope and rarely syncope may occur due to bradyarrhythmias, especially in inferior MI.
- Occasionally, no pain. A marked ↓ in BP with associated symptoms, along with electrocardiogram (ECG) findings, should suffice in making the diagnosis.
- Painless infarcts (in about 10% of patients), especially in diabetics or the elderly. In these patients, associated symptoms are often prominent and serve as clues to diagnosis.
- Over 50% of patients have a history of angina or prior infarction.
- Approximately, 33% of patients with acute infarction have no major risk factors: Death of a parent or sibling less than age 55, hypercholesterolemia, cigarette smoking, hypertension, or diabetes. Absence of these factors should not influence the diagnosis.

Physical Signs

- Patient appears apprehensive, anxious, cold, clammy.
- Area of chest pain may be indicated with a clenched fist.
- Tachycardia 100–120/minute. An ↑ in BP due to increased sympathetic tone is observed in slightly more than half of patients with anterior infarction.

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- Bradycardia less than 60 beats/minute and a ↓ in BP in about two-thirds of inferior infarcts; many of these patients become hypotensive, sometimes profoundly.
- S4 gallop is common; S3 and S4 if in CHF or cardiogenic shock.
- Murmur of mitral regurgitation (MR) due to papillary muscle dysfunction.
- Crepitations, more prominent over the lower third of the lung fields, may be present.
- Elevated jugular venous pressure due to left and right CHF or a very high venous pressure in the presence of right ventricular infarction or cardiac tamponade.
- Frequently, there are no abnormal physical signs, and this finding in a patient with suggestive symptoms should not ↓ the level of suspicion that the patient may have an MI.

Although sophisticated tests evolved in the 1980s to improve diagnostic accuracy, they are of limited value in the era of thrombolysis. Thus, a relevant history and correct interpretation of the ECG are of paramount importance in the implementation of early thrombolytic therapy, which will be of greatest benefit if given very early after symptom onset (<60 minutes).

Electrocardiogram

Diagnostic Features of ST-segment Elevation Myocardial Infarction

- ST-segment elevation of greater than 1 mm in more than two-limb leads (Figs. 3.1 to 3.3), or
- At least 2 mm ST elevation in more than two precordial leads (Figs. 3.4 and 3.7).

The above criteria, which have been used in most clinical trials of thrombolytic therapy, have become internationally standard and are considered diagnostic in patients with symptoms suggestive of acute MI. Where symptoms are not typical, the response to nitroglycerin is ascertained. Also, minimal ST-segment elevation in black patients must be reassessed to exclude the occasional normal variant. There is clear recognition that Q waves may evolve early or late and cannot be relied upon for early diagnosis. Thus, the terms "transmural" and "nontransmural" have been abandoned and Q wave or non-Q wave infarction cannot be categorized in the early phase. The best differentiating feature is ST-segment elevation, which is present in 95% of patients with acute coronary thrombotic occlusion.

In addition, later ECG signs of infarction include:

- Diminution of R waves (poor R wave progression)
- Evolving Q waves (Fig. 3.2)
- The simultaneous presence of reciprocal ST-segment depression is important to confirm the electrocardiographic diagnosis (Figs. 3.1 to 3.3).

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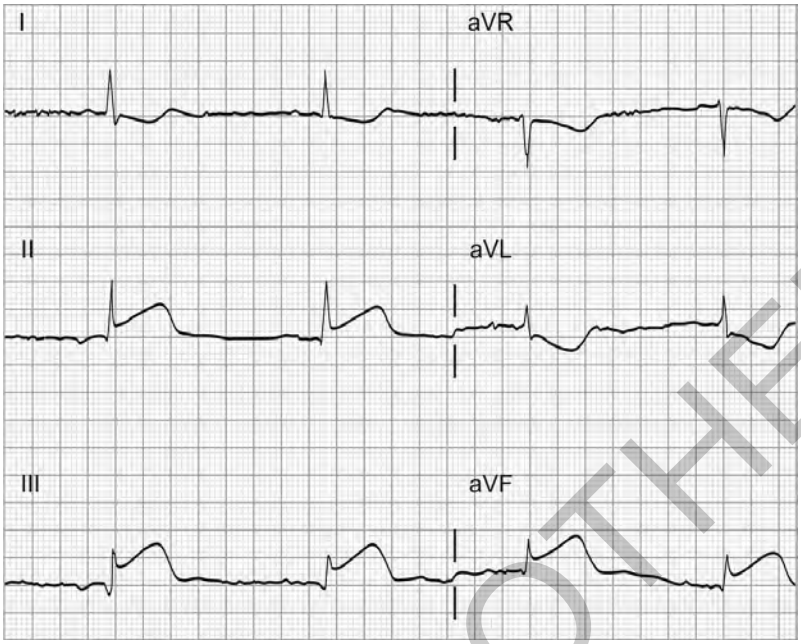


Fig. 3.1: Acute inferior myocardial infarction. ST elevation in three inferior leads; note reciprocal depression in leads I and aVL.
Source: Khan MG. On Call Cardiology, 3rd edition. Philadelphia: WB Saunders, Elsevier Science; 2006. p. 84.

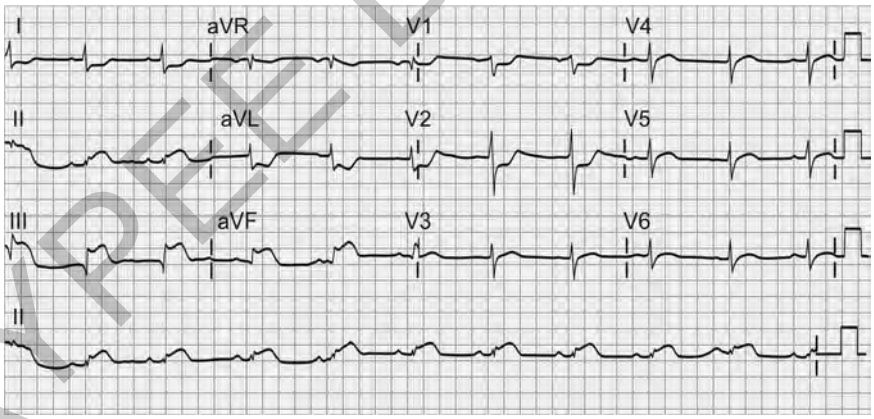


Fig. 3.2: Acute inferior myocardial infarction (STEMI); abnormally shaped high ST segment in inferior leads. Note the reciprocal depression in leads I, aVL, V1 and V2. Reciprocal depression is not diagnostic of infarction but is an important confirmatory sign that strongly supports the diagnosis.

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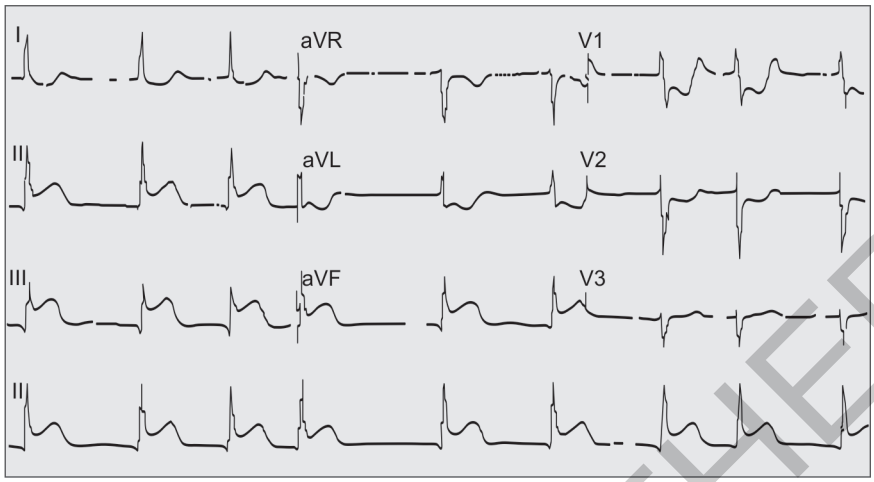


Fig. 3.3: Acute inferior myocardial infarction: ST elevation in leads II, III, and aVF. Note reciprocal depression in leads V1, V2 and aVL.

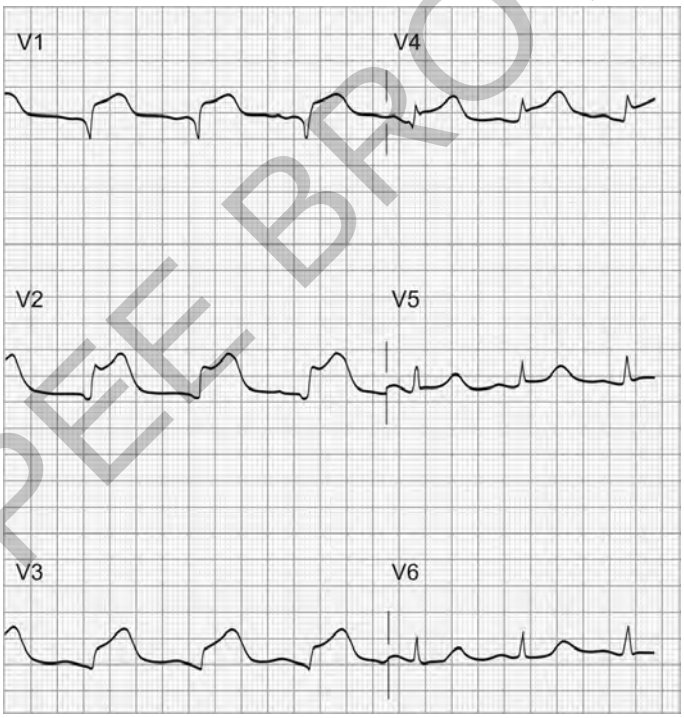


Fig. 3.4: Abnormal ST elevation greater than 2 mm in three precordial leads V1-V3 and V4: anterior STEMI.

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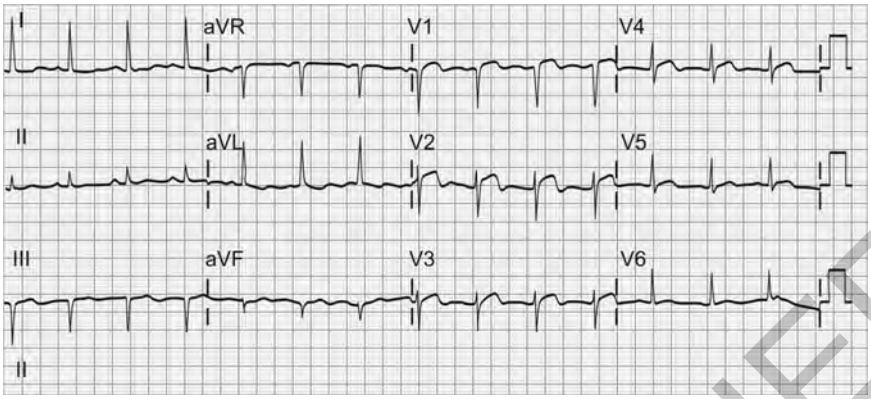


Fig. 3.5: Abnormal ST-segment elevation V1-V6 diagnostic for acute anterior MI.

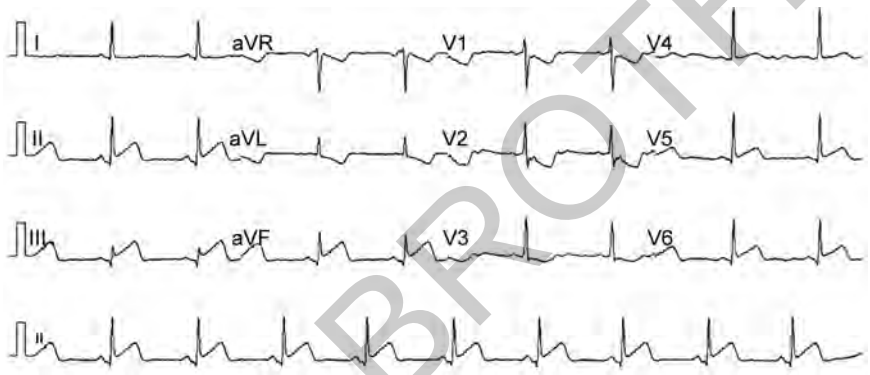


Fig. 3.6: ST-segment elevation leads II, III, aVF and anterolateral leads V5 and V6 acute inferolateral infarction. Note reciprocal ST depression leads aVL, V1-V3 help to confirm the diagnosis of acute MI.
Source: Khan GM. A new electrode placement method for obtaining 12-lead ECGs. Open Heart. 2015;2:e000226.

- Patients who are developing non-Q wave infarction often manifest ST depression, or T wave change.

If the first ECG is not diagnostic of acute injury/infarction, but the patient is strongly suspected of having an acute coronary syndrome, the ECG is repeated q 15 minutes until diagnostic changes are observed or until the creatine kinase (CK) and/or CK-MB results or troponins are reported.

Mimics of Acute MI Pattern on ECGs

An incorrect diagnosis may lead to unneeded coronary angiograms.
Mimics include:

- Early repolarization pattern
- In some young individuals, especially African Americans, the ST segment is elevated in V3 to V5 associated with minor T wave inversion

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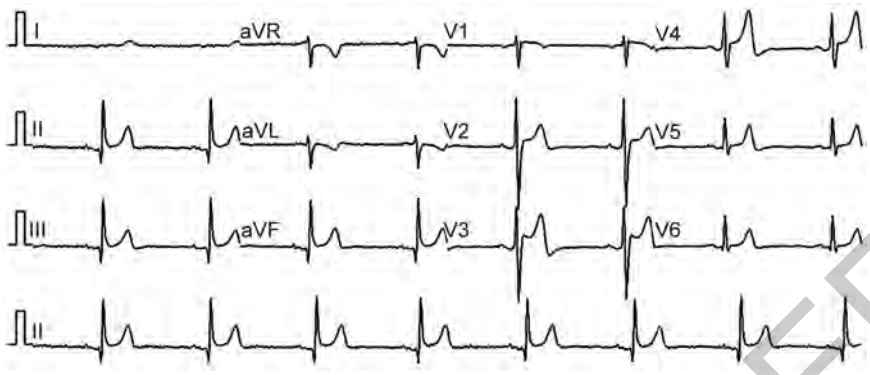


Fig. 3.7: Routine ECG from a 24-year-old male with no heart disease. Sinus bradycardia, ST segment concave (not convex) elevation II, III, aVF, and normal variant in V2, V3, V4; can be misdiagnosed as acute pericarditis or ST-segment elevation myocardial infarction (STEMI). Note the ST segment in V3 has a small notch at its commencement and is concave and not the convex shape of STEMI. There is reciprocal depression in aVR. If the ST elevation in II, III, aVF were due to inferior STEMI, there should be some reciprocal depression in I and aVL.

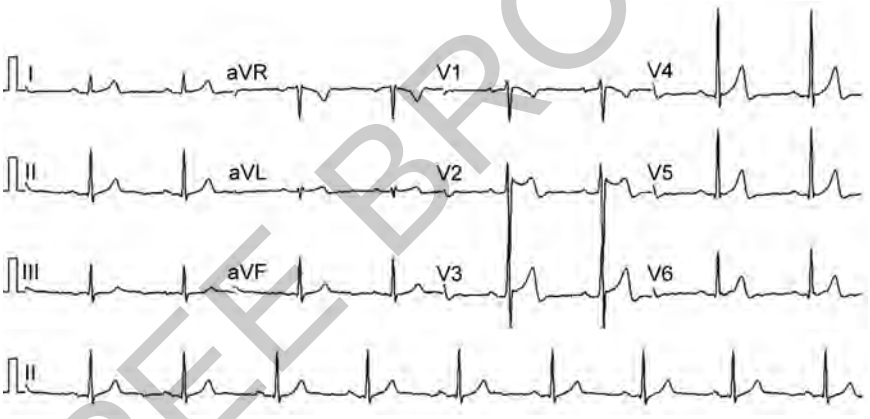


Fig. 3.8: ST-segment elevation V2 and V3; normal variant in a healthy 26-year-old man. Rarely observed in women.

as a normal variant; the ST segment tends to be slightly coved and may mimic acute MI, and caution is required to assess the clinical findings (Fig. 3.8).

- Acute pericarditis: Elevation in lead I is accompanied by elevation in leads II, III, and aVF; the ST elevation is concave, as opposed to convex upward with an injury current of infarction.
  - Reciprocal ST depression and PR-segment elevation in aVR is a typical finding with pericarditis.
  - Characteristic features of acute pericarditis: ST-segment elevation in most leads; I, II, aVL, aVF, V5 and V6, with reciprocal ST depression

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and PR-segment elevation in aVR. In addition, note sinus tachycardia and prominent PR-segment depression commonly seen with acute pericarditis.

- *Takotsubo stress cardiomyopathy*: If the ECG is equivocal and there is a strong clinical impression that acute MI is present, valuable confirmatory information may be obtained from an echocardiogram that is diagnostic of Takotsubo stress cardiomyopathy.
- Because the initial abnormality may not be fully diagnostic in up to 40% of cases, it is imperative to correlate the findings with accurate historical details. In patients with chest pain, new or presumably new Q waves in two leads with ST elevation are diagnostic in over 85% of cases.
- Q waves are fully developed in 4–12 hours and may manifest as early as 2 hours from onset of chest discomfort or associated symptoms.
- Evolutionary ST-T changes occur during 12–24 hours but may be delayed up to 30 hours.
- Inferior MI ST elevation in lead II, III and aVF with evolving Q waves and reciprocal depression in V1-V3. The latter depression may be due to reciprocal changes, but there is evidence to suggest that in some patients it is due to left anterior descending (LAD) disease. The evolutionary changes in repolarization that occur with inferior infarction evolve more rapidly than with anterior infarcts.
- Tachycardia may ↑ ischemic injury, causing elevation of the ST segment that must be differentiated from extension of infarction or pericarditis. Importantly, reciprocal depression does not occur in pericarditis.

Nondiagnostic ECG

Acute MI may be present with ECG changes that are nonspecific in 10–20% of cases and may result from:

- Slow evolution of ECG changes. The tracing may remain normal for several hours.
- Old infarction masking the ECG effect of a new infarct.
- Inferior MI associated with left anterior hemiblock in which R waves are expected to be small in leads II, III, aVF.
- Left bundle branch block (LBBB).
- Apical infarction.
- Posterolateral infarction is not usually associated with ST elevation or Q waves.

ST elevation of infarction from the following:

- Acute pericarditis where the ST segment elevation is not confined to leads referable to an anatomic segmental blood supply. Thus, elevation in lead I is accompanied by elevation in leads II, III, aVF; the ST elevation is concave, as opposed to convex, upward with an injury current of infarction; and reciprocal depression is absent.

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- Early repolarization changes may mimic infarction but are often observed mainly in leads V5 and V6 with a subtle "fish hook" configuration. This feature is common in blacks.
- Left ventricular (LV) aneurysm, in which there may be permanent ST elevation.

ECG and Location of Infarction Sites

- Anteroseptal: ST elevation V1, V2, V3
- Anterior: ST-segment elevation V2-V4
- Extensive anterior: V1-V6, 1 aVL
- Anterolateral: V4-V6, 1 aVL
- Inferior: 2, 3, aVF
- Posterior infarction: Tall R waves V1, V2, upright T waves occasionally ST depression V1-V2 and often inferior or inferolateral infarct signs
- Right ventricular infarction: ST-segment elevation V2, V4R, often associated with inferior infarction.

ECG and Size of Infarction

The extent of ST-segment elevation gives clues to infarct size, but the correlation is not close. The site of infarction influences mortality but is not as paramount as the size of infarction, which can be reasonably ascertained from the number of leads showing ST elevation, as follows:

- Small MI: 2 or 3 leads
- Moderate: 4 or 5 leads
- Large: 6 or 7 leads
- Extensive: 8 or 9 leads.

Echocardiography in Acute Myocardial Infarction

Echocardiography is not required routinely in an uncomplicated MI, especially where the history and ECG are typical or with non-Q wave infarction.

Indications

- Patients with cardiogenic shock often require assessment to determine the presence of mechanical complications: septal rupture, severe MR, myocardial rupture, and tamponade. Color Doppler can provide quick results and, with the unconscious patient, a transesophageal echocardiogram (TEE) is helpful and accurate.
- To distinguish acute severe MR from papillary muscle rupture.

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INTERNAL MEDICINE
Diagnosis and Therapy

Salient Features

- The practical substance of this textbook is aimed at family physicians, internists, residents and interns who assess and prescribe for patients' internal medicine problems including cardiology
- Up-to-date information and in-depth coverage in areas of internal medicine relevant to hospitals and office practices
- One million diabetics die annually in India and 1.5 million in China. M Gabriel Khan has recently shown that diabetics have high blood sitosterol and is the most likely cause of heart attacks, stroke and peripheral vascular disease. A new treatment for diabetes should evolve.

M Gabriel Khan MD FRCP (London) FACC FRCPC MB BCh (Queen's Belfast) is Former Associate Professor, Department of Medicine, Division of Cardiology, University of Ottawa; and Cardiologist, The Ottawa Hospital (1968–2017), Ottawa, Ontario, Canada. His recent research culminated in a novel discovery that should save the lives of many diabetics who die because of heart stroke. He emphasized that sitosterol causes atheromatous blockage of arteries, and is the first investigator to show that diabetics have high blood sitosterol. He has become an internationally acclaimed cardiologist through his writings: *Cardiac Drug Therapy, Rapid ECG Interpretation, Heart Disease Diagnosis and Therapy, On Call Cardiology,* and *Encyclopedia of Heart Diseases*.

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