

4

The ECG in patients with chest pain

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HISTORY AND EXAMINATION

There are many causes of chest pain. All the non-cardiac conditions can mimic a myocardial infarction, and so the ECG can be extremely useful when making a diagnosis. However, the ECG is less important than the history and physical examination, because the ECG can be normal in the first few hours of a myocardial infarction.

Acute chest pain can be caused by:

- Myocardial infarction
- Pulmonary embolism
- Pneumothorax
- Other causes of pleuritic pain
- Pericarditis
- Aortic dissection

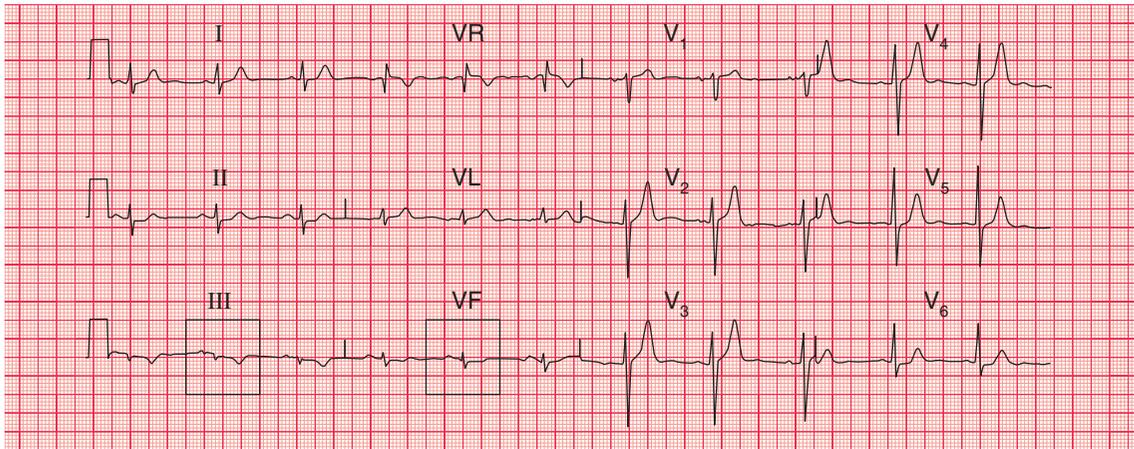


Fig. 4.1 Non-specific ST segment/T wave changes

Note

- Sinus rhythm
- Normal axis
- Normal QRS complexes
- ST segments probably normal, though possibly depressed in leads III and VF
- T wave inverted in lead III (possibly a normal variant) and flattened in VF



Inverted T wave in lead III



Flattened T wave in lead VF

- Ruptured oesophagus
- Oesophagitis
- Collapsed vertebra
- Herpes zoster.

Chronic or recurrent chest pain may be:

- Angina
- Nerve root pain
- Muscular pain
- Oesophageal reflux
- Nonspecific pain.

The ECG in Figure 4.1 was recorded in an A & E department from a 44-year-old man with rather vague chest pain. He was thought to have a viral illness and his ECG was considered to be within normal limits. He was allowed home, and died later that day. The postmortem examination showed a myocardial infarction which was probably a few hours old, and corresponded with his A & E attendance.

Acute chest pain

The typical pain of *myocardial infarction* is easy to recognize, the features being:

- central
- radiates to neck, jaw, teeth, arm(s) or back
- severe
- associated with nausea, vomiting and sweating.

Unfortunately not all patients have typical pain, and pain can even be absent.

Pulmonary embolism:

- causes pain similar to myocardial infarction if the embolus is central
- causes pleuritic pain if the embolus is peripheral
- is associated with breathlessness or haemoptysis
- can cause haemodynamic collapse.

Other lung disease, such as *infection* or *pneumothorax*, can be recognized from the pleuritic nature of the pain. This will be:

- worse on breathing
- often associated with a cough.

Pericardial pain can mimic both cardiac ischaemia and pleuritic pain, but can be recognized because it is relieved by sitting up and leaning forward.

Aortic dissection typically causes a 'tearing' pain (as opposed to the 'crushing' sensation of a myocardial infarction), and usually radiates to the back.

Oesophageal rupture follows vomiting.

Spinal pain is affected by posture, and associated root pain follows the nerve root distribution.

Shingles (herpes zoster) catches everyone out until the rash appears, although tenderness of the skin may provide a clue.

The physical examination of a patient with chest pain may reveal nothing other than the signs associated with the pain itself (anxiety, sinus tachycardia, restlessness, a cold and sweaty skin), but some specific signs are worth looking for:

- Left ventricular failure suggests myocardial infarction.
- A raised jugular venous pressure suggests myocardial infarction or pulmonary embolus.
- A pleural friction rub suggests pulmonary embolism or infection.
- A pericardial friction rub suggests pericarditis (?viral, ?secondary to myocardial infarction) or aortic dissection.
- Aortic regurgitation suggests aortic dissection.
- Unequal pulses or blood pressure in the arms suggests aortic dissection.
- Bony tenderness suggests musculoskeletal pain.

Chronic chest pain

The main differential diagnosis is between angina and the chest pain that is common in middle-aged men, but for which no clear diagnosis is usually made. This pain is sometimes called 'atypical chest pain', but this is a dangerous diagnostic label because it implies that there is a diagnosis (by implication, cardiac ischaemia) but that the symptoms are 'atypical'. Some of these pains are

musculoskeletal, Tietze's syndrome of pain from the costochondral junctions being the most obvious, but in most cases the best diagnostic label is 'chest pain of unknown cause'. This indicates a possible need for later re-evaluation.

The important features in the history that point to a diagnosis of angina are that the pain:

- is predictable
- usually occurs after a constant amount of exercise
- is worse in cold or windy weather
- is induced by emotional stress
- is induced by sexual intercourse
- is relieved by rest, and rapidly relieved by a short-acting nitrate.

The physical signs to look for are:

- Evidence of risk factors (high blood pressure, cholesterol deposits, signs of smoking)
- Any signs of cardiac disease (aortic stenosis, an enlarged heart, signs of heart failure)
- Anaemia
- Signs of peripheral vascular disease (which would suggest that coronary disease is also present).

THE ECG IN THE PRESENCE OF CHEST PAIN

Remember that the ECG can be normal in the early stages of a myocardial infarction. Having said that:

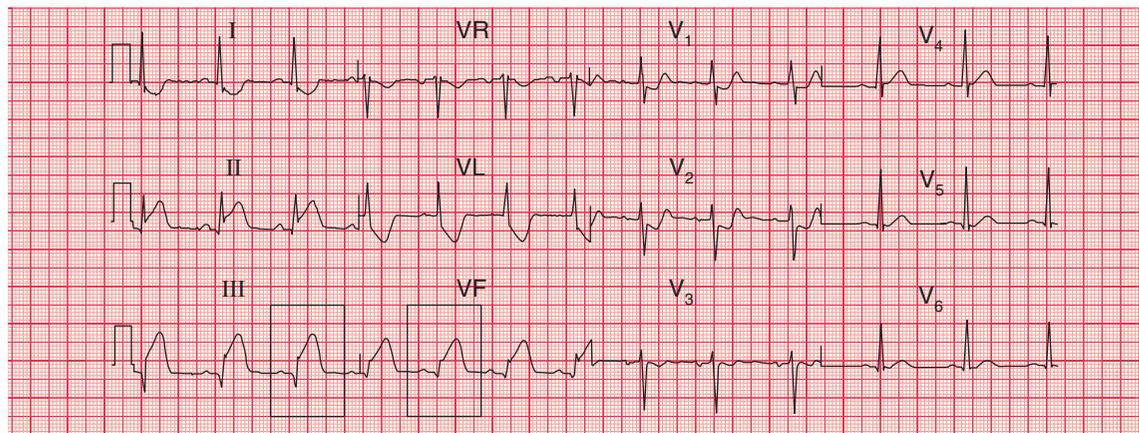
- An abnormal ECG is necessary to make a diagnosis of myocardial infarction before treatment is started.
- An ECG will demonstrate ischaemia in patients with angina *provided that* the patient has pain at the time the ECG is recorded.
- With pulmonary embolus there may be classical ECG changes, but these are often not present.
- With pericarditis, ECG changes, if present at all, are very nonspecific.

THE ECG IN PATIENTS WITH MYOCARDIAL INFARCTION

The diagnosis of a myocardial infarction depends on the history and examination, on the measurement of biochemical markers of cardiac muscle damage (especially the troponins) and on the ECG. A rise in troponin I or troponin T levels in patients with a history suggestive of a myocardial infarction is now taken to mean that infarction has occurred, but treatment still depends on the ECG. The term 'acute coronary syndrome' is now used to include:

- Myocardial infarction with ST segment elevation on the ECG
- Myocardial infarction (as shown by a troponin rise) with only T wave inversion or ST segment depression
- Chest pain with ischaemic ST segment depression but no troponin rise (what used to be called 'unstable angina')
- Sudden death due to coronary disease.

Stable angina and 'chest pain of unknown cause' remain entirely proper diagnostic labels for those patients who are admitted to hospital with chest pain, but for whom the term 'acute coronary syndrome' is inappropriate.



The development of ECG changes in myocardial infarction

The sequence of features characteristic of 'full thickness', or 'ST segment elevation', myocardial infarction is:

- Normal ECG
- ST segment elevation
- Development of Q waves
- ST segment returns to the baseline
- T waves become inverted.

The ECG leads that show the changes typical of a myocardial infarction depend on the part of the heart affected.

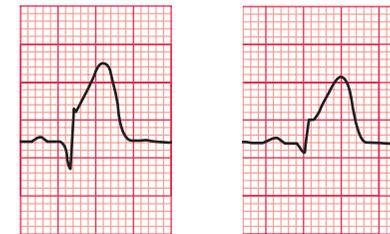
Inferior infarction

Figures 4.2, 4.3 and 4.4 show traces taken from a patient with a typical history of myocardial infarction: on admission to hospital, 3 h later, and 2 days later. The main changes are in the inferior leads II, III, and VF. Here the ST segments are initially raised, but then Q waves appear and the T waves become inverted.

Fig. 4.2 Acute inferior infarction

Note

- Sinus rhythm
- Normal axis
- Small Q waves in leads II, III, VF
- Raised ST segments in leads II, III, VF
- Depressed ST segments in leads I, VL, V₂, V₃
- Inverted T waves in leads I, VL, V₃



Raised ST segments in leads III and VF

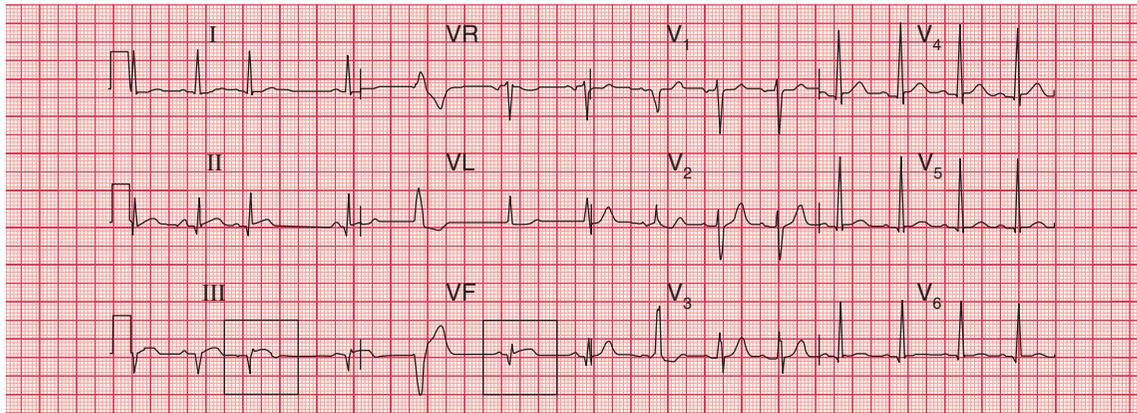
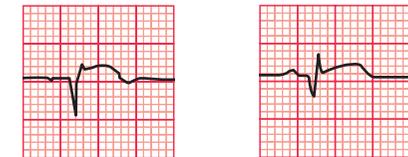


Fig. 4.3 Evolving inferior infarction

Note

- Same patient as in Figures 4.2 and 4.4
- Sinus rhythm with ventricular extrasystoles
- Normal axis
- Deeper Q waves in leads II, III, VF
- ST segments returning to normal, but still elevated in inferior leads
- Less ST segment depression in leads I, VL, V₃

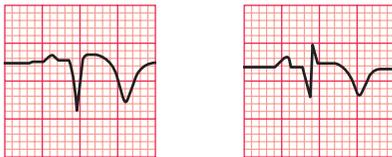


Deeper Q waves in leads III and VF

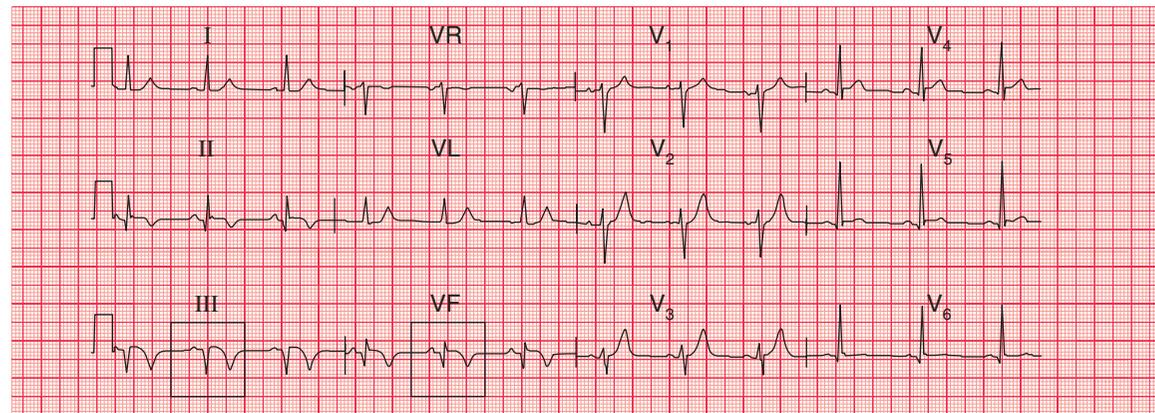
Fig. 4.4 Evolving inferior infarction

Note

- Same patient as in Figures 4.2 and 4.3
- Sinus rhythm
- Normal axis
- Q waves in leads II, III, VF
- ST segments nearly back to normal
- T wave inversion in leads II, III, VF
- Lateral ischaemia has cleared (as shown by ST segments in lateral leads)



Q waves, normal ST segments, and inverted T waves in leads III and VF



Anterior and lateral infarction

The changes of anterior infarction are seen in leads V_2 – V_5 . Lead V_1 , which lies over the right ventricle, is seldom affected (Fig. 4.5).

The lateral wall of the left ventricle is often damaged at the same time as the anterior wall, and then leads I, VL and V_6 show infarction changes. Figures 4.6 and 4.7 show the records of a patient with an acute anterolateral infarction, initially with raised ST segments and then with T wave inversion in the lateral leads. In the ECG in Figure 4.7 left axis deviation has appeared, indicating damage to the left anterior fascicle.

Figure 4.8 shows a record taken 3 days after a lateral infarction, with Q waves and inverted T waves in leads I, VL, and V_6 .

The ECG in Figure 4.9 was recorded several weeks after an anterolateral myocardial infarction. Although the changes in leads I and VL appear 'old', having an isoelectric ST segment, there is still ST segment elevation in leads V_3 – V_5 . If the patient had just been admitted with chest pain these changes would be taken to

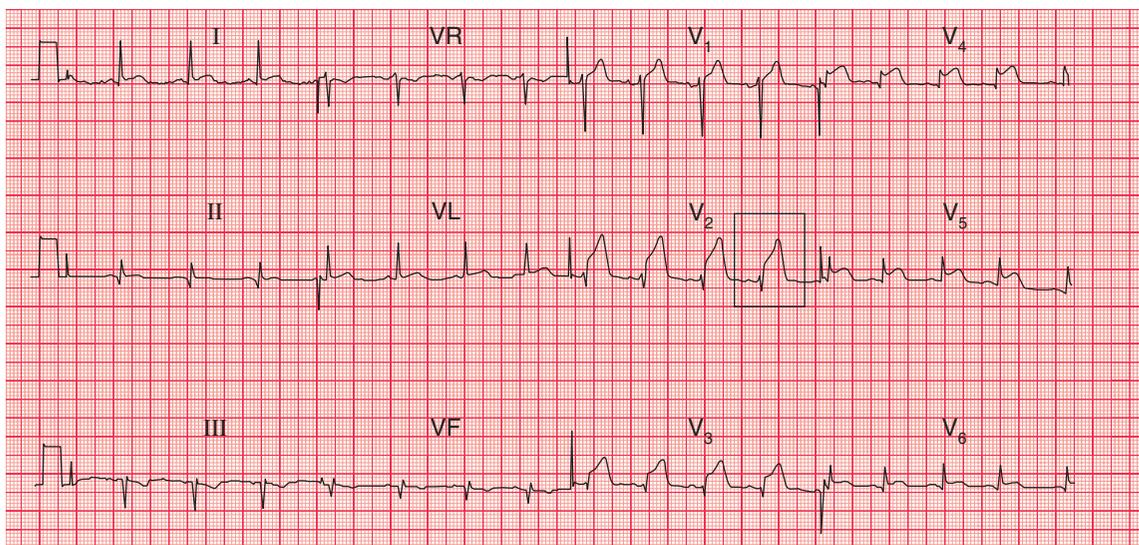


Fig. 4.5 Anterior infarction

Note

- Sinus rhythm
- Normal axis
- Raised ST segments in leads V_2 – V_5



Raised ST segment in lead V_2

indicate an acute infarction, but this patient had had pain more than a month previously. Persistent ST segment elevation is quite common after an anterior infarction: it sometimes indicates the development of a left ventricular aneurysm, but it is not reliable evidence of this.

An old anterior infarction often causes only what is called 'poor R wave progression'. Figure 4.10 shows the record from a patient who had had an anterior infarction some years previously. A normal ECG would show progressive increase in the size of the R wave from lead V_1 to V_5 or V_6 . In this case the R wave remains very small in leads V_3 and V_4 , but becomes a normal size in V_5 . This loss of 'progression' indicates the old infarction.

The time taken for the various ECG changes of infarction to occur is extremely variable, and the ECG is an unreliable way of deciding when an infarction occurred. Serial records showing progressive changes are the only way of timing the infarction from the ECG.

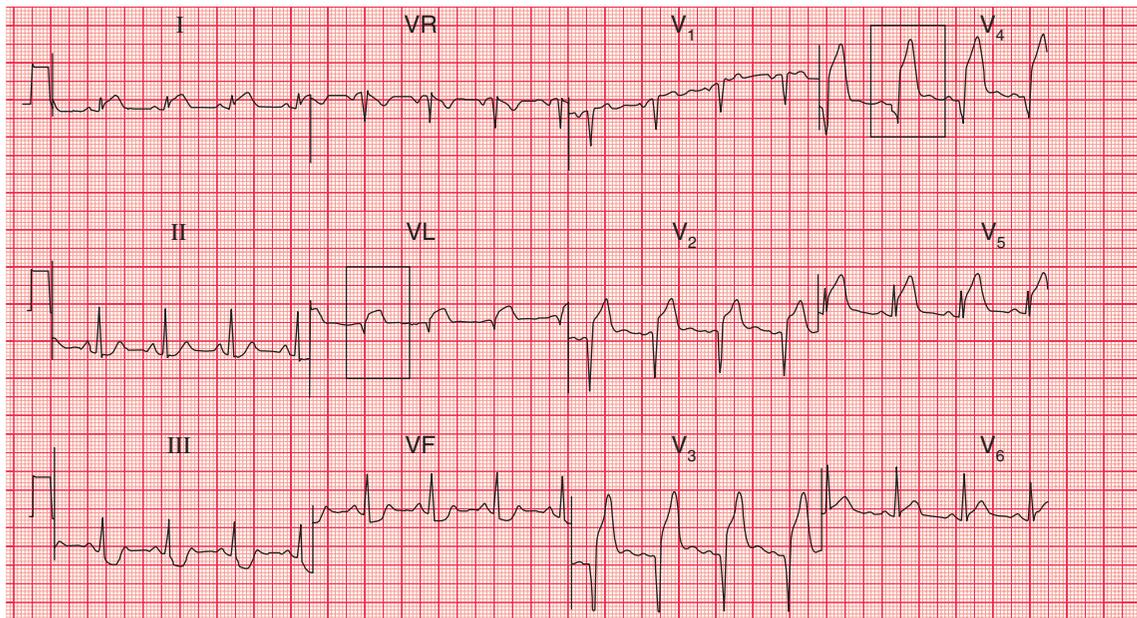


Fig. 4.6 Acute anterolateral infarction

Note

- Sinus rhythm
- Normal axis
- Q waves in leads VL, V₂-V₄
- Raised ST segments in leads I, VL, V₂-V₅



Raised ST segments in leads VL and V₄

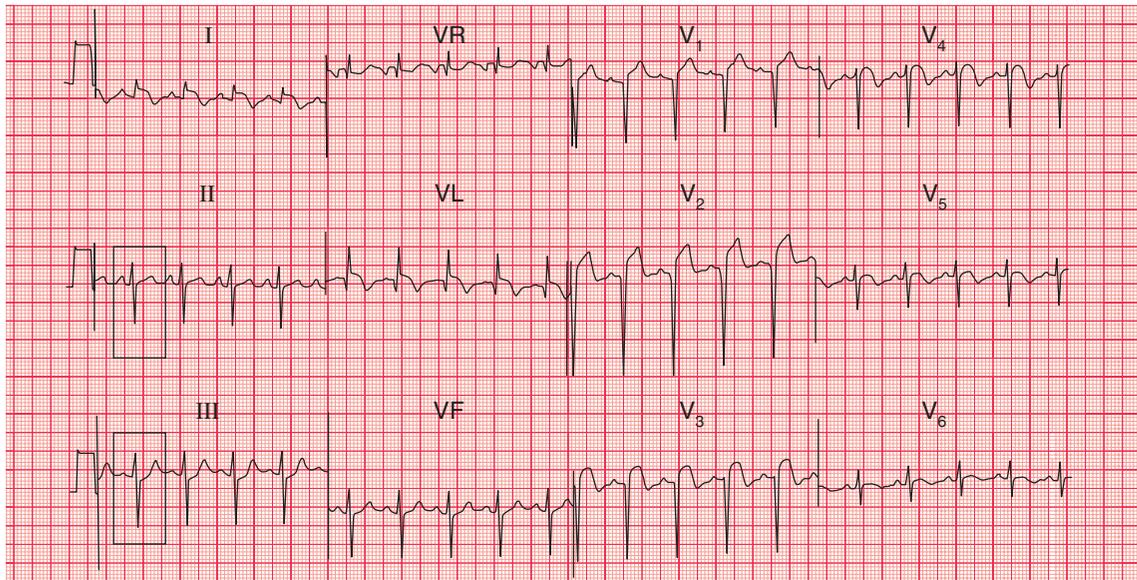
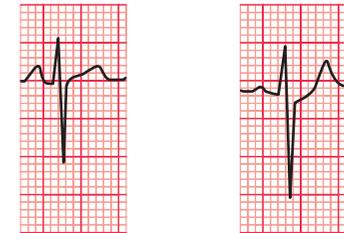


Fig. 4.7 Acute anterolateral infarction with left axis deviation

Note

- Sinus rhythm
- Left axis deviation
- ST segments now returning to normal
- T wave inversion in leads I, VL, V₄, V₅

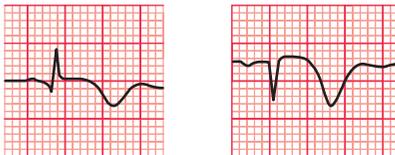


S waves in leads II and III: left axis deviation

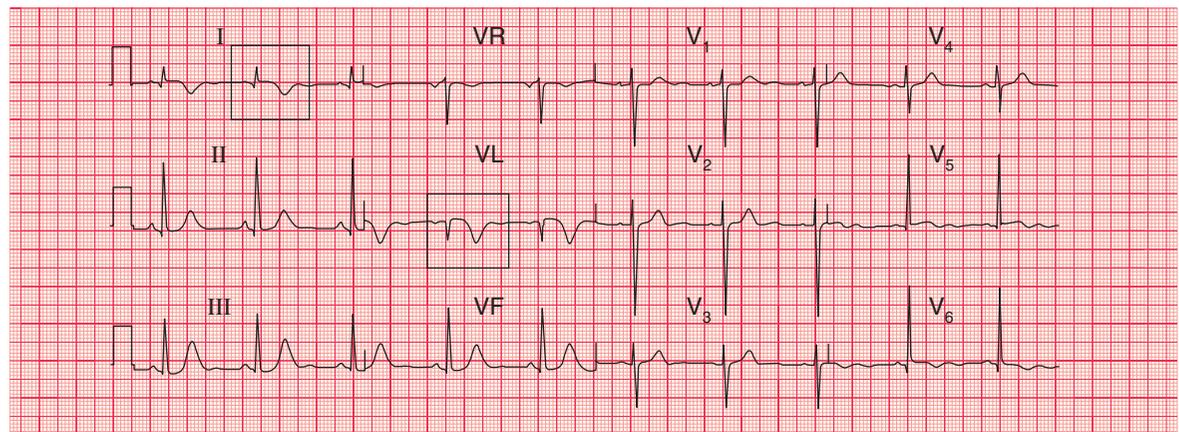
Fig. 4.8 Lateral infarction (after 3 days)

Note

- Sinus rhythm
- Normal axis
- Q waves in leads I, VL, ?V₆ (could be septal)
- ST segments isoelectric
- Inverted T waves in leads I, VL, V₆



Inverted T waves in leads I and VL



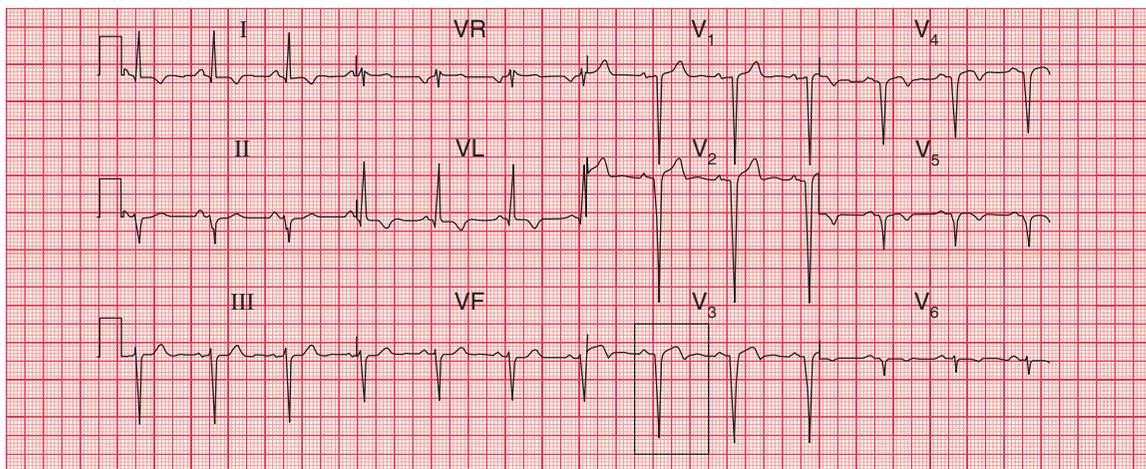


Fig. 4.9 Anterolateral infarction, ?age

Note

- Sinus rhythm
- Left axis deviation
- Q waves in leads I, II, V₂-V₅
- Raised ST segments in leads V₃-V₅
- Inverted T waves in leads I, VL, V₄-V₆

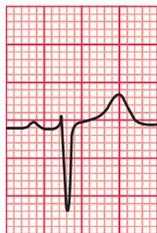


Raised ST segment in lead V₃

Fig. 4.10 Old anterior infarction

Note

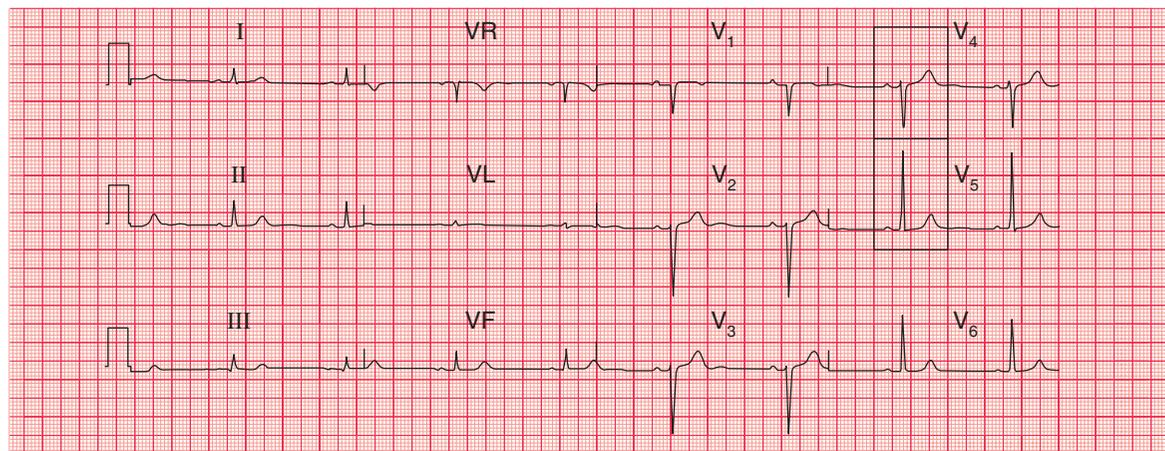
- Sinus rhythm
- Normal axis
- Small R waves in leads V₃-V₄, large R waves in V₅: this is 'poor R wave progression'



Small R wave in lead V₄



Tall R wave in lead V₅



Posterior infarction

It is possible to 'look at' the back of the heart by placing the V lead on the back of the left side of the chest, but this is not done routinely because it is inconvenient and the complexes recorded are often small.

An infarction of the posterior wall of the left ventricle can, however, be detected in the ordinary 12-lead ECG because it causes a dominant R wave in lead V₁. The shape of the QRS complex recorded from lead V₁ depends on the balance of electrical forces reaching the ECG electrode. Normally the right ventricle is being depolarized towards lead V₁, so causing an upward movement (an R wave) on the record; at the same time the posterior wall of the left ventricle is being depolarized, with the wave of excitation moving away from the electrode and so

causing a downward movement (an S wave) on the record. The left ventricle is more muscular than the right and therefore exerts a greater influence on the ECG, so in lead V₁ the QRS complex is normally predominantly downward, i.e. there is a small R wave and a deep S wave. In a posterior infarction, the rearward-moving electrical forces are lost so lead V₁ 'sees' the unopposed forward-moving depolarization of the right ventricle and records a predominantly upright QRS complex.

Figure 4.11 shows the first record from a patient with acute chest pain. There is a dominant R wave in lead V₁ and ischaemic ST segment depression (see p. 267) in leads V₂–V₄. When the chest electrodes were moved to the left axilla and back, to the V₇–V₁₂ positions, raised ST segments with Q waves typical of an acute infarction were seen.

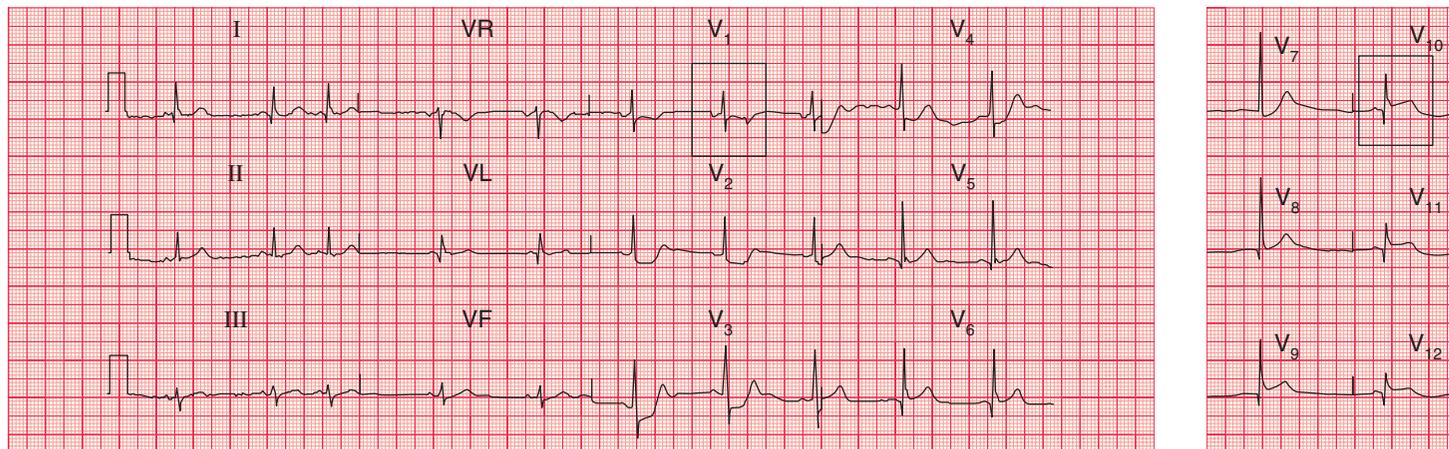


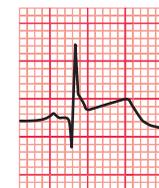
Fig. 4.11 Posterior infarction

Note

- Sinus rhythm with atrial extrasystoles
- Normal axis
- Dominant R waves in lead V₁ suggest posterior infarction
- ST segment depression in leads V₂–V₄
- Q waves and ST segment elevation in leads V₁₀–V₁₂ (posterior leads)



Dominant R wave in lead V₁



Q wave and raised ST segment in lead V₁₀

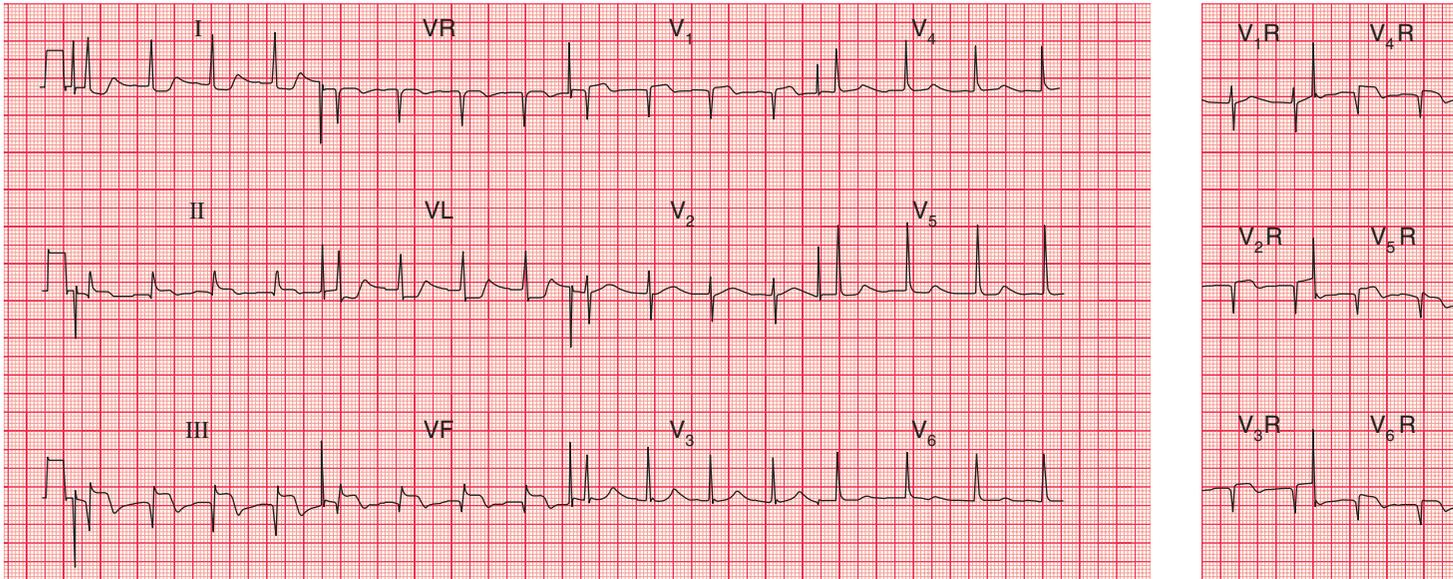


Fig. 4.12 Inferior and right ventricular infarction

Note

- Sinus rhythm
- Normal axis
- Raised ST segments in leads II, III, VF
- Raised ST segments in leads V₂R–V₅R
- Q waves in leads III, VF, V₂R–V₆R

Right ventricular infarction

Inferior infarction is sometimes associated with infarction of the right ventricle. Clinically, this is suspected in a patient with an inferior infarction when the lungs are clear but the jugular venous pressure is elevated. The ECG will show a raised ST segment in leads recorded from the right side of the heart. The positions of the leads correspond to those on the left side as follows: V₁R is in the normal V₂ position; V₂R is in the normal V₁ position; V₃R etc. are on the right side, in positions corresponding to V₃ etc. on the left side. Figure 4.12 is from a patient with an acute right ventricular infarct.