# Myocardial Infarction and Ischemia

# 3

Myocardial infarction occurs when oxygen delivery to the myocardium is inadequate for metabolic requirements and remains inadequate to the point of cellular death. This is in contrast to myocardial ischemia, wherein there is also inadequate oxygen delivery to the myocardium, but it is only temporary and is not prolonged or severe enough to lead to cell death. There is clearly a spectrum of response to inadequate oxygen delivery to the myocardium, ranging from mild ischemia through moderate and finally severe ischemia, and, if not reversible, ultimately to myocardial infarction (Fig. 3.1). The electrocardiographic appearance one observes depends on where on this spectrum the patient's oxygen compromise is located and which portion of the myocardium is affected.

## Ischemia

Ischemia is usually manifested by *reversible ST segment depression*. The reversibility of the changes is critical to the diagnosis of ischemia. The changes are reversible within a period of minutes, not weeks or months as may be the case with the changes of infarction. The ST segment depression may be downsloping, horizontal, or upsloping in configuration (Fig. 3.2). The most specific configuration for ischemia is downsloping ST segment depression, but this may occur in other situations as well (e.g., left ventricular hypertrophy—see Chap. 6) so it is not completely specific for ischemia. What is most specific for ischemia is *reversible* downsloping ST segment depression associated with symptoms of angina (e.g., substernal pressing chest pain, dyspnea, diaphoresis). Horizontal ST segment depression is not as specific as downsloping ST depression for ischemia, and upsloping ST depression is the least specific. But horizontal and even upsloping ST segment depressions which are associated with symptoms and are reversible do indicate ischemia. Upsloping ST segment depression can sometimes be seen at rapid heart rates without symptoms and does not indicate ischemia, but rather is due to atrial repolarization. These changes are called $T_A$ waves.

Two additional manifestations of ischemia should be mentioned. A less common form of ischemia than that described above is called Prinzmetal's angina, and is associated with coronary artery spasm with or without underlying fixed obstructive atherosclerotic disease. In Prinzmetal's angina, the ST segments are elevated, rather than depressed, and revert to normal over a period of minutes after the vasospasm-induced ischemia is resolved. The distinction between Prinzmetal's angina and transmural infarction, which is also associated with ST segment elevation (see below), is the rapid reversibility of the ST changes with Prinzmetal's angina as opposed to the persisting elevation in ST segments (hours to days) with transmural infarction. Finally, in rare circumstances, ischemia may be reflected on the electrocardiogram (EKG) by T wave inversions. This is very uncommon, and T wave inversions are more likely to be related to subendocardial infarction (see below). Nevertheless, when T wave inversions occur, are associated with symptoms of angina, and are reversible within minutes when symptoms resolve, that probably indicates ischemia.

## Myocardial Infarction

There are two types of myocardial infarction, and the names have changed over the course of time, with three pairs of terms (Table 3.1).

The oldest designation of the three is (1) transmural and (2) subendocardial, or non-transmural. These two categories imply an anatomical correlation between the appearance of the EKG and the extent of the damage to the heart wall. This implication, however, is not always correct, and hence the attraction to alternative terminology is based on the EKG changes. When the EKG shows the changes of transmural infarction, anatomical studies sometimes but not always confirm an infarction through the *entire thickness* ("trans-") of the heart *wall* ("-mural"). Likewise, when the EKG shows a subendocardial infarction, the damage may not be limited to

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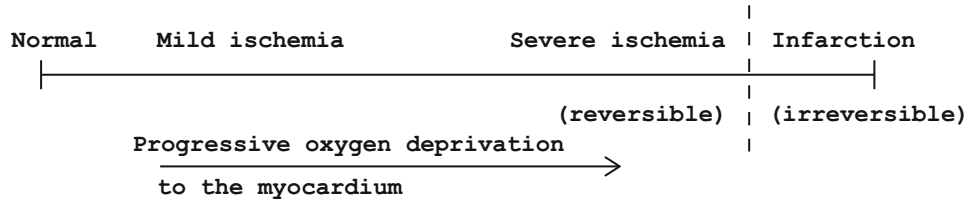



**Fig. 3.1** Spectrum of injury from cardiac oxygen deprivation

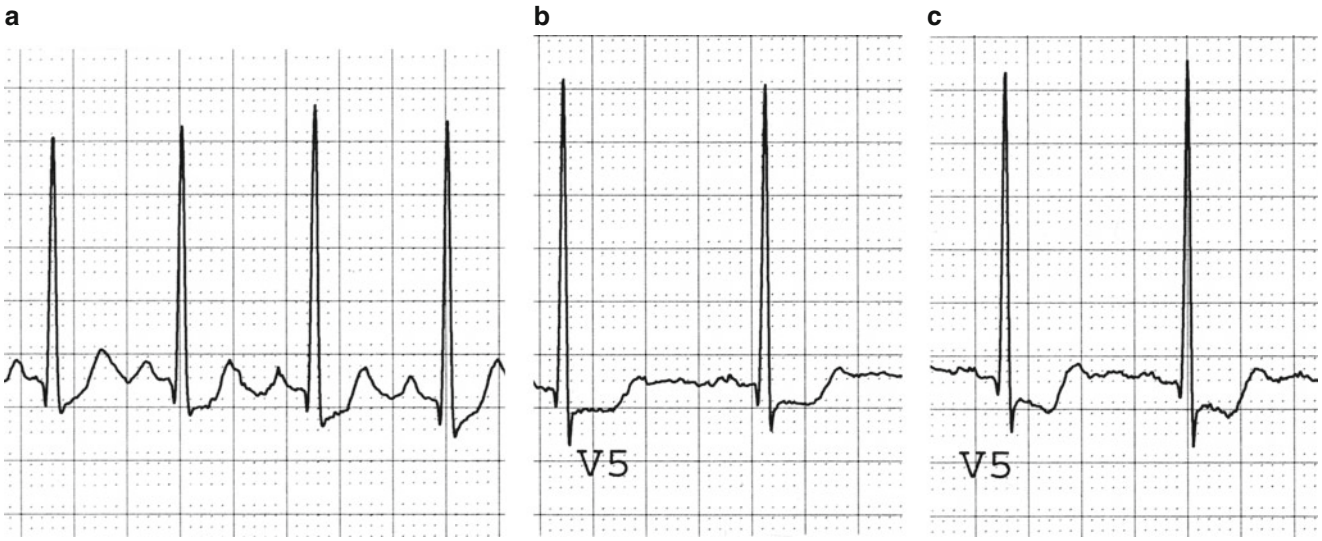



**Fig. 3.2** Ischemia. (**a**) Upsloping ST segment depression. (**b**) Horizontal ST segment depression. (**c**) Downsloping ST segment depression

**Table 3.1** Designations of myocardial infarctions

| Transmural | Subendocardial |
|---|---|
| Q wave | Non-Q wave |
| ST elevation | Non-ST elevation |

the subendocardium, but instead may be transmural, based on anatomical correlations [1]. In general, an anatomical transmural infarction associated with the electrocardiographic changes characteristic of "subendocardial" infarction is smaller in magnitude than a transmural infarction with typical transmural changes on the EKG.

Because of the discordance between anatomical findings and the electrocardiographic changes indicating "transmural" or "subendocardial" infarction, the terminology for infarctions changed to first "Q wave," then "ST elevation" myocardial infarction (STEMI) vs. "non-Q wave" or "non-ST elevation" myocardial infarction (non-STEMI), respectively [2]. Under this classification, the anatomical overtones are dropped, but the association of Q wave or ST elevation infarctions with higher in-hospital mortality, reduced ejection fraction, greater evidence of left ventricular failure, and less postinfarction exercise-induced ischemia compared to non-Q wave or non-ST elevation infarctions is similar to the findings in previous studies comparing "transmural" and "non-transmural" or "subendocardial" infarctions, respectively [3]. STEMIs have a higher in-hospital mortality rate than non-STEMI [4]. That non-STEMIs carry a serious prognosis, however, is clear. Reports suggest that the overall long-term mortality of myocardial infarction patients is the same if not greater after non-STEMI compared to STEMI [3, 5–8]. Non-STEMIs probably represent smaller infarctions with a larger area of myocardium still at risk for future infarction [3, 7]. This notion is supported by studies showing a higher frequency of reinfarction in patients with non-STEMIs compared to patients with STEMIs. The reinfarctions frequently occurred within weeks of the initial infarction and carried a poor prognosis [9, 10].

## Subendocardial/Non-Q Wave/Non-ST Elevation Myocardial Infarction

The electrocardiographic change of a non-STEMI is T wave inversion (Fig. 3.3). There is usually little or no depression of the ST segments. Over a period of weeks to months, the acute changes of T wave inversion *may* revert back to normal, with no residual change to indicate a previous infarction. Alternatively, the T wave inversion may persist in some degree indefinitely.

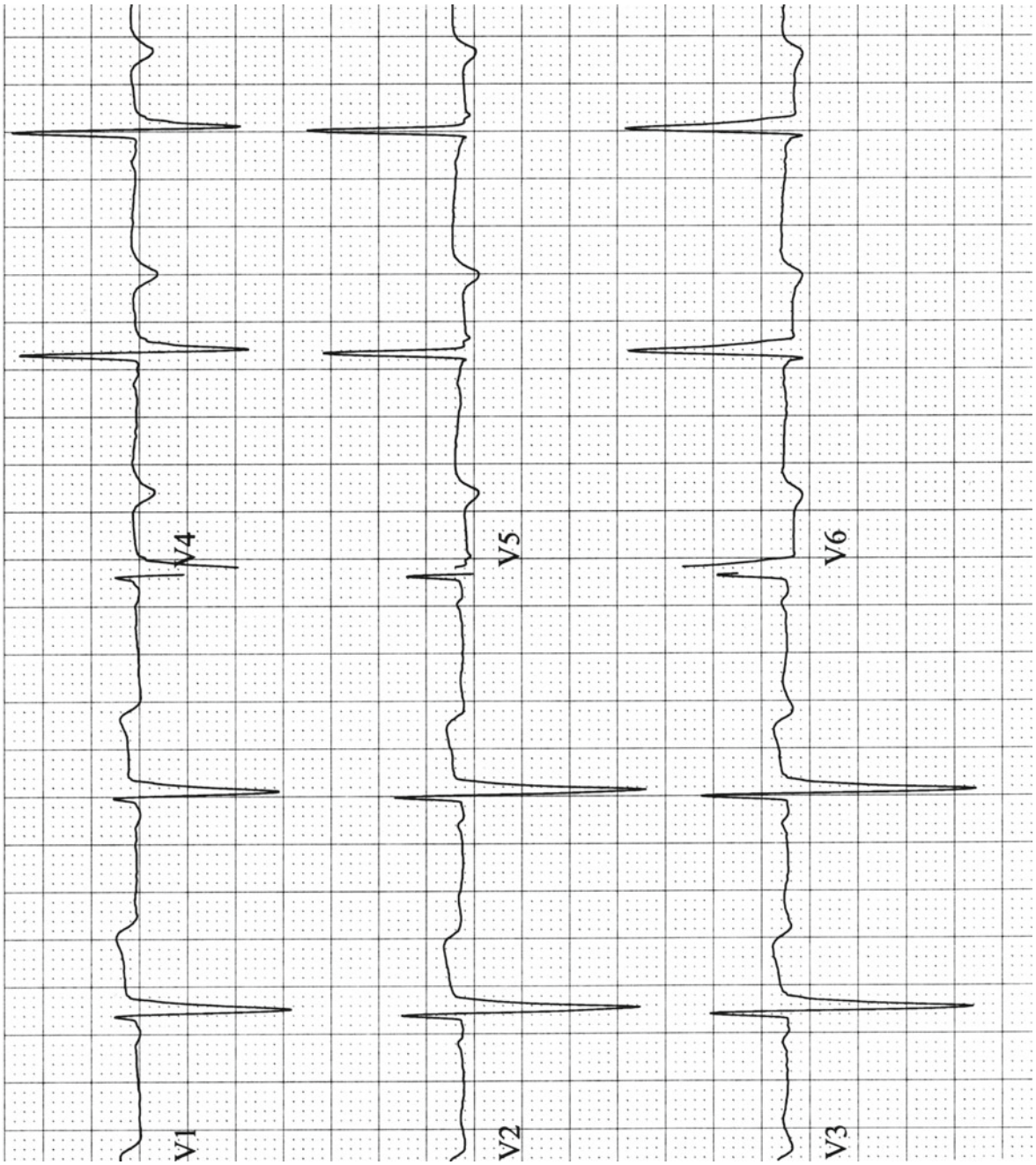



**Fig. 3.3** Subendocardial infarction. These are the precordial leads in a patient with an acute anterior subendocardial infarction. Note that the ST segments are essentially normal, but the T waves are biphasic in $V_3$ and inverted in $V_{4–6}$

## Transmural/Q Wave/ST Elevation Myocardial Infarction

The electrocardiographic changes associated with an acute STEMI follow a more complicated pattern (Fig. 3.4). The initial change is ST segment elevation, occurring usually within minutes of the interruption of oxygen delivery. The next change is the development of "pathological Q waves," Q waves that are new and are of at least 0.04 s duration. The Q waves appear within several hours of the infarction. Over the next few days, the T waves become inverted and the ST segments become less elevated, usually reverting to normal. Over a period of weeks to months, the T wave inversions may resolve, leaving only the Q waves as the residual manifestation of the infarction. This series of changes is known as the evolution of an acute STEMI. Occasionally, the Q waves that remain are not wide and deep and may be indistinguishable from the q waves that can be found as a normal variant. Thus, following either a STEMI or a non-STEMI, it is possible for the EKG to revert to a pattern that is within normal limits and does not reflect the previous infarction. This is more likely to occur with non-STEMI than STEMI, but it can occur with both.

*Very rarely* the first changes on the EKG seen with an acute STEMI are peaked T waves, the so-called hyperacute T waves. This is so rare as to only deserve mention for completeness. It is *not* a common finding, and when it does occur, it is soon supplanted by the far more typical ST segment elevations described earlier.

Sometimes patients are seen with myocardial infarction, suspected by the patient's history and confirmed by a typical and significant elevation of cardiac enzymes, but without any electrocardiographic abnormalities [2]. These "electrically silent" or "normal electrocardiographic" infarctions are uncommon.

## Location of Infarction/Ischemia

There are three general EKG locations for infarctions or ischemia: inferior, anterior, and posterior. The portion of the heart that is affected by infarction or ischemia may be suspected by the electrocardiographic leads in which the changes are found. Even if the correlation is imperfect between the leads reflecting ischemia or infarction and the part of the heart affected, the designation of the location quickly identifies the leads showing changes. As discussed in Chap. 2, leads II, III, and aVF are positive in the legs and negative in the arms, and therefore are most reflective of changes in the inferior wall of the heart and are called the inferior leads. Leads I and aVL, along with the precordial leads, reflect changes in the anterior wall of the heart. Therefore, when there is ischemia or infarction in the inferior wall of the heart, the electrocardiographic changes described earlier in this chapter are typically seen in the inferior leads—II, III, and aVF. When there is anterior wall ischemia or infarction, the changes are typically seen in leads I, aVL, and the precordial leads. Occasionally a distinction is made between the anteroseptal and anterolateral portions of the anterior wall. "Anteroseptal" and "anterolateral" are simply subsegments of the anterior wall, with changes limited to $V_{1–3}$ or $V_{4–6}$, respectively. I believe this subdivision of the anterior wall in reference to myocardial injury is of little practical importance, but an anteroseptal infarction with precordial changes limited to $V_{1–3}$ probably does suggest a less extensive infarction than when the changes are seen in most or all of the precordial leads. The posterior wall of the heart is not reflected directly by any of the standard electrocardiographic leads because none of them are placed on the patient's back. Instead, posterior wall injury is indirectly reflected by changes in leads $V_{1–2}$. Since those two leads are directly anterior, they best show changes affecting the posterior wall of the heart, but the changes are reversed from the usual changes of damage described above. An acute posterior STEMI is reflected by ST segment depression and a tall R wave in $V_{1–2}$ as opposed to ST segment elevation and a Q wave. This is because $V_{1–2}$ are anterior leads, and in reflecting posterior wall injury these leads would be expected to have opposite changes compared to those seen when the area of injury is directly below the leads. Another way to look at this situation is that the complexes in $V_{1–2}$ can be rotated around the baseline as an axis when considering the posterior wall of the heart, so ST segment depression becomes ST segment elevation, and the R wave becomes a Q wave in posterior infarction (Fig. 3.5). The presence of ST segment depression in $V_{1–4}$ in association with changes of acute inferior STEMI is usually due to inferoposterior or posterolateral wall involvement of the infarction rather than a "reciprocal change" [11] (see below).

Even though the area of the heart damaged in an infarction can be suggested on the EKG, ascribing the event to the coronary artery involved is less predictable than identifying the area of the heart that is damaged. *Generally*, the right coronary artery serves the inferior wall of the heart, the left anterior descending serves the anterior wall, and the circumflex serves the posterior wall with minor contributions to the inferior or lateral wall. Thus, in the setting of an inferior infarction, the right coronary artery is probably involved, while in an anterior infarction the left anterior descending artery is probably involved [12]. The common conduction problems with those infarctions can be predicted because the right coronary artery generally supplies the inferior wall of the heart and frequently sends a branch to the atrioventricular (AV) node, while the left anterior descending artery, usually involved in an anterior infarction, sends "septal perforator" arteries into the interventricular septum where the bundle of His and the bundle branches are located (Fig. 3.6). Thus, inferior infarctions are more frequently associated with AV node dysfunction, while anterior infarctions are more typically associated with dysfunction of the bundle system.

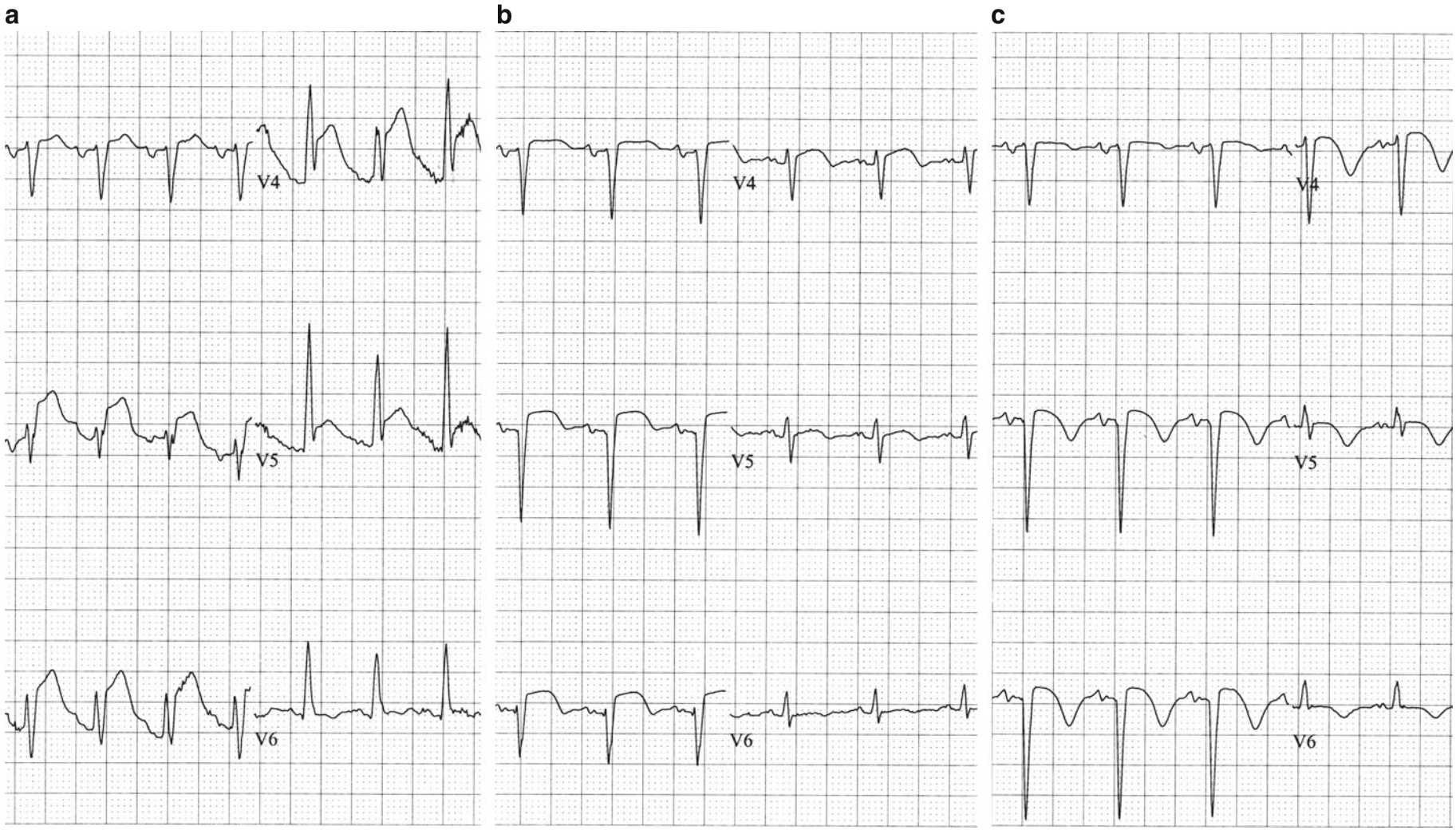



**Fig. 3.4** Evolution of a transmural infarction. Precordial leads in a patient with an acute anterior transmural myocardial infarction. (**a**) At initial presentation the patient has acute ST segment elevation, only a small, nonpathological q wave in $V_{5–6}$, and no T wave inversions. (**b**) Four hours later there is a new Q wave in $V_2$ and less ST elevation remains. (**c**) One day after initial tracing the Q waves are in $V_{2-3}$, slight ST elevation persists, and the T waves are now inverted in $V_{2–6}$

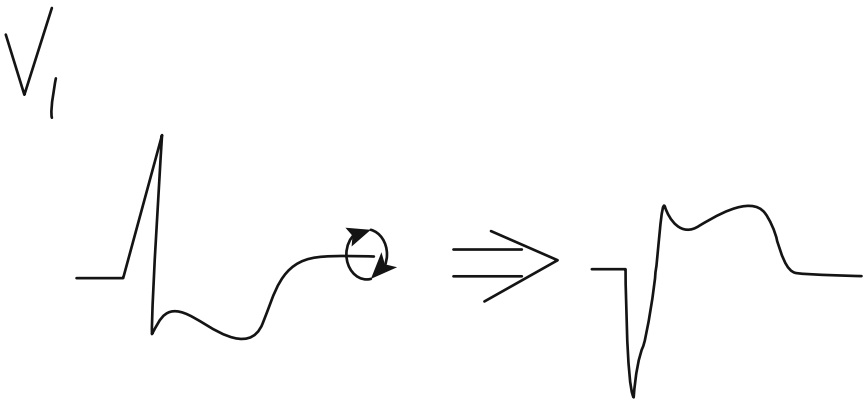



**Fig. 3.5** Posterior infarction (diagrammatic). Lead $V_1$ has a tall R wave and ST depression. When the complex is rotated around the baseline as an axis, the R wave becomes a Q wave and the ST segment depression becomes ST segment elevation, now typical of an acute transmural myocardial infarction

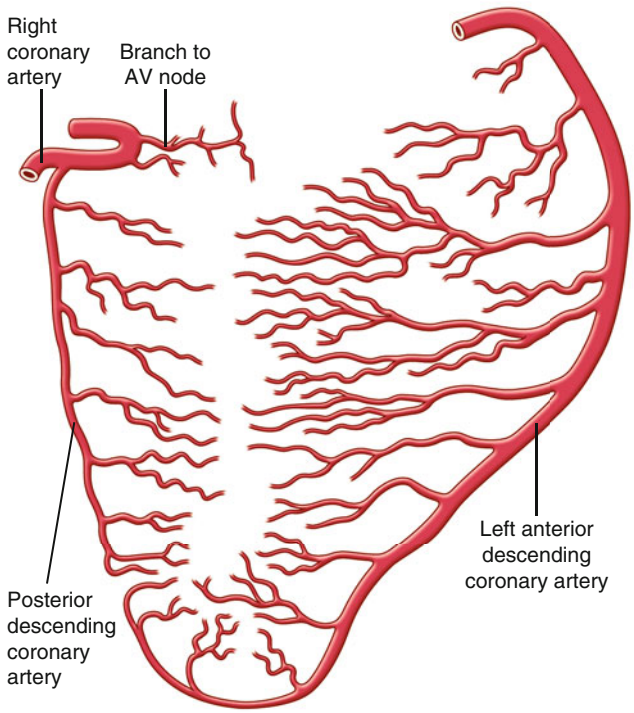



**Fig. 3.6** Blood supply to the conduction system. The right coronary artery gives off a branch to the AV node, while the septal perforators off the left anterior descending artery are primarily responsible for blood supply to the interventricular system where the bundle system is located

## Reciprocal Changes

Reciprocal changes are seen with STEMIs, never non-STEMIs, and are sometimes present with Prinzmetal's angina (see below). Reciprocal changes consist of ST segment depressions (reminiscent of ischemic changes) in leads "opposite" from those that show the typical ST segment elevations described above for STEMIs (sometime called "injury currents").

For example, in inferior STEMIs, with ST segment elevations in II, III, and aVF, there may be ST segment depressions as reciprocal changes in leads I and aVL, and perhaps in some precordial leads. On the other hand, in anterior STEMIs, with ST elevations in I, aVL and some precordial leads, there may be ST depressions in II, III, and aVF as reciprocal changes (Fig. 3.7).

Reciprocal changes are primarily observed in the limb leads and less often in the precordial leads and are seen more often in inferior than anterior infarctions [13, 14]. The degree of ST depression as a reciprocal change is proportional to the magnitude of ST elevation seen as an acute change in STEMIs [15]. Reciprocal changes are seen in approximately 50% of STEMIs, and their presence is associated with a more extensive infarction than when they are absent [16]. Accordingly, the prognosis for patients who have transmural infarction associated with reciprocal change is worse than that for patients whose infarctions are not associated with reciprocal change. The term "reciprocal change" implies secondary electrical change in the leads "opposite" those showing the primary acute injury, rather than ischemia in another part of the heart in addition to the acute infarction [14–16].

## ST Elevation: Differential Diagnosis

Several conditions can cause ST segment elevation. To this point we have covered three such conditions: (1) acute STEMI, (2) Prinzmetal's angina, and (3) junctional ST segment elevation/early repolarization (Fig. 3.8 and Chap. 1). While most cases of Prinzmetal's angina are "idiopathic," hypomagnesemia has been reported to cause spasm of coronary arteries [17, 18]. There are two other conditions that can lead to ST segment elevation: (1) acute pericarditis, and (2) ventricular aneurysm.

Acute pericarditis is typically associated with ST segment elevation that resembles early repolarization. But the ST elevations are diffusely present in almost all the leads, not just in an inferior or anterior distribution. Additionally, there may be PR segment depression but there are never reciprocal changes with acute pericarditis.

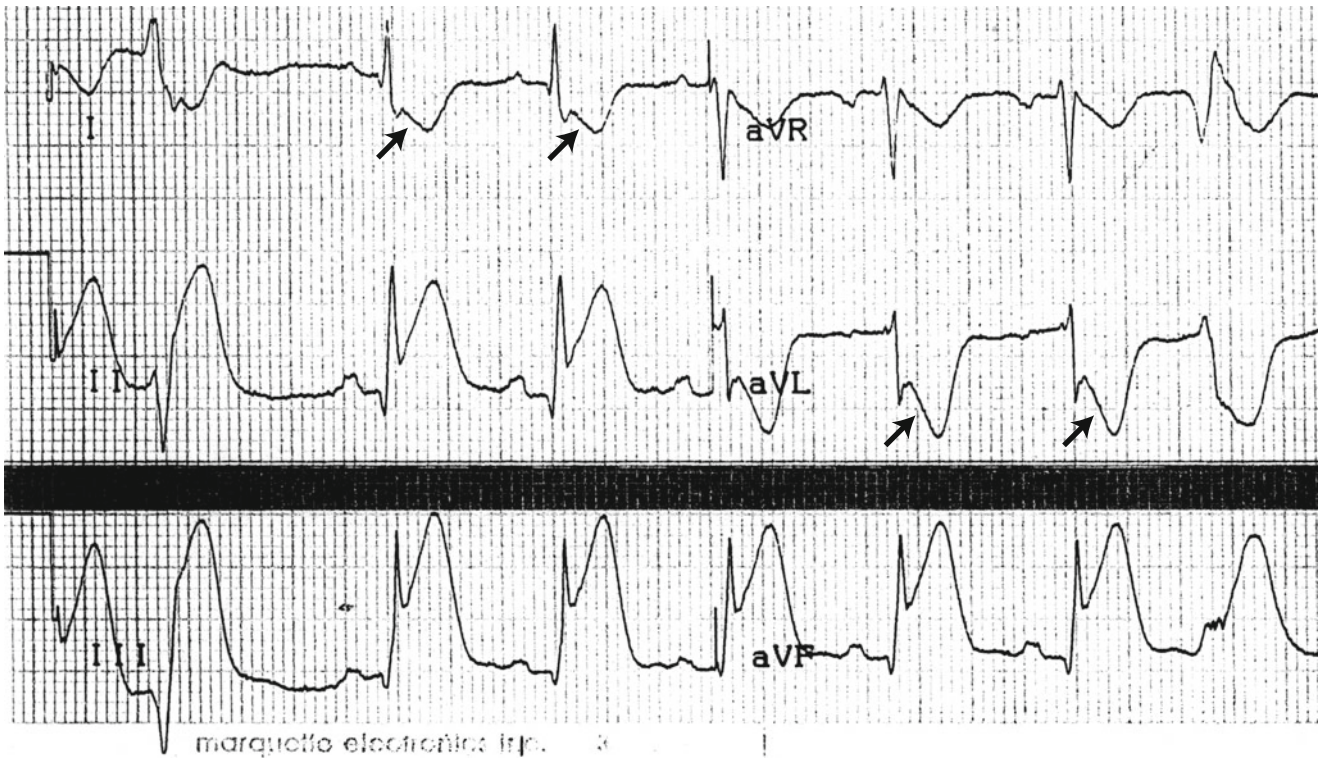



**Fig. 3.7** Reciprocal changes. These are the limb leads of a patient with an acute inferior transmural myocardial infarction. There is marked ST segment elevation in II, III, and aVF, but reciprocal ST segment depression in leads I and aVL (*arrows*)

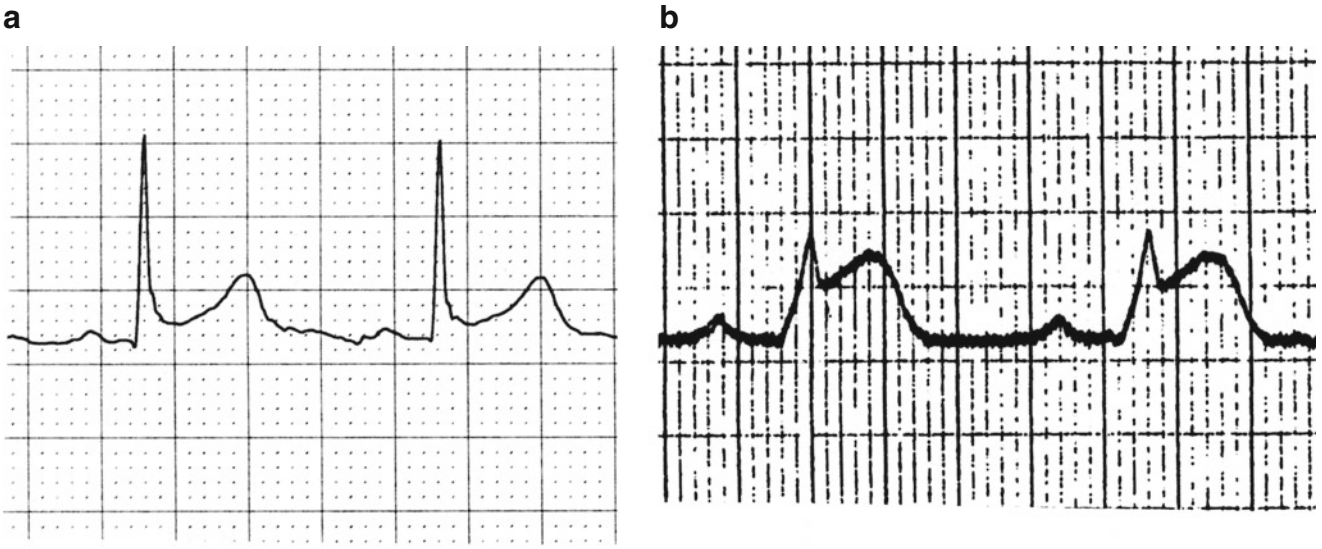
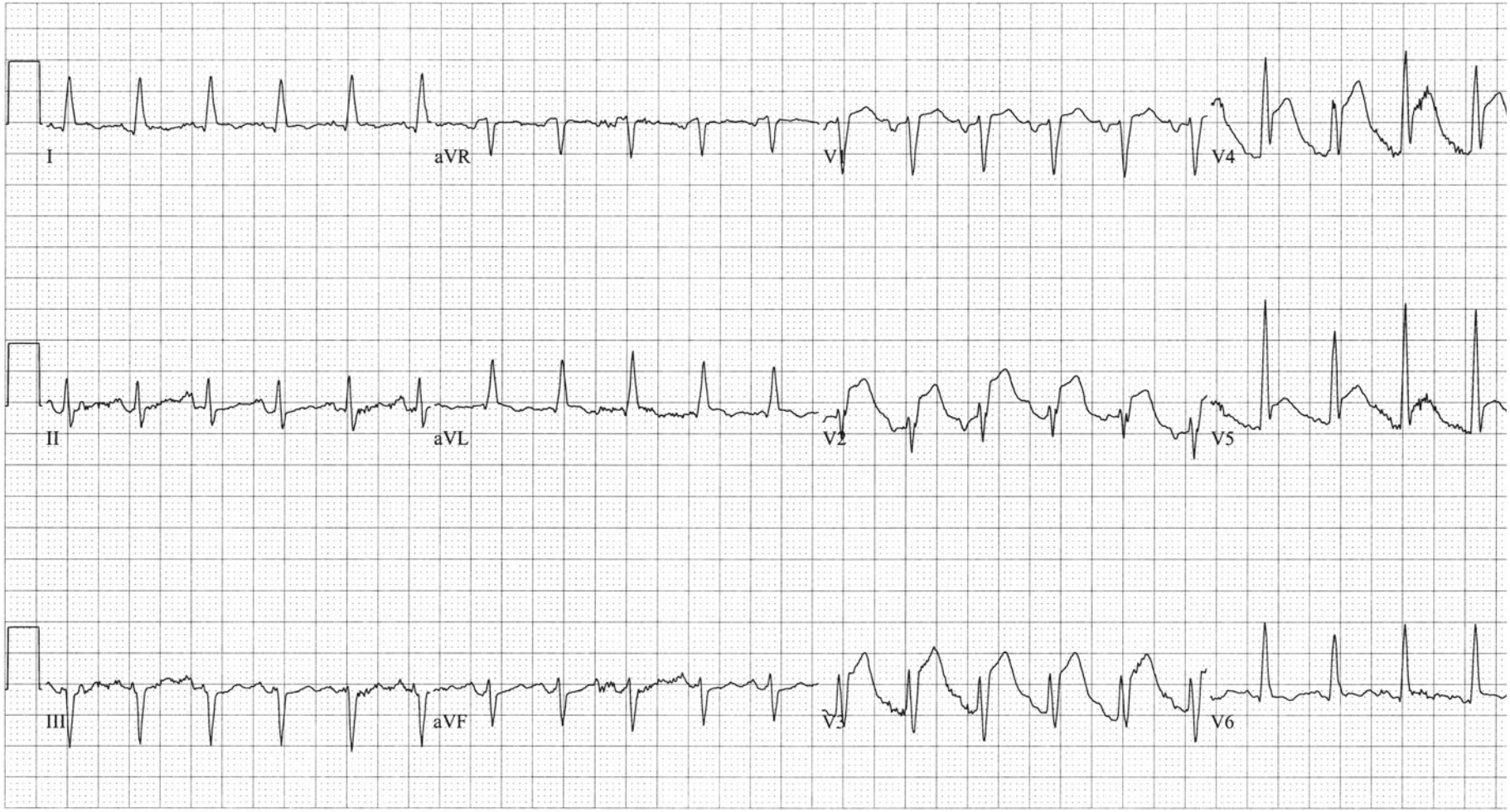



**Fig. 3.8** Early repolarization (**a**) vs. acute transmural myocardial infarction (**b**). Note the sharp angle where the ST segment comes off the QRS complex with the infarction, while early repolarization has a smoothly curved ST segment

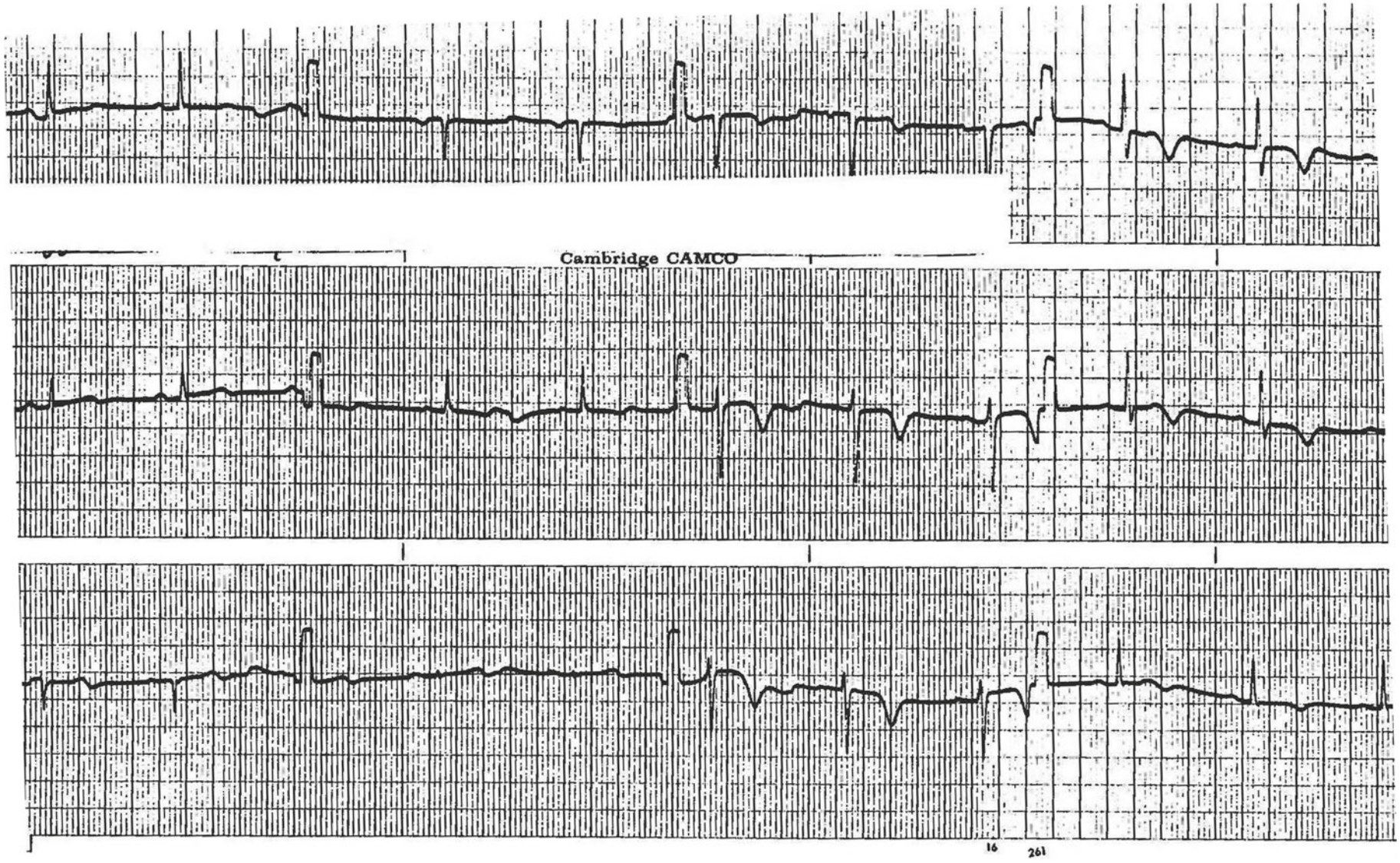
Ventricular aneurysms are usually due to previous STEMI, most commonly involving the anterior wall of the heart. The ST segments are typically elevated in the same leads associated with the previous STEMI and where the current ventricular aneurysm is located. It appears as if the acute ST elevations of a STEMI never resolved, in contrast to the usual evolution of myocardial infarction. So when ST segment elevations do not resolve following an acute STEMI, one should suspect the development of a ventricular aneurysm.

## Exercise Tracings

### Exercise Tracing 3.1



**Exercise Tracing 3.2**



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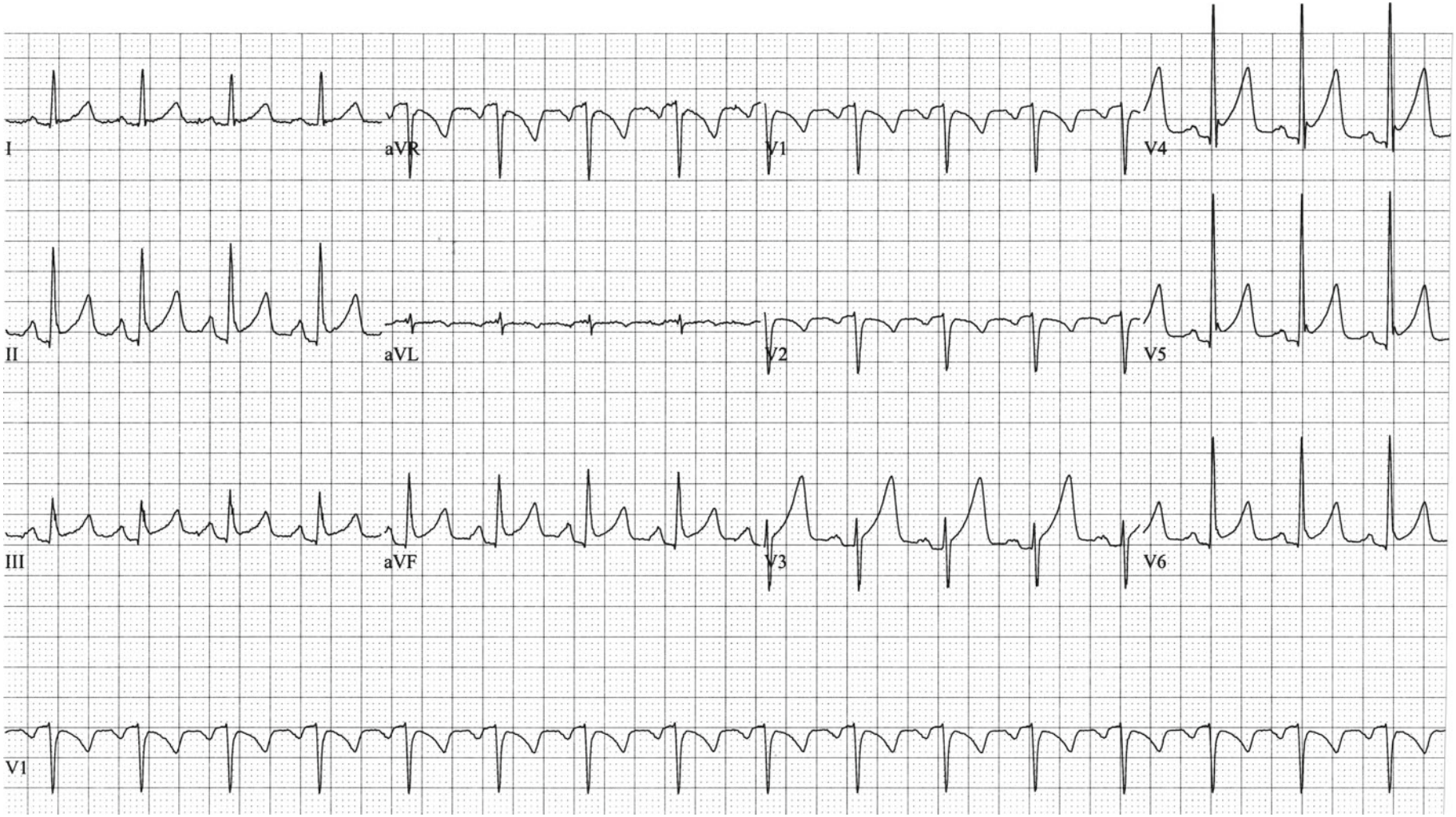

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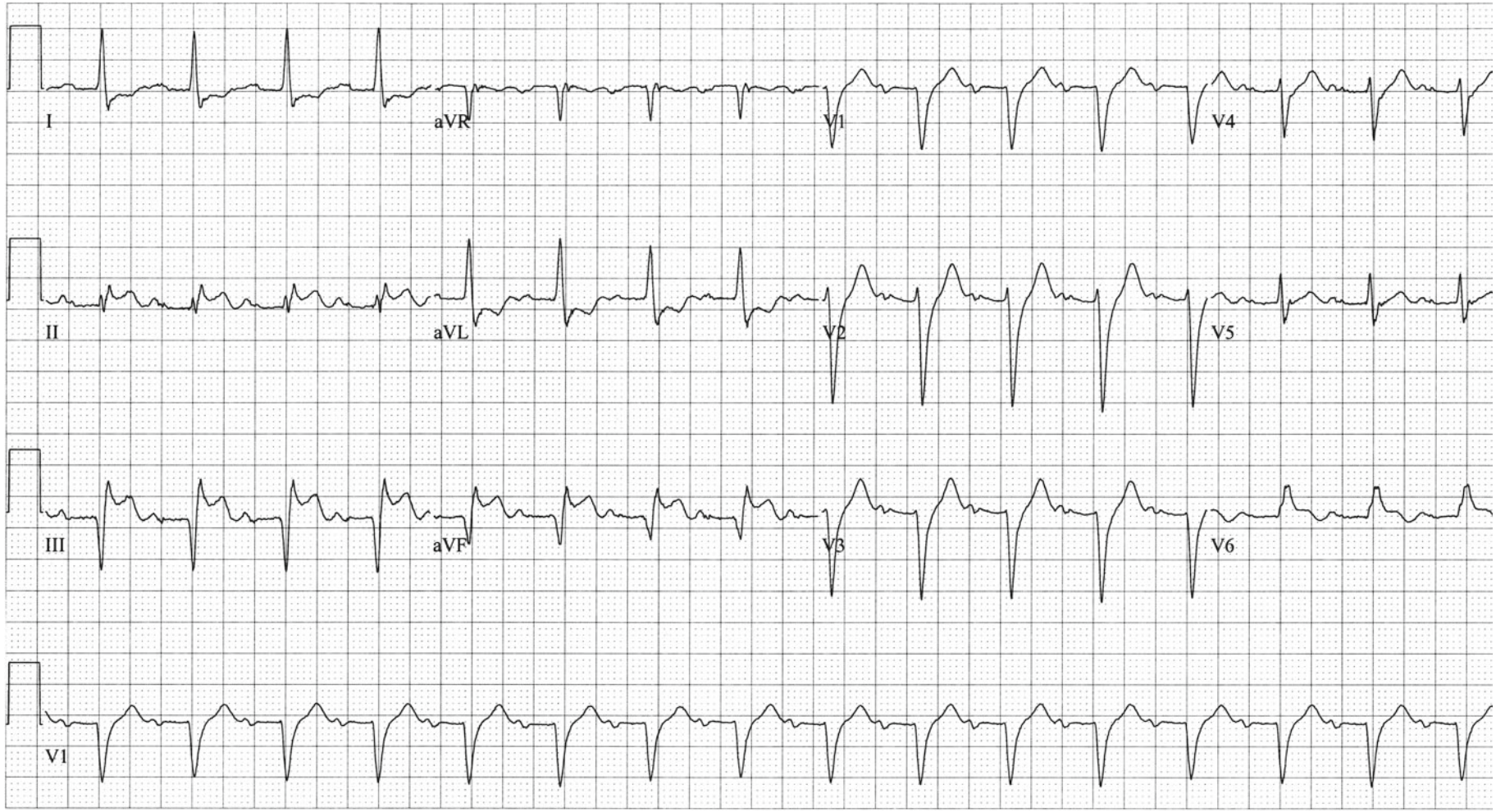
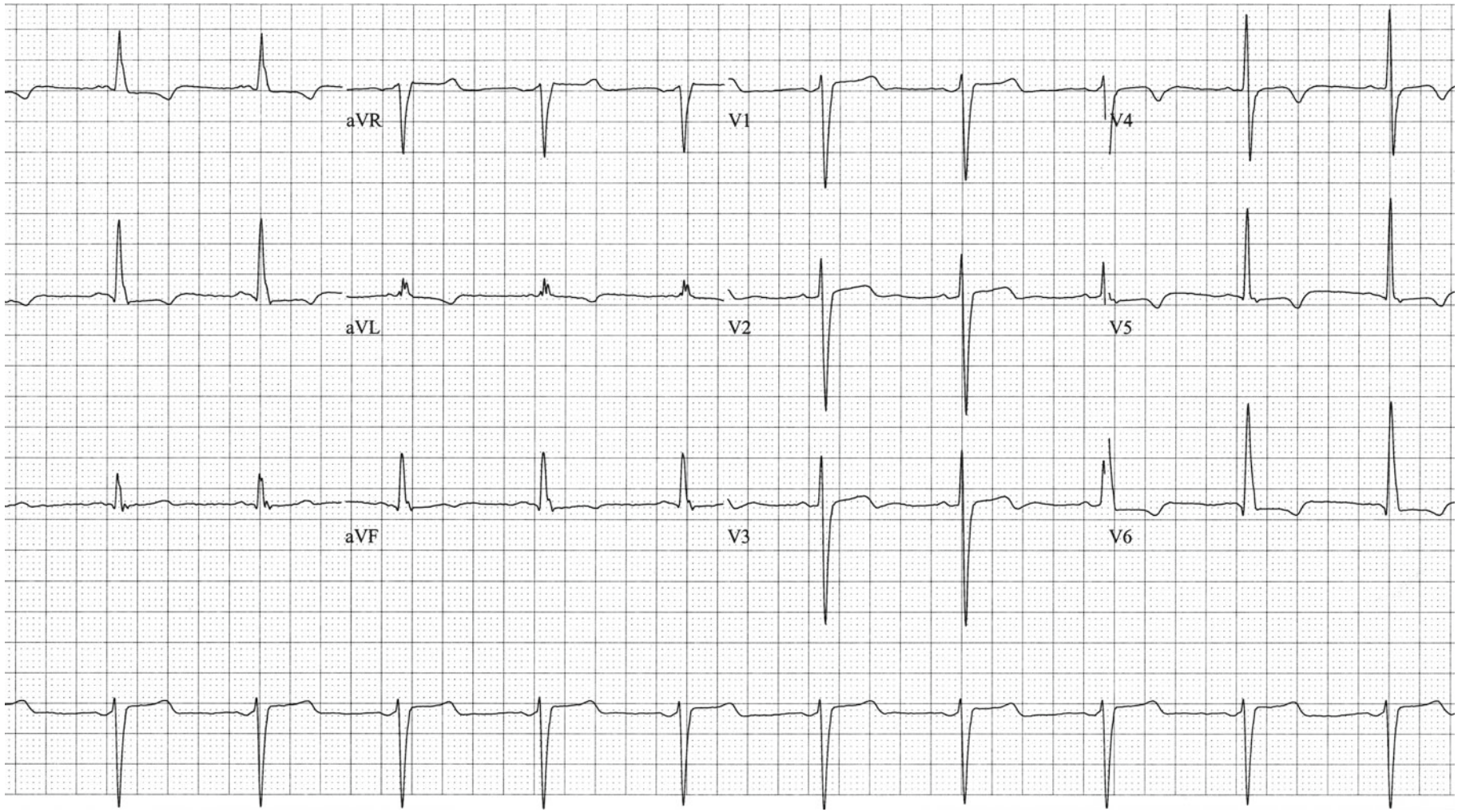


**Exercise Tracing 3.5**



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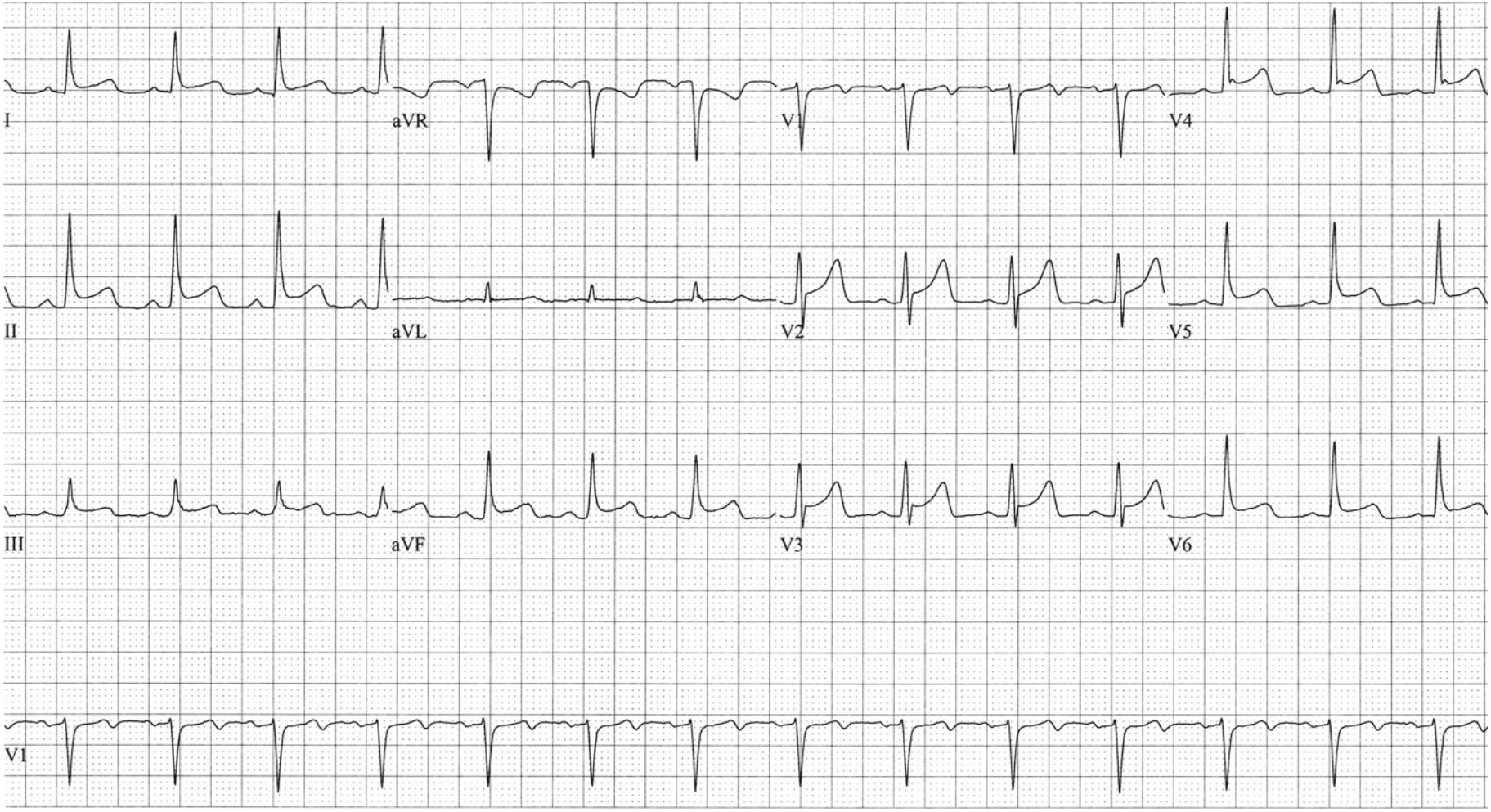
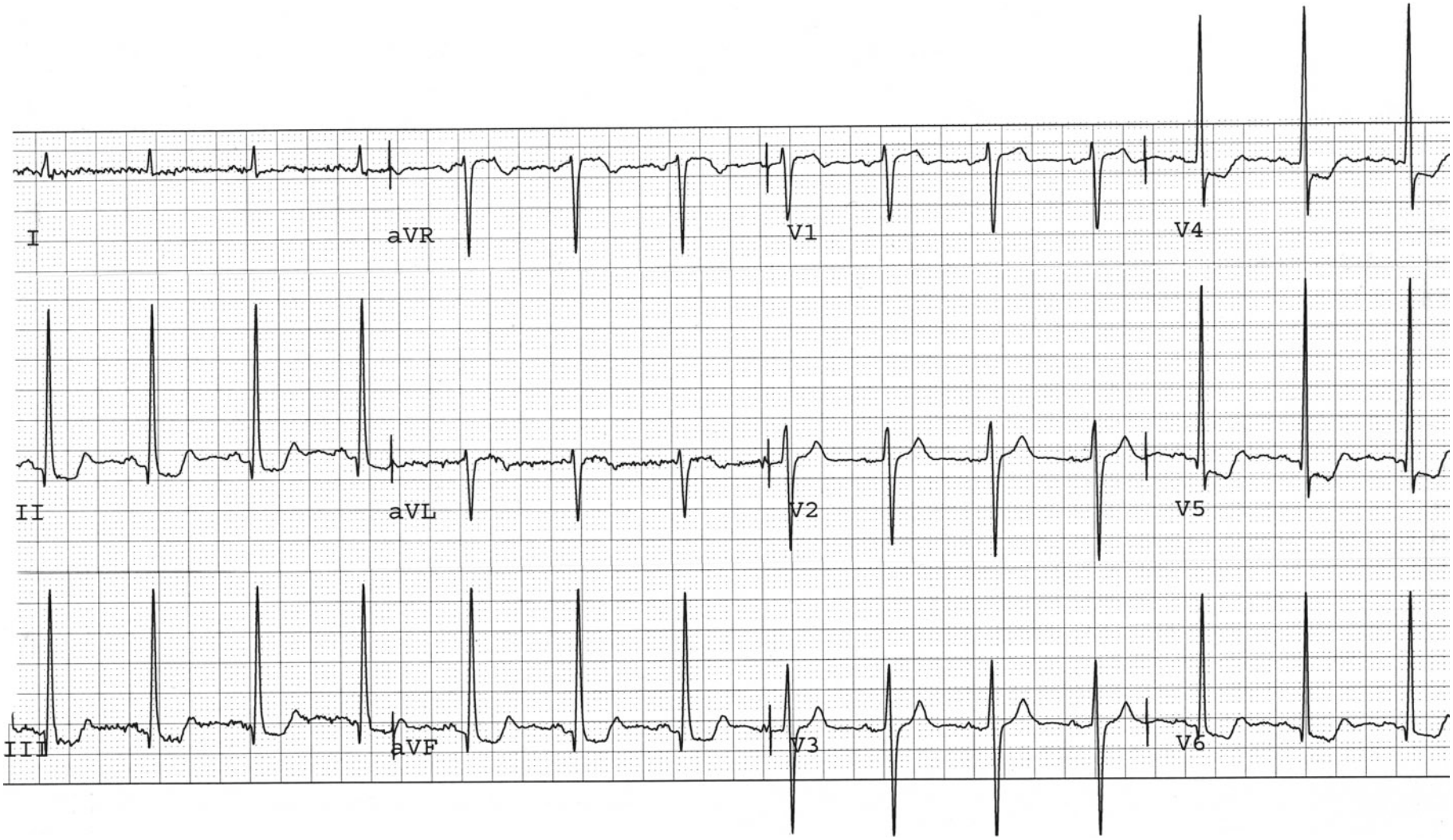


**Exercise Tracing 3.7**



## Interpretations of Exercise Tracings

| Exercise Tracing 3.1 | |
|---|---|
| RATE: | A 130 V 130 |
| RHYTHM: | Sinus tachycardia |
| AXIS: | –20° |
| INTERVALS: | PR 0.15 QRS 0.10 QT 0.30 |
| WAVEFORM: | ST elevation in V1-V5; inverted T waves in I, aVL |
| SUMMARY: | Abnormal due to acute anterior wall ST elevation myocardial infarction, leftward axis deviation |

| Exercise Tracing 3.2 | |
|---|---|
| RATE: | A 64 V 64 |
| RHYTHM: | Normal sinus rhythm |
| AXIS: | 0° |
| INTERVALS: | PR 0.16 QRS 0.08 QT 0.38 |
| WAVEFORM: | T wave biphasic in I, aVL, inverted in $V_{2–6}$ |
| SUMMARY: | Abnormal due to acute subendocardial anterior myocardial infarction |

| Exercise Tracing 3.3 | |
|---|---|
| RATE: | A 102 V 102 |
| RHYTHM: | Sinus tachycardia |
| AXIS: | +60° |
| INTERVALS: | PR 0.16 QRS 0.07 QT 0.34 |
| WAVEFORM: | Diffuse ST elevation; PR segment depression in the inferior leads, especially lead II; inverted P waves in $V_{1-2}$ |
| SUMMARY: | Abnormal due to ST changes and PR depression compatible with pericarditis; left atrial abnormality |

| Exercise Tracing 3.4 | |
|---|---|
| RATE: | A 105 V 105 |
| RHYTHM: | Sinus tachycardia |
| AXIS: | 0° |
| INTERVALS: | PR 0.25 QRS 0.10 QT 0.30 |
| WAVEFORM: | ST elevation in II, III, aVF; ST depression in I and aVL; Q III, aVF, Q in III, aVF |
| SUMMARY: | Abnormal due to acute inferior ST elevation myocardial infarction with reciprocal changes, first degree AV block (see Chap. 4)" |

| Exercise Tracing 3.5 | |
|---|---|
| RATE: | A 65 V 65 |
| RHYTHM: | Normal sinus rhythm |
| AXIS: | 50° |
| INTERVALS: | PR 0.15 QRS 0.11 QT 0.44 |
| WAVEFORM: | T wave inversions in I, II, aVL, $V_{4–6}$; non-pathological q waves in II, III, aVF, $V_{5–6}$ |
| SUMMARY: | Abnormal due to acute subendocardial anterolateral myocardial infarction, inferior infarction, left axis deviation, prolonged QT interval and U waves subendocardial myocardial infarction |

| Exercise Tracing 3.6 | |
|---|---|
| RATE: | A 86 V 86 |
| RHYTHM: | Normal sinus rhythm |
| AXIS: | +50° |
| INTERVALS: | PR 0.16 QRS 0.09 QT 0.36 |
| WAVEFORM: | Smooth ST elevation I, II, II, aVF, $V_{2–6}$; early R wave development $V_{2–3}$ |
| SUMMARY: | Abnormal due to acute pericarditis |

| Exercise Tracing 3.7 | |
|---|---|
| RATE: | A 88 V 88 |
| RHYTHM: | Normal sinus rhythm |
| AXIS: | +80° |
| INTERVALS: | PR 0.16 QRS 0.09 QT 0.35 |
| WAVEFORM: | ST depression II, III, aVF, $V_{4–6}$; tall R waves lead II and $V_5$ |
| SUMMARY: | Abnormal due to inferolateral ischemia [NOTE: Even though this EKG meets the criteria for left ventricular hypertrophy (see Chap. 6), these ST segment depressions were quickly reversible, which confirms the diagnosis of ischemia] |

## References


1. Freifeld AG, Shuster EH, Bulkley BH. Nontransmural versus transmural myocardial infarction: a morphological study. Am J Med. 1983;75:423–32.
2. Zema MJ. Q wave, S-T segment, and T wave myocardial infarction. Am J Med. 1985;78:391–8.
3. Hutter Jr AM, DeSanctis RW, Flynn T, Yeatman LA. Nontransmural myocardial infarction: a comparison of hospital and late clinical course of patients with that of matched patients with transmural anterior and transmural inferior myocardial infarction. Am J Cardiol. 1981;48:595–602.
4. Chin CT, Chen AY, Wang TY, Alexander KP, Mathews R, Rumsfeld JS, et al. Risk adjustment for in-hospital mortality of contemporary patients with acute myocardial infarction: the Acute Coronary Treatment and Interventions Outcome Network (ACTION) Registry®—Get With The Guidelines (GWTG)™ acute myocardial infarction mortality model and risk score. Am Heart J. 2011;161:113–22.
5. Connolly DC, Elveback LR. Coronary heart disease in residents of Rochester, Minnesota. VI. Hospital and post hospital course of patients with transmural and subendocardial myocardial infarction. Mayo Clin Proc. 1985;60:375–81.
6. Cannom DS, Levy W, Cohen LS. The short- and long-term prognosis of patients with transmural and nontransmural myocardial infarction. Am J Med. 1976;61:452–8.
7. Furman MI, Dauerman HL, Goldberg RJ, Yarzebski J, Lessard D, Gore JM. Twenty-two year (1975-1997) trends in the incidence, in-hospital and long-term case fatality rates from initial Q-wave and non-Q-wave myocardial infarction: a multi-hospital, community-wide perspective. J Am Coll Cardiol. 2001;37:1571.
8. Montalescot G, Dallongeville J, Van Belle E, Rouanet S, Baulac C, Degrandsart A, et al. STEMI and NSTEMI: are they so different? 1 year outcomes in acute myocardial infarction as defined by the ESC/ACC definition (the OPERA registry). Eur Heart J. 2007;28:1409–17.
9. Hollander G, Ozick H, Greengart A, Shani J, Lichstein E. High mortality early reinfarction with first non-transmural myocardial infarction. Am Heart J. 1984;108:1412–6.

10. Marmor A, Sobel BE, Roberts R. Factors presaging early recurrent myocardial infarction ("extension"). Am J Cardiol. 1981;48:603–10.
11. Goldberg HL, Borer JS, Jacobstein JG, Kluger J, Scheidt SS, Alonso DR. Anterior S-T segment depression in acute inferior myocardial infarction: indicator of posterolateral infarction. Am J Cardiol. 1981;48:1009–15.
12. Blanke H, Cohen M, Schlueter GU, Karsch KR, Rentrop KP. Electrocardiographic and coronary arteriographic correlations during acute myocardial infarctions. Am J Cardiol. 1984;54:249–55.
13. Dewhurst NG, Muir AL. Clinical significance of "reciprocal" S-T segment depression in acute myocardial infarction: relative contributions of infarct size and ischemia at a distance. Am J Med. 1985;78:765–70.
14. Tzivoni D, Chenzbraun A, Karen A, Benhorin J, Gottlieb S, Lonn E, et al. Reciprocal electrocardiographic changes in acute myocardial infarction. Am J Cardiol. 1985;56:23–6.
15. Gibson RS, Crampton RS, Watson DD, Taylor GJ, Carabello BA, Holt ND, et al. Precordial ST-segment depression during acute inferior myocardial infarction: clinical, scintigraphic and angiographic correlations. Circulation. 1982;66:732–41.
16. Billadello JJ, Smith JL, Ludbrook PA, Tiefenbrunn AJ, Jaffe AS, Sobel BE, et al. Implications of "reciprocal" ST segment depression associated with acute myocardial infarction identified by positron tomography. J Am Coll Cardiol. 1983;2: 616–24.
17. Khardori R, Cohen B, Taylor D, Soler NG. Electrocardiographic finding simulating acute myocardial infarction in a compound metabolic aberration. Am J Med. 1985;78:529–32.
18. Turlapaty PDMV, Altura BM. Magnesium deficiency produces spasm of coronary arteries: relationship to etiology of sudden death ischemic heart disease. Science. 1980;208:198–200.