From a clinical viewpoint, the division of acute myocardial infarction into ST-segment elevation and non-ST elevation types is useful since the efficacy of acute reperfusion therapy is limited to the former group.

The ECG leads are usually more helpful in localizing regions of ST elevation than non-ST elevation ischemia. For example, acute transmural anterior (including apical and lateral) wall ischemia is reflected by ST elevations or increased T-wave positivity in one or more of the precordial leads (V_1–V_6) and leads I and aVL. Inferior wall ischemia produces changes in leads II, III, and aVF. "Posterior" wall ischemia (usually associated with lateral or inferior involvement) may be indirectly recognized by *reciprocal* ST depressions in leads V_1 to V_3 (thus constituting an ST elevation "equivalent" acute coronary syndrome). Right ventricular ischemia usually produces ST elevations in right-sided chest leads (Fig. 268-5). When ischemic ST elevations occur as the earliest sign of acute infarction, they typically are followed within a period ranging from hours to days by evolving T-wave inversions and often by Q waves occurring in the same lead distribution. Reversible transmural ischemia, for example, due to coronary vasospasm (Prinzmetal's variant angina and possibly the Tako-tsubo "stress" cardiomyopathy syndrome), may cause transient ST-segment elevations without development of Q waves, as may very early reperfusion in acute coronary syndromes. Depending on the severity and duration of ischemia, the ST elevations may resolve completely in minutes or be followed by T-wave inversions that persist for hours or even days. Patients with ischemic chest pain who present with deep T-wave inversions in multiple precordial leads (e.g., V_1–V_4, I, and aVL) with or without cardiac enzyme elevations typically have severe obstruction in the left anterior descending coronary artery system (Fig. 268-12). In contrast, patients whose baseline ECG already shows abnormal T-wave inversions may develop T-wave normalization (*pseudonormalization*) during episodes of acute transmural ischemia.

With infarction, depolarization (QRS) changes often accompany repolarization (ST-T) abnormalities. Necrosis of sufficient myocardial tissue may lead to decreased R-wave amplitude or abnormal Q waves (even in the absence of transmurality) in the anterior or inferior leads (Fig. 268-13). Previously, abnormal Q waves were considered markers of transmural myocardial infarction, whereas subendocardial infarcts were thought not to produce Q waves. However, careful ECG-pathology correlative studies have indicated that transmural infarcts may occur without Q waves and that subendocardial (nontransmural) infarcts sometimes may be associated with Q waves. Therefore, infarcts are more appropriately classified as "Q-wave" or "non-Q-wave." The major acute ECG changes in syndromes of ischemic heart disease are summarized schematically in Fig. 268-14. Loss of depolarization forces due to posterior or lateral infarction may cause reciprocal increases in R-wave amplitude in leads V_1 and V_2 without diagnostic Q waves in any of the conventional leads. Atrial infarction may be associated with PR-segment deviations due to an atrial current of injury, changes in P-wave morphology, or atrial arrhythmias. In the weeks and months after infarction, these ECG changes may persist or begin to resolve. Complete normalization of the ECG after Q-wave infarction is uncommon but may occur, particularly with smaller infarcts. In contrast, ST-segment elevations that persist for several weeks or more after a Q-wave infarct usually correlate with a severe underlying wall motion disorder (akinetic or dyskinetic zone), although not necessarily a frank ventricular aneurysm. ECG changes due to ischemia may occur spontaneously or may be provoked by various exercise protocols (stress electrocardiography; Chap. 293).

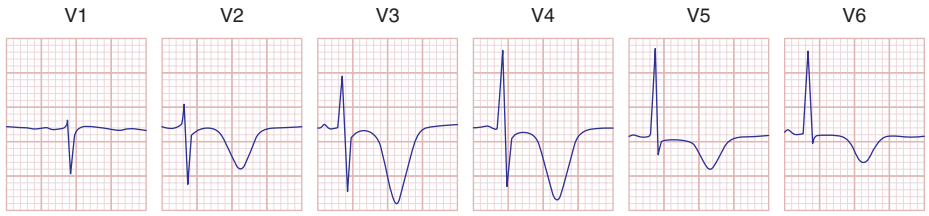

FIGURE 268-12 **Severe anterior wall ischemia** (with or without infarction) may cause prominent T-wave inversions in the precordial leads. This pattern (sometimes referred to as Wellens T waves) is usually associated with a high-grade stenosis of the left anterior descending coronary artery.

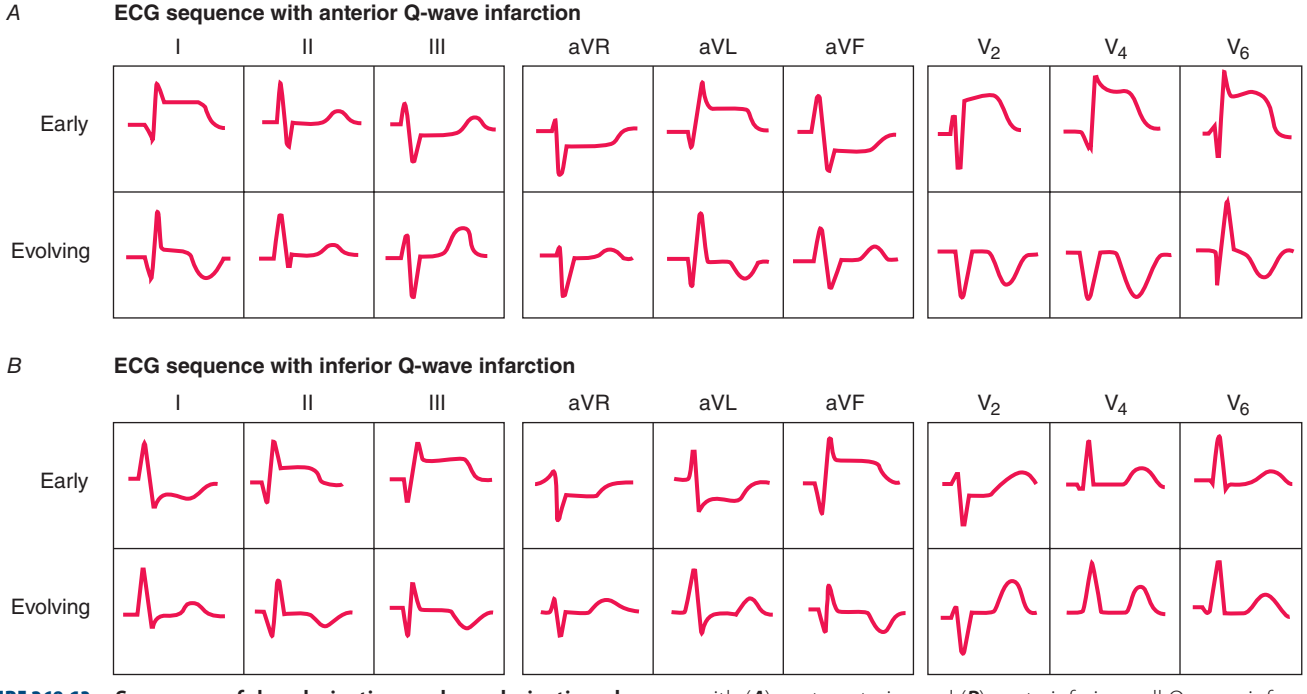

FIGURE 268-13 **Sequence of depolarization and repolarization changes** with (***A***) acute anterior and (***B***) acute inferior wall Q-wave infarctions. With anterior infarcts, ST elevation in leads I and aVL and the precordial leads may be accompanied by reciprocal ST depressions in leads II, III, and aVF. Conversely, acute inferior (or posterolateral) infarcts may be associated with reciprocal ST depressions in leads V_1 to V_3. *(After AL Goldberger et al: Goldberger's Clinical Electrocardiography: A Simplified Approach, 8th ed. Philadelphia, Elsevier/Saunders, 2013.)*