

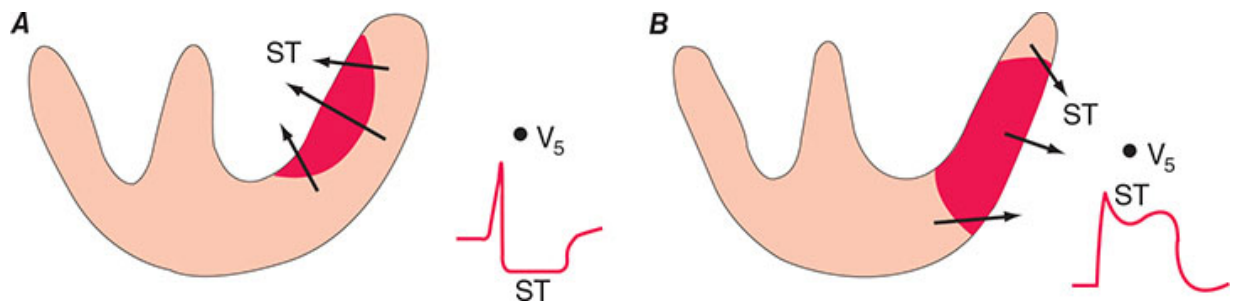


FIGURE 240-11 Acute ischemia causes a current of injury. A. With predominant subendocardial ischemia, the resultant ST vector will be directed toward the inner layer of the affected ventricle and the ventricular cavity. Overlying leads therefore will record ST depression. **B.** With ischemia involving the outer ventricular layer (transmural or epicardial injury), the ST vector will be directed outward. Overlying leads will record ST elevation.

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 (V₁–V₆) and leads I and aVL. Inferior wall ischemia produces changes in leads II, III, and aVF. “Posterior” wall ischemia (almost always associated with lateral or inferior involvement) may be indirectly recognized by *reciprocal* ST depressions in leads V₁ to V₃ (thus constituting an ST elevation “equivalent” acute coronary syndrome). Acute right ventricular ischemia usually produces ST elevations in right-sided chest leads (Fig. 240-5). 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 angina) may cause transient ST-segment elevations without development of Q waves. 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., V₁–V₄, I, and aVL) with or without cardiac enzyme elevations typically have severe