TABLE 4-1 ELECTROCARDIOGRAPHIC MANIFESTATIONS OF ATRIAL ABNORMALITIES AND VENTRICULAR HYPERTROPHY

LEFT ATRIAL ABNORMALITY
P-wave duration ≥0.12 second Notched, slurred P wave in leads I and II Biphasic P wave in lead V_1 with a wide, deep, negative terminal component
RIGHT ATRIAL ABNORMALITY
P-wave duration ≤0.11 second Tall, peaked P waves of ≥2.5 mm in leads II, III, and aVF
LEFT VENTRICULAR HYPERTROPHY
Voltage criteria R wave in lead aVL ≥12 mm R wave in lead I ≥15 mm S wave in lead V_1 or V_2 + R wave in lead V_5 or V_6 ≥35 mm Depressed ST segments with inverted T waves in the lateral leads Left axis deviation QRS duration ≥0.09 second Left atrial enlargement
RIGHT VENTRICULAR HYPERTROPHY
Tall R waves over right precordium (R-to-S ratio in lead V_1 >1.0) Right axis deviation Depressed ST segments with inverted T waves in leads V_1 to V_3 Normal QRS duration (if no right bundle branch block) Right atrial enlargement

TABLE 4-2 ELECTROCARDIOGRAPHIC MANIFESTATIONS OF FASCICULAR AND BUNDLE BRANCH BLOCKS

LEFT ANTERIOR FASCICULAR BLOCK
QRS duration ≤0.1 second Left axis deviation (more negative than −45 degrees) rS pattern in leads II, III, and aVF qR pattern in leads I and aVL
RIGHT POSTERIOR FASCICULAR BLOCK
QRS duration ≤0.1 second Right axis deviation (+90 degrees or greater) qR pattern in leads II, III, and aVF rS pattern in leads I and aVL Exclusion of other causes of right axis deviation (e.g., chronic obstructive pulmonary disease, right ventricular hypertrophy)
LEFT BUNDLE BRANCH BLOCK
QRS duration ≥0.12 second Broad, slurred, or notched R waves in lateral leads (I, aVL, V_5, and V_6) QS or rS pattern in anterior precordium leads (V_1 and V_2) ST-T-wave vectors opposite to terminal QRS vectors
RIGHT BUNDLE BRANCH BLOCK
QRS duration ≥0.12 second Large R′ wave in lead V_1 (rsR′) Deep terminal S wave in lead V_6 Normal septal Q waves Inverted T waves in leads V_1 and V_2

T-wave inversion opposite the QRS deflection (Fig. 4-6A). Given the abnormal sequence of ventricular activation with LBBB, many ECG abnormalities, such as Q-wave MI and left ventricular hypertrophy, are difficult to evaluate. In some cases, acute MI is apparent even with LBBB. An LBBB typically indicates underlying myocardial disease—most commonly fibrosis due to ischemic injury or hypertrophy. With RBBB, the interventricular septum depolarizes normally from left to right, the initial QRS deflection remains unchanged, and ECG abnormalities such as Q-wave MI can still be interpreted. After septal activation, the left ventricle depolarizes, followed by the right ventricle. The ECG is characterized by a wide QRS complex; a large R′ wave in lead V_1 (R-S-R′); and deep S waves in leads I, aVL, and V_6, representing delayed right ventricular activation (see Fig. 4-6B). Although RBBB may be associated with underlying cardiac disease, it may also appear as a normal variant or be seen intermittently when heart rate is elevated. In the latter case, it is referred to as *rate-related bundle branch block.*

Myocardial Ischemia and Infarction

Myocardial ischemia and MI may be associated with abnormalities of the ST segment, T wave, and QRS complex. Myocardial ischemia primarily affects repolarization of the myocardium and is often associated with horizontal or down-sloping ST-segment depression and T-wave inversion. These changes may be transient, such as during an anginal episode or an exercise stress test, or they may be long-lasting in the setting of unstable angina or MI. T-wave inversion without ST-segment depression is a nonspecific finding and must be correlated with the clinical findings. Localized ST-segment elevation suggests more extensive myocardial injury and is often associated with acute MI (Fig. 4-7). Vasospastic or Prinzmetal angina may be associated with reversible ST-segment elevation without MI. ST-segment elevation may occur in other settings not related to acute ischemia or infarction. Persistent, localized ST-segment elevation in the same leads as pathologic Q waves is consistent with a ventricular aneurysm. Acute pericarditis is associated with diffuse ST-segment elevation and PR depression. Diffuse J-point elevation in association with upward-coving ST segments is a normal variant common among young men and is often referred to as *early repolarization.*

A Q wave is one of the criteria used to diagnose MI. Infarcted myocardium is unable to conduct electrical activity, and electrical forces are directed away from the surface electrode overlying the infarcted region, producing a Q wave on the surface ECG. Knowing which region of the myocardium each lead represents enables the examiner to localize the area of infarction (Table 4-3). A pathologic Q wave has a duration greater than or equal to 0.04 second or a depth one fourth or more of the height of the corresponding R wave.

Not all MIs result in the formation of Q waves. Small R waves can return many weeks to months after an MI.

Abnormal Q waves, or *pseudoinfarction,* may be associated with nonischemic cardiac disease, such as ventricular preexcitation, cardiac amyloidosis, sarcoidosis, idiopathic or hypertrophic cardiomyopathy, myocarditis, and chronic lung disease.

Abnormalities of the ST Segment and T Wave

A number of drugs and metabolic abnormalities may affect the ST segment and T wave (Fig. 4-8). Hypokalemia may result in prominent U waves in the precordial leads and prolongation of the QT interval. Hyperkalemia may result in tall, peaked T waves. Hypocalcemia typically lengthens the QT interval, whereas hypercalcemia shortens it. A commonly used cardiac medication, digoxin, often results in diffuse, scooped ST-segment depression. Minor or *nonspecific* ST-segment and T-wave abnormalities may occur in many patients and have no definable cause. In these