This chapter and the next (forming one unit) examine one of the most important topics in clinical electrocardiography and clinical medicine—the diagnosis of myocardial ischemia and infarction* (ischemic heart disease), including ST segment elevation myocardial infarction (STEMI). Basic terms and concepts are briefly discussed first.

**MYOCARDIAL ISCHEMIA**

Myocardial cells require oxygen and other nutrients to function. Oxygenated blood is supplied by the coronary arteries. If severe narrowing or complete blockage of a coronary artery causes the blood flow to become inadequate, ischemia of the heart muscle develops. Ischemia means literally “to hold back blood.”

Myocardial ischemia may occur transiently. For example, patients who experience angina pectoris with exercise are having transient myocardial ischemia. If the ischemia is more severe, necrosis of a portion of heart muscle may occur. Myocardial infarction (MI) refers to myocardial necrosis (or, in nontechnical parlance—a “heart attack”).

This discussion focuses primarily on ischemia and infarction of the left ventricle, the predominant pumping chamber of the heart. The important clinical topic of right ventricular infarction is also discussed briefly.

**TRANSMURAL AND SUBENDOCARDIAL ISCHEMIA**

A simplified cross-sectional diagram of the left ventricle is presented in Figure 8-1. Notice that the left ventricle consists of an outer layer (epicardium or subepicardium) and an inner layer (subendocardium). This distinction is important because myocardial ischemia may be limited to just the inner layer, or it may affect virtually the entire thickness of the ventricular wall (transmural ischemia).

**MYOCARDIAL BLOOD SUPPLY**

The cardiac blood supply is delivered by the three main coronary arteries and their branches (Fig. 8-2). The right coronary artery supplies both the inferior (diaphragmatic) portion of the heart and the right ventricle. The left main coronary artery is

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*The terms infarction and infarct are used interchangeably in this book and clinically.

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short and divides into the left anterior descending coronary artery, which generally supplies the ventricular septum and a large part of the left ventricular free wall, and the left circumflex coronary artery, which supplies the lateral wall of the left ventricle. This circulation pattern may be variable. Sometimes, for example, the circumflex artery also supplies the inferoposterior portion of the left ventricle. MIs tend to be localized to the region (e.g., anterior or inferior) of the left ventricle supplied by one of these arteries or their branches.

The serial, typical effects involving STEMI and Q wave MI on the ECG are examined in this chapter. Chapter 9 discusses the diversity of ECG patterns associated with non–ST segment elevation ischemia and non–Q wave infarctions.

**ST SEGMENT ELEVATION, TRANSMURAL ISCHEMIA, AND ACUTE MYOCARDIAL INFARCTION**

“Transmural” MI is characterized by ischemia and ultimately necrosis of a portion of the entire (or nearly the entire) thickness of the left ventricular wall. Most patients who present with acute MI have underlying atherosclerotic coronary artery disease. The pathophysiology of acute STEMI and subsequent evolving Q wave MI most often relates to occlusion of one of the coronary arteries by a ruptured atherosclerotic plaque, followed by the formation of a clot at this site. The clot in the “culprit artery” is composed of platelets and fibrin, blocking the blood flow downstream.

Other factors can cause or contribute to acute STEMI, including cocaine, coronary artery dissections (spontaneous or induced during interventional procedures), coronary emboli, and other factors.

Not surprisingly, large transmural MIs generally produce changes in both myocardial depolarization (QRS complex) and myocardial repolarization (ST-T complex).

The earliest ECG changes seen with an acute transmural ischemia/infarction typically occur in the ST-T complex in sequential phases:

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**Figure 8-2.** The three major coronary arteries that supply blood to the heart.
1. The *acute* phase is marked by the appearance of ST segment elevations and sometimes tall positive (*hyperacute*) T waves in multiple (usually two or more) leads. The term “STEMI” refers to this phase.

2. The *evolving* phase occurs hours or days later and is characterized by deep T wave inversions in the leads that previously showed ST elevations.

Transmural MIs can also be described in terms of the location of the infarct. *Anterior* means that the infarct involves the anterior or lateral wall of the left ventricle, whereas *inferior* indicates involvement of the inferior (diaphragmatic) wall of the left ventricle (Fig. 8-3). The anatomic location of the infarct determines the leads in which the typical ECG patterns appear. For example, with an acute anterior wall MI, the ST segment elevations and tall hyperacute T waves appear in one or more of the anterior leads (chest leads V1 to V6 and extremity leads I and aVL) (Fig. 8-4). With an inferior wall MI the ST segment elevations and tall hyperacute T waves are seen in inferior leads II, III, and aVF (Fig. 8-5).

An important (but not always present) feature of the ST-T changes seen with STEMI is their *reciprocity*. The anterior and inferior leads tend to show inverse patterns. Thus in an anterior infarction with ST segment elevations in two or more of leads V1 to V6, I, and aVL, ST segment depression is often seen in leads II, III, and aVF. Conversely, with an acute inferior wall infarction, leads II, III, and aVF show ST segment elevation, with reciprocal ST depressions often seen in one or more of leads V1 to V3, I, and aVL. Reciprocal changes are illustrated in Figures 8-4 and 8-5.

The ST segment elevation seen with acute MI is called a *current of injury* and indicates that damage has occurred to the epicardial (outer) layer of the heart as a result of severe ischemia. The exact reasons that acute MI produces ST segment elevation are complex and not fully understood. Normally the ST segment is isoelectric (neither positive nor negative) because no net current flow is occurring at this time. MI alters the electrical charge on the myocardial cell membranes in a number of ways. As a result, current flow becomes abnormal (current of injury) and produces ST segment deviations.

**Figure 8-3.** Myocardial infarctions are most generally localized to either the anterior portion of the left ventricle (A) or the inferior (diaphragmatic) portion of the walls of this chamber (B).

**Figure 8-4.** A, Acute phase of an anterior wall infarction: ST segment elevations and new Q waves. B, Evolving phase: deep T wave inversions. C, Resolving phase: partial or complete regression of ST-T changes (and sometimes of Q waves). In A and B, notice the reciprocal ST-T changes in the inferior leads (II, III, and aVF).
The ST segment elevation seen with acute MI may have different shapes and appearances (Fig. 8-6). Notice that the ST segment may be plateau-shaped or dome-shaped. Sometimes it is obliquely elevated.

The ST segment elevations (and reciprocal ST depressions) are the earliest ECG signs of infarction, and are generally seen within minutes of blood flow occlusion. Tall, positive (hyperacute) T waves may also be seen at this time (Figs. 8-7 and 8-8).

These T waves have the same significance as the ST elevations. In some cases, hyperacute T waves actually precede the ST elevations.

Guidelines for assessing whether ST segment (and usually J point) elevations are due to acute ischemia have been suggested. However, strict criteria are limited because of false-positives (due to normal variants, left ventricular hypertrophy, etc., as described in Chapter 9) and false-negatives (e.g., T wave positivity may precede ST elevations or the ST elevations may be less than 1-2 mm).

Clinicians should be aware that ST changes in acute ischemia may evolve with the patient under observation. If the initial ECG is not diagnostic of STEMI but the patient continues to have symptoms consistent with myocardial ischemia, serial ECGs at 5- to 10-minute intervals (or continuous 12-lead ST segment monitoring) should be performed.

After a variable time lag (usually hours to a few days) the elevated ST segments start to return to the baseline. At the same time the T waves become inverted in leads that previously showed ST segment elevations. This phase of T wave inversions is called the evolving phase of the infarction. Thus with an anterior wall infarction the T waves become inverted in one or more of the anterior leads (V1 to V6, I, aVL). With an inferior wall infarction the T waves become inverted in one or more of the inferior leads (II, III, aVF). (These T wave inversions are illustrated in Figs. 8-4 and 8-5.)
CHAPTER 8  ST Segment Elevation, Transmural Ischemia, and Acute Myocardial Infarction

Figure 8-7. Chest leads from a patient with acute anterior ST segment elevation myocardial infarction (STEMI). A, In the earliest phase of the infarction, tall, positive (hyperacute) T waves are seen in leads V₂ to V₅. B, Several hours later, marked ST segment elevation is present in the same leads (current of injury pattern), and abnormal Q waves are seen in leads V₁ and V₂.

Figure 8-8. Hyperacute T waves with anterior ST segment elevation myocardial infarction (STEMI). This patient was complaining of severe chest pain. Notice the very tall (hyperacute) T waves in the chest leads. In addition, slight ST segment elevations are present in lead aVL and reciprocal ST depressions are seen in leads II, III, and aVF. Notice the atrial premature beat (APB) in lead V₄.
QRS Changes: Q Waves of Infarction
MI, particularly when large and transmural, often produces distinctive changes in the QRS (depolarization) complex. The characteristic depolarization sign is the appearance of new Q waves.

Why do certain MIs lead to Q waves? Recall that a Q wave is simply an initial negative deflection of the QRS complex. If the entire QRS complex is negative, it is called a QS complex:

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\text{\textbf{QRS Changes: Q Waves of Infarction}}
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A Q wave (negative initial QRS deflection) in any lead indicates that the electrical voltages are directed away from that particular lead. With a transmural infarction, necrosis of heart muscle occurs in a localized area of the ventricle. As a result the electrical voltages produced by this portion of the myocardium disappear. Instead of positive (R) waves over the infarcted area, Q waves are often recorded (either a QR or QS complex).

As discussed in the next chapter, the common clinical tendency to equate pathologic Q waves with transmural necrosis is an oversimplification. Not all transmural infarcts lead to Q waves, and not all Q wave infarcts correlate with transmural necrosis.

In summary, abnormal Q waves are characteristic markers of infarction. They signify the loss of positive electrical voltages caused by the death of heart muscle.

The new Q waves of an MI generally appear within the first day or so of the infarct. With an anterior wall infarction these Q waves are seen in one or more of leads V3 to V6, I, and aVL (see Fig. 8-4). With an inferior wall MI the new Q waves appear in leads II, III, and aVF (see Fig. 8-5).

ECG LOCALIZATION OF INFARCTIONS
As mentioned earlier, MIs are generally localized to a specific portion of the left ventricle, affecting either the anterior or the inferior wall. Anterior infarctions are sometimes designated as anteroseptal, strictly anterior, or anterolateral/anteroapical, depending on the leads that show signs of the infarction (Figs. 8-9 to 8-11).

Anterior Wall Q Wave Infarctions
The characteristic feature of an anterior wall Q wave infarct is the loss of normal R wave progression in the chest leads. Recall that normally the height of the R wave increases progressively as you move from lead V1 to lead V6. An anterior infarct interrupts this progression, and the result may be pathologic Q waves in one or more of the chest leads. In clinical practice cardiologists often subdivide anterior MIs into a number of subsets depending on the leads showing Q waves. Clinicians should be aware that these ECG localizations may not correspond exactly with imaging or postmortem findings and that different authors may not use exactly the same definitions.

“Anteroseptal” Q Wave Infarctions
Remember from Chapter 4 that the ventricular septum is depolarized from left to right and that leads V1 and V2 show small positive (r) waves (septal r waves). Now consider the effect of damaging the septum. Obviously, septal depolarization voltages are lost. Thus the r waves in leads V1 and V2 may disappear and an entirely negative (QS) complex appears.

The septum is supplied with blood by the left anterior descending coronary artery. Septal infarction generally suggests that this artery or one of its branches is occluded.

“Strictly” Anterior Q Wave Infarctions
Normally leads V3 and V4 show RS- or Rs-type complexes. If an infarction occurs in the anterior wall of the left ventricle, the positive R waves that reflect the voltages produced by this muscle area are lost. Instead, Q waves (as part of QS or QR complexes) are seen in leads V3 and V4. A strictly anterior infarct generally results from occlusion of the left anterior descending coronary artery.
Anterolateral or Anteroapical Q Wave Infarctions

An infarction of the anterolateral or apical wall of the left ventricle produces changes in the more laterally situated chest leads (V₅ and V₆). With such infarctions, abnormal Q waves, as part of QS or QR complexes, appear in leads V₅ and V₆ (see Fig. 7-7). The infarcts are often caused by occlusion of the left circumflex coronary artery, but they may also result from occlusion of the left anterior fascicular block.
descending coronary artery or even a branch of a dominant right coronary artery.

ST elevations and pathologic Q waves localized to leads I and aVL are often ascribed to a “high lateral” MI. The “culprit artery” in such cases is usually an occluded diagonal branch of the left anterior descending or branch of the left circumflex coronary.

**Differentiating Anterior Wall Infarctions: General Comments and Caveats**

As noted, the foregoing classification of anterior infarctions is not absolute, and infarct types often overlap. To avoid ambiguity, you can simply describe Q wave MIs by simply referring to any infarct that shows ECG changes in one or more of leads I, aVL, and V₁ to V₆ as anterior and then specifying the leads that show Q waves and ST-T changes.

Not surprisingly, anterior infarctions associated with large Q waves in leads V₁ to V₅ or V₆ usually represent extensive damage and substantially reduced left ventricular function (ejection fraction) (see Fig. 8-11).

**Inferior Wall Infarctions**

Infarction of the inferior (diaphragmatic) portion of the left ventricle is indicated by changes in leads II, III, and aVF (Figs. 8-12 to 8-14). These three leads, as shown in the diagram of the frontal plane axis, are oriented downward or inferiorly (see Fig. 5-1). Thus they record voltages from the inferior
portion of the ventricle. An inferior wall infarction may produce abnormal Q waves in leads II, III, and aVF. This type of infarction is generally caused by occlusion of the right coronary artery. Less commonly it occurs because of a left circumflex coronary obstruction.

**Posterior Infarctions**

Infarctions can occur in the posterior (back) surface of the left ventricle. These infarctions may be difficult to diagnose because characteristic abnormal ST elevations may not appear in any of the 12 conventional leads. Instead, tall R waves and ST depressions may occur in leads V1 and V2 (reciprocal to the Q waves and ST segment elevations that would be recorded at the back of the heart). During the evolving phase of these infarctions, when deep T wave inversions appear in the posterior leads, the anterior chest leads show reciprocally tall positive T waves (Fig. 8-15).

In most cases of posterior MI the infarct extends either to the lateral wall of the left ventricle, producing characteristic changes in lead V6, or to the inferior wall of that ventricle, producing characteristic changes in leads II, III, and aVF (see Fig. 8-15). Because of the overlap between inferior, lateral, and posterior infarctions, the more general terms inferoposterior or posterolateral are used, depending on which leads are involved.

**Right Ventricular Infarctions**

A related topic is right ventricular infarction. Clinical and autopsy studies have shown that patients with an inferoposterior infarct not uncommonly have associated right ventricular involvement. In one postmortem study, right ventricular infarction was found in about one of four cases of inferoposterior MI but not in cases of anterior MI. Clinically, patients with a right ventricular infarct may have elevated central venous pressure (distended neck veins) because of the abnormally high diastolic filling pressures in the right side of the heart. If the damage to the right ventricle is severe, hypotension and even cardiogenic shock may occur.

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**Figure 8-12.** Acute inferolateral wall ST segment elevation myocardial infarction (STEMI). Notice the prominent ST elevations in leads II, III, and aVF, as well, more subtly, in V5 and V6. The reciprocal ST depressions are in leads I and aVL, and V1 to V2. The latter finding may be reciprocal to lateral or posterior ischemia. (Used with permission from Nathanson LA, McClenen S, Safran C, Goldberger AL: ECG Wave-Maven: Self-Assessment Program for Students and Clinicians. http://ecg.bidmc.harvard.edu.)
result. Atrioventricular conduction disturbances are not uncommon in this setting. The presence of jugular venous distention in patients with acute inferoposterior wall MIs should always suggest this diagnosis. Many of these patients also have ST segment elevations in leads reflecting the right ventricle, such as V1 and V3R to V5R, as shown in Figure 8-16 (see also Chapter 3).

Recognition of right ventricular infarction is of major clinical importance. Careful volume expansion may be critical in improving cardiac output in patients who are hypotensive and have a low or normal pulmonary capillary wedge pressure despite elevated central venous pressure.

**Classical Sequence of ST-T Changes and Q Waves with STEMI**

To this point ventricular depolarization (QRS complex) and repolarization (ST-T complex) changes produced by an acute MI have been discussed separately. As shown in Figures 8-4 and 8-5, these changes often occur sequentially.

Ordinarily, the earliest sign of transmural ischemia is ST segment elevations (with reciprocal ST depressions). The ST elevations (current of injury pattern) usually persist for hours to days. During this same period, Q waves often begin to appear in the leads that show ST elevations. Once these changes have occurred, the ST segments start to return to
the isoelectric baseline and the T waves become inverted during the evolving phase.

In the weeks or months after an infarct, what should you expect to happen to the Q waves and the ST-T changes just described? The answer is that you cannot make any certain predictions. In most cases the abnormal Q waves persist for months and even years after the acute infarction. Occasionally, however, the abnormal Q waves diminish in size and even disappear entirely. In some cases, abnormal T wave inversions persist indefinitely. In others, improvement occurs, but minor nonspecific ST-T abnormalities such as slight T wave flattening may persist (see Figs. 8-4 and 8-5).

**Normal and Abnormal Q Waves: A Brief Overview**

A frequently encountered diagnostic problem is deciding whether Q waves are abnormal. Not all Q waves are indicators of MI. For example, a Q wave is normally seen in lead aVR. Furthermore, small “septal” q waves are normally seen in the left chest leads (V4 to V6) and in one or more of leads I, aVL, aVF.
PART I  Basic Principles and Patterns

II, III, and aVF. Recall from Chapter 4 the significance of these septal q waves. The ventricular septum depolarizes from left to right. Left chest leads record this spread of voltages toward the right as a small negative deflection (q wave) that is part of a qR complex in which the R wave represents the spread of left ventricular voltages toward the lead. When the electrical axis is horizontal, such qR complexes are seen in leads I and aVL. When the electrical axis is vertical, qR complexes appear in leads II, III, and aVF.

These normal septal q waves must be differentiated from the pathologic Q waves of infarction. Normal septal q waves are characteristically narrow and of low amplitude. As a rule, septal q waves are less than 0.04 sec in duration. A Q wave is generally abnormal if its duration is 0.04 sec or more in lead I, all three inferior leads (II, III, aVF), or leads V3 to V6.

What if Q waves with duration of 0.04 sec or more are seen in leads V1 and V2? A large QS complex can be a normal variant in lead V1 and rarely in leads V1 and V2. However, QS waves in these leads may be the only evidence of an anterior septal MI. An abnormal QS complex resulting from infarction sometimes shows a notch as it descends, or it may be slurred instead of descending and rising abruptly (see Fig. 8-9). Further criteria for differentiating normal from abnormal Q waves in these leads lie beyond the scope of this book.

Figure 8-16. Acute right ventricular ischemia with inferior wall infarction. A, Q waves and ST segment elevations in leads II, III, and aVF are accompanied by ST segment elevations (arrow) in the right precordial leads (V3R and V1). The ST-T changes in lead V6 are consistent with lateral wall ischemia. The ST segment depressions in leads I and aVL are probably reciprocal to inferior lead ST elevations. B, Follow-up tracing obtained the next day, showing diminution of the ST changes. (From Goldberger AL: Myocardial Infarction: Electrocardiographic Differential Diagnosis, 4th ed. St. Louis, Mosby, 1991.)
What if a wide Q wave is seen in lead aVL, or Q waves are present in leads III and aVF? These waveforms can also occur normally. Although a discussion of the precise criteria for differentiating normal from abnormal Q waves in these leads is also beyond the scope of this book, the following can be taken as general rules:

- An inferior wall MI should be diagnosed with certainty only when abnormal Q waves are seen in leads II, III, and aVF. If prominent Q waves appear just in leads III and aVF, the likelihood of MI is increased by the presence of abnormal ST-T changes in all three inferior extremity leads.
- An anterior wall MI should not be diagnosed from lead aVL alone. Look for abnormal Q waves and ST-T changes in the other anterior leads (I and V1 to V6).

Furthermore, just as not all Q waves are abnormal, all abnormal Q waves are not the result of MI. For example, slow R wave progression in the chest leads, sometimes with actual QS complexes in the right to middle chest leads (e.g., V1 to V3), may occur with left bundle branch block (LBBB), left ventricular hypertrophy, amyloidosis, and chronic lung disease in the absence of MI, in addition to multiple other factors. Prominent noninfarction Q waves are often a characteristic feature in the ECGs of patients with hypertrophic cardiomyopathy (Fig. 8-17). Noninfarction Q waves also occur with dilated cardiomyopathy (see Fig. 11-4). As mentioned previously, the ECGs of normal people sometimes have a QS wave in lead V1 and rarely in leads V1 and V2. Prominent Q waves in the absence of MI are sometimes referred to as a pseudoinfarct pattern (see Chapter 24).

**VENTRICULAR ANEURYSM**

After a large MI a ventricular aneurysm develops in some patients. An aneurysm is a severely scarred portion of infarcted ventricular myocardium that does not contract normally. Instead, during ventricular systole the aneurysmic portion bulges outward while the rest of the ventricle is contracting. Ventricular aneurysm may occur on the anterior or inferior surface of the heart.

The ECG may be helpful in making the diagnosis of ventricular aneurysm subsequent to an MI. Patients with ventricular aneurysm frequently have persistent ST segment elevations after an infarct. As mentioned earlier, the ST segment elevations seen with acute infarction generally resolve within several days. The persistence of ST segment elevations for several weeks or more is suggestive of a ventricular aneurysm (Fig. 8-18). However, the absence of persisting ST segment elevations does not rule out the possibility of an aneurysm.

Ventricular aneurysms are of clinical importance for several major reasons. They may lead to congestive heart failure. They may be associated with serious ventricular arrhythmias. A thrombus may form in an aneurysm and break off, resulting in a stroke or some other embolic complication.
MULTIPLE INFARCTIONS
Not infrequently, patients may have two or more MIs at different times. For example, a new anterior wall infarct may develop in a patient with a previous inferior wall infarction. In such cases the ECG initially shows abnormal Q waves in leads II, III, and aVF. During the anterior infarct, new Q waves and ST-T changes appear in the anterior leads. (The ECG of a patient with multiple infarcts [anterior and inferior] is presented in Fig. 8-19.)

“SILENT” MYOCARDIAL INFARCTION
Most patients with an acute MI have symptoms. They may experience the classic syndrome of crushing substernal chest pain, or they may have atypical pain (e.g., a sensation like indigestion, upper back pain, or jaw pain). Sometimes, however, patients may experience few if any symptoms (“silent” MI). Therefore, it is not unusual for an ECG to show abnormal Q waves that indicate a previous infarction in a patient without a clinical history of definite MI.

DIAGNOSIS OF MYOCARDIAL INFARCTION IN THE PRESENCE OF BUNDLE BRANCH BLOCK
The diagnosis of infarction is more difficult when the patient’s baseline ECG shows a bundle branch block pattern or a bundle branch block develops as a complication of the MI. Then the ECG picture becomes more complex.
CHAPTER 8  Diagnosis of Myocardial Infarction In the Presence of Bundle Branch Block

Right Bundle Branch Block with Myocardial Infarction
The diagnosis of an MI can be made relatively easily in the presence of right bundle branch block (RBBB). Remember that RBBB affects primarily the terminal phase of ventricular depolarization, producing a wide R' wave in the right chest leads and a wide S wave in the left chest leads. MI affects the initial phase of ventricular depolarization, producing abnormal Q waves. When RBBB and an infarct occur together, a combination of these patterns is seen: The QRS complex is abnormally wide (0.12 sec or more) as a result of the bundle branch block, lead V1 shows a terminal positive deflection, and lead V6 shows a wide S wave. If the infarction is anterior, the ECG shows a loss of R wave progression with abnormal Q waves in the anterior leads and characteristic ST-T changes. If the infarction is inferior, pathologic Q waves and ST-T changes are seen in leads II, III, and aVF. (An anterior wall infarction with a RBBB pattern is shown in Fig. 8-20.)

Left Bundle Branch Block with Myocardial Infarction
The diagnosis of LBBB in the presence of MI is considerably more complicated and confusing than that of RBBB. The reason is that LBBB interrupts both the early and the late phases of ventricular stimulation (see Chapter 7). It also produces secondary ST-T changes. As a general rule, LBBB hides the diagnosis of an infarct. Thus a patient with a chronic LBBB pattern who develops an acute MI may not show the characteristic changes of infarction described in this chapter.

Occasionally, patients with LBBB manifest primary ST-T changes indicative of ischemia or actual infarction. The secondary T wave inversions of uncomplicated LBBB are seen in leads V4 to V6 (with prominent R waves). The appearance of T wave inversions in leads V1 to V3 (with prominent S waves) is a primary abnormality that cannot be ascribed to the bundle branch block itself (Fig. 8-21).

The problem of diagnosing infarction with LBBB is further complicated by the fact that the LBBB pattern has several features that resemble those seen with infarction. Thus an LBBB pattern can mimic an infarct pattern. As discussed in Chapter 7, LBBB typically shows slow R wave progression in the chest leads because of the reversed way the ventricular septum is activated (i.e., from right to left, the opposite of what happens normally). Consequently, with LBBB a loss of the normal septal R waves is seen in the right chest leads. This loss of normal R wave progression simulates the pattern seen with an anterior wall infarct.

Figure 7-5 shows an example of LBBB with slow R wave progression. In this case, anterior wall infarction was not present. Notice that the ST segment elevations in the right chest leads resemble the pattern seen during the hyperacute or acute phase of an infarction. ST segment elevation in the right chest leads is also commonly seen with LBBB in the absence of infarction.
Figure 8-21. A, Typical left bundle branch block pattern. Notice the slow R wave progression in the right precordial leads and the discordance of QRS and ST-T vectors reflected by the ST segment elevations in the right precordial leads and the ST depressions with T wave inversions in the left precordial leads. B, Subsequently the ECG from this patient showed the development of primary T wave inversions in leads V1 to V3 (arrows) caused by anterior ischemia and probable infarction. (From Goldberger AL: Myocardial Infarction: Electrocardiographic Differential Diagnosis, 4th ed. St. Louis, Mosby, 1991.)
As a general rule, a patient with an LBBB pattern should not be diagnosed as having had an MI simply on the basis of poor R wave progression in the right chest leads or ST elevations in those leads. However, the presence of Q waves as part of QR complexes in the left chest leads (V5 and V6) with LBBB generally indicates an underlying MI (Fig. 8-22). In addition, the appearance of ST segment elevations in the left chest leads or in other leads with prominent R waves suggests ischemia (see Fig. 8-22, lead V5), as do ST segment depressions in the right leads or other leads with an rS or a QS morphology. (The discussion of the ECG with ischemia and infarction continues in Chapter 9, which focuses on subendocardial ischemia and non-Q wave MI patterns.)