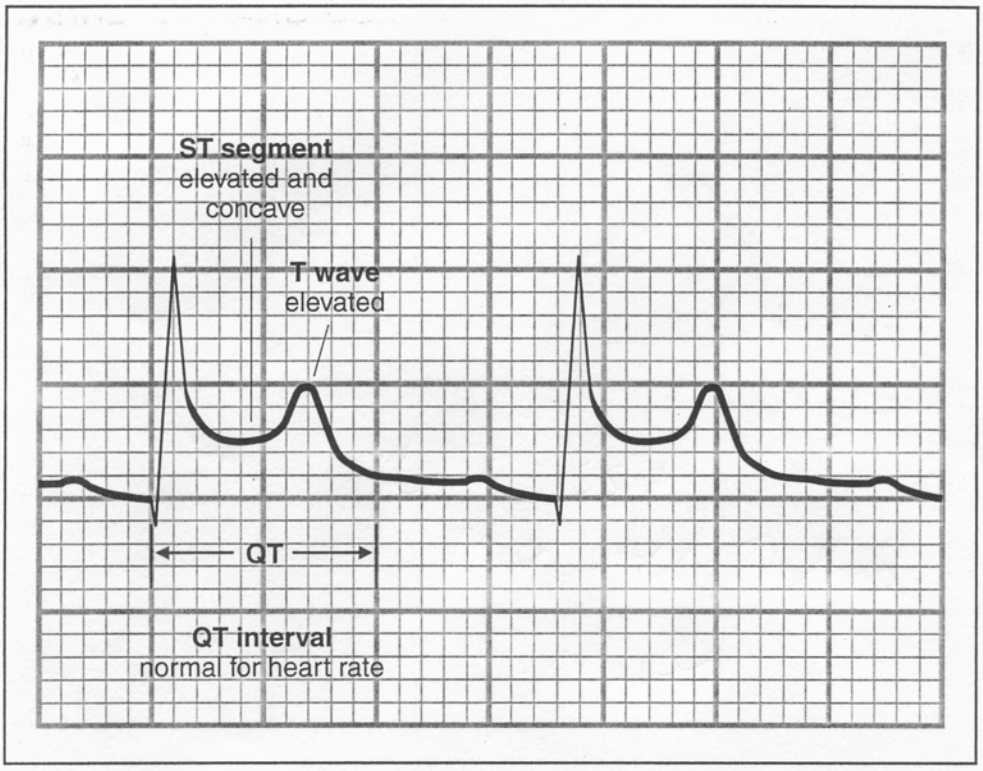

Figure 14-5 Pericarditis.

- Acute MI
- Trauma
- Connective tissue disorders
- Allergic and hypersensitivity diseases
- Metabolic disorders

Unlike acute MI, which pericarditis may mimic, pericarditis usually occurs in younger patients without cardiac risk factors who are not suspected of having coronary artery disease. The signs and symptoms of acute pericarditis include the following:

- Chest pain
- Dyspnea
- Tachycardia
- Fever
- Malaise
- Weakness
- Chills

The chest pain, which can mimic that of acute MI, is sharp and severe, with radiation to the neck, back, left shoulder, and, rarely, to the arm. Characteristically, it is present along the sternum, made worse by lying flat, and relieved by sitting or leaning forward. Often, the pain is pleuritic and made worse by breathing, especially during inspiration. Unlike the pain of acute MI, the pain may last for hours or even days.

A pericardial friction rub, resulting from the inflammation of the pericardial surface, is heard and even palpated along the lower left sternal border. Characteristic ECG findings are present in 90% of the patients with acute pericarditis.

◆ ECG CHARACTERISTICS

QRS Complexes

Abnormal Q waves and QS complexes are absent. In pericarditis with pleural effusion, the QRS complexes are of low voltage. When pleural effusion becomes severe, cardiac tamponade may occur, causing the QRS complexes to alternate between normal and low amplitude, coincident with respiration (electrical alternans).

ST Segments

ST segment elevation is the primary ECG abnormality in acute pericarditis. Although the ST segments are somewhat concave, they appear quite similar to the elevated ST segments present in acute MI.

The ST segments are usually elevated in most, if not all, leads except leads aVR and V_1, because pericarditis usually affects the entire myocardial surface of the heart. In lead aVR, the ST segment is either normal or slightly depressed. This wide distribution of the ST segment elevation in pericarditis helps to differentiate it from acute MI in which there is a more limited distribution of ST segment elevation. Occasionally, pericarditis will be local-

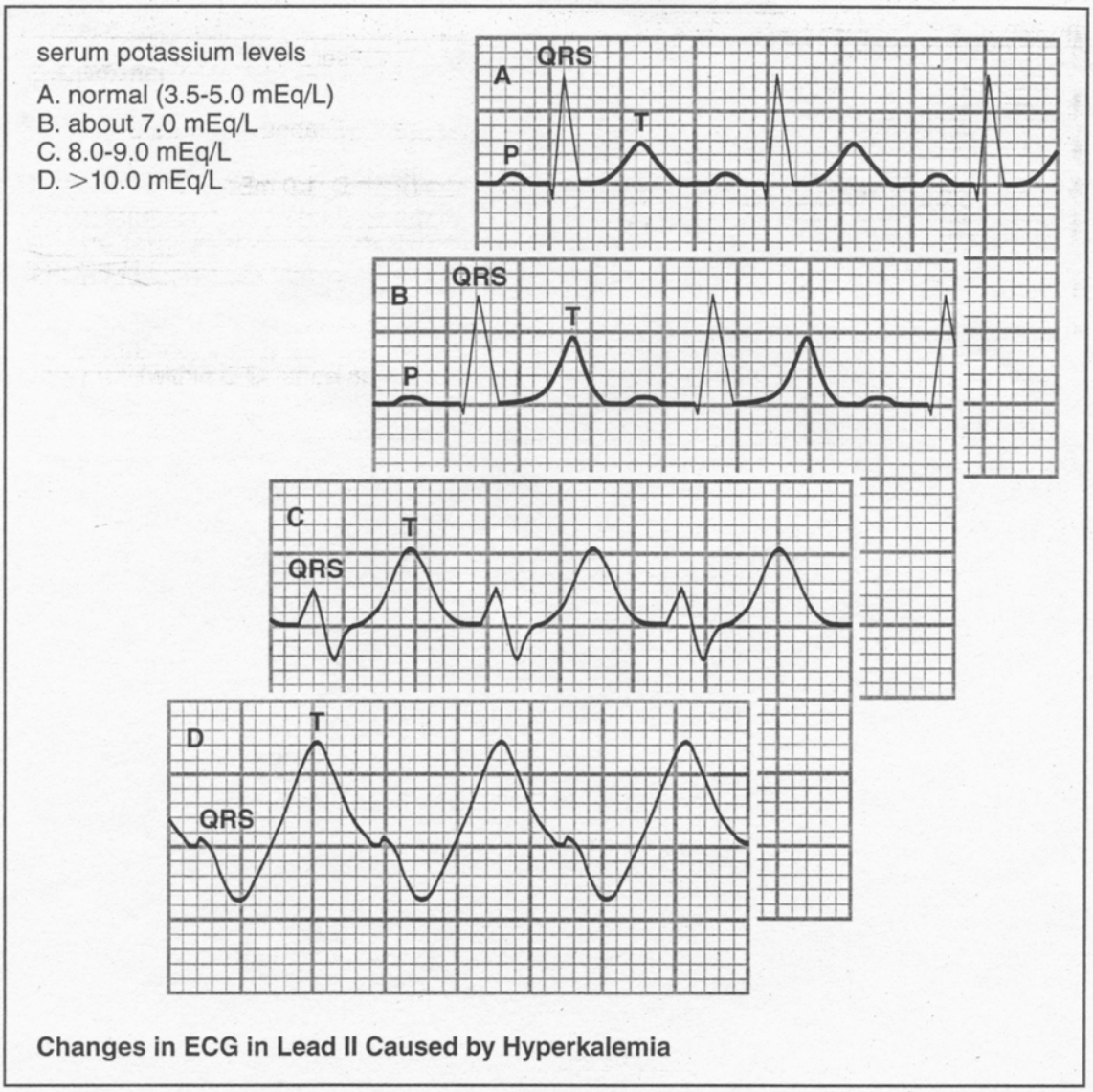

Figure 14-6 Hyperkalemia.

Hypokalemia

◆ PATHOPHYSIOLOGY

Hypokalemia (Figure 14-7) is the deficiency of serum potassium below the normal levels of 3.5 to 5.0 mEq/L. The most common cause of hypokalemia is loss of potassium in body fluids through vomiting, gastric suction, and excessive use of diuretics. Hypokalemia may also result from low serum magnesium levels (hypomagnesemia). Incidentally, the ECG characteristics of hypomagnesemia resemble those of hypokalemia.

Symptoms of hypokalemia are polyuria in mild cases and muscle weakness in more severely affected patients. Digitalis in the presence of hypokalemia may precipitate serious ventricular arrhythmias, including the torsade de pointes form of ventricular tachycardia. The diagnosis of hypokalemia is often made by the characteristic ECG changes caused by low serum potassium. Characteristic ECG changes occur at various levels of hypokalemia.

◆ ECG CHARACTERISTICS

P Waves

The P waves become typically tall and symmetrically peaked with an amplitude of 2.5 mm or greater in leads II, III, and aVF in severe hypokalemia of about 2 mEq/L or less. Because these P waves resemble P pulmonale, they are called "pseudo P pulmonale."

QRS Complexes

The QRS complexes begin to widen when the serum potassium level drops to about 3.0 mEq/L.

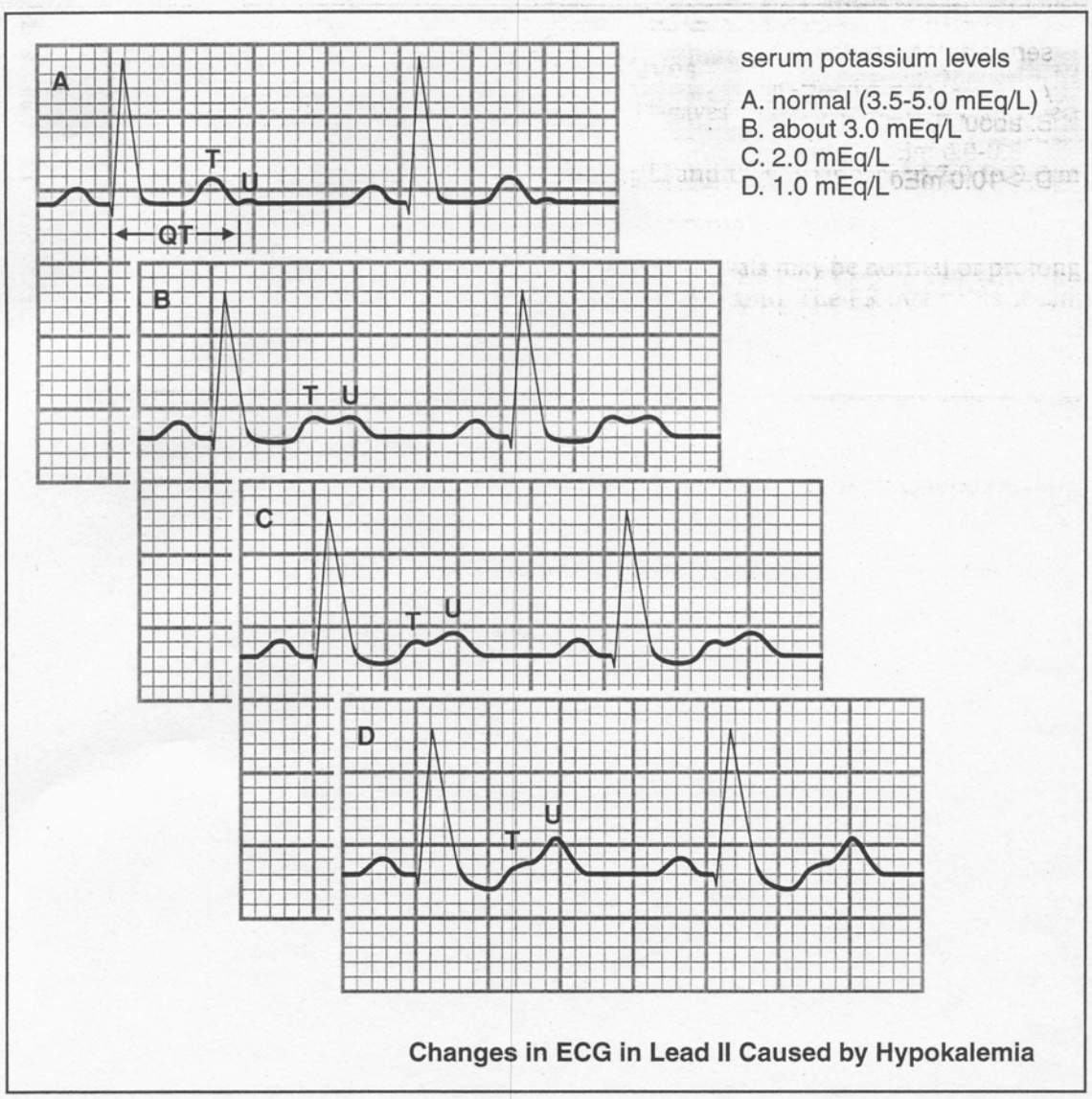

Figure 14-7 Hypokalemia.

ST Segments

The ST segments may become depressed by 1 mm or more.

T Waves

The T waves begin to flatten when the serum potassium level drops to about 3.0 mEq/L and continue to become smaller as the U waves increase in size. The T waves may either merge with the U waves or become inverted.

U Waves

The U waves begin to increase in size, becoming as tall as the T waves, when the serum potassium level drops to about 3.0 mEq/L; they become taller than the T waves at about 2 mEq/L. The U wave is considered to be "prominent" when it is equal to or taller than the T wave in the same lead. The U waves reach "giant" size and fuse with the T waves at 1 mEq/L.

QT Intervals

The QT intervals may appear to be prolonged when the U waves become prominent and fuse with the T waves.

Notes

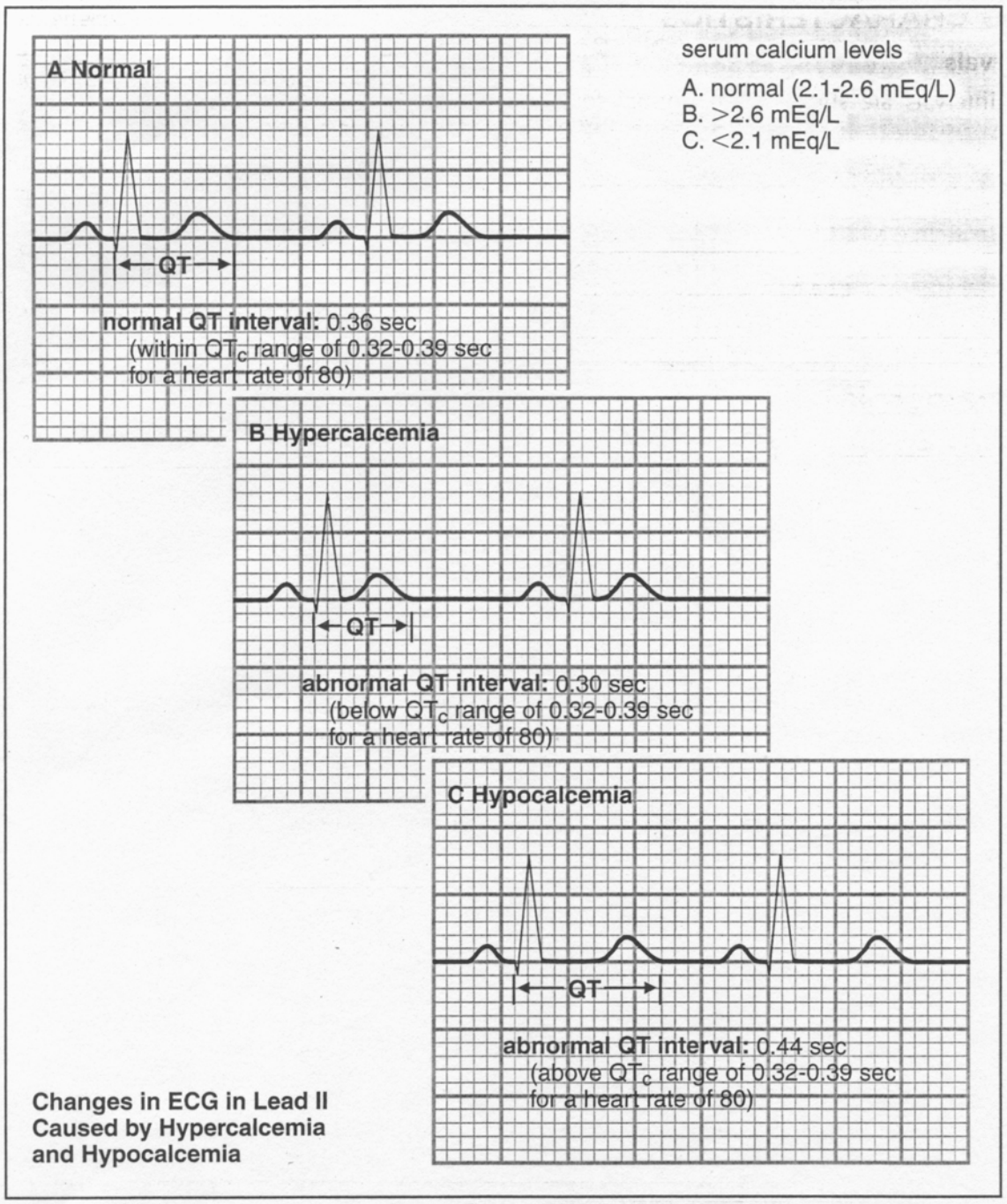

Figure 14-8 Hypercalcemia and hypocalcemia.

Hypercalcemia

◆ PATHOPHYSIOLOGY

Hypercalcemia (Figure 14-8, *B*) is the excess of serum calcium above the normal levels of 2.1 to 2.6 mEq/L (or 4.25 to 5.25 mg/100 mL). Common causes of hypercalcemia include the following:

- ◆ Adrenal insufficiency
- ◆ Hyperparathyroidism
- ◆ Immobilization
- ◆ Kidney failure
- ◆ Malignancy
- ◆ Sarcoidosis
- ◆ Thyrotoxicosis
- ◆ Vitamin A and D intoxication

Severe hypercalcemia is life threatening. Digitalis in the presence of hypercalcemia may precipitate serious arrhythmias.

◆ ECG CHARACTERISTICS

QT Intervals

The QT intervals are shorter than normal for the heart rate.

Notes

Hypocalcemia

◆ PATHOPHYSIOLOGY

Hypocalcemia (Figure 14-8, *C*) is the shortage of serum calcium below the normal levels of 2.1 to 2.6 mEq/L (or 4.25 to 5.25 mg/100 mL). Common causes of hypocalcemia include the following:

- Chronic steatorrhea
- Diuretics (such as furosemide or ethacrynic acid)
- Hypomagnesemia (possibly because of release of parathyroid hormone)
- Osteomalacia in adults and rickets in children
- Hypoparathyroidism
- Pregnancy
- Respiratory alkalosis and hyperventilation

◆ ECG CHARACTERISTICS

ST Segments

The ST segments are prolonged.

QT Intervals

The QT intervals are prolonged beyond the normal limits for the heart rate because of the prolongation of the ST segments.

Notes

DRUG EFFECT

Digitalis

◆ PATHOPHYSIOLOGY

Digitalis administered within therapeutic range produces characteristic changes in the ECG (Figure 14-9). In addition, when given in excess, digitalis toxicity occurs, causing excitatory or inhibitory effects on the heart and its electrical conduction system.

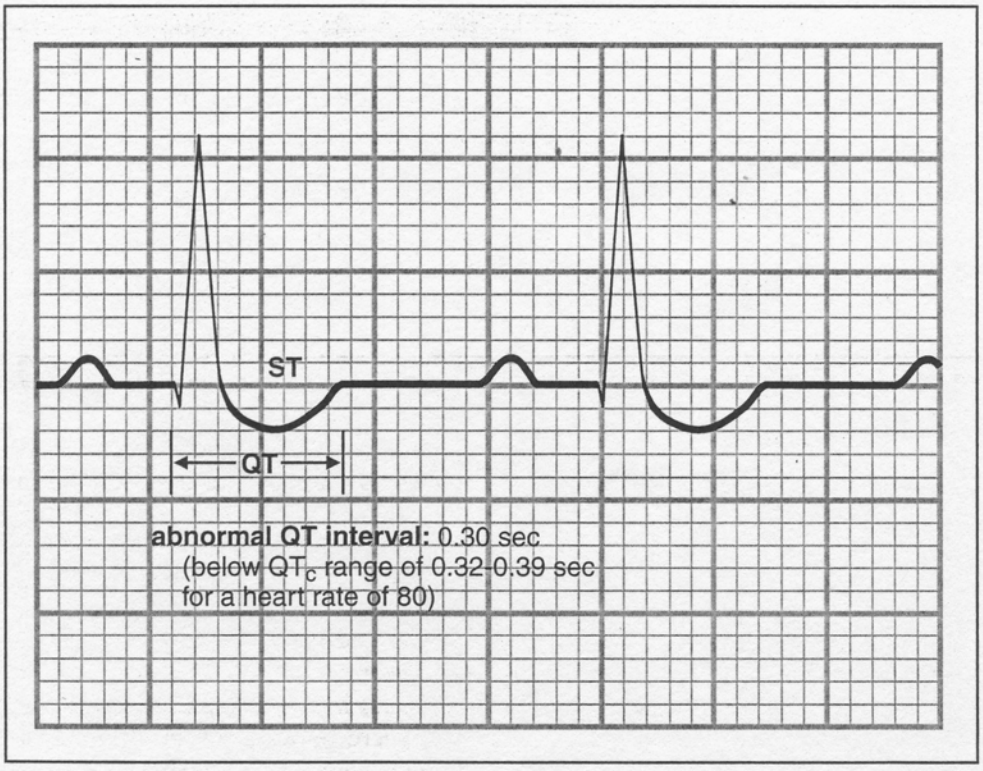

Figure 14-9 Digitalis effect.

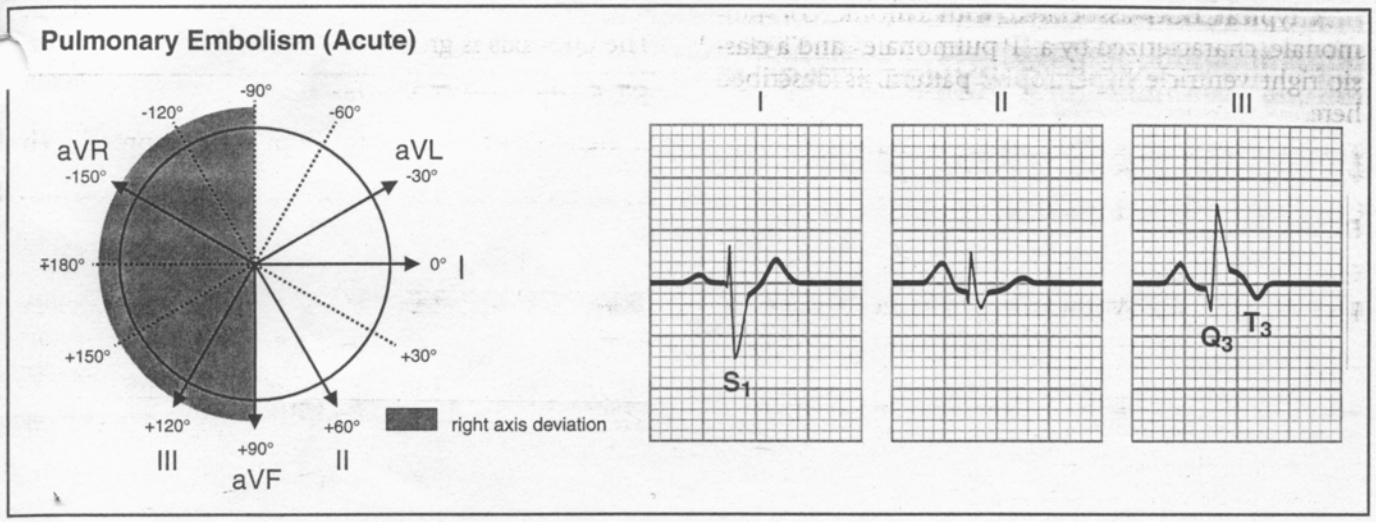

Figure 14-13 Pulmonary embolism.

- Cyanosis
- Distended neck veins (right-sided congestive heart failure)
- Forceful pulsation, seen and palpated, in the second left intercostal space with a systolic pulmonic murmur (dilated pulmonary artery)
- Hypotension, shock, and, rarely, cardiac arrest

Because of the increased pressure in the pulmonary artery (pulmonary hypertension) caused by the major obstruction of blood flow through the pulmonary circulation, the right ventricle and atrium become distended, unable to function properly, leading to right heart failure. This condition is called *acute cor pulmonale.*

In minimal pulmonary embolization, the ECG may be normal. However, in massive acute pulmonary embolization (acute cor pulmonale), the ECG shows a "P pulmonale" and a characteristic $S_1Q_3T_3$ pattern described below.

◆ ECG CHARACTERISTICS

P Waves

Changes indicative of right atrial enlargement are present (i.e., tall, symmetrically peaked P waves [P pulmonale] in leads II, III, and aVF and sharply peaked biphasic P waves in leads V_1 and V_2).

QRS Complexes

An S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III (the $S_1Q_3T_3$ pattern) may occur acutely. In addition, a right bundle branch block may also occur.

QRS Axis

The QRS axis is greater than +90°.

ST Segments/T Waves

A right ventricular "strain" pattern may be present (inverted T waves in leads V_1-V_3).

Notes

Chronic Cor Pulmonale

◆ PATHOPHYSIOLOGY

Chronic cor pulmonale (Figure14-14) is the enlargement of the right ventricle (dilatation and/or hypertrophy) commonly accompanied by right heart failure. It is usually the end stage result of prolonged pulmonary hypertension that occurs with many diseases of the lung, including COPD and recurrent pulmonary embolization.

Chronic cor pulmonale is often associated with atrial arrhythmias, including the following:

- Premature atrial contractions
- Wandering atrial pacemaker
- Multifocal atrial tachycardia
- Atrial flutter
- Atrial fibrillation

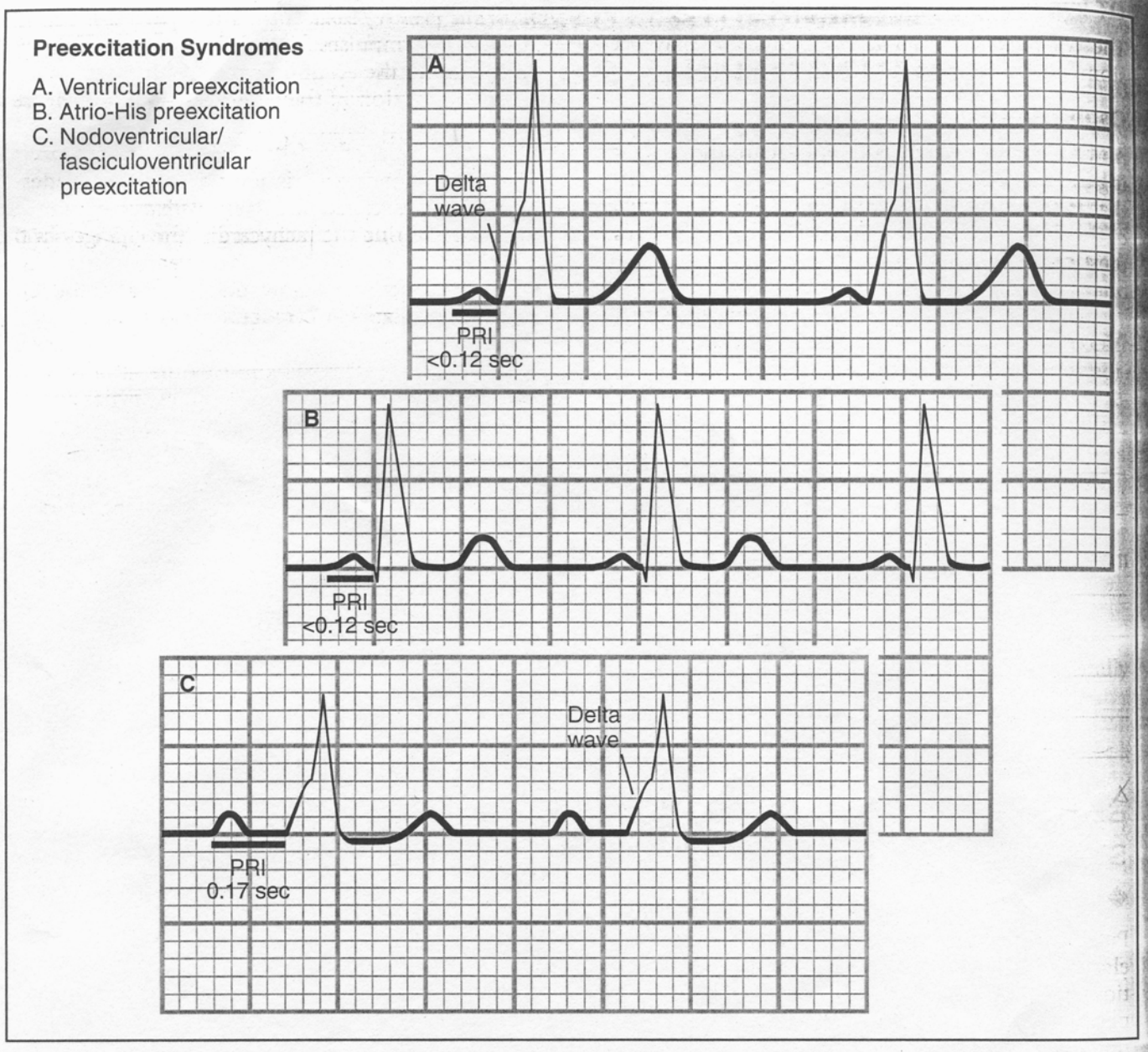

Figure 14-17 Preexcitation syndromes.

Nodoventricular/Fasciculoventricular Preexcitation (Nodoventricular/Fasciculoventricular Fibers)

PR Intervals. The PR intervals are usually normal, 0.12 second or greater.

QRS Complexes. The duration of the QRS complexes are greater than 0.10 second and abnormally shaped, with a delta wave (the slurring of the onset of the QRS complex) in both of these preexcitation syndromes.

◆ CLINICAL SIGNIFICANCE

Because of the wide and distorted QRS complexes associated with ventricular and nodoventricular/fasciculoventricular preexcitation, an ECG with such QRS complexes may be mistaken for a bundle branch block, ventricular hypertrophy, or MI. When the heart rate is rapid, the P waves are superimposed on the preceding T waves, causing, for example, a supraventricular tachycardia to resemble ventricular tachycardia.

Notes