Cardiac Action Potential

Figure 1-11 Cardiac action potential of myocardial cells.

trical charges within the cell and a drop in the membrane potential to about 0 mV.

◆ **Phase 2.** Phase 2 is the prolonged phase of slow repolarization (plateau phase) of the action potential of the myocardial cell, allowing it to finish contracting and begin relaxing. During phase 2, the membrane potential remains about 0 mV because of a very slow rate of repolarization. In a complicated exchange of ions across the cell membrane, calcium slowly enters the cell through the slow calcium channels as potassium continues to leave the cell and sodium enters it slowly.

◆ **Phase 3.** Phase 3 is the terminal phase of rapid repolarization, during which the inside of the cell becomes markedly negative and the membrane potential once again returns to about −90 mV, its resting level. This is caused primarily by the flow of potassium from the cell. Repolarization is complete by the end of phase 3.

◆ **Phase 4.** At the onset of phase 4 (the period between action potentials), the membrane has returned to its resting potential and the inside of the cell is once again negative (−90 mV) with respect to the outside. But there is still an excess of sodium in the cell and an excess of potassium outside. At this point, a mechanism known as the *sodium-potassium pump* is activated, transporting the excess sodium out of the cell and potassium back in. Because of this mechanism and the impermeability of the cell membrane to sodium during phase 4,

the myocardial cell normally maintains a stable membrane potential between action potentials.

Refractory and Supernormal Periods

The time between the onset of depolarization and the end of repolarization is customarily divided into periods during which the cardiac cells can or cannot be stimulated to depolarize. These are the refractory periods (absolute and relative) and the supernormal period (Figure 1-12).

The refractory period of cardiac cells (e.g., those of the ventricles) begins with the onset of phase 0 of the cardiac action potential and ends just before the end of phase 3. On the ECG, it extends from the onset of the QRS complex to about the end of the T wave.

The refractory period is further divided into the absolute and relative refractory periods. The absolute refractory period (ARP) begins with the onset of phase 0 and ends midway through phase 3 at about the peak of the T wave, occupying over two thirds of the refractory period. During this period, the cardiac cells, having completely depolarized, are in the process of repolarizing. Because they have not repolarized to their threshold potential, the cardiac cells cannot be stimulated to depolarize. In other words, myocardial cells cannot contract, and the cells of the electrical conduction system cannot conduct an electrical impulse during the absolute refractory period.
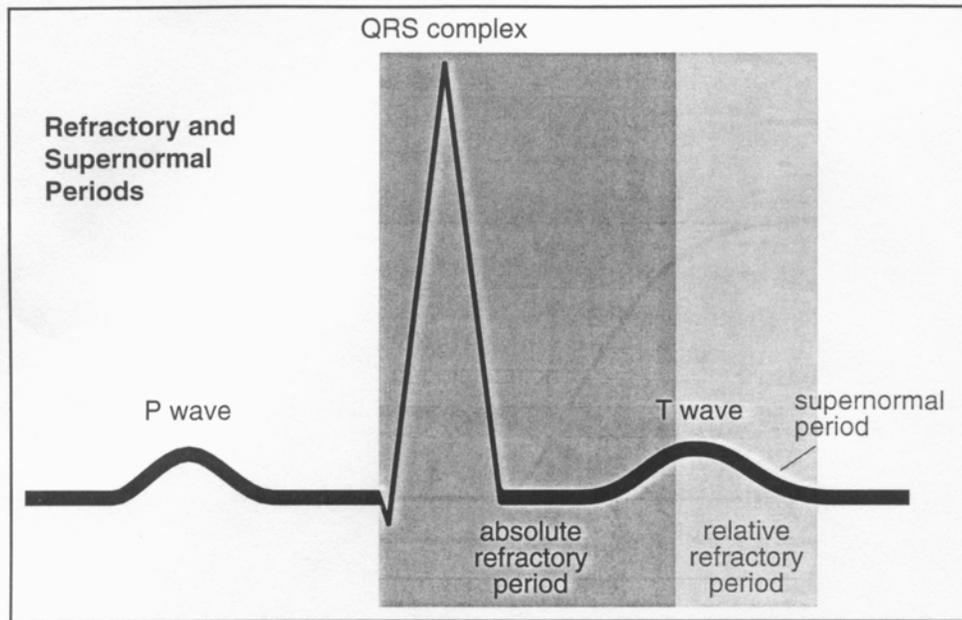
Figure 1-12 Refractory and supernormal periods.

The relative refractory period (RRP) extends through most of the second half of phase 3, corresponding to the downslope of the T wave. During this period, the cardiac cells, having repolarized to their threshold potential, can be stimulated to depolarize if the stimulus is strong enough. This period is also called the *vulnerable period of repolarization.*

During a short portion of phase 3 near the end of the T wave, just before the cells return to their resting potential, a stimulus weaker than is normally required can depolarize cardiac cells. This portion of repolarization is called the *supernormal period.*

Excitability and Automaticity

The capability of a resting, polarized cardiac cell to depolarize in response to an electrical stimulus is called the *property of excitability.* All cardiac cells have this property.

The capability of a cardiac cell to depolarize spontaneously during phase 4—to reach threshold potential and to depolarize completely without being externally stimulated—is called the *property of automaticity.* (This could also be called the *property of self-excitation.*)

Spontaneous depolarization depends on the ability of the cell membrane to become permeable to sodium during phase 4, thus allowing a steady leakage of sodium ions into the cell. This causes the resting membrane potential to become progressively less negative. As soon as the threshold potential is reached, rapid depolarization of the cell (phase 0) occurs.

The rate of spontaneous depolarization is dependent on the slope of phase 4 depolarization (Figure 1-13). The steeper the slope of phase 4 depolarization, the faster is the rate of spontaneous depolarization and the rate of impulse formation (the firing rate). The flatter the slope is, the slower the firing rate.

Certain of the specialized cells in the electrical conduction system normally have the property of automaticity. These cells, the pacemaker cells, are located in the SA node, in some areas of the internodal atrial conduction tracts and AV node, and throughout the bundle of His, bundle branches, and Purkinje network. The pacemaker cells of the SA node, having the fastest firing rate, are normally the dominant (or primary) pacemaker cells of the heart. The pacemaker cells in the rest of the electrical conduction system hold the property of automaticity in reserve should the SA node fail to function properly or electrical impulses fail to reach them for any reason, such as a disruption in the electrical conduction system. For this reason, these pacemaker cells are called *latent* (or *subsidiary* or *escape*) *pacemaker cells.* Myocardial cells, which do not normally have the capability to depolarize spontaneously during phase 4, are called *non-pacemaker cells.*

Increase in sympathetic activity and administration of catecholamines increase the slope of phase 4 depolarization, resulting in an increase in the automaticity of the pacemaker cells and their firing rate. On the other hand, an increase in parasympathetic activity and administration of such drugs as lidocaine, procainamide, and quinidine decrease the slope of phase 4 depolarization, causing a decrease in

Conduction through the His-Purkinje system precedes ventricular contraction. The electrical impulse spreads rapidly through the right and left bundle branches and the Purkinje fibers to reach the ventricular muscle. The Purkinje fibers are very large and transmit impulses at a speed about 6 times faster than that in the usual cardiac muscle and 150 times faster than that in some portions of the AV fibers.[7]

The electrical impulse spreads from the endocardium to the myocardium, finally reaching the epicardial surface. The ventricular walls are stimulated to contract in a twisting motion that wrings blood out of the ventricular chambers and forces it into arteries. The Purkinje fibers have an intrinsic pacemaker ability of 20 to 40 beats/min (Figure 2-17).

A summary of the conduction system can be found in Table 2-2.

CAUSES OF DYSRHYTHMIAS

Enhanced Automaticity

Enhanced automaticity is an abnormal condition in which cardiac cells not normally associated with the property of automaticity begin to depolarize spontaneously or when escape pacemaker sites increase their firing rate beyond that which is considered normal. Enhanced automaticity may occur as the result of catecholamine administration (epinephrine), administration of atropine sulfate, digitalis toxicity, acidosis, alkalosis, hypoxia, myocardial ischemia or infarction, hypokalemia, or hypocalcemia.

Examples of rhythms associated with enhanced automaticity include atrial flutter; atrial fibrillation; supraventricular tachycardia; premature atrial, junctional, or ventricular complexes; ventricular tachycardia or ventricular fibrillation; junctional tachycardia; accelerated idioventricular rhythm, and accelerated junctional rhythm.

Reentry

Reentry is the propagation of an impulse through tissue already activated by that same impulse.[1] An electrical impulse is delayed or blocked (or both) in one or more divisions of the electrical conduction system while the impulse is conducted normally through the rest of the conduction system. This results in the delayed electrical impulse entering cardiac cells that have just been depolarized by the normally conducted impulse (Figure 2-18).

If the area the delayed impulse stimulates is relatively refractory, the impulse can cause depolarization of those cells, producing a single premature beat or repetitive electrical impulses, resulting in short periods of tachydysrhythmias. Common causes of reentry

Reentry: Propagation of an impulse through tissue already activated by that same impulse.
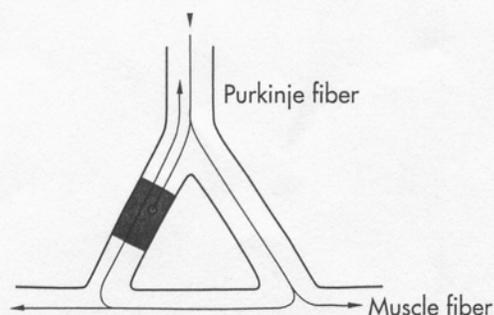

FIGURE **2-18**
Reentry.