highest rate of spontaneous impulse formation. With its faster rate of electrical discharge or depolarization than other parts of the conduction system, the SA node serves as pacemaker and controls heart rate and rhythm.

Initiation of an electrical impulse depends on the movement of sodium and calcium ions into a myocardial cell and movement of potassium ions out of the cell. Normally, the cell membrane becomes more permeable to sodium and opens pores or channels to allow its rapid movement into the cell. Calcium ions follow sodium ions into the cell at a slower rate. As sodium and calcium ions move into cells, potassium ions move out of cells. The movement of the ions changes the membrane from its resting state of electrical neutrality to an activated state of electrical energy buildup. When the electrical energy is discharged (depolarization), muscle contraction occurs.

Some cells in the cardiac conduction system depolarize in response to the entry of calcium ions rather than entry of sodium ions. In these calcium-respondent cells, which are found mainly in the SA and atrioventricular (AV) nodes, the electrical impulse is conducted more slowly and recovery of excitability takes longer than in sodium-respondent cells. Overall, activation of the SA and AV nodes depends on a slow depolarizing current through calcium channels, and activation of the atria and ventricles depends on a rapid depolarizing current through sodium channels. These two types of conduction tissues are often called slow and fast channels, respectively, and they differ markedly in their responses to drugs that affect conduction of electrical impulses.

The ability of a cardiac muscle cell to respond to an electrical stimulus is called *excitability* or *irritability*. The stimulus must reach a certain intensity or threshold to cause contraction. After contraction, sodium and calcium ions return to the extracellular space, potassium ions return to the intracellular space, muscle relaxation occurs, and the cell prepares for the next electrical stimulus and contraction.

Following contraction there is also a period of decreased excitability (called the *absolute refractory period*) during which the cell cannot respond to a new stimulus. Before the resting membrane potential is reached, a stimulus greater than normal can evoke a response in the cell. This period is called the *relative refractory period.*

## Conductivity

Conductivity is the ability of cardiac tissue to transmit electrical impulses. Although the electrophysiology of a single myocardial cell can assist understanding of the process, the orderly, rhythmic transmission of impulses to all cells is needed for effective myocardial contraction.

Normally, electrical impulses originate in the SA node and are transmitted to atrial muscle, where they cause atrial contraction, and then to the AV node, bundle of His, bundle branches, Purkinje fibers, and ventricular muscle, where they cause ventricular contraction. The cardiac conduction system is shown in Figure 52–1.

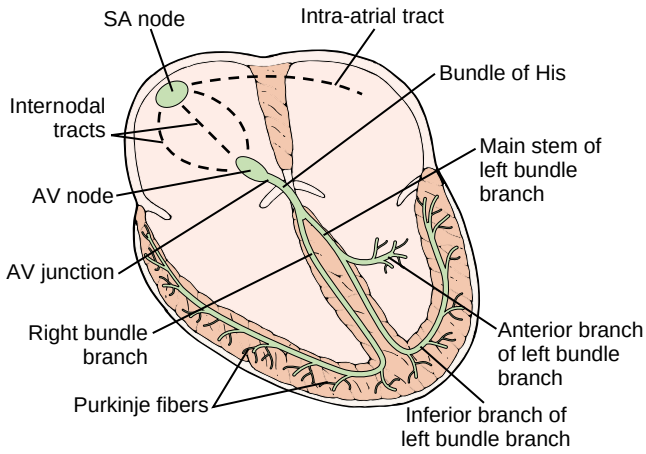


**Figure 52–1** The conducting system of the heart. Impulses originating in the SA node are transmitted through the atria, into the AV node to the bundle of His, and by way of Purkinje fibers through the ventricles.

## CARDIAC DYSRHYTHMIAS

Cardiac dysrhythmias can originate in any part of the conduction system or from atrial or ventricular muscle. They result from disturbances in electrical impulse formation (automaticity), conduction (conductivity), or both. The characteristic of automaticity allows myocardial cells other than the SA node to depolarize and initiate the electrical impulse that culminates in atrial and ventricular contraction. This may occur when the SA node fails to initiate an impulse or does so too slowly. When the electrical impulse arises anywhere other than the SA node, it is an abnormal or ectopic focus. If the ectopic focus depolarizes at a rate faster than the SA node, the ectopic focus becomes the dominant pacemaker. Ectopic pacemakers may arise in the atria, AV node, Purkinje fibers, or ventricular muscle. They may be activated by hypoxia, ischemia, or hypokalemia. Ectopic foci indicate myocardial irritability (increased responsiveness to stimuli) and potentially serious impairment of cardiac function.

A common mechanism by which abnormal conduction causes dysrhythmias is called *reentry excitation.* During normal conduction, the electrical impulse moves freely down the conduction system until it reaches recently excited tissue that is refractory to stimulation. This causes the impulse to be extinguished. The SA node then recovers, fires spontaneously, and the conduction process starts over again. Reentry excitation means that an impulse continues to reenter an area of the heart rather than becoming extinguished. For this to occur, the impulse must encounter an obstacle in the normal conducting pathway. The obstacle is usually an area of damage, such as myocardial infarction. The damaged area allows conduction in only one direction and causes a circular movement of the impulse (Fig. 52–2).

Dysrhythmias may be mild or severe, acute or chronic, episodic or relatively continuous. They are clinically significant if they interfere with cardiac function (ie, the heart's abil-