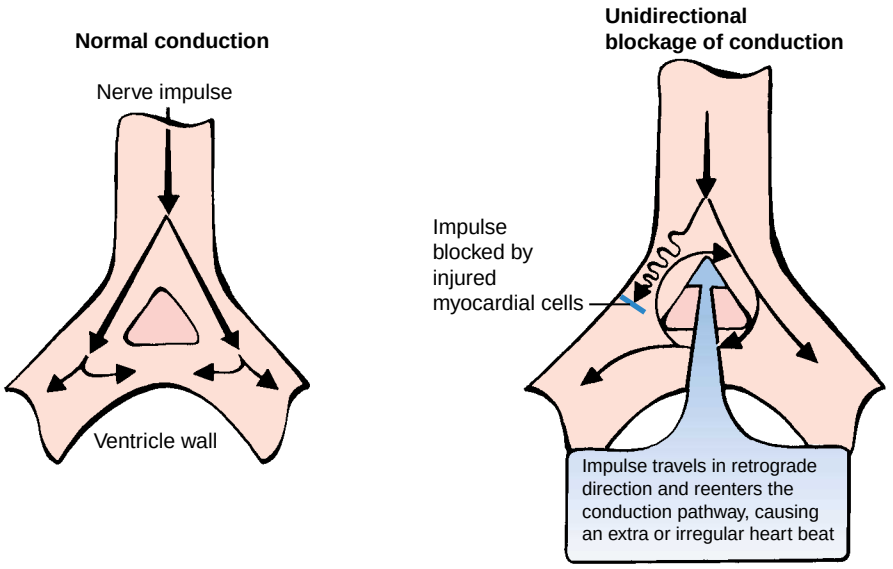

Figure 52–2 Reentry excitation of dysrhythmias.

ity to pump sufficient blood to body tissues). The normal heart can maintain an adequate cardiac output with ventricular rates ranging from 40 to 180 beats per minute. The diseased heart, however, may not be able to maintain an adequate cardiac output with heart rates below 60 or above 120. Dysrhythmias are usually categorized by rate, location, or patterns of conduction. Common types of dysrhythmias are described in Box 52–1.

ANTIDYSRHYTHMIC DRUGS

Antidysrhythmic drugs alter the heart's electrical conduction system. Atropine for bradydysrhythmias is discussed in Chapter 21; digoxin and its use in treating atrial fibrillation are discussed in Chapter 51. The focus of this chapter is the drugs used for tachydysrhythmias. These drugs are described in the following sections and listed in Drugs at a Glance: Antidysrhythmic Drugs.

Clinical use of antidysrhythmic drugs for tachydysrhythmias has undergone significant changes. One change is that the goal of drug therapy is to prevent or relieve symptoms or prolong survival, not just suppress dysrhythmias. This change resulted from studies in which clients treated for some dysrhythmias had a higher mortality rate than clients who did not receive antidysrhythmic drug therapy. The higher mortality rate was attributed to prodysrhythmic effects (ie, worsening existing dysrhythmias or causing new dysrhythmias). Overall, there is decreasing use of class I drugs (eg, quinidine) and increasing use of class II (beta blockers) and class III drugs (eg, amiodarone).

Another change is the greater use of nonpharmacologic management of dysrhythmias. These methods include destroying dysrhythmogenic foci in the heart with radio waves (radiofrequency catheter ablation) or surgical procedures and implanting devices for sensing, cardioverting, defibrillating, or pacing (eg, the implantable cardioverter–defibrillator or ICD).

Indications for Use

Antidysrhythmic drug therapy commonly is indicated in the following conditions:

1. To convert atrial fibrillation (AF) or flutter to normal sinus rhythm (NSR)
2. To maintain NSR after conversion from AF or flutter
3. When the ventricular rate is so fast or irregular that cardiac output is impaired. Decreased cardiac output leads to symptoms of decreased systemic, cerebral, and coronary circulation.
4. When dangerous dysrhythmias occur and may be fatal if not quickly terminated. For example, ventricular tachycardia may cause cardiac arrest.

Mechanisms of Action

Drugs used for rapid dysrhythmias mainly *reduce automaticity* (spontaneous depolarization of myocardial cells, including ectopic pacemakers), *slow conduction* of electrical impulses through the heart, and *prolong the refractory period* of myocardial cells (so they are less likely to be prematurely activated by adjacent cells). Several different groups of drugs perform one or more of these actions. They are classified according to their mechanisms of action and effects on the conduction system, even though they differ in other respects. Additionally, some drugs have characteristics of more than one classification.

CLASSIFICATIONS AND INDIVIDUAL DRUGS

Class I Sodium Channel Blockers

Class I drugs block the movement of sodium into cells of the cardiac conducting system. This results in a membrane-