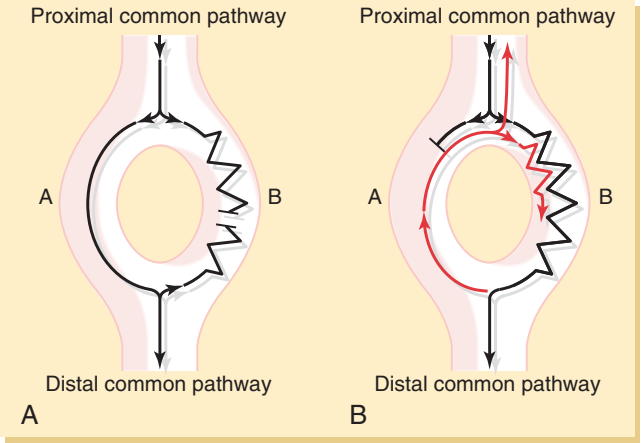

FIGURE 9-2 Mechanism of reentry. Reentry requires two distinct pathways with different refractoriness and a region of slowed conduction. One pathway *(A)* has normal rapid conduction but a long refractory period. The second pathway *(B)* has slowed conduction but a relatively shorter refractory period. To initiate reentry, conduction must fail down one pathway in the antegrade direction but then permit later retrograde reactivation of this pathway. This is referred to as a *unidirectional block*. A fixed or functional obstacle must maintain separation of the two pathways. Although drawn schematically as a circular loop, the anatomy of circuits is often complex and circuitous and is different in different arrhythmia mechanisms. **A,** In normal rhythm, the circuit is activated in an antegrade direction down both pathways. However, because of slowed conduction in the *B* limb, distal activation is mediated by the faster *A* pathway, which arrives first and may activate the slowly conducting pathway in a retrograde direction. This retrograde conduction is electrocardiographically concealed (invisible), collides with the antegrade wave front, and is extinguished, and no tachycardia results. **B,** Reentry is usually initiated by a premature beat originating independently of the circuit. The premature beat fails to propagate down the rapidly conducting *A* limb due to differential refractoriness of the two limbs, but it is able to propagate down the slowly conducting *B* pathway, where it may encounter substantial delay due to increased conduction time with prematurity (i.e., decremental conduction), allowing recovery of the previously blocked rapidly conducting *A* limb. This permits the rapidly conducting *A* limb to act as a return path and for ultimate reentrant reactivation of the slowly conducting *B* pathway, initiating sustained reentrant tachycardia in the circuit.

abnormalities may point to susceptibility to symptomatic bradycardia. Delta waves confirm an accessory pathway and direct the evaluation of arrhythmic symptoms toward the diagnosis of Wolff-Parkinson-White (WPW) syndrome while localizing the accessory pathway.

Evidence for hereditary cardiomyopathies and cardiac ion channel disorders that predispose to sudden death may be detected on a baseline ECG. Patients with arrhythmogenic right ventricular (RV) dysplasia may have epsilon waves and inverted T waves in the right precordial leads. QT interval prolongation or shortening may indicate congenital or acquired long QT or short QT syndrome, respectively. Brugada syndrome can be diagnosed based on coved ST-segment elevation in leads V_1 and V_2.

A 12-lead ECG obtained during arrhythmic symptoms can establish the cause of a patient's symptoms. A symptom ECG is most useful in discriminating between SVT and VT. The specific mechanism underlying narrow complex tachycardia can often be inferred from the ECG. Documentation of QRS morphology during VT on a 12-lead ECG aids in localizing the site of origin, identifying the VT mechanism, and guiding catheter ablation.

Ambulatory Monitoring

Although a 12-lead ECG obtained during arrhythmic symptoms is ideal, it is difficult to obtain in practice because of the transient and intermittent nature of these symptoms. Ambulatory recording devices permit electrocardiographic monitoring over longer periods to establish symptom-rhythm correlations.

Three types of monitoring devices are available. *Holter monitors* provide continuous electrogram storage for 24 to 48 hours. Holter monitoring is helpful for patients with frequent symptoms. The prolonged sampling period provides useful information about heart rate variability, rate control with atrial fibrillation (AF), AF burden, asymptomatic arrhythmias, and the frequency of ventricular ectopy.

External event monitors or loop recorders, which can be worn for 30 days, store electrograms when triggered by patients for symptoms or are autoactivated based on heart rate detection above or below a programmed threshold value. Some external loop monitors have algorithms to detect AF. Episode storage varies from seconds to minutes. After events are recorded, patients transmit the data by telephone. External loop recorders are intended to identify cardiac rhythm disturbances underlying infrequent symptoms.

For patients with arrhythmia symptoms occurring less than once per month, *implantable loop recorders* may be useful. These small devices implanted in a subcutaneous pocket in the left chest record patient-triggered and autotriggered ECGs based on programmed heart rate parameters. With a 3-year anticipated battery longevity, implantable loop recorders are valuable in establishing the cause of recurrent infrequent syncope.

Electrophysiologic Testing

To perform electrophysiologic studies, temporary transvenous pacing catheters are positioned in multiple locations in the heart, permitting pacing and recording of intracardiac electrograms. Catheters are typically placed in the right atrium, the right ventricle, close to the bundle of His, and in the coronary sinus for left atrial recording and pacing. Electrophysiologic studies can define the mechanism of tachyarrhythmias and guide therapy. In patients with prior MI, induction of VT may assist in determining patient susceptibility to life-threatening arrhythmias and inform decisions regarding defibrillator implantation. Electrophysiologic testing also can evaluate sinus node function and AV conduction.

Pharmacologic Therapy

Antiarrhythmic drugs are traditionally divided according to the Singh–Vaughan Williams classification, which categorizes agents based on their primary physiologic effect (Table 9-1). When this classification system was first proposed, knowledge of electrophysiologic mechanisms was limited. Although the simplicity of categorizing antiarrhythmic drugs according to Singh–Vaughan Williams classes I through IV is appealing, the system has many limitations. As hybrid classifications, class I and III agents block ion channels, and class II and IV drugs block receptors. Some drugs cross classes and have several mechanisms of action. There