inactivation is time dependent. The fast-response cells found in atrial myocytes, ventricular myocytes, and the His-Purkinje system display slow phase 4 depolarization and do not typically display automaticity. The resting E_m is more negative, and the fast Na^+ current drives rapid phase 0 depolarization and rapid conduction. Recovery from inactivation in these cells is voltage dependent.

The sinus node typically displays the fastest phase 4 depolarization. Other cardiac tissues have the capacity to depolarize spontaneously, and subsidiary pacemakers may take over when sinus rates slow and under conditions of increased automaticity. Typically, the AV node, located above the AV ring, serves as the heart's secondary pacemaker, with a spontaneous rate of depolarization of 40 to 50 beats per minute. Automaticity of cardiac myocytes is increased when the slope of phase 4 depolarization increases, with a shift of threshold potentials to more negative values, or in the presence of more positive maximal diastolic potentials.

The sinus node is the primary intrinsic pacemaker, and spontaneous depolarization leads to action potential generation, with normal resting rates of 60 to 100 beats per minute. Depolarization then spreads through the atria to the AV node, where conduction slows, introducing a delay between atrial and ventricular activation, and then to the His-Purkinje system fibers, which originate at the AV node with the bundle of His and split to form the left bundle branch and the right bundle branch, rapidly conducting depolarization to the ventricular myocardium. Cardiac myocytes are joined by electrical synapses called *gap junctions*, which permit the flow of intracellular current from cell to cell.

Classification of Arrhythmias

Mechanistically, cardiac arrhythmias can be broadly divided into disorders of action potential formation and disorders of impulse conduction. Clinically, arrhythmias are classified as bradycardias and tachycardias, with further categorization according to arrhythmia origin. This information is used to guide evaluation and management strategies.

Electrophysiologic Mechanisms of Arrhythmias

Automaticity is a normal function of pacemaker cells, occurring during phase 4 depolarization. *Enhanced automaticity* occurs when pacemaker cells depolarize at a faster rate due to an increased slope of phase 4 depolarization, a shift of threshold potential to a more negative value, or a shift of the maximal diastolic potential to a more positive value. These changes may occur with sympathetic stimulation. Enhanced automaticity may be normal (e.g., appropriate sinus tachycardia) or abnormal (e.g., inappropriate sinus tachycardia). Spontaneous depolarization occurring in nonpacemaker cardiac myocytes is called *abnormal automaticity*. Conditions as ischemia, electrolyte abnormalities, and sympathetic stimulation may produce abnormal automaticity. Premature atrial and ventricular depolarizations, atrial tachycardia, and ventricular tachycardia (VT) may result.

Triggered activity occurs when secondary cardiac depolarizations are initiated by prior depolarizations. If these secondary depolarizations reach threshold potential, they may generate action potentials during or immediately after phase 3 of the action potential. *Early afterdepolarizations* (EADs) are observed when triggered depolarization occurs during phase 3 of the action potential. Inciters of EADs include QT-prolonging drugs, hypokalemia, and bradycardia. Patients with congenital long QT syndrome (LQTS) are prone to develop EADS, resulting in *torsades de pointes* (TdP).

When triggered activity occurs during phase 4, *delayed afterdepolarizations* (DADs) result. DADs are exaggerated at rapid heart rates and observed with digoxin toxicity and high-level catecholamine states, conditions that are associated with intracellular calcium overload. DADs are thought to be the chief arrhythmic mechanism underlying catecholaminergic polymorphic VT (CPVT).

Reentry is the dominant mechanism underlying clinical tachyarrhythmias. Reentry describes the reexcitation of a localized region of cardiac tissue by the same impulse, requiring bifurcating conduction pathways with different velocities and refractory periods. To permit reentry, unidirectional block in one pathway and slowed conduction in the other are required. Reentry is further categorized as anatomic, circling around a fixed anatomic obstacle, or functional, in which the inexcitable center of a reentrant circuit is not fixed but functionally refractory. Figure 9-2 illustrates reentry as an arrhythmic mechanism. The two pathways join proximally and distally. Pathway *A* conducts rapidly but has a long refractory period. Pathway *B* is slowly conducting but has a shorter refractory period. A normally timed impulse enters the two pathways through the proximal common pathway, conducting rapidly down *A* and slowly down *B*. As the impulse from pathway *A* reaches the distal common pathway, while continuing distally, it may also turn around to activate *B* retrogradely. This impulse collides with the slowly conducting antegrade impulse in pathway *B*, extinguishing the impulse. However, a sufficiently premature stimulus may enter the proximal common pathway, finding pathway *A* with its long refractory period inexcitable, traveling slowly down pathway *B*, and finally reaching the distal common pathway. Due to the slow conduction velocity in pathway *B*, pathway *A* may no longer be refractory, and the impulse may successfully travel retrograde up pathway *A*, potentially repeatedly activating the circuit. Reentry is the most common mechanism producing supraventricular tachycardia (SVT) and VT.

For a deeper discussion on this topic, please see Chapter 61, "Principles of Electrophysiology," in Goldman-Cecil Medicine*, 25th Edition.*

GENERAL APPROACH TO MANAGEMENT

Diagnostic Procedures

Electrocardiography

The baseline 12-lead electrocardiogram (ECG) is essential for the initial evaluation of patients with arrhythmic symptoms. The baseline ECG may indicate an underlying structural heart disease, with Q waves or fractionated QRS complexes suggesting prior myocardial infarction (MI). Slow sinus rates or AV conduction