therapy. Class I sodium channel–blocking agents (e.g., flecainide, propafenone, disopyramide) are options for subjects without significant structural heart disease, but they have negative inotropic and proarrhythmic effects that warrant avoidance in patients with coronary artery disease or heart failure. The class III agents sotalol and dofetilide can be administered to patients with coronary artery disease or structural heart disease but have approximately a 3% risk of inducing excessive QT prolongation and torsades des pointes. Dofetilide should be initiated only in a hospital with ECG monitoring, and many physicians take this approach with sotalol as well. Dronedarone increases mortality in patients with heart failure. All of these agents have modest efficacy in patients with paroxysmal AF, of whom approximately 30–50% will benefit. Amiodarone is more effective, maintaining sinus rhythm in approximately two-thirds of patients. It can be administered to patients with heart failure and coronary artery disease. Over 20% of patients experience toxicities during long-term therapy.

### CATHETER AND SURGICAL ABLATION FOR ATRIAL FIBRILLATION

Catheter ablation avoids antiarrhythmic drug toxicities but has procedural risks and requires an experienced center. For patients with previously untreated but recurrent paroxysmal AF, catheter ablation has similar efficacy to antiarrhythmic drug therapy and is superior to antiarrhythmic drugs for patients who have recurrent AF despite drug treatment. The procedure involves cardiac catheterization, transatrial septal puncture, and radiofrequency ablation or cryoablation to electrically isolate the regions around the pulmonary veins, abolishing the effect of triggering foci to interact with the left atrial AF substrate. Extensive areas of ablation are required, and gaps in healed ablation areas necessitate a repeat procedure in 20–50% of patients. Sinus rhythm is maintained for more than 1 year after one procedure in approximately 60% of patients and in 70–80% of patients after multiple procedures. Some patients become more responsive to antiarrhythmic drugs.

There is a 2–7% risk of major complications, including stroke (0.5–1%), cardiac tamponade (1%), phrenic nerve paralysis, bleeding from femoral access sites, and fluid overload with heart failure, that can emerge 1–3 days after the procedure. It is important to recognize the potential for delayed presentation of some complications. Ablation within the pulmonary veins can lead to pulmonary vein stenosis, presenting weeks to months after the procedure with dyspnea or hemoptysis. Esophageal ulcers can form immediately after the procedure and may rarely lead to a fistula between the left atrium and esophagus (estimated incidence of 0.1%) that presents as endocarditis and stroke 10 days to 3 weeks after the procedure.

Catheter ablation is less effective for persistent AF. More extensive ablation is often required, including areas that likely support reentry in regions outside the pulmonary venous antra, but individual strategies are debated. More than one ablation procedure is often required to maintain sinus rhythm.

Surgical ablation of AF is typically performed concomitant with cardiac valve or coronary artery surgery and less commonly as a stand-alone procedure; however, for patients with persistent AF, surgical or hybrid procedures may have higher single-procedure efficacy. Risks include sinus node injury requiring pacemaker implantation. Surgical removal of the left atrial appendage may reduce stroke risk, although thrombus can form in the remnant of the appendage or if the appendage is not completely ligated.

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# 277 Ventricular Arrhythmias

Roy M. John, William G. Stevenson

Arrhythmias that originate in the ventricular myocardium or His-Purkinje system include premature ventricular beats, ventricular tachycardias that can be sustained or nonsustained, and ventricular fibrillation. Arrhythmia may emerge from a focus of myocardial or Purkinje cells capable of automaticity, or triggered automaticity, or from reentry through areas of scar or a diseased Purkinje system. Ventricular arrhythmias are often associated with structural heart disease and are an important cause of sudden death (Chap. 327). They also occur in some structurally normal hearts, in which case they are usually benign. Evaluation and management are guided by the risk of arrhythmic death, which is assessed based on symptoms, type of arrhythmia, and associated underlying heart disease.



### DEFINITIONS

Ventricular arrhythmias are characterized by their electrocardiographic appearance and duration. Conduction away from the ventricular focus through the ventricular myocardium is slower than activation of the ventricles over the Purkinje system. Hence, the QRS complex during ventricular arrhythmias will be wide, typically >0.12 s.

*Premature ventricular beats* (also referred to as *premature ventricular contractions* [PVCs]) are single ventricular beats that fall earlier than the next anticipated supraventricular beat (Fig. 277-1). PVCs that originate from the same focus will have the same QRS morphology and are referred to as unifocal (Fig. 277-1*A*). PVCs that originate from different ventricular sites have different QRS morphologies and are referred to as multifocal (Fig. 277-1*B*). Two consecutive ventricular beats are *ventricular couplets*.

*Ventricular tachycardia* (VT) is three or more consecutive beats at a rate faster than 100 beats/min. Three or more consecutive beats at slower rates are designated an *idioventricular rhythm* (Fig. 277-1*C*). VT that terminates spontaneously within 30 s is designated *nonsustained* (Fig. 277-2), whereas *sustained VT* persists longer than 30 s or is terminated by an active intervention, such as administration of an intravenous medication, external cardioversion, or pacing or a shock from an implanted cardioverter-defibrillator.

*Monomorphic VT* has the same QRS complex from beat to beat, indicating that the activation sequence is the same from beat to beat and that each beat likely originates from the same source (Fig. 277-3*A*). The initial site of ventricular activation largely determines the sequence of ventricular activation. Therefore, the QRS morphology of PVCs and monomorphic VT provides an indication of the site of origin within the ventricles (Fig. 277-4). The likely origin often suggests whether an arrhythmia is idiopathic or associated with structural disease. Arrhythmias that originate from the right ventricle or septum result in late activation of much of the left ventricle, thereby producing a prominent S wave in $V_1$ referred to as a left bundle branch block–like configuration. Arrhythmias that originate from the free wall of the left ventricle have a prominent positive deflection in $V_1$, thereby producing a right bundle branch block–like morphology in $V_1$. The frontal plane axis of the QRS is also useful. An axis that is directed inferiorly, as indicated by dominant R waves in leads II, III, and AVF, suggests initial activation of the cranial portion of the ventricle, whereas a frontal plane axis that is directed superiorly (dominant S waves in II, III, and AVF) suggests initial activation at the inferior wall.

Very rapid monomorphic VT has a sinusoidal appearance, also called *ventricular flutter*, because it is not possible to distinguish the QRS complex from the T wave (Fig. 277-3*B*). Relatively slow *sinusoidal* VTs have a wide QRS indicative of slowed ventricular conduction (Fig. 277-3*C*). Hyperkalemia, toxicity from excessive effects of drugs that blocks sodium channels (e.g., flecainide, propafenone, or