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259 Approach to Ventricular Arrhythmias

TABLE 258-3 Recommendations for Catheter Ablation in Patients with Atrial Fibrillation (AF) LEVEL OF EVIDENCE RECOMMENDATIONS CLASS

A In patients with symptomatic AF in whom antiarrhythmic drugs have been ineffective, contraindicated, not tolerated or not preferred, and continued rhythm control is desired, catheter ablation is useful to improve symptoms. PART 6 Disorders of the Cardiovascular System

A In selected patients (generally younger with few comorbidities) with symptomatic paroxysmal AF in whom rhythm control is desired, catheter ablation is useful as first-line therapy to improve symptoms and reduce progression to persistent AF.

A In patients with symptomatic or clinically significant atrial flutter, catheter ablation is useful for improving symptoms. 2a B In patients who are undergoing ablation for AF, ablation of additional clinically significant supraventricular arrhythmias can be useful to reduce the likelihood of future arrhythmia. 2a B In patients (other than younger with few comorbidities) with symptomatic paroxysmal or persistent AF who are being managed with a rhythm-control strategy, catheter ablation as first-line therapy can be useful to improve symptoms. Source: Reproduced with permission from JA Joglar et al: 2023 ACC/AHA/ACCP/ HRS Guideline for the Diagnosis and Management of Atrial Fibrillation: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol 83:109, 2024. of atrioesophageal fistula is important because delayed diagnosis leads to likely death. Diagnosis is made by chest CT scan with water-soluble oral and IV contrast. Endoscopy should be avoided in patients with a suspected fistula because of the risk of air/ esophageal fluid embolus. Definitive repair of the atrioesophageal fistula with emergent surgery is required. Pulsed-field ablation is associated with a significantly reduced risk of pulmonary vein stenosis, phrenic nerve injury, and left atrial esophageal fistula. Surgical ablation of AF is most frequently performed concomitant with cardiac valve or coronary artery surgery and less commonly as a stand-alone procedure. There is no significant difference in the rate of freedom from AF between patients who undergo pulmonary vein isolation and those who undergo the biatrial maze procedure. However, for patients with persistent AF, surgical or hybrid procedures (a combination of a surgical and catheter-based approach, most often in separate procedures) appear to have comparable efficacy to catheter ablation. Risks include sinus node injury requiring pacemaker implantation and higher morbidity with surgical ablation. 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. **RISK FACTORS FOR AND LIFESTYLE IMPACT ON ATRIAL FIBRILLATION** There is strong evidence that AF is associated with a sedentary lifestyle, obesity, hypertension, smoking, alcohol use, and sleep apnea. Aggressive treatment of these risk factors can substantially reduce AF episodes in some patients and is warranted in all patients, as additional benefits to the patient are likely beyond AF improvement. The amount of exercise appears to have a complex relationship with the risk of AF development. In males, a U-shaped curve exists, where AF risk is high among those with sedentary lifestyles and those who participate extensively in endurance athletics such as long-distance running or cycling. Moderate exercise appears to confer a lower risk of AF. On the other hand, in females, a linear relationship exists between exercise and AF risk, with risk of AF decreasing continuously with increasing exercise activity. Although caffeine intake is often invoked as a risk for AF development or as a trigger for AF episodes in patients with a known AF diagnosis, large cohort studies have demonstrated, in contrast, a modest decrease in AF risk with modest caffeine intake. Other proposed risk factors are

being evaluated, including psychological stress. Genetic predisposition to AF is seen in those with first-degree relatives with AF, and a small subset of AF patients can be determined have a familial form of AF. There is emerging emphasis on an integrated approach to management of AF patients, with coordinated management of risk factor modification, stroke prevention, rate control, rhythm control, and management of associated comorbidities of critical importance. The previous classification of AF, which was based only on arrhythmia duration (i.e., paroxysmal, persistent, and long-standing persistent), although useful, tended to emphasize therapeutic interventions. A more recent classification using four stages (i.e., at risk of AF, pre-AF, AF, and permanent AF) has been proposed and recognizes AF as a disease continuum that requires a variety of strategies at the different stages, including prevention, lifestyle and risk factor modification, screening, and therapy.

■ ■ **FURTHER READING** Joglar JA et al: 2023 ACC/AHA/ACCP/HRS Guideline for the Diagnosis and Management of Atrial Fibrillation: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol* 83:109, 2024. Packer DL et al: Effect of catheter ablation vs antiarrhythmic drug therapy on mortality, stroke, bleeding, and cardiac arrest among patients with atrial fibrillation: The CABANA randomized clinical trial. *JAMA* 321:1261, 2019. William H. Sauer, Usha B. Tedrow

Approach to Ventricular Arrhythmias There are myriad types of ventricular arrhythmias (VAs), ranging from benign to life-threatening, occurring both in patients with normal hearts and in those with structural heart disease. An understanding of an approach to these arrhythmias is critical to being appropriately parsimonious with benign forms, while understanding timely workup and management of the malignant forms. **TYPES OF VAs** VAs can arise from focal sites of origin or from reentrant circuits. Focal VAs can originate from myocardial or specialized Purkinje cells capable of automaticity or triggered activity. Reentrant VAs often involve areas of scar such as old myocardial infarction or a cardiomyopathic process. Less commonly, diseased Purkinje conduction pathways can also result in reentrant circuits. VAs are characterized by their electrocardiographic appearance and duration. Conduction away from the ventricular focus or reentrant circuit exit, propagating through the ventricular myocardium, is slower than activation of the ventricles over the normal Purkinje system. For this reason, the QRS complex duration during VAs will be wide, typically >0.12 s, though there are unusual situations that can arise with narrow QRS duration as well. Premature ventricular beats (also referred to as premature ventricular contractions or

premature ventricular complexes [PVCs]) are single ventricular beats that occur earlier than the next anticipated supraventricular beat (Fig. 259-1). PVCs that originate from the same focus will have the same QRS morphology and are referred to as unifocal (Fig. 259-1A). PVCs that originate from different ventricular sites have different QRS morphologies and are referred to as multifocal (Fig. 259-1B). 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

1000 ms I Art. Pr. A B C FIGURE 259-1 A. Unifocal premature ventricular contractions (PVCs) at bigeminal frequency. Trace shows electrocardiogram lead 1 and arterial pressure (Art. Pr.). Sinus rhythm beats are followed by normal arterial waveform. The arterial pressure following premature beats is attenuated (arrows) and imperceptible to palpation. The pulse in this patient is registered at half the heart rate. B. Multifocal PVCs. The two PVCs shown have different morphologies. C. Example of accelerated idioventricular rhythm. (See text for details.) slower rates are designated an idioventricular rhythm. VT that terminates spontaneously within 30 s is designated nonsustained, whereas sustained VT persists for >30 s or is terminated by an active intervention, such as administration of an intravenous medication, external I II aVR V1 V4 aVL III aVF V1 II
FIGURE 259-2 Repetitive monomorphic nonsustained ventricular tachycardia (VT) of right ventricular outflow tract origin. The VT has a left bundle branch block pattern with inferior axis with tall QRS complexes in the inferior leads.

CHAPTER 259 Approach to Ventricular Arrhythmias cardioversion, antitachycardia pacing, or a shock from an implanted cardioverter-defibrillator (Fig. 259-2). Monomorphic VT has the same QRS complex from beat to beat, indicating that the activation sequence is the same from beat to beat V5 V2 V3 V6

and that each beat likely originates from the same source (Fig. 259-3A). 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. 259-4). 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 V1 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 V1, thereby producing a right bundle branch block-like morphology in V1. 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.

PART 6 Disorders of the Cardiovascular System 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. 259-3B). Relatively slow sinusoidal VTs have a wide QRS indicative of slowed ventricular conduction (Fig. 259-3C). Hyperkalemia, toxicity from excessive effects of drugs that block sodium channels (e.g., flecainide, propafenone, or tricyclic antidepressants), and severe global myocardial ischemia are possible causes. Polymorphic VT has a continually changing QRS morphology indicating a changing ventricular activation sequence. Polymorphic VT that occurs in

the context of congenital or acquired prolongation of the QT interval often has a waxing and waning QRS amplitude, creating a characteristic shifting axis referred to as torsades des pointes after the classic ballet sequence (Fig. 259-3D). Ventricular fibrillation (VF) has continuous irregular activation with no discrete QRS complexes. Monomorphic or polymorphic VT may transition to VF in susceptible patients. Cardiac ischemia is the most common cause of VF (Fig. 259-3E). The term idiopathic ventricular arrhythmia generally refers to PVCs or VT that occurs in patients with a normal electrocardiogram (ECG), without structural heart disease, and not associated with an underlying genetic syndrome or risk of sudden death.

CLINICAL MANIFESTATIONS Common symptoms of VAs include palpitations, dizziness, exercise intolerance, episodes of lightheadedness, syncope, or sudden cardiac arrest leading to sudden death if not resuscitated. VAs can also be asymptomatic and encountered unexpectedly as an irregular pulse or heart sounds on examination or may be seen on a routine ECG, exercise test, or cardiac ECG monitoring. Occasionally when every other beat is a PVC (bigeminy), pulse measurements for heart rate can be erroneously low (pseudobradycardia) because the PVCs may not generate a separate pulse wave. Syncope is a concerning symptom, particularly when occurring without prodrome, during exercise, or in the setting of abnormal ECG or structural heart disease. Such episodes can be due to VT that produces severe hypotension, warranting concern for risk of cardiac arrest and sudden death with arrhythmia recurrence. Although benign processes such as reflex-mediated neurocardiogenic (vasovagal) episodes and orthostatic hypotension are the most common causes of syncope, it is important to consider the possibility of underlying heart disease or a genetic syndrome causing VT. When these are suspected, hospitalization for further evaluation and monitoring is often appropriate. Sustained VT may present as a wide QRS complex tachycardia that must be distinguished from supraventricular tachycardia with aberrancy (Chap. 253). Symptoms can be minor but more commonly include hypotension with syncope and even imminent cardiac arrest, particularly in patients with structural heart disease. Sustained VT may degenerate to VF, most commonly if it is rapid and polymorphic. Many patients who are at risk for VT have known heart disease, and many have an implantable cardioverter-defibrillator (ICD). In patients with an ICD, VT episodes may cause transient lightheadedness, palpitations, or syncope followed by a shock from the ICD (see below).

FIGURE 259-3 A. Monomorphic ventricular tachycardia (VT) with dissociated P waves (short arrows). B. Ventricular flutter. C. Sinusoidal VT due to electrolyte disturbance or drug effects. D. Polymorphic VT resulting from prolongation of QT interval (torsade de pointe VT). E. Ventricular fibrillation. (See text for details.)

EVALUATION OF PATIENTS WITH DOCUMENTED OR SUSPECTED VENTRICULAR ARRHYTHMIAS There are several important considerations that guide evaluation of patients with documented or suspected cardiac arrhythmias. First, establish whether a VA is the cause of the symptoms or clinical presentation. Second, determine whether the arrhythmia is associated with a cardiac disease, and establish the prognostic significance of that disease and, in particular, whether it is associated with a risk of sudden cardiac death. Finally, define the likelihood of arrhythmia recurrence and the symptoms and risk imposed by the recurrence. The risks of cardiac arrest and sudden cardiac death are largely determined by the cause of the arrhythmia and the associated underlying heart disease. The diagnosis of VAs can be established by recording the arrhythmia on an ECG, by an ambulatory or implanted cardiac monitor, by an implanted rhythm management device such as a pacemaker or ICD, or in some cases, initiation of the arrhythmia during an electrophysiologic

II III II, III AVF = Inferior axis superior origin LV RV V1 = LBBB Septal or RV origin V1 = RBBB LV origin II III II, III AVF = Superior axis inferior origin

FIGURE 259-4 Site of ventricular tachycardia origin based on QRS morphology. (See text for details.) LBBB, left bundle branch block; LV, left ventricle; RBBB, right bundle branch block; RV, right ventricle.

study. A 12-lead ECG of the arrhythmia should be obtained when possible and often provides clues to the potential site of origin and possible presence of underlying heart disease (see above) (Fig. 259-4). For patients with sustained wide-complex tachycardia, initial management is guided by the patient's hemodynamic stability. The approach to sustained wide-complex tachycardia is discussed in Chap. 261. The management of VT that causes cardiac arrest is discussed in Chap. 317. Once hemodynamic stability is restored, further management is guided by the possibility of a recurrence and the risk imposed by a recurrence.

■ **EVALUATION OF THE PATIENT WITH ARRHYTHMIA SYMPTOMS** When symptoms are intermittent, initial evaluation aims to establish symptom severity, provocative factors, and presence of underlying heart disease. Syncope or near syncope raises concern that an arrhythmia is causing episodes of hypotension and that there may be a risk of cardiac arrest. Symptoms that occur with exertion suggest arrhythmias that are provoked by sympathetic stimulation but can also be related to exertional ischemia in patients with coronary artery disease, although nonarrhythmia causes must also be considered. A past history of any cardiac disease is important. A review of all medications is relevant. Medications that prolong the QT interval predispose to polymorphic VT (Chap. 262). Adrenergic stimulants can provoke PVCs. Family history should determine the presence of premature coronary artery disease, cardiomyopathy, or cardiac arrhythmias, particularly a history of sudden death. Family history may also suggest that the possibility of a genetic cause of an arrhythmia warrants careful consideration. Details of premature deaths are relevant. Sudden death victims are often said to have died of a "massive heart attack" despite absence of definite confirmation of thrombotic myocardial infarction and when other causes such as arrhythmia may have been possible. The physical examination focuses on evidence of structural heart disease with assessment of pulse, jugular venous pressure lung fields, and cardiac auscultation. Stigmata of neuromuscular disease or dysmorphic features may suggest a genetic arrhythmia syndrome.

A 12-lead ECG should be obtained even if the patient is not having symptoms at the time of evaluation. Occasionally, premature ventricular contractions will be detected. Patients with benign idiopathic arrhythmias usually have a completely normal ECG during sinus rhythm. Any ECG abnormality warrants further evaluation. Particularly relevant findings include Q waves that indicate prior myocardial infarction, which may have been silent, and ventricular hypertrophy, which may indicate hypertrophic cardiomyopathy or other ventricular disease. An ECG finding is the major diagnostic manifestation of several genetic arrhythmia syndromes in patients without structural heart disease, including the long QT syndrome, Brugada syndrome, and short QT syndrome.

CHAPTER 259 Approach to Ventricular Arrhythmias V1 If there is suspicion of structural heart disease, cardiac imaging is warranted to assess ventricular function and structure. Transthoracic echocardiography is most frequently employed for initial evaluation. Depressed ventricular function increases concern for a risk of sudden death and warrants further evaluation to establish the cause, which may be cardiomyopathy, coronary artery disease, or valvular heart disease. Ventricular thickening may indicate hypertrophic cardiomyopathy or infiltrative diseases such as amyloidosis. Cardiac magnetic resonance imaging with gadolinium contrast imaging provides

similar assessment but also can detect areas of ventricular scar, evident as regions of delayed hyperenhancement, which are usually present in patients who have sustained monomorphic VT (Fig. 259-5). The nature and location of abnormalities are helpful in assessing the type of heart disease. Evaluation to exclude atherosclerotic coronary artery disease should be performed in patients at risk, guided by age and other risk factors.

■ ■ TREATMENT OPTIONS FOR VENTRICULAR ARRHYTHMIAS Treatment of VAs is guided by the severity and frequency of symptoms. For those with structurally normal hearts and normal ECGs, reassurance and removal of aggravating factors (e.g., caffeine or alcohol) may be all that is needed. For arrhythmias associated with a sudden death risk, ICD implantation is usually indicated and will provide a “safety net” to terminate life-threatening VT or VF, preventing sudden death but without preventing the arrhythmia. When suppression of the arrhythmia is required, antiarrhythmic drug therapy or catheter ablation is a major consideration.

■ ■ ANTIARRHYTHMIC DRUGS Use of antiarrhythmic drugs is based on consideration of the risks and potential benefit for the individual patient. Efficacy and side effects for the individual patient are not always predictable and are assessed by individual therapeutic trial. Causes of drug intolerance are mostly noncardiac and minor but can sometimes be severe enough to limit their use. Cardiac adverse events, however, include the potential for “proarrhythmia,” whereby a drug can increase the frequency of arrhythmia or cause a new arrhythmia. Aggravation of bradyarrhythmias is also a common concern. Although antiarrhythmic drugs are classified based on their actions on receptors or ion channels, most have multiple effects, affecting more than one channel.

■ ■ α -ADRENERGIC BLOCKERS Many VAs are sensitive to sympathetic stimulation, and β -adrenergic stimulation may also interact with the electrophysiologic effects of many membrane-active antiarrhythmic drugs. The safety of β -blocking agents makes them the first choice of therapy for most VAs. β -Blockers are particularly useful for exercise-induced arrhythmias and idiopathic arrhythmias but have limited efficacy for most arrhythmias associated

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FIGURE 259-5 Imaging studies of the left ventricle (LV) used to assist ablation for ventricular tachycardia (VT). Left panel is a magnetic resonance image of a longitudinal section demonstrating thinning of the anterior wall and late gadolinium enhancement in a subendocardial scar (white arrows). The middle panel shows a two-dimensional image of the LV in long axis corresponding to the sector through the mid-LV (arrow in figure on right panel) obtained by an intracardiac echocardiography probe positioned in the right ventricle. An electroanatomic three-dimensional map of the LV in the left anterior oblique projection is displayed in the right panel. The purple areas depict areas of normal voltage (>1.5 mV). Blue, green, and yellow represent progressively lower voltages, with the red areas indicating scar (<0.5 mV). Channels of viable myocardium with slow conduction within the scar are identified with the light blue dots. Areas of ablation delivered to regions involved in reentrant VT are indicated by maroon dots.

with heart disease. Bradyarrhythmias and negative inotropic effects are the major cardiac adverse effects.

■ ■ CALCIUM CHANNEL BLOCKERS The nondihydropyridine calcium channel blockers diltiazem and verapamil can be effective for some idiopathic VTs. The risk of proarrhythmia is low, but they have negative inotropic and vasodilatory effects that can aggravate hypotension.

■ ■ SODIUM CHANNEL-BLOCKING AGENTS Drugs whose major effect is mediated through sodium channel blockade include mexiletine, quinidine, disopyramide, flecainide, and propafenone, which are available for chronic oral therapy. Blockade of the fast inward sodium current has been referred to as a class I antiarrhythmic drug effect. Antiarrhythmic actions are the result of depressing cardiac conduction and membrane excitability. Conduction slowing can be manifest as a prolongation of QRS duration. Lidocaine, quinidine, and procainamide are available

as intravenous formulations. Quinidine, disopyramide, and procainamide also have potassium channel-blocking effects that may prolong the QT interval (class III antiarrhythmic drug action), contributing to their antiarrhythmic effect. Quinidine also blocks a particular potassium current, I_{to} , the blockade of which can be important in Brugada syndrome. These agents have potential proarrhythmic effects and, with the possible exception of quinidine, also have negative inotropic effects that may have contributed to the increased mortality observed when some were administered chronically to patients with prior myocardial infarction. Long-term therapy is generally avoided in patients with structural heart disease but may be used to reduce symptomatic arrhythmias in patients with ICDs. ■ ■ **POTASSIUM CHANNEL BLOCKING AGENTS** Sotalol and dofetilide block the delayed rectifier potassium channel I_{Kr} , thereby prolonging action potential duration (QT interval) and the cardiac refractory period, known as the class III antiarrhythmic drug effect. Sotalol also has nonselective β -adrenergic-blocking activity. It has been shown to have a modest effect on reducing ICD shocks due to ventricular and atrial arrhythmias. Proarrhythmia due to the polymorphic VT torsade de pointe that is associated with QT prolongation occurs in 3–5% of patients. Both sotalol and dofetilide are excreted via the kidneys, necessitating dose adjustment or avoidance in renal insufficiency. These drugs must be avoided in patients with other risk factors for torsades de pointes, including QT prolongation, other administered medications that prolong the QT interval, hypokalemia, and significant bradycardia. ■ ■ **AMIODARONE** Amiodarone blocks multiple cardiac ionic currents and has sympatholytic activity. It is the most effective antiarrhythmic drug for suppressing VAs. It is administered intravenously for life-threatening

arrhythmias. During chronic oral therapy, electrophysiologic effects develop over several days. It is more effective than sotalol in reducing ICD shocks and is often used for VAs in patients with heart disease. Bradyarrhythmias are the major cardiac adverse effect. Ventricular proarrhythmia can occur, but torsades de pointes VT is rare. Noncardiac toxicities are a major problem and contribute to drug discontinuation in at least a third of patients during long-term therapy. Hyper- and hypothyroidism are related to the iodine content of the drug. Pneumonitis or pulmonary fibrosis occurs in ~1% of patients. Photosensitivity is common, and neuropathy and ocular toxicity can occur. Systematic monitoring is recommended during chronic therapy including assessment for thyroid, liver, and pulmonary toxicity. Intravenous administration of amiodarone via a peripheral vein for >24 h can cause severe peripheral thrombophlebitis. Dronedarone has structural similarities to amiodarone but without the iodine moiety. Efficacy for VAs is poor, and dronedarone increases mortality in patients with heart failure, so dronedarone is not typically used for treatment of VAs. ■ ■ **IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS** ICDs detect sustained VT, largely based on heart rate, and then terminate the arrhythmia. In transvenous devices, VF is terminated by a shock applied between a lead in the right ventricle and the ICD pulse generator. The lead can provide pacing for bradycardia if needed. This transvenous form of ICD has the disadvantages of vascular occlusion, risk of lead fracture, endocarditis in the event of infection, and difficulty with removal. Monomorphic VT can also be terminated by a burst of rapid pacing faster than the VT, known as antitachycardia pacing (ATP) (Fig. 259-6A). If ATP fails or is not a programmed treatment, as is often the case for rapid VT or VF, a shock is delivered (Fig. 259-6B). ICDs can also be subcutaneous or extravascular, without a transvenous lead. The rhythm is also sensed by this lead, in a manner similar to a surface ECG. The lead is placed overlying the left chest with a coil parallel and next to or underneath the sternum. No matter the type of ICD, shocks are uncomfortable and distressing if the patient is conscious, sometimes leading to a posttraumatic stress disorder (PTSD). The most common ICD complication is the delivery of unnecessary therapy (either ATP or shocks)

in response to an inappropriately detected rapid supraventricular tachycardia or electrical noise as a result of an ICD lead fracture or electromagnetic interference from an external source. ICDs record and store electrograms from arrhythmia episodes that can be retrieved by interrogation of the ICD, which can be performed remotely and communicated via the internet. This assessment is critical after an ICD shock to determine the arrhythmia diagnosis and exclude unnecessary therapy. Device infection is an important problem long term and occurs in ~1% of patients. This risk may be less for subcutaneous or extravascular implants. ICDs decrease mortality in patients at risk for sudden death due to structural heart diseases. In all cases, ICDs are recommended only if there is also an expectation for survival of at least a year with acceptable

A Anti-tachycardia pacing **B** ICD shock **FIGURE 259-6** Implantable cardioverter-defibrillator (ICD) and therapies for ventricular arrhythmias. **A.** A monomorphic ventricular tachycardia (VT) is terminated by a burst of pacing impulses at a rate faster than VT (antitachycardia pacing). **B.** A rapid VT is converted with a high-voltage shock (arrow). The chest x-ray in the panel **C** shows the components of an ICD capable of biventricular pacing (yellow arrows). ICD generator in the subcutaneous tissue of the left upper chest, pacing leads in the right atrium and the left ventricular (LV) branch of the coronary sinus (LV lead), and a pacing/defibrillating lead in the right ventricle (RV lead) are shown. functional capacity. The exception is in cases of patients with endstage heart disease who are awaiting cardiac transplantation outside the hospital or who have left bundle branch block QRS prolongation such that they are likely to have improvement in ventricular function with cardiac resynchronization therapy from a biventricular ICD or conduction system pacing (Fig. 259-6C). In these cases, an ICD may be warranted despite a guarded prognosis. A wearable ICD system with electrodes incorporated into a vest and an external battery pack is also available for short-term use in patients pending a decision regarding a permanent implanted system, or when a permanent system cannot be implanted for other reasons such as an ongoing infection. Despite prompt termination of VT or VF by an ICD, the occurrence of these arrhythmias predicts subsequent increased mortality and risk of heart failure. Occurrence of VT or VF should therefore prompt assessment for potential causes including worsening heart failure, electrolyte abnormalities, and ischemia. Repeated shocks, even if appropriate, often induce posttraumatic stress disorder. Antiarrhythmic drug therapy, most commonly amiodarone, or catheter ablation is often required for suppression of recurrent arrhythmias. Antiarrhythmic drug therapy can alter the VT rate and the energy required for defibrillation, thereby necessitating programming changes in the ICD's algorithms for detection and therapy. ■ ■ **CATHETER ABLATION FOR VT** Catheter ablation is usually performed by applying radiofrequency (RF) current to cause thermal injury by resistive heating of cardiac tissue responsible for the arrhythmia. An electrode catheter with an electroanatomic mapping system is used to map local electrical activity to identify the ventricular myocardium that is causing the arrhythmia, referred to as the arrhythmia substrate. The size and location of the arrhythmia substrate determine the ease and likely effectiveness of the procedure, as well as the potential complications. When the arrhythmia originates from the endocardium, as is most commonly the case, it can be reached from an endovascular approach via a femoral vein or artery. Less commonly, arrhythmias originate from the subepicardium, and percutaneous pericardial puncture, similar to pericardiocentesis,

CHAPTER 259 Approach to Ventricular Arrhythmias Atrial lead ICD LV lead RV lead **C** is required to insert a catheter into the pericardial space for mapping and ablation. In patients with scar-related VT due to prior infarction or cardiomyopathy, ablation typically targets abnormal regions in the scar or region of fibrosis. Because these scars often contain multiple reentry circuits over relatively

large regions, extensive areas of ablation can be required, and these areas are often identified as regions of low voltage displayed on electroanatomic reconstructions of the ventricle (Fig. 259-5). Catheter ablation is often performed in patients with recurrent VAs associated with poor cardiac function, and the procedure-related mortality in this situation is 0.5–3%. Outcomes are better for patients with prior infarction and VT than for patients with nonischemic cardiomyopathies in which the scar locations are more variable and often intramural or subepicardial. Ablation can be lifesaving for patients with very frequent or incessant VT. Methods of delivering ablative energy to intramural areas or areas requiring very extensive ablation are under development. These include needle catheters capable of delivering ablative energy into intramural sources, and bipolar ablation where radiofrequency energy is delivered across two ablation catheters. Stereotactic body radiation therapy (SBRT), classically used for treating thoracic tumors, has been used to direct radiation therapy to a specific portion of the scar substrate to noninvasively ablate VT with encouraging early studies. Idiopathic VTs and PVCs that occur in the absence of structural heart disease usually originate from a small focus, for which catheter ablation typically has a higher success rate for preventing recurrent arrhythmia. Long-term arrhythmia-free survival in these patients is excellent. ARRHYTHMIA SURGERY When antiarrhythmic drug therapy and catheter ablation fail or are not an option, surgical cryoablation, often combined with aneurysmectomy, can be effective therapy for recurrent VT due to prior myocardial infarction and has also been used successfully in a few patients with nonischemic heart disease. Few centers now maintain the expertise for this therapy, though some use this therapy as an adjunct to ventricular assist device implantation.

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