Ventricular Arrhythmias
State of the Art

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INTRODUCTION

Epidemiology of Ventricular Arrhythmias

Sudden cardiac death (SCD) accounts for approximately 300,000 deaths in the United States per year and in most cases is the final result of ventricular tachycardia (VT) or ventricular fibrillation (VF).1 Ventricular arrhythmias must be recognized and treated promptly because of the high risk of acute mortality. In patients in the cardiac intensive care unit, 8% have episodes of nonsustained VT, 2% develop sustained VT, and 5% are treated for VF.2 SCD is a significant cause of long-term mortality in ischemic heart disease, accounting for 22% of all causes of death.3 Nonischemic conditions associated with VT, VF, and SCD include genetic conditions, cardiomyopathies, and idiopathic VT in structurally normal hearts. VT and VF can be challenging to manage, particularly if they recur despite initial therapy. The diversity of pharmacotherapy, devices, catheter ablation techniques, and other interventions increases the complexity of management.

Prognostic Significance

Sustained VT and VF have a high acute mortality and adversely affect long-term prognosis despite treatment. Even asymptomatic nonsustained VT confers higher long-term mortality if programmed stimulation induces sustained VT.4 VT or VF within 48 hours of the onset of an acute myocardial infarction (MI), although it significantly increases in-hospital mortality, does not adversely affect long-term prognosis.5 VT or VF immediately after cardiac surgery not only increases in-hospital mortality but also worsens long-term prognosis.6

IMMEDIATE CONSIDERATIONS

In patients with suspected VT, rapid but accurate recognition of the rhythm is imperative. Although...
treating hemodynamic instability is the first priority in patient care, hemodynamic instability does not confirm the diagnosis of VT. Wide complex tachycardia in patients with a history of coronary artery disease or structural heart disease is most likely VT; even so, the 12-lead electrocardiogram (ECG) must be systematically analyzed to reach the correct diagnosis. Other aspects of evaluating patients with VT are described in Table 1.

Noncardiac artifact obscuring a narrow complex rhythm must be excluded. Then, the QRS complexes can be classified as monomorphic (constant in form) or polymorphic (variable in form). Polymorphic VT also requires analysis of the preceding rhythm. In the setting of a prolonged corrected QT interval (QTc), polymorphic VT is consistent with torsades de pointe (TdP) and usually initiates with a short cycle length after a prolonged cardiac cycle.

Several ECG features, summarized in Fig. 1, differentiate monomorphic VT from supraventricular tachycardia (SVT) with aberrant conduction. Particularly useful for differentiating VT from SVT with aberrancy are the validated criteria developed by Brugada and colleagues, which are 98.7% sensitive and 96.5% specific for VT and 96% sensitive and 98.7% specific for SVT with aberrancy. Fig. 2 shows the application of these criteria to an ECG showing VT.

After morphologic criteria are applied, SVT conducting through an accessory pathway remains difficult to distinguish from VT, even with additional criteria. The ECG is also useful for determining the exit site of VT and for distinguishing endocardial from epicardial origin.

### NONPHARMACOLOGIC THERAPEUTIC MODALITIES

**External Defibrillation and Cardioversion**

Early defibrillation with a rectilinear biphasic automatic external defibrillator improves initial success of defibrillation for out-of-hospital VF arrest. Thus, national guidelines recommend early biphasic defibrillation for VF followed by epinephrine and amiodarone administration if the patient is difficult to

### Table 1

**Initial assessment of a patient with VT**

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Assessment</th>
<th>Goal</th>
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<tbody>
<tr>
<td>Vital signs</td>
<td>Hemodynamic stability</td>
<td>If hemodynamically unstable, treat with urgent DCCV or defibrillation</td>
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<tr>
<td>12-lead ECG</td>
<td>Tachycardia diagnosis</td>
<td>Differentiate VT from SVT with aberrancy; determine VT exit site</td>
</tr>
<tr>
<td>History</td>
<td>Symptoms (eg, chest pain indicating ongoing ischemia)</td>
<td>Identify cause and triggers</td>
</tr>
<tr>
<td>Current medications</td>
<td>Antiarrhythmics, digoxin, QTc-prolonging medications</td>
<td>Identify pharmacologic contribution to a proarrhythmic state</td>
</tr>
<tr>
<td>Family history</td>
<td>Family history of SCD</td>
<td>Determine risk of inherited predisposition to SCD</td>
</tr>
<tr>
<td>Physical examination</td>
<td>Canon A waves, Murmurs, sternotomy scar</td>
<td>Indicate AV dissociation, indicate existing structural heart disease</td>
</tr>
<tr>
<td>Laboratory tests</td>
<td>Electrolytes, creatinine, troponin, thyroid-stimulating hormone, toxicology assays</td>
<td>Identify metabolic, ischemic, or pharmacologic contributions to a proarrhythmic state</td>
</tr>
<tr>
<td>Imaging</td>
<td>Chest roentgenography, echocardiography, Coronary angiography, Computed tomography, magnetic resonance imaging</td>
<td>Indicated in all patients with VT to assess for structural heart disease, Indicated if VT occurs secondary to ischemia, Indicated in special cases when particular cardiomyopathies are suspected</td>
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**Abbreviations:** A, atrial; AV, atroventricular; DCCV, direct current cardioversion; QTc, corrected QT interval.
defibrillate.\textsuperscript{15} In VT, direct current cardioversion (DCCV) must be synchronized, because a shock occurring in a partially repolarized ventricle precipitates VF. In polymorphic VT, defibrillation is required, because synchronization is usually not possible. For comfort, hemodynamically stable conscious patients should be sedated before synchronized DCCV.

- Biphasic waveform defibrillation improves initial success of defibrillation.
- In VT, unsynchronized DCCV can precipitate VF.

Fig. 1. Criteria for diagnosing a wide QRS complex tachycardia as VT. AV, atrioventricular; LBBB, left bundle branch block; RBBB, right bundle branch block.

Fig. 2. 12-lead ECG showing monomorphic VT. This ECG shows a regular monomorphic tachycardia with a wide QRS of 160 milliseconds and an atypical QRS morphology in the precordial leads. The Brugada criteria can be applied. Step 1: There is an R wave in V1, so continue to step 2. Step 2: the longest RS interval in a precordial lead is slightly more than 100 milliseconds in V5 and is thus consistent with VT, even although no atrioventricular dissociation (step 3) is visible. Step 4: R is greater than R\textsuperscript{0} in V1, and S is greater than R in V6, meeting morphologic criteria for VT in V1 and V6.
Implantable Cardioverter-Defibrillator and Pacing

Numerous trials have shown the importance of implantable cardioverter-defibrillator (ICD) placement in high-risk patients to prevent SCD. The ICD treats but does not prevent recurrent VT. Because ICD shocks cause significant discomfort, inappropriate shocks should be acutely stopped by placing a magnet over the device while awaiting reprogramming. The ICD also can perform antitachycardia pacing, which creates a paced waveform that collides with the VT wavefront and terminates it.

- ICD implantation significantly reduces mortality from SCD in high-risk patients.
- Inappropriate ICD therapy can be halted by applying a magnet over the device.

Left Cardiac Sympathetic Denervation

Left stellate ganglion resection completely removes sympathetic input to the heart. It effectively prevents VT that recurs despite β-blockade in long QT syndrome (LQTS), catecholaminergic polymorphic VT (CPVT), and other types of VT. Stellate ganglion blockade with lidocaine injection is feasible in unstable patients with electrical storm (ES).

Radiofrequency Catheter Ablation

Catheter ablation techniques are rapidly advancing. VT is mapped by entrainment, pacing, or an electroanatomic system that determines catheter position in a magnetic field. Radiofrequency current ablates the endocardium identified as part of the reentry circuit. Another technique, substrate ablation, identifies slow conduction zones around myocardial scars and isolated potentials; these areas are ablated to prevent VT. The 24-month rate of freedom from recurrent VT or VF after catheter ablation is 48% to 88% in recent trials.

VT from the left ventricular endocardium can be ablated via either transseptal access from the left atrium or retrograde arterial access through the aortic valve. If a patient fails endocardial ablation or has an epicardial site of VT origin, epicardial ablation can be performed through percutaneous pericardial access or surgical thoracotomy. Rarely, if both endocardial and epicardial ablation fail or are not possible, ethanol injection in distal coronary arteries may successfully ablate midmyocardial sites; but this technique should be reserved for refractory cases because of the low success rate and risk of complications from the ethanol-induced infarction. Intracardiac echocardiography is integrated with electroanatomic mapping, reducing fluoroscopy time. Cryoablation can be performed to reduce the risk of permanent damage when ablation is performed near a coronary artery or the phrenic nerve.

Potential complications include a 2.8% risk of thrombosis, which increases with more extensive ablations but is reduced by irrigated catheters, antiplatelet medications, intraprocedural anticoagulation, and intracardiac echocardiography. There is a 1% risk of perforation and pericardial tamponade during endocardial ablation and 3.4% during epicardial ablation. Intracardiac echocardiography and fluoroscopic evaluation of the cardiac silhouette are important means of early detection of an accumulating pericardial effusion during ablation. If a large effusion develops, or if epicardial ablation is performed, a pericardial drain is placed until it drains less than 20 mL over 4 to 6 hours. Anticoagulation postprocedure is initiated for patients requiring extensive ablation, patients with atrial fibrillation or severely reduced ejection fraction, or patients who have a hemodynamic support or ventricular assist device (VAD). Antiarrhythmic medications are continued unless the patient experiences marked adverse effects from them. Intensive care unit admission postprocedure is recommended for patients requiring a prolonged procedure (>6 hours), patients requiring an intra-aortic balloon pump or a percutaneous hemodynamic support device such as an Impella (Abiomed, Danvers, MA), patients with a pericardial drain, patients with recurrent VT after unsuccessful ablation, and patients with significant fluid overload after a procedure.

PHARMACOTHERAPY

Intravenous Medications for Acute Management

Procainamide blocks both sodium and potassium channels, slowing and terminating VT. As a negative inotrope, it can cause hypotension; therefore, it is recommended for stable VT in patients with normal systolic function. Lidocaine is a sodium channel blocker that is potentially effective for VT during acute ischemia. Amiodarone has a complex mechanism of action and is superior to lidocaine for treating shock-resistant VF and VT refractory to procainamide. It shows significant long-term thyroid, pulmonary, and hepatic toxicities, which should be considered before initiating long-term oral therapy. Table 2 lists the recommended dosing and acute adverse effects of these medications.

- Procainamide is preferred for pharmacologic cardioversion of stable VT in patients with normal systolic function.
- Lidocaine is useful for VT during acute ischemia; it does not cause QTc prolongation.
Amiodarone is the most effective antiarrhythmic for VT but requires time to load.

Long-Term Oral Therapy

In a randomized controlled trial of sotalol, amiodarone with β-blockers, or β-blockers alone, amiodarone with a β-blocker (metoprolol, carvedilol, or bisoprolol) significantly reduced defibrillator shocks more than sotalol or β-blockers alone.38 There was no significant mortality difference, and in patients receiving amiodarone, 5% experienced pulmonary adverse events and 5.7% developed thyroid abnormalities. β-Blockers by themselves reduce SCD in patients with MI 39 and improve survival in patients with treated VT or VF.40

- A combination of β-blockers and amiodarone is the most successful long-term medical strategy for reducing defibrillator shocks.
- β-blockers improve survival in patients after VT or VF is treated.

Although right ventricular outflow tract (RVOT) and left ventricular outflow tract (LVOT) VT are typically sensitive to diltiazem and verapamil,41 giving calcium channel blockers acutely during VT when the mechanism of VT is unknown is contraindicated because of the risk of hemodynamic collapse.15

Other oral antiarrhythmics may suppress VT that recurs despite treatment with sotalol or amiodarone with β-blockers. Mexiletine is a sodium channel blocker that counteracts the sodium channel gain-of-function mutation in LQTS type 3. Quinidine has a significant side effect profile but may be particularly effective in Brugada syndrome. Flecainide and other class 1C sodium channel blockers are associated with increased mortality in patients with coronary artery disease.42 However, flecainide can be used in VT associated with structurally normal hearts.43 Dofetilide and ranolazine are not approved to treat ventricular arrhythmias but have significantly reduced recurrent VT and VF in small studies.44,45

Table 2
Acute and maintenance dosing of intravenous antiarrhythmic medications

<table>
<thead>
<tr>
<th>Antiarrhythmic</th>
<th>Dosing</th>
<th>Acute Adverse Reactions</th>
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<tbody>
<tr>
<td>Procainamide</td>
<td>Load: 17 mg/kg</td>
<td>Hypotension</td>
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<tr>
<td></td>
<td>Maximum rate: 50 mg/min</td>
<td>Hold if QRS prolongs &gt;50%</td>
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<tr>
<td></td>
<td>Maintenance: 1–4 mg/min</td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Load: 1–3 mg/kg</td>
<td>Reduce dose in heart failure</td>
</tr>
<tr>
<td></td>
<td>Rate: 20–50 mg/min</td>
<td>Monitor for neurotoxicity: delirium, seizures, or paresthesias</td>
</tr>
<tr>
<td></td>
<td>Maintenance: 1–4 mg/min</td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Load: 150 mg over 10 min if blood pressure is normal; 300 mg over 19 min if hypotensive Maintenance: 1 mg/min for 6 h, then 0.5 mg/min for 18 h</td>
<td>Caution in cardiogenic shock TdP is rare Use with pacing if patient is severely bradycardic</td>
</tr>
</tbody>
</table>

VT IN PATIENTS WITH STRUCTURAL HEART DISEASE

VT in Patients with Acute MI

Acute MI significantly increases the risk for VT and VF. Ischemia prolongs the QTc and can trigger polymorphic VT.46 Acutely, polymorphic VT requires defibrillation, because it is unlikely to allow hemodynamic stability. The patient should then receive urgent coronary reperfusion and other evidence-based therapies for MI. Both lidocaine and amiodarone are used for VT; neither is clearly superior to the other.47 Lidocaine should not be used prophylactically in patients with MI.15 Neither long-term antiarrhythmics nor ICD placement are required for VT during acute MI.5

VT in Revascularized Patients with Ischemic Cardiomyopathy

Numerous trials show that ICD implantation decreases mortality in patients with ischemic cardiomyopathy at high risk.16–18 In patients with VT occurring 1 month or more after MI, recurrence can be successfully prevented with catheter ablation before initiation of long-term antiarrhythmics.27,48

- Catheter ablation of VT before initiation of long-term antiarrhythmic therapy is reasonable in patients with sustained VT and a previous MI.

VT in Patients with Nonischemic Cardiomyopathy

The mechanisms of VT in nonischemic cardiomyopathy are diverse, including bundle branch
reentry, reentry around fibrosis, activation around focal inflammation (as in sarcoidosis), and epicardial involvement. Acutely, VT should be treated with DCCV and amiodarone, because most other antiarrhythmics are negative inotropes. VT with a typical bundle branch block should be evaluated for bundle branch reentry tachycardia, because ablation of the right bundle branch successfully treats bundle branch reentry.\textsuperscript{49} Catheter ablation for recurrent VT in patients with dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia (ARVD), hypertrophic cardiomyopathy, or cardiac sarcoidosis is associated with variable success rates.\textsuperscript{50–53}

- Catheter ablation of VT in nonischemic cardiomyopathy has variable success rates depending on the mechanism and substrate of VT.

**VT in Patients After Cardiac Surgery**

VT and VF occur in some patients immediately after coronary artery bypass grafting and valvular surgeries. Sympathetic stimulation, electrolyte abnormalities, ischemia from vein graft closure, atrial dysrhythmias, injury from cannulation, and preexisting substrate all contribute to initiation of VT. Prophylactic amiodarone prevents both atrial arrhythmias and VT,\textsuperscript{54} although no mortality benefit has been shown.\textsuperscript{55} VT after cardiac surgery increases both in-hospital and long-term mortality,\textsuperscript{2} but ICD placement is not indicated.\textsuperscript{56}

- Amiodarone prophylaxis reduces postoperative VT and VF.

**VT in Patients with VADs**

Increasing numbers of patients with advanced heart failure are being supported with VADs. In these patients, VT and VF cause hemodynamic instability from right heart failure, reducing left ventricular preload, which can cause a suction event. Catheter ablation in patients with a VAD is feasible; mapping has shown that VT originates from the scar in 75% of patients and less commonly from the inflow cannula site.\textsuperscript{57}

- VT in patients with a VAD comes predominantly from the preexisting substrate rather than from the inflow cannula site.

**VT in Patients After Orthotopic Heart Transplantation**

VT in orthotopic heart transplant recipients is rare, and acute rejection must be ruled out. β-Blockers should be used cautiously after heart transplantation because of the risk of sinus node dysfunction.\textsuperscript{58} Amiodarone, lidocaine, and mexiletine have been used for VT in heart transplant patients.\textsuperscript{59}

- Rejection must be ruled out in patients with orthotopic heart transplantation and VT.

**VT in Patients with Congenital Heart Disease**

Any congenital anomaly involving the ventricles predisposes patients to VT, especially tetralogy of Fallot, transposition of the great arteries, single ventricle, and congenital aortic stenosis. The predominant mechanism is reentry around fibrosis and surgical scar, which is responsive to catheter ablation.\textsuperscript{60}

**VT in Patients with Brugada Syndrome**

Brugada syndrome is diagnosed by coved ST segment increase in leads V1 to V3 either spontaneously or provoked by sodium channel blockade. \textsuperscript{Fig. 3} shows an ECG consistent with Brugada syndrome. Impaired sodium channel function prolongs the epicardial action potential, reversing the direction of repolarization and predisposing the ventricle to reentry. Isoproterenol prevents recurrent VT by increasing calcium current and should be titrated to increase the heart rate by 20% and to normalize the ST segments.\textsuperscript{61} Quinidine prevents recurrent VF by normalizing the direction of repolarization.\textsuperscript{62}

- In patients with Brugada syndrome presenting with VT, isoproterenol infusion should be initiated after DCCV and titrated until the ST segments normalize.

**VT in Patients with TdP**

TdP is polymorphic VT in the setting of a prolonged QTc in which a premature ventricular depolarization follows a prolonged cardiac cycle, as in \textsuperscript{Fig. 4}.\textsuperscript{9} TdP can be secondary to electrolyte abnormalities, bradyarrhythmias, or medication effects. After defibrillation, temporarily pacing faster than the intrinsic rate prevents long-short cycles from occurring, and pacing is more successful than isoproterenol and lidocaine in preventing recurrent TdP.\textsuperscript{9} Drugs that prolong the QTc must be discontinued, and potassium and magnesium levels must be maintained in normal ranges.

- Pacing prevents recurrent TdP while awaiting clearance of offending medications.
VT in Patients with Congenital LQTS

Congenital LQTS is caused by one of many mutations affecting repolarization. β-Blockers are an essential therapy for most types, and mexiletine is useful in LQTS type 3. Treatment of VT in LQTS requires pacing after DCCV; isoproterenol in patients with LQTS is potentially harmful and can precipitate recurrent VT. Permanent pacemaker placement may be required in LQTS to allow the use of adequate doses of β-blockers.\(^6\)

Left stellate ganglion resection is 91% effective for preventing recurrent VT.\(^2\)

- β-blockade combined with pacing prevents recurrent VT in LQTS.

VT in Patients with CPVT

CPVT occurs when a mutant ryanodine receptor allows calcium to leak into the cytoplasm and cause delayed afterdepolarizations. β-Blockers

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Fig. 3. 12-lead ECG showing covered ST segment elevation in leads V1 to V3 in a patient with Brugada syndrome.

Fig. 4. 12-lead ECG showing the initiation of TdP with a long-short sequence occurring in the setting of a prolonged QT interval.
are essential but may not prevent recurrent VT in all patients, and many patients require an ICD.64 Left stellate ganglion resection is particularly effective for preventing recurrent VT.21

**VT in Patients with Idiopathic VT**

VT occurring in the absence of structural heart disease, metabolic or pharmacologic provocation, or ion channel dysfunction is considered idiopathic. Idiopathic VT most commonly originates in the RVOT or LVOT and is suppressed by diltiazem or verapamil.41 Structural heart disease (particularly ARVD causing right ventricular VT) must be carefully excluded. Catheter ablation is often successful in treating recurrent VT. Idiopathic VT rarely causes hemodynamic compromise or SCD and is unlikely to present to the cardiac intensive care unit.

- Patients with RVOT VT should be evaluated to exclude ARVD.

**ES**

ES is defined as 3 or more episodes of VT or VF within 24 hours; electrical stability deteriorates rapidly, causing high mortality. A diversity of substrates and triggers can cause ES, and no single therapy is effective in all patients. VT refractory to 1 antiarrhythmic may be suppressed by another. After DCCV or defibrillation, amiodarone infusion is typically begun,36,37 to which a sodium channel blocker can be added if VT recurs. β-Blockade with esmolol, metoprolol, or propranolol can be titrated as blood pressure tolerates. In patients unable to tolerate β-blockade, sympathetic input to the heart can be blocked centrally with general anesthesia such as propofol65 and peripherally with injection of the left stellate ganglion with lidocaine.22 In some patients, percutaneous hemodynamic support with Impella (Abiomed, Danvers, MA) or TandemHeart (CardiacAssist, Pittsburgh, PA) is necessary. Fig. 5 shows Impella placement. Catheter ablation is successful for terminating ES in 48% of patients after the first ablation and in 84% after multiple attempts.67 Fig. 6 shows an algorithm for treating ES.

**SUMMARY**

VT and VF commonly occur in the cardiac intensive care unit but have many potential causes. Acutely, synchronized DCCV or pharmacologic cardioversion likely terminate VT, but definitive treatment is complex. Determining the cause, correcting reversible causes, and defining the substrate are the goals of initial evaluation. ICD programming, β-blockers, antiarrhythmics, and catheter ablation should be considered to prevent recurrence. For patients with ES, additional modalities, including general anesthesia, left stellate ganglion blockade, hemodynamic support, and additional catheter ablation, may be considered.

**REFERENCES**


