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aVR I aVL II aVF III V1 FIGURE 260-4 Accelerated idioventricular rhythm. Shown is an example of a slow regular wide-complex rhythm. Fusion beats are seen on complexes 4 and 10, which are more positive in lead V1 and narrower than the rest of the beats. These features are consistent with an accelerated idioventricular rhythm. myocardium can cause AIVR. Idioventricular rhythms are common during acute MI and may emerge during sinus bradycardia. Often, they are not symptomatic, but hemodynamic compromise may occur with the loss of atrioventricular synchrony in susceptible patients. Atropine may be administered to increase the sinus rates if this is a concern. This rhythm is also common in patients with cardiomyopathies or sleep apnea. It can also be idiopathic, often emerging when the sinus rate slows during sleep. Therapy should target any underlying cause and correction of bradycardia. Specific antiarrhythmic therapy for asymptomatic idioventricular rhythm is not necessary. FUTURE DIRECTIONS Recently, it has been appreciated that inflammation plays a role in the genesis of PVCs in specific patients with inflammatory cardiomyopathies and even in inherited cardiomyopathies. The roles of early identification of this process and targeted treatment are areas of active research. ■ ■ FURTHER READING Al-Khatib SM et al: 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Heart Rhythm 15:e73, 2018. Callans DJ: Josephson's Clinical Cardiac Electrophysiology: Techniques and Interpretations, 7th ed. Philadelphia, Wolters Kluwer, 2024. Cronin EM et al: 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias. EP Euro pace 21:1143, 2019. Jalife J, Stevenson W (eds): Zipes and Jalife's Cardiac Electrophysiology: From Cell to Bedside, 8th ed. Philadelphia, Elsevier, 2022. Zeppenfeld K et al: 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: Developed by the task force for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death of the European Society of Cardiology (ESC) Endorsed by the Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J 43:3997, 2022.

V1 V4 CHAPTER 261 V2 V5 V3 V6 Sustained Ventricular Tachycardia William H. Sauer, Usha B. Tedrow

Sustained Ventricular Tachycardia Sustained monomorphic ventricular tachycardia (VT) is a ventricular arrhythmia with a wide QRS lasting for at least 30 s or requiring an intervention such as

antitachycardia pacing from a defibrillator or a cardioversion for termination. Each QRS complex resembles the others, indicating either a focal site of origin or a repetitive exit from a fixed arrhythmia reentry circuit. In structural heart disease, the arrhythmia substrate is most often an area of patchy replacement fibrosis due to infarction, fibrosis, inflammation, or prior cardiac surgery that creates anatomic or functional reentrant pathways. Less commonly, VT is related to reentry or automaticity in diseased conduction pathways in the Purkinje system. While scar-related reentrant VTs are associated with risk of sudden death, idiopathic VT is a more benign form of VT that occurs in structurally normal hearts and can be due to a focal region of automaticity in the myocardium or reentry involving a portion of the Purkinje system. The clinical presentation varies depending on the rate of the arrhythmia, underlying cardiac function, and autonomic adaptation in response to the arrhythmia. Rapid VT can produce hypotension that may present as syncope, particularly in patients with significant ventricular dysfunction. In contrast, patients with normal cardiac function might tolerate sustained VT, even presenting with simple palpitations, despite rapid rates. Monomorphic VT that is rapid or associated with structural heart disease may eventually deteriorate to ventricular fibrillation (VF), which may be the initial cardiac rhythm recorded at the time of resuscitation of an out-of-hospital cardiac arrest.

DIAGNOSIS Sustained monomorphic VT (Table 261-1) has to be distinguished from other causes of uniform wide QRS tachycardia. These include supraventricular tachycardia with left or right bundle branch block

TABLE 261-1 Sustained Ventricular Arrhythmias

- Idiopathic ventricular tachycardia (VT) without structural heart disease
 - Outflow tract origin
 - Right ventricular (RV) outflow tract: left bundle branch block pattern in V1 with inferior axis (tall QRS in inferior leads) and late transition in the precordial leads
 - Left ventricular (LV) outflow tract: similar inferiorly directed axis but PART 6 Disorders of the Cardiovascular System with early precordial transition with prominent R wave in V2–V3
 - LV fascicular VT: Typical right bundle branch block pattern in V1 with sharp intrinsicoid deflection and left axis deviation (arising from left posterior fascicle in its most common form)
 - Papillary muscle VT
 - Posteromedial: atypical right bundle branch block pattern in V1 with monophasic R wave and left axis deviation
 - Anterolateral: atypical right bundle branch block pattern in V1 with positive deflection in lead III and negative deflection in lead I
- Ischemic cardiomyopathy
 - Monomorphic VT is common with prior large myocardial infarction
 - Polymorphic VT and ventricular fibrillation (VF) should prompt ischemia evaluation
- Nonischemic cardiomyopathy
 - Fibrotic scars can cause monomorphic VT, especially with sarcoidosis or other inflammatory cardiomyopathies, Chagas' disease, and familial arrhythmogenic cardiomyopathies such as Lamin A/C genetic cardiomyopathy
 - Polymorphic VT and VF can also occur independently or related to degeneration of monomorphic VT
- Arrhythmogenic RV cardiomyopathy
 - Monomorphic VT usually of RV origin (left bundle branch morphology in V1)
 - Polymorphic VT and VF can occur independently or related to degeneration of monomorphic VT
- Repaired tetralogy of Fallot
 - Monomorphic VT of RV origin (usually left bundle branch morphology in V1)
- Hypertrophic cardiomyopathy
 - Polymorphic VT or ventricular fibrillation
 - Less commonly, monomorphic VT associated with myocardial scars, particularly apical aneurysms
- Genetic arrhythmia syndromes
 - Long QT syndrome
 - Torsades des pointes VT
 - Brugada syndrome
 - Ventricular fibrillation episodes, often nocturnal
 - Catecholaminergic polymorphic VT
 - Polymorphic VT or bidirectional VT
 - Short QT and early repolarization syndromes
 - Ventricular fibrillation
- Idiopathic polymorphic VT or ventricular fibrillation
 - Usually triggered by recurrent premature ventricular contractions; the most common site of origin is the left posterior fascicle (right bundle branch block/left anterior fascicular block pattern) aberrant conduction, supraventricular tachycardias

conducted to the ventricles over an accessory pathway, and rapid cardiac pacing, appropriate or inappropriate, in a patient with a ventricular pacemaker or defibrillator. In the presence of known heart disease, VT is the most likely diagnosis of a wide QRS tachycardia, independent of QRS morphology. When left ventricular (LV) function is depressed or there is evidence of structural myocardial disease, scar-related reentry is the most likely cause of sustained monomorphic VT. Scars are suggested by pathologic Q waves on the electrocardiogram (ECG), segmental LV or right ventricular wall motion abnormalities on echocardiogram or nuclear imaging, and areas of delayed gadolinium enhancement during magnetic resonance imaging (MRI). Hemodynamic stability during the arrhythmia does not help distinguish between VT and other mechanisms of wide-complex tachycardia. A number of ECG criteria have been evaluated to distinguish supraventricular tachycardia with aberrancy from VT. The presence of

VT versus Supraventricular Tachycardia (SVT) with Aberrancy

Yes	No
AV dissociation	VT
aVR = R or Rs	No
rS or Rs in any of V1 to V6	VT
Monophasic R wave or Rs complex in aVR	No
Concordance from V1 to V6 of monophasic R or S waves	No
VT	Possible SVT with aberrancy

FIGURE 261-1 Algorithm for differentiation of ventricular tachycardia (VT) from supraventricular tachycardia with aberration. AV, atrioventricular; ventriculoatrial (VA) dissociation is a reliable marker for VT, provided the atrial rate is slower than the ventricular rate. Sometimes, P waves can be difficult to define, and the VA relationship cannot be assessed in a patient with an ongoing atrial arrhythmia such as atrial fibrillation. A P wave following each QRS does not exclude VT because 1:1 conduction from ventricle to atrium can occur. A monophasic R wave or Rs complex in aVR or concordance from V1 to V6 of monophasic R or

S waves is also relatively specific for VT (Fig. 261-1). A number of other QRS morphology criteria have also been described, but all have limitations and are not very reliable in patients with severe heart disease. In patients with known bundle branch block, the same QRS morphology during tachycardia as during sinus rhythm suggests supraventricular tachycardia rather than VT, but even this is not absolutely reliable. Patients with reentry involving the bundle branches of the Purkinje system can have a VT morphology that resembles their native QRS in sinus rhythm. An electrophysiologic study is sometimes required for definitive diagnosis. Occasionally, noise and movement artifacts on telemetry recordings can simulate VT, and prompt recognition of this can avoid unnecessary tests and interventions.

TREATMENT AND PROGNOSIS Initial management follows Advanced Cardiac Life Support (ACLS) guidelines. If hypotension, impaired consciousness, or pulmonary edema is present, QRS synchronous electrical cardioversion should be performed, ideally after sedation if the patient is conscious. For stable tachycardia, a trial of adenosine is reasonable as this may clarify a supraventricular tachycardia with aberrancy. Adenosine should not be used if the patient has a heart transplant or if the wide-complex rhythm is irregular or unstable. Intravenous amiodarone is the drug of choice if heart disease is present. Following restoration of sinus rhythm, hospitalization and evaluation to define underlying heart disease are required. Assessment of cardiac biomarkers for evidence of myocardial infarction (MI) is appropriate, but acute MI is rarely a cause of sustained monomorphic VT. Elevations in troponin or creatine kinase (CK)-MB are more likely to indicate myocardial injury that is secondary to hypotension and ischemia from fixed coronary lesions during the VT rather than an acute coronary event. Subsequent management is determined by the underlying heart disease and frequency of VT. If VT recurs frequently or is incessant, administration of antiarrhythmic medications or catheter ablation may be required to restore stability. More commonly, sustained monomorphic VT occurs as a

single episode but with a high risk of recurrence. Implantable cardioverterdefibrillators (ICDs) are warranted for secondary prevention of sudden death in patients who present with sustained VT associated with structural heart disease (Fig. 261-2).

aVR I aVL II aVF III V1 V5 FIGURE 261-2 Monomorphic ventricular tachycardia in a patient with prior myocardial infarction. Shown is a wide-complex tachycardia. Complexes 3, 6, 9, and 18 are narrower and are examples of fusion beats, proving ventriculoatrial (VA) dissociation and proving that this rhythm is in fact ventricular tachycardia. SUSTAINED MONOMORPHIC VT IN SPECIFIC DISEASES ■ ■CORONARY ARTERY DISEASE Patients who present with sustained monomorphic VT associated with coronary artery disease typically have a history of a remote prior large MI. Patients typically present years after the acute infarct with a remodeled ventricle and markedly depressed LV function. Even when there is biomarker evidence of acute MI, a preexisting scar from previous MI should be suspected as the cause of the VT. Infarct scars provide a durable substrate for sustained VT, and up to 70% of patients have a recurrence of the arrhythmia within 2 years. Scar-related reentry is not usually dependent on recurrent acute myocardial ischemia, so coronary revascularization is unlikely to prevent recurrent VT, although it may be appropriate for treatment of angina or other indications. Depressed ventricular function, which is a risk factor for sudden death, is usually present. Implantation of an ICD is clearly indicated for secondary prevention provided that there is a reasonable expectation of survival for 1 year with acceptable functional status. Compared with antiarrhythmic drug therapy, ICDs reduce annual mortality from 12.3 to 8.8% and lower arrhythmic deaths by 50% in patients with hemodynamically significant sustained VT or a history of cardiac arrest. Antiarrhythmic drugs may have some utility for palliation of VT symptoms and prevention of ICD therapies, such as shocks and antitachycardia pacing; however, without an ICD, these drugs do not improve survival. Following ICD implantation, patients with depressed ejection fraction remain at risk for clinical heart failure, recurrent ischemic events, and recurrent VT, with a 5-year mortality that exceeds 30%. Attention to guideline-directed medical therapy for patients with heart failure and coronary artery disease, including β -adrenergic blocking agents and angiotensin-converting enzyme inhibitors, is important. ICD therapies, whether shocks or antitachycardia pacing, constitute an adverse event for the patient and are associated with increased rates of heart failure, mortality, and psychological stress. For this reason, recurrent VT episodes in patients with an ICD warrant treatment with medications or catheter ablation. In a randomized study of catheter ablation versus escalated medical therapy (Ventricular Tachycardia Ablation versus Escalation of Antiarrhythmic Drugs [VANISH]), patients receiving catheter ablation fared better than those receiving increasing doses of antiarrhythmic drugs, in particular, amiodarone.

V1 V4 CHAPTER 261 V2 V5 Sustained Ventricular Tachycardia V3 V6 Another randomized trial (BERLIN VT) examined a preventative versus deferred ablation strategy in patients who had not yet failed an antiarrhythmic drug. This trial was stopped early for futility, with more procedural complications but fewer VT episodes in the catheter ablation group. For this reason, the most recent consensus statement most strongly recommends catheter ablation for patients with ischemic cardiomyopathy failing or intolerant of antiarrhythmic drugs but also allows for consideration of catheter ablation when long-term therapy with an antiarrhythmic drug (such as amiodarone, which has significant long-term toxicities) is not desired (Table 261-2). ■ ■NONISCHEMIC DILATED CARDIOMYOPATHY Sustained monomorphic VT associated with nonischemic cardiomyopathy is usually due to scar-related reentry. The etiology of scar is often

unclear, but progressive replacement fibrosis is the likely cause. Patients with nonischemic cardiomyopathy (NICM) have historically been presumed to have a postviral etiology, although increasingly, genetic causes are found in many. Inflammatory etiologies (myocarditis, sarcoidosis) are also increasingly appreciated. On cardiac MRI, scars are detectable as areas of delayed gadolinium enhancement and are more often intramural (Fig. 261-3) or subepicardial in location as compared with patients with prior MI. Scars that cause VT are often located adjacent to a valve annulus and can occur in either ventricle. Any cardiomyopathic process can cause scars and VT, but cardiac sarcoidosis, Chagas' disease, and cardiomyopathy due to Lamin A/C mutations are particularly associated with monomorphic VT. An ICD is indicated for patients with a history of sustained VT, syncope, or New York Heart Association class II or III heart failure symptoms, with additional drugs or catheter ablation for control of recurrent VT. In addition, for patients with malignant familial arrhythmogenic cardiomyopathies, an ICD may be considered earlier in the clinical course. Overall, there are fewer studies of catheter ablation for VT in NICM. Reported success rates are lower than VT ablation in ischemic cardiomyopathy in most observational series. Additionally, inability to reproduce the clinical VT at ablation attempts and epicardial and intramural reentry circuits are important causes of failure of endocardial VT ablation in NICM. Imaging with MRI or computed tomography (CT) scans with late contrast administration to define areas of fibrosis can be useful to guide ablation (Table 261-2).

TABLE 261-2 Summary of Randomized Controlled Studies Assessing Catheter Ablation of Ventricular Tachycardia in Patients with Structural Heart Disease

STUDY	YEAR	STUDY PERIOD	FOLLOW-UP (months)	CA	OC	INCLUSION CRITERIA	CONTROL ARM	SMASH-VT
2000–2006								
Prior MI, ICD for VF or unstable VT Medical therapy 67 ± 10								
32 ± 9	22.5 ± 5.5	PART 6	Disorders of the Cardiovascular System	VTACH	2010	2002–2006		
Prior MI, LVEF ≤50%, ICD indicated for stable VT CALYPSO,								
2012–2014								
IHD, ≥1 ICD shock or ≥3 ATP therapies for monomorphic VT in last 6 months VANISH,								
2009–2015								
Prior MI, ICD in situ, ≥1 episode of VT while on a class I/III AAD SMS, 2017 2002–2010								
IHD, LVEF ≤40%, unstable VT ICD + medical therapy 67 ± 8								
31 ± 7	27.0 ± 13.2	BERLIN-VT,						
2015–2018								
Prior MI, LVEF 30–50%, ICD in situ for life-threatening VT PARTITA,								

2000–2006

Prior MI, ICD for VF or unstable VT Medical therapy 67 ± 10

32 ± 9 22.5 ± 5.5 PART 6 Disorders of the Cardiovascular System VTACH, 2010 2002–2006

Prior MI, LVEF ≤50%, ICD indicated for stable VT CALYPSO,

2012–2014

IHD, ≥1 ICD shock or ≥3 ATP therapies for monomorphic VT in last 6 months VANISH,

2009–2015

Prior MI, ICD in situ, ≥1 episode of VT while on a class I/III AAD SMS, 2017 2002–2010

IHD, LVEF ≤40%, unstable VT ICD + medical therapy 67 ± 8

31 ± 7 27.0 ± 13.2 BERLIN-VT,

2015–2018

Prior MI, LVEF 30–50%, ICD in situ for life-threatening VT PARTITA,

2012-2021

Cardiomyopathy, had first ICD shock SURVIVE-VT,

2010-2017

Prior MI, sustained VT causing ICD shock or syncope PAUSE-SCD,

2015-2020

LVEF <50%, ICD indicated for secondary prevention or inducible monomorphic VT on EPS

Abbreviations: AAD, antiarrhythmic drug; ATP, antitachycardia pacing; BERLIN VT, Preventive Ablation of Ventricular Tachycardia in Patients with Myocardial Infarction; CA, catheter ablation; CALYPSO, Catheter Ablation for Ventricular Tachycardia in Patients with an Implantable Cardioverter Defibrillator; EPS, electrophysiology study; ICD, implantable cardioverter-defibrillator; ICM, ischemic cardiomyopathy; IHD, ischemic heart disease; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NR, not reported; OC, other care; PARTITA, Does Timing of VT Ablation Affect Prognosis in Patients with an Implantable Cardioverter-Defibrillator?; PAUSE-SCD, Pan-Asia United States Prevention of Sudden Cardiac Death Catheter Ablation Trial; SMASH VT, Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia; SMS, Substrate Modification Study; SURVIVE-VT, Substrate Ablation Versus Antiarrhythmic Drug Therapy for Symptomatic Ventricular Tachycardia; VANISH, Ventricular Tachycardia Ablation Versus Escalated Antiarrhythmic Drug Therapy in Ischemic Heart Disease; VF, ventricular fibrillation; VT, ventricular tachycardia; VTACH, Ventricular Tachycardia Ablation in Coronary Heart Disease. Source: Reproduced with permission from SA Virk, S Kumar: Catheter ablation of ventricular tachycardia in patients with structural heart disease: A meta-analysis. JACC Clin Electrophysiol 9: 255; 2023. ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a rare genetic disorder most commonly due to mutations in genes encoding for cardiac desmosomal proteins; however, it is increasingly appreciated FIGURE 261-3 Cardiac magnetic resonance image (MRI). Shown is an MRI of the heart with the right ventricle on the left and the left ventricle on the right. Between the ventricles (arrows) is a stripe of late gadolinium enhancement, indicating midmyocardial fibrosis in the interventricular septum. This type of scar pattern is often seen in patients with nonischemic cardiomyopathies and ventricular tachycardia.

AGE (years) MALE (%) ICM (%) BASELINE LVEF (%) ICD + medical therapy 66 ± 8

34 ± 9 22.5 ± 9 AAD therapy $63 \pm \text{NR}$

$30 \pm \text{NR}$

Escalation of AAD therapy 69 ± 8

31 ± 11 27.9 ± 17.1 Medical therapy until third ICD shock (then VT ablation) 66 ± 10

41 ± 6 13.2 ± 9.5 Medical therapy 68 ± 9

32 ± 9 24.2 (8.5-24.4) AAD therapy 70 ± 9

ICD + medical therapy 55 (46–64)

40 (30–49) 31.3 (20.1–40) that other cardiomyopathic processes may produce a similar phenotype. Approximately 50% of patients have a familial transmission with autosomal dominant inheritance. A less common, autosomal recessive form is classically associated with cardiocutaneous syndromes that include Naxos disease and Carvajal syndrome. The former is most commonly related to mutations in plakophilin-2 and plakoglobin, while the latter is most commonly due to a mutation in desmoplakin. Patients are typically diagnosed between the second and fifth decades with palpitations, syncope, or cardiac arrest owing to sustained monomorphic VT, although polymorphic VT can also occur. Fibrosis and fibrofatty replacement most commonly involve the right ventricular myocardium and provide the substrate for reentrant VT that usually has a left bundle branch block-like configuration in ECG lead V1, consistent with the right ventricular origin, and can resemble idiopathic VT. The sinus rhythm ECG suggests the disease in >85% of patients, most often showing T-wave inversions in V1–V3. Delayed activation of the right ventricle may cause a widened QRS (>110 ms) in the right precordial leads (V1–V3) and a prolonged S-wave upstroke in those leads and, occasionally, a notched deflection at the end of the QRS known as an epsilon wave. Cardiac imaging may show right ventricular enlargement or areas of abnormal motion or reveal areas of scar on contrast-enhanced MRI. LV involvement can occur and can occasionally precede manifest right ventricular disease. Clinical heart failure is rare except in late stages, and survival to advanced age can be anticipated provided that VT can be controlled. An ICD is recommended. When VT is exercise induced, it may respond to β -adrenergic blockers and limiting exercise. Sotalol, flecainide, and amiodarone have been used to reduce ventricular arrhythmias. Catheter ablation prevents or reduces VT episodes in 70% of patients, but epicardial mapping and ablation are often required. ADULT CONGENITAL HEART DISEASE Among all patients with adult congenital heart disease (ACHD), sustained monomorphic VT is quite rare. However, the most common substrate for sustained VT is seen in those with repairs of a ventricular

septal defect, in particular tetralogy of Fallot (TOF). The prevalence of VT after TOF repair is estimated to be 3–14%, and risk of sudden cardiac death may reach as high as 1% per year in adulthood by the fourth or fifth decade of life. The greatest risk for ventricular arrhythmias is posed via two potential mechanisms: (1) those who have undergone repair involving a ventriculotomy and (2) those with long-standing hemodynamic overload causing ventricular dysfunction and/or hypertrophy independent of surgical incisions. Monomorphic VT in TOF most commonly occurs in stereotyped circuits due to reentry around areas of surgically created scar in the right ventricle. Factors associated with VT risk include age >5 years at the time of repair, high-grade ventricular ectopy, inducible VT on an electrophysiologic study, abnormal right ventricular hemodynamics, and sinus rhythm QRS duration >180 ms. An ICD is usually warranted for patients who have a spontaneous episode of VT, but ICDs are also considered for patients with multiple risk factors. Catheter ablation or antiarrhythmic drug therapy is used to control recurrent episodes. BUNDLE BRANCH REENTRY VT Reentry through the Purkinje system occurs in ~5% of patients with monomorphic VT in the presence of structural heart disease. The reentry circuit typically revolves retrograde via the left bundle and anterograde down the right bundle, thereby producing VT that has a left bundle branch block configuration. The VT QRS morphology may closely resemble the QRS morphology in sinus rhythm. Catheter ablation of the right bundle branch abolishes this VT.

Bundle branch reentry is usually associated with severe underlying heart disease. Other scar-related VTs are often present and often require additional therapy. IDIOPATHIC MONOMORPHIC VT

Idiopathic VT in patients without structural heart disease usually presents with palpitations, lightheadedness, and, rarely, syncope. Episodes can be provoked either by sympathetic stimulation or acute withdrawal of sympathetic tone, as in the immediate postexercise period. The QRS morphology of the arrhythmia suggests the diagnosis (see below). The sinus rhythm ECG is normal. Family history suggests no familial cardiomyopathy or sudden death. Cardiac imaging, including echocardiography and cardiac MRI, shows normal ventricular function and no evidence of ventricular scar. Occasionally, a patient with structural heart disease is found to have concomitant idiopathic VT unrelated to it.

FIGURE 261-4 Idiopathic monomorphic ventricular tachycardia (VT). This is a 12-lead electrocardiogram showing the onset of idiopathic VT in a young patient without structural heart disease. The VT has a left bundle branch block configuration in V1 and an inferiorly directed axis consistent with an outflow tract origin. Note that the narrow (normal sinus) beats have a normal QRS configuration, consistent with the patient's lack of structural heart disease.

the structural disease, in which case, the underlying disease should be treated as per the guidelines, separate from the VT. Repeated bursts of nonsustained VT, which may occur incessantly, are known as repetitive monomorphic VT and can cause a tachycardia-induced cardiomyopathy with depressed ventricular function that recovers after suppression of the arrhythmia. Sudden death in isolated idiopathic VT is rare, and an ICD is not recommended.

CHAPTER 261 Outflow tract VTs originate from a focus near the pulmonic or aortic valve annuli, usually with features consistent with triggered automaticity. The arrhythmia may present with sustained VT, nonsustained VT, or premature ventricular contractions (PVCs). Most originate in the right ventricular outflow tract, which gives rise to VT that has a left bundle branch block configuration in V1 and an axis that is directed inferiorly, with tall R waves in II, III, and aVF. Idiopathic VT can also arise in the LV outflow tract or in sleeves of myocardium that extend along the aortic root. LV origin is suspected when lead V1 or V2 has prominent R waves. Although this typical outflow tract QRS morphology favors idiopathic VT, some cardiomyopathies, notably ARVC, can cause PVCs or VT from this region. Excluding these diseases is an initial focus of evaluation (Fig. 261-4). Sustained Ventricular Tachycardia LV fascicular VT, sometimes referred to as Belhassen's VT or verapamil-sensitive VT, is the second most common form of idiopathic VT after outflow tract VTs. It often presents with sustained VT that has a right bundle branch block-like configuration and is negative in the inferior leads. It is often exercise induced and occurs more often in men than women. The mechanism was originally thought to be focal but has been demonstrated to be due to a small reentry circuit in or near the septal ramifications of the LV Purkinje system. There can be an LV false tendon associated with this rhythm. Despite its reentrant nature, the course of this type of VT is typically benign. Other sites of origin for idiopathic VT exist, including papillary muscles, mitral and tricuspid valve annuli, and the moderator band in the right ventricle. Even focal sites from the epicardial surface have been described. The presence of VT from these more unusual sites should prompt even more careful assessment for structural heart disease.

MANAGEMENT OF IDIOPATHIC VT Treatment is required for symptoms or when frequent or incessant arrhythmias depress ventricular function. Symptoms can be controlled with medications including beta blockers, calcium channel blockers,

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