Sustained VentricularTachycardia

KEY CLINICAL POINTS

1. See source text for full details

FIGURES IN THIS CHAPTER

RAW CONTENT

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I aVR V1 V4

II aVL V2 V5

III aVF V3 V6

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 V and narrower than the rest of the beats. These features are consistent with an accelerated idioventricular rhythm.

1

myocardium can cause AIVR. Idioventricular rhythms are common

during acute MI and may emerge during sinus bradycardia. Often, they 261 Sustained Ventricular

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.

Sustained Monomorphic 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

William H. Sauer, Usha B. Tedrow

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.

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

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 Europace 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.

VT versus Supraventricular Tachycardia (SVT)

TABLE 261-1 Sustained Ventricular Arrhythmias

with Aberrancy

1. Idiopathic ventricular tachycardia (VT) without structural heart disease

A. Outflow tract origin

• Right ventricular (RV) outflow tract: left bundle branch block pattern in

Yes

V with inferior axis (tall QRS in inferior leads) and late transition in the AV dissociation VT

1 precordial leads

• Left ventricular (LV) outflow tract: similar inferiorly directed axis but No

with early precordial transition with prominent R wave in V–V

2 3 aVR aVR

B. LV fascicular VT: Typical right bundle branch block pattern in V with sharp Yes

1 aVR = R or Rs VT

intrinsicoid deflection and left axis deviation (arising from left posterior

fascicle in its most common form)

C. Papillary muscle VT

No

• Posteromedial: atypical right bundle branch block pattern in V with

1 V V V V V V

monophasic R wave and left axis deviation Yes 1 2 3 4 5 6

No rS or Rs in VT

• Anterolateral: atypical right bundle branch block pattern in V with

positive deflection in lead III and negative deflection in lead I 1 any of V 1 to V 6

2. Ischemic cardiomyopathy

No

• Monomorphic VT is common with prior large myocardial infarction V 1 V 2 V 3 V 4 V 5 V 6

• Polymorphic VT and ventricular fibrillation (VF) should prompt ischemia

Possible SVT with aberrancy

evaluation

VT still possible

3. Nonischemic cardiomyopathy

FIGURE 261-1 Algorithm for differentiation of ventricular tachycardia (VT) from

• Fibrotic scars can cause monomorphic VT, especially with sarcoidosis

supraventricular tachycardia with aberration. AV, atrioventricular.

or other inflammatory cardiomyopathies, Chagas' disease, and

familial arrhythmogenic cardiomyopathies such as Lamin A/C genetic

cardiomyopathy ventriculoatrial (VA) dissociation is a reliable marker for VT, provided

• Polymorphic VT and VF can also occur independently or related to the atrial rate is slower than the ventricular rate. Sometimes, P waves

degeneration of monomorphic VT can be difficult to define, and the VA relationship cannot be assessed in

4. Arrhythmogenic RV cardiomyopathy a patient with an ongoing atrial arrhythmia such as atrial fibrillation. A

• Monomorphic VT usually of RV origin (left bundle branch morphology in V) P wave following each QRS does not exclude VT because 1:1 conduc-

1

• Polymorphic VT and VF can occur independently or related to degeneration tion from ventricle to atrium can occur. A monophasic R wave or Rs

of monomorphic VT complex in aVR or concordance from V to V of monophasic R or

1 6

5. Repaired tetralogy of Fallot S waves is also relatively specific for VT (Fig. 261-1). A number of

• Monomorphic VT of RV origin (usually left bundle branch morphology in V) other QRS morphology criteria have also been described, but all have

1

6. Hypertrophic cardiomyopathy limitations and are not very reliable in patients with severe heart

• Polymorphic VT or ventricular fibrillation disease. In patients with known bundle branch block, the same QRS

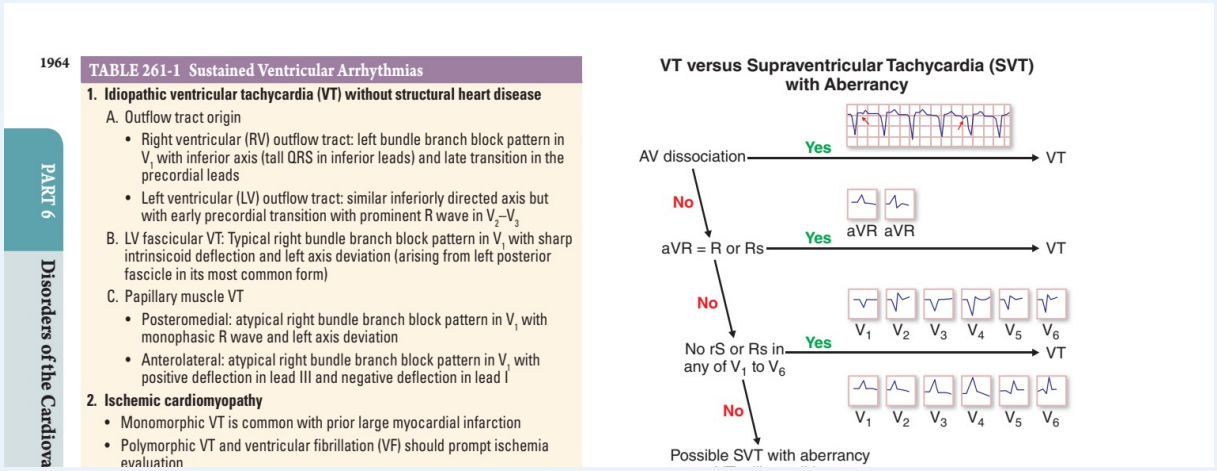
• Less commonly, monomorphic VT associated with myocardial scars, morphology during tachycardia as during sinus rhythm suggests

particularly apical aneurysms supraventricular tachycardia rather than VT, but even this is not abso-

7. Genetic arrhythmia syndromes lutely reliable. Patients with reentry involving the bundle branches of

the Purkinje system can have a VT morphology that res

FLOWCHARTS & ALGORITHMS — FROM HARRISON'S



1964

PART 6 Disorders of the Cardiova

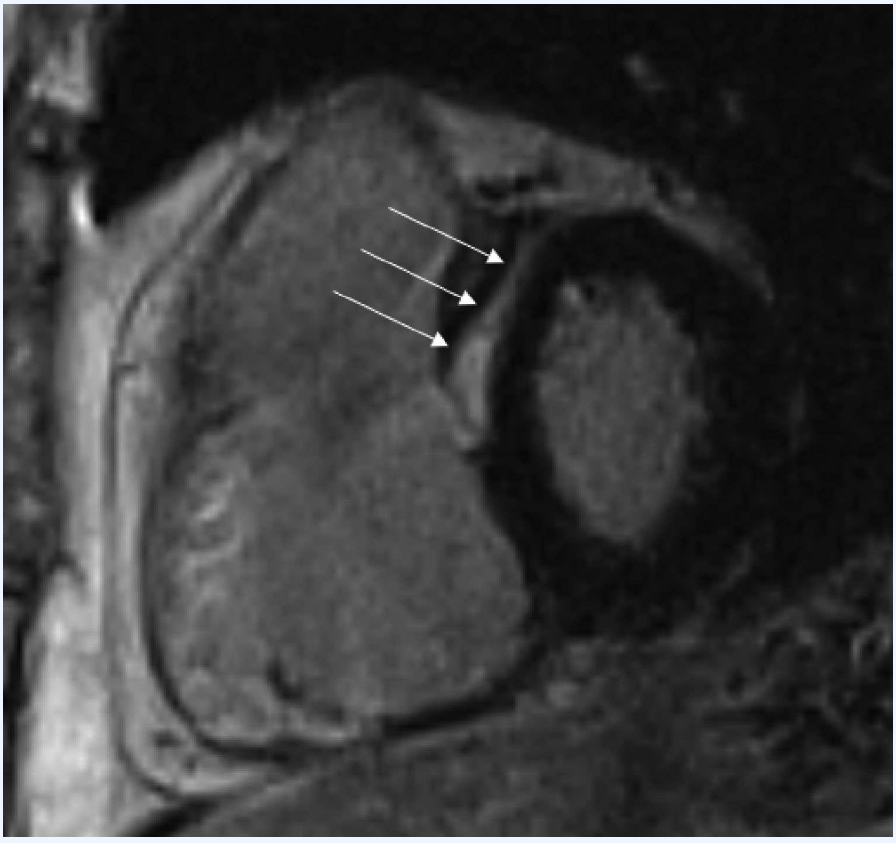
TABLE 261-1 Sustained Ventricular Arrhythmias

1. **Idiopathic ventricular tachycardia (VT) without structural heart disease**
   - A. Outflow tract origin
     - Right ventricular (RV) outflow tract: left bundle branch block pattern in V_1 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 with early precordial transition with prominent R wave in V_2–V_3
   - B. LV fascicular VT: Typical right bundle branch block pattern in V_1 with sharp intrinsicoid deflection and left axis deviation (arising from left posterior fascicle in its most common form)
   - C. Papillary muscle VT
     - Posteromedial: atypical right bundle branch block pattern in V_1 with monophasic R wave and left axis deviation
     - Anterolateral: atypical right bundle branch block pattern in V_1 with positive deflection in lead III and negative deflection in lead I
2. **Ischemic cardiomyopathy**
   - Monomorphic VT is common with prior large myocardial infarction
   - Polymorphic VT and ventricular fibrillation (VF) should prompt ischemia evaluation

Harrison's 22e · Flowchart 1

FIGURE 261-1 Algorithm for differentiation of ventricular tachycardia (VT) from supraventricular tachycardia with aberration. AV, atrioventricular.

FIGURES & ILLUSTRATIONS — FROM HARRISON'S

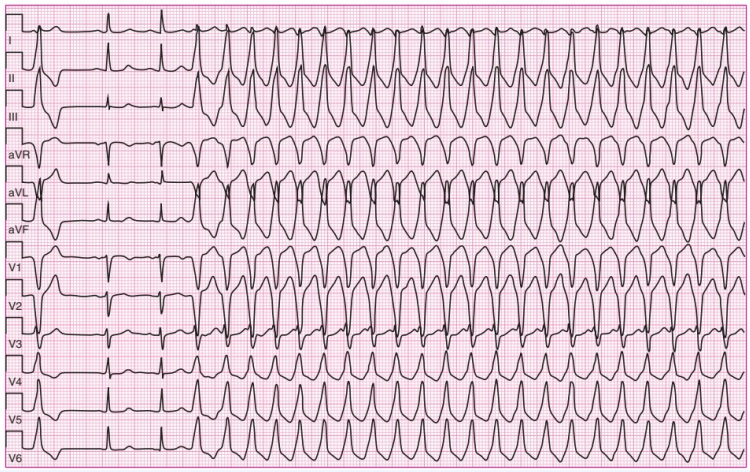


Harrison's 22e · Figure 1

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.

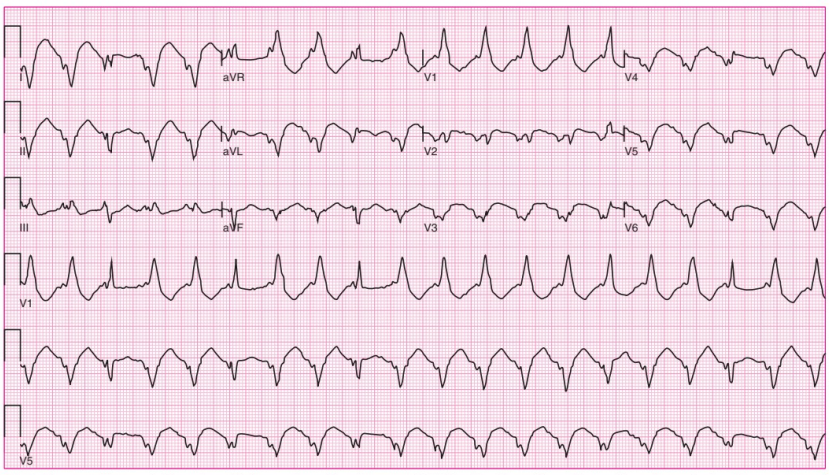
heart disease is found to have concomitant idiopathic VT unrelated to

with medications including beta blockers, calcium channel blockers,



Harrison's 22e · Figure 2

FIGURE 261-4 Idiopathic monomorphic ventricular tachycardia (VT). This is a 12-lead structural heart disease. The VT has a left bundle branch block configuration in V and an 1 (normal sinus) beats have a normal QRS configuration, consistent with the patient's lack



1965

CHAPTER 261 Sustained Ventricular Tachycardia

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.

Harrison's 22e · Figure 3

FIGURE 261-2 Monomorphic ventricular tachycardia in a patient with prior myocardial narrower and are examples of fusion beats, proving ventriculoatrial (VA) dissociation and SUSTAINED MONOMORPHIC VT IN

Harrison's 22e · Figure 4

FIGURE 260-4 Accelerated idioventricular rhythm. Shown is an example of a slow more positive in lead V and narrower than the rest of the beats. These features are 1