Ventricular Arrhythmias

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KEYWORDS

• Ventricular tachycardia • Ventricular fibrillation • Catheter ablation
• Implantable cardioverter-defibrillator

KEY POINTS

- Ventricular tachycardia (VT) is the most common form of wide complex tachycardia and associated with a high mortality rate.
- The electrocardiogram is paramount in diagnosis, but distinguishing VT from supraventricular tachycardia with aberrant conduction may be difficult; the diagnosis of VT should be assumed until proven otherwise.
- Catheter ablation of VT is an effective treatment modality typically used after antiarrhythmic drug failure, but recurrent VT is not uncommon.
- The implantable cardioverter-defibrillator aids in the acute termination of ventricular arrhythmia.
- Its pacing and sensing algorithms are helpful in improving the diagnostic yield and long-term management of patients with ventricular arrhythmia.

INTRODUCTION

Ventricular tachycardia (VT) is the most common cause of wide complex tachycardia.[1] It is associated with ischemic and nonischemic cardiomyopathies, cardiac channelopathies, and toxic metabolic conditions, or may exist as an idiopathic process in structurally normal hearts. Of the more than 130 million emergency department (ED) visits in the United States each year, cardiac dysrhythmias were the fifth most common reason for presentation in patients between the ages of 65 and 84 years, accounting for more than 278,000 visits.[2] Sustained VT occurs much less frequently, however, accounting for only 0.05% of ED visits in 1 series,[3] but occurs at a much higher frequency in critical care settings, with an incidence of 2% to 7%.[4,5]

Clinical presentations of ventricular tachyarrhythmias may range from the asymptomatic to implantable cardioverter-defibrillator (ICD) shocks, cardiogenic shock, and cardiac arrest. Syncope or near syncope secondary to cerebral hypoperfusion is relatively common, occurring in 30% of patients with sustained VT of more than 30 minutes' duration in individuals not presenting with myocardial ischemia or sudden death. Patients were more likely to present with syncope when the VT was greater than 200 bpm and chest pain in those with a background history of coronary artery disease (33% vs 12%).[6] Similar findings were seen in another series, in which 77% of patients in sustained VT were hemodynamically stable upon evaluation.[3] Hemodynamic compromise is variable and due to a number of different factors, including a rate-related decrease in diastolic filling time, incomplete myocardial relaxation, and a dyssynchronous ventricular activation pattern resulting in decreased pump function, loss of atrial transport, and possibly mitral regurgitation.[7]

Ventricular arrhythmia often results in hospital admission. Those with a hemodynamically stable VT are most commonly admitted to a

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non–intensive care unit setting (72%), but rarely discharged directly from the ED (16%), which mostly results from extenuating clinical circumstances, such as patient refusal or goals of care that necessitate a less aggressive clinical approach. Patients with a hemodynamically unstable VT are more likely admitted to an intensive care unit setting versus a general ward (41% vs 35%), with the remaining unstable patients not surviving past their initial ED evaluation,[3] thus highlighting the severity of the condition.

Definitions of Ventricular Arrhythmia

- Sustained VT: persists for greater than 30 seconds and/or requiring termination owing to hemodynamic compromise in less than 30 seconds.
- Monomorphic VT: single QRS morphology and stable cycle length.
- Polymorphic VT: QRS morphologies and cycle length vary considerably.
- VT storm: Fewer than 2 to 3 episodes of hemodynamically significant VT or 3 or more shocks in 24 hours.
- Incessant VT: unable to maintain sinus rhythm.
- Ventricular fibrillation (VF): rapid (usually >300 bpm) disorganized low-amplitude polymorphic ventricular activity.

DIAGNOSIS OF VENTRICULAR ARRHYTHMIA

Differentiation of Ventricular Tachycardia from Supraventricular Tachycardia on the 12-Lead Electrocardiograph

An important distinction in patients presenting with wide complex tachycardia is the differentiation of VT from supraventricular tachycardia (SVT). Hemodynamic instability is not useful in the differentiation but prior myocardial infarction, heart failure, and recent angina pectoris have a positive predictive value for the diagnosis of VT of 98%, 100%, and 100%, respectively.[8]

Multiple algorithms for the ECG diagnosis of VT have been proposed but none are 100% specific.[9] Common criteria used include:

- Absence of an RS complex in the precordial leads[10];
- An RS interval of greater than 0.1 second in any precordial lead[10];
- Atrioventricular dissociation with or without fusion with or without capture beats[10,11]; and
- VT is more likely in a right bundle branch block configuration when[11]:
  - QRS duration is greater than 0.14 second;
  - There are QR, R, or RSr' configurations in lead V1; and
  - The RS ratio is 1 or greater or there is a QS wave in lead V6.
- VT is more likely in a left bundle branch block configuration when[11]:
  - The QRS complex duration is greater than 0.16 second;
  - The initial R wave is greater than 0.03 second;
  - There is slurring or notching of the down stroke of the S wave or QRS complex onset to nadir of S wave is greater than 0.07 sec in lead V1; and
  - There is any Q wave in lead V6.
- When the diagnosis remains uncertain, it is preferable to assume VT rather than SVT. The 12-lead ECG is also useful in determining the exit site of VT and whether it is endocardial or epicardial, an important distinction when considering catheter ablation (**Fig. 1**).

A 12-Lead Electrocardiograph After Termination of Ventricular Tachycardia

Repeat ECG should be obtained immediately after termination of VT. This can provide useful clues as to the underlying etiology. For example,

- Q waves indicative of prior infarction;
- ST segment changes consistent with acute ST elevation myocardial infarction or myocardial ischemia;
- ECG changes suggestive of hyperkalemia or drug intoxication;
- Left ventricular hypertrophy and T wave changes suggestive of hypertrophic cardiomyopathy;
- Brugada's syndrome (**Fig. 2**);
- Prolonged QTc; and
- Epsilon waves indicative of arrhythmogenic right ventricular dysplasia.

Specific Etiologies of Ventricular Arrhythmia

1. Idiopathic VT without structural heart disease
   - Right ventricular outflow tract tachycardia: most commonly presents with frequent premature ventricular contractions, but can develop both repetitive monomorphic salvoes of VT and sustained rapid VT, often provoked by exertion. The ECG morphology is a left bundle branch block, inferior axis. Unlike arrhythmogenic right ventricular dysplasia, the right ventricle is not dilated or scarred. Beta-blockers may ameliorate symptoms, but patients with symptomatic high premature ventricular contraction burden (>10,000/24 hours) and sustained VT are good candidates for catheter ablation. Ablation is usually