tricyclic antidepressants), and severe global myocardial ischemia are 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 "twisting about the points" appearance referred to as *Torsade de Pointes* (Fig. 277-3*D*).

Ventricular fibrillation (VF) has continuous irregular activation with no discrete QRS complexes (Fig. 277-3*E*). Monomorphic or polymorphic VT may transition to VF in susceptible patients.

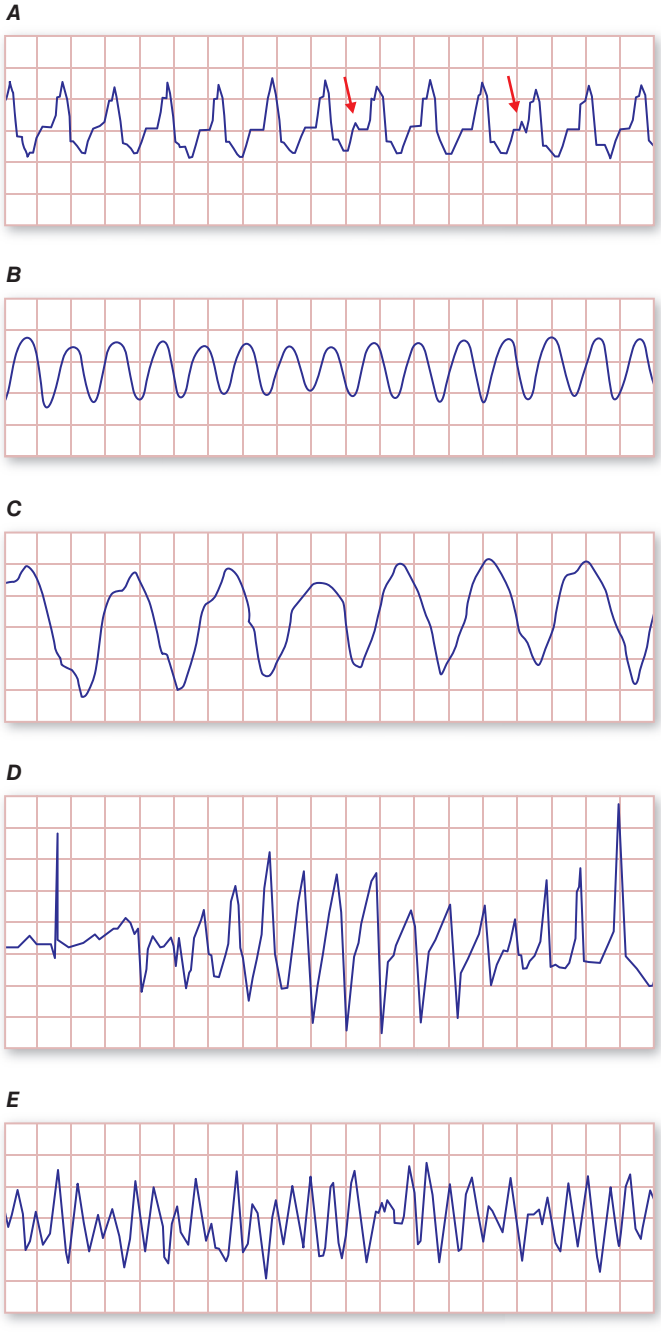

FIGURE 277-3 Examples of types of ventricular tachycardia (VT). ***A.*** Monomorphic VT with dissociated P waves (*short arrows*). ***B.*** Ventricular flutter. ***C.*** Sinusoidal VT due to electrolyte disturbance or drug effects. ***D.*** Polymorphic VT resulting from prolongation of QT interval (torsade de pointes VT). ***E.*** Ventricular fibrillation.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

Common symptoms of ventricular arrhythmias include palpitations, dizziness, exercise intolerance, episodes of lightheadedness, syncope, or sudden death. These arrhythmias can be asymptomatic and encountered unexpectedly as an irregular pulse or heart sounds on examination, or seen on a routine electrocardiogram (ECG), exercise test, or cardiac ECG monitoring.

Syncope is a concerning symptom that can be due to an episode of VT with hypotension. Syncope due to a ventricular arrhythmia often indicates that there is a significant risk for subsequent cardiac arrest and sudden death with arrhythmia recurrence. Although benign causes of syncope, such as reflex-mediated neurocardiogenic (vasovagal) syncope and orthostatic hypotension, are generally more common, it is important to consider the possibility of 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 with cardiac arrest, often with degeneration of the VT to VF. Occasionally a sustained VT will be hemodynamically tolerated and present with diminished exercise capacity or exacerbation of heart failure. Many patients who are at risk for VT have known heart disease and may have an implantable cardioverter-defibrillator (ICD). In patients with an ICD, spontaneous episodes of VT may elicit an episode of transient lightheadedness, palpitations, or syncope that may be followed by a shock from the ICD (see below).

The diagnosis of ventricular arrhythmias is established by recording of the arrhythmia on an ECG or, in some cases, initiation of the arrhythmia during an electrophysiologic study (Table 277-1). 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). When the arrhythmia is intermittent with days to weeks between symptoms, prolonged ambulatory monitoring to capture the ECG at the time of symptoms is required to make the diagnosis. Continuous ambulatory monitoring or looping event recording monitors are options. Exercise testing should be considered in patients with exercise-induced symptoms.

APPROACH TO THE PATIENT: Documented or Suspected Ventricular Arrhythmias

Initial assessment focuses on hemodynamic stability and evaluation for underlying heart disease. A family history of sudden death or cardiomyopathy suggests the possibility of a genetic basis for the arrhythmia and greater risk. The electrocardiogram can provide important clues. Patients with benign idiopathic arrhythmias usually have a completely normal ECG during sinus rhythm.

Cardiac imaging is warranted to assess ventricular function and look for evidence of depressed ventricular function indicative of a cardiomyopathy or ventricular hypertrophy that may indicate hypertrophic cardiomyopathy. Cardiac magnetic resonance imaging (MRI) with late gadolinium enhancement can detect areas of ventricular scar, which are usually present in patients who are at risk for sustained monomorphic VT (Fig. 277-5). Evaluation to exclude atherosclerotic coronary artery disease should be performed in patients at risk, guided by age and other risk factors.

SPECIFIC ARRHYTHMIAS

PVCs and Nonsustained VT Ventricular extrasystoles (Fig. 277-1*A*) can be due to automaticity or reentry (Chap. 278e). PVCs can be a sign of increased sympathetic tone; myocardial ischemia; hypoxia; electrolyte abnormalities, particularly hypokalemia; or underlying heart disease. During myocardial ischemia or in association with other heart disease, PVCs can be a harbinger of sustained VT or