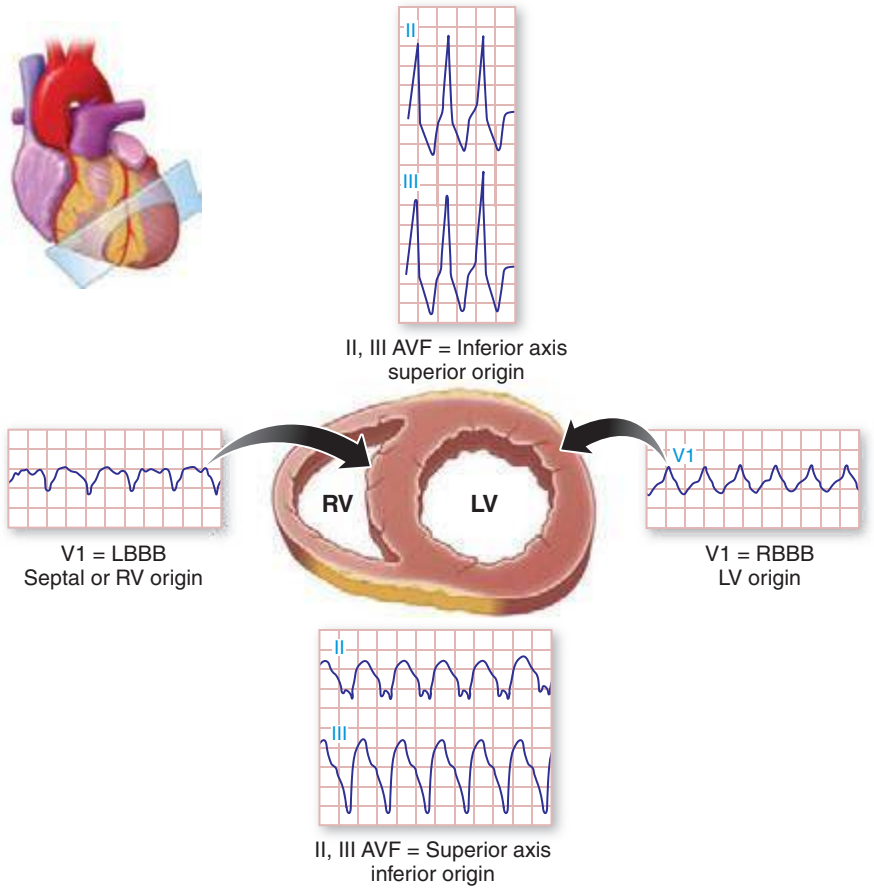

FIGURE 277-4 Site of VT origin based on QRS morphology. LBBB, left bundle branch block; LV, left ventricle; RBBB, right bundle branch block; RV, right ventricle.

VF. In patients with heart disease, a higher frequency of ectopy and complexity (couplets and nonsustained VT) are associated with more severe disease and, in those with heart failure, with increased mortality. However, suppression of these arrhythmias with antiarrhythmic drugs does not improve survival. In the absence of cardiac disease, PVCs and nonsustained VT generally have a benign prognosis. PVCs that occur at a bigeminal frequency may not generate sufficient cardiac output for a radial pulse and hence may register at rates half that of the heart rate (Fig. 277-1*A*). Very frequent PVCs can depress ventricular function (see below).

EVALUATION AND MANAGEMENT When encountered during acute illness or as a new finding, evaluation should focus on detection and correction of potential aggravating factors and causes, specifically myocardial ischemia, ventricular dysfunction, and electrolyte abnormalities, most commonly hypokalemia. Underlying heart disease should be defined.

The ECG characteristics of the arrhythmia are often suggestive of whether structural heart disease is present. PVCs with smooth uninterrupted contours and sharp QRS deflections suggest an ectopic focus in relatively normal myocardium, whereas broad notching and slurred QRS deflections suggest a diseased myocardial substrate. The most frequent site of origin for idiopathic ventricular arrhythmias is the right ventricular outflow tract, giving rise to PVCs or VT that have a left bundle branch block configuration, with an inferiorly directed frontal plane axis as discussed below (Fig. 277-2). However, QRS morphology alone is not reliable as an indicator of disease or subsequent risk. Nonsustained VT is usually monomorphic with rates less than 200 beats/min and typically lasts less than 8 beats (Fig. 277-2). Nonsustained VT that is very rapid, polymorphic, or with a first beat that occurs prior to the peak of the T wave ("short-coupled") is uncommon and should prompt careful evaluation for underlying disease or genetic syndromes associated with sudden death.

A family history of sudden death should prompt evaluation for genetic syndromes associated with sudden death, including cardiomyopathy, long QT syndrome, and arrhythmogenic right ventricular cardiomyopathy (see below). Any abnormality on the 12-lead ECG warrants further evaluation **(Fig. 277-6)**. Repolarization abnormalities are seen in a number of genetically determined syndromes associated with sudden death, including the long QT syndrome, Brugada syndrome, arrhythmogenic right ventricular cardiomyopathy (ARVC), and hypertrophic cardiomyopathy. An echocardiogram is often necessary to assess ventricular function, wall motion abnormalities, and valvular heart disease. Cardiac magnetic resonance (CMR) imaging is also useful for this purpose and for the detection of ventricular scarring that is the substrate for sustained VT (Fig. 277-5). Exercise stress testing should be performed in patients with effort-related symptoms and in those at risk for coronary artery disease.

IDIOPATHIC PVCS AND NONSUSTAINED VT For PVC and nonsustained VT in the absence of structural heart disease or a genetic sudden death syndrome, no specific therapy is needed unless the patient has significant symptoms or evidence that frequent PVCs are depressing ventricular function (see below). Reassurance that the arrhythmia is benign is often sufficient to allow the patient to cope with the symptoms, which will often wax and wane in frequency over years. Avoiding stimulants, such as caffeine, is helpful in some patients. If symptoms require treatment, β-adrenergic blockers and nondihydropyridine calcium channel blockers (verapamil and diltiazem) are sometimes helpful **(see Table 276-3)**. If these fail, more potent antiarrhythmic drugs or catheter

TABLE 277-1 DIAGNOSTIC TESTS FOR VENTRICULAR ARRHYTHMIAS

I.	12-Lead ECG
	A. Should be obtained for PVCs, nonsustained VT, and monomorphic VT when possible
	B. QRS morphology suggests ventricular region of origin
	V1 – dominant S = septum or RV
	V1 – dominant R = LV
	Superior axis = inferior wall origin
	Inferior axis = outflow region or anterior wall
II.	Ambulatory monitoring
	A. 24- to 48-h continuous Holter monitor
	Useful for evaluation of daily symptoms to quantitate PVCs
	B. Event recorder: can be used for weeks at a time
	Useful for evaluation of infrequent symptoms
	Some require patient activation and will miss asymptomatic arrhythmias
III.	Exercise testing
	A. Useful for evaluating exercise-induced arrhythmias and symptoms
	B. QT interval response to exercise may be abnormal in long QT syndrome
IV.	Invasive electrophysiology study
	A. Can establish definitive diagnosis of VT versus supraventricular tachycardia with aberrancy or ventricular preexcitation
	B. Can provoke some arrhythmias that are otherwise infrequent
	C. Allows potential catheter ablation
	D. Procedural risks determined by vascular access, whether ablation is performed, and the location of the arrhythmia substrate

Abbreviation: RV, right ventricle. See text for other abbreviations.