therapy to protect against sudden death in patients at high risk and are recommended for those with LV ejection fraction <0.35 and New York Heart Association class II and III heart failure, in whom they reduce mortality by 20%, from 36% to 29%, over 5 years.

OTHER CARDIAC DISEASES Ventricular ectopy is associated with increased mortality in patients with *hypertrophic cardiomyopathy* (Chap. 287) or with *congenital heart disease* (Chap. 282) associated with right ventricular or LV dysfunction. In these patients, management is similar to that for patients with ventricular dysfunction. Pharmacologic suppression of the arrhythmia has not been shown to improve mortality. ICDs are indicated for patients considered at high risk for sudden cardiac death.

PVC-INDUCED VENTRICULAR DYSFUNCTION Very frequent ventricular ectopy and repetitive nonsustained VT (Fig. 277-2) can depress ventricular function, possibly through an effect similar to chronic tachycardia or by inducing ventricular dyssynchrony. Depression of ventricular function rarely occurs unless PVCs account for more than 10–20% of total beats over a 24-h period. Often the PVCs are idiopathic and unifocal, most commonly originating from the LV papillary muscles or outflow tract regions where they can be targeted for ablation. The distinction between PVC-induced ventricular dysfunction as compared to a cardiomyopathic process causing ventricular dysfunction and arrhythmia is difficult and in some cases can be made only retrospectively by observing an improvement in ventricular function after the arrhythmia is suppressed with an antiarrhythmic drug, such as amiodarone, or by catheter ablation.

Idioventricular Rhythms Three or more ventricular beats at a rate slower than 100 beats/min are termed *idioventricular rhythm* (Fig. 277-1*C*). Automaticity is the likely mechanism. Idioventricular rhythms are common during acute MI (Chap. 295) and may emerge during sinus bradycardia. Atropine may be administered to increase the sinus rates if the loss of atrioventricular synchrony leads to hemodynamic compromise. 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 therapy for asymptomatic idioventricular rhythm is not necessary.

Sustained Monomorphic VT Sustained monomorphic VT presents as a wide QRS tachycardia that has the same QRS configuration from beat to beat, indicating an identical sequence of ventricular depolarization for each beat (Fig. 277-3A). VT originates from a stable focus or reentry circuit. In structural heart disease, the substrate is often an area of patchy replacement fibrosis due to infarction, inflammation, or prior cardiac surgery that creates anatomical or functional reentry pathways (Fig. 277-5). Less commonly, VT is related to reentry or automaticity in a diseased Purkinje system. In the absence of structural heart disease, idiopathic VT can present as sustained monomorphic VTs that are due to focal automaticity or reentry involving a portion of the Purkinje system.

The clinical presentation can vary depending on the rate of the arrhythmia, underlying cardiac function, and autonomic adaptation in response to the arrhythmia. Whereas patients with normal cardiac function might tolerate rapid VTs, those with severe LV dysfunction often experience symptoms of hypotension, even if VT is not particularly fast. Monomorphic VT may deteriorate to VF, which may be the initial cardiac rhythm recorded at the time of resuscitation.

DIAGNOSIS Sustained monomorphic VT has to be distinguished from other causes of uniform wide QRS tachycardia. These include supraventricular tachycardia with left or right bundle branch block aberrant conduction, supraventricular tachycardias conducted to the ventricles over an accessory pathway (Chap. 276), and rapid cardiac pacing in a patient with a pacemaker or defibrillator. In the presence of known heart disease, VT is the most likely diagnosis of a wide QRS tachycardia. Hemodynamic stability during the arrhythmia does not exclude VT. A number of ECG criteria have been evaluated. The presence of AV dissociation is usually a reliable marker for VT (Fig. 277-7), but P waves can be difficult to define. 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 V_1 to V_6 of monophasic R or S waves is also relatively specific for VT (Fig. 277-7). 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 is not absolutely reliable. An electrophysiologic study is sometimes required for definitive diagnosis. Rarely, noise and movement artifacts on telemetry recordings can simulate VT; prompt recognition can avoid unnecessary tests and interventions.

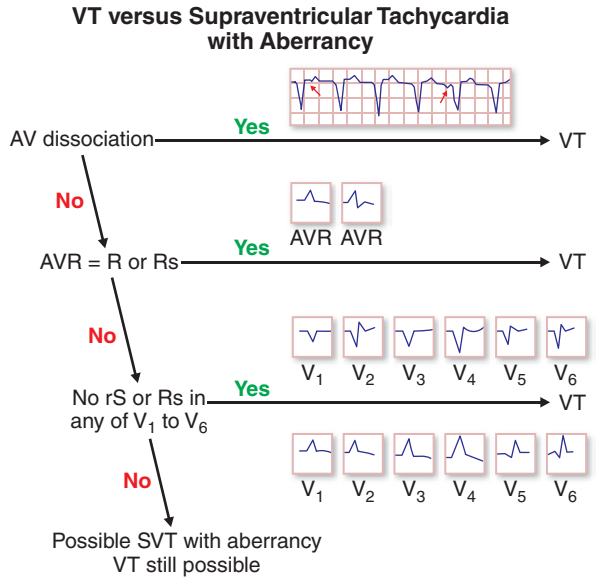

FIGURE 277-7 Algorithm for differentiation of ventricular tachycardia (VT) from supraventricular tachycardia (SVT) with aberration. AV, atrioventricular.

When LV function is depressed or there is evidence of structural myocardial disease, scar-related reentry is the most likely diagnosis. Scars are suggested by pathologic Q waves on the ECG, segmental left or right ventricular wall motion abnormalities on echocardiogram or nuclear imaging, and areas of delayed gadolinium enhancement during MRI (Fig. 277-5).

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 (Chap. 276). 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 MI is appropriate, but acute MI is rarely a cause of sustained monomorphic VT, and elevations in troponin or creatine kinase (CK)-MB are more likely to indicate myocardial damage that is secondary to hypotension and ischemia from the VT. 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 an isolated episode, but with a risk of recurrence. ICDs are usually considered for VT associated with structural heart disease.

Sustained Monomorphic VT in Specific Diseases • ***CORONARY ARTERY DISEASE*** Patients who present with sustained VT associated with coronary artery disease typically have a history of prior large MI and present years after