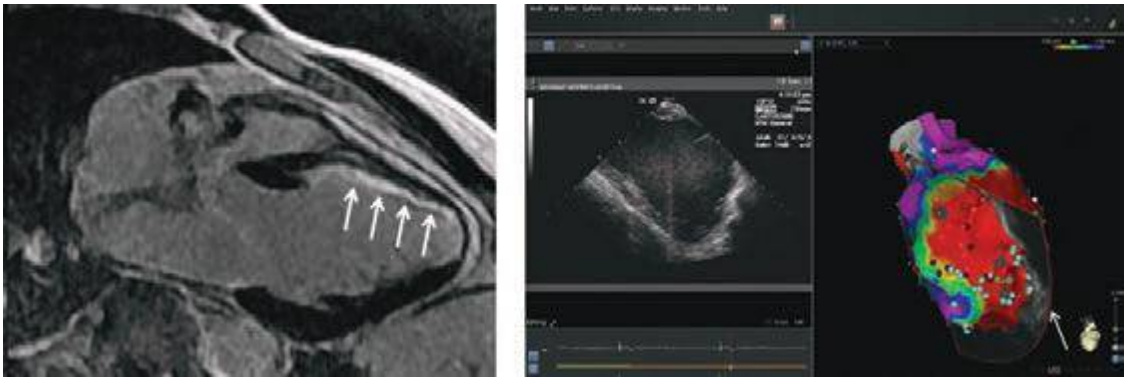

FIGURE 277-5 Imaging studies of the left ventricle (LV) used to assist ablation for ventricle tachycardia (VT). *Left panel* is a magnetic resonance image of a longitudinal section demonstrating thinning of the anterior wall and late gadolinium enhancement in a subendocardial scar (*white arrows*). The *middle panel* shows a two-dimensional image of the LV in long axis corresponding to the sector through the mid LV (*arrow, right panel*) obtained by an intracardiac echo probe positioned in the right ventricle. An electroanatomic three-dimensional map of the LV in the left anterior oblique projection is displayed in the right panel. The purple color depicts areas of normal voltage (>1.5 mV). Blue, green, and yellow represent progressively lower voltages with the red areas indicating scar (<0.5 mV). Channels of viable myocardium with slow conduction within the scar are identified with the light blue dots. Areas of ablation delivered to regions involved in reentrant VT are indicated by maroon dots.

ablation can be considered. The antiarrhythmic agents flecainide, propafenone, mexiletine, and amiodarone can be effective, but the potential for side effects warrants careful consideration. Catheter ablation can be effective if the arrhythmia occurs with sufficient frequency or is readily provoked such that its origin can be identified for ablation in a similar manner to that for idiopathic monomorphic VT as discussed below. Benefit must be carefully weighed against the procedure-related risks (see below).

PVCS AND NONSUSTAINED VT ASSOCIATED WITH ACUTE CORONARY SYNDROMES During and early after acute myocardial infarction (MI), PVCs and nonsustained VT are common and can be an early manifestation of ischemia and a harbinger of subsequent VF. Treatment with β-adrenergic blockers and correction of hypokalemia and hypomagnesemia reduce the risk of VF. Routine administration of the antiarrhythmic drugs such as lidocaine has not been shown to reduce mortality and is not indicated for suppression of PVCs or asymptomatic nonsustained VT.

Following recovery from acute MI, frequent PVCs (typically >10 PVCs per hour), repetitive PVCs with couplets, and nonsustained VT are markers for depressed ventricular function and increased mortality, but routine antiarrhythmic drug therapy to suppress these arrhythmias is not warranted. Treatment with the sodium channel blocker flecainide increased mortality. Amiodarone therapy reduces sudden death, but does not improve total mortality. Therefore, amiodarone is an option for treatment of symptomatic arrhythmias in this population when the potential benefit outweighs its potential toxicities. β-Adrenergic blockers reduce sudden death but have limited effect on spontaneous arrhythmias.

For survivors of an acute MI, an ICD reduces mortality in certain high-risk groups: patients who have survived >40 days after the acute MI and have a left ventricular (LV) ejection fraction of ≤0.30 or who have an ejection fraction <0.35 and have symptomatic heart failure (functional class II or III); and patients >5 days after MI who have a reduced LV ejection fraction, nonsustained VT, and inducible sustained VT or VF on electrophysiologic testing. ICDs do not reduce mortality when routinely implanted soon after MI or in patients after recent coronary artery revascularization surgery.

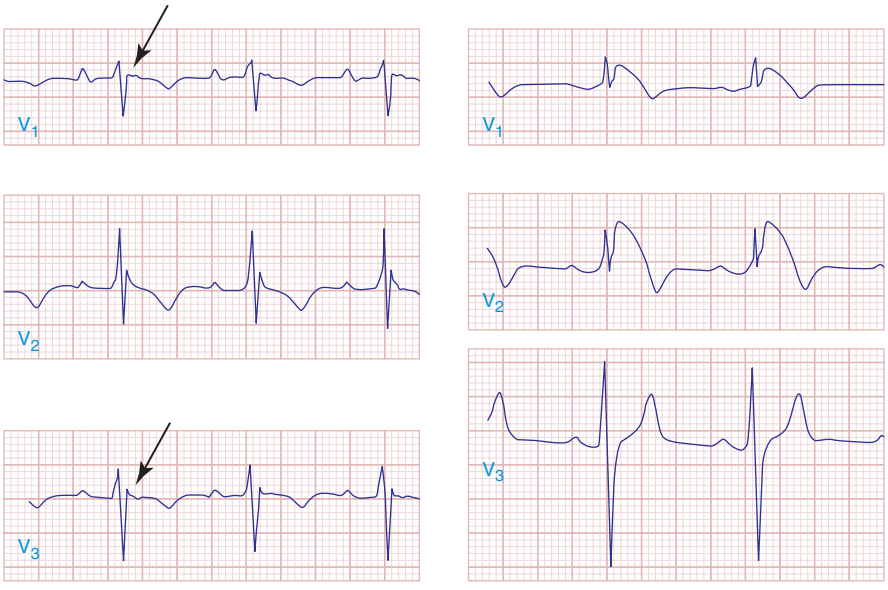

FIGURE 277-6 Precordial chest leads V_1–V_3 showing typical abnormalities of arrhythmogenic right ventricular cardiomyopathy (ARVC) (*A*) and Brugada syndrome (*B*). In ARVC, there is T inversion and delayed ventricular activation manifest as epsilon waves (*arrows*). Panel B shows ST elevation in V_1 and V_2 typical of the Brugada syndrome. *(Figures reproduced from F Marchlinski: The tachyarrhythmias. In Longo DL et al [eds]: Harrison's Principles of Internal Medicine, 18th edition. New York, McGraw-Hill, 2012, pp 1878–1900.)*

PVCS AND NONSUSTAINED VT ASSOCIATED WITH DEPRESSED VENTRICULAR FUNCTION AND HEART FAILURE PVCs and nonsustained VT are common in patients with depressed ventricular function and heart failure and are markers for disease severity and increased mortality, but antiarrhythmic drug therapy to suppress these arrhythmias has not been shown to improve survival. Antiarrhythmic drugs whose major action is blockade of the cardiac sodium channel (flecainide, propafenone, mexiletine, quinidine, and disopyramide) are avoided in patients with structural heart disease because of a risk of proarrhythmia, negative inotropic effects, and increased mortality. Therapy with the potassium channel blockers, e.g., dofetilide, does not reduce mortality. Amiodarone suppresses ventricular ectopy and reduces sudden death but does not improve overall survival. ICDs are the major