TABLE 9-5 DIFFERENTIATION OF VENTRICULAR TACHYCARDIA FROM SUPRAVENTRICULAR TACHYCARDIA WITH ABERRANCY

HELPFUL FEATURES	IMPLICATIONS
Positive QRS concordance	Diagnostic of VT
AV dissociation, capture beats, or fusion beats	Diagnostic of VT
Atypical RBBB (monophasic R, QR, RS, or triphasic QRS in V_1; R:S ratio <1, QS or QR, monophasic R in V_6)	Suggests VT
Atypical LBBB (R >30 min or R to S [nadir or notch] >60 min in V_1 or V_2; R:S ratio <1, QS or QR in V_6)	Suggests VT
Shift of axis from baseline	Suggests VT
History of CAD	Suggests VT
QRS during tachycardia identical to QRS during sinus rhythm	Suggests SVT
Termination with adenosine	Suggests SVT

AV, Atrioventricular; CAD, coronary artery disease; LBBB, left bundle branch block; RBBB, right bundle branch block; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

TABLE 9-6 CAUSES OF SUDDEN CARDIAC DEATH

NONCARDIAC CAUSES	
Central nervous system hemorrhage	Bradyarrhythmias, sick sinus syndrome
Massive pulmonary embolus	Aortic stenosis
Drug overdose	Tetralogy of Fallot
Hypoxia secondary to lung disease	Pericardial tamponade
Aortic dissection or rupture	Cardiac tumors
CARDIAC CAUSES	Complications of infective endocarditis
Ventricular fibrillation	Hypertrophic cardiomyopathy (arrhythmia or obstruction)
Myocardial ischemia or injury	Myocardial ischemia
Long QT syndrome	Atherosclerosis
Short QT syndrome	Prinzmetal angina
Brugada syndrome	Kawasaki arteritis
Arrhythmogenic right ventricular dysplasia	
Ventricular tachycardia	

disturbances. The right ventricular outflow tract (RVOT) is the most common origin of idiopathic VT, which is likely caused by triggered activity. This form of VT (or PVCs) is usually sensitive to catecholamines and may terminate with adenosine (i.e., adenosine-sensitive VT). Another common form of idiopathic VT originates from the left ventricular conduction system (i.e., fascicular VT) and may be verapamil sensitive. Idiopathic VTs are common targets for successful catheter ablation.

Nonsustained VT usually does not require specific therapy unless the patient is symptomatic. The Cardiac Arrhythmia Suppression Trial treated PVCs and NSVT after the acute phase of MI with class I antiarrhythmic drugs, and the trial demonstrated increased mortality rates when the arrhythmias were treated. If VT is attributed to reversible causes such as electrolyte disturbances or acute ischemia, the underlying mechanism should be treated. VT not due to reversible causes may be treated with β-blockers, antiarrhythmic drug therapy (e.g., amiodarone), or catheter ablation. If urgent treatment is required due to hemodynamic instability, direct current cardioversion is performed. It should be synchronized to the QRS complex if a regular morphology exists; otherwise, it should be nonsynchronized. Performing direct current cardioversion during the refractory period (T wave) of MMVT may degrade the rhythm to VF. An ICD often is used in patients who survive VT or VF to quickly treat recurrent episodes. Endocardial and epicardial catheter ablation has become an effective treatment for VT.

Prevention of Sudden Cardiac Death

SCD is defined as death within 1 hour of the onset of symptoms. It may result from a variety of cardiac or noncardiac conditions (Table 9-6). SCD is one of the most common causes of death, with 400,000 events occurring annually in the United States. The most common cause of SCD is VT or VF. Cardiac conditions that increase the risk of SCD include LQTS, hypertrophic cardiomyopathy, Brugada syndrome, arrhythmogenic RV dysplasia, and nonischemic or ischemic cardiomyopathy. The most common cardiac condition that may lead to SCD is acute or distant MI.

The successful treatment of SCD due to VF usually requires rapid access to cardioversion; if treatment is delayed by more than 5 to 10 minutes, permanent brain injury is common. AEDs can reduce the time to defibrillation and improve survival when placed in public areas, although they have been less effective when installed in private residencies, even for patients at risk for SCD.

ICDs used in the treatment of SCD have improved mortality rates. Patients who are at high risk for SCD are often offered an ICD to enable rapid defibrillation before the onset of anoxic brain injury. If a patient survives the first episode of SCD due to documented or presumed VT or VF from nonreversible or unknown causes, he or she is offered an ICD. ICDs are extremely successful in the detection and treatment of VT or VF. They do not always prevent loss of consciousness because it takes 15 to 20 seconds to treat the arrhythmia, and low cardiac output may cause syncope before restoration of normal rhythm, especially if several cardioversions are required.

The earliest ICD trials examined their use in the secondary prevention of SCD (i.e., treating patients who had already survived an episode of cardiac arrest). The largest study was the Antiarrhythmics Versus Implantable Defibrillators (AVID) trial, which randomized patients with a history of poorly tolerated sustained VT or cardiac arrest to empirical amiodarone or ICD implantation. In this trial and several others, ICD therapy was associated with a lower risk of arrhythmic and all-cause death compared with antiarrhythmic therapy.

Several trials have examined the use of ICDs for the primary prevention of SCDs (i.e., treating patients who are at risk for SCD). The first was the Multicenter Automatic Defibrillator Implantation Trial (MADIT), which enrolled patients with a prior MI and an ejection fraction of 35% or less who had frequent ventricular ectopy and inducible VT at electrophysiologic testing. The study demonstrated a substantial mortality reduction with ICD therapy. MADIT-II enrolled patients with a prior MI and an ejection fraction of 30% or less in the chronic phase, without requiring invasive testing. A significant mortality benefit was associated with ICD therapy.

The Sudden Cardiac Death in Heart Failure trial enrolled a broader population consisting of patients with ischemic and nonischemic cardiomyopathy, symptomatic heart failure, and an ejection fraction of 35% or less. A survival benefit was found for patients treated with an ICD compared with conventional therapy or empirical amiodarone therapy. The degree of benefit was similar for patients with ischemic or nonischemic