TABLE 277-3 CAUSES OF QT PROLONGATION AND TORSADE DE POINTES VENTRICULAR TACHYCARDIA

1. Congenital long QT syndromes (see text for details)
   - Long QT syndrome type 1: Reduced repolarizing current I_{Ks} due to mutation in *KCNQ1* gene
   - Long QT syndrome type 2: Reduced repolarizing current I_{Kr} due to mutation in *KCNH2* gene
   - Long QT syndrome type 3: Delayed inactivation of the I_{Na} due to mutations in *SCN5A* gene
   - Others: Several other types of long QT syndromes have been described; long QT types 1, 2, and 3 account for 80–90% of cases
2. Acquired prolongation of QT interval
   - Electrolyte abnormalities
     - Hypokalemia
     - Hypomagnesemia
     - Hypocalcemia
   - Drugs
     - Antiarrhythmic drugs
       - Class IA: Quinidine, disopyramide, procainamide
       - Class III: Sotalol, amiodarone (QT prolongation common but torsade ventricular tachycardia is rare), ibutilide, dofetilide, almokalant
     - Antibiotics
       - Macrolides: Erythromycin, clarithromycin, azithromycin
       - Fluoroquinolones: Levofloxacin, moxifloxacin, gatifloxacin
       - Trimethoprim-sulfamethoxazole
       - Clindamycin
       - Pentamidine
       - Chloroquine
       - Antifungals: Ketoconazole, itraconazole
       - Antivirals: Amantadine
     - Antipsychotics
       - Haloperidol, phenothiazines, thioridazine, trifluoperazine, sertindole, zimelidine, ziprasidone
       - Tricyclic and tetracyclic antidepressants
     - Antihistamines (histamine 1-receptor antagonists)
       - Terfenadine, astemizole, diphenhydramine, hydroxyzine
     - Cholinergic antagonists: Cisapride, organophosphates
     - Citrate (massive blood transfusions)
     - Cocaine
     - Methadone
     - Fluoxetine (in conjunction with other drugs that prolong QT)
   - Cardiac conditions
     - Myocardial ischemia and infarction
     - Myocarditis
     - Marked bradycardia
     - Stress cardiomyopathy
   - Endocrine disorders
     - Hypothyroidism
     - Hyperparathyroidism
     - Pheochromocytoma
     - Hyperaldosteronism
   - Intracranial disorders
     - Subarachnoid hemorrhage
     - Thalamic hematoma
     - Cerebrovascular accident
     - Encephalitis
     - Head injury
   - Nutritional disorders
     - Anorexia nervosa
     - Starvation
     - Liquid protein diets
     - Gastroplasty and ileojejunal bypass
     - Celiac disease

sustained VT, nonsustained VT, or PVCs often provoked by exercise or emotional upset. Repeated bursts of nonsustained VTs, which may occur incessantly, are known as repetitive monomorphic VTs and can cause tachycardia-induced cardiomyopathy with depressed ventricular function that recovers after suppression of the arrhythmia (Fig. 277-2). Most originate in the right ventricular outflow tract, which gives rise to VT that has a left bundle branch block configuration in V_1 and an axis that is directed inferiorly, with tall R waves in leads II, III, and AVF (Fig. 277-2). Idiopathic VT can also arise in the LV outflow tract or in sleeves of myocardium that extend along the aortic root. LV origin is suspected when lead V_1 or V_2 has prominent R waves. Although this typical outflow tract QRS morphology favors idiopathic VT, some cardiomyopathies, notably ARVC, can cause PVCs or VT from this region. Excluding these diseases is an initial focus of evaluation.

LV intrafascicular VT presents with sustained VT that has a right bundle branch block–like configuration. It is often exercise-induced and occurs more often in men than women. The mechanism is reentry in or near the septal ramifications of the LV Purkinje system. This VT can be terminated by intravenous administration of verapamil.

MANAGEMENT OF IDIOPATHIC VT Treatment is required for symptoms or when frequent or incessant arrhythmias depress ventricular function. β-Adrenergic blockers are first-line therapy. Nondihydropyridine calcium channel blockers (diltiazem and verapamil) are sometimes effective. Catheter ablation is warranted for severe symptoms or when beta blockers or calcium channel blockers are not effective or not desired. Efficacy and risks of catheter ablation vary with the specific site of origin of the VT, being most favorable for arrhythmias originating in the right ventricular outflow tract.

LV fascicular VT can be terminated by intravenous administration of verapamil, although chronic therapy with oral verapamil is not always effective. Catheter ablation is recommended if β-adrenergic blockers or calcium channel blockers are ineffective or not desired.

Polymorphic VT Sustained polymorphic VT can be seen with any form of structural heart disease (Table 277-2). However, unlike sustained monomorphic VT, polymorphic VT does not always indicate a structural abnormality or focus of automaticity. Reentry with continually changing reentrant paths, spiral wave reentry, and multiple automatic foci are potential mechanisms (Chap. 278e). Sustained polymorphic VT usually degenerates into VF. Polymorphic VT is typically seen in association with acute MI or myocardial ischemia, ventricular hypertrophy, and a number of genetic mutations that affect cardiac ion channels (Table 277-3).

POLYMORPHIC VT ASSOCIATED WITH ACUTE MI/MYOCARDIAL ISCHEMIA Acute MI or ischemia is a common cause of polymorphic VT and should be the initial consideration in management. Approximately 10% of patients with acute MI develop VT that degenerates to VF, related to reentry through the infarct border zone. The risk is greatest in the first hour of acute MI. Following resuscitation as per the ACLS guidelines, management is as for acute MI (Chap. 295). β-Adrenergic blockers, correction of electrolyte abnormalities, and prompt myocardial reperfusion are required. Repeated episodes of polymorphic VT suggest ongoing myocardial ischemia and warrant assessment of adequacy of myocardial reperfusion. Polymorphic VT and VF that occur within the first 48 h of acute MI are associated with greater in-hospital mortality, but those who survive past hospital discharge are not at increased risk for arrhythmic sudden death. Long-term therapy for postinfarct ventricular arrhythmia is determined by residual LV function, with an ICD being indicated for persistent severe LV dysfunction (LV ejection fraction <0.35).