Arrhythmias

Introduction

Disruptions in cardiac rhythm or rate occur in seven basic patterns: early beats, bigeminal beats, grouped beats, pauses, bradycardia, tachycardia, and chaotic rhythms. This section provides an approach to arrhythmias and discusses the diagnosis and management of specific rhythm disorders.

Approach to the Patient With Bradycardia

Clinical Presentation and Evaluation

Bradycardia (heart rate <50/min) may be asymptomatic or associated with light-headedness, syncope, exertional intolerance, dyspnea, or fatigue. It can be a normal finding or result from disease in the sinus node, atrioventricular (AV) node, or His-Purkinje system or from dysfunction of the autonomic system.

Diagnostic evaluation consists of a thorough history, physical examination, focused laboratory testing (electrolyte levels, thyroid function testing), and resting 12-lead ECG. Severe or unstable conduction abnormalities that require urgent intervention must be identified. The evaluation should also include investigation for extrinsic and reversible causes of bradycardia, including ischemia, myocarditis, endocarditis, hypothyroidism, infectious diseases, neurologic events, electrolyte disturbances, and medication use (especially AV nodal blockers and parasympathomimetics). Echocardiography is frequently performed, and exercise stress testing to assess chronotropic competence as well as ambulatory ECG monitoring may be helpful. Clues from the history and physical examination (e.g., rash suggestive of Lyme disease) may dictate further testing. In patients with nocturnal bradycardia or conduction disturbances, sleep apnea should be considered and, if appropriate, evaluated and treated. Isolated nocturnal bradyarrhythmias are unlikely to require a permanent pacemaker.

Sinus Bradycardia

Sinus bradycardia is defined as the presence of sinus rhythm with a heart rate below 50/min. Sinus bradycardia may be appropriate in trained athletes and during sleep. Inappropriate or pathologic sinus bradycardia is most commonly caused by sinus node dysfunction due to age-related myocardial fibrosis. Less commonly, sinus node dysfunction may result from right coronary ischemia, hypothyroidism, intracranial hypertension, postoperative scarring or fibrosis from cardiothoracic surgery, or infiltrative or inflammatory disorders (e.g., sarcoidosis). The most common extrinsic cause is medication use (β-blockers, donepezil, neostigmine, pyridostigmine).

Atrioventricular Block

AV block may be classified as first degree, second degree, or third degree. First-degree AV block is defined by a delay in AV conduction (PR interval >200 ms). In large cohort studies, first-degree AV block has been associated with an increased risk for atrial fibrillation (AF) and all-cause mortality.

In second-degree AV block, only some P waves conduct to the ventricles. Mobitz type 1 second-degree (Wenckebach) AV block is characterized electrocardiographically by a PR interval that progressively prolongs until a QRS complex is dropped, resulting in grouped beating (**Figure 16**). Mobitz type 2 second-degree AV block is typified by intermittent nonconducted P waves with unchanging PR intervals (**Figure 17**). When 2:1 block is present, the Mobitz type cannot be determined definitively. However, the distinction between types is important—Mobitz type 2 AV block usually occurs below the AV node and has a higher risk for progression to complete heart block. High-degree AV block refers to the presence of more than one successive nonconducted P wave, resulting in several consecutive P waves without QRS complexes.

In third-degree AV block, also termed complete heart block, no P waves conduct to the ventricles. AV dissociation is observed on the ECG (**Figure 18**).

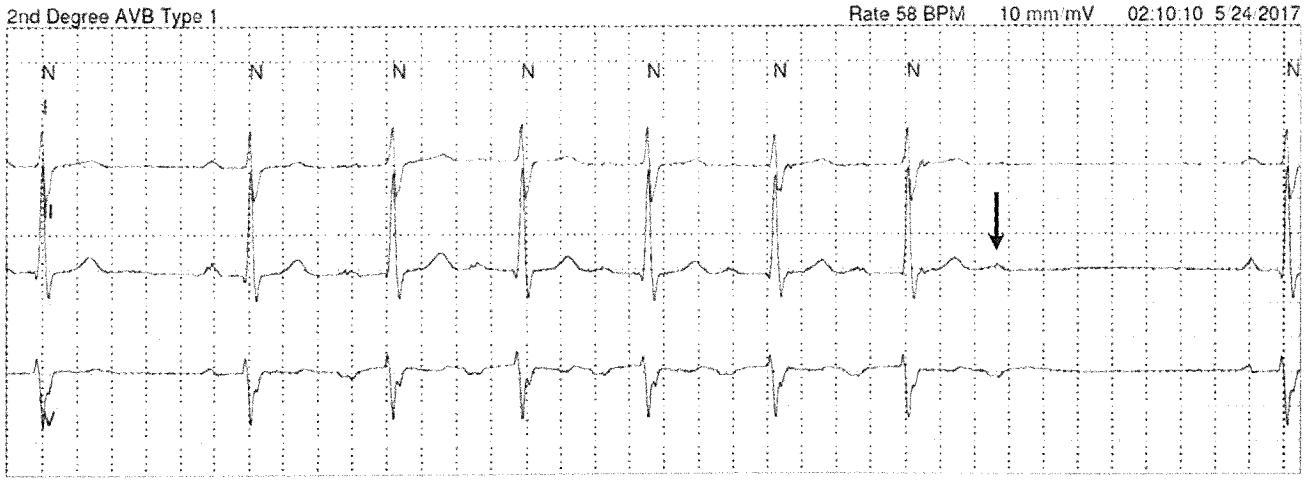

FIGURE 16. ECG showing Mobitz type 1 second-degree atrioventricular block (Wenckebach block), which manifests as a progressive prolongation of the PR interval until there is a dropped ventricular beat (*arrow*).

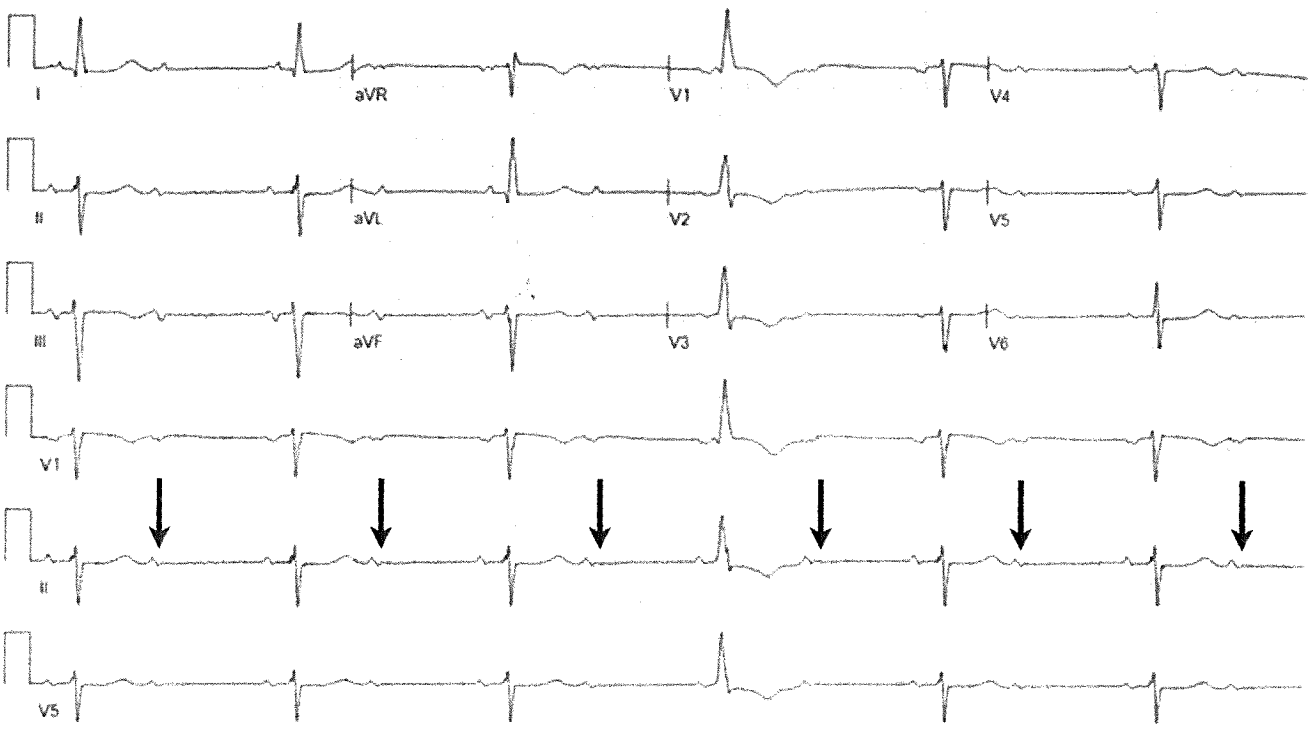

FIGURE 17. ECG showing Mobitz type 2 second-degree atrioventricular block. P waves are blocked intermittently (*arrows*), and the PR interval is fixed. Although 2:1 block can be a manifestation of Mobitz type 1 second-degree atrioventricular block, note the wide QRS complexes, which are more consistent with block below the compact atrioventricular node.

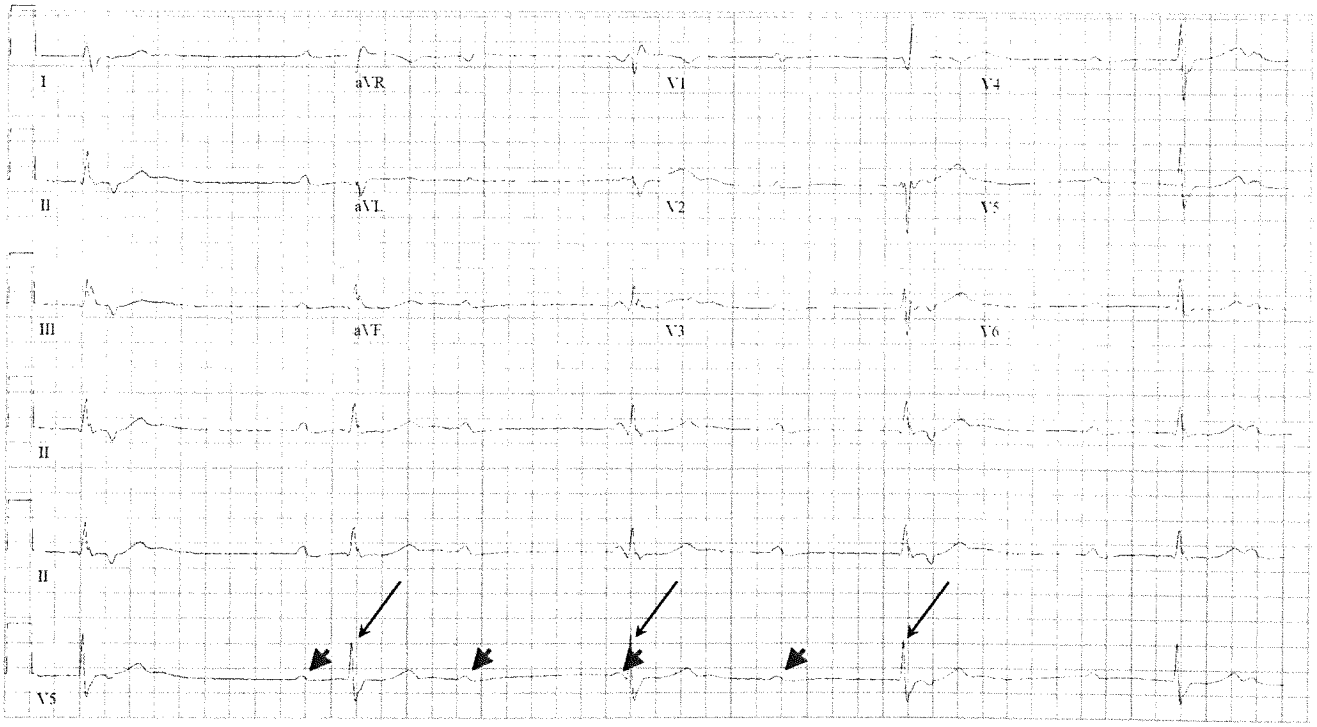

FIGURE 18. In this ECG, the P waves (*short arrows*) and the QRS complexes (*long arrows*) are not associated with each other, indicating the presence of complete heart block.

Treatment

In patients with symptomatic bradycardia and hemodynamic distress, atropine should be administered. If atropine is ineffective, chronotropic drug infusions (e.g., dopamine or epinephrine) can be given until transcutaneous pacing or a temporary pacing wire (preferred) can be implemented. Temporary pacing is indicated for transient conditions causing hemodynamically unstable bradycardia or asystole.

In hemodynamically stable patients, reversible and extrinsic causes of bradycardia should always be addressed before more invasive measures, such as permanent pacing, are considered. Common indications for permanent pacing include:

- Symptomatic bradycardia without reversible cause
- Permanent AF and symptomatic bradycardia
- Alternating bundle branch block (signifies high-risk conduction disease)
- Complete heart block, high-degree AV block, or Mobitz type 2 second-degree AV block, regardless of symptoms

Patients with stable left bundle branch block or right bundle branch block with or without a prolonged PR interval do not require permanent pacing because intraventricular conduction delays have a low risk for progressing to complete heart block (1%-3% per year).

Types of implanted cardiac electronic devices, their functions, and their general indications are reviewed in **Table 16**. **Figure 19** shows a leadless pacemaker in the region of the right ventricle.

KEY POINTS

- Permanent pacing is indicated for symptomatic bradycardia without reversible cause.
- Patients who have atrioventricular (AV) and infranodal conduction disturbances with a high risk for progressing to complete heart block or asystole, such as alternating bundle branch block, high-degree AV block, or Mobitz type 2 second-degree AV block, should receive a permanent pacemaker.

Approach to the Patient With Tachycardia

Clinical Presentation and Evaluation

Patients with tachycardia (heart rate >100/min) may be asymptomatic or experience tachypalpitations, a sensation of skipped beats, light-headedness, dizziness, chest discomfort, dyspnea, exertional intolerance, fatigue, progressive heart failure, near-syncope, or syncope. In asymptomatic patients, tachycardia may be discovered incidentally.

TABLE 16. Cardiac Implantable Electronic Devices for Treatment of Cardiac Rhythm Disorders

			Functions		
Device	**Components**	**Indications**	**Pacemaker Function**	**Antitachycardia Pacing**	**Defibrillation**
Transvenous pacemaker	Pulse generator and intravascular leads (single or dual chamber)	Sinus node dysfunction, AV block, nonreversible symptomatic bradycardia	Yes	No	No
Leadless pacemaker (see Figure 19)	Pulse generator with tines implanted directly into the cardiac chamber; no leads	Atrial fibrillation with bradycardia, paroxysmal bradycardia (e.g., brief sinus node dysfunction or AV block)	Yes (atrial sensing and ventricular pacing)	No	No
Implantable cardioverter-defibrillator	Defibrillator and intravascular leads (single or dual chamber)	Monitoring and treatment of ventricular arrhythmias	Yes	Yes	Yes
Subcutaneous implantable cardioverter-defibrillator	Defibrillator and a single lead that are entirely under the skin (extravascular); no transvenous leads	Monitoring and treatment of ventricular arrhythmias	No	No	Yes
Cardiac resynchronization therapy–pacing (CRT-P)	Pulse generator and intravascular leads, including a pacing lead in the coronary sinus to pace the left ventricle	Restoring electrical synchrony in select patients with symptomatic heart failure	Yes	No	No
Cardiac resynchronization therapy–defibrillator (CRT-D)	Defibrillator and intravascular leads, including a pacing lead in the coronary sinus to pace the left ventricle	Restoring electrical synchrony between the ventricles in patients with heart failure; monitoring and treating ventricular arrhythmias	Yes	Yes	Yes

AV = atrioventricular.

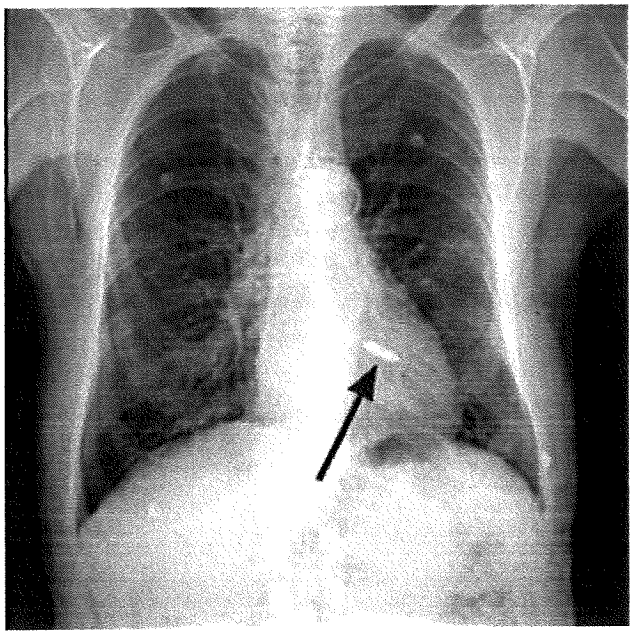

FIGURE 19. Chest radiograph (posteroanterior view) showing a leadless pacemaker in the region of the right ventricle, which could be verified to be retrosternal on a lateral film. Prior sternotomy and significant ascending aortic calcification are also noted.

Documentation of tachycardia on ECG and correlation with symptoms is the key component of the diagnostic evaluation, and a 12-lead ECG should be obtained in all patients with stable tachycardia. A 12-lead ECG recorded during symptoms, although often not possible to obtain, is far superior to most forms of ambulatory monitoring in terms of diagnostic value (see Diagnostic Testing in Cardiology for strategies in selecting an appropriate monitoring device). Based on ECG findings, tachyarrhythmias are traditionally categorized as supraventricular or ventricular. Supraventricular arrhythmias involve conduction through the AV node and are characterized by normal-appearing QRS complexes unless complicated by an aberrant ventricular condition (e.g., bundle branch block). Ventricular arrhythmias originate below the AV node and are characterized by abnormal-appearing and widened QRS complexes.

In addition to a thorough history with medication review and physical examination, thyroid function testing and echocardiography may be considered in select patients with tachycardia.

Antiarrhythmic Drugs

Antiarrhythmic agents have traditionally been organized according to primary mechanism of action using the Vaughan-Williams classification system (**Table 17**), although most antiarrhythmic drugs exert their effects through several mechanisms. Class I and class III agents are the most effective antiarrhythmic drugs; however, due to their membrane-active effects, they carry some paradoxical risk of inducing arrhythmia.

Flecainide and propafenone are the most commonly used class I agents (IC); they are primarily used to treat atrial arrhythmias and usually in conjunction with AV nodal blockers to prevent 1:1 atrial flutter. Toxicity can manifest as QRS widening (**Figure 20** and **Figure 21**). Class IC agents are

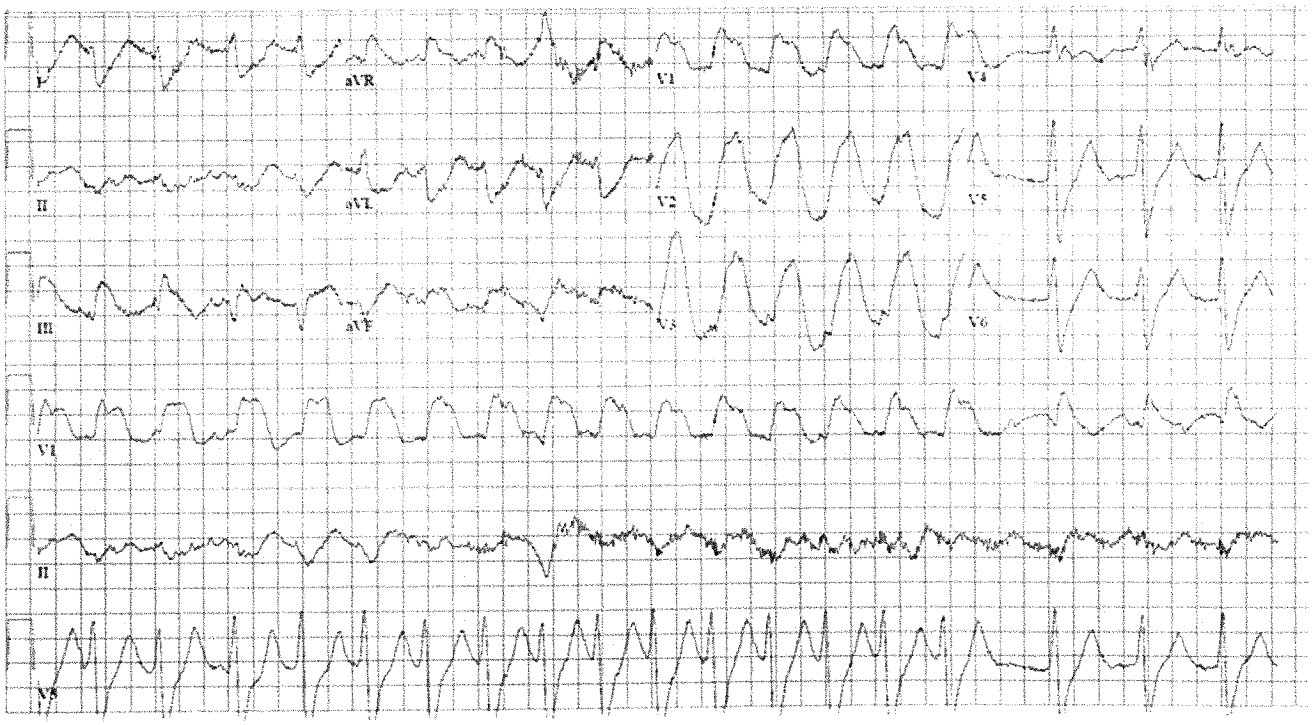

FIGURE 20. ECG in a patient with significant flecainide toxicity, manifesting with severely prolonged QRS duration, with broad and very unusual morphology in the setting of underlying atrial fibrillation or flutter.

TABLE 17. Commonly Used Antiarrhythmic Medications

Classification	Mechanism of Action	Examples	Primary Potential ECG Effects	Use	Side Effects	Contraindications
Class IB	Sodium channel blockade	Lidocaine, mexiletine	QRS widening	Ventricular arrhythmias	Headache, dizziness, or other neurologic symptoms (both drugs) Seizures (lidocaine toxicity)	Advanced liver disease
Class IC	Sodium channel blockade	Flecainide, propafenone	QRS widening	Atrial fibrillation, SVT	Headache, dizziness, or other neurologic symptoms	Ischemic or structural heart disease, sinus node dysfunction, second- or third-degree AV block or bundle branch disease without a pacemaker
Class II	β-Adrenergic blockade	Metoprolol, propranolol, carvedilol, atenolol, bisoprolol, nadolol	Decreased heart rate, prolonged PR interval	Rate control of atrial arrhythmias, SVT	Fatigue, drowsiness, dizziness, hair loss, cold hands and feet, depression, erectile dysfunction, bronchospasm	Severe asthma, cardiogenic shock, second- or third-degree AV block, preexcitation
Class III	Potassium channel blockade	Sotalol, dofetilide	QT prolongation	Atrial fibrillation, atrial flutter, ventricular arrhythmias	Headache, dizziness, bradycardia, fatigue, dyspnea; rarely, torsades de pointes (sotalol) Headache, dizziness, diarrhea; rarely, torsades de pointes (dofetilide)	Renal insufficiency, QT prolongation, bradycardia, or AV block without a pacemaker
Class IV	Calcium channel blockade (nondihydropyridines)	Verapamil, diltiazem	Decreased heart rate, prolonged PR interval	SVT, rate control of atrial arrhythmias	Dizziness, constipation, dependent edema, nausea	Significant sinus node dysfunction, second- or third-degree AV block without a pacemaker, preexcitation
Multichannel blockers	Several mechanisms, including potassium, sodium, and calcium channel blockade	Amiodarone, dronedarone	Many effects: decreased heart rate; prolonged PR, QRS, or QT interval	Atrial arrhythmias (both drugs), ventricular arrhythmias (amiodarone)	Fatigue, dizziness, nausea, vomiting, constipation or diarrhea, tremor, liver and lung toxicities (both drugs) Thyroid and eye toxicities (amiodarone)	Advanced liver, lung, or thyroid disease (amiodarone) Advanced liver disease, permanent atrial fibrillation, recent decompensated or advanced heart failure (NYHA functional class III-IV) (dronedarone)
Adenosine receptor agonists	A_1-receptor agonist	Adenosine	Brief AV block	Termination of SVT (intravenous only)	Flushing, dyspnea, chest pain, hypotension, dizziness, nausea	Severe asthma (can worsen)
Cardiac glycoside	Increases vagal activity	Digoxin	Slows AV node conduction	Rate control of atrial fibrillation	Nausea, vomiting, dizziness, blurry vision and yellow halos, thrombocytopenia, PAT with AV block (toxicity)	Advanced kidney impairment (requires dose adjustment)

AV = atrioventricular; PAT = paroxysmal atrial tachycardia; NYHA = New York Heart Association; SVT = supraventricular tachycardia.

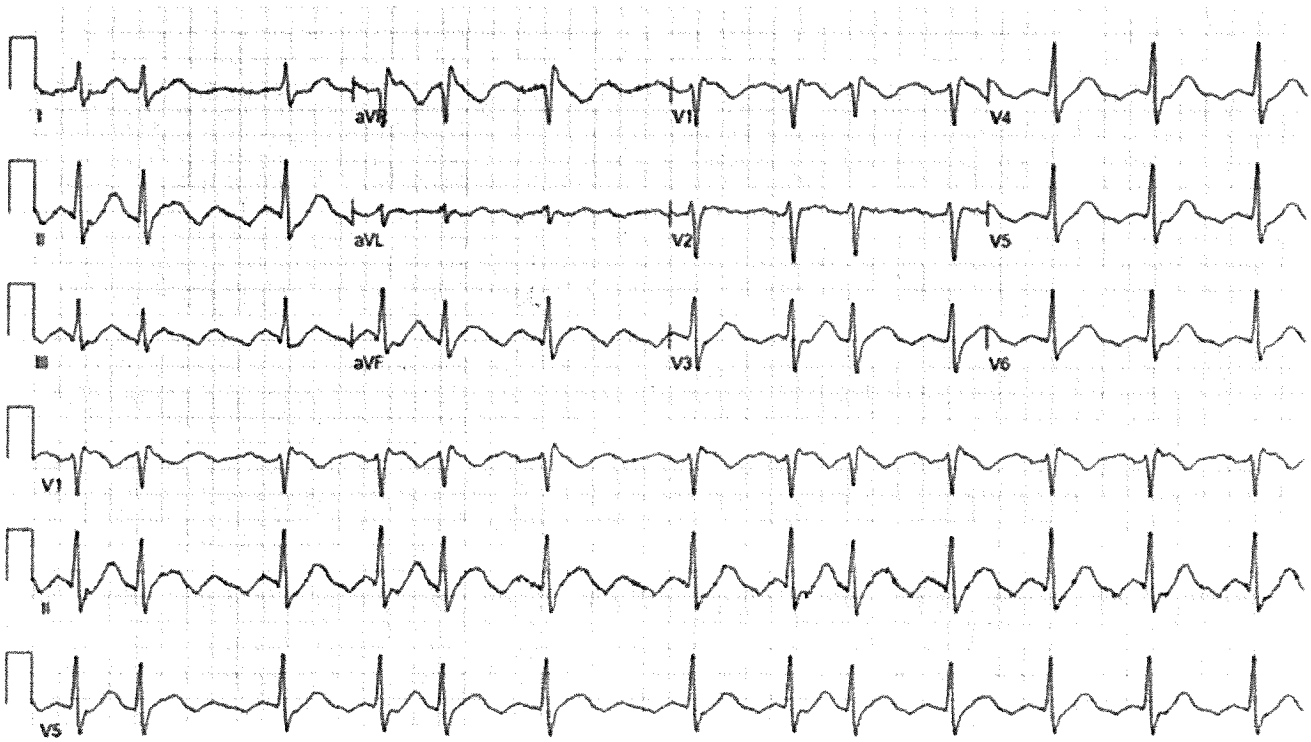

FIGURE 21. ECG in a patient taking unusually high propafenone doses, manifesting with QRS prolongation and broad, relatively slow waves of atrial flutter (best seen in leads II and III).

contraindicated in patients with ischemic or structural heart disease because of the risk for promoting ventricular arrhythmias and death.

Class II agents (β-blockers) and class IV agents (nondihydropyridine calcium channel blockers) are commonly used to inhibit arrhythmia induction and AV conduction (to decrease rate) in patients with supraventricular or atrial arrhythmias.

Class III agents sotalol and dofetilide are used to treat atrial and ventricular arrhythmias. Class III antiarrhythmic therapy typically is initiated in an inpatient setting, with regular assessment of the corrected QT interval (QTc) and caution exercised in patients with kidney disease. Dofetilide is particularly notorious for common and dangerous drug-drug interactions. Amiodarone, a class III multichannel blocker, is frequently used to treat patients with recurrent ventricular tachycardia (VT) or AF. Amiodarone has a low risk of pro-arrhythmia; however, it is associated with thyroid, liver, lung, and eye toxicities as well as neurologic side effects. Thyroid and liver function should be monitored every 6 months, and pulmonary function testing and ophthalmologic examination should be performed annually. Amiodarone interacts with many drugs, including warfarin, statins, and digoxin. Dronedarone, another class III multichannel blocker, can be used in patients with paroxysmal AF and no overt heart failure.

Digoxin and adenosine are excluded from the Vaughan-Williams classification. Digoxin is a positive inotropic agent that also increases vagal activity, leading to a lower resting heart rate. It can be used for rate control in patients with AF. Adenosine is used in the acute treatment of arrhythmias to interrupt AV conduction and terminate supraventricular tachycardia (SVT). Administering adenosine can also help in determining the type of arrhythmia.

Sinus Tachycardia

Sinus tachycardia (sinus rhythm with a heart rate >100/min) is the most common tachycardia and is typically the result of physiologic demand or distress, including exercise, pain, acute illness, hypovolemia, or anxiety. Diagnostic evaluation and treatment are guided by the underlying cause.

Inappropriate sinus tachycardia (IST) is a disorder characterized by an elevated resting heart rate, with exaggerated increases in heart rate with light activity. The sinus rate typically decreases during sleep. IST frequently presents in women in their second to fourth decade. Symptoms vary and can include palpitations, light-headedness, syncope (or near-syncope), dyspnea, and fatigue. Most importantly, the diagnosis of IST is based on the exclusion of secondary causes of tachycardia, such as hyperthyroidism, anemia, pheochromocytoma, and structural heart disease. First-line therapy is removal of aggravating factors and exercise therapy. In patients with bothersome and persistent symptoms, pharmacologic therapy can be considered, but the condition frequently improves over time.

Postural orthostatic tachycardia syndrome (POTS) is another condition that often presents with tachycardia. POTS is a form of dysautonomia characterized by orthostatic intolerance

and excessive tachycardia, particularly with standing. Diagnostic criteria for POTS include an increase in heart rate of 30/min or more or an increase to greater than 120/min within 10 minutes of standing. The diagnosis is often confirmed with tilt-table testing. Behavioral modification, compression stockings, exercise training, and increased fluid intake are important components of therapy. Medical therapy for POTS is highly variable and may include β-blockers, ivabradine (off-label use), fludrocortisone, selective serotonin reuptake inhibitors (off-label use), midodrine, and pyridostigmine (off-label use).

KEY POINT

- Sinus tachycardia is the most common tachycardia and is typically the result of physiologic demand or distress, including exercise, pain, fever, hypovolemia, and anxiety.

Supraventricular Tachycardias

Clinical Presentation

SVTs are rapid heart rhythms that arise from the atrium or require conduction through the AV node. AF and atrial flutter are technically SVTs, although the term is generally reserved for a narrow group of arrhythmias described herein. SVTs can affect all age groups but are frequently seen in younger patients. Prevalence is higher in women than in men. SVTs usually occur in the absence of structural heart disease, although echocardiography should be performed to exclude underlying cardiac dysfunction or structural defects. Patients often have repeated episodes of tachycardia and may report palpitations, a sensation of pounding in the neck, fatigue, light-headedness, chest discomfort, dyspnea, presyncope, and, less commonly, syncope.

The ECG typically demonstrates a narrow-complex tachycardia; however, wide QRS complexes (>120 ms) may be present in cases of bundle branch block, aberrancy, pacing, or anterograde accessory pathway conduction (antidromic tachycardia).

Vagal maneuvers, including the Valsalva maneuver or carotid sinus massage, are first-line therapy to restore sinus rhythm acutely in patients with SVT. Adenosine can be used to terminate SVT and simultaneously help diagnose its mechanism. Tachycardias that terminate with adenosine are typically AV node dependent (atrioventricular nodal reentrant tachycardia [AVNRT] and atrioventricular reciprocating tachycardia [AVRT]), whereas continued atrial activity (P waves) during AV block is consistent with atrial flutter or atrial tachycardia.

Atrioventricular Nodal Reentrant Tachycardia

AVNRT accounts for two thirds of all cases of SVT, not including cases of AF and atrial flutter. It is caused by a reentrant circuit within the AV node that uses both the fast and slow pathways. AVNRT is characterized by a short RP interval with a retrograde P wave inscribed very close to the QRS complex.

AVNRT may be terminated with vagal maneuvers or adenosine. AV nodal blockers (β-blockers or calcium channel blockers) are used to prevent recurrent AVNRT. In patients with recurrent AVNRT and those who do not tolerate or prefer to avoid long-term medical therapy, catheter ablation should be considered. Catheter ablation of AVNRT has a high success rate, although it is associated with a 1% risk for injury to the AV node necessitating pacemaker implantation.

Atrioventricular Reciprocating Tachycardia

AVRT is an accessory pathway–mediated tachycardia that is often observed as preexcitation (delta wave) on ECG (**Figure 22**). Early ventricular activation over the accessory pathway causes

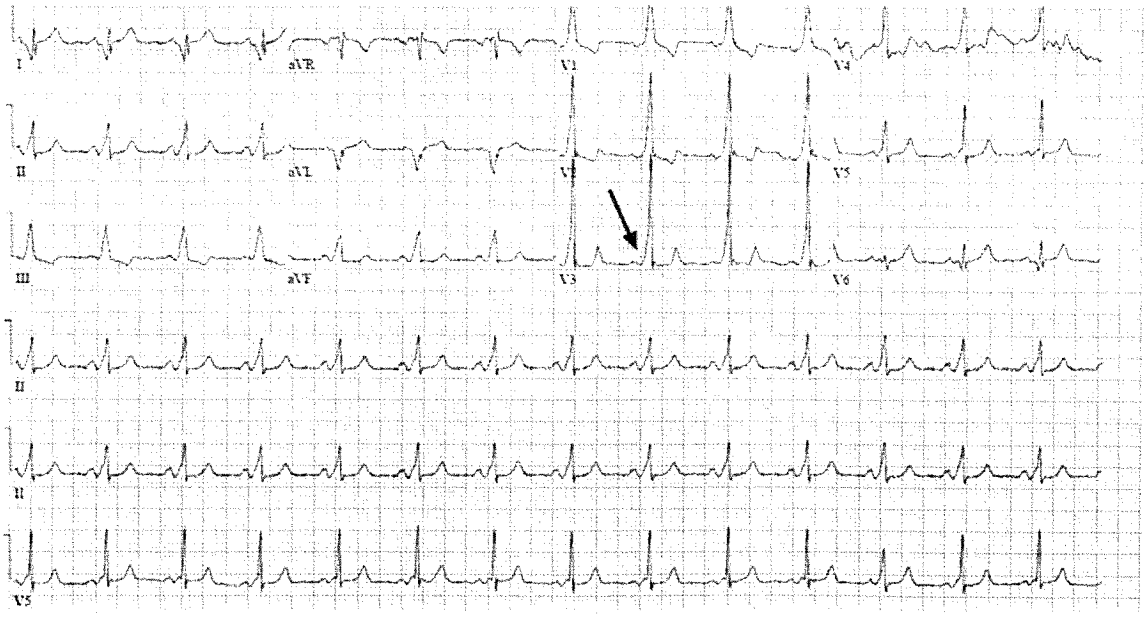

FIGURE 22. ECG demonstrating sinus rhythm with preexcitation as indicated by the presence of a delta wave (*arrow*). The slurring of the QRS upstroke represents premature depolarization of the ventricular tissue adjacent to the accessory pathway.

shortening of the PR interval, and the initial part of the QRS complex is slurred because of premature ventricular depolarization in the myocardial tissue adjacent to the accessory pathway. In AVRT, conduction is anterograde over the AV node (orthodromic, narrow-complex AVRT) in 90% to 95% of cases; conduction is anterograde over the accessory pathway (antidromic, wide-complex AVRT) in the remaining cases.

Wolff-Parkinson-White (WPW) syndrome is defined by symptomatic AVRT with evidence of preexcitation on resting ECG. AF occurs in up to one-third of patients with WPW syndrome. Rapid conduction over an accessory pathway in AF can result in ventricular fibrillation (VF) and sudden cardiac death (SCD), although this occurs in less than 1% of cases of WPW syndrome.

Risk stratification for SCD can be performed with exercise testing, although more frequently, patients are referred for electrophysiology testing for both risk stratification and curative ablation. Catheter ablation is first-line therapy for patients with WPW syndrome. The success rate for ablation is high but is dictated by the location of the accessory pathway. Antiarrhythmic therapy is second-line therapy.

In asymptomatic patients with preexcitation on ECG, management is controversial. Invasive testing is generally not required unless the patient has a high-risk occupation, such as a commercial airline pilot.

Premature Atrial Contractions and Atrial Tachycardia

Premature atrial contractions (PACs) are early isolated beats that arise from the atria. They are exceedingly common, and their frequency increases with age. During ambulatory ECG monitoring, only 1% of persons have no PACs. High PAC burden is associated with increased risk for AF. Symptomatic PACs are typically treated with β-blockers or calcium channel blockers.

Atrial tachycardia can arise in the presence or absence of structural heart disease. β-Blocker or calcium channel blocker therapy is first-line treatment for symptomatic atrial tachycardia. Second-line treatment is catheter ablation or antiarrhythmic drug therapy. Ablation success rates are generally lower in patients with atrial tachycardia than in patients with other SVTs.

Multifocal atrial tachycardia is typified by three or more P-wave morphologies and a heart rate greater than 100/min (**Figure 23**). It is usually seen in patients with severe pulmonary disease.

KEY POINTS

- Vagal maneuvers may restore sinus rhythm in patients with supraventricular tachycardia.
- Patients with recurrent supraventricular tachycardia are treated with atrioventricular nodal blockers (β-blockers or calcium channel blockers) or catheter ablation.
- First-line therapy for patients with Wolff-Parkinson-White syndrome is catheter ablation; antiarrhythmic drugs are second-line therapy.

Atrial Fibrillation

AF is characterized by disorganized atrial activity with an irregularly irregular ventricular response on ECG (**Figure 24**). It is the most common sustained arrhythmia. Lifetime risk for AF is 25% in patients older than 40 years. Incidence is strongly associated with and increases with age, with 10% of persons older than 80 years affected. AF is associated with an increased risk for adverse cardiac events, including a fivefold increased

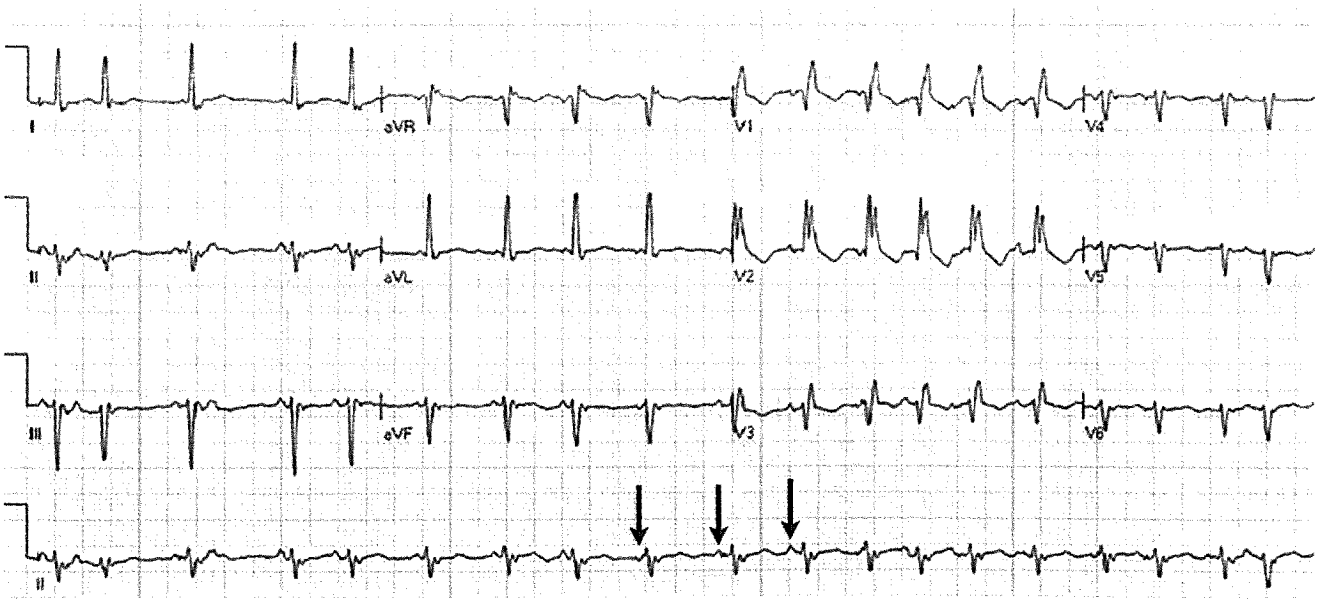

FIGURE 23. ECG showing multifocal atrial tachycardia typified by three or more P-wave morphologies (*arrows*).

anticoagulation is routinely indicated in these patients. Implantable loop recorders to detect subclinical AF may be indicated in selected patients with embolic stroke of undetermined source.

KEY POINTS

- Urgent cardioversion to sinus rhythm is indicated in patients with atrial fibrillation who have hypotension, acute myocardial ischemia, or decompensated heart failure, regardless of atrial fibrillation duration.
- Current guidelines recommend calculation of the CHA_2DS_2-VASc score for stroke risk stratification in patients with nonvalvular atrial fibrillation; patients with a CHA_2DS_2-VASc score of 2 or greater in men or 3 or greater in women should be treated with oral anticoagulation to prevent stroke.
- Direct-acting oral anticoagulants are recommended in preference to warfarin in patients with atrial fibrillation, excluding those with moderate to severe mitral stenosis or a mechanical valve prosthesis.
- In patients with atrial fibrillation, rate plus rhythm control offers superior symptom management compared with rate control alone.

Atrial Flutter

Atrial flutter is an organized macro-reentrant tachycardia with discrete regular atrial activity on ECG, usually with an atrial rate of 250/min to 300/min. Typical atrial flutter is characterized electrocardiographically by a sawtooth pattern with inverted flutter waves in leads II, III, and aVF and positive flutter waves in lead V_1 (**Figure 25**). Typical atrial flutter, the dominant form in patients without prior cardiac disease, is the result of counterclockwise reentry around the tricuspid annulus. Atypical flutter is primarily seen among patients with prior ablation for AF or prior cardiac surgery. In atypical flutter, the circuit is usually in other locations in the right and left atria.

Management of anticoagulation in the setting of chronic atrial flutter is similar to that for AF; however, a rhythm control strategy is favored in atrial flutter because rate control may be difficult and often requires high doses of more than one AV nodal blocker. Catheter ablation is the definitive treatment for typical atrial flutter because of a very high success rate (>95%) and low complication rate. Oral anticoagulation in patients with atrial flutter without ablation is approached in the same manner as in patients with AF.

KEY POINT

- Catheter ablation is the definitive treatment for typical atrial flutter, with a high success rate (>95%) and low risk for complications.

Ventricular Arrhythmias

Premature Ventricular Contractions

Premature ventricular contractions (PVCs) occur in up to 75% of healthy persons. Symptoms include palpitations or the perception of skipped beats. Forceful beats are caused by increased cardiac filling during the pause following the PVC. PVCs are

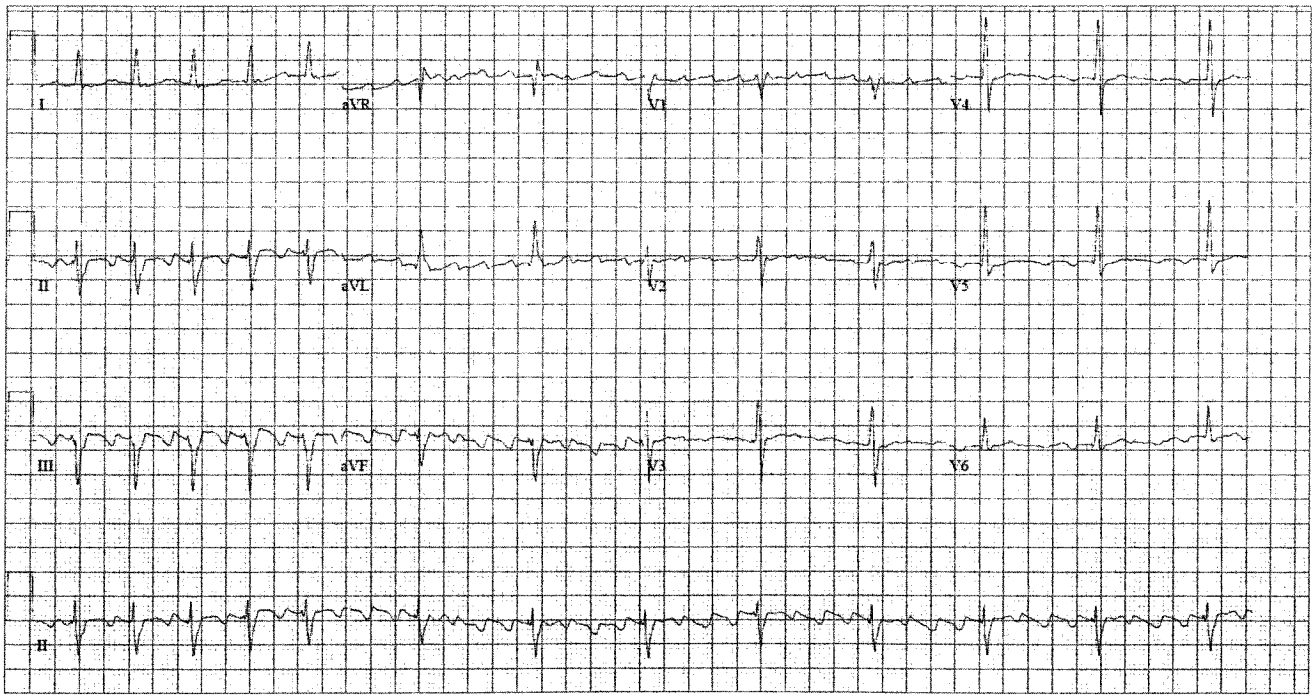

FIGURE 25. In this ECG demonstrating typical atrial flutter, negatively directed sawtooth waves are seen in the inferior leads (II, III, and aVF), and positive waves are seen in lead V_1. In the bottom rhythm strip, 2:1 and 4:1 conduction patterns are most easily seen.

more common in patients with hypertension, LV hypertrophy, previous MI, and other forms of structural heart disease, such as nonischemic cardiomyopathy.

In the absence of high-risk features (syncope, family history of premature SCD, structural heart disease), reassurance is often appropriate, and medical therapy is unnecessary. However, PVCs require treatment when symptoms are bothersome or frequent (>10% of all beats or 10,000 PVCs per day). PVC-induced cardiomyopathy may result from frequent PVCs (generally >10%-15% of beats), although it occurs only in a minority of patients (see Heart Failure).

First-line treatment for PVC suppression is β-blocker or calcium channel blocker therapy. β-Blockers are preferred in patients with ventricular dysfunction. Alternative antiarrhythmic therapy may be used if PVCs persist despite β-blockade or calcium channel blockade. The selection of an antiarrhythmic medication for PVC suppression depends on many factors, including age, kidney function, cardiac structure, and comorbid conditions. Catheter ablation should be considered in patients with continued frequent PVCs despite medical therapy, patients who cannot tolerate medical therapy, and patients who develop PVC-induced cardiomyopathy.

Ventricular Tachycardia

Clinical Presentation

VT is a wide-complex tachycardia (QRS complex ≥120 ms). The differential diagnoses for wide-complex tachycardia include SVT with aberrancy, preexcited tachycardia (antidromic tachycardia), ventricular paced rhythms, and most commonly, VT. In adult patients with known structural heart disease, 95% of wide-complex tachycardias are VT, and additional ECG or clinical criteria are often unnecessary. However, in patients without known structural heart disease, several important clinical and ECG features can distinguish VT from other conditions. Key features of VT on ECG include AV dissociation, fusion beats, and capture beats (**Figure 26**). When the origin of a wide-complex tachycardia is in question, VT should be assumed.

Ventricular arrhythmias most commonly occur in patients with structural heart disease, including both ischemic and nonischemic cardiomyopathy, in whom the presence of abnormal conduction and/or myocardial scar tissue facilitates the development of VT. In these patients, sustained VT (≥30 seconds) can lead to hypotension, syncope, VF, and cardiac arrest; however, on occasion, VT can be well tolerated. Thus, the absence of hemodynamic compromise does not exclude VT as a diagnosis.

VT in the absence of structural heart disease (idiopathic VT) typically arises from the ventricular outflow tracts, fascicles, and papillary muscles. Patients with idiopathic VT usually present with palpitations in the third to fifth decades of life. Episodes of syncope are uncommon. Arrhythmic events are often triggered by stress, emotion, or sleeplessness.

Evaluation and Management

Patients with VT and hemodynamic instability should undergo immediate direct current cardioversion (see Sudden Cardiac Arrest for a discussion of advanced cardiac life support).

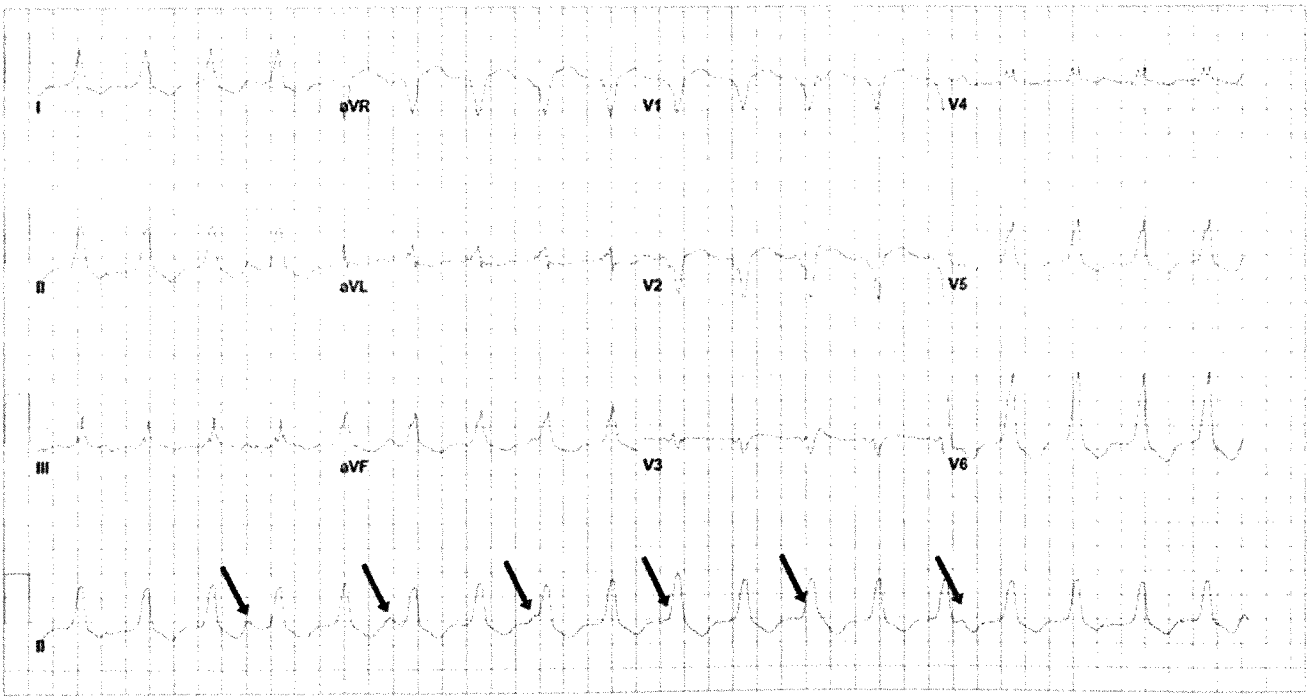

FIGURE 26. ECG demonstrating a regular monomorphic wide-complex tachycardia in a left bundle branch block pattern. The presence of atrioventricular dissociation confirms the diagnosis of ventricular tachycardia. The arrows identify nonconducting P waves.

Intravenous amiodarone should be administered if the VT persists or recurs after cardioversion. Patients with VT or VF with ST-elevation MI should undergo emergency revascularization. In patients with hemodynamically stable VT not in the setting of an acute MI, intravenous procainamide can be useful for VT termination, or intravenous amiodarone or sotalol may be considered.

Evaluation with resting ECG, exercise treadmill testing (to provoke arrhythmias), and cardiac imaging (to identify structural heart disease) is indicated in patients with VT. Cardiac magnetic resonance (CMR) imaging generally allows for tissue characterization, making it an important modality in the evaluation of myocardial diseases that may manifest as VT, including arrhythmogenic right ventricular cardiomyopathy, myocardial fibrosis/scarring, cardiac sarcoidosis, and other infiltrative cardiomyopathies (e.g., amyloidosis). CMR imaging can also clarify the extent and pattern of myocardial scarring.

Patients with ischemic cardiomyopathy who present with VT should be considered for angiography and revascularization, if appropriate, primarily to reduce ischemic burden rather than to treat the arrhythmia. Patients with cardiomyopathy and heart failure should receive guideline-directed medical therapy to minimize risk for ventricular arrhythmia. In patients with recurrent VT despite β-blocker therapy, antiarrhythmic drug therapy or catheter ablation may be considered. Contemporary evidence suggests ablation to be more effective than medical therapy. Implantable cardioverter-defibrillator (ICD) placement is indicated for secondary prevention of SCD in patients with structural heart disease or cardiomyopathy who have sustained VT/VF, provided that clearly reversible causes have been excluded (e.g., acute coronary ischemia, cocaine ingestion).

In patients with idiopathic VT, calcium channel blockers, especially verapamil, and β-blockers are first-line therapy. Catheter ablation can be considered if symptoms continue despite these therapies. ICD placement is generally unnecessary in idiopathic VT because of the benign prognosis and high efficacy of other therapies.

KEY POINTS

HVC
- Premature ventricular contractions (PVCs) without high-risk features (syncope, family history of premature sudden cardiac death, structural heart disease) are managed with reassurance; treatment is reserved for bothersome symptoms or frequent PVCs.
- Idiopathic ventricular tachycardia occurs in patients without structural heart disease and is unlikely to cause syncope or sudden cardiac death; β-blockers and calcium channel blockers are first-line therapy, with catheter ablation reserved for symptoms refractory to drug therapy.
- Ventricular tachycardia in the setting of structural heart disease often requires aggressive rhythm control along with implantable cardioverter-defibrillator implantation for secondary prevention.

Inherited Syndromes Characterized by Sudden Cardiac Death

Patients younger than 40 years without ischemic or structural heart disease who have unexplained cardiac arrest, unexplained near drowning, or recurrent high-risk syncope should be evaluated for inherited arrhythmia and/or cardiomyopathy syndromes. Unexplained premature death (age <35 years) or sudden death (age <40 years) in a first-degree family member also should raise suspicion for an inherited arrhythmia syndrome and prompt referral to a cardiovascular specialist, with genetic counseling and testing as indicated by clinical findings. The diagnosis of inherited arrhythmia syndromes can be complex because of variable penetrance and expressivity of these disorders. Characteristic findings and treatments for some common syndromes are reviewed in **Table 20**.

Genetic long QT syndrome is among the most common inherited arrhythmias, affecting between 1 in 1000 and 1 in 5000 persons (**Figure 27**). However, the presence of a prolonged QTc (>440 ms in men, >460 ms in women) alone is insufficient to diagnose long QT syndrome. QT prolongation can have many causes, most of which are acquired, such as medication use, structural heart disease, and electrolyte abnormalities. Drugs that have been implicated in QT prolongation include antiarrhythmic agents, antibiotics (including some macrolides and fluoroquinolones), antipsychotic drugs, and antidepressants. Given great variability in QTc across populations as well as variable penetrance of and subtypes of long QT syndrome, diagnosis is complex and should be referred to a specialist. A list of drugs categorized by their potential to cause QT prolongation is available at https://crediblemeds.org. Treatment includes β-blockers (first line); avoidance of QT-prolonging drugs; and, for selected patients, ICD implantation, surgical sympathectomy, and exercise restriction.

Brugada syndrome is distinguished by right precordial ECG abnormalities, including ST-segment coving (concave or linear downsloping ST segment) in leads V_1 through V_3 with or without right bundle branch block, VF, and cardiac arrest (**Figure 28**). Brugada syndrome has an increased prevalence in men and persons of Asian descent. Arrhythmic events (including SCD) in patients with Brugada syndrome are more common at night during sleep. Abnormalities on ECG can be intermittent and may be elicited by fever or pharmacologic challenge with sodium channel blockade, such as procainamide infusion.

Hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) can often present as SCD in young persons. Hypertrophic cardiomyopathy and arrhythmic risk stratification are discussed in Myocardial Disease. ARVC/D usually appears between puberty and young adulthood; however, it can also be identified in older age. Patients with ARVC/D typically present with frequent ventricular ectopy and/or monomorphic VT, although in severe cases, patients can present with heart failure. The diagnosis is established by ECG abnormalities, family history,

TABLE 20. Inherited Syndromes Characterized by Sudden Cardiac Death

Disorder	Presenting Symptoms and Characteristic Findings	Potential Treatments[a]
Long QT syndrome	Syncope during sleep, auditory triggers, and/or during exercise (depending on subtype); QTc usually >460 ms; torsades de pointes	β-Blockers, avoidance of QT-prolonging drugs; selected patients: ICD, sympathectomy, exercise restriction
Brugada syndrome	Syncope during sleep, VF, coved ST-segment elevation in early precordial leads (V_1 through V_3)	ICD, avoidance or management of triggers (drugs, fever), catheter ablation
Catecholaminergic polymorphic VT	Syncope, polymorphic or bidirectional VT during exercise or emotional distress	β-Blockers, verapamil, flecainide, ICD, exercise abstinence (uniform)
ARVC/D	Syncope, palpitations, T-wave inversions in leads V_1 through at least V_3, monomorphic VT, frequent PVCs, and abnormal right ventricular size and function on echocardiography or CMR imaging	ICD, β-blockers, antiarrhythmic medications, catheter ablation, exercise abstinence (uniform)
Hypertrophic cardiomyopathy	Syncope, VF during exercise, increased QRS voltage with or without repolarization abnormalities on ECG	ICD, β-blockers, disopyramide, catheter ablation, surgical myectomy

ARVC/D = arrhythmogenic right ventricular cardiomyopathy/dysplasia; CMR = cardiac magnetic resonance; ICD = implantable cardioverter-defibrillator; PVC = premature ventricular contraction; QTc = corrected QT interval; VF = ventricular fibrillation; VT = ventricular tachycardia.

[a]Treatment recommendations for ICD placement in inherited arrhythmia syndromes are guided by risk stratification with criteria that are often disease-specific. Additionally, antiarrhythmic drugs are often required for recurrent ventricular arrhythmias.

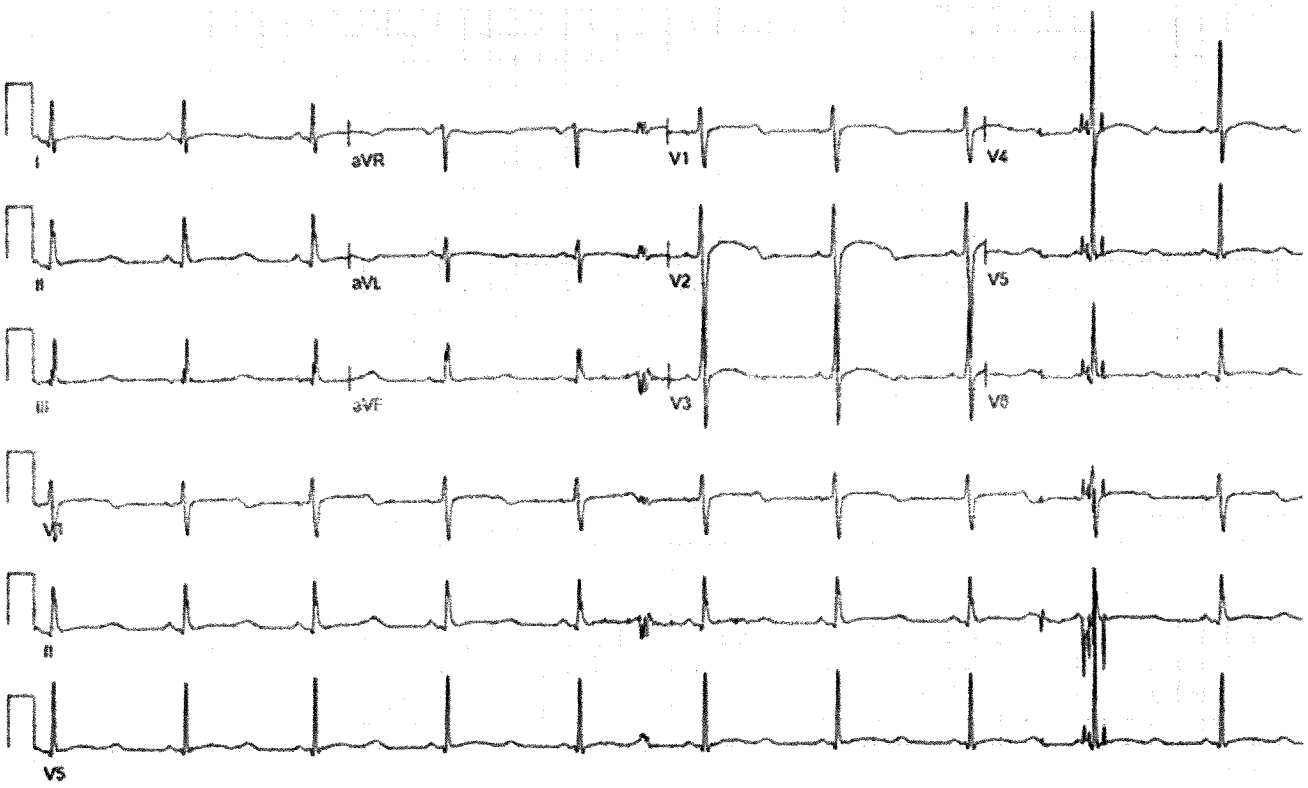

FIGURE 27. ECG demonstrating significant prolongation of the QT interval as well as abnormal morphology, which is best seen in the early precordial leads. Of note, when the heart rate is less than 60/min, the absolute QT interval is routinely used instead of the corrected QT interval (QTc) for heart rate. A QT or QTc greater than 500 ms is a risk factor for adverse events.

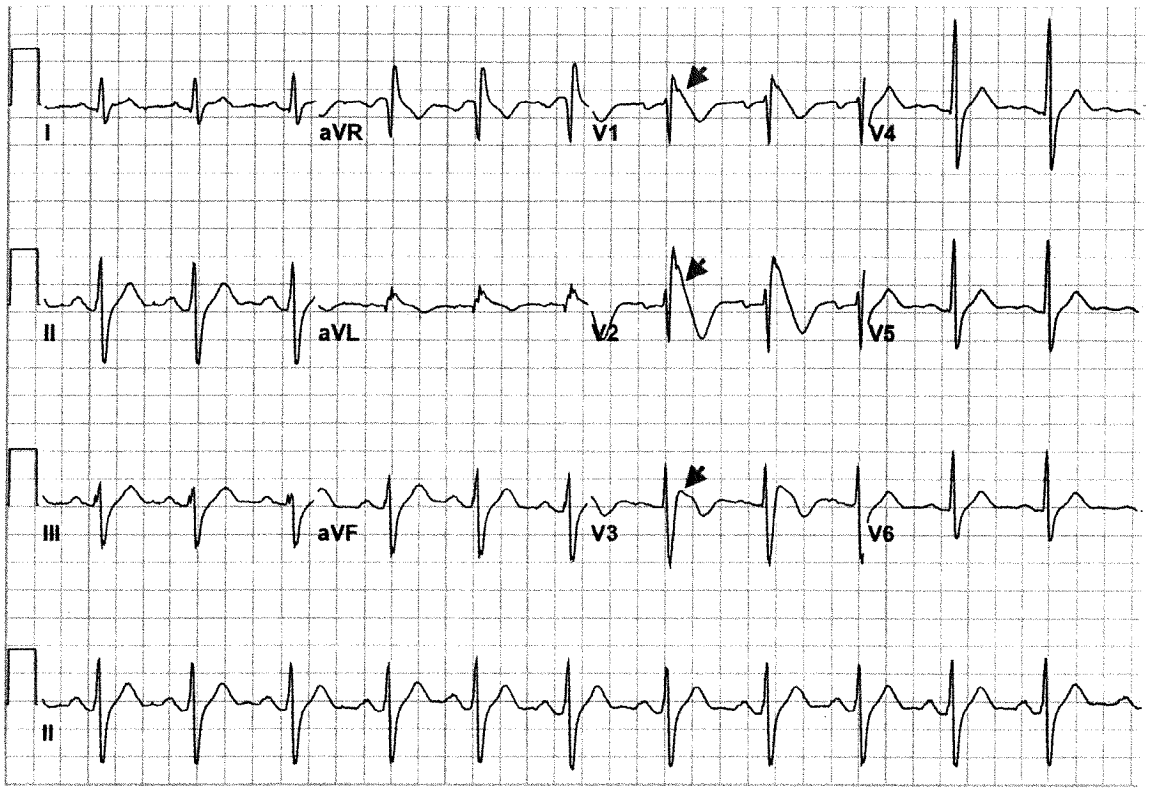

FIGURE 28. ECG demonstrating a type 1 Brugada pattern, ≥2 mm J-point elevation, ST-segment coving (concave or linear downsloping ST segment) (*arrows*), and T-wave inversions in leads V_1 through V_3.

arrhythmias, and structural abnormalities of the right ventricle. CMR imaging can demonstrate enlargement (segments of poorly contracting heart muscle), focal aneurysms, and wall motion abnormalities in the right ventricle (hypokinesis). ARVC/D is usually progressive, and patients should abstain from vigorous exercise. Patients with ARVC/D and cardiac arrest or risk factors (nonsustained VT, inducible VT) should be offered an ICD. β-Blockers are first-line therapy for ventricular arrhythmias, although antiarrhythmic therapy with sotalol or amiodarone or catheter ablation is often required for recurrent VT.

KEY POINT

- Unexplained premature death or sudden death in a first-degree family member should raise suspicion for an inherited arrhythmia syndrome and prompt referral to a cardiovascular specialist.

Sudden Cardiac Arrest

Epidemiology and Risk Factors

SCD is defined as a fatal event or collapse within 1 hour of symptom onset in a person without recent acute illness. In patients in whom death was unwitnessed, SCD is considered to have occurred if the patient was known to be alive and well within the last 24 hours. VT and VF are the most common causes of SCD.

In the United States, more than 350,000 episodes of SCD occur each year. The annual risk for SCD is 1:1000 in the general population. The highest incidence occurs in patients with preexisting structural heart disease, although LV function is normal in most patients who experience SCD. Risk factors include heart failure, diminished LV function, previous MI, unexplained syncope, LV hypertrophy, nonsustained ventricular arrhythmia, chronic kidney disease, and sleep apnea. It is important to distinguish between MI precipitating death and nonischemic SCD when a family history of cardiac disease is obtained.

Acute Management

Cardiac arrest necessitates immediate cardiopulmonary resuscitation (CPR) and advanced cardiac life support (**Figure 29**). Interruptions in chest compressions should be minimized, and defibrillation should occur as soon as possible in patients with a shockable rhythm because time to defibrillation is an important determinant of likelihood of survival to hospital discharge.

The presence or absence of a shockable rhythm guides management after CPR initiation. In patients with out-of-hospital arrest, early cardiac catheterization has value in (1) providing early diagnosis of the etiology of arrest, (2) facilitating early intervention when applicable, and (3) providing opportunities for advanced hemodynamic support (e.g., extracorporeal membrane oxygenation). Any reversible causes, such as tamponade, should be identified and treated.