Chapter

6 Bradyarrhythmias

The normal pacemaker of the heart is the **sinoatrial node**, located in the right atrium. Its impulse conducts through the atria to **the atrioventricular node**, where the electrical impulse is normally slowed (0.12 to 0.20 second). The impulse then travels to the **bundle of His**, which branches into three separate bundles (right, left anterior, left posterior). The sinus node normally generates 60 to 100 impulses per minute. This rate may be increased by sympathetic stimulation or decreased by cholinergic (vagal) stimulation.

Bradyarrhythmias, or abnormally slow heart rates, are generally defined as heart rates of less than 60 beats per minute (bpm). A slow heart rate itself is not necessarily a concern; many professional athletes have heart rates in the 40 bpm range without any difficulty or evidence of disease. In general, bradyarrhythmias arise by one of two mechanisms:

- Impulse formation
- Conduction

ETIOLOGY

Bradyarrhythmias may arise from a specific cardiac disease or as a general response to systemic disease, resulting in a delay in impulse formation or conduction. **Systemic causes** include:

- Hypoxia
- Increased intracranial pressure
- Hypothermia
- Hypothyroidism
- Hyperkalemia

Cardiac diseases associated with bradyarrhythmias are infiltrative heart disease (sarcoid, amyloid), degenerative disease of the cardiac conduction system, ischemic heart disease, Lyme disease, and rheumatic heart disease.

Degenerative disease of the conduction system is seen most often in the elderly. It may be isolated to the conduction system (Lenegre's disease) or represent generalized calcification of the cardiac skeleton that includes aortic and mitral valve involvement (Lev's disease).

Commonly used cardiac drugs slow cardiac conduction and may result in bradyarrhythmias:

- Beta blockers
- Calcium channel blockers
- Digoxin

CLINICAL MANIFESTATIONS

History

The presence of symptoms with an associated bradyarrhythmia often determines the extent of treatment. Symptoms, which may be the result of cardiac or central nervous system hypoperfusion, include:

- Syncope
- Near-syncope or light-headedness
- Dyspnea (due to congestive heart failure)
- Angina pectoris

Nonspecific symptoms, such as fatigue, must not be overinterpreted in the setting of bradycardia.

Physical Examination

Vital signs determine the severity of the bradyarrhythmia and assist in identifying the cause. Hypotension (systolic blood pressure <90) is evidence of hemodynamic instability and requires emergency treatment when associated with symptoms.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a bradyarrhythmia may be classified by the regularity of the heart rate.

Regular Rate
- Sinus bradycardia
- Complete heart block
- 2:1 AV block
- Sinus arrest with escape rhythm
- "Regularized" slow atrial fibrillation

Irregular Rate
- Sick sinus syndrome (sinus node dysfunction)
- Second-degree AV block (type I or II)
- Slow atrial fibrillation

DIAGNOSTIC EVALUATION

An **electrocardiogram** is the first diagnostic test. One should first look for the **presence of P waves**. The absence of a P wave may indicate failure of the SA node to fire (sinus arrest) or failure of the SA node to excite the atria (sinus exit block). These two conditions are indistinguishable by surface electrocardiogram and constitute the **sick sinus syndrome**. The entire PQRST complex is absent (Figure 6-1). If the syndrome is advanced, a regular escape rhythm (junctional or ventricular) may appear. P waves are also completely absent in atrial fibrillation, where no organized atrial activity is present. Uncontrolled atrial fibrillation usually results in an irregular tachycardia with heart rates of 80 to 140 bpm, but intrinsic AV node disease or nodal agents (beta blockers, calcium channel blockers) may slow the rate to 40 to 60 bpm. Therefore, an irregular bradycardia with no P waves is diagnostic of **slow atrial fibrillation**. In **regularized atrial fibrillation**, complete heart block is present at the AV node, and a lower escape pacemaker begins to fire (resulting in a rate of 40 to 60 bpm). This particular rhythm is seen in digoxin toxicity.

If a P wave is identified, the **PR interval** should be measured for each beat. In second-degree AV block, a QRS complex fails to follow a P wave. In **type I (Wenckebach) second-degree block**, the PR intervals progressively lengthen each beat before a P wave fails to conduct (Figure 6-2). This usually represents AV nodal disease. In **type II second-degree block**, the dropped beat occurs suddenly without warning,

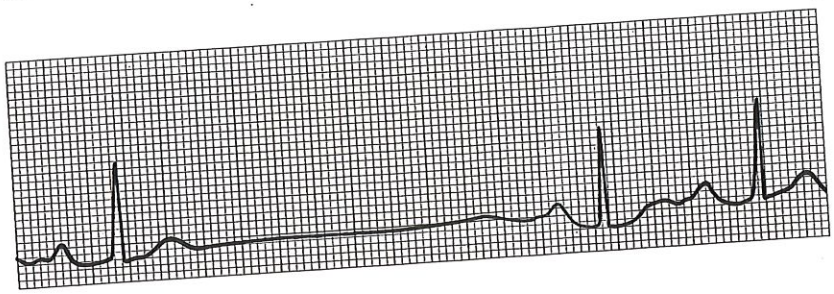

Figure 6-1 • Sick sinus syndrome.

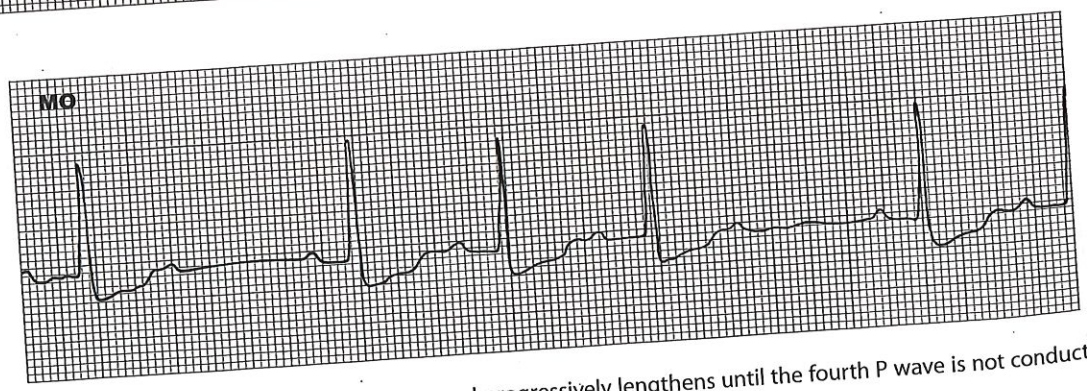

Figure 6-2 • Type I second-degree block. The PR interval progressively lengthens until the fourth P wave is not conducted.

signifying infranodal disease (Figure 6-3). Type II block has a higher risk of progression to complete heart block than type I block. If there is no relationship between the P waves and the QRS complex (variable PR interval), then **complete (third-degree) heart block** is likely to be present (Figure 6-4). P waves, firing at a certain rate, fail to conduct to the ventricles, and a slower pacemaker begins to fire. Both atrial and ventricular rates are regular but are unrelated to each other in complete heart block.

TREATMENT

Treatment of the bradyarrhythmias consists of treating the underlying cause (ischemia, infection, drugs, metabolic causes), if possible, and correcting the rhythm with medication or an extrinsic pacemaker. In treating the underlying cause, patients with evidence of cardiac ischemia should be treated with appropriate medications. Drugs that increase AV nodal block should be discontinued, and a digoxin level should be obtained in patients taking this medication. If hemodynamically stable (normal blood pressure, no congestive heart failure or central nervous system dysfunction), patients may be observed for resolution of the bradyarrhythmia.

Emergent treatment with **atropine** is indicated in the patient with:

- Bradycardia causing hypotension
- Congestive heart failure
- Syncope

Atropine 0.5 mg may be given intravenously or delivered through an endotracheal tube. It is most effective in bradyarrhythmias caused by increased vagal tone.

Cardiac pacemakers are used when a symptomatic bradycardia fails to resolve on its own or in response to atropine. They are also used when progression to complete heart block is likely. Pacemakers may be either temporary or permanent. If the aggravating factor is reversible (e.g., cardiac ischemia, Lyme disease), then a temporary pacemaker may be inserted. Permanent pacemakers are used for patients who have degenerative conduction disease that will not improve. **Transcutaneous pacemakers** have replaced temporary intravenous pacemakers in many instances. Cutaneous electrodes are applied to the chest and back, and the heart is paced by applied current. The transcutaneous pacemaker may be used as a bridge to definitive therapy or as a safety net for patients who are stable but may progress to higher-degree block.

The indications for a transvenous pacemaker are often debated. The more accepted indications for **permanent pacemaker** placement include:

- Complete heart block with symptoms
- Sinus node dysfunction with symptoms
- Bifascicular block with intermittent type II second-degree AV block

Other indications for a pacemaker are situation dependent. For example, type II second-degree AV block in the setting of a **myocardial infarction** generally requires a pacemaker because progression to complete heart block is common. Type I second-

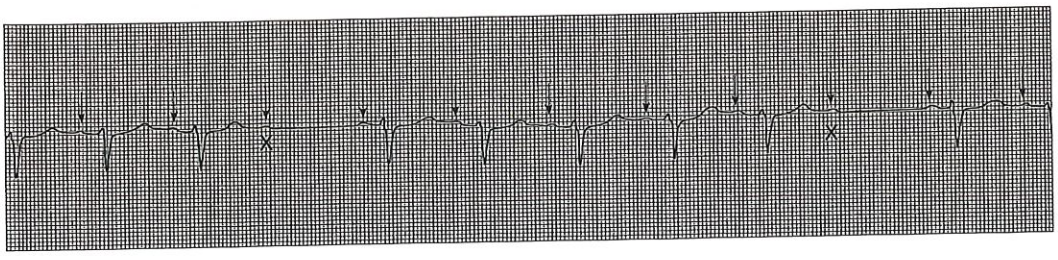

Figure 6-3 • Type II second-degree block. The "X" indicates a P wave suddenly not conducted.

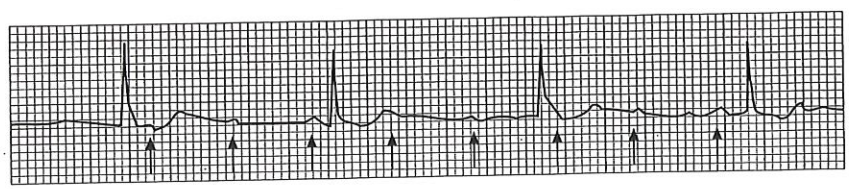

Figure 6-4 • Complete AV block. Arrows indicate regular P waves with no relation to QRS.

degree block (commonly seen in inferior myocardial infarctions) may be observed since it often resolves.

Pacemakers are **not indicated** for asymptomatic type I second-degree AV block or asymptomatic sinus node dysfunction.

Patients with asymptomatic bradyarrhythmias should avoid drugs that may exacerbate the condition. Patients who develop symptoms should be considered for a pacemaker. Some patients may benefit from electrophysiologic studies to determine whether the conduction disturbance is at or below the AV node. These patients include those with 2:1 AV block (Figure 6-5), in whom it is impossible to classify the disturbance as type I or type II. Patients with syncope and bundle branch or bifascicular block also may benefit from electrophysiologic studies.

KEY POINTS

1. A bradyarrhythmia is defined by the heart rate (<60 bpm) and the hemodynamic consequences (symptoms, hypotension).
2. Digoxin toxicity should be considered in patients presenting with atrial fibrillation and a regular ventricular response.
3. Type I second-degree block has progressively increasing PR intervals before a dropped QRS complex. This block often represents underlying AV nodal disease.
4. Pacemakers should not be inserted in patients with asymptomatic sinus node dysfunction.

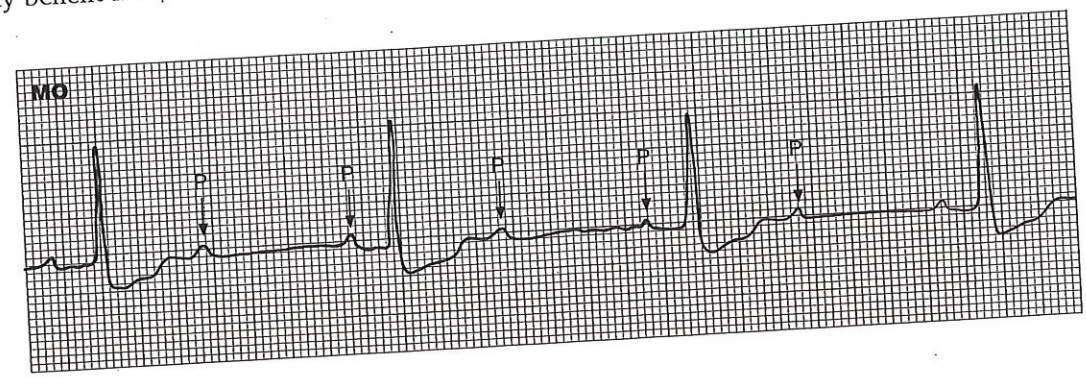

Figure 6-5 • Patients with 2:1 AV block.

Chapter

7 Tachyarrhythmias

Tachyarrhythmias are defined as heart rates exceeding 100 beats per minute (bpm). These rhythm disturbances are best divided into narrow complex tachycardias (QRS duration <0.12 second), which are almost always supraventricular in origin, and wide complex tachycardias, which may be supraventricular or ventricular. This classification helps to determines the best treatment approach to a given arrhythmia.

MECHANISMS OF ARRHYTHMOGENESIS

Tachyarrhythmias occur by one of two mechanisms: abnormal impulse formation (enhanced automaticity or triggered activity) and abnormal impulse propagation (reentry). **Enhanced automaticity** refers to an increased inherent rate of depolarization; that is, certain cardiac cells simply start beating faster. **Triggered activity** relates to electrical oscillations of a cell's membrane potential during or just after repolarization. These oscillations may reach threshold potential and result in premature depolarization.

Reentry is the most common cause of tachyarrhythmias and occurs when two conduction pathways exist (Figure 7-1). One pathway is rapidly conducting but slowly repolarizing (the fast pathway), whereas the other is slowly conducting and rapidly repolarizing (the slow pathway). As a premature impulse enters the loop, it finds the fast pathway refractory and descends down the slow pathway. When it reaches the distal end of the slow pathway, it continues distally and also enters the fast pathway, which is no longer refractory. The impulse travels retrograde up the fast pathway and reenters the loop when it reaches the proximal aspect of the slow pathway. The tachyarrhythmia is perpetuated by recurrent reentry into the circuit. Treatments that interrupt this circuit abruptly terminate the tachycardia.

Tachycardias may arise from the atria, ventricles, or atrioventricular node (junctional). The specific tachycardias are discussed below.

RISK FACTORS

Risk factors for supraventricular tachycardia include:

- Hyperthyroidism (atrial fibrillation, ectopic atrial tachycardia)
- Hypertension (atrial fibrillation and atrial flutter)
- Mitral valve disease (atrial fibrillation)
- Chronic obstructive lung disease (multifocal atrial tachycardia)
- Post cardiac surgery (nonparoxysmal junctional tachycardia)

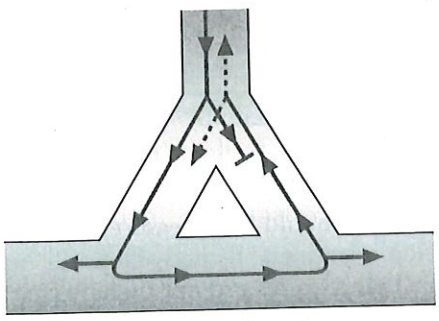

Figure 7-1 • Mechanism of reentry.

TABLE 7-1
Examples of Antiarrhythmics

Type IA	Type IC	Type III
Procainamide	Flecainide	Sotalol
Quinidine	Encainide	Amiodarone
Disopyramide	Propafenone	

Risk factors for ventricular tachycardia (VT) include:

- Prior myocardial infarction (monomorphic VT)
- Ischemia (polymorphic VT, ventricular fibrillation)
- Long QT syndrome (polymorphic VT/torsade de pointes)
- Type IA and III antiarrhythmics (Table 7-1), phenothiazines, tricyclic antidepressants (torsade de pointes)
- Hypomagnesemia, hypo- or hyperkalemia (polymorphic VT, ventricular fibrillation)

Digoxin, especially at toxic levels, can result in several characteristic arrhythmias, including:

- Paroxysmal atrial tachycardia with 2:1 AV block
- Regularized atrial fibrillation
- Bidirectional VT

CLINICAL MANIFESTATIONS

History

The patient with a tachyarrhythmia may be asymptomatic, but frequently presents with:

- Dizziness or syncope
- Palpitations
- Diaphoresis
- Chest pain

These symptoms are not specific for a particular arrhythmia and therefore do not help in distinguishing between rhythms.

Physical Examination

Blood pressure should be measured immediately. **Hypotension** signifies a hemodynamically unstable arrhythmia that requires prompt therapy (see "Treatment"). **Signs of AV dissociation**, such as cannon A waves and variability of the first heart sound, may help identify ventricular tachycardia; AV dissociation is rarely present in supraventricular tachycardias.

DIFFERENTIAL DIAGNOSIS

Tachyarrhythmias should be categorized as supraventricular or ventricular in origin (on the basis of the presence or absence of P waves, and the width and morphology of the QRS on ECG) and as regular or irregular in rhythm (this classification is used in the "Diagnostic Evaluation" section as well).

Regular supraventricular tachycardias include:

- Sinus tachycardia
- Ectopic atrial tachycardia
- Atrial flutter
- AV nodal reentrant tachycardia (AVNRT)
- AV reentrant tachycardias (AVRT; e.g., Wolff-Parkinson-White syndrome)
- Junctional tachycardia

Irregular supraventricular tachycardias include:

- Atrial fibrillation
- Multifocal atrial tachycardia
- Atrial flutter with variable AV block

Ventricular tachyarrhythmias include:

- Ventricular tachycardia
- Ventricular fibrillation
- Torsade de pointes

DIAGNOSTIC EVALUATION

Figure 7-2 presents a general algorithm for diagnosis of tachyarrhythmias.

Electrocardiogram

The ECG is most helpful when obtained during the tachyarrhythmia; however, the baseline ECG may also be of diagnostic utility. For instance, in Wolff-Parkinson-White (WPW) syndrome the baseline ECG in sinus rhythm demonstrates a short PR interval (<0.12 second) and a delta wave, a slurring of the initial deflection of the QRS complex (Figure 7-3). The ECG during tachycardia should be carefully analyzed in regard to the regularity of the rhythm, the width and morphology of the QRS complex, and the presence of P waves. Narrow QRS complex tachy-

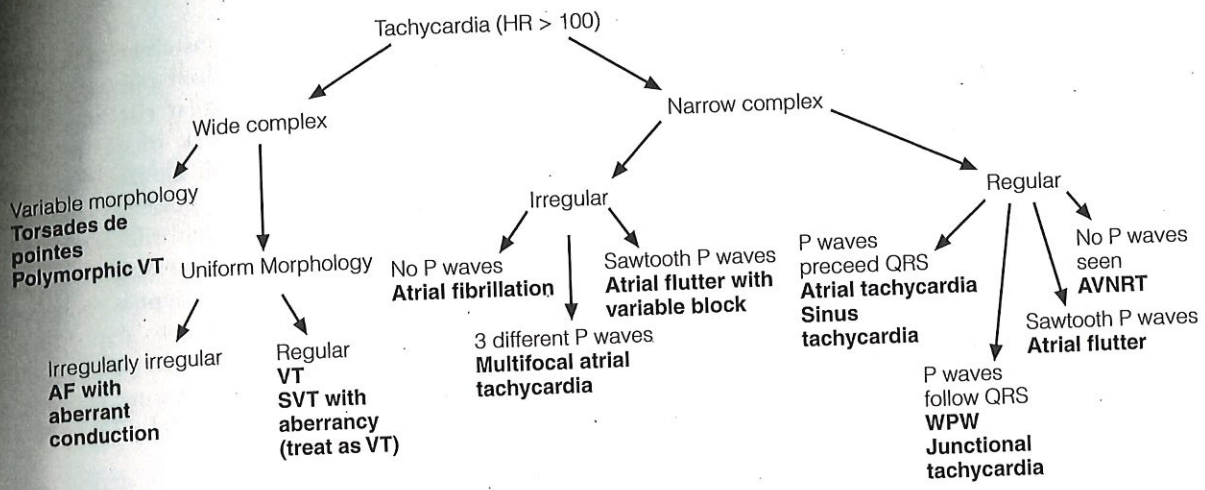

Figure 7-2 • An approach to tachycardia. The diagram shows a general algorithm for diagnosis; exceptions do occur. AF, atrial fibrillation; AVNRT, atrioventricular node reentrant tachycardia; SVT, supraventricular tachycardia; VT, ventricular tachycardia; WPW, Wolff-Parkinson-White syndrome.

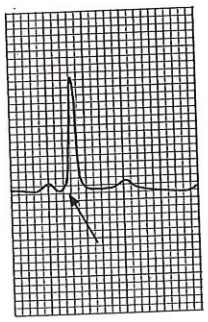

Figure 7-3 • Wolff-Parkinson-White syndrome.

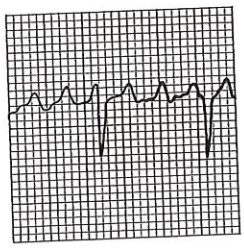

Figure 7-4 • Atrial flutter.

cardias are almost always SVT, whereas wide complex tachycardias may be VT or SVT with aberrancy.

Regular Narrow Complex Tachycardia

Once the tachyarrhythmia is classified as regular narrow complex, evidence of atrial activity (e.g., P waves, flutter waves) should be sought for. P waves that **precede** the QRS complex are seen in sinus tachycardia and atrial tachycardia. P waves **will not be visible** or **will follow** the QRS complex in AVNRT, AVRT, and junctional tachycardia. The flutter waves of atrial flutter are described as "sawtooth," are seen best in the inferior leads (II, III, aVF) and in V_1, occur at a rate of 250 to 350 bpm (Figure 7-4), and conduct to the ventricles most commonly in a 2:1 pattern.

Irregular Narrow Complex Tachycardia

Irregular narrow complex tachycardia also requires identifying atrial activity. Flutter waves, at 300 bpm, may be conducted to the ventricles with variable (2:1, 3:1, or 4:1) AV block, thus resulting in an irregular rhythm. The presence of **P waves of three or more different morphologies** defines multifocal atrial tachycardia. The absence of P waves with an irregular ventricular response is consistent with atrial fibrillation.

Wide Complex Tachycardia

Wide complex tachycardias may be VT or SVT with aberrant conduction. Aberrant conduction during SVT may occur as a result of a baseline conduction abnormality (e.g., bundle branch block), a rate-related conduction abnormality, or conduction through an aberrant pathway (e.g., a bypass tract). Differentiating between VT and SVT with aberrancy can be difficult, but is essential for the selection of appropriate therapy. ECG features that suggest VT include:

- Evidence of AV dissociation (P waves with no relation to QRS complex)
- QRS duration >160 msec
- Shift in QRS axis from baseline ECG
- Atypical bundle branch patterns

In the patient with known heart disease, wide complex tachycardias are most often VT (Figure 7-5). When in doubt, it is best to err on the side of treating for VT. Torsade de pointes (French for "twisting of the points") is a form of polymorphic VT. It has a characteristic appearance on ECG (Figure 7-6).

Other Laboratory Studies

Underlying exacerbating conditions, such as **hypokalemia** or **hypomagnesemia**, should be excluded. A **digoxin level** should be ordered for patients taking this drug, especially when the rhythm is atrial tachycardia with 2:1 block or nonparoxysmal junctional tachycardia. **Thyroid-stimulating hormone** level should be obtained in patients with AF to exclude hyperthyroidism.

TREATMENT: GENERAL PRINCIPLES

Synchronized countershock (cardioversion) is indicated in all patients in whom the tachyarrhythmia is associated with hemodynamic instability (hypotension, congestive heart failure, chest pain, decreased level of consciousness). For regular rhythms (atrial flutter, AVNRT), an initial shock of 50 joules is appropriate. For AF and VT with a pulse, the initial energy level is 100 joules. In pulseless rhythms, **unsynchronized countershock (defibrillation)** is performed starting at 200 joules. The exception to use of defibrillation in the unstable patient is torsade de pointes, in which **external pacing** to override the rhythm is the treatment of choice. In patients who fail initial attempts at cardioversion, sequential shocks with increasing energy (200 J, 300 J, 360 J) should be tried.

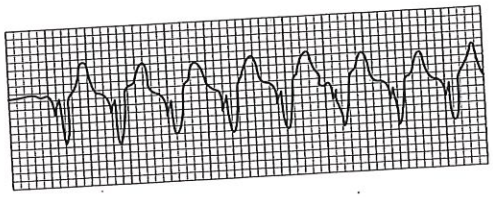

Figure 7-5 • Ventricular tachycardia.

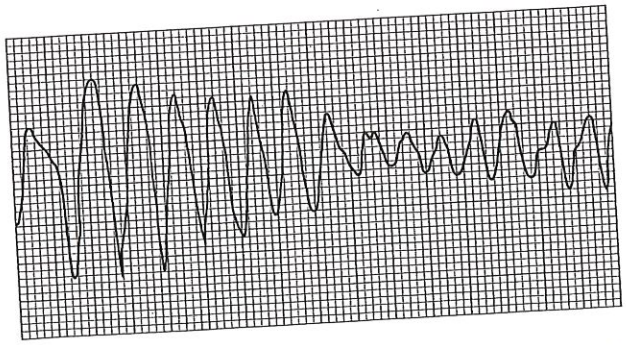

Figure 7-6 • Torsade de pointes.

In patients with hemodynamically stable SVT, maneuvers or medications that block the AV node are the treatments of choice. These include:

- Vagal maneuvers (Valsalva, carotid sinus massage)
- Adenosine
- Beta-blocking agents (e.g., metoprolol, atenolol)
- Calcium channel antagonists (e.g., verapamil, diltiazem)
- Digoxin

For rhythms that are independent of the AV node (e.g., AF, atrial flutter, atrial tachycardia), these therapies will slow or block conduction through the AV node, thereby decreasing the ventricular response to the SVT and unmasking the atrial activity that may have previously been obscured by the rapid ventricular rate. For rhythms that are dependent on the AV node (AVNRT, AVRT), these therapies may terminate the arrhythmia. It is generally recommended to try vagal maneuvers before pharmacotherapy if the patient is stable. Pharmacotherapy is initially given intravenously to achieve rapid control of the heart rate associated with SVT. Adenosine is often used as a diagnostic (and therapeutic) agent for narrow complex tachycardias owing to its marked effect on the AV node and its short duration of action ($t_{1/2} \cong 6$ sec). Digoxin is less useful in the acute setting because of its delayed onset of action. Beta blockers may be best in situations in which adrenergic drive is contributing to the arrhythmia.

For patients with hemodynamically stable VT, lidocaine is the drug of choice and is initially given as a bolus (1 mg/kg), followed by an infusion of 1–4 mg/min. Side effects include confusion and seizures. Intravenous amiodarone is the drug of choice for hemodynamically unstable VT (used in

conjunction with defibrillation) and can be considered in patients with stable VT.

TREATMENT: SPECIFIC ARRHYTHMIAS

Sinus tachycardia, multifocal atrial tachycardia, and junctional tachycardia are managed over the long term by correcting the underlying disease. Conditions that may require drug or interventional treatments are discussed in this section.

Atrial Fibrillation

The three main treatment concerns with AF are:

- Rate control
- Rhythm control
- Prevention of embolic events (i.e., CVA)

As noted earlier, the ventricular response to AF may be controlled with beta blockers, calcium channel antagonists, or digoxin. Atrial fibrillation predisposes to intra-atrial thrombus formation and subsequent thromboembolism. This accounts for the 5% to 6% per year risk of CVA associated with AF, irrespective of whether it is paroxysmal or chronic AF. For this reason, anticoagulation with warfarin should be considered for almost all patients with atrial fibrillation (patients <60 years old with no other medical problems may be treated with aspirin). An international normalized ratio of 2 to 3 should be the goal of treatment.

Atrial fibrillation may be converted to sinus rhythm by electrical cardioversion or by type IA or type III antiarrhythmic agents (see Table 7-1). Patients should be anticoagulated at the time of cardioversion and for at least 3 weeks after. Unfortunately, AF is a recurrent problem in many patients, although continued treatment with antiarrhythmic agents increases the likelihood of maintaining sinus rhythm (amiodarone is the most effective agent in this regard). Increased left atrial size (>50 mm) and long duration of atrial fibrillation (>6 months) are predictors of relapse.

Atrial Flutter

Atrial flutter is treated similarly to atrial fibrillation: rate control, rhythm control, and anticoagulation.

Supraventricular Reentrant Tachycardias

Patients with AVNRT or AVRT may be treated with AV nodal blocking agents, both acutely during the arrhythmia and to prevent recurrences. Catheter-based radiofrequency ablation of one arm of the reentrant loop offers definitive treatment, with a success rate of approximately 90%. This option may be best for young patients with recurrent symptoms and avoids lifelong drug therapy. Complications, such as procedure-related AV block or thromboembolism, may occur in rare instances.

Specific note should be made of AVRT. This arrhythmia is a reentrant rhythm that uses the AV node as one limb of the reentrant loop and an accessory pathway between the atria and ventricle as the other. The most common form of AVRT is WPW syndrome. In sinus rhythm, atrial activity may reach the ventricles either through the AV node or via the accessory pathway. This may be evident on ECG by the presence of a delta wave (see Figure 7-3), representing ventricular preexcitation. Patients with WPW are prone to developing both AVRT and atrial fibrillation. The acute treatment of AVRT involves AV nodal blockade; however, the acute treatment of AF in patients with WPW should not include AV blockade as this will preferentially shunt the atrial activity down the accessory pathway at very rapid rates. The appropriate treatment is procainamide or electrical cardioversion.

Ventricular Tachycardia

Ischemic heart disease often is the underlying cause of VT and should be appropriately evaluated and treated. Asymptomatic nonsustained VT (NSVT) in patients with preserved LV systolic function is usually treated with beta-blocking agents. Patients with more malignant ventricular arrhythmias (symptomatic NSVT, sustained VT, VF) have a high risk of subsequent sudden cardiac death and require more aggressive therapy. In general, antiarrhythmic therapy is inadequate in this setting, and placement of an implantable cardioverter-defibrillator (ICD) is often required. In patients who are felt to be at intermediate risk, electrophysiological studies may help to guide therapy.

Torsade de Pointes

As noted earlier, torsade de pointes is a polymorphic ventricular tachycardia and is almost always associated with a prolonged QT interval. **Causes of prolonged QT interval** are:

- Medications (type IA and III antiarrhythmics [see Table 7-1], phenothiazines, tricyclic antidepressants)
- Electrolyte disorders (hypokalemia, hypomagnesemia, hypocalcemia)
- Congenital long QT syndrome
- Ischemia

This rhythm tends to be self limited but may be associated with hemodynamic instability if prolonged, and may progress to ventricular fibrillation. Torsade de pointes is unique among the VTs in that acute treatment is with intravenous magnesium and overdrive pacing. Phenytoin is the antiarrhythmic of choice in this setting. Removal of the inciting agent is essential.

KEY POINTS

1. Digitalis toxicity can lead to paroxysmal atrial tachycardia with 2:1 block or nonparoxysmal junctional tachycardia.
2. The WPW syndrome is characterized by a short PR interval, delta wave, and AVRT.
3. AVNRT and AVRT should be treated initially with AV nodal blocking agents. Radiofrequency ablation should be considered for long-term management.
4. Torsade de pointes is a multifocal ventricular tachycardia, associated with a long QT interval. Treatment is overdrive pacing and magnesium.
5. Adenosine is the drug of choice to treat regular narrow complex tachycardias after vagal maneuvers have been unsuccessful.
6. Treatment of atrial fibrillation and atrial flutter involves rate control, rhythm control, and prevention of thromboembolism.