The term dysrhythmia denotes any abnormality in cardiac rhythm. In this chapter, we review dysrhythmias outside the context of cardiac arrest specific toxidromes. We then review the physiology of normal and abnormal cardiac impulse formation and conduction. Finally, we discuss the diagnostic tools for evaluating the patient with a known or suspected dysrhythmia, including the history, physical examination, and surface electrocardiogram (ECG). For each rhythm disturbance, we identify the treatment options for prehospital and emergency department (ED) management.

**CARDIAC CELLULAR ELECTROPHYSIOLOGY**

The function of individual cells in the conductive and contractile tissues of the heart depends on an intact resting membrane potential. Na⁺, K⁺, and Ca²⁺ ions create the membrane potential and regulate conduction and contractility. The membrane potential is the result of a differential concentration of Na⁺ and K⁺ on either side of the cell membrane, measuring approximately −90 mV in normal resting, nonpacemaker cells. This electrical gradient exists mainly because of the Na⁺-K⁺ exchange pump and the natural concentration-dependent flow of K⁺ out of the cell. Adenosine triphosphate (ATP) fuels Na⁺ transport out to the extracellular fluid, with Mg²⁺ used as a cofactor (Fig. 79-1). This process creates an osmotic gradient, allowing Ca²⁺ to be passively exchanged for Na²⁺ and promoting conduction as well as myofibril contraction. The resting membrane potential is generated from the flow of K⁺ down a concentration gradient toward the extracellular fluid. The cell membrane is far more permeable to potassium than sodium ions, resulting in a net loss of intracellular positive charge.

In normal nonpacemaker cells, the application of an electrical stimulus causes the membrane potential to become less negative, termed depolarization. When the membrane potential reaches −70 mV, specialized Na⁺ channels open, causing a rapid influx of positive charge into the cell. This “fast” channel activity further decreases the membrane potential and is augmented at 30 to 40 mV by a second “slow” channel that allows Ca²⁺ influx. When these channels close, resting potential is restored by the sodium-potassium pump, an event known as repolarization (Fig. 79-2).

In nonpacemaker cells, additional depolarization from a second electrical stimulus is not possible when the membrane potential is more positive than −60 mV. This period is termed the effective refractory period (Fig. 79-3). At a membrane potential of −60 to −70 mV, a strong impulse can cause a response that is likely to be propagated, although abnormally; this response represents the relative refractory period. At a membrane potential of −70 mV or less, virtually all fast channels are ready for activity if properly stimulated (see Fig. 79-3).

Pacemaker cells differ from non–impulse-generating cells in two ways: their resting membrane potential is less negative, and they can spontaneously depolarize via slow Na⁺ influx. Pacemaker cells exist normally within the sinoatrial (SA) and atrioventricular (AV) nodes and can also be found on the atrial surfaces of the AV valves and within the His-Purkinje system. Nonpacemaker cells may undergo spontaneous depolarization under pathologic conditions, especially during ischemia.

Afterdepolarizations are fluctuations in membrane potential that occur as the resting potential is restored. These fluctuations may precipitate another depolarization, either just before full resting potential is reached (early afterdepolarizations) or after full resting potential is reached (delayed afterdepolarizations). Early afterdepolarizations are associated with high resting membrane potentials and are more likely with slower heart rates. Delayed afterdepolarizations can arise from ischemia, catecholamine excess, or electrolyte disturbances (especially K⁺, Mg²⁺, and Ca²⁺) and are enhanced by faster heart rates.

**ANATOMY AND CONDUCTION**

The SA node is an area of specialized impulse-generating tissue located at the junction of the right atrium and the superior vena cava. Its blood supply is from the right coronary artery (RCA) in 55% of patients and the left circumflex artery (LCA) in 45%. The normal SA node produces spontaneous depolarizations at a faster rate than other pacemakers and functions as the dominant pacemaker. The SA node maintains a rate of 60 to 90 beats/min in most adults. Hypothermia and vagal stimulation slow the sinus rate, whereas hyperthermia and sympathetic stimulation increase the rate. In the setting of low or absent parasympathetic tone—for example, with certain drugs or after heart transplantation—the resting sinus rate tends to be faster.

In the absence of normal SA node activity, other myocardial tissues may assume the role of pacemaker. The AV node has an intrinsic impulse-generating rate of 45 to 60 beats/min. Infranodal pacemakers, found within the His bundle, the Purkinje system, and the bundle branches, maintain intrinsic rates ranging from 30 to 45 beats/min. Other atrial and ventricular tissues may assume pacemaker-like activity under conditions of ischemia or metabolic derangement. Rates of ectopic pacemaker activity vary widely depending on the underlying pathology.

Figure 79-4 correlates the surface ECG tracings with tissue electrical events. Impulses generated from within the SA node itself, imperceptible on the surface ECG, are propagated through the
atrial tissue to the AV node. Atrial depolarization is characterized by the P wave on the surface ECG.

The AV node is an area of specialized tissue between the atria and the ventricles of the heart, located in the posterior-inferior region of the interatrial septum. Its blood supply is from a branch of the RCA in 90% of patients (right dominant) and from the LCA in the remaining 10% (left dominant). Transmission of impulses within the AV node is slower than in parts of the conducting system (Table 79-1) because of a dependence on slow-channel ion influx for membrane depolarization. In some patients, pathologic “accessory pathways” connect atrial and ventricular tissues. These accessory pathways do not share the normal conduction delay of the AV node and may allow for rapid ventricular rates. Preexcitation refers to the early depolarization of ventricular myocardium when accessory pathways are used instead of the normal conduction system.

On the surface ECG, the PR interval (normally 0.10 to 0.20 second) represents the time it takes for conduction of a sinus impulse through the atria and AV node and into the ventricles. Impulses originating in lower atrial tissues are associated with a shortened PR interval, as are impulses conducted to the ventricles via accessory pathways outside the AV node. PR prolongation is usually a result of nodal or supranodal conduction system disease.

After passing through the AV node, impulses propagate down the His bundle to the three main bundle branch fascicles: the right bundle branch (RBB), the left anterior-superior bundle (LASB), and the left posterior-inferior bundle (LPIB). The RBB and LASB are typically supplied by the left anterior descending (LAD) artery, whereas the LPIB may be supplied by either the RCA or the LCA. After conduction down the three main bundle branches, impulses are delivered to the Purkinje fibers, which propagate impulses to myocardial tissues in a swift and orderly fashion, allowing for coordinated ventricular contraction. If an impulse arrives prematurely, it may be conducted aberrantly (if bundles are relatively refractory), or it may be blocked (if the bundles are completely refractory).

On the surface ECG, the QRS complex represents ventricular depolarization, and the normal QRS interval is 0.09 seconds or less, with a duration of 0.12 seconds or greater being clearly abnormal. The T wave corresponds to ventricular repolarization and varies in duration depending, among other things, on the length of the cardiac cycle. The total time of ventricular depolarization and repolarization is represented by the QT interval. Shorter cycle lengths (i.e., shorter R-R intervals) result in a shorter repolarization time. Conversely, longer cardiac cycles are associated with longer repolarization times.

### MECHANISMS OF DYSRHYTHMIA FORMATION

There are three electrophysiologic mechanisms: enhanced automaticity, triggered activity, and reentry. Enhanced automaticity includes spontaneous depolarization of nonpacemaker cells or a lower threshold for depolarization in normal pacemaker cells (Fig. 79-5). Both types of enhanced automaticity can occur in the setting of ischemia, electrolyte disturbances, or drugs. Examples of enhanced automaticity include idioventricular rhythms in the setting of acute myocardial infarction, and atrial tachycardias or junctional tachycardias (JTs) seen with digitalis toxicity.

Triggered activity occurs as the result of early or delayed afterdepolarizations. The classic dysrhythmia associated with early afterdepolarization is torsades de pointes. Triggered dysrhythmias caused by delayed afterdepolarizations are frequently found in the setting of intracellular Ca\(^{2+}\) overload as occurs with digitalis toxicity or with reperfusion therapy for acute myocardial infarction. Many ectopic atrial, junctional, and ventricular rhythms fall into this category and are amenable to treatment with medications that slow the heart rate or interfere with calcium entry into the cell.

Reentry dysrhythmias occur as a consequence of abnormal conduction (Fig. 79-6). For a reentry mechanism, two alternate pathways for conduction must be present and one path must have a longer refractory period. The unequal responsiveness of the limbs creates a functional unidirectional block such that when the impulse exits one limb, it may then reenter the other in retrograde fashion. The cycle is then repeated, creating a self-sustaining or “circus movement” tachycardia that can appear orderly or disorderly (i.e., fibrillatory).

Reentry mechanisms are responsible for most narrow-complex tachycardias and many ventricular tachycardias (VTs). Treatment is predicated on altering conduction in one or both limbs of the reentry circuit.

### CLASSIFICATION OF ANTIDYSRHYTHMIC DRUGS

Medications used to treat dysrhythmias are classified into four major categories on the basis of their electrophysiologic effects (Box 79-1). Other agents fall outside this classification system and are discussed separately.

Class I agents exert their major effects on the fast Na\(^{+}\) channels, resulting in membrane stabilization. The subclasses IA, IB, and IC are distinguished on the basis of differential effects on depolarization, repolarization, and conduction. Class II agents are the beta-adrenergic antagonists, which depress SA node automaticity and slow AV node conduction. Beta-blockers can also suppress conduction in ischemic myocardial tissue. Class III agents prolong repolarization and refractory period duration, predominantly via

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**Figure 79-1.** Flow of various ions across the myocardial cell membrane. Na\(^{-}\)-K\(^{+}\) pump exchanges three Na\(^{+}\) ions for each two K\(^{+}\) ions, generating a net negative flow of 10 mV. The flow of K down the concentration gradient (dark arrow) generates another 80 mV of current. The Na\(^{-}\)-Ca\(^{2+}\) exchange adds little to the resting potential. ATPase, adenosine triphosphatase. (From Marriott HJL, Conover MB: Advanced Concepts in Dysrhythmias, 2nd ed. St Louis, Mosby, 1989.)
their effects on K⁺ channels. Class IV agents are the Ca²⁺ channel blockers, which slow conduction through the AV node and suppress other calcium-dependent dysrhythmias. Other agents important in the emergency treatment of dysrhythmias include magnesium sulfate, digitalis, and adenosine.

All antidysrhythmics can cause “prodysrhythmic effects.” This occurs most often in patients with existing structural heart disease and in patients receiving new or higher doses of antidysrhythmic agents. The class I and III agents are associated with prodysrhythmic effects in up to 15% of patients.¹,²

**Class IA Agents**

Class IA agents slow conduction through the atria, AV node, and His-Purkinje system and suppress conduction via accessory pathways. Class IA agents also exhibit anticholinergic and mild negative inotropic effects, with procainamide having the least effect on contractility. Procainamide is the most commonly used class IA agent in the emergency treatment of ventricular and supraventricular dysrhythmias. Intravenous procainamide is administered at a rate of 20 to 30 mg/min in stable patients until the dysrhythmia is terminated, hypotension occurs, or the QRS complex widens (to 50% of the pretreatment width), or up to a total dose of 18 to 20 mg/kg (12 mg/kg if congestive heart failure is present). The peripheral vasodilatory effects cause the hypotension, and the risk of prodysrhythmia is approximately 5%.
Class IB Agents

Class IB agents slow conduction and depolarization less than other class I agents, and they shorten repolarization rather than prolonging it. Class IB agents have little effect on accessory pathway conduction. Lidocaine is the sole class IB agent used in emergency rhythm management. Lidocaine is useful for suppressing dysrhythmias from enhanced automaticity such as VT. Lidocaine also suppresses SA and AV node function and is associated with asystole in the setting of myocardial ischemia.

Class IC Agents

The class IC agents profoundly slow depolarization and conduction and have pronounced antidysrhythmic properties. Up to 15% of patients treated with class IC agents experience new or increased ventricular dysrhythmias. Class IC agents are approved only for oral use in the United States.

Flecainide

Flecainide is used in the treatment of supraventricular tachycardias (SVTs) and certain forms of VT outside the setting of acute ischemia. Initiation of oral therapy requires continuous ECG monitoring. Flecainide has high oral bioavailability, but its half-life is variable, and the therapeutic index (in terms of QRS and QT prolongation) is narrow. Long-term use of flecainide in those with a previous myocardial infarction is associated with an increased risk of fatal dysrhythmia.

Propafenone

Propafenone shares properties with both class IA and IC agents, with intermediary effects on sodium channels. It also possesses some beta-adrenergic and calcium channel–blocking properties.
Propafenone is used for the oral treatment of atrial fibrillation and ventricular dysrhythmias. Compared with flecainide, propafenone has a lower observed prodysrhythmic rate at therapeutic doses.

### Classification of Antidysrhythmic Drugs

**Class I**
- Sodium (fast) channel blockers. Slow depolarization with varying effects on repolarization. These “membrane-stabilizing” drugs have prominent antiectopic effects.

**Class IA**
- Moderate slowing of depolarization and conduction. Prolong repolarization and action potential duration.
  - Procainamide
  - Quinidine
  - Disopyramide

**Class IB**
- Minimally slow depolarization and conduction. Shorten repolarization and action potential duration.
  - Lidocaine
  - Phenytoin
  - Ticainide
  - Mexiletine

**Class IC**
- Markedly slow depolarization and conduction. Prolong repolarization and action potential duration.
  - Flecainide
  - Encainide
  - Lorcainide
  - Propafenone (shares properties with class IA agents)
  - Vernakalant (atrial specific, investigational)

**Class II**
- Beta-adrenergic blockers
  - Propranolol
  - Esmolol
  - Metoprolol
  - Atenolol

**Class III**
- Antifibrillatory agents. Prolong action potential duration and refractory period duration with antifibrillatory properties.
  - Bretylium (historical significance)
  - Amiodarone
  - Dofetilide
  - Ibutilide*
  - Sotalol†
  - Dronedarone
  - Azimilide

**Class IV**
- Calcium (slow) channel blockers
  - Verapamil
  - Diltiazem

**Miscellaneous**
- Digitalis
- Magnesium sulfate
- Adenosine

*Shares activity with class I agents.
†Shares activity with class II agents.

Setting of acute myocardial ischemia, beta-blockers play a role in preventing ventricular dysrhythmias.

Side effects of beta-blockers include bronchospasm, bradycardia, hypotension, and heart failure. All beta-blockers are active at both beta_1_ and beta_2_ receptors (Table 79-2) to varying degrees. Those with more prominent beta_2_ effects are said to be cardioselective. Relative contraindications to the use of beta-blockers include asthma or chronic obstructive lung disease, advanced congestive heart failure, and third-trimester pregnancy. Beta-blockers should not be used in patients with preexisting bradycardia or heart block beyond first-degree. Intravenous beta-blockers should be used cautiously in conjunction with calcium channel blockers because of the risk of additive side effects. Acute side effects of beta-blockers include bronchospasm, heart failure, excessive bradycardia, and hypotension.

**Esmolol**
Esmolol is a beta_1_-selective agent attractive in the emergency setting because of its rapid onset of action and short elimination half-life (minutes). An intravenous bolus of 500 µg of esmolol per kilogram followed by a continuous infusion starting at 50 µg/kg/min is a common starting approach, with titration to need and effect.

**Metoprolol**
Metoprolol is available in oral and intravenous preparations. Although not approved for dysrhythmia treatment in the United States, metoprolol (5–10 mg intravenously [IV] every 10–15 minutes titrated to response) will slow atrial and nodal fast rhythms and is often used for these purposes.

**Class III Agents**
All class III agents prolong the refractory period by blocking K+ channels, and they have variable effects on the QT interval. In general, class III agents are alternatives to the class I agents for the treatment of many ventricular and atrial dysrhythmias.

**Bretylium**
Bretylium was once the most commonly used class III agent, but its hemodynamic side effects were unfavorable, and it is no longer available in the United States.

**Amiodarone**
Amiodarone is approved for the treatment of both ventricular and supraventricular dysrhythmias. In addition to features in common
with all class III agents, amiodarone has numerous other effects, including actions that are similar to those of class IA, class II, and class IV agents.

The serum half-life of amiodarone is 25 hours after a single intravenous dose and up to 50 days during long-term oral use. Because of the unusual pharmacokinetics, oral regimens vary widely. The acute side effects of amiodarone are primarily limited to hypotension, bradycardia, and heart failure (Box 79-2). There is an additive risk of bradycardia and hypotension when amiodarone is used in conjunction with calcium channel or beta-adrenergic blockers. Rates of prodysrhythmia are relatively low (1-3%). Long-term amiodarone use, however, is associated with significant extracardiac side effects, including irreversible lung and thyroid disease. Amiodarone alters the pharmacokinetics of numerous other drugs, including digoxin and warfarin.

**Ibutilide**

Ibutilide has a unique mechanism of action characterized by induction of a slow inward Na\(^+\) current, thereby prolonging the refractory period. Intravenous ibutilide is approved for cardioversion of atrial fibrillation and atrial flutter.\(^1\) Ibutilide is associated with QT interval prolongation, and polymorphic VT is more common with amiodarone although still relatively rare (0.9-2.5%).

**Sotalol**

Sotalol is beta-adrenergic receptor blocker with type III antidysrhythmic properties via inhibition of K\(^+\) channels. It is used orally for suppression of supraventricular and ventricular dysrhythmias. The incidence of QT prolongation and risk of polymorphic VT require that initiation of sotalol therapy occur in a monitoring setting.

**Dofetilide**

Dofetilide is a pure class III agent that blocks K\(^+\) efflux, increasing the refractory period. It is approved for chemical cardioversion and maintenance of sinus rhythm in patients with atrial fibrillation or flutter,\(^1\) but because of its prodysrhythmic effects, it has limited usefulness.

**Dronedarone**

Structurally related to amiodarone, dronedarone displays class III properties plus those of other antidysrhythmic classes. The incidence of polymorphic VT associated with dronedarone is very low. Dronedarone is approved for oral use to maintain sinus rhythm in patients with atrial fibrillation or flutter but is contraindicated in patients with severe or recent heart failure decompensation.\(^2\) Long-term effects and the ideal populations for the use of this drug remain unclear.\(^3\)

**Class IV Agents**

Class IV agents block the slow Ca\(^{2+}\) channels, slowing conduction within the AV node and suppressing the SA node to a lesser degree. Like beta-blockers, their primary use is in patients with SVTs. Of the two commonly used class IV agents, diltiazem is more frequently used for ventricular rate control in patients with atrial fibrillation or flutter.

All class IV agents are associated with peripheral vasodilation. Verapamil has the least effect on peripheral vascular tone, and diltiazem has an effect between that of verapamil and purely peripherally acting calcium channel blockers, such as nifedipine. In the acute setting, intravenous calcium salts (1 g slow intravenous delivery) can attenuate peripheral vasodilatory effects. Class IV drugs should not be used in patients with second- or third-degree AV block and should be used with close monitoring in those with first-degree block.

**Diltiazem**

Intravenous diltiazem 0.25 to 0.35 mg/kg over 2 minutes controls the ventricular response rate in 90% of patients with atrial fibrillation and atrial flutter and is associated with minimal hypotension. If the intravenous bolus is successful, a continuous infusion (5-15 mg/hr initially, titrated to need) or an oral dose (60-90 mg initially) will help sustain the response.

**Verapamil**

Verapamil terminates or controls the ventricular response rate in 80 to 90% of tachycardic rhythms.

Intravenous verapamil is given at a dose of 0.1 mg/kg over 1 to 2 minutes; for the average healthy adult, this translates to a dose of 5 to 10 mg. In elderly patients or those with preexisting borderline hypotension (systolic blood pressure of 90-110 mm Hg), a smaller dose (0.05 mg/kg, or 2.5-mg increments) is preferred.

**Miscellaneous Agents**

**Digoxin**

Digitalis compounds have a variety of effects on myocardial cells. Digoxin inhibits the ATP-dependent Na\(^+-K^+\) exchange pump, increasing intracellular Na\(^+\) concentrations and decreasing intracellular K\(^+\) concentrations. The resultant increase in intracellular Ca\(^{2+}\) concentration accounts for the positive inotropic effects of digitalis. Digoxin can behave as an excitant, a depressant, or both. Excitatory effects of digoxin have to do with enhanced automaticity and triggered activity, particularly at high therapeutic or toxic doses. At the same time, digoxin slows AV node conduction at therapeutic doses via lengthening of the refractory period.
Adverse Effects of Digitalis

**Common Effects**
- Gastrointestinal intolerance (nausea, vomiting, abdominal pain, diarrhea, anorexia)
- Fatigue
- Drowsiness
- Visual color disturbances
- Headache
- Depression
- Apathy

**Less Common Effects**
- Psychosis
- Cardiac symptoms
- Heart block
- Increased ectopy
- Combined block and ectopy (multifocal atrial tachycardia with block or complete atrioventricular block with accelerated junctional rhythm, usually in overdose setting)
- Ventricular tachycardia

Digoxin (0.25-0.5 mg IV) can control the ventricular rate in patients with SVTs, including atrial fibrillation and atrial flutter. Because of its delayed onset of action and narrow therapeutic window, however, digitalis is not a first-line agent for emergency therapy. It is not true that digoxin promotes conversion to a sinus rhythm any more than other rate-controlling agents. Side effects of digoxin are listed in Box 79-3 and are aggravated by hypokalemia, hypercalcemia, hypomagnesemia, increased catecholamines, and acid-base disturbances. Digoxin overdose therapy is covered elsewhere.

**Magnesium**

Magnesium can aid control of many tachydysrhythmias. Magnesium (1-2 g IV) plays a central role in terminating torsades de pointes and as an aid in VT therapy. Aside from these indications, magnesium is considered at best an adjunctive, second- or third-line agent in other settings.

**Adenosine**

Adenosine is a naturally occurring purine nucleoside used for the termination of narrow-complex tachydysrhythmias. Administered as an intravenous bolus, adenosine causes an abrupt slowing of AV conduction in both anterograde and retrograde pathways. Adenosine usually has an onset of action of 5 to 20 seconds and a duration of effect of 30 to 40 seconds. Except in rare cases of catecholamine-induced ventricular dysrhythmias, adenosine has little or no effect on infranodal conduction. For this reason, adenosine is often used as a diagnostic agent in patients with wide-complex tachydysrhythmias when the cause is unclear. An initial dose of 6 mg as a rapid bolus for adults weighing 50 kg or greater is recommended, with flush through a large peripheral vein. If no response is seen within 1 to 2 minutes, the second dose is doubled (12 mg) and administered. If no effect is seen after a final 12-mg dose, the rhythm should be reassessed and another agent used. Pediatric doses are 0.05 mg/kg initially with doubling at similar intervals up to a total dose of 0.25 mg/kg. Side effects coincide with the onset of clinical effects and occur in up to a third of patients but are usually minor. These include flushing, dyspnea, chest pressure, nausea, headache, dizziness, transient bradycardia or heart block, and hypotension (seen rarely, from the vasodilatory properties). These side effects usually resolve rapidly without treatment, although many patients are intensely uncomfortable for a short period. Asystole can occur but is usually transient though unsettling to those observing.

Because of its short duration of action, adenosine is not an effective rate control agent for atrial fibrillation or flutter, although it can help unmask these rhythms when not apparent on the initial surface ECG. Similarly, some narrow-complex tachydysrhythmias that terminate with adenosine will recur later given the short duration of action.

**APPROACH TO DYSRHYTHMIA RECOGNITION AND MANAGEMENT**

Dysrhythmias are classified according to their electrophysiologic origin, ECG appearance, and underlying ventricular rate. Although overlap exists, the following categorization is useful:
- Bradycardias
- Extrasystoles
- Narrow-complex (QRS less than 0.12 second) tachycardias (regular and irregular)
- Wide-complex (QRS 0.12 second or greater) tachycardias (regular and irregular)

Classically, the approach to any specific dysrhythmia is defined on the basis of the clinical stability. *Unstable patients* demonstrate evidence of severe or multiple end-organ features of hypoperfusion, such as altered sensorium, respiratory distress, hypotension, syncope, or chest pain suggestive of myocardial ischemia. *Stable patients* may be asymptomatic or have mild symptoms, such as light-headedness, dyspnea on exertion, palpitations, or mild anxiety. In practice, clinical stability is a continuum; in the absence of profound altered sensorium or hypotension, a clear line distinguishing stable and unstable patients is often not present.

It is important to consider whether a dysrhythmia is the cause or an effect of a clinical presentation; for example, rapid atrial fibrillation may be causing hypotension or resulting from profound volume depletion. Failure to consider the clinical situation can lead to an inappropriate focus on diagnosing and treating the rhythm to the detriment of the patient. With recognition of this need to incorporate the overall clinical picture, treatment of those with a dysrhythmia and clear instability is empirical and assumes the rhythm is the cause, whereas stable patients can be approached in a more systematic and thoughtful manner to identify the cause and choose the most appropriate therapy.

**Initial Assessment of Stable Patients**

The approach to the patient with a stable dysrhythmia begins with gathering evidence from the history, physical examination, 12-lead ECG, and a rhythm strip. The nature of any symptoms, including the timing, velocity of onset (gradual vs. abrupt), and duration, is important. For the patient with “palpitations,” questions about the rate and regularity of the heartbeat are often asked, but having the patient tap out the rhythm with a finger can often be more valuable. Inquiring about precipitating events and associated symptoms such as dizziness, chest pain, dyspnea, or syncope is critical to understanding the context of any dysrhythmia. The past history—of rhythm disturbances or a prior history of ischemic or structural heart disease—and a thorough medication history are essential. For example, a new and symptomatic wide-complex tachycardia in a patient with known ischemic heart disease is much more often VT than a supraventricular event. Occasionally the family history can be helpful, particularly if there are first-degree relatives with a history of dysrhythmia, unexplained syncope, or sudden death—all of which suggest an inherited disorder such as an accessory pathway or Brugada’s syndrome.

Aside from palpating the pulse and listening to the heart sounds, the physical examination should focus on detecting subtle
Ventricular Rhythm

Multiple Rhythm: QRS best seen in inferior leads or V detect such things as the presence or absence of P waves (often adequate for diagnosis, but multiple leads are often required to with a suspected dysrhythmia. Use of a single ECG lead is often dysrhythmias is often at the onset or termination of the rhythm, observed. Because the most useful information about paroxysmal syndrome, the 12-lead ECG is essential for evaluation of any patient’s rhythm on a continuous cardiac monitor.

Basic Electrocardiographic Observations during Dysrhythmia Analysis

1. Ventricular rate: Fast (>100 complexes/min), slow (<60 complexes/min), or normal (60-100 complexes/min).
2. Rhythm: Regular, completely irregular (irregularly irregular or chaotic), regular with occasional irregularity, or grouped impulses. Calipers and long strips are recommended to detect subtle irregularity.
3. QRS width: Prolonged (>0.12 s), borderline (0.09-0.12 s), or normal. If determined without electrocardiogram being physically present (e.g., prehospital radio medical command), it is helpful to ask for QRS duration in number of small boxes from printed rhythm strip (each box equals 0.04 s) to ensure accuracy.
4. P wave presence and relationship to QRS complexes: This may require mapping of P waves with calipers to detect those falling within QRS complex or T wave.
5. Rhythm changes: Examine these areas closely for clues.
6. Multiple leads, especially chest leads or esophageal lead if difficulties with P wave visualization are experienced.
7. Comparison with previous tracings (if available) is often valuable.

Figure 79-7. Note the P waves before the QRS complexes in lead aVF.

Box 79-4

<table>
<thead>
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</tbody>
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Pseudodysrhythmias

Loose leads, muscle contraction, shivering, tremors, and other patient movement can produce artifactual findings on a monitor, rhythm strip, or 12-lead ECG (Fig. 79-8). Such pseudodysrhythmias can mimic and are often mistaken for serious dysrhythmias, including ventricular fibrillation, illustrating the need to avoid making decisions on the basis of ECG tracings without incorporating the clinical context.

SPECIFIC DYSRHYTHMIAS

Sinus Bradycardia, Sinoatrial and Atrioventricular Block

Bradycardia is defined as a ventricular rate of less than 60 beats/min, although in practice rates above 50 beats/min are not usually a concern. Bradycardia may occur because of depression of the sinus node discharge or because of a conduction system block; when the ventricular rate falls, a subsidiary pacemaker elsewhere in the atrium, AV junction, or ventricle may assume the dominant role, resulting in an escape rhythm.

Sinus Bradycardia

Sinus bradycardia is characterized by a P wave with normal morphology and a fixed P-P interval that is equal to the R-R interval,
Sinus Dysrhythmia

Sinus dysrhythmia is similar to sinus bradycardia and is managed in the same manner; it is diagnosed when a variable P-P interval is seen (Fig. 79-10). It is considered a normal variant and is seen frequently in children and young adults.

Sinus Arrest and Sinoatrial Exit Block

Aside from sinus bradycardia and sinus dysrhythmia, disturbances in sinus rhythm are characterized by an absence of atrial depolarization, diagnosed by missing P waves on the ECG (Fig. 79-11). A lack of atrial depolarization can occur because of either failure of the sinus node to generate an impulse (sinus arrest) or failure of impulse conduction out of the SA node (SA exit block). When incomplete block (i.e., one or more missing P waves) exists, a compensatory pause equal to the P-P interval implies the presence of SA exit block, whereas a shorter pause more likely represents sinus arrest. Also, with SA exit block, it is not uncommon to see dropped P waves in regularly occurring patterns, representing 2:1,
Atrioventricular Block

AV block results from impaired conduction through the atria, AV node, or proximal His-Purkinje system. First- and second-degree AV blocks represent an incomplete conduction disturbance, whereas third-degree block indicates a complete interruption.

First-Degree Atrioventricular Block.

First-degree AV block is characterized by prolonged conduction of atrial impulses without the loss of any single impulse. This can occur at the level of the atria, the AV node (most common), or the His-Purkinje system. On the ECG, first-degree AV block is defined by a prolonged PR interval (>0.20 second), typically with a narrow QRS complex (Fig. 79-12). First-degree AV block is a normal variant in up to 2% of healthy young adults but is also seen in pathologic conditions associated with depression of AV node conduction. First-degree AV block requires no specific treatment, but nodal depressing agents should be given with caution in this setting.

Second-Degree Atrioventricular Block.

Second-degree AV block is characterized by one or more (but not all) sinus impulses failing to reach the ventricles. The conduction ratio is defined as the ratio of the number of P waves to the number of QRS complexes over a period of time (e.g., 3:2, 2:1). A 2:1 conduction ratio does not 3:1, or 4:1 block. Both sinus arrest and SA exit block are often manifestations of intrinsic SA node disease or looming SSS, but they can also be seen under conditions of increased vagal tone, both benign and pathologic. When the patient is symptomatic, the approach to treatment is similar to that for sinus bradycardia.

Sick Sinus Syndrome

Sick sinus syndrome refers to a group of dysrhythmias from disease of the sinus node and its surrounding tissues, including sinus bradycardia, sinus arrest, and SA exit block. A variant of SSS known as bradycardia-tachycardia syndrome is characterized by one or more of these bradydysrhythmias alternating with a tachydysrhythmia, typically atrial fibrillation. SSS is most common in elderly adults, a result of fibrotic degeneration of the cardiac conduction system. It is also associated with cardiomyopathies, connective tissue diseases, and certain drugs. In the acute setting, SSS is suspected when an elderly patient has syncope and the ECG demonstrates sinus impulse abnormalities. Long-term management often requires permanent pacemaker placement for symptomatic bradycardia, in addition to any conventional therapy for atrial fibrillation.
always mean worse conduction system disease than a 4:2 ratio, and not all 2:1 conduction is pathologic. For example, an atrial impulse rate of 300 complexes per minute (common in atrial flutter) presented to the AV node usually results in conduction of half the impulses, producing a ventricular rate of 150 beats/min. This conduction ratio does not represent AV dysfunction or important block because the AV node is responding normally and preventing excessive ventricular stimulation. Conversely, a sinus rhythm at a rate of 70 P waves per minute with a 2:1 ratio (a ventricular rate of 35 beats/min) represents profound AV block and dysfunction. The term high-grade second-degree block is best applied to conduction disturbances that prevent physiologic ventricular response rates and not solely to higher conduction ratios. Second-degree AV block can be further classified into two types on the basis of the underlying pathophysiology and ECG appearance (Table 79-3).

**Type I Second-Degree Atrioventricular Block.** Type I second-degree AV block, also called Wenckebach or Mobitz I AV block, is associated with a conduction disturbance within the AV node. The surface ECG is characterized by a progressive lengthening of the PR interval until an impulse is not conducted (“dropped beat”). The progressive lengthening of the PR interval gives the appearance of successive P waves retreating into the preceding QRS complexes (Fig. 79-13). By definition, the longest R-R interval (i.e., following the dropped beat) is less than twice the length of the shortest. On a rhythm strip, type I second-degree AV block gives the appearance of “grouped beating,” especially pairs or trios (i.e., 3:2 or 4:3 block), but occasionally in larger multiples. Grouped beating is not unique to type I second-degree AV block; it occurs in a variety of other conditions (Box 79-5), including SA exit block, type II second-degree AV block, and extrasystoles with or without block.

Type I second-degree AV block occurs in acute and chronic conditions associated with increased vagal tone and usually requires no treatment. In well-conditioned adults, type I second-degree AV block may be a normal variant. In the setting of an acute inferior myocardial infarction, this type of AV block is generally transient and is associated with a good outcome.

**Type II Second-Degree Atrioventricular Block.** Type II second-degree AV block, or Mobitz II block, represents a conduction block just below the level of the AV node and is never a
normal variant. On the surface ECG, conduction of atrial impulses is sporadic and typically periodic, but the PR interval does not widen (Fig. 79-14). The QRS complex is intrinsically narrow, but concomitant infranodal conduction disturbances (i.e., bundle branch blocks) are not uncommon with type II second-degree AV block.

Type II second-degree AV block can occur at conduction ratios similar to those seen with type I second-degree block but can also occur at higher conduction ratios (e.g., 3:1, 4:1, or higher). When the conduction ratio is 2:1, it may be impossible to distinguish type I from type II second-degree AV block on the basis of the surface ECG because there is no progression of PR intervals. In general, the presence of a prolonged PR interval makes type I block more likely, whereas the presence of wide QRS complexes makes type II block more likely. A rhythm strip demonstrating periods of block with higher or lower conduction ratios can be useful in distinguishing between type I and type II AV block.

Type II second-degree AV block generally carries a worse prognosis than type I second-degree AV block. In acute myocardial infarction, type II AV block is associated with anterior wall ischemia and often progresses to complete AV block. The presence of any form of new type II second-degree AV block, even if asymptomatic, requires prolonged observation or admission for monitoring and preparation for temporary pacing. For patients with signs of hypoperfusion at rest, immediate temporary pacing—either transcutaneous or transvenous—is the treatment of choice. Atropine typically has no effect on conduction disturbances below the AV node and is a temporizing measure during preparation for pacing when required.

Third-Degree Atrioventricular Block. Third-degree AV block (also known as complete AV block or complete heart block) is characterized by absent conduction of all atrial impulses (Fig. 79-15) accompanied by a slow escape rhythm. The width and frequency of QRS complexes depends on the site of the escape-rhythm pacemaker. Pacemakers above the His bundle are associated with a

| Box 79-5 Causes of Grouped Impulses |

- Wenckebach mechanism (usually at atrioventricular node, but can occur elsewhere)
- Atrial tachycardia or flutter with alternating conduction
- Frequent extrasystoles
- Nonconducted atrial trigeminy
- Concealed or interpolated extrasystoles

Figure 79-14. A, Second-degree atrioventricular (AV) block, type II. In this example, 3:1 conduction is shown. B, Second-degree AV block with 2:1 conduction. From the rhythm strip alone, it is difficult to categorize this as type I or II block. (A, From Goldberger AL, Goldberger E: Clinical Electrocardiography, 2nd ed. St Louis, Mosby, 1981.)

Figure 79-15. Complete (third-degree) atrioventricular block. Note that there is no constant relationship of P waves to QRS complexes even though some are noted in close proximity.
narrow-complex QRS at a rate of 45 to 60 beats/min, whereas pacemakers at or below the His bundle produce a wide-complex QRS at a rate of 30 to 45 beats/min.

The hallmark of complete heart block is AV dissociation (i.e., no electrocardiographic relationship between atrial and ventricular activity), with an R-R interval that is longer than the P-P interval. Conversely, the presence of AV dissociation with an R-R interval that is shorter than the P-P interval (e.g., as occurs with accelerated junctional rhythms and VTs) does not imply third-degree heart block. When the atrial rate and the escape rate are similar (or isorhythmic), AV dissociation is difficult to appreciate unless a long rhythm strip is examined and the P waves and QRS complexes are closely tracked. One particular challenge in diagnosing AV dissociation occurs when atrial fibrillation is accompanied by complete heart block; the fibrillatory waves, which may not be apparent, are followed by a slow and regular ventricular response (so-called regularized atrial fibrillation). This specific dysrhythmia is classically associated with digoxin toxicity.

Third-degree AV block can be congenital, but newly symptomatic third-degree block is commonly acquired, either because of senescent degeneration of the electrical conduction system or as a result of acute ischemia, drug therapy, or other pathologic conditions (e.g., Lyme disease).

In the field or ED, the approach to third-degree heart block depends on the cause and symptoms. Patients with newly acquired or symptomatic third-degree AV block should be admitted to the hospital. For patients with third-degree heart block who are markedly symptomatic (i.e., signs of hypoperfusion at rest), temporary transvenous or transcutaneous pacing is best until either a reversible cause is addressed (e.g., ST elevation myocardial infarction, beta-blocker overdose) or a permanent pacemaker is placed. Atropine is usually ineffective.

**Extrasystoles**

An extrasystole is an electrical impulse originating from an ectopic atrial or ventricular focus. Depending on the site of origin and the timing of the impulse, there may not be an associated mechanical contraction. The terms “premature atrial contraction” and “premature ventricular contraction” are misleading but widespread and used here for ease. The extrasystole and its preceding impulse are referred to as a **couplet**, and the **coupling interval** refers to the period between these two beats.

**Bigeminy** (Fig. 79-16) occurs when there is an extrasystole after every native beat, so that every other impulse is extrasystolic; **trigeminy** (every third beat) and **quadrigeminy** (every fourth beat) are similar. Most extrasystoles are the result of enhanced automaticity, whether in the atria, AV node, His-Purkinje system, or ventricles.

**Premature Atrial Contractions**

Premature atrial contractions (PACs; Fig. 79-17) are common and usually have little clinical significance. On occasion, a PAC can be the precipitant of a more important dysrhythmia, such as atrial fibrillation, atrial flutter, or SVT. PACs can be distinguished on the ECG by the presence of an abnormal P wave early within a cardiac cycle, although sometimes the P wave may be difficult to detect if it is buried within the preceding T wave.

Most PACs will depolarize the sinus node, resetting its refractory period. Because of this, the P-P interval between two sinus beats surrounding a PAC will be less than twice the intrinsic P-P cycle length (see Fig. 79-17). If a PAC reaches the AV node or the infranodal conducting system during its absolute refractory period, there will be no ventricular depolarization. A nonconducted (or blocked) PAC typically results in a noncompensatory pause (i.e., R-R interval less than twice the intrinsic R-R cycle; Fig. 79-18) because the sinus node is reset. Blocked PACs are among the most common causes of pauses seen on an ECG but can be easily overlooked if one is not careful to inspect for ectopic P waves.

If a PAC reaches the infranodal conducting system during its relative refractory period, the QRS complex may appear wide or **aberrant**, typically with a right bundle branch block (RBBB) pattern. Because the refractory period depends on the previous cycle length, an early-arriving PAC that follows a long cardiac cycle...
Premature Ventricular Contractions

Premature ventricular contractions (PVCs) occur in wide a variety of pathologic and nonpathologic states. Occasional PVCs are commonly seen in healthy adults, more so under conditions associated with catecholamine excess, such as pain, anxiety, and use of stimulants (e.g., caffeine, nicotine, cocaine, amphetamines). Pathologic conditions associated with frequent PVCs include myocardial infarction, electrolyte disturbances, and medication toxicity. Although usually not requiring any intervention, frequent PVCs may occasionally herald the onset of VT—for example, in the setting of ST elevation myocardial infarction or in patients with a prolonged QT interval.

A PVC appears as a wide–QRS complex extrasystole without a preceding P wave (Fig. 79-19). Most PVCs capture the AV node, making it refractory to the next arriving atrial impulse. Because retrograde conduction of a PVC rarely extends far enough to capture and reset the SA node, however, atrial impulses continue to arrive at the AV node at the intrinsic sinus rate. As a result, the R-R interval surrounding a PVC ends up being equal to twice the intrinsic R-R interval length (see Fig. 79-19), a phenomenon known as a compensatory pause. Rarely, a PVC will capture the SA node, resulting in a noncompensatory pause, or will fail to capture the AV node, leaving the underlying rhythm completely unaffected (a so-called interpolated PVC; Fig. 79-20).

The morphology of a PVC depends on the origin of the impulse, with a left bundle branch block (LBBB) appearance resulting from an extrasystolic focus in the right ventricle and vice versa. Multiform (or “multifocal”) PVCs come from more than one source.
QRS often

and have variable morphologies. When a PVC occurs at or around the time that a supraventricular impulse is set to depolarize the ventricle, the result is a fusion QRS complex (Fig. 79-21). Table 79-4 notes common features of PACs and PVCs.

Therapy for PVCs is directed toward correcting any precipitating condition, be it a catecholamine excess, a drug effect, an electrolyte imbalance, or cardiac ischemia (Box 79-6). When occurring in isolation, symptomatic PVCs can be treated with low-dose beta-blocker therapy, but this is rarely an emergency need. Although lidocaine will suppress PVCs, it is not recommended in the absence of VT owing to limited clinical benefit and the risk of asystole.

**Narrow-Complex Tachycardia**

Narrow-complex tachycardias are defined by a QRS complex duration of 0.12 second or less on surface ECG with a ventricular rate of greater than 100 beats/min. The term supraventricular tachycardia is a confusing one because it is sometimes used in reference specifically to AV reentry tachycardia, but in other contexts it can denote any tachycardia originating at or above the AV node.

ECG features that help in distinguishing between different narrow-complex tachycardias include the appearance of P waves and the regularity or irregularity of the R-R interval. For example, a narrow-complex tachycardia with absent P waves and an irregular R-R interval is almost certainly atrial fibrillation. With rapid tachycardias, however, evidence of atrial depolarization is often obscured by ventricular repolarization; for example, with a regular, narrow-complex tachycardia at a rate of 150 beats/min, it can be difficult to distinguish sinus tachycardia from atrial flutter or a JT. Vagal maneuvers or adenosine can be used to transiently slow AV nodal conduction and expose evidence of atrial depolarization and aid diagnosis. Alternatively, the patient may convert to sinus rhythm, in which case AVNRT is both diagnosed and treated.

**Sinus Tachycardia**

Sinus tachycardia is characterized electrocardiographically by a regular, narrow-complex tachycardia with normal P waves preceding each QRS complex (Fig. 79-22). In adults, sinus tachycardia rarely exceeds a rate of 170 beats/min, but in infants and young children, it is not unusual to see rates above 200 to 225 beats/min.
Sinus tachycardia can be seen in any context associated with sympathetic excess, whether endogenous (e.g., pain, anxiety, fever, hyperthyroidism) or exogenous (stimulants and other drugs). The approach to the patient with sinus tachycardia centers on identifying and addressing the cause(s); rarely is specific rhythm therapy indicated.

Atrial Tachycardia

Atrial tachycardia (AT) is an atrial rhythm occurring at a rate of more than 100 beats/min arising from a non–sinus node site (or sites) within the left or right atrium. The hallmark of AT is the presence morphologically abnormal P waves on the surface ECG (Fig. 79-23). If the site of origin is close to the sinus node,
Atrial fibrillation is usually identified by electrical chaos; it starts from chaotic depolarization of atrial tissues caused by multiple microreentry circuits, generating 300 to 600 atrial impulses per minute. This chaotic activity reduces cardiac output from a loss of coordinated atrial contractions and from a rapid ventricular rate, both of which may limit the diastolic filling and stroke volume of the ventricles. Patients with atrial fibrillation are at risk for developing left atrial thrombi, especially in the left atrial appendage, and consequent embolic events.

Atrial fibrillation may be paroxysmal, persistent, or permanent, and long-term approaches to management depend on many factors, including chronicity, symptomatology, underlying heart disease, and other comorbidities.

Electrocardiographically, the hallmark of atrial fibrillation is a “irregularly irregular” QRS pattern (Fig. 79-25). Although atrial fibrillation is not the sole cause of an irregular ventricular rhythm, it is by far the most common (Box 79-7). Atrial fibrillatory waves appear “coarse” or “fine” on the basis of their amplitude and are often best appreciated in the inferior leads or lead V1.
Typically, the ventricular rate in atrial fibrillation is limited by the refractory period of the AV node, with response rates rarely exceeding 150 to 170 beats/min and often slower, particularly in the presence of nodal blocking agents. Atrial fibrillation in an adult with a ventricular rate exceeding 200 beats/min strongly suggests the presence of an accessory conduction pathway and has important implications for management (see later). Frequently, this rapid atrial fibrillation with an accessory path will have a wide QRS complex; if the irregularity of ventricular depolarization is not sought, it is easy to mistake this for VT. When a wide QRS complex is seen at rates below 200 beats/min but with ventricular chaos, an existing or acquired bundle branch block is likely present.

The Ashman phenomenon refers to aberrant ventricular conduction of an early-arriving atrial impulse following a relatively long R-R interval, as a result of a partially refractory His bundle. Such aberrantly conducted impulses are often seen in atrial fibrillation but can occur in any irregular rhythm in which long-short cycle sequences occur, and they typically assume an RBB block pattern (Fig. 79-26). Ashman beats can be mistaken for PVCs or paroxysmal VT.

Atrial fibrillation is most commonly associated with underlying heart disease or hypertension (Box 79-8), but it can also occur in isolation (so-called lone atrial fibrillation) or as a manifestation of hyperthyroidism. As many as one third of patients with congestive heart failure also have atrial fibrillation.

The presentation of patients with atrial fibrillation is variable. For example, patients without underlying cardiopulmonary disease may tolerate atrial fibrillation with ventricular rates of 150 to 170 at rest and complain only of palpitations or exercise intolerance. On the other hand, a patient with left ventricular dysfunction and new or worsened rate control may experience dyspnea at rest even with a modest rate increase. In a patient with preexisting atrial fibrillation and a new rapid ventricular rate, it is key to seek evidence that the rapid rate is a response to some other hemodynamic stress, such as decompensated heart failure, sepsis or hypovolemic shock, massive pulmonary embolism, or cardiac tamponade. Failure to recognize and take into account the underlying cause of instability may result in counterproductive attempts at rate control or cardioversion.

For stable patients with new or recurrent rapid atrial fibrillation, administration of a nodal blocking agent with a goal of achieving a target ventricular rate of 120 beats/min or less is a first step. Intravenous calcium channel blockers (diltiazem and verapamil) or beta-blockers (metoprolol) offer the advantage of being easily titrated and can be followed by an oral agent before discharge. Nodal agents should not be used for rate control in the setting of accessory pathway conduction because AV conduction (with retrograde conduction into the accessory pathway) may be the only thing preventing the ventricular rate from accelerating even further and degenerating into ventricular fibrillation. The debate as to whether asymptomatic patients with persistent atrial fibrillation benefit additionally from rhythm control, either via cardioversion or by other means (e.g., ablation), is ongoing without a definitive answer. In choosing rate control for the long term, the ideal target rate is debated, with some advocating less than 80 beats/min at rest instead of the common goal of 110 beats/min or less; no clear evidence of either target being superior exists yet.

For stable patients with new-onset or newly recurrent atrial fibrillation—defined as having a duration of 72 hours or less—ED cardioversion is an option. If atrial fibrillation has been present longer or for an uncertain interval in the absence of ongoing anticoagulation, however, cardioversion is not recommended to avoid the risk (1-4% at 30 days) of systemic embolization. The choice of electrical versus pharmacologic cardioversion is dependent on institutional factors and patient preference, although published success rates are higher (80% or greater) with electrical conversion.

Among patients with new or recurrent atrial fibrillation of less than 48 to 72 hours’ duration, up to 50% will convert spontaneously to sinus rhythm within 24 hours. Prehospital cardioversion is not recommended.

Various agents are available for pharmacologic cardioversion of patients with stable atrial fibrillation in the ED, including the class IA, IC, and III antidysrhythmics (Box 79-9). In practice,
intravenous procainamide, amiodarone, and ibutilide are the agents most commonly used in ED setting. Although differences in success rates exist among agents, the overall response is 40 to 65% for drug-based ED cardioversion. The class IC antidysrhythmics are contraindicated in patients with structural or ischemic heart disease. For atrial fibrillation with accessory pathway conduction, procainamide is recommended because it has no effect on AV conduction.

For patients for whom electrical cardioversion is selected, adequate procedural sedation should be used. An initial attempt with 100 J (biphasic preferred) is usually adequate for cardioversion of atrial fibrillation, but occasionally a second attempt at 100 to 200 J is required.

Many patients with atrial fibrillation, whether frequent paroxysmal or permanent, benefit from long-term anticoagulation as prophylaxis against stroke. Although long-term anticoagulation typically falls beyond the scope of the ED, it is important that any patient for whom anticoagulation may be appropriate be referred for timely follow-up to address this issue.

Atrial fibrillation alone is not an indication for hospital admission. Observation or admission should be considered for symptomatic patients with atrial fibrillation that is complicated by significant underlying cardiopulmonary disease, in those in whom duration is unknown and follow-up uncertain, in those in whom cardioversion fails or the rhythm returns, and in all patients with concomitant new noncardiac illnesses.

**Atrial Flutter**

Atrial flutter is defined by atrial depolarization occurring at a regular rate of 250 to 350 beats/min (300 beats/min is classic) caused by an atrial reentry mechanism (Fig. 79-27). So-called flutter waves are distinguished by their broad, sawtooth appearance on the surface ECG. The ventricular rate in atrial flutter is often rapid, but in the absence of a bypass tract (in which 1:1 conduction is possible), the conduction ratio is limited by the refractory period of the AV node. With 2:1 conduction, the ventricular rate is approximately 150 beats/min, often making flutter waves themselves difficult to appreciate and allowing the rhythm to be mistaken for sinus tachycardia. Often the conduction ratio will change from beat to beat, called atrial flutter with variable conduction, with a resultant irregular ventricular rate; this is hard to distinguish from atrial fibrillation.

Atrial flutter can be associated with structural heart disease, particularly valvular heart disease and cardiomyopathies. The acute management of atrial flutter is similar to that of atrial fibrillation with a few special considerations. Because AV conduction occurs at fixed ratios in atrial flutter, the administration of beta-blocker or calcium channel blocker therapy can result in an abrupt rate change, making it more challenging to titrate therapy to a desired target rate. Atrial flutter is more sensitive to DC cardioversion (up to 90% conversion rate) than atrial fibrillation, and usually requires lower energy (20-50 J) for conversion to sinus rhythm.13 Conversely, atrial flutter is more resistant to chemical cardioversion (less than 50%) than new-onset nonvalvular atrial fibrillation.

**Atrioventricular Nodal Reentrant Tachycardia**

Also known by the less precise term paroxysmal supraventricular tachycardia (PSVT), AVNRT is characterized by a regular narrow-complex rhythm and a ventricular rate of 160 beats/min or greater (Fig. 79-28). It is the most common nonsinus tachydysrhythmia in young adults. As its name suggests, AVNRT occurs as the result of a reentry circuit within the AV node, with normal conduction (narrow QRS) down the bundles of His, and with retrograde conduction (inverted P waves typically buried within the QRS) up into the atria (Fig. 79-29).

The onset of AVNRT is typically abrupt and frequently arises in the context of strenuous exercise or emotional stress. Termination is also abrupt, either spontaneously or following vagal maneuvers that patients are taught or just adopt instinctively. Most patients with AVNRT are asymptomatic, but hemodynamic instability is unusual in the absence of underlying cardiopulmonary disease.

If vagal maneuvers fail to restore sinus rhythm, first-line field or ED therapy for AVNRT is adenosine (6 mg rapid large-bore intravenous bolus followed by a flush; repeat with 12 mg if no effect on rate). This approach is successful in 85 to 90% of cases. In refractory cases, diltiazem, esmolol, or metoprolol may be used. Rarely, synchronized cardioversion (at 100-200 J, synchronized and biphasic preferred) is required to terminate AVNRT that is refractory to pharmacologic therapy or in a patient with hemodynamic instability.

Most patients can be discharged once AVNRT has been terminated with adenosine or vagal maneuvers. Patients with frequent recurrences are candidates for prophylaxis (typically with a beta-blocker or calcium channel blocker) or ablation therapy.

**Junctional Tachycardia**

In contrast to the bursts seen in AVNRT, JTs (also known as nonparoxysmal or sustained junctional tachycardia) show sustained ventricular rates but rarely exceed 130 beats/min. JTs are commonly associated with structural heart disease, metabolic disturbances, or drug toxicity. Treatment is aimed at addressing underlying conditions, although a cautious trial of nodal blockade is not unreasonable.

**Preexcitation and Accessory Pathway Syndromes**

Preexcitation refers to depolarization of the ventricular myocardium by circumvention of the AV node via an accessory pathway or bypass tract linking the atria to the ventricles. Accessory pathways lend themselves to reentry tachycardias and very rapid ventricular rates. Wolff-Parkinson-White (WPW) syndrome is the classic accessory pathway syndrome, characterized by paroxysmal tachycardia and the following three resting ECG features (Fig. 79-30):
Figure 79-27. A, Atrial flutter with 2:1 conduction and isolated premature ventricular contraction. B, Atrial flutter with 2:1 conduction. Lead II helps identify characteristic flutter waves.

Continued
Figure 79-27, cont’d. C, Atrial flutter with 1:1 conduction. This is rare and can be mistaken for ventricular tachycardia (lead II).

Figure 79-28. Paroxysmal supraventricular tachycardia. Note the narrow, regular QRS complexes.
tachycardia is indistinguishable clinically from AVNRT unless the
arrhythmia syndrome associated with paroxysmal narrow-complex tachycardia, a short PR interval, and a normal QRS complex without a delta wave. The treatment parallels that for the WPW syndrome.

Wide-Complex Tachycardias

Wide-complex tachycardia refers to any tachydysrhythmia accompanied by a QRS duration of 0.12 second or more. Wide-complex tachycardias can derive from the ventricles themselves (i.e., VT) or can result from a tachycardia originating at or above the AV node accompanied by aberrant AV conduction, caused by either an accessory pathway or a preexisting or rate-related conduction delay, such as a bundle-branch block. The presence of AV dissociation or fusion beats on the 12-lead ECG clearly points to VT; all else being equal, older age and a prior history of myocardial infarction make VT much more likely than a supraventricular tachycardia with aberrancy. On the other hand, an irregular tachycardia with a wide QRS approximating a bundle-branch morphology is most likely atrial fibrillation with aberrant conduction. For stable patients, there are decision tools that can be used to distinguish VT with relatively good accuracy (Table 79-5 and Fig. 79-32).

In practice, it can be difficult to make the distinction between VT versus SVT and abnormal conduction when confronted with a patient displaying a wide-complex tachycardia. Many of the decision rules rely on an ability to discern subtleties of the QRS morphology, and none has perfect discriminatory power. As a general rule, because failure to treat VT appropriately can be disastrous for the patient, VT should be presumed initially to be VT. Hemodynamic stability does not help to exclude VT.

The Brugada ECG criteria search for four pieces of evidence of VT from among those listed previously; as soon as one is found,18,20,21 the diagnosis of VT is made. The rhythm needs to be regular for these to be used (chaos suggests atrial fibrillation with altered conduction). The sequential criteria are as follows (see Figs. 79-32 and 79-33):

1. Absence of any RS complexes in the chest leads
2. RS duration (measured from beginning of R to deepest part of S wave) greater than 100 msec
3. AV dissociation (often present but overlooked; may be best appreciated in inferior limb leads and V1; Fig. 79-34)
4. Specific VT morphologic criteria (see Fig. 79-33)

Only when none of the Brugada criteria are present is a supraventricular cause diagnosed. Although the original investigators found excellent sensitivity (98.7%) and specificity (96.5%) in detecting VT, follow-up investigations have not duplicated this level of accuracy in ED patients (sensitivity 92-94%). Often, physicians either cannot complete the assessment or cannot agree on the findings. In patients receiving class I agents, the Brugada criteria are less reliable.
QRS usually and V6 to identify SVT, then seeking AV dissociation in the remainder, first through classic RBBB or LBBB morphologies in V1 patients with both ischemic and nonischemic cardiomyopathy.

Unstable patients with a wide-complex tachycardia should be treated with cardioversion (100 J, synchronized and biphasic if possible). For stable patients in whom pharmacologic treatment is sought, procainamide or amiodarone are attractive choices because they can effectively convert both ventricular and supraventricular dysrhythmias. A diagnostic and therapeutic trial of adenosine is reasonable if no irregularity is noted and if the ventricular rate is less than 250 beats/min; this is done based on the facts that most SVTs will either slow or terminate with this intervention and that any ill effects of adenosine will be fleeting.

The Griffith criteria use a three-step approach to identify aberrancy, first through classic RBBB or LBBB morphologies in V1 and V6 to identify SVT, then seeking AV dissociation in the remainder to identify VT (see Figs. 79-32 and 79-34). This approach has a reported sensitivity of 92%, although specificity may be less than seen with the Brugada approach (though head-to-head in-practice comparative data do not exist).

Unstable patients with VT refractory to drug therapy should undergo intervention and that any ill effects of adenosine will be fleeting.

**Box 79-10**

**Diseases Associated with Wolff-Parkinson-White Syndrome**

- Idiopathic*
- Cardiomyopathy (especially hypertrophic)
- Transposition of great vessels
- Endocardial fibroelastosis
- Mitral valve prolapse
- Tricuspid atresia
- Ebstein's disease

*Most common.

**Table 79-5**

<table>
<thead>
<tr>
<th>Features Helpful in Distinguishing Ventricular Tachycardia from Supraventricular Tachycardia (VT) with Abnormal Conduction</th>
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<tr>
<td>VENTRICULAR TACHYCARDIA</td>
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<tr>
<td>Clinical features</td>
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<tr>
<td>History of myocardial infarction</td>
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<td>Previous history of ventricular tachycardia</td>
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<td>Physical examination</td>
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<tr>
<td>Variation in arterial pulse</td>
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<td>Variable first heart sound</td>
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<td>Electrocardiogram</td>
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<td>AV dissociation</td>
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<td>QRS &gt;0.14 s</td>
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<td>Extreme LAD artery (30 degrees)</td>
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<td>No response to vagal maneuvers</td>
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<td>Specific QRS patterns</td>
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<td>V2: S, rS, or qR</td>
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<tr>
<td>Identical to previous ventricular tachycardia tracing*</td>
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<td>Concordance of positivity or negativity*</td>
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**Table 79-5 footnotes:**

1. ASHD, arteriosclerotic heart disease; AV, atrioventricular; CABG, coronary artery bypass graft; LAD, left anterior descending; SVT, supraventricular tachycardia.
2. *If proven by electrophysiologic studies or by a preponderance of evidence.
3. †Main deflection of QRS complex either positive or negative in every precordial lead.
4. ‡Most common.

**Figure 79-31.** Wolff-Parkinson-White (WPW) paths and associated rhythms. AV, atrioventricular. (A, From Watanabe Y, Dreifus LS. Cardiac Dysrhythmias. New York, Grune & Stratton, 1977.)

**Ventricular Tachycardia**

VT originates within or below the His bundle. **Nonsustained** VT refers to short episodes (<30 seconds) that revert spontaneously, whereas **sustained** VT refers to more prolonged episodes. Reentry mechanisms are the most common cause of VT, although automatic and triggered mechanisms also occur. Most patients with VT have underlying heart disease.

**Monomorphic ventricular tachycardia** is the most common form of VT and is characterized by morphologically consistent QRS complexes, usually in a regular pattern and at a rate of 150 to 200 beats/min (Fig. 79-35). **Polymorphic ventricular tachycardia** is seen with varying QRS morphologies and suggests more severe underlying disease (Figs. 79-36 and 79-37). VT is prevalent in patients with both ischemic and nonischemic cardiomyopathy.

For stable patients with VT, amiodarone (3-5 mg/kg IV over minutes) or lidocaine (1.0-1.5 mg/kg IV bolus, up to 3 mg/kg maximum and followed by an infusion) are first-line choices in the field or ED, with successful termination of up to 90%. Procainamide (30-50 mg/min IV up to a total of 18 mg/kg or until VT is terminated) is a second-line agent. Unstable patients or those with VT refractory to drug therapy should undergo...
### Figure 79-32

**A.** Brugada four-step approach for differentiating ventricular tachycardia (VT) and wide-QRS supraventricular tachycardia (SVT). Only when the response to all four questions is negative is a supraventricular rhythm with abnormal conduction diagnosed. As soon as a single “yes” answer is noted, VT is diagnosed. **B.** Griffith approach, in which aberrant conduction is sought in leads V₁ and V₆ (right bundle branch block [RBBB] or left bundle branch block [LBBB] classic appearances), followed by a search for atrioventricular (AV) dissociation. If classic RBBB and LBBB patterns are absent, or if the remainder of leads have no AV dissociation, VT is diagnosed. (A, From Brugada P, et al: A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation 83:1649, 1991. A, Redrawn from Griffith MJ, et al: Ventricular tachycardia as default diagnosis in broad complex tachycardia. Lancet 343:386, 1994.)

### Figure 79-33

The morphology associated with the fourth criterion in the Brugada system: **A,** in patients with a right bundle branch–appearing complex; **B,** in patients with a left bundle branch–appearing complex.
Figure 79-34. A and B, Ventricular tachycardia. Note atrioventricular dissociation. C, Intermittent, nonsustained ventricular tachycardia. Atrioventricular dissociation is evident. (Courtesy Edward Curtis, MD.)
Figure 79-35. Ventricular tachycardia. A, RS complexes are present in chest leads, but RS duration is greater than 100 msec. Although the Brugada criteria indicate that no further analysis is necessary, atrioventricular dissociation is also evident, and QRS morphology in lead V₆ is consistent with ventricular tachycardia. B, Some RS complexes are present, RS duration is not greater than 100 msec, and atrioventricular dissociation is difficult to appreciate; morphologic criteria for ventricular tachycardia are fulfilled because S is notched in V₁ and QR is present in V₆. C, Diagnosis is based on morphologic criteria because S is notched in V₁ and V₂ and QS is present in V₆. (Courtesy Edward Curtis, MD.)
synchronized cardioversion with 100 J (biphasic preferred), with escalating doses (up to 200 J biphasic or 360 J monophasic) as needed.

All patients with new or symptomatic VT should be admitted, the exception being those who have implanted defibrillators that are functioning appropriately (see Chapter 80).

**Torsades de Pointes**

*Torsades de pointes* is literally translated as “twisting of the points” and is a paroxysmal form of polymorphic VT that meets the following clinical criteria (see Fig. 79-37):

1. Ventricular rate greater than 200 beats/min
2. Undulating QRS axis, with the polarity of the complexes appearing to shift about the baseline
3. Paroxysms of less than 90 seconds

Torsades de pointes occurs in the setting of a prolonged QT interval, a reflection of abnormal ventricular repolarization. A prolonged QT interval can be congenital or acquired. Women are at a greater risk for torsades de pointes. Acquired torsades de pointes is much more common than congenital and is termed “pause dependent,” triggered by a slow heart rate.

Acquired QT prolongation is often multifactorial (Box 79-11). Common triggers include electrolyte disturbances (hypokalemia and hypomagnesemia) and many different drugs (notably class IA and IC agents but also many others; see Box 79-11), especially when used in combination. Treatment of torsades de pointes in

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**Classification and Causes of Prolonged QT Syndromes That Produce Torsades de Pointes**

<table>
<thead>
<tr>
<th><strong>Pause Dependent (Acquired)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug induced: Class IA and IC antidysrhythmics; many phenothiazines and butyrophenones (notably haloperidol and droperidol), cyclic antidepressants, antibiotics (especially macrolides), organophosphates, antihistamines, antifungals, antiseizure and antiemetic agents</td>
</tr>
<tr>
<td>Electrolyte abnormalities: hypokalemia, hypomagnesemia, hypocalcemia (rarely)</td>
</tr>
<tr>
<td>Diet related: starvation, low protein</td>
</tr>
<tr>
<td>Severe bradycardia or atrioventricular block</td>
</tr>
<tr>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Contrast injection</td>
</tr>
<tr>
<td>Cerebrovascular accident (especially intraparenchymal)</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Adrenergic Dependent (Tachycardia Prompted)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congenital</strong></td>
</tr>
<tr>
<td>Jervell and Lange-Nielsen syndrome (deafness, autosomal recessive)</td>
</tr>
<tr>
<td>Romano-Ward syndrome (normal hearing, autosomal dominant)</td>
</tr>
<tr>
<td>Sporadic (normal hearing, no familial tendency)</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
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<table>
<thead>
<tr>
<th><strong>Acquired (Rare)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrovascular disease (especially subarachnoid hemorrhage)</td>
</tr>
<tr>
<td>Autonomic surgery: radical neck dissection, carotid endarterectomy, truncal vagotomy</td>
</tr>
</tbody>
</table>
Brugada’s Syndrome

Brugada’s syndrome is also associated with ventricular dysrhythmias and syncope or sudden cardiac death in the absence of structural heart disease. Brugada’s syndrome results from an inherited disorder of sodium channels and is most commonly present in males during young adulthood. The Brugada syndrome ECG pattern is characterized by a downward coved or humped (saddleback) ST segment elevation in leads V1 to V3 (Fig. 79-38), sometimes simulating an RBBB appearance. The ST segment findings may be transient or elicited only with pharmacologic stimulation.
Any patient with unexplained syncope and a Brugada-pattern ECG requires admission for consideration of an implanted defibrillator. For patients in whom a Brugada-pattern ECG is noted incidentally, there is no consensus on treatment, but referral to a cardiologist is advisable.

### KEY CONCEPTS

- Electrical therapy is used for any unstable patient in whom the rhythm is the cause of symptoms—pacing if the heart rate is slow, countershock with sedation if fast.
- A regular, new-onset, symptomatic wide-complex tachycardia should be assumed to be VT until proven otherwise.
- Type II second-degree AV block is never a normal variant and implies a conduction block below the AV node. When the conduction ratio is 2:1, type II block should be assumed until proven otherwise.
- The possibility of an accessory pathway should be considered in any tachycardia exceeding a rate of 200 to 250, regardless of the QRS complex morphology, and nodal blocking agents should not be used without careful consideration.
- One should look closely for irregularity in tachycardia over 200 beats/min; this and underlying atrial fibrillation can be missed if R-R intervals at fast rates are not carefully tracked.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.
References