Chapter 18: Cardiac Rhythm Disturbances

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Content Update

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Atrial Fibrillation and Flutter, Disposition: Many patients with atrial fibrillation/flutter that receive rate control or rhythm conversion in the emergency department do not need hospital admission and can be safely discharged from the ED with follow-up arranged. While there is significant institutional and regional variation in disposition practices, we have provided a summary of reasonable practice indicating criteria for discharge and admission and options for outpatient anticoagulation.

GENERAL CONSIDERATIONS

INITIAL APPROACH TO THE STABLE PATIENT

The focused evaluation of the patient includes determining the presenting complaint(s), obtaining the medical history, identifying medication use, performing a physical examination, initiating continuous cardiac rhythm monitoring, reviewing the 12-lead ECG, and analyzing the cardiac rhythm on the rhythm monitor, a printer strip, or the ECG.

Presenting symptoms may include palpitations, lightheadedness, fatigue, or weakness. Ischemic symptoms, such as chest pain, nausea, dyspnea, or lightheadedness, may be due to dysrhythmia-induced ischemia.

The medication history includes prescribed medications, herbals, recreational drugs, and caffeine-containing beverages. Especially note recently started new medications or increased medication doses. Symptoms of hyperthyroidism should be sought. Patients with a family history of sudden death, syncope, or dysrhythmias and those with organic heart disease have a higher risk of cardiac dysrhythmias and complications. Panic or anxiety is a diagnosis of exclusion in tachycardic ED patients.

INITIAL APPROACH TO THE UNSTABLE PATIENT

An unstable patient needs rapid assessment and treatment to prevent cardiovascular collapse. Instability means that the dysrhythmia is (1) impairing cardiac output and threatening vital organ function or (2) has the potential to suddenly deteriorate into cardiac arrest (**Table 18-1**).[1] Establish an IV line, initiate cardiac rhythm monitoring, obtain an ECG, and be prepared for drug or electrical therapy.

Table 18–1

Instability Indicators in the Patient with Cardiac Dysrhythmias

Hypotension: e.g., systolic blood pressure <90 mm Hg (<12 kPa)
Systemic hypoperfusion
Altered mentation
Ischemic chest pain
Respiratory distress
Extremely rapid ventricular rate: e.g., rate over 200 beats/min in adult

Dysrhythmia-induced chest pain results from coronary hypoperfusion, and dyspnea results from pulmonary edema, usually with objective evidence: ST segment abnormalities, rales on examination, or low oxygen saturation. As the ventricular rate exceeds 200 beats/min, severe systemic hypoperfusion often results and the RR interval narrows proportionally, increasing the opportunity for malignant ventricular dysrhythmias.

GENERAL APPROACH TO BRADYDYSRHYTHMIAS

Bradydysrhythmia describes rhythms with a ventricular rate slower than 60 beats/min in the adult. Age-appropriate heart rates define pediatric bradydysrhythmia. Bradydysrhythmias can be broadly categorized as bradycardias (atria and ventricles beat at the same slow rate) and atrioventricular (AV) blocks (ventricles beat slower than the atria).[1] The bradycardias include sinus bradycardia, junctional rhythm, idioventricular rhythm, and hyperkalemia-related sinoventricular rhythm. Bradydysrhythmias due to AV blocks include second-degree (usually type II) and third-degree AV block, as well as atrial fibrillation and atrial flutter with a slow ventricular response.

The most common bradycardia is sinus bradycardia, followed by junctional rhythm, and less commonly idioventricular rhythm. These dysrhythmias are found in both stable and unstable patients. Atrial fibrillation and flutter with slow ventricular response are uncommon. If the patient is unstable, the vast majority of AV blocks are third-degree heart block followed much less frequently by second-degree AV block. If the patient is stable, second-degree type I AV block is most frequently seen, third-degree AV block is less common, whereas second-degree type II AV block is quite rare.[2-4]

Bradydysrhythmias result from conditions that affect the automaticity and refractoriness of cardiac cells as well as the conduction of impulses within the cardiac electrical system.[1,4] About 80% of bradydysrhythmias are caused by factors external to the cardiac electrical system including acute coronary syndrome, drug effects or overdose, and hypoxia with cardiac hypoperfusion.[2,3]

Emergent treatment of bradydysrhythmia is not required unless (1) the heart rate is slower than 50 beats/min accompanied by hypotension or hypoperfusion and/or (2) the bradydysrhythmia is due to structural disease of the infranodal conduction system. This first group requires resuscitative treatment while evaluating the cause. The second group of patients does not require immediate treatment but should be closely monitored, with pacing readily available while arranging definitive care.

Medications used to increase heart rate in symptomatic bradycardias include atropine, β-adrenergic agonists, and glucagon (**Table 18-2**; see chapter 19, "Pharmacology of Antiarrhythmics and Antihypertensives").[1,4] Atropine enhances the automaticity of the sinoatrial (SA) node and potentiates conduction through the AV node by direct vagolytic activity. Atropine is usually effective for sinus bradycardia and junctional rhythms but is not useful (nor particularly harmful) in idioventricular

rhythms and second-degree type II and third-degree AV block.[2,3] β-Adrenergic agents stimulate both chronotropic and inotropic cardiac activity, as well as enhancing electrical conduction within the AV node and infranodal system, thus their potential to produce ischemia and ectopy. Glucagon stimulates inotropic and chronotropic cardiac activity independent of the β-adrenergic receptors. Glucagon is primarily used for bradycardias due to cardiotoxicity from β-blocker or calcium channel blocker overdose. Effectiveness of drug treatment for bradycardia varies, and in general, these agents are best used as a temporary bridge to cardiac pacing.

Table 18–2

Drug Treatment for Cardiac Dysrhythmias in Adults

Drugs for Bradydysrhythmias		
Atropine	0.5-milligram IV push, may repeat every 3–5 min until desired heart rate is achieved or to total dose of 3 milligrams (0.04 milligram/kg)	Most effective for bradydysrhythmias due to sinus and AV nodal disease
Dopamine	IV infusion at rate 2–20 micrograms/kg per min, titrate to desired heart rate	May precipitate myocardial ischemia and ectopy
Epinephrine	IV infusion at rate 2–10 micrograms/min, titrate to desired heart rate	May precipitate myocardial ischemia and ectopy
Glucagon	3–10 milligrams IV infused over 1–2 min, followed by an IV continuous infusion of 1–5 milligrams/h	Used for cardiotoxicity associated with β-blocker and calcium channel blocker overdose Nausea and vomiting are often limiting side effects Tachyphylaxis may develop during infusion
Drugs to Block AV Nodal Conduction		
Adenosine	6-milligram rapid IV push; if after 2 min the dysrhythmia persists, repeat rapid IV push with 12 milligrams; may repeat once more if dysrhythmia persists	Effective in terminating narrow QRS complex reentrant tachydysrhythmias involving the AV node
Verapamil	2.5–5 milligrams IV bolus over 2–3 min; if after 15 min the dysrhythmia persists, may repeat with dose of 5–10 milligrams	Effective in terminating narrow QRS complex reentrant tachydysrhythmias involving the AV node and reducing ventricular rate in atrial fibrillation or flutter
Diltiazem	15–20 milligrams IV bolus over 2 min, followed by IV infusion at 5–10 milligrams/h	
Esmolol	500 micrograms/kg IV bolus over 1 min, followed by IV infusion starting at 50 micrograms/kg per min; titrate infusion to desired heart rate	
Metoprolol	5-milligram IV bolus; may repeat 5 milligrams IV every 5 min up to total dose of 15 milligrams	
Propranolol	30 micrograms/kg IV over 1 min; may repeat same dose every 2 min, up to total dose of 100 micrograms/kg	
Drugs to Terminate Tachydysrhythmias		
Procainamide	15–17 milligrams/kg IV over 30 min, followed by IV infusion at 1–4 milligrams (20–80 micrograms/kg) per min; or 20-50 milligrams/min; or 100 milligrams IV q 5 min	Used in wide-complex tachydysrhythmias and new-onset atrial fibrillation Median time to conversion of new-onset atrial fibrillation about 1 h

		Caution in patients with AMI and LV dysfunction Infuse initial dose at rate of 20 milligrams/min to reduce adverse effects
Amiodarone	Stable patient: 150 milligrams IV over 10 min; may repeat same dose every 10 min up to total dose of 2 grams OR use IV infusion 0.5 milligram/min Ventricular fibrillation or pulseless ventricular tachycardia: 300 milligrams IV bolus; may repeat with additional dose of 150 milligrams IV bolus	Used in wide-complex tachydysrhythmias and new-onset atrial fibrillation Preferred in setting of AMI or LV dysfunction Contraindicated in pregnancy
Lidocaine	1 milligram/kg IV over 60 s; may repeat 0.5 milligram/kg IV every 5–10 min, up to 300 milligrams in a 1 h period; followed by infusion of 1–4 milligrams/min	Third-line agent for ventricular tachycardia and ventricular fibrillation
Magnesium sulfate	2 grams IV over 2 min, followed by infusion of 1–2 grams/h	Used in torsades de pointes with long QT interval
Ibutilide	Weight <60 kg: 10 micrograms/kg IV over 10 min Weight >60 kg: 1 milligram IV over 10 min	Used for conversion of new-onset atrial fibrillation or flutter Median time to conversion 20–30 min
Flecainide	200 milligrams PO < 70 kg 300 milligrams PO > 70 kg; or 2 milligrams/kg IV over 10 min*	Used for conversion of new-onset atrial fibrillation or flutter Median time to conversion up to 4 h Avoid in patients with ACS or cardiomyopathy
Propafenone	450 milligrams PO < 70 kg 600 milligrams PO > 70 kg; or 2 milligrams IV over 10 min*	Used for conversion of new-onset atrial fibrillation or flutter Median time to conversion 2 h Avoid in patients with ACS, cardiomyopathy, or severe COPD
Vernakalant*	3 milligrams/kg IV infusion over 10 min; if dysrhythmia persists after 15 min, a second infusion of 2 milligrams/kg IV over 10 min can be given	Used for conversion of new-onset atrial fibrillation or flutter Median time to conversion 8–11 min Avoid in patients with hypotension, ACS within 30 days, severe aortic stenosis, and prolonged QT interval

Abbreviations: ACS = acute coronary syndrome; AMI = acute myocardial infarction; AV = atrioventricular; COPD = chronic obstructive pulmonary disease; LV = left ventricle.

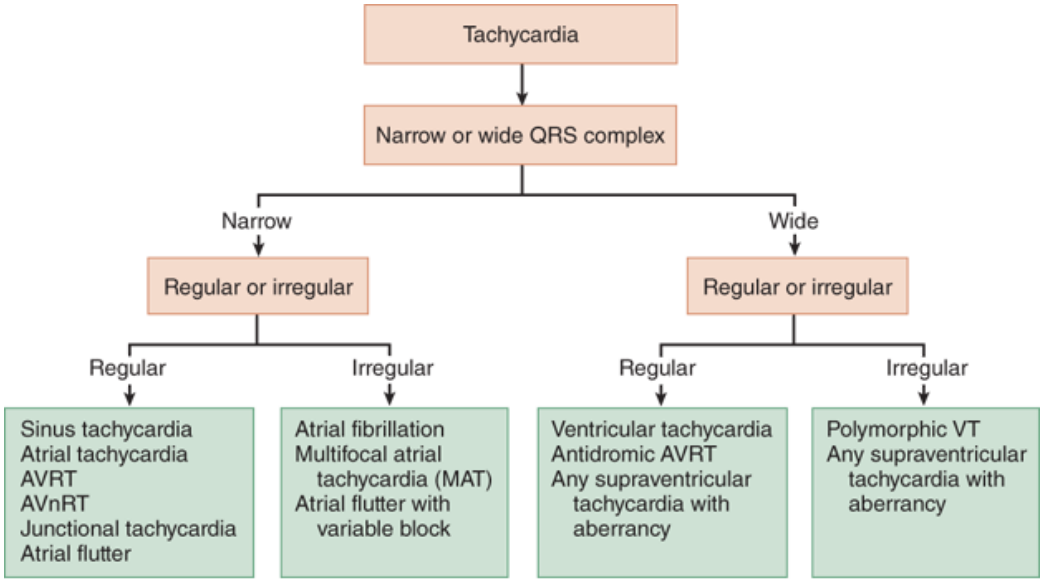
* Not available in the United States.

Transcutaneous pacing can be applied quickly and is the most appropriate pacing method for the acutely symptomatic patient (see chapter 33, "Cardiac Pacing and Implanted Defibrillation"). Transvenous pacing requires considerable physician expertise and specialized equipment for insertion and proper placement. Disease-specific therapies can also be effective in reversing a toxin-induced bradycardia (e.g., treatment of hyperkalemia or toxicity from calcium channel blockers, β-blockers, or digitalis).

GENERAL APPROACH TO TACHYDYSRHYTHMIAS

Tachydysrhythmia describes rhythms with a ventricular rate greater than 100 beats/min in an adult, with age-appropriate limits in children. Tachycardias are categorized as supraventricular or ventricular (**Figure 18–1**). Supraventricular tachycardias originate from a focus within or above the AV node and most often present with a narrow QRS complex; thus, they are termed *narrow-complex tachycardias*. Ventricular tachycardias, resulting from a focus below the AV node in the ventricular myocardium, usually demonstrate a widened QRS complex and are referred to as *wide-complex tachycardias*. This classification scheme does have limitations; a supraventricular rhythm can present with a widened QRS complex due to aberrant ventricular conduction, the widened QRS complex resulting from a fixed (i.e., preexisting) bundle-branch block, rate-related conduction block, ventricular preexcitation syndrome (i.e., Wolff-Parkinson-White [WPW] syndrome), or toxic-metabolic condition.[5] The normal QRS complex duration is less than 120 milliseconds for older children and adults; therefore, a wide-complex tachycardia possesses a QRS complex width greater than 120 milliseconds.

FIGURE 18–1.
Tachycardia classification. AVnRT = atrioventricular nodal reentrant tachycardia; AVRT = atrioventricular reentrant tachycardia; VT = ventricular tachycardia.



Common narrow-complex tachycardias include sinus tachycardia, atrial fibrillation, atrial flutter, and paroxysmal supraventricular tachycardia; less common narrow-complex tachycardias include multifocal atrial tachycardia, atrial tachycardias, and preexcited tachycardias seen in accessory pathway syndromes including WPW syndrome.[6] Wide-complex tachycardias include ventricular tachycardia and supraventricular tachycardia with aberrant conduction.[5,7] Ventricular tachycardia further is subdivided into monomorphic and polymorphic forms; the polymorphic category includes the subtype called *torsade de pointes*.[8]

Treatment for symptomatic tachycardia is primarily intravenous medications for the stable patient and electrical therapy for the unstable patient (see chapter 23, "Defibrillation and Cardioversion"). The QRS width, often indicating the portion of the heart where the dysrhythmia originates, guides therapeutic choices.

Narrow-Complex Tachycardia

Sinus tachycardia and multifocal atrial tachycardia are best managed by treating the underlying cause, rather than the dysrhythmia specifically. Other narrow-complex tachycardias require specific antidysrhythmic treatment by a combination of

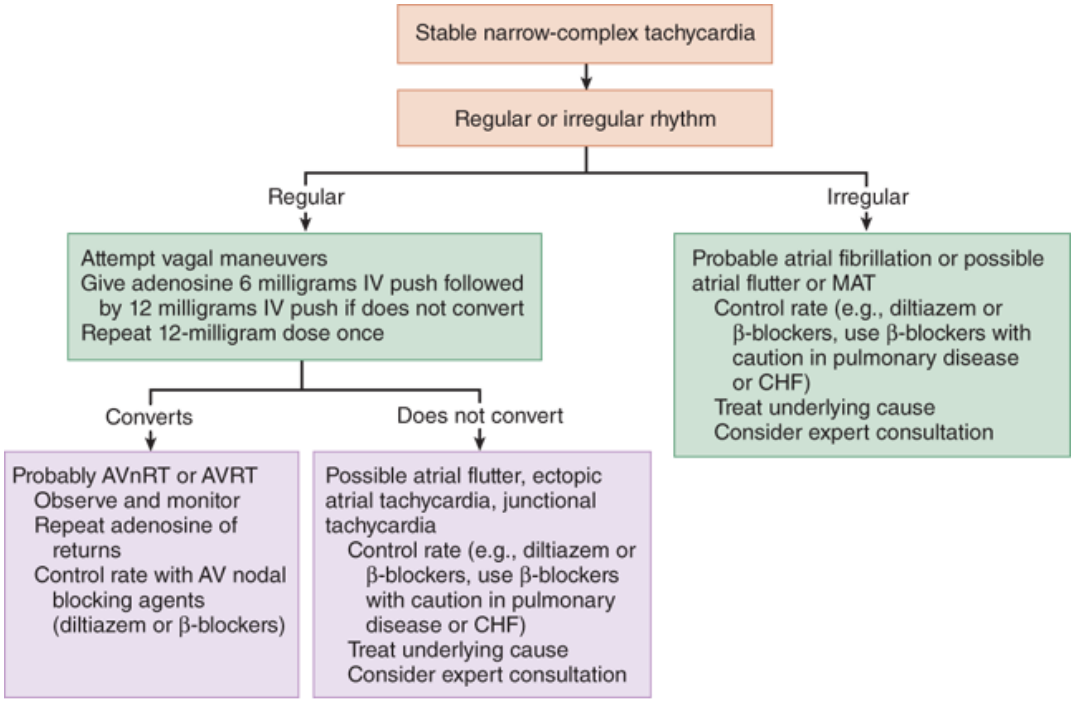
vagal maneuvers (**Table 18-3**), medications (Table 18-2), and electrical cardioversion (**Figure 18–2**).[9] Basic supportive therapy in most patients involves an IV fluid bolus to expand the circulating intravascular volume and supplemental oxygen.

Table 18–3

Vagal Maneuvers

Carotid sinus massage	Listen for bruit first; do not massage an artery with a bruit Massage only one side at a time Massage for 20 s or less
Valsalva maneuver	Have patient hold breath and strain against closed glottis while tightening abdominal wall muscles Hold for as long as practical, ideally >20 s Increased vagal tone seen during release phase after breath hold
Diving reflex	More effective in infants than adults Place bag of ice and water on face for 15–30 s

FIGURE 18–2.
Treatment of narrow-complex tachycardia. AV = atrioventricular; AVnRT = atrioventricular nodal reentrant tachycardia; AVRT = atrioventricular reentrant tachycardia; CHF = congestive heart failure; MAT = multifocal atrial tachycardia; VT = ventricular tachycardia.



Vagal maneuvers heighten parasympathetic tone and may slow electrical conduction in the heart to a degree that abolishes sustained reentry. If applied early, vagal maneuvers can convert about 20% of patients presenting with reentrant tachycardias, such as paroxysmal supraventricular tachycardia and narrow-complex tachycardia associated with WPW syndrome. Effective vagal maneuvers include carotid sinus massage, the release phase of the Valsalva maneuver, and the diving reflex; the response to these vagal maneuvers is enhanced by placing the patient supine.[10-12]

Adenosine is a very-short-acting agent that blocks conduction through the AV node and can interrupt sustained reentry when the AV node is part of the circuit (Table 18-2). The AV nodal blocking effect of adenosine is very transient, although quite profound, so a brief period of AV nodal blockade with near-immediate recurrence of the reentrant supraventricular tachycardia is not a treatment failure but a consequence of the medication's short duration of effect. In such situations, repeat adenosine with a higher dose (12 milligrams).[13]

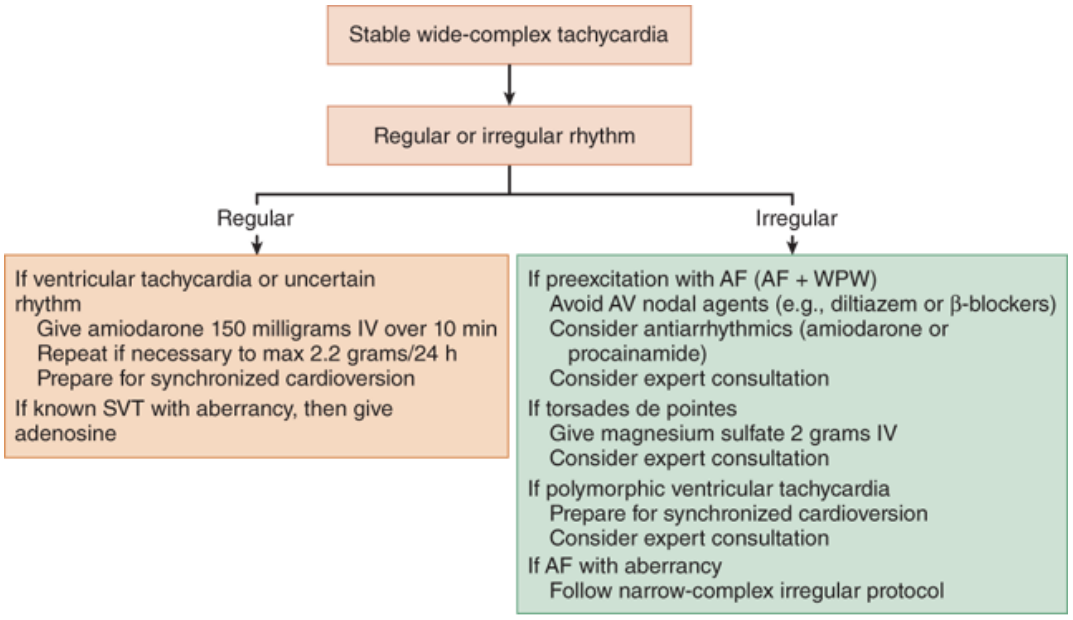
β-Blockers and calcium channel blockers slow conduction through the AV node and can convert some supraventricular tachycardias, such as reentrant supraventricular tachycardias, and slow the ventricular response in others, such as atrial fibrillation or flutter. Esmolol, an intravenous β-blocker with a short duration of effect, can be used when temporary AV nodal blockade is desired and a longer period of action is not anticipated (as in conversion of paroxysmal supraventricular tachycardia) or when the patient is unstable and the ability to titrate the degree of drug effect is important. Metoprolol is a longer acting β-blocker used in more stable patients, typically for ventricular rate control in patients with atrial fibrillation. Verapamil is a calcium channel blocker used for conversion of reentrant supraventricular tachycardias, and although it can be used for ventricular rate control, there is potential for hypotension, so diltiazem, which does not have as much potential to induce hypotension, is the calcium channel blocker recommended for ventricular rate control.

Synchronized electrical cardioversion can be used in narrow-complex tachycardias when patients are unstable or do not respond to pharmacologic measures (see chapter 23).

Wide-Complex Tachycardia

In the stable patient, pharmacologic agents used to terminate a wide-complex tachycardia include procainamide, amiodarone, lidocaine, and magnesium (Table 18-2 and **Figure 18–3**). **For rapid treatment, amiodarone is the antiarrhythmic of choice, given as an IV bolus**. Procainamide is effective for stable ventricular tachycardia in patients with preserved left ventricular dysfunction, given as an IV infusion.[14] Lidocaine is a less effective alternative.[14] Magnesium is used for tachydysrhythmias associated with QT interval prolongation, such as torsade de pointes.

FIGURE 18–3.
Treatment of wide-complex tachycardia. AF = atrial fibrillation; AV = atrioventricular; SVT = supraventricular tachycardia; WPW = Wolff-Parkinson-White syndrome.



Electrical cardioversion is the preferred treatment for wide-complex tachycardia with hemodynamic instability, myocardial ischemia, or failure of pharmacologic treatment (see chapter 23).[7,8]

NON-TACHYCARDIC IRREGULAR DYSRHYTHMIAS

SINUS ARRHYTHMIA

Description

While some variation in the rate of sinus (or SA) node electrical discharge is normal, sinus arrhythmia is present when the variation in the SA node discharge rate is greater than 120 milliseconds between the longest and shortest P to P wave intervals (**Figure 18–4**). There should be a consistent P-wave morphology indicating that the electrical impulses are all originating from the same atrial pacemaker, usually the SA node (**Table 18-4**). Two or more different P wave morphologies suggest atrial ectopy, wandering atrial pacemaker, or other nonsinus focus.

FIGURE 18–4.
Sinus arrhythmia.

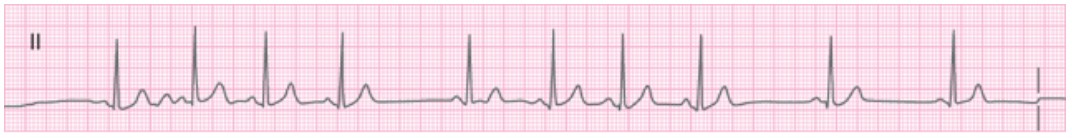


Table 18–4

ECG Features of Sinus Arrhythmia

Upright P waves in leads I, II, and III
Consistent P wave–QRS complex relationship
Consistent P-wave morphology from beat-to-beat
Variation in the sinus node discharge rate >120 ms between longest and shortest P to P wave intervals

Clinical Significance

Sinus arrhythmia is a normal finding in children and young adults but is less common in the middle-aged and elderly. Sinus arrhythmia is most commonly a respiro-phasic phenomenon; the sinus node rate accelerates during inspiration and decelerates during expiration. This variation is thought to be due to changes in vagal tone occurring with respiration, termed *the Bainbridge reflex*. Any condition or medication that alters vagal tone may exaggerate an underlying sinus arrhythmia. During long intervals of sinus arrhythmia, junctional escape beats may occur.

Treatment

None is required.

SINOATRIAL BLOCK

Description

The SA node electrical discharge must be conducted into the atria to pace the heart during sinus rhythm. If sinus node discharges are delayed or blocked in their outward propagation (exit block), then SA block is present. SA block is divided into first-, second-, and third-degree varieties, much like the classification system used for AV nodal blockade.

In **first-degree SA block,** the impulse is delayed in its conduction out of the sinus node into the atria, a condition that cannot be recognized on the clinical 12-lead ECG; this entity is diagnosed in the electrophysiology laboratory. In **second-degree SA block,** some impulses get through and some are blocked. **Second-degree SA block** can be suspected whenever an expected P wave and the corresponding QRS complex are absent. Usually, the interval between normal P waves encompassing the missing beat is a simple multiple of the existing P to P rate. **Third-degree SA block** occurs when the sinus node discharge is completely blocked and no P wave originating from the sinus is seen. In addition to third-degree SA block, absence of a P wave may also be caused by (1) sinus node failure, (2) a sinus node stimulus inadequate to activate the atria, and (3) atrial unresponsiveness.

Clinical Significance

SA block usually arises from myocardial disease (acute rheumatic fever, acute inferior MI, or other causes of myocarditis) or drug toxicity (digoxin, quinidine, salicylates, β-blockers, or calcium channel blockers). In rare individuals, vagal stimulation can produce SA block.

Treatment

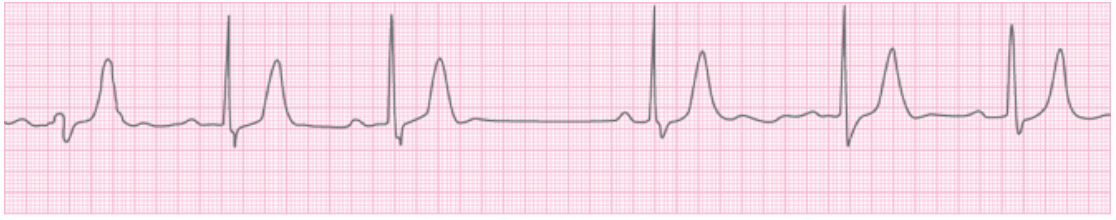
Treatment depends on the underlying cause, associated dysrhythmias, and whether symptoms of hypoperfusion are present. Sinus node discharge rate and SA conduction can be facilitated by atropine when clinically required; however, ischemia may result from a rhythm that is accelerated. Cardiac pacing is indicated for recurrent or persistent symptomatic bradycardia.

SINUS ARREST (PAUSE)

Description

Sinus pause is a failure of impulse formation within the sinus node. In sinus arrest, the P to P wave interval encompassing the missing beat has no relation to the underlying SA node discharge rate (**Figure 18–5**).

FIGURE 18–5.
Sinus pause.



Clinical Significance

The same conditions that produce SA block produce sinus arrest, especially digoxin toxicity and aging disease of the SA node. Brief periods of sinus arrest may occur in healthy individuals due to increased vagal tone. If sinus arrest is prolonged, AV junctional escape beats often occur.

Treatment

Treatment is often unneeded and when considered depends on the underlying cause, associated dysrhythmias, and whether symptoms of hypoperfusion are present. If sinus arrest is symptomatic, atropine usually will increase the SA node discharge rate. Cardiac pacing is indicated for recurrent or persistent symptomatic bradycardia.

SICK SINUS SYNDROME

Description

Sick sinus syndrome (sometimes referred to as the *tachycardia-bradycardia syndrome*) is a heterogeneous disorder consisting of abnormalities of supraventricular impulse generation and conduction that produce a wide variety of intermittent supraventricular tachy- and bradydysrhythmias (**Table 18-5**). The tachydysrhythmias are usually atrial fibrillation, junctional tachycardia, paroxysmal supraventricular tachycardia, and atrial flutter. The bradydysrhythmias are marked sinus bradycardia, prolonged sinus arrest, and SA block, usually associated with AV nodal conduction abnormalities and inadequate junctional escape rhythms.

Table 18–5

ECG Features of Sick Sinus Syndrome

Intermittent combination of bradydysrhythmias and tachydysrhythmias
Bradydysrhythmias
Sinus bradycardia
Sinus arrest
SA block
Tachydysrhythymias
Atrial fibrillation
Atrial flutter
Paroxysmal supraventricular tachycardia

Abbreviation: SA = sinoatrial.

Clinical Significance

Sick sinus syndrome is most commonly seen in elderly patients and is associated with a variety of cardiac diseases that can affect the SA and AV nodes, including ischemic disorders, myocarditis and pericarditis, rheumatologic disease, metastatic tumors, surgical damage, or cardiomyopathies.

Symptoms of sick sinus syndrome are due to the effects of fast or slow heart rate. Common symptoms include syncope or near-syncope, palpitations, dyspnea, chest pain, and cerebrovascular ischemic events. Conditions that increase vagal tone (abdominal pain, increased intracranial pressure), thyrotoxicosis, and hyperkalemia may exacerbate the abnormalities of sick sinus syndrome and increase symptoms. Medications such as digoxin, quinidine, procainamide, disopyramide, nicotine, β-blockers, and calcium channel blockers also can increase symptoms.

Ambulatory ECG monitoring or electrophysiologic studies are usually necessary for diagnosis, because intermittent dysrhythmias may not be evident during the examination.

Treatment

By definition, symptomatic patients will have both tachy- and bradydysrhythmias, and each of the elements may need to be addressed. Aggressive pharmacologic treatments and ablation procedures to reduce tachycardia carry risks of worsening bradycardia, AV block, and pauses. Transcutaneous pacing may be required if the patient is being pharmacologically treated for atrial tachydysrhythmias in the setting of sick sinus syndrome. Permanent pacemaker implantation is frequently indicated.

PREMATURE ATRIAL CONTRACTIONS

Description

Premature atrial contractions originate from ectopic pacemakers anywhere in the atrium other than the SA node (**Figure 18–6**) with characteristic ECG features (**Table 18-6**). The ectopic P wave may not be conducted through the AV node if the premature atrial contraction reaches the AV node during the absolute refractory period. When a premature atrial contraction reaches the AV node during the relative refractory period, it may be conducted with a delay, as demonstrated on the ECG by a longer PR interval than seen with a sinus beat. Nonconducted premature atrial contractions are the most common cause of pauses in cardiac rhythm. Premature atrial contractions may occur in a pattern, such as every other beat (atrial bigeminy), every third beat (atrial trigeminy), and so on.

FIGURE 18–6.
Premature atrial contractions in an atrial trigeminy pattern.

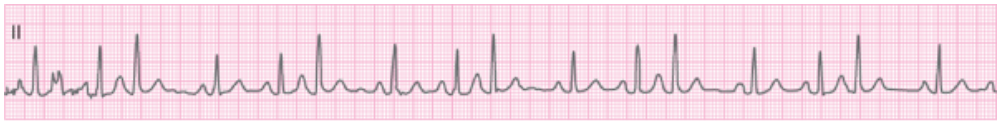


Table 18–6

ECG Features of Premature Atrial Contractions

P waves that appear sooner (prematurely) than expected sinus beat
Ectopic P waves
With different shape and axis than the SA node–initiated P wave
That may or may not be conducted through the AV node
Interval between normal P waves encompassing the premature atrial contraction is less than twice the existing P to P cycle length (a noncompensatory pause)

Abbreviations: AV = atrioventricular; SA = sinoatrial.

Most premature atrial contractions are conducted with baseline QRS complexes, but some may be conducted aberrantly through the infranodal system, particularly if they reach a bundle branch during the refractory period. The premature atrial contraction will often depolarize the SA node ("reset"), so the interval between normal P waves encompassing the premature atrial contraction will not be twice the existing P to P interval, creating a shorter pause than the fully compensatory pauses seen after most premature ventricular contractions.

Clinical Significance

Premature atrial contractions are common at all ages and usually do not indicate underlying heart disease. Increased rates of premature atrial contractions are seen in patients with chronic heart or lung disease. Chemical agents that enhance either sympathetic tone (e.g., cocaine, amphetamines, caffeine, nicotine) or parasympathetic tone (e.g., digoxin) may lead to a higher frequency of premature atrial contractions in an individual patient. Premature atrial contractions can precipitate sustained atrial tachycardia, flutter, or fibrillation under certain circumstances.

Treatment

No specific treatment is necessary. If the premature atrial contractions are symptomatic, discontinue any precipitating toxins and treat any underlying disorder that is contributing to symptoms.

PREMATURE JUNCTIONAL CONTRACTIONS

Description

Premature junctional contractions are due to an ectopic pacemaker within the AV node or common AV bundle with characteristic ECG features (**Table 18-7**).

Table 18–7

ECG Features of Premature Junctional Contractions

Ectopic P wave
With different shape, axis, and amplitude from SA node–initiated P waves
That may occur before or after QRS complex
QRS complex with similar morphology to SA node–initiated QRS complex

Abbreviation: SA = sinoatrial.

Because the premature junctional contraction is usually conducted back into the atria, the SA node is usually affected by the ectopic depolarization, and the postectopic pause is noncompensatory, but a compensatory pause is seen if the premature junctional contraction is not conducted in retrograde fashion. Premature junctional contraction may be isolated, multiple (as in bigeminy or trigeminy), or multifocal.

Clinical Significance

Premature junctional contractions are uncommon in healthy hearts and are typically seen in patients with heart failure, digitalis toxicity, ischemic heart disease, and myocardial ischemia (especially of the inferior wall).

Treatment

No specific treatment is usually required. Treatment of the underlying disorder is appropriate.

PREMATURE VENTRICULAR CONTRACTIONS

Description

Premature ventricular contractions occur when electrical impulses originate from single or multiple areas in the ventricles. ECG characteristics (**Figure 18–7** and **Table 18-8**) are used to differentiate premature ventricular contractions from other premature beats.

FIGURE 18–7.
Sinus rhythm with premature ventricular contractions (PVCs). **A**. Sinus rhythm with a single PVC. **B**. Sinus rhythm with multiple unifocal PVCs. **C**. Sinus rhythm with multifocal PVCs (three different PVC morphologies in this single lead, indicated by #1, #2, and #3).

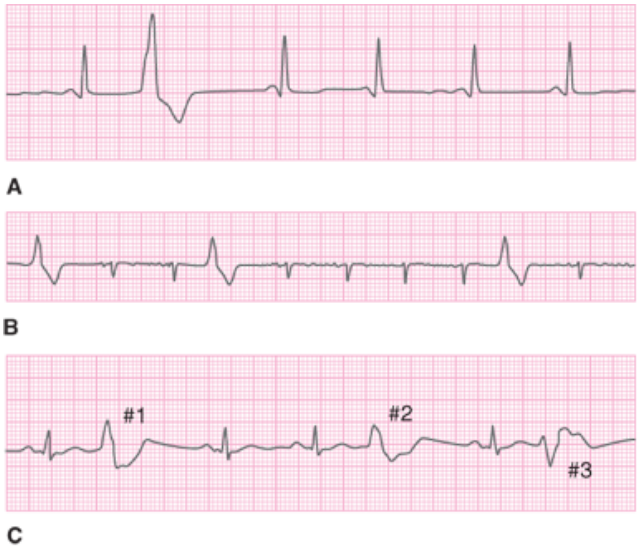


Table 18–8

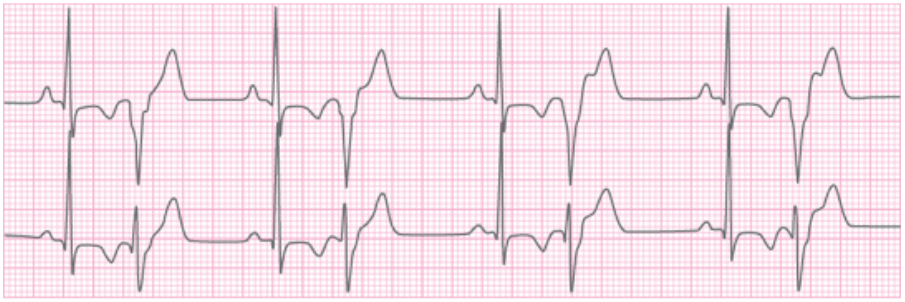
ECG Features of Premature Ventricular Contractions

Absence of P wave prior to the QRS complex
Occasional retrograde P wave following QRS complex
Abnormally widened QRS complex with different morphology from SA node–initiated QRS complex
Commonly a compensatory postectopic pause following the premature ventricular contraction to next SA node–initiated beat
ST segments and T waves that are directed opposite the major QRS complex deflection

Abbreviation: SA = sinoatrial.

Most premature ventricular contractions do not affect the spontaneous discharge of the SA node, so the interval between normal sinus P waves encompassing the premature ventricular contraction is twice the previous P to P interval, termed a fully compensatory postectopic pause. This fully compensatory pause occurs because the SA node discharges during the refractory period of either the AV node or His bundles induced by the premature ventricular contraction. Less commonly, a premature ventricular contraction may be interpolated between two sinus beats. Many premature ventricular contractions occurring in a bigeminal or trigeminal pattern have a fixed coupling interval (within 40 milliseconds) from the preceding sinus beat (**Figure 18–8**). Occasionally, a **ventricular fusion beat** occurs when both supraventricular and ventricular impulses depolarize the ventricular myocardium almost simultaneously. The QRS configuration of a fusion beat has characteristics of both the normally conducted beat and the ectopic one.

FIGURE 18–8.
Premature ventricular contractions producing ventricular bigeminy.

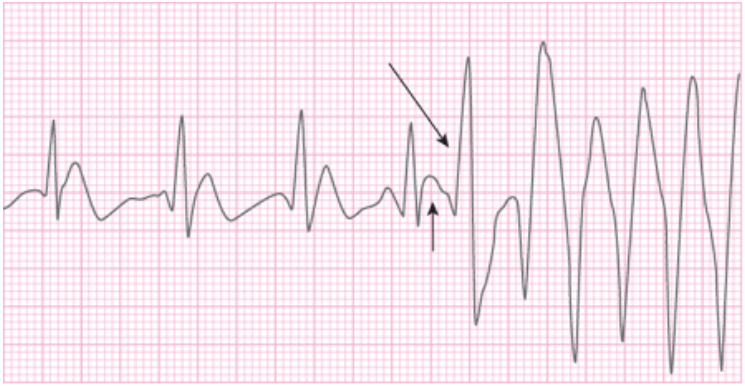


The degree (quantity and quality) of the premature ventricular contractions is categorized as follows: (1) an occasional premature ventricular contraction seen on the rhythm strip is called an isolated premature ventricular contraction (Figure 18–7A), (2) multiple premature ventricular contractions of similar morphology are called unifocal premature ventricular contractions (Figure 18–7B), and (3) multiple premature ventricular contractions with different morphology are called multifocal premature ventricular contractions (Figure 18–7C), implying more than one ventricular focus is producing ectopy. Periods of sustained unifocal premature ventricular contractions may occasionally be seen, often with a fixed ratio and coupling interval to sinus beats.

Clinical Significance

Premature ventricular contractions are very common and related to factors that alter the electrophysiology of cardiac tissue or to pathologic conditions of the myocardium itself. Sometimes, infrequent or rare premature ventricular contractions may be observed in patients without any evidence of heart disease. Premature ventricular contractions may trigger sustained runs of ventricular tachycardia (**Figure 18–9**).[15]

FIGURE 18–9.
Sinus rhythm in an ST-segment elevation myocardial infarction patient with a premature ventricular contraction (PVC) initiating ventricular tachycardia. Note the R wave (*large arrow*) of a PVC falling on the T wave (*small arrow*) of the last sinus beat. This R-on-T event produces ventricular tachycardia.



There is a correlation between the severity of underlying coronary artery disease and the degree of ventricular ectopy, and in addition, ventricular ectopy is an independent risk factor for sudden cardiac death.[16,17] In acute coronary syndrome, premature ventricular contractions indicate the underlying electrical instability of the heart, but patterns of premature ventricular contractions ("warning dysrhythmias") are not reliable predictors of subsequent ventricular fibrillation.

Treatment

Review the ECG for evidence of ischemia or infarction, chamber enlargement, QT interval prolongation, or Brugada syndrome. Assess for potentially reversible conditions such as hypoxia, drug effect, or electrolyte abnormalities. In general, treat the underlying cause.[18,19] Typical recommendations for stress reduction and elimination of stimulants such as caffeine or nicotine are not consistently effective.[19] Patients with greater than three premature ventricular contractions in a row are considered to have nonsustained ventricular tachycardia, which can be a marker for sustained tachydysrhythmias and sudden cardiac death. If this is a new dysrhythmia, initiate emergency cardiac investigation.

Pharmacologic suppression of isolated premature ventricular contractions with antiarrhythmic medications in the acute setting does not confer improved survival for the acute condition. Attempts to suppress premature ventricular contractions with long-term oral antidysrhythmics increase mortality due to the dangerous prodysrhythmic properties of the medications themselves.[18,19] Implantable cardioverter defibrillators are used in patients with premature ventricular contractions that have potential to trigger malignant ventricular dysrhythmias or cardiac arrest.

BRADYDYSRHYTHMIAS

SINUS BRADYCARDIA

Description

Sinus bradycardia is when the SA node discharge rate falls below 60 beats/min and AV conduction remains intact with a constant PR interval (**Figure 18–10**). The ECG characteristics are identical to sinus rhythm with the exception of a slow heart rate (**Table 18-9**).

FIGURE 18–10.
Sinus bradycardia.

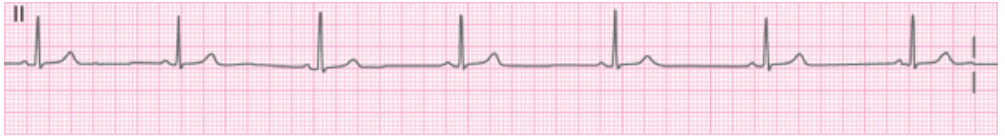


Table 18–9

ECG Features of Sinus Bradycardia

Normal SA node–initiated P waves: consistent morphology among all P waves with upright amplitude in leads I, II, and III
Normal PR interval: 120–200 milliseconds
1:1 atrioventricular conduction: a QRS complex for each P wave with consistent association
Rate <60 beats/min and regular

Abbreviation: SA = sinoatrial.

Table 18–10

ECG Features of Junctional Rhythm

Absence of normal (sinus-mediated) P waves with normal PR interval
Rare retrograde P wave (usually an inverted P wave and immediately adjacent to QRS complex, pre or post)
Narrow QRS complex
Regular rate
Ventricular rate
Between 40 and 60 beats/min for junctional rhythm
Between 60 and 100 beats/min for accelerated junctional rhythm
>100 beats/min for junctional tachycardia

Clinical Significance

Sinus bradycardia represents a reduction of the SA node discharge rate. Sinus bradycardia can be (1) physiologic (in well-conditioned athletes, during sleep, or with vagal stimulation), (2) pharmacologic (β-blockers, digoxin, opioids, calcium channel blockers), or (3) pathologic (hypoxia, acute inferior wall myocardial ischemia or infarction, increased intracranial pressure, carotid sinus hypersensitivity, hypothyroidism).

Treatment

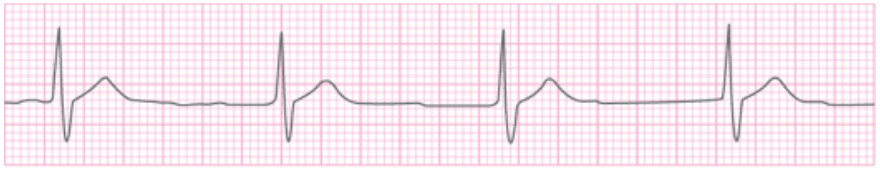
Sinus bradycardia usually does not require specific treatment unless the heart rate is slower than 50 beats/min and there is evidence of hypoperfusion. Correct underlying causes. Use atropine in the unstable patient, followed by transcutaneous cardiac pacing and infusions of dopamine or epinephrine if there is no response to atropine.[1]

JUNCTIONAL RHYTHM

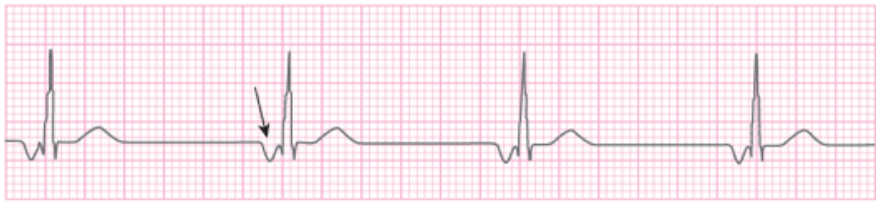
Description

Under normal circumstances, the SA node discharges at a faster rate than the AV node, so the pacemaker function of the AV node and all other slower pacemakers are suppressed. If SA node discharges slow or fail to reach the AV node, junctional escape beats will produce a rhythm (**Figure 18–11**) usually at a rate between 40 and 60 beats/min. If the junctional beats continue in sequence, then a junctional rhythm is present. In most cases, junctional escape beats do not conduct retrograde into the atria, so a QRS complex without a P wave is usually seen (Figure 18–11A); rarely, the junctional escape beat does conduct retrograde into the atria, producing the retrograde P wave; a P wave usually inverted and found immediately prior to or following the QRS complex (Figure 18–11B).

FIGURE 18–11.
Junctional rhythm. **A**. Junctional rhythm. **B**. Junctional rhythm with retrograde P waves (*arrow*).



A



B

At times, enhanced AV nodal automaticity overrides the sinus node and produces an accelerated junctional rhythm with a rate of 60 to 100 beats/min or junctional tachycardia with a rate greater than 100 beats/min. Usually, the enhanced junctional pacemaker captures both the atria and ventricles.

Clinical Significance

Junctional escape beats may occur whenever there is a long enough pause in the impulses reaching the AV node, as with sinus bradycardia, slow phase of sinus arrhythmia, or during the pause after premature beats. Sustained junctional escape rhythms may be seen with heart failure, myocarditis, hypokalemia, or digitalis toxicity.

Accelerated junctional rhythm, including junctional tachycardia, may occur from medication toxicity, acute rheumatic fever, or inferior myocardial ischemia. With medication toxicity (particularly digitalis compounds) in a patient being treated for atrial fibrillation, the rate is usually between 70 and 130 beats/min and the ECG is characterized by regular QRS complexes superimposed on atrial fibrillatory waves.

Treatment

Isolated, infrequent junctional escape beats usually do not require specific treatment. If sustained junctional escape rhythms are producing symptoms, the underlying cause should be treated. Atropine can be used to accelerate the SA node discharge rate and enhance AV nodal conduction. Accelerated junctional rhythm and junctional tachycardia usually do not produce significant symptoms.

IDIOVENTRICULAR RHYTHMS

Description

Idioventricular rhythms are of ventricular origin (**Figure 18–12**), manifesting as regular widened QRS complexes without evidence of atrial activity (**Table 18-10**). An idioventricular rhythm has a ventricular rate of 30 to 50 beats/min, and the accelerated idioventricular rhythm has a ventricular rate of 50 to 75 beats/min. Idioventricular rhythm tends to appear in nonsustained fashion with runs of short duration, ranging from 3 to 30 consecutive beats, and will typically begin with a fusion beat.

FIGURE 18–12.
Idioventricular rhythm with a ventricular rate of approximately 30 beats/min.

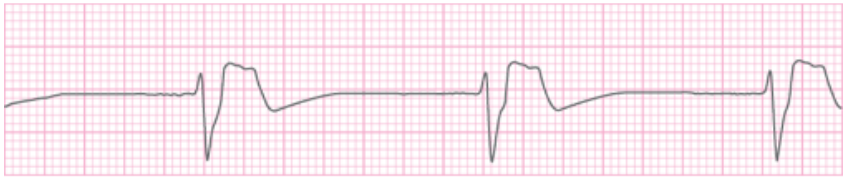


Table 18–10

ECG Features of Idioventricular Rhythm

Widened QRS complex
QRS complexes occurring regularly
No evidence of atrial activity: no P waves
Ventricular rate
30–50 beats/min for idioventricular rhythm
50–75 beats/min for accelerated idioventricular rhythm
Often in nonsustained fashion with runs of short duration: 3–30 consecutive beats

Clinical Significance

Idioventricular rhythm is seen most commonly in the setting of an ST-segment elevation myocardial infarction. An accelerated idioventricular rhythm that appears during successful fibrinolysis of an occluded coronary artery is termed a *reperfusion dysrhythmia*. Although there is some association with ventricular tachycardia, there is no apparent association with ventricular fibrillation. Idioventricular rhythms, particularly the slower versions, can produce dizziness, weakness, syncope, chest pain, and dyspnea; profound hypoperfusion may occur. Accelerated idioventricular rhythm itself usually produces no symptoms, but the loss of atrial contraction and subsequent fall in cardiac output can produce hemodynamic deterioration.

Treatment

With idioventricular rhythm producing hypoperfuson, drugs to increase the heart rate are appropriate. Atropine is recommended, although the likelihood of successful treatment is low. Cardiac pacing is often needed, starting via the transcutaneous route. In most cases of accelerated idioventricular rhythm, treatment is not necessary. If accelerated idioventricular rhythm is the only functioning pacemaker, **suppression with antiarrhythmic agents may lead to asystole**. If sustained accelerated idioventricular rhythm produces symptoms secondary to a decrease in cardiac output, pacing is recommended.

ATRIOVENTRICULAR BLOCKS

First-degree AV block is characterized by a delay in AV conduction manifested by a prolonged PR interval. Second-degree AV block is characterized by intermittent AV conduction: some atrial impulses reach the ventricles, and others are blocked. Third-degree AV block is characterized by the complete blockage of atrial impulses to the ventricles.

AV blocks are divided into nodal and infranodal blocks because of the clinical significance and prognostic differences.[1] Nodal AV block (block within the AV node) is usually due to reversible depression of conduction, is often self-limited, and generally has a stable infranodal escape pacemaker pacing the ventricles. Infranodal blocks (block below the AV node) usually are due to organic disease of the His bundle or bundle branches; often the damage is irreversible. They generally have a slow and unstable ventricular escape rhythm pacing the ventricles, and they frequently have a bad prognosis.

FIRST-DEGREE ATRIOVENTRICULAR BLOCK

Description

In first-degree AV block, each atrial impulse is conducted to the ventricles but less rapidly than normal, as noted by a prolonged PR interval, greater than 200 milliseconds. There is a P wave for each QRS complex; this association is consistent from one beat to the next (**Figure 18–13** and **Table 18-11**). The AV node is usually the site of conduction delay, although this block may occur at an infranodal level.

FIGURE 18–13.
Sinus rhythm with first-degree atrioventricular block (PR interval, 300 milliseconds).

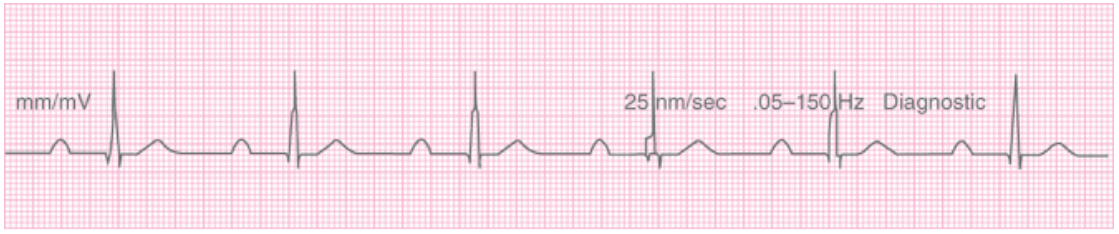


Table 18–11

ECG Features of First-Degree Atrioventricular Block

Consistent P wave to QRS complex relationship
Prolongation of PR interval >200 ms

Clinical Significance

First-degree AV block occasionally is found in normal hearts. Other common causes include increased vagal tone of any cause, medication toxicity, inferior myocardial infarction, and myocarditis. Patients with first-degree AV block without evidence of organic heart disease appear to have no difference in mortality compared with matched controls. In the setting of an acute coronary syndrome event, its appearance can indicate an increased chance of progression to complete heart block.

Treatment

Usually none is required. Close monitoring in the patient with acute myocardial ischemia is indicated due to the potential for progression to complete heart block.

SECOND-DEGREE MOBITZ TYPE I (WENCKEBACH) ATRIOVENTRICULAR BLOCK

Description

In second-degree Mobitz type I (Wenckebach) block, there is progressive prolongation of AV conduction (and the PR interval) until an atrial impulse is completely blocked; when an atrial impulse is blocked, no accompanying QRS complex is seen (**Figure 18–14** and **Table 18-12**). Conduction ratios indicate the ratio of atrial to ventricular depolarizations; for instance, a 4:3 ratio indicates that three of four atrial impulses are conducted into the ventricles. Usually, only one atrial impulse is blocked. After the dropped beat, the AV conduction returns to normal, and the cycle usually repeats itself with the same conduction ratio (fixed ratio) or a different conduction ratio (variable ratio). This type of block almost always occurs at the level of the AV node and is often due to reversible depression of AV nodal conduction.

FIGURE 18–14.
Second-degree, type I atrioventricular block. Note the nonconducted P waves (*arrows*).

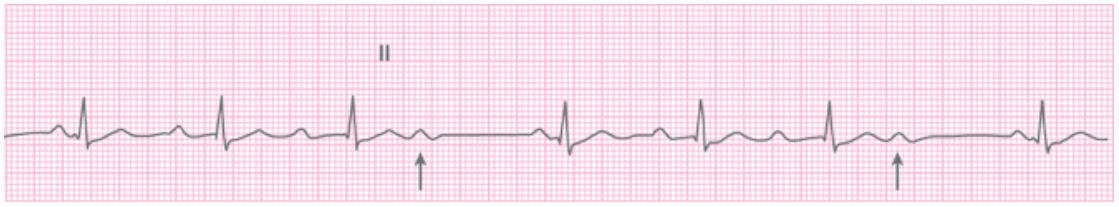


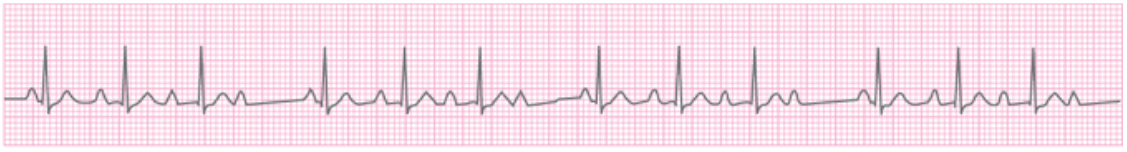
Table 18–12

ECG Features of Second-Degree Mobitz Type I (Wenckebach's) Atrioventricular Block

Progressive prolongation of PR interval until an atrial impulse is completely blocked: a P wave without accompanying QRS complex
After the nonconducted beat, cycle repeats
Grouped beating

Second-degree type I, or Wenckebach, block occurs because each successive depolarization produces prolongation of the refractory period of the AV node. When the next atrial impulse comes upon the node, it is earlier in the relative refractory period, and conduction occurs more slowly relative to the previous stimulus. This process is progressive until an atrial impulse reaches the AV node during the absolute refractory period, and conduction is blocked altogether. The pause allows the AV node to recover, and the cycle repeats. This cyclic occurrence produces a pattern of "grouped beating" and is apparent over successive cycles (**Figure 18–15**).

FIGURE 18–15.
A. Second-degree, type I atrioventricular block. **B**. The cardiac rhythm strip illustrates the concept of grouped beating, or clustering of QRS complexes indicated by the ovals in the rhythm strip.



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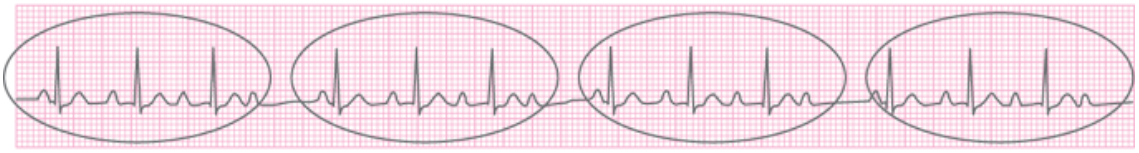


B

Clinical Significance

This block is often transient and usually associated with an inferior myocardial ischemia, medication toxicity, or myocarditis, or after cardiac surgery. It may occur when a normal AV node is exposed to very rapid atrial rates. This block can also be a normal variant, not indicative of acute or chronic heart disease.

Treatment

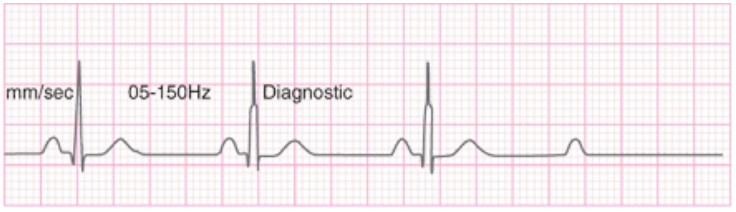
Specific treatment is usually not necessary unless very slow ventricular rates produce signs of hypoperfusion, where most patients will respond to atropine. The need for an increased rate and increased perfusion must be balanced with the increased myocardial work in the acutely ischemic patient.

SECOND-DEGREE MOBITZ TYPE II ATRIOVENTRICULAR BLOCK

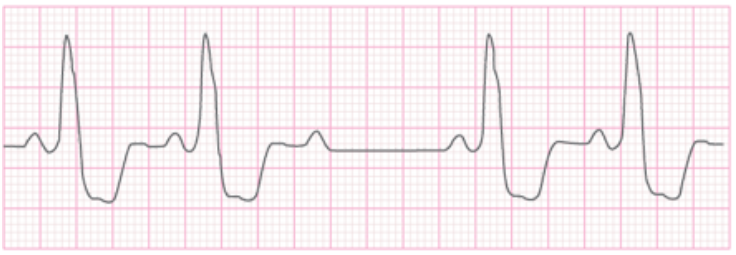
Description

In second-degree Mobitz type II block, the PR interval remains constant across the rhythm strip, both before and after the nonconducted atrial beats (**Figure 18–16** and **Table 18-13**). Each P wave is associated with a QRS complex until a nonconducted atrial depolarization (i.e., P wave) is noted without accompanying QRS complex. Mobitz II blocks usually occur in the infranodal conducting system, often with coexistent fascicular or bundle-branch blocks, and the QRS complexes therefore are usually wide. Even if the QRS complexes are narrow, the block is generally in the infranodal system. High-grade AV block is noted when more than one consecutive P wave is not conducted (Figure 18–16C).

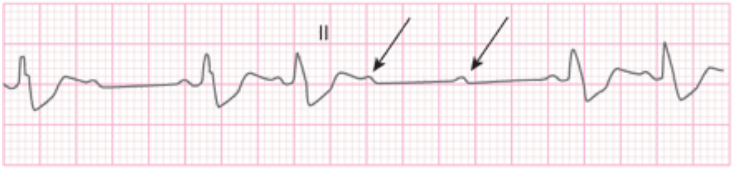
FIGURE 18–16.
Three examples of second-degree, type II atrioventricular (AV) block: (**A**) with narrow QRS complex rhythm; (**B**) with wide QRS complex rhythm; and (**C**) with wide QRS complex rhythm and high-grade AV block indicated by two or more consecutive nonconducted P waves (*arrows*).



A



B



C

Table 18–13

ECG Features of Second-Degree Mobitz Type II Atrioventicular Block (AVB)

PR interval remains constant
Each P wave is associated with a QRS complex until a non-conducted atrial depolarization (i.e., P wave) is noted without accompanying QRS complex
QRS complexes are usually widened though a narrow complex is occasionally seen
High grade AVB is noted when more than one consecutive P wave is not conducted

When second-degree AV block occurs with a fixed conduction ratio of 2:1, it is not possible to differentiate between type I (Wenckebach) or type II block. If the QRS complex is wide, the block is more likely to be in the infranodal system. If the QRS complex is narrow, then the block is in the AV node or infranodal system with about equal incidence; it is recommended that the "worst case scenario" be assumed in such presentations and to consider the "untypable" second-degree AV block a type II blockage.

Clinical Significance

Type II blocks imply structural damage to the infranodal conducting system, are usually permanent, and may progress suddenly to complete heart block, notably with concomitant acute myocardial ischemia.

Treatment

Patients should have transcutaneous cardiac pacing pads applied in the ED in anticipation of possible need. Start emergent pacing when slow ventricular rates produce symptoms of hypoperfusion. Atropine can be tried but the effect is

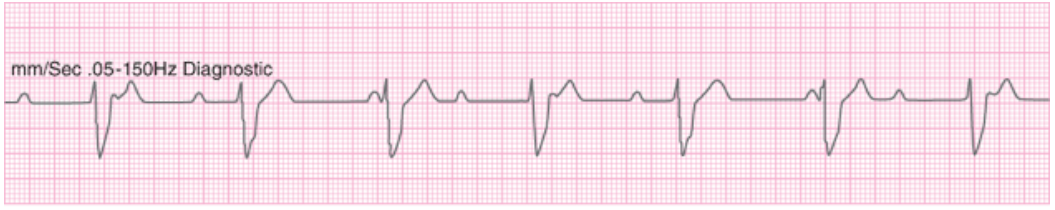
inconsistent.[2,3] Most patients, especially in the setting of acute myocardial ischemia, will require eventual transvenous cardiac pacing.

THIRD-DEGREE ATRIOVENTRICULAR BLOCK (COMPLETE HEART BLOCK)

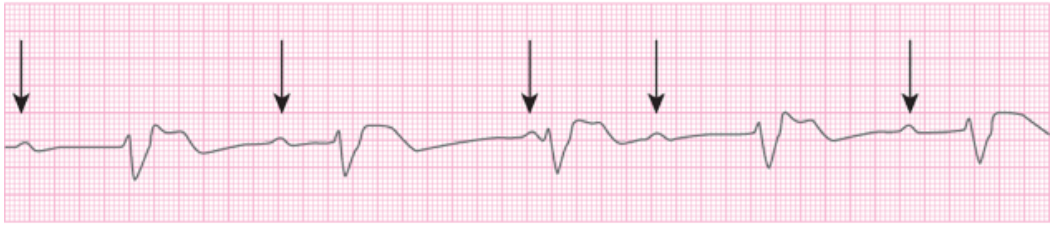
Description

In third-degree AV block, there is no AV conduction (**Figure 18–17** and **Table 18-14**). An escape pacemaker (manifested by the QRS complex) paces the ventricles at a rate slower than the atrial rate manifested by the P wave. When third-degree AV block occurs in the AV node, a junctional escape pacemaker takes over with a ventricular rate of 40 to 60 beats/min. The QRS complexes are narrow because the rhythm originates above the bifurcation of the bundle of His. When third-degree AV block occurs at the infranodal level, the ventricles are driven by a ventricular escape rhythm at a rate slower than 40 beats/min. Third-degree AV blocks at the His bundle level can have a narrow or wide QRS complex, whereas blocks in the bundle branches or elsewhere in the Purkinje system invariably have escape rhythms with wide QRS complexes.

FIGURE 18–17.
Two examples of third-degree atrioventricular block. Both have an atrial rate of 83 with ventricular escape rate of 50. Not all P waves are visible; some are hidden by the QRS complex or T wave.



A



B

Table 18–14

ECG Features of Third-Degree Atrioventricular Block (Complete Heart Block)

No association of P wave with QRS complexes
Atrial rate greater than ventricular rate
QRS complexes are usually widened; occasional narrow QRS complexes are seen
Ventricular rate is regular

Clinical Significance

Nodal third-degree AV block (i.e., with a narrow QRS complex) develops in up to 8% of inferior acute myocardial infarction patients and may last for several days. Infranodal third-degree AV blocks (i.e., with a wide QRS complex) indicate structural damage to the infranodal conducting system, as seen with an extensive anterior acute myocardial infarction. The ventricular escape pacemaker is usually inadequate to maintain cardiac output and the patient is unstable, with periods of ventricular asystole. When third-degree block is seen in acute myocardial infarction, mortality is increased even with pacing.

Treatment

Infrequently, patients with complete heart block will present with minimal to no symptomatology; these patients require monitoring and admission. Manage the symptomatic patient with either medication and/or pacing. Nodal blocks may respond to atropine; infranodal blocks are unlikely to respond to atropine or other medications that can enhance AV nodal conduction. Patients should have transcutaneous cardiac pacer pads applied in the ED. If there is no or incomplete response to atropine, use transcutaneous cardiac pacing, recognizing that transvenous pacing is eventually necessary in most patients.

NARROW-COMPLEX TACHYCARDIAS

SINUS TACHYCARDIA

Description

Sinus tachycardia (**Figure 18–18** and **Table 18-15**), the most common narrow-complex tachycardia encountered in the ED, is defined as sinus rhythm with rates greater than 100 beats/min.

FIGURE 18–18.
Sinus tachycardia at a rate of 110 beats/min.

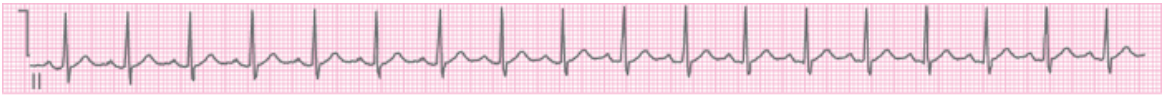


Table 18–15

ECG Features of Sinus Tachycardia

Normal sinus P waves and PR interval
1:1 atrioventricular conduction
Atrial rate usually between 100 and 160 beats/min

Clinical Significance

Sinus tachycardia should be considered a reactive rhythm, occurring in response to a triggering condition. Generally, sinus tachycardia is a benign rhythm without any end-organ dysfunction.

Treatment

Because sinus tachycardia is often a compensatory mechanism resulting from a physiologic stress, address the underlying cause, not the rhythm.

ATRIAL FIBRILLATION AND ATRIAL FLUTTER

Description

After sinus tachycardia, atrial fibrillation is the next most frequent narrow-complex tachycardia encountered in the ED; atrial flutter is a less common dysrhythmia. Atrial fibrillation occurs when there are multiple, small areas of atrial myocardium continuously discharging and contracting.[20] There is no uniform atrial depolarization and contraction but, rather, only a quivering of the atrial chamber walls, resulting in less than effective ventricular filling and diminished cardiac output.

The ECG hallmarks of atrial fibrillation (**Figure 18–19** and **Table 18-16**) include the absence of discernible P waves and an irregularly irregular ventricular rhythm. With the chaotic atrial activity, distinct P waves are not noted; rather either a flat or chaotic baseline is seen, most prominent in lead V_1. The irregularly irregular ventricular rhythm results from the atrial chaos and the variable conduction of impulses through the AV node to the ventricle. The atrial rate in atrial fibrillation is often greater than 600 beats/min, whereas the ventricular rate is markedly lower due to the refractory period of the AV node; in atrial fibrillation where the AV node is unaffected by disease or medications, the ventricular rate is typically 120 to 170 beats/min. Illnesses or medications may reduce AV node conduction and markedly slow ventricular response. A very rapid ventricular response (>200 beats/min) may be seen in patients with accessory or bypass tracts (discussed later in this chapter).

FIGURE 18–19.
Three examples of atrial fibrillation with irregular ventricular response.

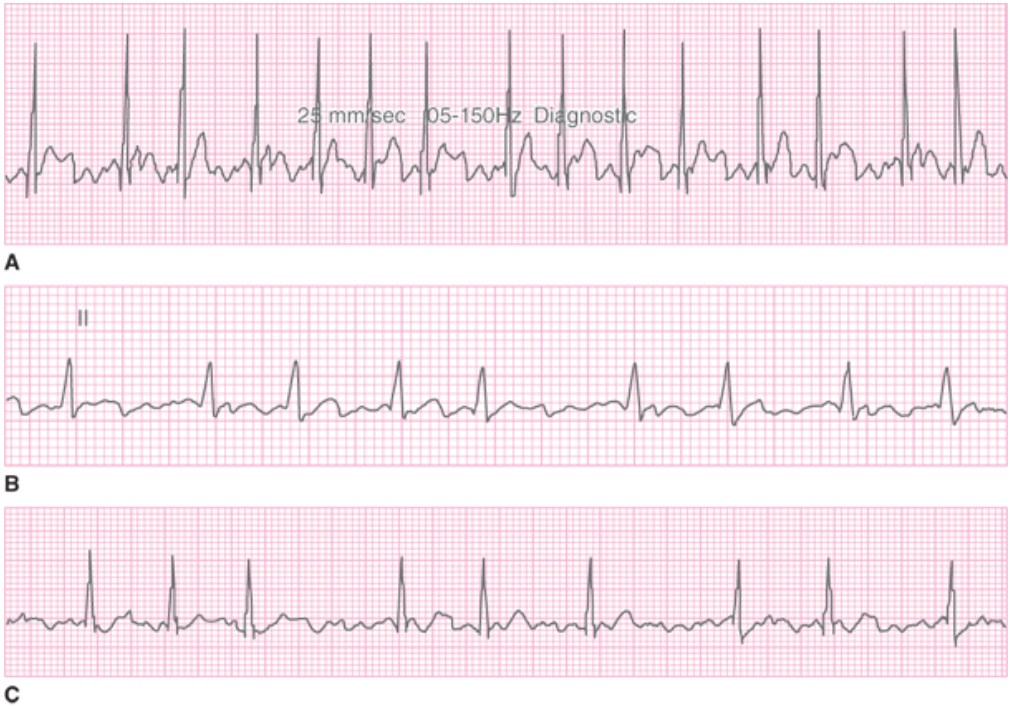


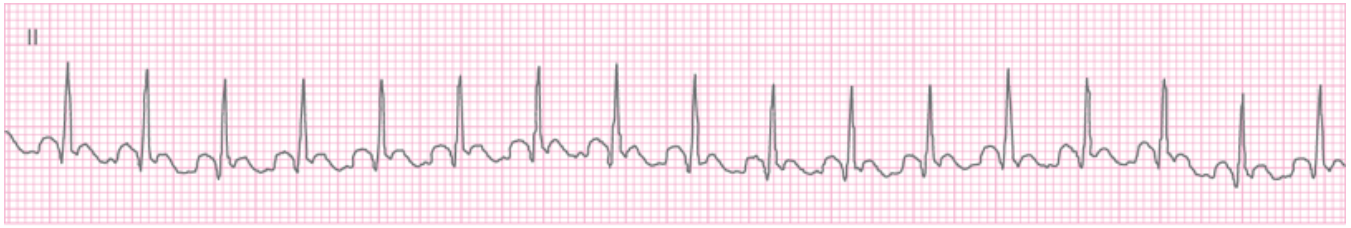
Table 18–16

ECG Features of Atrial Fibrillation

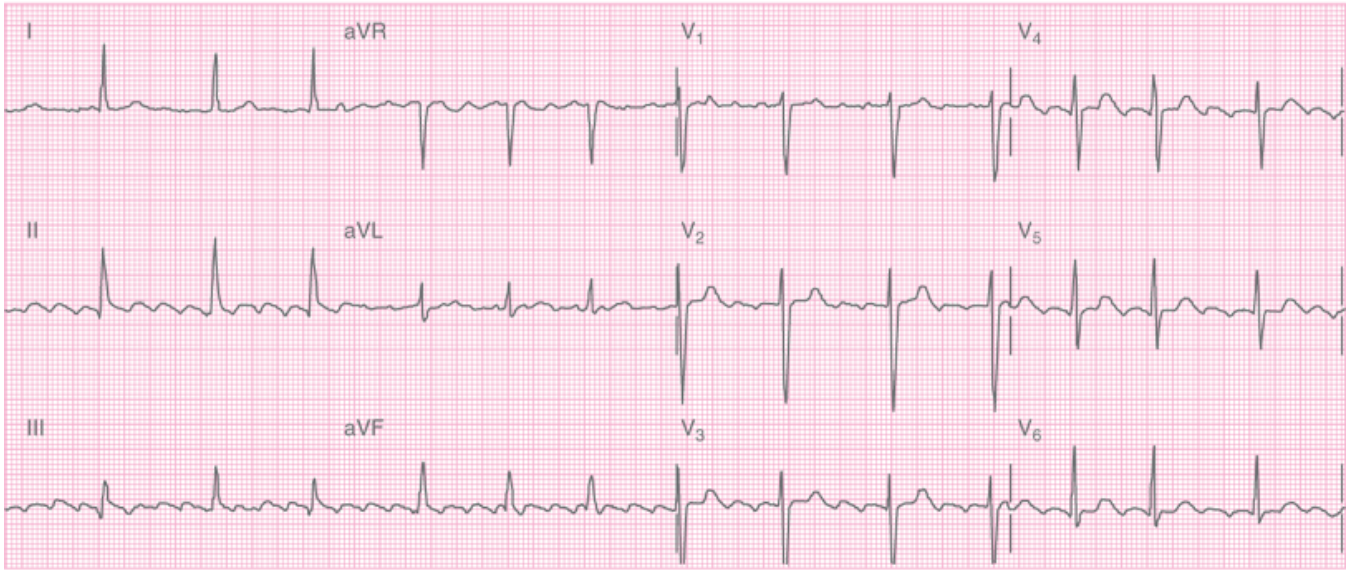
Absence of discernible P waves with flat or chaotic isoelectric baseline
QRS complexes narrow unless preexisting bundle-branch block or preexcitation syndrome
Irregularly irregular ventricular rhythm

In contrast to atrial fibrillation, atrial flutter most often is a regular rhythm (**Figure 18–20** and **Table 18-17**); in rare cases, it can be irregular. P waves are present and of a single morphology, typically a downward deflection, called *flutter waves* resembling a saw blade with a "sawtooth" pattern, best seen in the inferior ECG leads and lead V_1. Most commonly, the atrial rate is regular, classically around 300 beats/min, varying between 250 and 350 beats/min. The ventricular rhythm is frequently regular and is a function of the AV block. AV ratios of 2:1 are common and produce a ventricular rate around 150 beats/min, whereas a 3:1 AV ratio will result in a ventricular rate of 100 beats/min. Although the degree of AV conduction is often fixed, it may also be variable and create an irregular ventricular response. **A regular narrow-complex tachycardia at an approximate rate of 150 beats/min strongly suggests atrial flutter with 2:1 conduction**.

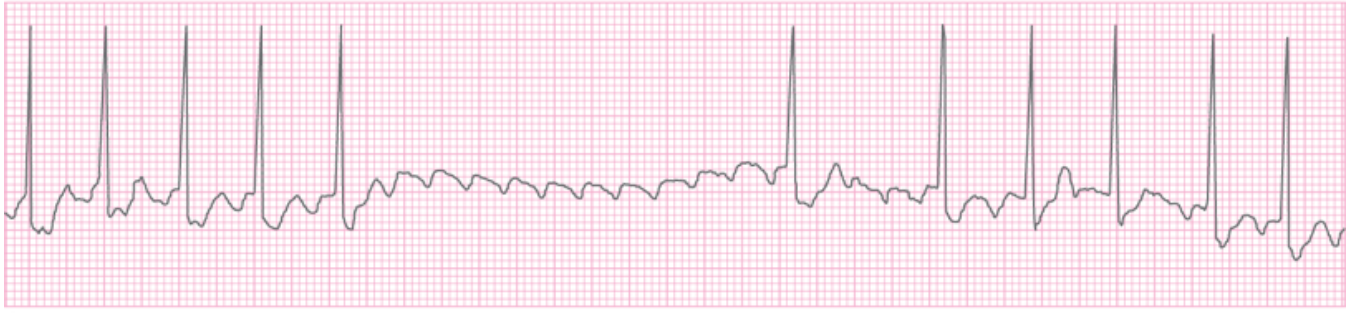
FIGURE 18–20.
Atrial flutter. **A**. Regular, narrow-complex tachycardia at a ventricular rate of 155 beats/min. **B**. Atrial flutter with flutter waves most visible in leads 2, 3, and AVF. **C**. Atrial flutter response to carotid sinus massage inducing transient AV block and unmasking flutter waves.



A



B



C

Table 18–17

ECG Features of Atrial Flutter

Identifiable P waves
Single morphology
Negative amplitude ("flutter waves" in "sawtooth pattern") best seen in inferior ECG leads and V1
Atrial rate is regular, usually at 300 beats/min
QRS complexes narrow unless preexisting bundle-branch block
Ventricular rate regular although occasional irregularity can be seen
Ventricular rate often 150 beats/min

Clinical Significance

Atrial fibrillation is usually associated with ischemic or valvular heart disease; less common causes include congestive cardiomyopathy, myocarditis, alcohol binge ("holiday heart"), thyrotoxicosis, and blunt chest trauma.[20,21] Left atrial enlargement is a common feature of patients with chronic atrial fibrillation. Atrial fibrillation can be paroxysmal (lasting less than 7 days, terminating either spontaneously or with treatment), persistent (sustained longer than 7 days or requiring treatment to terminate), long-standing persistent (lasting continuously longer than 1 year), or permanent (long-standing where a decision has been made not to try to restore normal sinus rhythm).[20] New or recent onset is applied to symptomatic patients presenting to the ED without a prior history of atrial fibrillation.

The clinical consequences of atrial fibrillation include loss of atrial contraction, potential for rapid ventricular rates, and risk of arterial embolism. In patients with compromised cardiac function, left atrial contraction contributes significantly to left ventricular filling, so the loss of effective atrial contraction, as in atrial fibrillation, may produce heart failure in these patients. A rapid ventricular rate can impact ventricular filling as well as coronary and systemic perfusion. Atrial fibrillation increases the risk of venous and atrial thrombosis, with potential for pulmonary and systemic arterial embolism. If an atrial thrombus has formed, conversion from atrial fibrillation to sinus rhythm can propel a portion of the thrombosis out into the systemic circulation, and this risk increases with duration of the dysrhythmia. Observational studies note that conversion from new-onset atrial fibrillation that has been present for 12 hours or less carries a 0.3% risk of arterial embolism, whereas that risk is about 1% for durations of 12 to 48 hours before conversion.[22] For patients with heart failure and diabetes mellitus, conversion from new-onset (duration <48 hours) atrial fibrillation carries a risk of thromboembolic events as high as 9.8%.[23] Conversely, the incidence of thromboembolic events is 0.2% in patients age <60 years and no heart failure.[23] After a duration of >48 hours, the risk of conversion-induced thromboembolic events is increased across all patient groups and a period of anticoagulation is recommended prior to conversion.[20,24]

Treatment

Treatment of atrial fibrillation in the ED involves three issues: ventricular rate control, rhythm conversion, and anticoagulation to prevent arterial embolism.[24-26] Treatment varies according to patient stability, duration of symptoms, and chronicity of atrial fibrillation (paroxysmal, persistent, or permanent). Review prior records to identify past episodes or treatment for atrial fibrillation. For new-onset atrial fibrillation, consider checking thyroid function.[27] For patients on warfarin,

check the prothrombin time. Calculate either the $CHADS_2$ or CHA_2DS_2-VASc score to risk-stratify the potential for future arterial embolic complications; a $CHADS_2$ score of 0 or a CHA_2DS_2-VASc score of 0 or 1 identify low-risk patients (**Table 18-18**).[20,21]

Table 18–18

$CHADS_2$ and CHA_2DS_2-VASc scores

Criteria	$CHADS_2$	CHA_2DS_2-VASc
Congestive heart failure	1	1
Hypertension	1	1
Age ≥75 y	1	2
Diabetes mellitus	1	1
Stroke, TIA, or thromboembolism	2	2
Vascular disease (CAD, PAD)	–	1
Age 65–74 y	–	1
Sex (female)	–	1
Range	0–6	0–9

Abbreviations: CAD = coronary artery disease; PAD = peripheral artery disease; TIA = transient ischemic attack.

For patients with paroxysmal atrial fibrillation or acute medical conditions producing atrial fibrillation, a period of observation and treatment in the ED is appropriate as the atrial fibrillation may spontaneously convert.[12,28] Up to 70% of otherwise healthy ED patients evaluated for acute-onset atrial fibrillation will spontaneously convert within 48 to 72 hours.[29] Ventricular rate control may help control symptoms until conversion. Also, it is more difficult to achieve rate or rhythm control of atrial fibrillation in patients with acute underlying medical illness, and such attempts are associated with an increased incidence of adverse events.[30]

For patients with recent-onset atrial fibrillation and a rapid ventricular response that is producing hypotension, myocardial ischemia, or pulmonary edema, treat with urgent electrical cardioversion.[20,24,25,31] When possible, first determine if the patient has long-standing atrial fibrillation because electrical cardioversion is not likely to succeed, and instead, initiate ventricular rate control treatment. If the patient is at increased risk for embolic complications ($CHADS_2$ or CHA_2DS_2-VASc scores ≥1, mechanical heart value, or rheumatic valvular disease), consider anticoagulation with heparin before or immediately after electrical cardioversion and continue that as a bridge to oral anticoagulants.[31]

For stable low-risk patients in the ED with new-onset atrial fibrillation, either rate-control or rhythm-conversion strategies are appropriate (**Table 18-19**).[20,24,25] The rate-control approach consists of initiating medications that block the AV node to control the ventricular response, initiating oral anticoagulants to prevent thromboembolism (if appropriate), and re-evaluation

after 3 to 4 weeks for elective cardioversion. The rhythm-conversion approach uses electrical or pharmacologic methods to convert the patient back into sinus rhythm while in the ED.

Table 18–19

Typical ED Management of Atrial Fibrillation/Flutter[tin_ch18fn9]

DILTIAZEM	**Metoprolol**
1. Diltiazem, 15-20 milligrams IV bolus over 2 minutes; may repeat bolus if inadequate rate response.	1. 5 mg IV; may repeat every 5 minute to total of 15 mg
2. Once satisfactory rate response, begin IV infusion 5-10 milligrams/hr	2. Once satisfactory rate response, given oral metoprolol, as 12.5 mg po per 5 mg IV metoprolol
3. Begin transition to oral diltiazem, 60-90 milligrams po for 5 or 10 milligrams drip rate, respectively	
4. Stop IV infusion 2 hours after oral dose	
CARDIOVERSION REQUIREMENTS	
Atrial Flutter	May require as little as 25-50 j
Atrial Fibrillation	150-200 j

* See chapter 19, tables 19-6, 19-7, and 19-19 for detailed discussion.

Control of the ventricular response is done using a calcium channel blocker (diltiazem) or β-blockers (metoprolol and esmolol) with limited data favoring more effective acute rate control with diltiazem.[32] If β-blockers or calcium channel blockers are ineffective, intravenous procainamide or amiodarone is an option to slow the ventricular response. The goal for rate control is a ventricular rate of <100 beats/min at rest.[25]

Electrical cardioversion using 150 to 200 J can terminate atrial fibrillation allowing for sinus rhythm to resume.[24,31] Conversion to and retention of sinus rhythm is more likely when atrial fibrillation is of short duration (<48 hours) and the atria are not greatly dilated. Observational analysis also notes that administration of rate-control or rhythm-conversion medications prior to electrical cardioversion attempts is associated with a reduced rate of successful conversion to sinus rhythm.[33] Although ED electrical cardioversion is effective in many atrial fibrillation patients[34] and observational studies indicate shorter ED length of stay,[26] there has been mixed acceptance of this approach.[35-37] As noted above, a significant portion of patients with new-onset atrial fibrillation will spontaneously convert to sinus rhythm within 24 hours of onset and evaluation.[12,28,29,38,39] This rate of spontaneous conversion coupled with the results of atrial fibrillation trials demonstrating that rate-control is similar to rhythm-control in terms of several key endpoints indicates no proven benefit for conversion of all new atrial fibrillation patients to sinus rhythm while in the ED.[24,38,39] The patient with new-onset atrial fibrillation who is stable can certainly be managed with rate-control alone, either as an inpatient or outpatient depending overall clinical condition.

The antiarrhythmics procainamide, ibutilide, flecainide, propafenone, and vernakalant can chemically convert atrial fibrillation to sinus rhythm.[24,40,41] Of the five, ibutilide has the highest consistent success rate for conversion. Ibutilide

should not be given in the presence of hypokalemia, prolonged QT interval, or history of heart failure, as torsade de pointes may be initiated. This risk of torsade de pointes persists for 4 to 6 hours after the ibutilide is given. Pharmacologic conversion therapy is best avoided if duration is unknown or greater than 48 hours, allowing for heart clot detection and anticoagulation prior to any attempt.

Patients with recurrent paroxysmal atrial fibrillation are sometimes given oral medications (usually flecainide or propafenone) to be taken at the onset of the dysrhythmia; this "pill in a pocket" approach is successful in selected outpatients.[24] This approach should only be used in patients with paroxysmal episodes after SA or AV node dysfunction, conduction disturbance (bundle branch block, Brugada syndrome), and structural heart disease have been excluded.[2]

Atrial flutter is managed in the same fashion as atrial fibrillation: either rhythm conversion or ventricular rate-control with β-blockers or calcium channel blockers.[24] Atrial flutter is very responsive to electrical cardioversion; as little as 25 to 50 J is often effective. In general, patients in atrial flutter tend to better tolerate the dysrhythmia hemodynamically than patients with atrial fibrillation. This "hemodynamic toleration" results from the organized atrial contraction seen with atrial flutter, as opposed to the lack of organized atrial contraction with atrial fibrillation. Despite organized atrial activity, there is a risk of arterial embolism with atrial flutter,[42] and corresponding recommendations for anticoagulation are based on the same criteria used for atrial fibrillation.[25]

Patients with chronic atrial fibrillation and planned cardioversion should be anticoagulated for 3 to 4 weeks, assuming clinical stability allows such an approach.[20] Patients with permanent atrial fibrillation are at increased risk for embolic stroke, and oral anticoagulation can reduce that occurrence. The benefits of oral anticoagulation are counterbalanced by the potential adverse effects, usually hemorrhagic events. Clinical scoring tools, such as the CHA_2DS_2-VASc, can be used to guide the decision to initiate long-term oral anticoagulation therapy in atrial fibrillation patients.[20,21]

Disposition

Significant regional variation for hospitalization after an ED visit for atrial fibrillation has been reported, reflecting the interplay between patient clinical, socioeconomic, and hospital characteristics.[43] Admission is indicated when the patient's clinical status identifies distress with the need for continued therapy; such as heart failure with pulmonary congestion and continued respiratory distress, or ongoing myocardial ischemia.[44] Occasionally, admission is advised when ventricular rate control cannot be achieved and the patient has persistent symptoms.[44] Conversely, 80% or more of patients with atrial fibrillation can be discharged from the ED after rate or rhythm control with low risk for stroke or death within 30 days.[45] For patients with atrial flutter converted to sinus rhythm with electrical cardioversion, about 90% can be discharged with a very low incidence of stroke within the following year.[46] A protocol is useful to ensure that the patient with persistent dysrhythmia is discharged with appropriate medications and plan for follow up (Table 18-20 and Table 18-21).[47]

Table 18-20.

Example Protocol for Discharge of Patient with Persistent Atrial Fibrillation or Flutter

Patient is breathing comfortably on room air with SaPO2 > 95% and BP is at baseline
Ventricular rate has been controlled to a rate less than 100 bpm at rest
For nonvalvular atrial fibrillation patients not converted to sinus rhythm, determine risk for embolic stroke by calculating the CHA_2DS_2-VASc score CHA_2DS_2-VASc = 0: oral anticoagulation not recommended CHA_2DS_2-VASc = 1: either no anticoagulation or oral anticoagulant are acceptable CHA_2DS_2-VASc ≥ 2: oral anticoagulation recommended, consult follow-up specialist for specific agent
Consult with internist or cardiologist to arrange early follow-up (within 7 days) and choose medications for rate and/or rhythm control and oral anticoagulants upon discharge (Table 18-21)
Arrange outpatient echocardiography for patients with new-onset atrial fibrillation or flutter
Discharge patient with prescriptions for rate and/or rhythm control, and oral anticoagulant if indicated, in quantity only sufficient to last until follow-up appointment

Table 18-21.

Rate Control and Oral Anticoagulation Medications used on Discharge of Patients with Persistent Atrial Fibrillation or Flutter

	Typical Initial Dose	Comments
Rate Control Medications		
Diltiazem	30 mg PO QID (120 mg/d) non-sustained release form, increase dose if needed for ventricular rate control up to 480 mg/d; typical maintenance dose 360 mg/d	More effective than metoprolol in controlling heart rate and reducing dysrhythmia-related symptoms Avoid in patients with heart failure with reduced ejection fraction Can switch to a slow-release/extended release dose when rate control dose established
Metoprolol	25 mg PO BID (50 mg/d) nonsustained release form, increase dose if needed for ventricular rate control up to 200 mg/d; typical maintenance dose 100 mg/d	Can switch to a slow-release/extended release dose when rate control dose established
Oral Anticoagulants		
Warfarin	5 mg PO daily	Half-life 20 – 60 hours, mean 40 hours Preferred regimen if CrCl < 15 mL/min
Enoxaparin	1 mg/kg SC q 12h Reduce dose if CrCl <30 mL/min	See text comments about bridging parenteral anticoagulation
Dabigatran etexilate mesylate	150 mg PO BID (if CrCl >30 mL/min) 75 mg PO BID (if CrCl 15 - 30 mL/min)	Half-life 12-17 hours
Rivaroxaban	20 mg PO daily with evening meal (if CrCl >50 mL/min)	Half-life 5-9 hours

	15 mg PO daily with evening meal (if CrCl 15 - 50 mL/min)	
Apixaban	5 mg PO BID 2.5 mg PO BID in patients with at least 2 of the following characteristics: age ≥80 years, body weight ≤60 kg, or serum creatinine ≥1.5 mg/dL	Half-life during repeat oral dosing 12 hours

Oral anticoagulation is used to reduce the incidence of embolic stroke in patients with atrial fibrillation or flutter.[48] Practical factors such as co-morbidities, convenience, and cost should be considered when selecting the specific oral anticoagulant. For patients at short-term high risk for thromboembolism (e.g., CHA2DS2-VASc ≥ 7, prior stroke, intracardiac thrombus, biosynthetic valve, mitral stenosis), bridging therapy with a parenteral anticoagulant, such as enoxaparin, is reasonable when starting warfarin. Enoxaparin is started at the same time as the warfarin and continued until the INR is within the therapeutic range – 2.0 to 3.0 for nonvalvular atrial fibrillation. For patients at low risk for short-term thromboembolism (e.g., nonvalvular atrial fibrillation and no prior history of thromboembolism), the incidence of a thromboembolic event during the few days required to achieve therapeutic anticoagulation upon starting warfarin is so low as to obviate the need for bridging parenteral anticoagulation. It is best to consult with the appropriate specialist who will be following the patient after ED discharge to discuss the specific agent to be used.

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

Description

Paroxysmal supraventricular tachycardia most frequently results from sustained reentry occurring with the AV node, with an ectopic atrial focus accounting for the remaining 15% to 20%. In paroxysmal supraventricular tachycardia (**Figure 18–21** and **Table 18-22**), the QRS complex is of normal width, rapid, and regular. P waves are "buried" within the QRS complex in about 70% of cases. In the others, a P wave (so-called "retrograde" P wave) is found immediately adjacent before, during, or after the QRS complex without a measurable PR interval.

FIGURE 18–21.
Paroxysmal supraventricular tachycardia.

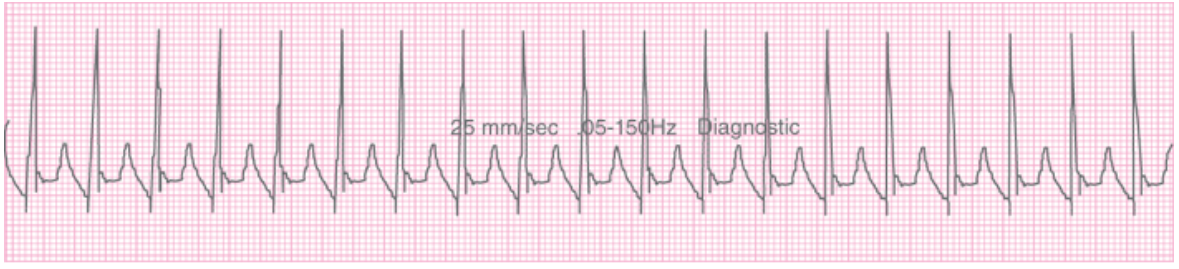


Table 18–22

ECG Features of Paroxysmal Supraventricular Tachycardia

Absence of normal (sinus-mediated) P waves with normal PR interval
Rare retrograde P wave (usually inverted P wave and immediately adjacent to QRS complex, pre or post)
Narrow QRS complex, usually <100 ms in duration
Ventricular rate usually 170–180 beats/min; the rate can range from as low as 130 beats/min to as high as 300 beats/min

Clinical Significance

Paroxysmal supraventricular tachycardia is seen more frequently in females, with a peak in the late teenage and young adult years. The majority of patients are without active cardiovascular disease.[49] Patients may be able to describe the abrupt onset of this reentrant dysrhythmia and also note when it self-terminates. Palpitations, lightheadedness, and dyspnea are common symptoms.

Treatment

If applied early in the dysrhythmia course, vagal maneuvers are often effective (Table 18-3).[10,11] Attention to technique is important to maximize success rate. If there is no response to vagal maneuvers, adenosine IV is recommended to convert to sinus rhythm.[9] It is the rare patient who requires β-blocker or calcium channel blocker. In patients with recalcitrant paroxysmal supraventricular tachycardia or who are unstable, use electrical cardioversion to convert the dysrhythmia.

MULTIFOCAL ATRIAL TACHYCARDIA

Description

Multifocal atrial tachycardia is an irregular rhythm resulting from at least three different atrial ectopic foci competing to pace the heart. The electrocardiographic characteristics (**Figure 18–22** and **Table 18-23**) require at least three distinct P-wave morphologies. Due to irregularity and the chaotic appearance of atrial depolarization, multifocal atrial tachycardia is often confused with atrial fibrillation or atrial flutter.

FIGURE 18–22.
Multifocal atrial tachycardia. Note multiple P-wave morphologies with irregular tachycardia rhythm.

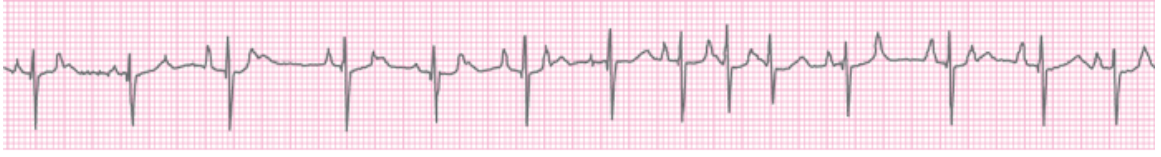


Table 18–23

ECG Features of Multifocal Atrial Tachycardia

At least 3 distinct P-wave morphologies in a single ECG lead
No consistent P to P, PR, or R to R intervals
Irregularly irregular rhythm with rates usually 100–180 beats/min
Normal QRS complex unless preexisting bundle-branch blocks
Frequently confused with atrial fibrillation or flutter

Clinical Significance

Multifocal atrial tachycardia is found most often in elderly patients with decompensated chronic lung disease, but may also complicate heart failure or sepsis.

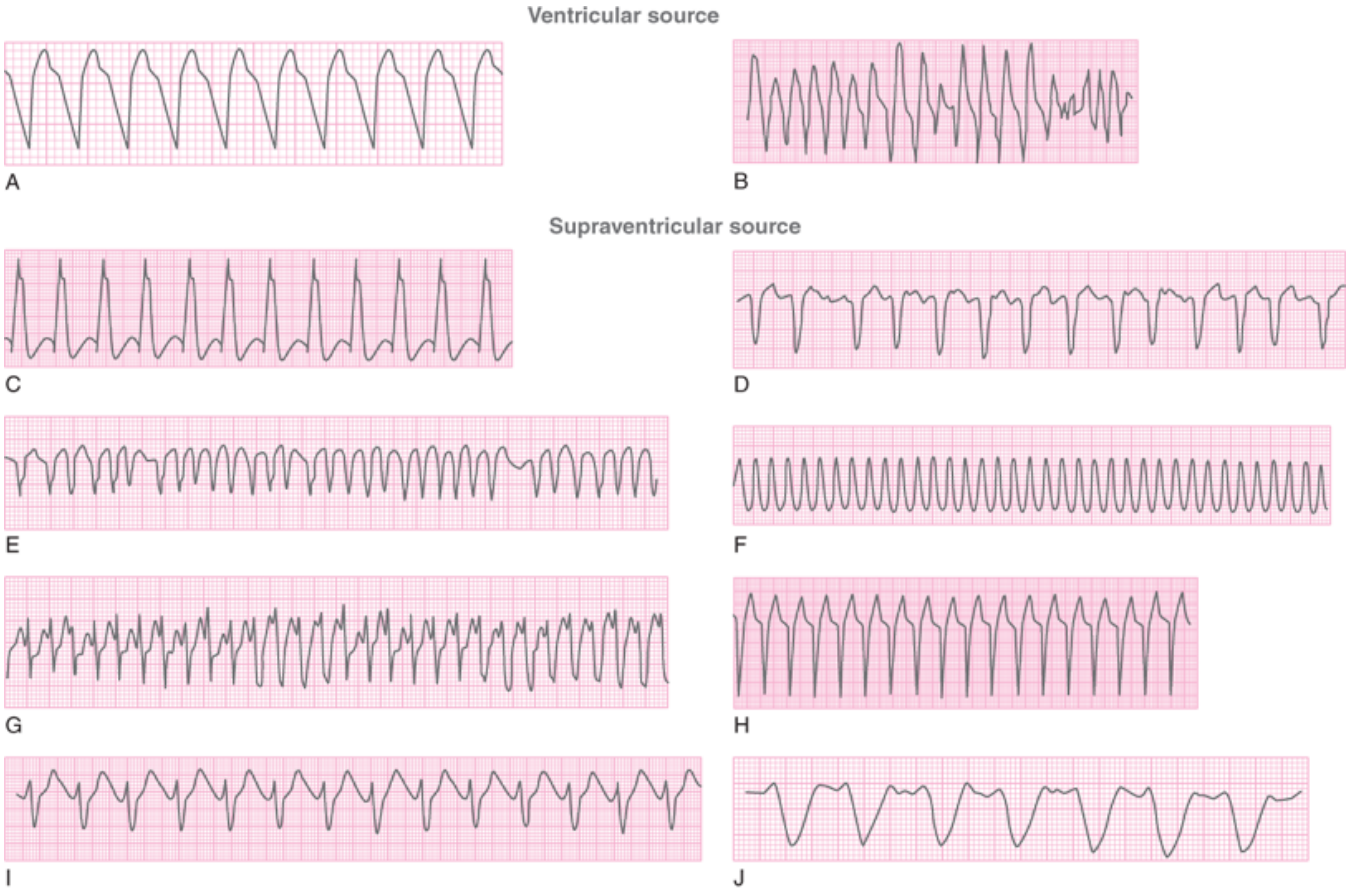
Treatment

Treatment is directed toward the underlying disorder. With decompensated lung disease, oxygen and bronchodilators improve pulmonary function and arterial oxygenation and decrease atrial ectopy. Antidysrhythmic treatment is not indicated, and cardioversion has no effect.

WIDE-COMPLEX TACHYDYSRHYTHMIAS

Wide-complex tachydysrhythmias are defined as a rhythm with a QRS complex duration greater than 120 milliseconds and a rate over 100 beats/min. Both ventricular tachycardia and supraventricular tachydysrhythmias with aberrant ventricular conduction present as wide-complex tachydysrhythmias (**Figure 18–23**).[5]

FIGURE 18–23.
Wide complex tachydysrhythmias. **A**. Ventricular tachycardia (monomorphic). **B**. Ventricular tachycardia (polymorphic). **C**. Sinus tachycardia with bundle-branch block. **D**. Atrial fibrillation with preexisting bundle-branch block. **E**. Preexcited atrial fibrillation in the Wolff-Parkinson-White syndrome. **F**. Atrioventricular reentrant tachycardia in the Wolff-Parkinson-White syndrome. **G**. Paroxysmal supraventricular tachycardia with rate-related bundle-branch block in an infant. **H**. Paroxysmal supraventricular tachycardia with fixed (preexisting) bundle branch block. **I**. Sodium channel blocker toxicity with wide-complex tachycardia. **J**. Wide-complex tachycardia in patient with severe hyperkalemia.



Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
www.accessmedicine.com

Traditional teaching, derived largely from coronary care units and electrophysiology laboratories, presents that 80% of wide-complex tachycardias are ventricular tachycardia.[50] However, in the ED, there is a range of pathophysiologic conditions that can produce delayed ventricular depolarization that when combined with a sinus tachycardia induced by acute illness result in a wide-complex tachycardia.[7] The conduction abnormality can be the result of a bundle-branch block (new-onset, preexisting, or rate-related), metabolic abnormality (e.g., hyperkalemia), adverse medication effect (e.g., sodium channel blockade), or ventricular preexcitation syndrome. All supraventricular tachycardias can present as a wide-complex tachycardia if intraventricular conduction is abnormal. In addition, two tachydysrhythmias seen in WPW syndrome will present with a widened QRS complex: the antidromic version of the AV nodal reentrant tachycardia and atrial fibrillation or flutter with ventricular depolarization predominantly or exclusively via the accessory tract.

VENTRICULAR TACHYCARDIA

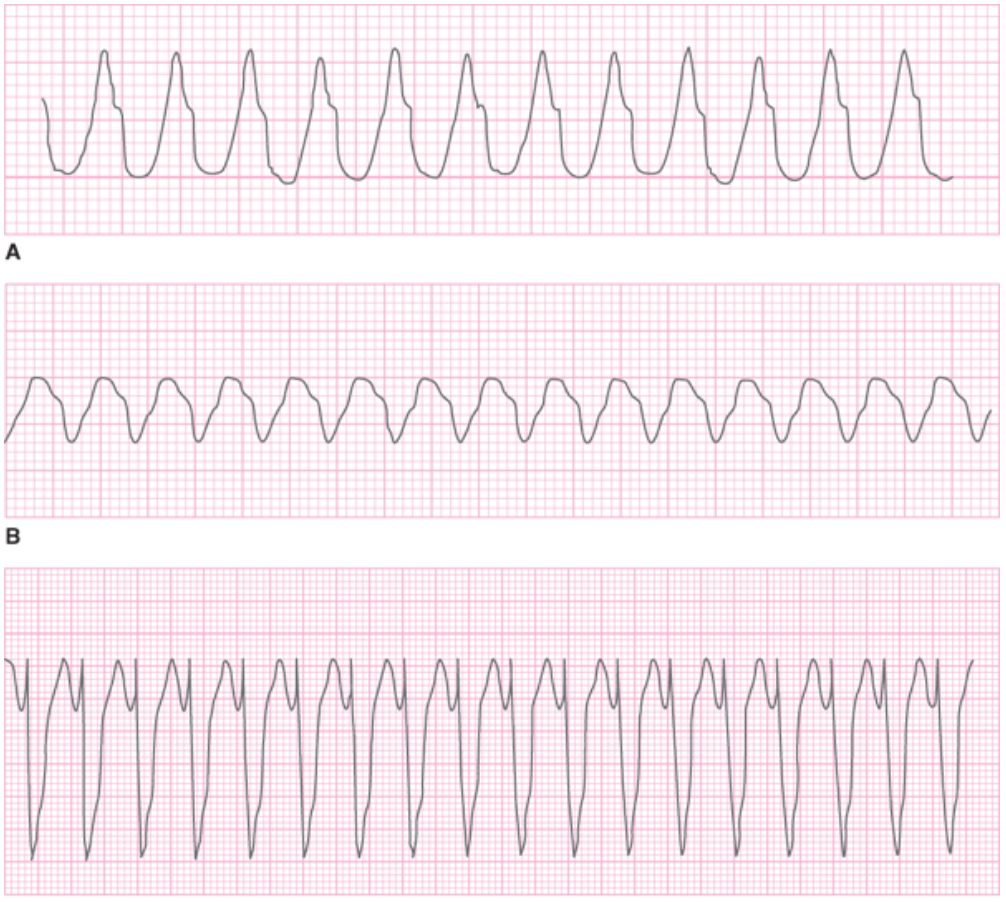
Description

Ventricular tachycardia is the occurrence of three or more consecutive depolarizations from a ventricular ectopic pacemaker at a rate faster than 100 beats/min.[8] Ventricular tachycardia can occur in a nonsustained manner,[51] with short episodes, lasting several beats, with spontaneous termination, or it can occur in a sustained fashion, with longer episodes that typically require treatment.[8] Ventricular tachycardia can be further characterized according to hemodynamic effect (stable vs unstable), morphology (monomorphic vs polymorphic), or clinical presentation (perfusing vs pulseless as during cardiac arrest).

The electrocardiographic features (**Figure 18–24** and **Table 18-24**) vary between the monomorphic and polymorphic varieties. Morphologic ventricular tachycardia is the most commonly encountered form of ventricular tachycardia, accounting for about 70% of cases, and is usually very regular with rates most often in the 140 to 180 beats/min range. Medications,

such as amiodarone, may slow the rate of ventricular tachycardia; such patients may present with ventricular rates of 110 to 130 beats/min. On rare occasions, monomorphic ventricular tachycardia may present with an irregular rhythm, but only minimally irregular as compared to an aberrantly conducted atrial fibrillation, which ordinarily manifests marked irregularity.

FIGURE 18–24.
Three examples of monomorphic ventricular tachycardia. **A**. Ventricular tachycardia with a rate of 270 beats/min. **B**. Ventricular tachycardia with a rate of 220 beats/min. **C**. Ventricular tachycardia with a rate of 180 beats/min.



A

B

C

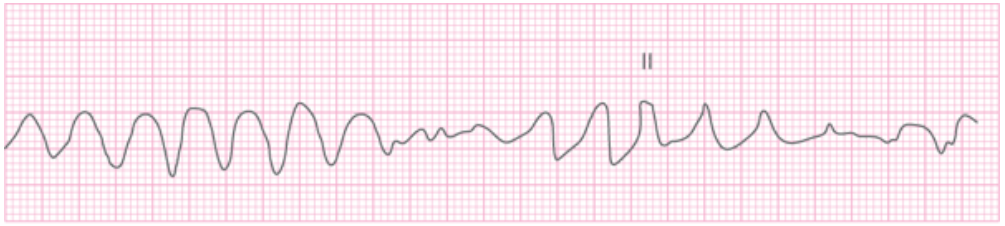
Table 18–24

ECG Features of Monomorphic and Polymorphic Ventricular Tachycardia

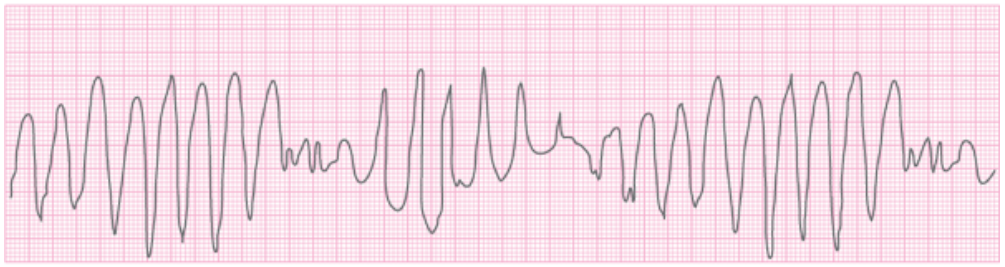
Monomorphic
No P waves associated with QRS complex, occasional dissociated P wave
Rapid and regular rhythm
Rate usually 140–180 beats/min (range, 120–300 beats/min)
Widened QRS complex >100–120 ms with consistent beat-to-beat morphology
Polymorphic
No P waves associated with QRS complex; may have occasional dissociated P wave
Rapid and irregular rhythm
Rate 140–180 bpm (range, 120–300 beats/min)
Widened QRS complex >100–120 ms with inconsistent beat-to-beat morphology

Polymorphic ventricular tachycardia has varying QRS morphology in any single ECG lead (**Figure 18–25** and Table 18-24). Variations in both the RR interval and electrical axis are also features of this form of ventricular tachycardia. Torsade de pointes, a specific subtype of polymorphic ventricular tachycardia, occurs in the setting of delayed myocardial repolarization manifested on the sinus rhythm ECG by a prolongation of the QT interval.[52] The French term *torsade de pointes* means "twisting of the points" and describes the appearance of the QRS complex as it varies in appearance (Figure 18–25B).

FIGURE 18–25.
Two examples of polymorphic ventricular tachycardia. **A**. Polymorphic ventricular tachycardia. **B**. Polymorphic ventricular tachycardia of the torsade de pointes subtype, with the characteristic pattern of progressively changing QRS complex amplitude and direction.



A



B

Clinical Significance

Ventricular tachycardia is rare in patients without underlying heart disease.[5] The most common causes of ventricular tachycardia are chronic ischemic heart disease and acute myocardial infarction, comprising about 50% of all cases of symptomatic ventricular tachycardia. Less common causes of ventricular tachycardia include dilated or hypertrophic cardiomyopathy, valvular heart disease (including mitral valve prolapse), inherited ion channel abnormalities, and drug toxicity. Hypoxia, alkalosis, and electrolyte abnormalities, especially hyperkalemia, exacerbate the propensity for ventricular ectopy and tachycardia.

Torsade de pointes occurs when repolarization is delayed, and cardiac after-potentials can initiate this form of polymorphic ventricular tachycardia. Delayed repolarization is seen in inherited (congenital long QT syndrome) and acquired (primarily drug toxicity) circumstances.[52] Torsade de pointes often occurs in bursts of up to 30 cycles before spontaneously stopping. Sustained torsade de pointes is uncommon and can potentially degenerate into ventricular fibrillation.

It is a misconception that patients with ventricular tachycardia are clinically unstable. Ventricular tachycardia cannot be reliably differentiated from supraventricular tachycardia with aberrant conduction on the basis of clinical symptoms or vital signs. Differentiation using the 12-lead ECG can be challenging and, at times, impossible (see discussion in next section).

Treatment

The management of ventricular tachycardia is based on the patient's clinical stability. The most extreme form of instability is cardiac arrest; these patients should receive prompt electrical defibrillation, chest compressions, and other advanced life support measures. Patients with a pulse but compromised by the ventricular tachycardia should undergo electrical cardioversion coupled with administration of a procedural analgesia and sedation if clinical status allows time to administer. In the stable patient, pharmacologic agents are first-line therapy.[53]

Drug therapy for ventricular tachycardia includes procainamide, amiodarone, lidocaine, and magnesium. Procainamide is an effective agent for stable ventricular tachycardia in patients with preserved left ventricular dysfunction.[14] Based on limited human comparative studies, procainamide is superior to lidocaine for converting patients with stable ventricular tachycardia.[14] The primary disadvantage of procainamide is the relatively slow dosing because rapid infusions can cause hypotension.

For patients with tenuous hemodynamic status, amiodarone is the antiarrhythmic of choice with lidocaine as the less effective alternative. If pharmacologic interventions are unsuccessful, then sedation-assisted electrical (synchronized) cardioversion is the next option.

Magnesium is primarily used as an antiarrhythmic agent in patients with known hypomagnesemia, QT interval prolongation, polymorphic ventricular tachycardia, or torsade de pointes.[54] Case reports describe the benefits of isoproterenol or phenytoin in torsade de pointes, but controlled studies are lacking.[55-57] Definitive treatment of torsade de pointes is ventricular pacing at a rate necessary to prevent the bursts of polymorphous ventricular tachycardia. Pacing is continued until the cause of QT prolongation (drug toxicity, electrolyte abnormality) is ameliorated.

UNDIFFERENTIATED WIDE-COMPLEX TACHYCARDIA

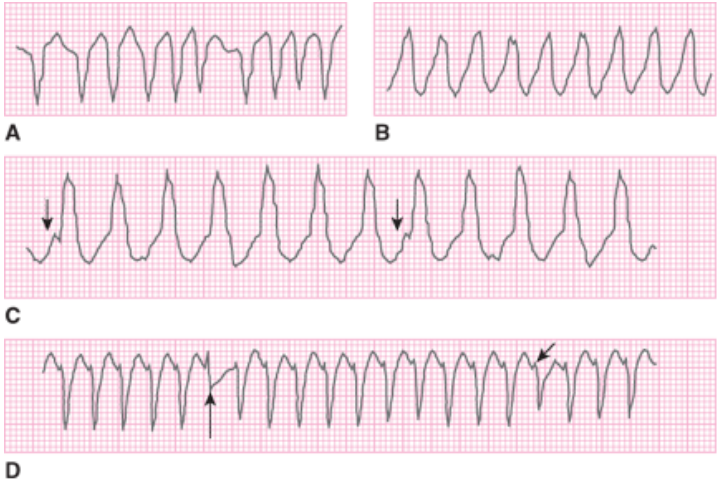
Description

A regular, wide-complex tachycardia can be either of ventricular origin (ventricular tachycardia) or due to a supraventricular tachycardia with aberrant conduction of the electrical impulse through the ventricles. The differentiation between ectopic beats of ventricular origin and those of aberrantly conducted supraventricular origin can be difficult, more so in sustained tachycardias with widened QRS complexes.

Clinical Features

Traditional teaching emphasizes five electrocardiographic features as helpful in differentiating ventricular tachycardia from supraventricular tachycardia with aberrant conduction: irregularity, AV dissociation, fusion and capture beats, QRS duration, and QRS complex concordance (**Figure 18–26**).[5,7] Ventricular tachycardia is usually very regular; if irregular, the degree of irregularity is minimal (Figures 18–23A and 18–26C). Marked irregularity is strongly suggestive of atrial fibrillation with bundle-branch block; of course, torsade de pointes is also irregular, but the changing polarity of the QRS complex is usually quite apparent.

FIGURE 18–26.
ECG considerations in the distinction of supraventricular tachycardia with aberrant ventricular conduction from ventricular tachycardia. **A**. Irregular rhythm in Wolff-Parkinson-White–related atrial fibrillation. **B**. Regular rhythm in ventricular tachycardia. **C**. Atrioventricular dissociation in ventricular tachycardia. The *arrows* indicate P waves, indicative of atrioventricular dissociation, which is strongly suggestive of ventricular tachycardia. **D**. Capture (*large arrow*) and fusion (*small arrow*) beats that are indicative of ventricular tachycardia.



AV dissociation, where the atria and ventricles have separate independent pacemakers (Figure 18–26C), if noted, is strongly suggestive of ventricular tachycardia. In cases of ventricular tachycardia without retrograde conduction to the atria, the SA node continues to initiate atrial depolarization. Because atrial depolarization is completely independent of ventricular activity, the resulting P waves will be dissociated from the QRS complexes. AV dissociation is not common; it is noted in only approximately 10% of ventricular tachycardia patients.

Capture and fusion beats (Figure 18–26D) are potentially useful and suggest ventricular tachycardia. In the patient with ventricular tachycardia, an independent atrial impulse may occasionally cause ventricular depolarization via the normal conducting system; such a supraventricular impulse, if conducted and able to trigger a depolarization within the ventricle, will result in a different QRS complex morphology—different than the other wide QRS complex beats. If the resulting QRS complex occurs earlier than expected and is narrow, the complex is called a capture beat; the supraventricular impulse electrically captures the ventricle, producing a narrow complex. The presence of capture beats strongly supports a diagnosis of ventricular tachycardia. Fusion beats occur when a sinus beat conducts to the ventricles via the AV node and joins, or fuses, with a ventricular beat originating from the abnormal ectopic focus. These two electrical "beats" combine, resulting in a QRS complex of intermediate width and differing morphology when compared to the other beats of ventricular tachycardia. As with capture beats, the presence of fusion beats is strongly suggestive of ventricular tachycardia. Fusion and capture beats occur infrequently and are seen in fewer than 10% of patients with ventricular tachycardia.

Ventricular tachycardia usually has a wider QRS complex than does supraventricular tachycardia with aberrancy, so extreme QRS durations >160 milliseconds argue in favor of ventricular tachycardia. Concordance describes that the polarity of the QRS complexes is consistent across the precordium, either all negative or positive from leads V_1 to V_6. Negative concordance, with all the precordial QRS complexes having a negative or downward deflection, suggests ventricular tachycardia.

In addition to these traditional characteristic, features of the QRS complex on the 12-lead ECG have been analyzed as differentiators of ventricular tachycardia versus supraventricular tachycardia with aberrancy. Since 1991, five different algorithms have been created, studied, and compared (**Table 18-25**).[58-62] The approach varies: the Brugada, Griffith, and Lau approaches use multiple leads and criteria; the Vereckei algorithm uses only lead aVR, although with four criteria; and the Pava algorithm only uses lead II and only one criterion. The Griffith approach is reversed from the other four; it assumes that the wide-complex tachycardia is ventricular in origin and then looks for evidence that favors supraventricular with aberrancy. Published comparisons are mixed regarding the sensitivity and specificity of these different algorithms, and neither has proven consistently superior.[63-67] Simplicity favors the Vereckei or Pava approaches.

Table 18–25

Methods for Wide-Complex Tachycardia Differentiation

Author and Year	Favors Ventricular Tachycardia (VT)	Favors Supraventricular Tachycardia with Aberrant Conduction (SVTAC)
Brugada, 1991[58]	VT diagnosed if, analyzed in sequence, any one of these 4 criteria is present: Absence of RS complexes in all precordial leads R to S interval >100 ms in one or more precordial leads AV dissociation Morphologic criteria for VT in leads V_1 and V_6 (V_1: monophasic R, qR, QS, or RS; and V_6: rS, QS, qR, or S > R)	SVTAC diagnosed if no to all 4 criteria for VT
Griffith, 1994[59]	VT diagnosed if no to either criteria for SVTAC	SVTAC diagnosed if both criteria are present: QRS morphology classic for bundle-branch block: LBBB (rS or QS in leads V_1 and V_2; time to S wave nadir in lead V_1 or V_2 >70 ms; R wave and no Q wave in V_6) *or* RBBB (rSR' in lead V_1; RS in lead V_6; R wave larger than S wave in V_6) No atrioventricular dissociation is seen
Lau, 2000[60]	Bayesian analysis starting with pretest odds for VT then multiply by likelihood ratio for VT associated with each of these factors: QRS duration QRS frontal plane axis V_1 morphology with a RBBB pattern V_1 and V_2 morphology with a LBBB pattern Interval of initial R-wave deflection in V_6 V_6 morphology VT diagnosed if posttest odds >3	SVTAC diagnosed if posttest odds <0.33

Author and Year	Favors Ventricular Tachycardia (VT)	Favors Supraventricular Tachycardia with Aberrant Conduction (SVTAC)
Vereckei, 2008[61]	VT diagnosed if, analyzed in sequence, any of these 4 criteria are present in lead aVR: Initial R wave Initial r or q wave >40 ms Notch present on the initial descending limb of a predominately negative QRS Slow conduction at beginning of QRS: ratio of vertical distance travelled in voltage during the initial 40 ms (v_i) and terminal 40 ms (v_t); ratio of v_i/v_t <1	SVTAC diagnosed if none of the 4 criteria for VT are present
Pava, 2010[62]	VT diagnosed if time from isoelectric to peak of R wave in lead II is >50 ms	SVTAC diagnosed if not

Abbreviations: LBBB = left bundle-branch block; RBBB = right bundle-branch block.

Treatment

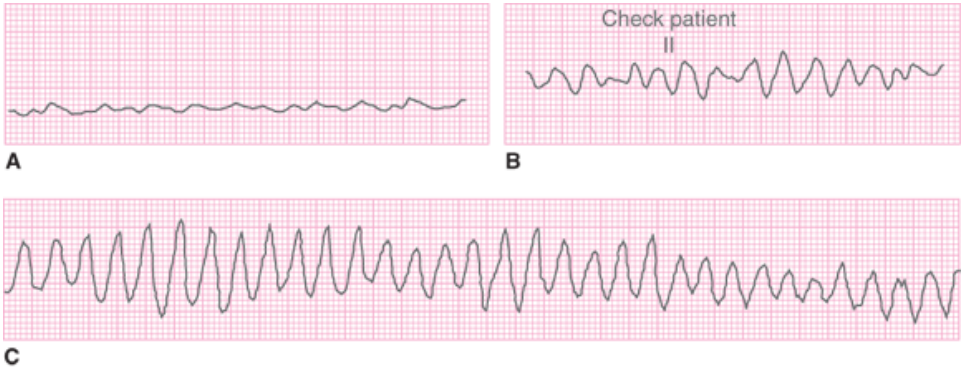
When in doubt, it is safer to assume a new and symptomatic wide-complex tachycardia is ventricular in origin than the converse. Maintain a focus on treating the entire patient, not solely the ECG. If rapid treatment is needed, use IV amiodarone. In stable patients, response to adenosine can be helpful in differentiating ventricular from supraventricular wide-complex tachycardias.[68] Not all wide-complex tachycardias require antiarrhythmic agents or urgent electrical therapy. Treatment of the causative syndrome is often the most appropriate action.

VENTRICULAR FIBRILLATION

Description

Ventricular fibrillation is disorganized depolarization and chaotic contraction of small areas of ventricular myocardium absent any effective mechanical cardiac activity; there is no cardiac output. The ECG of ventricular fibrillation shows a fine, irregular pattern without discernible P waves or organized QRS complexes. The irregular pattern itself can be coarse, intermediate, or fine in amplitude (**Figure 18–27**).

FIGURE 18–27.
Three examples of ventricular fibrillation. **A**. Coarse amplitude. **B**. Fine amplitude. C. Coarse amplitude mimicking ventricular tachycardia.



Clinical Significance

Ventricular fibrillation is seen most commonly in patients with severe ischemic heart disease, with or without an acute myocardial infarction. Primary ventricular fibrillation occurs suddenly and without preceding hemodynamic deterioration, whereas secondary ventricular fibrillation occurs after a prolonged period of left ventricular failure and/or circulatory shock. In addition, direct stimulation of the myocardium by catheters or other instruments, electrocution, and direct blunt chest trauma (also known as *comotio cordis*) can induce ventricular fibrillation.

Treatment

Treatment of pulseless ventricular tachycardia or fibrillation is with electrical defibrillation along with chest compressions and other advanced life support measures. If resuscitation is successful at restoring spontaneous circulation, address any dysrhythmic triggers including acute ischemia, metabolic derangements, or toxicologic insult.

DYSRHYTHMIA ASSOCIATED CONDUCTION ABNORMALITIES

WOLFF-PARKINSON-WHITE SYNDROME

Description

WPW syndrome is a form of ventricular preexcitation involving an accessory conduction pathway that bypasses the AV node and creates a direct electrical connection between the atria and ventricles.[69] WPW patients are prone to a variety of supraventricular tachydysrhythmias (**Table 18-26**). Ventricular fibrillation is very rare and usually only occurs in the setting of therapeutic misadventure.

Table 18–26

Dysrhythmias Seen in Symptomatic Wolff-Parkinson-White Syndrome

Atrioventricular reciprocating tachycardia (AVRT)
Narrow QRS complex tachycardia (orthodromic AVRT) – 65%
No P wave
Narrow QRS complexes
No delta wave
Rapid and regular
Ventricular rates ranging from 160–220 beats/min
Difficult to distinguish from paroxysmal supraventricular tachycardia
Wide QRS complex tachycardia (antidromic AVRT) – 10%
No P wave
Widened QRS complexes with consistent QRS complex morphology
Delta wave
Rapid and regular
Ventricular rates ranging from 160–220 beats/min
Difficult to distinguish from ventricular tachycardia
Atrial fibrillation – 25%
No P wave
Widened QRS complexes with varying, bizarre QRS complex morphologies
Delta wave
Rapid and irregular
Ventricular rates consistently >200 beats/min

Clinical Features

The classic triad of ECG findings (**Figure 18–28** and **Table 18-27**) seen in WPW is visible during sinus rhythm and is usually not detectable during tachydysrhythmias. The PR interval is shortened in sinus rhythm because the impulse moving

through the accessory pathway is not subject to the physiologic slowing within the AV node. The ventricle is activated by two separate pathways, resulting in a fused, or widened, QRS complex. The initial part of the complex, the delta wave, represents aberrant activation through the accessory pathway, while the terminal portion of the QRS complex represents normal activation through the His-Purkinje system from impulses traveling through the AV node. Due to altered ventricular depolarization, secondary repolarization changes reflected in altered ST segments and T waves that are generally directed opposite (discordant) to the major delta wave and QRS complex. The delta wave may create Q waves that can mimic ECG changes associated with ischemic heart disease.

FIGURE 18–28.
A. A 12-lead ECG of patient with the Wolff-Parkinson-White (WPW) syndrome in sinus rhythm. **B** and **C**. Single P-QRS-T complex; note the shortened PR interval, delta wave, and widened QRS complex. **D**. The direction of cardiac impulse conduction in the WPW syndrome patient in sinus rhythm. The *two arrows* indicate the direction of the impulse, moving from atrial (A) tissues to ventricular (V) tissues via the accessory pathway (AP) and atrioventricular node (AVN). The impulse arrives at the ventricular tissues via the AVN and moves through ventricular tissues via the right bundle branch (RBB) and left bundle branch (LBB); the impulse also arrives at ventricular tissues via the AP and moves through ventricular tissues using cell-to-cell conduction. SAN = sinoatrial node.

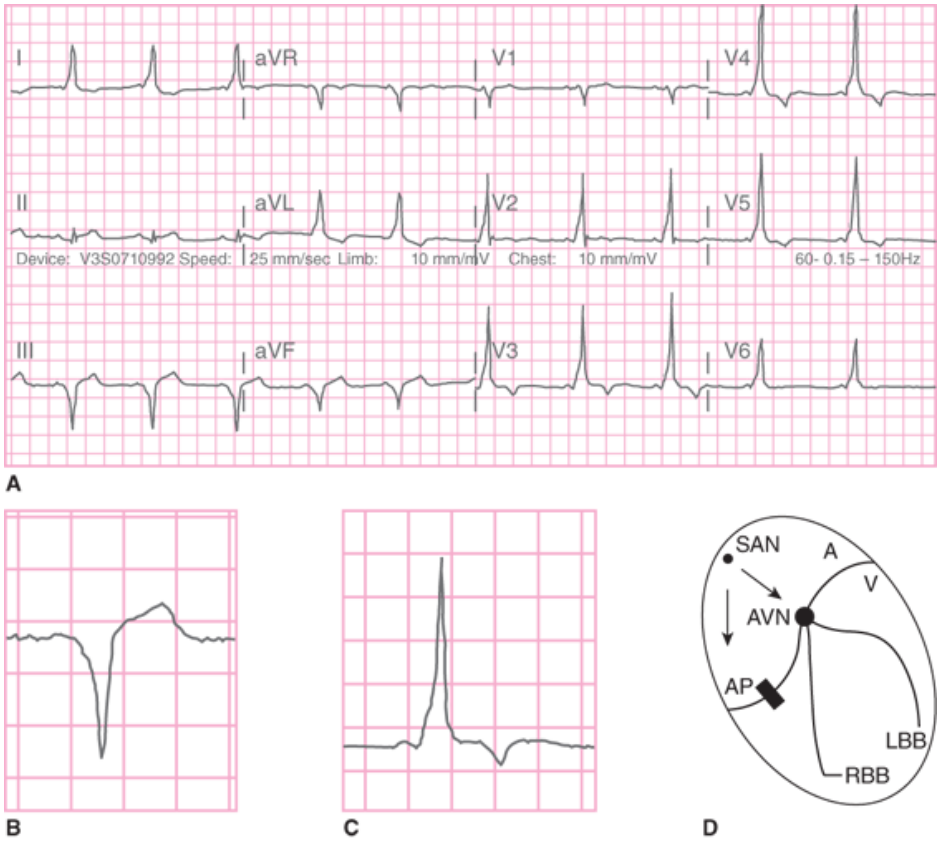


Table 18–27

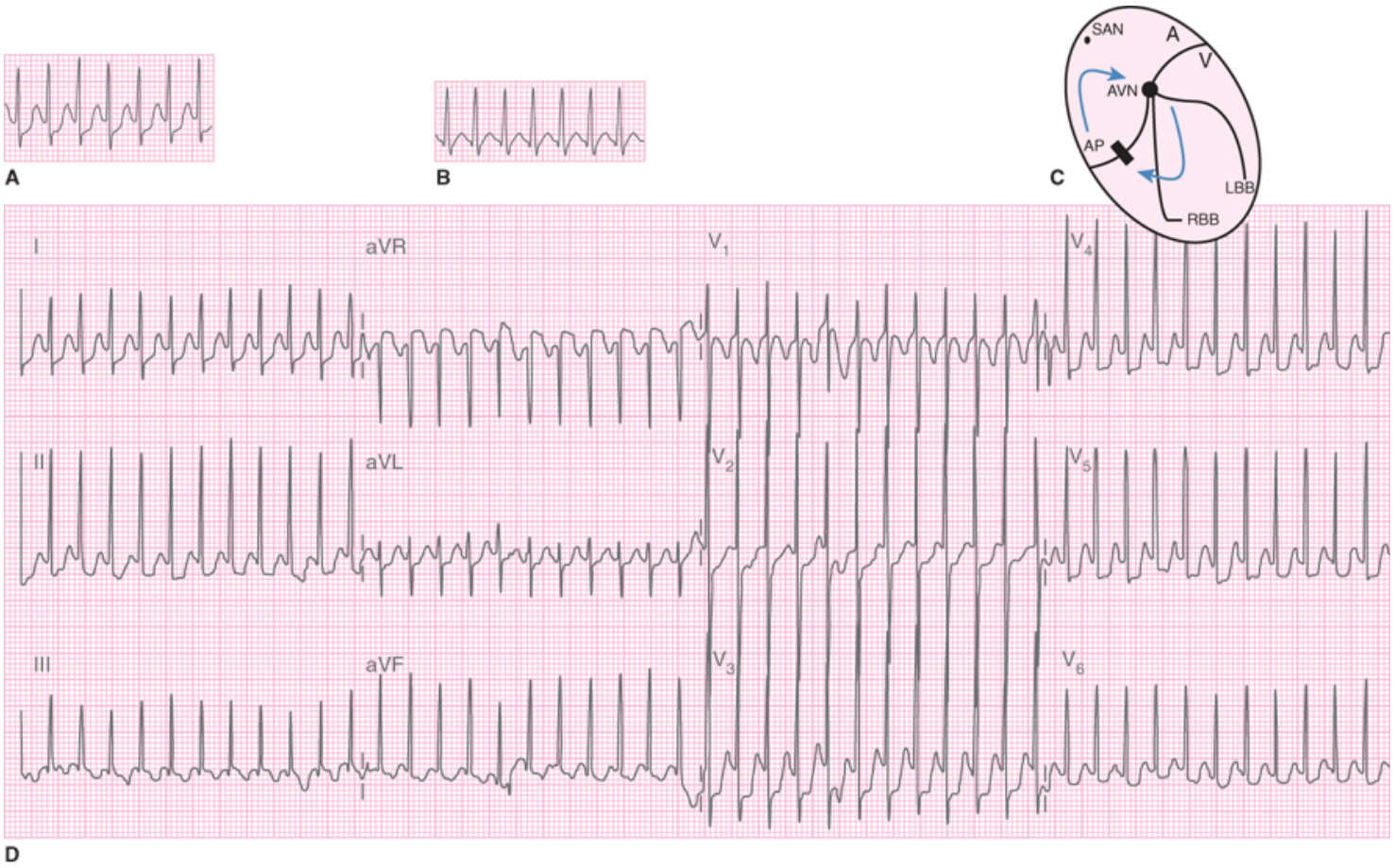
The ECG Triad of Wolff-Parkinson-White Syndrome During Sinus Rhythm

PR interval <120 ms
Slurring of the initial QRS complex, known as a delta wave
Widened QRS complex

An incidental ECG may identify WPW in an otherwise asymptomatic individual. While such asymptomatic individuals will never develop tachydysrhythmias, a portion of patients do, and some rare patients experience ventricular fibrillation and cardiac arrest.[70] Electrophysiologic studies can identify patients with multiple accessory tracts and short refractory periods in those tracts and, thus, who are at increased risk for ventricular fibrillation and cardiac arrest.[71]

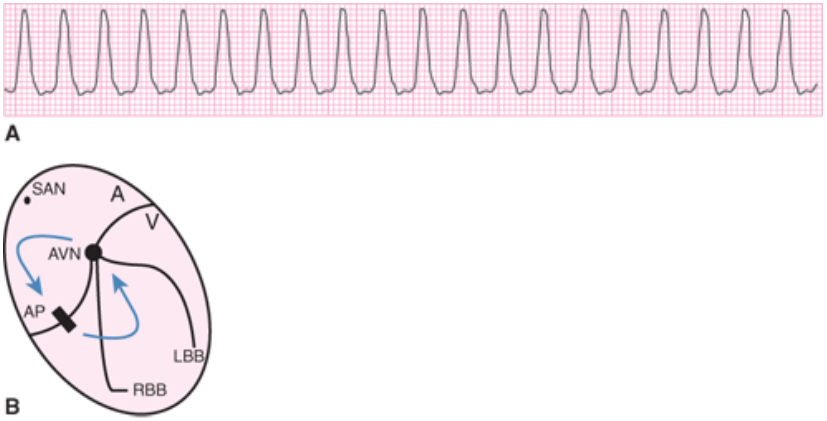
The most frequent dysrhythmia seen in the WPW patient is a reentrant tachycardia termed AV reciprocating tachycardia. In AV reciprocating tachycardia, the reentry circuit involves the AV node and the accessory pathway. AV reciprocating tachycardia is either orthodromic (anterograde conduction through the AV node) or antidromic (retrograde conduction through the AV node). During orthodromic or anterograde AV reciprocating tachycardia (**Figure 18–29** and Table 18-27), the atrial stimulus is conducted to the ventricle through the AV node with a return of the impulse back to the atria through the accessory pathway. Since the ventricles are stimulated solely via the normal His-Purkinje system, the QRS complexes are narrow and there is no delta wave. Orthodromic AV reciprocating tachycardia accounts for approximately 65% of dysrhythmias seen in WPW patients and is difficult to distinguish from AV nodal reentrant tachycardia (i.e., typical paroxysmal supraventricular tachycardia).

FIGURE 18–29.
Narrow-complex tachycardia (orthodromic atrioventricular [AV] reciprocating tachycardia [AVRT]), the most common dysrhythmia of the Wolff-Parkinson-White syndrome. **A** and **B**. Rapid, regular, narrow QRS complexes without delta waves. **C**. The direction of cardiac impulse conduction in orthodromic AVRT; anterograde from atrial (A) tissues to ventricular (V) tissues through the AV node (AVN) and returning to the atrial tissues from ventricular tissues via the accessory pathway (AP). **D**. A 12-lead ECG demonstrating orthodromic AVRT. AP = accessory pathway; LBB = left bundle branch; RBB = right bundle branch; SAN = sinoatrial node.



In approximately 10% of symptomatic WPW patients, an antidromic (retrograde) AV reciprocating tachycardia occurs (**Figure 18–30** and Table 18-26). In antidromic AV reciprocating tachycardia, the reentrant circuit conducts in the opposite direction, with anterograde conduction down the accessory pathway and return of the impulse retrograde to the atria via the AV node. With this pathway, the QRS complexes are wide; the ECG displays a very rapid, wide-complex tachycardia that looks like ventricular tachycardia.

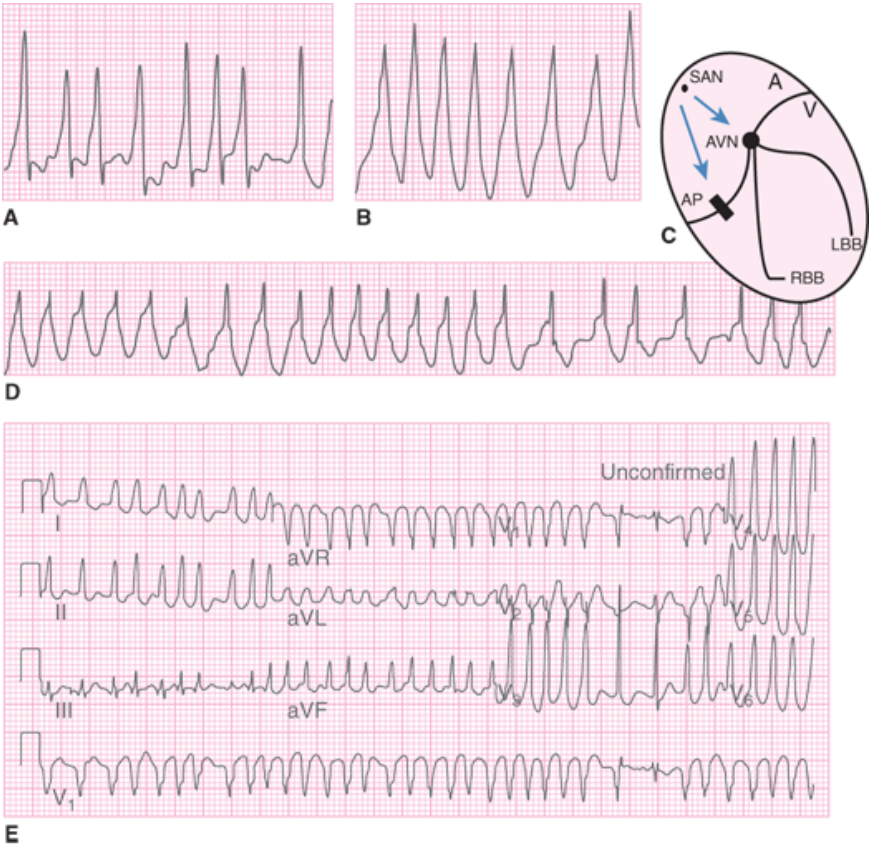
FIGURE 18–30.
Antidromic atrioventricular (AV) reciprocating tachycardia (AVRT; wide-complex tachycardia). **A**. Wide-complex tachycardia that mimics ventricular tachycardia. **B**. The direction of cardiac impulse conduction in a Wolff-Parkinson-White syndrome patient during antidromic AVRT. The *two arrows* indicate the direction of the impulse movement, anterograde from atrial (A) tissues to ventricular (V) tissues through the accessory pathway (AP) and returning to the atrial tissues from ventricular tissues via the AV node (AVN). LBB = left bundle branch; RBB = right bundle branch; SAN = sinoatrial node.



Atrial fibrillation occurs in up to 25% of WPW patients with symptomatic dysrhythmias. The electrocardiographic features (**Figure 18–31** and Table 18-26) of atrial fibrillation with WPW syndrome are a very fast ventricular rate due to the ability of the accessory pathway to conduct impulses into the ventricle faster than through the AV node and significant beat-to-beat variation in the QRS complex morphology resulting from a combination of the two impulses arriving at the ventricle and fusing to form a composite depolarization. This wide-complex dysrhythmia may appear relatively regular and can be misdiagnosed as ventricular tachycardia.[72]

FIGURE 18–31.

Atrial fibrillation in Wolff-Parkinson-White (WPW) syndrome. **A**, **B**, and **D**. Rapid, wide, irregular QRS complex tachycardia with varying QRS complex morphologies. **C**. The direction of cardiac impulse conduction in a WPW syndrome patient during atrial fibrillation. The *two arrows* indicate the direction of the impulse movement, both anterograde from atrial (A) tissues to ventricular (V) tissues through both the accessory pathway (AP) and the atrioventricular node (AVN). The larger of the two arrows indicates that the majority of impulses travel to the ventricular via the AP. **E**. A 12-lead ECG demonstrating a rapid, wide, irregular QRS complex tachycardia with varying QRS complex morphologies. LBB = left bundle branch; RBB = right bundle branch; SAN = sinoatrial node.



Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
www.accessmedicine.com

Treatment

Treat all three tachydysrhythmias in the WPW patient according to the patient's clinical stability and the electrocardiographic features of the dysrhythmia. Use electrical cardioversion in patients with hemodynamic instability.

In the stable patient, therapy is guided by the QRS complex width and the regularity of the rhythm. In patients with a regular, narrow QRS complex tachycardia (i.e., orthodromic AV reciprocating tachycardia), the first intervention is vagal maneuver (Table 18-3). If this intervention fails, the next step would be intravenous adenosine (Table 18-2). If adenosine fails, then longer acting AV nodal blocking agents, such as β-blockers and calcium channel blockers are indicated. In refractory cases, procainamide is an effective agent that blocks conduction in the accessory conduction pathway and can terminate this reentrant tachycardia. If all medications are unsuccessful, the patient can be electrically cardioverted with appropriate sedation.

In stable patients with a wide QRS complex tachycardia, either regular (i.e., orthodromic AV reciprocating tachycardia) or irregular (atrial fibrillation), procainamide should be administered.[73] If unsuccessful, electrical cardioversion with appropriate sedation should be considered. Some antiarrhythmic medications can paradoxically enhance condition via the accessory tract, potentially enabling excessively rapid atrial activity to trigger ventricular fibrillation. Also, by blocking conduction through the AV node in WPW patients with atrial fibrillation, more of the rapid atrial depolarizations are able to initiate ventricular depolarization because competition from the normal conducting pathway is impaired, although evidence of adverse clinical outcome by such action is weak.[74] **Agents that can enhance conduction in the accessory tract and/or block conduction in the AV node should be avoided in WPW patients with wide-complex irregular tachycardias; these include adenosine, amiodarone, β-blockers, and calcium channel blockers.[20]**

After conversion, observe the patient with continuous cardiac rhythm monitoring and a repeat ECG. In general, patients who have rare episodes of tachydysrhythmias and who are stable after conversion can be discharged home with routine follow-

up. Patients with frequent tachydysrhythmia episodes or who have an episode of atrial fibrillation with a very rapid ventricular response should be promptly referred for ablation of the accessory pathway. Patients who experience loss of consciousness during a known or possible tachydysrhythmia should be kept for observation due to the potential for repeat episode or cardiac arrest. Asymptomatic patients in whom WPW appears as an incidental finding should be referred to a cardiologist for further evaluation.[75]

BRUGADA SYNDROME

Description

Brugada syndrome is an inherited disorder of myocardial depolarization that predisposes young individuals to malignant ventricular dysrhythmias and sudden death.[76-78] Congenital Brugada syndrome is due to mutations in the genes responsible for transmembrane sodium, calcium, or potassium ion channels in the heart.[79] Eight genetic mutations have been identified as producing a *channelopathy* that results in the Brugada syndrome. The prevalence of Brugada syndrome varies dramatically among different ethnic groups; it is highest in East and Southeast Asian populations and relatively lower in groups originating from Western Europe.

Clinical Features

The majority of patients with Brugada syndrome are asymptomatic and are only found via an incidental ECG. Symptomatic patients present with palpitations, near to complete syncope, or seizures due to ventricular tachydysrhythmias. Characteristic ECG changes are necessary for the diagnosis, but such changes are variable and not always evident (**Table 18-28** and **Figure 18–32**).[80] Fever and provocative testing with a sodium channel blocker (e.g., flecainide) may provoke surface ECG abnormalities associated with Brugada syndrome. The type 1 pattern is considered diagnostic when combined with appropriate clinical or family history (**Table 18-29**). Types 2 and 3 are considered suggestive but not diagnostic of Brugada syndrome. Because types 2 and 3 may convert to type 1, either spontaneously or with provocative testing, further evaluation is recommended if there is an appropriate history.

Table 18–28

ECG Patterns Associated with Brugada Syndrome in ECG Leads V_1 to V_3

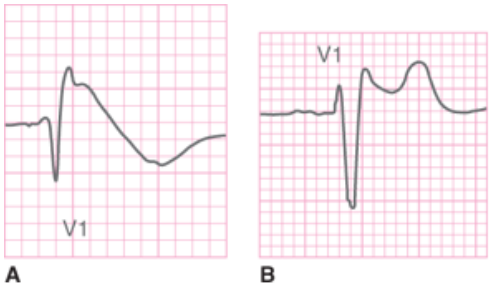
Type 1	Coved-shaped ST-segment elevation >2 mm Followed by an inverted T wave
Type 2	ST segment elevation >2 mm With a trough in the ST segment at least 1 mm deep Followed by a positive or biphasic T wave Producing a "saddleback" pattern
Type 3	Coved-shaped or saddleback pattern ST segment With 1- to 2-mm elevation

Table 18–29

Risk Stratification in Brugada Syndrome

Personal history	Near or complete syncope Seizure Nocturnal agonal respiration
Aborted sudden cardiac death	Polymorphous ventricular tachycardia Ventricular fibrillation
Family history	Family history of unexplained sudden cardiac death Family history of coved-shaped ST segment (type 1) ECG pattern
Electrophysiologic study	Inducibility of ventricular tachycardia or fibrillation with programmed stimulation

FIGURE 18–32.
Brugada ECG pattern. **A**. Type 1. **B**. Type 2.



Risk stratification is done to identify patients with Brugada syndrome at significant risk for malignant ventricular tachydysrhythmias.[81] In patients with aborted sudden cardiac death, the risk of recurrence of ventricular fibrillation is over 50% within 5 years.

Treatment

An important role for the emergency provider is to recognize this ECG pattern, especially in patients who present with symptoms, and to refer to for appropriate follow up.[82] Avoid exacerbating the underlying pathophysiology; do not use medications that possess sodium channel blockade effects (http://www.brugadadrugs.org). Educate the patient on the importance of treating fever, with even minor infections. The only proven therapy to terminate malignant ventricular arrhythmias and prevent sudden death is implantable cardioverter defibrillator placement. Quinidine, a class Ia antiarrhythmic, is sometimes used as an adjunct with implantable cardioverter defibrillator placement to reduce the incidence of dysrhythmias. Side effects (hepatitis, thrombocytopenia, and diarrhea) and the potential to induce torsade de pointes are cautions to quinidine's routine use.

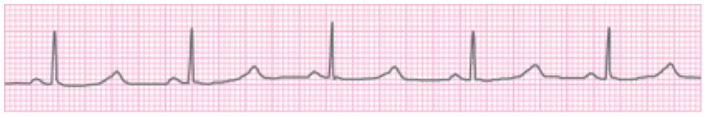
LONG QT SYNDROME

Description

The long QT syndromes are characterized by prolongation of the QT interval (**Figure 18–33**) and a predisposition to ventricular tachydysrhythmias, including torsade de pointes and sudden cardiac death.[83-85] Congenital long QT syndrome is an inherited disorder due to mutations in cation channel genes (*channelopathies*). Of the 13 described variants of

congenital long QT syndrome, the three most common, accounting for about 90% of all cases, are mutations in a cardiac potassium channel for long QT syndrome types 1 and 2 and the sodium ion channel for long QT syndrome type 3.[85] With increased awareness and identification, the prevalence of congenital long QT syndrome is estimated to be 1 in 2000 live births. Acquired long QT syndrome can occur from electrolyte abnormalities (hypokalemia, hypomagnesemia), medication effects, and other disease states, such as acute coronary syndrome and severe left ventricular dysfunction.[86,87]

FIGURE 18–33.
Long QT interval; QTc is 550 milliseconds.



Clinical Features

The normal QT interval corrected for heart rate (QTc) is up 440 milliseconds in adult males and 460 milliseconds in adult females. The risk of dysrhythmias associated with long QT syndrome increases as the QTc prolongs, with moderate risk when the QTc is between 480 and 499 milliseconds and significantly higher risk when the QTc is greater than 500 milliseconds.[27] Syncope is the most common symptom, and torsade de pointes is the most common dysrhythmia. A diagnostic scoring system has been created to assist in identifying patients with congenital long QT syndrome (**Table 18-30**).[88]

Table 18–30

Diagnostic Score for Congenital Long QT Syndrome

Feature		Points
ECG*		
QTc	≥480 ms	3
	460–479 ms	2
	450–459 (male) ms	1
QTc ≥480 ms at 4th min of recovery from exercise stress test		1
T-wave alternans		1
Notched T waves in three ECG leads		1
Heart rate <2nd percentile for age (<50 in adult age 20–60)		0.5
Clinical history		
Syncope *or* episode of torsade de pointes (not both)	Torsade de pointes	2
	Syncope provoked by stress	2
	Syncope not provoked by stress	1
Congenital deafness		0.5
Family history		
Family members with definite long QT syndrome		1
Immediate family member <30 years old with unexplained sudden cardiac death		0.5
Scoring: Range, 0 to 10; 1.5 to 3 points = intermediate probability; ≥3.5 points = high probability		

* In absence of medications or conditions known to affect QT interval or T wave.

Treatment

An important role for the emergency provider is to recognize this ECG pattern, especially in patients who present with syncope, and to refer for appropriate follow-up.[82] Avoid exacerbating the underlying pathophysiology; do not use medications that possess channel blockade effects, impair cardiac repolarization, prolong the QT interval, and provoke tachydysrhythmias (https://crediblemeds.org/healthcare-providers/).[89] Correct underlying electrolyte abnormalities, especially those of potassium and magnesium. β-Blockers are the initial treatment for symptomatic patients with congenital

long QT syndrome.[90] Propranolol and nadolol are the most effective β-blockers, and patients with congenital long QT syndrome type 1 are the most responsive.

Lifestyle modifications are beneficial in congenital long QT syndrome.[91] Exercise is a trigger for lethal dysrhythmic events, especially in patients with congenital long QT syndrome type 1, where swimming is notably dangerous. Patients with congenital long QT syndrome type 2 are sensitive to decreased serum potassium and are at risk for lethal cardiac events triggered by emotions or being startled, as being roughly aroused from rest or sleep.

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