Patients with cardiac arrhythmias often present to the emergency department. The patient’s clinical presentation determines the urgency with which the assessment and management should proceed. Patients with serious signs and symptoms (ie, shock, hypotension, congestive heart failure (CHF), severe shortness of breath, altered level of consciousness, ischemic chest pain, or acute myocardial infarction) require immediate treatment. With stable patients, more time is afforded for review of the 12-lead electrocardiogram (ECG) and rhythm strip to diagnose the cardiac arrhythmia. Review of available prior ECGs may also assist in arrhythmia diagnosis.

**ELEVEN HELPFUL HINTS FOR EMERGENCY DEPARTMENT ARRHYTHMIAS**

1. Obtain as much information as available. Always look at all 12 leads and be sure of name, date, age, correct lead placement, and standardization.
2. Know what each lead looks like normally (Figure 35–1); eg, lead I (and usually lead II and aVF) should look like the textbook PQRST except no Q wave. In lead I, the P, QRS, and T should all be upright, the intervals should be normal and the PR and ST baselines should be isoelectric.
3. A regular tachycardia with a rate close to 150 should prompt a search for atrial flutter.
4. Precise diagnosis of wide complex tachycardias (WCTs) can be difficult. If ventricular rate is irregular consider atrial fibrillation (AF) or atrial flutter with variable conduction and underlying bundle branch block (BBB).
5. Do not rely on computer readings. They may or may not be correct.
6. Single-lead rhythm strips may not have enough information. If time permits, always obtain a 12-lead ECG.
7. You cannot have too many ECGs. Serial ECGs are important. Sinus tachycardia rates tend to change over time.
8. Arrhythmias are common in acute ST elevation myocardial infarctions.
9. Tachyarrhythmias are divided into narrow or wide QRS width and then into regular or irregular.
Figure 35-1. Normal ECG.
10. Arrhythmia classifications and terminologies can be confusing and they change as new information becomes available.

11. If the heart rhythm is slow and the patient is hypotensive with signs of poor perfusion, assume transthoracic or transvenous pacing will be needed.

A NOTE ON CARDIOVERSION AND DEFIBRILLATION

No consensus exists on correct pad positioning and current ACLS guidelines endorse both the conventional or sternal apical positioning (one pad on the superior–anterior right chest just below the level of the clavicle and one pad on the inferolateral left chest) and the anteroposterior (the anterior pad as in the conventional method and the posterior pad on the right or left upper back). However, some authors feel that anteroposterior placement with the anterior pad over the right atrium and the posterior pad at the tip of the left scapula optimizes cardioversion of atrial tachyarrhythmias while placement of the anterior pad over the ventricles and posterior pad again at the tip of the left scapula works well for ventricular arrhythmias. All currently manufactured defibrillators use biphasic waveforms so unless you are using an older machine, the energy setting will range from 0 Joules (J) to 200 J. All energy doses mentioned in this chapter will be for biphasic defibrillators. In addition to disease-specific energy recommendations, there are device-specific recommendations for the different biphasic defibrillator models for first shock energy dose in some situations. Notably, in ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) the initial shock is 120 for devices using a rectilinear waveform and 150–200 J for devices using a truncated exponential waveform ranging from 120 to 200 J. ACLS guidelines recommend that IF THE OPERATOR IS UNSURE of device-specific recommendations then the defibrillator’s highest energy level should be used in this setting; this will be 200 J for all biphasic units and 360 J if you happen to still have a monophasic unit. The bottom line is that if you are uncertain on the energy dose in any emergent situation where electricity is required for an adult your best bet is turn the energy up as high as it will go as even maximal doses of energy are felt to be relatively safe.

TACHYARRHYTHMIAS

Immediate synchronized cardioversion should be performed on all unstable patients with tachyarrhythmias. The specific arrhythmia diagnosis (supraventricular or ventricular) does not need to be made immediately because initial management is the same. Patients with polymorphic ventricular tachycardia (PMVT) of 30 seconds or more and all unstable patients should be treated with immediate defibrillation.

In stable patients, the initial medical management will be guided by the underlying rhythm and a detailed history and physical examination. In recent years, the more traditional approach to categorize patients as either stable or unstable has been modified. Hemodynamically stable patients can be further subdivided into those with preserved or impaired cardiac function. Findings of impaired cardiac function in a patient who is otherwise stable may alter the pharmacologic treatment.

SUPRAVENTRICULAR ARRHYTHMIAS

1. Sinus Tachycardia

Clinical Findings

(See Appendix, Figure 35–3.) Sinus tachycardia occurs when the sinus rate is faster than 100 beats/min. Usually the rate is 101–160 beats/min. Young, healthy adults can accelerate their heart rate up to 180–200 beats/min, particularly during exercise. Young children have been noted to have sinus rates up to 220 beats/min. Sinus tachycardia should not be viewed as a primary arrhythmia but more as a response to an underlying illness or condition. It is often normal in infancy and early childhood but can occur as a result of a number of conditions including pain, fever, stress, hyperadrenergic states, anemia, hypovolemia, hypoxia, myocardial ischemia, pulmonary edema, shock, and hyperthyroidism. Certain medications and illicit drugs can also cause tachycardia. The P wave in sinus tachycardia should have a positive axis in the frontal plane, ie, the P wave should be positive in lead I and aVF.

Treatment and Disposition

The treatment of sinus tachycardia is directed at the underlying cause. This may include correction of dehydration with intravenous fluids, analgesic or antipyretic administration, or supplemental oxygen to correct hypoxia. Treatment aimed at correcting the heart rate rather than the underlying condition may be harmful if the tachycardia is compensatory and is supporting the cardiac output. Gradual slowing of the heart rate with treatment of the underlying condition or during carotid sinus massage may help to differentiate sinus tachycardia from other supraventricular arrhythmias. Adenosine administration with a 12-lead rhythm strip is helpful in differentiating from other causes of tachyarrhythmias. Further management, including the need for hospitalization, depends on the underlying condition.

2. Paroxysmal Supraventricular Tachycardia

Clinical Findings

(See Appendix, Figures 35–6 to 35–11.) Paroxysmal supraventricular tachycardia (PSVT) is a general term that refers to a number of tachyarrhythmias that arise from above the bifurcation of the His bundle. Approximately 90% of these arrhythmias occur as a result of a reentrant mechanism; the remaining 10% occur as a result of increased automaticity.
Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common form of PSVT, accounting for 50–60% of cases. The heart rate is usually 180–200 beats/min and is characterized by sudden onset and sudden termination. Because the reentrant mechanism occurs within the AV node itself, virtually simultaneous excitation of the atria and ventricles occurs. As a result, the P waves occur concurrent with the QRS complexes and are difficult to visualize on the ECG. Often, patients with AVNRT do not have underlying heart disease. Common precipitating factors include alcohol, caffeine, and sympathomimetic amines. Patients with AVNRT usually present in their third or fourth decade of life, and the majority (approximately 70%) are female.

Atrioventricular reciprocating tachycardia (AVRT) accounts for 30% of PSVT. In most cases, the impulse travels down the AV node and follows a retrograde path up the accessory bypass tract. Because activation of the ventricles occurs through normal conduction pathways, the accessory pathway is concealed, and the QRS morphology is normal. Consider AVRT if the heart rate is faster than 200 beats/min or if P waves are seen following the QRS complex.

Sinus node reentry and intraatrial reentry are uncommon causes of PSVT, accounting for approximately 5% of cases. In these arrhythmias, the heart rate is usually 130–140 beats/min. More often, patients with these arrhythmias have underlying heart disease.

Automatic atrial tachycardia is another uncommon arrhythmia, accounting for less than 5% of cases of PSVT. The heart rate is usually 160–250 beats/min but may be as slow as 140 beats/min. In this case, the underlying mechanism is increased automaticity rather than reentry. Automatic atrial tachycardia is commonly associated with underlying heart disease. This arrhythmia is difficult to treat and may be refractory to standard measures including cardioversion.

PSVT can be classified as AV nodal dependent or independent. This strategy may prove useful in formulating treatment options. AVNRT and AVRT are AV nodal dependent, meaning that the AV node is involved in the reentrant circuit. For these rhythms, pharmacologic management is designed to decrease conduction through the AV node.

**Treatment**

**A. Unstable Patients**

Patients with PSVT who are hemodynamically unstable require immediate synchronized DC cardioversion. Current recommendations are to start with low-energy levels (50–100 J) and then to increase the initial dose by 50 J as needed until sinus rhythm is restored. If clinical circumstances permit, administer intravenous sedatives. Avoid the common error of delaying emergency cardioversion to perform other patient care activities. If immediate cardioversion is unavailable, physical maneuvers that cause vagal stimulation can be attempted.

Adenosine, β-blocker, or calcium channel blocker may be administered.

**B. Stable Patients**

Tachycardia associated with PSVT is usually well tolerated unless the patient has underlying heart disease or left ventricular dysfunction.

1. **Physical maneuvers**—In stable patients, physical maneuvers causing vagal stimulation can be attempted prior to medication administration. Maneuvers that stimulate the vagus nerve such as the Valsalva maneuver (expiration against a closed glottis), Mueller maneuver (deep inspiration against a closed glottis), cold water facial immersion, and carotid sinus massage are at times effective in terminating PSVT that results from AV nodal and sinoatrial (SA) nodal dependent mechanisms. Perform carotid sinus massage only after auscultation for carotid bruits.

2. **Pharmacologic treatment**—If vagal stimulation is contraindicated or ineffective, adenosine is considered first-line medical therapy for conversion of PSVT. In general, pharmacologic agents with AV nodal blocking properties such as adenosine, β-blockers, calcium channel blockers, and digoxin are used for the acute management and prevention of AV nodal dependent PSVT. Other antiarrhythmic agents, such as procainamide and amiodarone, which exert effects at various levels of the cardiac conduction system are used for the management and prevention of AV nodal independent PSVT. Antiarrhythmic medications may be considered for conversion of PSVT when AV nodal blocking agents are unsuccessful.

**a. Adenosine**—Adenosine is an endogenous nucleoside that slows conduction through the AV node and is successful in terminating more than 90% of PSVTs resulting from AV nodal reentry mechanisms (AVNRT and AVRT). Adenosine may also be effective in terminating sinus node reentry tachycardia but is usually ineffective in terminating automatic atrial tachycardia. Often adenosine will cause a transient AV block, briefly exposing the underlying atrial activity. Administration of a medication with more prolonged effect on the AV node (β-blockers or calcium channel blockers) may provide a more sustained reduction in ventricular rate.

Administer adenosine rapidly, and follow each dose immediately with a 20-cc saline flush. Although current recommendations are to administer an initial intravenous dose of 6 mg over 1–3 seconds repeated at 2 and 4 minutes with 12-mg doses if this does not terminate the PSVT, many clinicians choose to forgo the initial 6-mg dose and will increase the dose to 18-mg if the 12-mg dose does not produce AV...
blockade. The 18-mg dose has been shown to be both safe and effective. Common side effects include unexplainable feeling of impending doom, facial flushing, hyperventilation, dyspnea, and chest pain. These side effects are often transient owing to the short half-life of adenosine (less than 5 seconds). Prewarning to the patient of these symptoms is helpful. The effects of adenosine are antagonized by caffeine and theophylline and potentiated by dipyridamole and carbamazepine. Heart transplant patients may be overly sensitive to the effects of adenosine; if necessary, use smaller doses. Because adenosine can provoke bronchospasm, use caution if it is being administered to patients with a history of reactive airway disease.

Adenosine can also be administered to a stable patient with a wide QRS complex tachycardia suspected to be supraventricular in origin. Adenosine is preferred over calcium channel blockers in patients with hypotension or impaired cardiac function and in patients concomitantly receiving β-adrenergic blocking agents.

b. β-BLOCKING AGENTS—β-blockers such as metoprolol or esmolol slow SA node impulse formation and slow conduction through the AV node. These medications should be used with caution in patients with a history of severe reactive airway disease and CHF.

Metoprolol is an alternative to calcium channel blockers, and is administered intravenously at a dose of 5 mg every 5 minutes for three doses. Esmolol is an ultrashort-acting β-blocker that has the advantage of a brief half-life (~10 minutes) and a rapid onset of action. Administer a loading dose of 0.5 mg/kg over 1 minute. This is followed by a maintenance infusion of 50 μg/kg/min. If the response is inadequate, another dose of 0.5 mg/kg can be administered after 4 minutes and the maintenance infusion increased to 100 μg/kg/min. When heart rate control is achieved, reduce the maintenance infusion to 25 μg/kg/min.

c. CALCIUM CHANNEL BLOCKERS—Calcium channel blockers such as diltiazem or verapamil are effective in converting PSVT to sinus rhythm. The efficacy of diltiazem and verapamil in terms of conversion rates, rapidity of response, and safety profile appear similar. These medications decrease SA and AV node conduction and cause prolongation of the AV node refractory period. Calcium channel blockers also decrease myocardial contractility and peripheral vascular resistance. Use calcium channel blockers with caution in patients with left ventricular dysfunction or CHF. Avoid these medications in patients with WCT of unknown origin, ventricular tachycardia (VT), or tachycardia with ventricular preexcitation. Hypotension is the most concerning side effect of intravenous administration and occurs in 10–15% of patients.

Verapamil—The initial dose of verapamil is 5–10 mg administered intravenously over 1–2 minutes. Additional doses of 5–10 mg can be administered every 15 minutes as needed until the desired effect is achieved or a total of 30 mg has been administered.

Diltiazem—The initial dose of diltiazem is 0.25 mg/kg administered intravenously over 2 minutes (20 mg for the average adult). If necessary, a dose of 0.35 mg/kg can be administered in 15 minutes. After conversion, a maintenance infusion can be started at 5–10 mg/h and can be increased to a maximum of 15 mg/h if needed.

The choice between β-blockers and calcium channel blockers depends on multiple factors, but both should not be given intravenously to the same patient. Both have rapid onset (minutes) and both should be used with caution in severe COPD and severe CHF. Medication that the patient is currently taking and physician preference are considerations. In patients with hyperthyroidism and congenital heart disease, β-blockers are the best choice.

d. DIGOXIN—Digoxin administration will increase vagal tone while reducing sympathetic activity. As a result, conduction through the AV node is slowed. Digoxin may be administered as an intravenous bolus dose of 0.5 mg. Additional doses of 0.25 mg may be given as needed every 4–6 hours, with a total dose not to exceed 1.25 mg in 24 hours. The immediate benefit of digoxin is lessened by its slow onset of action. When used in combination, digoxin may allow for lower doses of subsequently administered antiarrhythmic agents. Avoid digoxin in patients with AF with ventricular preexcitation.

e. AMIODARONE—Amiodarone is a class III antiarrhythmic agent with sodium- and potassium-channel blocking properties and β-blocking and calcium channel blocking properties. By virtue of its β-blocking and calcium channel blocking properties, amiodarone slows conduction through the AV node. In patients with impaired cardiac function or CHF, treatment options narrow. Amiodarone has a solid safety profile and may be an effective alternative agent in this situation. Amiodarone can be administered as a slow intravenous infusion of 150 mg over 10 minutes. This is followed by a maintenance infusion of 1 mg/min for 6 hours and then 0.5 mg/min. Additional bolus doses of 150 mg can be repeated as needed for resistant or recurrent PSVT up to a total daily dose of 2 g.

f. PROCAINAMIDE—Procainamide is a class IA antiarrhythmic agent with sodium channel blocking properties. Procainamide will slow conduction through both the AV node and, if present, an accessory bypass tract. Procainamide can be considered for patients with PSVT refractory to AV nodal blocking agents. The recommended loading dose of procainamide is 17 mg/kg administered as a slow intravenous infusion at a rate of 20–30 mg/min (1 g for an average adult). Stop the initial infusion if the arrhythmia is suppressed, hypotension develops, or the QRS complex widens by more than 50% of its original duration. After arrhythmia suppression, start a maintenance infusion at 1–4 mg/min.
Hospitalization should be considered for patients in PSVT with accompanying serious signs and symptoms, patients requiring emergency cardioversion, patients in PSVT with ventricular preexcitation, and patients with arrhythmias refractory to standard treatment. Outpatient follow-up care should be provided for the otherwise healthy patient with a transient episode of PSVT converted to sinus rhythm in the emergency department.

### 3. Atrial Fibrillation

#### Clinical Findings

(See Appendix, Figures 35–12 and 35–13.) In AF, the atrial rate is disorganized and is 300–600 beats/min. AF is characterized by an irregularly irregular ventricular rate with the absence of discernible P waves.

AF is the most common sustained cardiac arrhythmia in adults. It is estimated that AF affects more than 2 million persons in the United States; its prevalence increases with age, approaching 10% in those older than 80 years. AF can occur in the absence of underlying heart disease or may be associated with a number of conditions, including chronic hypertension, valvular disease, cardiomyopathy, myocardial ischemia, myocarditis, pericarditis, or congenital heart disease. AF may also occur in the presence of other systemic disorders, including hyperthyroidism, pulmonary embolism, hypoxia, and excess consumption of alcohol or caffeine.

Patients with nonvalvular AF have approximately a 5% annual incidence of stroke as a result of a thromboembolic event. This risk increases fourfold in patients with mitral stenosis and increases dramatically in older patients, approaching 30% in patients aged 80–89 years.

#### Treatment

Acute management of AF includes ventricular rate control and prevention of thromboembolic complications. Additional management considerations include restoration and maintenance of sinus rhythm.

**A. Unstable Patients**

Patients in AF with a rapid ventricular response who are hemodynamically unstable require immediate synchronized DC cardioversion. Recommendations are to start between 100 and 200 J biphasic and then to increase the dose in stepwise fashion as needed until sinus rhythm is restored.

**B. Stable Patients**

In stable patients with a rapid ventricular response, the initial goal is rate control. This can usually be achieved with β-blockers, calcium channel blockers, or digoxin. β-blockers may prove most helpful in patients with hyperthyroidism but are relatively contraindicated in patients with acute decompensated CHF. Diltiazem and verapamil can often slow the ventricular rate and have the added benefit of antianginal effects and blood pressure control in hypertensive patients. In more than 90% of patients, a reduction in heart rate of at least 20% is noted. Diltiazem appears to be safe for use in patients with mild CHF. Digoxin can also help control the ventricular rate in patients with AF and may be useful in patients with left ventricular dysfunction. Its slower onset of action as compared to other agents makes it less useful for acute rate control. In patients with mild to moderate CHF, the administration of amiodarone may prove useful. Intravenous amiodarone can also be considered an alternative agent for rate control when the above agents fail. The specific medication choice will often be dictated by the urgency of the situation, the medication profile, physician preference, and the patient’s underlying condition.

1. **Anticoagulants**—Prophylactic anticoagulation with warfarin has been shown to significantly reduce the incidence of stroke in patients with AF. If new-onset AF is of undetermined duration or greater than 48 hours duration, initiation of anticoagulation is necessary. Current recommendations include anticoagulation for 3 weeks, followed by elective cardioversion and then continued outpatient anticoagulation for four more weeks. An alternative strategy is initial anticoagulation with unfractionated or low-molecular-weight heparin followed by transesophageal echocardiography to evaluate the left atrial appendage for the presence of clot. If no clot is identified, the patient may safely undergo cardioversion, followed by anticoagulation for 4 weeks. If a left atrial appendage clot is identified by transesophageal echocardiography, recommendations include anticoagulation for 3 weeks, followed by cardioversion and then continued anticoagulation for four additional weeks. In patients with AF of less than 48 hours duration, anticoagulation is not recommended.

2. **Antiarrhythmics**—Various antiarrhythmic agents, including amiodarone, procainamide, and sotalol (class III), are used to chemically convert AF. Pharmacologic or electrical cardioversion may be considered in selected stable emergency department patients with AF of less than 48 hours duration. Remodeling, both anatomic and electrically, occurs soon after the onset of AF. Postponing cardioversion could lead to an increased resistance to attempts at conversion.

#### Disposition

Patients with chronic rate-controlled AF do not require hospital admission. In patients with new-onset AF, hospitalization is often required for ventricular rate control, initiation of anticoagulation, and sometimes for initiation of antiarrhythmic therapy. If a patient presents with thromboembolic complications, hospital admission will also be necessary.
4. Atrial Flutter
► Clinical Findings
(See Appendix, Figure 35–14) In atrial flutter, the atrial rate is usually 250–350 beats/min. It is the most common underdiagnosed tachyarrhythmia. Sawtooth flutter waves may sometimes be seen on ECG, but should not be relied upon. Typically, atrial flutter will present with 2:1 AV conduction. For this reason, it is important to consider atrial flutter in the differential diagnosis of a regular tachycardia at approximately 150 beats/min, even in the absence of flutter waves. Atrial flutter is most commonly identified as negative waves in II, III, and aVF with positive flutter waves in lead V1.

If atrial flutter is suspected, several options are available to better identify atrial activity. Vagal maneuvers or administration of adenosine with a 12-lead rhythm strip may unmask flutter waves.

► Treatment
Acute management of atrial flutter includes ventricular rate control and prevention of thromboembolic complications. Additional management considerations include restoration and maintenance of sinus rhythm.

A. Unstable Patients
Patients in atrial flutter with a rapid ventricular response who are hemodynamically unstable require immediate synchronized DC cardioversion. Current recommendations are to start with between 50 and 100 J biphasic and then increase the energy dose in stepwise fashion as needed until sinus rhythm is restored.

B. Stable Patients
In stable patients with a rapid ventricular response, the initial goal is rate control. Adequate heart rate control can be achieved with the administration of either β-blockers or calcium channel blockers. Digoxin is often less effective acutely because of its slow onset of action. Amiodarone and diltiazem are alternatives for rate control in the stable patient with impaired cardiac function or CHF.

The stroke risk for patients in atrial flutter is less than that of AF. The same anticoagulation guidelines exist for atrial flutter as in AF.

► Disposition
Patients with chronic rate-controlled atrial flutter do not require hospital admission. In patients with new-onset atrial flutter, hospitalization is often required for ventricular rate control, initiation of anticoagulation, and sometimes for initiation of antiarrhythmic therapy.

5. Multifocal Atrial Tachycardia
► Clinical Findings
(See Appendix, Figure 35–15) In multifocal atrial tachycardia (MAT) the heart rate is typically 100–130 beats/min. The characteristic ECG finding is at least three different P wave morphologies. The rhythm often appears irregular and can at times be confused with AF. Varying PR intervals may also be noted. When the rate is slower than 100 beats/min, the term wandering atrial pacemaker is applied. Unless underlying aberrant conduction is present, the QRS complexes are narrow. Severe underlying chronic obstructive pulmonary disease accounts for approximately 60–85% of cases. Theophylline and digoxin levels should be checked since toxicity of these drugs can cause MAT.

► Treatment
The initial treatment of MAT is directed at correcting the underlying cause. As with AF, the initial goal of therapy is to achieve heart rate control. Because MAT does not respond to electrical cardioversion, pharmacologic intervention may be required.

Magnesium may be effective in converting MAT and can be administered as a 2 g intravenous bolus over 1 minute. This is followed by a 2 g/h infusion for 5 hours. Magnesium can still be effective if serum magnesium levels are in the normal range. Potassium repletion may be helpful in patients who are hypokalemic.

Amiodarone, digoxin, or diltiazem may be considered as alternative agents for rate control, especially when the patient exhibits findings of CHF.

► Disposition
Patients may require hospitalization for MAT if the heart rate is difficult to control or for further management of the underlying condition.

6. Preexcitation Arrhythmias
► Clinical Findings
(See Appendix, Figures 35–11 and 35–13) Patients with Wolff-Parkinson-White (WPW) syndrome have an accessory pathway. Anatomical location varies and the pathways can be AV (Kent), atrio-His (James), intranodal, and nodoventricular (Mahain). On the ECG, a short PR interval (less than 120 ms) and the presence of a δ wave (initial upward slurring of the QRS complex) signify ventricular preexcitation.

A variety of arrhythmias may occur in patients with WPW syndrome; approximately 70% is orthodromic AVRT. In this case, the cardiac impulse travels down the AV node (antegrade conduction) and stimulates the ventricles through the normal conduction pathways. The accessory
AV bypass tract serves as the retrograde limb of the circuit. In the absence of aberrant ventricular conduction or a fixed BBB, the morphology of the QRS complex is narrow without evidence of ventricular preexcitation (absent δ wave). The bypass pathway is considered concealed if the short PR and δ wave are not present on the baseline ECG.

Rarely, antidromic AVRT occurs whereby the accessory AV pathway acts as the antegrade limb of the circuit and the AV node as the retrograde limb. Antidromic AVRT will produce a wide QRS complex tachycardia and may masquerade as VT. The tachycardia may be extremely rapid (with ventricular rate 220–300), leading to ventricular fibrillation (VF) as a result of an R-on-T phenomenon.

AF is the second most common arrhythmia associated with WPW syndrome. AF with ventricular preexcitation has a high potential to precipitate hemodynamic compromise. AF with a rapid ventricular rate is characterized by an irregular tachycardia and a wide QRS complex resulting from ventricular preexcitation.

**Treatment**

Patients with orthodromic AVRT who are hemodynamically unstable require immediate synchronized DC cardioversion. Current recommendations are to start between 50 J and 100 J biphasic and then to increase the initial dose in stepwise fashion as needed until sinus rhythm is restored. In patients with known WPW syndrome presenting with a narrow complex regular tachycardia, orthodromic AVRT can be assumed. In stable patients, the medical treatment will be the same as in AVNRT. Pharmacologic treatment with adenosine, β-adrenergic blocking agents, or calcium channel blockers can be administered as deemed necessary and appropriate for the individual case. In general, the treatment of orthodromic AVRT with AV nodal blocking agents is safe. The risk of enhancing antegrade conduction down the bypass tract is very low.

Treatment of AF with ventricular preexcitation (antidromic AVRT) is different from that of orthodromic AVRT. If the patient is hemodynamically unstable, immediate synchronized DC cardioversion starting at 100–200 J is warranted. The use of AV nodal blocking agents, specifically β-blockers, calcium channel blockers, and digoxin, is contraindicated. If conduction through the AV node is slowed, conduction down the accessory pathway may be enhanced, possibly degenerating to VF. Because procainamide will slow conduction through both the AV node and the accessory pathway, it is the medication of choice when AF with a rapid ventricular response is associated with ventricular preexcitation. Procainamide is also the medication of choice in antidromic AVRT. Amiodarone can be used as an alternative agent in treating AF with ventricular preexcitation and findings of CHF.

**Disposition**

Hospitalization is not required for patients who are asymptomatic with evidence of ventricular preexcitation on the ECG (sinus rhythm, short PR, and a δ wave). Consider hospitalizing patients who have serious signs and symptoms or those requiring cardioversion. In addition, hospitalization is recommended for patients with AF and ventricular preexcitation or antidromic AVRT. Patients who present with stable orthodromic AVRT may be discharged with close outpatient follow-up after pharmacologic conversion in the emergency department.

**VENTRICULAR ARRHYTHMIAS**

1. **Ventricular Tachycardia**

**Clinical Findings**

(See Appendix, Figures 35–16 and 35–17) Ventricular tachycardia is the most common cause of wide QRS complex tachycardia. The term VT is used when six or more consecutive ventricular beats occur. The ventricular rate is usually 150–220 beats/min, although rates slower than 120 beats/min may occur. Nonsustained VT is characterized by an episode lasting less than 30 seconds. Sustained VT is characterized by an episode lasting longer than 30 seconds, associated with hemodynamic compromise, or requiring therapeutic intervention for termination. WCT refers to a regular tachycardia with a QRS complex greater than 0.12 seconds (120 ms) in duration. WCT most often occurs as a result of either VT or SVT with aberrant conduction (underlying or rate-dependent BBB).

In more than 75% of patients presenting in the emergency department with regular WCT, the underlying arrhythmia is VT. The presence of structural heart disease, coronary artery disease, prior myocardial infarction, or CHF strongly suggests VT. Certain ECG findings favor VT over SVT with aberrant conduction. These findings include a QRS complex wider than 160 ms, the presence of fusion beats, and evidence of AV dissociation. AV dissociation occurs in about 20% of patients with VT and confirms the diagnosis (this is usually seen with ventricular rates less than 150). A common clinical error that must be avoided is to assume that
WCT is SVT with aberrant conduction. All cases of WCT of unknown origin should be managed as VT.

Electrical storm is a somewhat rare but well described entity that consists of recurrent ventricular tachycardia, usually with an implanted defibrillator that discharges repeatedly. Patients with this condition have a high mortality and will likely need sedation as well as sympathetic blockade to control the recurrent dysrhythmias. Anti-arrhythmics use is usually required and IV amiodarone is the drug of choice.

Ten Tips for the Diagnosis of Regular WCT

1. A WCT is most likely VT.
2. Consider toxicity—always think of hyperkalemia, tricyclic antidepressants, and digoxin. Treatment is different and cardioversion is not helpful.
3. If unstable, treat immediately with cardioversion.
4. Ask two questions: Prior MI? Tachycardia new since MI? Answering yes increases likelihood of VT to >90%.
5. Twelve-lead ECG is always best, if possible, before, during, and after treatment. Save all tracings.
6. Old ECGs are invaluable when looking for similar BBB patterns.
7. There are many algorithms for determining VT (vs SVT with BBB, aberrancy) and none are 100% accurate. The rules are difficult to remember and interpret. VT is likely if the following are identified:
   a. RS absent in all precordial leads (seen in less than 25% of VT). If cannot find RS (only QS, QR, monophasic R, or rSR complexes) this favors VT.
   b. Onset of R to nadir of S >.10 ms in any pre-cordial lead.
   c. AV disassociation.
   d. Fusion beats, capture beats.
   e. Concordance—all positive or all negative pre-cordial lead deflections.
   f. Frontal plane QRS axis—usually abnormal.
   g. If RBBB-like, then look for monophasic R or RSR’ in V1 and for R/S <1 in V6. If LBBB-like, then look for wide R (>30 ms), onset of R to nadir of S > 100 ms in V1 or V2, and QR or QS in V6.
8. If still unsure treat for VT.
9. Best treatment is cardioversion.
10. Stabilize rhythm before admission.

Treatment

A. Unstable Patients

Patients with VT or WCT of unknown origin who are hemodynamically unstable with serious signs and symptoms require immediate synchronized DC cardioversion. Recommendations are to start with 50–100 J and then increase the initial dose by 50 J as needed until sinus rhythm is restored.

B. Stable Patients

Traditionally, patients with stable VT are administered an antiarrhythmic agent for chemical cardioversion. A number of medications are available. The choice for a particular patient is often based on physician preference and experience, findings of preserved or impaired cardiac function, and the underlying cause of the VT.

1. Lidocaine—Lidocaine is a class Ib antiarrhythmic with sodium channel blocking properties. Because it can be administered rapidly with few side effects, some authors consider it the agent of choice for ventricular arrhythmias associated with acute myocardial ischemia or infarction. The recommended intravenous loading dose is 1.0–1.5 mg/kg. If required, a second bolus dose of 0.75–1.5 mg/kg can be administered in 5–10 minutes. If ventricular ectopy persists, an additional bolus dose of 0.5–0.75 mg/kg can be administered every 5–10 minutes to a maximum dose of 3 mg/kg. After rhythm suppression, start a maintenance infusion at 2–4 mg/min. Lidocaine has the lowest incidence of toxicity of all currently used antiarrhythmic medications.

2. Other drugs—Procainamide is an alternative agent to lidocaine for the treatment of stable monomorphic VT. Amiodarone may be preferable to other antiarrhythmic agents for VT in patients with CHF. Although recommended, amiodarone’s efficacy may not be fast enough for use on an emergency basis.

Disposition

Hospitalization is recommended for all patients who present with VT.

2. Polymorphic Ventricular Tachycardia (Including Torsades de Pointes)

Clinical Findings

(See Appendix, Figure 35–18) Polymorphic ventricular tachycardia is a form of VT with varying QRS complex morphology. The rhythm is often irregular and hemodynamically unstable, and it can degenerate to VF.

Torsades de pointes is a form of PMVT associated with a prolonged QT interval on the baseline ECG. The rhythm is often described as having a twisting-on-point appearance and can be either paroxysmal or sustained. The heart rate is usually 200–250 beats/min. Hereditary long QT syndromes associated with torsades de pointes include Lange-Nielsen syndrome and Romano-Ward syndrome. Torsades de pointes may also occur as a result of numerous medication interactions. A complete list of medications that have been reported to prolong the QT interval is available at www.qtdrugs.org.
PMVT can also occur in the absence of a prolonged QT interval. In this case, cardiac ischemia or underlying structural heart disease is often the cause.

**Treatment and Disposition**

Patients with PMVT who are hemodynamically unstable with serious signs and symptoms require immediate cardioversion or defibrillation. Recommendations are to start with 200 J. To prevent recurrence, discontinue all agents that can prolong the QT interval.

Magnesium is the medication of choice for the management of torsades de pointes associated with congenital and acquired forms of long QT syndrome. It may be effective even when serum levels are normal. A 2-g intravenous dose can be administered as a slow push over 5 minutes. Follow the bolus dose by a maintenance infusion of 1–2 g/h. Consider supplemental potassium as an adjunctive therapy to maintain serum potassium levels in the high normal range. Temporary transvenous pacing at rates around 100 beats/min may be useful to prevent recurrences, especially in patients with bradycardia or pauses. Hospitalization is recommended for all patients who present with PMVT.

### 3. Ventricular Fibrillation

**Clinical Findings**

(See Appendix, Figure 35–19) Ventricular fibrillation is characterized by an irregular ventricular rhythm with no discernible distinction between the QRS complex, ST segment, and T waves. VF is a common cause of sudden cardiac death and remains a significant contributor to mortality in the first 24 hours after an acute myocardial infarction. In the absence of early bystander cardiopulmonary resuscitation and initiation of advanced cardiac life support, including defibrillation, survival rates are poor.

**Treatment and Disposition**

Witnessed VF or pulseless VT, is treated with immediate treatment is asynchronous defibrillation followed by CPR for 2 minutes before rhythm check. If VT or pulseless VT persists, repeat defibrillation followed by either epinephrine or vasopressin and continued CPR for 2 minutes. If VT or pulseless VT still persists again repeat defibrillation followed by either amiodarone or lidocaine and CPR for 2 minutes. All patients who have been successfully resuscitated from VF or pulseless VT should be started on a drip of the last antiarrhythmic administered and admitted to the intensive care unit for close observation. If an acute coronary syndrome is suspected as the cause of the arrest, the patient may require cardiac catheterization for evaluation and treatment. Chapter 9 offers a more in-depth discussion of the management of cardiac arrest.
CARDIAC ARRHYTHMIAS

CHAPTER 35

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by PP intervals that gradually shorten while the PR interval remains constant. This cycle terminates with a blocked P wave. The length of the pause is shorter than twice the preceding PP cycle.

3. Second-degree Sinoatrial Block (Mobitz Type II)

Second-degree Mobitz type II SA block is characterized by fixed pauses. On the ECG, the PP interval remains constant and is then followed by a blocked P wave. The PP interval, including the blocked P wave, will be twice the length of the normal PP interval.

4. Third-degree Sinoatrial Block

Third-degree SA block may be difficult to distinguish from sinus arrest. Patients with either conduction disturbance present with variable pauses on the ECG until an escape rhythm occurs or sinus rhythm is restored.

5. Sick Sinus Syndrome

Clinical Findings

Sick sinus syndrome is a manifestation of sinus node dysfunction. Patients with the syndrome may present with a wide range of bradyarrhythmias. Numerous arrhythmias are associated with sick sinus syndrome, including marked sinus bradycardia, sinus pause, sinus arrest, and SA block. On occasion, patients may also present with ventricular or atrial tachyarrhythmias.

Treatment and Disposition

Treatment may be indicated when pauses of more than 2–3 seconds occur or if the patient is symptomatic. Administration of atropine or initiation of temporary cardiac pacing may be required. Symptomatic patients will require hospital admission, often for permanent pacemaker placement.

ATRIOVENTRICULAR BLOCK

AV block refers to a group of conduction disturbances within the AV junctional tissue. In general, AV block is characterized by prolonged conduction time or a failure to conduct impulses through the AV node. The conduction disturbance can be partial (first- or second-degree AV block) or complete (third-degree AV block). In general, the hemodynamic effects will depend on the ventricular rate and the presence of underlying heart disease. AV conduction blocks are traditionally classified as first-, second-, or third-degree heart block.

1. First-degree Atrioventricular Block

(See Appendix, Figure 35–25) First-degree AV block is the most common conduction disturbance and is characterized by a PR interval that is prolonged for greater than 0.2 seconds. In general, the PR interval is constant, and each atrial impulse

SINUS BRADYCARDIA

Clinical Findings

(See Appendix, Figure 35–4) Sinus bradycardia occurs when the sinus rate is slower than 60 beats/min. Usually the rate is 45–59 beats/min, but on rare occasion it may be as slow as 35 beats/min. Sinus bradycardia is commonly associated with sinus arrhythmia and is often a normal finding in young, healthy, athletic individuals. Sinus bradycardia is often benign and does not necessarily indicate sinus node dysfunction. Although commonly physiologic, sinus bradycardia may be pathologic when patients experience symptoms of cerebral hypoperfusion or when the heart rate does not increase appropriately with activity or exercise. Certain underlying conditions have been associated with a slowing of the heart rate, including hypothermia, hypothyroidism, and increased intracranial pressure. In addition, a number of different medications, including β-blockers, calcium channel blockers, clonidine, digoxin, and lithium, can cause bradycardia.

Treatment and Disposition

Usually no treatment is required for asymptomatic sinus bradycardia. When serious signs and symptoms are present, medical management, pacemaker placement, and hospital admission are indicated.

SINUS ARREST

Sinus arrest is defined as a failure of sinus node impulse formation. On the ECG, random periods of absent cardiac activity may be noted. Unless escape beats occur, lengthy pauses are noted. When pauses occur, patients may complain of dizziness or lightheadedness or may have syncope. If untreated, pauses longer than 2.5 seconds may progress to asystole.

SINOATRIAL BLOCK

SA block differs from sinus arrest in that SA block is a form of exit block rather than failure of impulse formation. Like sinus arrest, SA block may occur as a result of a number of conditions, including acute myocardial infarction, myocarditis, fibrosis of the SA node, excessive vagal tone, and digoxin toxicity. Analogous to AV block, SA block can be classified into first-, second-, and third-degree heart block.

1. First-degree Sinoatrial Block

First-degree SA block does not produce any ECG changes. The diagnosis can be made only through electrophysiologic testing.

2. Second-degree Sinoatrial Block (Mobitz Type I)

(See Appendix, Figure 35–24) Second-degree Mobitz type I SA block, also known as SA Wenckebach, is characterized

ATRIAL VELOCITY BLOCK
is conducted to the ventricles. First-degree AV block can be a normal variant in young or athletic individuals due to excessive vagal tone. First-degree AV block is also common in elderly patients without underlying heart disease. It may occur in patients with myocarditis, mild digoxin toxicity, and inferior wall myocardial infarction secondary to AV nodal ischemia.

2. Second-degree Atrioventricular Block (Mobitz Type I)

(See Appendix, Figure 35–26) Second-degree Mobitz type I AV block is also known as Wenckebach AV block. This type of block is characterized by a progressive lengthening of the PR interval followed by a nonconducted P wave leading to a dropped QRS complex. Classically, the PP interval remains constant except when sinus arrhythmia is present. The RR interval will have a characteristic cycle throughout the conduction disturbance. The RR interval that includes the blocked P wave is the longest in duration. This is then followed by RR intervals that subsequently become shorter until the next P wave is blocked.

On a rhythm strip, grouped beating is often evident and can further help distinguish second-degree from third-degree AV block. The blocked P waves may occur frequently or periodically, and may or may not occur with regularity. Because Mobitz type I AV block is at the level of the AV node, the QRS complex is normal in configuration unless aberrant ventricular conduction or an underlying BBB exists. In general, Mobitz type I AV block does not usually produce hemodynamically significant symptoms. It can be seen in patients with acute myocardial infarction (usually inferior wall) and does not commonly progress to complete heart block (CHB). If CHB does occur, the escape rhythm pacemaker is usually located in the AV junctional tissue and is often fast enough to maintain an adequate cardiac output.

3. Second-degree Atrioventricular Block (Mobitz Type II)

(See Appendix, Figure 35–28) Second-degree Mobitz type II AV block is characterized by a constant PR interval, either normal or prolonged, that is followed by a nonconducted P wave. In Mobitz type II AV block, the QRS complex is usually wide. This occurs because Mobitz type II AV block represents an infranodal block. At times, every other P wave is blocked. This is described as 2:1 AV conduction. When this occurs, one cannot distinguish between Mobitz type I or type II AV block (see Appendix, Figure 35–27). Mobitz type II AV block is common in patients with acute myocardial infarction (usually anterior wall) and can suddenly progress to CHB resulting in syncope.

4. Third-degree Atrioventricular Block (Complete Heart Block)

Clinical Findings

(See Appendix, Figures 35–29 to 35–31) Third-degree AV block, or CHB, is characterized by independent atrial and ventricular activity. As a result of complete AV block, no atrial impulses are conducted through the AV node. The ventricular rate is determined by the intrinsic escape rhythm, AV junctional escape (usually 45–60 beats/min), or an idioventricular escape rhythm (usually 30–40 beats/min). The atrial rate may be sinus in origin or may be from an ectopic atrial focus. In CHB the atrial rate is typically faster than the ventricular rate. As noted with second-degree AV block, the hemodynamic consequences depend on the ventricular rate and the presence of underlying heart disease. Syncope or CHF commonly accompany acute acquired CHB. Complete AV block is most commonly caused by coronary artery disease or by degeneration of the cardiac conduction system.

Treatment

A. Unstable Patients

Emergency cardiac pacing is indicated for patients with hemodynamically unstable bradycardia, especially for patients who have failed medical therapy, patients with malignant escape rhythms, and patients in bradyasystolic arrest. Transcutaneous cardiac pacing is the initial intervention because of its ease of application, compared to temporary transvenous pacing. In unstable patients, medical management can be initiated, although at times its utility is only temporary.

B. Stable Patients

1. Atropine—Atropine is an anticholinergic medication with parasympatholytic properties leading to enhanced SA node automaticity and AV node conduction. The initial intravenous dose of atropine is 0.5–1.0 mg, which can be repeated every 5 minutes to a total dose of 0.04 mg/kg (3 mg for the average adult). The maximal dose produces complete vagal blockade. Atropine is recommended for, but not limited to, patients with symptomatic bradycardia or relative bradycardia, bradycardia with malignant escape rhythms, and asystole.

Rarely, a paradoxical reduction in heart rate has been observed in patients with advanced AV block after administration of atropine. Therefore, use atropine with caution in patients with infranodal AV block (Mobitz type II, and CHB with wide QRS complexes). Other rarely encountered side effects of atropine administration include worsening of cardiac ischemia in patients with an acute myocardial infarction, or the development of a ventricular tachyarrhythmia. These adverse effects are uncommon, but knowledge of such responses may assist with proper patient selection. Atropine is not effective in the management of the heart transplant patient with symptomatic bradycardia because of surgical denervation of the vagus nerve.

2. Isoproterenol—Isoproterenol is a nonspecific β-adrenergic agonist that causes an increase in heart rate and cardiac contractility. The combined effects lead to increases in cardiac output and systolic blood pressure and decreases...
in systemic and pulmonary vascular resistance and diastolic blood pressure. As a result, no significant change in mean arterial pressure occurs. Myocardial oxygen demand is increased as a result of the increased heart rate and contractility. In addition, isoproterenol causes smooth muscle relaxation and bronchodilation. Isoproterenol may be used to treat symptomatic bradycardia in heart transplant patients. The initial intravenous dose of isoproterenol is 1 μg/min, titrated slowly until the desired hemodynamic effects are achieved. The maximum infusion rate is 4 μg/min.

3. Dopamine—Dopamine is an endogenous catecholamine with dose-related effects. At doses of 3.0–7.5 μg/kg/min, it has β-agonist properties resulting in increased heart rate and cardiac output. The β-agonist effects are less pronounced than those of isoproterenol. Dopamine is the preferred catecholamine for symptomatic bradycardia refractory to atropine.

4. Aminophylline—Aminophylline, a methylxanthine derivative, is a competitive antagonist of adenosine. Conduction disturbances during an acute myocardial infarction may be partially mediated by the endogenous release of adenosine. Aminophylline can be administered intravenously at a dose of 5–6 mg/kg infused over 5 minutes. A maintenance infusion may be required and can be initiated at 0.5 mg/kg/h.

5. Glucagon—Glucagon stimulates cyclic adenosine monophosphate production. It may be beneficial in the treatment of bradycardia associated with β-blocker or calcium-channel-blocker toxicity. An initial intravenous dose of 0.05–0.15 mg/kg is recommended, although optimal doses have not been determined.

Disposition

Hospitize all patients who have symptomatic bradycardia. Discontinue medications with AV nodal blocking properties. Although some patients with advanced AV conduction blocks will be asymptomatic, it is recommended that all patients with newly diagnosed second-degree Mobitz type II AV block and CHB be hospitalized.

Often patients with Wenckebach AV block will be asymptomatic. Treatment is usually not necessary unless symptoms occur. In general, no treatment is necessary for patients with first-degree AV block. At times, hospitalization will be necessary to treat the underlying condition such as myocardial ischemia or digoxin toxicity.

IDIOVENTRICULAR RHYTHM

Clinical Findings

Idioventricular rhythm refers to the occurrence of six or more consecutive ventricular escape beats. The rate of an idioventricular escape rhythm is usually 30–40 beats/min. The duration of the QRS complex often exceeds 0.16 seconds. The morphology of the QRS complex is similar to that in premature ventricular contractions (PVCs) but varies depending on the location of the ectopic ventricular focus. Escape rhythms often develop in response to severe bradycardia or an advanced AV block. If the rate is 50–100 beats/min, the rhythm is called accelerated idioventricular rhythm (AIVR). AIVR can also be seen after administration of thrombolytic therapy for acute myocardial infarction and may serve as a marker of reperfusion.

Treatment and Disposition

Treatment may be indicated if the ventricular escape rhythm is unable to maintain adequate cerebral perfusion or if the patient is unstable. If ventricular escape beats occur in response to advanced AV block, it could be dangerous to abolish the escape rhythm. In this case, the escape rhythm may be helping to maintain adequate perfusion. Management is directed at treating the underlying AV block. If AIVR occurs secondary to reperfusion, no treatment is generally needed. Because an idioventricular escape rhythm often occurs as a result of advanced AV block, the majority of patients will require hospitalization.

ATRIOVентRICULAR JUNCTIONAL RHYTHM

Clinical Findings

AV junctional escape rhythm refers to the occurrence of six or more consecutive junctional escape beats. The ventricular rate is usually 45–60 beats/min. AV junctional rhythm, like AV junctional premature beats, may originate from any location in the AV junctional tissue. Because the origin of the rhythm is the AV junctional tissue, the QRS complex is narrow unless the patient has a preexisting BBB. If the junctional escape rhythm is faster than 60 beats/min, the term AV junctional tachycardia is applied. If this rhythm is present, digoxin toxicity should be ruled out.

Treatment and Disposition

Patients with sinus bradycardia and occasional or intermittent AV junctional escape beats do not generally require intervention. Treatment including hospitalization will depend on the underlying cause of the cardiac arrhythmia.


Sherbino J, Verbeek PR, MacDonald RD, Sawadowsky BV, McDonald AC, Morrison LJ: Prehospital transcutaneous cardiac pacing for symptomatic bradycardia or bradyasystolic cardiac arrest:
It is estimated that more than 100,000 implantable cardioverter-defibrillators (ICDs) and more than 200,000 permanent cardiac pacemakers are implanted in the United States annually. These devices have dramatically reduced death from sudden cardiac death and other arrhythmias. However, they occasionally fail and emergency medicine physicians should be familiar with both normal pacemaker and AICD common malfunctions. It is estimated that permanent pacemakers have a 6% yearly incidence of malfunction and although many of these malfunctions will be identified during routine evaluation, some malfunctions will occur unexpectedly, resulting in an emergency department visit.

Types of Pacemakers

Pacemakers are either single-chamber (right atrium or right ventricle) dual-chamber (right atrium and right ventricle) or biventricular (right atrium, right ventricle and left ventricle) devices. In single-chamber pacemakers, a single lead paces and senses in the same chamber, most often the right ventricle. In dual-chamber pacemakers, one pacing and sensing lead is in the right atrium and the other is in the right ventricle. The biventricular pacemaker is similar to the dual chamber units except that there is also a left ventricular lead. Biventricular pacing is used with increasing frequency to optimize treatment of CHF with conduction delay or dysynchrony.

Since 1990, almost all pacemaker leads are bipolar. Bipolar leads have two electrodes on the same pacing lead, a distal cathode, and a proximal anode located approximately 1 cm apart near the distal tip of the pacemaker lead. Bipolar leads produce a small electrical field between the two electrodes. This produces a small, sometimes barely noticeable, pacing spike on the ECG. Older pacemaker leads were unipolar in design. The cathode was located at the distal end of the lead and the pulse generator served as the anode. Unipolar leads produce a larger electrical field and give rise to larger pacemaker spikes on the ECG. Unipolar leads are more likely to sense noncardiac electrical events such as pectoralis muscle activity. This can result in inappropriate inhibition of pacemaker activity (myopotential inhibition). The introduction of bipolar leads has virtually eliminated this type of oversensing malfunction.

Types of ICDs

Since receiving US Food and Drug Administration approval in 1985, ICDs have undergone significant technologic advances. Initially, devices were implanted in the abdominal wall and epicardial patches were sewn in place via a median sternotomy. Newer third-generation devices are smaller, and most are implanted in the subpectoral fascia using a transvenous lead system, similar to permanent pacemaker systems. As compared to earlier models, third-generation devices have more advanced tachycardia detection and termination features with longer battery life (7–8 years). The advanced tachycardia termination features include antitachycardia pacing (ATP), low-energy cardioversion, and high-energy defibrillation. Newer ICDs are also capable of rate-responsive dual-chamber back-up pacing.

COMPLICATIONS OF IMPLANTABLE CARDIAC PACEMAKERS AND ICDS

Venous Access

Although uncommon, the majority of venous access complications occur early after implantation. Venous access complications include bleeding, pneumothorax, hemothorax, and rarely air embolism. Venous thrombosis is another rare complication of pacemaker placement. Patients may present with unilateral upper extremity pain and swelling.

Pacemaker and ICD Pocket Site

Usually placed in the left subclavicular area, early device pocket site complications include bleeding with hematoma formation, wound dehiscence, or infection. Early pocket site infections are usually caused by Staphylococcus aureus. Late complications (greater than 30 days after implantation) can include pacemaker site erosion, keloid formation, pacemaker migration, and infection. Late infections are usually caused by Staphylococcus epidermidis. Approximately 6% of patients with permanent pacemakers develop pocket site infections.

Lead Complications

A number of complications can occur with endocardial pacemaker and ICD leads. Lead dislodgement is uncommon for pacemakers; rates are less than 2% for ventricular leads and less than 5% for atrial leads. ICD lead dislodgement approaches 10%. If lead dislodgement is suspected, obtain posteroanterior and lateral chest radiographs and compare them with prior chest X-ray. Lead fracture or insulation break may also occur. Lead fractures generally occur at three sites: (1) close to the pulse generator, (2) at the venous entry site, and (3) with the heart. Lead fractures may be diagnosed by chest X-ray or by pacemaker interrogation.

Cardiac perforation is another uncommon but potentially serious lead complication. Suspect perforation in the...
patient with a new paced right bundle branch block (RBBB) pattern on ECG, intercostal muscle or diaphragmatic con- tractions (hiccups), pericardial effusion, or tamponade. Cardiac perforation may also be identified by a plain chest radiograph demonstrating the tip of the pacemaker lead outside the cardiac silhouette. Echocardiography may be invaluable in diagnosing a pericardial effusion. Most cases (80%) of perforation occur within the first 4 days of pacemaker insertion. Another uncommon lead complication is Twiddler’s syndrome. This occurs when a patient wiggles or rotates the pacemaker generator, eventually dislodging the pacemaker leads.

**DEVICE MALFUNCTION**

### General Considerations

The most common pacemaker malfunctions are sensing abnormalities. Sensing malfunctions are further subdivided into undersensing or oversensing. Undersensing occurs when the pacemaker fails to sense intrinsic electrical cardiac activity (P wave or QRS complex). On the ECG, a pacing spike is preceded by an intrinsic P wave or QRS complex. Oversensing or crosstalk—misinterpretation by one lead of the signal generated by the other lead—can cause the pacemaker to inappropriately inhibit a pacing stimulus. On the ECG, this is evident by a pause that is longer than the programmed pacemaker rate.

Other pacemaker malfunctions include failure to pace and failure to capture. Failure to pace is characterized by an absence of an appropriate pacing stimulus. Failure to capture occurs when a pacing stimulus fails to depolarize the myocardium. Physiologic failure to capture may occur if the pacing stimulus occurs during the ventricular refractory period (within 300 ms after a native depolarization). This is not a malfunction, but reprogramming may still be necessary.

Lead complications are common causes of pacemaker malfunction. An increase in the pacing threshold may also cause sensing malfunctions and failure to capture. This can occur as a result of fibrosis at the lead tip, hyperkalemia, hypoxemia, myocardial ischemia, and antiarrhythmic drug toxicity.Battery depletion or component failure may result in failure to pace or undersensing. Electromagnetic interference from electrocautery or magnetic resonance imaging (MRI) can lead to over-sensing. Patients with implantable cardiac pacemakers should not undergo MRI. Variable effects have been documented, including pacemaker motion, function modification, heating of the pacemaker generator, and induction of voltage or current in the pacing leads.

Pacemaker-mediated tachycardia (PMT) is an uncommon complication that can occur with dual-chamber pacemakers. PMT can be triggered by a PVC with ventricular-to-atrial (VA) conduction. Retrograde atrial activity triggers a ventricular paced beat. As the ventricular paced beat undergoes VA conduction, another ventricular paced beat is triggered and the cycle continues. PMT will be evident by sustained pacing at the upper limit of the programmed pacing rate (100–140 beats/min). The ECG will characteristically reveal a wide complex paced tachycardia. PMT is often not life-threatening because the heart rate does not usually result in hemodynamic instability. Runaway pacemaker is another rare cause of a wide QRS complex paced tachycardia. In this case, the malfunctioning pulse generator discharges at a rate above its preset upper limit.

### Clinical Findings

Patients may present with a number of symptoms suggestive of pacemaker or ICD malfunction. These include dizziness, lightheadedness, near syncope, syncope, palpitations, short-ness of breath, or chest pain. The symptoms most concerning are those associated with cerebral hypoperfusion. Patients may present after blunt chest trauma or external defibrillation leading to pacemaker malfunction. Bradycardia may be an indicator or malfunction because the lower limit of fixed rate pacing is typically 50–60 beats/min. This may occur as a result of oversensing or failure to pace. The upper limit of rate responsive pacemakers is generally 100–140 beats/min. A paced rhythm at this rate may or may not be pacemaker malfunction.

Although uncommon, frequent or recurrent shocks may represent an ICD malfunction. An increased frequency of shocks may be caused by a number of conditions including an increased frequency of ventricular arrhythmias, device inefficacy, or an ICD sensing malfunction. The most common cause of an increased frequency of ICD shocks is an increased frequency of VT or VF. Ventricular arrhythmias can occur as a result of worsening left ventricular dysfunction, myocardial ischemia, or changes in antiarrhythmic therapy. An ICD sensing malfunction may lead to double counting of the T waves or inappropriate recognition of SVT as VT. Lead complications may also cause inappropriate ICD shocks.

Occasionally a patient may present with a sustained ventricular arrhythmia without ICD intervention. Although rare, this may occur as a result of a failure to detect the arrhythmia or exhaustion of therapies. In patients with ICDs, antibradyarrhythmia pacing malfunctions will be similar to those experienced by patients with implantable cardiac pacemakers.

Evaluation of the patient suspected of having ICD or pacemaker malfunction includes a 12-lead ICG and rhythm strip. If available, a comparison ECG may be helpful. A chest radiograph should also be obtained. Lab testing, specifically of potassium, magnesium, creatinine, thyroid screening, and antiarrhythmic levels, may be necessary.

A systematic approach to the evaluation of the 12-lead ECG and rhythm strip may help to identify pacemaker malfunction. The ECG should be evaluated to determine the presence or absence of appropriate pacing spikes. A normally functioning pacemaker should be inhibited from firing when
the patient’s intrinsic rate is faster than the programmed rate. Pacemaker function cannot be evaluated when the intrinsic rate is faster than the programmed rate. When properly inhibited, no pacing spikes are seen on the ECG.

Magnet application may provide information regarding battery depletion or malfunction. When applied correctly over a pacemaker or ICD generator, the magnet triggers a reed switch, which inactivates the sensing function. Pacemakers should revert to an asynchronous pacing mode at a rate (magnet rate) preset by the manufacturer. A magnet rate that is slower than the manufacturer’s preset rate suggests battery depletion. If no pacemaker spikes occur after magnet application, lead fracture or another malfunction may be the cause. When applied over an ICD, all antitachycardia functions (ATP and shock therapies) are disabled. Antibradycardia pacing functions are unaffected. Although most pacemakers and ICDs respond immediately when a magnet is applied correctly, no industry standard and responses are somewhat manufacturer-dependent. MRI is contraindicated in patients with both implantable pacemakers and ICDs. The strong magnetic field may damage the generator and interfere with normal device functioning.

Major ICD functions include sensing, detection, provision of therapy to terminate VT or VF, and pacing for bradyarrhythmia. When a tachycardia is detected two therapies are possible. First, ATP, which commonly consists of burst pacing at a rate 6–10 beats faster than the ventricular rate, is usually attempted. ATP may be felt but is not painful. Second, if ATP does not terminate the tachyarrhythmia, then high-energy shocks (1–40 J) will be delivered between the right ventricle coil electrode and the ICD casing and/or another electrode. These shocks are painful if the patient is conscious.

**Treatment and Disposition**

Treat venous access complications accordingly. Admit for parenteral antibiotics any patients suspected of having pocket site infections.

For patients presenting with pacemaker malfunction leading to symptomatic bradycardia, institute pharmacologic treatment or emergency pacing measures. If transcutaneous cardiac pacing is initiated, place the anterior pacing pad as far away from the pacemaker generator as possible. In the setting of symptomatic bradycardia, a magnet can also be applied to revert to asynchronous pacing. If a patient requires synchronized DC cardioversion or defibrillation, place the paddles or pads as far from the pulse generator as possible.

In the emergency department, treatment of PMT may be undertaken by a number of different maneuvers. First, a magnet may be applied to terminate the tachycardia. If a magnet is unavailable or unsuccessful, chest wall stimulation using a transcatheter pacemaker can be attempted. The required stimulus is usually 10–20 mA. This is less than the stimulus generally required for transcutaneous pacing. If unsuccessful, isometric exercises can be tried. Finally, chest thumps have had success in terminating PMT; no more than two are recommended. Each of the mentioned techniques is designed to affect the sensing function of the pacemaker, inhibit ventricular pacing, and terminate PMT. If these are unsuccessful, cardiology consultation for pacemaker interrogation and reprogramming will be necessary.

Runaway pacemaker is a rarely encountered problem. Pharmacologic intervention or magnet application can be attempted but will most likely be unsuccessful. Definitive treatment may require disconnecting the pacemaker leads or removal of the pulse generator.

Obtain cardiology consultation for patients suspected of having pacemaker malfunction. Unless the pacemaker can be interrogated in the emergency department, the majority of patients with suspected pacemaker malfunction will require hospitalization. For ICD malfunction resulting in frequent inappropriate shocks, temporary device deactivation may be necessary. Similar to cardiac pacemakers, magnet application should trigger a magnetically activated reed switch. This disables all antitachycardia functions (ATP and shock therapies). Antibradycardia pacing functions are unaffected. Although most ICDs are immediately deactivated when a magnet is applied correctly, responses are somewhat manufacturer-dependent. Deactivation is not commonly performed, however, because the most common reason for frequent shocks is an increase in the frequency of VT or VF.

If recurrent ventricular arrhythmias result in frequent ICD shocks, antiarrhythmic administration and sedation may be necessary. If the ventricular arrhythmia is incessant, external cardioversion or defibrillation may be needed. Place the defibrillator pads or paddles as far from ICD generator as possible. Older ICDs with epicardial electrodes have been reported to increase the defibrillation threshold by preventing externally applied current from passing into the myocardium. This may decrease the likelihood of successful defibrillation.


APPENDIX: COMMONLY ENCOUNTERED CARDIAC ARRHYTHMIAS

**Normal Sinus Rhythm**
(Figure 35–2) The heart rate is 60–100 beats/min. There is a constant and normal PR interval, and the P wave will be upright in lead II and inverted in lead aVR.

**Sinus Tachycardia**
(Figure 35–3) The heart rate is faster than 100 beats/min. Usually the rate is 101–160 beats/min. The P wave morphology is the same as in normal sinus rhythm.

**Sinus Bradycardia**
(Figure 35–4) The heart rate is slower than 60 beats/min. Usually the rate is 45–59 beats/min. Sinus bradycardia is commonly associated with sinus arrhythmia. The P wave morphology is the same as in normal sinus rhythm.

**Sinus Arrhythmia**
(Figure 35–5) The heart rate is usually 45–100 beats/min. The P wave morphology is the same as in normal sinus rhythm. The PP or RR cycles vary by 0.16 seconds or more. Most commonly, sinus arrhythmia occurs in relation to the respiratory cycle. The sinus rate will gradually increase with inspiration and slow with expiration.

**Automatic Atrial Tachycardia**
(Figure 35–6) The heart rate is usually 160–250 beats/min but may be as slow as 140 beats/min. The P wave morphology is usually different from that of normal sinus rhythm. The PP and RR cycles are regular in most cases. When the atrial rate is slower than 200 beats/min, 1:1 AV conduction is commonly noted. When the atrial rate is faster than 200 beats/min, the ventricular rate is often half the atrial rate because of the refractoriness of the AV node.

**Atrioventricular Nodal Reentrant Tachycardia**
(Figures 35–7 to 35–10) The heart rate is usually 180–200 beats/min. The P waves occur concurrent with the QRS complex and are often difficult to visualize on the ECG.

**Atrioventricular Reciprocating Tachycardia**
(Figure 35–11) The heart rate is usually faster than 200 beats/min. Because activation of the ventricle occurs through normal conduction pathways, the accessory pathway is concealed and the QRS morphology is normal.

**Atrial Fibrillation**
(Figures 35–12 and 35–13) The atrial rate is disorganized and is 400–650 beats/min. The ventricular rate is irregularly irregular. No P waves are discernible on ECG.

**Atrial Flutter**
(Figures 35–14) The atrial rate is usually 250–350 beats/min. Characteristic sawtooth flutter waves may be seen on the ECG, particularly in lead II. Variable AV conduction may be noted. Typically, 2:1 AV conduction occurs, resulting in a ventricular rate of approximately 150 beats/min.
Figure 35-4. Sinus bradycardia at a rate of 45 beats/min.

Figure 35-5. Sinus arrhythmia. The heart rate varies between 60 and 80 beats/min.

Figure 35-6. Automatic atrial tachycardia at a rate of 140 beats/min.

Figure 35-7. Atrioventricular nodal reentrant tachycardia at a rate of 175 beats/min. Note the absence of clearly discernible P waves.
**Figure 35–8.** A: AV nodal reentrant tachycardia with a left bundle branch block at a rate of 155 beats/min. B: The baseline ECG in the same patient showing sinus rhythm with a LBBB at a rate of 95 beats/min. Note that the 11th beat is a premature ventricular contraction.

**Multifocal Atrial Tachycardia**

(Figure 35–15) The heart rate is typically 100–130 beats/min. The characteristic ECG finding is at least three different P wave morphologies. Varying PR intervals may also be noted.

**Ventricular Tachycardia**

(Figures 35–16 and 35–17) The ventricular rate is usually 180–250 beats/min, although rates slower than 160 beats/min may occur. The QRS complex is wide (greater than 0.12 s in duration) and often bizarre in appearance. Fusion beats...
**Figure 35–9.** A: AV nodal reentrant tachycardia at a rate of 150 beats/min. B: Seconds later after the administration of adenosine, the same patient converts to sinus rhythm.

**Figure 35–10.** Paroxysmal supraventricular tachycardia at a rate of 150 beats/min in a patient who is hemodynamically unstable. After the seventh beat, the patient is cardioverted with 50 J to sinus rhythm.

**Figure 35–11.** A: AV reciprocating tachycardia at a rate of 250 beats/min. B: The same patient after pharmacologic conversion showing sinus rhythm with ventricular preexcitation.
Figure 35–12. A: Atrial fibrillation with a controlled ventricular response. B: Atrial fibrillation at a ventricular rate of 130 beats/min.

Figure 35–13. A: Atrial fibrillation with ventricular preexcitation. B: The same patient after pharmacologic conversion showing sinus rhythm with ventricular preexcitation.
or AV dissociation may be noted. If AV dissociation is present, the diagnosis of VT is confirmed.

- **Polymorphic Ventricular Tachycardia (Torsades de Pointes)**
  (Figure 35–18) The heart rate is usually 200–250 beats/min. Torsades de pointes is described as having a twisting-on-point appearance.

- **Ventricular Fibrillation**
  (Figure 35–19) VF is characterized by an irregularly irregular ventricular rhythm with no discernible distinction between the QRS complex, the ST segment, and T waves.

- **Premature Atrial Contractions**
  (Figure 35–20) A premature atrial contraction (PAC) may originate from anywhere in the atria except the sinus node. The P wave morphology is usually different from that of normal sinus rhythm. It is common to see a postectopic pause after a PAC. The QRS complex is narrow unless aberrantly conducted.

- **Premature Ventricular Contractions**
  (Figure 35–21) A PVC may originate from anywhere in the ventricles. The QRS complex is 0.12 second or longer in duration and resembles either a LBBB or RBBB. Uniform PVCs originate from the same foci and have the same appearance. Multiform PVCs have different morphology because they originate from different ventricular foci.

- **Idioventricular Rhythm**
  (Figure 35–22) The ventricular rate is usually 30–40 beats/min. The morphology of the QRS complexes will be similar to PVCs but will vary depending on the location of the ventricular foci. If the ventricular rate is 50–100 beats/min, the rhythm is called AIVR.

- **Atrioventricular Junctional Rhythm**
  (Figure 35–23) The ventricular rate is usually 45–60 beats/min. The QRS complex is narrow unless aberrantly conducted. If the junctional rhythm is faster than 60 beats/min, the term AV junctional tachycardia is applied.
Figure 35–16. The rhythm strip shows a run of ventricular tachycardia; the rate is 150 beats/min. After 16 beats the ventricular tachycardia spontaneously converts to sinus tachycardia.

Figure 35–17. Ventricular tachycardia at a rate of 145 beats/min.

Figure 35–18. Polymorphic ventricular tachycardia.

Figure 35–19. Ventricular fibrillation. After six beats, sinus rhythm degenerates into ventricular fibrillation.
▲ Figure 35–20. Sinus rhythm with premature atrial contractions in a bigeminal pattern. The configuration of the P waves of the premature atrial contractions are different from that of normal sinus rhythm.

▲ Figure 35–21. A: Sinus rhythm with frequent premature ventricular complexes in a pattern of bigeminy. B: Sinus rhythm with frequent premature ventricular complexes in a pattern of trigeminy.

▲ Figure 35–22. A: Atrial fibrillation with an idioventricular escape rhythm. B: Accelerated idioventricular rhythm at a rate of 50 beats/min.
Third-degree Atrioventricular Block (Complete Heart Block)
(Figures 35–29 to 35–31) The PP interval (atrial rate) is usually shorter (faster) than the RR interval (ventricular rate). Because no atrial impulses are conducted through the AV node, no relationship exists between the atrial and ventricular activity.

Sinoatrial Block
(Figure 35–24) SA block is characterized by blocked P waves, evident by a long PP interval. The PP intervals before the blocked P wave may gradually shorten (SA Wenckebach), or the PP intervals may be constant (second-degree Mobitz type II SA block).

First-degree Atrioventricular Block
(Figure 35–25) The PR interval is constant but characteristically prolonged greater than 0.2 second.

Second-degree Atrioventricular Block (Mobitz Type I)
(Figure 35–26) There is progressive lengthening of the PR interval followed by a nonconducted P wave leading to a dropped QRS complex. Classically, the PP interval remains constant. The RR interval that includes the blocked P wave is the longest in duration.

Second-degree Atrioventricular Block (Mobitz Type II)
(Figure 35–27) When every other P wave is blocked, one cannot distinguish between Mobitz type I or Mobitz type II AV block. This is described as 2:1 AV conduction.

Second-degree Atrioventricular Block
(Figure 35–28) The PR interval is regular and can be either normal or prolonged. Periodically, a P wave is not conducted, leading to a dropped QRS complex.

Single-chamber Ventricular Pacing
(Figures 35–32 and 35–33) When the intrinsic heart rate is faster than the programmed pacemaker rate, the pacemaker is inhibited from firing. When the intrinsic rate is slower, the pacemaker is triggered, taking over as the dominant pacemaker of the heart.

Dual-chamber Atrioventricular Pacing
(Figures 35–34 and 35–35) The pacemaker is capable of pacing and sensing the atria and ventricles. Depending on the intrinsic rate, the pacemaker can either be triggered or inhibited.

Failure to Capture
(Figure 35–36) Failure to capture occurs when an appropriate pacemaker discharge fails to depolarize the myocardium. Physiologic failure to capture can occur if the pacing stimulus occurs during the ventricular refractory period.
**Figure 35–25.** Sinus rhythm with first-degree AV block. The PR interval is 0.44 s.

**Figure 35–26.** Sinus bradycardia with second-degree Mobitz type I AV block. Note the progressive lengthening of the PR interval until a QRS complex is dropped.

**Figure 35–27.** Sinus rhythm with second-degree AV block.

**Figure 35–28.** Sinus rhythm with second-degree Mobitz type II AV block. Note the variable AV conduction.

**Figure 35–29.** Third-degree AV block. The atrial rate is 92 beats/min and the ventricular rate is 50 beats/min.
**Figure 35–30.** Third-degree AV block. The atrial rate is 88 beats/min and the ventricular rate is 30 beats/min.

**Figure 35–31.** Third-degree AV block with an accelerated idioventricular escape rhythm with a ventricular rate of 60 beats/min.

**Figure 35–32.** Asynchronous ventricular pacing. In this case, the intrinsic heart rate is slower than the programmed pacemaker rate. When this occurs, the pacemaker is triggered, taking over as the dominant pacemaker of the heart.

**Figure 35–33.** VVI pacing. Although the pacemaker spikes are difficult to appreciate, beats 3–8 are ventricular paced beats. When the intrinsic heart rate is faster than the programmed rate, the pacemaker is inhibited from firing.

**Figure 35–34.** AV sequential pacing in a dual-chamber pacemaker. In this case, the pacemaker will pace both the atria and ventricles when no intrinsic cardiac activity is sensed.
Figure 35–35. Dual-chamber pacemaker functioning in the VAT mode. The pacemaker paces the ventricles and senses the atria. If intrinsic atrial depolarizations are sensed, a ventricular pacing spike is triggered. This is evident on the ECG by the presence of atrial tracking.

Figure 35–36. A: Single-chamber ventricular pacemaker showing failure to capture. The underlying rhythm is second-degree Mobitz type I AV block. A ventricular pacing spike occurs after the fifth atrial complex (P wave). This pacing spike fails to depolarize the ventricular myocardium. B: Dual-chamber pacemaker showing failure to capture. Beat 3 shows an atrial pacing spike that fails to depolarize the atrial myocardium. The pacemaker then proceeds to pace the ventricle. Beats 1, 2, and 4–9 show an atrial pacing spike with capture followed by normal AV conduction. (Part A reproduced, with permission, from Garson A: Stepwise approach to the unknown pacemaker ECG. Am Heart J 1990;119:924.)

Figure 35–37. A: Undersensing. The fifth beat is a premature ventricular contraction (PVC). The next beat is a ventricular paced beat. Note that the paced beat occurs soon after the PVC, indicating a failure to sense the preceding complex. B: The first and second beats are paced and the third and fourth beats show normal AV conduction. There is a longer than expected pause between the fourth and fifth beats. This occurs secondary to ventricular oversensing. (Reproduced, with permission, from Garson A: Stepwise approach to the unknown pacemaker ECG. Am Heart J 1990;119:924.)
**Failure to Sense (Undersensing)**

(Figure 35–37A) Undersensing occurs when the pacemaker fails to detect intrinsic electrical cardiac activity. On the ECG, a P wave or QRS complex is inappropriately followed by a pacing spike.

**Oversensing**

(Figure 35–37B) Oversensing is the inappropriate inhibition of a pacing stimulus. On the ECG, it is evident by a pause that is longer than the programmed pacemaker rate.