



# Acute Dysrhythmias

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## LEARNING OBJECTIVES

1. Distinguish among the different bradyarrhythmias and develop optimal treatment strategies.
2. Design treatment modalities for atrial tachycardias.
3. Assess unique pharmacologic considerations for the treatment of Wolff–Parkinson–White syndrome.
4. Develop strategies for the detection and management of non-ACLS ventricular tachycardia.

### ABBREVIATIONS IN THIS CHAPTER

AAD	Antiarrhythmic drug
AF	Atrial fibrillation
AFI	Atrial flutter
AT	Atrial tachycardia
AV	Atrioventricular
AVNRT	Atrioventricular nodal reentrant tachycardia
AVRT	Atrioventricular reentrant tachycardia
CAD	Coronary artery disease
CTI	Cavotricuspid isthmus
DCCV	Direct-current cardioversion
HF	Heart failure
ICD	Internal cardioverter-defibrillator
LVAD	Left-ventricular-assist device
PPM	Permanent pacemaker
PVC	Premature ventricular contraction
RVR	Rapid ventricular response
SA	Sinoatrial
SCD	Sudden cardiac death
SVT	Supraventricular tachycardia
TdP	Torsades de pointes
VA	Ventricular arrhythmia
VT	Ventricular tachycardia
WPW	Wolff–Parkinson–White syndrome

[Table of other common abbreviations.](#)

## INTRODUCTION

A dysrhythmia can be defined as any abnormality of physiologic rhythm, either atrial or ventricular. Dysrhythmias associated with either location can significantly increase morbidity, and ventricular arrhythmias (VAs) can be life threatening. Recent research has focused mainly on procedural treatments such as ablative therapy; beta-blockers, nondihydropyridine calcium channel blockers; and antiarrhythmics continue to be the main pharmacological treatment options for acute dysrhythmias. There has been little drug development, but several recent major studies give clinicians evidence on how to best implement the therapies in practice so as to maximize efficacy and safety outcomes. This chapter focuses on the treatment of common acute dysrhythmias not covered in ACLS.

## BRADYARRHYTHMIAS

In general, bradyarrhythmias refer to a group of abnormal rhythms such as sinus bradycardia, sick sinus syndrome, atrioventricular (AV) block, and conduction disorders that result in heart rates lower than the normal range of 60–100 beats per minute. While the National Institutes of Health defines bradycardia as a heart rate of less than 60 beats/minute in non-well-trained athletes, the 2018 ACC/AHA/HRS guidelines define bradycardia as a sinus rate of less than 50 beats/minute (Kusumoto 2018). Table 1 describes the different types of bradyarrhythmias, including ECG diagnostic characteristics (Kadish 2001). The initial evaluation of bradycardia begins with an ECG evaluation and a thorough patient assessment. Regardless of rhythm type, the patient's symptomology drives treatment initiation with presenting symptoms often including syncope or presyncope, transient dizziness, heart failure (HF) symptoms, and altered mental status (Epstein 2013). Because traditional symptoms of dizziness

and syncope may be difficult to identify in the critically ill patient population, assessments should focus on clinical manifestations of cerebral hypoperfusion resulting from slow heart rate (Goldschlager 1988).

Many underlying conditions can be associated with bradyarrhythmias, varying from cardiac abnormalities to infectious processes. Box 1 identifies common causes of bradyarrhythmias, some of which could be readily reversible.

### Sinus Bradycardia and Sick Sinus Syndrome

For the purposes of this chapter, evaluation and treatment of sinus bradycardia and sick sinus syndrome (now referred

to as *sinus node dysfunction*) will be discussed together and referred to as *acute bradycardia*. Upon recognition of acute bradycardia, evaluation should include assessment of organ perfusion, ECG, and presence of reversible causes. Medications associated with bradycardia are listed in Box 2. If the patient has moderate or severe symptoms of hypoperfusion, treatment should be initiated. Figure 1 identifies an algorithmic approach to treatment in acute bradycardia. Specific information regarding dosing, mechanism of action, and specific pharmacologic considerations is given in Table 2.

The treatment of acute bradycardia begins with an initial assessment of clinical manifestations, focusing on signs of hypoperfusion, and a quick evaluation of potentially reversible causes. Hypoperfusion can manifest as transient dizziness or light-headedness to more-moderate or severe symptoms such as syncope, dyspnea on exertion, altered mental status, and hemodynamic instability. Patients presenting with bradycardia associated with moderate-to-severe symptoms should begin treatment immediately, focusing on increasing the heart rate and improving perfusion.

Atropine is often the first-line agent for treatment of acute bradycardia. Atropine blocks muscarinic receptors and thereby facilitates sinoatrial conduction and an increase in sinus node automaticity (Brady 1999). If the patient remains bradycardic with symptoms after atropine administration, beta agonist therapy should be initiated with either a continuous infusion of dopamine or epinephrine to increase heart rate and contractility. Dopamine and epinephrine are most commonly used for acute bradycardia; other beta agonists such as dobutamine or isoproterenol should be used cautiously in these scenarios. From a practical standpoint, clinicians are usually more familiar with dopamine and epinephrine, and those infusions are typically more readily available for quick administration in acute situations when compared with dobutamine or isoproterenol. Dobutamine and isoproterenol can also cause vasodilation, which could lead to increasing of hypotension in some patients (Ogawa 1991). Whereas all beta agonists should be used cautiously in patients with possible coronary ischemia, isoproterenol should be avoided because it increases myocardial oxygen demand via beta-1 stimulation and decreases coronary perfusion because of beta-2 effects (Kurland 1979, Kusomoto 2018).

Patients should be evaluated for any potential drug toxicities that could be reversible, including nondihydropyridine calcium channel blockers, beta-blockers, and digoxin (Figure 2). Even though many clinicians rapidly identify common pharmacological causes of bradycardia such as beta-blockers, digoxin, and nondihydropyridine calcium channel blockers, it is important to recognize that many other medications could be associated with either inducing or exacerbating bradycardia (see Box 2).

#### BASELINE KNOWLEDGE STATEMENTS

Readers of this chapter are presumed to be familiar with the following:

- Signs and symptoms associated with dysrhythmias
- Basic pharmacologic management of patients with dysrhythmias, as recommended by the Heart Rhythm Society / American College of Cardiology / American Heart Association
- Basic hemodynamic concepts relevant to hemodynamics and dysrhythmias
- Basic pharmacologic properties of medications used for the treatment of dysrhythmias, including beta-blockers, calcium channel blockers, and antiarrhythmics

[\*Table of common laboratory reference values.\*](#)

#### ADDITIONAL READINGS

The following free resources are available for readers wishing additional background information on this topic.

- Page RL, Joglar JA, Caldwell MA, et al. [2015 ACC/AHA/HRS Guideline for the management of adult patients with supraventricular tachycardia](#). *Circulation* 2016;133:e506-e74.
- January CT, Wann LS, Calkins H, et al. [2019 AHA/ACC/HRS focused update of the 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation](#). *Circulation* 2019;140:e125-e51.
- January CT, Wann LS, Alpert JS, et al. [2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation](#). *J Am Coll Cardiol* 2014;64:2246-80.
- Al-Khatib SM, Stevenson WG, Ackerman MJ, et al. [2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death](#). *J Am Coll Cardiol* 2018;72:e91-e220.

**Table 1.** Types of Bradyarrhythmias

Arrhythmia	Description
Sick sinus syndrome (sinus node dysfunction) <sup>a</sup>	<ul style="list-style-type: none"> <li>• Sinus bradycardia: Sinus rate &lt; 50 beats/minute</li> <li>• Sinus pause: Sinus node depolarizes more than 3 seconds between atrial depolarization after most recent depolarization</li> <li>• Sinus node arrest: No evidence of sinus node depolarization</li> <li>• Ectopic atrial bradycardia: Atrial depolarization caused by an atrial pacemaker other than the sinus node at a rate of &lt; 50 beats/minute</li> <li>• Sinoatrial exit block: Presence of blocked conduction between the sinus node and adjacent atrial tissue</li> <li>• Tachycardia–bradycardia (“tachy-brady”) syndrome: Sinus bradycardia, ectopic atrial bradycardia, or sinus pause alternating with times of abnormal atrial tachycardia, atrial flutter, or atrial fibrillation</li> <li>• Chronotropic incompetence: Inability of the body to appropriately increase heart rate in response to increased demand or activity</li> <li>• Isorhythmic dissociation: Depolarization of the atria that is slower than ventricular depolarization</li> </ul>
Atrioventricular block	<ul style="list-style-type: none"> <li>• <i>First-degree atrioventricular block</i>: P waves associated with 1:1 atrioventricular conduction and a PR interval &gt; 200 milliseconds</li> <li>• <i>Second-degree atrioventricular block</i>: P waves with a constant rate (&lt; 100 beats/minute) when atrioventricular conduction is present but not 1:1 <ul style="list-style-type: none"> <li>◦ <i>Mobitz type I</i>: P waves at a constant rate (&lt; 100 beats/minute) with a periodic single nonconducted P wave associated with P waves before and after the nonconducted P wave with <b>inconstant</b> PR intervals</li> <li>◦ <i>Mobitz type II</i>: P waves at a constant rate (&lt; 100 beats/minute) with a periodic single nonconducted P wave associated with P waves before and after the nonconducted P wave with <b>constant</b> PR intervals</li> </ul> </li> <li>• <i>Third-degree atrioventricular block</i> (complete heart block): No evidence of atrioventricular conduction</li> </ul>

<sup>a</sup>*Sinus node dysfunction* and *sick sinus syndrome* will be referred to as *acute bradycardia*.

Information from Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guidelines on the evaluation and management of patients with bradycardia and cardiac conduction delay. J Am Coll Cardiol 2018. doi: <https://doi.org/10.1016/j.jacc.2018.10.044>.

### Box 1. Conditions Associated with Bradycardia

Autonomic disturbances	Metabolic
<ul style="list-style-type: none"> <li>• Carotid sinus hypersensitivity</li> <li>• Neurally mediated syncope/presyncope</li> <li>• Physical conditioning</li> <li>• Situational syncope (cough, defecation, glottis stimulation, medical procedures, vomiting)</li> <li>• Sleep (with or without sleep apnea)</li> </ul>	<ul style="list-style-type: none"> <li>• Acidosis</li> <li>• Hyperkalemia</li> <li>• Hypokalemia</li> <li>• Hypothermia</li> <li>• Hypothyroidism</li> <li>• Hypoxia</li> </ul>
Cardiomyopathy (ischemic or nonischemic)	Rheumatologic disease
Congenital heart disease	<ul style="list-style-type: none"> <li>• Rheumatoid arthritis</li> <li>• Scleroderma</li> <li>• Systemic lupus erythematosus</li> </ul>
Degenerative fibrosis	Surgical or procedural trauma
Infection/inflammation	<ul style="list-style-type: none"> <li>• Cardiac procedures such as ablation or cardiac catheterization</li> <li>• Congenital heart disease surgery</li> <li>• Septal myomectomy for hypertrophic obstructive cardiomyopathy</li> </ul>
<ul style="list-style-type: none"> <li>• Chagas disease</li> <li>• Diphtheria</li> <li>• Infectious endocarditis</li> <li>• Lyme disease</li> <li>• Myocarditis</li> <li>• Sarcoidosis</li> <li>• Toxoplasmosis</li> </ul>	Valve surgery (including percutaneous valve replacement)
Ischemia/infarction	

Information from Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guidelines on the evaluation and management of patients with bradycardia and cardiac conduction delay. J Am Coll Cardiol 2018. doi: <https://doi.org/10.1016/j.jacc.2018.10.044>.

## Box 2. Medications Associated with Bradycardia

### Cardiovascular Medications

- Adenosine
- Amiodarone
- Beta blockers (including eye drops)
- Clonidine
- Digoxin
- Dronedarone
- Flecainide
- Ivabradine
- Methyldopa
- Nondihydropyridine calcium channel blockers
- Procainamide
- Propafenone
- Reserpine
- Quinidine
- Sotalol
- Ticagrelor

### Neurologic Medications

- Donepezil
- Lithium
- Opioid analgesics
- Phenothiazines
- Phenytoin
- Selective serotonin reuptake inhibitor
- Tricyclic antidepressants

### Herbal Supplements

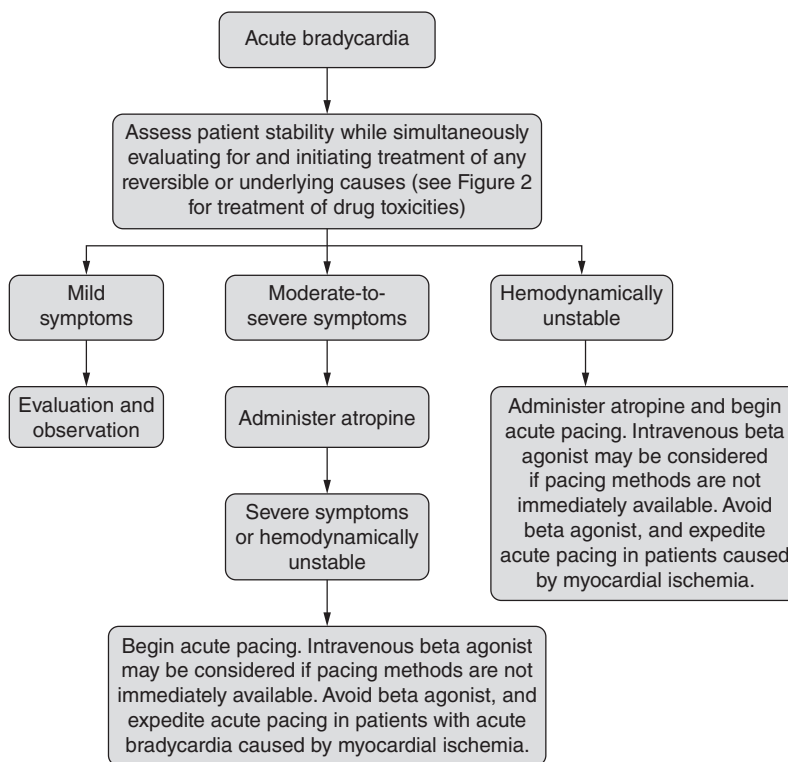
- Foxglove
- Ginseng
- Hawthorne
- Indian snakeroot
- Khella seed
- Kudzu root
- Lily of the valley
- Licorice root

- Lobelia
- Monkshood
- Motherwort
- Oleander
- Queen of the Night
- Skullcap
- Squill
- Strophanthus
- Valerian
- Wolfsbane

### Miscellaneous Medications

- Cannabis
- Dexmedetomidine
- Propofol
- Succinylcholine

Information from Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guidelines on the evaluation and management of patients with bradycardia and cardiac conduction delay. J Am Coll Cardiol 2019;74:e51-e156.



**Figure 1.** Algorithmic approach to treatment in acute bradycardia.

Information from Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guidelines on the evaluation and management of patients with bradycardia and cardiac conduction delay. J Am Coll Cardiol 2019;74:e51-e156.

**Table 2.** Drug Therapy for Acute Bradycardia

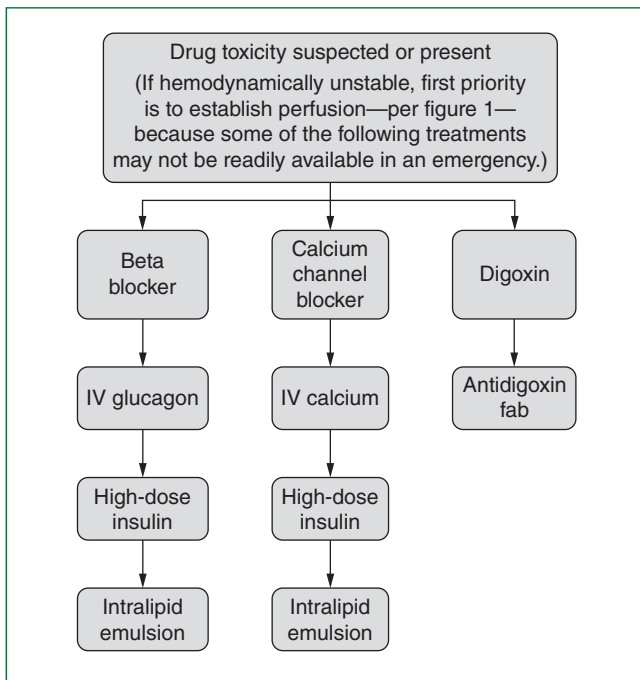
Drug	Dose	Mechanism of Action	Potential Adverse Effects	Considerations
Aminophylline	250 mg IV over 30 minutes	Inhibits the suppressive effects of adenosine on the SA node	<ul style="list-style-type: none"> <li>Headache</li> <li>Irritability</li> <li>Nausea</li> <li>Tremor</li> </ul>	<ul style="list-style-type: none"> <li>Generally well tolerated</li> <li>Dose utilized is less than typical loading dose for COPD (5.7 mg/kg)</li> <li>Minimal data to support use</li> </ul>
Atropine	0.5 mg – 1 mg IV every 3–5 minutes up to a maximum dose of 3 mg	Blocks acetylcholine at muscarinic receptors, leading to an increase in cardiac output and heart rate	<ul style="list-style-type: none"> <li>Tachycardia</li> <li>Urinary retention</li> <li>Delayed gastric emptying</li> </ul>	<ul style="list-style-type: none"> <li>Can exacerbate preexisting anticholinergic symptoms</li> </ul>
Dopamine	5–20 mcg/kg/min IV infusion	Stimulates both adrenergic and dopaminergic receptors	<ul style="list-style-type: none"> <li>Tachycardia</li> <li>Hypertension</li> </ul>	<ul style="list-style-type: none"> <li>Often readily available as a premix infusion</li> </ul>
Calcium	Calcium chloride 10%: 1–2 g IV every 10–20 minutes Calcium gluconate 10%: 3–6 g IV every 10–20 minutes	Increase calcium entry into the myocardium via nonblocked channels	<ul style="list-style-type: none"> <li>Hypercalcemia</li> </ul>	<ul style="list-style-type: none"> <li>Reserve calcium chloride for central administration to avoid extravasation</li> </ul>
Digoxin-specific antibody fragments	Empiric dose for acute toxicity: 10–20 vials IV (i.e., 40 mg per vial) Dose calculation based on serum concentration: [(concentration-ng/mL) x (patient weight in kg)] / 100 = required number of vials	Binds directly to digoxin	<ul style="list-style-type: none"> <li>Hypokalemia</li> <li>Worsening heart failure</li> <li>Rapid ventricular rate</li> <li>Allergic reactions (rare)</li> </ul>	<ul style="list-style-type: none"> <li>High cost</li> <li>One vial binds approximately 0.5 mg of digoxin</li> <li>Dose may be repeated</li> <li>Administer over at least 30 minutes</li> <li>Total drug levels will remain elevated after antibody administration for 24 hours</li> </ul>
Epinephrine	2–10 mcg/min IV infusion	Stimulates adrenergic receptors	<ul style="list-style-type: none"> <li>Tachycardia</li> <li>Hypertension</li> </ul>	<ul style="list-style-type: none"> <li>Clinicians usually unfamiliar with dosing as continuous infusion vs. IV pushes during cardiac arrest</li> </ul>

(Continued)

**Table 2.** Drug Therapy for Acute Bradycardia (Continued)

Drug	Dose	Mechanism of Action	Potential Adverse Effects	Considerations
Glucagon	3–5 mg IV bolus (up to 10 mg) followed by infusion of 3–5 mg/h if patient responded to initial bolus dose	Directly increases cardiac-cyclic-adenosine-monophosphate-enhancing inotropy and chronotropy	<ul style="list-style-type: none"> <li>Dose-dependent nausea and vomiting</li> <li>Hyperglycemia</li> </ul>	<ul style="list-style-type: none"> <li>Increased risk of aspiration in patients with decreased mental status</li> </ul>
High-dose insulin therapy	Regular Insulin: 0.5–1 unit/kg/h continuous infusion along with dextrose 0.5–1 g/kg/h to maintain serum glucose concentrations of 100–250 mg/dL	Enhances carbohydrate use and energy production by myocardial cells, resulting in improved contractility	<ul style="list-style-type: none"> <li>Hypokalemia</li> <li>Hypoglycemia</li> <li>Volume overload caused by free water administration by means of dextrose infusions</li> </ul>	<ul style="list-style-type: none"> <li>Very labor-intensive for bedside staff</li> <li>Requires blood glucose measurements every 10–20 minutes</li> <li>Insulin infusion should be maintained at the 0.5–1 unit/kg/h rate, and dextrose boluses and infusions should be given to support blood glucose levels in the 100–250 mg/dL range</li> </ul>
Intralipid emulsion 20% injection	100 mL of 20% intralipid IV push over 1–2 minutes and repeat if necessary. May be followed with a continuous infusion at 0.25–0.5 mL/kg/min for 30–60 minutes	Proposed mechanisms of action include <ul style="list-style-type: none"> <li>Lipid sink theory: lipophilic molecules partition into lipemic plasma compartment, becoming unavailable in the tissue</li> <li>Direct activation of myocardial calcium channels</li> <li>Utilizing fatty acids as an energy source of myocardial cells</li> </ul>	<ul style="list-style-type: none"> <li>Fat embolism</li> <li>Interference with lab analyses because of lipemic blood</li> </ul>	<ul style="list-style-type: none"> <li>Most useful in lipophilic drug overdoses such as propranolol, diltiazem, or verapamil</li> <li>Limited data to support routine use</li> </ul>

Information from Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guidelines on the evaluation and management of patients with bradycardia and cardiac conduction delay. J Am Coll Cardiol 2018.



**Figure 2.** Treatment of drug toxicities associated with bradycardia.

Information from Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guidelines on the evaluation and management of patients with bradycardia and cardiac conduction delay. *J Am Coll Cardiol* 2019;74:e51-e156.

If beta-blocker toxicity is suspected, glucagon may be used as initial therapy despite limited data (Bailey 2003). If the patient fails to respond to glucagon therapy, high-dose insulin therapy has been studied as treatment of severe bradycardia. Although therapy has been associated with increases in heart rate and hemodynamic status, the evidence to support its use consists of animal studies, case reports, and case series. High-dose insulin therapy requires significant bedside commitment from nurses, providers, and pharmacy staff because it has to be carefully monitored—and can be associated with hypoglycemia and hypokalemia. Treatment with intralipid fat emulsion could be considered in cases of lipophilic beta-blocker overdose such as propranolol or nondihydropyridine calcium channel blocker overdose.

Intravenous calcium should be administered in the case of a suspected nondihydropyridine calcium channel blocker overdose (St-Onge 2014). Data supporting the use of calcium are limited to case series and systemic reviews; however, they do support a general improvement in heart rate and blood pressure with a minimal adverse-effect profile (Ramoska 1993). The choice of which calcium salt, gluconate, or chloride should be determined based on current intravenous access. Calcium gluconate may be administered by either peripheral or central access, and calcium chloride should be reserved

for central-venous-access administration. High-dose insulin therapy and intralipid emulsion administration may also be considered in these cases as well.

Digoxin toxicity with serum levels of more than 2 mcg/L or hyperkalemia is associated with an increased risk of death (Eddleston 2000). The use of digoxin-specific antibodies is associated with a high response rate of 80% – 90%, especially in the acute setting (Chan 2014). Patients with lower digoxin levels can also manifest signs and symptoms of toxicity. The decision to administer digoxin-specific antibody should be made based on bradycardia-associated symptoms or hemodynamic compromise in the setting of known or suspected digoxin toxicity (Kusumoto 2018). It is important to note that digoxin levels obtained after antibody administration will be unreliable because the assay detects both bound and unbound drug. If a digoxin level is wanted for evaluating post-antibody administration, a free digoxin level should be obtained. Patients presenting with acute digoxin toxicity often present with hyperkalemia caused by increases in extracellular potassium concentrations by means of inhibition of the sodium-potassium ATP-ase pump. Care should be taken to avoid rapidly correcting hyperkalemia because once treatment with digoxin antibody has been initiated, potassium begins shifting back into the cell, and serum concentrations of potassium decrease. Up front, treatment of hyperkalemia in this instance is often associated with clinically significant hypotension after administration of digoxin antibody. Hypokalemia caused by diuretic use or acute GI illnesses can make the patient more vulnerable to the effects of digoxin toxicity, and it is often seen in cases of chronic toxicity (Lip 1993).

Acute bradycardia related to autonomic denervation is often seen in patients post-heart-transplant. Atropine therapy does not treat bradycardia in the denervated heart and may cause worsening bradycardia. In a study of 25 patients who underwent heart transplant, 20% of patients experienced paradoxical heart block or sinus arrest after standard doses of atropine for bradycardia (Bernheim 2004). Methylxanthines such as theophylline and aminophylline are positive chronotropic agents that inhibit the suppressive effects of adenosine on the sinoatrial (SA) node (Lou 2013). Those agents may be used to restore sinus rate and/or prevent the need for permanent pacemakers (PPMs) in patients with heart transplants (Bertolet 1996, Heinz 1993, Redmond 1993). Methylxanthines, too, may be considered for the treatment of acute bradycardia caused by autonomic dysfunction in patients with spinal cord injuries. In those cases, bradycardia is related to unopposed parasympathetic stimulation, which is usually unresponsive to atropine or adrenergic agonist treatment (Kusomoto 2018). Data supporting the use of methylxanthines in this indication are sparse and include three case series totaling six patients which demonstrated an increase in HR and avoidance of PPM placement (Pasnoori 2004, Sadaka 2010, Schulz-Stübner 2005).

Patients who are hemodynamically unstable and refractory to other therapies—including intravenous beta agonist therapy—may require temporary transcutaneous pacing to maintain perfusion and alleviate symptoms until a transvenous pacemaker can be placed. It is important to note that transcutaneous pacing can be uncomfortable and painful for the patient. Pharmacists should make sure that patients receive analgesia and anxiolytic therapy while the patients are being paced. Patients are typically hemodynamically unstable at this time, and so, short-acting analgesic agents such as fentanyl may help them tolerate the procedure better. If clinically appropriate, adding an anxiolytic agent on top of analgesia would be beneficial. If a patient is intubated, appropriate postintubation analgesia and sedation should be evaluated and adjusted based on the addition of a painful procedure.

### Atrioventricular Block

The presence of a conduction block is identified based on ECG findings but is further classified by the anatomic site of the block: AV node, labor intensive (within the His bundle), and infra-Hisian (below the His bundle). Similar patient evaluation and treatment modalities should be used for AV blocks as with acute bradycardias. Reversible causes should be identified and treated, and temporary treatment modalities should be used for increasing perfusion and decreasing symptoms. In an evaluation of causation with medication regimens, patients with AV blocks are often found to have underlying hypertension, arrhythmias, heart failure, and other cardiac diseases that necessitate the use of such agents as beta-blockers, antiarrhythmic drugs (AADs), and nondihydropyridine calcium channel blockers. On the surface, it might seem that this represents a case of reversible AV block, but case series suggest that when AV nodal blocking medications are used at therapeutic doses, their discontinuation rarely results in block reversal (Knudsen 2013, Osmonov 2012, Zeltser 2004). In addition, a PPM is indicated for patients with compelling indications so as to allow continued use of such medications.

It is reasonable to try atropine therapy as a first-line intervention for AV block. Atropine therapy is most useful for AV nodal level blocks and bradycardia caused by vagal tone excess; however, atropine is unlikely to correct AV block at the His bundle or the His–Purkinje level (Kusumoto 2018). Because of its short duration of action, atropine usually serves as a bridge therapy to either beta agonist infusions or temporary pacing measures.

Infusions such as dopamine and epinephrine improve AV nodal and His–Purkinje conduction. In the setting of complete AV block, dopamine and epinephrine infusions may enhance the automaticity of subsidiary AV junctional and ventricular pacemakers (Dhingra 1973, Hatle 1971). The positive effects of beta agonist infusions must be weighed

against the potential for induction of ventricular dysrhythmias and coronary ischemia. As with acute bradycardia, patients with second- or third-degree AV block that is associated with symptoms or hemodynamic instability and refractory to other interventions are candidates for temporary pacing. If the block is caused by a reversible condition, it is reasonable to consider temporary transvenous pacing. Many patients, however, may require PPM placement based on underlying physiology and concomitant medication requirements.

## SUPRAVENTRICULAR ARRHYTHMIAS

### Supraventricular Tachycardia

*Supraventricular tachycardia* (SVT) is an umbrella term describing tachycardias that initiate from the His Bundle or above. Atrial fibrillation is generally not included within the SVT characterization. Types of SVT are described in Table 3. Supraventricular tachycardia is generally paroxysmal, and incidence increases with age. Individuals older than 65 years of age have more than five times the risk of developing paroxysmal SVT (Orejarena 1998).

The most-common symptoms at presentation are palpitations, light-headedness, dizziness, shortness of breath, and chest pain. True syncope can occur but is rare. The symptoms of SVT must be differentiated from symptoms of anxiety disorders or any condition associated with heightened awareness of sinus tachycardia (i.e., postural orthostatic tachycardia) (Page 2016). It is important to obtain a 12-lead ECG to determine whether the rhythm is dependent on the AV node. If there is AV node involvement, then AV nodal blocking agents are more likely to be effective. Alternatively, if the rhythm is not dependent on the AV node, then antiarrhythmic agents may be more appropriate. In a situation in which the QRS duration is more than 120 milliseconds (ms), it is also critical to discriminate VT from SVT with aberrancy, from preexisting bundle-branch block, and from preexcitation. Several criteria-based algorithms aid with making that differentiation, but they are beyond the scope of this chapter (Page 2016).

Treatments of the different types of SVT may vary if a diagnosis is established. In general, synchronized direct-current cardioversion (DCCV) is recommended in any case of hemodynamic instability (e.g., hypotension, acutely altered mental status, signs of shock, chest pain, acute heart failure symptoms). Conscious patients should receive adequate sedation prior to cardioversion (Link 2015). Vagal maneuvers are considered first-line treatment for termination if the patient is conscious and relatively stable (Waxman 1980). Valsalva maneuvers and/or carotid sinus massage can be performed quickly while pharmacologic therapies are being prepared. Vagal maneuvers will generally be ineffective in rhythms that do not involve the AV node (Figure 3).



**Table 3.** Types of SVT

Arrhythmia	Definition	Notes
<b>Atrial tachycardia</b>		
Focal AT	SVT arising from a focal atrial site	<ul style="list-style-type: none"> <li>• Regular, discrete P waves</li> <li>• Often an isoelectric segment between P waves</li> </ul>
Multifocal AT	Irregular SVT with patterns of atrial activation at different rates	<ul style="list-style-type: none"> <li>• <math>\geq 3</math> distinct morphologies of P waves</li> <li>• Commonly associated with pulmonary disease, coronary artery disease, valvular heart disease, or hypomagnesemia</li> </ul>
<b>Atrial flutter</b>		
Typical	AT involving a circuit that rotates around the tricuspid valve in a counterclockwise direction (up the septum and down the free wall)	<ul style="list-style-type: none"> <li>• CTI-dependent flutter</li> <li>• Characterized by predominantly negative flutter waves in the inferior leads (sawtooth waves)</li> </ul>
Atypical	Reentrant AT that is not dependent on conduction through the cavotricuspid isthmus	<ul style="list-style-type: none"> <li>• Can be idiopathic but commonly occurs in patients with atrial scarring from prior heart surgery or ablation</li> </ul>
AVNRT	Reentrant pathway involving two distinct (fast and slow) pathways	
Accessory pathways: extranodal AV pathway that connects atrium to ventricle		
Manifest accessory pathway	Pathway that conducts anterogradely	
Concealed accessory pathway	Pathway that conducts retrogradely	
Wolff–Parkinson–White syndrome	Documented SVT or SVT symptoms with ventricular preexcitation during sinus rhythm	

AT = atrial tachycardia; AV = atrioventricular; AVNRT = atrioventricular nodal reentrant tachycardia

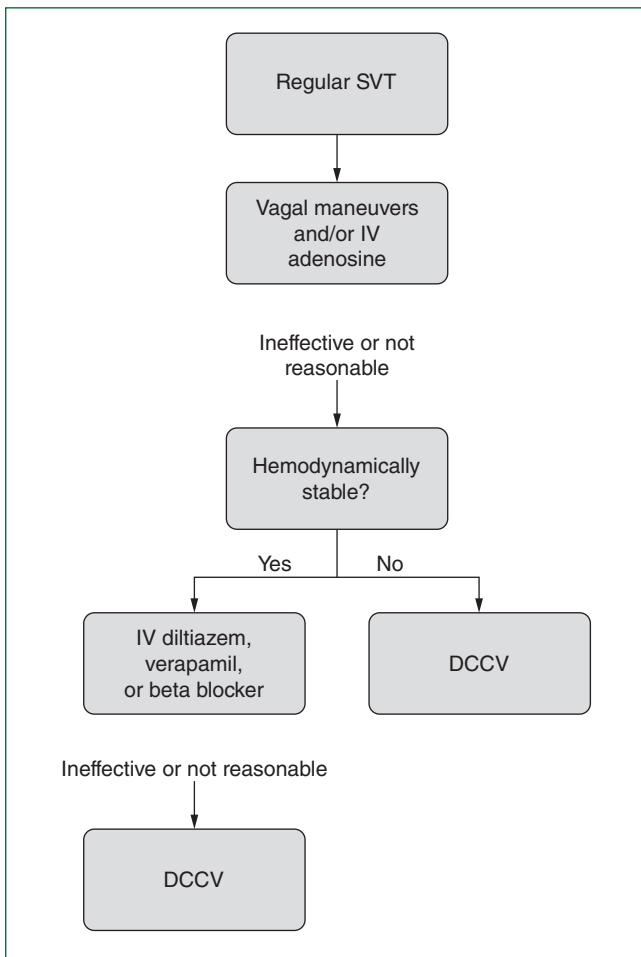
Information from Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: executive summary. *Circulation* 2016;133:e471-e505.

Adenosine slows conduction time through the AV node and produces transient AV nodal block. It is often successful at terminating SVT caused by atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT) (Gausche 1994). It may also be helpful as a diagnostic tool because the induced AV nodal blockade slows the ventricular rate and reveals the unaffected arrhythmia if not AV node dependent (DiMarco 1985). Patients should be educated about side effects such as chest pain, chest discomfort, transient cardiac pause, shortness of breath, and flushing. The short half-life of less than 10 seconds limits the severity of those adverse effects. Adenosine should be administered as a rapid bolus followed by a saline flush. Continuous ECG monitoring is recommended to assess rhythm response and help with diagnosis. Specific information regarding dosing, mechanism of action, and specific pharmacologic considerations is given in Table 4.

Diltiazem and verapamil have demonstrated some success in SVT termination when given intravenously in hemodynamically stable patients without systolic heart

failure (Brady 1996). They can be given as intravenous boluses; however, a slow infusion of up to 20 minutes may decrease the potential for hypotension (Lim 2002). These agents should be avoided in patients with reduced ejection fraction or acute myocardial infarction. There are only limited data on the efficacy of beta-blockers. A comparative study of diltiazem and esmolol showed that diltiazem was more effective (Gupta 1999). However, given their similar mechanisms of action and safety profiles, it is reasonable to try intravenous beta-blockers in hemodynamically stable patients.

Once acute stability has been achieved, electrophysiology testing with ablation is generally considered first-line therapy because it serves as a definitive cure without need for chronic medication. Registry studies report success rates of 93% – 97%, with a recurrence rate of 5% – 8% and low incidence of complications (e.g., stroke or pericardial effusion) for common SVTs such as AVNRT (Calkins 1999, Spector 2009).



**Figure 3.** Acute supraventricular tachycardia management algorithm.

Information from Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: executive summary. *Circulation* 2016;133:e471-e505.

All patients should be taught how to perform vagal maneuvers in case of return of SVT. Patients should forcefully exhale against a closed airway for 10–30 seconds while in a supine position. Some patients also find it helpful to apply an ice-cold, wet towel to the face (Page 2016).

Some patients may prefer not to undergo ablation, and a small percentage may experience symptom recurrence despite ablation. In that case, therapy with AV nodal blockers can reduce the repetition of episodes. Antiarrhythmic agents are also reasonable options; choice may be based on concomitant diseases, renal function, liver function, drug-drug interactions, and patient and provider preference (Page 2016). When compared with propranolol and verapamil, digoxin similarly decreased episodes of SVT but required doses higher than commonly used. Therefore its use should be reserved for patients who cannot take AV nodal blockers or AADs (Winniford 1984).

## Atrial Flutter

Atrial flutter (Afl) is a reentrant AT characterized by regular atrial rate and consistent P-wave morphology. Atrial rates typically range from 250 to 330 beats/minute but may be slower in patients with scarring or who are taking AADs. AFI can occur in clinical settings similar to those associated with AF and can be triggered by AT or AF. It is common for AF and AFI to coexist in the same patient. Atrial flutter is subclassified as either typical or atypical (see Table 3). In general, they are similarly managed acutely. Once acute stability has been achieved, catheter ablation is generally considered first-line therapy (Page 2016). Typical atrial flutter (CTI-dependent) ablations are successful in 97% of cases. Catheter ablation of atypical flutter requires more-extensive mapping, and success rates are lower (Spector 2009).

First-line acute treatment of AFI is in the form of synchronized DCCV for both symptom resolution and prevention of tachycardia-mediated cardiomyopathy (Botkin 2003). If cardioversion is unavailable, either a rate or a rhythm control approach is reasonable. It is often difficult to achieve adequate rate control in AFI. Diltiazem is preferred over verapamil because of its efficacy and lower incidence of hypotension (Ellenbogen 1991). Esmolol is often the preferred beta-blocker because of its rapid onset; however, other beta-blockers are reasonable (Platia 1989). Patients should be monitored for hypotension and bradycardia.

Ibutilide successfully converts AFI in 60% of cases (Stambler 1996). Concurrent therapy with intravenous magnesium may increase the efficacy—and reduce the incidence—of torsades de pointes (TdP) (Patsilinos 2010, Steinwender 2010). Continuous ECG monitoring is recommended during administration and for at least 4 hours afterward in order to document conversion as well as monitor for QTc prolongation. Dofetilide is useful for both the conversion and maintenance of sinus rhythm in hospitalized patients who are clinically stable (Page 2016). Amiodarone should be reserved for patients with heart failure or those who are refractory to other agents because of the risk of potential side effects (Delle Karth 2001).

## ATRIAL FIBRILLATION

Atrial fibrillation can present with a wide spectrum of symptoms: some patients are asymptomatic, and others may be severely affected. Severe symptoms include dyspnea at rest, angina, presyncope, and syncope. Some patients present with embolic events or heart failure symptoms, and AF is detected during the workup. Cardioversion is recommended in patients with hemodynamic instability. Electrical cardioversion and pharmacologic cardioversion have not been compared in controlled trials, but evidence from studies comparing AAD with placebo suggest lower rates of cardioversion (Naccarelli 2000). Flecainide, propafenone, and ibutilide are each useful for acute pharmacologic cardioversion. Dofetilide may be initiated for conversion and maintenance of sinus rhythm in

**Table 4.** Acute Drug Therapy for Dysrhythmias

Drug	Initial dose	Subsequent or maintenance dose	Mechanism of action	Potential adverse effects	Precautions and interactions
<i>Nucleoside</i>					
Adenosine	6 mg rapid IV bolus over 1–2 sec, followed by rapid saline flush	If no result within 1–2 min, increase to 12 mg. Can repeat x 1. The safe use of 18 mg has been reported	Slows conduction time through AV node, interrupting reentry pathways	Transient AV block, flushing, chest pain, hypotension, dyspnea	<ul style="list-style-type: none"> <li>AV block greater than first degree or SA node dysfunction (without PPM)</li> </ul>
<i>Beta blockers</i>					
Esmolol	500 mcg/kg IV bolus over 1 min	50–300 mcg/kg/min infusion	Competitively blocks response to $\beta_1$ -adrenergic stimulation with little or no effect on $\beta_2$ -receptors except at high doses	Hypotension, worsening HF, bronchospasm, bradycardia	<ul style="list-style-type: none"> <li>AV block greater than first degree or SA node dysfunction (without PPM)</li> <li>Decompensated HF</li> <li>Cardiogenic shock</li> <li>Hypotension</li> <li>Bronchospasm</li> </ul>
Metoprolol	2.5–5 mg IV bolus over 2 min	Can repeat 2.5–5 mg IV bolus in 5 min, up to 3 doses			
Propranolol	1 mg IV over 1 min	Can repeat 1 mg IV at 2-min intervals, up to 3 doses	Competitively blocks response to $\beta_1$ - and $\beta_2$ -adrenergic stimulation		
<i>Nondihydropyridine calcium channel blockers</i>					
Diltiazem	0.25 mg/kg IV bolus over 2 min	5–15 mg/h infusion	Inhibits calcium ion from entering the slow channels or selects voltage-sensitive areas of vascular smooth muscle and myocardium during depolarization; slows automaticity and conduction of AV node	Hypotension, worsening HF, bradycardia	<ul style="list-style-type: none"> <li>AV block greater than first degree or SA node dysfunction (without PPM)</li> <li>Decompensated HF</li> <li>Hypotension</li> </ul>
Verapamil <sup>a</sup>	5–10 mg IV bolus over 2 min	Can repeat 10 mg after 30 min; then 0.005 mg/kg/min infusion			
<i>Cardiac glycosides</i>					
Digoxin	0.25–0.5 mg IV bolus	Repeat 0.25 mg IV bolus up to maximum of 12 mg/kg or 1.5 mg over 24 h, given q6–8 h	Direct suppression of AV node conduction to increase effective refractory period and decrease conduction velocity	Anorexia, nausea, vomiting, visual changes	<ul style="list-style-type: none"> <li>Renal dysfunction</li> <li>AV block greater than first-degree or SA node dysfunction (without PPM)</li> <li>Multiple drug-drug interactions (DDIs)</li> </ul>
<i>Class I antiarrhythmic agents</i>					
Flecainide	50 mg PO q12h	200 mg PO q12h	Slows depolarization by blocking fast inward Na <sup>+</sup> current, prolongs effective refractory period	AFI with 1:1 AV conduction, worsening HF	<ul style="list-style-type: none"> <li>Sinus or AV conduction disease (without PPM)</li> <li>Structural heart disease</li> <li>Coronary artery disease (CAD)</li> </ul>

(Continued)

**Table 4.** Acute Drug Therapy for Dysrhythmias (Continued)

Procainamide	10–17 mg/kg IV (lean body weight) at max rate of 50 mg/min	1–4 mg/min IV	Decreases conduction velocity and may depress myocardial contractility by increasing stimulation threshold of the ventricle and His–Purkinje system	Hypotension, worsening HF, TdP	<ul style="list-style-type: none"> <li>Blood dyscrasias</li> <li>Lupuslike syndrome</li> <li>Hypotension</li> <li>Renal dysfunction</li> <li>Many DDIs</li> </ul>
Propafenone	150 mg PO q8h (immediate release [IR]) 225 mg PO q12h (sustained release [SR])	300 mg PO q8h (IR) 425 mg PO q12h (SR)	Slows depolarization by blocking fast inward Na <sup>+</sup> current, prolongs effective refractory period	AFI with 1:1 AV conduction, worsening HF	<ul style="list-style-type: none"> <li>Sinus or AV conduction disease (without PPM)</li> <li>Structural heart disease</li> <li>CAD</li> </ul>
<i>Class III antiarrhythmic agents</i>					
Amiodarone	150 mg IV over 10 min	1 mg/min x 6 h; then 0.5 mg/min x 18 h	Competitively blocks response to $\alpha_1$ , $\beta_1$ , and $\beta_2$ -adrenergic stimulation. Affects Na, K, and Ca channels and prolongs action potential and refractory period	Hypotension, bradycardia, phlebitis, QT prolongation, TdP	<ul style="list-style-type: none"> <li>Sinus or AV conduction disease (without PPM)</li> <li>Inflammatory lung disease</li> <li>Hepatic dysfunction</li> <li>Multiple DDIs</li> </ul>
Dofetilide	125–500 mcg PO q12h based on renal function and QT interval	N/A	Blockade of rapid potassium current. Increases action potential duration caused by delayed repolarization	Headache, dizziness, TdP	<ul style="list-style-type: none"> <li>Do not use if QTc &gt; 440 ms or history of TdP</li> <li>Multiple DDIs</li> <li>Initiate in hospital with continuous ECG</li> </ul>
Ibutilide	≥ 60 kg: 1 mg IV over 10 min < 60 kg: 0.01 mg/kg Consider IV magnesium infusion	Repeat 1 mg (or 0.01 mg/kg if < 60 kg) once after 10 min	Prolongs action potential duration and increases atrial and ventricular refractoriness by activation inward Na current. Inhibits rapid potassium current	QT prolongation, TdP, AV block	<ul style="list-style-type: none"> <li>Do not use if QTc &gt; 440 ms or history of TdP</li> </ul>
Sotalolol	80 mg q12h	160 mg q12h	Blockade of rapid potassium current. Increases action potential duration caused by delayed repolarization. Competitively blocks response to $\beta_1$ - and $\beta_2$ -adrenergic stimulation	QT prolongation, TdP, AV block	<ul style="list-style-type: none"> <li>Do not use if QTc &gt; 450 ms or history of TdP</li> <li>Renal dysfunction</li> <li>Sinus or AV nodal dysfunction</li> <li>Decompensated HF</li> <li>Bronchospasm</li> </ul>

<sup>a</sup>Verapamil may cause pulmonary edema in patients with hypertrophic cardiomyopathy

hospitalized patients who are relatively stable (Singh 2000). Initial dose is based on renal function, and patients must remain hospitalized and on continuous telemetry for five doses. Dofetilide may be used in patients with structural heart disease such as HF (Torp-Pedersen 1999).

A pill-in-the-pocket approach with flecainide or propafenone can be taken so as to convert AF of short duration (< 48 hours). The efficacy and safety of that approach should first be performed in a monitored setting—typically, an emergency department. Structural heart disease or ischemic heart disease must be ruled out prior to administration. Patients must be reliably aware of when they go into AF so they can promptly take the dose. And the use of an AV nodal blocker—if not on chronically—should be considered in order to prevent a rapid ventricular rate if the AF converts to AFI with 1:1 conduction (Alboni 2004).

Ibutilide has a success rate of 45%–50% in converting AF to sinus rhythm. Conversion generally occurs within 30 minutes, and rates are higher in patients with normal left atrial size or shorter arrhythmia durations. The risk of polymorphic VT has been reported as high as 8%, although it is usually nonsustained (Stambler 1996). High-dose intravenous magnesium (5 g pre- and postibutilide administration) has been shown to increase both efficacy and safety through reduction of TdP (Patsilinkos 2010). Magnesium reduces atrial automaticity and inhibits AV node conduction (Rasmussen 1988).

Despite its relative efficacy and because of its noncardiac toxicities (e.g., liver, lungs, thyroid), amiodarone should be reserved for patients with contraindications or who are refractory to other AADs. Amiodarone is considered safe for use in patients with HF. When administered intravenously, amiodarone has an effect on heart rate within 4 hours and improves ventricular rate in 74% of patients by 24 hours. On the other hand, oral amiodarone may require days to achieve effective rate control (Delle Karth 2001). Chronic administration of oral amiodarone is associated with multiple toxicities and requires consistent monitoring throughout use.

Based on the thought that waiting to see whether the medication can pharmacologically convert the rhythm is a better predictor of maintenance of sinus rhythm, some practitioners prefer to delay DCCV until after the AAD has been loaded. The data regarding that practice are limited (Malhotra 2014). Regardless, the strategy should be reserved for patients who are hemodynamically stable. In a recent study, patients presenting to an emergency department with recent-onset (< 36 hours), symptomatic AF were randomized to either a wait-and-see strategy (with rate control medications) or early cardioversion (with pharmacology +/- electrical). The wait-and-see strategy was noninferior to early cardioversion in obtaining sinus rhythm at 4 weeks. Spontaneous cardioversion often occurred in the wait-and-see group (Pluymaekers 2019).

## Postoperative AF

Postoperative AF (POAF) is diagnosed when an ECG or telemetry demonstrates AF for 30 or more seconds. The incidence of POAF varies widely based on the procedure performed but is much higher in cardiac surgeries versus noncardiac surgeries. Risk factors for development of POAF are the same as those for AF in the nonsurgical setting. In addition, a cardiac surgical procedure may produce inflammation (i.e., pericarditis), which increases the vulnerability of the atrium. It is unclear how much the arrhythmia actually contributes to mortality, but POAF is clearly associated with increased length of stay and risk of thromboembolism (Frendl 2014).

Patients chronically taking beta-blockers before surgery should continue therapy postoperatively if at all possible. A large meta-analysis demonstrated that acute withdrawal of beta-blockers before cardiac surgery increases the risk of developing POAF (Burgess 2006). Data supporting prophylactic beta-blocker initiation prior to surgery are limited. Trials have demonstrated a reduction in POAF but a high incidence of hypotension and bradycardia (Devereaux 2008). Intravenous magnesium has demonstrated success in preventing POAF in patients with low serum levels (Rostron 2005).

Patients considered to be at intermediate-to-high risk of developing POAF may warrant more-aggressive prevention strategies. Diltiazem may be considered for patients with preserved ejection fraction and who are not taking beta-blockers (Amar 2000). In addition, the efficacy of amiodarone in preventing POAF in such patients is well established. The PAPABEAR trial randomized patients undergoing nonemergency cardiac surgery to oral amiodarone (10 mg/kg daily) or placebo beginning 6 days prior to surgery and through 6 days after surgery. Atrial tachyarrhythmias occurred in fewer amiodarone patients (16.1% versus 29.5%,  $p < 0.001$ ). Adverse effects such as bradycardia, corrected QT interval (QTc) prolongation, and skin rash occurred more frequently in the amiodarone group, but there were no differences in postoperative complications or 6-month readmission (Mitchell 2005). Other dosing regimens have been studied and shown to be effective, and therefore, guidelines emphasize the importance of loading amiodarone given its large volume of distribution. The use of digoxin to prevent POAF is not recommended (Frendl 2014).

Colchicine has been evaluated for prevention of POAF given its anti-inflammatory properties. A substudy of the COPPS trial demonstrated less POAF in patients who received colchicine beginning on postoperative day 3. In addition, both the duration of POAF and length of hospital stay were reduced with colchicine (Imazio 2011). Despite those encouraging results, the delayed initiation of colchicine represented a major limitation because the majority of POAF cases occur within 48 hours of surgery.

The COPPS-2 trial was designed to address those limitations. Three-hundred sixty patients were randomized to

either colchicine (beginning 48–72 hours before cardiac surgery and continuing for 1 month) or placebo. Development of POAF was a secondary end point, and no differences were observed. The incidence of adverse effects was twice that seen in the original COPPS trial—mainly because of GI intolerance. The authors attributed the discrepancy to the increased vulnerability of patients during the perioperative phase (Imazio 2014). A meta-analysis of three trials demonstrated that perioperative colchicine was associated with decreased incidences of POAF. Gastrointestinal intolerance was higher in those receiving colchicine but did not result in early treatment discontinuation (Lee 2016). Large-scale studies with primary end points of POAF are needed to establish the true role of colchicine.

If new-onset POAF occurs, potential triggers such as bleeding, pneumothorax, airway issues, and myocardial ischemia should be managed appropriately. Catecholaminergic inotropic agents should be weaned and discontinued if hemodynamics allow. Electrolytes and fluid balance should be optimized. Cardioversion is indicated in patients with hemodynamic instability.

Data comparing rate and rhythm control strategies in hemodynamically stable patients are limited, and thus, either approach is reasonable. There is a high rate of spontaneous conversion to sinus rhythm within 24 hours of onset, which favors an initial rate control approach. Intravenous esmolol, metoprolol, or diltiazem generally has the same efficacy as that seen in nonsurgical AF. Digoxin or amiodarone may be used in cases of hypotension and/or heart failure. If AF persists beyond 24–48 hours, rhythm control may be attempted with any AAD that is not contraindicated based on comorbidities (Frenzl 2014).

### Accessory Pathways

An accessory pathway is defined as a pathway that directly connects the atrium to the ventricle, bypassing normal conduction through the AV node and the His–Purkinje system (see Table 3). A pathway is considered manifest if it conducts in the anterograde (normal, forward) direction, demonstrating early activation (preexcitation), with a delta wave on the ECG. Concealed pathways conduct in the retrograde (reverse) direction and therefore do not cause preexcitation on the ECG. The most common accessory pathway is orthodromic (normal direction) AVRT, wherein the circuit travels in the retrograde direction from the ventricle to the atrium and then through the AV node in the anterograde direction (Page 2016). A diagnosis of Wolff–Parkinson–White (WPW) syndrome is reserved for patients who have symptoms consistent with SVT and who demonstrate ventricular preexcitation on a resting sinus ECG. Rapid accessory pathway conduction during AF can result in sudden cardiac death (SCD) in patients with manifest accessory pathways. Unfortunately, SCD may be the first presentation of WPW syndrome. The risk of SCD

associated with WPW syndrome appears highest in the first 2 decades of life.

Synchronized DCCV is recommended in the case of hemodynamic instability. Patients may perform vagal maneuvers for acute conversion, and they should be educated about proper ways to perform when diagnosed (Smith 2013, Wen 1998). Adenosine is effective in converting orthodromic AVRT in 90%–95% of patients (Delaney 2011, DiMarco 1985). Electrical cardioversion should be available because adenosine can precipitate an AF that may conduct rapidly to the ventricle and trigger a ventricular fibrillation.

Ibutilide or procainamide decreases ventricular rate by slowing conduction over the accessory pathway in patients with preexcited AF (Glatter 2001). Digoxin should be avoided in such patients because it can shorten the refractoriness of the accessory pathway and increase ventricular rate (Sellers 1977). In addition, amiodarone, beta-blockers, and nondihydropyridine calcium channel blockers should be avoided in preexcited AF because they can enhance conduction over the accessory pathway by slowing conduction through the AV node (Boriani 1996, Shiraishi 2002).

## ANTICOAGULATION

Patients with AF and/or AFI should be considered to have the same risks of thromboembolism. Thus, the recommendations for anticoagulation with regard to either pharmacological or electrical cardioversion of patients are the same. Cardioversion may be performed in dysrhythmia of duration of less than 48 hours. Patients with CHA<sub>2</sub>DS<sub>2</sub>-VASc scores of 2 or more in men and 3 or more in women should receive anticoagulation as soon as possible before cardioversion, followed by long-term anticoagulation. Patients with CHA<sub>2</sub>DS<sub>2</sub>-VASc scores of 0 in men or 1 in women may be anticoagulated prior to cardioversion, but they do not require postcardioversion oral anticoagulation (January 2019). Anticoagulation options include intravenous heparin, low-molecular-weight heparin, factor Xa inhibitor, and direct thrombin inhibitor. If AF duration is more than 48 hours and if it is clinically feasible, performing a transesophageal echocardiogram prior to cardioversion to rule out left-atrial-appendage thrombus is preferred (January 2014).

Long-term anticoagulation is indicated in patients with AF and elevated CHA<sub>2</sub>DS<sub>2</sub>-VASc scores of 2 or more in men or 3 or more in women. Options include warfarin or a DOAC (dabigatran, rivaroxaban, apixaban, or edoxaban) (January 2019) (Table 5). Based on such properties as shorter drug onset and offset of action; more-predictable pharmacokinetics; fewer drug, food, and disease interactions; and less-strict monitoring requirements, direct-acting oral anticoagulants (DOACs) serve as simplified long-term anticoagulation management in patients with AF/AFI. Warfarin remains a viable option for patients unable or unwilling to take a DOAC. The availability

of reversible agents for DOACs—including idarucizumab for direct thrombin inhibitors (dabigatran) and andexanet alfa for factor Xa inhibitors (apixaban and rivaroxaban), respectively—has increased the comfort level for many providers and patients (January 2019).

The need to bridge anticoagulation has long been considered controversial with regard to balancing the risk of bleeding as well as thromboembolism. The BRIDGE trial compared bridging anticoagulation therapy—with low-molecular-weight heparin—with placebo in patients treated with warfarin who were undergoing elective invasive procedures. The incidence of arterial thromboembolism was 0.4% in the no-bridging group and 0.3% in the bridging group ( $p=0.01$  for noninferiority). The incidence of major bleeding was significantly higher in the bridging group (Douketis 2015). Although acute emergencies do not usually offer the opportunity for a bridging option, it is important to know that the practice of bridging has decreased significantly since publication of the BRIDGE trial. The practice is generally reserved for those at very high risk (i.e., mechanical valve, history of stroke, high CHA<sub>2</sub>DS<sub>2</sub>-VASc score).

**Table 5.** Anticoagulation for Atrial Fibrillation/Atrial Flutter

Drug	Dose
<i>Heparins</i>	
Enoxaparin	1 mg/kg subq q12h
Heparin	Weight-based dosing, institution-specific nomograms recommended
<i>Factor Xa inhibitors</i>	
Apixaban	5 mg PO q12h 2.5 mg PO q12h if patient has at least 2 of the following: Age ≥ 80 Weight < 60 kg SCr > 1.5 mg/dL
Edoxaban	CrCl > 95 mL/min: Do not use CrCl 50–95 mL/min: 60 mg daily CrCl 15–50 mL/min: 30 mg daily CrCl < 15: Avoid use
Rivaroxaban	CrCl > 50 mL/min: 20 mg daily with largest meal CrCl 15–50 mL/min: 15 mg daily with largest meal CrCl < 15 mL/min: Avoid use
<i>Direct thrombin inhibitor</i>	
Dabigatran	CrCl > 30 mL/min: 150 mg q12h CrCl 15–30 mL/min: 75 mg q12h CrCl < 15 mL/min: Avoid use

## PREMATURE VENTRICULAR CONTRACTIONS

Premature ventricular contractions (PVCs) result when the ventricular myocytes spontaneously depolarize to create extra systole that is out of sync with the cardiac cycle (Cantillon 2013). PVCs can often be incidental findings during routine monitoring. Patients may complain of palpitations, shortness of breath, chest pain, fatigue, and dizziness, but many experience periodic PVCs without any accompanying symptoms. The decision to treat PVCs is based on whether a patient has structural heart disease and/or whether the symptoms are affecting the patient's quality of life (Pedersen 2014).

In assessing treatment strategies, it is important that the clinician complete a thorough physical exam and ECG evaluation. Box 3 gives a brief overview of underlying causes, including disease states and medications (Cantillon 2013). All of those should be evaluated and treated and any offending medication therapies discontinued. Avoidance of triggers such as caffeine is reasonable—especially in patients who demonstrate significant correlation between the trigger and symptoms. Even though trigger avoidance and behavior modifications should be encouraged, data have yet to show benefits in symptoms or outcomes (DeBacker 1979).

Once underlying causes have been evaluated and appropriate assessments have been made, the decision about how to best treat PVCs depends on whether the patient has structural heart disease. In the absence of structural heart disease, PVCs are often untreated given their benign nature. If the patient exhibits intolerable symptoms or a high PVC

### Box 3. Underlying Causes of PVCs

#### Diseases

- Adrenal gland conditions
- Obstructive sleep apnea
- Pulmonary hypertension
- Sarcoidosis
- Sex hormone abnormalities
- Structural heart disease
  - CAD
  - CHF
  - Congenital heart disease
  - Hypertrophic cardiomyopathy
  - MI
  - Prolonged QT syndromes
  - Valvular heart disease
- Thyroid disease

#### Pharmacological causes

- Alpha-, beta-, dopamine-receptor agonists
- Amphetamine
- Cocaine
- Methamphetamine
- Milrinone
- OTC sympathomimetic agents

burden, treatment with a beta-blocker, diltiazem, or verapamil can be initiated. It is important to know that the efficacy of those medications is small, with only 10% – 15% of patients achieving more than 90% PVC suppression (Stec 2012). AAD therapy may be considered in those patients if they are unresponsive to previous therapy and have high PVC burdens or intolerable symptoms.

Coronary artery disease and resultant tissue injury and tissue death are common causes of spontaneous ventricular ectopy (Bigger 1977, Eldar 1992). Tissue reperfusion during coronary revascularization also increases the likelihood of reentrant VAs (Cantillon 2013). Both the CAST and MADIT II trials confirmed that attempts to suppress those ectopic arrhythmias did not improve outcomes and are instead associated with increased mortality in structural heart disease and ischemic heart disease (CAST 1989, Moss 2002). In patients with CAD and other forms of structural heart disease, the treatment of PVCs should be initiated only if patients exhibit significant symptoms, are hemodynamically unstable, or have high burdens—especially in the setting of impaired LV function. If treatment is indicated, beta-blockers should be used as first-line agents. If beta-blocker therapy is contraindicated or the patient is experiencing significant PVCs despite optimal beta-blocker therapy, amiodarone may be initiated. Catheter ablation is indicated if patients continue having significant symptoms and/or very high PVC burdens despite medical therapy. Studies indicate high rates of ablation efficacy, with reported rates of PVC suppression in 74% – 100% of patients, with low complication rates (~1%) (Pedersen 2014).

## VENTRICULAR TACHYCARDIA WITH A PULSE

### LVAD-Associated VT

The development of VAs is extremely common in patients with left-ventricular-assist devices (LVADs) as a result of relative electrophysiological-instability status postimplantation (Harding 2001). Ventricular arrhythmias are most common in the immediate postoperative period and generally decrease in frequency during the subsequent weeks to months. Even though the presence of a preimplantation VA is the most powerful predictor for postimplantation VA, one study found that new-onset monomorphic VT occurred in as many as 18% of patients who had left-ventricular-assist devices—without previous VT (Ziv 2005).

The development of VA in patients with left-ventricular-assist devices can also depend on other underlying factors such as ischemia, fibrosis, positive inotrope or vasopressor therapy, mechanical induction from inflow cannula, and suction events (Vollkron 2007). The direct impact of VA in patients with left-ventricular-assist devices is highly variable, with some patients reporting good tolerance of symptoms of weakness and palpitations but no syncope (Oz 1994). In an evaluation of the impact of VA in patients with left-ventricular-assist

devices, it is very difficult to correlate a relationship with mortality given the complex underlying clinical conditions and the pathophysiologies of the patients. In 2015, a meta-analysis of nine studies encompassing 1179 patients identified pre-LVAD VA as a major risk factor for mortality in the LVAD population (Makki 2015). Even though the data are compelling, they should be considered only as hypothesis generating, because other evaluations—that included data from the INTERMACS registry—found that arrhythmias were not found to be predictors of mortality (Kirklin 2015).

Despite the expected complication of VA post LVAD implantation, no great data exist to guide treatment, and therefore, clinicians must rely on case reports or smaller observational studies to evaluate options. Data regarding the management of VA in patients with structural heart disease and ICDs are often used for extrapolating the management of LVAD VA. Even though no AAD therapy has shown better survival over ICD placement, pharmacotherapy could be used in patients with ICDs and VAs so as to improve symptoms, decrease ICD interventions, and prevent hemodynamic instability, which is especially important in patients with LVADs (Gopinathannair 2019). Given that VA is generally well tolerated in the LVAD population, it is important to balance the risks associated with AAD therapy with any potential benefit. Many patients with left-ventricular-assist devices are already on beta-blocker therapy, titrated to the optimal outcome dose based on underlying cardiovascular conditions. In fact, conflicting data exist regarding the potential benefit of beta-blockers in VA treatment in patients with left-ventricular-assist devices. One study of 42 such patients found a strong association between beta-blocker nonuse and the risk of developing VA (odds ratio, 7.04 [p=0.001]), and another study—of 23 such patients—found no identified predictors of VA, including beta-blocker usage (Andersen 2009, Refaat 2008). The most-robust data to guide therapy come from the OPTIC (Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients) trial, which compared three interventions: beta-blocker monotherapy, beta-blocker in conjunction with amiodarone, and sotalol monotherapy. Although the study did not include the LVAD population, it demonstrated a significantly decreased rate of recurrent VT in the amiodarone plus beta-blocker group and the sotalol monotherapy group compared with beta-blocker monotherapy (Connolly 2006).

To look specifically at the LVAD population, an evaluation of 15 patients in whom amiodarone had been initiated as secondary prevention demonstrated a 60% arrhythmia-free survival (Raasch 2012). However, a larger, multicenter evaluation of 488 patients with left-ventricular-assist devices found that baseline amiodarone use was independently associated with an increase in mortality (HR, 1.77 [95% CI, 1.1–2.8]; p=0.018) (Gopinathannair 2019). If therapy is needed acutely, amiodarone or a sodium channel blocker such as lidocaine or procainamide could be used temporarily to alleviate symptoms. In the absence of clear data to support or refute the use



of amiodarone, the long-term toxicities of amiodarone must be weighed against the potential benefits of initiating therapy. Catheter ablation is another option for the treatment of VA in patients when AAD therapy was either not tolerated or was undesired by the patient or was ineffective. Short-term success ranges from 77% to 86% (Gopinathannair 2019).

### VT/ICD Storm

Electrical storm is characterized by three or more episodes of sustained VT, ventricular fibrillation, or appropriate ICD shocks within a 24-hour time period (Al-Khatib, 2018). Occasionally, the patient's presentation may be less of an emergency (i.e., VT that is slower than the programmed detection rate of the ICD). Such patients may complain of palpitations, chest pain, or presyncope but be relatively hemodynamically stable at presentation. Amiodarone has demonstrated efficacy in reducing the frequency of VT in numerous trials. As mentioned previously, the OPTIC trial demonstrated benefit from amiodarone combined with beta-blocker and sotalol monotherapy (Connolly 2006, Hohnloser 2006).

Procainamide and amiodarone were compared in the PROCAMIO study. Patients with tolerated monomorphic wide QRS tachycardia were assigned to intravenous procainamide (10 mg/kg over 20 minutes) or amiodarone (5 mg/kg over 20 minutes). The primary end point of major cardiac adverse events occurred in 9% of the procainamide and 41% of the amiodarone patients ( $p=0.006$ ). The tachycardia also terminated earlier in the procainamide group. It is important to note that procainamide was used in patients with structural heart disease and that the incidence of the primary end point was less in the procainamide group (Ortiz 2017).

Beta-blockers are used for reducing the adrenergic surge associated with VT. Propranolol appears to be more effective than metoprolol. A single-center, double-blind study of patients with ICDs who experienced electrical storm randomized patients to propranolol (40 mg PO q6h) or metoprolol (50 mg PO q6h) for the first 48 hours. All patients received intravenous amiodarone. The time to termination of VA was shorter in patients receiving propranolol (3 versus 18 hours). In addition, 27 of 30 patients who received propranolol were arrhythmia free within 24 hours (versus 16 of 30 who received metoprolol) (Chatzidou 2018).

### CONCLUSION

Despite advances in ablation technology, beta-blockers, nondihydropyridine calcium channel blockers, and antiarrhythmics continue to be the main pharmacological treatment options for acute dysrhythmias. There has been little drug development for acute dysrhythmias in the past 10 years, but recent studies continue to clarify ways of best identifying high-risk patients and of optimizing management. Medication choice involves considerations of relative efficacy, comorbidities, and adverse effects.

### Practice Points

- The treatment of acute bradycardia should include efforts to immediately maintain perfusion and to determine possible treatment of reversible causes.
- Patients who are hemodynamically unstable based on any type of tachycardia should receive DCCV.
- Beta-blockers and nondihydropyridine calcium channel blockers may be used for rate control in patients with atrial tachyarrhythmias and no contraindications.
- To make sure there are no comorbidity contraindications, careful consideration should be given when adding AAD.
- In a patient who has been experiencing an AT for less than 48 hours, the need for anticoagulation before and after cardioversion is determined by calculating the CHA<sub>2</sub>DS<sub>2</sub>-VASc score.
- Anticoagulation bridging should be reserved for patients at very high risk of a thrombotic event.
- Ensure that underlying causes have been evaluated prior to initiation of therapy for patients with PVCs.

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# Self-Assessment Questions

## Questions 1 and 2 pertain to the following case.

J.M., a 60-year-old woman (weight 85 kg) with ischemic cardiomyopathy (EF 30%), presents to the ED with dyspnea at rest, fatigue, and palpitations. Her vital signs include: BP 105/75 mm Hg, HR 142 beats/minute, respiratory rate 22 breaths/minute, and O<sub>2</sub> saturation 95% on 2 L nasal cannula. Physical examination reveals: 12 cm jugular vein distention (JVD), crackles bilaterally, irregular rhythm, and 3+ pitting edema. J.M. is alert and oriented x 4. Pertinent labs include potassium 3.9 mmol/L, SCr 1.6 mg/dL (baseline), and magnesium 1.9 mg/dL; ECG demonstrates a regular narrow complex tachycardia. J.M.'s home drugs include lisinopril 20 mg daily, metoprolol succinate 50 mg daily, and furosemide 20 mg twice daily.

- Which one of the following is best to recommend as first-line therapy for J.M.?
  - Adenosine 6 mg IV bolus
  - Metoprolol 5 mg IV bolus
  - Amiodarone 150 mg IV bolus
  - Diltiazem 10 mg IV bolus
- Two hours after initial therapy and monitoring, J.M. is back in supraventricular tachycardia (SVT). Her vital signs are: BP 80/55 mm Hg and HR 155 beats/minute. She is lethargic and confused. Which one of the following is best to recommend for J.M.?
  - Adenosine 6 mg IV bolus
  - Amiodarone 150 mg IV bolus
  - Cardioversion
  - Diltiazem 20 mg IV bolus
- An 18-year-old man has recently been diagnosed with Wolff–Parkinson–White syndrome (WPW) and is scheduled for ablation in a month. He presents to the ED in a rapid irregularly irregular rhythm. Which one of the following is best to recommend for this patient?
  - Propranolol 1 mg IV
  - Diltiazem 20 mg IV
  - Procainamide 15 mg/kg IV
  - Digoxin 0.5 mg IV
- A 74-year-old man (weight 75 kg, height 70 in) with non-ischemic cardiomyopathy (EF 20%) presents to the ED with complaints of chest pain and shortness of breath. His vital signs are: BP 125/90 mm Hg, HR 148 beats/minute, respiratory rate 20 breaths/minute, and O<sub>2</sub> sat 95% on 2 L nasal cannula. Physical examination reveals irregularly irregular rhythm, clear to auscultation, trace LE edema. Pertinent labs include potassium 4.2 mmol/L, SCr 1.8 mg/dL, and magnesium 2.1 mg/dL; ECG reveals atrial fibrillation. The patient's home drugs include losartan 50 mg daily, carvedilol 25 mg twice daily, furosemide 40 mg daily, and spironolactone 25 mg daily. Which one of the following is best to recommend to manage this patient's atrial fibrillation?
  - Diltiazem
  - Sotalol
  - Flecainide
  - Amiodarone
- A 65-year-old woman is postoperative day 1 mitral valve replacement surgery. Overnight she develops atrial fibrillation (AF) with rapid ventricular response (RVR). The patient is hemodynamically stable and has no history of AF. Her EF is 50%. Which one of the following is best to recommend for this patient's new-onset AF?
  - Digoxin
  - Propafenone
  - Amiodarone
  - Diltiazem
- A 58-year-old man with a history of CAD, paroxysmal AF, and GERD presents to the ED after a bike collision. He has a radial fracture that will require surgical repair. The patient's home drugs include omeprazole, apixaban, lisinopril, and carvedilol. Pertinent labs include: potassium 3.6 mmol/L, magnesium 1.9 mg/dL, and SCr 1.1 mg/dL. Surgery is planned for the next day. The team asks you about bridging anticoagulation. Which one of the following is best to recommend for this patient?
  - Hold apixaban and begin enoxaparin
  - Hold apixaban and begin heparin
  - Continue apixaban uninterrupted
  - Hold apixaban and do not bridge
- A 58-year-old man presents with ICD firing. He was fishing with a friend when he experienced sudden dizziness followed by a shock and lost consciousness. Interrogation reveals VT and a total of 4 shocks delivered. The patient's medical history is significant for hypertension, CAD (LAD stent 2 years ago), ischemic cardiomyopathy (EF 25%), and diabetes. His vital signs include: BP 101/74 mm Hg, HR 98 beats/minute, respiratory rate 18 breaths/minute, and O<sub>2</sub> saturation 95% on 2 L nasal cannula. Physical examination reveals: regular rhythm, lungs clear to auscultation, trace LE edema. Pertinent labs include potassium 3.1 mmol/L, SCr 1.7 mg/dL (baseline), and magnesium 1.5 mg/dL; ECG demonstrates normal sinus rhythm (NSR). His medications include: aspirin 81 mg daily, atorvastatin 80 mg daily, carvedilol 12.5 mg twice daily, metformin 1000 mg twice daily, lisinopril 10 mg daily, and spironolactone 25 mg daily. As you

enter the room with the team, the patient goes into VT at a rate of 140 beats/minute. His ICD shock zone is set at 170 beats/minute. He is anxious but otherwise alert. Which one of the following is best to recommend for this patient?

- A. Allow ICD to shock
  - B. IV Amiodarone
  - C. IV Lidocaine
  - D. IV Procainamide
8. In a study of patients with atrial fibrillation and heart failure, flecainide was associated with greater mortality than placebo. Death occurred in 5.9% of flecainide-treated patients and 2.2% of patients treated with placebo. What is the number needed to harm associated with flecainide use in this study?
- A. 12
  - B. 17
  - C. 27
  - D. 45
9. A 64-year-old woman presents to the ED feeling fatigued and tired for the past week. Her medical history is significant for hypertension, depression, and asthma. Her vital signs include: BP 82/58 mm Hg, HR 34 beats/minute, respiratory rate 16 breaths/minute, and O<sub>2</sub> saturation 98% on room air; ECG demonstrates first-degree AV block. The patient is able to maintain her airway and can answer questions. She states that she feels horrible, nauseated, and just wants to sleep. Her home drugs include: hydrochlorothiazide 25 mg daily, citalopram 20 mg daily, fluticasone 110 mcg 2 puffs twice daily, and albuterol 2 puffs every 4 hours as needed. Which one of the following is best to recommend for this patient?
- A. Atropine 0.5 mg IV
  - B. Dopamine 5 mcg/kg/min IV infusion
  - C. Normal Saline 20 mL/kg IV bolus
  - D. Epinephrine 1 mg IV

**Questions 10–13 pertain to the following case.**

A rapid response call is issued for J.B., a 71-year-old woman admitted to the general medicine floor for a UTI. According to the nurse, the patient's heart rate has dropped down into the 30s for the past few minutes. J.B.'s medical history is significant for COPD, hypertension, CAD (RCA stent 7 years ago), ischemic cardiomyopathy (EF 25%), and hyperlipidemia. Her vital signs include: BP 90/60 mm Hg, HR 32 beats/minute, respiratory rate 20 breaths/minute, and O<sub>2</sub> saturation 96% on 4 L nasal cannula. Physical examination reveals lungs with slight rales in the bases bilaterally and trace LE edema. J.B. complains of a little shortness of breath and feels tired but otherwise feels fine. Her medications include: aspirin 81 mg daily, atorvastatin 40 mg daily, metoprolol succinate 100 mg

daily, lisinopril 10 mg daily, furosemide 20 mg daily, potassium chloride 10 meq daily, tiotropium 18 mcg inhaled twice daily, and ceftriaxone 1 g IV daily.

10. Given her clinical picture, which one of the following is best to recommend to support J.B. and the rapid response team?
- A. Initiate chest compressions because her HR is too low to maintain cerebral perfusion pressure.
  - B. Because she is maintaining adequate circulation at this time, review her medication administration record for potential causes of bradycardia.
  - C. Initiate treatment with epinephrine as a bridge to transvenous pacing.
  - D. Initiate treatment with a 20 mL/kg bolus of normal saline to treat her blood pressure in the setting of a UTI.
11. Upon review of J.B.'s medication administration record and a discussion with the primary nurse, you identify that the patient received 200 mg of metoprolol tartrate 2 hours ago instead of her schedule 100 mg of metoprolol succinate. J.B. is now starting to feel a little more tired and her responses to the team's questions are a bit delayed. Her vital signs include: BP 80/50 mm Hg, HR 27 beats/minute, respiratory rate 16 breaths/minute, and O<sub>2</sub> saturation 96% on 6 L nasal cannula. Which one of the following is best to recommend for J.B.?
- A. Contact nephrology to initiate hemodialysis for an acute metoprolol overdose.
  - B. Contact the cardiology service to discuss implantation of a permanent pacemaker.
  - C. Initiate therapy with IV glucagon.
  - D. Initiate therapy with a phenylephrine infusion to increase blood pressure.
12. J.B. is now responding intermittently to questions with one-word answers. Her current vital signs include BP 60/40, HR 20 beats/minute, respiratory rate 12 breaths/minute, and O<sub>2</sub> sat 95% on 6 L nasal cannula. Which one of the following is best to recommend initiating for J.B.?
- A. Dopamine infusion
  - B. IV high dose insulin therapy
  - C. IV calcium
  - D. Transvenous pacing
13. J.B. is currently on a dopamine infusion at 15 mcg/kg/minute with the following vital signs: BP 80/60 mm Hg, HR 35 beats/minute, respiratory rate 12 breaths/minute, and O<sub>2</sub> saturation 96% on 100% nonrebreather mask. She has become a bit more alert after the dopamine initiation but is still answering questions with one or two words. Her dopamine is increased to 20 mcg/kg/min with no clinical change in her status and the decision is made to

start transcutaneous pacing while setting up for insertion of a transvenous pacer. Which one of the following is best to recommend for J.B.?

- A. Intubate and start a midazolam infusion.
  - B. Intubate and start a dexmedetomidine infusion.
  - C. Administer a dose of fentanyl.
  - D. Administer a dose of morphine.
14. A 38-year-old man presents to the ED with complaints of racing heartbeat. He states that every once in a while it feels like his heart is beating out of his chest and he gets very uncomfortable. His medical history is significant for ADHD and migraine headaches. His vital signs include: BP 112/70 mm Hg, HR 65 beats/minute, respiratory rate 14 breaths/minute, and O<sub>2</sub> saturation 98% on room air. Physical exam is unremarkable. The patient's home drugs include: dextroamphetamine and amphetamine salts 5 mg twice daily and ibuprofen as needed for headaches. While talking with the patient about his home drugs he has 3 PVCs and complains that his heart just raced. The decision is made to observe him in the ED for the next 6 hours to complete a cardiac work up, and he continues to have several episodes of PVCs. The patient reveals that he stopped taking the dextroamphetamine and amphetamine salt a week ago because he was worried that it was causing his heart to race. His cardiac

work up reveals no underlying structural heart disease. The patient becomes very tearful at this time and states that he is really scared that something is wrong because this is happening all the time. Which one of the following is best to recommend for this patient?

- A. Initiate therapy with metoprolol.
  - B. Initiate therapy with amiodarone.
  - C. Do not initiate any therapy at this time.
  - D. Consult cardiology to initiate catheter ablation.
15. A 62-year-old man who is 3 days post-surgery for LVAD placement has had two runs of VT over the past 24 hours. During these episodes he states he did feel "a little funny" but reports no weakness, dizziness, or shortness of breath. His medical history includes: HFrEF, hypertension, hyperlipidemia, diabetes, and gout. The patient's medications include: atorvastatin 40 mg daily, losartan 100 mg daily, aspirin 81 mg daily, carvedilol 25 mg twice daily, warfarin 5 mg daily, and insulin glargine 20 units daily. Which one of the following is best to recommend for this patient?
- A. Discontinue carvedilol and initiate amiodarone.
  - B. Initiate sotalol.
  - C. Initiate amiodarone.
  - D. Initiate no new therapy.