Part 7.3: Management of Symptomatic Bradycardia and Tachycardia

Cardiac arrhythmias are a common cause of sudden death. ECG monitoring should be established as soon as possible for all patients who collapse suddenly or have symptoms of coronary ischemia or infarction. To avoid delay, apply adhesive electrodes with a conventional or automated external defibrillator (AED) or use the "quick-look" paddles feature on conventional defibrillators. For patients with acute coronary ischemia, the greatest risk for serious arrhythmias occurs during the first 4 hours after the onset of symptoms (see Part 8: "Stabilization of the Patient With Acute Coronary Syndromes").[1]

Principles of Arrhythmia Recognition and Management

The ECG and rhythm information should be interpreted within the context of total patient assessment. Errors in diagnosis and treatment are likely to occur if ACLS providers base treatment decisions solely on rhythm interpretation and neglect clinical evaluation. Providers must evaluate the patient's symptoms and clinical signs, including ventilation, oxygenation, heart rate, blood pressure, and level of consciousness, and look for signs of inadequate organ perfusion. These guidelines emphasize the importance of clinical evaluation and highlight principles of therapy with algorithms that have been refined and streamlined since the 2000 edition of the guidelines.[2] The principles of arrhythmia recognition and management in adults are as follows:

- If bradycardia produces signs and symptoms (eg, acute altered mental status, ongoing severe ischemic chest pain, congestive heart failure, hypotension, or other signs of shock) that persist despite adequate airway and breathing, prepare to provide pacing. For symptomatic high-degree (second-degree or third-degree) atrioventricular (AV) block, provide transcutaneous pacing without delay.
- If the tachycardic patient is unstable with severe signs and symptoms related to tachycardia, prepare for immediate cardioversion.
- If the patient with tachycardia is stable, determine if the patient has a narrow-complex or wide-complex tachycardia and then tailor therapy accordingly.
- You must understand the initial diagnostic electrical and drug treatment options for rhythms that are unstable or immediately life-threatening.
- Know when to call for expert consultation regarding complicated rhythm interpretation, drugs, or management decisions.

A comprehensive presentation of the evaluation and management of bradyarrhythmias and tachyarrhythmias is beyond the scope of these guidelines. For further information see the following sources:

- American College of Cardiology/American Heart Association/European Society of Cardiology Guidelines for the Management of Patients With Supraventricular Arrhythmias,[3] available at the following sites: *www.acc.org, www.americanheart.org,* and *www.escardio.org.*
- *ACLS: Principles and Practice,* Chapters 12 through 16.[4]

There are 3 major sections in Part 7.3. The first 2 sections, "Bradycardia" and "Tachycardia," begin with evaluation and treatment and provide an overview of the information summarized in the ACLS bradycardia and tachycardia algorithms. To simplify these algorithms, we have included some recommended drugs but not all possible useful drugs. The overview presents information about the drugs cited in the algorithms. The third section, "Antiarrhythmic Drugs," provides more detailed information about a wider selection of drug therapies.

Bradycardia

See the Bradycardia Algorithm, Figure 1. Box numbers in the text refer to the numbered boxes in the algorithm.

Evaluation

Bradycardia is generally defined as a heart rate of <60 beats per minute (Box 1). A slow heart rate may be physiologically normal for some patients, and heart rates >60 beats per minute may be inadequate for others. This bradycardia algorithm focuses on management of clinically significant bradycardia (ie, bradycardia that is inadequate for clinical condition).

Initial treatment of any patient with bradycardia should focus on support of airway and breathing (Box 2). Provide supplementary oxygen, place the patient on a monitor, evaluate blood pressure and oxyhemoglobin saturation, and establish intravenous (IV) access. Obtain an ECG to better define the rhythm. While initiating treatment, evaluate the clinical status of the patient and identify potential reversible causes.

The provider must identify signs and symptoms of poor perfusion and determine if those signs are likely to be caused by the bradycardia (Box 3). Signs and symptoms of bradycardia may be mild, and asymptomatic patients do not require treatment. They should be monitored for signs of deterioration (Box 4A). Provide immediate therapy for patients with hypotension, acute altered mental status, chest pain, congestive heart failure, seizures, syncope, or other signs of shock related to the bradycardia (Box 4).
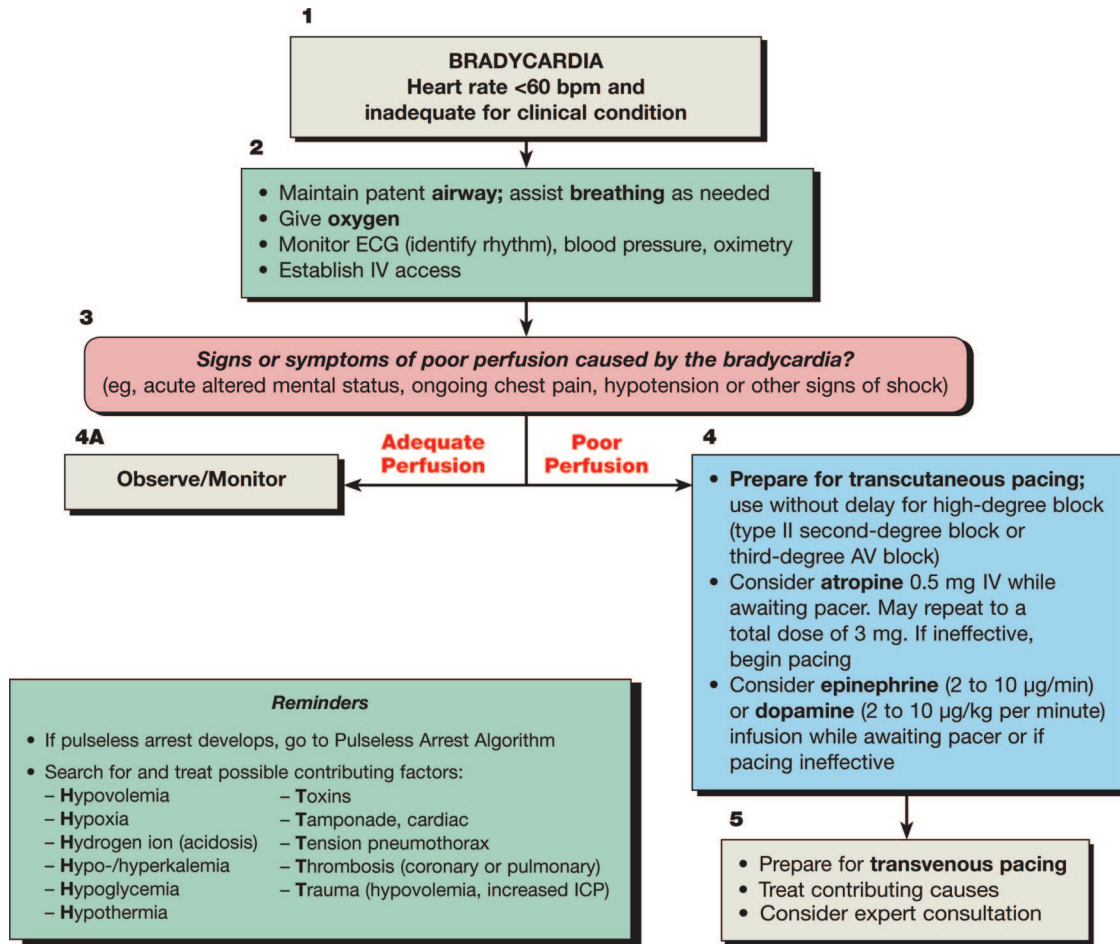
Figure 1. Bradycardia Algorithm.

AV blocks are classified as first, second, and third degree. They may be caused by medications or electrolyte disturbances, as well as structural problems resulting from acute myocardial infarction and myocarditis. A first-degree AV block is defined by a prolonged PR interval (>0.20 second) and is usually benign. Second-degree AV block is divided into Mobitz types I and II. In Mobitz type I block, the block is at the AV node; the block is often transient and may be asymptomatic. In Mobitz type II block, the block is most often below the AV node at the bundle of His or at the bundle branches; the block is often symptomatic, with the potential to progress to complete (third-degree) AV block. Third-degree heart block may occur at the AV node, bundle of His, or bundle branches. When third-degree AV block is present, no impulses pass between the atria and ventricles. Third-degree heart block can be permanent or transient, depending on the underlying cause.

Therapy (Box 4)

Be prepared to initiate transcutaneous pacing quickly in patients who do not respond to atropine (or second-line drugs if these do not delay definitive management). Pacing is also recommended for severely symptomatic patients, especially when the block is at or below the His-Purkinje level (ie, type II second-degree or third-degree AV block).

Atropine

In the absence of reversible causes, atropine remains the first-line drug for acute symptomatic bradycardia (Class IIa). In 1 randomized clinical trial in adults (LOE 2)[5] and additional lower-level studies (LOE 4),[6,7] IV atropine improved heart rate and signs and symptoms associated with bradycardia. An initial dose of 0.5 mg, repeated as needed to a total of 1.5 mg, was effective in both in-hospital and out-of-hospital treatment of symptomatic bradycardia.[5–7] Transcutaneous pacing is usually indicated if the patient fails to respond to atropine, although second-line drug therapy with drugs such as dopamine or epinephrine may be successful (see below).

Use transcutaneous pacing without delay for symptomatic high-degree (second-degree or third-degree) block. Atropine sulfate reverses cholinergic-mediated decreases in heart rate and should be considered a temporizing measure while awaiting a transcutaneous pacemaker for patients with symptomatic high-degree AV block. Atropine is useful for treating symptomatic sinus bradycardia and may be beneficial for any type of AV block at the nodal level.[7]

The recommended atropine dose for bradycardia is 0.5 mg IV every 3 to 5 minutes to a maximum total dose of 3 mg. Doses of atropine sulfate of <0.5 mg may paradoxically result in further slowing of the heart rate.[8] Atropine administration should not delay implementation of external pacing for patients with poor perfusion.

Use atropine cautiously in the presence of acute coronary ischemia or myocardial infarction; increased heart rate may worsen ischemia or increase the zone of infarction.

Atropine may be used with caution and appropriate monitoring following cardiac transplantation. It will likely be ineffective because the transplanted heart lacks vagal innervation. One small uncontrolled study (LOE 5)[9] documented paradoxical slowing of the heart rate and high-degree AV block when atropine was administered to patients after cardiac transplantation.

Avoid relying on atropine in type II second-degree or third-degree AV block or in patients with third-degree AV block with a new wide-QRS complex. These patients require immediate pacing.

Pacing

Transcutaneous pacing is a Class I intervention for symptomatic bradycardias. It should be started immediately for patients who are unstable, particularly those with high-degree (Mobitz type II second-degree or third-degree) block. Some limitations apply. Transcutaneous pacing can be painful and may fail to produce effective mechanical capture. If cardiovascular symptoms are not caused by the bradycardia, the patient may not improve despite effective pacing.

Transcutaneous pacing is noninvasive and can be performed by ECC providers at the bedside. Initiate transcutaneous pacing immediately if there is no response to atropine, if atropine is unlikely to be effective, or if the patient is severely symptomatic. Verify mechanical capture and reassess the patient's condition. Use analgesia and sedation for pain control, and try to identify the cause of the bradyarrhythmia.

If transcutaneous pacing is ineffective (eg, inconsistent capture), prepare for transvenous pacing and consider obtaining expert consultation.

Alternative Drugs to Consider

These drugs are not first-line agents for treatment of symptomatic bradycardia. They may be considered when the bradycardia is unresponsive to atropine and as temporizing measures while awaiting the availability of a pacemaker. To simplify the algorithm, we have listed epinephrine and dopamine as alternative drugs to consider (Class IIb); they are widely available and familiar to ACLS clinicians. In this section we also summarize evidence in support of other drugs that may be considered.

Epinephrine

Epinephrine infusion may be used for patients with symptomatic bradycardia or hypotension after atropine or pacing fails (Class IIb). Begin the infusion at 2 to 10 μg/min and titrate to patient response. Assess intravascular volume and support as needed.

Dopamine

Dopamine hydrochloride has both α- and β-adrenergic actions. Dopamine infusion (at rates of 2 to 10 μg/kg per minute) can be added to epinephrine or administered alone. Titrate the dose to patient response. Assess intravascular volume and support as needed.

Glucagon

One case series (LOE 5)[10] documented improvement in heart rate, symptoms, and signs associated with bradycardia when IV glucagon (3 mg initially, followed by infusion at 3 mg/h if necessary) was given to in-hospital patients with drug-induced (eg, β-blocker or calcium channel blocker overdose) symptomatic bradycardia not responding to atropine.

Tachycardia

This section summarizes the management of a wide variety of tachyarrhythmias. Following the overview of tachyarrhythmias and summary of the initial evaluation and treatment of tachycardia, common antiarrhythmic drugs used in the treatment of tachycardia are presented.

Classification of Tachyarrhythmias

The tachycardias can be classified in several ways based on the appearance of the QRS complex. Professionals at the ACLS level should be able to recognize and differentiate between sinus tachycardia, narrow-complex supraventricular tachycardia (SVT), and wide-complex tachycardia. Because ACLS providers may be unable to distinguish between supraventricular and ventricular rhythms, they should be aware that most wide-complex (broad-complex) tachycardias are *ventricular* in origin.

- Narrow–QRS-complex (SVT) tachycardias (QRS <0.12 second) in order of frequency
  — Sinus tachycardia
  — Atrial fibrillation
  — Atrial flutter
  — AV nodal reentry
  — Accessory pathway–mediated tachycardia
  — Atrial tachycardia (ectopic and reentrant)
  — Multifocal atrial tachycardia (MAT)
  — Junctional tachycardia

- Wide–QRS-complex tachycardias (QRS ≥0.12 second)
  — Ventricular tachycardia (VT)
  — SVT with aberrancy
  — Pre-excited tachycardias (*advanced* recognition rhythms using an accessory pathway)

Irregular narrow-complex tachycardias are probably atrial fibrillation or possibly atrial flutter or MAT. The management of atrial fibrillation and flutter is discussed in the section "Irregular Tachycardias," below.

Initial Evaluation and Treatment of Tachyarrhythmias

The evaluation and management of tachyarrhythmias is depicted in the ACLS Tachycardia Algorithm (Figure 2). Box numbers in the text refer to numbered boxes in this algorithm. Note that the "screened" boxes (boxes with text that is noticeably lighter, ie, Boxes 9, 10, 11, 13, and 14) indicate therapies that are intended for in-hospital use or with expert consultation available.

This algorithm summarizes the management of the tachycardic patient with pulses (Box 1). If pulseless arrest develops at any time, see the ACLS Pulseless Arrest Algorithm in Part 7.2: "Management of Cardiac Arrest."
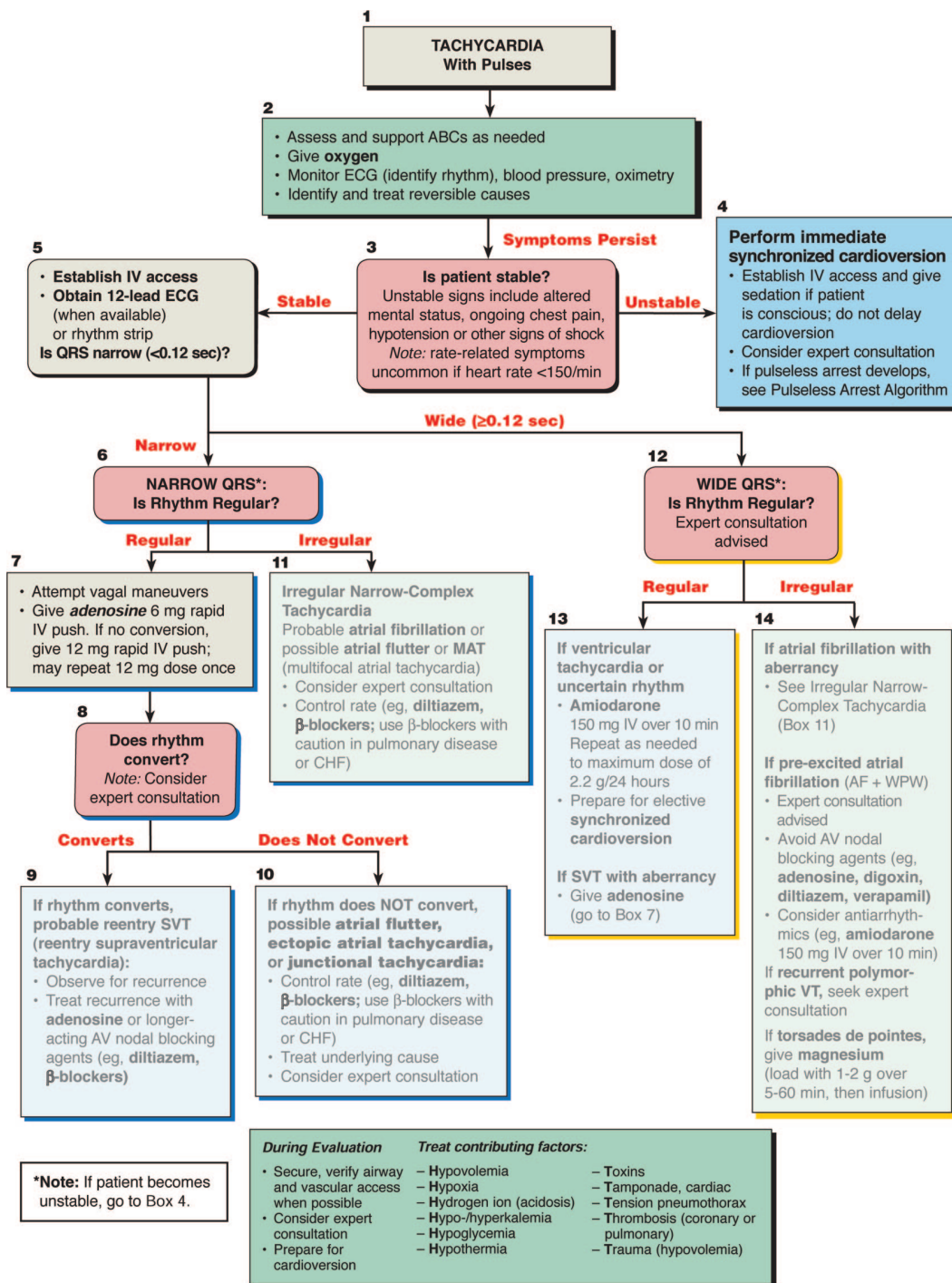
1
TACHYCARDIA
With Pulses

2
- Assess and support ABCs as needed
- Give **oxygen**
- Monitor ECG (identify rhythm), blood pressure, oximetry
- Identify and treat reversible causes

Symptoms Persist

4
Perform immediate synchronized cardioversion
- Establish IV access and give sedation if patient is conscious; do not delay cardioversion
- Consider expert consultation
- If pulseless arrest develops, see Pulseless Arrest Algorithm

3
Is patient stable?
Unstable signs include altered mental status, ongoing chest pain, hypotension or other signs of shock
Note: rate-related symptoms uncommon if heart rate <150/min

Stable   Unstable

5
- **Establish IV access**
- **Obtain 12-lead ECG** (when available) or rhythm strip
Is QRS narrow (<0.12 sec)?

Narrow   Wide (≥0.12 sec)

6
NARROW QRS*:
Is Rhythm Regular?

Regular   Irregular

12
WIDE QRS*:
Is Rhythm Regular?
Expert consultation advised

Regular   Irregular

7
- Attempt vagal maneuvers
- Give *adenosine* 6 mg rapid IV push. If no conversion, give 12 mg rapid IV push; may repeat 12 mg dose once

11
Irregular Narrow-Complex Tachycardia
Probable **atrial fibrillation** or possible **atrial flutter** or MAT (multifocal atrial tachycardia)
- Consider expert consultation
- Control rate (eg, **diltiazem, β-blockers;** use β-blockers with caution in pulmonary disease or CHF)

13
If **ventricular tachycardia or uncertain rhythm**
- Amiodarone
  150 mg IV over 10 min
  Repeat as needed to maximum dose of 2.2 g/24 hours
- Prepare for elective **synchronized cardioversion**

If **SVT with aberrancy**
- Give **adenosine** (go to Box 7)

14
If **atrial fibrillation with aberrancy**
- See Irregular Narrow-Complex Tachycardia (Box 11)

If **pre-excited atrial fibrillation** (AF + WPW)
- Expert consultation advised
- Avoid AV nodal blocking agents (eg, **adenosine, digoxin, diltiazem, verapamil**)
- Consider antiarrhythmics (eg, **amiodarone** 150 mg IV over 10 min)

If **recurrent polymorphic VT**, seek expert consultation

If **torsades de pointes**, give **magnesium** (load with 1-2 g over 5-60 min, then infusion)

8
Does rhythm convert?
Note: Consider expert consultation

Converts   Does Not Convert

9
If rhythm converts, probable reentry SVT (reentry supraventricular tachycardia):
- Observe for recurrence
- Treat recurrence with **adenosine** or longer-acting AV nodal blocking agents (eg, **diltiazem, β-blockers**)

10
If rhythm does NOT convert, possible **atrial flutter, ectopic atrial tachycardia,** or **junctional tachycardia:**
- Control rate (eg, **diltiazem, β-blockers;** use β-blockers with caution in pulmonary disease or CHF)
- Treat underlying cause
- Consider expert consultation

*Note: If patient becomes unstable, go to Box 4.

During Evaluation
- Secure, verify airway and vascular access when possible
- Consider expert consultation
- Prepare for cardioversion

Treat contributing factors:
- **Hypo**volemia
- **Hypo**xia
- **H**ydrogen ion (acidosis)
- **Hypo-/hyperkalemia**
- **Hypo**glycemia
- **Hypo**thermia
- **Toxins**
- **Tamponade, cardiac**
- **Tension** pneumothorax
- **Thrombosis** (coronary or pulmonary)
- **Trauma** (hypovolemia)

Figure 2. ACLS Tachycardia Algorithm.

The provider must assess the patient while supporting the airway and breathing, administering oxygen (Box 2), obtaining an ECG to identify the rhythm, and monitoring blood pressure and oxyhemoglobin saturation. The provider should establish IV access when possible and identify and treat reversible causes of the tachycardia.

If signs and symptoms persist despite provision of supplementary oxygen and support of airway and ventilation, the provider should determine if the patient is unstable and if signs of cardiovascular compromise are related to the tachycardia (Box 3). If the patient demonstrates rate-related cardiovascular compromise, with signs and symptoms such as altered mental status, ongoing chest pain, hypotension, or other signs of shock, provide immediate synchronized cardioversion (Box 4—see below). Serious signs and symptoms are uncommon if the ventricular rate is <150 beats per minute in patients with a healthy heart. Patients with impaired cardiac function or significant comorbid conditions may become symptomatic at lower heart rates. If the patient is unstable with narrow-complex reentry SVT, you may admin-

ister adenosine while preparations are made for synchronized cardioversion (Class IIb), but do not delay cardioversion to administer the drug or to establish IV access.

If the patient with tachycardia is stable (ie, no serious signs or symptoms related to the tachycardia), the provider has time to obtain a 12-lead ECG and evaluate the rhythm (Box 5) and determine treatment options. Stable patients may await expert consultation because treatment has the potential for harm.

Synchronized Cardioversion and Unsynchronized Shocks (Box 4)

Synchronized cardioversion is shock delivery that is timed (synchronized) with the QRS complex. This synchronization avoids shock delivery during the relative refractory period of the cardiac cycle (some call it the "vulnerable period"), when a shock could produce VF.[11] The energy (shock dose) used for synchronized cardioversion is lower than the doses used for unsynchronized shocks (ie, doses for attempted defibrillation). Low-energy shocks should always be delivered as *synchronized* shocks because delivery of low energy unsynchronized shocks is likely to induce VF. If cardioversion is needed and it is impossible to synchronize a shock (eg, the patient's rhythm is irregular), use high-energy unsynchronized shocks (defibrillation doses).

Synchronized cardioversion is recommended to treat (1) unstable SVT due to reentry, (2) unstable atrial fibrillation, and (3) unstable atrial flutter. These arrhythmias are caused by reentry, an abnormal rhythm circuit that allows a wave of depolarization to travel in a circle. Delivery of a shock can stop these rhythms because it interrupts the circulating (reentry) pattern. Synchronized cardioversion is also recommended to treat unstable monomorphic (regular) VT.

If possible, establish IV access before cardioversion and administer sedation if the patient is conscious. But do not delay cardioversion. Consider expert consultation. For further information about defibrillation and cardioversion, see Part 5: "Electrical Therapies."

The recommended initial dose for cardioversion of atrial fibrillation is 100 J to 200 J with a monophasic waveform. A dose of 100 J to 120 J is reasonable with a biphasic waveform. Escalate the second and subsequent shock doses as needed.

Cardioversion of atrial flutter and other SVTs generally requires less energy. An initial energy of 50 J to 100 J monophasic damped sine (MDS) waveform is often sufficient. If the initial 50-J shock fails, increase the dose in a stepwise fashion.[12] More data is needed before detailed comparative dosing recommendations for cardioversion with biphasic waveforms can be made.

Cardioversion is not likely to be effective for treatment of junctional tachycardia or ectopic or multifocal atrial tachycardia because these rhythms have an automatic focus, arising from cells that are spontaneously depolarizing at a rapid rate. Delivery of a shock generally cannot stop these rhythms. In fact, shock delivery to a heart with a rapid automatic focus may increase the rate of the tachyarrhythmia.

The amount of energy required for cardioversion of VT is determined by the morphologic characteristics and the rate of the VT.[13] If the patient with monomorphic VT (regular form

and rate) is unstable but has a pulse, treat with synchronized cardioversion. To treat monomorphic VT using a monophasic waveform, provide an initial shock of 100 J. If there is no response to the first shock, increase the dose in a stepwise fashion (eg, 100 J, 200 J, 300 J, 360 J). These recommendations are consistent with the recommendations in the *ECC Guidelines 2000*.[2] There is insufficient data to recommend specific biphasic energy doses for treatment of VT.

If a patient has polymorphic VT and is unstable, treat the rhythm as VF and deliver high-energy *unsynchronized* shocks (ie, defibrillation doses). Although synchronized cardioversion is preferred for treatment of an organized ventricular rhythm, for some irregular rhythms, such as polymorphic VT, synchronization is not possible. If there is any doubt whether monomorphic or polymorphic VT is present in the *unstable* patient, do not delay shock delivery to perform detailed rhythm analysis—provide high-energy unsynchronized shocks (ie, defibrillation doses). Use the ACLS Pulseless Arrest Algorithm (see Part 7.2: "Management of Cardiac Arrest").

Regular Narrow-Complex Tachycardia (Boxes 7, 8, 9, 10)

Sinus Tachycardia

Sinus tachycardia is common and usually results from a physiologic stimulus, such as fever, anemia, or shock. Sinus tachycardia occurs when the sinus node discharge rate is >100 times per minute in response to a variety of stimuli or sympathomimetic agents. No specific drug treatment is required. Therapy is directed toward identification and treatment of the underlying cause. When cardiac function is poor, cardiac output can be dependent on a rapid heart rate. In such compensatory tachycardias, stroke volume is limited, so "normalizing" the heart rate can be detrimental.

Supraventricular Tachycardia (Reentry SVT)

Evaluation

Reentry SVT is a regular tachycardia that is caused by reentry, an abnormal rhythm circuit that allows a wave of depolarization to travel in a circle. The often abrupt onset and termination of this tachyarrhythmia led to its original name, paroxysmal supraventricular tachycardia (PSVT). The rate of reentry SVT exceeds the typical upper limits of sinus tachycardia at rest (>120 beats per minute) with or without discernible P waves. The rhythm is considered to be of supraventricular origin if the QRS complex is narrow (<120 milliseconds or <0.12 second) or if the QRS complex is wide (broad) and bundle branch aberrancy is *known* to be present. Reentry SVT may include AV nodal reentrant tachycardia or AV reentry tachycardia.

Therapy

Vagal Maneuvers. Vagal maneuvers and adenosine are the preferred initial therapeutic choices for the termination of stable reentry SVT (Box 7). Vagal maneuvers alone (Valsalva maneuver or carotid sinus massage) will terminate about 20% to 25% of reentry SVT[14]; adenosine treatment is required for the remainder. In 1 study (LOE 4)[15] of stable reentry SVT in younger patients, vagal maneuvers were often unsuccessful.

Adenosine. If reentry SVT does not respond to vagal maneuvers, give 6 mg of IV adenosine as a rapid IV push (Class I). Give adenosine rapidly over 1 to 3 seconds through a large (eg, antecubital) vein followed by a 20-mL saline flush and elevation of the arm. If the rate does not convert within 1 to 2 minutes, give a 12-mg bolus. Give a second 12-mg bolus if the rate fails to convert within 1 to 2 minutes after the first 12-mg bolus.

Five prospective controlled nonrandomized cohort studies (LOE 2[16]; LOE 3[17–20]) showed that adenosine is safe and effective in converting SVT in both the in-hospital and out-of-hospital settings. Although 2 randomized clinical trials (LOE 3)[17,21] documented a similar SVT conversion rate between adenosine and calcium channel blockers, adenosine was more rapid with fewer severe side effects than verapamil. Amiodarone can achieve nearly 100% efficacy in the inhibition of induced sustained reentrant SVT (LOE 6).[22]

Adenosine is safe and effective in pregnancy.[23] Adenosine, however, does have several important drug interactions. Larger doses may be required for patients with a significant blood level of theophylline, caffeine, or theobromine. The initial dose should be reduced to 3 mg in patients taking dipyridamole or carbamazepine, those with transplanted hearts, or if given by central venous access. Side effects with adenosine are common but transient; flushing, dyspnea, and chest pain are the most frequently observed.[24]

If the rhythm does convert (Box 9), it was probably reentry SVT. Monitor the patient for recurrence and treat any recurrence with adenosine or control the rate with a longer-acting AV nodal blocking agent (eg, diltiazem or β-blocker).

Calcium Channel Blockers and β-Blockers. If adenosine fails to convert reentry SVT (Box 10), attempt rate control with a nondihydropyridine calcium channel blocker (ie, verapamil or diltiazem) or β-blocker as a second-line agent (Class IIa).[25–27] These drugs act primarily on nodal tissue either to slow the ventricular response to atrial arrhythmias by blocking conduction through the AV node or to terminate the reentry SVT that depends on conduction through the AV node.

Verapamil and, to a lesser extent, diltiazem may decrease myocardial contractility and critically reduce cardiac output in patients with severe left ventricular dysfunction. Calcium channel blockers that affect the AV node (including verapamil and diltiazem) are considered harmful when given to patients with atrial fibrillation or atrial flutter associated with known pre-excitation (Wolff-Parkinson-White [WPW]) syndrome. β-Blockers should be used with caution in patients with pulmonary disease or congestive heart failure.

For verapamil, give a 2.5 to 5 mg IV bolus over 2 minutes (over 3 minutes in older patients). If there is no therapeutic response and no drug-induced adverse event, repeated doses of 5 to 10 mg may be administered every 15 to 30 minutes to a total dose of 20 mg. An alternative dosing regimen is to give a 5-mg bolus every 15 minutes to a total dose of 30 mg. Verapamil should be given *only* to patients with narrow-complex reentry SVT or arrhythmias known with certainty to be of supraventricular origin. It should not be given to patients with impaired ventricular function or heart failure.

For diltiazem, give a dose of 15 to 20 mg (0.25 mg/kg) IV over 2 minutes; if needed, in 15 minutes give an IV dose of 20 to 25 mg (0.35 mg/kg). The maintenance infusion dose is 5 to 15 mg/h, titrated to heart rate.

A wide variety of β-blockers may be given for treatment of supraventricular tachyarrhythmias. More detailed information is provided below. Side effects of β-blockers can include bradycardias, AV conduction delays, and hypotension.

Wide- (Broad-) Complex Tachycardia (Boxes 12, 13, 14)

Evaluation

The first step in the management of any tachycardia is to determine if the patient's condition is stable or unstable (Box 3). An unstable patient with wide-complex tachycardia is presumed to have VT, and immediate cardioversion is performed (Box 4 and see above).

If the patient is stable, the second step in management is to obtain a 12-lead ECG (Box 5) to evaluate the QRS duration (ie, narrow or wide). At this point the provider should consider the need to obtain expert consultation. If the patient becomes unstable at any time, proceed with synchronized cardioversion. If the patient develops pulseless arrest or is unstable with polymorphic VT, treat as VF and deliver high-energy unsynchronized shocks (ie, defibrillation doses).

Wide-complex tachycardias are defined as those with a QRS ≥ 0.12 second. The most common forms of wide-complex tachycardia are

- VT
- SVT with aberrancy
- Pre-excited tachycardias (associated with or mediated by an accessory pathway)

The third step in management of a tachycardia is to determine if the rhythm is regular or irregular (Box 12). A *regular* wide-complex tachycardia is likely to be VT or SVT with aberrancy. An irregular wide-complex tachycardia may be atrial fibrillation with aberrancy, pre-excited atrial fibrillation (ie, atrial fibrillation with WPW syndrome), or polymorphic VT. Polymorphic VT may represent torsades de pointes (see below). Providers should consider the need for expert consultation when treating wide-complex tachycardias.

Therapy for Regular Wide-Complex Tachycardias (Box 13)

If the wide-complex regular tachycardia is thought to be SVT, adenosine is recommended. The dose used (6 mg rapid IV push; providers may follow the first dose with a 12-mg bolus and a second 12-mg bolus if the rate fails to convert) is the same as that for reentry SVT (see above for more information).

Synchronized cardioversion is appropriate for treatment of monomorphic (regular) wide-complex tachycardia, particularly if the patient is symptomatic (eg, signs of altered level of consciousness). If the rhythm is identified as likely VT in a stable patient, IV antiarrhythmic drugs may be effective. If antiarrhythmics are administered, we recommend amiodarone (Class IIa). Give 150 mg IV over 10 minutes; repeat as needed to a maximum dose of 2.2 g IV per 24 hours. Alternative drugs for wide-complex regular tachycardias are procainamide and sotalol (see below).

Evidence in support of amiodarone comes from 3 observational studies (LOE 5)[28–30] that indicate that amiodarone is effective for the termination of shock-resistant or drug-refractory VT. One randomized parallel study (LOE 2)[31] indicated that aqueous amiodarone is more effective than lidocaine in the treatment of shock-resistant VT. Amiodarone administration is also supported by extrapolated evidence (LOE 7) from studies of out-of-hospital cardiac arrest with shock-refractory VF/VT, which showed that amiodarone improved survival to hospital admission (but not discharge) compared with placebo[32] or lidocaine.[33]

Irregular Tachycardias

Atrial Fibrillation and Flutter

Evaluation
An irregular narrow-complex or wide-complex tachycardia is most likely atrial fibrillation with an uncontrolled ventricular response. Other diagnostic possibilities include MAT. We recommend a 12-lead ECG and expert consultation if the patient is stable.

Therapy
Management (Box 11) should focus on control of the rapid ventricular rate (rate control) and conversion of hemodynamically unstable atrial fibrillation to sinus rhythm (rhythm control). Patients with atrial fibrillation for >48 hours are at increased risk for cardioembolic events and must first undergo anticoagulation before rhythm control. Electric or pharmacologic cardioversion (conversion to normal sinus rhythm) should *not be attempted* in these patients unless the patient is unstable or the absence of a left atrial thrombus is documented by transesophageal echocardiography.

Magnesium (LOE 3),[34] diltiazem (LOE 2),[35] and β-blockers (LOE 2)[36,37] have been shown to be effective for rate control in the treatment of atrial fibrillation with a rapid ventricular response in both the prehospital (LOE 3)[38] and hospital settings.

Ibutilide and amiodarone (LOE 2)[39–41] have been shown to be effective for rhythm control in the treatment of atrial fibrillation in the hospital setting.

In summary, we recommend expert consultation and initial *rate* control with diltiazem, β-blockers, or magnesium for patients with atrial fibrillation and a rapid ventricular response. Amiodarone, ibutilide, propafenone, flecainide, digoxin, clonidine, or magnesium can be considered for *rhythm* control in patients with atrial fibrillation of ≤48 hours duration.

If a pre-excitation syndrome was identified before the onset of atrial fibrillation (ie, a delta wave, characteristic of WPW, was visible during normal sinus rhythm), expert consultation is advised. Do not administer AV nodal blocking agents such as adenosine, calcium channel blockers, digoxin, and possibly β-blockers to patients with pre-excitation atrial fibrillation or atrial flutter (Box 14) because these drugs can cause a paradoxical increase in the ventricular response to the rapid atrial impulses of atrial fibrillation.

Polymorphic (Irregular) VT (Box 14)
Polymorphic (irregular) VT requires immediate treatment because it is likely to deteriorate to pulseless arrest. Providers

should consider consultation with an expert in arrhythmia management.

Pharmacologic treatment of recurrent polymorphic VT is determined by the presence or absence of a long QT during sinus rhythm. If a long QT interval is observed during sinus rhythm (ie, the VT is torsades de pointes), the first step is to stop medications known to prolong the QT interval. Correct electrolyte imbalance and other acute precipitants (eg, drug overdose or poisoning—see Part 10.2: "Toxicology in ECC").

Although magnesium is commonly used to treat torsades de pointes VT (polymorphic VT associated with long QT interval), it is supported by only 2 observational studies (LOE 5)[42,43] showing effectiveness in patients with prolonged QT interval. One adult case series (LOE 5)[44] showed that isoproterenol or ventricular pacing can be effective in terminating torsades de pointes associated with bradycardia and drug-induced QT prolongation. Magnesium is unlikely to be effective in terminating polymorphic VT in patients with a normal QT interval (LOE 5),[43] but amiodarone may be effective (LOE 4).[45]

If the patient with polymorphic VT is or becomes unstable (ie, demonstrates altered level of consciousness, hypotension, or other signs of shock, such as severe pulmonary edema), provide high-energy (ie, defibrillation dose) *unsynchronized* shocks. Although synchronized cardioversion is always preferred for an organized ventricular rhythm, synchronization is not possible for some arrhythmias. The many QRS configurations and irregular rates present in polymorphic VT make it difficult or impossible to reliably synchronize to a QRS complex. A good rule of thumb is that if your eye cannot synchronize to each QRS complex, neither can the defibrillator/cardioverter.

If there is any doubt whether monomorphic or polymorphic VT is present in the *unstable* patient, do not delay shock delivery for detailed rhythm analysis—provide high-energy unsynchronized shocks (ie, defibrillation doses). Current research confirms that it is reasonable to use selected energies of 150 J to 200 J with a biphasic truncated exponential waveform or 120 J with a rectilinear biphasic waveform for the initial shock. For second and subsequent biphasic shocks use the same or higher energy (Class IIa). Providers should use the biphasic device-specific dose; the default dose is 200 J. If a monophasic defibrillator is used, use a dose of 360 J for all unsynchronized shocks (for further information see Part 5: "Electrical Therapies: Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing"). Lower energy levels should not be used for these unsynchronized shocks because low-energy shocks have a high likelihood of provoking VF when they are given in an unsynchronized mode.

After shock delivery the healthcare provider should be prepared to provide immediate CPR (beginning with chest compressions) and follow the ACLS Pulseless Arrest Algorithm if pulseless arrest develops. For further information see Part 7.2: "Management of Cardiac Arrest."

Antiarrhythmic Drugs

Adenosine
Adenosine is an endogenous purine nucleoside that briefly depresses AV node and sinus node activity. Adenosine is recommended for the following indications:

- For defined, stable, narrow-complex AV nodal or sinus nodal reentry tachycardias.[16–21] The most frequent example of these is reentry SVT (Class I). Adenosine will not terminate arrhythmias such as atrial flutter, atrial fibrillation, or atrial or ventricular tachycardias, because these arrhythmias are not due to reentry involving the AV or sinus node. Adenosine will not terminate the arrhythmia but may produce transient AV or retrograde (ventriculoatrial) block clarifying the underlying rhythm.
- For unstable reentry SVT while preparations are made for cardioversion (Class IIb).
- For undefined, stable, narrow-complex SVT as a combination therapeutic and diagnostic maneuver.
- For stable, wide-complex tachycardias in patients with a recurrence of a known reentry pathway that has been previously defined.

Amiodarone IV

IV amiodarone is a complex drug with effects on sodium, potassium, and calcium channels as well as α- and β-adrenergic blocking properties. Amiodarone is recommended for tachyarrhythmias, with the following indications:

- For narrow-complex tachycardias that originated from a reentry mechanism (reentry SVT) if the rhythm remains uncontrolled by adenosine, vagal maneuvers, and AV nodal blockade in patients with preserved or impaired ventricular function (Class IIb)[22]
- Control of hemodynamically stable VT, polymorphic VT with a normal QT interval, and wide-complex tachycardia of uncertain origin (Class IIb)[28–31]
- To control rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias (Class IIb)[22]

Administer 150 mg of IV amiodarone over 10 minutes, followed by a 1 mg/min infusion for 6 hours and then a 0.5 mg/min maintenance infusion over 18 hours. Supplementary infusions of 150 mg can be repeated every 10 minutes as necessary for recurrent or resistant arrhythmias to a maximum manufacturer-recommended total daily IV dose of 2.2 g. One study found amiodarone to be effective in patients with atrial fibrillation when administered at relatively high doses of 125 mg/h for 24 hours (total dose 3 g).[41] In patients known to have severely impaired heart function, IV amiodarone is preferable to other antiarrhythmic agents for atrial and ventricular arrhythmias.

The major adverse effects of amiodarone are hypotension and bradycardia, which can be prevented by slowing the rate of drug infusion.

Calcium Channel Blockers: Verapamil and Diltiazem

Verapamil and diltiazem are nondihydropyridine calcium channel blocking agents that slow conduction and increase refractoriness in the AV node. These actions may terminate reentrant arrhythmias and control ventricular response rate in patients with a variety of atrial tachycardias. These medications are indicated in the following circumstances:

- For stable, narrow-complex, reentry mechanism tachycardias (reentry SVT) if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers (Class IIa)[25–27]
- For stable, narrow-complex, automaticity mechanism tachycardias (junctional, ectopic, multifocal) if the rhythm is not controlled or converted by adenosine or vagal maneuvers
- To control rate of ventricular response in patients with atrial fibrillation or atrial flutter (Class IIa)[35,38]

IV verapamil is effective for terminating narrow-complex reentry SVT, and it may also be used for rate control in atrial fibrillation. The initial dose of verapamil is 2.5 to 5 mg IV given over 2 minutes. In the absence of a therapeutic response or a drug-induced adverse event, repeat doses of 5 to 10 mg may be administered every 15 to 30 minutes to a total dose of 20 mg. An alternative dosing regimen is to give a 5-mg bolus every 15 minutes to a total dose of 30 mg. Verapamil should be given *only* to patients with narrow-complex reentry SVT or arrhythmias known with certainty to be of supraventricular origin. It should not be given to patients with impaired ventricular function or heart failure.

Diltiazem at a dose of 0.25 mg/kg, followed by a second dose of 0.35 mg/kg, seems to be equivalent in efficacy to verapamil.[25–27] Verapamil and, to a lesser extent, diltiazem may decrease myocardial contractility and critically reduce cardiac output in patients with severe left ventricular dysfunction. Calcium channel blockers that affect the AV node (eg, verapamil and diltiazem) are considered harmful when given to patients with atrial fibrillation or atrial flutter associated with known pre-excitation (WPW) syndrome.

β-Adrenergic Blockers

β-Blocking agents (atenolol, metoprolol, labetalol, propranolol, esmolol) reduce the effects of circulating catecholamines and decrease heart rate and blood pressure. They also have various cardioprotective effects for patients with acute coronary syndromes. For acute tachyarrhythmias, these agents are indicated for rate control in the following situations:

- For narrow-complex tachycardias that originate from either a *reentry mechanism* (reentry SVT) or an *automatic focus* (junctional, ectopic, or multifocal tachycardia) uncontrolled by vagal maneuvers and adenosine in the patient with preserved ventricular function (Class IIa)
- To control rate in atrial fibrillation and atrial flutter in the patient with preserved ventricular function[36,37]

The recommended dose of atenolol (β_1) is 5 mg *slow* IV (over 5 minutes). If the arrhythmia persists 10 minutes after that dose and the first dose was well tolerated, give a second dose of 5 mg slow IV (over 5 minutes).

Metoprolol (β_1) is given in doses of 5 mg by slow IV/IO push at 5-minute intervals to a total of 15 mg.

An alternative agent is propranolol (β_1 and β_2 effects) 0.1 mg/kg by slow IV push divided into 3 equal doses at 2- to 3-minute intervals. The rate of administration should not

exceed 1 mg/min. May repeat total dose in 2 minutes if necessary.

IV esmolol is a short-acting (half-life 2 to 9 minutes) β_1-selective β-blocker that is administered in an IV loading dose of 500 μg/kg (0.5 mg/kg) over 1 minute, followed by a 4-minute infusion of 50 μg/kg per minute (0.05 mg/kg per minute) for a total of 200 μg/kg. If the response is inadequate, a second bolus of 0.5 mg/kg is infused over 1 minute, with an increase of the maintenance infusion to 100 μg/kg (0.1 mg/kg) per minute (maximum infusion rate: 300 μg/kg [0.3 mg/kg] per minute).

Side effects related to β-blockade include bradycardias, AV conduction delays, and hypotension. Cardiovascular decompensation and cardiogenic shock after β-adrenergic blocker therapy are infrequent complications. Contraindications to the use of β-adrenergic blocking agents include second-degree or third-degree heart block, hypotension, severe congestive heart failure, and lung disease associated with bronchospasm. These agents may be harmful for patients with atrial fibrillation or atrial flutter associated with known pre-excitation (WPW) syndrome.

Ibutilide
Ibutilide is a short-acting antiarrhythmic that acts by prolonging the action potential duration and increasing the refractory period of cardiac tissue. This agent may be used in the following circumstances:

- For acute pharmacologic rhythm conversion of atrial fibrillation or atrial flutter in patients with normal cardiac function when duration of the arrhythmia is ≤48 hours (Class IIb).[39]
- To control rate in atrial fibrillation or atrial flutter in patients with preserved ventricular function when calcium channel blockers or β-blockers are ineffective.
- For acute pharmacologic rhythm conversion of atrial fibrillation or atrial flutter in patients with WPW syndrome and preserved ventricular function when the duration of the arrhythmia is ≤48 hours. But the intervention of choice for this indication is DC cardioversion.

Ibutilide seems most effective for the pharmacologic conversion of atrial fibrillation or atrial flutter of relatively brief duration. For adults weighing ≥60 kg, ibutilide is administered intravenously, diluted or undiluted, as 1 mg (10 mL) over 10 minutes. If the first dose is unsuccessful in terminating the arrhythmia, a second 1-mg dose can be administered at the same rate 10 minutes after the first. In patients weighing <60 kg, an initial dose of 0.01 mg/kg is recommended.

Ibutilide has minimal effects on blood pressure and heart rate. Its major limitation is a relatively high incidence of ventricular arrhythmias (polymorphic VT, including torsades de pointes). Correct hyperkalemia or low magnesium before administration. Monitor patients receiving ibutilide *continuously* for arrhythmias at the time of its administration and for *at least 4 to 6 hours* thereafter. Ibutilide is contraindicated in baseline QT$_C$ (QT interval corrected for heart rate) of >440 msec.

Lidocaine
Lidocaine is one of a number of antiarrhythmic drugs available for treatment of ventricular ectopy, VT, and VF. At this time there is good evidence that alternative agents are superior to lidocaine in terminating VT.[46] Lidocaine may be considered in the following conditions (although it is not considered the drug of choice):

- For *stable monomorphic VT* in patients with preserved ventricular function (Class Indeterminate). Alternative agents are preferred.
- For *polymorphic VT* with *normal baseline QT interval* when ischemia is treated and electrolyte imbalance is corrected.
- If ventricular function is preserved: lidocaine may be administered.
- If ventricular function is impaired: use amiodarone as an antiarrhythmic agent. If unsuccessful, perform DC cardioversion.
- Lidocaine can be used for polymorphic VT with a prolonged baseline QT interval that suggests torsades de pointes.

Initial doses ranging from 0.5 to 0.75 mg/kg and up to 1 to 1.5 mg/kg may be used. Repeat 0.5 to 0.75 mg/kg every 5 to 10 minutes to a maximum total dose of 3 mg/kg. A maintenance infusion of 1 to 4 mg/min (30 to 50 μg/kg per minute) is acceptable.

Toxic reactions and side effects include slurred speech, altered consciousness, muscle twitching, seizures, and bradycardia.

Magnesium
Magnesium is recommended for the treatment of torsades de pointes VT with or without cardiac arrest, but it has not been shown to be helpful for treatment of non-torsades pulseless arrest. Low-level evidence suggests that magnesium is effective for rate control (LOE 3)[34] in patients with atrial fibrillation with a rapid ventricular response (LOE 2),[40] so it may be considered for this arrhythmia.

Give magnesium sulfate in a dose of 1 to 2 g diluted in D$_5$W over 5 to 60 minutes. Slower rates are preferable in the stable patient. A more rapid infusion may be used for the unstable patient.

Procainamide
Procainamide hydrochloride suppresses both atrial and ventricular arrhythmias by slowing conduction in myocardial tissue. One randomized trial (LOE 2)[47] indicated that procainamide is superior to lidocaine in terminating spontaneously occurring VT. Procainamide may be considered in the following situations:

- As one of several drugs that may be used for treatment of stable monomorphic VT in patients with preserved ventricular function (Class IIa)[46]
- One of several equivalent drugs that can be used for control of heart rate in atrial fibrillation or atrial flutter in patients with preserved ventricular function
- One of several drugs that can be used for acute control of heart rhythm in atrial fibrillation or atrial flutter in patients

with known pre-excitation (WPW) syndrome and pre-served ventricular function

- One of several drugs that can be used for AV reentrant, narrow-complex tachycardias such as reentry SVT if rhythm is uncontrolled by adenosine and vagal maneuvers in patients with preserved ventricular function

Procainamide hydrochloride for non-VF/VT arrest may be given in an infusion of 20 mg/min until the arrhythmia is suppressed, hypotension ensues, the QRS complex is prolonged by 50% from its original duration, or a total of 17 mg/kg (1.2 g for a 70-kg patient) of the drug has been given. Bolus administration of the drug can result in toxic concentrations and significant hypotension. The maintenance infusion rate of procainamide hydrochloride is 1 to 4 mg/min, diluted in D_5W or normal saline. This should be reduced in the presence of renal failure.

Procainamide should be used cautiously in patients with preexisting QT prolongation. In general it should be used with caution if at all in combination with other drugs that prolong the QT interval (consider obtaining expert consultation). Monitor the ECG and blood pressure continuously during administration of procainamide.

Sotalol

Sotalol is not a first-line antiarrhythmic. Sotalol hydrochloride is an antiarrhythmic agent that, like amiodarone, prolongs action potential duration and increases cardiac tissue refractoriness. It also has nonselective β-blocking properties. One randomized controlled trial (LOE 1)[48] indicated that sotalol is significantly more effective than lidocaine for terminating acute sustained VT. This agent may be used in the following circumstances with expert consultation:

- To control rhythm in atrial fibrillation or atrial flutter in patients with pre-excitation (WPW) syndrome and preserved ventricular function when the duration of the arrhythmia is ≤48 hours. But the intervention of choice for this indication is DC cardioversion.
- For monomorphic VT.

IV sotalol is usually administered at a dose of 1 to 1.5 mg/kg body weight, then infused at a rate of 10 mg/min. Side effects include bradycardia, hypotension, and arrhythmia. The incidence of torsades de pointes following a single dose of sotalol for treatment of VT is reportedly 0.1%.[45] Use of IV sotalol is limited by the need to infuse it relatively slowly.

Summary

The goal of therapy for bradycardia or tachycardia is to rapidly identify and treat patients who are hemodynamically unstable. Pacing or drugs, or both, may be used to control symptomatic bradycardia. Cardioversion or drugs, or both, may be used to control symptomatic tachycardia. ALS providers should closely monitor stable patients pending expert consultation and should be prepared to aggressively treat those who develop decompensation.

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