Bradycardia

Overview

Bradycardia is generally defined as any rhythm disorder with a heart rate less than 60/min but for assessment and management of a patient with symptomatic bradycardia, it is typically defined as having a heart rate less than 50/min.

Managing bradycardia requires

- Differentiating between signs and symptoms caused by the slow rate vs those that are unrelated
- Correctly diagnosing the presence and type of atrioventricular (AV) block
- Using atropine as the drug intervention of first choice
- Deciding when to initiate transcutaneous pacing (TCP)
- Deciding when to start epinephrine or dopamine to maintain heart rate and blood pressure
- Knowing when to seek expert consultation about complicated rhythm interpretation, drugs, or management decisions or when to consider transvenous pacing
- Knowing the techniques and cautions for using TCP

Rhythms for Bradycardia

- Sinus bradycardia
- First-degree AV block
- Second-degree AV block: block of some, but not all, atrial impulses before they reach the ventricles. This block can be further classified as Mobitz type I or Mobitz type II second-degree AV block.
    - –Mobitz type I AV block:
        - Also known as *Wenckebach phenomenon*, typically occurs at the AV node. It is characterized by successive prolongation of the PR interval until an atrial impulse is not conducted to the ventricles (Figure 26B). The P wave corresponding to that atrial impulse is not followed by a QRS complex. The cycle of progressive lengthening of the PR interval until failure of conduction of the atrial impulse to the ventricles often repeats.
    - –Mobitz type II second-degree AV block (Figure 26C):
        - Occurs below the level of the AV node. It is characterized by intermittent nonconduction of P waves (atrial impulses to the ventricle) with a constant PR interval on conducted beats. There can be a consistent ratio of atrial to ventricular depolarizations, eg, 2 P waves to 1 QRS complex.
- Third-degree AV block

You should know the major AV blocks because important treatment decisions are based on the type of block (Figure 26). Complete (or third-degree) AV block is generally the most clinically significant block because it is most likely to cause cardiovascular collapse and require immediate pacing. Recognizing a stable bradycardia due to AV block is a primary goal, and recognizing the type of AV block is secondary.

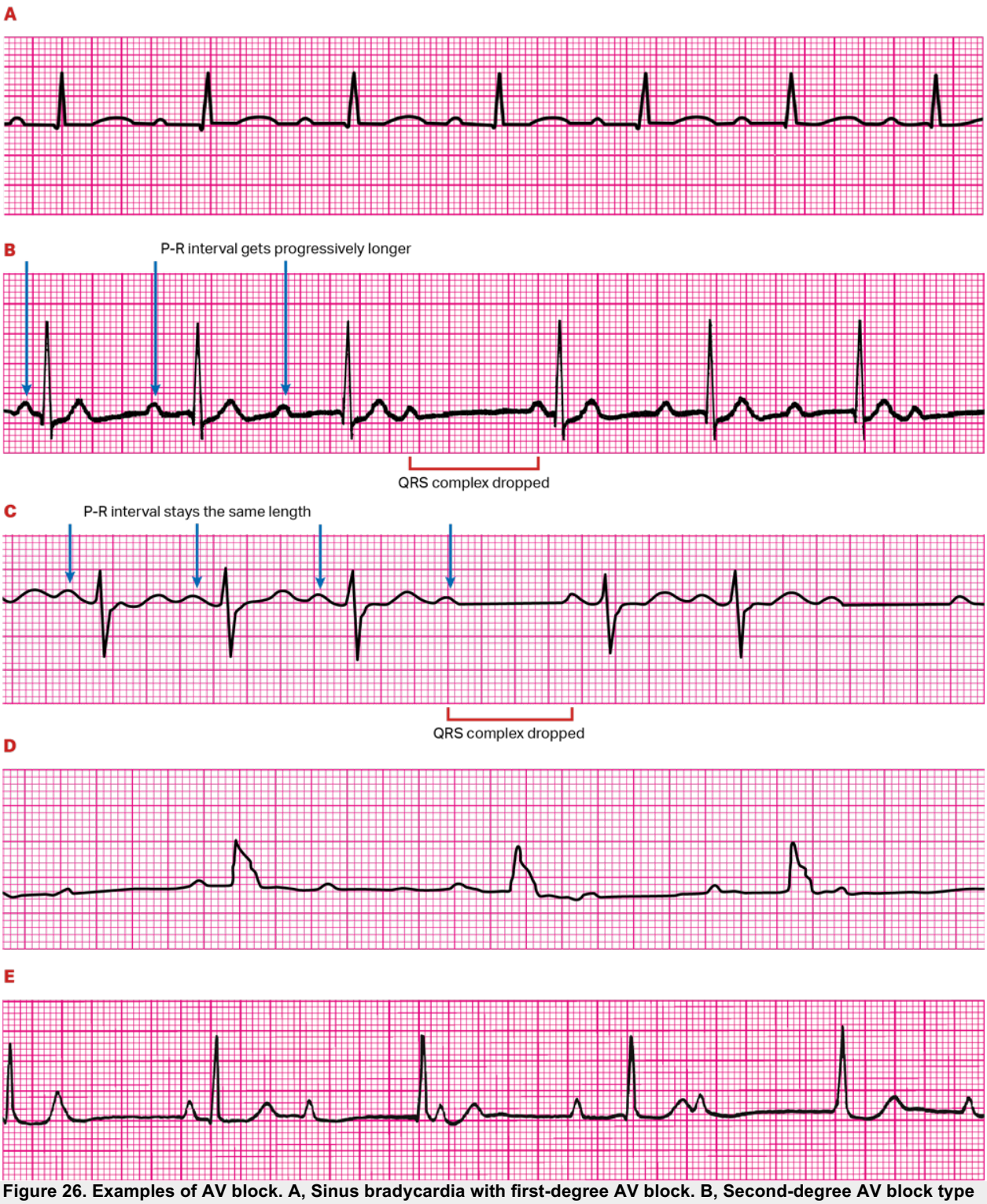

Figure 26. Examples of AV block. A, Sinus bradycardia with first-degree AV block. B, Second-degree AV block type I. C, Second-degree AV block type II. D, Complete AV block with a ventricular escape pacemaker (wide QRS: 0.12 to 0.14 second). E, Third-degree AV block with a junctional escape pacemaker (narrow QRS: less than 0.12 second).

Drugs for Bradycardia

Drugs for bradycardia include

- Atropine

- Dopamine (infusion)
- Epinephrine (infusion)

Symptomatic Bradycardia

Bradycardia may have multiple causes, including some that are physiologic and require no assessment or therapy. For example, a healthy, well-trained athlete may have a resting heart rate less than 50/min.

In contrast, some patients have heart rates in the normal range, but these rates are inappropriate or insufficient for them. This is called a *functional* or *relative bradycardia*. For example, a heart rate of 70/min may be relatively too slow for a patient in cardiogenic or septic shock.

The key to managing symptomatic bradycardia is determining which signs or symptoms are due to the decreased heart rate. An unstable bradycardia exists clinically when 3 criteria are present:

1. The heart rate is slow.
2. The patient has symptoms.
3. The symptoms are due to the slow heart rate.

Signs and Symptoms

Unstable bradycardia leads to serious signs and symptoms that include

- Hypotension
- Acutely altered mental status
- Signs of shock
- Ischemic chest discomfort
- Acute heart failure

Managing Bradycardia: The Bradycardia Algorithm

The Adult Bradycardia Algorithm (Figure 27) outlines the steps for assessing and managing a patient who presents with unstable bradycardia with a pulse. Implementing this algorithm begins with identifying bradycardia (Step 1), which is typically when the heart rate is less than 50/min. First steps include the components of the BLS Assessment and the Primary Assessment.

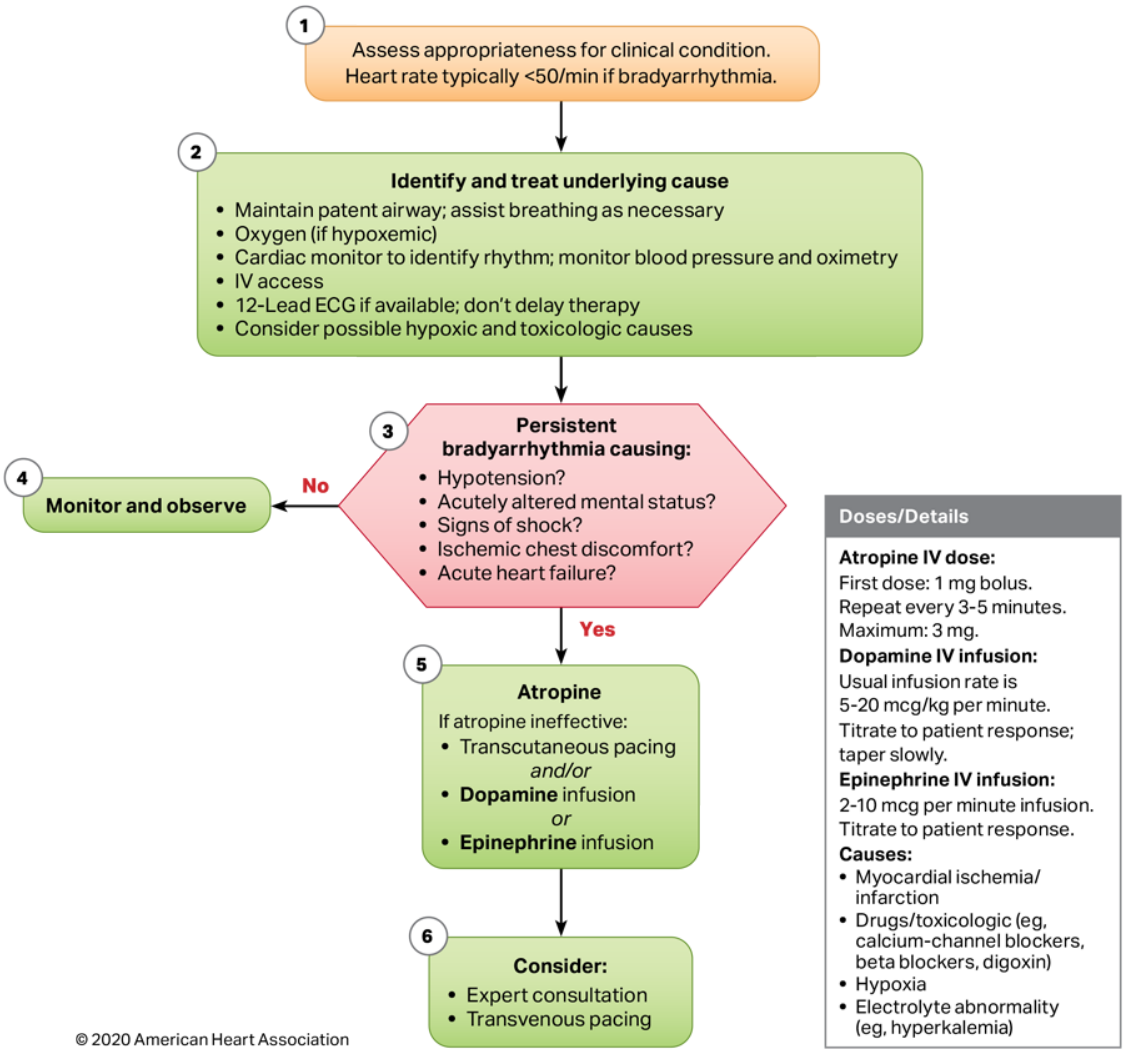

Figure 27. Adult Bradycardia Algorithm.

Identify and treat underlying causes (Step 2):

- Maintain patent airway; assist breathing as necessary.
- Give oxygen (if hypoxemic).
- Use a cardiac monitor to identify rhythm. Monitor blood pressure and oximetry.
- Establish IV access.
- Obtain a 12-lead ECG if available (Step 2).
- Consider possible hypoxic and toxicologic causes.

In the differential diagnosis, the primary decision point in the algorithm is to determine if the patient has signs or symptoms of poor perfusion and if these are caused by the bradycardia (Step 3). If there are no signs of poor perfusion, monitor and observe (Step 4). If there are signs of poor perfusion, administer atropine (Step 5). If atropine is ineffective, prepare for TCP and/or consider dopamine or epinephrine infusion (Step 5). If indicated, seek expert consultation and consider transvenous pacing (Step 6).

The severity of the patient's condition determines the treatment sequence in the algorithm, and you may need to implement multiple interventions simultaneously. If cardiac arrest develops, go to the Adult Cardiac Arrest Algorithm.

Applying the Adult Bradycardia Algorithm

In this case, a patient presents with symptoms of bradycardia. You conduct appropriate assessment and interventions as outlined in the Adult Bradycardia Algorithm while searching for and treating possible contributing factors.

Identify Bradycardia

Identify whether the heart rate is

- Bradycardia by definition, ie, heart rate typically less than 50/min
- Inadequate for the patient's condition (functional or relative)

Identify and Treat Underlying Causes

Perform the Primary Assessment, including the following:

- **A:** Maintain patent airway.
- **B:** Assist breathing as necessary; give oxygen in case of hypoxia; monitor oxygen saturation.
- **C:** Monitor blood pressure, oximetry, and heart rate; obtain and review a 12-lead ECG; establish IV access.
- **D and E:** Conduct a problem-focused history and physical examination; search for possible hypoxic and toxicologic causes, and treat possible contributing factors.

Critical Concepts: Bradycardia

- *Bradycardia can be a sign of life-threatening hypoxia.*
- *Bradycardia associated with hypertension can be a sign of a life-threatening increase in intracranial pressure, especially in the setting of stroke or brain injury.*

Are Signs or Symptoms Caused by Persistent Bradyarrhythmia

Look for these adverse signs and symptoms of the bradycardia:

- **Symptoms: acutely altered mental status, signs of shock, ischemic chest discomfort**
- **Signs: hypotension, acute heart failure**
- Are the signs and symptoms related to the slow heart rate?

Sometimes the symptom is not due to the bradycardia. For example, hypotension associated with bradycardia may be due to myocardial dysfunction rather than the bradycardia. Keep this in mind when you reassess the patient's response to treatment.

Critical Concepts: Bradycardia

The key clinical question is whether the bradycardia is causing the patient's symptoms or some other illness is causing the bradycardia.

Assess for Adequate Perfusion?

You must now decide if the patient has adequate or poor perfusion.

- If the patient has **adequate perfusion**, monitor and observe (Step 4).
- If the patient has persistent bradyarrhythmia causing **poor perfusion**, proceed to Step 5.

Treatment Sequence Summary

If the patient has poor perfusion secondary to bradycardia, treat as follows:

- Give atropine as first-line treatment: atropine 1 mg IV—may repeat to a total dose of 3 mg IV.
- *If atropine is ineffective,* provide transcutaneous pacing and/or dopamine 5 to 20 mcg/kg per minute infusion (chronotropic or heart rate dose) or epinephrine 2 to 10 mcg/min infusion.

The severity of the patient's clinical presentation determines the treatment sequence. For patients with unstable bradycardia, move quickly through this sequence. These patients may be in pre–cardiac arrest and may need multiple interventions simultaneously.

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 where the location of the block is likely to be in infranodal tissue (such as in the bundle of His or more distal conduction system).

Treatment Sequence: Atropine

If you find no immediately reversible causes, atropine remains the first-line drug for acute stable bradycardia. Atropine sulfate acts by reversing cholinergic-mediated decreases in the heart rate and AV node conduction. Dopamine and epinephrine may be successful as an alternative to TCP.

For bradycardia, give atropine 1 mg IV every 3 to 5 minutes (maximum total dose of 3 mg IV). Note that atropine doses of less than 0.5 mg IV may further slow the heart rate.

Use atropine cautiously in the presence of acute coronary ischemia or myocardial infarction (MI). An atropine-mediated increase in heart rate may worsen ischemia or increase infarct size.

Do not rely on atropine in Mobitz type II second-degree or third-degree AV block or in patients with third-degree AV block with a new wide QRS complex. These bradycardias likely will not respond to reversal of cholinergic effects by atropine; preferably, treat them with TCP or β-adrenergic support as temporizing measures while the patient is prepared for transvenous pacing. Atropine administration should not delay external pacing or β-adrenergic infusion for patients with impending cardiac arrest.

A β-adrenergic infusion (ie, dopamine, epinephrine) is not a first-line agent for treating unstable bradycardia, but it can be used as an alternative when a bradycardia is unresponsive to treatment with atropine. You can also use a β-adrenergic infusion as a temporizing measure while the patient is prepared for transvenous pacing.

Vasopressors do not increase survival from bradycardia. Because these medications can improve aortic diastolic blood pressure, coronary artery perfusion pressure, and the rate of ROSC, the AHA continues to recommend their use.

Alternative drugs may also be appropriate in special circumstances such as the overdose of a β-blocker or calcium channel blocker. Do not wait for a maximum dose of atropine if the patient presents with second-degree or third-degree block; rather, move to a second-line treatment after 2 to 3 doses of atropine.

Treatment Sequence: TCP

TCP may be useful to treat unstable bradycardia. TCP is noninvasive and can be performed by ACLS providers. Consider immediate pacing in unstable patients with high-degree heart block when IV access is not available. It is reasonable to initiate TCP in unstable patients who do not respond to atropine.

After initiating TCP, confirm electrical and mechanical capture (Figure 28). Because heart rate is a major determinant of myocardial oxygen consumption, set the pacing to the lowest effective rate based on clinical assessment and symptom resolution. Reassess the patient for symptom improvement and hemodynamic stability. Give analgesics and sedatives for pain control. Note that many of these drugs may further decrease blood pressure and affect the patient's mental status. Try to identify and correct the cause of the bradycardia.

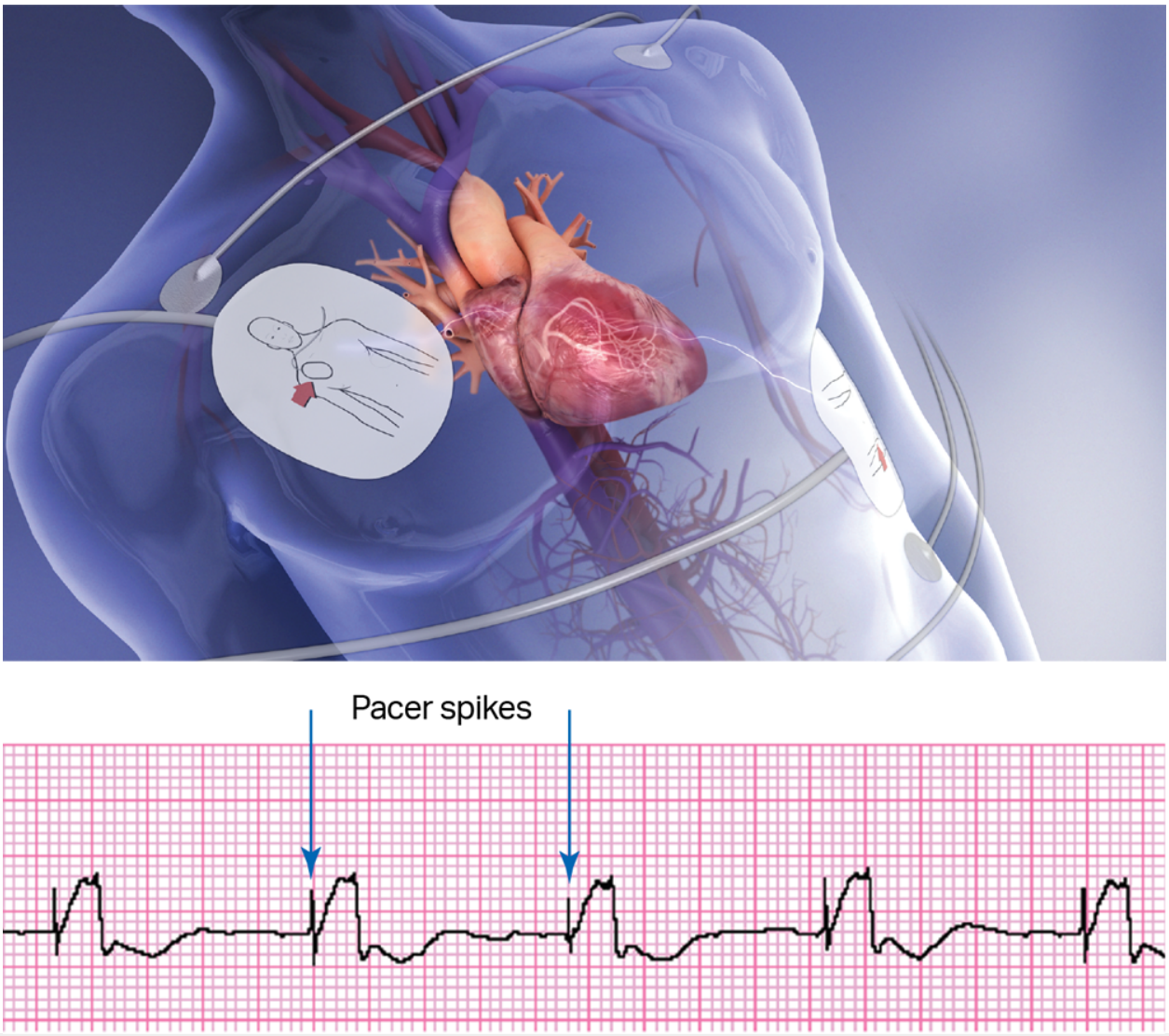

Figure 28. Transcutaneous pacing.

TCP has its limitations—it can be painful and may not produce effective electrical and mechanical capture. If bradycardia is not causing the symptoms, TCP may be ineffective despite capture. For these reasons, consider TCP as an emergent bridge to transvenous pacing in patients with significant sinus bradycardia or AV block.

If you chose TCP as the second-line treatment and it is also ineffective (eg, inconsistent capture), begin an infusion of dopamine or epinephrine and prepare for possible transvenous pacing by obtaining expert consultation.

Sedation and Pacing

Most conscious patients should be sedated before pacing. If the patient is in cardiovascular collapse or rapidly deteriorating, you may need to start pacing without prior sedation, particularly if sedation drugs are not immediately available. Evaluate the need for sedation in light of the patient's condition and need for immediate pacing. A review of sedation drugs is beyond the scope of this course, but the general approach could include the following:

- Give a parenteral narcotic for analgesia.
- Give parenteral benzodiazepine for anxiety and muscle contractions.
- Use a chronotropic infusion once available.
- Obtain expert consultation for transvenous pacing.

Treatment Sequence: Epinephrine, Dopamine

Although β-adrenergic agonists with rate-accelerating effects are not first-line agents for treating stable bradycardia, they are alternatives to TCP or in special circumstances, such as overdose with a β-blocker or calcium channel blocker.

Because epinephrine and dopamine are vasoconstrictors as well as chronotropes, healthcare providers must assess the patient's intravascular volume status and avoid hypovolemia when using these drugs. Dobutamine (a β-adrenergic agonist) is appropriate when vasoconstriction is not desired.

Either epinephrine infusions or dopamine infusions may be used for patients with stable bradycardia, particularly if associated with hypotension, for whom atropine may be inappropriate or after atropine fails.

Begin epinephrine infusion at a dose of 2 to 10 mcg/min and titrate to patient response; begin dopamine infusion at 5 to 20 mcg/kg per minute and titrate to patient response. At lower doses, dopamine has a more selective effect on inotropy and heart rate; at higher doses (greater than 10 mcg/kg per minute infusion), it also has vasoconstrictive effects.

Next Actions

After considering the treatment sequence in Step 5, you may need to

- Consider expert consultation—but do not delay treatment if the patient is unstable or potentially unstable.
- Prepare the patient for transvenous pacing.

Transcutaneous Pacing

Many devices can pace the heart by delivering an electrical stimulus, causing electrical depolarization and subsequent cardiac contraction, and TCP delivers pacing impulses to the heart through the skin via cutaneous electrodes. Most defibrillator manufacturers have added a pacing mode to manual defibrillators. Performing TCP is often as close as the nearest defibrillator, but you should know the indications, techniques, and hazards for using TCP.

Indications and Precautions

Indications for TCP are as follows:

- Hemodynamically unstable bradycardia (eg, hypotension, acutely altered mental status, signs of shock, ischemic chest discomfort, acute heart failure hypotension)
    - –Unstable clinical condition likely due to the bradycardia
- Bradycardia with stable ventricular escape rhythms

Precautions for TCP are as follows:

- TCP is contraindicated in severe hypothermia.
- Conscious patients require analgesia for discomfort unless delay for sedation will cause or contribute to deterioration.
- Do not assess the carotid pulse to confirm mechanical capture; electrical stimulation causes muscular jerking that may mimic the carotid pulse.

Technique

Perform TCP by following these steps:

1. Place pacing electrodes on the chest according to package instructions.
2. Turn the pacer on.
3. Set the demand rate to 60 to 80/min. You can adjust this rate up or down (based on patient clinical response) once pacing is established.

4. Set the current milliamperes output 2 mA above the dose at which consistent capture is observed (safety margin).

External pacemakers have either *fixed* rates (asynchronous mode) or *demand* rates.

Assess Response to Treatment

Signs of hemodynamic impairment include hypotension, acutely altered mental status, signs of shock, ischemic chest discomfort, acute heart failure, or other signs of shock related to the bradycardia. The goal of therapy is to improve these signs and symptoms rather than target a precise heart rate. Start pacing at a rate of 60 to 80/min. Once pacing is initiated, adjust the rate based on the patient's clinical response.

Consider giving atropine before pacing in mildly symptomatic patients. Do not delay pacing for unstable patients, particularly those with high-degree AV block. Atropine may increase heart rate, improve hemodynamics, and eliminate the need for pacing. If atropine is ineffective or likely to be ineffective, or if IV access or atropine administration is delayed, begin pacing as soon as it is available.

Patients with ACS should be paced at the lowest heart rate that allows clinical stability. Higher heart rates can worsen ischemia because heart rate is a major determinant of myocardial oxygen demand. Ischemia, in turn, can precipitate arrhythmias.

If unstable bradycardia does not respond to atropine, consider a chronotropic drug infusion to stimulate heart rate as an alternative to pacing:

- Epinephrine: administer at 2 to 10 mcg/min infusion and titrate to patient response.
- Dopamine: administer at 5 to 20 mcg/kg per minute infusion and titrate to patient response.

Bradycardia With Escape Rhythms

A bradycardia may lead to secondary bradycardia-dependent ventricular rhythms. When a patient's heart rate falls, an electrically unstable ventricular area may "escape" suppression by higher and faster pacemakers (eg, sinus node), especially in the setting of acute ischemia. These ventricular rhythms often fail to respond to drugs. With severe bradycardia, some patients will develop wide-complex ventricular beats that can precipitate VT or VF. Pacing may increase the heart rate and eliminate bradycardia-dependent ventricular rhythms. However, an accelerated idioventricular rhythm (sometimes called AIVR) may occur in the setting of inferior wall MI. This rhythm is usually stable and does not require pacing.

Patients with ventricular escape rhythms may have normal myocardium with disturbed conduction. After correcting electrolyte abnormalities or acidosis, use pacing to stimulate effective myocardial contractions until the conduction system recovers.

Standby Pacing

Acute ischemia of conduction tissue and pacing centers can cause several bradycardic rhythms in ACS. Patients who are clinically stable may decompensate suddenly or become unstable over minutes to hours due to worsening conduction abnormalities, and these bradycardias may deteriorate to complete AV block and cardiovascular collapse. To prepare for this clinical deterioration, place TCP electrodes on any patient with acute myocardial ischemia or infarction associated with the following rhythms:

- Symptomatic sinus node dysfunction with severe and symptomatic sinus bradycardia
- Asymptomatic Mobitz type II second-degree AV block
- Asymptomatic third-degree AV block
- Newly acquired left, right, or alternating bundle branch block or bifascicular block in the setting of AMI