

Bradycardia

CODE SCENARIO

A code is called for a 78-year-old man who was admitted to the hospital for syncope of unknown etiology. He was resting comfortably in a monitored bed when the monitor alarm suddenly signaled an abnormal heart rate. The nurse finds the man unresponsive with a heart rate of 42 bpm. The code team arrives and finds the man unconscious in bed. The team leader assesses for a pulse and feels a faint carotid pulse at about 40 bpm corresponding to the QRS complexes on the monitor (Figure 12-1).

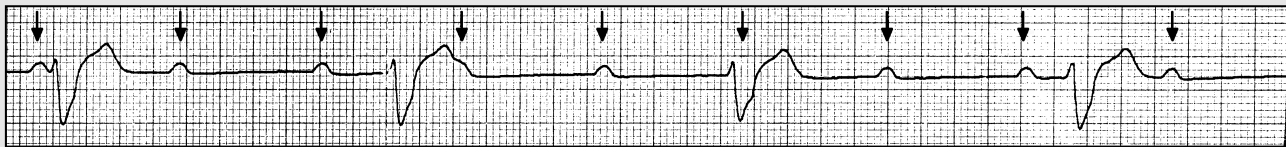


FIGURE 12-1

1. What is the cardiac rhythm?
 - a. Pulseless electrical activity
 - b. Type I second-degree heart block (Wenckebach)
 - c. Type II second-degree heart block
 - d. Third-degree heart block

The rhythm strip demonstrates P-waves that are dissociated from the QRS complexes (denoted by arrows in figure), which is indicative of third-degree, or complete, heart block. Pathologic bradycardias (e.g., those that result in hypoperfusion) can be classified into one of several types. First-degree heart block is defined as a prolonged PR interval and does not result in hypoperfusion. Type I second-degree heart block, commonly known as Wenckebach, is defined as progressive PR prolongation until a QRS complex is entirely dropped. Similar to first-degree block, Wenckebach does not compromise cardiac output. Both type II second-degree heart block and third-degree heart block are malignant arrhythmias frequently associated with hemodynamic compromise. Type II second-degree heart block is characterized by fixed PR intervals with intermittently dropped QRS complexes. This rhythm is a precursor to third-degree heart block, in which there is complete dissociation of P-waves from the QRS complexes, as seen in this patient.

2. What is the first appropriate action for this patient?
 - a. Initiate cardiopulmonary resuscitation (CPR) with bag-valve-mask (BVM) ventilation and cardiac compressions
 - b. Establish large-bore IV access
 - c. Intubate the patient
 - d. Administer atropine 1 mg IV push

In resuscitating this patient, several things should be occurring simultaneously. Ideally, a code cart containing standard code medications, a defibrillator, and airway equipment is immediately available. Realistically, it may take several minutes for the code cart to be wheeled into the patient's room. The patient should be placed on high-flow oxygen via a nonrebreather mask until a BVM can be obtained. Someone should be responsible for connecting the defibrillator and, in this case, preparing for

transcutaneous pacing. Because this patient has been admitted to the hospital, IV access is probably already established. Large-bore access is not a high priority immediately. CPR is not necessary in a patient who has a palpable pulse, albeit bradycardiac and weak. Intubation may be indicated, but it will take several minutes, and the most immediate priority in this patient is reversing the bradycardia that is likely the etiology of the patient's mental status decline. Atropine 1 mg IV push should be given as soon as possible. Ideally one person should be managing the airway and preparing for intubation, another person should be preparing the defibrillator for transcutaneous pacing, and a third should be preparing the code medications. In this case both atropine and epinephrine should be readily available. The patient is administered 1 mg of atropine IV and his pulse increases to 52 bpm. The patient becomes more alert and his blood pressure is 82/42, but his heart rate dips back down to the low 40s again.

3. What is the next appropriate step?
- Administer epinephrine 1 mg IV push
 - Initiate transcutaneous pacing
 - Start dopamine drip at 10 $\mu\text{g}/\text{kg}/\text{min}$
 - Defibrillate at 50 J biphasic

The effect of atropine in the setting of bradycardia is to block parasympathetic receptors, and thus augment sympathetic drive. Atropine acts primarily at the sinus node and atrioventricular (AV) node. However, in third-degree heart block, the ventricular rate is determined by the escape pacemaker of the AV node or ventricular escape beats. Therefore, atropine is often ineffective in third-degree heart block. Epinephrine has the effect of increasing sympathetic drive, thus promoting escape pacemakers in the AV node and ventricle. Epinephrine also directly increases cardiac contractility and cardiac output. The best way to reverse this patient's hypoperfusion is to reestablish a pacemaker that fires at greater than 60 bpm. If this is not accomplished by atropine alone, then the next best step is to start transcutaneous pacing. Epinephrine may transiently increase heart rate and blood pressure, but bradycardia will likely recur within several minutes as the drug wears off. Epinephrine imparts a massive sympathetic surge and workload on a heart that likely has baseline impairment. If the patient remains hypotensive, dopamine can be started to increase inotropy.

Transcutaneous pacing is initiated and set at a rate of 60 bpm, and a dopamine drip is started at 10 $\mu\text{g}/\text{kg}/\text{min}$ (Figure 12-2). The patient becomes more alert and a repeat blood pressure is 108/68. The patient is transferred to the ICU where a right subclavian central venous line is placed and transvenous pacing is initiated.



FIGURE 12-2 Transcutaneously paced rhythm.

Answers: 1-d; 2-d; 3-b

DEFINITION

Bradycardia is defined as a heart rate of less than 60 bpm. This may be perfectly normal in young, healthy adults or in patients who are taking AV nodal blocking agents. Bradycardia is considered pathologic if it results in decreased cardiac output to the point where

it causes symptoms (Box 12-1). Pathologic bradycardia occurs most commonly in elderly people with underlying cardiac conduction abnormalities. It occurs less commonly in cases of toxic ingestion (e.g., overdose of AV nodal blockers, digoxin, organophosphates, and others). It is important to remember that the heart rate is determined by the pulse and not what is

BOX 12-1 Common symptoms and signs associated with symptomatic bradycardia

- Lightheadedness
- Fatigue/weakness
- Syncope
- Nausea
- Diaphoresis
- Shortness of breath
- Weak pulses
- Hypotension

TABLE 12-1 Characteristics of heart blocks

Type of heart block	Characteristics
First-degree	PR prolongation > 200 msec (five small boxes)
Second-degree type I (Wenckebach)	Progressive PR prolongation until a QRS complex is dropped
Second-degree type II	Fixed PR interval with intermittently dropped QRS complex
Third-degree	AV dissociation (P-waves are completely dissociated from QRS complexes)

displayed on the monitor. For example, a patient may be in bigeminy (e.g., alternating sinus beat with an ectopic ventricular beat) with a displayed heart rate of 80. But if the ectopic ventricular contractions are not strong enough to effect forward blood flow, then the actual pulse rate is 40 and the patient is bradycardic.

ETIOLOGY

Symptomatic bradycardia is the result of one of two possible physiologic disturbances. The first is the depression of the dominant pacemaker, or the sinus node. The second is the result of a conduction system delay, most typically at the AV node. Either form of bradycardia may result in the firing of secondary pacemakers in the heart, resulting in “escape” beats. These secondary pacemakers include the AV node (junctional escape beats) and ventricle (ventricular escape beats).

Sinus Node Dysfunction (Sick Sinus Syndrome)

Sinus node dysfunction has several manifestations, including, sinus pauses, bradycardia, and tachycardia-bradycardia syndrome. A sinus pause is a period greater than 5 seconds during which there is no atrial depolarization, which appears as a period of asystole on the monitor. This may result in symptoms such as

lightheadedness, blurred vision, nausea, or syncope. Tachycardia-bradycardia syndrome is manifested by periods of tachycardia, often rapid atrial fibrillation, followed by periods of profound bradycardia. Sinus node dysfunction is most commonly the result of acute or chronic cardiac ischemia, dilated cardiomyopathy, or medications.

AV Block

AV block occurs when conduction through the atria, AV node, and proximal His-Purkinje system is impaired. Table 12-1 summarizes the increasing degrees of AV block based on ECG criteria.

First-degree AV block is characterized by PR interval prolongation (more than 200 msec or 5 small boxes) and is generally a benign finding. Figure 12-3 depicts a rhythm strip with first-degree heart block. Second-degree heart block is divided into type I (Wenckebach) and type II.

Type I second-degree AV block (Wenckebach) is caused by a conduction disturbance within the AV node. It is characterized by the progressive lengthening of the PR interval until the impulse is not conducted, resulting in a dropped QRS complex (Figure 12-4). A resetting of the original PR interval follows each dropped beat. Similar to first-degree AV block, type I second-degree AV block is infrequently clinically significant, except that it may eventually progress to type II second-degree or to third-degree heart block.

**FIGURE 12-3 First-degree heart block: PR interval is greater than 200 msec.**

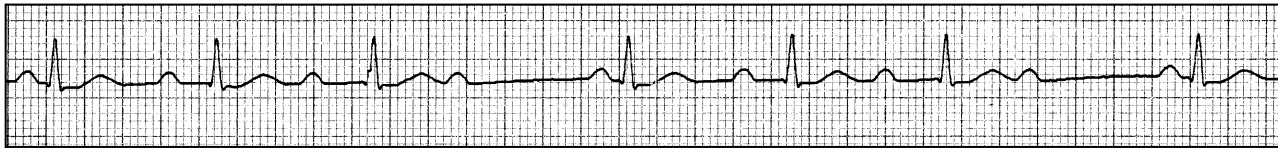


FIGURE 12-4 Second-degree type I heart block (Wenckebach); note that the PR interval progressively lengthens until a QRS complex is not conducted at all.

Additionally, type I second-degree AV block may be seen in patients with inferior myocardial ischemia.

Type II second-degree AV block and third-degree AV block are more malignant arrhythmias that are associated with a conduction disturbance below the level of the AV node. In type II second-degree AV block, QRS complexes are intermittently dropped with intervening complexes that have normal PR intervals (Figure 12-5). Type II second-degree heart block often precedes third-degree heart block. Third-degree heart block is characterized by the complete absence of atrial impulse conduction through the AV node. Although P-waves (which represent atrial depolarization) occur at a regular rate on the ECG, none of the atrial impulses are conducted to the ventricles. Therefore, for the ventricle to depolarize, it must be triggered by a ventricular pacemaker, which typically triggers at a rate of 30 to 40 bpm. This results in complete dissociation of P-waves and QRS complexes, with the rate of P-waves being significantly faster than the rate of QRS complexes (Figure 12-6).

TREATMENT

Asymptomatic Patients Can Wait

The treatment of bradycardia depends on the stability of the patient. Signs and symptoms of inadequate

tissue perfusion, including mental status changes, hypotension, chest pain, and difficulty breathing, indicate instability. Prompt treatment is warranted in these patients (Box 12-2). In stable patients, the treatment depends on the etiology of the bradycardia. For stable sick sinus syndrome, the patient can be closely monitored for further decompensation. These patients usually require permanent pacemaker placement by a cardiologist in the cath lab. Patients with first-degree or type I second-degree AV block require no treatment unless they develop symptoms. Conversely, given the unstable nature of type II second-degree and third-degree AV block, transvenous pacing is often required emergently until a permanent pacemaker can be implanted.

Think Cardiac Ischemia

Acute myocardial infarction (MI) is a common cause of bradycardia; therefore, obtaining a 12-lead ECG early is important. Occlusion of the right coronary artery, causing ST-segment elevation in the inferior leads, often results in second- and third-degree heart block. Inferior MI is also frequently associated with increased parasympathetic tone. Symptoms of inferior MI tend to be vagal in nature, and include nausea, diaphoresis, abdominal discomfort, and bradycardia. Inferior MI may also be associated with right ventricular

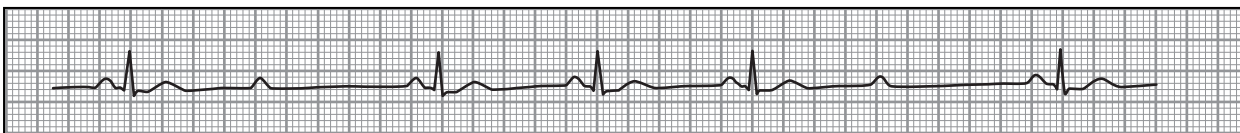


FIGURE 12-5 Second-degree type II heart block; note that the second and sixth P-waves are not followed by a QRS complex.

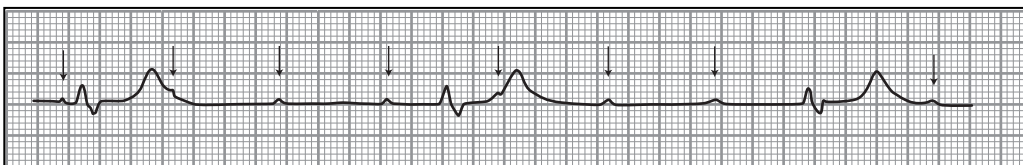


FIGURE 12-6 Third-degree heart block; arrows indicate P-waves that are dissociated from the QRS complexes.

BOX 12-2 Critical steps in treating bradycardia

1. Identify symptomatic bradycardia
2. Establish ABCs, IV, O₂, monitor
3. Apply transcutaneous pacing pads and defibrillator leads
4. Identify specific heart blocks
5. Identify associated cardiac ischemia
6. Reach for atropine as first-line drug
7. Establish transcutaneous pacing if atropine is ineffective
8. Start vasopressors if patient continues to be hypotensive
9. Know when transvenous pacing is indicated

infarction. Symptomatic bradycardia in these patients often responds to a fluid bolus, which increases right ventricular filling and thus increases cardiac output.

Acute anterior ST-segment elevation MI, or occlusion of the left coronary or left anterior descending artery, can cause third-degree heart block due to ischemia of the AV node. These patients require immediate cardiac catheterization. If cardiac catheterization is not available, systemic thrombolysis and transcutaneous pacing should be initiated.

Unstable Patients Require Intervention

Unstable patients with bradycardia require immediate stabilization (see bradycardia algorithm on page 75).

BOX 12-3 Atropine

Atropine is a compound found in *Atropa belladonna* and other plants. As a tertiary amine, atropine is a tertiary amine and therefore lipid soluble and thus is widely distributed throughout the body. This results in a long duration of action. Atropine blocks muscarinic receptors, making it the prototypical anticholinergic agent. Its effect on the heart is mediated by competitive M₂ receptor blockade in the AV node resulting in uninhibited sympathetic tone that increases AV conduction and heart rate.

Other anticholinergic effects of atropine include decreased salivation (especially in children), mydriasis (this effect lasts more than 72 hours), decreased gut motility, and decreased bladder tone (urinary retention). It is important to remember that pupils will be dilated and fixed after the administration of atropine; therefore, fixed pupils should not be misinterpreted as irreversible brain death. Another key point is that atropine acts at the AV node; therefore, atropine is not useful in bradycardias caused by type II second-degree and third-degree AV block in which the conduction block is below the AV node.

BOX 12-4 Dopamine

If the maximum dose of atropine (0.04 mg/kg or about 2–3 mg) has been administered without effect, a dopamine infusion should be started at 5 µg/kg/min. The dose can be titrated up to 20 µg/kg/min as needed to restore heart rate and blood pressure. Dopamine is a catecholamine that stimulates beta-adrenergic receptors, causing an increase in cardiac contractility and chronicity. Dopamine at higher doses also stimulates alpha-adrenergic receptors, resulting in peripheral vasoconstriction and increased blood pressure.

The first line of therapy is atropine 0.5–1.0 mg IV given in 3- to 5-minute intervals up to a total dose of 0.04 mg/kg. While atropine is being administered, the external pacing pads should be placed on the patient and transcutaneous pacing initiated. If the patient remains hypotensive after these measures, then dopamine or epinephrine drips should be started. These patients should receive transvenous pacing as soon as possible, and the underlying cause of bradycardia should be sought out and treated expeditiously. See Boxes 12-3, 12-4, and 12-5 for more information on atropine, dopamine, and transcutaneous pacing.

BOX 12-5 Transcutaneous pacing

Transcutaneous pacing is a lifesaving intervention that is critical to treating symptomatic bradycardia. Most current defibrillators perform transcutaneous pacing, which provides electrical pacing impulses to the heart through the skin by cutaneous electrodes. The anode and cathode electrodes are contained in the large defibrillator pads that are also used for electrical defibrillation. Of note, the three defibrillator limb leads must also be attached to perform transcutaneous pacing.

Once the leads are attached, the operator sets a pacing rate, typically 60 to 80 bpm. The electrical current is then titrated up until electrical capture is obtained. Starting with the dial at zero, the electrical current is gradually increased until contraction of external musculature occurs. This is often uncomfortable for an alert patient, and therefore sedation is required. As the current is increased, the operator monitors the cardiac rhythm. Capture is characterized by the presence of ventricular depolarizations following pacing impulses. Once capture occurs, the electrical current required to achieve capture is noted (typically 60 to 100 mAmp) and maintained. The peripheral pulse rate should correspond with the set pacer rate if capture is occurring properly.

KEY POINTS

- Treat only symptomatic bradycardia in the acute stage
 - First-degree and type I second-degree AV block do not require treatment if asymptomatic
 - Type II second-degree and third-degree AV block will require transvenous pacing
- Be wary of asymptomatic bradycardias that may deteriorate
 - Type II second-degree AV block
 - Third-degree AV block
- Remember the treatment sequence for symptomatic bradycardia
 - Atropine
 - Transcutaneous pacing
 - Dopamine
 - Epinephrine

ALGORITHM: BRADYCARDIA

