Chapter 12
Bradyarrhythmia in the ER

Carlos Jerjes-Sánchez, Jose Gildardo Paredes-Vázquez, Mariana Vanoye Tamez, David Rodríguez, Raul del Toro-Mijares, and Mauricio Vázquez Guajardo

12.1 The Scope of the Problem

Bradyarrhythmias are defined by a heart rate below 60 beats per minute (bpm) and are not always a pathologic condition with life-threatening aspects. They are caused by an abnormal function at any point in the conduction system of the heart. They are a less frequent variant of cardiac arrhythmias presenting to the emergency room (ER) than tachyarrhythmias but require the same meticulous assessment and risk stratification, because of their broad clinical and prognostic spectrum. The first step in the clinical assessment must be the determination of hemodynamic stability or instability, depending on vital signs, mental status, and other symptoms. This will establish the initial management of the patient. The electrocardiogram (ECG) is the gold standard for the diagnosis and should be performed in the first 10 minutes of admission to the ER. It can be performed at the bedside of the patient, and its thorough analysis provides important information for clinical decision-making. On the other hand, it is important that other cardiac and metabolic causes of the bradyarrhythmia should be considered during the initial approach, as their recognition and treatment would resolve the acute event.

12.2 Prevalence

A bradyarrhythmia is an uncommon complaint in the ER. Unfortunately, no clinical records are available with accurate information about their real prevalence in ER. This condition can be divided according to its etiology. The primary causes are due to an intrinsic defect in the conduction system, whereas the secondary causes include extrinsic alterations of the conduction system. One study found that only 15% of unstable patients with bradyarrhythmia that presents to the ER are due to a primary defect in the conduction system. On the other hand, secondary causes are more common and account for about 85% of cases. Acute myocardial infarction

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is the most common secondary cause of bradyarrhythmia (40% of cases) and has been reported as responsible for up to 25% of hemodynamically unstable patients at ER. The most common drug cause of bradyarrhythmia is an overdose of beta-adrenergic blockers.

12.3 High-Clinical Suspicion in the ER

Any patient that presents at ER with palpitations, chest pain, dizziness, lightheadedness, dyspnea, or syncope has a high probability of having a bradyarrhythmia, and an ECG must be done within the first 10 minutes from admission to the ER. It can also present as shortness of breath, exercise intolerance, or fatigue.

12.4 Risk Factors

The clinical features related to the presence of bradycardia are the following:

- Past medical history of:
  - Atrial fibrillation
  - Cardiomyopathies
  - Structural heart disease
  - Ischemic heart disease
  - Sudden cardiac death episode
  - Unexplained syncope
  - Metabolic diseases with electrolyte abnormalities
  - Sleep disorders (i.e., sleep apnea)
- Alcohol abuse
- Drug abuse
- Use of herbal medication with unknown effect
- Use of potassium or magnesium modifying drugs
- Use of beta-blockers, digoxin, calcium channel blockers, or other medications that inhibit AV node or sinus node
- The family history of sudden cardiac death

12.5 Clinical Presentation

Clinical presentation and physical findings of bradyarrhythmias are highly variable, depending on the type and the heart rate. The spectrum varies from light dizziness to syncope with subsequent hypoperfusion, organ failure, and cardiac arrest. Dizziness and weakness are the most frequent complaint. However, these symptoms usually are not enough to make the patient go to the ER. The main symptom that makes the patients attend the ER is syncope [1].

Main clinical characteristics [2, 3]:

- Syncope
- Fatigue
- Weakness
- Dizziness
- Cognitive impairment
- Dyspnea
- Palpitations
- Chest pain
- Nausea
- Lightheadedness
- Diaphoresis

Bradycardias can be broadly divided into two main categories: nodal sinus dysfunction (SND) and atrioventricular (AV) block. The clinical presentation will be determined by the primary or secondary cause of the bradycardia (see Table 12.1), associated comorbidities, baseline medication, ventricular rate, and blood pressure compensation. The prognosis is related with the reversibility and the severity of the primary bradycardia cause. The ventricular rate is related to the presence of junctional of ventricular rhythm that reaches the depolarization threshold in these tissues.

The most common causes of reversible bradycardia are [3]:

- Myocardial infarction
- Athletic training
- Drug toxicity
  - Beta-blockers, non-dihydropyridine calcium channel blockers, digoxin, antiarrhythmic drugs, lithium, methyldopa, risperidone, cisplatin, interferon
- Toxin overdose (i.e., toluene, organophosphates, tetrodotoxin, cocaine)
- Electrolytes imbalance
- Hypothyroidism
- Hypothermia
- Hypercarbia
- Acidosis
- Sleep apnea
- Hypovolemic shock
- Guillain-Barre syndrome

12.6 Physical Examination

12.6.1 Clinical Instability

The first step in the clinical approach in ER is the evaluation of hemodynamic stability depending on the following indicators [3]:

- Hypotension (systolic blood pressure < 90 mmHg)
- Systemic hypoperfusion

Table 12.1 Main conditions associated with bradyarrhythmia [3, 4]

System	Clinical conditions
Cardiovascular	Acute coronary syndrome
	Nonischemic cardiomyopathy
	Degenerative fibrosis of conductive system
	New-onset AF associated with system conductive disorders
	Myocarditis
	Infectious endocarditis
	Cardiac amyloidosis
	Post-valvular surgery, catheterization, or septal myomectomy
Infection/ inflammation	Chagas disease
	Lyme disease
	Diphtheria
	Meningitis
	Intracranial tumors
	Sarcoidosis
	Toxoplasmosis
Infiltrative disorders	Systemic amyloidosis
	Hemochromatosis
	Lymphoma
Rheumatic disorders	Rheumatoid arthritis
	Scleroderma
	Systemic lupus erythematosus
Severe metabolic disorders	Metabolic acidosis
	Hyperkalemia
	Hypokalemia
	Hypothermia
	Hypothyroidism
	Adrenal disease
	Hypoxia
Vagotonic – -associated with increased vagal tone	Sleep apnea
	High-level athletic conditioning
	Neurocardiogenic syncope

- Altered mental status
- Acute congestive heart failure
- Ischemic chest pain
- Shock

12.6.2 Clinical Stability

An extended clinical examination should be performed, with special attention to vital signs, heart and lung auscultation, and pulse examination. The ER physicians must have a high clinical suspicion of any other cardiovascular cause of bradycardia

that requires further evaluation and treatment (e.g., acute coronary syndrome, severe hypoxemia, etc.).

12.7 Diagnosis Approach

The diagnostic approach of bradyarrhythmias is focused on the early diagnosis of the cause and the recognition of those etiologies that require immediate treatment.

The cornerstone of the diagnosis is the ECG. It must be performed in the first 10 minutes after patient's arrival to the ER, and it is the best tool to differentiate the different types of bradyarrhythmia.

The primary evaluation of the patient includes vital signs, medical history (emphasizing in medication use), complete physical exam, and laboratory test needed to rule out other cardiovascular or metabolic causes, like acute coronary syndrome and severe heart failure, which can be life-threatening.

After the acute management in the ER, the diagnostic approach to determine the specific cause of the bradyarrhythmia and to establish the chronic management can also be performed at the ER or continued as an outpatient depending on the circumstances and symptoms of the patient (see Fig. 12.1).

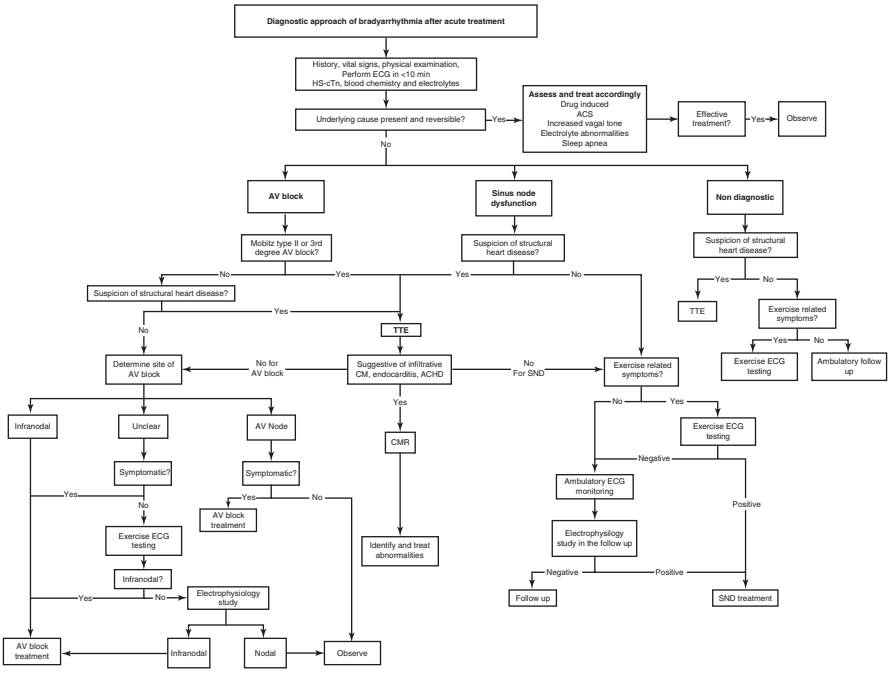


Fig. 12.1 Diagnostic approach of bradyarrhythmias after acute treatment. ECG electrocardiogram, HS-cTn high-sensitive cardiac troponin, ACS acute coronary syndromes, AV atrioventricular, TTE transthoracic echocardiogram, CM cardiomyopathy, ACHD adult congenital heart disease, SND sinus node dysfunction

12.8 Electrocardiogram

The ECG is the most important diagnostic tool and is the mandatory first step in emergency attention care.

The first step in the ECG interpretation is recognizing the presence or absence of normal atrial activity and AV conduction. This is determined by the presence of a p wave, ventricular depolarization (QRS complex) after every atrial depolarization, and normal duration between atrial and ventricular depolarization (PR interval).

The electrocardiographic findings presented in the different types of bradyarrhythmias are described in Table 12.2.

12.9 Imaging

12.9.1 Chest X-ray

A chest-X-ray is not required for the diagnostic assessment of patients with arrhythmia unless another cardiac or pulmonary cause is highly suspected. In patients with heart failure history or with comorbidities, a chest X-ray is mandatory.

12.9.2 Transthoracic and Transesophageal Echocardiogram

A transthoracic echocardiogram is required primarily to rule out structural heart diseases like cardiomyopathies, endocarditis, and adult congenital heart diseases. Transesophageal echocardiography is indicated only in patients with high clinical suspicion of infective endocarditis but is not a routinary recommendation [3].

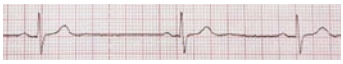
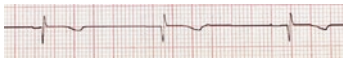
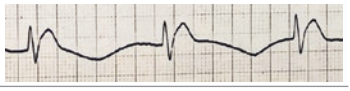
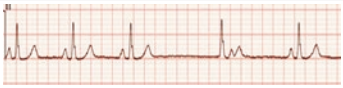
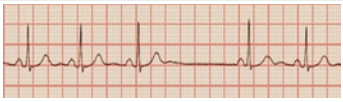
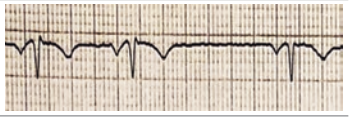
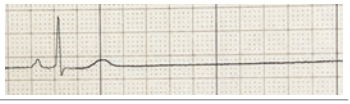
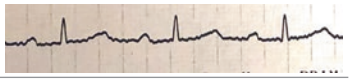
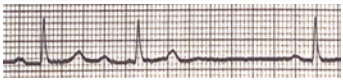
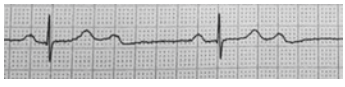
12.9.3 Cardiac Computed Tomography

Cardiac computed tomography is not required for the diagnostic assessment of patients with arrhythmia unless another cardiac or pulmonary cause is highly suspected (e.g., pulmonary embolism or aortic dissection).

12.10 Laboratory Evaluation

The laboratory workup is oriented toward the identification of electrolytic abnormalities and to ruling out other possible and potentially life-threatening causes of bradycardia like acute myocardial infarction. The laboratory tests are listed below:

Table 12.2 Electrocardiogram findings in the principal bradyarrhythmias [1]

Bradyarrhythmia	Electrocardiogram characteristics	Example
Sinus bradycardia	P wave positive in leads I, II, and III, atrioventricular conduction = 1:1, normal PR interval, heart rate <60 bpm	
Junctional rhythm	Absence of normal P wave, rare retrograde P wave inverted and adjacent to QRS complex, narrow QRS complex, regular rate, HR 40–60 bpm	
Idioventricular rhythm	Widened QRS complex, regularly QRS complexes, no P waves, HR 30–50 bpm	
Sinoatrial exit block second-degree type I Wenckebach	P-P interval progressively shortens before pause, duration of the pause is less than two P-P intervals	
Sinoatrial exit block second-degree type II	The absence of normally expected P wave. The interval without P waves is equal approximately two, three, or four times the normal P-P interval	
Sinus pause	The absence of P wave >3 s after a normal sinus complex, no multiple of the P-P interval	
Sinus arrest	The absence of P wave after a normal sinus complex, no multiple of the P-P interval	
Tachy-bradycardia syndrome	Sinus bradycardia, ectopic atrial bradycardia, or sinus pause alternating with abnormal atrial tachycardia, atrial flutter or AF	
1st grade AV block	Consistent P wave to QRS complex relationship, fix PR interval > 200 ms	
2nd grade AV block type I Wenckebach	Progressive prolongation of PR interval until an atrial impulse is completely blocked: a P wave without accompanying QRS complex. After non-conducted beat, cycle repeats	
2nd grade AV block type 2 Möbitz type II	Fix PR interval > 200 ms, P wave associated with a QRS complex until a P wave is not accompanied by QRS complex	
3rd grade AV block (complete)	AV dissociation (P wave not associated with QRS complex, atrial rate greater than ventricular rate. P wave defluxion during QRS	

- Chemistry profile
- Electrolytes
- High-sensitive troponin I
- D-dimer
- Type B brian natriuretic peptide
- Toxicologic test
- Thyroid profile

12.11 Differential Diagnosis

Differential diagnoses among bradyarrhythmia are the following:

- Sinus node dysfunction
  - Sinus bradycardia
  - Tachycardia-bradycardia syndrome
  - Sinoatrial (SA) exit block
    - First-degree SA block
    - Second-degree type I SA block
    - Second-degree type II SA block
    - Third-degree SA block
  - Sinus pauses
  - Sinus arrest
- AV block
  - 1st grade AV block
  - 2nd grade AV block type I
  - 2nd grade AV block type 2
  - 3rd grade AV block (complete)

Sinus bradycardia is a normal sinus depolarization with a heart rate < 60 bpm that reaches the atrial tissue with no interference. It is the most common clinical presentation of bradycardia secondary to drugs and has an excellent response to the acute treatment.

Tachycardia-bradycardia syndrome is an intermittent cause of bradyarrhythmias included in the spectrum of sinus node dysfunction. It is characterized by sinus bradycardia, ectopic atrial bradycardia, or sinus pause alternating with periods of abnormal atrial tachycardia, atrial flutter, or atrial fibrillation. It is usually presented in older patients with ischemic or inflammatory causes of cardiac insult.

SA exit blocks are included in the sinus node dysfunction classification. In these cases, the impulse is generated by the sinus node, but there is a block in the conduction between the sinus node and atrial tissue. This explains why the main ECG findings are multiple P-P intervals, "group-beating" of atrial depolarizations, and

sinus pauses. The block degrees are described in the same fashion that it is in AV block. The definitive diagnostic test is an electrophysiological study. This bradyarrhythmia arises from myocardial disease and drug toxicity and less frequently due to vagal stimulation.

Sinus arrest pause occurs when sinus node depolarization happens more than 3 seconds after the last atrial depolarization. It is secondary to the failure of nodal sinus discharge that results in the absence of atrial depolarization and periods of ventricular asystole or escape rhythm. P-P intervals are not multiple of the P-P interval without the bradycardia. The clinical scenarios are the same as those seen in SA blocks.

Sinus arrest has the same pathophysiology as the sinus pause, but with no evidence of sinus node depolarization during the period of bradyarrhythmia. It is related to atrial infarction and inflammatory disorders [1, 3, 4].

12.12 Treatment

The acute treatment of bradyarrhythmias in ER is based on the hemodynamic stability of the patient (see Fig. 12.2).

The initial treatment includes continuous ECG monitoring, vital signs, and oximetry and must be performed in a facility that can provide a complete cardiac resuscitation.

In unstable patients, the first-line therapy is atropine 0.5 mg IV with possible subsequent doses every 3–5 minutes until symptoms resolved.

In patients with clinical stability, the goal of the initial management is monitoring the bradycardia until it is resolved and concomitantly identifying the possible cause of the arrhythmia.

It is important to keep in mind the possibility of drug toxicity because the treatment can be more efficient with the specific antidote. The main medications related to bradycardia that have specific medical therapy are [3]:

- Non-dihydropyridine calcium channel blockers: treat with 10% calcium chloride 1–2 grams IV every 10–20 minutes or an infusion of 0.2–0.4 mL/kg/h. Another option is 10% calcium gluconate 3–6 grams IV every 10–20 minutes or an infusion of 0.6–1.2 mL/kg/h.
- Beta-blockers: treat with glucagon 3–10 mg IV with an infusion of 3–5 mg/h. If the bradycardia persists and the symptoms are moderate to severe, insulin 1 unit/kg IV bolus followed by an infusion of 0.5 units/kg/h is indicated.
- Digoxin: treat with specific digoxin antibody fragment one vial IV over 30 minutes with possible extra dose, depending on the amount ingested (that every vial binds 0.5 mg of digoxin).

In patients with severe symptoms and AV block, it is important to recognize three conditions that identify bronchodilator drugs, aminophylline, and theophylline, as a treatment of choice to abolish bradycardia symptoms and clinical instability [3].

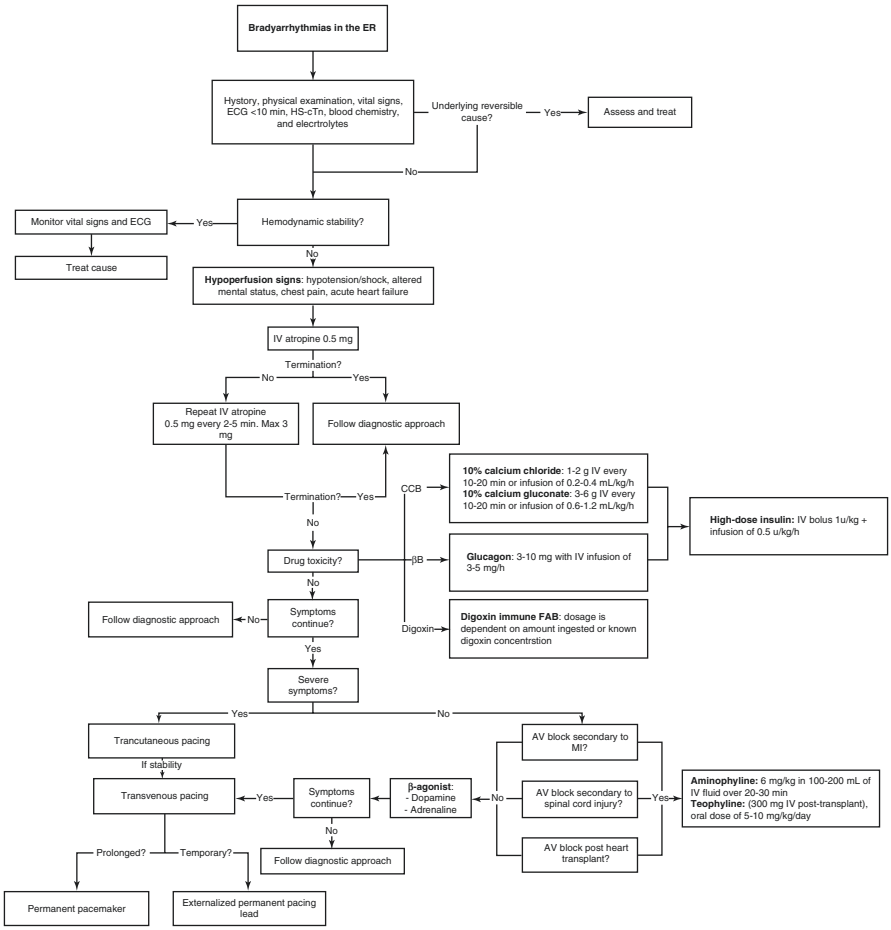


Fig. 12.2 Treatment approach of bradyarrhythmias. ECG electrocardiogram, HS-cTn high-sensitivity cardiac troponin

- Myocardial infarction and AV block
  - Aminophylline 250 mg IV in bolus
- Spinal cord injury
  - Aminophylline 6 mg/kg in 100–200 mL of IV fluid over 20–30 minutes
  - Theophylline 5–10 mg/kg/day per oral titrated to effect
- Post-heart transplant
  - Aminophylline 6 mg/kg in 100–200 mL of IV fluid over 20–30 minutes
  - Theophylline 300 mg IV followed by an oral dose of 5–10 mg/kg/day titrated to effect

In patients with unstable bradycardia unresponsive to atropine, drug antidote, or specific treatment for specific clinical conditions, the next step is beta-agonist therapy with epinephrine or dopamine with dose titration based on stability and symptoms.

If the beta-agonist is not enough to solve the bradyarrhythmia and its symptoms, the last step in the acute treatment is pacing therapy.

The pacing therapy has different modalities [2, 3]:

- Transcutaneous pacing
  - Preferred in unstable patients with severe symptoms and not time for different pacing modalities
- Transvenous pacing
  - Preferred in unstable patients with moderate symptoms that can tolerate the procedure of implantation.
  - This modality is mandatory when the patient with transcutaneous pacing is stable and the symptoms resolved, but the pacing is still needed for an undetermined duration.
- Externalized permanent pacing lead
  - Used in patients with transvenous pacing that need prolonged pacing or permanent pacing, and the permanent pacemaker implantation is not feasible.
- Permanent pacemaker implantation
  - In patients with an irreversible cause of bradycardia and the clinical condition permit the implantation procedure

Specific recommendations in different clinical conditions of American and European guidelines are presented in Table 12.3.

12.13 Additional Clinical Practice Takeaway

- The atropine in the context of acute myocardial infarction must be used with caution because of the rise in the oxygen demand that atropine produces.
- The transcutaneous pacing is the treatment of choice in patients that the transvenous pacing delays the prompt therapy.
- Digoxin is not a drug that can be removed from the blood patient with hemodialysis.
- The further diagnostic approach with imagen studies is not indicated in the acute management in the ED.
- Atropine is the gold standard medical therapy in symptomatic bradyarrhythmia.
- High-degree AV block as chronic condition must be treated with permanent pacemaker independently of the presence of symptoms.
- Consider invasive or noninvasive mechanical ventilation in patients with bradyarrhythmia, respiratory distress, and severe hypoxemia (oxygen saturation < 90%).

Table 12.3 Current international guideline recommendations [3, 5]

European guidelines	COR	LOE
Persistent bradycardia		
Sinus node disease: pacing is indicated when symptoms can clearly be attributed to bradycardia	I	B
Sinus node disease: pacing may be indicated when symptoms are likely to be due to bradycardia, even if the evidence is not conclusive	IIb	C
Sinus node disease: pacing is not indicated in patients with sinus bradycardia, which is asymptomatic or due to reversible causes	III	C
Acquired AV block: pacing is indicated in patients with third- or second-degree type 2 ABV block irrespective of symptoms	I	C
Acquired AV block: pacing should be considered in patients with second-degree type 1 AV block which causes symptoms or is found to be located at intra- or infra-His levels at EPS	IIa	C
Acquired AV block: pacing is not indicated in patients with AV block which is due to reversible causes	III	C
Intermittent documented bradycardia		
Sinus node disease (including brady-tachy form): pacing is indicated in patients affected by sinus node disease who have the documentation of symptomatic bradycardia due to sinus arrest or sinus-atrial block	I	B
Intermittent/paroxysmal AV block (including AF with slow ventricular conduction): pacing is indicated in patients with intermittent/paroxysmal intrinsic third- or second-degree AV block	I	C
Reflex asystolic syncope: pacing should be considered in patients ≥40 years with syncopes and documented symptomatic pause/s due to sinus arrest or AV block or the combination of the two	IIa	B
Asymptomatic pauses (sinus arrest or AV block): pacing should be considered in patients with a history of syncope and documentation of asymptomatic pauses >6 s due to sinus arrest, sinus-atrial block or AV block	IIa	C
Pacing is not indicated in reversible causes of bradycardia	III	C
AHA/ACC guidelines		
In patients with newly identified LBBB, second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or third-degree atrioventricular block with or without apparent structural heart disease or coronary artery disease, transthoracic echocardiography is recommended	I	B-NR
In patients with documented or suspected bradycardia or conduction disorder during sleep, screening for symptoms of sleep apnea syndrome is recommended with subsequent confirmatory testing directed by clinical suspicion	I	B-NR
In patients with sleep-related bradycardia or conduction disorder and documented obstructive sleep apnea, treatment directed specifically at the sleep apnea (e.g., continuous positive airway pressure and weight loss) is recommended	I	B-NR
In patients with SND associated with symptoms or hemodynamic compromise, atropine is reasonable to increase sinus rate	IIa	C-LD
In patients with SND associated with symptoms or hemodynamic compromise who are at low likelihood of coronary ischemia, isoproterenol, dopamine, dobutamine, or epinephrine may be considered to increase heart rate and improve symptoms	IIb	C-LD
In patients who have undergone heart transplant without evidence for autonomic reinnervation, atropine should not be used to treat sinus bradycardia.	III	C-LD

Table 12.3 (continued)

European guidelines	COR	LOE
In patients with bradycardia associated with symptoms or hemodynamic compromise because of calcium channel blocker overdose, intravenous calcium is reasonable to increase heart rate and improve symptoms.	IIa	C-LD
In patients with bradycardia associated with symptoms or hemodynamic compromise because of beta-blocker or calcium channel blocker overdose, glucagon is reasonable to increase heart rate and improve symptoms	IIa	C-LD
In patients with bradycardia associated with symptoms or hemodynamic compromise because of beta-blocker or calcium channel blocker overdose, high-dose insulin therapy is reasonable to increase heart rate and improve symptoms	IIa	C-LD
In patients with bradycardia associated with symptoms or hemodynamic compromise in the setting of digoxin toxicity, digoxin Fab antibody fragment is reasonable to increase heart rate and improve symptoms	IIa	C-LD
In patients with bradycardia associated with symptoms or hemodynamic compromise attributable to digoxin toxicity, dialysis is not recommended for removal of digoxin	III	C-LD
In post-heart transplant patients, aminophylline or theophylline is reasonable to increase heart rate if clinically indicated	IIa	C-LD
In patients with SND associated with symptoms or hemodynamic compromise in the setting of acute spinal cord injury, aminophylline or theophylline is reasonable to increase heart rate and improve symptoms	IIa	C-LD
In patients with persistent hemodynamically unstable SND refractory to medical therapy, temporary transvenous pacing is reasonable to increase heart rate and improve symptoms until a PPM is placed or the bradycardia resolves	IIa	C-LD
In patients with SND with severe symptoms or hemodynamic compromise, temporary transcutaneous pacing may be considered to increase heart rate and improve symptoms until a temporary transvenous or PPM is placed or the bradycardia resolves	IIb	C-LD
In patients with symptoms that are directly attributable to SND, permanent pacing is indicated to increase heart rate and improve symptoms	I	C-LD
In patients who develop symptomatic sinus bradycardia as a consequence of guideline-directed management and therapy for which there is no alternative treatment and continued treatment is clinically necessary, permanent pacing is recommended to increase heart rate and improve symptoms	I	C-EO
For patients with tachy-brady syndrome and symptoms attributable to bradycardia, permanent pacing is reasonable to increase heart rate and reduce symptoms attributable to hypoperfusion	IIa	C-EO
Patients with transient or reversible causes of atrioventricular blocks, such as Lyme carditis or drug toxicity, should have medical therapy and supportive care, including temporary transvenous pacing if necessary, before determination of the need for permanent pacing	I	B-NR
In selected patients with symptomatic second-degree or third-degree atrioventricular block who are on chronic stable doses of medically necessary antiarrhythmic or beta-blocker therapy, it is reasonable to proceed to permanent pacing without further observation for drug washout or reversibility	IIa	B-NR

(continued)

Table 12.3 (continued)

European guidelines	COR	LOE
For patients with second-degree or third-degree atrioventricular block believed to be at the atrioventricular nodal level associated with symptoms or hemodynamic compromise, atropine is reasonable to improve atrioventricular conduction, increase ventricular rate, and improve symptoms	IIa	C-LD
For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise and who have a low likelihood for coronary ischemia, beta-adrenergic agonists, such as isoproterenol, dopamine, dobutamine, or epinephrine, may be considered to improve atrioventricular conduction, increase ventricular rate, and improve symptoms	IIb	B-NR
For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise in the setting of acute inferior MI, intravenous aminophylline may be considered to improve atrioventricular conduction, increase ventricular rate, and improve symptoms	IIb	C-LD
For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise that is refractory to medical therapy, temporary transvenous pacing is reasonable to increase heart rate and improve symptoms	IIa	B-NR
For patients who require prolonged temporary transvenous pacing, it is reasonable to choose an externalized permanent active fixation lead over a standard passive fixation temporary pacing lead	IIa	B-NR
For patients with second-degree or third-degree atrioventricular block and hemodynamic compromise refractory to antibradycardic medical therapy, temporary transcutaneous pacing may be considered until a temporary transvenous or PPM is placed or the bradyarrhythmia resolves	IIb	B-R
In patients with a first-degree atrioventricular block or second-degree Mobitz type I (Wenckebach) or 2:1 atrioventricular block which is believed to be at the level of the atrioventricular node, with symptoms that do not temporally correspond to the atrioventricular block, permanent pacing should not be performed	III	C-LD
In patients with symptomatic atrioventricular block attributable to a known reversible cause in whom the atrioventricular block does not resolve despite treatment of the underlying cause, permanent pacing is recommended	I	C-LD
In patients with acquired second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or third-degree atrioventricular block not attributable to reversible or physiologic causes, permanent pacing is recommended regardless of symptoms	I	B-NR
In patients with permanent AF and symptomatic bradycardia, permanent pacing is recommended	I	C-LD

COR class of recommendation, *LOE* level of evidence

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