Sinus bradycardia

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INTRODUCTION

Sinus bradycardia is a rhythm in which the rate of impulses arising from the sinoatrial (SA) node is lower than expected.

The normal adult heart rate, arising from the SA node, has been considered historically to range from 60 to 100 beats per minute, with sinus bradycardia being defined as a sinus rhythm with a rate below 60 beats per minute. The heart rate reflects a complex interplay between the sympathetic and parasympathetic nervous systems. It is affected by numerous factors, including age and physical conditioning (table 1) [1,2]. Sinus arrhythmia, the fluctuation in sinus rate with respiratory cycles, often accompanies sinus bradycardia. (See "Normal sinus rhythm and sinus arrhythmia".)

The etiology, clinical presentation, evaluation, and management of sinus bradycardia will be reviewed here. Primary sinus node dysfunction (ie, sinus node dysfunction) is discussed in detail separately. (See "Sinus node dysfunction: Epidemiology, etiology, and natural history" and "Sinus node dysfunction: Clinical manifestations, diagnosis, and evaluation" and "Sinus node dysfunction: Treatment".)

DEFINITION AND ECG FEATURES

Normal sinus rhythm (NSR) is the characteristic rhythm of the healthy heart. NSR is considered to be present in adults if the heart rate is between 60 and 100 beats per minute, the P wave vector on the electrocardiogram (ECG) is normal (ie, consistent with SA nodal impulse origin), and the rate is largely regular (waveform 1). The normal sinus P wave demonstrates right atrial followed by left atrial depolarization and high to low atrial activation giving rise to an upright P wave in leads I, II, and aVL, and a negative P wave in lead aVR.

By conventional definition, bradycardia indicates a heart rate less than 60 beats per minute with a normal P wave vector on the surface ECG. As such, sinus bradycardia is typically thought of as sinus rhythm occurring at a rate of less than 60 beats per minute, although one professional society has advocated a rate of less than 50 beats per minute (waveform 2) [3]. A rate less than 50 beats per minute may be a more pragmatic definition, as most patients with sinus rates in the 50s are asymptomatic. It is important to note that the rate at which a patient is labeled as having bradycardia is somewhat age dependent.

ETIOLOGY

Sinus bradycardia occurs in healthy patients as an adaptive response, particularly in well-conditioned persons or while sleeping, but it can also occur as a pathologic response in a variety of conditions.

It is very important to recognize that in normal healthy children and adults, sinus bradycardia is a frequent and normal finding, particularly during sleep when rates may transiently drop as low as 30 beats per minute, and pauses of up to two seconds are not uncommon [4-7]. Sinus bradycardia may also be seen in the absence of heart disease in the following settings:

- Well-conditioned athletes, particularly in endurance athletic activities, where bradycardia is generally attributed to increased vagal tone induced by exercise conditioning [8-12].
- Some older adult patients, although this may represent an early manifestation of sinus node dysfunction (SND) [13].
- Sinus bradycardia may represent a manifestation of rare familial forms of SND that have been described. Mutations in two genes, HCN4 and SCN5A, have been associated with sinoatrial (SA) node dysfunction as well as other hereditary arrhythmic disorders such as the Brugada syndrome, atrial fibrillation, and progressive cardiac conduction disease [14-16].

Sinus bradycardia can be seen in a variety of pathophysiologic settings (table 2), including:

- **Sinus node dysfunction** – Sinus bradycardia may be the first manifestation of SA node dysfunction [17,18]. Primary disease of the SA node is discussed in detail separately. (See "Sinus node dysfunction: Clinical manifestations, diagnosis, and evaluation".)

- **Medications** – A number of drugs can depress the SA node and slow the heart rate as either an expected response to or side effect of therapy, or as a toxicity related to overdose. These include:

  - Parasympathomimetic agents (eg, acetylcholine, carbachol, acetylcholinesterase inhibitors)
  - Sympatholytic drugs (eg, beta blockers, methyldopa, clonidine)
  - Opioids and sedatives
  - Cimetidine
  - Digitalis
  - Non-dihydropyridine calcium channel blockers (diltiazem and verapamil)
  - Ivabradine
  - Amiodarone and other antiarrhythmic drugs
  - The chronic hepatitis C drugs sofosbuvir and daclatasvir in patients receiving amiodarone [19]
  - Lithium
  - Chemotherapeutic agents (eg, thalidomide, lenalidomide, paclitaxel)
  - Organophosphate compounds [20]

A 2020 scientific statement from the American Heart Association details drugs associated with bradycardia [21].

- **Acute myocardial infarction** – Sinus bradycardia occurs in 15 to 25 percent of patients with acute myocardial infarction, particularly those involving the right coronary artery as it supplies the SA node in approximately 60 percent of people. Increased vagal activity is primarily responsible, and the bradycardia is typically transient. If treatment is necessary because of hemodynamic compromise or ischemia, sinus bradycardia usually responds well to intravenous atropine (0.6 to 1.0 mg in the majority of cases). (See "Supraventricular arrhythmias after myocardial infarction", section on 'Sinus bradycardia'.)

- **Obstructive sleep apnea** – Individuals with obstructive sleep apnea frequently have sinus bradycardia that may be severe (<30 beats per minute) during apneic episodes [22]. Therapies to improve the apnea frequently alleviate the bradycardia [23]. (See "Obstructive sleep apnea and cardiovascular disease in adults", section on 'Other arrhythmias'.)

- **Exaggerated vagal activity** – Vasovagal responses may be associated with a profound bradycardia due to heightened parasympathetic activity and sympathetic withdrawal on the SA node. There are a variety of stimuli for vagal activation, including carotid sinus stimulation, vomiting, coughing, and Valsalva maneuver. The combination of the slow heart rate and an associated decline in peripheral vascular resistance is often sufficient to produce presyncope or syncope. (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation".)

- **Increased intracranial pressure and other central nervous system conditions** — Increased intracranial pressure should be excluded when sinus bradycardia occurs in a patient with neurologic dysfunction. Stroke is another neurological condition that can display sinus bradycardia. Sinus bradycardia is also seen with trauma to the cervical or thoracic spine where sympathetic denervation of the heart leaves an unopposed parasympathetic tone [24]. (See "Evaluation and management of elevated intracranial pressure in adults", section on 'Clinical manifestations'.)

- **Infectious causes** – Infectious agents associated with relative sinus bradycardia include Lyme disease, Chagas disease, legionella, psittacosis, Q fever, typhoid fever, typhus, babesiosis, malaria, leptospirosis, yellow fever, dengue fever, viral hemorrhagic fevers, trichinosis, and Rocky Mountain Spotted fever [25,26].

- **Other causes** – Other causes of sinus bradycardia include hypothyroidism, anorexia nervosa, hypothermia, and severe prolonged hypoxia. Sinus bradycardia is also seen in the long QT syndrome and in the catecholaminergic polymorphic ventricular tachycardia syndrome, two forms of genetic channelopathies.

CLINICAL PRESENTATION

In the vast majority of patients, sinus bradycardia itself does not directly cause symptoms, although a patient with comorbid conditions that might be exacerbated by reduced cardiac output (eg, angina, heart failure) may present with worsening symptoms related to the comorbidity.

Symptoms related to the slow heart rate itself can occur, including lightheadedness, presyncope or syncope, worsening of angina pectoris or heart failure, cognitive slowing, and exercise intolerance. Symptoms may be subtle, with many patients noting only fatigue, which is frequently ascribed to aging rather than bradycardia. There is no specific heart rate below

which all patients develop symptoms, as cardiac output will vary depending upon the ability to increase stroke volume related to underlying conditioning or comorbidities.

Sinus bradycardia associated with pathology of the sinoatrial node is part of the sinus node dysfunction (SND). SND often does not respond appropriately to exercise (chronotropic incompetence), and fatigue or dyspnea on exertion may be the presenting feature. SND and chronotropic incompetence are discussed in detail separately. (See "Sinus node dysfunction: Epidemiology, etiology, and natural history" and "Prognostic features of stress testing in patients with known or suspected coronary disease", section on 'Heart rate response to exercise'.)

EVALUATION

Confirm sinus bradycardia — Sinus bradycardia is generally confirmed by ECG after a slow pulse is identified on physical examination, with the diagnosis usually being easy to establish from the surface ECG. An upright P wave in leads I, II, and aVL, and a negative P wave in lead aVR, indicates a sinus origin of the bradycardia. It is vital to exclude other causes of bradyarrhythmias such as atrioventricular (AV) block.

Differential diagnosis — Sinus bradycardia should be distinguished from other bradyarrhythmias (table 3) resulting in a reduced heart rate (ie, second- or third-degree AV block, junctional escape rhythm, ventricular escape rhythm). This is easily done by establishing the 1:1 relationship between P waves and QRS complexes on the surface ECG. Patients with more than one P wave for every QRS complex have second- or third-degree AV block, while patients with no discernible P waves will have an escape rhythm (either junctional or ventricular in origin).

The heart rate is often clinically assessed by detection of the pulse with a plethysmograph system, and conditions other than sinus bradycardia can cause a reduction in measured pulse rate (for example, ventricular bigeminy, in which the premature ventricular complex beats result in diminished pulse pressure, which may not be detected). In such cases of ineffective bigeminy, a falsely low heart rate can be recorded. Thus, it is important to always obtain an ECG when new or unexpected bradycardia is identified.

Further evaluation — For the majority of patients with sinus bradycardia, the underlying cause can usually be determined from history and physical examination alone. In addition to the measurement of a full set of vital signs, including temperature and pulse oximetry, important features to elicit in a history and examination include:

- Quantity of exercise performed and level of fitness
- Exposure to medications and toxins
- Signs of infectious exposure (eg, tick or insect bites) or other systemic conditions (eg, eating disorders, hypothyroidism)

The evaluation of a patient who presents with sinus bradycardia requires a comprehensive evaluation where the history is probably the most important component of the evaluation (algorithm 1). The aim of the initial evaluation is to establish the presence or absence of symptoms, and any evidence of hemodynamic compromise as a result of the bradycardia. This may include hypotension, chest discomfort, altered mental status, or shortness of breath. The presence of hemodynamic compromise demands immediate attention to the cause of the bradycardia and its amelioration.

Once hemodynamic compromise has been excluded, the clinician will have to exclude diseases, cardiovascular or other, associated with sinus bradycardia and, most importantly, drugs associated with sinus bradycardia (algorithm 1). The list of medications, cardiovascular and otherwise, that suppress the sinus node is extensive.

Symptoms may be subtle such as lightheadedness, unexplained falls, or exertional dyspnea due to chronotropic incompetence, or they may be pronounced such as syncope. These symptoms point to underlying sinus node disease, which may or may not be exacerbated by drugs that suppress the sinus node. Sinus bradycardia in a healthy, athletic individual requires no further evaluation or intervention. On the other hand, sinus bradycardia in an older individual may indicate sinus node dysfunction (SND) [27]. Sinus bradycardia may be associated with ischemic heart disease if the blood supply to the sinus node (right coronary artery or, in some patients, the left circumflex artery) is compromised. Patients with congestive heart failure have slower heart rates than their healthy counterparts [28]. Sinus bradycardia in an individual with a history of atrial fibrillation and/or conduction disease, especially if associated with a family history of sinoatrial node dysfunction, should raise the possibility of a hereditary form of SND [15].

MANAGEMENT

For asymptomatic patients with sinus bradycardia, treatment is neither indicated nor required. The following approach should be followed when symptoms occur (algorithm 1):

- For patients with symptoms who have evidence of hemodynamic instability, we administer atropine (1.0 mg intravenous [IV] push, which can be repeated every three to five minutes,

if needed, to a total dose of 3 mg) [29-31].

- If symptoms do not improve following atropine, proceed with temporary cardiac pacing and/or IV dopamine or epinephrine infusion [29-31]. (See "Temporary cardiac pacing" and "Advanced cardiac life support (ACLS) in adults", section on 'Bradycardia'.)

- If beta adrenergic or calcium channel blocker overdose is suspected, administer IV glucagon. The glucagon dose is 3 to 10 mg IV bolus given over three to five minutes; the bolus may repeat once if no response (increase in heart rate). If there is a response to bolus glucagon therapy, start a continuous infusion at 3 to 5 mg per hour titrated according to response [30].

- If symptoms improve, the patient should be monitored with continuous cardiac telemetry.

- For patients who are hemodynamically stable, the following should be considered:

  - Patients with signs or symptoms of acute myocardial ischemia or infarction should be treated accordingly. (See "Overview of the acute management of non-ST-elevation acute coronary syndromes" and "Overview of the acute management of ST-elevation myocardial infarction".)

  - Patients with evidence of another systemic condition associated with sinus bradycardia (eg, hypothyroidism, infection, etc) should be treated accordingly. (See "Treatment of primary hypothyroidism in adults" and "Lyme carditis".)

  - Patients in whom a medication is suspected to be causing the symptomatic bradycardia should have the medication withheld. If the medication is mandatory for the treatment of a comorbid condition (eg, beta blockers for severe angina), a permanent pacemaker may be required [30]. If the symptoms resolve and heart rate improves following the withdrawal of the suspected offending agent, no additional immediate treatment is required.

  - Patients with no other evidence of a potential cause should be evaluated for SND. (See "Sinus node dysfunction: Clinical manifestations, diagnosis, and evaluation".)

PROGNOSIS

There is no adverse prognostic significance to sinus bradycardia in otherwise healthy subjects. In subjects over the age of 40, for example, there is no adverse effect on longevity [32].

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Bradycardia (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Definition** – Sinus bradycardia, most commonly defined as sinus rhythm with a rate below 60 beats per minute (waveform 2), occurs in normal children and adults, particularly during sleep or at rest, in well-conditioned athletes, and in some older adult patients. Sinus bradycardia can also be seen in a variety of pathophysiologic settings, most commonly due to medication effects/toxicities or primary sinoatrial disease (table 2). (See 'Definition and ECG features' above and 'Etiology' above.)

- **Symptoms** – In the vast majority of patients, sinus bradycardia itself does not cause symptoms. However, a slow heart rate can cause symptoms such as fatigue, lightheadedness, and presyncope or syncope. Patients with comorbid conditions that might be exacerbated by reduced cardiac output (eg, angina, heart failure) may present with worsening symptoms related to the comorbidity. (See 'Clinical presentation' above.)

- **Evaluation** – The aim of the initial evaluation is to establish the presence or absence of symptoms, and any evidence of hemodynamic compromise as a result of the bradycardia (algorithm 1). Once hemodynamic compromise has been excluded, the clinician will have to exclude diseases, cardiovascular or other, associated with sinus bradycardia and, most importantly, drugs associated with sinus bradycardia. (See 'Further evaluation' above.)

- **Management** – For asymptomatic patients with sinus bradycardia, treatment is neither indicated nor required. The approach to the treatment of patients with symptomatic sinus bradycardia is presented within the text and the adjoining algorithm (algorithm 1). (See 'Management' above.)

- **Prognosis** – There is no adverse prognostic significance to asymptomatic sinus bradycardia in otherwise healthy subjects. (See 'Prognosis' above.)

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Topic 1075 Version 31.0

GRAPHICS

Normal heart rates in adults based on age and sex

Age (years)	HR (beats per minute)								
	All			Male			Female		
	N	Mean	1%-99%	N	Mean	1%-99%	N	Mean	1%-99%
20-29	6086	67	43-98	3127	64	42-99	2959	69	46-99
30-39	9569	69	46-100	4605	67	44-99	4964	70	48-100
40-49	15,392	69	46-101	7104	68	45-101	8288	70	48-102
50-59	18,578	68	46-102	9936	68	45-102	8642	69	47-102
60-69	16,585	67	44-102	9457	65	42-102	7128	68	46-101
70-79	8432	65	43-101	4509	64	42-102	3923	67	44-101
80-89	2259	65	44-101	1001	63	41-98	1258	67	46-102
90-99	119	70	43-146	58	64	43-95	81	72	44-147

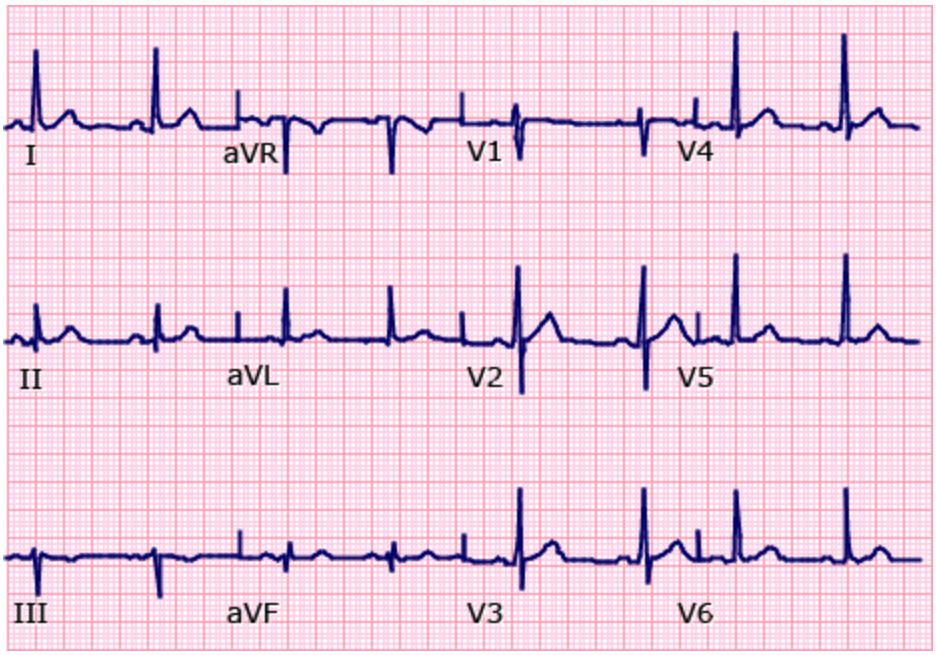
Normal heart rate values (with range from 1st to 99th percentile) for heart rate (beats/minute) in 77,276 healthy adults according to age and gender.

%: percent; HR: heart rate.

Data from: Mason JW, Ramseth DJ, Chanter DO, et al. Electrocardiographic reference ranges derived from 79,743 ambulatory subjects. J Electrocardiol 2007; 40:228.

Graphic 77746 Version 4.0

Normal ECG in sinus rhythm



Normal sinus rhythm at a rate of 71 beats/minute, a P wave axis of 45°, and a PR interval of 0.15 seconds.

ECG: electrocardiogram.

Courtesy of Morton Arnsdorf, MD.

Graphic 58149 Version 6.0

Single lead electrocardiogram (ECG) showing sinus bradycardia

Marked sinus bradycardia at a rate of 25 to 30 beats/min. The normal P waves (upright in lead II) and PR interval are consistent with a sinus mechanism with normal atrioventricular (AV) conduction.

Courtesy of Ary Goldberger, MD.

Graphic 52675 Version 4.0

Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Causes of bradycardia

Intrinsic	Extrinsic
Idiopathic degenerative disorder	Drugs
Ischemic heart disease	Antiarrhythmic agents
Chronic ischemia	Class IA - quinidine, procainamide
Acute myocardial infarction	Class IC - propafenone, flecainide
Hypertensive heart disease	Class II - β-blockers
Cardiomyopathy	Class III - sotalol, amiodarone, dronedarone
Trauma	Class IV - diltiazem, verapamil
Surgery for congenital heart disease	Cardiac glycosides
Heart transplant	Antihypertensive agents
Inflammation	Clonidine, reserpine, methyldopa
Collagen vascular disease	Antipsychotic agents
Rheumatic fever	Lithium, phenothiazines, amitriptyline
Pericarditis	Autonomically mediated
Infection	Vasovagal syncope (cardioinhibitory)
Viral myocarditis	Carotid sinus hypersensitivity
Lyme disease (Borrelia burgdorfer I)	Hypothyroidism
Neuromuscular disorder	Intracranial hypertension
Friedreich ataxia	Hypothermia
X-linked muscular dystrophy	Hyperkalemia
Familial disorder	Hypoxia
	Anorexia nervosa

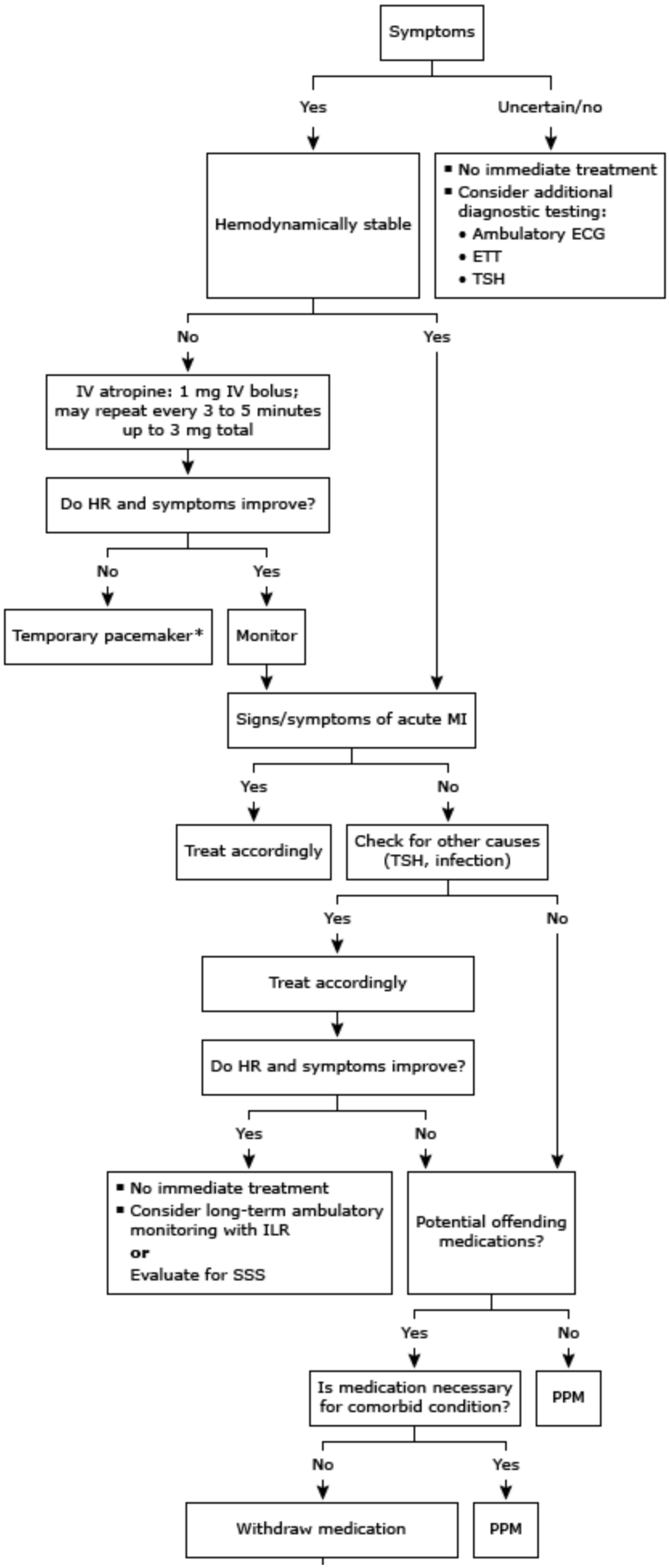
Graphic 65521 Version 11.0

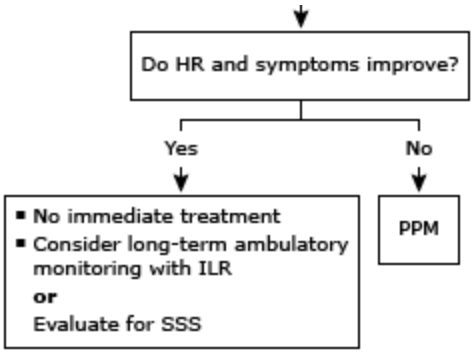
Major etiologies of bradyarrhythmias

Sinus bradycardia and its variants - including sinoatrial block
Atrioventricular heart block or dissociation - can occur with sinus rhythm or atrial fibrillation or flutter
Second or third degree AV block
Isorhythmic AV dissociation and related variants
Wandering atrial pacemaker
Junctional (AV nodal) escape rhythms - can occur with sinus rhythm or atrial fibrillation or flutter
Ventricular escape (idioventricular) rhythms

Graphic 50037 Version 1.0

Algorithm for the diagnosis and management of sinus bradycardia


Symptoms
Yes
Uncertain/no
Hemodynamically stable
No immediate treatment
Consider additional diagnostic testing:
Ambulatory ECG
ETT
TSH
No
Yes
IV atropine: 1 mg IV bolus; may repeat every 3 to 5 minutes up to 3 mg total
Do HR and symptoms improve?
No
Yes
Temporary pacemaker*
Monitor
Signs/symptoms of acute MI
Yes
No
Treat accordingly
Check for other causes (TSH, infection)
Yes
No
Treat accordingly
Do HR and symptoms improve?
Yes
No
No immediate treatment
Consider long-term ambulatory monitoring with ILR
or
Evaluate for SSS
Potential offending medications?
Yes
No
Is medication necessary for comorbid condition?
PPM
No
Yes
Withdraw medication
PPM



ECG: electrocardiogram; ETT: exercise tolerance test; TSH: thyroid stimulating hormone; IV: intravenous; MI: myocardial infarction; HR: heart rate; ILR: implantable loop recorder; SSS: sick sinus syndrome; PPM: permanent pacemaker.

* If atropine is ineffective, IV infusion of dopamine (5 to 20 mcg/kg/minute) or epinephrine (2 to 10 mcg/minute) may also be considered prior to temporary pacing.

Graphic 103718 Version 2.0

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