

2018 Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay

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2018 Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, and the Heart Rhythm Society

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The purpose of this ACC/AHA/HRS guideline is to provide guidance to clinicians for the management of patients with bradycardia, or symptoms thought to be associated with bradycardia or cardiac conduction system disorders. Although background on the pathophysiology and epidemiology of bradycardia and cardiac conduction disorders is summarized, this guideline is not intended to be an exhaustive review.

The following resource contains Figures and Tables from the 2018 Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay. The resource is only an excerpt from the Guideline and the full publication should be reviewed for more figures and tables as well as important context.

2018 Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay

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Top 10 Take-Home Messages (1 of 2)

1

Sinus node dysfunction is most often related to age-dependent progressive fibrosis of the sinus nodal tissue and surrounding atrial myocardium leading to abnormalities of sinus node and atrial impulse formation and propagation and will therefore result in various bradycardic or pause-related syndromes.

2

Both sleep disorders of breathing and nocturnal bradycardias are relatively common, and treatment of sleep apnea not only reduces the frequency of these arrhythmias but also may offer cardiovascular benefits. The presence of nocturnal bradycardias should prompt consideration for screening for sleep apnea, beginning with solicitation of suspicious symptoms. However, nocturnal bradycardia is not in itself an indication for permanent pacing.

3

The presence of left bundle branch block on electrocardiogram markedly increases the likelihood of underlying structural heart disease and of diagnosing left ventricular systolic dysfunction. Echocardiography is usually the most appropriate initial screening test for structural heart disease, including left ventricular systolic dysfunction.

4

In sinus node dysfunction, there is no established minimum heart rate or pause duration where permanent pacing is recommended. Establishing temporal correlation between symptoms and bradycardia is important when determining whether permanent pacing is needed.

5

In patients with acquired second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or third-degree atrioventricular block not caused by reversible or physiologic causes, permanent pacing is recommended regardless of symptoms. For all other types of atrioventricular block, in the absence of conditions associated with progressive atrioventricular conduction abnormalities, permanent pacing should generally be considered only in the presence of symptoms that correlate with atrioventricular block.

"Top Ten Messages" is continued in the next page.



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Top 10 Take-Home Messages (2 of 2)

6

In patients with a left ventricular ejection fraction between 36% to 50% and atrioventricular block, who have an indication for permanent pacing and are expected to require ventricular pacing >40% of the time, techniques that provide more physiologic ventricular activation (e.g., cardiac resynchronization therapy, His bundle pacing) are preferred to right ventricular pacing to prevent heart failure.

7

Because conduction system abnormalities are common after transcatheter aortic valve replacement, recommendations on postprocedure surveillance and pacemaker implantation are made in this guideline.

8

In patients with bradycardia who have indications for pacemaker implantation, shared decision-making and patient-centered care are endorsed and emphasized in this guideline. Treatment decisions are based on the best available evidence and on the patient's goals of care and preferences.

9

Using the principles of shared decision-making and informed consent/refusal, patients with decision-making capacity or his/her legally defined surrogate has the right to refuse or request withdrawal of pacemaker therapy, even if the patient is pacemaker dependent, which should be considered palliative, end-of-life care, and not physician-assisted suicide. However, any decision is complex, should involve all stakeholders, and will always be patient specific.

10

Identifying patient populations that will benefit the most from emerging pacing technologies (e.g., His bundle pacing, transcatheter leadless pacing systems) will require further investigation as these modalities are incorporated into clinical practice.



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Table of Definitions (1 of 3)

Table 3

Term	Definition or Description
Sinus Node Dysfunction (with accompanying symptoms)	<ul style="list-style-type: none"> • Sinus bradycardia: Sinus rate <50 bpm. • Ectopic atrial bradycardia: Atrial depolarization attributable to an atrial pacemaker other than the sinus node with a rate <50 bpm • Sinoatrial exit block: Evidence that blocked conduction between the sinus node and adjacent atrial tissue is present. Multiple electrocardiographic manifestations including “group beating” of atrial depolarization and sinus pauses. • Sinus pause: Sinus node depolarizes >3 s after the last atrial depolarization • Sinus node arrest: No evidence of sinus node depolarization • Tachycardia-bradycardia (“tachy-brady”) syndrome: Sinus bradycardia, ectopic atrial bradycardia, or sinus pause alternating with periods of abnormal atrial tachycardia, atrial flutter, or AF. The tachycardia may be associated with suppression of sinus node automaticity and a sinus pause of variable duration when the tachycardia terminates. • Chronotropic Incompetence: Broadly defined as the inability of the heart to increase its rate commensurate with increased activity or demand, in many studies translates to failure to attain 80% of expected heart rate reserve during exercise. • Isorhythmic dissociation: Atrial depolarization (from either the sinus node or ectopic atrial site) is slower than ventricular depolarization (from an atrioventricular nodal, His bundle, or ventricular site).
Atrioventricular Block	<ul style="list-style-type: none"> • <i>First-degree atrioventricular block:</i> P waves associated with 1:1 atrioventricular conduction and a PR interval >200 ms (this is more accurately defined as atrioventricular delay because no P waves are blocked) • <i>Second-degree atrioventricular block:</i> P waves with a constant rate (<100 bpm) where atrioventricular conduction is present but not 1:1 <ul style="list-style-type: none"> ◦ Mobitz type I: P waves with a constant rate (<100 bpm) with a periodic single nonconducted P wave associated with P waves before and after the nonconducted P wave with inconstant PR intervals ◦ Mobitz type II: P waves with a constant rate (< 100 bpm) with a periodic single nonconducted P wave associated with other P waves before and after the nonconducted P wave with constant PR intervals (excluding 2:1 atrioventricular block) ◦ 2:1 atrioventricular block: P waves with a constant rate (or near constant rate because of ventriculophasic sinus arrhythmia) rate (<100 bpm) where every other P wave conducts to the ventricles ◦ Advanced, high-grade or high-degree atrioventricular block: ≥2 consecutive P waves at a constant physiologic rate that do not conduct to the ventricles with evidence for some atrioventricular conduction

Atrioventricular Block (Table 3) will continue in the next page.



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Table of Definitions (2 of 3)

Table 3 (*continued*)

Term	Definition or Description
Atrioventricular Block (Continued)	<ul style="list-style-type: none"> • <i>Third-degree atrioventricular block (complete heart block)</i>: No evidence of atrioventricular conduction • <i>Vagally mediated atrioventricular block</i>: Any type of atrioventricular block mediated by heightened parasympathetic tone • <i>Infranodal block</i>: Atrioventricular conduction block where clinical evidence or electrophysiologic evidence suggests that the conduction block occurs distal to the atrioventricular node
Conduction Tissue Disease	<ul style="list-style-type: none"> • RBBB (as defined in adults): <ul style="list-style-type: none"> ◦ Complete RBBB: <ol style="list-style-type: none"> 1. QRS duration \geq120 ms 2. rsr', rsR', rSR', or rarely a qR in leads V₁ or V₂. The R' or r' deflection is usually wider than the initial R wave. In a minority of patients, a wide and often notched R wave pattern may be seen in lead V₁ and/or V₂. 3. S wave of greater duration than R wave or $>$40 ms in leads I and V₆ in adults. 4. Normal R peak time in leads V₅ and V₆ but $>$50 ms in lead V₁ ◦ Incomplete RBBB: Same QRS morphology criteria as complete RBBB but with a QRS duration between 110 and 119 ms. • LBBB (as defined in adults): <ul style="list-style-type: none"> ◦ Complete LBBB: <ol style="list-style-type: none"> 1. QRS duration \geq120 ms in adults. 2. Broad notched or slurred R wave in leads I, aVL, V₅, and V₆ and an occasional RS pattern in V₅ and V₆ attributed to displaced transition of QRS complex. 3. Absent q waves in leads I, V₅, and V₆, but in the lead aVL, a narrow Q wave may be present in the absence of myocardial pathology. 4. R peak $>$60 ms in leads V₅ and V₆ but normal in leads V₁, V₂, and V₃, when small initial R waves can be discerned in the precordial leads. 5. ST and T waves usually opposite in direction to QRS ◦ Incomplete LBBB: <ol style="list-style-type: none"> 1. QRS duration between 110 and 119 ms in adults 2. Presence of left ventricular hypertrophy pattern 3. R peak time $>$60 ms in leads V₄, V₅, and V₆ 4. Absence of Q wave in leads I, V₅, and V₆ • Nonspecific intraventricular conduction delay (as defined in adults): QRS duration $>$110 ms where morphology criteria for RBBB or LBBB are not present

Conduction Tissue Disease (Table 3) will continue in the next page.



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Table of Definitions (3 of 3)

Table 3 (continued)

Term	Definition or Description
Conduction Tissue Disease <i>(Continued)</i>	<ul style="list-style-type: none"> • Left anterior fascicular block <ul style="list-style-type: none"> ◦ QRS duration <120 ms. ◦ Frontal plane axis between -45° and -90° ◦ qR (small r, tall R) pattern in lead aVL ◦ R-peak time in lead aVL of ≥ 45 ms ◦ rS pattern (small r, deep S) in leads II, III, and aVF • Left posterior fascicular block <ul style="list-style-type: none"> ◦ QRS duration <120 ms ◦ Frontal plane axis between 90° and 180° in adults. Because of the more rightward axis in children up to 16 years of age, this criterion should only be applied to them when a distinct rightward change in axis is documented. ◦ rS (small r, deep S) pattern in leads I and aVL. ◦ qR (small q, tall R) pattern in leads III and aVF.

Maximum predicted heart rate for age calculated as 220 – age (y).

AF indicates atrial fibrillation; bpm, beats per minute; LBBB, left bundle branch block; and RBBB, right bundle branch block.



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Evaluation of Bradycardia and Conduction Disease Algorithm

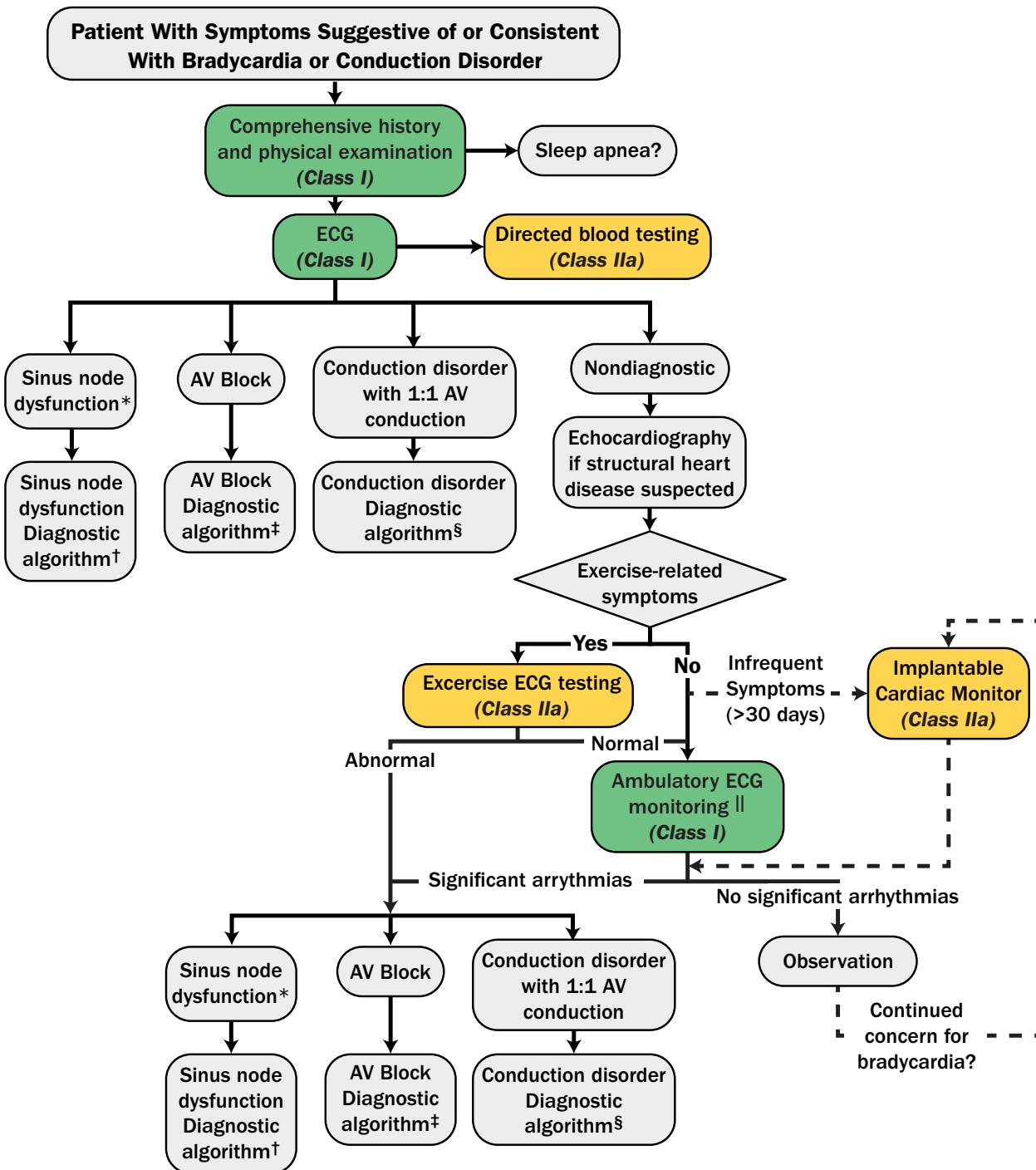


Figure 1

*Sinus bradycardia, ectopic atrial rhythm, junctional rhythm, sinus pause

[†]Refer to Figure 2 on page 11.

[†]Refer to Figure 3 on page 20.

§ Refer to Figure 8 on page 24

¹¹ Monitor choice based on the frequency of symptoms

AV indicates atrioventricular; and ECG, electrocardiogram.

Dashed lines indicate possible optional strategies based on the specific clinical situation.



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Common Potentially Reversible or Treatable Causes of Sinus Node Dysfunction

Table 7

Common Potentially Reversible or Treatable Causes of Sinus Node Dysfunction	
Acute myocardial ischemia or infarction	
Athletic training	
Atrial fibrillation	
Cardiac surgery	<ul style="list-style-type: none"> • Valve replacement, maze procedure, coronary artery bypass graft
Drugs or toxins*	<ul style="list-style-type: none"> • Toluene, organophosphates, tetrodotoxin, cocaine
Electrolyte abnormality	<ul style="list-style-type: none"> • Hyperkalemia, hypokalemia, hypoglycemia
Heart transplant: acute rejection, chronic rejection, remodeling	
Hypervagotonia	
Hypothermia	<ul style="list-style-type: none"> • Therapeutic (post-cardiac arrest cooling) or environmental exposure
Hypothyroidism	
Hypovolemic shock	
Hypoxemia, hypercarbia, acidosis	<ul style="list-style-type: none"> • Sleep apnea, respiratory insufficiency (suffocation, drowning, stroke, drug overdose)
Infection	<ul style="list-style-type: none"> • Lyme Disease, Legionella, psittacosis, Typhoid fever, Typhus, listeria, malaria, Leptospirosis, Dengue fever, viral hemorrhagic fevers, Guillain-Barre
Medications*	<ul style="list-style-type: none"> • Beta-blockers, non-dihydropyridine calcium channel blockers, digoxin, antiarrhythmic drugs, lithium, methyldopa, risperidone, cisplatin, interferon

*Partial list



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Initial Evaluation of Suspected or Documented Sinus Node Dysfunction Algorithm

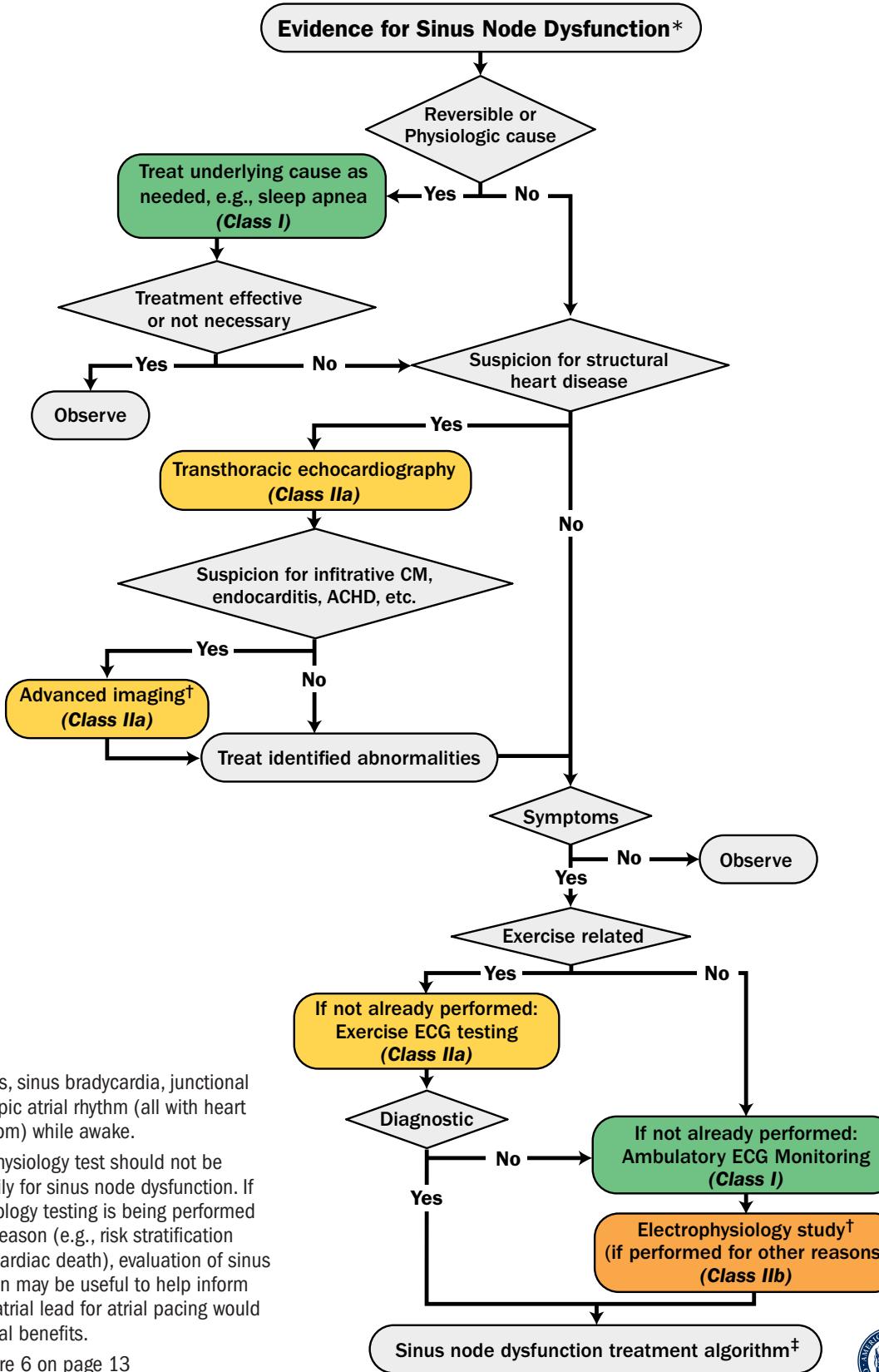


Figure 2

*Sinus pauses, sinus bradycardia, junctional rhythm, ectopic atrial rhythm (all with heart rates <50 bpm) while awake.

†The electrophysiology test should not be done primarily for sinus node dysfunction. If electrophysiology testing is being performed for another reason (e.g., risk stratification for sudden cardiac death), evaluation of sinus node function may be useful to help inform whether an atrial lead for atrial pacing would have potential benefits.

‡Refer to Figure 6 on page 13



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Acute Pacing Algorithm

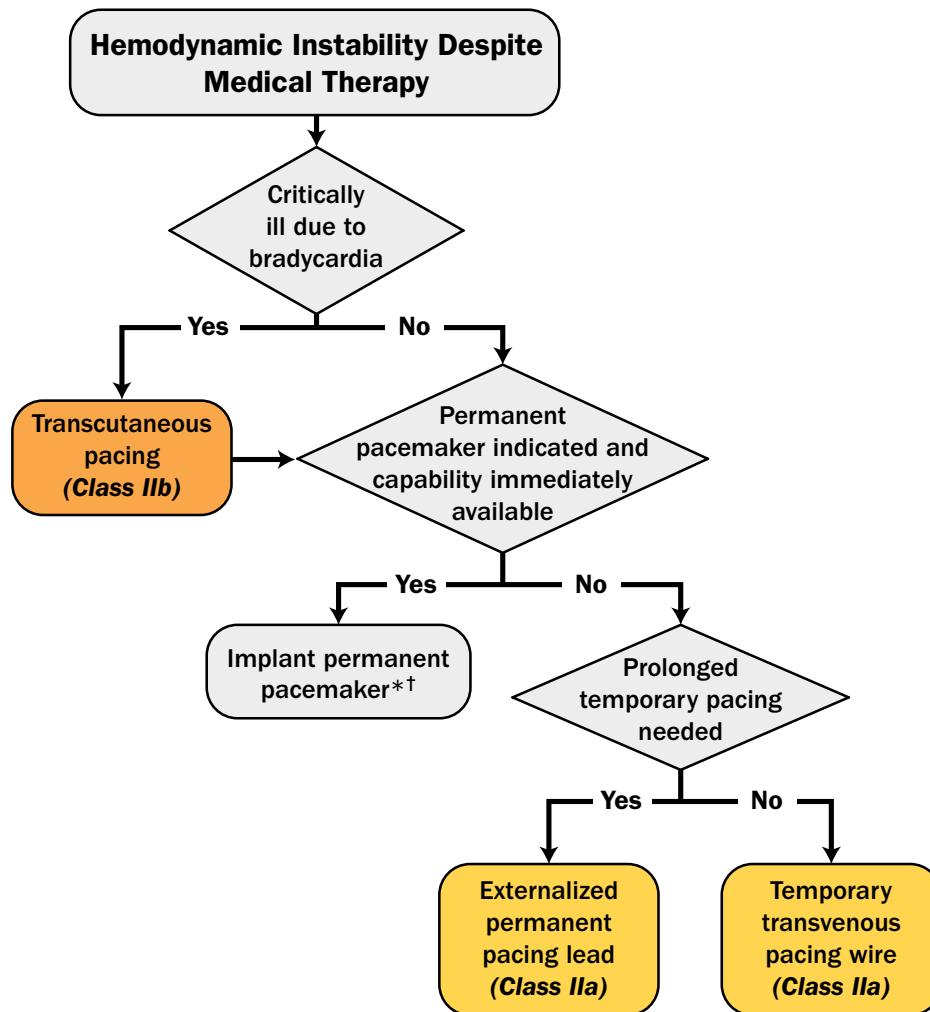


Figure 5

*Refer to Figure 6 on page 13 for Chronic SND and Figure 7 on page 23 for Chronic AV Block

†Careful management of anesthesia to avoid or minimize the use of drugs associated with bradycardia is required.



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Chronic SND Management Algorithm

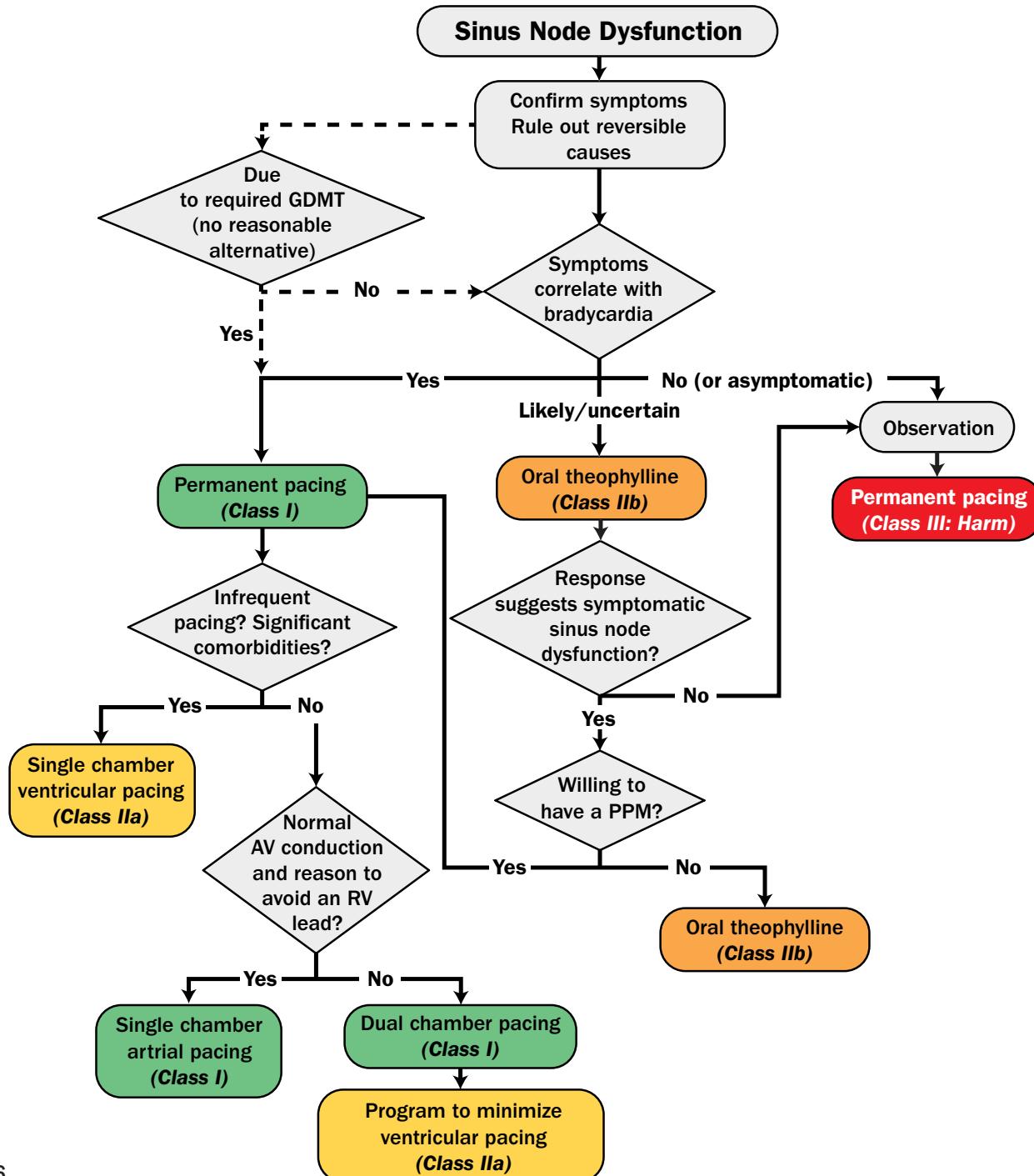


Figure 6

*Symptomatic patients with very infrequent need for pacing for rate support or patients with significant comorbidities.

AV indicates atrioventricular; GDMT, guideline-directed management and therapy; PPM, permanent pacemaker; and RV, right ventricular.

Dashed lines indicate possible optional strategies based on the specific clinical situation.

Recommendation for Sleep Apnea Evaluation and Treatment in Patients with Documented or Suspected Bradycardia or Conduction Disorders

COR	LOE	Recommendations
I	B-NR	1. 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	2. 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.
IIa	B-NR	3. In patients who have previously received or are being considered for a permanent pacemaker for bradycardia or conduction disorder, screening for sleep apnea syndrome is reasonable.



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Recommendations for Cardiac Imaging in Bradycardia or Conduction Disorders

COR	LOE	Recommendations
I	B-NR	1. In patients with newly identified left bundle branch block, second degree Mobitz type II AV block, high-grade AV block, or third degree AV Block with or without apparent structural heart disease or coronary artery disease, transthoracic echocardiography is recommended.
IIa	B-NR	2. In selected patients presenting with bradycardia or conduction disorders other than left bundle branch block, second degree Mobitz type II AV block, high-grade AV block, or third-degree AV Block, transthoracic echocardiography is reasonable if structural heart disease is suspected.
IIa	C-LD	3. In selected patients with bradycardia or bundle branch block, disease-specific advanced imaging (e.g., transesophageal echocardiography, computed tomography, cardiac magnetic resonance imaging, or nuclear imaging) is reasonable if structural heart disease is suspected yet not confirmed by other diagnostic modalities.
III: No Benefit	B-NR	4. In the evaluation of patients with asymptomatic sinus bradycardia or first degree AV block and no clinical evidence of structural heart disease, routine cardiac imaging is not indicated.

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Recommendations for General Principles of Chronic Therapy/Management of Bradycardia Attributable to Sinus Node Dysfunction

COR	LOE	Recommendations
III: Harm	C-LD	1. In asymptomatic individuals with sinus bradycardia or sinus pauses that are secondary to physiologically elevated parasympathetic tone, permanent pacing should not be performed.
III: Harm	C-LD	2. In patients with sleep-related sinus bradycardia or transient sinus pauses occurring during sleep, permanent pacing should not be performed unless other indications for pacing are present.
III: Harm	C-LD	3. In patients with asymptomatic sinus node dysfunction, or in those in whom the symptoms have been documented to occur in the absence of bradycardia or chronotropic incompetence, permanent pacing should not be performed.

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Medications that Can Induce/Exacerbate Bradycardia or Conduction Disorders

Table 4

Anti-hypertensive	Anti-arrhythmic	Psychoactive	Other
<ul style="list-style-type: none"> Beta Adrenergic Receptor Blockers (including beta adrenergic blocking eye drops used for glaucoma) Clonidine Methyldopa Non-dihydropyridine calcium channel blockers Reserpine 	<ul style="list-style-type: none"> Adenosine Amiodarone Dronedarone Flecainide Procainamide Propafenone Quinidine Sotalol 	<ul style="list-style-type: none"> Donepezil Lithium Opioid analgesics Phenothiazine antiemetics and antipsychotics Phenytoin Selective Serotonin Reuptake Inhibitors Tricyclic Antidepressants 	<ul style="list-style-type: none"> Anesthetic Drugs (propofol) Cannabis Digoxin Ivabradine Muscle relaxants (e.g. succinylcholine)



Conditions Associated with Bradycardia and Conduction Disorders

Table 5

Intrinsic	Extrinsic
Cardiomyopathy (ischemic or nonischemic)	Autonomic Perturbation
Congenital heart disease	<ul style="list-style-type: none"> • Carotid Sinus Hypersensitivity
Degenerative Fibrosis	<ul style="list-style-type: none"> • Neurally-Mediated Syncope /Presyncope
Infection/Inflammation	<ul style="list-style-type: none"> • Physical Conditioning
<ul style="list-style-type: none"> • Chagas Disease • Diphtheria • Infectious Endocarditis • Lyme Disease • Myocarditis • Sarcoidosis • Toxoplasmosis 	<ul style="list-style-type: none"> • Situational Syncope
Infiltrative Disorders	<ul style="list-style-type: none"> ◦ Cough ◦ Defecation ◦ Glottic stimulation ◦ Medical Procedures ◦ Micturition ◦ Vomiting
<ul style="list-style-type: none"> • Amyloidosis • Hemochromatosis • Lymphoma 	• Sleep (with or without sleep apnea)
Ischemia/infarction	Metabolic
Rheumatological Conditions	<ul style="list-style-type: none"> • Acidosis
<ul style="list-style-type: none"> • Rheumatoid Arthritis • Scleroderma • Systemic Lupus Erythematosus 	<ul style="list-style-type: none"> • Hyperkalemia • Hypokalemia • Hypothermia • Hypothyroidism • Hypoxia
Surgical or Procedural Trauma	
<ul style="list-style-type: none"> • Cardiac procedures such as ablation or cardiac catheterization • Congenital Heart Disease surgery • Septal myomectomy for hypertrophic obstructive cardiomyopathy • Valve Surgery (including percutaneous valve replacement) 	Adapted with permission from Mangrum JM, DiMarco JP. The evaluation and management of bradycardia. N Engl J Med. 2000;342: 703-9. Vogler J, Breithardt G, Eckardt L. Bradyarrhythmias and conduction blocks. Rev Esp Cardiol (Engl Ed). 2012;65:656-67.

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Acute Bradycardia Algorithm

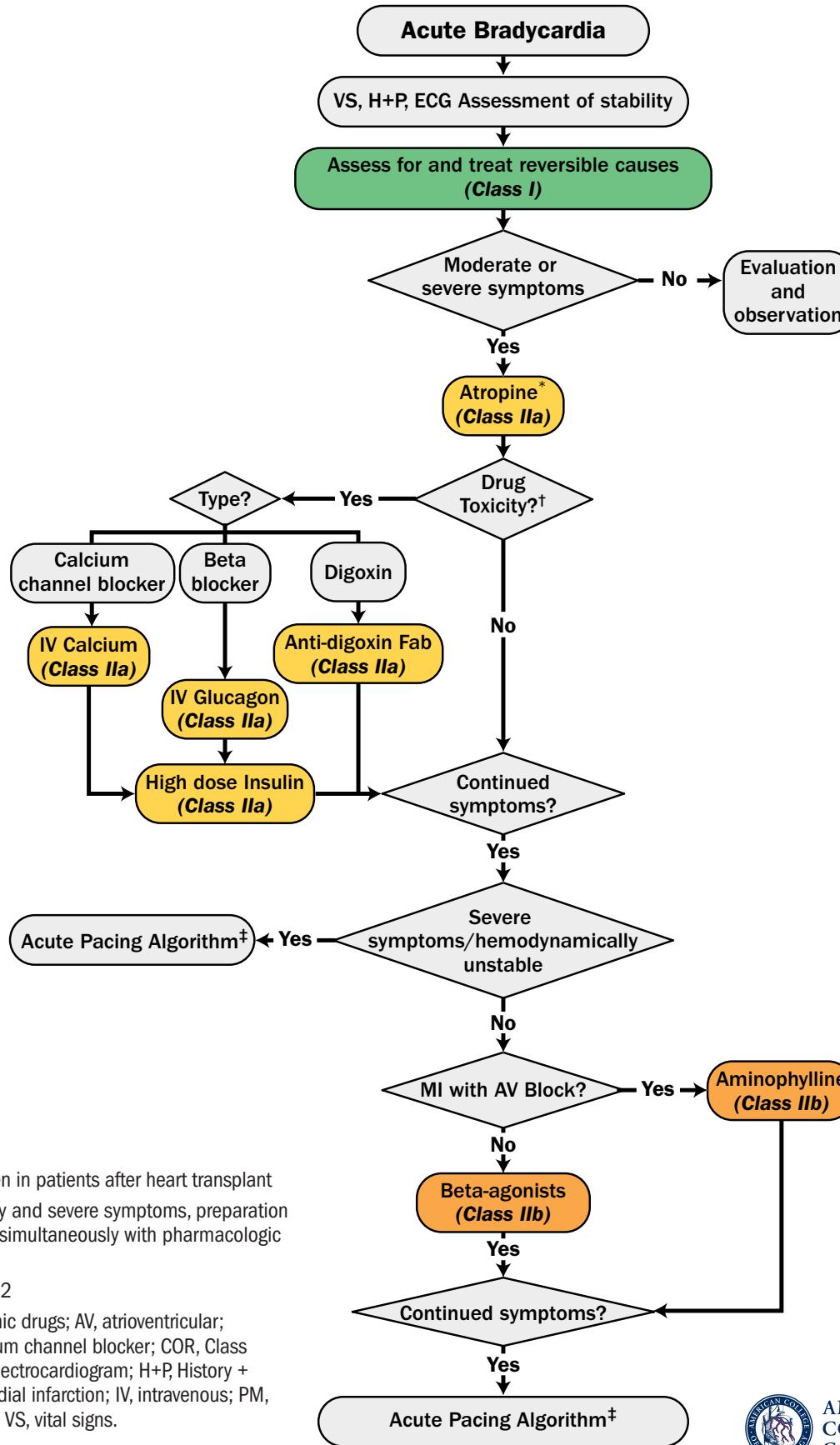


Figure 4

*Atropine should not be given in patients after heart transplant

†In patients with drug toxicity and severe symptoms, preparation for pacing should proceed simultaneously with pharmacologic treatment of drug toxicity

‡Refer to Figure 5 on page 12

AADs indicates anti-arrhythmic drugs; AV, atrioventricular; BB, beta Blocker; CCB, calcium channel blocker; COR, Class of Recommendation; ECG, electrocardiogram; H+P, History + Physical; IMI, inferior myocardial infarction; IV, intravenous; PM, pacemaker; S/P, status post; VS, vital signs.



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Initial Evaluation of Suspected AV Block Algorithm

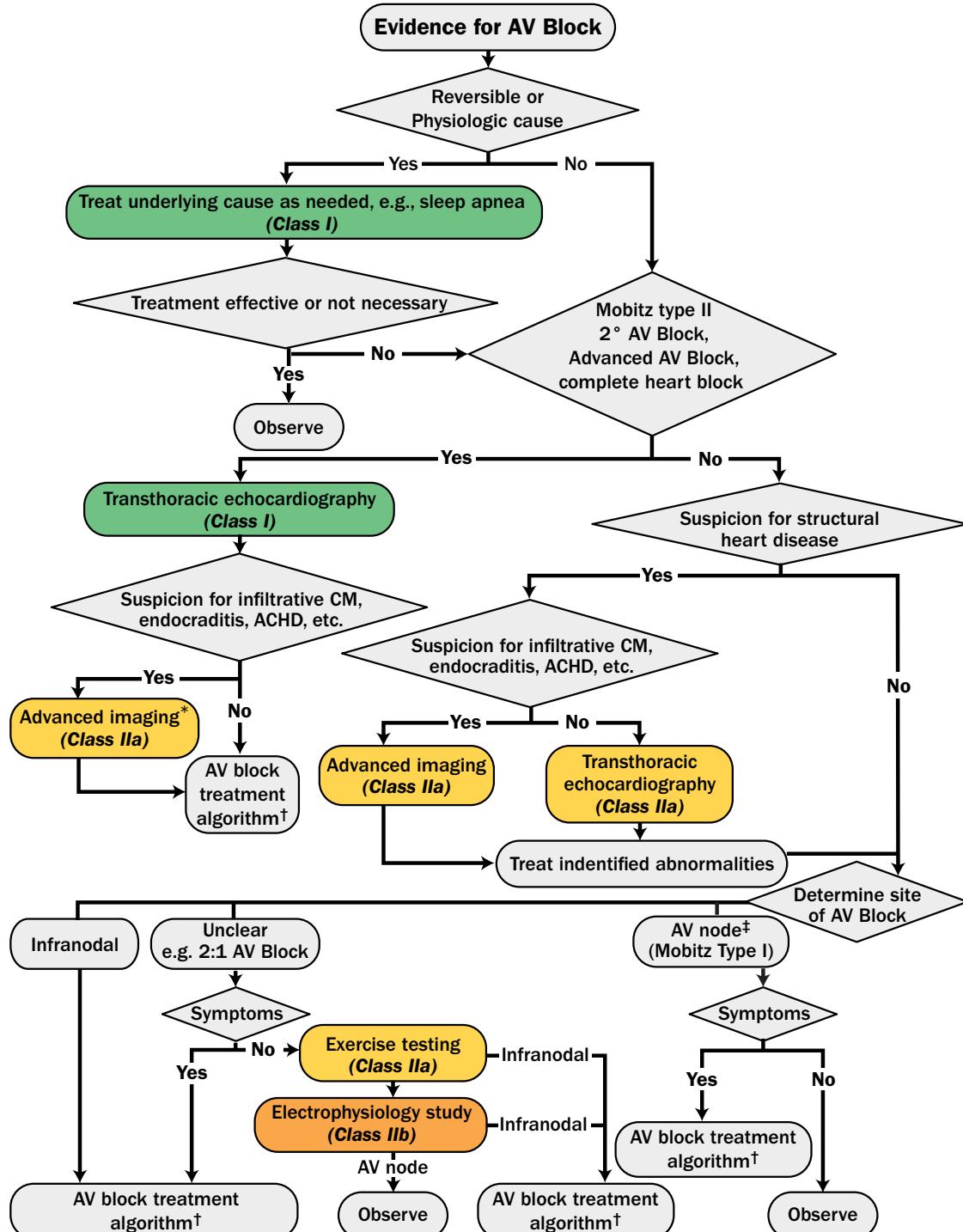


Figure 3

AV indicates atrioventricular; ACHD, adult congenital heart disease; and CM, cardiomyopathy.

*Targeted Advanced Imaging- Magnetic Resonance Imaging (MRI): Amyloidosis, myocarditis, hemochromatosis, sarcoidosis, CHD, sinus of Valsalva aneurysm, aortic dissection, Arrhythmogenic Right Ventricular Cardiomyopathy; Fluoro-Deoxy-Glucose (fludeoxyglucose)-Positron Emission Tomography (FDG PET): Sarcoidosis; 99m Technetium pyrophosphate (Tc PYP) or 99m Technetium 3,3-diphosphono-1,2-propanodicarboxylic acid (TC-DPD): Transthyretin (TTR) Amyloidosis; Cardiac Computed Tomography (CT): CHD, sinus of Valsalva aneurysm, aortic dissection, Arrhythmogenic Right Ventricular Cardiomyopathy; Echo longitudinal strain: Amyloidosis; Transesophageal Echocardiogram (TEE): Endocarditis, sinus of Valsalva aneurysm, aortic dissection, CHD

†Refer to Figure 7 on page 23

‡The AV node is more likely the site of block with 2nd degree Mobitz type I AV block and a narrow QRS complex or severe 1st degree AV block (>0.30s) with a narrow QRS complex



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Acute Medical Management of Bradycardia Attributable to SND or Atrioventricular Block

Table 8

Medication	Dosage	Comments
Symptomatic sinus bradycardia or AV block		
Atropine	0.5-1 mg IV (may be repeated every 3-5 minutes to a maximum dose of 3 mg)	
Dopamine	5 to 20 mcg/kg IV per minute, starting at 5 mcg/kg/min and increasing by 5 mcg/kg/minute every 2 minutes	Dosages of >20 mcg/kg per minute may result in vasoconstriction or arrhythmias
Isoproterenol	20-60 mcg IV bolus followed doses of 10-20 mcg, or infusion of 1-20 mcg/minute based on heart rate response	Monitor for potential development of ischemic chest pain
Epinephrine	2-10 mcg/minute IV or 0.1-0.5 mcg/kg/minute IV titrated to desired effect	
Second or Third Degree Atrioventricular Block Associated with Acute Inferior Myocardial Infarction		
Aminophylline	250 mg IV bolus	
Calcium Channel Blocker Overdose		
10% calcium chloride	1-2 gm IV every 10-20 minutes or an infusion of 0.2-0.4 mL/kg/hr	
10% calcium gluconate	3-6 gm IV every 10-20 minutes or an infusion at 0.6-1.2 mL/kg/hr	
Beta-blocker or Calcium Channel Blocker Overdose		
Glucagon	3-10 mg IV with infusion of 3-5 mg/hr	
High dose insulin therapy	IV bolus of 1 unit/kg followed by an infusion of 0.5 units/kg/hour.	Follow glucose and potassium levels
Digoxin overdose		
Digoxin Antibody Fragment	Dosage is dependent on amount ingested or known digoxin concentration	<ul style="list-style-type: none"> One vial binds approximately 0.5 mg of digoxin. Administer over at least 30 min. May be repeated
Post-heart Transplant		
Aminophylline	6 mg/kg in 100-200 mL of IV fluid over 20-30 minutes	
Theophylline	300 mg IV, followed by oral dose of 5-10 mg/kg per day titrated to effect	<ul style="list-style-type: none"> Therapeutic serum levels range from 10 to 20 mcg/mL Usual post-transplant dosages average 450 mg ± 100 mg a day
Spinal Cord Injury		
Aminophylline	6 mg/kg in 100-200 mL of IV fluid over 20-30 minutes	
Theophylline	Oral dose of 5-10 mg/kg per day titrated to effect	Effective dosages often result in serum levels below the usual effective range of 10-20 mcg/mL

IV indicates intravenous; and MI, myocardial infarction



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Etiology of Atrioventricular Block

Table 9

Congenital/Genetic
<ul style="list-style-type: none"> • Congenital AV block (associated with maternal systemic lupus erythematosus) • Congenital heart defects (e.g., L-TGA) • Genetic (e.g., SCN5A mutations)
Infectious
<ul style="list-style-type: none"> • Lyme carditis • Bacterial endocarditis with perivalvar abscess • Acute rheumatic fever • Chagas disease • Toxoplasmosis
Inflammatory/Infiltrative
<ul style="list-style-type: none"> • Myocarditis • Amyloidosis • Cardiac sarcoidosis • Rheumatologic disease: Systemic sclerosis, SLE, RA, reactive arthritis (Reiter's syndrome) • Other cardiomyopathy-idiopathic, valvular
Ischemic
<ul style="list-style-type: none"> • Acute MI • Coronary ischemia without infarction—unstable angina, variant angina • Chronic ischemic cardiomyopathy
Degenerative
<ul style="list-style-type: none"> • Lev's and Lenegre's diseases
Vagotonic-associated with Increased Vagal Tone
<ul style="list-style-type: none"> • Sleep, obstructive sleep apnea • High-level athletic conditioning • Neurocardiogenic
Metabolic/Endocrine
<ul style="list-style-type: none"> • Acid-base disorders • Poisoning/overdose (e.g., mercury, cyanide, carbon monoxide, mad honey) • Thyroid disease (both hypothyroidism and hyperthyroidism) • Adrenal disease (e.g., pheochromocytoma, hypoaldosteronism)
Other Diseases
<ul style="list-style-type: none"> • Neuromuscular diseases (e.g., myotonic dystrophy, Kearns-Sayre syndrome, Erb's dystrophy) • Lymphoma
Iatrogenic
<ul style="list-style-type: none"> • Medication related <ul style="list-style-type: none"> ◦ Beta blockers, verapamil, diltiazem, digoxin ◦ Antiarrhythmic drugs ◦ Neutraceuticals • Catheter ablation • Cardiac surgery, especially valve surgery • TAVR, alcohol septal ablation

RA indicates rheumatoid arthritis; MI, Myocardial Infarction; SLE, systemic lupus erythematosus; and TAVR, Transcatheter aortic valve replacement.



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Management of Bradycardia or Pauses due to Chronic AV Block Algorithm

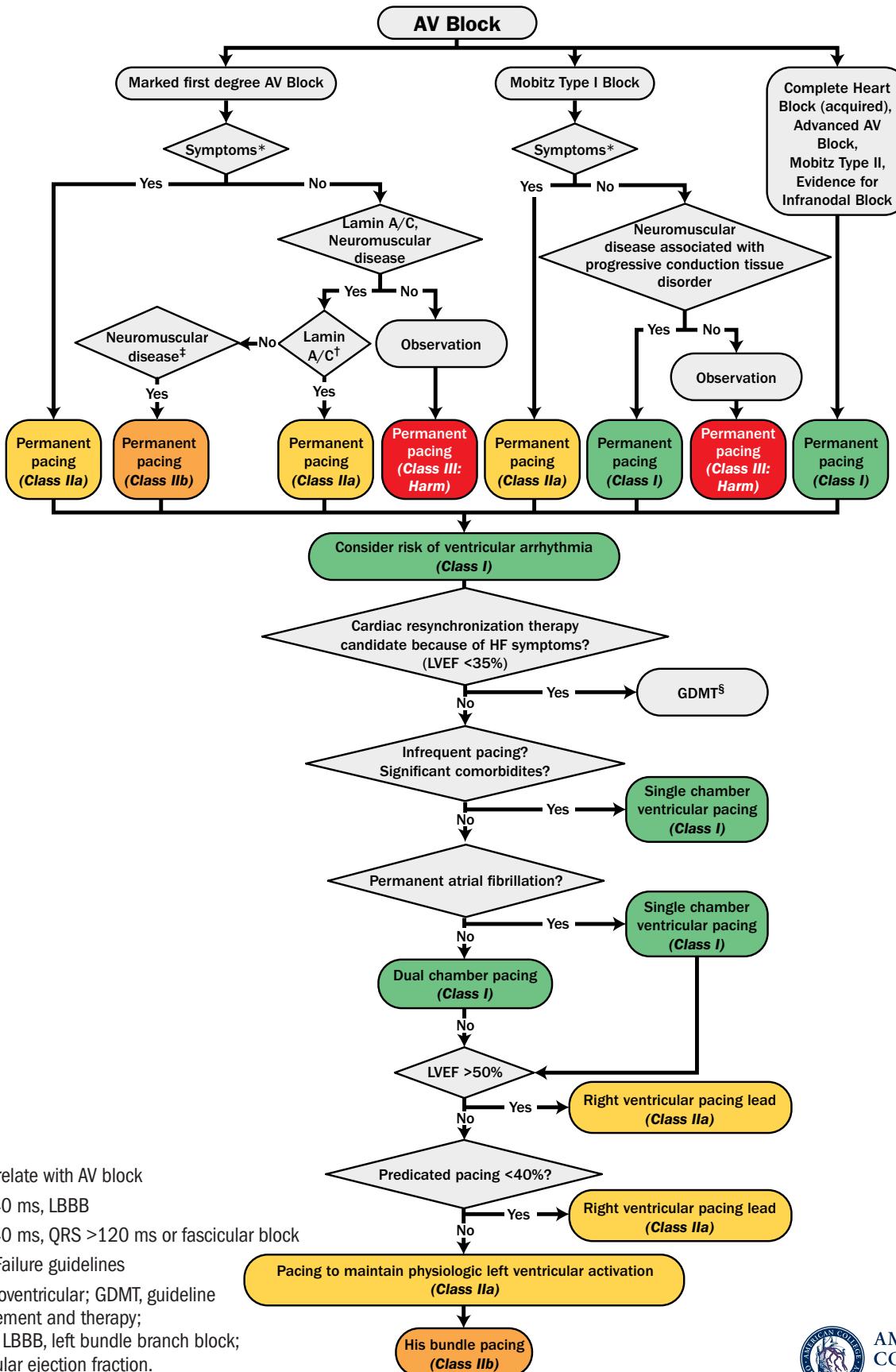


Figure 7

*Symptoms correlate with AV block

†PR interval >240 ms, LBBB

‡PR interval >240 ms, QRS >120 ms or fascicular block

\$Refer to Heart Failure guidelines

AV indicates atrioventricular; GDMT, guideline directed management and therapy;

HF, heart failure; LBBB, left bundle branch block;

LVEF, left ventricular ejection fraction.



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Evaluation of Conduction Disorders Algorithm

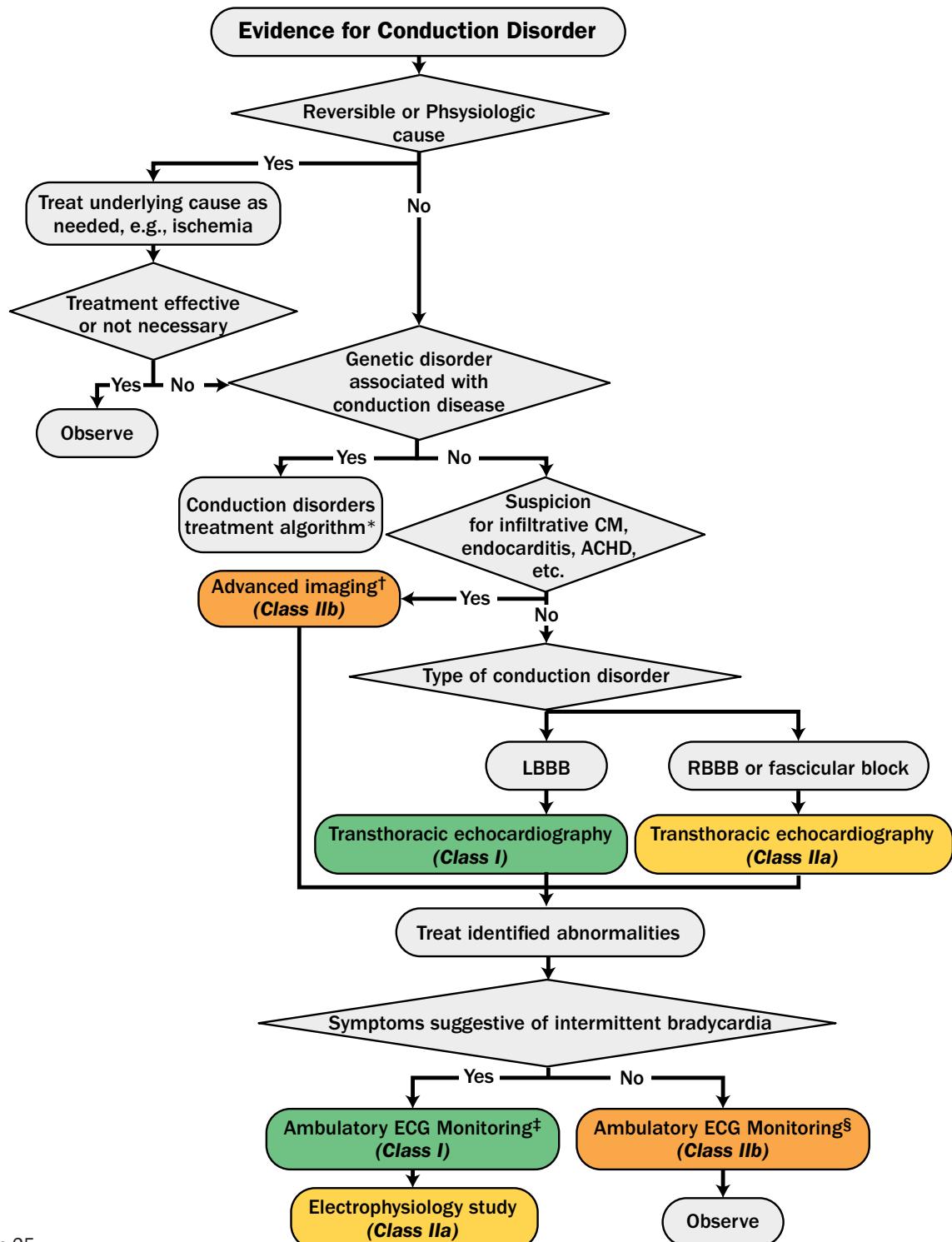


Figure 8

*Refer to Figure 9 on page 25

†Advanced imaging could include magnetic resonance imaging, computed tomography, or transesophageal echocardiography.

‡Monitor choice based on the frequency of symptoms.

§Extensive conduction disease (e.g. First degree AV block combined with LBBB)

ACHD indicates adult congenital heart disease; CM, cardiomyopathy; ECG, electrocardiogram; LBBB, left bundle branch block; and RBBB, right bundle branch block.



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Management of Conduction Disorders Algorithm

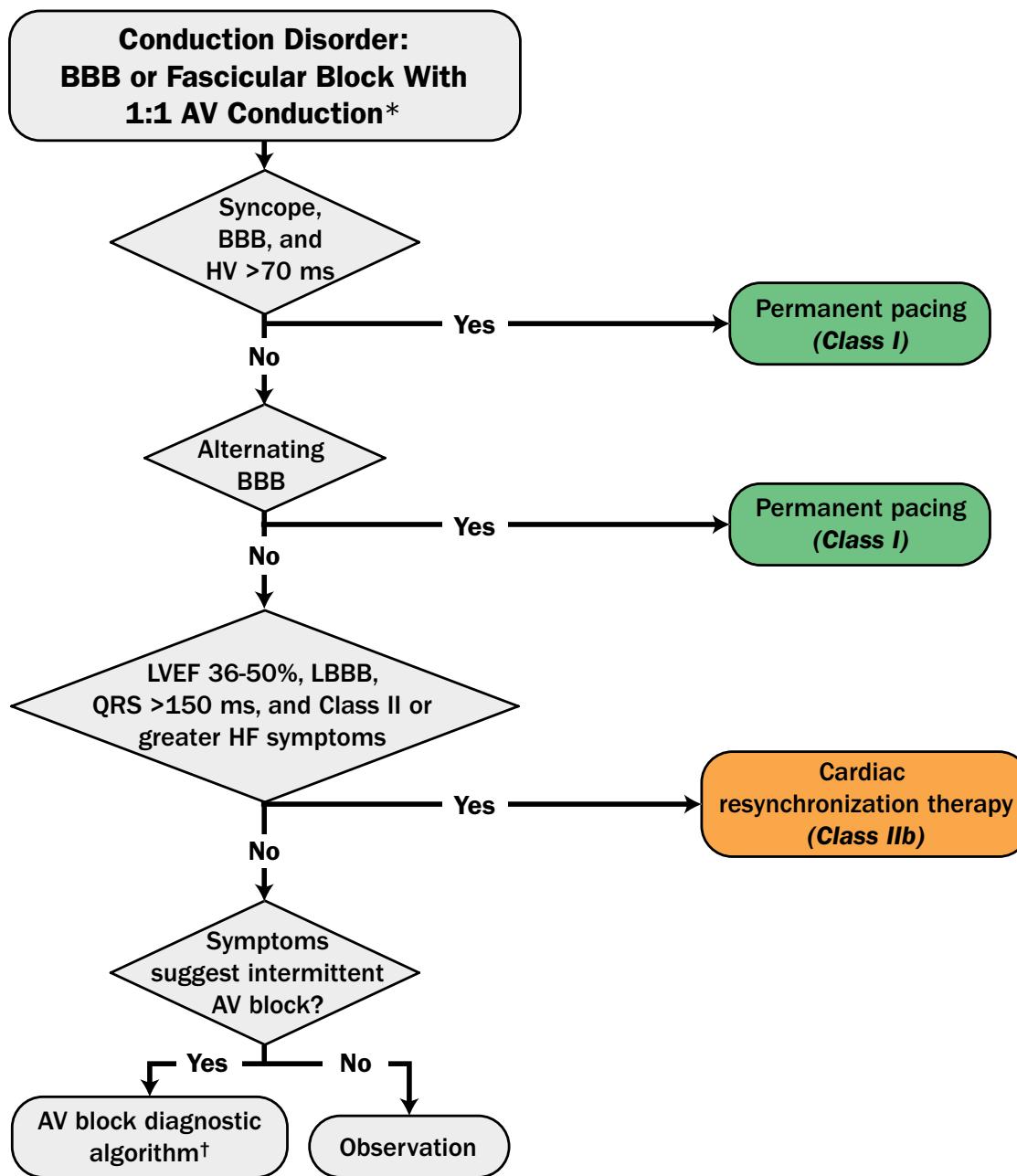


Figure 9

*For severe first degree AV block or first degree AV block with an accompanying neuromuscular disease, also refer to Figure 7 on page 23, the AV block algorithm.

†See Figure 3 on page 20

AV indicates atrioventricular; BBB, bundle branch block; HF, heart failure; LBBB, left bundle branch block; and LVEF, left ventricular ejection fraction.



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Recommendations for Conduction Disturbances after Transcatheter Aortic Valve Replacement

COR	LOE	Recommendations
I	B-NR	1. In patients who have new atrioventricular block after transcatheter aortic valve replacement associated with symptoms or hemodynamic instability that does not resolve, permanent pacing is recommended before discharge.
IIa	B-NR	2. In patients with new persistent bundle branch block after transcatheter aortic valve replacement, careful surveillance for bradycardia is reasonable.
IIb	B-NR	3. In patients with new persistent left bundle branch block after transcatheter aortic valve replacement, implantation of a permanent pacemaker may be considered.

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Recommendations for Shared Decision Making for Pacemaker Implantation in the Setting of Guideline-Based Indications for Bradycardia Pacing

COR	LOE	Recommendations
I	C-LD	1. In patients with symptomatic bradycardia or conduction disorder, clinicians and patients should engage in a shared decision making approach in which treatment decisions are based not only on the best available evidence, but also on the patient's goals of care, preferences, and values.
I	C-LD	2. Patients considering implantation of a pacemaker or with a pacemaker that requires lead revision or generator change should be informed of procedural benefits and risks, including the potential short and long-term complications and possible alternative therapy, if any, in light of their goals of care, preferences, and values.
III: No Benefit	C-LD	3. In patients with indications for permanent pacing but also with significant co-morbidities such that pacing therapy is unlikely to provide meaningful clinical benefit, or if patient goals of care strongly preclude pacemaker therapy, implantation or replacement of a pacemaker should not be performed.

