2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay: Executive Summary

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2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay: Executive Summary

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

Developed in Collaboration With the American Association for Thoracic Surgery, the Pediatric & Congenital Electrophysiology Society, and the Society of Thoracic Surgeons

Endorsed by the American Association for Thoracic Surgery, the Pediatric & Congenital Electrophysiology Society, and the Society of Thoracic Surgeons

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Top 10 Take-Home Messages

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.
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.

Preamble

Since 1980, the American College of Cardiology (ACC) and American Heart Association (AHA) have translated scientific evidence into clinical practice guidelines with recommendations to improve cardiovascular health. These guidelines, which are based on systematic methods to evaluate and classify evidence, provide a foundation for the delivery of quality cardiovascular care. The ACC and AHA sponsor the development and publication of clinical practice guidelines without commercial support, and members volunteer their time to the writing and review efforts.

Clinical practice guidelines provide recommendations applicable to patients with or at risk of developing cardiovascular disease. The focus is on medical practice in the United States, but these guidelines are relevant to patients throughout the world. Although guidelines may be used to inform regulatory or payer decisions, the intent is to improve quality of care and align with patients' interests. Guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances, and should not replace clinical judgment.

Recommendations for guideline-directed management and therapy, which encompasses clinical evaluation, diagnostic testing, and both pharmacological and procedural treatments, are effective only when followed by both practitioners and patients. Adherence to recommendations can be enhanced by shared decision-making between clinicians and patients, with patient engagement in selecting interventions on the basis of individual values, preferences, and associated conditions and comorbidities.

The ACC/AHA Task Force on Clinical Practice Guidelines strives to ensure that the guideline writing committee both contains requisite expertise and is representative of the broader medical community by selecting experts from a broad array of backgrounds representing different geographic regions, sexes, races, ethnicities, intellectual perspectives/biases, and scopes of clinical practice, and by inviting organizations and professional societies with related interests and expertise to participate as partners or collaborators. The ACC and AHA have rigorous policies and methods to ensure that documents are developed without bias or improper influence. The complete policy on relationships with industry and other entities (RWI) can be found at http://www.acc.org/guidelines/about-guidelines-and-clinical-documents/relationships-with-industry-policy.

Beginning in 2017, numerous modifications to the guidelines have been and continue to be implemented to make guidelines shorter and enhance "user friendliness." Guidelines are written and presented in a modular knowledge chunk format, in which each chunk includes a table of recommendations, a brief synopsis, recommendation-specific supportive text and, when appropriate, flow diagrams or additional tables. Hyperlinked references are provided for each modular knowledge chunk to facilitate quick access and review. More structured guidelines—including word limits ("targets") and a web guideline supplement for useful but noncritical tables and figures—are 2 such changes. This Preamble is an abbreviated version, with the detailed version available at: http://jaccjacc.acc.org/Clinical_Document/Bradycardia_GL_Web_Supplement.pdf.

The reader is encouraged to consult the full-text guideline (P-1) for additional guidance and details about bradycardia and cardiac conduction delay, because the executive summary contains mainly the recommendations.

Glenn N. Levine, MD, FACC, FAHA
Chair, ACC/AHA Task Force on Clinical Practice Guidelines

1. Introduction

1.1. Methodology and Evidence Review

The recommendations listed in this guideline are, whenever possible, evidence based. An initial extensive evidence review, which included literature derived from research involving human subjects, published in English, and indexed in MEDLINE (through PubMed), EMBASE, the Cochrane Library, the Agency for Healthcare Research and Quality, and other selected databases relevant to this guideline, was conducted from January 2017 to September 2017. Key search words included but were not limited to the following: *AV block, bradycardia, bundle branch block, conduction disturbance, left bundle branch block, loop recorder, pauses, permanent pacemaker, sick sinus syndrome, sinus node dysfunction,* and *temporary pacemaker*. Additional relevant studies published through January 2018, during the guideline writing process, were also considered by the writing committee and added to the evidence tables when appropriate. The final evidence tables are included in the Online Data Supplement (http://jaccjacc.acc.org/Clinical_Document/Bradycardia_GL_Online_Data_Supplement.pdf) and summarize the evidence used by the writing committee to formulate recommendations. References selected and published in the present document are representative and not all-inclusive.

As noted in the detailed version of the Preamble, an independent evidence review committee was commissioned to perform a formal systematic review of 1 critical clinical question related to bradycardia, the results of which were considered by the writing committee for incorporation into this guideline. Concurrent with this process, writing committee members evaluated study data relevant to the rest of the guideline. The findings of the evidence review committee and the writing committee members were formally presented and discussed, and then recommendations were developed. The systematic review, titled "Impact of Physiologic Versus Right Ventricular Pacing Among Patients With Left Ventricular Ejection Fraction Greater Than 35%: A Systematic Review for the 2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay" is published in conjunction with this guideline (S1-1) and its respective data supplements are available online (http://jaccjacc.acc.org/Clinical_Document/Bradycardia_Systematic_Review_Online_Data_Supplement.pdf). The evidence review committee report informed recommendations in Section 6.4.4.1.

1.2. Organization of the Writing Committee

The writing committee consisted of cardiac electrophysiologists, clinicians, cardiologists, surgeons, an anesthesiologist, and a lay/patient representative. The writing committee included representatives from the ACC, AHA, Heart Rhythm Society (HRS), American Association for Thoracic Surgery (AATS), Pediatric & Congenital Electrophysiology Society (PACES), and the Society of Thoracic Surgeons (STS).). Appendix 1 of the present document lists writing committee members' relevant RWI. For the purposes of full transparency, the writing committee members' comprehensive disclosure information is available online (http://jaccjacc.acc.org/Clinical_Document/Bradycardia_GL_Comprehensive_Author_RWI.pdf).

1.3. Document Review and Approval

This document was reviewed by 2 official reviewers each nominated by the ACC, AHA, and HRS; 1 official lay reviewer nominated by the AHA; 1 organizational reviewer each from the AATS, PACES, and STS; and 31 individual content reviewers. Reviewers' RWI information was distributed to the writing committee and is published as an abbreviated table in this document (Appendix 2). The reviewers' detailed RWI information is available online (http://jaccjacc.acc.org/Clinical_Document/Bradycardia_GL_Comprehensive_Reviewer_RWI.pdf).

This document was approved for publication by the governing bodies of the ACC, the AHA, and the HRS; and was endorsed by the American Association for Thoracic Surgery, the Pediatric & Congenital Electrophysiology Society, and the Society of Thoracic Surgeons.

1.4. Scope of the Guideline

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. This guideline supersedes the pacemaker recommendations made in the "ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities" (S1.4-1, S1.4-2) and "2012 ACCF/AHA/HRS Focused Update Incorporated Into the ACCG/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities (S1.4-2). The guideline will be useful to general internists, family physicians, emergency physicians, anesthesiologists, surgeons, cardiologists, and arrhythmia specialists. This document is aimed at the adult population (>18 years of age) and offers no specific recommendations in pediatric patients, although some of the evidence review included pediatric patients. 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. Rather, it focuses on practical clinical evaluation and management. Specific objectives and goals include:

- Describe the clinical significance of bradycardia with respect to mortality, symptoms (e.g., syncope, impaired functional capacity), and exacerbations of associated disorders (e.g., ischemia, heart failure, provoked tachyarrhythmias).
- Address inherited and acquired disorders of the sinus node, atrioventricular node, His-Purkinje fibers, and intramyocardial conducting tissue, including the effects of medications, aging, metabolic derangements, trauma, radiation, infiltrative, ischemic, and inflammatory disorders, infectious and toxic agents and iatrogenic factors.
- Delineate the clinical presentation and general approach to clinical evaluation of patients with overt or suspected bradycardias or conduction diseases.
- Comprehensively evaluate the evidence supporting recommendations for the selection and timing of available diagnostic testing modalities, including monitoring devices and electrophysiologic testing.
- Define the evidence base supporting recommendations for the use of available treatment modalities, including lifestyle interventions, pharmacotherapy and external and implanted device-based therapies, with particular attention to indications for temporary and permanent pacing.
- Address special considerations that may be applicable to distinct populations based on age (>18 years of age), comorbidities or other relevant factors.
- Identify knowledge gaps, pertinent trials in progress and directions for future research.

Table 1 lists other guidelines and pertinent documents that the writing committee considered for this guideline. The listed documents contain relevant information for the management of patients with bradycardia or cardiac conduction system disorder.

Table 1. Associated Guidelines and Related References

Title	Organization	Publication Year (Reference)
Guidelines		
Ventricular arrhythmias and sudden cardiac death	ACC/AHA/HRS	2017 (S1.4-3)
Syncope	ACC/AHA/HRS	2017 (S1.4-4)
Stable ischemic heart disease	ACC/AHA/AATS/PCNA/S	2014* (S1.4-5)

	CAI/STS	2012 (S1.4-6)
Atrial fibrillation	AHA/ACC/HRS	2014 (S1.4-7)
Perioperative cardiovascular evaluation and management of patients undergoing non-cardiac surgery	ACC/AHA	2014 (S1.4-8)
Non–ST-elevation acute coronary syndromes	AHA/ACC	2014 (S1.4-9)
Heart failure	ACC/AHA	2013 (S1.4-10)
ST-elevation myocardial infarction	ACC/AHA	2013 (S1.4-11)
Device-based therapy for cardiac rhythm abnormalities	ACC/AHA/HRS	2013 (S1.4-2)
Coronary artery bypass graft surgery	ACC/AHA	2011 (S1.4-12)
Hypertrophic cardiomyopathy	ACC/AHA	2011 (S1.4-13)
Percutaneous coronary intervention	ACC/AHA/SCAI	2011 (S1.4-14)
Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care—Part 9: post-cardiac arrest care	AHA	2010 (S1.4-15)
Other related references		
Expert consensus statement on cardiovascular implantable electronic device lead management and extraction	HRS	2017 (S1.4-16)
Management of cardiac involvement associated with neuromuscular diseases	AHA	2017 (S1.4-17)
Expert consensus statement on magnetic resonance imaging	HRS	2017 (S1.4-18)
Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Task Force 9: arrhythmias and conduction defects	ACC/AHA	2015 (S1.4-19)
Expert consensus statement on the diagnosis and treatment of postural tachycardia syndrome, inappropriate sinus tachycardia, and vasovagal syncope	HRS	2015 (S1.4-20)
Expert consensus statement on the recognition and management of arrhythmias in adult congenital heart disease	PACES/HRS	2014 (S1.4-21)
Expert consensus statement on the use of implantable cardioverter-defibrillator therapy in patients who are not included or not well represented in clinical trials	HRS/ACC/AHA	2014 (S1.4-22)
Expert consensus statement on the diagnosis and management of arrhythmias associated with cardiac sarcoidosis	HRS	2014 (S1.4-23)
Cardiac pacing and cardiac resynchronization therapy	ESC	2013 (S1.4-24)
Expert consensus statement on pacemaker device and mode selection	HRS/ACCF	2012 (S1.4-25)
Expert consensus statement on the state of genetic testing for the channelopathies and cardiomyopathies	HRS/EHRA	2011 (S1.4-26)
Expert consensus statement on the management of cardiovascular implantable electronic devices (CIEDs) in patients nearing end of life or requesting withdrawal of therapy	HRS	2010 (S1.4-27)
Recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement	AHA/ACCF/HRS	2009 (S1.4-28)
Recommendations for the standardization and interpretation of the electrocardiogram: part V: electrocardiogram changes associated with cardiac chamber hypertrophy: a scientific statement	AHA/ACCF/HRS	2009 (S1.4-29)

*Focused Update.

AATS indicates American Association for Thoracic Surgery; ACC, American College of Cardiology; ACCF, American College of Cardiology Foundation; AHA, American Heart Association; EHRA, European Heart Rhythm Association; ESC, European Society of Cardiology; HRS, Heart Rhythm Society; PACES, Pediatric & Congenital Electrophysiology Society; PCNA, Preventive Cardiovascular Nurses Association; SCAI, Society for Cardiovascular Angiography and Interventions; and STS, Society of Thoracic Surgeons.

1.5. Class of Recommendation and Level of Evidence

Recommendations are designated with both a class of recommendation (COR) and a level of evidence (LOE). The class of recommendation indicates the strength of recommendation, encompassing the estimated magnitude and certainty of benefit in proportion to risk. The level of evidence rates the quality of scientific evidence supporting the intervention on the basis of the type, quantity, and consistency of data from clinical trials and other sources (Table 2) (S1.5-1).

Table 2. Applying Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care* (Updated August 2015)

CLASS (STRENGTH) OF RECOMMENDATION	
CLASS I (STRONG)	**Benefit >>> Risk**

Suggested phrases for writing recommendations:
▪ Is recommended
▪ Is indicated/useful/effective/beneficial
▪ Should be performed/administered/other
▪ Comparative-Effectiveness Phrases†:
  ○ Treatment/strategy A is recommended/indicated in preference to treatment B
  ○ Treatment A should be chosen over treatment B	

CLASS IIa (MODERATE)	**Benefit >> Risk**

Suggested phrases for writing recommendations:
▪ Is reasonable
▪ Can be useful/effective/beneficial
▪ Comparative-Effectiveness Phrases†:
  ○ Treatment/strategy A is probably recommended/indicated in preference to treatment B
  ○ It is reasonable to choose treatment A over treatment B	

CLASS IIb (WEAK)	**Benefit ≥ Risk**
Suggested phrases for writing recommendations: ▪ May/might be reasonable ▪ May/might be considered ▪ Usefulness/effectiveness is unknown/unclear/uncertain or not well established	
CLASS III: No Benefit (MODERATE) *(Generally, LOE A or B use only)*	**Benefit = Risk**
Suggested phrases for writing recommendations: ▪ Is not recommended ▪ Is not indicated/useful/effective/beneficial ▪ Should not be performed/administered/other	
CLASS III: Harm (STRONG)	**Risk > Benefit**
Suggested phrases for writing recommendations: ▪ Potentially harmful ▪ Causes harm ▪ Associated with excess morbidity/mortality ▪ Should not be performed/administered/other	

LEVEL (QUALITY) OF EVIDENCE‡	
LEVEL A	
▪ High-quality evidence‡ from more than 1 RCT ▪ Meta-analyses of high-quality RCTs ▪ One or more RCTs corroborated by high-quality registry studies	
LEVEL B-R	**(Randomized)**
▪ Moderate-quality evidence‡ from 1 or more RCTs ▪ Meta-analyses of moderate-quality RCTs	
LEVEL B-NR	**(Nonrandomized)**
▪ Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies ▪ Meta-analyses of such studies	
LEVEL C-LD	**(Limited Data)**
▪ Randomized or nonrandomized observational or registry studies with limitations of design or execution ▪ Meta-analyses of such studies ▪ Physiological or mechanistic studies in human subjects	
LEVEL C-EO	**(Expert Opinion)**
Consensus of expert opinion based on clinical experience	

COR and LOE are determined independently (any COR may be paired with any LOE).

A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

* The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).

† For comparative-effectiveness recommendations (COR I and IIa; LOE A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

‡ The method of assessing quality is evolving, including the application of standardized, widely used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee.

COR indicates Class of Recommendation; EO, expert opinion; LD, limited data; LOE, Level of Evidence; NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.

1.6. Abbreviations

Abbreviation	Meaning/Phrase
ACHD	adult congenital heart disease
AF	atrial fibrillation
CRT	cardiac resynchronization therapy
ECG	electrocardiogram
EPS	electrophysiology study
LBBB	left bundle branch block
MI	myocardial infarction
SND	sinus node dysfunction

2. Epidemiology and Definitions

2.1. Definitions

See Table 3.

Table 3. Table of Definitions

Term	Definition or Description
Sinus node dysfunction (with accompanying symptoms)	• 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 (S2.1-1). 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 (S2.1-2)	• *First degree atrioventricular block:* 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) • *Second degree atrioventricular block:* P waves with a constant rate (<100 bpm) where atrioventricular conduction is present but not 1:1 o 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 o 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) o 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 o 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 • *Third-degree atrioventricular block (complete heart block):* No evidence of atrioventricular conduction • *Vagally mediated atrioventricular block:* Any type of atrioventricular block mediated by heightened parasympathetic tone • *Infranodal block:* atrioventricular conduction block where clinical evidence or electrophysiologic evidence suggests that the conduction block occurs distal to the

	atrioventricular node
Conduction tissue disease (S2.1-2)	• RBBB (as defined in adults): o Complete RBBB 1. QRS duration ≥120 ms 2. rsr′, rsR′, rSR′, or rarely a qR in leads V_1 or V_2. 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 V_1 and/or V_2. 3. S wave of greater duration than R wave or >40 ms in leads I and V_6 in adults 4. Normal R peak time in leads V_5 and V_6 but >50 ms in lead V_1 o Incomplete RBBB: Same QRS morphology criteria as complete RBBB but with a QRS duration between 110 and 119 ms • LBBB (as defined in adults): o Complete LBBB: 1. QRS duration ≥120 ms in adults 2. Broad notched or slurred R wave in leads I, aVL, V_5, and V_6 and an occasional RS pattern in V_5 and V_6 attributed to displaced transition of QRS complex 3. Absent Q waves in leads I, V_5, and V_6, but in the lead aVL, a narrow Q wave may be present in the absence of myocardial pathology 4. R peak time >60 ms in leads V_5 and V_6 but normal in leads V_1, V_2, and V_3, when small initial R waves can be discerned in the precordial leads 5. ST and T waves usually opposite in direction to QRS o Incomplete LBBB: 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 V_4, V_5, and V_6 4. Absence of Q wave in leads I, V_5, and V_6 • Nonspecific intraventricular conduction delay (as defined in adults): QRS duration >110 ms where morphology criteria for RBBB or LBBB are not present • Left anterior fascicular block: o QRS duration <120 ms o Frontal plane axis between −45° and −90° o qR (small r, tall R) pattern in lead aVL o R-peak time in lead aVL of ≥45 ms o rS pattern (small r, deep S) in leads II, III, and aVF • Left posterior fascicular block: o QRS duration <120 ms o 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. o rS (small r, deep S) pattern in leads I and aVL o qR (small q, tall R) pattern in leads III and aVF

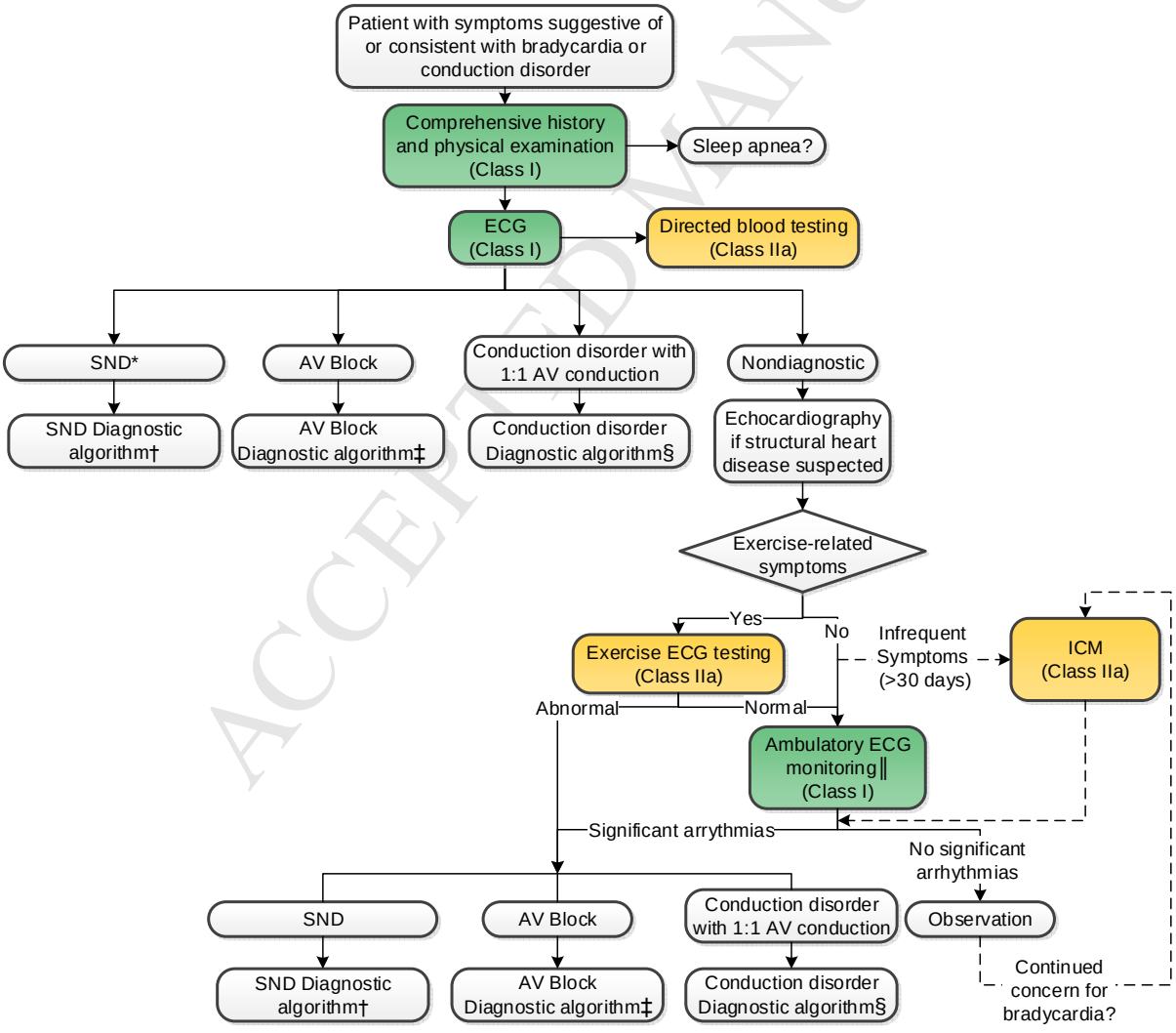
Maximum predicted heart rate for age calculated as 220 – age (y).
AF indicates atrial fibrillation; bpm, beats per minute; LBBB, left bundle branch block; and RBBB, right bundle branch block.

3. General Evaluation of Patients With Documented or Suspected Bradycardia or Conduction Disorders

3.1. History and Physical Examination of Patients With Documented or Suspected Bradycardia or Conduction Disorders

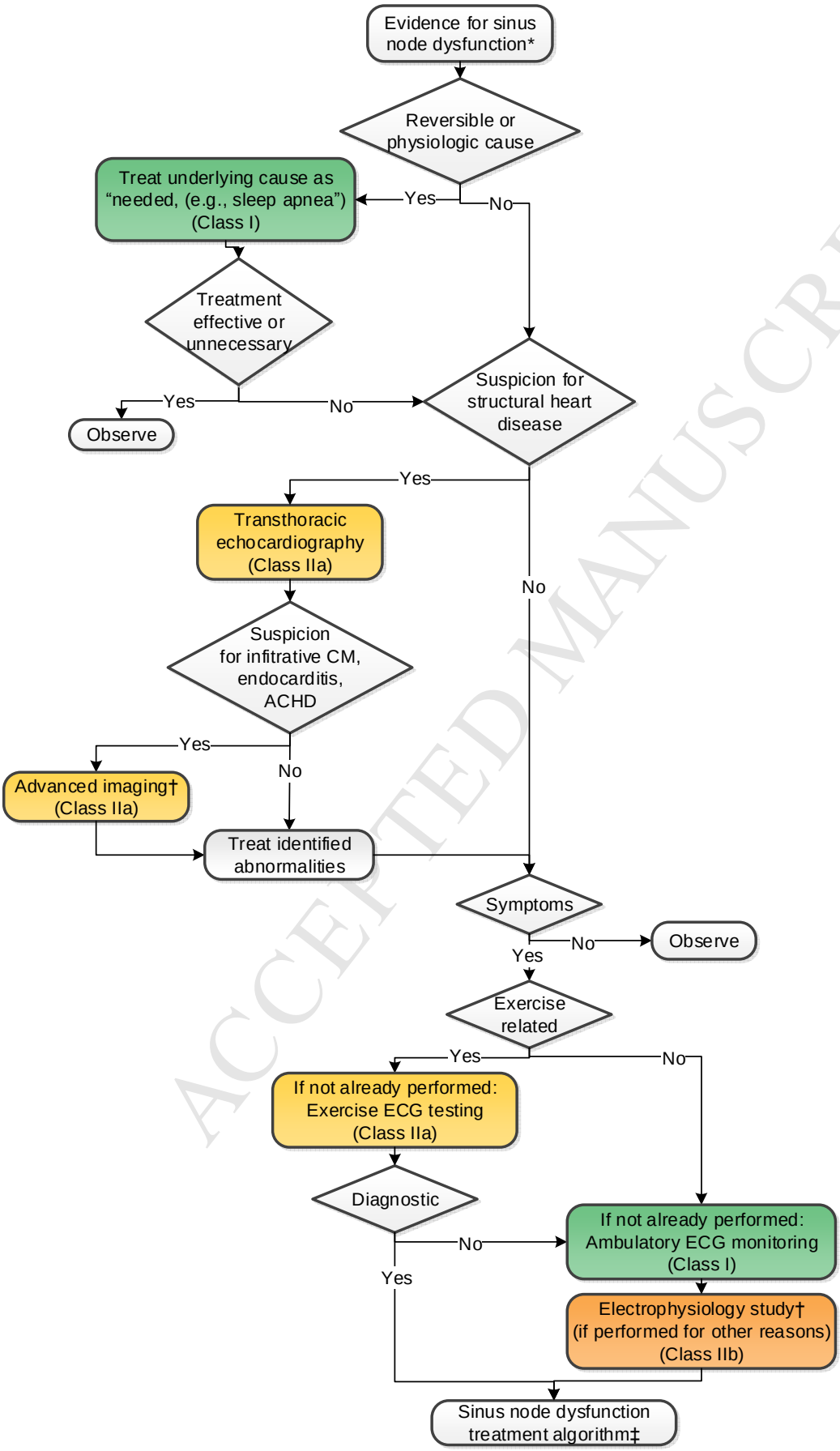
Recommendation for History and Physical Examination in Patients With Documented or Suspected Bradycardia or Conduction Disorders		
COR	**LOE**	**Recommendation**
I	**C-EO**	**1. In patients with suspected bradycardia or conduction disorders a comprehensive history and physical examination should be performed.**

Figure 1. Evaluation of Bradycardia and Conduction Disease Algorithm



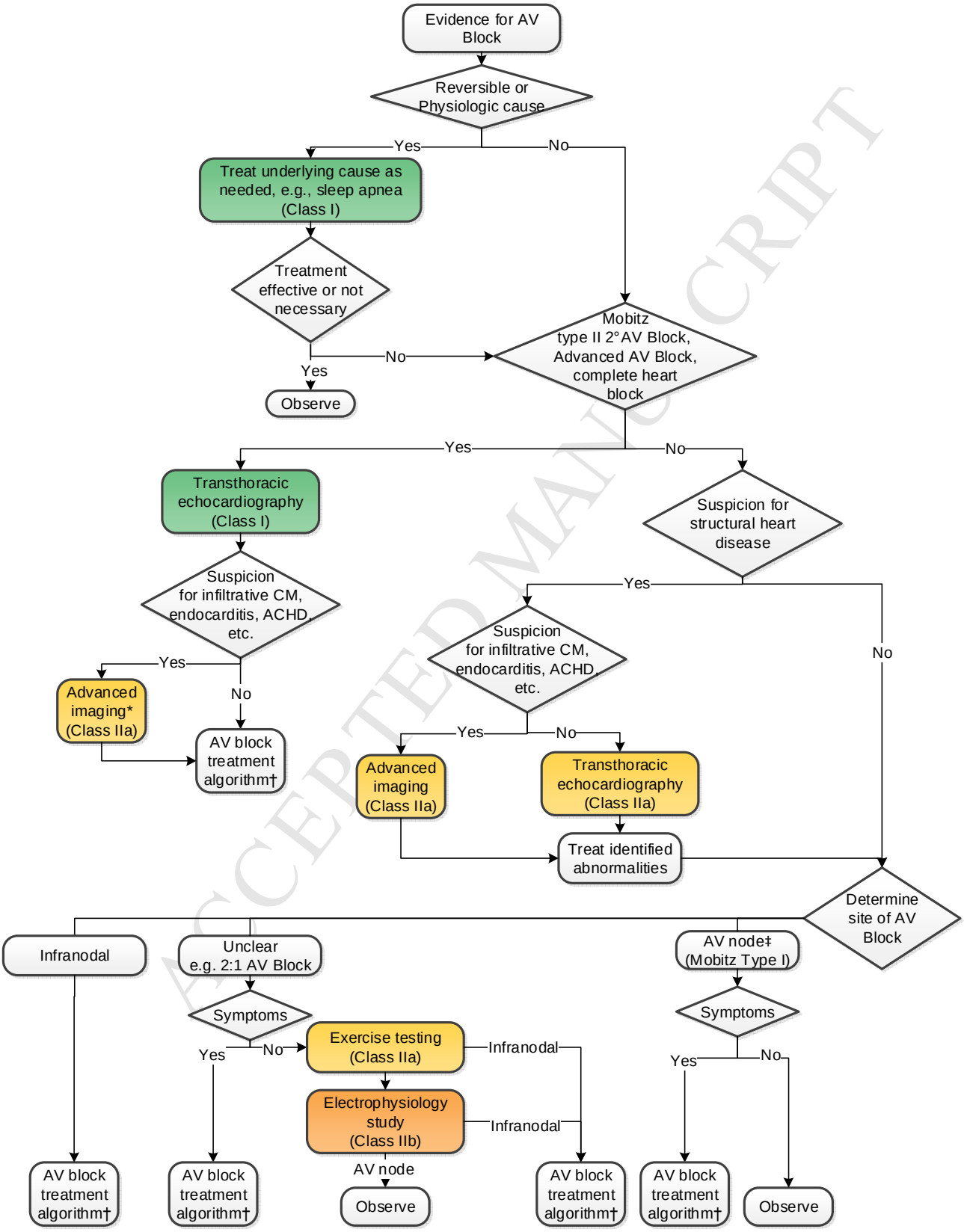
Colors correspond to Class of Recommendation in Table 2.
See Section 4 in the full-text guideline for discussion.
Dashed lines indicate possible optional strategies based on the specific clinical situation.
*Sinus bradycardia, ectopic atrial rhythm, junctional rhythm, sinus pause.
†Refer to Section 3.3.2. Figure 2.
‡Refer to Section 3.3.2. Figure 3.
§Refer to Section 6.1. Figure 8.
‖Monitor choice based on the frequency of symptoms.
AV indicates atrioventricular; and ECG, electrocardiogram/electrocardiographic.

Figure 2. Initial Evaluation of Suspected or Documented Sinus Node Dysfunction Algorithm



Colors correspond to Class of Recommendation in Table 2.
See Section 4 in the full-text guideline for discussion.
*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 Section 4.3.4.1., Figure 6.
ACHD indicates adult congenital heart disease; CM, cardiomyopathy; and ECG, electrocardiogram/electrocardiographic.

Figure 3. Initial Evaluation of Suspected Atrioventricular Block Algorithm



Colors correspond to Class of Recommendation in Table 2.
*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 Section 5.3., Figure 7.
‡The atrioventricular node is more likely the site of block with second-degree Mobitz type I atrioventricular block and a narrow QRS complex or severe first-degree atrioventricular block (>0.30 s) with a narrow QRS complex.
AV indicates atrioventricular; ACHD, adult congenital heart disease; CHD, congenital heart disease; and CM, cardiomyopathy.

Table 4. Medications That Can Induce/Exacerbate Bradycardia or Conduction Disorders

Antihypertensive	Antiarrhythmic	Psychoactive	Other
• Beta adrenergic receptor blockers (including beta adrenergic blocking eye drops used for glaucoma) • Clonidine • Methyldopa • Non-dihydropyridine calcium channel blockers • Reserpine	• Adenosine • Amiodarone • Dronedarone • Flecainide • Procainamide • Propafenone • Quinidine • Sotalol	• Donepezil • Lithium • Opioid analgesics • Phenothiazine antiemetics and antipsychotics • Phenytoin • Selective serotonin reuptake inhibitors • Tricyclic antidepressants	• Anesthetic drugs (propofol) • Cannabis • Digoxin • Ivabradine • Muscle relaxants (e.g., succinylcholine)

Table 5. Conditions Associated With Bradycardia and Conduction Disorders

Intrinsic
Cardiomyopathy (ischemic or nonischemic)
Congenital heart disease
Degenerative fibrosis
Infection/inflammation • Chagas disease • Diphtheria • Infectious endocarditis • Lyme disease • Myocarditis • Sarcoidosis • Toxoplasmosis
Infiltrative disorders • Amyloidosis • Hemochromatosis • Lymphoma
Ischemia/infarction
Rheumatological conditions

• Rheumatoid arthritis • Scleroderma • Systemic lupus erythematosus
Surgical or procedural trauma • Cardiac procedures such as ablation or cardiac catheterization • Congenital heart disease surgery • Septal myomectomy for hypertrophic obstructive cardiomyopathy • Valve surgery (including percutaneous valve replacement)
Extrinsic
Autonomic perturbation • Carotid sinus hypersensitivity • Neurally-mediated syncope/presyncope • Physical conditioning • Situational syncope o Cough o Defecation o Glottic stimulation o Medical procedures o Micturition o Vomiting • Sleep (with or without sleep apnea)
Metabolic • Acidosis • Hyperkalemia • Hypokalemia • Hypothermia • Hypothyroidism • Hypoxia

Adapted with permission from Mangrum and DiMarco (S3.1-1) and Vogler et al. (S3.1-2).

3.2. Noninvasive Evaluation

3.2.1. Resting ECG in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendation for Electrocardiogram (ECG) in Patients With Documented or Suspected Bradycardia or Conduction Disorders Referenced studies that support the recommendation are summarized in Online Data Supplement 1.		
COR	**LOE**	**Recommendation**
I	**B-NR**	**1. In patients with suspected bradycardia or conduction disorder, a 12-lead ECG is recommended to document rhythm, rate, and conduction, and to screen for structural heart disease or systemic illness (S3.2.1-1–S3.2.1-4).**

3.2.2. Exercise Electrocardiographic Testing in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendations for Exercise Electrocardiographic Testing in Patients With Documented or Suspected Bradycardia or Conduction Disorders Referenced studies that support recommendations are summarized in Online Data Supplement 2.		
COR	**LOE**	**Recommendations**
IIa	**B-NR**	**1. In patients with suspected chronotropic incompetence, exercise electrocardiographic testing is reasonable to ascertain the diagnosis and provide information on prognosis (S3.2.2-1, S3.2.2-2).**
IIa	**C-LD**	**2. In patients with exercise-related symptoms suspicious for bradycardia or conduction disorders, or in patients with 2:1 atrioventricular block of unknown level, exercise electrocardiographic testing is reasonable (S3.2.2-3, S3.2.2-4).**

3.2.3. Ambulatory Electrocardiography in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendation for Ambulatory Electrocardiography in Patients With Documented or Suspected Bradycardia or Conduction Disorders Referenced studies that support the recommendation are summarized in Online Data Supplement 3.		
COR	**LOE**	**Recommendation**
I	**B-NR**	**1. In the evaluation of patients with documented or suspected bradycardia or conduction disorders, cardiac rhythm monitoring is useful to establish correlation between heart rate or conduction abnormalities with symptoms, with the specific type of cardiac monitor chosen based on the frequency and nature of symptoms, as well as patient preferences (S3.2.3-1–S3.2.3-12).**

Table 6. Cardiac Rhythm Monitors

Types of Monitor	Device Description	Patient Selection
Nonphysician prescribed smartphone-based systems	• Commercially available smartphone–based systems • Can record a rhythm strip when the patient has symptoms or continuously depending on the technology	Patient access to the technology
Holter monitor	• Continuous recording for 24–72 h; up to 2 wk with newer models • Symptom rhythm correlation can be achieved through a patient event diary and patient-activated annotations	Symptoms frequent enough to be detected within a short period (24–72 h) of monitoring
Patient-activated, transtelephonic monitor (event monitor)	A recording device that transmits patient-activated data (live or stored) via an analog telephone line to a central remote monitoring station (e.g., physician office)	• Frequent, spontaneous symptoms likely to recur within 2–6 wk • Limited use in patients with incapacitating symptoms
External loop recorder (patient or auto triggered)*	• A device that continuously records and stores rhythm data over weeks to months • Patient activated, or auto triggered (e.g., to record asymptomatic arrhythmias) to provide a recording of events antecedent to (3–14 min), during, and after (1–4 min) the triggered event • Newer models are equipped with a cellular telephone, which transmits triggered data automatically over a wireless network to a remote monitoring system	Frequent, spontaneous symptoms potentially related to bradycardia or conduction disorder, likely to recur within 2–6 wk
External patch recorders	• Patch device that continuously records and stores rhythm data, with patient-trigger capability to allow for symptom-rhythm correlation • No leads or wires, and adhesive to chest wall/sternum • Various models record from 2–14 d • Offers accurate means of assessing burden of AF • Patient activated, or auto triggered (e.g., to record asymptomatic arrhythmias) to provide a recording of events antecedent to, during, and after the triggered event	• Can be considered as an alternative to external loop recorder • Given that it is leadless, can be accurately self-applied, and is largely water resistant, it may be more comfortable and less cumbersome than an external loop recorder, potentially improving compliance • Unlike Holter monitors and other external monitors, it offers only 1-lead recording
Mobile cardiac outpatient telemetry	• Device that records and transmits data (up to 30 d) from preprogrammed arrhythmias or patient activation to a communication hub at the patient's home • Significant arrhythmias are detected; the monitor automatically transmits the patient's electrocardiographic data through a wireless network to the central monitoring station, which is attended by trained technicians 24 h/d	• Spontaneous symptoms, potentially related to bradycardia or conduction disorder, that are too brief, too subtle, or too infrequent to be readily documented with patient activated monitors • In high-risk patients whose rhythm requires real-time monitoring

*Higher yield in patients who are able to record a diary to correlate with possible arrhythmia.

Adapted with permission from Shen et al. (S3.2.3-13).
AF indicates atrial fibrillation.

3.2.4. Imaging in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendations for Cardiac Imaging in Bradycardia or Conduction Disorders Referenced studies that support recommendations are summarized in Online Data Supplements 3 and 4.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. In patients with newly identified left bundle branch block (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 (S3.2.4-1–S3.2.4-10).**
IIa	**B-NR**	**2. In selected patients presenting with bradycardia or conduction disorders other than LBBB, second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or third-degree atrioventricular block, transthoracic echocardiography is reasonable if structural heart disease is suspected (S3.2.4-3, S3.2.4-11–S3.2.4-13).**
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 (S3.2.4-14–S3.2.4-22).**
III: No Benefit	**B-NR**	**4. In the evaluation of patients with asymptomatic sinus bradycardia or first-degree atrioventricular block and no clinical evidence of structural heart disease, routine cardiac imaging is not indicated (S3.2.4-22–S3.2.4-24).**

3.2.5. Laboratory Testing in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendation for Laboratory Testing in Patients With Documented or Suspected Bradycardia or Conduction Disorders		
COR	**LOE**	**Recommendation**
IIa	**C-LD**	**1. In patients with bradycardia, laboratory tests (e.g., thyroid function tests, Lyme titer, potassium, pH) based on clinical suspicion for a potential underlying cause are reasonable (S3.2.5-1–S3.2.5-4).**

3.2.6. Genetic Testing in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendations for Genetic Testing in Documented or Suspected Bradycardia or Conduction Disorders

COR	LOE	Recommendations
I	C-EO	1. In patients in whom a conduction disorder-causative mutation has been identified, genetic counseling and mutation-specific genetic testing of first-degree relatives is recommended to identify similarly affected individuals.
IIb	C-EO	2. In patients with inherited conduction disease, genetic counseling and targeted testing may be considered to facilitate cascade screening of relatives as part of the diagnostic evaluation.

3.2.7. Sleep Apnea Evaluation and Treatment in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendation for Sleep Apnea Evaluation and Treatment in Patients With Documented or Suspected Bradycardia or Conduction Disorders Referenced studies that support recommendations are summarized in Online Data Supplement 5.		
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 (S3.2.7-1–S3.2.7-11).
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 (S3.2.7-12–S3.2.7-16).
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 (S3.2.7-10, S3.2.7-11).

3.3. Invasive Testing

3.3.1. Implantable Cardiac Monitor in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendation for Implantable Cardiac Monitor in Patients With Documented or Suspected Bradycardia or Conduction Disorders Referenced studies that support the recommendation are summarized in Online Data Supplement 6.		
COR	**LOE**	**Recommendation**
IIa	**C-LD**	**1. In patients with infrequent symptoms (>30 days between symptoms) suspected to be caused by bradycardia, long-term ambulatory monitoring with an implantable cardiac monitor is reasonable if initial noninvasive evaluation is nondiagnostic (S3.3.1-1–S3.3.1-3).**

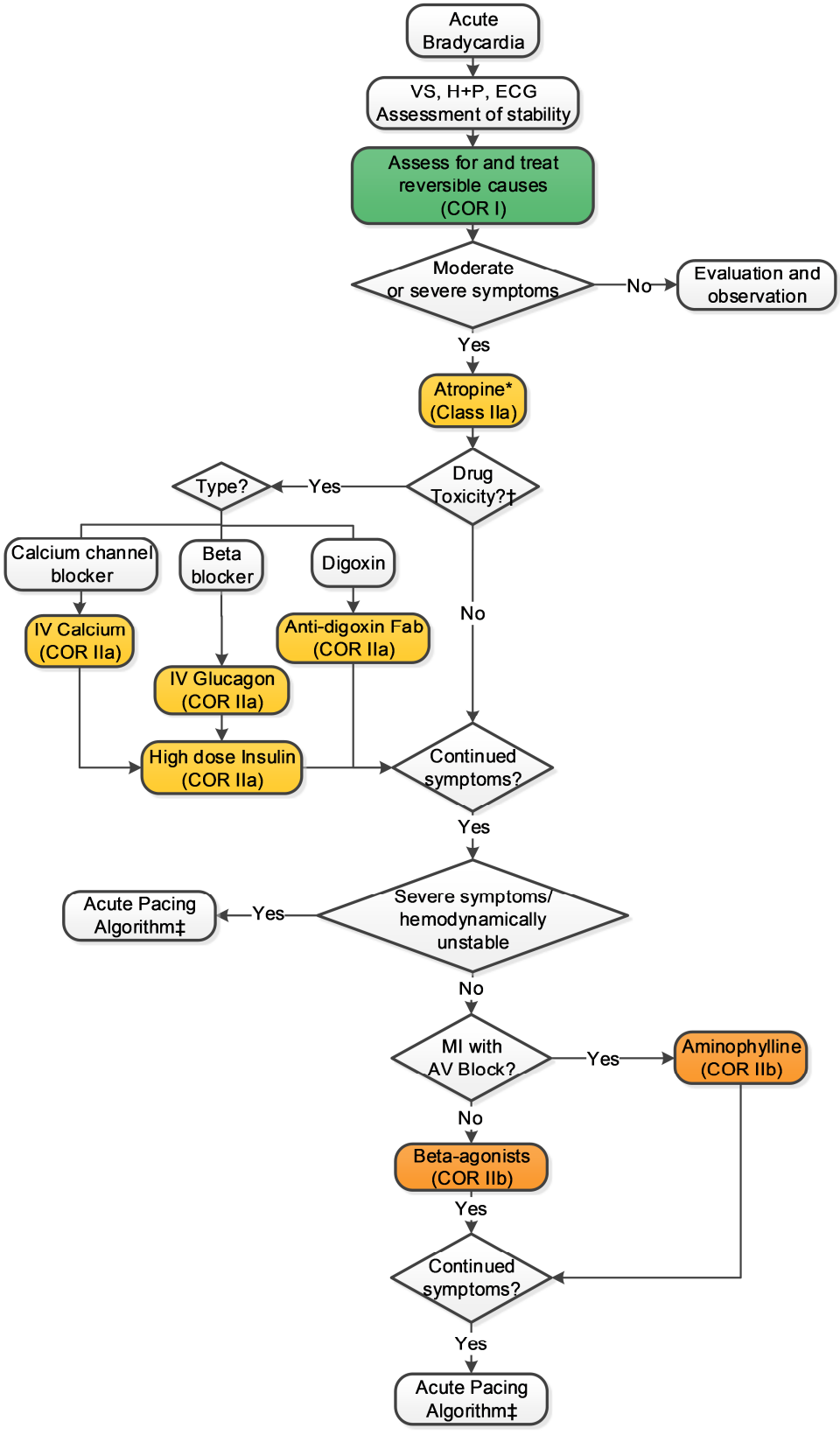
3.3.2. Electrophysiology Study in Patients With Documented or Suspected Bradycardia or Conduction Disorders

Recommendation for Electrophysiology Testing in Patients With Documented or Suspected Bradycardia or Conduction Disorders Referenced studies that support recommendations are summarized in Online Data Supplement 7.		
COR	**LOE**	**Recommendation**
IIb	**C-LD**	**1. In patients with symptoms suspected to be attributable to bradycardia, an electrophysiology study (EPS) may be considered in selected patients for diagnosis of, and elucidation of bradycardia mechanism, if initial non-invasive evaluation is nondiagnostic (S3.3.2-1–S3.3.2-5).**

4. Bradycardia Attributable to Sinus Node Dysfunction

4.1. Acute Management of Sinus Node Dysfunction

Figure 4. Acute Bradycardia Algorithm



Colors correspond to Class of Recommendation in Table 2.
See Sections 5.3. and 6.3. in the full-text guideline for discussion.
*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 Section 4.1.3., Figure 5.
AADs indicates anti-arrhythmic drugs; AV, atrioventricular; BB, beta blocker; CCB, calcium channel blocker; COR, Class of Recommendation; ECG, electrocardiographic; H+P, history and physical examination; IMI, inferior myocardial infarction; IV, intravenous; PM, pacemaker; S/P, status post; and VS, vital signs.

4.1.1. Acute Management of Reversible Causes of Sinus Node Dysfunction

Recommendation for Acute Management of Reversible Causes for Bradycardia Attributable to Sinus Node Dysfunction		
COR	**LOE**	**Recommendation**
I	**C-EO**	**1. In symptomatic patients presenting with sinus node dysfunction (SND), evaluation and treatment of reversible causes is recommended.**

Table 7. Common Potentially Reversible or Treatable Causes of SND (S4.1.1-1)

Acute myocardial ischemia or infarction (S4.1.1-2–S4.1.1-4)
Athletic training (S4.1.1-5)
Atrial fibrillation (S4.1.1-6)
Cardiac surgery • Valve replacement (S4.1.1-7, S4.1.1-8), maze procedure (S4.1.1-7), coronary artery bypass graft (S4.1.1-9, S4.1.1-10)
Drugs or toxins* • Toluene, organophosphates, tetrodotoxin, cocaine (S4.1.1-11)
Electrolyte abnormality • Hyperkalemia (S4.1.1-12), hypokalemia (S4.1.1-13), hypoglycemia (S4.1.1-14)
Heart transplant (S4.1.1-15): Acute rejection, chronic rejection, remodeling (S4.1.1-16, S4.1.1-17)
Hypervagotonia (S4.1.1-18, S4.1.1-19)
Hypothermia • Therapeutic (post-cardiac arrest cooling (S4.1.1-20)) or environmental exposure (S4.1.1-21)
Hypothyroidism (S4.1.1-22)
Hypovolemic shock (S4.1.1-23)
Hypoxemia, hypercarbia, acidosis (S4.1.1-24) • Sleep apnea, respiratory insufficiency (suffocation, drowning (S4.1.1-25), stroke (S4.1.1-26), drug overdose)
Infection (S4.1.1-27) • Lyme disease (S4.1.1-28), legionella, psittacosis, typhoid fever, typhus, listeria (S4.1.1-29), malaria, leptospirosis, Dengue fever, viral hemorrhagic fevers, Guillain-Barre (S4.1.1-30)
Medications* • Beta blockers, non-dihydropyridine calcium channel blockers, digoxin (S4.1.1-31), antiarrhythmic drugs, lithium (S4.1.1-32), methyldopa, risperidone, cisplatin, interferon

*Partial list.
SND indicates sinus node dysfunction.

4.1.2. Acute Medical Therapy for Bradycardia

4.1.2.1. Atropine and Beta-Agonists for Bradycardia to SND

Recommendations for Atropine and Beta-Agonists for Bradycardia Attributable to SND Referenced studies that support recommendations are summarized in Online Data Supplements 8, 9, 10, and 11.		
COR	**LOE**	**Recommendations**
IIa	**C-LD**	**1. In patients with SND associated with symptoms or hemodynamic compromise, atropine is reasonable to increase sinus rate (S4.1.2.1-1–S4.1.2.1-4).**
IIb	**C-LD**	**2. 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 (S4.1.2.1-5–S4.1.2.1-11).**
III: Harm	**C-LD**	**3. In patients who have undergone heart transplant without evidence for autonomic reinnervation, atropine should not be used to treat sinus bradycardia (S4.1.2.1-12, S4.1.2.1-13).**

Table 8. Acute Medical Management of Bradycardia Attributable to SND or Atrioventricular Block

Medication	**Dosage**	**Comments**
Symptomatic sinus bradycardia or atrioventricular block		
Atropine	0.5-1 mg IV (may be repeated every 3-5 min to a maximum dose of 3 mg) (S4.1.2.4-8–S4.1.2.4-12)	
Dopamine	5 to 20 mcg/kg/min IV. starting at 5 mcg/kg/min and increasing by 5 mcg/kg/min every 2 min (S4.1.2.4-13)	Dosages of >20 mcg/kg/min may result in vasoconstriction or arrhythmias
Isoproterenol	20-60 mcg IV bolus followed doses of 10-20 mcg, or infusion of 1-20 mcg/min based on heart rate response (S4.1.2.4-14–S4.1.2.4-20)	Monitor for potential development of ischemic chest pain
Epinephrine	2-10 mcg/min IV or 0.1-0.5 mcg/kg/min IV titrated to desired effect (S4.1.2.4-19, S4.1.2.4-21)	
Second- or third-degree atrioventricular block associated with acute inferior MI		
Aminophylline	250-mg IV bolus	
Calcium channel blocker overdose		
10% calcium chloride	1-2 g IV every 10-20 min or an infusion of 0.2-0.4 mL/kg/h (S4.1.2.4-22–S4.1.2.4-24)	
10% calcium gluconate	3-6 g IV every 10-20 min or an infusion at 0.6-1.2 mL/kg/h (S4.1.2.4-22–S4.1.2.4-24)	
Beta-blocker or calcium channel blocker overdose		
Glucagon	3-10 mg IV with infusion of 3-5 mg/h (S4.1.2.4-25, S4.1.2.4-26)	
High dose insulin therapy	IV bolus of 1 unit/kg followed by an infusion of 0.5 units/kg/h (S4.1.2.4-24, S4.1.2.4-27, S4.1.2.4-28).	Follow glucose and potassium levels
Digoxin overdose		
Digoxin antibody fragment	Dosage is dependent on amount ingested or known digoxin concentration (S4.1.2.4-29–S4.1.2.4-36)	• 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 min	
Theophylline	300 mg IV, followed by oral dose of 5-10 mg/kg/d	• Therapeutic serum levels range from 10-

	titrated to effect	20 mcg/mL • Usual posttransplant dosages average 450 mg±100 mg/d
Spinal cord injury		
Aminophylline	6 mg/kg in 100-200 mL of IV fluid over 20-30 min (S4.1.2.4-7)	
Theophylline	Oral dose of 5-10 mg/kg/d titrated to effect (S4.1.2.4-6)	Effective dosages often result in serum levels below the usual effective range of 10-20 mcg/mL

IV indicates intravenous; MI, myocardial infarction; and SND, sinus node dysfunction.

4.1.2.2. Therapy of Beta Blocker and Calcium Channel Blocker Mediated Bradycardia Attributable to SND or Atrioventricular Block

Recommendations for Therapy of Beta-Blocker and Calcium Channel Blocker Mediated Bradycardia Referenced studies that support recommendations are summarized in Online Data Supplement 12.		
COR	**LOE**	**Recommendations**
IIa	**C-LD**	**1. 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 (S4.1.2.2-1–S4.1.2.2-3).**
IIa	**C-LD**	**2. 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 (S4.1.2.2-4, S4.1.2.2-5).**
IIa	**C-LD**	**3. 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 (S4.1.2.2-6, S4.1.2.2-7).**

4.1.2.3. Therapy of Digoxin Mediated Bradycardia Attributable to Either SND or Atrioventricular Block

Recommendations for Therapy of Digoxin Mediated Bradycardia Attributable to SND or Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 13, 14, and 15.		
COR	**LOE**	**Recommendations**
IIa	**C-LD**	**1. 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 (S4.1.2.3-1–S4.1.2.3-8).**
III: No Benefit	**C-LD**	**2. In patients with bradycardia associated with symptoms or hemodynamic compromise attributable to digoxin toxicity, dialysis is not recommended for removal of digoxin (S4.1.2.3-9).**

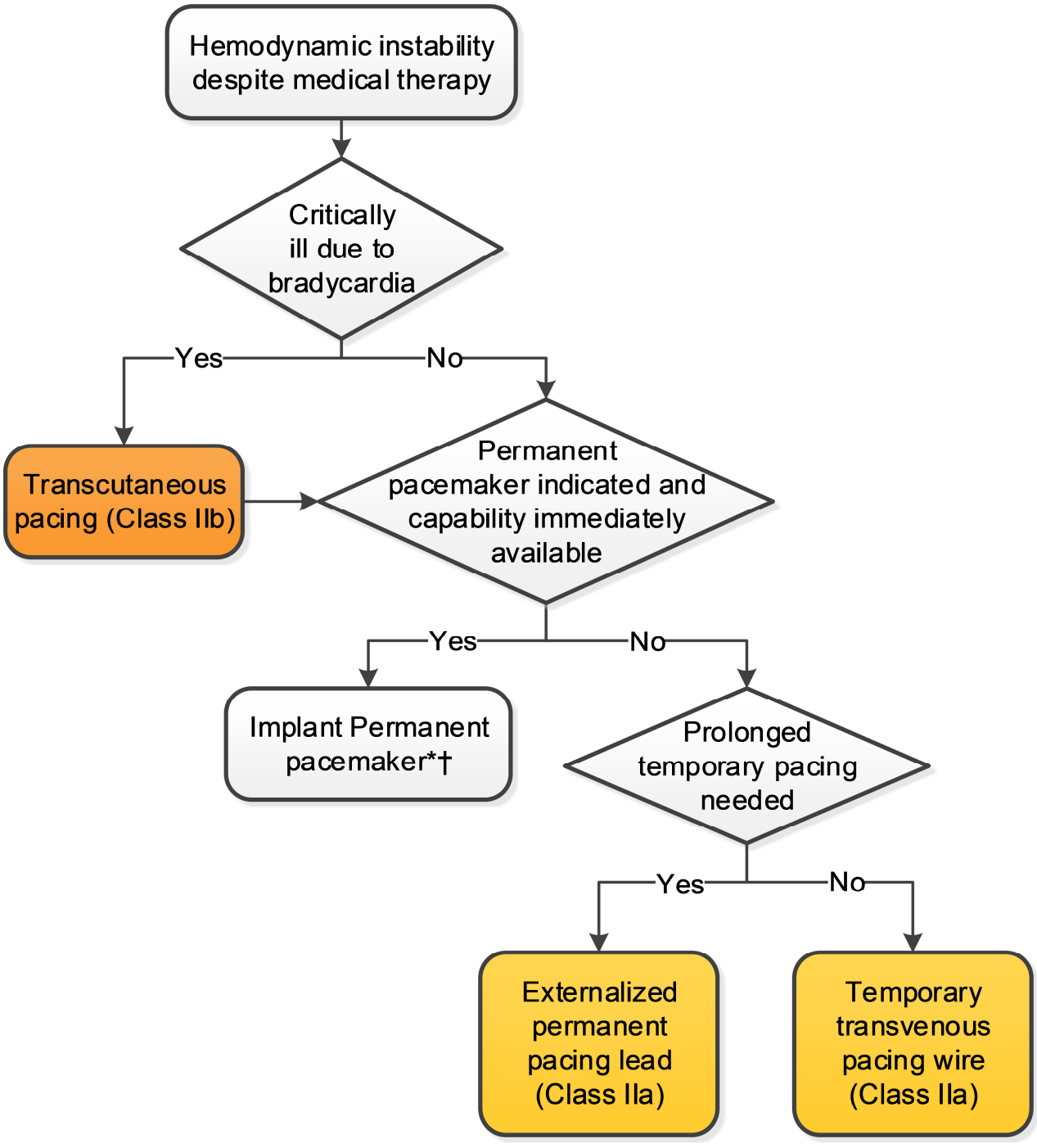
4.1.2.4. Aminophylline or Theophylline for Bradycardia Attributable to SND

Recommendations for Theophylline/Aminophylline for Bradycardia Attributable to SND Referenced studies that support recommendations are summarized in Online Data Supplements 16 and 17.		
COR	**LOE**	**Recommendations**
IIa	**C-LD**	**1. In post-heart transplant patients, aminophylline or theophylline is reasonable to increase heart rate if clinically indicated (S4.1.2.4-1–S4.1.2.4-4).**
IIa	**C-LD**	**2. 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 (S4.1.2.4-5–S4.1.2.4-7).**

4.1.3. Temporary Pacing for Bradycardia Attributable to SND

Recommendations for Temporary Pacing for Bradycardia Attributable to SND Referenced studies that support recommendations are summarized in Online Data Supplements 18, 19, 20, and 21.		
COR	**LOE**	**Recommendations**
IIa	**C-LD**	**1. 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 permanent pacemaker is placed or the bradycardia resolves (S4.1.3-1–S4.1.3-15).**
IIb	**C-LD**	**2. 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 permanent pacemaker is placed or the bradycardia resolves (S4.1.3-16–S4.1.3-21).**
III: Harm	**C-LD**	**3. In patients with SND with minimal and/or infrequent symptoms without hemodynamic compromise, temporary transcutaneous or transvenous pacing should not be performed (S4.1.3-1, S4.1.3-2, S4.1.3-8, S4.1.3-9, S4.1.3-11, S4.1.3-12, S4.1.3-14, S4.1.3-22).**

Figure 5. Acute Pacing Algorithm



Colors correspond to Class of Recommendation in Table 2.
See Sections 5.4. and 6.3. in the full-text guideline for discussion.
*Refer to Section 4.3.4.1., Figure 6 for chronic SND and Section 5.3., Figure 7 for chronic atrioventricular block
†Careful management of anesthesia to avoid or minimize the use of drugs associated with bradycardia is required.

4.2. Chronic Therapy/Management of Bradycardia Attributable to SND

4.2.1. General Principles of Chronic Therapy/Management of Bradycardia Attributable to SND

Recommendations for General Principles of Chronic Therapy/Management of Bradycardia Attributable to SND		
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 (S4.2.1-1–S4.2.1-7).**
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 (S4.2.1-1–S4.2.1-7).**
III: Harm	**C-LD**	**3. In patients with asymptomatic SND, 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 (S4.2.1-5–S4.2.1-7).**

4.2.2. Transient/Reversible Causes (Including Medications) of Bradycardia Attributable to SND

Recommendation for Transient/Reversible Causes of Sinus Bradycardia		
COR	**LOE**	**Recommendation**
I	**C-EO**	**1. Patients presenting with symptomatic SND secondary to a reversible cause should first be managed by directing the therapy at eliminating or mitigating the offending condition.**

4.2.3. Additional Testing of Bradycardia Attributable to SND

Recommendations for Additional Testing of Bradycardia Attributable to SND		
COR	**LOE**	**Recommendations**
IIb	**C-EO**	**1. In patients with symptoms suggestive of bradycardia (e.g., syncope, lightheadedness) who are also undergoing an EPS for another indication, evaluation of sinus node function as part of the EPS may be considered.**
IIb	**C-EO**	**2. In symptomatic patients with suspected SND, EPS for the assessment of sinus node function may be considered when the diagnosis remains uncertain after initial noninvasive evaluations (S4.2.3-1–S4.2.3-5).**
III: No Benefit	**C-LD**	**3. In patients with asymptomatic sinus bradycardia, an EPS should not be performed unless other indications for electrophysiological testing exist (S4.2.3-6, S4.2.3-7).**

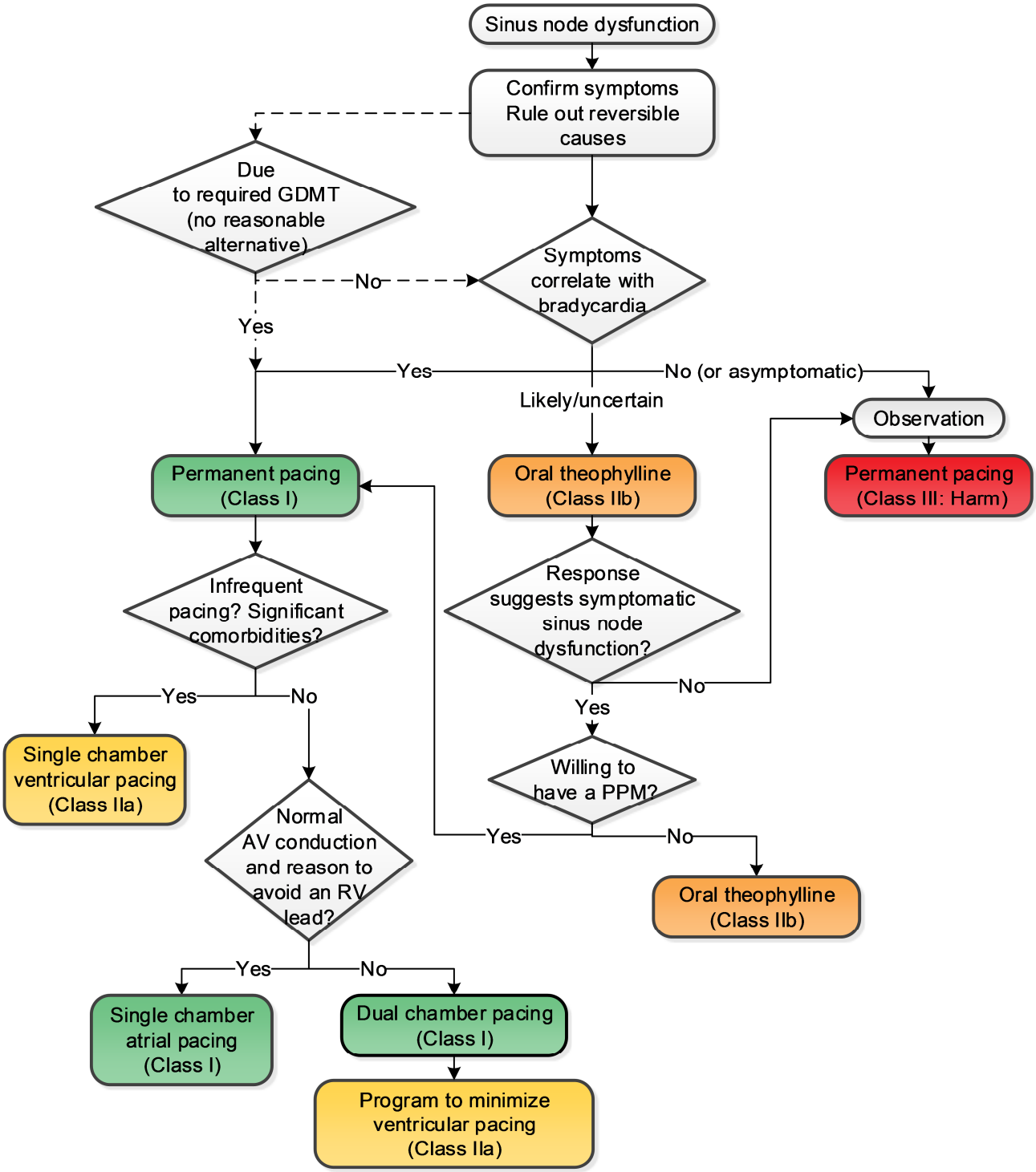
4.3.4. Permanent Pacing for Chronic Therapy/Management of Bradycardia Attributable to SND

Recommendations for Permanent Pacing for Chronic Therapy/Management of Bradycardia Attributable to SND Referenced studies that support recommendations are summarized in Online Data Supplements 24 and 25.		
COR	**LOE**	**Recommendations**
I	C-LD	1. In patients with symptoms that are directly attributable to SND, permanent pacing is indicated to increase heart rate and improve symptoms (S4.3.4-1, S4.3.4-2).
I	C-EO	2. 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.
IIa	C-EO	3. 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	4. In patients with symptomatic chronotropic incompetence, permanent pacing with rate-responsive programming is reasonable to increase exertional heart rates and improve symptoms.
IIb	C-LD	5. In patients with symptoms that are likely attributable to SND, a trial of oral theophylline may be considered to increase heart rate, improve symptoms, and help determine the potential effects of permanent pacing (S4.3.4-3, S4.3.4-4).

4.3.4.1. Permanent Pacing Techniques and Methods for Chronic Therapy/Management of Bradycardia Attributable to SND

Recommendations for Permanent Pacing Techniques and Methods for Chronic Therapy/Management of Bradycardia Attributable to SND Referenced studies that support recommendations are summarized in Online Data Supplement 25.		
COR	**LOE**	**Recommendations**
I	B-R	1. In symptomatic patients with SND, atrial-based pacing is recommended over single chamber ventricular pacing (S4.3.4.1-1–S4.3.4.1-4).
I	B-R	2. In symptomatic patients with SND and intact atrioventricular conduction without evidence of conduction abnormalities, dual chamber or single chamber atrial pacing is recommended (S4.3.4.1-5).
IIa	B-R	3. In symptomatic patients with SND who have dual chamber pacemakers and intact atrioventricular conduction, it is reasonable to program the dual chamber pacemaker to minimize ventricular pacing (S4.3.4.1-6).
IIa	C-EO	4. In symptomatic patients with SND in which frequent ventricular pacing is not expected or the patient has significant comorbidities that are otherwise likely to determine the survival and clinical outcomes, single chamber ventricular pacing is reasonable.

Figure 6. Chronic SND Management Algorithm



Colors correspond to Class of Recommendation in Table 2.
See Sections 4.3. and 5.5. in the full text guideline for discussion.
Dashed lines indicate possible optional strategies based on the specific clinical situation.
*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.

5. Bradycardia Attributable to Atrioventricular Block

5.1. Pathophysiology, Etiology, and Classification of Bradycardia Attributable to Atrioventricular Block

Table 9. Etiology of Atrioventricular Block

Congenital/genetic
• Congenital AV block (associated with maternal systemic lupus erythematosus) • Congenital heart defects (e.g., L-TGA) • Genetic (e.g., SCN5A mutations)
Infectious
• Lyme carditis • Bacterial endocarditis with perivalvar abscess • Acute rheumatic fever • Chagas disease • Toxoplasmosis
Inflammatory/infiltrative
• Myocarditis • Amyloidosis • Cardiac sarcoidosis • Rheumatologic disease: Systemic sclerosis, SLE, RA, reactive arthritis (Reiter's syndrome) • Other cardiomyopathy-idiopathic, valvular
Ischemic
• Acute MI • Coronary ischemia without infarction—unstable angina, variant angina • Chronic ischemic cardiomyopathy
Degenerative
• Lev's and Lenegre's diseases
Vagotonic-associated with increased vagal tone
• Sleep, obstructive sleep apnea • High-level athletic conditioning • Neurocardiogenic
Metabolic/endocrine
• 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
• Neuromuscular diseases (e.g., myotonic dystrophy, Kearns-Sayre syndrome, Erb's dystrophy) • Lymphoma
Iatrogenic

• Medication related
  o Beta blockers, verapamil, diltiazem, digoxin
  o Antiarrhythmic drugs
  o 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.

5.2. Acute Management

5.2.1. Acute Management of Reversible Causes of Bradycardia Attributable to Atrioventricular Block

Recommendations for Acute Management of Reversible Causes of Bradycardia Attributable to Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplement 26.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. Patients with transient or reversible causes of atrioventricular block, such as Lyme carditis or drug toxicity, should have medical therapy and supportive care, including temporary transvenous pacing if necessary, before determination of need for permanent pacing (S5.2.1-1–S5.2.1-5).**
IIa	**B-NR**	**2. 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 (S5.2.1-6–S5.2.1-9).**
IIa	**B-NR**	**3. In patients with second-degree or third-degree atrioventricular block associated with cardiac sarcoidosis, permanent pacing, with additional defibrillator capability if needed and meaningful survival of greater than 1 year is expected, without further observation for reversibility is reasonable (S5.2.1-10, S5.2.1-11).**
IIb	**C-LD**	**4. In patients with symptomatic second-degree or third-degree atrioventricular block associated with thyroid function abnormalities but without clinical myxedema, permanent pacing without further observation for reversibility may be considered (S5.2.1-12).**

5.2.2. Acute Medical Therapy for Bradycardia Attributable to Atrioventricular Block

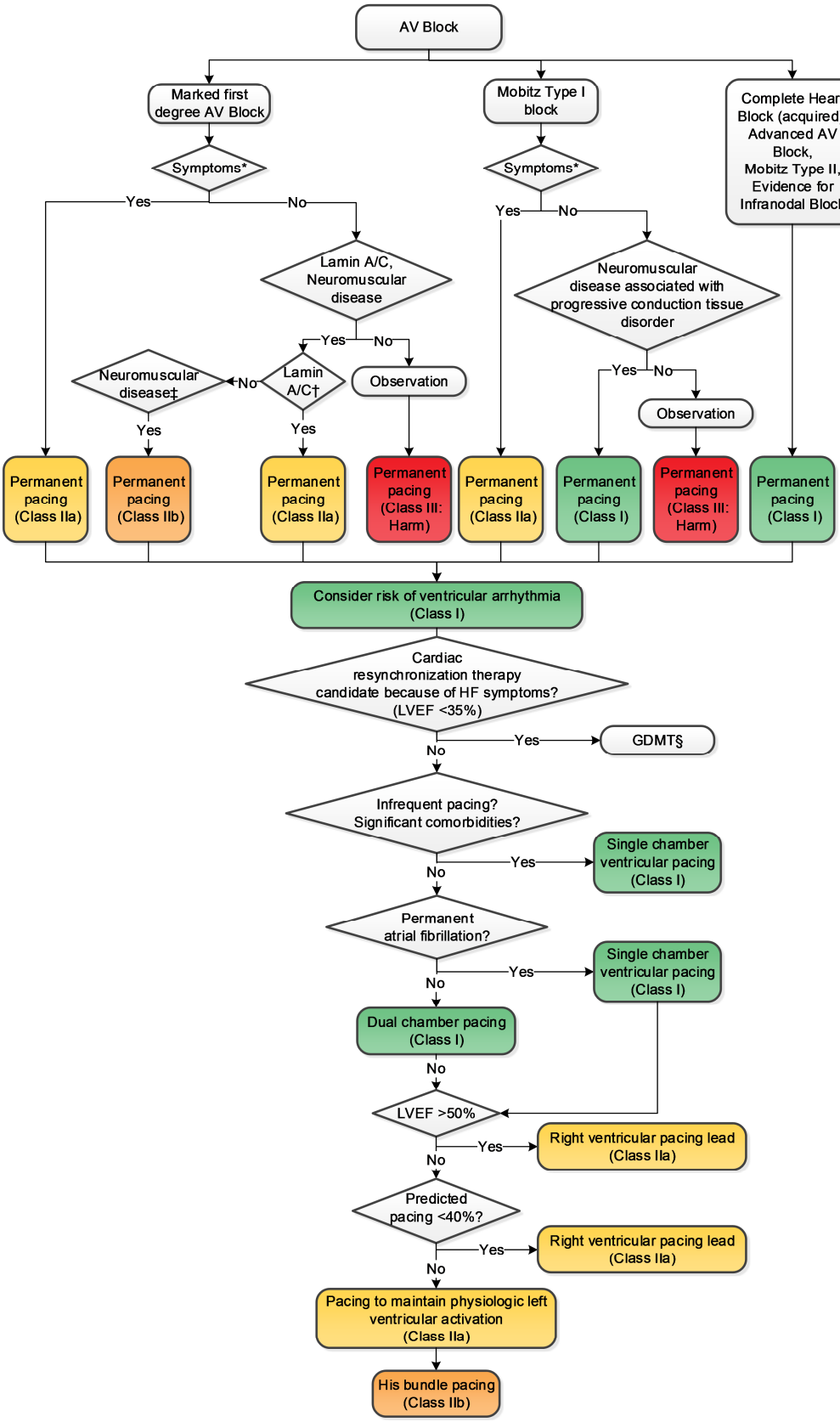
Recommendations for Acute Medical Therapy for Bradycardia Attributable to Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 27 and 28.		
COR	**LOE**	**Recommendations**
IIa	**C-LD**	**1. For patients with second-degree or third-degree atrioventricular 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 (S5.2.2-1–S5.2.2-3).**
IIb	**B-NR**	**2. For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise and who have 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 (S5.2.2-3–S5.2.2-7).**
IIb	**C-LD**	**3. For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise in the setting of acute inferior myocardial infarction (MI), intravenous aminophylline may be considered to improve atrioventricular conduction, increase ventricular rate, and improve symptoms (S5.2.2-8–S5.2.2-11).**

5.2.3. Temporary Pacing for Atrioventricular Block

Recommendations for Temporary Pacing for Bradycardia Attributable to Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 29 and 30.		
COR	**LOE**	**Recommendations**
IIa	**B-NR**	**1. 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 (S5.2.3-1–S5.2.3-7).**
IIa	**B-NR**	**2. 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 (S5.2.3-8–S5.2.3-14).**
IIb	**B-R**	**3. 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 permanent pacemaker is placed or the bradyarrhythmia resolves (S5.2.3-15–S5.2.3-20).**

5.3. Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block

Figure 7. Management of Bradycardia or Pauses Attributable to Chronic Atrioventricular Block Algorithm


AV Block
Marked first degree AV Block
Mobitz Type I block
Complete Heart Block (acquired), Advanced AV Block, Mobitz Type II, Evidence for Infranodal Block
Symptoms*
Symptoms*
Yes
No
Yes
No
Lamin A/C, Neuromuscular disease
Neuromuscular disease associated with progressive conduction tissue disorder
Yes
No
Yes
No
Neuromuscular disease‡
No
Lamin A/C†
Observation
Yes
No
Observation
Yes
Yes
Permanent pacing (Class IIa)
Permanent pacing (Class IIb)
Permanent pacing (Class IIa)
Permanent pacing (Class III: Harm)
Permanent pacing (Class IIa)
Permanent pacing (Class I)
Permanent pacing (Class III: Harm)
Permanent pacing (Class I)
Consider risk of ventricular arrhythmia (Class I)
Cardiac resynchronization therapy candidate because of HF symptoms? (LVEF <35%)
Yes
GDMT§
No
Infrequent pacing? Significant comorbidities?
Yes
Single chamber ventricular pacing (Class I)
No
Permanent atrial fibrillation?
Yes
Single chamber ventricular pacing (Class I)
No
Dual chamber pacing (Class I)
No
LVEF >50%
Yes
Right ventricular pacing lead (Class IIa)
No
Predicted pacing <40%?
Yes
Right ventricular pacing lead (Class IIa)
No
Pacing to maintain physiologic left ventricular activation (Class IIa)
His bundle pacing (Class IIb)

Colors correspond to Class of Recommendation in Table 2.
Refer to Section 6.4. in the full-text guideline for discussion.
*Symptoms correlate with atrioventricular block.
†PR interval >240 ms, LBBB.
‡PR interval >240 ms, QRS >120 ms or fascicular block.
§Refer to heart failure guidelines (S5.3-1, S5.3-2).
AV indicates atrioventricular; GDMT, guideline directed management and therapy; HF, heart failure; LBBB, left bundle branch block; and LVEF, left ventricular ejection fraction.

5.3.1. General Principles of Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block

Recommendations for General Principles of Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 31, 32, 33, and 34.		
COR	**LOE**	**Recommendations**
III: Harm	**C-LD**	**1. In patients with 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 (S5.3-1–S5.3-7).**
III: Harm	**C-LD**	**2. In asymptomatic patients with 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, permanent pacing should not be performed (S5.3-4–S5.3-10).**

5.3.2. Transient/Potentially Reversible Causes of Atrioventricular Block

Recommendations for Potentially Reversible or Transient Causes of Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 34, 35, 36, and 37.		
COR	**LOE**	**Recommendations**
I	**C-LD**	**1. 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 (S5.3.2-1–S5.3.2-3).**
III: Harm	**C-LD**	**2. In patients who had acute atrioventricular block attributable to a known reversible and non-recurrent cause, and have had complete resolution of the atrioventricular block with treatment of the underlying cause, permanent pacing should not be performed (S5.3.2-1, S5.3.2-4–S5.3.2-9).**
III: Harm	**C-LD**	**3. In patients with asymptomatic vagally mediated atrioventricular block, permanent pacing should not be performed (S5.3.2-6–S5.3.2-10).**

5.3.3. Additional Testing for Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block

Recommendations for Additional Testing for Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 37 and 38.		
COR	**LOE**	**Recommendations**
IIa	**B-R**	**1. In patients with symptoms (e.g., lightheadedness, dizziness) of unclear etiology who have first-degree atrioventricular block or second-degree Mobitz type I atrioventricular block on ECG, ambulatory electrocardiographic monitoring is reasonable to establish correlation between symptoms and rhythm abnormalities (S5.3.3-1–S5.3.3-4).**
IIa	**C-LD**	**2. In patients with exertional symptoms (e.g., chest pain, shortness of breath) who have first-degree or second-degree Mobitz type I atrioventricular block at rest, an exercise treadmill test is reasonable to determine whether they may benefit from permanent pacing (S5.3.3-5, S5.3.3-6).**
IIb	**B-NR**	**3. In selected patients with second-degree atrioventricular block, an EPS may be considered to determine the level of the block and to determine whether they may benefit from permanent pacing (S5.3.3-7–S5.3.3-9).**
IIb	**C-LD**	**4. In selected patients with second-degree atrioventricular block, carotid sinus massage and/or pharmacological challenge with atropine, isoproterenol, or procainamide may be considered to determine the level of the block and to determine whether they may benefit from permanent pacing (S5.3.3-10–S5.3.3-12).**

5.3.4. Permanent Pacing

Recommendations for Permanent Pacing for Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 34, 39, and 40.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. 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 (S5.3.4-1–S5.3.4-7).**
I	**B-NR**	**2. In patients with neuromuscular diseases associated with conduction disorders, including muscular dystrophy (such as myotonic dystrophy type 1) or Kearns-Sayre syndrome, who have evidence of second-degree atrioventricular block, third-degree atrioventricular block, or an HV interval of 70 ms or greater, regardless of symptoms, permanent pacing, with additional defibrillator capability if needed and meaningful survival of greater than 1 year is expected, is recommended (S5.3.4-8–S5.3.4-15).**
I	**C-LD**	**3. In patients with permanent atrial fibrillation (AF) and symptomatic bradycardia, permanent pacing is recommended (S5.3.4-2, S5.3.4-16, S5.3.4-**

		17).
I	**C-LD**	**4. In patients who develop symptomatic atrioventricular block 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 (S5.3.4-18–S5.3.4-24).**
IIa	**B-NR**	**5. In patients with an infiltrative cardiomyopathy, such as cardiac sarcoidosis or amyloidosis, and second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or third-degree atrioventricular block, permanent pacing, with additional defibrillator capability if needed and meaningful survival of greater than 1 year is expected, is reasonable (S5.3.4-25–S5.3.4-30).**
IIa	**B-NR**	**6. In patients with lamin A/C gene mutations, including Limb Girdle and Emery Dreifuss muscular dystrophies, with a PR interval greater than 240 ms and LBBB, permanent pacing, with additional defibrillator capability if needed and meaningful survival of greater than 1 year is expected, is reasonable (S5.3.4-31–S5.3.4-33).**
IIa	**C-LD**	**7. In patients with marked first-degree or second-degree Mobitz type I (Wenckebach) atrioventricular block with symptoms that are clearly attributable to the atrioventricular block, permanent pacing is reasonable (S5.3.4-34–S5.3.4-37).**
IIb	**C-LD**	**8. In patients with neuromuscular diseases, such as myotonic dystrophy type 1, with a PR interval greater than 240 ms, a QRS duration greater than 120 ms, or fascicular block, permanent pacing, with additional defibrillator capability if needed and meaningful survival of greater than 1 year is expected, may be considered (S5.3.4-9–S5.3.4-13, S5.3.4-15).**

5.3.4.1. Permanent Pacing Techniques and Methods for Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block

Recommendations for Permanent Pacing Techniques and Methods for Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block Referenced studies that support recommendations are summarized in Online Data Supplements 39 and 40 and the Systematic Review.		
COR	**LOE**	**Recommendations**
I	**A**	**1. In patients with SND and atrioventricular block who require permanent pacing, dual chamber pacing is recommended over single chamber ventricular pacing (S5.3.4.1-1–S5.3.4.1-7).**
I	**A**	**2. In select patients with atrioventricular block who require permanent pacing in whom frequent ventricular pacing is not expected, or who have significant comorbidities that are likely to determine clinical outcomes and that may limit the benefit of dual chamber pacing, single chamber ventricular pacing is effective (S5.3.4.1-1–S5.3.4.1-6, S5.3.4.1-8–S5.3.4.1-10).**
I	**B-R**	**3. For patients in sinus rhythm with a single chamber ventricular pacemaker who develop pacemaker syndrome, revising to a dual chamber pacemaker is recommended (S5.3.4.1-1, S5.3.4.1-2, S5.3.4.1-5, S5.3.4.1-8–S5.3.4.1-10).**
IIa	**B-R^{SR}**	**4. In patients with atrioventricular block who have an indication for permanent pacing with a left ventricular ejection fraction between 36% and 50% and are expected to require ventricular pacing more than 40% of the time, it is reasonable to choose pacing methods that maintain physiologic ventricular activation (e.g., cardiac resynchronization therapy [CRT] or His bundle pacing) over right ventricular pacing (S5.3.4.1-7, S5.3.4.1-11–S5.3.4.1-19)**
IIa	**B-R**	**5. In patients with atrioventricular block who have an indication for permanent pacing with a left ventricular ejection fraction between 36% and 50% and are expected to require ventricular pacing less than 40% of the time, it is reasonable to choose right ventricular pacing over pacing methods that maintain physiologic ventricular activation (e.g., CRT or His bundle pacing) (S5.3.4.1-15, S5.3.4.1-16, S5.3.4.1-20, S5.3.4.1-21).**
IIb	**B-R^{SR}**	**6. In patients with atrioventricular block at the level of the atrioventricular node who have an indication for permanent pacing, His bundle pacing may be considered to maintain physiologic ventricular activation (S5.3.4.1-19, S5.3.4.1-22–S5.3.4.1-25).**
III: Harm	**C-LD**	**7. In patients with permanent or persistent AF in whom a rhythm control strategy is not planned, implantation of an atrial lead should not be performed (S5.3.4.1-26, S5.3.4.1-27).**

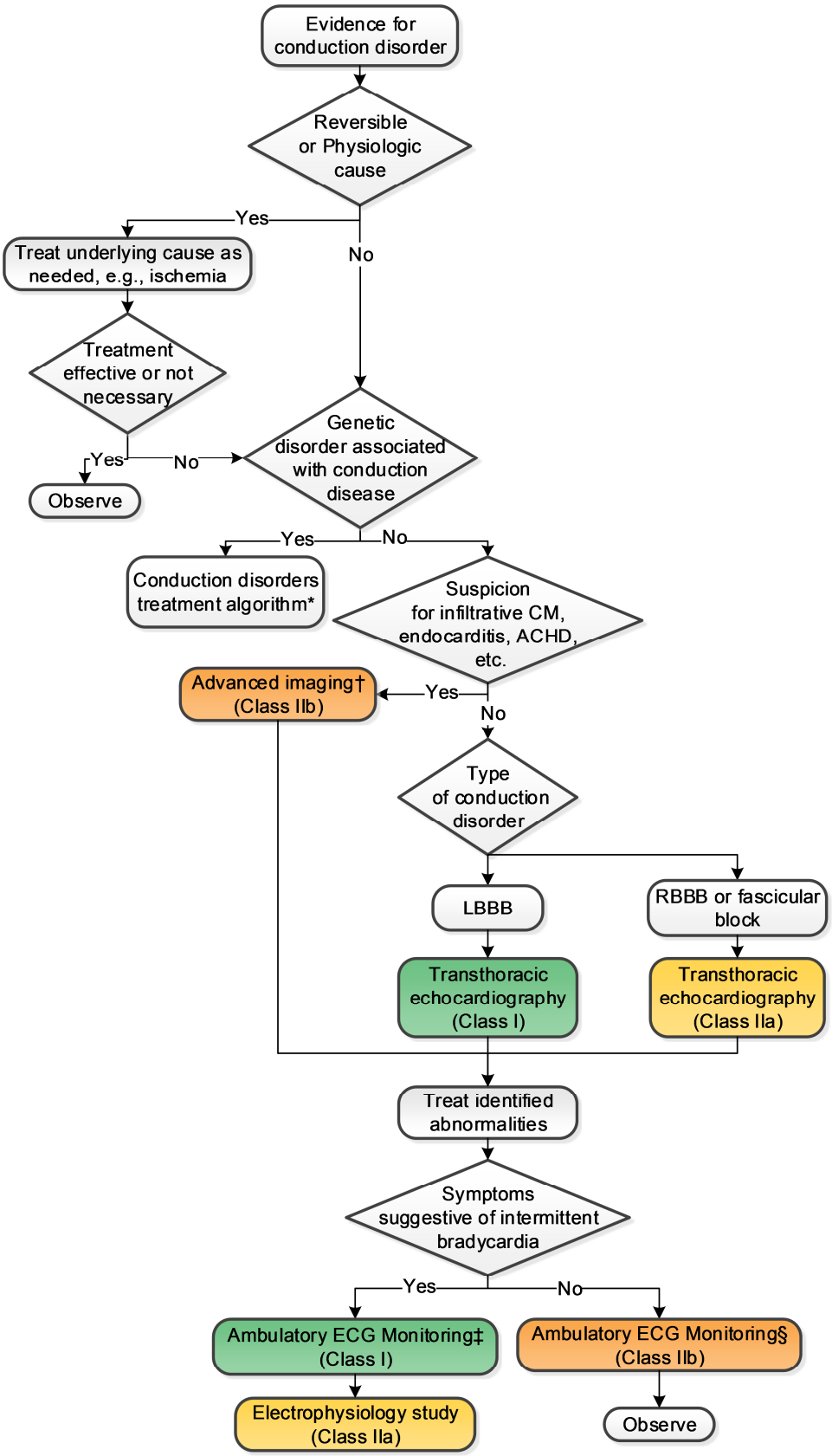
SR indicates systematic review.

6. Conduction Disorders (With 1:1 Atrioventricular Conduction)

6.1. Evaluation of Conduction Disorders

Recommendations for Evaluation of Conduction Disorders (With 1:1 Atrioventricular Conduction and Normal PR Interval) Referenced studies that support recommendations are summarized in Online Data Supplements 41 and 42.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. In patients with newly detected LBBB, a transthoracic echocardiogram to exclude structural heart disease is recommended (S6.1-1–S6.1-3).**
I	**C-LD**	**2. In symptomatic patients with conduction system disease, in whom atrioventricular block is suspected, ambulatory electrocardiographic monitoring is useful (S6.1-4–S6.1-11).**
IIa	**B-NR**	**3. In selected patients presenting with intraventricular conduction disorders other than LBBB, transthoracic echocardiography is reasonable if structural heart disease is suspected (S6.1-3, S6.1-12, S6.1-13).**
IIa	**B-NR**	**4. In patients with symptoms suggestive of intermittent bradycardia (e.g., lightheadedness, syncope), with conduction system disease identified by ECG and no demonstrated atrioventricular block, EPS is reasonable (S6.1-14).**
IIa	**C-LD**	**5. In selected patients with LBBB in whom structural heart disease is suspected and echocardiogram is unrevealing, advanced imaging (e.g., cardiac MRI, computed tomography, or nuclear studies) is reasonable (S6.1-15).**
IIb	**C-LD**	**6. In selected asymptomatic patients with extensive conduction system disease (bifascicular or trifascicular block), ambulatory electrocardiographic recording may be considered to document suspected higher degree of atrioventricular block (S6.1-4, S6.1-6).**
IIb	**C-LD**	**7. In selected asymptomatic patients with LBBB in whom ischemic heart disease is suspected, stress testing with imaging may be considered (S6.1-2).**

Figure 8. Evaluation of Conduction Disorders Algorithm

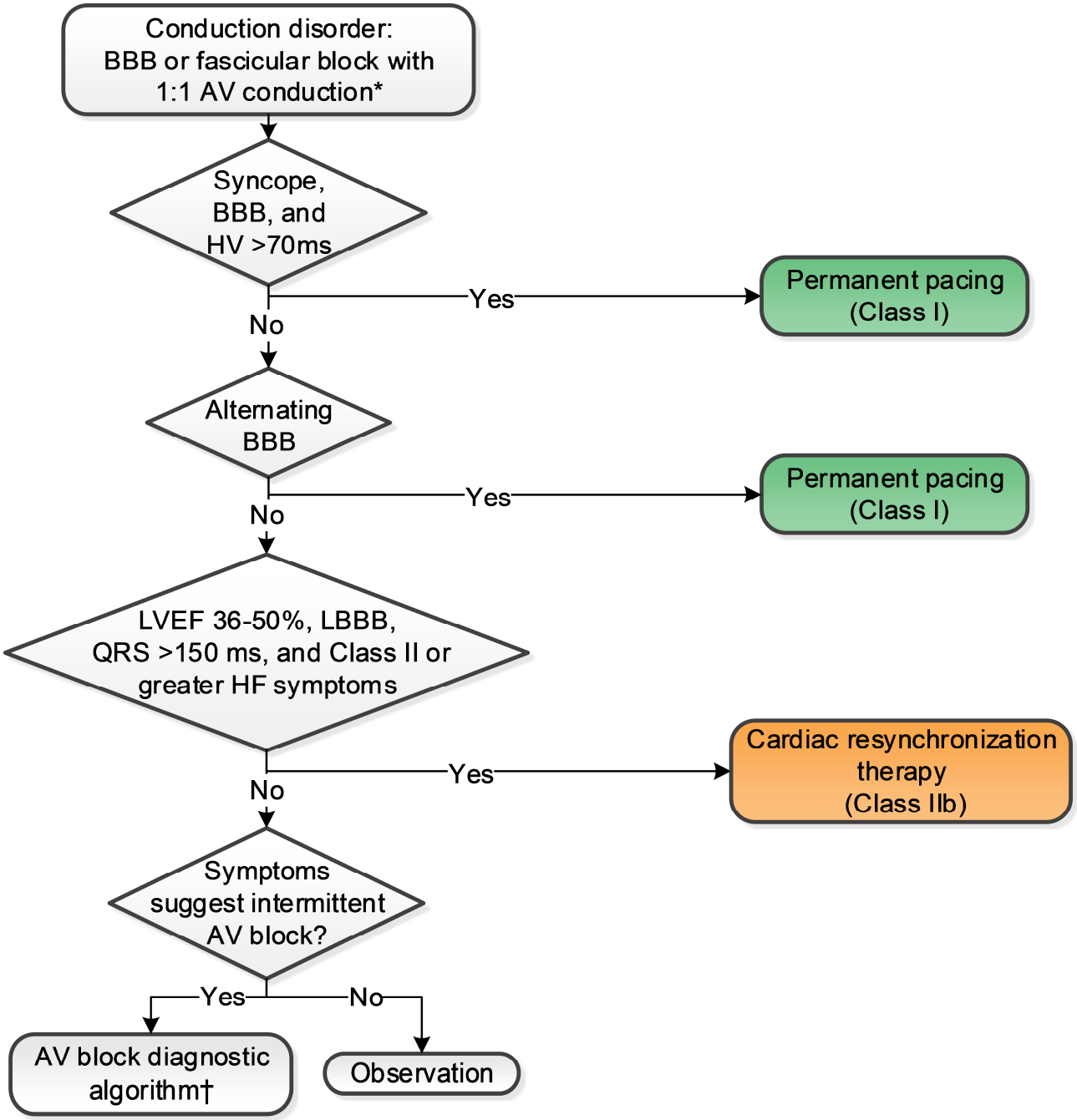

Evidence for conduction disorder
Reversible or Physiologic cause
Yes
No
Treat underlying cause as needed, e.g., ischemia
Treatment effective or not necessary
Yes
No
Observe
Genetic disorder associated with conduction disease
Yes
No
Conduction disorders treatment algorithm*
Suspicion for infiltrative CM, endocarditis, ACHD, etc.
Advanced imaging† (Class IIb)
Yes
No
Type of conduction disorder
LBBB
RBBB or fascicular block
Transthoracic echocardiography (Class I)
Transthoracic echocardiography (Class IIa)
Treat identified abnormalities
Symptoms suggestive of intermittent bradycardia
Yes
No
Ambulatory ECG Monitoring‡ (Class I)
Ambulatory ECG Monitoring§ (Class IIb)
Electrophysiology study (Class IIa)
Observe

Colors correspond to Class of Recommendation in Table 2.
See Section 7.4. in the full-text guideline for discussion.
*Refer to Section 6.2., Figure 9.
†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 atrioventricular block combined with LBBB).
ACHD indicates adult congenital heart disease; CM, cardiomyopathy; ECG, electrocardiogram/electrocardiographic; LBBB, left bundle branch block; and RBBB, right bundle branch block.

6.2. Management of Conduction Disorders (With 1:1 Atrioventricular Conduction)

Recommendations for Management of Conduction Disorders (With 1:1 Atrioventricular Conduction and Normal PR Intervals) Referenced studies that support recommendations are summarized in Online Data Supplements 41 and 42.		
COR	**LOE**	**Recommendations**
I	**C-LD**	**1. In patients with syncope and bundle branch block who are found to have an HV interval 70 ms or greater or evidence of infranodal block at EPS, permanent pacing is recommended (S6.2-1, S6.2-2)**
I	**C-LD**	**2. In patients with alternating bundle branch block, permanent pacing is recommended (S6.2-3).**
IIa	**C-LD**	**3. In patients with Kearns-Sayre syndrome and conduction disorders, permanent pacing is reasonable, with additional defibrillator capability if appropriate and meaningful survival of greater than 1 year is expected (S6.2-4, S6.2-5).**
IIb	**C-LD**	**4. In patients with Anderson-Fabry disease and QRS prolongation greater than 110 ms, permanent pacing, with additional defibrillator capability if needed and meaningful survival of greater than 1 year is expected, may be considered (S6.2-6, S6.2-7).**
IIb	**C-LD**	**5. In patients with heart failure, a mildly to moderately reduced left ventricular ejection fraction (36%–50%), and LBBB (QRS ≥150 ms), CRT therapy may be considered (S6.2-8, S6.2-9).**
III: Harm	**B-NR**	**6. In asymptomatic patients with isolated conduction disease and 1:1 atrioventricular conduction, permanent pacing is not indicated (in the absence of other indications for pacing) (S6.2-10–S6.2-15).**

Figure 9. Management of Conduction Disorders Algorithm



Colors correspond to Class of Recommendation in Table 2.
*For severe first-degree atrioventricular block or first-degree atrioventricular block with an accompanying neuromuscular disease, also refer to Section 5.3., Figure 7, the atrioventricular block algorithm.
†See Section 3.3.2., Figure 3.
AV indicates atrioventricular; BBB, bundle branch block; HF, heart failure; LBBB, left bundle branch block; and LVEF, left ventricular ejection fraction.

7. Special Populations

7.1. Perioperative Management

7.1.1. Patients at Risk for Bradycardia During Noncardiac Surgery or Procedures

Recommendations for Patients at Risk for Bradycardia During Noncardiac Surgery or Procedures Referenced studies that support recommendations are summarized in Online Data Supplements 42, 44, and 45.		
COR	**LOE**	**Recommendations**
IIa	**B-NR**	**1. In patients who are thought to be at high risk for the development of intraoperative or periprocedural bradycardia because of patient characteristics or procedure type, placement of transcutaneous pacing pads is reasonable (S7.1.1-1–S7.1.1-3).**
III: Harm	**B-NR**	**2. In patients with LBBB who require pulmonary artery catheterization for intraoperative monitoring, routine prophylactic temporary transvenous pacing should not be performed (S7.1.1-4, S7.1.1-5).**

7.1.2. Postoperative Bradycardia and Conduction Disorders After Cardiac Surgery

7.1.2.1. Coronary Artery Bypass

Recommendations for Pacing After Isolated Coronary Artery Bypass Surgery Referenced studies that support recommendations are summarized in Online Data Supplement 47.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. In patients who have new postoperative SND or atrioventricular block associated with persistent symptoms or hemodynamic instability that does not resolve after isolated coronary artery bypass surgery, permanent pacing is recommended before discharge (S7.1.2.1-1–S7.1.2.1-9).**
IIa	**B-NR**	**2. In patients undergoing isolated coronary artery bypass surgery, routine placement of temporary epicardial pacing wires is reasonable (S7.1.2.1-5, S7.1.2.1-10, S7.1.2.1-11).**
IIb	**C-EO**	**3. In patients undergoing coronary artery bypass surgery who will likely require future CRT or ventricular pacing, intraoperative placement of a permanent epicardial left ventricular lead may be considered.**

7.1.2.2. Surgery for AF

Recommendations for Pacing After Surgery for AF Referenced studies that support recommendations are summarized in Online Data Supplement 48.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. In patients undergoing surgery for AF, routine placement of temporary epicardial pacing wires is recommended (S7.1.2.2-1–S7.1.2.2-4).**
I	**B-NR**	**2. In patients who have new postoperative SND or atrioventricular block associated with symptoms or hemodynamic instability that does not resolve after surgery for AF, permanent pacing is recommended before discharge (S7.1.2.2-1–S7.1.2.2-4).**
IIb	**C-EO**	**3. In patients undergoing surgery for AF who will likely require future CRT or ventricular pacing, intraoperative placement of a permanent epicardial left ventricular lead may be considered.**

7.1.2.3. Valvular Surgery

7.1.2.3.1. Surgical Aortic Valve Replacement or Repair

Recommendations for Pacing After Aortic Valve Surgery Referenced studies that support recommendations are summarized in Online Data Supplement 48.		
COR	**LOE**	**Recommendations**
I	**C-LD**	**1. In patients undergoing surgical aortic valve replacement or repair, routine placement of temporary epicardial pacing wires is recommended (S7.1.2.3.1-1–S7.1.2.3.1-3).**
I	**B-NR**	**2. In patients who have new postoperative SND or atrioventricular block associated with persistent symptoms or hemodynamic instability that does not resolve after aortic valve replacement, permanent pacing is recommended before discharge (S7.1.2.3.1-1–S7.1.2.3.1-5).**
IIb	**C-EO**	**3. In patients undergoing aortic valve surgery who will likely require future CRT or ventricular pacing, intraoperative placement of a permanent epicardial left ventricular lead may be considered.**

7.1.2.3.2. Mitral Valve Surgery

Recommendations for Pacing After Mitral Valve Surgery Referenced studies that support recommendations are summarized in Online Data Supplement 48.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. In patients who have new postoperative SND or atrioventricular block associated with persistent symptoms or hemodynamic instability that does not resolve after mitral valve repair or replacement surgery, permanent pacing is recommended before discharge (S7.1.2.3.2-1, S7.1.2.3.2-2).**
IIa	**C-LD**	**2. In patients undergoing mitral valve surgery, routine placement of temporary epicardial pacing wires is reasonable (S7.1.2.3.2-1–S7.1.2.3.2-3).**
IIb	**C-EO**	**3. In patients undergoing surgical mitral valve repair or replacement who will likely require future CRT or ventricular pacing, intraoperative placement of a permanent epicardial left ventricular lead may be considered.**

7.1.2.3.3. Tricuspid Valve Surgery

Recommendations for Pacing After Tricuspid Valve Surgery Referenced studies that support recommendations are summarized in Online Data Supplement 48.		
COR	**LOE**	**Recommendations**
I	**C-LD**	**1. In patients undergoing tricuspid valve surgery, routine placement of temporary epicardial pacing wires is recommended (S7.1.2.3.3-1–S7.1.2.3.3-4).**
I	**B-NR**	**2. In patients who have new postoperative SND or atrioventricular block associated with symptoms or hemodynamic instability that does not resolve after tricuspid valve surgery, permanent pacing is recommended before discharge (S7.1.2.3.3-1–S7.1.2.3.3-4).**
IIa	**C-LD**	**3. In patients who are undergoing tricuspid valve replacement or tricuspid repair with high risk for postoperative atrioventricular block, intraoperative placement of permanent epicardial leads at the time of cardiac surgery is reasonable (S7.1.2.3.3-1–S7.1.2.3.3-5).**

7.1.2.4. Transcatheter Aortic Valve Replacement

Recommendations for Conduction Disturbances After Transcatheter Aortic Valve Replacement Referenced studies that support recommendations are summarized in Online Data Supplement 49.		
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 (S7.1.2.4-1–S7.1.2.4-4).**
IIa	**B-NR**	**2. In patients with new persistent bundle branch block after transcatheter aortic valve replacement, careful surveillance for bradycardia is reasonable (S7.1.2.4-5, S7.1.2.4-6).**
IIb	**B-NR**	**3. In patients with new persistent LBBB after transcatheter aortic valve replacement, implantation of a permanent pacemaker may be considered (S7.1.2.4-4, S7.1.2.4-7–S7.1.2.4-10).**

7.1.2.5. Heart Transplant, Surgical Myectomy, and Alcohol Septal Ablation

7.1.2.5.1. Surgical Myectomy and Alcohol Septal Ablation for Hypertrophic Cardiomyopathy

Recommendations for Patients Undergoing Surgical Myectomy or Alcohol Septal Ablation for Hypertrophic Cardiomyopathy Referenced studies that support recommendations are summarized in Online Data Supplements 51 and 52.		
COR	**LOE**	**Recommendations**
I	**B-NR**	**1. In patients with second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or persistent complete atrioventricular block after alcohol septal ablation or surgical myectomy, permanent pacing is recommended before discharge (S7.1.2.5.1-1–S7.1.2.5.1-4).**
IIa	**B-NR**	**2. In selected patients with hypertrophic cardiomyopathy who require permanent pacing for rate support after alcohol septal ablation or surgical myectomy and are at high risk for sudden cardiac death and meaningful survival of greater than 1 year is expected, selecting a device with defibrillator capabilities is reasonable (S7.1.2.5.1-5–S7.1.2.5.1-7).**
IIb	**C-LD**	**3. In patients with hypertrophic cardiomyopathy who undergo alcohol septal ablation and who are at risk for developing late atrioventricular block, prolonged ambulatory electrocardiographic monitoring may be considered (S7.1.2.5.1-1, S7.1.2.5.1-2, S7.1.2.5.1-4, S7.1.2.5.1-7, S7.1.2.5.1-8).**
IIb	**C-LD**	**4. In patients with hypertrophic cardiomyopathy, evaluation of ventriculoatrial conduction by EPS at the time of alcohol septal ablation may be considered for identifying future risk of atrioventricular block (S7.1.2.5.1-9).**

7.2. Bradycardia Management for Adult Congenital Heart Disease

Recommendations for Management of Bradycardia in Adults With Adult Congenital Heart Disease Referenced studies that support recommendations are summarized in Online Data Supplement 53.		
COR	**LOE**	**Recommendations**
I	B-NR	1. In adults with adult congenital heart disease (ACHD)and symptomatic SND or chronotropic incompetence, atrial based permanent pacing is recommended (S7.2-1–S7.2-6).
I	B-NR	2. In adults with ACHD and symptomatic bradycardia related to atrioventricular block, permanent pacing is recommended (S7.2-7–S7.2-9).
I	B-NR	3. In adults with congenital complete atrioventricular block with any symptomatic bradycardia, a wide QRS escape rhythm, mean daytime heart-rate below 50 bpm, complex ventricular ectopy, or ventricular dysfunction, permanent pacing is recommended (S7.2-10, S7.2-11).
I	B-NR	4. In adults with ACHD and postoperative second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or third-degree atrioventricular block that is not expected to resolve, permanent pacing is recommended (S7.2-12, S7.2-13).
IIa	B-NR	5. In asymptomatic adults with congenital complete atrioventricular block, permanent pacing is reasonable (S7.2-7–S7.2-11).
IIa	B-NR	6. In adults with repaired ACHD who require permanent pacing for bradycardic indications, a bradycardia device with atrial antitachycardia pacing capabilities is reasonable (S7.2-14, S7.2-15).
IIa	C-EO	7. In adults with ACHD with preexisting sinus node and/or atrioventricular conduction disease who are undergoing cardiac surgery, intraoperative placement of epicardial permanent pacing leads is reasonable.
IIb	B-NR	8. In adults with ACHD and pacemakers, atrial-based permanent pacing for the prevention of atrial arrhythmias may be considered (S7.2-3–S7.2-5, S7.2-16).
III: Harm	B-NR	9. In selected adults with ACHD and venous to systemic intracardiac shunts, placement of endocardial pacing leads is potentially harmful (S7.2-17, S7.2-18).

7.3. Management of Bradycardia in Patients With an Acute MI

Recommendations for Management of Bradycardia in the Context of Acute MI Referenced studies that support recommendations are summarized in Online Data Supplement 54.		
COR	**LOE**	**Recommendations**
I	B-NR	1. In patients presenting with an acute MI, temporary pacing is indicated for medically refractory symptomatic or hemodynamically significant bradycardia related to SND or atrioventricular block (S7.3-1–S7.3-4).
I	B-NR	2. Patients who present with SND or atrioventricular block in the setting of an acute MI should undergo a waiting period before determining the need for permanent pacing (S7.3-1, S7.3-4–S7.3-7).
I	B-NR	3. In patients presenting with an acute MI with second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, alternating bundle branch block, or third-degree atrioventricular block (persistent or infranodal), permanent pacing is indicated after a waiting period (S7.3-7, S7.3-8).
IIa	B-NR	4. In patients with an acute MI with symptomatic or hemodynamically significant sinus bradycardia or atrioventricular block at the level of the atrioventricular node, the administration of atropine is reasonable (S7.3-9–S7.3-11).
III: Harm	B-NR	5. In patients with an acute MI and transient atrioventricular block that resolves, permanent pacing should not be performed (S7.3-1, S7.3-4, S7.3-7, S7.3-12–S7.3-16).
III: Harm	B-NR	6. In patients with an acute MI and a new bundle branch block or isolated fascicular block in the absence of second-degree or third-degree atrioventricular block, permanent pacing should not be performed (S7.3-17–S7.3-19).

7.4. Neurologic Disorders

7.4.1. Epilepsy

Recommendation for Patients With Epilepsy and Symptomatic Bradycardia Referenced studies that support the recommendation are summarized in Online Data Supplement 55.		
COR	**LOE**	**Recommendation**
IIa	C-LD	1. In patients with epilepsy associated with severe symptomatic bradycardia (ictal bradycardia) where antiepileptic medications are ineffective, permanent pacing is reasonable for reducing the severity of symptoms (S7.4.1-1–S7.4.1-4).

8. Evaluation of the Risks for Ventricular Arrhythmias in Patients Who Require Permanent Pacing

Recommendation for Management of Bradycardia and Conduction Tissue Disease in Patients Who Require Pacing Therapy and May Also Be at Risk for Ventricular Arrhythmias Referenced studies that support the recommendation are summarized in Online Data Supplement 56.		
COR	**LOE**	**Recommendation**
I	**B-NR**	**1. In patients who require permanent pacing therapy, before implantation, an assessment of the risk of future ventricular arrhythmias and need for an implantable cardioverter defibrillator should be performed (S8-1–S8-7).**

9. Shared Decision-Making

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 (S9-1–S9-6).**
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 (S9-1–S9-6).**
III: No Benefit	**C-LD**	**3. In patients with indications for permanent pacing but also with significant comorbidities 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 (S9-1–S9-6).**

10. Discontinuation of Pacemaker Therapy

Recommendation for Discontinuation of Pacemaker Therapy		
COR	**LOE**	**Recommendation**
IIa	**C-LD**	**1. In patients who present for pacemaker pulse generator replacement, or for management of pacemaker related complications, in whom the original pacing indication has resolved or is in question, discontinuation of pacemaker therapy is reasonable after evaluation of symptoms during a period of monitoring while pacing therapy is off (S10-1, S10-2).**

Presidents and Staff

Key Words: ACC/AHA Clinical Practice Guidelines ■ ablation ■ ambulatory electrocardiography ■ aminophylline ■ atrioventricular block ■ atropine ■ AV block ■ beta-adrenergic agonist ■ bradyarrhythmia ■ bradycardia ■ bundle branch block ■ cardiac pacing ■ cardiac resynchronization therapy ■ cardiac sinus pause ■ cardiac surgery ■ congenital heart disease ■ digoxin antibodies Fab fragments ■ electrocardiogram ■ glucagon ■ heart block ■ Holter monitoring ■ intraoperative ■ isoproterenol ■ lamin A-C ■ left bundle branch block ■ muscular dystrophies ■ myocardial infarction ■ myotonic dystrophy ■ pacemaker ■ pacing ■ preoperative ■ quality of life ■ right bundle branch block ■ sarcoidosis ■ shared decision making ■ sick sinus syndrome ■ sinus arrest ■ sinus bradycardia syndrome ■ sinus node dysfunction ■ spinal cord injuries ■ syncope ■ theophylline ■ transcatheter aortic valve replacement.

Appendix 1. Author Relationships With Industry and Other Entities (Relevant)—2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay (July 2018)

Committee Member	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusals by Section*
Fred M. Kusumoto *(Chair)*	Mayo Clinic—Professor of Medicine	None	None	None	None	None	None	None
Mark H. Schoenfeld *(Vice Chair)*	Yale University School of Medicine—Clinical Professor of Medicine	None	None	None	None	None	None	None
Coletta C. Barrett	American Heart Association—Chairman of the Board	None	None	None	None	None	None	None
James R. Edgerton	The Heart Hospital Baylor—Director of Education	None	None	None	None	None	None	None
Kenneth A. Ellenbogen	VCU Medical Center—Director, Clinical Electrophysiology	• Biotronik† • Boston Scientific† • Janssen Pharmaceuticals • Medtronic† • Pfizer† • St. Jude Medical†	• Biosense Webster† • Biotronik† • Boston Scientific† • Medtronic† • St. Jude Medical†	None	• Biosense Webster† • Boston Scientific† • Medtronic† • Sanofi-Aventis† • Medtronic (DSMB)†	• Biosense Webster† • Boston Scientific† • Medtronic† • Sanofi-Aventis	None	4.3.1, 4.3.2, 5, 6, 7, 8, 9, 11, 13
Michael R. Gold	Medical University of South Carolina—Director, Division of Cardiology and Professor of Medicine	• Boston Scientific† • Medtronic • St. Jude Medical	None	None	• Boston Scientific‡ • St. Jude Medical‡	None	None	4.3.1, 5, 6, 7, 8, 9, 11, 13
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*Writing committee members are required to recuse themselves from voting on sections to which their specific relationships with industry and other entities may apply. Section numbers pertain to those in the full-text guideline.
†Significant relationship.
‡No financial benefit.

CMS reported payments from Cardiofocus to Dr. Kusumoto in 2016 and 2017. Dr. Kusumoto has established that the study he participated in ended in 2014 and was published in 2015.
CMS reported consulting payments to Dr. Lee from Abbott, Cryolife and Maquet in 2016. Dr. Lee has established that his participation with the companies ended in January 2015.
CMS reported research payments from Medtronic to Dr. McLeod in 2016 and 2017. Dr. McLeod is disputing the payments.

ACC indicates American College of Cardiology; AHA, American Heart Association; DSMB, data safety monitoring board; HRS, Heart Rhythm Society; UT, University of Texas; VA, Veterans Affairs; and VCU, Virginia Commonwealth University.

Appendix 2. Abbreviated Reviewer Relationships With Industry and Other Entities— 2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay (August 2018)*

Reviewer	Representation	Employment	Comprehensive RWI?
Yong-Mei Cha	Official Reviewer—AHA	Mayo Clinic, Division of Cardiovascular Diseases	No
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Richard J. Kovacs	Official Reviewer—ACC Science and Quality Committee	Indiana University School of Medicine, Krannert Institute of Cardiology—Professor of Clinical Medicine	Yes
Kevin F. Kwaku	Official Reviewer—AHA	Dartmouth-Hitchcock Medical	No
Daniel M. Philbin Jr	Official Reviewer—ACC Board of Governors	New England Heart Institute	Yes
Peter A. Brady	Organizational Reviewer—HRS	Mayo Clinic, Mayo Foundation	No
Ratika Parkash	Organizational Reviewer—HRS	Dalhousie University and Nova Scotia Health Authority—Professor of Medicine, Division of Cardiology (Arrhythmia); Director of Research, Division of Cardiology	Yes
Jonathan Philpott	Organizational Reviewer—STS	Mid-Atlantic Cardiothoracic Surgeons	Yes
Kevin Shannon	Organizational Reviewer—PACES	Mattel Children's Hospital at UCLA—Clinical Professor, Division of Pediatric Cardiology	Yes
Gus J. Vlahakes	Organizational Reviewer—AATS	Harvard Medical School and Massachusetts General Hospital—Professor of Surgery	No
Nazem Akoum	Content Reviewer	University of Washington	No
Sana M. Al-Khatib	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Duke Clinical Research Institute— Professor of Medicine	Yes
Joshua A. Beckman	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Vanderbilt University Medical Center— Director, Section of Vascular Medicine	Yes
Kim K. Birtcher	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	University of Houston College of Pharmacy—Clinical Professor	Yes
Mitchell I. Cohen	Content Reviewer—PACES	Pediatric Cardiology Associates	No
Freddy Del-Carpio Munoz	Content Reviewer—ERC Member	Mayo Clinic	No
Bernard Dennis	Content Reviewer—ACC/AHA Lay Reviewer	Dennis Associates, LLC	No
Anita Deswal	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Michael E. DeBakey VA Medical Center—Chief, Cardiology; Baylor College of Medicine—Professor of Medicine	Yes
Andrew E. Epstein	Content Reviewer	The Hospital of the University of Pennsylvania—Professor of Medicine	Yes
Michael E. Field	Content Reviewer	University of Wisconsin School of Medicine and Public	No

		Health—Director, Clinical Electrophysiology and Cardiac Arrhythmia Service	
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Federico Gentile	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Centro Cardiologico Gentile	No
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Bulent Gorenek	Content Reviewer	Eskisehir Osmangazi University Cardiology Department—Professor of Cardiology	Yes
Mohamed H. Hamdan	Content Reviewer	University of Wisconsin-Madison— Chief of Cardiovascular Medicine	Yes
Glenn N. Levine	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Baylor College of Medicine— Professor of Medicine; Michael E. DeBakey Medical Center—Director, Cardiac Care Unit	Yes
Mark S. Link	Content Reviewer	UT Southwestern Medical Center	Yes
Daniel L. Lustgarten	Content Reviewer	University of Vermont School of Medicine	Yes
Siva K. Mulpuru	Content Reviewer—ERC Member	Mayo Clinic—Associate Professor	Yes
Patrick T. O'Gara	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Harvard Medical School—Professor of Medicine; Brigham and Women's Hospital—Director, Strategic Planning	Yes
Brian Olshansky	Content Reviewer	Professor of Medicine	Yes
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*Detailed reviewer disclosures can be found at this link [insert link here].

AATS indicates American Association for Thoracic Surgery; ACC, American College of Cardiology; AHA, American Heart Association; CCEP, Clinical Cardiac Electrophysiology; ERC, evidence review committee; HRS, Heart Rhythm Society; LLC, limited liability company, NYU, New York University; PACES, Pediatric & Congenital Electrophysiology Society; STS, the Society of Thoracic Surgeons; UCLA, University of California, Los Angeles; UT, University of Texas; and VA, Veterans Affairs.

References

Preamble

P-1. Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS 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. J Am Coll Cardiol. 2018;In press.

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