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REVIEW ARTICLE

Pacemakers

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Abstract

Pacemakers are critical in the management of bradyarrhythmias. The authors review pacemaker fundamentals, indications, and device types, as well as the role of pacemakers in heart failure, emerging advancements, and current limitations.

C. Corey Hardin, M.D. Ph.D., Editor

Introduction

Pacemakers have transformed the management of bradycardia and heart block, offering the only definitive therapy for nonreversible bradyarrhythmias. Cardiac pacing restores appropriate heart rates and heart-rate response to normalize hemodynamics and reestablish effective circulation. Over 1 million pacemakers are implanted annually worldwide, a number expected to increase amid an aging population and increased prevalence of cardiovascular comorbidities.[1,2] Advances in pacemaker technology have improved patient outcomes and expanded their use across a broad spectrum of cardiovascular disorders. From conventional single- and dual-chamber devices to innovations, such as leadless pacemakers, biventricular pacing to deliver cardiac-resynchronization therapy (CRT), and His-bundle and left bundle-branch area pacing, pacemakers are a cornerstone of modern cardiovascular care.[3,4] This review examines the fundamental principles of pacemakers, current indications for pacemaker implantation, types of devices, the role of pacemakers in heart-failure management, emerging advancements in pacemaker therapy, and the ongoing challenges and limitations within this evolving field.

Basic Pacemaker Principles

The traditional transvenous pacemaker consists of three primary components: a pulse generator that produces electrical impulses, leads that deliver these impulses to the heart, and electrodes that both sense the intrinsic heart rhythm and deliver the electrical impulses to the myocardium when necessary. The pulse generator is typically implanted in the infraclavicular region in a prepectoral position, although subpectoral implantation is occasionally used.

NOMENCLATURE

In 1974, the American Heart Association and the American College of Cardiology introduced a coding system to describe pacemaker function, which was later refined by the North American Society of Pacing and Electrophysiology and the British Pacing and

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Table 1. NBG Code for Pacing Nomenclature.*

Position I	Position II	Position III†	Position IV	Position V
Chamber(s) paced	Chamber(s) sensed	Response to sensing	Rate modulation	Multisite pacing
A=Atrium	A=Atrium	I=Inhibited	R=Rate modulation	A=Atrium
V=Ventricle	V=Ventricle	T=Triggered	O=None	V=Ventricle
D=Dual	D=Dual	D=Dual		D=Dual
O=None	O=None‡	O=None‡		O=None

* The NGB code uses up to five letters to denote various functions. When fewer than five characters are present, omitted positions are assumed to be "O", or absent.
† I denotes inhibited pacing, where detection of a spontaneous beat prevents pacing; T denotes triggered pacing in response to a sensed event; and D denotes dual response, where atrial sensing inhibits atrial pacing but triggers ventricular pacing after a programmable delay (mimicking the PR interval), or where ventricular pacing is inhibited if intrinsic ventricular activity is detected during the delay.
‡ O in positions II and III indicates no sensing, allowing the device to pace at a fixed rate regardless of intrinsic rhythm.

Electrophysiology Group.[5] This system, known as the NBG code for pacing nomenclature, uses up to five letters to denote various functions, as shown in Table 1. When fewer than five characters are present, omitted positions are assumed to be "O", or absent.

PACING MODES

Selecting the optimal pacing takes into account the patient's underlying arrhythmia, exercise capacity, chronotropic response, left ventricular function, and comorbidities. The primary single-chamber pacing modes are atrial demand pacing (AAI/AAIR) and ventricular demand pacing (VVI/VVIR). In atrial demand pacing, the atrium is paced and sensed, with pacing output inhibited when intrinsic atrial activity is detected. Ventricular demand pacing involves pacing and sensing in the ventricle, with pacing output inhibited when a ventricular event is sensed. VVI/VVIR pacing is versatile, protecting against bradycardias of any origin, but may cause atrioventricular (AV) dyssynchrony, leading to pacemaker syndrome.

The principal pacing modes for dual-chamber pacemakers are DDD/DDDR and DDI/DDIR. Both modes involve atrial and ventricular sensing and pacing; however, they differ in their response to atrial activity. In DDI/DDIR mode, a sensed atrial event does not initiate a ventricular paced response after the programmed AV delay, resulting in the absence of atrial tracking and loss of AV synchrony. In contrast, DDD/DDDR mode maintains AV synchrony by coordinating atrial and ventricular activity.

Although asynchronous pacing modes (AOO, VOO, or DOO), where the pacemaker delivers fixed-rate pacing without sensing the intrinsic rhythm, are rarely used in the long term, they are frequently employed during surgery or magnetic resonance imaging (MRI), when sensing may be unreliable. For example, electrocautery signals may be misinterpreted as intrinsic cardiac activity, leading to inappropriate inhibition of pacing. However, asynchronous pacing carries the risk of competition between intrinsic and paced rhythms, with the theoretical potential for a paced impulse to fall on the vulnerable period of a native T wave, precipitating ventricular arrhythmias. This theoretical risk is reduced by pacing at faster rates.

Contemporary Pacemakers

TRANSVENOUS PACEMAKERS

Recent advances in pacing technology have expanded the options beyond traditional systems, underscoring the need to tailor pacemaker selection to optimize patient outcomes.

A summary of these devices is provided in Figure 1. Transvenous pacemakers, which remain the most commonly implanted, include single-chamber systems (with a lead in either the atrium or ventricle) and dual-chamber systems (with leads in both chambers).

LEADLESS PACEMAKERS

An important innovation is the leadless pacemaker, a self-contained device implanted directly into the heart. This approach eliminates the need for transvenous leads, thereby reducing lead-associated risks such as infection and venous stenosis.[6] Initially limited to right ventricular pacing, leadless pacemakers now offer the capability to sense atrial and ventricular activity (VDDR) and provide AAIR, VVIR, and DDDR.[7] These devices are typically implanted percutaneously via a transfemoral approach, although transjugular access may be used when necessary.[8] The safety and efficacy of leadless pacemakers have been

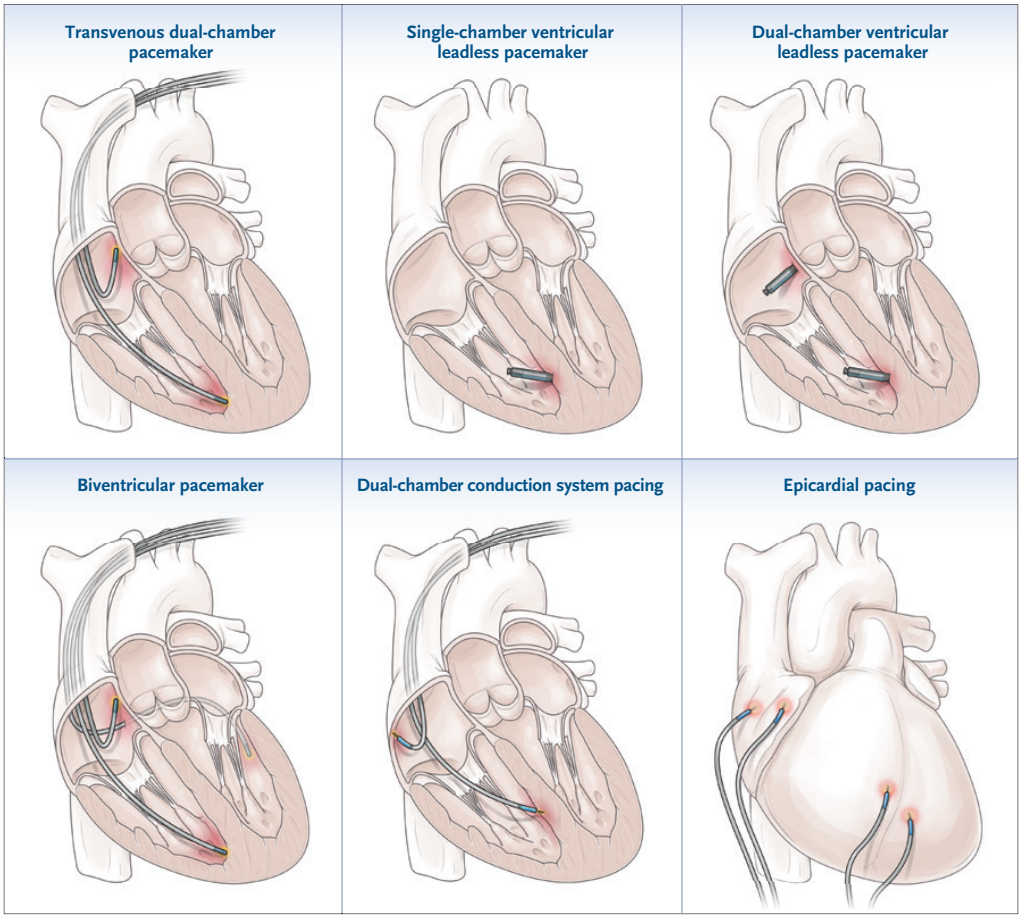

Figure 1. Types of Pacemakers.

This figure depicts the various types of pacemakers approved for clinical use.

demonstrated in multiple prospective, nonrandomized, multicenter studies.[9-14] A summary of transvenous versus leadless pacemakers is presented in Table 2.[15-21]

CARDIAC RESYNCHRONIZATION THERAPY

Although traditional and leadless pacemakers can maintain AV synchrony, they cannot provide interventricular synchrony, as pacing occurs exclusively from the right ventricle. To address this limitation, cardiac physiological pacing, which encompasses both CRT and conduction-system pacing (CSP), was developed. CRT involves pacing both ventricles to improve ventricular synchrony. The left ventricle is paced via a coronary-sinus lead, an epicardial lead, or a leadless device within the left ventricle.[22,23] This approach is particularly beneficial in patients with systolic heart failure and left bundle-branch block, as discussed in detail in the following section on conduction system pacing.[22,24,25] However, coronary-sinus lead placement is not always feasible (e.g., owing to venous stenosis, scar tissue, high pacing thresholds, diaphragmatic stimulation, or congenital anomalies), and the additional lead can increase the risk of venous obstruction and earlier battery depletion.[26]

CONDUCTION SYSTEM PACING

More recently, CSP has emerged as an additional or alternative option to CRT, with the aim of activating the His–Purkinje system and improving ventricular synchrony. His-bundle pacing — one approach to CSP — involves positioning the lead in the proximal septum to capture the His bundle.[27] Although promising, this type of pacing has been limited by technical challenges, including lead stability with higher rates of intervention for lead dislodgement, atrial oversensing, and high capture thresholds resulting in more rapid battery depletion.[28-30] While previous investigations have focused on CRT-indicated patients with a left bundle-branch block,[31,32,33] the His–CRT trial is evaluating

Table 2. Characteristics of Transvenous and Leadless Pacemakers.

Characteristic	Transvenous Pacemaker	Leadless Pacemaker
Implant complications	Valve injury or damage Pocket hematoma Lead dislodgement Hemo/pneumothorax	Vascular injury Groin bleeding or hematoma Device embolization Cardiac perforation or tamponade
Infection	High risk	Low risk
Extraction	Significant experience	Limited experience
Atrial pacing	Capable	Capable
Cardiac resynchronization	Capable	No
Recovery	Prolonged	Short
Battery life*	Up to 17 years (median, 10.8)	Up to 27 years (median, 12.1)
Remote monitoring	Capable	Only one type (Micra) currently capable
MRI-conditional	Yes	Yes
Cost	Low	High

*Battery life is less for dual-chamber leadless pacemakers when they are programmed to communicate, with a mean of 6.4 years for the atrial device and 11.3 years for the ventricular device.[15] MRI denotes magnetic resonance imaging.

His-bundle pacing in CRT-indicated patients with a right bundle-branch block.[34] Left bundle-branch area pacing (LBBAP) has emerged as another form of CSP with the goal of restoring native conduction by capturing the left bundle-branch to improve interventricular synchrony. LBBAP involves placing a lead in the proximal right ventricular septum, distal to the His bundle, and confirming left bundle-branch capture through testing. LBBAP has achieved high success rates, with QRS narrowing below 120 milliseconds, and is associated with improvements in left ventricular ejection fraction and heart-failure symptoms.[35] Compared with His-bundle pacing, LBBAP has demonstrated lower complication rates, better pacing thresholds, and reduced atrial oversensing; however, it carries a higher risk of septal perforation owing to the more distal and deeper positioning of the lead in the septum.[36] Ongoing large randomized clinical trials evaluating LBBAP are summarized in Table 3. Although pacing alternative atrial sites as an alternative means of physiological pacing has been postulated to reduce the progression of atrial arrhythmias, this did not bear out in a meta-analysis of randomized clinical trials.[37] However, interest in Bachman's Bundle pacing has recently been reinvigorated using an electrogram-guided approach, which is being actively investigated.[38]

EPICARDIAL PACEMAKERS

For patients in whom venous-access limitations preclude the use of transvenous or leadless pacemakers, epicardial leads (atrial, right ventricular, and/or left ventricular) may be surgically placed via a minithoracotomy at the fourth or fifth intercostal space, anterior to the midaxillary line. Unlike transvenous leads, epicardial leads offer more flexible positioning, as they are not confined to the anatomic branches of the left ventricular venous circulation. However, given their higher complication risk and lack of clear benefit over transvenous leads, epicardial leads are generally reserved for patients who are not candidates for transvenous leads.[39,40]

ADDITIONAL FEATURES

Modern pacemakers offer several advanced features beyond the basic pacing modes. Rate-responsive sensors adjust the pacing rate in response to patient activity through various mechanisms, including the detection of body motion, minute ventilation, or changes in right ventricular impedance.[41,42] Traditional right ventricular pacing causes the right ventricle to contract before the left ventricle, simulating the effects of a left bundle-branch block. This results in ventricular dyssynchrony, which in some patients can cause or worsen heart failure and increase the frequency of atrial fibrillation.[43-45] Specialized pacing modes to minimize ventricular pacing have been developed to address this.[46-49] All modern pacemakers also offer remote monitoring and MRI compatibility, as discussed in the following section on pacemaker indications.

Pacemaker Indications

Guidelines for implantation of cardiac pacemakers have been published jointly by the American College of Cardiology, the American Heart Association, and the Heart Rhythm Society (HRS), with similar guidelines established by the European Society of Cardiology.[3,4] These guidelines

Table 3. Summary of Ongoing Large Randomized Clinical Trials Evaluating Left Bundle-Branch Area Pacing.*

Trial Name	ClinicalTrials.gov ID	Trial Site(s)	Estimated Enrollment	Follow-Up	Estimated Completion	Trial Group	Primary Outcome(s)
Left versus Left Randomized Clinical Trial	NCT05650658	12 locations across the United States and Canada	2136	5.5 years	June 2029	His or LBBAP versus CRT in patients with LVEF≤50% and either a wide QRS (≥130 milliseconds) or with current or anticipated >40% pacing who are already receiving current standard heart-failure medical therapy	A combined clinical end point of all-cause mortality and hospitalization for heart failure
Conduction-System Pacing with Left Bundle-Branch Pacing as Compared to Standard Right Ventricular Pacing	NCT05015660	Single center in Canada	1300	3 years	January 2027	LBBAP versus standard right ventricular pacing in patients with high-degree AV block	Time to cardiovascular death, time to first heart-failure event, and worsening LVESVi at 2 years
Protection of Cardiac Function with Left Bundle-Branch Pacing in Patients with AV Block (OptimPacing)	NCT04624763	Single center in China	683	3 years	June 2029	LBBAP versus right ventricular pacing	Combined all-cause mortality, hospitalization for heart failure and/or occurrence of pacing-induced cardiomyopathy
Physiological Ventricular Pacing versus Managed Ventricular Pacing for Persistent AF Prevention in Prolonged AV Interval (PhysioVP-AF)	NCT05367037	Single center in Italy	640	3 years	December 2028	His or LBBAP versus dual-chamber pacing with the addition of algorithms for ventricular pacing avoidance in patients with sinus-node disease or paroxysmal type 1 or 2 second-degree AV block	Freedom from persistent AF and a composite of death from cardiovascular disease, heart failure, or pacing system upgrading to conduction-system pacing or to CRT
Impact of Left Bundle-Branch Area Pacing versus Right Ventricular Pacing in AV Block (LEAP-Block)	NCT04730921	8 locations across China	458	2 years	December 2025	LBBAP versus right ventricular pacing in patients with AV block	Time to a first event of composite all-cause death, hospitalization for heart failure, and an upgrade to CRT as a result of pacing-induced heart failure
Preventive Effect of Left Bundle-Branch Area Pacing versus Right Ventricular Pacing on All-Cause Death, Heart-Failure Progression, and Ventricular Dyssynchrony in Patients with Substantial Ventricular Pacing (PROTECT-SYNC)	NCT05585411	8 locations across South Korea	450	2 years	November 2026	LBBAP versus right ventricular pacing in patients with bradyarrhythmias who require a high burden of ventricular pacing (>40%)	Composite of all-cause death, heart-failure hospitalization, occurrence of pacing-induced cardiomyopathy, and an upgrade to CRT
Conduction-System Pacing versus Biventricular Pacing in Systolic Dysfunction and Wide QRS: Mortality, Heart-Failure Hospitalization or Cardiac Transplant (CONSYST-CRT II)	NCT06105580	Single center in Spain	320	1 year	November 2027	His bundle or LBBAP versus CRT in patients with left bundle-branch block, QRS≥130, and LVEF≤35%	Composite end point of all-cause mortality, cardiac transplant, or heart-failure hospitalization

* AF denotes atrial fibrillation; AV, atrioventricular; CRT, cardiac-resynchronization therapy; LBBAP, left bundle-branch area pacing; LVEF, left ventricular ejection fraction; and LVESVi, left ventricular end-systolic volume index.

Table 4. Summary of U.S. and European Guideline Recommendations for Common Indications for Permanent Pacing.*

Disorder	ACC/AHA/HRS Class†	ACC/AHA/HRS Level‡	ESC Class†	ESC Level§	Indication for Permanent Pacing	Recommended Pacing Mode
Sinus-node dysfunction	I	C-LD	I	A	Symptoms that are directly attributable to sinus-node dysfunction	AAI if there is no evidence of AV-node disease DDD in setting of concomitant AV-node disease DDDR if there is chronotropic incompetence DDIR if episodes of supraventricular tachycardia present
	I	C-EO	–	–	Symptomatic sinus bradycardia due to guideline-directed management and therapy for which there is no alternative treatment and continued treatment is clinically necessary	
	IIa	C-EO	I	B	Tachy-brady syndrome with symptoms attributable to bradycardia	
	IIa	C-EO	IIa	B	Symptomatic chronotropic incompetence	
	–	–	IIb	C	Sinus-node dysfunction with asymptomatic pauses >6 seconds in the setting of unexplained syncope	
	–	–	IIb	C	Sinus bradycardia with symptoms suggestive of bradycardia but without conclusive evidence	
	III	C-LD	–	–	In asymptomatic individuals with sinus bradycardia or pauses that are secondary to physiologically elevated parasympathetic tone	
	III	C-LD	–	–	Sleep-related sinus bradycardia or sinus pauses occurring during sleep	
	III	C-LD	III	C	Sinus-node dysfunction that is asymptomatic or due to transient causes, or in those in whom the symptoms have been documented to occur in the absence of bradycardia or chronotropic incompetence	
AV block	I	B-NR	I	C	Acquired paroxysmal or permanent second-degree Mobitz type II, infranodal 2:1, high-grade AV block, or third-degree AV block not attributable to reversible or physiological causes	VVI if no organized atrial activity present VVIR if there is chronotropic incompetence DDD if sinus-node function is preserved DDDR if there is chronotropic incompetence
	I	C-LD	–	–	Symptomatic AV block in patients receiving essential evidence-based therapies with no effective alternative	
	I	C-LD	–	–	Permanent atrial fibrillation with symptomatic bradycardia	
	I	B-NR	I	C	Neuromuscular diseases associated with conduction disorders (e.g., myotonic dystrophy type 1) or Kearns–Sayre syndrome, with evidence of second- or third-degree AV block or an HV interval ≥70 milliseconds	
	IIa	C-LD	IIa	C	Marked first-degree AV block (PR interval >300 milliseconds) or second-degree Mobitz I AV block that is symptomatic or found to be at the intra- or infra-Hisian level on EPS	

(*Continued*)

Table 4. (*Continued*) Summary of U.S. and European Guideline Recommendations for Common Indications for Permanent Pacing.*

Disorder	ACC/AHA/HRS Class†	ACC/AHA/HRS Level‡	ESC Class†	ESC Level§	Indication for Permanent Pacing	Recommended Pacing Mode
	IIa	B-NR	IIa	C	Infiltrative cardiomyopathies (e.g., cardiac sarcoidosis) with second-degree Mobitz type II, infranodal 2:1, or third-degree AV block	
	IIa	B-NR	IIa	C	Lamin A/C gene mutations, including limb-girdle and Emery–Dreifuss muscular dystrophies, with a PR interval >240 milliseconds and left bundle-branch block	
	IIb	C-LD	IIb	C	Patients with neuromuscular diseases, such as myotonic dystrophy type 1, with a PR interval >240 milliseconds, QRS duration >120 milliseconds, or fascicular block	
	III	C-LD	III	C	AV block due to transient causes (e.g., vagally mediated AV block) that can be corrected and prevented	
	III	C-LD	–	–	Asymptomatic first-degree AV block or second-degree Mobitz type I or 2:1 AV block believed to be at the level of the AV node	
Acute myocardial infarction	I	B-NR	I	C	Acute myocardial infarction with second-degree Mobitz type II AV block, high-grade AV block, third-degree AV block, or alternating bundle-branch block after a waiting period ≥ 5 days	VVI if no organized atrial activity present VVIR if there is chronotropic incompetence DDD if sinus-node function is preserved DDDR if there is chronotropic incompetence
Bundle-branch block	I	C-LD	I	B	Alternating bundle-branch block	VVI if no organized atrial activity present VVIR if there is chronotropic incompetence DDD if sinus-node function is preserved DDDR if there is chronotropic incompetence
	I	C-LD	I	B	Syncope and bundle-branch block or bifascicular block with an HV interval ≥70 milliseconds, second- or third-degree intra- or infra-Hisian block during incremental atrial pacing, or abnormal response to pharmacological challenge	
Congenital heart disease	I	B-NR	I	C	Adults with symptomatic sinus-node dysfunction, chronotropic incompetence, AV, or any symptomatic bradycardia with a wide QRS escape rhythm, pauses >3× the cycle length of the ventricular escape rhythm, mean daytime heart rate below 50 beats per minute, prolonged QT interval, complex ventricular ectopy, or ventricular dysfunction attributed to hemodynamic compromise due to bradycardia	Same as AV block
	IIa	B-NR	IIb	C	Asymptomatic congenital complete or high-grade AV block	

(*Continued*)

Table 4. (*Continued*) Summary of U.S. and European Guideline Recommendations for Common Indications for Permanent Pacing.*

Disorder	ACC/AHA/HRS Class†	ACC/AHA/HRS Level‡	ESC Class†	ESC Level§	Indication for Permanent Pacing	Recommended Pacing Mode
Neurocardiogenic syncope	IIb	B-R	I	A	To reduce recurrent syncope in patients aged >40 years with severe, unpredictable, recurrent syncope who have: spontaneous documented symptomatic asystolic pause(s) >3 seconds or asymptomatic pause(s) >6 seconds due to sinus arrest or AV block; cardioinhibitory carotid-sinus syndrome; or asystolic syncope during tilt testing	DDD if sinus node function is preserved DDDR if there is chronotropic incompetence

* ACC denotes American College of Cardiology; AHA, American Heart Association; AV, atrioventricular; EPS, electrophysiology study; ESC, European Society of Cardiology; HRS, Heart Rhythm Society; and HV, His-ventricular.

† Class I (green) recommendations are recommended or indicated, IIa (yellow) should be considered, IIb (orange) may be considered, and III (red) is not recommended.

‡ For the U.S. guidelines, level A (teal blue) is high-quality evidence from more than one randomized controlled trial or meta-analyses, or one or more randomized controlled trials corroborated by high-quality registry studies; B-R (sky blue) is moderate-quality evidence from one or more randomized controlled trials or meta-analyses; B-NR (sky blue) is moderate-quality evidence from one or more well-designed and executed nonrandomized studies or meta-analyses of such studies; C-LD (pale blue) is evidence derived from randomized or nonrandomized studies with limitations of design or execution, or meta-analyses of these studies; and C-EO (pale blue) is consensus of expert opinion.

§ For the European guidelines, level A (teal blue) is data derived from multiple randomized clinical trials or meta-analyses; level B (dusty teal) is data derived from a single randomized clinical trial or large nonrandomized studies; and level C (frost blue) is the consensus of opinion of the experts and/or small studies, retrospective studies, and registries.

are divided into three classes: class I are conditions in which permanent pacing is definitely beneficial, useful, and effective; class II are conditions in which permanent pacing may be indicated, but with conflicting evidence and/or differences of opinion; and class III are conditions in which permanent pacing is not useful or effective, and, in some cases, may be harmful. Both guidelines' recommendations for common disorders requiring permanent pacing are summarized in Table 4.

SINUS-NODE DYSFUNCTION

The pathophysiology of sinus-node dysfunction involves complex electrophysiologic and structural remodeling. The decision to implant a permanent pacemaker for sinus-node dysfunction is based entirely on the presence of symptoms. While heart rates below 40 beats per minute and pauses exceeding 4 seconds are more likely to be associated with symptoms, there is no absolute threshold for heart rate or pause duration that necessitates pacemaker implantation, particularly if the bradycardia occurs during sleep. The class I to III indications for pacemaker implantation in sinus-node dysfunction are summarized in Table 4.

ATRIOVENTRICULAR BLOCK

AV block is most often an acquired condition arising from various etiologies, including infectious, inflammatory, degenerative, ischemic, or iatrogenic causes. The decision to proceed with pacemaker implantation hinges primarily on the severity and irreversibility of the AV block. Furthermore, the underlying etiology, such as infiltrative or genetic cardiomyopathies, neuromuscular disorders, or congenital conduction abnormalities, plays an important role in determining the need for permanent pacing. The class I to III indications for pacemaker implantation in AV block are summarized in Table 4. An algorithm for determining the need for single- or dual-chamber pacing and the recommended pacing modes in sinus-node dysfunction and AV block is shown in Figure 2.

OTHER ARRHYTHMIAS REQUIRING PACING

Conduction abnormalities frequently arise in the context of myocardial infarction. In the acute phase, high-grade AV block, such as second-degree Mobitz II, infranodal 2:1, and third-degree AV block, is typically managed with temporary cardiac pacing if hemodynamic compromise occurs, with permanent pacing reserved for cases in which AV block persists. AV block following anterior myocardial infarction is less likely to resolve than that occurring with inferior myocardial infarction. Pacemaker implantation is also indicated in patients with alternating bundle-branch block or those with syncope and bundle-branch block with a His-ventricular interval greater than or equal to 70

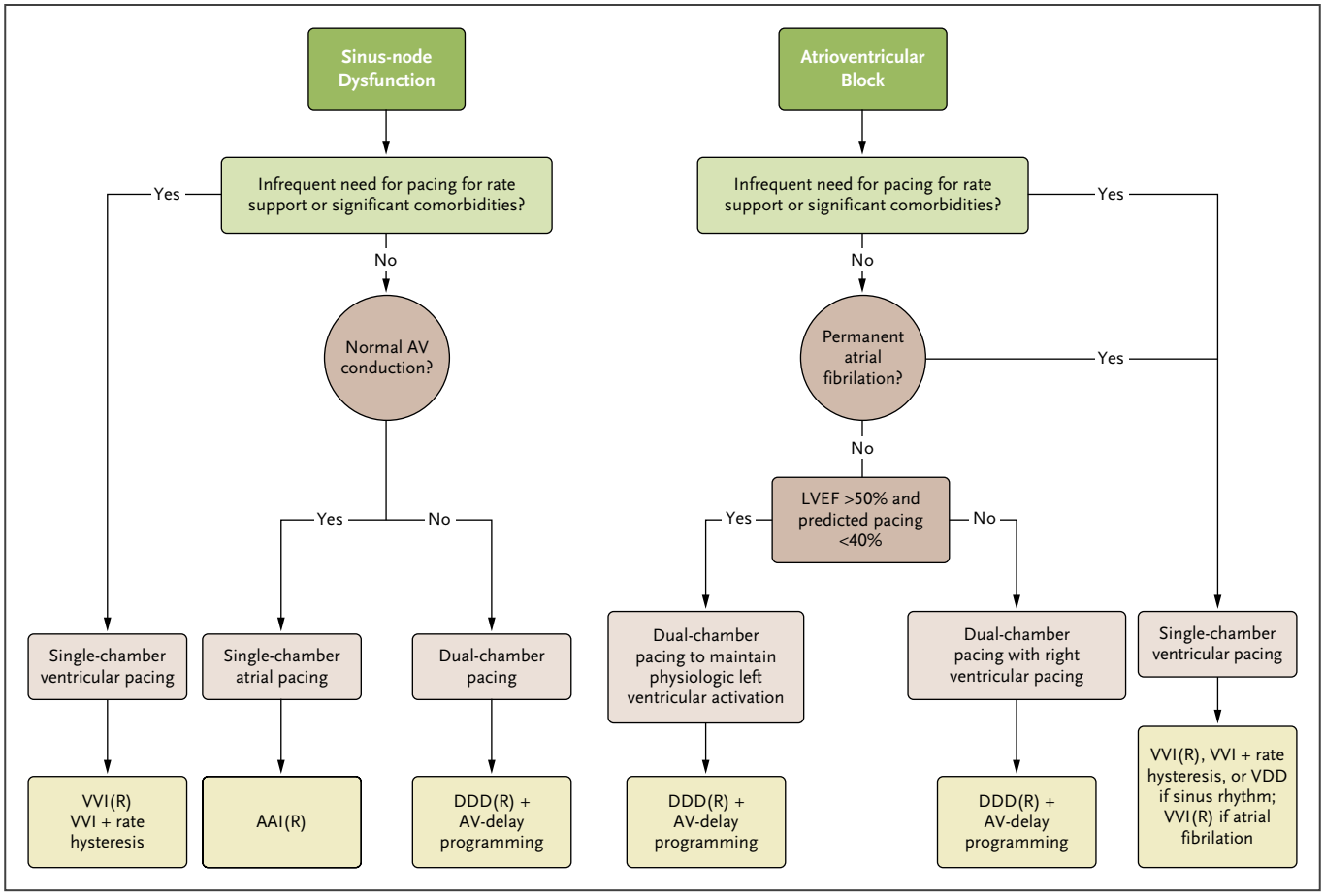

Figure 2. Algorithm to Determine Need for Single- versus Dual-Chamber Pacing and Recommended Pacing Modes in Sinus-Node Dysfunction and Atrioventricular Block.

(R) indicates that the programming of rate response is preferred in the case of chronotropic incompetence. AV-delay programming and other algorithms, such as rate hysteresis, are used to minimize ventricular pacing. AV denotes atrioventricular; and LVEF, left ventricular ejection fraction.

milliseconds, or if infranodal block is demonstrated on an electrophysiology study. As previously discussed, neuromuscular, infiltrative, and genetic conditions associated with AV block are strong indications for permanent pacing. The role of permanent pacing in asymptomatic patients with congenital complete heart block and a structurally normal heart is less well defined. Pacemaker implantation is recommended if high-risk features are present, such as a wide QRS complex, mean daytime heart rate below 50 beats per minute, complex ventricular ectopy, or ventricular dysfunction, but may be reasonable even in the absence of these risk factors. In addition, pacemakers are indicated in select patients with neurally mediated syncope when syncopal episodes are clearly linked to a significant cardioinhibitory or bradycardic response. The U.S. and European guidelines regarding common indications for permanent pacing and recommended pacing modes are summarized in Table 3.

Pacemakers in Heart-Failure Management

Evidence for CRT in patients with moderate to severe heart failure (New York Heart Association [NYHA] class II to IV), left bundle-branch block, and a reduced ejection fraction (≤35%) is well established.[50] Large clinical trials, such as the Multicenter Automatic Defibrillator Implantation Trial (MADIT) with CRT and the Resynchronization–Defibrillation for Ambulatory Heart Failure Trial (RAFT),

have shown a significant reduction in heart-failure events and hospitalization with biventricular pacing as an adjunct therapy to pharmacological management of heart failure.[51,52] For patients meeting criteria for an implantable cardioverter defibrillator and biventricular pacing, a defibrillator lead is placed in lieu of a right ventricular pacing lead, hence the important distinction between CRT-Pacemaker and CRT-Defibrillator. Improvement in heart-failure event rate after CRT device implantation remains challenging to define and is an area of debate. Patients are often grouped into CRT "responders" and "nonresponders". Several clinical factors, including advanced heart failure, ischemic cardiomyopathy, persistence of mechanical dyssynchrony, atrial fibrillation, QRS under 150 milliseconds, and ventricular arrhythmias, are among the predictors of poor response to biventricular pacing.[53]

In 2013, the Biventricular versus Right Ventricular Pacing in Heart-Failure Patients with AV Block (BLOCK HF) trial expanded the indications for biventricular pacing by including patients with AV block and a left ventricular ejection fraction of 50% or less. There is a known risk of left ventricular dysfunction with ongoing right ventricular apical pacing, and biventricular pacing was demonstrated to be superior for patients with a borderline or already reduced ejection fraction.[54] Although the indications for CRT have expanded, careful patient selection remains crucial. Investigations have shown that biventricular pacing in patients with a narrow QRS (<130 milliseconds) does not reduce heart-failure hospitalizations or mortality and may even be associated with increased mortality.[55,56] This has led to an update of the guidelines to give a class III (no benefit) recommendation for CRT in patients in sinus rhythm who have an ejection fraction less than or equal to 35%, a non-left bundle-branch block pattern with QRS duration less than 150 milliseconds, and NYHA class I or II symptoms on guideline-directed medical therapy.[22]

More recently, LBBAP emerged as a new alternative for patients with heart failure in whom coronary sinus anatomy is challenging and a left ventricular lead may not be implanted successfully. Although CRT remains the mainstay of therapy for patients meeting criteria for biventricular pacing, the guidelines on CSP highlight the role of LBBAP when effective CRT pacing cannot be achieved. In addition, they provide a class IIb indication to consider CSP strategies for patients in sinus rhythm who have a midrange ejection fraction (36 to 50%), left bundle-branch block with QRS duration greater than 150 milliseconds, and NYHA class II to IV symptoms on guideline-directed medical therapy[22]

Complications and Limitations

The overall rate of any immediate complication associated with transvenous pacemaker implantation is reported as approximately 5 to 10%, although this varies considerably across the literature. In the long term, the risk of complications is reported as 1 to 2% per year and is mainly related to lead failure and infection. Dual-chamber pacing systems have a higher rate of implant complication than single-chamber systems, the most common being atrial lead dislodgement. Other well-reported complications include pneumothorax from vascular access, hematoma formation, cardiac perforation, device malfunction, and infection. Less common complications include deep vein thrombosis, tricuspid-valve injury, and pulmonary embolism.[18,19] Device upgrades and generator replacements are associated with an increased risk of complications. Although the rate of major complications following transvenous lead insertion was previously reported to be as high as 15%, the rate of lead malfunction has been shown to be less than 5% at 10 years.[21,57] Leadless pacing systems avoid several complications related to leads and generators. Although there were initial safety concerns associated with leadless pacemakers, related to both cardiac tamponade and battery depletion, they are now considered largely safe and effective alternatives to transvenous systems in the right patient population. The main complications associated with leadless pacemakers are vascular injury given the large size of the delivery catheter, cardiac perforation or tamponade, and device dislodgement.[20] Longitudinal safety data are still lacking given novel leadless technologies still entering the market.

With expanding indications for pacemaker implantation and advances in technology, the need for device and lead extraction due to lead failure, device-related infection, or the need for system upgrade is increasingly common. The 2017 HRS Expert Consensus Statement on Cardiovascular Implantable Electronic Device Lead Management and Extraction recommends complete device and lead removal for all patients with definite, device-related staphylococcal infection, valvular endocarditis, or recurrent or persistent bacteremia despite adequate antibiotic therapy. Other indications for extraction include chronic severe pain at the device insertion site, and specific vascular or thrombosis issues related to transvenous leads.[58] Device and lead extraction procedures should be planned with the assistance of a cardiothoracic surgeon given the risk of sternotomy in the event of vascular laceration and/or cardiac

avulsion. Other major complications associated with lead extraction include hemothorax, pulmonary embolism, and tricuspid-valve injury requiring intervention.[59]

Recent Advances and Future Directions

Modern pacemakers use lightweight, lithium-based batteries designed to last over a decade. However, as patients live longer, many people outlive their pacemaker's battery life and may require multiple generator replacements over their lifetime. Rechargeable pacemakers represent a promising innovation. Although early attempts with nickel–cadmium batteries were discontinued owing to reliability issues, recent developments include wireless external charging devices and self-recharging generators that convert biomechanical energy from body motion and gravity to sustain device power.[60-62] These technologies, currently under investigation, have the potential to extend battery longevity, reduce the need for generator replacements, lower costs, and minimize environmental impact from battery waste.

In addition to advancements in battery technology, modern pacemakers have been designed to be MRI-compatible, enabling patients to safely undergo MRI scans without the risk of device malfunction. For patients with pacemakers that are not MRI-compatible, scans can still be conducted in a 1.5 Tesla magnet under strict protocols, including pre- and postscan device interrogation. Although abandoned leads are generally contraindications for MRI, even in the presence of MRI-conditional devices, certain imaging centers have protocols allowing these to be conducted in specialized settings.[63]

Pacemaker indications may broaden in the future, as ongoing research explores the potential benefits of pacing in emerging populations.[39,64] Furthermore, leadless pacemakers have recently been used for LBBAP, which could impact how cardiac physiological pacing is achieved moving forward. This device is currently being studied in the prospective Leadless CSP feasibility study.[65]

Remote-monitoring capabilities have revolutionized pacemaker management, enabling near-real-time data transmission to clinicians and facilitating earlier detection of complications, arrhythmias (such as atrial fibrillation or ventricular tachycardia), and device malfunctions. This approach reduces hospital visits and enhances patient outcomes through more proactive care. Remote monitoring is now wireless, with a communicator or transmitter that patients take home with them that will transmit data without being dependent on user input[66,67] Looking forward, the integration of remote monitoring with predictive algorithms powered by artificial intelligence and automated alerts could further optimize patient care by accurately forecasting battery depletion and improving emergency response times. Remote adjustments of pacing parameters may also become feasible, reducing the need for in-person follow-ups and allowing for greater individualization of management. Moreover, artificial intelligence–driven approaches could support highly personalized pacing strategies by tailoring device settings to each patient's unique physiology and activity patterns.[68,69]

Conclusion

Pacemakers have transformed the management of bradyarrhythmias, providing lifesaving therapy for patients with nonreversible conduction disorders. Advances in pacemaker technology, including MRI-compatible devices, leadless systems, and CSP, have broadened the range of clinical applications and enhanced patient outcomes. Emerging innovations in battery-life extension, remote monitoring, and artificial intelligence–driven personalization promise to further refine pacemaker therapy, minimizing health care burden and enabling more individualized care. As this field continues to evolve, ongoing research and innovation will remain critical to addressing remaining limitations and enhancing the quality of life for patients worldwide.

Disclosures

Author disclosures are available at evidence.nejm.org.

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