Bradycardia pacing

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Abstract

The number of patients with cardiac implantable devices for rhythm management has soared and the indications for their use have increased. This article aims to provide a practical review of pacemaker indications, and an overview of pacemaker function and malfunction, to demystify a device that has become commonplace in medical practice.

Keywords Arrhythmia; bradycardia; indications; MRCP; pacemaker

Key points

- Sinoatrial and atrioventricular node dysfunction (intrinsic or extrinsic) is the main cause of bradycardia requiring pacing
- The choice of pacemaker, pacing site and mode depends not only on the indication for pacing, but also on patient-specific factors
- Leadless pacemakers are emerging as an alternative to transvenous leads, although long-term outcome data are awaited

Conduction system disease and pacemakers

Failure of the cardiac conduction system

Impulse generation from the sinoatrial node (SAN) propagates across the atrium to the atrioventricular (AV) node before rapid transit down the His–Purkinje network to the ventricles. Failure of impulse generation in the SAN or impulse propagation via the AV node/His–Purkinje network results in bradycardia; if this is sufficiently slow or prolonged, it results in symptoms.

Sinus node disease (SND)

SND is the most common reason for pacemaker implantation. It is most often idiopathic and seen in elderly individuals. SND can result in sinus bradycardia, chronotropic incompetence and sinus arrest. The frequent association of paroxysmal atrial fibrillation (AF) and sinus bradycardia, which can suddenly oscillate from one to the other, is termed tachy-brady syndrome. Of patients with SND, only 1% per year develop significant AV block.

AV node disease

Acquired AV block is the second most common indication for permanent pacemaker placement. Three degrees of heart block are described, according to site and severity (see Table 1 for a classification of heart block). Many disorders can cause acquired AV block, although it is usually idiopathic. Nodal conduction block can also result from ischaemia (after myocardial infarction), toxicity (β-adrenoceptor blockers, calcium channel antagonists, digoxin), infection (brucellosis, Lyme disease) and cardiac surgery (most commonly after aortic valve surgery, given the proximity of the AVN and aortic valve). Congenital heart block is uncommon and can occur in isolation, in association with structural congenital abnormalities or as a consequence of maternal lupus antibodies. The clinical course is highly variable and it may be identified on a routine electrocardiogram (ECG) in young patients with no symptoms.

Neurocardiogenic (vasovagal) syncope

Neurocardiogenic syncope (also known as vasovagal syncope) is a benign condition characterized by a self-limited episode of systemic hypotension, during which increased vagal tone results in reduction of cardiac filling, bradycardia and hypotension with ensuing loss of consciousness. Differential diagnoses include carotid sinus hypersensitivity (resulting from an extreme reflex response to carotid sinus stimulation) and orthostatic hypotension (failure of the autonomic reflex response). The mainstay of

Classification of heart block

First-degree	Fixed but prolonged PR interval (≥200 ms) 1:1 AV relationship
Second-degree	Some atrial activity is not conducted through to the ventricles (intermittent but complete failure of AV node conduction) Further classified into: • *Mobitz I* (Wenckebach) – progressive prolongation of PR interval until the P wave fails to be conducted and the cycle starts again • *Mobitz II* – PR interval is constant but ventricular activation fails either intermittently or in a fixed ratio (e.g. 2:1, 3:1, 4:1) to the P wave rate
Third-degree	Complete AV block or AV dissociation. Atrial activity is independent of ventricular activity (ventricular activity is provided by an escape rhythm of around 30–40 bpm)

Table 1

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ECG characteristics of nodal versus infranodal conduction abnormality

AV nodal pathology	Significant PR prolongation or Mobitz type I (AV Wenckebach)
Disease below the AV node (infra-Hisian)	Normal/minimally prolonged PR interval, Mobitz type II (fixed-degree AV block), QRS complex abnormalities (bundle branch block and/or fascicular block)

Table 2

management is educating the patient to avoid situations that predispose to syncope, coping skills and reassuring the patient and others that this is a benign condition; pacing for the bradycardia component is occasionally helpful in selected patients.

Pacing indications in conduction system disease

Two factors guide most decisions to implant a pacemaker: the association of symptoms with an arrhythmia and the location of the conduction abnormality:

- **Symptoms** – consider symptoms that have resulted from bradyarrhythmias (e.g. dizziness, lightheadedness, syncope, fatigue, poor exercise tolerance). Evidence of mild or intermittent sinus node dysfunction or conduction abnormalities is frequently found, and it is crucial to attempt to establish a direct correlation between symptoms and bradyarrhythmias by a careful history and ambulatory monitoring.
- **Location of the conduction abnormality** – the location of an AV conduction abnormality (within the AV node or His–Purkinje system) is an important determinant of both the probability and the likely pace of progression of conduction system disease. Disease below the AV node in the His–Purkinje system is generally considered to be less stable; as a result, permanent pacemaker placement is more likely to be recommended (Table 2).

Updated guidelines for cardiac pacemaker implantation have been published by a taskforce formed by the American College of Cardiology in collaboration with the American Heart Association[1]; these guidelines are broadly similar to those issued by the European Society of Cardiology/European Heart Rhythm

Pacemaker indications (adapted from ACC/AHA/HRS 2012 Updated guidelines for device-based therapy of cardiac rhythm abnormalities and ESC 2013 Guidelines on cardiac pacing and cardiac resynchronization)

Class I (good evidence)

Sinus node dysfunction where symptoms clearly relate to the bradycardia (including chronotropic incompetence, frequent sinus pauses, or heart rate <40 bpm)

Symptomatic sinus bradycardia due to required drug therapy that cannot be stopped (primarily anti-arrhythmic therapy in tachy-brady syndrome)

Complete (third-degree) AV block

Advanced second-degree AV block (block of two or more consecutive P-waves)

Mobitz II second-degree AV block with a widened QRS or chronic bifascicular block

} regardless of symptoms[a]

Symptomatic Mobitz I or Mobitz II second-degree AV block

Exercise-induced second or third-degree AV block (in the absence of myocardial ischaemia)

Alternating bundle branch block with or without symptoms

Significant carotid sinus hypersensitivity (syncope and >3 seconds of asystole following minimal carotid sinus massage)

Post-myocardial infarction

Third-degree AV block within or below the His–Purkinje system

Persistent second-degree AV block in the His–Purkinje system, with bilateral bundle branch block

Transient advanced infranodal AV block with associated bundle branch block

Class IIa/IIb (some evidence in favour though some divergence of opinion)

Sinus bradycardia (heart rates <40 bpm) in a patient with symptoms suggestive of bradycardia, but without a clearly demonstrated association between bradycardia and symptoms

Unexplained syncope in a patient with sinus node dysfunction

Chronic heart rates <40 bpm while awake in a minimally symptomatic patient

Asymptomatic Mobitz II second-degree AV block with a narrow QRS interval

First-degree AV block when there is haemodynamic compromise because of effective AV dissociation secondary to a very long PR interval

Bifascicular or trifascicular block associated with syncope that can be attributed to transient complete heart block, based upon the exclusion of other plausible causes of syncope (specifically ventricular tachycardia)

Unexplained syncope and bundle branch block

Patients with syncope without clear provocative events and with a hypersensitive cardio-inhibitory response of 3 seconds or longer

Patients with recurrent neurocardiogenic syncope associated with bradycardia, documented spontaneously or at the time of tilt-table testing

[a] In asymptomatic advanced AV node disease, it may be appropriate not to implant a pacemaker if the symptoms occur only with a reversible cause (e.g. due to obstructive sleep apnoea), or there is a narrow complex escape rhythm >40 bpm with no evidence of pauses on repeated monitor studies. However, as there is no robust evidence as to the safety of this approach, it is recommended strongly to consider pacing in complete heart block even with a fast escape rhythm.

Table 3

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