Bradycardia pacing

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Abstract

Since the first human pacemaker in the 1950s, there has been rapid development in the technology of cardiac devices for rhythm management and the equipment to facilitate their implantation. As a result, the number of patients with devices 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; pacemaker

Conduction system disease and pacemakers

Failure of the cardiac conduction system

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

Sinus node disease (SND)

Sinus node disease is the most common reason for pacemaker implant. It is most often idiopathic and most frequently seen in the elderly. SND refers to a broad array of abnormalities in the sinus node resulting in sinus bradycardia, chronotropic incompetence and sinus arrest. The frequent association of paroxysmal atrial fibrillation (AF) and sinus bradycardia, which may oscillate suddenly from one to the other, is termed 'tachy-brady syndrome'. Though abnormalities of AV conduction can often be demonstrated patients with SND, only 1% of patients per year will develop significant AV block.[1]

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 classification of heart block). Many disorders can cause acquired AV block, although it is most commonly idiopathic. Nodal conduction block can also result from ischaemia (after MI), toxicity (β-blockers, calcium channel antagonists, digoxin), infection (brucellosis, Lyme disease) and cardiac surgery (most commonly following aortic valve surgery, given the proximity of the AVN to the aortic valve). Congenital heart block is an uncommon condition that may 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 routine ECG in young patients with no symptoms.[2]

Neurocardiogenic (vasovagal) syncope

Syncope as a symptom is most often (50–60% cases) caused by neurocardiogenic 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 a 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 management is education of the patient to avoid situations that predispose to syncope, coping skills, and reassurance of the patient and others that this is a benign condition, though pacing for the bradycardia component is occasionally helpful in selected patients.[3]

Pacing indications in conduction system disease

Despite the many clinical situations in which permanent pacing is considered, two factors guide most decisions: the association of symptoms with an arrhythmia and the location of the conduction abnormality.

Symptoms: patients are often considered for pacemaker placement because of symptoms that may have resulted from bradyarrhythmias (e.g. dizziness, lightheadedness, syncope, fatigue, and poor exercise tolerance). Frequently, evidence of mild or intermittent sinus node dysfunction or conduction abnormalities is 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 conduction abnormality: the location of an atrioventricular (AV) conduction abnormality (within the AV node or in the 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 and as a result, permanent pacemaker placement is more likely to be recommended (Table 2).

Updated guidelines for implantation of cardiac pacemakers have been published by a task force formed by the American College of Cardiology in collaboration with the American Heart Association,[4] and these are broadly similar to those issued by the European Society of Cardiology/European Heart Rhythm Association.[5] Although occasional cases cannot be categorized

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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 AVN conduction). Further classified into *Mobitz I* (Wenckebach) — progressive prolongation of PR interval until P wave fails to be conducted and cycle starts again; or *Mobitz II* — PR interval constant but ventricular activation fails either intermittently or in a fixed ratio to the P wave rate (e.g. 2:1, 3:1, 4:1).
Third-degree	Complete AV block or AV dissociation. Atrial activity independent of ventricular activity (ventricular activity provided by escape rhythm of around 30–40 bpm).

Table 1

according to these guidelines, they are, for the most part, all-encompassing and have been widely endorsed (see Table 3).

Other indications for pacemakers

Neuromuscular diseases

A number of neuromuscular diseases including myotonic muscular dystrophy, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy are associated with AV block. Patients with these disorders have a class I indication for pacemaker placement once any evidence of second or third-degree block develops. This is true even if the patient is asymptomatic, because AV conduction disease can progress rapidly and unpredictably.

Long QT syndrome

Because of their ability to prevent bradycardia — and hence, ventricular arrhythmias — pacemakers have been used in high-risk patients with long QT syndrome. However, many such patients are now treated with an implantable cardioverter-defibrillator, which also has pacing capability.

Hypertrophic cardiomyopathy

Pacing for medically refractory, symptomatic hypertrophic cardiomyopathy with significant resting or provoked LV outflow obstruction is now considered a class IIb indication and is not generally recommended. It was previously thought that pacing in the right ventricular apex with a short AV delay would alleviate a proportion of the outflow tract gradient, but these patients would now be considered for either surgical myomectomy or alcohol septal ablation.

ECG characteristics of nodal vs infranodal conduction abnormality

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

Table 2

Drug-induced bradycardia

Many drugs have effects on the cardiac conduction system. These include β-blockers, which are widely used in heart failure and ischaemic heart disease, calcium channel blockers for angina, and most anti-arrhythmic therapies, as well as non-cardiological therapies such as antidepressants, anticonvulsants and antipsychotics. Where a therapeutic drug causes a conduction problem that would normally be an indication for pacing, it is recommended that a pacemaker should be implanted if that drug cannot safely be stopped.

Atrial fibrillation

Paroxysmal AF frequently co-exists with sinus node dysfunction, which is often exacerbated by medications, necessitating a combined approach of pacemaker implantation and anti-arrhythmic therapy. In persistent AF, where adequate rate control cannot be achieved (usually due to drug intolerances), symptoms may be improved by pacemaker implantation and radiofrequency ablation of the AV node ('pace and ablate'). This isolates the ventricles from the atria, so that the heart rate is entirely dependent on the pacemaker.[6]

Unexplained syncope/falls/atypical symptoms

In an ideal world, all patients would present with typical symptoms of bradycardia and a diagnostic ECG. However, this is often not the case, and clinical judgement remains important in determining whether pacing is indicated. For example, it is estimated that 15–20% of unexplained falls may be due to bradycardia, and when patients cannot remember the details this may represent retrograde amnesia. However, a randomized trial of pacemaker implantations in such patients did not reduce the frequency of falls.[7] Rather than perform 'empirical pacing' for unexplained or atypical symptoms, it is recommended that prolonged rhythm monitoring, usually with an implantable loop recorder (ILR) be performed to establish a diagnosis.

Pacemaker function

Components

A pacemaker consists of three components — battery (generally lithium iodide), circuitry (determines what the pacemaker can do) and lead connectors (international standard for all manufacturers ['IS1']).

Pacing leads

Pacing leads have an electrode at the tip, secured within the heart by either active or passive fixation (see Figure 1). Passive leads secure themselves within the myocardial trabeculae by use of tines (like a soft anchor), whereas active leads have a small helical screw that is deployed into the myocardium. Pacing leads have two basic but crucial functions:

- they are required to see or 'sense' a cardiac impulse and be able to pace appropriately
- they must be able to pace the myocardium in the absence of a cardiac impulse.

A pacing lead in good contact with the heart will see/sense a clear intracardiac signal and will require a lower voltage to

