INDICATIONS FOR PERMANENT PACEMAKERS

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Impairment of the function of the sinus node and atrioventricular conduction system are believed to occur usually because of degeneration of atrial cells or specialized conduction tissue in the heart, normally without any other overt heart disease. An increasing number of open heart operations are performed every year, leading to traumatic damage of conduction tissue; and as a consequence of corrective surgery for congenital heart disease, many of these patients need a permanent pacemaker. However, in most of the cases the definitive cause of bradycardia remains unknown.

Sometimes it is necessary to study the patient invasively in the electrophysiology laboratory to confirm or rule out impairment in intracardiac conduction. However, in a majority of cases, it is the clinical picture and the 12-lead ECG that are needed to make a decision for a permanent pacemaker. Occasionally, a pacemaker has to be implanted after extensive studies have failed to determine the cause of symptoms and when there is still lack of correlation of the symptoms with the findings in the ECG or ambulatory monitoring.

General Principles

The decision to implant a permanent pacemaker should be made only after the diagnosis of bradyarrhythmia is clear; it should be known which disorder is to be treated. The patient's symptoms and the correlation to ECG findings should be assessed. The patient should be functional and his/her demand for different types of pacemakers should be evaluated. On taking these factors into consideration, we can determine the right mode of pacing for a given patient and more importantly prevent the implantation of a pacemaker that may be harmful for him/her. It is extremely important to rule out all possible temporary causes for bradycardia such as medications that can have an effect on propagation or conduction of impulses, disturbances of electrolyte balance, acute ischemia, and endocrine disorders (hypothyroidism).

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In industrialized countries, 40% to 60% of permanent pacemakers are implanted due to disorders of the sinus node. Incidence of sick sinus syndrome (SSS) increases in older populations and it is very uncommon below the age of 60 y. In Western countries, it has been estimated that 150 to 200 people per one million develop symptomatic SSS, thus justifying the need for pacemaker implantation.

SSS is a clinical description of a condition where electrical propagation of the impulse in the node itself and/or its conduction in surrounding (atrial) tissue is impaired. The spectrum of findings related to this syndrome is very wide, ranging from inappropriate bradycardia to the classical picture of the so-called tachy-brady syndrome where supraventricular tachycardia is followed by a long period of bradycardia or even asystole.

The etiology of SSS is unknown, but it is believed to be caused by an autoimmune process leading to a diminished number of pacemaker cells in the right atrium. It is also known to be associated with...
various other cardiac diseases which include coronary artery disease, systemic disease, cardiomyopathies, and infections.

The decision to implant a pacemaker is easy if dysfunction of the sinus node is associated with clear symptoms such as syncope or near syncope. However, symptoms are very often unclear. Sometimes, the only symptom can be excessive fatigue, mental dysfunction, or exceptionally low-exercise tolerance. In these cases, extensive noninvasive and invasive studies should be required before correct diagnosis, and most importantly the need for a pacemaker can be defined. Various types of ambulatory ECG recordings are used to define a correlation between bradycardia and symptoms. Twenty-four-hour Holter recording is known to be negative in 70% to 80% of cases, and it is useful only if symptomatic arrhythmia (tachy or brady) is detected. Event recorders activated by the patient will probably prove to be more useful than conventional Holter recording.

To confirm that sinus node malfunction is present, all medications that have an effect on impulse formation or conduction should be terminated. This is often not possible and the decision to implant a pacemaker has to be made knowing that at least part of the manifestation of the syndrome may be attributable to these drugs. In exercise stress-testing, some patients with suspected SSS may demonstrate inadequate heart rate increase on exercise, confirming impaired nodal function.

Indications for Permanent Pacing in Atrioventricular Conduction Defects

Traditionally, atrioventricular (A V) conduction defects are divided as first-degree, second-degree, and third-degree A V blocks (total A V block). Anatomically speaking, these defects are located above the bundle of His (supra-His), in the bundle area (intra-His), and below the bundle of His (infra-His).

Second-degree A V block is further divided into Mobiz I (Wenckebach type) and Mobiz-2-like defect. In Wenckebach block, the PR interval increases from beat to beat and one atrial impulse is not followed by ventricular activation. In Mobiz 2, the PR interval remains constant until one P wave is not conducted. Wenckebach-type block is considered a benign phenomenon since the conduction defect is located at the level of atrioventricular junction. These patients are often asymptomatic. In Mobiz 2, the risk for complete A V block is higher because of the distal nature of the block, and the patients are often symptomatic. Other indicators for distal (usually infra-His) block are bundle-branch morphology of the QRS complex and exercise-related increase of block ratio (e.g., from 2:1 conduction to 4:1). Impairment of atrioventricular conduction can be totally asymptomatic or the patient can be seriously symptomatic from bradycardia or from ventricular arrhythmias.

A pacemaker is never needed in first-degree A V block and almost never in Wenckebach-type second-degree block. The prognosis of the patient with Wenckebach-type block is not impaired because it is not believed to progress to higher degrees of conduction defects as in Mobiz 2, where a pacemaker is usually indicated.

Sometimes young people with high nocturnal vagal tone may present Mobiz-2-like findings during ambulatory monitoring. Unless they do not have symptoms related to bradycardia during daytime, a pacemaker is generally not indicated. Patients with Mobiz 2 are often symptomatic, and this condition often progresses to grade 3 A V block. Therefore, these patients usually need a permanent pacemaker.

It is common practice to pace any person with a grade 3 A V block, regardless of symptoms. It is known, from the early era of cardiac pacing, that symptomatic patients with total disconnection of A V conduction benefit from pacing, both from the symptomatic and prognostic viewpoints.3

In congenital complete A V block, it is a common policy to pace only symptomatic patients unless there are signs of inadequate cardiac output, i.e., increased heart volume and poor growth. Implantation of a permanent pacemaker is not considered mandatory if the ventricular rate is over 40/min, the QRS complex is narrow, no ventricular irritability, and the heart rate response to exercise is normal. However, a recent Swedish study has shown that patients with asymptomatic congenital complete A V block encountered at any point of life may benefit from pacing compared to unpaced population; both mortality and morbidity being less in the paced group.4
Indications for Pacemakers

Intraventricular Conduction Defects

Sometimes impulse conduction is impaired below the atrioventricular node in two or three of the fasciculus of left or right bundle branch. Usually, ECG manifestations are either RBBB + LAHB or RBBB + LPHB (bifascicular block). In addition to these findings, PR time can be prolonged, indicating even more seriously impaired A V conduction. Alternating RBBB and LBBB is also a clear sign for significant damage to the conduction system. If a patient complains of syncopal or near-syncopal symptoms and the above-mentioned conduction defects are documented in 12-lead ECG, a pacemaker is often indicated, even without definitive evidence of bradycardia because intermittent total A V block is the most likely reason for the patient's symptoms.1

Expanding Indications for Permanent Pacemakers

In recent years, several new and still controversial indications for permanent pacemaker implantation have been proposed. In hypertrophic obstructive cardiomyopathy, the pathology involves dynamic pressure gradients over left ventricular outflow tract caused by abnormal movements in the septal musculature and the anterior mitral leaflet. Altering the activation of the interventricular septum with right ventricular pacing has been known to reduce this gradient. The use of dual-chamber pacing with A V delay, short enough to achieve full capture, is probably beneficial in the subset of patients who have failed conventional therapy which may also be an alternative to surgical treatment.5

In dilated cardiomyopathy, ventricular activation is abnormal, the QRS is prolonged (often more than 140 ms), and LBBB may be present. A long PR interval often predisposes to late diastolic (presystolic) mitral regurgitation. This prevents effective forward flow through the mitral valve during diastole. Prolonged functional systolic mitral regurgitation is also often present. These abnormalities in electrical activation and subsequent changes in hemodynamics are harmful for these patients. Dual-chamber pacing with shorter A V delay is possibly beneficial to a selected subset of patients.6 Long-term studies regarding the real value of this therapy are still underway.

The congenital long QT syndrome is an inherited anomaly of cardiac repolarization that often results in recurrent syncope and even sudden cardiac death caused by polymorphic ventricular tachycardia. Initial treatment of this syndrome includes beta-blocking agents, followed in some cases with left cervical sympathectomy if medical treatment fails. Protective effects of beta-blocking agents are believed to be due to protection of the heart from excessive sympathetic discharge. Bradycardia is known to be one of the triggering events and therefore the combined use of beta-blockers and pacing is an attractive method to prevent ventricular arrhythmias. Controlled studies are still needed to verify the beneficial effects of this therapy on survival.7 Neuromediated syncope is listed as a potential indication for pacing in the American Heart Association and American College of Cardiology guidelines for permanent pacing.1 This issue is far from resolved, but patients who have this syndrome with predominant bradycardiac component may benefit from dual-chamber pacing.

References