Regular Review

Cardiac pacing

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Rapid progress in the development of implantable cardiac pacemakers and other cardiostimulatory devices has made the selection of pacing units suited to specific clinical problems increasingly difficult: the continuing pace of research and development has meant that recommendations have to be constantly modified. The indications for pacing are based on careful evaluation of symptoms and the disturbances in rhythm, using ambulatory monitoring and electrophysiological studies, but in addition each individual patient has to be assessed with attention to his or her general medical, emotional, and mental state before the decision to implant a pacemaker is taken.

The indications for permanent pacing discussed in this review are confined to the management of chronic or intermittent disorders of cardiac rhythm; I shall not discuss those factors which cause transient interference with cardiac impulse formation and conduction, such as electrolyte disturbances or endocrine imbalance, infection, drugs, or the acute phase of myocardial infarction.

Patients with abnormalities of atrioventricular function may be asymptomatic or experience serious symptoms related to bradycardia or ventricular arrhythmias or both. Indeed, symptomatic bradycardia is one of the most important signs of the need for a pacemaker and is considered to be present when symptoms can be directly attributed to a slow heart rate: these comprise lightheadedness, dizziness, presyncope or frank syncope as manifestations of transient cerebral ischaemia, together with other general symptoms such as abnormal weakness, fatigability, or the presence of cardiac failure.

Atrioventricular block is classified clinically as first, second, and third degree. It may also be further defined anatomically as being supra His, intra His, or infra His. Second degree heart block is further classified into type I, with progressive prolongation of the P-R interval before a blocked beat, and type II, where there is no progressive prolongation of the P-R interval before the blocked beats.

Patients with complete heart block and syncope are known to have an improved survival with permanent pacing.¹ There is no evidence that pacemakers prolong survival in patients with isolated first degree atrioventricular block. The prognosis in type I second degree atrioventricular block, when due to atrioventricular nodal delay, tends to be benign.^{2,3} In patients with type II second degree heart block whether intra or infra His, however, symptoms are frequent, the prognosis is uncertain, and progression to complete heart block is common.^{2,4}

The current consensus is that pacing should be started if the patient has complete heart block at any anatomical level associated with symptomatic bradycardia, congestive heart failure, ventricular ectopy, or other arrhythmias that require treatment with drugs which suppress automaticity; documented periods of asystole of three seconds or longer,

or any escape rate of less than 40 beats/min in symptom free patients; a confusional state which clears up after temporary pacing; permanent or intermittent second degree atrioventricular block, regardless of the type or site of the block, when associated with symptomatic bradycardia; and any of atrial fibrillation or flutter, supraventricular tachycardia with complete heart block, advanced atrioventricular block, or symptomatic bradycardia which is not induced by a drug.

Opinion is less unanimous on the need for pacing in patients with permanent or intermittent asymptomatic complete heart block at any anatomical site with ventricular rates of 40 or faster, permanent or intermittent asymptomatic type II second degree block, and asymptomatic type I second degree block, whether intra His or infra His. All authorities agree that first degree atrioventricular block and asymptomatic type I second degree heart block supra His do not require permanent pacing.

A requirement for temporary pacing during acute myocardial infarction is not an indication for permanent pacing. The long term prognosis in those who have had atrioventricular block associated with a myocardial infarction is related primarily to the extent of myocardial injury and the character of intraventricular conduction disturbances rather than to the atrioventricular block.^{5,8} The incidence of sudden death is increased in patients with acute myocardial infarction who have intraventricular conduction defects (with the exception of isolated left anterior hemiblock).^{5,7} The deaths are not due necessarily to the development of high grade atrioventricular block, though the incidence of such block is higher in patients with abnormal intraventricular conduction after myocardial infarction.⁹

In contrast to the other indications for permanent pacing patients who have had a myocardial infarction and have atrioventricular block do not necessarily require symptoms in order to merit pacing. Patients with persistent second degree atrioventricular block or complete heart block after acute myocardial infarction should be paced before their discharge from hospital.^{7,9} Some authorities advocate pacing in patients after myocardial infarction if they have persistent first degree atrioventricular block in the presence of new bundle branch block¹⁰ and in those with transient atrioventricular block and associated bundle branch block. Most agree that pacing is not required after myocardial infarction in patients in whom atrioventricular disturbances are transient and are not associated with intraventricular conduction defects, in those with transient atrioventricular block in the presence of isolated left anterior hemiblock, and in those with left anterior hemiblock in the absence of atrioventricular block.

Fascicular block

Fascicular block refers to electrocardiographic evidence of

abnormal conduction below the atrioventricular node in two or three of the fascicles of the right and left bundle branches. Bifascicular and trifascicular blocks when accompanied by symptoms are associated with a high mortality and a significant incidence of sudden death.¹ While syncope may occur in patients with bifascicular block it does not appear to be associated with an increased incidence of sudden death,^{10,11} although it has been pointed out that pacing relieves the transient neurological symptoms.¹² It is known, however, that syncope is associated with an increased incidence of sudden death if associated with transient complete heart block.¹ Some, therefore, advocate prophylactic pacing in the presence of bifascicular and trifascicular block associated with syncope as they postulate that the symptom may be due to transient complete heart block.^{13,14} Nevertheless, the rate of progression of bifascicular block to complete heart block is low, and attempts have been made to identify clinical and laboratory variables which will point to the patients at high risk of death from future bradyarrhythmia as a consequence of fascicular block. Prolongation of the P-R interval is common in patients with bifascicular block but is most often due to delay at the level of the atrioventricular node.

His bundle electrocardiography allows measurement of the H-V interval, the time required for conduction of the impulse from the common His bundle to the ventricular myocardium. There is no correlation between the P-R and H-V intervals, nor does there appear to be a relation between the length of the P-R interval and the progression to complete heart block and incidence of sudden death.¹⁵ Asymptomatic patients with bifascicular block and prolonged H-V interval are sometimes considered suitable candidates for permanent pacing.^{16,17} It appears, however, that, though the prevalence of a prolonged H-V interval is high, the incidence of progression to complete heart block is low. Nevertheless, prolongation of the H-V interval is usually associated with serious cardiac disease and carries an increased mortality—though it is not specifically associated with sudden death.^{10,15,18} The probability that atrial pacing will induce heart block in patients with fascicular block is low¹⁹; indeed pacing often fails to induce distal block in such patients with documented abnormal conduction of the His-Purkinje system.²⁰ Failure to induce distal block is not necessarily evidence that the patient will not develop complete heart block at some time in the future.

What conclusions should be drawn in this controversial topic? Permanent pacing is indicated in patients with bifascicular block associated with intermittent complete heart block accompanied by symptoms and in patients with bifascicular block with intermittent type II heart block associated with symptoms. Permanent pacing is sometimes advocated for patients with bifascicular or trifascicular block with intermittent type II atrioventricular block which is asymptomatic and for patients who have bifascicular trifascicular block with syncope which has not been correlated with an episode of complete heart block but where no cause for the syncope can be otherwise determined. All agree that patients with fascicular blocks without atrioventricular block or symptoms and with fascicular blocks with first degree atrioventricular block without symptoms should not be considered for permanent pacing.

Sick sinus syndrome

The sick sinus syndrome consists of a variety of cardiac arrhythmias: sinus bradycardia, sinoatrial block, sinus arrest,

and paroxysmal supraventricular tachycardia alternating with bradycardia or asystole. Symptoms must be correlated with the occurrence of the specific arrhythmia. Symptoms related to bradycardia may be relieved by permanent pacing,²¹ but long term survival may not be improved.^{22,23} Sinus node dysfunction with documented symptomatic bradycardia is an indication for permanent pacing. This may sometimes occur as a result of essential drug treatment for which there is no acceptable alternative. Some will consider pacing in such patients even if no clear association has been shown with symptoms. Permanent pacing is not indicated in asymptomatic patients with dysfunction of the sinus node or in those in whom symptoms can be shown by Holter monitoring not to be associated with periods of bradycardia.

The carotid sinus syndrome is an uncommon cause of syncope. In patients with this syndrome syncope results from an exaggerated reflex response to carotid sinus stimulation. There are two components to the reflex—the cardioinhibitory element, manifested by slowing of the sinus rate or prolongation of the P-R interval or both and advanced atrioventricular block, and a vasodepressor element, which occurs secondary to a reduction in sympathetic activity leading to profound hypotension. The relative contribution of these two components of carotid sinus stimulation to the patient's symptoms needs careful evaluation before proceeding to pacing. A hyperactive response to carotid sinus stimulation may be assumed to exist in the presence of asystole due to sinus arrest or atrioventricular block lasting longer than three seconds or a substantial symptomatic decrease in systolic blood pressure. Changes of this kind in heart rate and blood pressure may occur in normal people and those with coronary artery disease.²⁴ In those with a pure excessive cardioinhibitory response to carotid stimulation pacing will relieve symptoms.²⁵ One fifth of the patients with this syndrome, however, tend to have a vasodepressor component and this must be defined. The consensus is that patients should be paced if they have recurrent syncope associated with events provoking carotid sinus stimulation and minimal carotid sinus pressure induces asystole of greater than three seconds—provided that they are not taking any drug which depresses the sinus node or atrioventricular conduction. Some advocate pacing patients with recurring syncope who do not have a history of provocative events but, none the less, show a hypersensitive cardioinhibitory response. Pacing is not indicated in those with recurrent syncope, lightheadedness, or dizziness in whom the vaso-depressor element of the reflex is the cause of symptoms, nor is it indicated in those with vague symptoms of dizziness or lightheadedness or both, even though they may have a hyperactive cardioinhibitory response to carotid sinus stimulation.

Two types of pulse generators are currently available for permanent implantation; single chamber pacemakers, which may be used in either the atrium or the ventricle, and dual chamber pacemakers, which are used in both chambers. The modern pacemaker is powered by a lithium battery capable of functioning for up to 15 years and can be programmed—though non-programmable pacemakers are still widely used. Programmability is a process by which the functions of the pulse generator can be altered non-invasively, thus yielding considerable versatility. The rate of pulse generation, output, sensitivity, and refractoriness may be altered to meet the specific needs of the patient after implantation. This facility avoids reoperation in as many as one fifth of patients. Some of the newer multiprogrammable

pacemakers are capable of interrogation and on command can provide information on pacemaker function and clinical performance.

Atrial pacemakers which are inhibited by sensed atrial activity can be used in symptomatic sinus node dysfunction once careful investigation has shown that atrioventricular conduction is normal. Ventricular pacing, which is the original pacing mode, is pacing from the ventricle with inhibition by sensing spontaneous ventricular activity. This mode is used in any symptomatic bradycardia, particularly when there is no substantial atrial haemodynamic contribution. This may be as high as 20%, and its loss may be important in patients with defective left ventricular function. Periods of alternating atrioventricular synchrony and asynchrony may lead to a fall in cardiac output resulting in episodic weakness or syncope in patients with pacemakers—the “pacemaker syndrome.” Ventricular pacing is unacceptable in patients who are known to be or may be susceptible to this syndrome and those who need a maximal atrial contribution, as in congestive cardiac failure.

The solution to these problems has come with dual chamber pacemakers. Models with different sensing capabilities are available. Some units offer ventricular pacing which is synchronous with sensed atrial activity and are inhibited by sensed ventricular activity. At a slow atrial rate, such pacemakers will function as a simple ventricular demand pacemaker. These units are used in patients who suffer from, or who seem likely to develop, the pacemaker syndrome and who clearly require an atrial contribution for an adequate haemodynamic state. Other models can pace both the atria and ventricles at a preselected minimal rate and are inhibited only by ventricular activity. These are of value in patients with symptomatic bradycardia due to sinus node dysfunction who require sequential atrioventricular contraction. These pacemakers should not be used in patients who have ineffective atrial contractions or who exhibit supraventricular arrhythmias. The most versatile of these units can pace both chambers and sense from those chambers; they inhibit atrioventricular output by sensed atrial and ventricular activity and ventricular output may be triggered by sensed atrial activity. Such units are of value in active or youthful patients with atrial rates which respond to physiological requirements and who need sequential atrioventricular contraction. They are also of value in patients with the pacemaker syndrome or with reduction in blood pressure of 20 mm Hg at the start of ventricular pacing at the time of implantation. These units cannot be used in patients with supraventricular arrhythmias or with ineffective atrial contractions.

The pharmacological management of many tachyarrhythmias remains unsatisfactory, and this has stimulated the development of antitachycardia pacemaking systems. Already ectopic atrial tachycardias, reciprocating atrial tachycardias, and type I atrial flutter (atrial rate 230–350 a minute) can be treated by various antitachycardia pacemakers. Developments are extremely rapid and problems—such as those posed by coexistent large atria, cardiac failure, and obstructive airways disease with multifocal atrial mechanisms—are all currently undergoing evaluation. The ideal antitachycardia pacing system should be an automatic demand pulse generator, which should automatically reactivate as soon as the arrhythmia is terminated. It must be able to distinguish arrhythmia from sinus tachycardia and to scan to deliver a critically timed stimulus in order to provoke arrhythmia termination. It should be capable of

being interrogated and it must be fully programmable. Ideally, too, it should be able to provide defibrillation.

Defibrillators

The automatic implantable defibrillator has now been available for several years and is undergoing clinical evaluation. As with antitachycardia pacemakers the device is undergoing continuous modification as problems arise. Electrophysiological testing is required before and during implantation. The range of indications for this device has been broadened beyond ventricular fibrillation: it can now deal with ventricular tachycardia. Higher energy defibrillators, with an output of 42 joules, have become available to deal with high defibrillation thresholds. The three major obstacles at present are the need for a satisfactory energy source (the lithium iodine cell is very quickly exhausted, yielding about 100 defibrillations an implant); a fail safe mechanism for the recognition of ventricular fibrillation so that spurious discharges do not ensue; and the prevention of myocardial damage by the defibrillatory shock. Early experience has suggested that such an implantable defibrillator can identify and correct lethal ventricular tachyarrhythmias, leading to an increase in one year survival in selected patients at high risk.²⁶

Clearly the technological developments and the broadening therapeutic applications of pacemakers demand a high level of skill from the clinicians concerned. They may, indeed, support the suggestion that cardiac pacing should be identified as a new subspecialty.²⁷

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