Permanent Pacing in the Absence of Heart Block
An Approach to the Management of Intractable Arrhythmias

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SUMMARY
Permanent pacemakers are useful in the management of certain patients with arrhythmias in the absence of heart block. Five patients are presented, four of whom obtained a satisfactory response to pacing. Continuous electrocardiographic monitoring by means of a magnetic tape system is useful in diagnosis as well as in the evaluation of the effects of therapy.

Additional Indexing Words:
ECG monitoring    Digitalis intoxication

It is well established that permanent artificial pacemakers reduce the morbidity and mortality associated with complete heart block and Stokes-Adams attacks.1-4 Pacemakers are also useful in the treatment of patients who do not have heart block. Temporary pacing has been used for the suppression of arrhythmias immediately following cardiac surgery and in other acute situations.5-6 Recently, several reports have indicated the value of permanent pacing in the treatment of supraventricular and ventricular tachycardias.7-15

The purpose of this report is to present experience with permanent pacemakers in the management of five patients presenting with a variety of problems including ventricular tachycardia and fibrillation, recurrent digitalis intoxication, and periods of asystole following paroxysmal supraventricular tachycardia. A valuable adjunct to the management of these patients has been a system for the continuous recording of the electrocardiogram on magnetic tape for subsequent high-speed playback and analysis.16

Report of Cases

Case 1

G. P., a 38-year-old Caucasian woman, was admitted to The Johns Hopkins Hospital on April 23, 1967, for evaluation of recurrent syncopal episodes of 18 months' duration. Five months prior to admission the syncopal episodes had become more frequent, occurring as often as five times a day.

Physical examination was unremarkable. The ECG demonstrated a short P-R interval, frequent unifocal premature ventricular contractions (PVCs) with occasional bigeminy, and nonspecific ST-T waves changes. Continuous electrocardiographic monitoring revealed bigeminy with episodes of trigeminy and paroxysmal ventricular tachycardia (fig. 1A and B).

Successive trials of atropine, quinidine sulfate, procainamide, and propranolol failed to influence
the basic arrhythmia or to abolish recurrent episodes. On May 25, right atrial pacing was established by means of a bipolar electrode catheter. A rate of 120/min was required to abolish the arrhythmia, but after 3 mg of propranolol given intravenously, the arrhythmias could be abolished by pacing at a rate of 96/min. The dose of propranolol was increased to 160 mg orally daily, and on May 31, a permanent transvenous pacing catheter was introduced into the apex of the right ventricle, and pacing was continued at 92/min with a Medtronic (model 5870C) pulse generator. The patient has remained asymptomatic to the present (August 1968) and continues to take propranolol, 160 mg/day, and quinidine, 1.2 g/day (fig. 1C).

**Case 2**

C. A., a 58-year-old Caucasian man, was admitted to The Johns Hopkins Hospital on October 9, 1967, because of a syncopal episode several hours previously. He had undergone mitral commissurotomy in 1959 and had been maintained on digitalis and diuretics until 12 days prior to admission when digitalis was discontinued because of bradycardia. Following this, congestive failure became manifest and was refractory to diuretics. In the week prior to admission he experienced two syncopal episodes of 2 to 3 minutes’ duration, which were preceded by lightheadedness and palpitations.

The physical findings of mitral regurgitation, pulmonary hypertension, and congestive failure were present. The ECG showed sinus rhythm without premature beats and evidence of an old inferior myocardial infarction.

The patient was placed on continuous monitoring by a magnetic tape system, and the tape revealed that 8 hours after admission several premature ventricular contractions appeared and were followed by ventricular fibrillation which terminated spontaneously in 2 to 3 minutes (fig. 2A and B). Intravenous administration of...
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Figure 2

Case 2. Lead II. (A and B) Continuous recording. Sinus bradycardia with development of premature ventricular contractions leading to ventricular fibrillation which terminated spontaneously. (C) Functioning pacemaker.

Figure 3

Case 3. Lead II. Continuous tracing showing ventricular tachycardia terminated by external countershock.

Lidocaine was unsuccessful in preventing ventricular irritability, and on October 17, two episodes of ventricular fibrillation occurred, one of which required countershock for termination. An attempt was made to initiate pacing by the transvenous route but this was unsuccessful. Thoracotomy, for insertion of myocardial electrodes, was complicated by recurrent ventricular fibrillation. After institution of pacing with a Medtronic (model 5870) pulse generator set at 118/min, no further ventricular fibrillation occurred. On October 27, the pacemaker rate was diminished to 92/min without difficulty (fig. 2C).
Following discharge, the patient remained asymptomatic until December 24, when pacing ceased and ventricular fibrillation recurred. At a university hospital in another city, one of the myocardial electrode wires was found to have broken, and the pacemaker was put into a unipolar mode of operation. An infection of the wound and pacemaker pocket developed, and the patient was transferred to The Johns Hopkins Hospital. On February 2, 1968, a transvenous pacing catheter was placed in the right ventricle and pacing was established with a Medtronic (model 5870C) pulse generator at a rate of 124/min. The following morning the patient vomited and ventricular fibrillation occurred which was refractory to resuscitative efforts.

Autopsy revealed the transvenous catheter to be in the apex of the right ventricle. Its tip was located directly under the site of implantation of the myocardial electrodes. The myocardium was scarred and thin in this area. Rheumatic scarring of the mitral valve and a healed inferior myocardial infarction were noted.

**Case 3**

C. S., a 63-year-old Caucasian man, was admitted to The Johns Hopkins Hospital on November 13, 1967, for treatment of recurrent ventricular tachycardia. He had been in good health until June 1967 when he had an anterolateral myocardial infarction. Subsequently, recurrent episodes of ventricular tachycardia developed, each of which required countershock for termination (fig. 3). The arrhythmia could be controlled with propranolol and procainamide, but on August 14, he developed congestive heart failure, which responded to diuretics. He was able to return to work until November 12, when ventricular tachycardia recurred requiring countershock. The electrocardiogram showed sinus bradycardia and an old anterolateral myocardial infarction.

On November 15, a permanent transvenous pacing catheter was introduced into the apex of the right ventricle and pacing was instituted at 79/min with a Medtronic (model 5870C) pulse generator. Several hours later, ventricular fibrillation occurred which was refractory to resuscitative efforts.

**Figure 4**

Case 4. Lead II. (A) First degree heart block. (B) Second degree heart block and ventricular irritability following digitalis administration. (C) Demand pacemaker functioning in fixed-rate mode. (D) Demand pacemaker functioning in stand-by mode.
tachycardia. Following a second episode of ventricular tachycardia, the pacemaker rate was increased to 120/min. Acute pulmonary edema ensued. Treatment with propranolol was temporarily discontinued and the pacing rate was reduced to 92/min.

The patient was discharged from the hospital on 80 mg of propranolol and 4.0 g of procainamide per day. Two weeks after discharge, paroxysmal ventricular tachycardia recurred and continued despite increased propranolol dosage of 120 mg/day. Dilantin in a dose of 400 mg/day was added to the regimen and the pacemaker rate was increased to 104/min. The arrhythmia continued, and on January 30, 1968, the patient became hypotensive and expired 3 hours later.

Autopsy revealed satisfactory placement of the pacing catheter. A healed anterolateral myocardial infarction was present. All branches of the left coronary artery were severely narrowed by atheroma.

Case 4

A.A., a 53-year-old Negro, was admitted to The Johns Hopkins Hospital on August 29, 1967, because of recurrent congestive heart failure. He had had numerous previous admissions for congestive heart failure associated with episodes of digitalis intoxication manifested by second degree heart block and ventricular irritability or paroxysmal atrial tachycardia (fig. 4A and B). He was known to have rheumatoid spondylitis and aortic insufficiency.

The physical findings were those of aortic regurgitation and congestive failure. The ECG revealed first degree heart block, frequent premature ventricular contractions, an old anterior myocardial infarction, left ventricular hypertrophy, and associated ST-segment and T-wave changes.

On September 14, because of digitalis sensitivity and persistent ventricular irritability, a temporary electrode catheter was placed in the apex of the right ventricle and pacing was initiated at 130/min. The ventricular irritability was abolished, and the congestive heart failure was successfully treated with digitalis. On October 2, a permanent transvenous pacing catheter was introduced into the apex of the right ventricle, and pacing was instituted with a Cordis Demand (Ventricor III) pulse generator at 90/min (fig. 4C and D). Subsequently, the patient has been free of congestive heart failure and digitalis intoxication.

Case 5

M.A., a 64-year-old Caucasian man, was admitted to The Johns Hopkins Hospital on January 13, 1968, for selective pacemaker implantation. He had noted the onset of episodes of lightheadedness in 1963 which increased in frequency and duration until March 1964 when syncope occurred. Paroxysmal atrial flutter-fibrillation was observed to be followed by sinus arrest with asystole causing syncope (fig. 5). He failed to respond to various drug regimens and episodes of near-syncope continued.

Physical examination was not remarkable. The ECG demonstrated first degree heart block and left axis deviation.

On January 15 a permanent transvenous pacing catheter was introduced into the apex of the right ventricle and connected to a Cordis Demand (Ectocor) pulse generator set at 70/min. Continuous monitoring revealed occasional episodes of atrial flutter with 2 to 1 block followed by asystole at which time the pacemaker would pace the heart at 70/min until sinus rhythm returned. Since discharge from the hospital, the patient has not noted lightheadedness or near-syncope.

Discussion

The prevention of ventricular tachycardia and ventricular fibrillation by pacemakers was originally reported by Zoll and associates\textsuperscript{17}
in patients with heart block. Although external electrodes were used, the therapeutic value of the method was clearly demonstrated and subsequently confirmed, following the development of permanent pacing systems.\textsuperscript{1-4}

In contrast, the use of pacemakers in the absence of heart block has been limited. Cohen and associates\textsuperscript{7} have used a permanent pacemaker for the prevention of sinus and nodal tachycardia. Temporary pacing has been used for the suppression of arrhythmias during acute situations, particularly myocardial infarction and following cardiac surgery.\textsuperscript{5, 6}

Recently, several reports have indicated the effectiveness of permanent pacemakers in the long-term treatment of paroxysmal ventricular tachycardia and ventricular fibrillation.\textsuperscript{8-15} Zipes and Orgain\textsuperscript{18} have reported a case of paroxysmal ventricular tachycardia with refractoriness to all forms of therapy, including pacing.

The use of pacemakers for the management of arrhythmias should be considered for those patients who are refractory to anti-arrhythmic drugs. It is often possible to obtain satisfactory control with drugs alone, especially in young patients with paroxysmal ventricular tachycardia who have no other evidence of heart disease.\textsuperscript{19} When this approach is not successful, a therapeutic trial of temporary pacing is warranted. The combination of anti-arrhythmic drugs and a pacemaker should also be considered as it may prove to be more effective than either alone. If a pacemaker is used, the dosage of drugs can frequently be reduced.\textsuperscript{8, 13, 15} A permanent pacemaker can also help in the management of patients with organic heart disease in whom digitalis, although indicated for congestive failure, aggravates an underlying heart block or ventricular irritable rhythm.

Continuous electrocardiographic monitoring on a magnetic tape system has been a valuable adjunct to the management of these patients. Taped records covering a period of many hours are of great value diagnostically and are also helpful in evaluating the effects of various therapeutic regimens.\textsuperscript{16}

Zoll and associates\textsuperscript{17} suggested that improved myocardial oxygenation may account for the decreased ventricular irritability. Physiological observations support this suggestion in that the acceleration of heart rate by pacing increases coronary blood flow and, hence, oxygen supply to the myocardium while cardiac output and, hence, oxygen requirement changes very little.\textsuperscript{20-24} An alternate, electrophysiological explanation for the decreased ventricular irritability is presented by Han and associates.\textsuperscript{25} These authors concluded that bradycardia enhances the development of premature ectopic beats because there is greater asynchrony of repolarization of adjacent excitable units associated with long cycle lengths. This enhancement of ventricular irritability has been confirmed by numerous other studies, both clinical and experimental\textsuperscript{8, 19, 26-28}

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**References**

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