Long-Term Pervenous Atrial Pacing

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SUMMARY

Long-term pervenous right atrial pacing has been used in five patients with intact atrioventricular (A-V) conduction for the treatment of refractory ventricular arrhythmias in two subjects and marked sinus bradycardia in three, two of whom also had paroxysmal supraventricular arrhythmias. The pervenous method was used to avoid a thoracotomy, and atrial pacing was chosen over ventricular pacing to preserve the normal A-V contraction sequence. Reliable atrial pacing was established in four cases, but one patient required ultimate conversion to a ventricular system because of irregular atrial capture. The most constant pacing was achieved by using a curved electrode with the tip positioned in the right atrial appendage.

Additional Indexing Words:
Atrial electrode Arrhythmia Refractory ventricular irritability
Sinus bradycardia

VENTRICAL PACING has provided a dramatic advance in the management of symptomatic complete heart block. There are occasions, however, when pacemaker therapy is indicated in patients with intact atrioventricular (A-V) conduction. Although ventricular pacing has been generally used for these purposes, the presence of normal A-V conduction permits stimulation of the heart from the atrium, thus taking advantage of the beneficial effect on cardiac performance that occurs when contraction of the atrium precedes that of the ventricle.

Pacing under such circumstances has been employed in at least three different clinical settings: (1) to overcome drug-refractory ventricular irritability by pacing the heart at a rapid rate; (2) to provide an adequate heart rate for patients with disease of the sinus node who have symptomatic sinus bradycardia or slow A-V junctional (nodal) rhythm, often associated with troublesome bouts of rapid supraventricular arrhythmias, and (3) occasionally to terminate certain atrial and reciprocating tachyarrhythmias.

Although atrial pacing is usually employed only temporarily, particularly for the suppression of refractory ventricular irritability, long-term atrial pacing is occasionally required. Implantation of electrodes into the wall of either atrium by thoracotomy has been reported, but there are relatively few instances of long-term atrial pacing by the pervenous technic. It is the purpose of this communication to describe the results of long-term pervenous right atrial stimulation in five patients treated by this method at the Massachusetts General Hospital. The pertinent features of these patients are listed in table 1.

Report of Cases

Case 1 (J. M., MGH 29-80-62) *

This 67-year-old woman with mitral regurgitation and atrial fibrillation entered the hospital on
### Table 1

**Data on Five Patients Treated with Long-Term Pervenous Atrial Pacing**

<table>
<thead>
<tr>
<th>Patient, age (yr), sex</th>
<th>Cardiac diagnosis</th>
<th>Indications for pacing</th>
<th>Atrial electrode (type)</th>
<th>Location of tip, date implanted</th>
<th>Impulse generator rate, pacing threshold (if measured)</th>
<th>Subsequent course</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 J.M. 67—F</td>
<td>RHD-MR</td>
<td>Refractory ventricular irritability</td>
<td>Bipolar</td>
<td>RA-SVC junction 6–3–66</td>
<td>Fixed rate 90 beats/min 5 ma</td>
<td>Reliable atrial pacing for 6 mo until persistent atrial fibrillation supervened</td>
</tr>
<tr>
<td>2 G.Cr. 58—M</td>
<td>Acute myocardial infarction</td>
<td>Refractory ventricular irritability</td>
<td>Bipolar</td>
<td>RA-SVC junction 5–12–67</td>
<td>Fixed rate 100 beats/min</td>
<td>Reliable atrial pacing for 17 mo. Died suddenly 10–17–68</td>
</tr>
<tr>
<td>3 G.Ch. 48—F</td>
<td>RHD-MR, AS, “sinus node disease”</td>
<td>Bradycardia and paroxysmal atrial fibrillation</td>
<td>Curved unipolar</td>
<td>RA 7–5–67</td>
<td>Fixed rate 95 beats/min</td>
<td>Mostly reliable atrial pacing with episodes of paroxysmal atrial fibrillation; died suddenly 17 mo later of an arrhythmia</td>
</tr>
<tr>
<td>4 L.C. 89—F</td>
<td>“Sinus node disease”</td>
<td>Bradycardia</td>
<td>Curved unipolar</td>
<td>RA 8–9–67</td>
<td>Fixed rate 85 beats/min 2.5 ma</td>
<td>Unreliable atrial pacing; changed to pervenous ventricular pacing system 5 mo later</td>
</tr>
<tr>
<td>5 R.F. 75—F</td>
<td>RHD-MS, “sinus node disease”</td>
<td>Bradycardia and paroxysmal atrial flutter and fibrillation</td>
<td>Curved unipolar</td>
<td>RA appendage 12–8–67</td>
<td>Fixed rate 95 beats/min 2.2 ma</td>
<td>Reliable atrial pacing for 16 mo when not in atrial flutter or fibrillation</td>
</tr>
</tbody>
</table>

Abbreviations: AS = aortic stenosis; ma = milliamperes; MR = mitral regurgitation; MS = mitral stenosis; RA = right atrial; RHD = rheumatic heart disease; SVC = superior vena cava.
April 28, 1966, for treatment of congestive heart failure. On May 8, 1966, recurrent ventricular tachycardia and ventricular fibrillation developed which required external massage and repeated direct current countershock. Lidocaine, procainamide, quinidine, and dyphenylhydantoin were all ineffective in controlling the arrhythmias, and she appeared moribund. A temporary pacing electrode was then passed into the right atrium under electrocardiographic monitoring, and rapid atrial pacing suppressed the arrhythmias. Repeated ventricular irritability developed during subsequent efforts to wean the patient from a respirator and could only be suppressed by continued rapid atrial pacing. Therefore, on June 3, 1966, a pervenous fixed-rate pacemaker was implanted through the right external jugular vein with the electrode tip positioned at the junction of the superior vena cava and right atrium.* Reliable atrial pacing was achieved. The stimulating threshold for atrial pacing was 5 ma and the subcutaneous impulse generator was adjusted to a maximal output setting of 7 ma with a pacing rate of 90 beats/ min. Intermittent stimulation of the right hemidiaphragm subsided after several weeks. Three months after the initial ventricular arrhythmia she was discharged to continue treatment with quinidine, a diuretic, and a low salt diet.

During the next 6 months atrial fibrillation recurred on several occasions with prompt atrial capture by the pacemaker on successful conversion with quinidine or countershock. Finally, further efforts to prevent the recurrence of atrial fibrillation proved unsuccessful, and the atrial pacemaker remained ineffective thereafter. The patient has remained in atrial fibrillation with treatment with digoxin to slow the ventricular response and quinidine to curb ventricular irritability.

The patient noted greater exercise tolerance in paced atrial rhythm than in atrial fibrillation at comparable ventricular rates. Studies of cardiac output, however, were not performed to document these clinical impressions. She has been well enough to live at home by herself and to perform light activities.

Case 2 (G. Cr., MGH #143-87-78)

This 58-year-old fireman had an acute anterior myocardial infarct on March 27, 1967, which was complicated by the development of complete A-V block 2 days later. At this point he was transferred to the Massachusetts General Hospital where physical examination revealed him to be weak, diaphoretic, and clammy. Blood pressure was 100/70 with a pulse rate of 55 beats/min, and there were signs of congestive failure. A temporary bipolar electrode was inserted into the right ventricle and satisfactory pacing was established. Shortly afterward A-V conduction resumed transiently and by the fourth day of his illness the block had completely resolved.

On the 14th day, ventricular fibrillation developed unexpectedly and was converted promptly with countershock. Despite additional procainamide, two more episodes of ventricular fibrillation, occurred, each successfully terminated with countershock. For the ensuing 2 weeks the irritability continued with a basic sinus rate of 80 to 90 beats/min. Ventricular bigeminy persisted with prolonged runs of ventricular tachycardia, which was slowed to a rate of 100 beats/min but never completely suppressed by a lidocaine infusion and quinidine. Congestive heart failure became more severe.

Because of difficulty in controlling the ventricular irritability with drug therapy, a wire electrode was inserted into the right atrium, and satisfactory atrial pacing at a rate of 100 beats/min was established. Administration of quinidine was continued; the ventricular irritability virtually disappeared, and diuresis ensued. Digitalis was cautiously administered and stable temporary pacing continued. Attempts to stop pacing resulted in the return of ventricular arrhythmia. Therefore, on May 12, 1967, 2 weeks after institution of temporary pacing, a fixed-rate pervenous pacemaker was implanted with the electrode inserted through the right cephalic vein and the tip positioned at the junction of the right atrium and superior vena cava. (Same equipment as used in case 1.) The rate was set at 100 beats/min. The stimulating threshold was not determined.

The patient continued to improve although occasional erratic response of the atrium to the pacemaker occurred because of competition from the sinus node and from occasional loss of contact between the electrode and the atrial wall. The ventricular irritability, however, was completely controlled, and he was discharged 2 months after admission to the hospital. He continued to pace regularly with occasional interference from sinus beats for 17 months. He died unexpectedly in his sleep on October 17, 1968. No autopsy was performed.

Case 3 (G. Ch., MGH #92-80-62)

This 48-year-old woman was admitted to the Massachusetts General Hospital on June 26, 1967, for treatment of recurrent arrhythmias.

At age 15, without a history of rheumatic fever, the patient was told that she had a heart murmur. At age 30 she began to have "skipped beats" and

*Chardack-Greatbatch Pulse Generator, Model 5870-C; Chardack Bipolar Cardiac Catheter Electrode Model 5818; Medtronic, Inc., Minneapolis, Minnesota.
Patient G.C. Lead 2

A. Atrial fibrillation 6-26-67

B. Sinus bradycardia 6-26-67

C. Atrial pacing 7-12-67

D. Atrial pacing 8-5-68

Figure 1

Electrocardiograms (lead 2) from case 3. Atrial pacing was instituted as treatment for alternating atrial fibrillation (A) and sinus bradycardia (B). Successful 1:1 capture was produced at the time of pacemaker implantation (C) and over a year later (D) whenever the patient was not in atrial fibrillation.

Quinidine was prescribed which she took intermittently thereafter. Three years before admission, she began to suffer from attacks of paroxysmal atrial fibrillation which caused her to feel weak, short of breath, and apprehensive (fig. 1A). When digitalis, quinidine, procainamide, diphénylfurantoin, or propranolol were administered sinus bradycardia was produced with a heart rate of 30 to 40 beats/min (fig. 1B). This produced marked weakness, nausea, and faintness. Recurrent atrial fibrillation became more and more difficult to control without producing symptomatic bradycardia, and she was admitted for consideration of pacemaker therapy.

On physical examination murmurs of mitral regurgitation, aortic stenosis, and aortic regurgitation were present. An electrocardiogram showed sinus bradycardia at a rate of 50 beats/min with occasional A-V junctional escape beats. Left ventricular and left atrial enlargement were present. Chest x-rays showed enlargement of the left ventricle, left atrium, and right ventricle with calcification in the region of the mitral valve.

A temporary bipolar pacing electrode was placed in the right atrium which was paced reliably with a threshold of 7 ma. At the conclusion of the procedure atrial fibrillation developed and the patient was given digoxin. By the next day regular rhythm had returned, and an electrocardiogram showed 1:1 atrial pacing at a rate of 95. An irregular pacing response noted when the patient sat up was suggestive that the electrode at this time was no longer in contact with the atrial wall.

On July 5, 1967, 5 days after temporary pacing had been started, a curved unipolar atrial electrode* was passed into her right atrium via the right cephalic vein and was attached to the negative pole of a variable-rate pacemaker† set at 95 beats/min. The other pole was connected to a coil of wire imbedded subcutaneously as an indifferent lead. Atrial fibrillation had developed and the pacing threshold could not be determined. By the next day sinus rhythm had returned, but atrial pacing was erratic. A chest roentgenogram showed that the electrode had withdrawn into the superior vena cava. The electrode was repositioned so that the tip lay just inside the right atrium below the superior vena cava, and reliable atrial capture followed (fig. 1C). The patient's heart remained in a paced atrial rhythm. Chest films showed a decrease in heart size following

*Cordis Corp., Miami, Florida.
†Chardack-Greatbatch Pulse Generator, Model 5570-C, Medtronic, Inc., Minneapolis, Minnesota.
stabilization of the cardiac rhythm with pacing.

The patient was discharged to continue treatment with digoxin, warfarin, and quinidine. Transient paroxysms of atrial fibrillation were successfully treated with additional quinidine, but became increasingly more troublesome. Occasional electrical conversion was required, but atrial pacing was then re-established when the arrhythmias stopped and was present most of the time (fig. 1D). In December 1968, she developed a rapid arrhythmia while visiting in another city and was admitted to a local hospital. Shortly after admission and before the rhythm could be satisfactorily identified, cardiac arrest occurred. Resuscitative measures failed. Permission for autopsy was refused.

Case 4 (L. C., MGH #111-92-96)

This 89-year-old woman was admitted to the Massachusetts General Hospital on July 25, 1967, for further treatment of congestive heart failure which had persisted for over 18 months despite use of digitals and diuretics. At no time had she had chest pain or syncopal attacks, but she had been troubled particularly by increasing weakness. An electrocardiogram showed sinus bradycardia at an average rate of 40 beats/min with frequent A-V junctional escape beats.

Treatment with ephedrine and sublingual isoproterenol was poorly tolerated. Consequently, a trial of temporary atrial pacing was instituted. The patient observed that she felt better when paced at a rate of about 80 beats/min. When the pacemaker was turned off, the original symptoms of weakness returned, and she felt faint. On August 9, 1967, a curved atrial unipolar electrode was inserted through the right cephalic vein with the tip directed against the lateral aspect of the right atrial wall. The electrode was then connected to the variable-rate pulse generator which was set for 85 beats/min (same equipment as in case 3). The patient continued to improve and was discharged.

Brief periods of erratic pacing occurred during the subsequent months, but most of the time pacing was regular and she noted increased exercise tolerance. Three months later incessant atrial pacing recurred along with subjective symptoms of weakness. Reliable pacing could be produced by changes in body position and was most constant when the patient lay on her right side but became unreliable when she lay on her back, left side, or sat up. No change in position of the electrode could be detected by fluoroscopy. Exploration of the pacemaker system revealed an atrial stimulating threshold of 2.5 ma. A unipolar fixed-rate pulse generator* with a greater output was then implanted. These efforts, however, did not eliminate the intermittent erratic pacing, and she was readmitted in January 1968, when a permanent pervenous ventricular stand-by pacemaker was implanted. Ventricular pacing was established at a rate of 80 beats/min and intermittent 1:1 retrograde conduction to the atria was observed. The patient improved with the regular ventricular pacing and was discharged 2 weeks later. Reliable pacing has persisted in the interim, but the patient continues to have periods of weakness and malaise.

Case 5 (R.F., MGH #76-13-70)

This 75-year-old woman was admitted to the hospital in December 1967 for treatment of alternating atrial flutter and sinus bradycardia.

Several years previously she developed paroxysmal atrial fibrillation due to rheumatic mitral stenosis, and in August 1966 closed mitral valvulotomy was performed without complications. Sinus rhythm was maintained for 4 months with quinidine but its use was discontinued when nausea occurred. She then developed atrial fibrillation and atrial flutter, the latter associated with 2:1 A-V conduction and a ventricular rate of about 130 beats/min. Sinus rhythm was restored on several occasions with the use of quinidine or countershock, but after conversion the electrocardiogram revealed marked sinus bradycardia with A-V junctional escape beats.

In view of these difficulties the patient was readmitted to the hospital and on December 8, 1967, a curved atrial unipolar electrode was passed into the right atrial appendage via the left cephalic vein and attached to a fixed-rate pacemaker set at 95 beats/min. The impulse generator was buried in the left pectoral region. Chest x-rays (fig. 2) revealed the electrode to be directed anteriorly with a slight curve to the left (same equipment as in case 3). The threshold for stimulation was 2.2 ma.

When the patient did not take the quinidine as prescribed atrial flutter recurred which could be easily converted with additional doses of the drug. She took her medicines irregularly and consequently has had occasional atrial fibrillation or atrial flutter. However, the atrial pacemaker has continued to function satisfactorily when the patient does not have supraventricular tachycardia. Subsequent chest x-rays have shown no significant electrode displacement.

Discussion

Long-term atrial pacing was instituted in these patients for two types of problems: (1)

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*Ventricor Pacemaker, Cordis Corp., Miami, Florida.

† Ectocor Pacemaker, Cordis Corp., Miami, Florida.
ventricular tachycardia and fibrillation refractory to usual pharmacologic treatment (cases 1 and 2); and (2) sinus node disease with symptomatic bradycardia in cases 3, 4, and 5, with the added complication of periodic atrial tachyarrhythmias in cases 3 and 5.

Short-term rapid atrial pacing has been widely employed to override refractory life-threatening ventricular irritability when A-V conduction is intact. Since the phenomena which give rise to such arrhythmias are usually transient, for example digitalis toxicity, myocardial infarction, and myocarditis, pacing is usually not necessary for more than a few days. However, in our first two cases, large doses of antiarrhythmic drugs did not stabilize the rhythm, and permanent pacing was required. In both instances long-term pacing seemed lifesaving even though the first patient eventually reestablished permanent atrial fibrillation, at which point atrial pacing was no longer effective.

The last three patients appear to have had disease of the sinus node, with cases 3 and 5 falling into the category described by Short as “the syndrome of alternating bradycardia and tachycardia.” These patients have episodes of bothersome supraventricular arrhythmias, but conversion with antiarrhythmic drugs results in symptomatic bradycardia because of a slow sinus rate as well as an inadequate escape rate from lower foci. With atrial pacing, even though episodes of tachycardia may persist, the pacemaker prevents marked slowing of the heart when the arrhythmia ceases and allows more liberal and effective use of digitalis and antiarrhythmic drugs.

In our patients with repetitive supraventricular arrhythmias, we had hoped that stimulating the atria at a rapid rate might reduce the frequency of such attacks. In both cases, the number of episodes of atrial arrhythmias diminished, but we could not determine whether this beneficial effect was due to pacing per se or to the fact that pacing permitted the use of larger doses of antiarrhythmic drugs.

Currently, ventricular pacing preferably of the demand or standby type, plus antiarrhythmic drugs is the more commonly used method of treating patients such as those described here. Long-term atrial pacing is seldom employed, but there are obvious advantages to this method if it can be successfully instituted and maintained. Although
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there is some difference of opinion concerning the hemodynamic advantages of sequential atrial and ventricular contraction in normal man, the important contribution of atrial transport to the enhancement of ventricular contraction has been repeatedly shown in patients with diseased hearts. Documentation of this augmenting effect is particularly extensive in patients with complete heart block. Atrial pacing has also proven to be extremely helpful in the immediate postoperative period in patients who have undergone cardiac surgery.

Another significant advantage of atrial over ventricular stimulation is that pacing from the atrium is much less likely to generate ventricular irritability. This factor may be important when artificial pacing is being used to override life-endangering ventricular arrhythmias. Furthermore, reciprocating arrhythmias may develop with ventricular pacing when antegrade and retrograde A-V conduction is intact.

Myocardial perforation, a small but definite hazard of transvenous ventricular pacing, has not occurred in our experience with atrial pacing.

The most reliable form of atrial pacing lies in the implantation of electrodes directly into the wall of either the left or the right atrium, but this obviously requires a thoracotomy with its attendant risks in the elderly patients who develop these problems. Pervenous pacing is much simpler and safer. However, pervenous atrial pacing also poses problems, the most important one being that of erratic pacing caused by difficulty in positioning the tip of the electrode securely in contact with the right atrial musculature. In our first two patients, standard pacing electrodes were placed with their tips at the junction of the right atrium and superior vena cava. These units provided reliable but not constant pacing. Also the pacing threshold seemed high, and in the first patient, troublesome temporary stimulation of the right hemidiaphragm occurred.

In an attempt to obtain a more secure position within the right atrium, electrodes with a curved tip were employed. These wires were originally designed as the atrial sensing electrode in a pervenous synchronous pacing system. However, even with this electrode pacing was not constant in cases 3 and 4, and indeed the pacing system in the latter patient had to be changed from an atrial to a ventricular mode. The most constant pacing was achieved in case 5, in which this same electrode was introduced through the left cephalic vein and then wedged anteriorly into the right atrial appendage where it has remained. Constant atrial pacing has been present between episodes of supraventricular arrhythmias. We will try for similar electrode placement in the future.

The right atrial appendage may not be the only site where satisfactory pervenous atrial pacing can be accomplished. Moss and associates and Gonzales recently obtained reliable pacing by positioning the electrode tip in the coronary sinus. Although promising, the assessment of the reliability and safety of this method awaits the passage of further time.

Acknowledgment

The authors would like to thank Dr. Peter M. Yurchak for permission to report on patient L.C. (case 4) and Mrs. Harriet Walker who typed the manuscript.

Addendum

Curved atrial electrodes have been successfully used in another patient since submission of this manuscript.

W.G. (MGH #155-47-50) is a 54-year-old man who had his second myocardial infarction in January 1969. He was transferred to the Massachusetts General Hospital for treatment of repetitive and refractory ventricular irritability in the presence of a slow sinus rhythm. A curved electrode was passed under fluoroscopic control into the right atrial appendage through the right jugular vein. Ventricular irritability markedly decreased with temporary atrial pacing at a rate of 105/min. Stimulation was constant despite normal body movements. On March 27, 1969, another curved electrode was inserted through a vein in the left pectoral region and directed into the region of the right atrial appendage for permanent pacing. Reliable atrial stimulation at a rate of 100/min was established. (Equipment was the same as in case 5). The patient returned to his home to continue treatment with digoxin, quinidine, and propranolol and to resume moderate physical activity. When last examined on July
2, 1969, constant 1:1 atrial pacing was present with occasional ventricular premature beats. (This patient was kindly referred by Drs. C. Hauser and W. N. St. John, Glens Falls, New York.)

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If one doubts the necessity for controls, reflect on the statement: “It has been conclusively demonstrated by hundreds of experiments that the beating of tom-toms will restore the sun after an eclipse.”—E. BRIGHT WILSON, Jr.: An Introduction to Scientific Research. New York, McGraw-Hill Book Co., Inc., 1952, p. 41.
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