Permanent Pervenous Atrial Pacing from the Coronary Vein
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SUMMARY
Permanent pervenous atrial pacing from the coronary vein has been attempted in 14 patients. Several types of arrhythmias including symptomatic bradycardia, "brady-tachy" syndrome, and refractory ventricular tachyarrhythmia-fibrillation have been successfully treated with pervenous atrial pacing in 10 patients for 1 to 30 mo. Long-term atrial pacing was unsuccessful in four patients; in two of these four this was due to high atrial pacing thresholds. To date there has been no evidence of pacemaker perforation or pacemaker-induced coronary vein thrombosis. It is concluded that permanent pervenous atrial pacing from the coronary vein is a reliable method of atrial pacing without resorting to thoracotomy.

Additional Indexing Words:
Demand atrial pacing Bradycardia-tachycardia syndrome Sinus Bradycardia Ventricular tachyarrhythmia Sinoatrial block

RECENTLY, long-term cardiac pacing has been used for the treatment of arrhythmias in patients with intact atrioventricular conduction.1-18 The desirability of using atrial pacing to maintain nearly normal hemodynamic and electrophysiologic relationships has been documented.19-28 However, the difficulty of maintaining a stable atrial pacing site without resort to thoracotomy8-18 has resulted in a limited application of atrial pacing on a long-term basis.14-18, 29

The purpose of this communication is to describe our experience with long-term pervenous left atrial pacing using an electrode catheter positioned in the proximal aspect of the coronary vein. A variety of arrhythmias including symptomatic supraventricular bradycardia, bradycardia-tachycardia syndrome, and recurrent ventricular tachyarrhythmia have been successfully managed using this technic.

Methods
Details of the implantation of the coronary venous left atrial pacemaker have been previously described from this laboratory.16, 18 In all patients the pacemaker electrode* was introduced through the thoracic portion of a cephalic vein. The left side has been the preferred approach for coronary vein catheterization. Adequacy of atrioventricular conduction was first assessed by means of atrial pacing using an external generator at frequencies between 70 and 130/min with the electrode placed in contact with the atrial endocardium. The P-R interval was measured directly.

Positioning of the pacemaker electrode in the coronary vein was done under fluoroscopic visualization. With the electrode tip formed into a "J" configuration, the electrode was cautiously advanced in a superior and posterior direction with respect to the tricuspid valve. Proper

*Medtronic catheter electrode, model 5816 or 5818. Medtronic, Inc., Minneapolis, Minnesota.
position in the coronary vein was documented by a typical left atrial P-wave configuration on the electrocardiogram and the orientation of the electrode under fluoroscopy. In all patients an attempt was made to obtain consistent pacing at an excitation threshold of less than 3 ma.

Following catheter positioning, the electrode was connected to an implantable pulse generator* and placed in a subcutaneous pocket in the left pectoral region. A 12-lead electrocardiogram was obtained on each patient, and the pacemaker artifact preceded P waves that were inverted in leads I and V6 with a mean vector oriented in the cephalad direction. Posteroanterior and lateral chest roentgenograms revealed that the electrodes were located posteriorly and inferiorly with respect to the left atrium.

In selected patients, unipolar and bipolar electrograms were recorded from the ring and tip electrodes of the bipolar catheter positioned in the coronary vein. The voltage of the atrial and ventricular complexes could be quantitated with respect to the threshold required to trigger a demand pulse generator.

Results

The overall results of successful permanent pervenous left atrial pacing from the coronary vein are presented in table 1. In 10 of 14 attempted placements, effective left atrial pacing has persisted without difficulty from 1 to 30 mo. In two of these 10 successful cases, elective battery replacement was performed at 18 and 23 mo (cases 1 and 9, respectively). Atrial pacing threshold at the time of battery change was less than 3 ma. No examples of coronary vein thrombosis or perforation have been observed.

Electrograms from the ring and tip electrodes have been recorded in four patients. Unipolar ring and tip electrograms as well as a bipolar ring-tip tracing from patient 3 are presented (fig. 1). In the proximal portion of the coronary vein the atrial electrogram reflects an early period of atrial depolarization and a later, presumably left atrial depolarization in the more distal portions of the vein. The same is true for the ventricular electrogram with respect to the initial and terminal phases of ventricular depolarization

*Medtronic pulse generator, model 5870-C (fixed rate) or 5841 (demand). Medtronic, Inc., Minneapolis, Minnesota.

Figure 1
Bipolar (top), unipolar tip (middle), and unipolar ring (bottom) electrograms recorded in patient 3 using a Medtronic model 5818 electrode catheter. Standardization: 10 mm = 1 mv. The bipolar atrial signal is of sufficient amplitude (>2 mv) to be sensed by the Medtronic demand generator (model 5841).

(fig. 2). The relative amplitudes of the atrial and ventricular electrograms determine whether the pacemaker will operate in the atrial or ventricular demand mode. The bipolar atrial signal was of sufficient magnitude (>2 ma) in three of the four patients in whom it was measured to meet the threshold requirement of the demand generator used in our laboratory. Atrial demand pacing has been accomplished by this pervenous technic (fig. 3).

Symptomatic Supraventricular Bradyarrhythmia

In two of the four patients with symptomatic sinus node disease, periods of asystole were of sufficient length to result in syncopal episodes. In addition, one had transient cerebral ischemic attacks, and one had intermittent orthostatic hypotension. Atropine (0.5 g to 1.0 mg orally every 4 to 6 hours) failed to result in significant improvement. In these four patients, the establishment of left atrial stimulation using a pervenously placed coronary vein electrode has resulted in acceptable heart rate and rhythm control for periods of 1
BIPOLAR ELECTROGRAM

DISTAL CORONARY VEIN

PROXIMAL CORONARY VEIN

Figure 2
Bipolar electrograms (BE) recorded from distal (left panel) and proximal (right panel) locations in the coronary vein simultaneously with ECG lead I. Record of a patient obtained during cardiac catheterization employing a U. S. Catheter Corporation bipolar electrode catheter (model 5651). The atrial and ventricular electrograms recorded at the distal site represent terminal (presumably left) atrial and ventricular depolarization, respectively. Both electrograms recorded at the proximal site represent an earlier portion of atrial and ventricular depolarization. The adequacy of the atrial or ventricular signal as a demand stimulus depends on the location of the electrode within the coronary vein and on the magnitude of the depolarization vector for the atrial or ventricular tissue underlying the electrode sites.

to 23 mo. Bradycardic symptomatology has been completely eliminated.

Case 3 (W.B. SMH #56-62-31)
A 70-year-old hypertensive male had documented sinoatrial node disease with ineffective secondary escape foci. Over a 7-year period he had experienced intermittent, 3 to 5 sec periods of sinus arrest associated with symptoms of lightheadedness, nausea, and fatigue and one episode of syncope. Oral atropine (0.5 mg every 6 hours), which resulted in dry mouth and urinary hesitancy, failed to control the patient's symptoms. A pervenous coronary vein pacemaker was implanted on October 31, 1969, with a demand stimulus rate of 72/min. Appropriate position was confirmed by electrocardiogram and chest roentgenograms. Subjective improvement has followed pacemaker implantation, and during the follow-up period lightheadedness and syncope have been eliminated. The patient is receiving no medications.

Bradycardia-Tachycardia Syndrome
Four patients (cases 5 to 8) manifested the so-called bradycardia-tachycardia syndrome with three of the four having atrial flutter and fibrillation and the fourth having both atrial and A-V junctional tachycardia. All four patients were receiving digitalis therapy and had received one or more antiarrhythmic drugs without significant reduction in the episodes of tachyarrhythmia. Two of the four patients required cardioversion because of prolonged episodes of tachyarrhythmia and in two the effect of atropine on the sinus rate failed to reduce the number of such episodes. Permanent pervenous atrial pacing has been accomplished in all four at frequencies of 73 to 94/min for periods from 6 to 10 mo. In addition to providing an acceptable heart rate
Table 1

**Patient Summary: Successful Long-Term Pervenous Atrial Pacing**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, sex</th>
<th>Heart disease</th>
<th>Arrhythmia</th>
<th>Rhythm Type</th>
<th>Rate (/min)</th>
<th>P-R (sec)</th>
<th>Symptoms</th>
<th>Duration (yr)</th>
<th>Pacemaker Implant date, generator rate, P-R, threshold</th>
<th>Follow-up Duration, medication, symptoms, comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. N.T.</td>
<td>64 M</td>
<td>HCVD CHD-DMI</td>
<td>Sinus bradycardia</td>
<td>35-40</td>
<td>0.14</td>
<td>Orthostatic dizziness, CHF</td>
<td>3</td>
<td>March 1968, fixed rate, 75/min, 0.14 sec, 2.0 ma</td>
<td>23 mo, no therapy, no arrhythmia, dizziness; CHF improved, generator change Jan. 1970</td>
<td></td>
</tr>
<tr>
<td>2. W.O'C.</td>
<td>60 M</td>
<td>HCVD CHD-DMI</td>
<td>Sinus bradycardia</td>
<td>40</td>
<td>0.19</td>
<td>Transient cerebral ischemia</td>
<td>1</td>
<td>Sept. 1969, demand, 72/min, 0.19 sec, 2.5 ma</td>
<td>6 mo, no therapy, no arrhythmia, no symptoms</td>
<td></td>
</tr>
<tr>
<td>3. W.B.</td>
<td>70 M</td>
<td>Idiopathic</td>
<td>Sinoatrial block</td>
<td>40</td>
<td>0.20</td>
<td>Dizziness, Syncope</td>
<td>7</td>
<td>Oct. 1969, demand, 73/min, 0.18 sec, 1.3 ma</td>
<td>4 mo, no therapy, no arrhythmia, no symptoms</td>
<td></td>
</tr>
<tr>
<td>4. W.W.</td>
<td>88 M</td>
<td>CHD</td>
<td>Sinoatrial block</td>
<td>45</td>
<td>0.24</td>
<td>Syncope, CHF</td>
<td>4</td>
<td>Feb. 1968, fixed rate, 100/min, 0.22 sec, 2.0 ma</td>
<td>1 mo, no therapy, no arrhythmia, no syncope; CHF improved, died - noncardiac cause, March 1968</td>
<td></td>
</tr>
<tr>
<td>5. M.B.</td>
<td>70 F</td>
<td>CHD</td>
<td>Sinus bradycardia</td>
<td>35-40</td>
<td>0.20</td>
<td>Tachycardia, CHF</td>
<td>1</td>
<td>April 1969, fixed rate, 79/min, 0.20 sec, 1.5 ma</td>
<td>10 mo, digitoxin, 1 episode - atrial flutter, spontaneous reversion to paced rhythm</td>
<td></td>
</tr>
</tbody>
</table>

**Sinus bradycardia-sinoatrial block**

**BradyCARDIA-TACHYCARDIA SYNDROME**
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Pacing Type</th>
<th>P-R Interval</th>
<th>Heart Rate</th>
<th>Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.</td>
<td>R.A.</td>
<td>53 M</td>
<td></td>
<td>ASD—repair</td>
<td>CHD-DMI</td>
<td>35-45</td>
<td>0.16</td>
<td>Tachycardia (angina)</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CHD-DMI</td>
<td></td>
<td>Atrial flutter</td>
<td></td>
<td>Syncope</td>
<td>7 mo, Procainamide; digoxin, no tachycardia, rare VPCs, no symptoms</td>
</tr>
<tr>
<td>7.</td>
<td>A.P.</td>
<td>74 F</td>
<td></td>
<td></td>
<td>CHD-DMI</td>
<td>55</td>
<td>0.20</td>
<td>Tachycardia (angina)</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinus bradycardia</td>
<td></td>
<td>A-V junctional tachyarrhythmia</td>
<td></td>
<td>CHF</td>
<td>7 mo, quinidine; digoxin, persistent episodes, of junctional tachycardia; terminated spontaneously or with paced atrial stimuli</td>
</tr>
<tr>
<td>8.</td>
<td>A.F.</td>
<td>60 M</td>
<td></td>
<td>Idiopathic</td>
<td>CHD-DMI</td>
<td>40-50</td>
<td>0.18</td>
<td>Tachycardia</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinoatrial block</td>
<td></td>
<td>Atrial flutter &amp; fibrillation</td>
<td></td>
<td></td>
<td>6 mo, digoxin; quinidine, no arrhythmia, no symptoms</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Refractory ventricular tachyarrhythmia and fibrillation</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>A.R.</td>
<td>69 M</td>
<td></td>
<td></td>
<td>CHD-AMI</td>
<td>65</td>
<td>0.24</td>
<td>Ventricular fibrillation</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinus rhythm</td>
<td></td>
<td>Refractory vent. tachyarrhythmia-fibrillation</td>
<td></td>
<td></td>
<td>29 mo, propranolol; digoxin, moderate number VPCs, generator change April 1969</td>
</tr>
<tr>
<td>10.</td>
<td>M.W.</td>
<td>61 M</td>
<td></td>
<td></td>
<td>CHD-DMI</td>
<td>70</td>
<td>0.24</td>
<td>Ventricular fibrillation</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinus rhythm</td>
<td></td>
<td>Refractory vent. tachyarrhythmia-fibrillation</td>
<td></td>
<td></td>
<td>3 mo, Dilantin, propranolol; digoxin, moderate number VPCs</td>
</tr>
</tbody>
</table>

*Excludes data on four patients in whom attempted coronary vein pacing was unsuccessful. See text.
†Fixed-rate generators — Medtronic model 5870-C; demand generators — Medtronic model 5841.
‡Following pacemaker insertion, transient periods of markedly prolonged P-R interval have been observed. No fluoroscopic evidence of electrode migration has been noted. P-R prolongation is presumed to be a manifestation of underlying coronary heart disease.
§Atrial pacing has been employed despite P-R prolongation because of superior hemodynamic results.

Abbreviations: HCVD = hypertensive cardiovascular disease; CHD = coronary heart disease; DMI = diaphragmatic myocardial infarction; CHF = congestive heart failure; ASD = atrial septal defect; VPC = ventricular premature contraction; AMI = anterior wall myocardial infarction.
ATRIAL DEMAND PACING—CORONARY VEIN

Figure 3

Atrial demand pacing from a coronary vein electrode. Lead II ECG (upper panel) documents atrial demand pacing in a patient in whom a Medtronic electrode (model 5818) and generator (model 5841) had been positioned. Because of inconstant capture, the electrode had to be repositioned to the right ventricle. An enlarged diagram (lower panel) indicates the significant findings on the electrocardiogram. The initial pacemaker artifact is followed by an atrially stimulated beat. The second pacemaker artifact fails to stimulate the atrium and is followed by an escape sinus beat. At the automatic cycle length of 650 msec, a pacemaker stimulus should have fallen in the downslope of the sinus P wave. Absence of such a pacemaker artifact indicates that the demand pacemaker was inhibited by atrial depolarization. The time of appearance of the subsequent pacemaker artifact is further evidence for atrial demand sensing.

and rhythm, atrial pacing has allowed more effective use of digitalis and suppressant therapy with no subsequent episodes of tachyarrhythmia in two of the four patients, and with marked reduction in the frequency of episodes in the other two patients.

Case 6 (R.A. SMH #41-40-43)

A 53-year-old man had documented coronary heart disease and underwent repair of atrial septal defect at another hospital in November 1968. Paroxysmal episodes of atrial flutter and fibrillation had been documented since 1963. Specific therapy was not employed until April 1968 when, in association with a slow unstable atrial pacemaker, the episodes of tachyarrhythmia became more frequent and resulted in dyspnea and coronary chest pain. Direct-current cardioversion of sustained episodes was accomplished on four occasions. However, attempts at suppression of arrhythmia with quinidine, procainamide, or propranolol, alone or in combination with digoxin, were unsuccessful. Moreover, suppressant therapy resulted in more sustained symptomatic bradycardyarrhythmia with inadequate nodal escape foci and frequent atrial and ventricular premature beats. On August 3, 1969, the patient was hospitalized, and all suppressant drugs were discontinued. Symptomatic bradycardyarrhythmia persisted and was punctuated by two episodes of atrial flutter with rapid ventricular response requiring cardioversion. This was undertaken only after prophylactic, temporary insertion of a ventricular pacemaker.

Because of the adequacy of A-V conduction manifest during episodes of tachyarrhythmia and the theoretical desirability of an atrial pacing site in controlling the atrial ectopic foci, a temporary pervenous coronary vein pacemaker was inserted. Left atrial pacing resulted in a stable rhythm at a rate of 90/min and allowed the administration of procainamide and digoxin therapy. A permanent demand coronary vein pacemaker was implanted on August 8, 1969, and in the follow-up period the only arrhythmia experienced by the patient has been occasional ventricular premature contractions. The patient has been able to return to full-time employment with infrequent angina.

Refractory Ventricular Tachyarrhythmia

In two patients ventricular tachyarrhythmia that deteriorated into ventricular fibrillation proved refractory to vigorous antiarrhythmia therapy in addition to careful attention to electrolyte balance and maintenance of cardiac compensation. In each instance the arrhythmia arose in the setting of an acute myocardial infarction and required d-c cardioversion or defibrillation. Use of atropine to achieve a more rapid sinus response than the control heart rates (65-70 beats/min) also failed to abolish the arrhythmia. Pervenous atrial pacing from the coronary vein at frequencies of 86 and 110/min in addition to procainamide or lidocaine resulted in control of the arrhythmia and provided a more satisfactory pulse pressure than ventricular pacing at comparable rates. In two other patients temporary atrial pacing from the coronary vein for periods of 1 to 2 weeks allowed control of drug refractory ventricular
tachyarrhythmia in the setting of an acute myocardial infarction.

Case 10 (M.W. SMH #60-24-86)

A 61-year-old man was hospitalized in November 1969 with an acute diaphragmatic myocardial infarction complicated by intermittent complete heart block. A permanent right ventricular demand pacemaker was implanted. In January 1970, the patient was readmitted with ventricular irritability complicated by hypotension (systolic BP, 80 mm Hg) and low cardiac output. Despite demand ventricular pacing at a frequency of 86/min and suppressant therapy including parenteral procainamide, propranolol, and lidocaine, direct current cardioversion was required for control of recurrent episodes of ventricular tachyarrhythmia. It was observed that sinus conducted beats with a P-R interval of 0.24 sec resulted in systolic cuff pressures 20 mm Hg greater than those generated by paced ventricular beats. Because of the persistent hypotension the pacing electrode was repositioned in the coronary vein and atrial pacing was accomplished without difficulty. Although the generator frequency was left unaltered (86 beats/min), the patient’s hemodynamic state improved considerably with atrial pacing and the systolic blood pressure stabilized at 100 mm Hg. Digoxin and procainamide therapy were maintained, and over a 1-mo period of observation, the recurrent episodes of ventricular tachyarrhythmia have been completely eliminated.

Unsuccessful Atrial Pacing From the Coronary Vein

Atrial pacing electrodes were inserted into the coronary vein in four other patients, but the catheters had to be repositioned in the right ventricle because of a variety of problems. In two patients, the threshold for pacing at the time of pacemaker implantation was in excess of 5 ma; inconstant atrial pacing subsequently developed. In one patient with an unsuspected presence of a left superior vena cava, the electrode could not be stably positioned in the coronary vein. It was assumed that the large size of the coronary vein and the angle at which the left superior vena cava entered the coronary vein prevented stabilization. In the fourth patient, electrode repositionment was necessitated by the subsequent development of intraventricular conduction delay at the rapid atrial paced rate.

Discussion

Permanent pervenous atrial pacing was undertaken for the control of three types of cardiac arrhythmia: (1) symptomatic sinus bradycardia with abnormal sinus node impulse formation and failure of secondary escape foci; (2) intermittent sinus bradycardia complicated by paroxysmal atrial or junctional tachyarrhythmia; and (3) drug refractory ventricular tachyarrhythmias.

In patients with symptomatic bradycardia due to disorders of sinus node impulse formation or propagation, or both, atrial pacing may be employed to provide an adequate rate and rhythm. By pacing from the atrium the hemodynamic temporal relationship between atrial and ventricular contraction and the excitation transmission over a normal intraventricular conduction pathway are maintained. With left atrial stimulation the P-R interval in all instances but one was equal to or shorter than the control value. In case 7, the exception to this observation, postulated intermittent A-V node ischemia resulted in temporary but marked P-R prolongation. The comparability of the atrial contribution to ventricular filling and function resulting from left and right atrial stimulation is currently being studied in our laboratory. In the clinical situation, however, the pulse pressure derived from left atrial pacing was clearly superior to that obtained from ventricular pacing at a similar rate. In addition, atrial pacing reduces the danger of competition between paced and spontaneous ventricular rhythm and eliminates the potential of mechanically induced ventricular ectopic beats and re-entrant ventricular arrhythmias.

In patients with the so-called bradytachy syndrome, the efficacy of pacemaker suppression may reside in a reduction in the prolonged diastolic period which predisposes to ectopic activity as well as an improvement in the homogeneity of recovery of excitability at more rapid rates. At rapid frequencies, intrinsic pacemaker activity from an ectopic focus may be inhibited by the mechanism of overdrive suppression. In addition, altera-
tion of critical timing relationships in re-entrant pathways may abolish re-entrant arrhythmias.40-44 This was illustrated by patient 7 in whom use of an induction coil to vary the timing of the pacemaker artifact resulted in abolition of a junctional tachyarrhythmia within 2 to 3 beats.

Many of the above-described mechanisms may be operative in preventing ventricular tachyarrhythmia. In addition, continued depolarization of the ectopic focus may result from rapid pacing. In one of these patients atrial pacing was consistently effective in controlling the arrhythmia while ventricular pacing was not. This suggests that the hemodynamic benefit of atrial pacing may be operative in addition to the electrophysiologic mechanisms described above.45, 46

Permanent demand atrial pacing using the pervenous approach has not been previously reported. The relative amplitudes of the atrial and ventricular electrograms recorded from the coronary vein site determine whether the demand pacemaker will operate as an atrial (fig. 3) or ventricular (fig. 4) demand unit. Since ventricular stimulation from distal coronary vein locations has been reported,47-50 the potential exists for atrial synchronized ventricular pacing from a single catheter positioned in the coronary vein. A dominant atrial electrogram recorded from the proximal ring electrode of a bipolar coronary vein catheter could be used to synchronize ventricular stimulation via the distal-tip electrode. This approach is presently under investigation.

**References**

5. Cohen LS, Buccino RA, Morrow AG, et al: Recurrent ventricular tachycardia and fibrillation treated with a combination of beta-
30. Mirosky M: Left atrial rhythm: Diagnostic criteria and differentiation from nodal arrhythmias. Amer J Cardiol 17: 203, 1966

37. **Lange C**: Action of driving stimuli from intrinsic and extrinsic sources on in situ cardiac pacemaker tissue. Circulation Research 17: 449, 1965


46. **Han J**: Ventricular vulnerability during acute coronary occlusion. Amer J Cardiol 24: 857, 1969


