Temporary Transvenous Catheter-Electrode Pacing of the Heart

By ROBERT C. TANCREDI, M.D., BEN D. MCCALLISTER, M.D., AND HAROLD T. MANKIN, M.D.

SUMMARY
Experience with 110 separate periods of transvenous cardiac pacing by means of a catheter-electrode in 91 patients has been reviewed. Indications for the use of the catheter-electrode included (1) complete heart block with and without Adams-Stokes syndrome, (2) other arrhythmias with and without cardiogenic syncope, (3) malfunction of previously implanted permanent pacemaker units, and (4) need for pacing during general surgical procedures in patients with a variety of rhythm disturbances. A case illustrating the combined use of drug therapy and catheter-electrode pacing in controlling paroxysmal ventricular tachycardia is presented.

Only six deaths occurred in spite of the serious heart disease in all 91 patients in this series. Two were related to complications of temporary transvenous intracardiac pacing. The major complications associated with the procedure included perforation of the heart, bacteremia, acute myocardial infarction, cephalic vein phlebitis, and ventricular tachyarrhythmia. Minor problems were primarily related to equipment failure or positional difficulties with the catheter-electrode and were usually of no serious consequence.

Additional Indexing Words:
Heart block
Cardiac perforation
Adams-Stokes syndrome
Myocardial infarction
Arrhythmias
Bacteremia

In 1952, Zoll\(^1\) successfully stimulated the human heart by means of electrodes applied externally to the chest wall. Although this technique was not suitable for long-term pacing of the heart, it began a new era in the treatment of cardiac arrhythmias. Furman and Robinson\(^2\) in 1958, first described the technique of intracardiac pacing in man. Electric stimulation and pacing of the heart were successfully accomplished by means of a unipolar catheter-electrode inserted transvenously into the right ventricular cavity. Certain undesirable features were associated with the use of the unipolar electrode, however, and Parsonnet and co-workers\(^3,4\) developed the dipolar catheter-electrode in 1962. Since that time the dipolar catheter-electrode has been used with considerable success in various patients requiring cardiac pacing for relatively short periods. In our institution, this method has now largely replaced other techniques of temporary cardiac stimulation, such as myocardial wires, sympathomimetic drugs, and external cardiac pacing.

Although the catheter-electrode provides an effective and dependable means of pacing the heart, certain complications associated with its use deserve further emphasis and prompted a review of our experience during a period of 30 months.

**Methods**

Clinical records of all patients who underwent intracardiac pacing by means of a catheter-electrode at the Mayo Clinic from October 1963 through March 1966 were reviewed. One hundred ten separate periods of intracardiac pacing in 91 different patients (66 males and 25 females) are represented in this series. The patients...
ranged in age from 5 to 85 years, with a mean age of 68 years. Six patients were less than 40 years old and 66 were between 60 and 80 years old.

The duration of catheter-electrode pacing ranged from several hours to 54 days. Twelve periods of pacing exceeded 10 days. Thirty-nine catheter-electrodes were inserted within 24 hours of operation for implantation of permanent internal pacemakers.

All catheter-electrode insertions were made under fluoroscopic guidance in the cardiac catheterization laboratory. While the patient was being taken to the laboratory, a portable, battery-powered, external pacemaker-defibrillation unit was connected to the patient for emergency use. The electrocardiogram was monitored continuously throughout placement of the catheter-electrode. The right external jugular vein was isolated by cutdown when possible, unless eventual implantation of a permanent transvenous pacemaker was planned for this location. However, if a suitable vein could not be located in the neck, the right antecubital vein was used (occasionally it may be necessary to use a saphenous vein). The dipolar catheter-electrode* was positioned in the pulmonary artery, and then a transistorized power pack† was connected to the proximal leads. The catheter-electrode was then slowly withdrawn into the right ventricular outflow tract until adequate pacing was initiated and maintained. Effective pacing was usually accomplished with a current of about 2.5 mA at rates of 70 to 75 beats/min, although settings were individualized. It has been our practice to set the power pack for approximately 2 mA above the minimal threshold level required for cardiac pacing in each patient. After successful pacing was established, the catheter-electrode was sutured in place and the wound was closed around the externalized portion. Various head and body positions were tested to ensure that adequate pacing persisted. All patients were returned to the medical intensive-care unit for continual observation and cardiac monitoring. Anticoagulants were not administered during intracardiac pacing unless their use was indicated for other reasons, and antibiotics were not given unless repeated manipulation of the catheter-electrode was necessary.

Gordon* and Lister and co-workers† have described methods for detecting failures in catheter-electrode pacing systems, and similar methods have been most useful in our experience.

* Bipolar platinum electrode (H5651), U. S. Catheter & Instrument Corp., Glens Falls, New York.
† External pacemaker (Model 5800), Medtronic, Inc., Minneapolis, Minnesota.

Indications for Transvenous Intracardiac Pacing

The indications for pacing in this series of patients are summarized in table 1. For discussion these have been divided into the following categories: (1) complete heart block, with and without Adams-Stokes syndrome, (2) other cardiac arrhythmias, with and without cardiovagal syncope, (3) malfunction of previously implanted permanent pacemaker units, and (4) temporary pacing during surgical procedures in patients with a variety of rhythm disturbances.

Complete Heart Block

With Adams-Stokes Syndrome

Forty-five patients underwent intracardiac pacing because of complete heart block with the Adams-Stokes syndrome (table 2). Paroxysmal ventricular tachyarrhythmias were noted in four of these patients.

Twenty-one of the 45 patients had no evidence of ischemic or valvular heart disease. Six of these 21 patients had moderately severe congestive heart failure associated with the complete heart block. Fifteen additional patients had a history of angina pectoris or previous myocardial infarction. Catheter-electrode pacing was used in all but one of these patients prior to and during operation for implantation of permanent internal pacemakers. The patient who did not undergo operation represents an early and unsuccessful attempt at pacing with a unipolar elec-

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Table 1

<table>
<thead>
<tr>
<th>Indication</th>
<th>Instances</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete heart block</td>
<td>54</td>
</tr>
<tr>
<td>With Adams-Stokes syndrome</td>
<td>46</td>
</tr>
<tr>
<td>Without Adams-Stokes syndrome</td>
<td>8</td>
</tr>
<tr>
<td>Other cardiac arrhythmias</td>
<td>14</td>
</tr>
<tr>
<td>With cardiovagal syncope</td>
<td>12</td>
</tr>
<tr>
<td>Without cardiovagal syncope</td>
<td>2</td>
</tr>
<tr>
<td>Difficulties with previously implanted permanent pacemaker</td>
<td>36</td>
</tr>
<tr>
<td>Temporary pacing during surgical procedures in patients with various arrhythmias</td>
<td>6</td>
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</table>
Table 2

Indications for Temporary Transvenous Pacing in Patients With Complete Heart Block

<table>
<thead>
<tr>
<th>Indication</th>
<th>Pt</th>
<th>Instances</th>
<th>PPM*</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>With Adams-Stokes syndrome</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No other heart disease</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Without congestive heart failure</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>0</td>
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<tr>
<td>With congestive heart failure</td>
<td>6</td>
<td>6</td>
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<td>Ischemic heart disease</td>
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<td>0</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td>4</td>
<td>5</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Acute bacterial endocarditis</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
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<td>45</td>
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<td>3</td>
</tr>
<tr>
<td><strong>Without Adams-Stokes syndrome</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina and congestive heart failure</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>0</td>
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<tr>
<td>Cerebral ischemia</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>After repair of tetralogy (digitalis)</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>2</td>
<td>2</td>
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<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>8</td>
<td>8</td>
<td>2</td>
<td>1</td>
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</table>

*Permanent pacemaker implanted later.

trode; control was achieved in this patient eventually by drug therapy alone.

Intrakardial pacing was initiated in four patients because of Adams-Stokes syndrome associated with acute myocardial infarction. One of these patients had had complete heart block with Adams-Stokes syndrome for several years prior to the infarction that necessitated hospitalization. A cardiac pacemaker was inserted, but the patient died despite technically adequate pacing. In the three remaining patients, the conduction disturbances developed for the first time soon after an acute myocardial infarction. One patient died in spite of technically adequate pacing. In the second patient, normal sinus rhythm returned in 6 days, and the clinical course was otherwise uncomplicated. The third patient, who had ventricular fibrillation prior to the insertion of a transvenous cardiac pacemaker, had two additional episodes of ventricular fibrillation in spite of procainamide therapy and technically adequate cardiac pacing. One of these episodes was successfully treated with intravenous injection of procainamide and direct-current countershock. The other episode subsided spontaneously when the catheter-electrode pacemaker was turned off. Normal sinus rhythm returned within 1 week, and there were no further complications.

Acquired calcific aortic stenosis was associated with complete heart block and Adams-Stokes syndrome in four patients. Catheter-electrode pacing was instituted prior to cardiac surgery, and in each of these patients successful prosthetic replacement of the aortic valve was accomplished without incident. Permanent internal pacemakers were implanted at the time of the aortic valve surgery in two of these patients. Normal sinus rhythm resumed in the immediate postoperative period in the other two patients. One of these two patients remained in permanent normal sinus rhythm, but the other patient subsequently returned 2 months later because of recurrence of complete heart block associated with Adams-Stokes syndrome. A catheter-electrode pacemaker was placed prior to implantation of a permanent pacemaker.

One patient in this group was hospitalized for treatment of acute bacterial endocarditis. Blood cultures revealed the presence of *Staphylococcus aureus*, and appropriate antibiotic therapy was started. Ten days after

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admission, complete heart block developed; a subsequent cardiac arrest responded well to resuscitative measures. Catheter-electrode pacing was begun shortly thereafter and was continued for 4 days, at which time the patient died despite technically adequate pacing.

**Without Adams-Stokes Syndrome**

Eight patients with complete heart block required intracardiac pacing for reasons other than Adams-Stokes syndrome. Five of these patients were known to have ischemic heart disease.

Five patients were noted to have symptoms possibly related to a low cardiac output without evidence of ventricular asystole or tachyarrhythmia. Three of these five patients had severe and progressive angina pectoris with signs and symptoms of increasing heart failure; two of the three patients showed marked improvement during a trial of pacing, and permanent pacemakers were subsequently implanted. The third patient did not improve during catheter-electrode pacing. The two other patients had symptoms suggesting cerebral ischemia (intermittent confusion, confusion, and disorientation), but a trial of cardiac pacing with the catheter-electrode did not improve the mental status. Because of the poor clinical response to temporary pacing in these three patients, permanent internal cardiac pacemakers were not implanted.

Complete heart block without Adams-Stokes syndrome occurred in one patient within 24 hours after surgical repair of a tetralogy of Fallot. Excessive digitalis was also implicated as a contributing factor, and catheter-electrode pacing was used until normal sinus rhythm resumed (approximately 24 hours).

Complete heart block without Adams-Stokes syndrome developed early in the clinical course of two patients with acute myocardial infarction. In one patient, complete heart block with a ventricular rate of 53 beats/min developed on the third day after an acute myocardial infarction. Hypotension (80 mm Hg systolic and 40 diastolic) and a low urinary output improved promptly after institution of catheter-electrode pacing. Normal sinus rhythm returned in 3 days, and the patient recovered without incident. The second patient died in spite of technically satisfactory cardiac pacing.

**Other Arrhythmias**

**With Cardiogenic Syncope**

In 12 patients with cardiogenic syncope, electrocardiograms revealed the presence of arrhythmias other than complete heart block (table 3). One patient in this group had first-degree atrioventricular (A-V) block with left

### Table 3

**Indications for Temporary Transvenous Pacing in Patients with Other Arrhythmias**

<table>
<thead>
<tr>
<th>Indication</th>
<th>Pt</th>
<th>Instances</th>
<th>PPM*</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>With cardiogenic syncope</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First-degree A-V block</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Second-degree A-V block</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2:1</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>3:2</td>
<td>1</td>
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<tr>
<td>Other</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular tachyarrhythmias</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
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<td><strong>Without cardiogenic syncope</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2:1 A-V block</td>
<td>1</td>
<td>1</td>
<td></td>
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</tr>
<tr>
<td>(therapeutic trial)</td>
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<tr>
<td>Paroxysmal ventricular tachycardia</td>
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<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>2</td>
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</tr>
</tbody>
</table>

*Permanent pacemaker implanted later.*
axis deviation and a right bundle-branch block; five patients had second-degree A-V block (2:1 or 3:2 block). In these six patients, temporary cardiac pacing was instituted prior to surgical implantation of permanent pacemakers.

The other six patients in this group had various types of supraventricular and ventricular arrhythmias. In one patient, an arrhythmia was first manifested by paroxysmal atrial tachycardia with a high-grade variable block which was not thought by the attending physician to be related to digitalis toxicity. Seizures occurred and were associated with atrial flutter and ventricular asystole. Transvenous pacing at a rate of 75 beats/min controlled the arrhythmia, but bradycardia with a ventricular rate of 28 beats/min occurred when the catheter-electrode was inadvertently transected while the dressings were being changed. Five seizures occurred prior to the insertion of a new catheter-electrode. A Cardack-Greatbatch permanent transvenous pacemaker was subsequently implanted and the arrhythmia was adequately controlled.

In another patient, a complex arrhythmia developed shortly after aortic valve replacement with a Starr-Edwards prosthesis. The atrial mechanism was a combined flutter-fibrillation pattern. Some regular sequences of ventricular beats occurred at rates of 30/min, but there were also sequences of tachycardia which were slowed by vagal stimulation (abolished by carotid sinus pressure). In spite of transvenous pacing at rates up to 130 beats/min, ventricular capture was not accomplished and sequences of tachycardia still occurred. When the pacemaker was turned off, ventricular asystole occurred in 5 to 15 seconds. After diphenylhydantoin therapy was instituted, the ventricular beats began to follow the pacemaker impulses. Pacing was discontinued a few days later and the patient was dismissed, the atrial fibrillation being well-controlled by digitalis and diphenylhydantoin.

Paroxysmal ventricular tachyarrhythmias were associated with cardiogenic syncope in four patients. A 14-year-old boy with a non-obstructive cardiomyopathy had a paroxysmal ventricular tachycardia which could be controlled only by quinidine therapy combined with catheter-electrode pacing at rates exceeding 130 beats/min. This was unsatisfactory for long-term pacing, and therapy with quinidine and diphenylhydantoin was continued in the hope of affecting some degree of control. The patient was dismissed without a permanent cardiac pacemaker. The second patient had paroxysmal ventricular tachycardia and fibrillation which could not be controlled with drug therapy but which did not recur during catheter-electrode pacing at a rate of 100 beats/min. A permanent pacemaker was then implanted and the patient has been free of ventricular tachyarrhythmias for approximately 28 months. (This patient was previously described in detail.) Intermittent episodes of ventricular tachycardia and fibrillation developed in a third patient after an acute myocardial infarction. Catheter-electrode pacing was instituted and the arrhythmia did not recur, but the patient subsequently died of shock secondary to his infarction. The fourth patient presented several unusual features which are briefly outlined in the following case report.

Report of Case

A 52-year-old man, with a history of diabetes mellitus and chronic rheumatoid spondylitis, was seen in the emergency room in October 1965, 1 hour after he had experienced chest pain, tachycardia, and syncope. A first-degree heart block (P-R interval, 0.30 sec) had been noted in 1963. During the 3 months prior to admission he had experienced six to eight brief episodes of tachycardia.

The electrocardiogram at admission (fig. 1) revealed varying degrees of heart block (first-degree block with a P-R interval of 0.44 sec and second-degree block with a 3:2 ventricular response). Six hours after admission, ventricular tachycardia developed with a rate of 215 beats/min (fig. 2). A single 100-watt-second direct-current shock converted the rhythm to a second-degree A-V block. Because of some concern that complete heart block may have preceded the episode, a transvenous catheter-electrode pacemaker was inserted into the right ventricular cavity via the right external jugular vein, but internal pacing was not initiated immediately. The ventricular
tachycardia recurred within 1 hour and again reverted to a second-degree A-V block after direct-current shock. At this time, the catheter-electrode pacemaker was turned on, and ventricular capture occurred with the pacemaker rate set at 100 beats/min. In spite of technically satisfactory pacing, however, another episode of ventricular tachycardia occurred 9 hours later; this responded to 50 mg of procainamide given intravenously. Oral quinidine therapy was then started (200 mg every 4 hours) and catheter-electrode pacing was continued. When the pacemaker was turned off, a second-degree varying A-V block was present, and ventricular extrasystoles were noted, similar in contour to those present at the time of the episodes of ventricular tachycardia.

At various times throughout the next 4 weeks a ventricular parasystolic focus and ventricular tachycardia recurred whenever the quinidine therapy or the pacing or both were discontinued. No enzymatic or electrocardiographic evidence of myocardial infarction was noted throughout our period of observation.

Six weeks after admission, a permanent internal cardiac pacemaker (Medtronic) was inserted by Dr. J. W. Kirklin, with the rate set at 100 beats/min. During the postoperative period (36 hours) ventricular tachycardia recurred (fig. 3) and responded to intravenous injection of procainamide. It was thought that poor gastrointestinal absorption of the quinidine after operation was probably responsible for recurrence of the

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**Figure 1**

Electrocardiogram on admission, showing various degrees of heart block.
Figure 2

Electrocardiogram 6 hours after hospital admission, showing ventricular tachycardia (ventricular rate, 215 beats/min).

arrhythmia, and an intramuscular preparation was given until the patient was eating well. One month after operation tachycardia of brief duration developed when administration of the quinidine was discontinued. The patient has taken quinidine (200 mg every 6 hours) regularly since then, and no further episodes of arrhythmia have occurred in a 23-month follow-up period.

Comment on Case. It is possible that rheumatoid involvement of the conduction system was responsible for the atrioventricular conduction disturbance in this patient. Cardiac pacing with the transvenous catheter-electrode permitted the use of cardiodepressant drugs to control a recurrent paroxysmal ventricular tachycardia in the presence of heart block. The optimal rate (100 beats/min) for controlling the arrhythmia was determined prior to insertion of the permanent pacemaker. Because of the unusual features in this case and some concern about the association of a recent myocardial infarction, a relatively long period (43 days) of transvenous pacing was necessary.

Without Cardiogenic Syncope

Two patients required intracardiac pacing because of cardiac arrhythmias which, although not associated with syncopal attacks, were severely incapacitating. The first patient, with a 2:1 heart block and bradycardia (30 to 35 beats/min), was given a 15-day trial of

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intracardiac pacing because of symptoms suggesting cerebral ischemia (confabulation, disorientation, and confusion). In spite of technically successful pacing for 15 days, no clinical improvement was noted, and the patient was subsequently dismissed without a permanent internal pacemaker (prior to dismissal, a pneumoencephalogram was interpreted as showing diffuse cortical atrophy). Several months later it was learned that this patient had a permanent pacemaker inserted at another institution. Within a few weeks, the patient's mental status improved to the extent that he could return to a busy law practice. This result was quite unexpected and unexplained in view of the unsuccessful trial of seemingly adequate transvenous pacing at our institution.

The second patient, who had a history of multiple myocardial infarctions, was advised to have surgery for bronchogenic carcinoma. Prior to the operation, however, severe angina decubitus and multiple episodes of paroxysmal ventricular tachycardia occurred and could not be controlled with drug therapy. Only moderate control was achieved by combining drug therapy with transvenous catheter-electrode pacing at various rates. Consequently, the operation was cancelled and x-ray therapy was administered for the carcinoma.

Complications with Previously Implanted Permanent Pacemakers

Twenty-seven patients were paced by means of catheter-electrodes on 36 separate occasions because of failure of permanent pacemaker units previously implanted for a variety of indications (table 4). Five patients were paced on two separate occasions and two patients were paced three different times. One patient died before a new pacemaker could be implanted. The remaining patients had transvenous catheter-electrode pacemakers placed prior to successful surgical repair or replacement of the defective pacemaker units.

Other Surgical Procedures

Six patients had protective transvenous cardiac pacing during various general surgical

<table>
<thead>
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<td>Indications for Temporary Transvenous Pacing in Patients with Difficulties with Previously Implanted Permanent Pacemakers</td>
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<tr>
<th>Original indication for pacemaker implantation</th>
<th>Pt</th>
<th>Instances</th>
<th>PPM*</th>
<th>Deaths</th>
</tr>
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<tr>
<td>Complete heart block</td>
<td>20</td>
<td>27</td>
<td>27</td>
<td>0</td>
</tr>
<tr>
<td>With Adams-Stokes syndrome</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Without Adams-Stokes syndrome</td>
<td>4</td>
<td>5</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Second-degree A-V block</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>27</td>
<td>36</td>
<td>35</td>
<td>1</td>
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</table>

*Permanent pacemaker repaired or replaced.

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procedures, including vaginal hysterectomy and repair of a cystocele, laryngectomy for carcinoma of the larynx, abdominoperineal resection of a carcinoma of the rectum, cholecystectomy and replacement of a power pack of a previously implanted pacemaker, aortography, and closure of a colonic stoma. Five of these patients had complete heart block and one patient had 2:1 A-V block with angina pectoris. All of the surgical procedures were carried out without incident.

**Problems and Complications**

In 72 (65%) of the 110 instances of catheter-electrode placement in this series, there were no complications. In the remaining 38, there were 47 complications associated with the use of the catheter-electrode. Minor difficulties were related primarily to placement of the catheter-electrode or equipment failure. Ten major complications occurred during catheter-electrode pacing in nine patients.

**Minor Problems**

Placement difficulties were encountered in 28 (25%) of the 110 instances of pacing. In 15 instances, intermittent failure to pace was recognized clinically and normal pacing usually resumed after appropriate changes, at the bedside, in the position of the catheter-electrode tip. However, in 11 instances, catheter manipulation under fluoroscopic guidance was required. In two instances, continuing positional difficulties necessitated insertion of a new catheter-electrode through a different vein. During the replacement of the catheter-electrode in one patient, the electrode entered what appeared to be the left ventricle, presumably through a patent foramen ovale, but pacing was initiated and continued without difficulty. Since we have abandoned routine use of the antecubital vein, intermittency of pacing because of malposition of the catheter-electrode has been much less frequent.

Equipment failure occurred nine different times and included broken electrodes and lead wires in addition to battery failures. In each instance, the cause of the pacemaker failure was quickly determined and corrected with- out serious consequence to the patient’s clinical course.

**Major Complications**

Perforation of the heart was documented in four patients and suspected in a fifth.

In the four documented instances, the patients were paced prior to (1 day to 3 weeks) and during operation for implantation of permanent internal pacemakers, and the perforations were found unexpectedly at operation. Preoperatively, one patient was noted to have intermittent failure to pace, which was corrected by changes, at the bedside, in the position of the catheter-electrode tip; the other three patients were paced without difficulty. The perforation occurred through the right ventricular outflow tract in two patients and through the right atrioventricular groove in another; there was no evidence of hemopericardium in any of these three patients. Several milliliters of dark blood was observed at operation in the pericardial sac of the fourth patient, but no perforation could be found; although the preoperative placement of the catheter-electrode had been uncomplicated, a small perforation must have occurred.

In the fifth patient, temporary catheter-electrode pacing was instituted because of failure of a permanent cardiac pacemaker. Three days later, intermittent failure to pace was noted. A friction rub developed on the seventh day, and the patient died 9 days after the transvenous pacemaker had been inserted. Perforation of the heart was strongly suspected, but permission for a postmortem examination was denied.

Serious infection, an *Aerobacter* bacteremia directly attributable to the presence of an indwelling catheter-electrode, occurred in one patient on the day after placement of the catheter-electrode. This patient also had an acute myocardial infarction shortly after the placement. In spite of therapeutic attempts the patient died 7 days later after a complicated hospital course.

Phlebitis of the cephalic vein complicated the clinical course for another patient. Although there was no clinical evidence of venous thrombosis, a later attempt to insert
a Chardack-Greatbatch permanent transvenous pacemaker was unsuccessful because of thrombosis of the internal jugular and subclavian veins. A permanent internal cardiac pacemaker was subsequently inserted without incident.

In one patient, ventricular fibrillation seemed to be precipitated when the power pack was turned on after placement of the catheter-electrode. (The arrhythmia was observed on a monitoring oscilloscope, but a permanent record was not obtained.) In a second patient, ventricular fibrillation had been present prior to insertion of the catheter-electrode, and two episodes of ventricular fibrillation occurred during the pacing. The recurrent arrhythmia was finally controlled by a combination of cardiac pacing and intravenous injection of procainamide. A definite causal relationship between the pacemaker impulse and the onset of ventricular fibrillation could not be established in either of these patients.

In summary, two patients died as a direct result of major complications associated with transvenous catheter-electrode pacing.

Discussion

The treatment of patients with complete heart block and Adams-Stokes syndrome by means of permanently implanted cardiac pacemakers has been well established. Our practice has been to initiate temporary transvenous pacing prior to operation in all of these patients and to continue temporary pacing until the permanent pacemaker is safely implanted and functioning properly. The clinical response to a trial of temporary transvenous cardiac pacing with various heart rates and catheter positions (atrium or ventricle) can furnish valuable information for the decision as to whether or not a patient with a complex arrhythmia may benefit from implantation of a properly selected internal pacemaker.

Acute myocardial infarction complicated by complete heart block has been associated with a poor prognosis in the past, with mortality rates generally quoted between 40 and 50%. It had been hoped that transvenous catheter-electrode pacing in selected patients would decrease the mortality. Bruce and co-workers\(^8\) reported successful pacing in eight of nine patients who had complete heart block during acute myocardial infarctions, but data from other reports are less impressive.\(^9\)\(^-\)\(^11\) Our series includes five patients who experienced complete heart block during acute myocardial infarctions. Two of these patients died; three patients were successfully paced until spontaneous reversion to normal sinus rhythm occurred in 3, 6, and 7 days, respectively. Further experience will be necessary to determine the usefulness of transvenous cardiac pacing in patients with complete heart block complicating acute myocardial infarction, but it does appear to be of benefit in some cases.

Perforation of the heart has been emphasized as a complication of transvenous cardiac pacing. Furman and associates\(^12\) first described an instance of ventricular perforation which occurred during transvenous pacing and resulted in the death of the patient. Subsequent reports have indicated that, in spite of the potentially serious hazards of perforation, it is rarely associated with any serious clinical difficulties. In the presence of a perforation, ventricular pacing may be uninterrupted, intermittent, or absent, and increased current may be necessary for effective depolarization of the myocardium. In our series, one patient died of complications secondary to a presumed perforation of the heart by the catheter-electrode (permission for autopsy not granted); four patients sustained documented perforations without any obvious difficulties.

Ventricular tachyarrhythmias may occur during intracardiac pacing with the transvenous catheter-electrode, and it may be difficult to determine whether or not the pacemaker stimulus itself was responsible for the development of the tachyarrhythmia. In addition, electrocution hazards\(^13\) have been emphasized, and, rarely, ventricular arrhythmias have been precipitated by use of improperly grounded monitoring or electrocardiographic equipment during catheter-electrode pacing.\(^14\) In our series, two patients...
had ventricular fibrillation shortly after activation of the power pack following placement of the transvenous catheter-electrode. Although Race and associates have demonstrated in animal experiments that the ventricular fibrillation threshold is usually more than 10 times the strength of a stimulus needed to pace the heart, this relationship may be altered in the diseased human myocardium. Consequently, it seems advisable to maintain transvenous pacing with as small a stimulus as possible—approximately 2 ma above the threshold level. We would reemphasize from our experience, that all patients with temporary transvenous catheter-electrode pacemakers should be continuously monitored because of the potential hazard of associated, serious cardiac arrhythmias.

Fortunately, although infection and sepsis related to the use of an indwelling catheter-electrode can be a serious complication, the incidence has remained low in most series. Adherence to careful aseptic techniques, both during initial catheter placement and at the time of necessary bedside adjustments, is also to be emphasized.

References
Temporary Transvenous Catheter-Electrode Pacing of the Heart
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