Right Atrial Stimulation

in
The Treatment of Supraventricular Tachyarrhythmias

SYNOPSIS
Pervenous electrical stimulation of the right atrium was successful in converting 15 episodes of supraventricular tachyarrhythmia in 9 of 15 patients. This included 13 conversions of atrial flutter, one each of atrial tachycardia and junctional tachycardia. The procedure was performed at the bedside, without complication, using intracardiac electography to direct catheter position. Indirect evidence from this series supports all three proposed mechanisms for the conversion of atrial flutter by atrial stimulation: 1) interruption of a re-entry circuit, 2) introduction of an impulse in the atrial vulnerable period creating atrial fibrillation, and 3) overdrive suppression of a rapid firing ectopic focus. The method has certain advantages over DC cardioversion: 1) there is less potential for serious post-conversion arrhythmias, 2) serum enzyme profiles are not altered, 3) concomitant digitalis therapy is possible, and 4) continuous intracardiac electrographic monitoring is available with the potential for repeated conversions.

METHODS AND MATERIALS
Conversion of supraventricular arrhythmias by atrial stimulation was attempted in fifteen patients. Thirteen patients had atrial flutter, one patient had AV junctional tachycardia and one patient had paroxysmal atrial tachycardia with variable AV block. In every patient the arrhythmia was thought to be of less than 72 hours duration.

A flow directed platinum tipped catheter was passed to the high right atrium via a subclavian or arm vein. The catheter was positioned using intracardiac electography without fluoroscopy. A portable chest film was obtained to guarantee that there was no looping of the wire in the right atrium.
TABLE 1
Characteristics of High Frequency Pacemaker

<table>
<thead>
<tr>
<th>Constant current squarewave</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allowable load: 0 to 1,000 ohms resistive or complex impedance</td>
</tr>
<tr>
<td>Output current: adjustable from 0 to 20 milliamps</td>
</tr>
<tr>
<td>Pulse width: 1 millisecond</td>
</tr>
<tr>
<td>Pulse rate: adjustable between 60 and 600 pulses per minute</td>
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</tbody>
</table>

The high frequency pacemaker was designed and constructed by Mr. William Temple of the Veterans Administration Hospital, Portland, Oregon.

The procedure was carried out at the bedside and precautions were taken to protect the patient from another electrical source that could inadvertently cause ventricular fibrillation. A DC cardioverter and resuscitation cart were immediately available. The pacing wire was usually left in place following conversion for monitoring up to 72 hours. All the patients had received therapeutic doses of digitalis.

Initial stimulation was performed with a conventional pacemaker** and in later cases with a specially designed high frequency pacemaker (Table 1). In all instances stimulation was begun at a slow rate, 50-60 impulses/min and low amperage (2mA), while observing the surface Lead II for ventricular capture. The rate was advanced slowly with constant monitoring for changes in rhythm. Subsequently the amperage was increased to 15-20 mA.

The average length of time for the procedure was thirty minutes, with a range of fifteen minutes to one and a half hours. In most instances existing central venous pressure catheters in the subclavian vein served as immediate avenues for the placement of the stimulating wire, however antecubital basilic veins and external jugular veins were also satisfactory in some cases. Technical problems arose in two patients in whom delays of about thirty minutes were experienced in obtaining a suitable venous route. There was one forty minute delay when difficulty in positioning the catheter in the right atrium occurred.

**Medtronic Demand Pacemaker 5840
RESULTS

In a total of 15 patients there were 15 successful conversions in 9 patients. Table 2 lists the pertinent data of the successful conversions. In five patients atrial flutter was converted directly to sinus rhythm. Figure 1 is representative of two of these patients (cases 1 and 2) in whom the stimulating rate was slow, around 80/min. Prior to atrial stimulation in case 2, DC cardioversion at 50 watts seconds had produced atrial fibrillation, which spontaneously reverted back to atrial flutter. Slow atrial stimulation was successful in extinguishing the flutter and establishing sinus rhythm. Rapid atrial stimulation at a rate of 600/min successfully converted the other three patients to sinus rhythm (cases 4, 7 and 9).

One episode of atrial flutter was converted to transient atrial fibrillation (case 3). Figure 2 demonstrates slow right atrial stimulation, which changes the rhythm to atrial fibrillation followed by spontaneous conversion to sinus rhythm in 15 minutes.

One patient, with mitral stenosis and an enlarged left atrium (case 6), was converted from atrial flutter to atrial fibrillation three times with rapid atrial stimulation (fig. 3). Each episode of atrial flutter was associated with a rapid ventricular rate which was difficult to control with digitalis. Rapid atrial stimulation, faster than the flutter rate (around 400/min), produced atrial capture.

If the pacemaker was slowed or turned off at this point, atrial flutter returned. When the rate was increased to 600, atrial fibrillation developed with slowing of the ventricular rate. The patient was maintained in atrial fibrillation after adjustment of antiarrhythmia drugs.

AV junctional tachycardia was diagnosed by right atrial electrography as seen in Figure 4 (case 5). Slow right atrial stimulation converted this rhythm to regular sinus rhythm after a brief episode of packer-induced AV dissociation.

One patient had several supraventricular tachyarrhythmias (case 8). He initially had paroxysmal atrial tachycardia with varying first and second degree AV block, as shown in Figure 5. The initial impression was that the arrhythmia was digitalis-induced. Rapid atrial stimulation initially converted the PAT to atrial fibrillation with slowing of the ventricular rate. Additional digitalis was given and the rhythm changed to atrial flutter. Further atrial stimulation converted the atrial flutter to atrial fibrillation four times, and digitalis was given to slow the ventricular rate to around 100. Sinus rhythm ensued two hours later.

In 6 patients, all of whom had atrial flutter, right atrial stimulation was unsuccessful in changing the rhythm. No difference in diagnosis, medication or technique distinguished these patients from those who had successful conversions. In the successful conversions, the rhythm was usually altered within the first two to three minutes of stimulation and there were no instances in which prolonged stimulation altered the rhythm. All of the conversions occurred at less than 5 mA.

Figure 2: Lead II (panel A) and the right atrial electrogram (panel B) demonstrate atrial flutter with 2:1 AV block. Right atrial stimulation (panel C) converts this to atrial fibrillation (panel D). Sinus rhythm occurred after 15 minutes and 0.2 mg ouabain.

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<table>
<thead>
<tr>
<th>LEAD II</th>
<th>RIGHT ATRIUM</th>
<th>R.A. STIMULATION</th>
<th>RIGHT ATRIUM</th>
<th>RIGHT ATRIUM</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
</tbody>
</table>

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DISCUSSION

The treatment of atrial flutter has traditionally centered around drug therapy and direct current cardioversion. Cardioversion, however, is not without significant complications and side effects, especially in the digitalized patient. In treating atrial flutter with digitalis, high doses are often required to slow the ventricular rate. While propranolol may be beneficial in slowing the ventricular rate, the myocardial depressant effects of the drug may be undesirable.

Atrial stimulation offers several advantages over direct current cardioversion for the treatment of supraventricular tachyarrhythmias. No anesthesia or sedation is necessary and discomfort is confined to venapuncture. There is less potential for serious post-conversion arrhythmias since the ventricular myocardium is not directly depolarized by the stimulus. Digitalis withdrawal is not necessary to conversion and no serious post-conversion arrhythmias have been encountered. Continuous monitoring is available if the wire is left in position and there is the potential for repeated conversion. There is no distortion of the serum enzyme profile in monitoring for acute myocardial infarction.

Disadvantages of atrial stimulation include technical problems associated with vein entry and catheter manipulation which occasionally may be time consuming. Percutaneous subclavian catheterization is not without some risk, however in many of the patients in our series a central venous catheter had previously been placed for monitoring of pressure. Atrial stimulation in the small number of series in the literature is less effective than direct current cardioversion. Our success rate is consistent with that reported in the literature.

Figure 3: Lead II (panel A) and right atrial electrogram show atrial flutter with 2:1 AV block. Panel C demonstrates rapid stimulation with atrial capture in the first portion and conversion to atrial fibrillation in the later portion. Both rapid stimulation and atrial fibrillation (panel C, D & E) demonstrate slowing of the ventricular rate.

A
LEAD II

B
RIGHT ATRIUM

C
R.A. STIMULATION

D
RIGHT ATRIUM

E
LEAD II
The greatest danger with this procedure is inadvertent pacing of the right ventricle, which could cause serious ventricular arrhythmias. Although fluoroscopic positioning of the wire would be ideal, critically ill patients may often experience unnecessary delay in preparation for fluoroscopy equipment and personnel. Accurate positioning of the catheter in the right atrium is provided by the intracardiac electrogram. A portable chest film is used to verify the catheter position. Stimulation is begun at a slow rate watching for inadvertent right ventricular capture.

Barold has described three possible mechanisms for the termination of atrial flutter by atrial stimulation.\(^3\) The first mechanism is that of interruption of a re-entry circuit. Durrer\(^{15}\) and Barold\(^{16}\) have reported various supraventricular tachyarrhythmias which can be initiated and terminated with a single impulse. Impulses delivered to the atrium at a rate less than its intrinsic rate may fall at an appropriate time to block a re-entry circuit, interrupt the arrhythmia and establish sinus rhythm. The conversion of atrial flutter directly to sinus rhythm in two cases in our series with slow atrial stimulation is compatible with this thesis.

The proposed mechanism is the introduction of a single impulse in the atrial vulnerable period, producing an unstable rhythm such as atrial fibrillation. In one patient of our series atrial fibrillation occurred with slow right atrial stimulation which subsequently converted to sinus rhythm, without further intervention.

The third mechanism is overdrive suppression of a single, rapid-firing ectopic focus. It would seem unlikely that slow atrial stimulation could suppress or change a rapid-firing ectopic focus, however impulses at a rate higher than the intrinsic atrial rate could overtake and suppress a rapid-firing focus. In one patient in our series (case 6), atrial capture was evident at rates faster than the intrinsic atrial rate, however if the stimulation was stopped or slowed the flutter returned.

*Figure 4:* Initial ECG and the high superior vena cava electrogram (panel A) show a regular supraventricular rhythm. In the right atrial electrogram (panel B), the atrial spike is seen buried in the QRS, typical of AV junctional tachycardia. Slow right atrial stimulation (panel C) converted this arrhythmia to sinus rhythm (panel E) after a brief episode of pacemaker induced AV dissociation.
Figure 5: Initial ECG, Lead II (panel A) shows the regular tachyarrhythmia in a patient taking 0.25 mg digoxin per day. The right atrial electrogram (panel B) shows atrial tachycardia with first and second degree AV block after 0.2 mg ouabain. Right atrial stimulation was carried out as digitalis excess was considered to be the etiology of the arrhythmia. However, following conversion to atrial fibrillation (panel C) the ventricular rate was rapid. The patient spontaneously converted to atrial flutter (panel D) and rapid stimulation changed the rhythm to atrial fibrillation four times. Digitalis was given to slow the ventricular rate to 100. Sinus rhythm occurred in two hours.

immediately. This is consistent with a single rapid-firing focus which could be suppressed temporarily during faster stimulation.

Rapid atrial stimulation may offer an advantage over conventional slow stimulation by increasing the numerical possibility of a single impulse interrupting a re-entry circuit or falling in the atrial vulnerable period. In two patients in our series rapid atrial stimulation produced atrial fibrillation and in one of these patients, it was observed to be definitely dependent on the high rate stimulation (case 6).

In the three patients in whom atrial fibrillation was induced by atrial stimulation, two converted to sinus rhythm without further intervention and in all cases the ventricular rate was reduced during atrial fibrillation.

The slowing of the ventricular rate with onset of atrial fibrillation or during rapid atrial stimulation due to concealed conduction in the AV conducting system. The refractory period of the AV nodal tissue is prolonged due to the increased number of impulses reaching it. By increasing the refractory period, fewer impulses are completely transmitted to the ventricles.

Gosslein found more consistent success using alternating current to convert supraventricular tachyarrhythmias to atrial fibrillation. A method of placing an appropriately timed impulse to the atrium, as described by Haft, may produce a higher percentage of conversion to atrial fibrillation. As demonstrated in our series and others, atrial fibrillation is often an unstable rhythm and will often spontaneously convert to sinus rhythm.

Atrial stimulation in the conversion of supraventricular tachyarrhythmias is a new method that needs further clinical trials and elaboration to judge its effectiveness. It offers an alternative method to the treatment of supraventricular tachyarrhythmias and has certain advantages to the traditional avenues of treatment.
TABLE 2 Patients with Successful Conversions

<table>
<thead>
<tr>
<th>Case</th>
<th>Age/Sex</th>
<th>Diagnoses</th>
<th>Initial Arrhythmia</th>
<th>Final Rhythm</th>
<th>Stimulation Rate (ppm)/mA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>77 M</td>
<td>Ischemic heart disease, pericarditis</td>
<td>Atrial flutter</td>
<td>sinus</td>
<td>85/3</td>
</tr>
<tr>
<td>2</td>
<td>23 M</td>
<td>Mitral stenosis</td>
<td>Atrial flutter</td>
<td>sinus</td>
<td>80/2</td>
</tr>
<tr>
<td>3</td>
<td>66 M</td>
<td>Septic shock, adrenal insufficiency</td>
<td>Atrial flutter</td>
<td>sinus</td>
<td>130/2</td>
</tr>
<tr>
<td>4</td>
<td>62 M</td>
<td>Acute myocardial infarction, shock</td>
<td>Atrial flutter</td>
<td>sinus</td>
<td>600/2</td>
</tr>
<tr>
<td>5</td>
<td>55 M</td>
<td>Acute myocardial infarction with congestive heart failure</td>
<td>AV junctional tachycardia</td>
<td>sinus</td>
<td>95/2</td>
</tr>
<tr>
<td>6</td>
<td>49 M</td>
<td>Mitral stenosis</td>
<td>Atrial flutter</td>
<td>atrial fibrillation (3 times)</td>
<td>600/2</td>
</tr>
<tr>
<td>7</td>
<td>70 F</td>
<td>Post-op abdominal aortic aneurysm, ischemic heart disease</td>
<td>Atrial flutter</td>
<td>sinus</td>
<td>600/4</td>
</tr>
<tr>
<td>8</td>
<td>25 M</td>
<td>Aortic insufficiency</td>
<td>PAT with block &amp; Atrial flutter</td>
<td>sinus</td>
<td>600/2</td>
</tr>
<tr>
<td>9</td>
<td>70 M</td>
<td>Chronic obstructive pulmonary disease, pneumonia, ischemic heart disease</td>
<td>Atrial flutter</td>
<td>sinus</td>
<td>600/4</td>
</tr>
</tbody>
</table>

REFERENCES