Treatment of Supraventricular Tachycardias by Rapid Atrial Stimulation

By John W. Lister, M.D., Lawrence S. Cohen, M.D., William H. Bernstein, M.D., and Philip Samet, M.D.

SUMMARY
In 10 patients, 24 episodes of supraventricular tachycardia were treated by rapid electrical stimulation of the right atrium. One episode of sinus tachycardia, eight episodes of atrial tachycardia, two episodes of atrial flutter, and 13 episodes of A-V junctional tachycardia occurred. In each case the diagnosis of the arrhythmia was documented by obtaining unipolar and bipolar intra-atrial electrograms.

In three cases of supraventricular tachycardia the ventricular rate was slowed as a result of an increased atrial rate. The increased atrial rate caused an increase of the functional refractory period of the A-V junction, and thus, fewer atrial impulses were transmitted to the ventricles.

In seven cases, 21 episodes of supraventricular tachycardia were terminated by rapid atrial stimulation. In six of these cases the tachycardia was converted to normal sinus rhythm either during or shortly after atrial stimulation. In one case, 12 episodes of A-V junctional tachycardia were converted to rhythms varying between normal sinus rhythm and a slow A-V junctional rhythm. In this case, after termination of the last episode of A-V junctional tachycardia, the rhythm was stabilized by atrial pacing.

Additional Indexing Words:
Atrial fibrillation Atrial flutter Intra-atrial electrograms

Supraventricular tachycardia is a common complication of heart disease. Cardioversion by electric countershock has often simplified the immediate treatment of supraventricular tachycardias.1-5 In patients receiving digitalis, however, cardioversion may be hazardous,6-9 and in supraventricular tachycardia that recurs at short intervals electric countershock is only a temporary measure.

The purpose of this report is to present 10 cases of supraventricular tachycardia in which rapid atrial stimulation was employed to either slow the ventricular rate or terminate the arrhythmia.

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Methods
The effect of rapid electrical stimulation of the right atrium was studied in 10 patients with supraventricular tachycardias. All patients had documented pre-existing heart disease, and their ages ranged from 62 to 83 years. Eight of the 10 patients were receiving maintenance doses of digitalis. In the other two cases, administration of digitalis had been discontinued 4 days prior to study. The pertinent data related to the patients studied are presented in table 1.

Under local anesthesia, a bipolar electrode catheter with electrodes 1 cm apart was placed in the right atrium. The catheter was positioned transvenously via a femoral vein percutaneous puncture or a surgically exposed antecubital vein. The catheters were positioned under fluoroscopic or electrographic control or both.10 Electrographic control alone was only used in the cases in which the patient could not be moved, and the portable image amplifier was not available. In each case, prior to and subsequent to atrial stimulation, the rhythm was analyzed by the simultaneous or sequential recording of a right intra-atrial unipolar and bipolar electrogram obtained from the electrode catheter and a
Table 1

Pertinent Data Concerning Patients Studied

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yr)</th>
<th>Type of heart disease</th>
<th>Functional classification*</th>
<th>Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>76</td>
<td>ASHD</td>
<td>IV-D</td>
<td>Sinus tachycardia</td>
</tr>
<tr>
<td>2</td>
<td>62</td>
<td>RHD</td>
<td>II-C</td>
<td>Atrial tachycardia</td>
</tr>
<tr>
<td>3</td>
<td>75</td>
<td>ASHD</td>
<td>4 days after MI</td>
<td>Atrial tachycardia</td>
</tr>
<tr>
<td>4</td>
<td>83</td>
<td>ASHD</td>
<td>II-C</td>
<td>Atrial tachycardia</td>
</tr>
<tr>
<td>5</td>
<td>77</td>
<td>ASHD</td>
<td>I-B</td>
<td>Atrial tachycardia</td>
</tr>
<tr>
<td>6</td>
<td>70</td>
<td>ASHD</td>
<td>I-B</td>
<td>Atrial flutter</td>
</tr>
<tr>
<td>7</td>
<td>63</td>
<td>ASHD</td>
<td>I-B</td>
<td>A-V junctional tachycardia</td>
</tr>
<tr>
<td>8</td>
<td>83</td>
<td>ASHD</td>
<td>I-B</td>
<td>Atrial tachycardia</td>
</tr>
<tr>
<td>9</td>
<td>72</td>
<td>ASHD</td>
<td>III-C</td>
<td>Atrial tachycardia</td>
</tr>
<tr>
<td>10</td>
<td>75</td>
<td>RHD</td>
<td>III-C</td>
<td>A-V junctional tachycardia</td>
</tr>
</tbody>
</table>

*Functional classification of the New York Heart Association.
†Cordis Corporation, Miami, Florida.
Abbreviations: ASHD = arteriosclerotic heart disease; RHD = rheumatic heart disease; MI = myocardial infarction.

standard lead electrocardiogram. After the rhythm had been delineated by the recording of the electrograms, the electrode catheter was connected to a Medtronic "R" wave coupled-pulse generator, (Model 5837*), and the atria were paced by single pulse stimulation or by paired pulse stimulation. The modes and exact rates of atrial stimulation are presented in table 2. Continuous atrial stimulation was maintained for 3 sec to 15 min. In one subject the functional refractory period of the A-V junction was tested by the placement of premature stimuli into the atrium. This was done by triggering the pacemaker discharge from the previous QRS complex at various intervals (fig. 1).

Intra-atrial unipolar and bipolar electrograms and standard lead electrocardiograms were simultaneously recorded by the connection of the catheter leads to an electrode distribution box which was connected to a multichannel oscilloscopic photographic recorder. The unipolar intra-atrial electrograms and standard lead electrocardiograms were recorded at filter settings of 0 to 20 cycles/sec. The bipolar intra-atrial electrograms were obtained at filter settings of 40 to 200 cycles/sec. When the intra-atrial electrograms and the standard lead electrocardiograms were obtained in sequence, a Sanborn electrocardiograph machine (Model 100) was used. The methods used in this study for the recording of atrial electrograms have been previously described in detail. Careful attention was paid to the proper grounding of all equipment.

Results

In 10 patients, 24 episodes of supraventricular tachycardia were treated by rapid electrical stimulation of the right atrium (table 2). One episode of sinus tachycardia, eight episodes of atrial tachycardia, two of atrial flutter, and 13 of A-V junctional tachycardia occurred. In each case the diagnosis of the arrhythmia was documented by obtaining unipolar and bipolar intra-atrial electrograms. Table 2 presents a detailed analysis of the arrhythmias, the mode of atrial stimulation, and the results of atrial stimulation.

In cases 1 to 3 the ventricular rate was decreased as a result of an increased atrial rate (figs. 1 to 3). In case 1, the patient was receiving a maintenance dose of digitalis; the rhythm was sinus tachycardia with occasional premature ventricular beats. The refractory period of the A-V junction was tested by the

*Medtronic, Inc., Minneapolis, Minnesota.
Table 2

Modes and Rates of Atrial Stimulation

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Medication</th>
<th>Control</th>
<th>Rate</th>
<th>After atrial stimulation</th>
<th>Rate</th>
<th>Comments</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>A-V conduction</td>
<td>Atrial</td>
<td>Ventricular</td>
<td>MOAS Rate</td>
<td>Int. (msec)</td>
</tr>
<tr>
<td>1</td>
<td>Digitalis</td>
<td>Sinus tachycardia</td>
<td>1:1</td>
<td>111</td>
<td>111</td>
<td>S.S.</td>
</tr>
<tr>
<td>2</td>
<td>Digitalis</td>
<td>Atrial tachycardia</td>
<td>1:1</td>
<td>118</td>
<td>118</td>
<td>P.S.</td>
</tr>
<tr>
<td>3</td>
<td>Digitalis</td>
<td>Atrial tachycardia</td>
<td>2:1</td>
<td>280</td>
<td>140</td>
<td>S.S.</td>
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<tr>
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<td>Digitalis</td>
<td>Atrial tachycardia</td>
<td>1:1</td>
<td>143</td>
<td>143</td>
<td>P.S.</td>
</tr>
<tr>
<td>5</td>
<td>Digitalis</td>
<td>Atrial tachycardia</td>
<td>1:1</td>
<td>166</td>
<td>166</td>
<td>S.S.</td>
</tr>
<tr>
<td>6</td>
<td>Digitalis</td>
<td>Atrial flutter</td>
<td>Variable</td>
<td>330</td>
<td>110</td>
<td>S.S.</td>
</tr>
<tr>
<td>7</td>
<td>Digitalis</td>
<td>A-V junctional tachycardia</td>
<td>1:1</td>
<td>158</td>
<td>158</td>
<td>S.S.</td>
</tr>
</tbody>
</table>
### TREATMENT OF SUPRAVENTRICULAR TACHYCARDIAS

Placement of premature atrial beats. The refractory period of the A-V junction was found to be short, less than 250 msec (fig. 1B). Atrial pacing at 166/min resulted in 1:1 A-V conduction (fig. 1C). In order to increase the refractory period of the A-V junction, lanatoside C 0.3 mg (Cedilanid) was administered intravenously. Twenty minutes after the administration of the lanatoside C, atrial pacing at a slightly greater rate (176/min) resulted in 2:1 A-V block and reduction of the ventricular rate from 111/min to 88/min (fig. 1D). In case 2, the ventricular rate was also slowed by increasing the atrial rate (fig. 2). In case 3, the ventricular rate was significantly decreased by conversion of atrial flutter to atrial fibrillation (figs. 3 and 4). In these instances, an increase in the number of impulses which arrived at the A-V junction resulted in a decrease of the ventricular rate.

In cases 1 and 2 the ventricular rate could probably have been better controlled if sustained atrial fibrillation could have been created. Repeated attempts to induce atrial fibrillation with paired pulse stimulation of the atria were unsuccessful. Up to 500 impulses/min were applied to the atria.

In cases 4 to 10, rapid atrial stimulation terminated the supraventricular tachycardia. In case 4, paired stimulation of the atria converted an atrial tachycardia to atrial fibrillation, and the ventricular rate was immediately slowed (fig. 5A to C). One-half hour subsequent to atrial stimulation the rhythm spontaneously converted to normal sinus rhythm (fig. 5D). In case 5, rapid atrial stimulation first converted an atrial tachycardia to atrial flutter, and then repeat atrial stimulation converted the atrial flutter to normal sinus rhythm (fig. 6).

In cases 6 and 7, rapid atrial stimulation converted an atrial flutter (fig. 7) and an A-V junctional tachycardia to normal sinus rhythm (fig. 8). In patient 8, during a 5-day period, four episodes of atrial tachycardia were terminated and converted to normal sinus rhythm by rapid atrial stimulation.

In case 6, after atrial stimulation, there were several A-V junctional beats prior to the

<table>
<thead>
<tr>
<th>Atrial tachycardia</th>
<th>Atrial tachycardia</th>
<th>A-V junctional tachycardia</th>
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</thead>
<tbody>
<tr>
<td>2:1</td>
<td>1:1</td>
<td>variable</td>
</tr>
<tr>
<td>Digitalis</td>
<td>Digitalis</td>
<td>Digitalis</td>
</tr>
<tr>
<td>330</td>
<td>329</td>
<td>200</td>
</tr>
<tr>
<td>S.S.</td>
<td>S.S.</td>
<td>S.S.</td>
</tr>
<tr>
<td>400</td>
<td>126</td>
<td>146</td>
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<tr>
<td>400</td>
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</table>

| Abbreviations: MOAS = mode of atrial stimulation; S.S. = single stimulation; P.S. = paired stimulation. Int. = interval between the pacemaker impulses of a pair of stimuli. N.S.R. = normal sinus rhythm; P.V.R. = paced ventricular rhythm; IV = intravenous. |
Case 1. Sinus tachycardia; ventricular rate slowed by lanatoside C administration and rapid atrial pacing.

(A) Simultaneous intra-atrial unipolar and bipolar electrograms, and lead II electrocardiogram. Sinus tachycardia of 111/min, with a P-R interval of 0.21 sec, is present. There is a single premature ventricular beat.

(B) Lead II electrocardiogram. (Four panels, I through IV.) In the first panel a pacemaker impulse 210 msec after a P wave fails to capture the atria. In panels II to IV pacemaker impulses 250, 270, and 300 msec after a P wave capture the atria and are conducted to the ventricles.

(C) Lead II electrocardiogram. Atrial pacing at 166/min results in 1:1 A-V conduction with progressive prolongation of the P-R interval and a ventricular rate of 166/min. The change in amplitude of the pacemaker impulses is caused by turning the pacemaker on and off.

(D) Lead II electrocardiogram. Twenty minutes after the IV administration of 0.3 mg of lanatoside C. Atrial pacing at 176/min results in 2:1 A-V block and a slowing of the ventricular rate to 88 beats/min.

Abbreviations in this and subsequent figures: AUE = intra-atrial unipolar electrogram; ABE = intra-atrial bipolar electrogram; L-2 = lead II electrocardiogram; PI = pacemaker impulse; arrows show initiation of atrial pacing; HR = heart rate; P-P = P-P interval in sec; paper speed, 50 mm/sec; time lines, 1 sec; P-R = P-R interval in seconds.
onset of normal sinus rhythm (fig. 7B and C). In cases 7 and 8, it was not possible to determine with certainty whether an intermediate rhythm occurred prior to conversion to normal sinus rhythm. Intermediate rhythms may have been concealed by the pacemaker stimuli.

Patient 9 had a Cordis Ectocor, ventricular demand pacemaker implanted 10 weeks prior to developing atrial tachycardia. The ventricular pacemaker was originally implanted because of sinus bradycardia. In this subject, rapid atrial stimulation broke the atrial tachycardia and resulted in a ventricular paced rhythm with retrograde ventriculo-atrial conduction (fig. 9). After atrial stimulation, the first three R-R intervals were irregular (fig. 9B). This probably represented transient atrial fibrillation.

In case 10, recurrent episodes of supraventricular tachycardia were repeatedly controlled by rapid atrial stimulation. Twelve
Discussion

For both man and the experimental animal it has been well documented that increasing the atrial rate by electrical pacing results in progressive prolongation of the P-R interval and is followed by increasing degrees of atrioventricular block. Lister and his associates have shown in patients with normal sinus rhythm and sinus tachycardia that paired and coupled atrial stimulation may be employed to slow the ventricular rate by producing 2 : 1 atrioventricular block.

The results of our study show that in cases of sinus and atrial tachycardia and atrial flutter, the ventricular rate can be significantly slowed by increasing the atrial rate or inducing atrial fibrillation by atrial stimulation. The ventricular rate in atrial tachycardia, atrial flutter, and atrial fibrillation is dependent upon (1) the number of impulses which
arrive at the atroventricular junction per minute, (2) the length of the functional refractory period of the atroventricular junction, and (3) how many of the atrial impulses partially penetrate the atroventricular junction per minute (repetitive concealed conduction). \(^{20-22}\) Thus, the ventricular rate may be slowed, in cases of sinus and atrial tachycardia, and atrial flutter and fibrillation by increasing the atrial rate or the functional refractory period of the atroventricular junction by the administration of pharmacological agents, or both.

Supraventricular tachycardia may be terminated by atrial stimulation, provided the atrial paced beats are not blocked above the site of origin of the tachycardia. Atrial stimulation resulting in conversion of supraventricular tachycardias to normal sinus rhythm has been reported by several investigators. These investigators have described the termination of atrial flutter by rapid atrial stimulation\(^{23}\) and the termination of reciprocal tachycardias by pairs of atrial stimuli.\(^{24, 25}\) In seven of the patients presented in this report, there were 21 episodes of ectopic supraventricular tachycardias terminated by atrial stimulation. In most instances, the atrial rate was significantly increased above the intrinsic atrial rate by atrial stimulation. However, in

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**Figure 4**

*Case 3. Records obtained prior to and after stimulation (see fig. 3). (A and B) Sequentially recorded lead II electrocardiogram and intra-atrial bipolar electrogram prior to atrial stimulation. Atrial flutter with 2:1 A-V block is present. The atrial rate is 280/min and the ventricular rate is 140/min. (C to E) Sequentially recorded aVF: intra-atrial unipolar and bipolar electrograms after atrial stimulation. Atrial fibrillation with an average ventricular rate of about 80/min. Altering the rhythm from atrial flutter to atrial fibrillation significantly slowed the ventricular rate.*

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**HR:** 140/min

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*Arrival at the atrioventricular junction per minute, (2) the length of the functional refractory period of the atrioventricular junction, and (3) how many of the atrial impulses partially penetrate the atrioventricular junction per minute (repetitive concealed conduction).\(^{20-22}\) Thus, the ventricular rate may be slowed, in cases of sinus and atrial tachycardia, and atrial flutter and fibrillation by increasing the atrial rate or the functional refractory period of the atrioventricular junction by the administration of pharmacological agents, or both.

Supraventricular tachycardia may be terminated by atrial stimulation, provided the atrial paced beats are not blocked above the site of origin of the tachycardia. Atrial stimulation resulting in conversion of supraventricular tachycardias to normal sinus rhythm has been reported by several investigators. These investigators have described the termination of atrial flutter by rapid atrial stimulation\(^{23}\) and the termination of reciprocal tachycardias by pairs of atrial stimuli.\(^{24, 25}\) In seven of the patients presented in this report, there were 21 episodes of ectopic supraventricular tachycardias terminated by atrial stimulation. In most instances, the atrial rate was significantly increased above the intrinsic atrial rate by atrial stimulation. However, in
two cases of junctional tachycardia, which were terminated by atrial stimulation, the pacemaker rate was slightly slower than the intrinsic rate of the tachycardia in one case, and in the other case, the pacemaker rate was identical to the intrinsic rate of the tachycardia.

The mechanisms probably responsible for atrial stimulation resulting in the termination of supraventricular and reciprocal tachycardias are: (1) overdrive and suppression of an ectopic supraventricular pacemaker focus;\textsuperscript{23, 26-28} (2) alteration in the supraventricular arrhythmia, that is, conversion of atrial flutter to atrial fibrillation, and the conditions present do not permit the new arrhythmia to be self-perpetuating; and (3) the interruption of a fixed circus circuit by atrial paced beats.

Atrial pacing to stabilize the rhythm was used in one case (case 10) after recurrent episodes of A-V junctional tachycardia had been terminated by rapid atrial stimulation. After suppression of the last episode of tachycardia, the rhythm varied between a slow A-V junctional rhythm and normal sinus rhythm. Atrial pacing at 115/min for 24 hr stabilized the rhythm while propranolol was administered orally. Ventricular and atrial pacing for prevention of recurrent ventricular
and supraventricular tachycardias has been previously reported. Silverman and his associates have implanted a permanent atrial pacemaker in a patient who had periods of sinus arrest. When atrioventricular conduction is intact, atrial pacing is a more physiological therapeutic approach to the treatment of bradycardias and possible prevention of tachycardias than ventricular pacing.

The intracardiac electrodes used for atrial stimulation are also used for the recording of intra-atrial electrograms. The intracardiac electrograms permit the exact determination of the atrial rate and the temporal relationship between atrial and ventricular activation. In cases of supraventricular tachycardia prior to rapid atrial stimulation, it is necessary to determine the atrial rate and the temporal relationship between atrial and ventricular activation. Initially, the pacemaker rate should be set at a rate slightly greater than the atrial rate. In cases of atrioventricular block and atrial fibrillation, rapid atrial stimulation will be of no avail. In atrial fibrillation the pacemaker stimuli are ineffective.

No complications thus far have been encountered in patients who were receiving digoxin and had supraventricular tachycardias which were slowed or terminated by rapid atrial stimulation. In patients receiving digitalis who have supraventricular tachycardias other than atrial fibrillation, treatment of the tachycardia by rapid atrial stimulation may, in time, prove to be safer than treatment of

Figure 6

Case 5. Conversion of an atrial tachycardia to an atrial flutter, and conversion of the atrial flutter to normal sinus rhythm. Paper speed 50 mm/sec; time lines, 1 sec.

(A, B, and D) Simultaneous AUE, ABE, and lead II. (A) Atrial tachycardia with 1:1 A-V conduction and a rate of 166 beats/min. (B) This record was obtained immediately after atrial stimulation at a rate of 200/min. The atrial stimulation converted the atrial tachycardia of 166/min to atrial flutter with 2:1 A-V block. The atrial rate is 300/min, and the ventricular rate is 150/min. (C) Lead II electrocardiogram. Paired stimulation of the atrium (PS) at 200/min. The interval between the stimuli of a pair of impulses is 130 msec. After atrial stimulation is terminated, normal sinus rhythm is present. (D) Normal sinus rhythm is present.

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Figure 7

Case 6. Conversion of atrial flutter to normal sinus rhythm. Paper speed, 50 mm/sec; time lines, 1 sec.
(A) Simultaneous AUE, ABE, and lead II. Atrial flutter with variable A-V conduction. The atrial rate is 330/min, and regular; the ventricular rate is 110/min and irregular.
(B and C) Continuous lead II electrocardiogram. The pacemaker rate is 375/min. Atrial stimulation reduces the ventricular rate from 110/min to 90/min. The first three beats after termination of atrial stimulation are A-V junctional beats. The first of these beats has no discernible P wave. The following two beats show retrograde atrial activation. The P-R interval of these two beats is short, 0.11 sec. After the three A-V junctional beats, the rhythm stabilizes into normal sinus rhythm. The pacemaker rate of discharge is continuously recorded. The pacemaker impulses (PI) change in contour when the pacemaker is turned on or off. The arrows on the lead II ECG show the initiation and termination of atrial stimulation.
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Case 7. Conversion of a junctional tachycardia to normal sinus rhythm. Paper speed, 50 mm/sec; time lines, 1 sec.

(A) Simultaneous AUE, ABE, and lead II. The tracings show junctional tachycardia of 158 beats/min. The P waves cannot be clearly defined on the intra-atrial unipolar electrogram and lead II electrocardiogram.

(B and C) Continuous lead II tracing. Atrial pacing at 158 impulses/min is instituted. Strip C shows the initial capture of the heart by the pacemaker. The P-R interval of 0.21 sec of the atrial paced beats indicates that the rhythm in strip A is a junctional tachycardia and not an atrial tachycardia with first degree heart block. At the end of strip B, the pacemaker was turned off for 0.68 sec, and no spontaneous beats occurred. Then, the pacemaker was turned on for three beats and again turned off. The first beat in strip C shows capture of the atrium by the pacemaker with a shortened P-R interval and aberrant ventricular activation. After the pacemaker is turned off there is immediate reversion to sinus rhythm.

(D) Simultaneous AUE, ABE, and lead II. The record shows normal sinus rhythm.

Figure 8
Case 9. Conversion of an atrial tachycardia to a ventricular paced rhythm with 1:1 ventriculo-atrial conduction. Patient has an implanted transvenous right ventricular Cordis Ectocor demand pacemaker. Paper speed 50 mm/sec; time lines, 1 sec.

(A) Simultaneous AUE, ABE, and lead II. The atrial rate is 252 beats/min; the ventricular rate is 126 beats/min and slightly irregular. The ventricular demand pacemaker is partially suppressed by the rapid ventricular rate and is firing at an irregular rate. There are eight ventricular paced beats (VPB) on this record. The spontaneous cycle length of the R-R interval is 0.48 sec and the P-P interval is 0.24 sec; 2:1 A-V block is present.

(B) Lead II electrocardiogram. Two short bursts of atrial stimulation at a rate of 252 impulses/min result in a transient prolongation of the R-R interval. The rhythm is not permanently altered by the atrial stimulation.

(C) Lead II electrocardiogram. A short burst of atrial stimulation of 400 impulses/min results in conversion of the atrial tachycardia to a regular ventricular paced rhythm. The R-R intervals of the first three beats after termination of the atrial stimulation are irregular.

(D) Simultaneous AUE, ABE, and lead II. The record shows a paced ventricular rhythm with 1:1 ventriculo-atrial conduction.

Figure 9
TREATMENT OF SUPRAVENTRICULAR TACHYCARDIAS

Figure 10

Case 10; 10:15 a.m. Conversion of a junctional tachycardia to a normal sinus rhythm. The arrows show when the pacemaker is turned on and off. Cycle length of pacemaker = 0.42 sec; pacemaker rate = 146/min.

(A) Intra-atrial electrogram. The ventricular rate is 150/min and regular. The P wave is buried in the QRS complex and deforms the QRS complex. The R' is caused by the P wave. (Compare the contour of the QRS complex to that of the QRS complex in F.)

(B to E) Continuous lead II electrocardiogram. The pacemaker rate is slightly slower than the intrinsic heart rate and the pacemaker impulses capture the heart only on occasion. Whenever the heart is captured by the pacemaker impulse, the heart rate is slowed (seventh beat in C and last beat in D). (E) After atrial stimulation was terminated, for four beats, the rhythm varied between a junctional and sinus rhythm and then stabilized in normal sinus rhythm.

(F) AUE. Normal sinus rhythm. Note, there is no longer an R' in the QRS complex.

the arrhythmia by cardioversion. In cases of recurrent episodes of supraventricular tachycardia other than atrial fibrillation recurring at short time intervals, rapid atrial stimulation rather than cardioversion appears to be the treatment of choice for the immediate termination of the arrhythmia or slowing of the ventricular rate. The right atrial electrode catheter is left in place and rapid atrial stimulation can be repetitively applied with no discomfort to the patient; anesthesia is not necessary. However, loss of atrial capture due
to catheter movement in the right atrium makes long-term atrial pacing through a transvenous catheter unreliable at this time.

Electrical stimulation of the atria has a wide range of clinical applications in the treatment of cardiac arrhythmias. The exact clinical indications for atrial stimulation remain to be more precisely defined.

References
10. Lister, J. W., Furman, S., Stein, E., Damato, A. N., Schwedel, J. B., and Escher, D. J. W.: Heart block: Method for the rapid determination of causes of pacing failure in

Figure 11
Case 10; 12:40 p.m. Slowing of the ventricular rate by an increase of the atrial rate by atrial pacing; conversion of a junctional tachycardia to a slower rhythm, and stabilization of the rhythm by atrial pacing. This record was obtained from the same patient as in figure 10. The arrows show when the pacemaker was turned on and off. Cycle length of pacemaker = 0.32 sec; pacemaker rate = 188/min.

(A) The first four beats show a recurrence of the junctional tachycardia of 150 beats/min. The atrium is paced at 188 beats/min. The fourth or fifth pacemaker impulse captures the atria. At this atrial rate, at first there is 3:2 A-V block and then 2:1 A-V block, resulting in a ventricular rate of 94/min. Pacing at this atrial rate was continuously maintained for 10 min.

(B and C) Continuous lead II electrocardiogram. Atrial pacing at 188 impulses/min, for 10 min has resulted in variable A-V block with variations in the P-R interval of the conducted beats. In B, after atrial pacing was terminated, a junctional rhythm of 75 beats/min was present. In C, several sinus beats occurred. In order to stabilize the rhythm, atrial pacing at 115 beats/min was instituted. The atrial pacing was maintained for 24 hr.


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